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AB 2588 Air Toxics Health Risk Assessment for Reporting Year 2009

University of California Campus
Davis, California

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University of California, Davis

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LIST OF ACRONYMS

AB	Assembly Bill
BPIP	Building Profile Input Program
CARB	California Air Resources Board
CATEF	California Toxic Emission Factor
CHCP	Central Heating and Cooling Plant
DPM	Diesel particulate matter
EI Guidelines	<i>Emission Inventory Criteria and Guidelines for the Air Toxics "Hot Spots" Program</i>
ERM	ERM-West, Inc.
GEP	Good engineering practice
g/hp-hr	Grams per horsepower-hour
g/s	Grams per second
HARP	Hotspots Analysis and Reporting Program
HRA	Health Risk Assessment
IC	Internal combustion
ISCST3	Industrial Source Complex Short-Term 3 (air dispersion model)
LFG	Landfill gas
LRDP	Long Range Development Plan
$\mu\text{g}/\text{m}^3$	Micrograms per cubic meter
$\mu\text{g}/\text{m}^2/\text{day}$	Micrograms per square meter per day
m/s	Meters per second
mg/kg-day	Milligrams of chemical per kilogram of body weight per day
MMBtu	Million British thermal units per hour
MEIR	Maximum exposed individual resident
MEIW	Maximum exposed individual worker
NMOC	Non-methane organic compound
OEHHA	California Office of Environmental Health Hazard Assessment
PMI	Point of maximum impact
REL	Reference exposure level (equivalent to RfC)
RfC	Reference air concentration
RfD	Reference dose
SF	Slope Factor
RfDi	Reference dose - inhalation exposure
RfDo	Reference dose - oral (ingestion) exposure

SCDS	South Campus Disposal Site
SFi	Inhalation exposure slope factor
SFo	Oral (ingestion) exposure slope factor
TAC	Toxic air contaminant
UC Davis	University of California at Davis
URF	Unit risk factor
USEPA	United States Environmental Protection Agency
UTM	Universal Transverse Mercator
YSAQMD	Yolo-Solano Air Quality Management District
ZOI	Zone of impact

EXECUTIVE SUMMARY

This report presents a Health Risk Assessment (HRA) Update to comply with the Air Toxics “Hot Spots” Information and Assessment Act of 1987 (Assembly Bill [AB] 2588 Hot Spots) for sources of toxic air contaminants (TACs) emitted from the University of California at Davis (UC Davis) campus. AB 2588 Hot Spots legislation established a statewide program for the inventory of TAC emissions from individual facilities. TACs are air pollutants for which ambient air quality standards have not been established, but are known or suspected to cause short-term (acute) and/or carcinogenic and long-term (chronic) non-carcinogenic adverse health effects.

The Yolo-Solano Air Quality Management District (YSAQMD) has authority for the AB 2588 Program in part of the Sacramento Air Basin, which includes the UC Davis campus. Staff at YSAQMD has communicated to UC Davis that they are required to prepare an AB 2588 HRA for reporting year 2009. New requirements under the California Air Resources Board (CARB) *Emission Inventory Criteria and Guidelines for the Air Toxics “Hot Spots” Program* (effective 26 September 2007) (EI Guidelines) (CARB 2007), require facilities operating diesel-fired internal combustion (IC) engines to prepare an updated AB 2588 HRA. This recent EI Guidelines document was developed in part to better align the AB 2588 program with CARB’s Stationary Diesel Air Toxics Control Measure (Title 17 of the California Code of Regulations, Section 93115), which was promulgated to regulate emissions from diesel-fired IC engines.

The methodologies used in the HRA followed those described in the *Assembly Bill 2588 Health Risk Assessment Protocol for Reporting Year 2009 for the University of California at Davis*, prepared by ERM-West, Inc., and approved by the YSAQMD. As recommended by staff at the California Air Resources Board (CARB) (CARB 2009), emissions from the facility were largely assumed to be the same as those from the 2005 reporting year, with the addition of diesel-fired IC engines and a new boiler at the Central Heating and Cooling Plant. A copy of the protocol is included in Appendix A. The health impacts examined in this HRA include cancer risks, as well as chronic and acute non-cancer health effects. Cancer risk is expressed in terms of the probability of contracting cancer at any exposure level, typically over a lifetime. The calculated **cancer risk** (expressed as the chances in one million) is always greater than zero when the exposure level is greater than zero. Non-cancer health effects (chronic and acute) are measured by the **hazard index**, the ratio of the reported concentration of an air toxic compound to an acceptable or “reference” exposure level (REL). For non-inhalation pathways, hazard indices are calculated as the ratio of calculated doses to acceptable or “reference” doses (RfDs). If the reported concentration or dose of a given chemical is less than its REL or RfD, then the hazard index will be less than 1.0.

Health effect calculations were performed at the off-site point of maximum impact (PMI), the location of a maximum exposed individual at an existing residential receptor (MEIR), and the location of a maximum exposed individual at an existing worker (occupational) receptor (MEIW). Health risks were also calculated at receptor locations representing on-campus housing, university staff, campus gathering locations, and other sensitive receptors (e.g., schools, daycare centers, hospitals, and nursing homes).

All potential health effects were calculated using the Tier 1 analysis from the Office of Environmental Human Health Assessment (OEHHHA) *Air Toxics Hot Spots Program Risk Assessment Guidelines: The Air Toxics Hot Spots Program Guidance Manual for Preparation of*

Health Risk Assessments (OEHHA 2003). The Tier 1 assessment assumes a continuous 70-year exposure for long-term health risks. The Tier 1 analysis, required to be conducted for AB 2588 HRAs, combines the 70-year exposure period with a standard point-estimate approach using the upper-bound exposure pathway parameters recommended in the OEHHA guidelines. As required by YSAQMD, the cancer burden is estimated by multiplying the number of people exposed by the individual cancer risk at the population centroid of each census block within the exposed population. For the purposes of this analysis, **chronic** non-cancer health effects are based on annual period of continuous exposure and **acute** non-cancer health effects are calculated for a one-hour exposure time. The key findings of this HRA are summarized in the table below, including the maximum PMI, MEIR, MEIW, sensitive receptor, and on-site receptor for the carcinogenic risk, as well as chronic and acute health effects. The cancer risk calculations at the PMI, MEIR, and at sensitive receptor locations are based on a continuous 70-year exposure time. The chronic hazard index and acute hazard index results are based on maximum annual and maximum hourly exposure periods, respectively. The calculated cancer burden is also included.

Summary of Potential Maximum Health Risks from UC Davis

Type of Estimated Health Impact	Cancer Risk (per million)	Chronic Hazard Index	Acute Hazard Index
Carcinogenic Risk at the Point of Maximum Downwind Impact (PMI)	2.2		
Chronic Non-Cancer Hazard Index at PMI		0.009	
Acute Non-Cancer Hazard Index at PMI			0.1
Maximum Exposed Individual Residential (MEIR)	2.0	0.009	0.08
Maximum Exposed Individual Worker (MEIW)	0.4	0.007	0.1
Off-Site Sensitive Receptor Location ¹	1.9	0.009	0.1
On-Campus Staff/Campus Gathering Location	0.8	0.01	0.2
On-Campus Student Housing	0.4	0.01	0.1
Population Cancer Burden ²	0.03 cases		

¹ A continuous 70-year exposure was assumed at all sensitive receptor locations, which would overstate risk to children or the elderly at these locations, hence health protective.

² This is not cancer risk, but rather an estimate of the expected number of cancer cases in the exposed population.

The AB 2588 law requires public notification in the area impacted by a given facility when individual cancer risk estimates exceed 10 in one million or a non-cancer hazard index exceeds 1.0. None of these levels were exceeded for the PMI, MEIR, or MEIW locations. Therefore, estimated maximum health risks from UC Davis activities during the 2009 reporting year were below public notification levels.

The cancer risk estimate at the PMI was dominated by diesel particulate matter from the campus IC engines (67 percent), the laboratory fume hoods (16 percent) and the incinerator (15 percent). The remaining 2 percent is contributed by other campus sources of TACs. Chemicals other than diesel particulate matter contributing to the cancer risk at this location include 9 percent from furans, 4 percent from dioxins, 4 percent from acrylamide, 3 percent from chloroform, and 3 percent from formaldehyde. The MEIR and MEIW cancer risk estimates were also dominated by the IC engines with secondary contributions from the laboratory fume hoods and the incinerator. The majority of the

cancer risk from these sources was attributable to diesel particulate matter, furans, dioxins, acrylamide, chloroform, and formaldehyde.

The chronic non-cancer health risks at the PMI, MEIR, and MEIW, and maximum off-site sensitive receptor location were highest for the respiratory target organ, with campus laboratories being the predominant source of the contributing emissions. Secondary contributions were from the IC engines. The majority of the PMI, MEIR, and MEIW chronic hazard index was attributable to glutaraldehyde, formaldehyde, hydrochloric acid, and diesel particulate matter.

Acute non-cancer health risk at the PMI, MEIR, MEIW, and the maximum sensitive receptor location was highest for the eye target organ and was predominantly attributable to the Primate Boiler #2 while combusting landfill gas, laboratory emissions, and natural gas-fired boilers. The majority of the acute hazard index at each of these locations was attributable to formaldehyde.

1.0 INTRODUCTION

On behalf of the University of California at Davis (UC Davis), ERM-West, Inc. (ERM) prepared this update to the *AB 2588 Air Toxics Health Risk Assessment for Reporting Year 2009* (HRA) to comply with the Air Toxics “Hot Spots” Information and Assessment Act of 1987 (Assembly Bill [AB] 2588 Hot Spots). This document presents the HRA methodology and results for toxic air contaminants (TACs) emitted from the UC Davis campus during the reporting year 2009. This HRA was prepared in compliance with AB 2588, administered by the Yolo-Solano Air Quality Management District (YSAQMD). The purpose of an AB 2588 HRA is to assess potential public health impacts associated with airborne emissions from routine operations. The sources analyzed in this HRA are the same as those included in the HRA prepared for reporting year 2005, with the addition of one boiler at the Central Heating and Cooling Plant (CHCP), and includes diesel-fired internal combustion (IC) engines located throughout the campus.

1.1 Site Location

The UC Davis campus is located in Davis, California, just west of Sacramento at the junction of Interstate 80 and Highway 113. The contact address for UC Davis is One Shields Avenue, Davis, California, 95616. The YSAQMD Plant identification number for UC Davis is 00025.

Figure 1 shows the general location of the UC Davis campus. The topography in this region is simple, with little variation in elevation. The elevation of the UC Davis campus is approximately 50 feet above mean sea level. Land use to the north, and some areas to the east of the campus are mixed commercial and residential. Agricultural land surrounds the campus to the west, south, and some areas to the east.

1.2 Health Risk Assessment Process

Public exposure to TACs released from UC Davis is predominantly through airborne emissions. Potential health risks resulting from these airborne emissions were assessed through multiple exposure pathways in accordance with guidance established by the California Office of Environmental Health Hazard Assessment (OEHHA). The AB 2588 “Hot Spots” law established a statewide program for the inventory of TAC emissions from individual facilities as well as requirements for risk assessment and public notification of potential health risks. This HRA report was based on methodology outlined in the *Air Toxics Hot Spots Program Risk Assessment Guidelines: The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments* (“OEHHA Guidelines”) (OEHHA 2003).

The HRA was conducted in three basic steps:

- **A hazard identification** was performed to determine pollutants of concern associated with UC Davis activities;
- **An exposure assessment** was conducted simulating the transport of pollutants using atmospheric dispersion modeling to locations of predicted exposure (or “receptors”); and
- **A risk characterization** was performed analyzing potential health risks from these calculated exposures, including the locations of maximum potential cancer and non-cancer health risks.

An overview of these steps is presented in the following subsections, with details provided in the remainder of this report.

1.2.1 Hazard Identification

As recommended by staff at the California Air Resources Board (CARB) (CARB 2009), emissions from the facility were largely assumed to be the same as those from the 2005 reporting year, with the addition of diesel-fired IC engines and a new boiler at the CHCP. On 16 April 2010, *Assembly Bill 2588 Health Risk Assessment Protocol for Reporting Year 2009 for the University of California at Davis* was submitted to the YSAQMD for approval of methods to be used in this HRA. A copy of the protocol and the YSAQMD approval letter are in Appendix A. As described in the protocol, emissions from the CHCP boilers, the primate boilers, and the landfill gas flare were updated based on 2009 operating information. The new CHCP boiler and emissions from the IC engines were added. Emissions from the small heaters and furnaces, kilns, chloroform remediation, veterinary incinerator, laboratory fume hoods, storage tanks, fuel dispensing, bulk solvent storage, and the wastewater treatment plant were not updated. This decision was based on the insignificant impacts reported in the 2005 AB 2588 HRA and that emissions of diesel particulate matter (DPM) were likely to dominate cancer risks. In total, 71 TACs (detailed in Appendix B) were modeled from various campus emission sources. A detailed description of the revised emission estimates for these TACs is included as part of this HRA in Section 2.0 and Appendix C.

1.2.2 Exposure Assessment

The exposure pathways included in this analysis were:

- Inhalation;
- Dermal absorption;
- Ingestion of soil with deposited pollutants;
- Exposure to pollutants potentially in mother's milk; and
- Exposure due to the consumption of homegrown produce.

Consideration of these pathways is consistent with the risk screening procedures contained in the OEHHA Guidelines (OEHHA 2003), as approved by the YSAQMD (see Appendix A).

For each exposure pathway, the dose of each chemical, in milligrams of chemical per kilogram of body weight per day (mg/kg-day), was calculated pursuant to the OEHHA Guidelines using the Hotspots Analysis and Reporting Program (HARP), Version 1.4a.

All health risks were calculated using the Tier 1 analysis from the OEHHA Guidelines. The Tier 1 assessment assumes a continuous 70-year exposure for long-term health risks. The Tier 1 analysis, required for AB 2588 HRAs, combines the 70-year exposure period with a standard point-estimate approach using upper-bound exposure pathway parameters recommended in the OEHHA Guidelines. This is described further in Section 4.0.

1.2.3 Risk Characterization

1.2.3.1 Cancer Risk

The OEHHA classifies 49 of the 71 chemicals evaluated in this HRA as confirmed or suspected human carcinogens. It is routinely assumed that the development of cancer as a result of chemical exposures takes place by the alteration of genetic code with no lower threshold for the

phenomena. That is, such chemicals are believed to present a cancer risk at any exposure level. Thus, **cancer risk** is expressed in terms of the chances (or probability) of contracting cancer at any exposure level. The calculated probability (typically expressed as the chances in one million) is always greater than zero when the exposure level is greater than zero. Under the AB 2588 program, public notification of potential cancer risks from a facility is required for estimated risks at **10 in one million or greater**.

Population exposure estimates of cancer risk can also be included in AB 2588 HRAs, subject to individual air district requirements. The YSAQMD required that a cancer burden calculation be included in the AB 2588 HRA for reporting year 2005 and therefore, has also been included as part of this AB 2588 HRA. The cancer burden is calculated by multiplying the number of people exposed by the individual cancer risk at the population centroid of each census block within a zone of impact (ZOI). The ZOI is defined by an isopleth surrounding the facility where receptors have a multipathway cancer risk of greater than 1 in one million. The result of this calculation is an estimate of the number of cancer cases expected from a 70-year exposure to currently estimated facility emissions.

1.2.3.2 Non-Cancer Risk

Fifty-six of the 71 chemicals in this HRA were evaluated for potential chronic non-cancer health effects, and 23 of the 71 chemicals were evaluated for potential acute non-cancer health effects.

Chronic toxicity is defined as the adverse biological effects caused by prolonged chemical exposure. These exposures may be continuous or repeated. Chronic effects usually occur at lower exposure levels than acute effects, primarily because of chemical accumulation in the body.

Acute toxicity is defined as adverse biological effects caused by brief chemical exposures of no more than 24 hours. Acute effects may range from simple eye or skin irritation to death. For most chemicals, eye or respiratory irritation is the main symptom of threshold acute exposures. The air concentration required to produce acute effects is typically higher than levels required to produce chronic effects, because the duration of exposure is shorter. Acute effects usually occur immediately or almost immediately after exposure begins and, if the exposure level is not high enough to cause serious injury or death, complete recovery usually follows soon after exposure ceases.

Non-cancer health risk of an inhaled air toxic is measured by the **hazard index**, the ratio of the reported concentration of an air toxic compound to an acceptable or "reference" exposure level (REL). For non-inhalation pathways, hazard indices are calculated as the ratio of calculated doses to acceptable or reference doses (RfDs). If the reported concentration or dose of a given chemical is less than its REL or RfD, then the hazard index will be less than 1.0. If more than one chemical is considered, it is assumed that multiple sub-threshold exposures could result in an adverse health effect. Thus, chemical-specific hazard indices are summed. Typically, for a given set of chemicals, hazard indices are summed for each organ system.

For any organ system, a total hazard index exceeding 1.0 indicates a potential health effect. The AB 2588 program uses total **hazard indices of 1.0 or greater** as the public notification criteria.

2.0 HAZARD IDENTIFICATION

The hazard identification involved the evaluation of all emission sources to determine if particular substances are used or generated that may cause health effects if released to the air. OEHHA Guidelines (OEHHA 2003 and updates) outline the compounds that are to be included in an AB 2588 HRA. Of the compounds included in the emissions calculations, Appendix B identifies the 71 chemicals required under AB 2588 guidance to be evaluated in this HRA, and whether they are associated with potentially carcinogenic risks or non-carcinogenic (acute or chronic) health effects. The toxicity factors used in this HRA are also given in Appendix B for each chemical, based on CARB- and OEHHA-approved toxicological factors, as contained in the HARP model.

The current campus emission sources included in this AB 2588 HRA for reporting year 2009 include:

- Laboratory operations
- Combustion sources:
 - CHCP boilers
 - Primate Center boilers
 - Veterinary medical incinerator
 - Numerous small boilers, each less than 5 million British thermal units (MMBtu) per hour in capacity
 - Walnut dryer and craft kilns
 - Diesel-fired IC engines
- Wastewater treatment plant
- Landfill (fugitive emissions plus flare emissions)
- Chloroform remediation operations
- Gasoline-dispensing operations
- Solvent-dispensing operations

Methodologies used to calculate emissions from these sources are described below. Emissions are summarized in tabular format in Appendix C.

2.1 Laboratory Emissions

2.1.1 Selection of Chemicals and Emissions Calculation Methodology

For the purposes of this AB 2588 HRA, and based on recommendations from CARB (CARB 2009), the same emission estimates were used as described in the *AB 2588 Air Toxics Health Risk Assessment for Reporting Year 2005* ("2005 AB 2588 HRA"; ERM 2008). A detailed description of the methodologies used to calculate laboratory fume hood emissions is included in that document. Maximum hourly emissions were calculated by applying a 5.18 factor to the annual average emissions. This factor is consistent with what was used in the 2005 AB 2588 HRA and

in various other university HRAs. The factor is based on ratios between maximum and average emission factors from other studies described in detail in Appendix D of the *University of California at Berkeley: Central Campus Health risk Assessment* (URS Corp. 2000). In a comment letter dated 10 November, 2008, OEHHA expressed concern that the use of the 5.18 factor may underestimate maximum hourly emission rates due to laboratory operating hours.

To specifically address OEHHA concerns regarding hours of operation, ERM conducted an additional analysis to estimate short-term laboratory emissions based on estimated annual laboratory operational hours. ERM has assumed that the hours of laboratory operations are equivalent to the number of instructional hours for the 2009 academic year. According to the *Academic & General Campus Calendars* (http://registrar.ucdavis.edu/html/academic_calendar.html), UC Davis includes three 10-week instructional quarters during the regular school year and two 6-week summer instructional sessions. Based on the academic calendar, there are 225 instructional days (including final exams) for the 2009 academic year. This estimate excludes weekends and holidays (including winter and spring break). Assuming 8-hours per instruction day, a total of 1,800 hours of laboratory operations were calculated. Based on 8,760 hours per year, a factor of 4.87 was calculated. Using this methodology and set of assumptions, the maximum hourly emissions can be calculated to be 4.87 times higher than the annual average emissions. Please note that the assumptions used do not account for the potential emissions during evening course instruction or from laboratory operations occurring due to research, and/or from hours (such as weekends) spent on independent work students may conduct to fulfill various course requirements. Therefore, the number of operational hours assumed may be an underestimate. This would lead to an over estimate of maximum hourly emissions. The two different methodologies for estimating maximum hourly emissions resulted in similar results. The factor of 5.18 appears to be a reasonable estimate for calculating maximum hourly chemical emissions due to laboratory operations.

2.2 Combustion Sources

2.2.1 Central Plant

There are four permitted boilers at the Central Plant. One of the boilers was installed during 2008 and is now operational. The three older boilers (CHCP Boiler #1, #2, and #3) are fired on natural gas with diesel as a back-up fuel. No diesel was used during reporting year 2009. The new boiler (CHCP Boiler #4) was fired both on natural gas and diesel (for testing purposes only) during 2009. TAC emissions from the natural gas and diesel combustion were calculated using emission factors from the California Toxic Emission Factor (CATEF) database available from the CARB (2006a, 2010). The emission factors were multiplied by the amount of fuel combusted to obtain emission estimates. Annual emissions were calculated based on actual annual fuel usage obtained from the UC Davis staff (UCD 2010). Hourly emissions were based on the rated heat input for each boiler. The emission factors and emission calculations are presented in Appendix C.

2.2.2 Primate Center Boilers

There are two boilers located at the Primate Research Center. Primate Center Boiler #1 fires natural gas and diesel as a backup fuel. No diesel was used during reporting year 2009. Boiler #2 fires both natural gas and landfill gas. Emissions from natural gas combustion were estimated using CATEF emission factors with metered 2009 natural gas usage. Emissions from the combustion of landfill gas (LFG) were estimated using emission factors from AP-42 (United States Environmental Protection Agency [USEPA] 1995a) with the measured 2009 LFG flow to the boiler. These emission calculations are consistent with the methodologies that were used in the 2005 AB 2588 HRA (ERM 2008). Appendix C contains tables summarizing emission factors and emission estimates.

2.2.3 Veterinary Medical Incinerator

The UC Davis veterinary medical incinerator is used to destroy both infectious and non-infectious carcasses. For the purposes of this analysis, as recommended by CARB (CARB 2009), emissions from this source remain the same as those used in the 2005 AB 2588 HRA. Estimated emissions from the Veterinary Medical Incinerator and the emission factors used are summarized in Appendix C.

2.2.4 Campus Boilers Less than 5 MMBtu/hr

There are more than 20 small natural gas-fired boilers located throughout the UC Davis campus. Emissions for these sources remain the same as those used in the 2005 AB 2588 HRA as recommended by the CARB staff (CARB 2009). The emissions were calculated using emission factors from the CATEF database (CARB 2006a) with metered 2005 natural gas usage. Appendix C contains tables summarizing emission factors and emission estimates.

2.2.5 Walnut Dryer and Craft Kilns

Emissions from the walnut dryer and craft kilns, which are located throughout the campus, were also estimated using emission factors from CATEF (CARB 2006a) and remain the same as those used in the 2005 AB 2588 HRA. The emission factors and emission calculations are summarized in Appendix C.

2.2.6 Diesel-Fired IC Engines

Emissions of diesel particulates for most IC engines were calculated based on source-specific emission factors obtained from UC Davis staff. For engines where no source-specific data were available, a conservative particulate emission factor of 1 gram per horsepower-hour (g/hp-hr) (Table 3.3-1 of Chapter 3.3, AP-42, USEPA 1995b) was used. Annual emissions were estimated based on actual hours of operation for both testing and actual emergencies for 2009. During emergency operations, it was assumed that the engine was operating at full rated capacity. During testing operations, it was assumed that the IC engines operated at 25 percent of the maximum capacity. Emissions from each engine were summed to calculate the total annual emissions from each IC engine. Maximum hourly emissions were based on the specific rated engine capacity. Appendix C summarizes emissions from the diesel-fired IC engines.

2.3 Wastewater Treatment Plant

Wastewater treatment plant emissions remain the same as those used in the 2005 AB 2588 HRA (ERM 2008). Emission estimates are summarized in Appendix C.

2.4 Campus Landfill

UC Davis operates a Class III (general municipal solid waste) landfill. LFG is created as buried waste decomposes. The precise composition of LFG varies from landfill to landfill, but is approximately 50-55 percent methane, 35-40 percent carbon dioxide, 5-10 percent nitrogen (and other gases, including oxygen and hydrogen sulfide), and trace amounts of non-methane organic compounds (NMOCs). TACs are contained chiefly in the NMOCs. The campus landfill has an LFG recovery system, which is a series of buried wells and pipes connected to the vacuum side of an air-moving system that collects LFG and draws it to a flare for combustion. Any uncollected LFG escapes to the atmosphere as fugitive emissions. Collected NMOCs are destroyed in the flare with high efficiency, on the order of 98-99 percent or greater (Table 2.4-3 of Chapter 2.4, AP-42, USEPA 1995c). UC Davis has not measured the "collection efficiency" of the LFG collection system installed at the campus landfill, as this is difficult to

measure. The LFG not captured by the LFG recovery system is emitted as fugitive emissions from the landfill surface. The same methodology used to estimate the recovery rate and the fugitive emissions in the 2005 AB 2588 HRA was also used in this analysis in conjunction with current LFG flow rates. The combined measured LFG flow rate to the LFG flare and Primate Center Boiler #2 for 2009 was 7,099,000 standard cubic feet per year. Appendix C contains calculations of TAC emissions from fugitive LFG and LFG combusted in the LFG flare and Primate Boiler #2.

2.5 Chloroform Remediation Operations

UC Davis currently operates a groundwater remediation system by the campus landfill, and two remediation systems at the South Campus Disposal Site (SCDS), one for groundwater and the other for soil, for the treatment of chloroform contamination. For the purposes of this AB 2588 HRA, emissions are assumed to remain the same as those used in the 2005 AB 2588 HRA. Please refer to that document for a detailed description of the emission calculation methodologies. The emissions are summarized in Appendix C.

2.6 Gasoline Dispensing Operations

Emissions from gasoline dispensing were calculated storage tanks at Agricultural Services, the Primate Center, Grounds Division, Pomology, the University Airport, and Fleet Services. Gasoline-dispensing emissions were assumed to remain unchanged since the 2005 AB 2588 HRA. TACs from gasoline vaporization were estimated using the CARB Speciation Profiles (CARB 2006b) and are summarized in Appendix C.

2.7 Storehouse/Receiving Bulk Solvent Storage

Emissions from the Storehouse would occur from the dispensing of solvents. The emissions were assumed to remain unchanged since the 2005 AB 2588 HRA. The emissions are summarized in Appendix C.

3.0 EXPOSURE ASSESSMENT

An exposure assessment was conducted using atmospheric modeling to simulate the transport of pollutants to locations of predicted exposure (or “receptors”). This section describes the atmospheric dispersion modeling conducted for this HRA and the exposure assumptions used at the various receptor types.

3.1 Air Dispersion Modeling

3.1.1 Model Selection

The atmospheric transport and dilution of emissions from the UC Davis campus sources were simulated by the USEPA-approved Industrial Source Complex Short Term 3 (ISCST3) model (USEPA 1995c). This mathematical model estimates dilution of emissions by diffusion and turbulent mixing with clean air as they move away from a source downwind. It can predict the resulting cumulative concentrations from many point, area, volume, and open pit sources at numerous specified locations of interest (“receptors”). The ISCST3 model is capable of predicting impacts in simple terrain (receptors at or below stack height), intermediate terrain (receptors between stack height and final plume rise centerline), and complex terrain (receptors above final plume rise centerline).

Land use north, and some areas to the east-northeast of the UC Davis campus is generally urban. However, land use to the west, south, and some areas to the east-southeast of the campus is predominantly agricultural. Because most of the surrounding land use is rural, rural dispersion coefficients were used.

Other technical options selected for the ISCST3 modeling were USEPA default options, as listed below:

- Final plume rise;
- Buoyancy induced dispersion;
- Stack tip downwash;
- Calm processing routine;
- Default wind speed profile exponents (rural); and
- Default vertical temperature gradients.

The following sections present a more expanded technical discussion of the dispersion modeling that was performed for this HRA, in accordance with the approved modeling protocol contained in Appendix A.

3.1.2 Source Data

A total of 223 point sources (stacks) and 12 area sources were used to represent emissions from the UC Davis campus. Source parameters are summarized in Appendix D.

3.1.2.1 Laboratory Sources

Existing rooftop vents associated with laboratory emissions were represented using 102 point sources. Because of the large number of buildings associated with laboratory emissions, a

single point source was used to represent emissions from each building. Stack parameters for all laboratory vents (existing and proposed) were based on information from the *Air Toxics Assessment for the University of California Davis 2003 Long Range Development Plan* (2003 LRDP HRA) (URS 2003) and the 2005 AB 2588 HRA (ERM 2008). Stack parameters are summarized in Appendix D.

3.1.2.2 Combustion Sources and Other Point Sources

Combustion sources and the Storehouse/Receiving Bulk Storage were modeled as point sources. Combustion sources included in the modeling are described in Section 2.2. Stack parameters for three of the four CHCP boilers, the boilers located at the Primate Center and the small boilers and heaters were obtained from the 2005 AB 2588 HRA. Stack height, stack inner diameter, and location for the new CHCP Boiler #4 were supplied by UC Davis staff. Flow rates were estimated using "F-Factor" combustion calculations. Stack parameters are summarized in Appendix D.

The Storehouse/Receiving Bulk Storage was modeled as one point source (building vent). Stack parameters were assumed to be the same as those assumed for all laboratory building vents.

Stack parameters for the landfill gas flare were obtained from the 2005 AB 2588 HRA. Emissions from the chloroform remediation systems were modeled as point sources, and stack parameters for these were obtained from the 2005 AB 2588 HRA.

3.1.2.3 Diesel-fired IC Engines

Eighty-four point sources were used to represent existing stationary and portable diesel-fired IC engines (emergency generators) located throughout campus. Stack parameters for 49 of the emergency generators were based on information obtained from the 2003 LRDP HRA (URS 2003). UC Davis staff supplied complete stack parameters for an additional 25 generators, with the exception of one stack height. For this generator, the stack height was based on the average height of the "known" stack heights for other campus generators. Partial stack parameters were supplied for an additional 11 generators, including stack height and diameter. Flow rates for these sources were estimated using "F-Factor" combustion calculations assuming a temperature equal to the average of other existing generators. Stack parameters for the final generator assumed an average stack height and diameter, and the flow rate was estimated using "F-Factor" combustion calculations. Stack parameters for the emergency generators in the modeling are summarized in Appendix D.

3.1.2.4 Area Sources

Conservatively, all area sources were assumed to have a ground-level release height. Area sources included those representing the landfill, the wastewater treatment plant, and gasoline dispensing. Modeling parameters are summarized in Appendix D.

3.1.3 Building Downwash

When sources are located near or on buildings or structures, the dispersion of the plume can be influenced. Under certain wind speeds, the wake produced on the lee side of the building can cause the plume to be pulled toward the ground near the building, resulting in higher concentrations close to the building. These effects are called building downwash.

The effects of building downwash have been considered in this modeling analysis. The USEPA provides specific guidance to determine whether or not a building potentially affects pollutant dispersion. According to that guidance, if a structure is located within a certain distance from the emission source (stack), downwash effects must be considered. Stack heights that minimize

downwash effects are designated good engineering practice (GEP) stack heights. The GEP formula height is defined as:

$$H_s = H_b + 1.5L_b$$

where:

H_s = GEP formula height

H_b = Building height

L_b = The lesser building dimension of the height, length, or width.

The existing emission stacks on campus are less than GEP formula height and thus were considered in the downwash analysis. Because of the complexity of the stack/building relationships on the UC Davis campus, the analysis included all buildings that could potentially influence each point source. Figure 2 illustrates the buildings and point source locations included in the downwash analysis.

The USEPA-approved Building Profile Input Program (BPIP) was used to provide input for the downwash analysis. This program calculates the GEP formula stack heights and direction-specific building dimensions for input to the ISCST3 model. BPIP requires the input of building corner coordinates and heights, and stack coordinates. The Universal Transverse Mercator (UTM) Coordinate System was used to identify building and source locations.

3.1.4 Meteorological Data

The ISCST3 model requires the input of an hourly meteorological dataset consisting of observations of wind speed, wind direction, temperature, atmospheric stability, and mixing height. A 5-year dataset was used for this HRA (1985-1989). The surface data were collected at the Sacramento Metropolitan Airport. Mixing height data were calculated using upper air data collected at the Oakland International Airport. The wind distribution is shown in Figure 3 as a "wind rose." The length of each spoke indicates the percentage of the time that the wind is blowing *from* that compass direction.

3.1.5 Receptor Locations

Receptors were placed at 50-meter increments along the campus boundaries. Additional receptors were located at 100-meter increments to a distance of 500 meters, at 250-meter increments to a distance of approximately 1 kilometer. Off-site discrete and/or sensitive receptors, including schools, daycare centers, hospitals, and nursing homes, were also identified to a distance of 5 miles. Receptors at two on-campus daycare centers and the Cowell Student Health Center are also included. Appendix E contains a table listing the sensitive receptors and locations in UTM Coordinates. It also includes a copy of the Offsite Receptor Report prepared for this HRA by Environmental Data Resources, Inc. Figure 4 illustrates the locations of sensitive receptors. Receptors were also placed at student residential locations and locations representative of on-campus gathering areas and administrative staff and workers.

The off-site receptor grid and on-site receptor locations are illustrated in Figure 5. After modeling was completed, results were reviewed. The PMI locations for cancer risk, chronic non-cancer health effects, and acute non-cancer health effects all occurred at the property boundary and the estimated health hazards decreased with distance from the UC Davis campus. Thus, no additional receptors were necessary. Receptor locations are indicated using UTM Coordinates, and receptor elevations were taken from digital elevation models digitized from United States Geological Survey maps.

3.2 Exposure Durations

The assessment of cancer risk and chronic non-cancer health effects used annual-average emissions, while assessment of acute non-cancer health effects used maximum short-term emissions. Also, for the acute analysis, it was conservatively assumed that all maximum short-term emissions would occur in the same hour.

For individuals at off-campus residential locations, the cancer risk calculations assumed that these individuals would never leave these locations for 70 years and that all existing university operations included in the dispersion modeling would operate over the 70-year period. At other specific receptor locations, including off-site workers, student housing, and representative locations for university staff and campus gathering areas, a continuous 70-year exposure is not appropriate. For these receptors, the following assumptions were applied:

- **Student Residents**

Adjust standard 70-year residential exposure assumption to a duration of 24 hours/day, 350 days/year for 9 years, or by a factor of 9/70.

- **Off-Site Workers and On-site University Staff/Gathering Areas**

Assume the HARP standard default for off-site workers, which includes:

- *Exposure duration 8 hours/day, 245 days/year for 40 years.*
- *For the inhalation pathway, a worker breathing rate of 149 liters per kilogram body weight per day.*

Note that off-site workers who are teachers would have less exposure due to their schedules; however, for the purposes of this assessment, they were treated as any other off-site worker.

Sensitive receptors include schools, daycare centers, hospitals, and nursing homes. The potential cancer risk for the non-working populations at these receptors assumed the standard 70-year residential exposure, which would overstate the risks to children and the elderly since these Tier 1 estimates assume a lifetime exposure. Chronic and acute non-cancer health effects were assessed from annual-average and short-term exposure estimates, respectively, without further adjustments from the standard Tier 1 assumptions and, as stated above, it was assumed for the acute analysis that all maximum short-term emissions would occur in the same hour. Furthermore, the non-cancer toxicity factors were established by the CARB and OEHHA to be protective of sensitive members of the population or those undergoing physiological change, which include children and the elderly. Therefore, it is believed that these analyses have accounted for the protection of sensitive individuals.

4.0 RISK CHARACTERIZATION

Human doses were calculated for the modeled environmental exposures over specified time periods via multiple environmental pathways. These environmental pathways included direct inhalation, soil ingestion, dermal (skin) absorption, consumption of homegrown produce, and mother's milk. The exposure algorithms follow air toxics HRA guidance published in the OEHHA Guidelines (OEHHA 2003).

All health risks were calculated using the Tier 1 analysis from the OEHHA Guidelines. The Tier 1 assessment assumes a continuous 70-year exposure for long-term health risks to determine the point of maximum impact (PMI) and the maximum exposed individual resident location (MEIR). The maximum exposed individual worker location (MEIW) used an adjusted exposure period consistent with the OEHHA Guidelines, as described in Section 3.2.

The Tier 1 analysis, required to be conducted for AB 2588 HRAs, combines the 70-year exposure period with a standard point-estimate approach using upper-bound exposure pathway parameters recommended in the OEHHA Guidelines. As was described in the protocol submitted to and approved by the YSAQMD (see Appendix A), the Tier 1 analysis performed for this HRA calculates cancer risk with the derived (adjusted) method, consistent with the CARB Interim Risk Management Policy (CARB 2003).

4.1 Exposure Pathways

The inhalation exposure pathway in this health risk assessment involves the direct inhalation of gaseous and particulate air pollutants. In addition, there is the potential for exposure via non-inhalation pathways due to the deposition of particulate pollutants. Potential non-inhalation exposure pathways include soil ingestion, dermal (skin) absorption, mother's milk, and ingestion of homegrown produce.

The ISCST3 dispersion model was run with a unit emission rate (1 gram per second [g/s]) for all sources to calculate normalized air concentrations (micrograms per cubic meter [$\mu\text{g}/\text{m}^3$] per g/s emissions) at each receptor point. The model outputs were then used in conjunction with source-specific emission rates for each toxic air contaminant as well as toxicity factors to calculate potential health effects. The calculations were performed using the HARP model, Version 1.4a. HARP uses ISCST3 dispersion model outputs along with site-specific emissions data and pollutant-specific toxicity factors. The HARP model implements the methodologies described in the OEHHA Guidelines (OEHHA 2003). Appendix F includes a CD with electronic input and output files from the ISCST3 dispersion modeling and the HARP modeling runs.

To estimate airborne concentrations, HARP uses the normalized modeled dilution factor (normalized concentration) for each receptor location from the ISCST3 output and multiplies them by pollutant and source-specific emission rates. For estimating non-airborne pollutant concentrations from particulate emissions, HARP first estimates air concentrations for each pollutant ($\mu\text{g}/\text{m}^3$) by multiplying particulate pollutant emission rates (g/s) by the maximum normalized concentration, and then uses the following equation to estimate pollutant deposition rates:

$$\text{Dep} = \text{GLC} * \text{Dep-rate} * 86,400$$

where:

$$\text{Dep} = \text{deposition on the affected soil area per day (micrograms per square meter per day } [\mu\text{g}/\text{m}^2/\text{day}]$$

GLC = estimated ground-level air concentrations ($\mu\text{g}/\text{m}^3$)

Dep-rate = vertical rate of deposition (meters per second [m/s])

The factor of 86,400 is the number of seconds in a day. OEHHA recommends a deposition rate value (Dep-rate) of 0.05 m/s for uncontrolled sources and 0.02 m/s for controlled sources (OEHHA 2003). It would be overly conservative to use an uncontrolled deposition rate for the combustion sources since the particulate matter from these sources are extremely small in size, thus the controlled deposition rate of 0.02 m/s (representative of fine particulate matter) was used in these calculations. These deposition estimates were then used in algorithms contained in HARP for the soil ingestion, dermal (skin) absorption, mother's milk, and homegrown produce pathways for the evaluation of the PMI and MEIR. Consistent with OEHHA Guidelines, the MEIW was evaluated with only the inhalation, soil ingestion, and dermal absorption pathways. In addition, the homegrown produce and mother's milk pathways would not apply to the on-campus student and on-campus worker receptors, as well as the off-site sensitive receptor locations. These were run with the inhalation, soil ingestion, and dermal absorption pathways as well.

4.2 Toxicity Assessment

4.2.1 Cancer Risk

The OEHHA classifies 49 of the 71 chemicals evaluated in this HRA as confirmed or suspected human carcinogens. It is routinely assumed that the development of cancer as a result of chemical exposures takes place by the alteration of genetic code with no lower threshold for the phenomena. That is, such chemicals are believed to present a cancer risk at any exposure level. Some chemicals could act by non-genetic mechanisms at certain exposure levels, or act in concert with other chemicals upon one or multiple exposures to cause cancer. Since carcinogenic mechanisms for all chemicals are not precisely understood, regulatory agencies have decided to regulate all known or potential human carcinogens as if they all act as no-threshold initiators of cancer.

It is known that cancer incidence increases greatly with age because of increased lifetime exposure to risk factors and because of changes in hormonal status and other biological factors. Exposures to agents such as radiation, cigarette smoke, and diet can present cancer risks or can interact with chemical exposures. However, observations in either humans or animals are rarely sufficient to characterize the variation in age accurately, so most health risk assessments are based on lifetime cancer risk. Thus, **cancer risk** is expressed in terms of the chances (or probability) of contracting cancer at any exposure level typically over a lifetime exposure. The calculated probability (typically expressed as the chances in one million) is always greater than zero when the exposure level is greater than zero. Under the AB 2588 program, public notification of potential cancer risks from a facility is required for estimated risks at **10 in one million or greater**.

Toxicologists for both the OEHHA and the USEPA have developed cancer slope factors (SFs) for various compounds based on epidemiological studies in humans (when available), or more commonly, from animal studies. Cancer slope factors are typically expressed either in terms of inhalation exposure (SF_i) or oral (ingestion) exposure (SF_o). They represent the potential risk of contracting cancer per daily dose of the chemical (in milligrams of chemical per kilogram of body weight per day [1/(mg/kg-day)]). These factors are sometimes referred to as dose-response relationships. The cancer toxicity factors used in this HRA are presented in Appendix B.

Cancer risk through the inhalation exposure pathway is calculated as:

$$\text{Cancer Risk (Inhalation)} = \frac{\text{Inhalation Dose} \times \text{Inhalation Slope Factor (SFi)}}{\text{Factor (SFi)}}$$

where:

$$\text{Inhalation Dose (mg/kg-day)} = \text{Ca} * (\text{IR} * \text{EF} * \text{ET}) / (\text{BW} * \text{AT} * 350 * 1000)$$

Ca = air concentration of pollutant ($\mu\text{g}/\text{m}^3$)

IR = inhalation rate (m^3/day)

EF = exposure frequency (days/yr)

ET = exposure time (days)

BW = body weight (kg)

AT = averaging time for toxic effect (days)

When exposure is assessed in terms of an airborne concentration, SFi can be expressed in terms of a unit risk factor (URF), which is the probability of contracting cancer over a specified time period assuming continuous exposure to a $1 \mu\text{g}/\text{m}^3$ airborne concentration. The URF is related to the SFi as follows:

$$\text{URF} = (\text{SFi} * \text{IR} * \text{ET}) / (\text{BW} * \text{AT} * 1000)$$

Cancer risk through the non-inhalation pathways is calculated from the sum of non-inhalation doses:

$$\text{Cancer Risk (Non-Inhalation)} = \text{Dose (sum of non-inhalation pathways)} \times \text{Oral Slope Factor (SFo)}$$

Calculation of dose through the different non-inhalation pathways is detailed in the OEHHA Guidelines. The HARP model was used for these dose calculations.

Population exposure estimates of cancer risk can also be included in AB 2588 HRAs, subject to individual air district requirements. The YSAQMD requires that a cancer burden calculation be included. The cancer burden is calculated by multiplying the number of people exposed by the individual cancer risk at the population centroid of each census block within a ZOI. The ZOI is defined by an isopleth surrounding the facility where receptors have a multipathway cancer risk of greater than 1 in one million. The result of this calculation is an estimate of the number of cancer cases expected from a 70-year exposure to current estimated facility emissions.

4.2.2 Non-Cancer Health Effects

Fifty-six of the 71 chemicals in this HRA were evaluated for potential chronic non-cancer health effects, and 23 of the 71 chemicals were evaluated for potential acute non-cancer health effects.

Chronic toxicity is defined as the adverse biological effects caused by prolonged chemical exposure. These exposures may be continuous or repeated. Chronic effects usually occur at lower exposure levels than acute effects primarily because of chemical accumulation in the body. Since chemical accumulation to toxic levels typically occurs slowly, symptoms of chronic effects usually do not appear until long after exposure commences. The highest no-effect exposure level is the RfD, which is expressed in terms of mass dose of the chemical per body weight per day ($\text{mg}/\text{kg}\text{-day}$). Below these thresholds, the body is capable of eliminating or detoxifying the chemicals rapidly enough to prevent accumulation. As with cancer toxicity

factors, RfDs are usually provided either in terms of inhalation exposure (RfDi) or oral (ingestion) exposure (RfDo). If exposure is expressed as an airborne concentration, these thresholds are sometimes expressed as reference air concentrations (s). This HRA uses the OEHHA term of REL for reference air concentration (RfC). The chronic REL and RfDi for a given chemical are related as follows:

$$\text{REL} = (\text{RfDi} \cdot \text{BW} \cdot \text{AT} \cdot 1000) / (\text{IR} \cdot \text{ET})$$

where:

REL = reference air concentration causing a toxicological response ($\mu\text{g}/\text{m}^3$)

RfDi = inhalation reference dose causing a toxicological response ($\text{mg}/\text{kg}\text{-day}$)

The other factors are the same as defined in Section 4.2.1.

Acute toxicity is defined as adverse biologic effects caused by brief chemical exposures of no more than 24 hours. Acute effects may range from simple eye or skin irritation to death. For most chemicals, eye or respiratory irritations are the main symptoms to threshold acute exposures. The air concentration required to produce acute effects is typically higher than levels required to produce chronic effects because the duration of exposure is shorter. Acute effects usually occur immediately or almost immediately after exposure begins and, if the exposure level is not high enough to cause serious injury or death, complete recovery usually follows soon after exposure ceases. A minimum exposure level is required to cause any acute effect. These threshold exposure levels (RfCs or RELs) correspond to levels that manifest milder acute health effects such as eye irritation or simple respiratory discomfort.

Non-cancer adverse health effects of an inhaled air toxic are measured by the **hazard index**, the ratio of the reported concentration of an air toxic compound to an acceptable or REL. For non-inhalation pathways, hazard indices are calculated as the ratio of calculated doses to acceptable or RfDs. If the reported concentration or dose of a given chemical is less than its REL or RfD, then the hazard index will be less than 1.0. The non-cancer toxicity factors used in this HRA are presented in Appendix B.

If more than one chemical is considered, it is assumed that multiple subthreshold exposures could result in an adverse health effect. Thus, chemical-specific hazard indices are summed. Typically, for a given set of chemicals, hazard indices are summed for each organ system. For any organ system, a total hazard index exceeding 1.0 indicates a potential health effect. The AB 2588 program uses total **hazard indices of 1.0 or greater** as the public notification criteria.

5.0 ESTIMATED HEALTH RISKS

Health risks are estimated by combining human exposure calculations with toxicological dose-response relationships. The calculation of potential airborne concentrations of 71 chemicals associated with campus operations was discussed in Section 3.0, and the methods used to estimate human doses to these chemicals for several environmental pathways and the assessment of potential health risk were described in Section 4.0. This section presents the resulting estimates of health risks when the estimated human exposures are combined with the dose-response relationships.

The Tier 1 approach used in this HRA uses the 70-year exposure period, although it is not likely that most people will reside at a single residence for 70 years. This assumption is used as a benchmark for comparing risks calculated from different facilities on the same basis and evaluating the effectiveness of regional control strategies. It is also useful as a risk management tool to assess potential risks to individuals remaining in the same area over a lifetime and exposed to multiple cumulative exposures. However, as a measure of the actual individual risk at any given location due to emissions from a single facility, these assumptions would tend to overestimate individual lifetime cancer risk. Section 6.0 describes sources of uncertainty in the risk estimates in more detail.

There are eight chemicals that contribute most significantly impact the results of either the cancer and/or non-cancer adverse health effects in this UC Davis HRA. Appendix G contains detailed chemical profiles; however, brief descriptions of the potential health effects are presented here.

The major contributor to both the cancer risk and the chronic non-cancer health effects was due to emissions of DPM from IC engines. The cancer risks associated with DPM exposures are primarily linked to lung and bladder cancer. Chronic non-cancer health effects are linked to respiratory system causing damage to the lungs.

Exposures to hydrochloric acid was one of the major contributors to the chronic HI and maximum impact locations. Long-term exposures can potentially effect the respiratory system, specifically, the nasal passage, the larynx, and the trachea.

Chloroform has a potential link to cancer of the bladder, rectum, large intestines, and other digestive tract organs. Symptoms of acute chloroform toxicity include fainting, vomiting, fatigue, headache, dizziness, respiratory depression and coma. Limited data on chronic effects indicate that chloroform inhalation may cause depression, gastrointestinal distress, increased risk of viral hepatitis and slight liver damage.

Cancer risks associated with exposures to chlorinated dibenzo-p-dioxins and dibenzofurans may be linked to cancers of the digestive system, lymphatic and hematopoietic system cancer, myeloma cancer, rectal cancer and leukemia. Non-cancer chronic health effects due to long-term exposures to chlorinated dibenzo-p-dioxins and dibenzofurans can cause damage to the liver, the reproductive system, fetal development, the endocrine system, respiratory and the hematopoietic (blood) system.

Acrylamide is a possible carcinogen linked to lung cancer and central nervous system cancer. Formaldehyde is also linked to lung cancer as well as known to cause increased rates of brain cancer and leukemia in embalmers who use it. Chronic non-cancer effects of formaldehyde include increased incidence of headaches and eye, nose and throat irritation. Acute effects include eye and upper respiratory tract irritation.

Glutaraldehyde chronic exposure effects include skin sensitivity resulting from dermatitis and irritation of the eyes and nose and occupational asthma. Hydrochloric acid chronic exposure effects include bleeding of the nose and gums, and ulceration of the upper respiratory system mucous membranes. Acute inhalation exposure may result in coughing, choking, sore throat, nasal discharge, burning of the respiratory tract and pulmonary edema. Nitric acid has acute health effects including corrosivity to the eyes, skin, nose, mucous membranes, respiratory tract, gastrointestinal tract or any other tissue with which it comes in contact.

5.1 Cancer Risk

The following presents a summary of the study results at the PMI, MEIR, and MEIW cancer risk locations. More detailed tables specifying source and chemical contributions can be found in Appendix H. Detailed chemical profiles of the chemicals contributing most significantly to cancer risk are provided in Appendix G.

This study calculated a PMI cancer risk (point of maximum impact regardless of land use) of **2.16 in one million**, occurring along the UC Davis northern property boundary along Russell Boulevard near the intersection of S. Campus Way. Figure 6 illustrates the locations of maximum impact. This risk is based on an assumption of a 350-day-per-year exposure over a 70-year period. Table 1 shows the breakdown of the estimated cancer risks by campus source category. The estimated cancer risk at this location was primarily due to emissions from the diesel-fired IC engines (66.8 percent), with secondary contributions laboratory fume hoods (15.9 percent) and the incinerator (14.8 percent). All other campus sources combined contributed the remaining 2.5 percent of the cancer risk at the PMI. The majority of the risk was attributable to DPM (66.7 percent), furans (9.5 percent), dioxins (4.7 percent), acrylamide (3.7 percent), chloroform (3.5 percent), and formaldehyde (2.7 percent).

The MEIR was calculated at **2.0 in one million**, and located north of the property boundary along Russell Boulevard at the corner of Oeste Drive. This risk is based on an assumption of a 350-day-per-year exposure over a 70-year period. Table 2 shows the breakdown of the estimated cancer risks by campus source category. The estimated cancer risk at this location was primarily due to emissions from the diesel-fired IC engines (66.1 percent), laboratory fume hoods (16.2 percent), and the incinerator (15.3 percent). All other campus sources combined contributed the remaining 2.4 percent of the cancer risk at the MEIR. The majority of the risk was attributable to DPM (66.3 percent), furans (9.8 percent), dioxins (4.8 percent), acrylamide (3.8 percent), chloroform (3.5 percent), and formaldehyde (2.8 percent). Appendix H includes tables detailing the results.

The MEIW was calculated at **0.4 in one million**, occurring at an assumed existing off-campus worker located at the corner of Russell Boulevard and Anderson Road. This risk is based on an assumption of an 8-hour-per-day, 245-day-per-year exposure over a 40-year period. Table 3 shows the breakdown of the estimated cancer risks by campus source category. The estimated cancer risk at this location was primarily due to emissions from diesel-fired IC engines (65.0 percent), laboratory fume hoods (16.7 percent), and the incinerator (15.8 percent). All other campus sources combined contributed the remaining 2.5 percent of the cancer risk at the MEIW. The majority of the risk was attributable to DPM (65.0 percent), furans (10.3 percent), dioxins (5.1 percent), acrylamide (3.8 percent), chloroform (3.6 percent), and formaldehyde (2.8 percent). Tables summarizing these findings can be found in Appendix H.

The maximum estimated cancer risk at an off-site sensitive receptor location was **1.94 in one million**, occurring at the Woodland Clinic Medical Group located north of campus along Russell Boulevard. This estimate was based on assuming a 350-day-per-year exposure over 70 years. For the non-worker population at this receptor location, this represents an overestimate. The maximum estimated on-campus risk for a university worker or at a campus gathering place was **0.8 in one million** based on an assumption of an 8-hour-per-day, 245-day-per-year

exposure over 40 years. A student living at an on-campus residence had a calculated cancer risk of **0.4 in one million**, assuming continuous exposure over a 9-year period.

Finally, cancer burden is calculated for populations within the ZOI or areas where the 70-year cancer risk is estimated to be at 1 in one million or higher. Figure 7 illustrates the extent of the ZOI. The estimated cancer burden in the population surrounding the UC Davis campus was estimated at 0.03 cases within the exposed population.

5.2 Non-Cancer Health Effects

As described in Section 4.2.2, the methodology used for assessing chronic non-cancer health effects is the calculation of non-cancer hazard indices for each target organ. Detailed chemical profiles of the chemicals contributing most significantly to non-cancer health effects are provided in Appendix G.

The maximum chronic hazard index at the PMI was calculated to be **0.009 for the respiratory system**, located north of the campus along Russell Boulevard, north of Russell Intramural Field. Table 1 shows the breakdown by campus source category. The estimated chronic health effect at this location was primarily due to emissions from laboratories (86.1 percent), with a secondary contribution from the diesel-fired IC engines (7.9 percent). All other campus sources combined contributed the remaining 1.6 percent of the maximum chronic hazard index at the PMI. The majority of the PMI chronic hazard was attributable to glutaraldehyde (43.1 percent), hydrochloric acid (31.2 percent), formaldehyde (14.4 percent), and DPM (7.9 percent).

The maximum chronic hazard index at the MEIR was calculated to be **0.009 for the respiratory system**, located north of the campus on the west corner of Russell Boulevard and College Park. Table 2 shows the breakdown by campus source category. The estimated chronic health effect was primarily due to emissions from campus laboratories (85.7 percent). A secondary contribution was from the diesel-fired IC engines (8.1 percent). All other campus sources combined contributed the remaining 1.7 percent of the maximum chronic hazard index at the MEIR. The majority of the MEIR chronic hazard was attributable to glutaraldehyde (43.0 percent), hydrochloric acid (31.1 percent), formaldehyde (14.4 percent), and DPM (8.1 percent).

The maximum chronic hazard index at the MEIW was calculated to be **0.007 for the respiratory system**, located north of the campus at the corner of Russell Boulevard and Anderson Road. The estimated chronic health effect was primarily due to emissions from campus laboratories (81.0 percent). A secondary contributing source includes the diesel-fired IC engines (11.5 percent). All other campus sources combined contributed the remaining 4.7 percent of the maximum chronic hazard index at the MEIW. The majority of the MEIW chronic hazard was attributable to glutaraldehyde (40.5 percent), hydrochloric acid (29.8 percent), formaldehyde (14.6 percent), and DPM (11.5 percent).

The maximum estimated chronic hazard indices at other receptors were **0.009** at an off-site sensitive receptor, **0.01** at a university worker location or on-campus gathering place, and **0.01** at an on-campus residence. Tables summarizing these findings are in Appendix H.

The maximum acute hazard index at the PMI was calculated to be **0.1 effecting the eyes**, and was located along the property boundary northeast of the Primate Center. Table 1 shows the breakdown by campus source category. The combustion of landfill gas from Primate Center Boiler #2 contributed 65.5 percent to this acute health effect. Emissions from the laboratory hoods contributed 19.9 percent and the CHCP boilers contributed 10.4 percent. The remaining sources contributed 4.2 percent to the total acute health effects. The majority of the acute impact was attributable to formaldehyde, contributing 91.1 percent.

The maximum acute hazard index at the MEIR was calculated to be **0.08 for the eyes**, located northeast of the Primate Center on Larue Way. Table 2 shows the breakdown by campus source category. Emissions from the Primate Center Boiler #2 fired on LFG contributed 44.5 percent to this acute health effect. Secondary contributing sources include the laboratory fume hoods at 33.3 percent and the CHCP at 16.2 percent. The remaining 6.0 percent was contributed from other sources on campus. The majority of the contribution to the acute effects at the MEIR was attributed to formaldehyde at 90.7 percent.

The maximum acute hazard index at the MEIW was calculated to be **0.1 for the eyes** located northeast of the Primate Center at the Grace Valley Christian Academy. Table 3 shows the breakdown by campus source category. The Primate Center Boiler #2 fired on LFG contributed 62.3 percent with secondary contributions from the laboratories (21.9 percent) and the CHCP boilers (11.5 percent). The remaining 4.3 percent of the impacts were from the remaining sources on campus. Most of the acute effects at the MEIW were attributed to formaldehyde (91.3 percent).

The maximum estimated acute hazard indices for other receptors were **0.1** at an off-site sensitive receptor, **0.2** at a university worker location or on-campus gathering place, and **0.1** at an on-campus residence. Tables summarizing these findings can be found in Appendix H.

5.3 Conclusions

The AB 2588 law requires public notification in the area impacted by a given facility when individual cancer risk estimates exceed 10 in one million or a non-cancer hazard index exceeds 1.0. None of these levels have been exceeded at the PMI, MEIR, or MEIW locations. Therefore, estimated maximum health risks from UC Davis activities during the 2009 reporting year were below public notification levels.

6.0 UNCERTAINTIES

Predictions of potential health risks related to UC Davis activities entails uncertainties because of gaps in scientific knowledge in the practice of exposure and risk assessment, as well as the need to simplify some aspects of the process for a manageable computational effort. In general, there are model and data uncertainties with respect to the assumed emissions, dispersion modeling, characteristics of the potentially exposed populations, and toxicological factors.

Because risk assessments are so often performed to set some regulatory limit on exposure for the protection of public health, the assumptions of risk assessments have tended to overestimate rather than underestimate risk. The methodologies used in this risk assessment followed the Tier 1 “point estimate” approach described in the OEHHA Guidelines (OEHHA 2003). Point-estimate risk values are based on a central tendency approach combined with 95 percent upper confidence limit exposure factors to arrive at single point health risk estimates, believed to be conservative upper-bound estimates. Sometimes, risk assessments follow a “stochastic approach,” presenting ranges of health risk rather than single numerical values to better convey the actual uncertainties involved. The 2003 OEHHA guidance offers alternative stochastic approaches to defining exposure factors that provide for a quantitative or semi-quantitative treatment of the risk estimate variability.

For this HRA, the standard Tier 1 regulatory approach of employing health-protective “point estimate” assumptions was used to provide a degree of maximum protection on environmental values. The resulting health risk predictions should be viewed as maximum estimates of the actual health risks. Although the assessment process includes assumptions that may individually either overestimate or underestimate impact, as described below, on balance, health risk impacts are probably overestimated by a substantial margin.

6.1 Emission Estimates

Emission estimates could be in error due to limits in scientific certainty. This bias could be toward underestimation or overestimation for any given source. Conservative (i.e., overpredictive) assumptions were applied where possible in the estimation of emissions. However, it is possible that all sources of emissions were not identified, and it was necessary to limit the number of substances included in the analysis. These latter two factors could lead to an underestimation of risk. The sources excluded from the HRA were determined to have a low emissions potential. It is believed that emission sources representing a significant emissions potential have been included in the HRA.

Literally hundreds of chemicals are used in UC Davis campus laboratories. In addition, chemicals are emitted from the landfill, wastewater treatment plant, campus combustion sources, and other campus sources. For practical reasons, it was necessary to limit the number of substances included in the analysis in order to complete the HRA within reasonable resource constraints. Yet, 71 chemicals are analyzed in this HRA, including 49 carcinogens. In most risk assessments, calculated health risks are dominated by only a handful of the evaluated chemicals. The 71 chemicals evaluated in this HRA include common chemicals addressed in most risk assessments, and are likely representative of the highest emitted TACs at UC Davis. While it is possible a chemical was missed that could be a significant contributor to health risk, this is believed unlikely. Thus, omission of substances from the HRA is unlikely to lead to a substantial underestimation of health risks based on the structure of this HRA.

Finally, the emission estimation methodologies that were used could have errors leading to underestimation or overestimation of emissions for any given chemical. For the laboratory emissions, an effort was made to use upper-bound evaporation estimates in the emission calculations. For large emitting sources including CHCP boilers, Primate Center boilers and the diesel-fired IC engines, actual 2009 fuel usage information supplied by UC Davis was used for the emissions calculations. For small boilers (<5 MMBtu/hr), the same emissions estimates calculated for the 2005 AB 2588 HRA were used. These data were assumed to be representative of typical annual operations, and could be higher or lower for any operation in any given year. USEPA and CARB emission factors used by regulatory agencies such as the YSAQMD were applied to the annual fuel use data and rated equipment capacities to arrive at emission estimates. These factors on balance tend to overestimate rather than underestimate potential emissions.

In summary, there are factors in the estimation of emissions that could lead to underestimation or overestimation of health risks. It is believed that the compounds chosen for analysis in this HRA are likely to have characterized the substantial majority of potential health risks, and that the emission calculation procedures used are not likely to have caused a significant underestimation of risk, and may well represent an overestimation.

6.2 Air Dispersion Modeling

In general, USEPA-approved dispersion models, such as the one used in this risk assessment, tend to overpredict concentrations rather than underpredict them. For example, all chemical emissions are assumed not to be transformed in the atmosphere. For certain pollutants, conversion to less toxic forms may occur sufficiently quickly to reduce concentrations from the conservative model predictions. Moreover, these models use assumptions about plume dispersion that tend to overpredict concentrations. In the modeling for this HRA, it was necessary to group multiple sources together (e.g., for many buildings, all laboratory emissions were modeled from one stack rather than from many stacks), which tends to overestimate risks because emissions are concentrated into a single plume rather than in several disperse, smaller plumes.

The surface-level meteorological data used in the dispersion modeling were obtained from the Sacramento Metropolitan Airport, located about 20 miles from Davis. The Sacramento data were the closest meteorological data available in a format necessary for the dispersion modeling. The general meteorological characteristics of Sacramento and Davis are similar. These surface-level meteorological data were augmented by "upper air" data from Oakland International Airport. "Upper air" data define limits for the "vertical mixing" of pollutants in the atmosphere. The Oakland station is the closest upper air reporting station to Davis. The protocol for this HRA proposed the use of the meteorological data described above, and the YSAQMD (2010) found these data to be representative of Davis. The use of these meteorological data combined with the conservatism of the ISCST3 model should have created an overall bias to overestimation rather than underestimation of health risks.

6.3 Exposure Assessment

The most important uncertainties concern the definitions of exposed populations and their exposure characteristics. The choice of a 70-year exposure period at residential exposure locations for lifetime risk estimates is very conservative in the sense that no person will actually spend 24 hours a day, 350 days a year, for over 70 years at exactly the point of highest toxicity-weighted annual average air concentrations. The greatest true exposure is likely to be at least two times, and perhaps more than 10 times lower than that calculated by this assumption. The average period of U.S. residency at any one location is about 9 years, and the 90th percentile of

residency (typically used by the USEPA in “reasonable maximum exposure” estimates) is about 30 years. In addition, the exposure assessment considered all feasible environmental exposure pathways, including inhalation, soil ingestion, dermal absorption, crop consumption, and mother’s milk.

For selected non-residential receptors included in the analysis for which a 70-year exposure assumption is not representative, assumptions were applied that likely overestimated long-term exposure. These included at every exposure location assessed: a 40-year working lifetime for university staff, faculty, and school/daycare workers; and a continuous 9-year exposure for students on-campus and in student housing. For short-term exposure, there is also likely overprediction because the analysis assumed that all campus operations involving the use of chemicals of short-term concern will occur at maximum hourly emission rates all at the same time.

6.4 Toxicity Assessment

All estimates of cancer and non-cancer toxicity for this HRA came from toxicologists with the State of California, and are among the most conservative compilations of toxicity information available. Toxicity estimates are derived either from observations in humans or from projection of information derived from experiments with laboratory animals. Human data are obviously more relevant for health risk assessments, but are often uncertain because of the difficulty of estimating exposures associated with the health effect of interest, insufficient numbers of people studied, relatively high occupational exposures must be extrapolated to low environmental exposures, or the population studied may be more or less susceptible than the population as a whole.

Cancer risk coefficients from human data are typically considered best estimates and are applied without safety factors. As discussed previously, cancer risk is typically considered proportional to pollutant concentration at any level of exposure (i.e., a linear, no-threshold model), which is conservative at low environmental doses. For non-cancer effects, the lowest exposure known to cause effects in humans is usually divided by uncertainty or safety factors to account for variations in susceptibility and other factors. When toxicity estimates come from animal data, they usually involve extra safety factors to account for possibly greater sensitivity in humans, and the less-than-human-lifetime observations in animals. Overall, the toxicity assumptions and criteria used in this HRA are biased toward overestimating risk.

6.5 Summary

Although this HRA includes both component features that overestimate and underestimate impacts, on balance, maximum individual health risks are probably overestimated. The amount of the bias is unknown, but could be substantial.

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- CARB. 2006a. *California Air Toxics Emission Factor Database*. <http://www.arb.ca.gov/ei/catef/catef.htm>. Webpage updated as of 25 February 2006. Accessed 1 March 2007.
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- URS Corporation (URS). 2003. *Air Toxics Health Risk Assessment for the University of California Davis 2003 Long Range Development Plan*. April.
- URS. 2000. *University of California Berkeley Central Campus Human Health Risk Assessment*. Prepared for UC Berkeley Physical and Environmental Planning. 28 June.

Figures

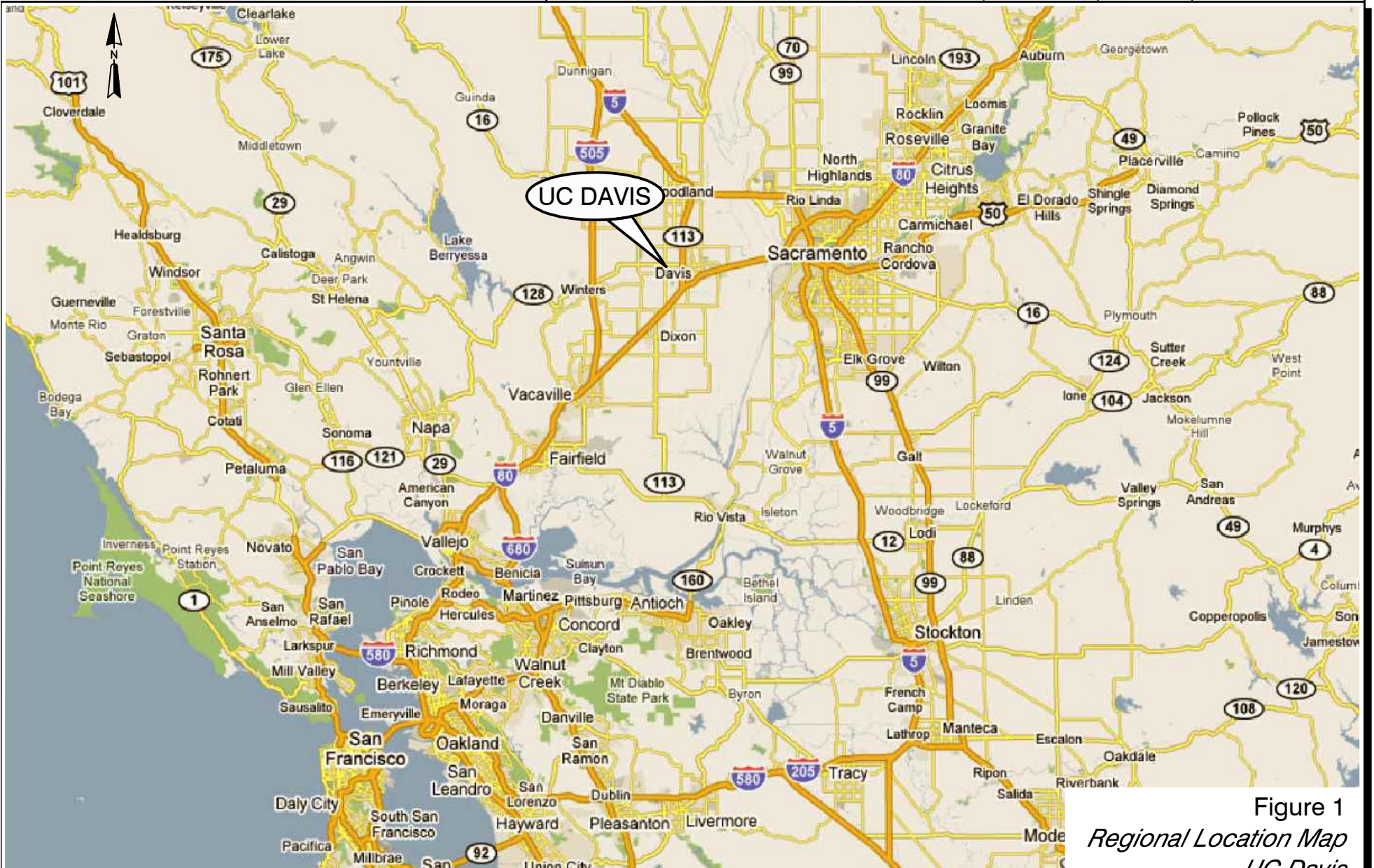


Figure 1
Regional Location Map
UC Davis
Davis, California

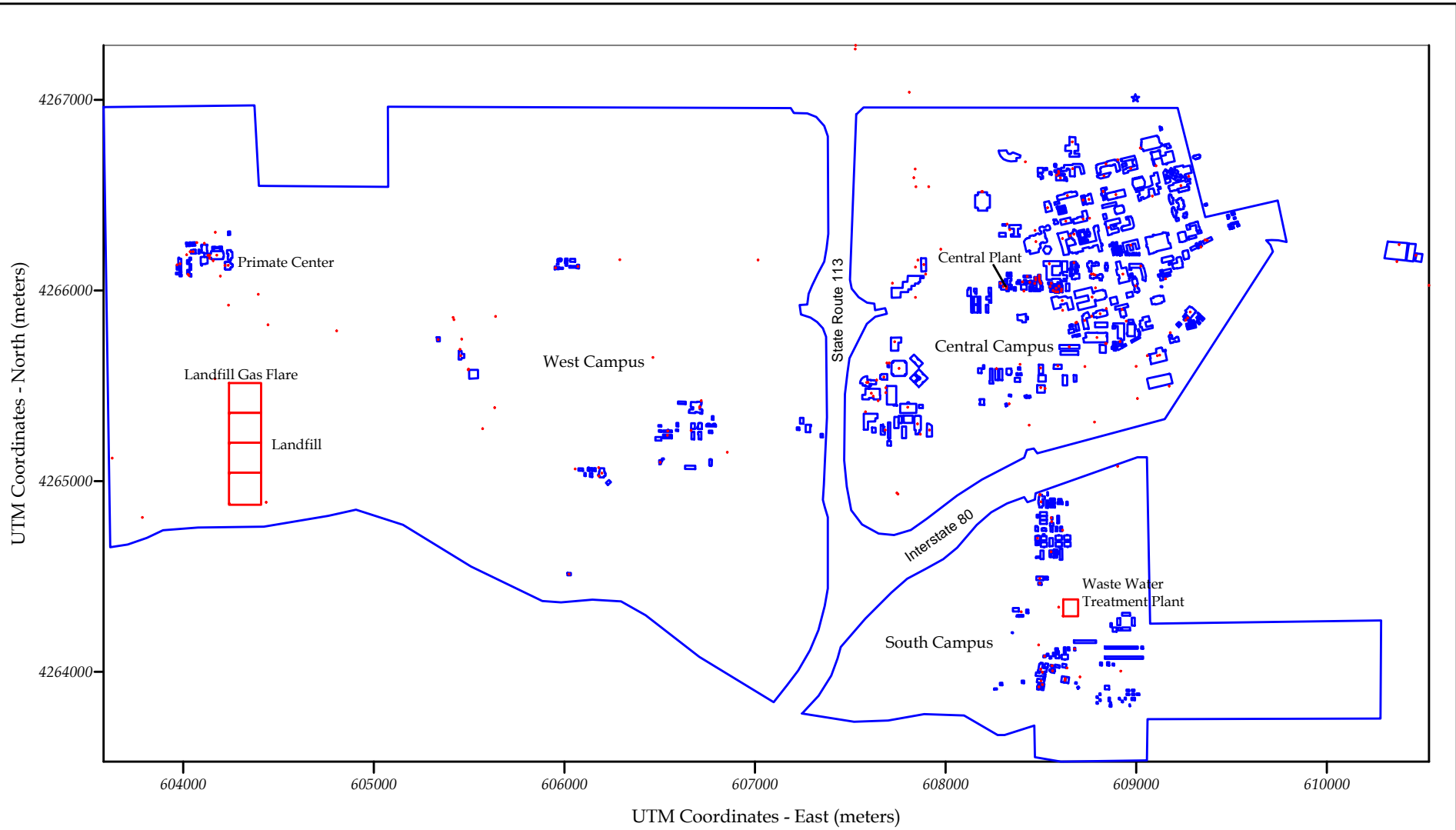
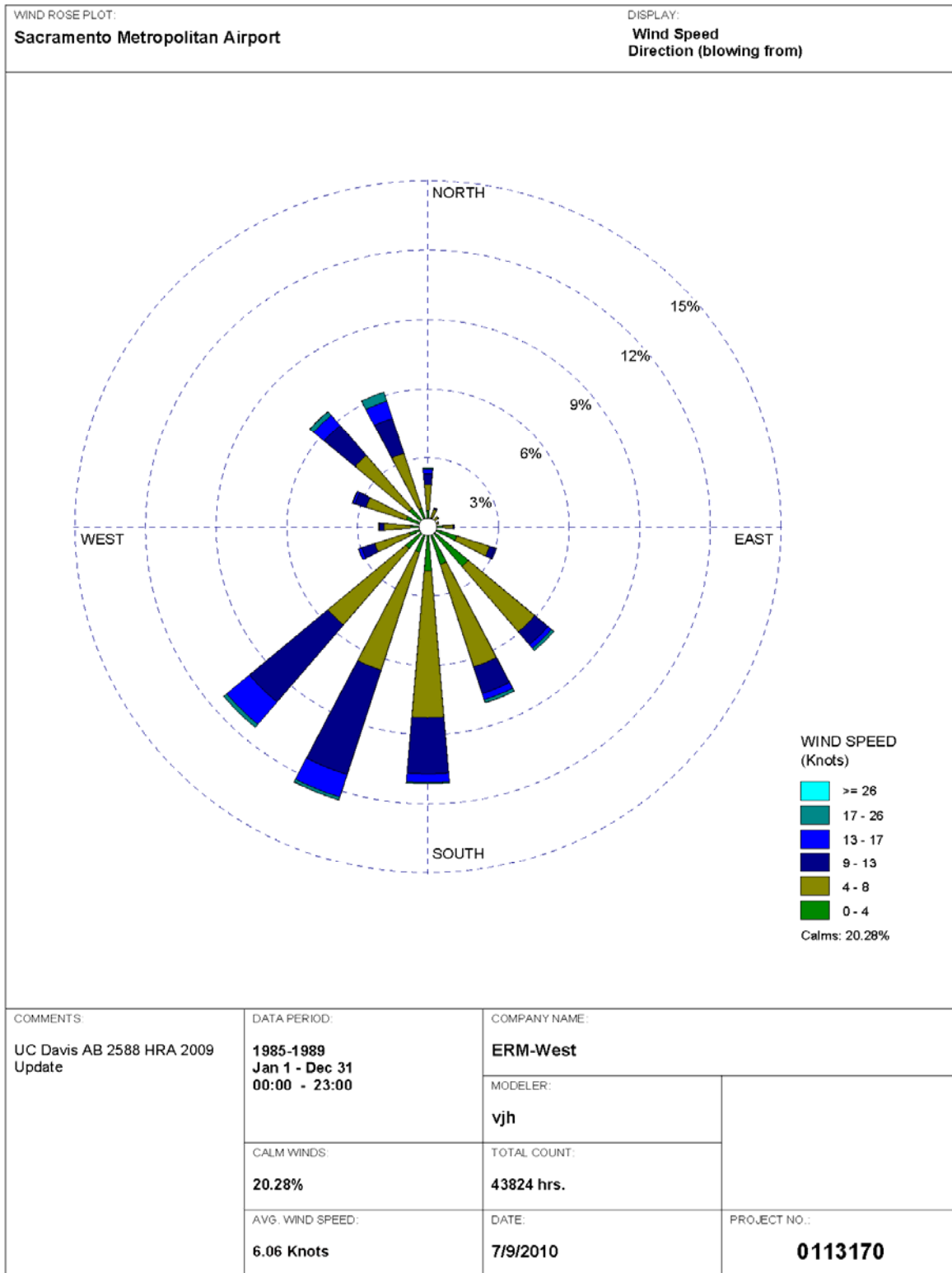


Figure 2

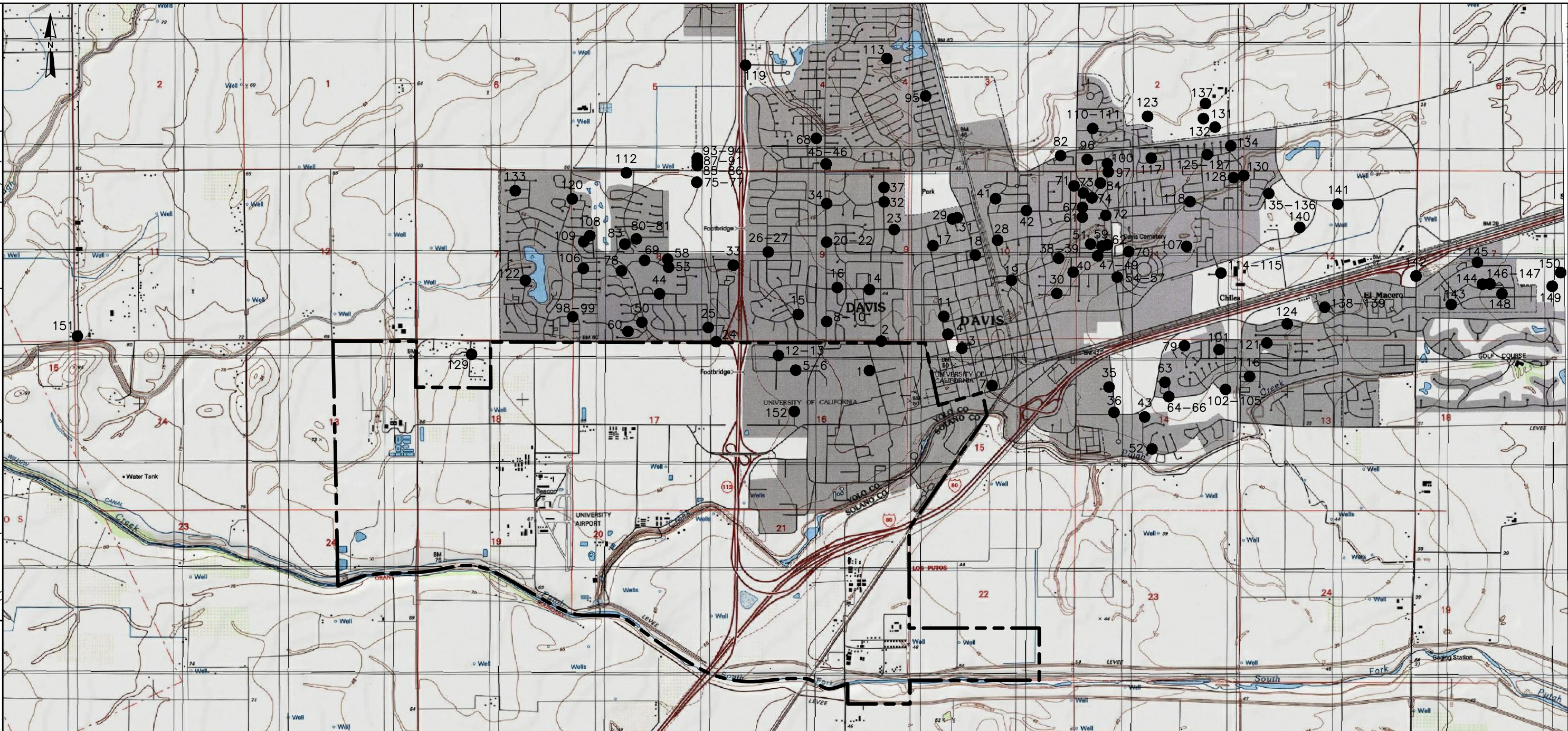
*Building and Modeled Source Locations
UC Davis
Davis, California*



WRPLOT View - Lakes Environmental Software

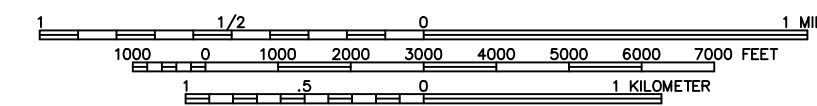
Figure 3
Five-Year Wind Rose
UC Davis
Davis, California

Project No. 0113170-02.dwg
 Date: 07/21/10
 Drawn By: R. Olson
 CAD File: C:\0113170\011317000-02.dwg



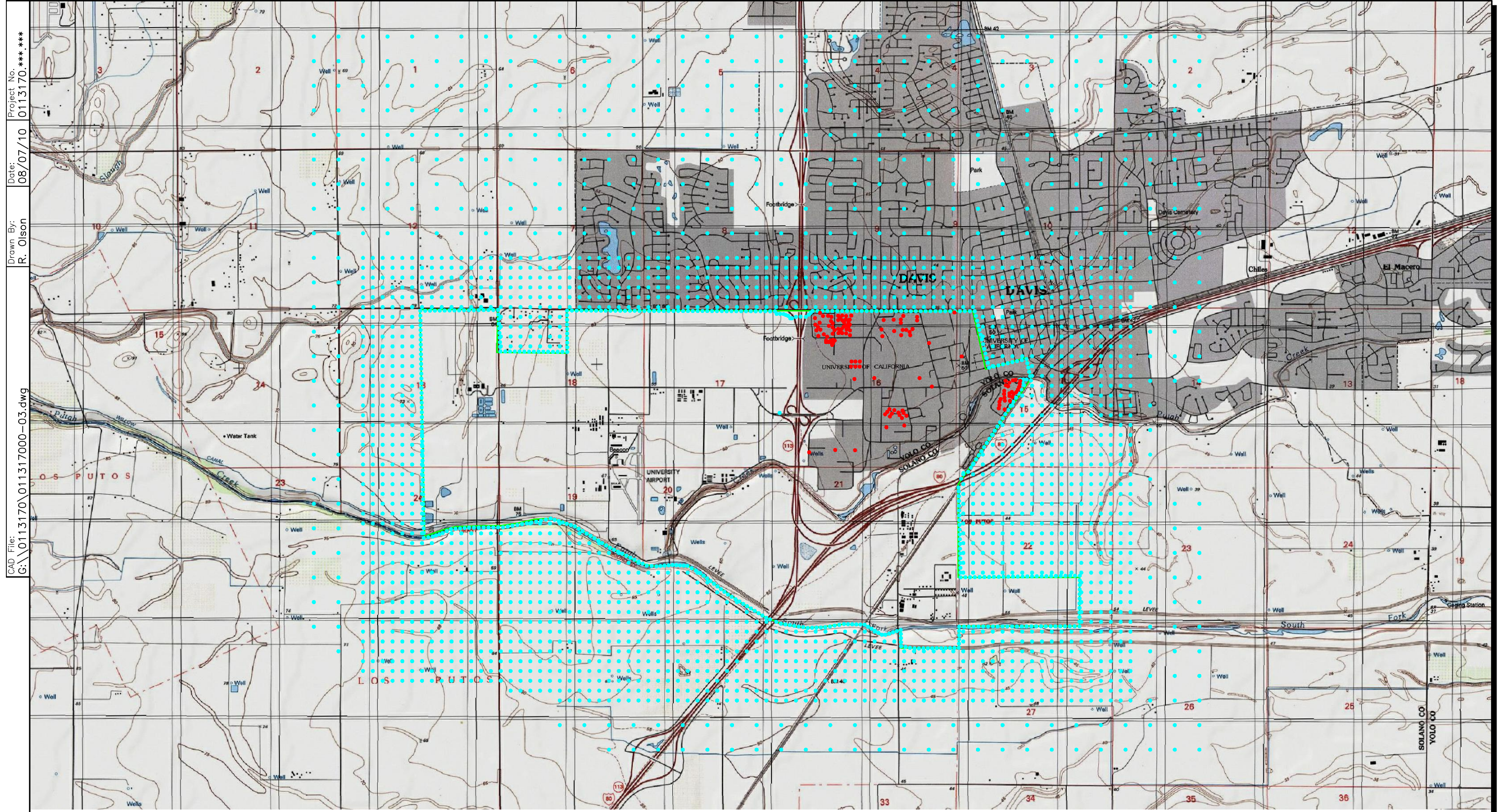
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1	COWELL STUDENT HEALTH CENTER	20	CESAR CHAVEZ STATE PRESCHOOL	39	VALLEY OAK SCHOOL AGE CHILD DEVELOPMENT CENTER	58	NAVARRO, ESPERANZA	77	UNIVERSITY RETIREMENT	96	BOUGHTON, KRISTEN	115	MONTESSORI COUNTRY DAY II	134	HONEYCUTT, BEOLA
2	WOODLAND CLINIC MEDICAL GROUP	21	CESAR CHAVEZ SCHOOL AGE CDC	40	BOWERS, LEANN	59	CONNOLLY, COLLEEN	78	A WORLD OF LEARNING	97	BIRCH LANE ELEMENTARY	116	BARAJAS, MARIA	135	DAVIS CHILDREN'S CENTER
3	COMMUNITY CHURCH NURSERY SCHOOL	22	CESAR CHAVEZ ELEMENTARY	41	STONE, ELISA	60	LAMBERT, PATRICIA	79	CHANG, DEE	98	PARKSIDE CHILDREN'S HOUSE	117	KUSS, LESLIE	136	FRED T. KOREMATSU ELEMENTARY SCHOOL AT MACE RANCH
4	DAVIS SCHOOL FOR INDEPENDENT STUDY	23	DAVIS SENIOR HIGH	42	OLIVER WENDELL HOLMES JUNIOR HIGH	61	BAKAY, DAVID AND SKOG, LESLYN	80	PATWIN SCHOOL AGE CHILD DEVELOPMENT CENTER	99	PARKSIDE CHILDREN'S HOUSE	118	PROGRESS RANCH - THE GROVE	137	DAHAL, SHAKUNTALA
5	LA RUE PARK CHILD DEVELOPMENT CENTER	24	APLEGATE NURSERY	43	THOMAS, DIANA	62	CUETARA, JULIE	81	PATWIN ELEMENTARY	100	BIRCH LANE SCHOOL-AGE CHILD DEVELOPMENT CENTER	119	DAVIS WALDORF SCHOOL	138	MERRYHILL COUNTRY SCHOOL
6	LA RUE PARK CHILD DEVELOPMENT CENTER	25	COOK, NOAH	44	RALPH WALDO EMERSON JUNIOR HIGH	63	PYTEL, JEANNIE	82	WELLNESS EXPRESS CLINIC	101	LETELIER, EDUARDO	120	TENDER LEARNING CARE	139	MERRYHILL COUNTRY SCHOOL - LAVIDA
7	YOLO HOSPICE	26	ROBERT E. WILLETT SCHOOL AGE CDC	45	PHYSICIANS CLINICAL LABORATORY	64	MERRYHILL SCHOOL	83	NOORISTANI, TAIBA	102	DAVIS PARENT NURSERY SCHOOL	121	ALEMI, NAJ	140	RAMOS, OLGA
8	DAVIS PHYSICAL THERAPY/MATRIX	27	ROBERT E. WILLETT ELEMENTARY	46	STEPHEN H FOSTER MD	65	MERRYHILL	84	HARZULA, RUTH	103	MARGUERITE MONTGOMERY CHILD DEVELOPMENT CENTER	122	ROMERO, LUCY	141	MOHAMED, SAYDA
9	RANSDELL LABORATORIES	28	MALHOTRA, SURINDER	47	COURTYARD HEALTHCARE CENTER	66	MERRYHILL SCHOOL #1036	85	SUTTER DAVIS HOSPITAL PULMONARY LAB	104	MARGUERITE MONTGOMERY CHILD DEVELOPMENT CENTER	123	SALAMATI, ROSHAN	142	UNIVERSITY COVENANT NURSERY SCHOOL
10	JAMES A KENNEDY MD	29	NORTH DAVIS ELEMENTARY	48	COURTYARD HEALTHCARE CENTER	67	HERNANDEZ, KAY	86	SUTTER DAVIS HOSPITAL	105	MARGUERITE MONTGOMERY ELEMENTARY	124	SAH, RANJANA	143	CARSON, JESSICA
11	KING (MARTIN LUTHER) HIGH (CONTINUATION)	30	WONG-XOQUIC, HEATHER	49	COURTYARD HEALTHCARE CENTER	68	VALCARENGHI, MICHELLE	87	ALICE VAN ALSTINE, MD	106	WILLIAMS, GINA	125	YOLO CRISIS NURSERY-FAMILIES FIRST INC.	144	PIONEER SCHOOL AGE CHILD DEVELOPMENT CENTER
12	RUSSELL PARK CHILD DEVELOPMENT CENTER (INFANTS)	31	NORTH DAVIS SCHOOL AGE CHILD DEVELOPMENT CENTER	50	BENNETT, MELE	69	MEDINA, ELIZABETH	88	WILLIAM HOCH MD	107	REYNOSO, GUADALUPE	126	YOLO CRISIS NURSERY-FAMILIES FIRST INC.	145	BIRYUKOVA, TAIYANA
13	RUSSELL PARK CHILD DEVELOPMENT CENTER	32	LEONARDO DAVINCI HIGH	51	RON A BERRYHILL	70	TROSTEL, TAM	89	JOHN D HERRIRED, MD	108	FARMER, KATIE	127	FAMILIES FIRST, INC.	146	DJUSD CHILDREN'S CENTER
14	DAVIS PARENT NURSERY SCHOOL #2	33	EBERLE, USA	52	SAH, B. DEVI	71	ALARCON-SOTO, SANDRA SOTO, JUAN	90	PETER E DROUBAY MD	109	HILLMAN, ANNE	128	MAROTTO, JOANNE	147	PIONEER ELEMENTARY
15	INTERNATIONAL PARENT-CHILD LEARNING CENTER	34	GAN HAVERIM PRESCHOOL	53	CHAVEZ, JOSEFINA	72	SAH, VINA	91	CHARLES A DERBY MD	110	MONTESSORI COUNTRY DAY	129	REDBUD MONTESSORI	148	DEO, GODAWARI
16	MOORE, JANET	35	SUTTER DAVIS VISITING NURSE ASSOC	54	SIERRA HEALTH CARE CONVALESCENT HOSP	73	HASSAN, MABEL	92	INTERNAL MEDICINE CONSULTANTS	111	MONTESSORI COUNTRY DAY	130	MONTESSORI COUNTRY DAY	149	YANCHER, LYNDIA & ROSS
17	ST JAMES ELEMENTARY SCHOOL	36	TPMG - DAVIS MOB	55	SIERRA HEALTH CARE CENTER	74	RAJBHANDARI, VIDYA	93	DAVIS COMMUNITY CLINIC, THE	112	WOODLAND HEALTHCARE-DAVIS MEDICAL GRP	131	POUDYAL, SHANTI	150	SAH, NORMA
18	DISCOVERY PRESCHOOL	37	SUTTER VISITING NURSE ASSOC - DAVIS	56	SIERRA HEALTHCARE CONVALESCENT	75	UNIVERSITY RETIREMENT COMM AT DAVIS	94	DAVIS COMMUNITY CLINIC	113	CHAVEZ, IRMA	132	THORESON, NITA	151	FAIRFIELD ELEMENTARY
19	MURRAY-CLARK, JAMIE	38	VALLEY OAK STATE PRESCHOOL	57	SIERRA HEALTH CARE CENTER	76	UNIVERSITY RETIREMENT	95	GREENAMYER, ALICIA	114	MONTESSORI COUNTRY DAY II	133	STEPHENS, MARGARET	152	HUTCHINSON CHILD DEVELOPMENT CENTER

LEGEND
 ● Sensitive Receptor Location
 - - - UC Davis Property Boundary



References:
 TOPO!® Software
 U.S.G.S. 7.5 Minute Series (Topographic) Quadrangle,
 Merrit, California Version: 1992; Current: 1992
 Davis, California Version: 1992; Current: 1992

Figure 4
 Sensitive Receptor Locations
 UC Davis
 Davis, California
 ERM 07/10



Project No. 0113170-03.dwg
 Date: 08/07/10
 Drawn By: R. Olson
 CAD File: G:\0113170\011317000-03.dwg

References:
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 U.S.G.S. 7.5 Minute Series (Topographic) Quadrangle,
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 Davis, California Version: 1997; Current: 1992

LEGEND

- On-Site Receptor Location
- Off-Site Receptor Location

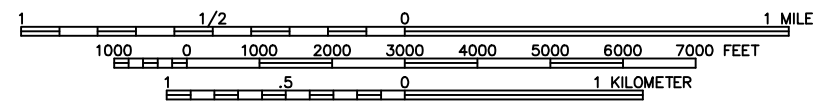
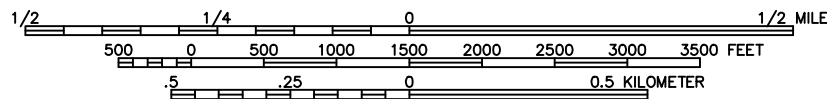
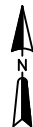
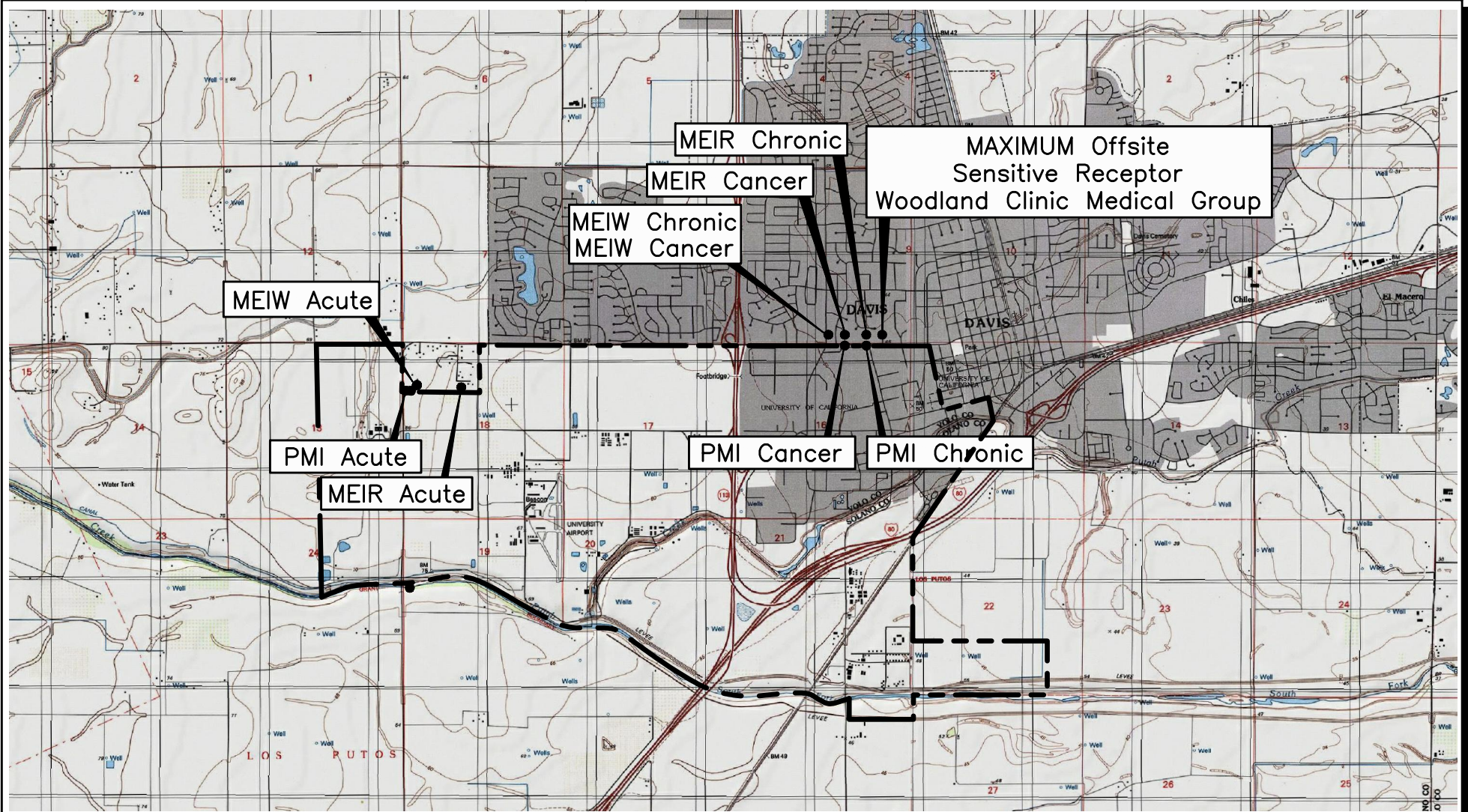
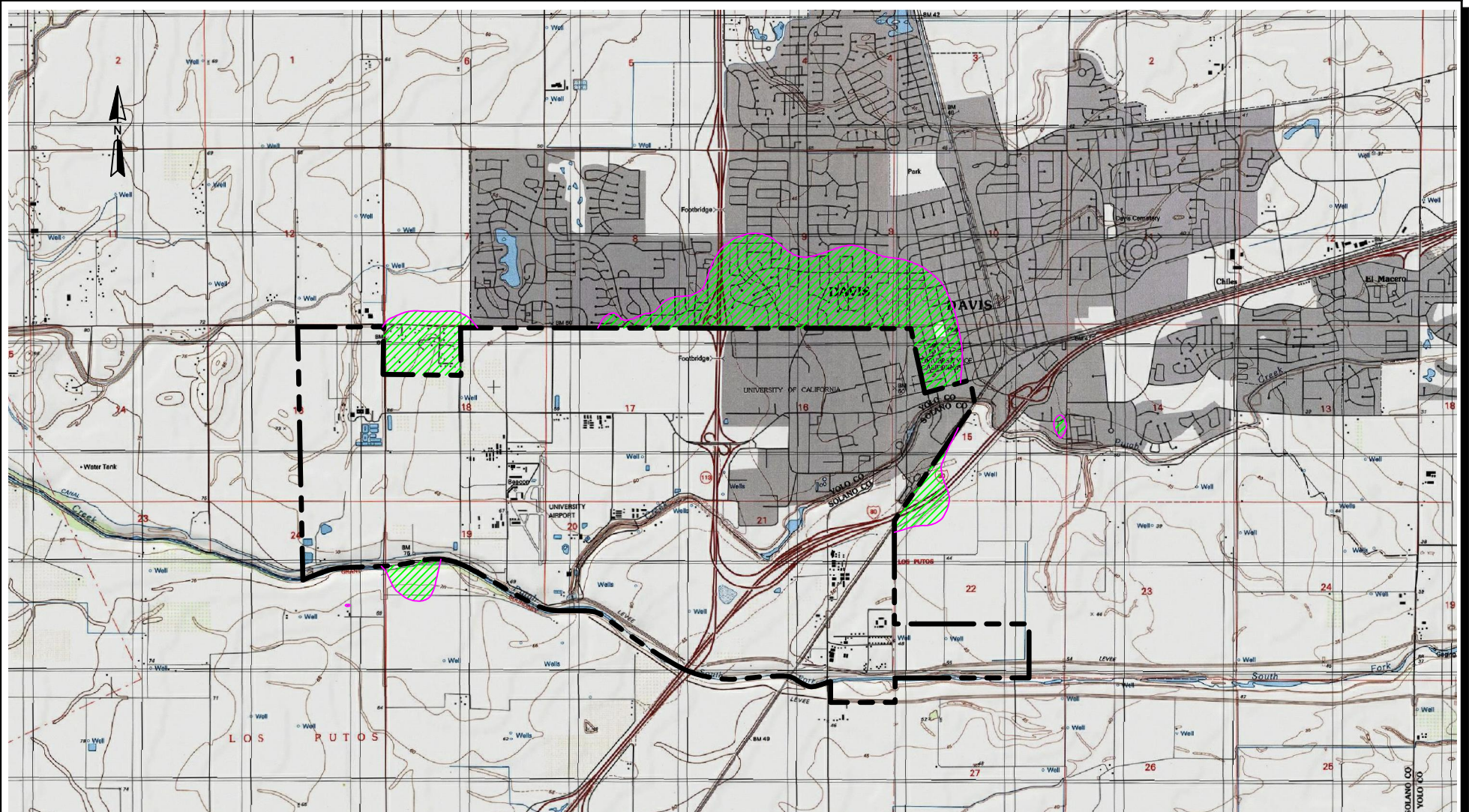


Figure 5
*Off-Site Receptor Grid and
 On-Site Receptor Locations*
 UC Davis
 Davis, California
 ERM 08/10





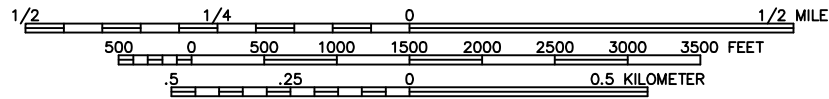
References:
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U.S.G.S. 7.5 Minute Series (Topographic) Quadrangle,
Merritt, California Version: 1992; Current: 1992
Davis, California Version: 1997; Current: 1992

Figure 6
*Location of Maximum Impacts
UC Davis
Davis, California*



LEGEND

-  Zone of Impact
-  UC Davis Property Boundary



References:
TOPO® Software
U.S.G.S. 7.5 Minute Series (Topographic) Quadrangle,
Merritt, California Version: 1992; Current: 1992
Davis, California Version: 1997; Current: 1992

Figure 7
Zone of Impact
(One in One Million Cancer Risk)
UC Davis
Davis, California

Tables

Table 1
Health Risks by Source Category at the Point of Maximum Impact (PMI)
UC Davis
Davis, California

Source Category	PMI - Cancer Risk		PMI - Chronic HI		PMI - Acute HI	
	Cancer Risk	Percent of Total	Chronic HI	Percent of Total	Acute HI	Percent of Total
Laboratories	3.44E-07	15.9%	8.08E-03	86.1%	2.22E-02	19.9%
Central Heating and Cooling Plant	8.86E-09	0.4%	1.7E-04	1.9%	1.2E-02	10.4%
Natural Gas Fired Boilers ¹	6.60E-09	0.3%	7.4E-05	1%	2.2E-03	2.0%
Primate Boiler # 2 (landfill gas combustion)	6.92E-09	0.3%	9.9E-06	0.1%	7.3E-02	65.5%
Heaters and Kilns	6.59E-09	0.3%	9.6E-07	0.01%	4.0E-04	0.4%
Landfill Fugitives and Landfill Flare	1.01E-08	0.5%	9.1E-06	0.1%	1.5E-03	1.3%
Incinerator	3.19E-07	14.8%	2.3E-04	2.5%	1.5E-04	0.1%
Diesel Internal Combustion Engine	1.44E-06	66.8%	7.4E-04	7.9%	0.0E+00	0.0%
Storehouse/Bulk Receiving Operations (solvent dispensing operations)	0.00E+00	0.0%	0.0E+00	0.0%	0.0E+00	0.0%
Chloroform Remediation Operations	1.30E-09	0.06%	0.0E+00	0.0%	0.0E+00	0.0%
Wastewater Treatment Plant	4.06E-09	0.2%	6.2E-05	0.7%	5.7E-05	0.1%
Gasoline Storage and Dispensing	4.77E-09	0.2%	4.0E-07	0.004%	8.8E-07	0.001%
Total Cancer Risk	2.16E-06		9.38E-03		1.12E-01	

¹ This source category includes small boilers less than 5 MMBtu/hr and Primate Center Boiler #1 and #2 during natural gas combustion.

Cancer Risk PMI UTM Coordinate: 608418 E, 4266959 N

Chronic HI PMI UTM Coordinate: 608868 E, 4266958 N

Acute HI PMI UTM Coordinate: 604397 E, 4266549 N

Table 2
Health Risks by Source Category at the Maximum Exposed Individual Resident Location (MEIR)
UC Davis
Davis, California

Source Category	MEIR - Cancer Risk		MEIR - Chronic HI		MEIR - Acute HI	
	Cancer Risk	Percent of Total	Chronic HI	Percent of Total	Acute HI	Percent of Total
Laboratories	3.27E-07	16.2%	7.43E-03	85.7%	2.51E-02	33.3%
Central Heating and Cooling Plant	8.70E-09	0.4%	1.7E-04	1.9%	1.2E-02	16.2%
Natural Gas Fired Boilers ¹	6.43E-09	0.3%	7.0E-05	1%	2.4E-03	3.1%
Primate Boiler # 2 (landfill gas combustion)	7.04E-09	0.3%	9.9E-06	0.1%	3.4E-02	44.5%
Heaters and Kilns	6.17E-09	0.3%	8.9E-07	0.01%	5.8E-04	0.8%
Landfill Fugitives and Landfill Flare	1.08E-08	0.5%	9.3E-06	0.1%	1.2E-03	1.6%
Incinerator	3.09E-07	15.3%	2.2E-04	2.6%	2.4E-04	0.3%
Diesel Internal Combustion Engine	1.34E-06	66.1%	7.0E-04	8.1%	0.0E+00	0.0%
Storehouse/Bulk Receiving Operations (solvent dispensing operations)	0.00E+00	0.0%	0.0E+00	0.0%	0.0E+00	0.0%
Chloroform Remediation Operations	1.27E-09	0.06%	0.0E+00	0.0%	0.0E+00	0.0%
Wastewater Treatment Plant	3.91E-09	0.2%	5.8E-05	0.7%	8.2E-05	0.1%
Gasoline Storage and Dispensing	4.83E-09	0.2%	3.8E-07	0.0%	1.2E-06	0.002%
Total Cancer Risk	2.02E-06		8.67E-03		7.52E-02	

¹ This source category includes small boilers less than 5 MMBtu/hr and Primate Center Boiler #1 and #2 during natural gas combustion.

Cancer Risk MEIR UTM Coordinate: 608400 E, 4267000 N

Chronic HI MEIR UTM Coordinate: 608900 E, 4267000 N

Acute HI MEIR UTM Coordinate: 604900 E, 4266600 N

Table 3
Health Risks by Source Category at the Maximum Exposed Individual Worker Location (MEIW)
UC Davis
Davis, California

Source Category	MEIW - Cancer Risk		MEIW - Chronic HI		MEIW - Acute HI	
	Cancer Risk	Percent of Total	Chronic HI	Percent of Total	Acute HI	Percent of Total
Laboratories	6.14E-08	16.7%	5.36E-03	81.0%	2.26E-02	21.9%
Central Heating and Cooling Plant	1.71E-09	0.5%	1.2E-04	1.9%	1.2E-02	11.5%
Natural Gas Fired Boilers ¹	1.27E-09	0.3%	1.1E-04	2%	2.3E-03	2.2%
Primate Boiler # 2 (landfill gas combustion)	9.33E-10	0.3%	1.1E-05	0.2%	6.4E-02	62.3%
Heaters and Kilns	1.09E-09	0.3%	7.0E-07	0.01%	4.3E-04	0.4%
Landfill Fugitives and Landfill Flare	2.32E-09	0.6%	1.5E-05	0.2%	1.2E-03	1.2%
Incinerator	5.80E-08	15.8%	1.9E-04	2.8%	1.8E-04	0.2%
Diesel Internal Combustion Engine	2.39E-07	65.0%	7.6E-04	11.5%	0.0E+00	0.0%
Storehouse/Bulk Receiving Operations (solvent dispensing operations)	0.00E+00	0.0%	0.0E+00	0.0%	0.0E+00	0.0%
Chloroform Remediation Operations	2.37E-10	0.06%	0.0E+00	0.0%	0.0E+00	0.0%
Wastewater Treatment Plant	6.93E-10	0.2%	5.6E-05	0.8%	6.8E-05	0.1%
Gasoline Storage and Dispensing	1.11E-09	0.3%	5.9E-07	0.01%	1.9E-06	0.002%
Total Cancer Risk	3.68E-07		6.61E-03		1.03E-01	

¹ This source category includes small boilers less than 5 MMBtu/hr and Primate Center Boiler #1 and #2 during natural gas combustion.

Cancer Risk MEIW UTM Coordinate: 608300 E, 4267000 N

Chronic HI MEIW UTM Coordinate: 608300 E, 4267000 N

Acute HI MEIW UTM Coordinate: 604500 E, 4266700 N

Appendix A
2009 AB 2588 HRA Protocol and
District Approval Letter

**Environmental
Resources
Management**

1277 Treat Boulevard
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16 April 2010

Mr. David B. Smith
Yolo-Solano Air Quality Management District
1947 Galileo Court, Suite 103
Davis, CA 95618



Subject: Assembly Bill 2588 Health Risk Assessment Protocol for
Reporting Year 2009 for the University of California at
Davis - One Shields Avenue, Davis, California 95616

Dear Mr. Smith:

ERM-West, Inc. (ERM) is currently under contract with University of California, Davis (UC Davis) to prepare an air toxics health risk assessment (HRA) for compliance with the Air Toxics "Hot Spots" Information and Assessment Act, Assembly Bill 2588 (AB2588). Based on communications between the Yolo Solano Air Quality Management District (YSAQMD) and UC Davis staff, it is understood that an AB2588 HRA will be required for reporting year 2009. This is mainly due to the recent updates to the California Air Resources Board (CARB) Emission Inventory Criteria and Guidelines for the Air Toxics "Hot Spots" Program (effective 26 September 2007; EI Guidelines).¹ The recent EI Guidelines, in part, were developed to better align the AB2588 program with CARB's Stationary Diesel Air Toxics Control Measure (ATCM; section 93115, title 17, California Code of Regulations)² developed to regulate emissions from diesel-fired internal combustion (IC) engines. The inclusion of diesel-fired IC-engines as part of AB2588 Emission Inventory is considered a "significant change in operation" and therefore, requires the facility to prepare an updated HRA for compliance with AB2588.

¹ California Air Resources Board (CARB). 2007. Emissions Inventory Criteria and Guidelines for the Air Toxics Program. September.

² CARB. 2007. Title 17, California Code of Regulations section 93115 3RB. Air Toxics Control Measures (ATCM). October.

EMISSIONS ESTIMATION

The HRA will include emissions from combustion sources, including diesel-fired IC engines, laboratory operations, the landfill, the wastewater treatment plant, and other miscellaneous sources of toxic air contaminants (TACs) located on the main campus. This modeling protocol presents ERM's proposed procedures to assess potential health risk from TAC emissions from the UC Davis main campus.

As mentioned above, the revised guidelines require that previously exempt diesel-fired IC engines be included in future AB2588 HRAs, as they will most likely dominate contributions to cancer risk estimates. ERM has been in contact with CARB regarding the options for completing the HRA. Due to the large number of diesel-fired IC engines located on the UC Davis campus, CARB and ERM concluded that the use of CARB's Screening Level Tables would not be practical. Because ERM has extensive experience using the Hotspots Analysis Reporting Program (HARP) model and is in possession of the 2005 HARP modeling files, the recommended approach is to add the existing diesel-fired IC-engines to the analysis and to utilize the 2005 analysis with a few minor modifications, which include:

- Update emissions from the Central Heating and Cooling Plant (CHCP) boilers based on 2009 operations;
- Update emissions from the Primate Center boilers based on 2009 operations;
- Update emissions from the landfill gas flare based on 2009 operations; and
- Add in source and emissions information for a new boiler located at the CHCP.

It is not proposed to update emissions from the following sources:

- Small heaters and furnaces;
- Kilns;
- Chloroform remediation systems;
- Laboratory fume hoods;

- Storage tanks and fuel dispensing units;
- Storehouse bulk solvent storage;
- Incinerator;
- Wastewater treatment plant; and
- Cooling towers.

The proposed approach is largely based on CARB staff recommendations and the results from the AB2588 HRA prepared for operating year 2005 (2005 HRA).³ The estimated cancer risk calculated at the Point of Maximum Impact (PMI) reported in the 2005 HRA was 1.4 in 1 million. This cancer risk assumes continuous exposure for 70 years, located at a non-residential location. The maximum cancer risk at any residential location was estimated to be 0.8 in 1 million. Based on the low cancer risk estimates in the 2005 HRA, high probability that emissions of diesel particulates (DPM) will drive future cancer risk estimates, and the likelihood that emissions from these sources would significantly change, it is recommended that source information and emission rate information from the 2005 HRA mostly remain unchanged, and that the sources of DPM be added to the existing modeling files.⁴ The dispersion modeling and health risk modeling can then be redone to calculate updated health hazards. This proposed methodology will be the most efficient and cost effective path for the UC Davis Campus to proceed.

DISPERSION MODELING APPROACH

Atmospheric dispersion modeling will be performed to estimate offsite, ground-level concentrations for the pollutants of concern. The analysis will follow methodologies outlined in *Air Toxics Hot Spots Risk Assessment*

³ ERM. 2008. *AB 2588 Air Toxics Health Risk Assessment for Reporting Year 2005 University of California Campus Davis, California*. April.

⁴ ERM. 2009. Telephone conversation between Vicki Hoffman, ERM, and Chris Halm, CARB, regarding methodologies for performing the AB2588 HRA for the UC Davis Main Campus.

Guidelines (Office of Environmental Health Hazard Assessment [OEHHA] 2003),⁵ and will mimic those used in the 2005 HRA.

Model Selection

The terrain within the modeling region (i.e., within 5 kilometers [km] of the project site) can be characterized as simple (i.e., flat terrain in all directions). However, for modeling purposes, elevations will be utilized to incorporate slight differences in elevations. To simulate the dispersion and subsequent ground-level concentrations of the pollutants of concern, HARP will be used. HARP utilizes the Industrial Source Complex Short Term, Version 3 (ISCST3) model for estimating pollutant concentration and dilution. ISCST3 was released by the United States Environmental Protection Agency (USEPA) in 1995. It can estimate pollutant concentrations for both simple and complex terrain. The ISCST3 model is a steady-state, multiple-source, Gaussian dispersion model, which allows for the use of many options to address unique modeling requirements. Technical options selected for the ISCST3 modeling are listed in Table 1. These are referred to as the regulatory default options in the ISCST3 User's Guide (USEPA 1995)⁶.

Table 1 Technical Options for the Proposed ISCST3 Modeling

Option	ISCST3
Final Plume Rise	Yes
Stack-Tip Downwash	Yes
Buoyancy-Induced Dispersion	Yes
Calms Processing Routine	Yes
Dispersion Coefficients	Rural

⁵ Office of Environmental Health Hazard Assessment. 2003. "Air Toxics Hot Spots Program Risk Assessment Guidelines." *The Air Quality Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments*. August.

⁶ USEPA. 1995. User's Guide for the Industrial Source Complex (ISC), Volume I, User Instructions. September.

Option	ISCST3
Default Wind Profile Exponents	Yes
Default Vertical Potential Temperature Gradient	Yes
Calculate Concentrations for both Simple and Complex Terrain	Yes

Meteorological Data

The ISCST3 model requires input of hourly meteorological data consisting of wind speed, wind direction, temperature, atmospheric stability, and mixing height. There are no data available in the immediate vicinity of the UC Davis Campus. It is proposed that 5 years of meteorological data (1985 - 1989) collected at Sacramento Executive Airport, located approximately 12 miles to the northeast, be used. This is the same data set used for modeling in the 2005 HRA. The data, received from the Sacramento Metropolitan Air Quality Management District (SMAQMD), are the most recent 5-year period available that are compatible with the ISCST3 model. The data include surface meteorological data collected at Sacramento Executive Airport combined with upper air data obtained from Oakland International Airport.

Receptor Grid

It is proposed that the same receptor grid be used as was developed for the 2005 HRA. The grid contains receptor locations at 50-meter increments along the UC Davis property boundary. Additional receptors area located at 100-meter increments to a distance of 500 meters, and at 250-meter increments to a distance of approximately 1 km. Discrete and/or sensitive receptors will also be identified for nearby locations, including schools, hospitals, daycare facilities, and convalescent centers. Sensitive receptors and their locations will be based on an updated Offsite Receptor Report to be obtained from Environmental Data Resources, Inc. Receptors will also be placed at on-campus locations, including dormitories, day care centers, gathering areas, and at on-campus worker locations. At this time, it is not anticipated that cancer burden calculations will be required. Receptor locations will be identified using Universal Transverse Mercator (UTM) Coordinates, and receptor

elevations will be obtained from digital elevation models (DEMs) digitized from United States Geological Survey (USGS) maps.

Source Parameters

Except for the added diesel-fired IC engines and the new CHCP boiler, stack locations will be identical to those used in the 2005 HRA. The majority of emission sources will be modeled as point sources, including the diesel-fired IC engines and the new CHCP boiler. Point source modeling parameters will include stack location, stack base elevation, stack height, stack internal diameter, stack gas exit velocity, stack gas exit temperature, and emission rates. Emissions from fugitive landfill gas, and other potential fugitive sources, such as some wastewater treatment plant operations and fugitive fuel vapor sources, will be modeled using area sources. Area source parameters include source length, width, release height, and emission rates.

Aerodynamic Downwash

Evaluation of building downwash on adjacent stack sources is deemed necessary, because most, if not all, of the stack source heights may be below Good Engineering Practice (GEP) heights. The formula for GEP height estimation is:

$$H_s = H_b + 1.50L_b$$

where: H_s = GEP stack height
 H_b = building height
 L_b = the lesser building dimension of the height, length, or width

The effects of aerodynamic downwash due to buildings and other structures will be accounted for by using wind direction-specific building parameters calculated by the USEPA-approved Building Parameter Input Program PRIME (BPIPRIME), and the algorithms included in the ISCST3 air dispersion model. Based on examination of plot plans for the relationship of sources to the location of facility structures, the locations and dimensions of emission sources, and facility structures will be entered into the BPIP software package that calculates the direction-

specific building dimensions for input into the ISCST3 model. A downwash analysis will be performed for each point source.

Health Risk Calculations

The HRA will present estimated offsite risk from emissions of TACs. Risk will be calculated for both short term (acute health effects) and long term (chronic health effects and cancer risk) exposures. To calculate health risk from the facility, ERM will use the HARP model, which includes the algorithms and methodologies outlined in the *Air Toxics Hot Spots Program Risk Assessment Guidelines* (OEHHA 2003).⁷ Since TAC emissions will include particulate matter, the following exposure pathways will be evaluated: inhalation; dermal; incidental ingestion; mother's milk; and locally grown produce. A Tier 1 analysis is proposed and is consistent with the CARB Interim Risk Management Policy (CARB 2003);⁸ maximum cancer risk will be calculated with the derived (adjusted) method.

REPORT FORMAT

The report will present methodologies used to calculate emissions and perform the UC Davis Campus AB2588 HRA. The HRA report will include emission estimates and results from atmospheric dispersion modeling and risk associated with the UC Davis main campus. Chemical emissions, including Chemical Abstracts Service (CAS) number, will be presented for both annual average (pounds per year) and maximum hourly (pounds per hour) emissions. Source parameters, emission rates, and calculated cancer risk and hazard indices for the points of maximum impact (PMI), maximum exposed individual at a residential receptor (MEIR), the maximum exposed individual at a worker (occupational) receptor (MEIW), and sensitive receptors will be provided in tabular form (with UTM Coordinates).

⁷ OEHHA. 2003. *Air Toxics Hot Spots Program Risk Assessment Guidelines*. The Air Quality Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments. August.

⁸ CARB. 2003. Air Resources Board Recommended Interim Risk Management Policy for Inhalation-Based Residential Cancer Risk.

Scaled maps will be provided showing facility location, emission sources, property boundary, and locations of modeled receptors. Maps will summarize the HRA results and illustrate the PMI locations and sensitive receptors. Isopleths will be provided summarizing cancer risk (greater than 1 in 1 million), the chronic hazard index (for each endpoint greater than 1.0), and the acute hazard index (for each endpoint greater than 1.0). Modeling input and output files will be included in electronic format.

If you have any questions or comments regarding the methodologies outlined in this protocol, please do not hesitate to call me at (925) 482-3236.

Sincerely,

A handwritten signature in black ink, appearing to read "Vicki J. Hoffman". The signature is fluid and cursive, with the first name "Vicki" being the most prominent.

Vicki J. Hoffman
Senior Air Quality Scientist

VJH/kl/0113170

cc: Aimee Pfohl, UC Davis



April 23, 2010

Ms. Vicki J. Hoffman
Senior Air Quality Scientist
Environmental Resources Management
1277 Treat Boulevard, Suite 500
Walnut Creek, CA 94596

RE: Health Risk Assessment Protocol (Protocol), University of California at Davis (Facility)
Facility Location: University of California at Davis Campus

This letter is to inform you that the District has received and approves the Protocol. The District has found the Protocol to be a suitable method for quantifying the potential risks of the Facility pursuant to the Air Toxics "Hot Spots" Information and Assessment Act of 1987.

When the Health Risk Assessment (HRA) is completed; it should be sent to the District. It will be reviewed by the District and the Office of Environmental Health Hazard Assessment.. The due date for the HRA is August 1, 2010.

If you have any questions, please feel free to contact me at (530)757-3662.

Sincerely,

David B. Smith
Supervising Air Quality Specialist

cc: Aimee Pfohl, UC Davis - Environmental Health and Safety

Appendix B
Chemicals Included in the Health
Risk Assessment

Appendix B
UC Davis AB2588 Health Risk Assessment
Pollutants Included in the Health Risk Assessment

No.	CAS No.	Chemical Name	Abbreviation	Cancer Potency	Cancer Potency	Chronic REL	Chronic REL	Acute REL
				Factor (Inhalation)	Factor (Oral)	(Inhalation)	(Oral)	
				ug/m ³	mg/kg-d	ug/m ³		
1	9901	Diesel Particulate Matter (DPM)	DieselExhPM	1.10E+00	*	5.00E+00	*	*
2	50000	Formaldehyde	Formaldehyde	2.10E-02	*	9.00E+00	*	5.50E+01
3	50328	Benzo[a]pyrene	B[a]P	3.90E+00	1.20E+01	*	*	*
4	53703	Dibenz[a,h]anthracene	D[a,h]anthracen	4.10E+00	4.10E+00	*	*	*
5	56235	Carbon tetrachloride	CCl4	1.50E-01	*	4.00E+01	*	1.90E+03
6	56553	Benz[a]anthracene	B[a]anthracene	3.90E-01	1.20E+00	*	*	*
7	67561	Methanol	Methanol	*	*	4.00E+03	*	2.80E+04
8	67630	Isopropyl alcohol	Isopropyl Alcoh	*	*	7.00E+03	*	3.20E+03
9	67663	Chloroform	Chloroform	1.90E-02	*	3.00E+02	*	1.50E+02
10	68122	Dimethyl formamide	DMF	*	*	8.00E+01	*	*
11	71432	Benzene	Benzene	1.00E-01	*	6.00E+01	*	1.30E+03
12	71556	Methyl chloroform (1,1,1-Trichloroethane)	1,1,1-TCA	*	*	1.00E+03	*	6.80E+04
13	75003	Ethyl chloride	Ethyl Chloride	*	*	3.00E+04	*	*
14	75014	Vinyl chloride	Vinyl Chloride	2.70E-01	*	*	*	1.80E+05
15	75070	Acetaldehyde	Acetaldehyde	1.00E-02	*	1.40E+02	*	4.70E+02
16	75092	Methylene chloride	Methylene Chlor	3.50E-03	*	4.00E+02	*	1.40E+04
17	75150	Carbon disulfide	CS2	*	*	8.00E+02	*	6.20E+03
18	75343	1,1-Dichloroethane	1,1-DiClEthane	5.70E-03	*	*	*	*
19	75354	Vinylidene chloride	Vinylid Chlorid	*	*	7.00E+01	*	*
20	78875	1,2-Dichloropropane	1,2-DiClPropane	6.30E-02	*	*	*	*
21	78933	Methyl ethyl ketone	MEK	*	*	*	*	1.30E+04
22	79016	Trichloroethylene	TCE	7.00E-03	*	6.00E+02	*	*
23	79061	Acrylamide	Acrylamide	4.50E+00	*	*	*	*
24	79345	1,1,2,2-Tetrachloroethane	TetraClEthane	2.00E-01	*	*	*	*
25	91203	Naphthalene	Naphthalene	1.20E-01	*	9.00E+00	*	*
26	100414	Ethyl benzene	Ethyl Benzene	8.70E-03	*	2.00E+03	*	*
27	106467	p-Dichlorobenzene	p-DiClBenzene	4.00E-02	*	8.00E+02	*	*
28	106934	Ethylene dibromide (EDB)	EDB	2.50E-01	*	8.00E-01	*	*
29	106990	1,3-Butadiene	1,3-Butadiene	6.00E-01	*	2.00E+01	*	*
30	107028	Acrolein	Acrolein	*	*	3.50E-01	*	2.50E+00
31	107062	Ethylene dichloride (EDC)	EDC	7.20E-02	*	4.00E+02	*	*
32	107131	Acrylonitrile	Acrylonitrile	1.00E+00	*	5.00E+00	*	*
33	108883	Toluene	Toluene	*	*	3.00E+02	*	3.70E+04
34	108907	Chlorobenzene	Chlorobenzn	*	*	1.00E+03	*	*
35	110543	Hexane	Hexane	*	*	7.00E+03	*	*
36	111308	Glutaraldehyde	Glutaraldehyd	*	*	8.00E-02	*	*

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No.	CAS No.	Chemical Name	Abbreviation	Cancer Potency	Cancer Potency	Chronic REL	Chronic REL	Acute REL
				Factor (Inhalation)	Factor (Oral)	(Inhalation)	(Oral)	
				ug/m ³	mg/kg-d	ug/m ³		
37	115071	Propylene	Propylene	*	*	3.00E+03	*	*
38	121448	Triethylamine	Triethylamine	*	*	2.00E+02	*	2.80E+03
39	123911	1,4-Dioxane	1,4-Dioxane	2.70E-02	*	3.00E+03	*	3.00E+03
40	127184	Perchloroethylene (Tetrachloroethene)	Perc	2.10E-02	*	3.50E+01	*	2.00E+04
41	193395	Indeno[1,2,3-cd]pyrene	In[1,2,3-cd]pyr	3.90E-01	1.20E+00	*	*	*
42	205992	Benzo[b]fluoranthene	B[b]fluoranthen	3.90E-01	1.20E+00	*	*	*
43	207089	Benzo[k]fluoranthene	B[k]fluoranthen	3.90E-01	1.20E+00	*	*	*
44	218019	Chrysene	Chrysene	3.90E-02	1.20E-01	*	*	*
45	302012	Hydrazine	Hydrazine	1.70E+01	*	2.00E-01	*	*
46	1330207	Xylenes (mixed)	Xylenes	*	*	7.00E+02	*	2.20E+04
47	1746016	2,3,7,8-Tetrachlorodibenzo-p-dioxin	2,3,7,8-TCDD	1.30E+05	1.30E+05	4.00E-05	1.00E-08	*
49	3268879	1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin	1-8OctaCDD	1.30E+01	1.30E+01	4.00E-01	1.00E-04	*
48	7439921	Lead	Lead	4.20E-02	8.50E-03	*	*	*
50	7439976	Mercury	Mercury	*	*	3.00E-02	1.60E-04	6.00E-01
51	7647010	Hydrochloric acid	HCl	*	*	9.00E+00	*	2.10E+03
52	7664393	Hydrogen fluoride	HF	*	*	1.40E+01	4.00E-02	2.40E+02
53	7697372	Nitric acid	Nitric Acid	*	*	*	*	8.60E+01
54	7783064	Hydrogen sulfide	H2S	*	*	1.00E+01	*	4.20E+01
55	7803512	Phosphine	Phosphine	*	*	8.00E-01	*	*
56	18540299	Chromium (hexavalent)	Cr(VI)	5.10E+02	*	2.00E-01	2.00E-02	*
57	19408743	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin	1-3,7-9HxCDD	1.30E+04	1.30E+04	4.00E-04	1.00E-07	*
58	35822469	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin	1-4,6-8HpCDD	1.30E+03	1.30E+03	4.00E-03	1.00E-06	*
59	39001020	1,2,3,4,6,7,8,9-Octachlorodibenzofuran	1-8OctaCDF	1.30E+01	1.30E+01	4.00E-01	1.00E-04	*
60	39227286	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin	1-4,7,8HxCDD	1.30E+04	1.30E+04	4.00E-04	1.00E-07	*
61	40321764	1,2,3,7,8-Pentachlorodibenzo-p-dioxin	1-3,7,8PeCDD	1.30E+05	1.30E+05	4.00E-05	1.00E-08	*
62	51207319	2,3,7,8-Tetrachlorodibenzofuran	2,3,7,8-TCDF	1.30E+04	1.30E+04	4.00E-04	1.00E-07	*
63	55673897	1,2,3,4,7,8,9-Heptachlorodibenzofuran	1-4,7-9HpCDF	1.30E+03	1.30E+03	4.00E-03	1.00E-06	*
64	57117314	2,3,4,7,8-Pentachlorodibenzofuran	2-4,7,8PeCDF	6.50E+04	6.50E+04	8.00E-05	2.00E-08	*
65	57117416	1,2,3,7,8-Pentachlorodibenzofuran	1-3,7,8PeCDF	6.50E+03	6.50E+03	8.00E-04	2.00E-07	*
66	57117449	1,2,3,6,7,8-Hexachlorodibenzofuran	1-3,6-8HxCDF	1.30E+04	1.30E+04	4.00E-04	1.00E-07	*
67	57653857	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin	1-3,6-8HxCDD	1.30E+04	1.30E+04	4.00E-04	1.00E-07	*
68	60851345	2,3,4,6,7,8-Hexachlorodibenzofuran	2-4,6-8HxCDF	1.30E+04	1.30E+04	4.00E-04	1.00E-07	*
69	67562394	1,2,3,4,6,7,8-Heptachlorodibenzofuran	1-4,6-8HpCDF	1.30E+03	1.30E+03	4.00E-03	1.00E-06	*
70	70648269	1,2,3,4,7,8-Hexachlorodibenzofuran	1-4,7,8HxCDF	1.30E+04	1.30E+04	4.00E-04	1.00E-07	*
71	72918219	1,2,3,7,8,9-Hexachlorodibenzofuran	1-3,7-9HxCDF	1.30E+04	1.30E+04	4.00E-04	1.00E-07	*

Appendix C
Emissions Estimates

Facility Wide Toxic Air Contaminant Emissions

CAS	Chemical	Hourly Emissions	Annual Emissions
		(lb/hr)	(lb/yr)
9901	Diesel Particulate Matter	25.62	245.21
50000	Formaldehyde	1.55	865.05
50328	Benzo[a]pyrene	0.00055	0.16
53703	Dibenz[a,h]anthracene	0.00055	0.15
56235	Carbon tetrachloride	0.019	32.59
56553	Benz[a]anthracene	0.00060	0.19
64175	Ethanol ¹	0.010	88.29
67561	Methanol	3.25	5535.17
67630	Isopropyl alcohol	0.78	1493.27
67641	Acetone ¹	0.0033	28.68
67663	Chloroform	0.40	725.74
68122	Dimethyl formamide	0.012	20.22
71432	Benzene	0.024	54.94
71556	Methyl chloroform (1,1,1-Trichloroethane)	0.0025	21.30
74840	Ethane, no health values ¹	0.22	1882.98
74873	Methyl chloride (Chloromethane) ¹	0.00051	4.31
74931	Methyl mercaptan, no health values ¹	0.00098	8.44
74986	Propane, no health values ¹	0.0040	34.47
75003	Ethyl chloride (Chloroethane)	0.00067	5.68
75014	Vinyl chloride	0.0038	32.33
75070	Acetaldehyde	0.013	15.87
75081	Ethyl mercaptan (ethanethiol) ¹	0.0012	9.98
75092	Methylene chloride (Dichloromethane)	0.66	1224.78
75150	Carbon disulfide	0.00036	3.11
75183	Dimethyl sulfide (methyl sulfide) ¹	0.0040	34.22
75274	Bromodichloromethane ¹	0.0043	36.15
75343	1,1-Dichloroethane	0.0019	16.39
75354	Vinylidene chloride	0.00016	1.37
75434	Dichlorofluoromethane (Freon 21) ¹	0.0022	19.00
75456	Chlorodifluoromethane (Freon 22) ¹	0.00073	6.18
75694	Trichlorofluoromethane (Freon 11) ¹	0.00087	7.36
75718	Dichlorodifluoromethane (Freon 12) ¹	0.016	133.78
78875	1,2-Dichloropropane	0.00017	1.43
78933	Methyl ethyl ketone (2-Butanone)	0.0058	50.64
79016	Trichloroethylene	0.0053	29.93
79061	Acrylamide	0.0018	3.10
79345	1,1,2,2-Tetrachloroethane	0.0015	13.13
83329	Acenaphthene ¹	0.00029	0.015
85018	Phenanthrene ¹	0.00087	0.37
86737	Fluorene ¹	0.00031	0.15
91203	Naphthalene	0.81	99.08
91576	2-Methyl naphthalene ¹	0.00018	0.0024
91587	2-Chloronaphthalene ¹	0.000024	0.00032
100414	Ethyl benzene	0.0060	34.57
100527	Benzaldehyde ¹	0.0095	20.28
106467	p-Dichlorobenzene	0.00026	2.18
106934	Ethylene dibromide (EDB)	0.0000016	0.013
106978	Butane ¹	0.0024	20.59
106990	1,3-Butadiene	0.0043	7.21
107028	Acrolein	0.0022	0.66
107062	Ethylene dichloride (EDC)	0.012	21.97
107131	Acrylonitrile	0.0030	24.01
108101	Methyl isobutyl ketone (Hexone) ¹	0.0030	26.50

Facility Wide Toxic Air Contaminant Emissions

CAS	Chemical	Hourly Emissions	Annual Emissions
		(lb/hr)	(lb/yr)
108883	Toluene	0.20	1300.20
108907	Chlorobenzene	0.00023	1.98
109660	Pentane ¹	0.0019	16.72
110543	Hexane	0.024	94.20
110827	Cyclohexane ¹	0.00040	3.49
111308	Glutaraldehyde	0.0073	12.28
115071	Propylene	0.0036	2.95
120127	Anthracene ¹	0.00015	0.11
121448	Triethylamine	0.0075	12.61
123911	1,4-Dioxane	0.021	34.85
127184	Perchloroethylene (Tetrachloroethene)	0.0092	73.90
129000	Pyrene ¹	0.00024	0.18
156605	t-1,2-Dichloroethene ¹	0.0023	19.40
191242	Benzo[g,h,i]perylene ¹	0.000030	0.0079
192972	Benzo[e]pyrene ¹	0.000018	0.00024
193395	Indeno[1,2,3-cd]pyrene	0.00055	0.16
198550	Perylene ¹	0.000035	0.00047
205992	Benzo[b]fluoranthene	0.00057	0.17
206440	Fluoranthene ¹	0.00025	0.20
207089	Benzo[k]fluoranthene	0.00067	0.18
208968	Acenaphthylene ¹	0.00041	0.31
218019	Chrysene	0.000075	0.055
302012	Hydrazine	0.00026	0.44
463581	Carbonyl sulfide ¹	0.00024	2.07
540841	2,2,4-Trimethylpentane ¹	0.00078	6.81
1330207	Xylenes (mixed)	0.19	1284.57
1746016	2,3,7,8-Tetrachlorodibenzo-p-dioxin	0.000000019	0.0000064
3268879	1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin	0.00000061	0.000046
7439921	Lead	0.00037	0.059
7439976	Mercury	0.0000043	0.0052
7647010	Hydrochloric acid	0.73	1059.22
7664393	Hydrogen fluoride	0.055	24.50
7697372	Nitric acid	0.37	620.39
7783064	Hydrogen sulfide	0.011	93.36
7803512	Phosphine	0.000016	0.028
10035106	Hydrogen bromide ¹	0.016	4.57
18540299	Chromium, hexavalent (& compounds)	0.0000089	0.0015
19408743	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin	0.000000026	0.000024
35822469	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin	0.00000010	0.000072
39001020	1,2,3,4,6,7,8,9-Octachlorodibenzofuran	0.000000076	0.000049
39227286	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin	0.000000016	0.000015
40321764	1,2,3,7,8-Pentachlorodibenzo-p-dioxin	0.000000027	0.000014
51207319	2,3,7,8-Tetrachlorodibenzofuran	0.000000043	0.000029
55673897	1,2,3,4,7,8,9-Heptachlorodibenzofuran	0.000000018	0.000017
57117314	2,3,4,7,8-Pentachlorodibenzofuran	0.000000050	0.000047
57117416	1,2,3,7,8-Pentachlorodibenzofuran	0.000000045	0.000031
57117449	1,2,3,6,7,8-Hexachlorodibenzofuran	0.000000046	0.000032
57653857	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin	0.000000054	0.000040
60851345	2,3,4,6,7,8-Hexachlorodibenzofuran	0.0000000061	0.0000057
67562394	1,2,3,4,6,7,8-Heptachlorodibenzofuran	0.00000027	0.00023
70648269	1,2,3,4,7,8-Hexachlorodibenzofuran	0.000000076	0.000072
72918219	1,2,3,7,8,9-Hexachlorodibenzofuran	0.0000000039	0.0000037

¹ There are no toxicity values for this chemical. Therefore, no further analysis was performed.

Toxic Air Contaminant Emissions from Large Boilers - Natural Gas Combustion

Source ID	CHCPBLR1	CHCPBLR2	CHCPBLR3	NEWBLRNG	PRIMBLR1	PCBLR2NG		
Annual Emissions from Natural Gas Fired Boilers								
	CHCP Boiler #1	CHCP Boiler #2	CHCP Boiler #3	CHCP Boiler #4	PC Boiler #1	PC Boiler #2	Total	
Natural Gas Throughput (MMscf/yr)¹	245.70	387.42	45.90	239.95	14.20	14.40	947.57	
Substance	Emissions Factor (lbs/MMscf)	Emissions (lb/yr)						
Acetaldehyde	8.87E-03	2.18E+00	3.44E+00	4.07E-01	2.13E+00	1.26E-01	1.28E-01	8.40E+00
Benzaldehyde ²	1.64E-02	4.03E+00	6.35E+00	7.53E-01	3.94E+00	2.33E-01	2.36E-01	1.55E+01
Benzene	4.31E-03	1.06E+00	1.67E+00	1.98E-01	1.03E+00	6.12E-02	6.21E-02	4.08E+00
Formaldehyde	2.21E-01	5.43E+01	8.56E+01	1.01E+01	5.30E+01	3.14E+00	3.18E+00	2.09E+02
NG Heating Value =		1020	Btu/scf					

Maximum Hourly Emissions from Natural Gas Fired Boilers								
	CHCP Boiler #1	CHCP Boiler #2	CHCP Boiler #3	CHCP Boiler #4	PC Boiler #1	PC Boiler #2	Total	
Max Natural Gas Throughput (MMBtu/hr)¹	126.1	126.1	90.3	180	12.51	12.6	547.61	
Substance	Emissions Factor (lbs/MMscf)	Emissions (lb/hr)						
Acetaldehyde	8.87E-03	1.10E-03	1.10E-03	7.85E-04	1.57E-03	1.09E-04	1.10E-04	4.76E-03
Benzaldehyde ²	1.64E-02	2.03E-03	2.03E-03	1.45E-03	2.89E-03	2.01E-04	2.03E-04	8.80E-03
Benzene	4.31E-03	5.33E-04	5.33E-04	3.82E-04	7.61E-04	5.29E-05	5.32E-05	2.31E-03
Formaldehyde	2.21E-01	2.73E-02	2.73E-02	1.96E-02	3.90E-02	2.71E-03	2.73E-03	1.19E-01

¹ Emission factors were obtained from the CARB CATEF database.

² There are no toxicity factors for this chemical. Therefore, no further analysis has been performed.

Sample Calculation:

Emissions (lbs/yr) = EF (lb/MMscf) * Natural Gas Throughput (MMscf/yr)

Emissions (lbs/hr) = EF (lb/MMscf) * Max Natural Gas Throughput (MMBtu/hr) / NG Heating Value (Btu/scf)

NG Heating Value = 1020

**Toxic Air Contaminant Emissions from CHCP Boiler #4 - Diesel
Combustion - Annual**

Substance	Emissions Factor (lbs/Mgal) ¹	Annual Emissions (lb/yr)
Diesel Throughput (Mgal/yr)		17.33
2-Chloronaphthalene ²	1.84E-05	3.19E-04
2-Methylnaphthalene ²	1.40E-04	2.43E-03
Acenaphthene ²	2.11E-04	3.66E-03
Acenaphthylene ²	6.50E-05	1.13E-03
Anthracene ²	2.39E-05	4.14E-04
Benzene	2.54E-03	4.40E-02
Benzo(a)anthracene	1.35E-05	2.34E-04
Benzo(a)pyrene	7.55E-06	1.31E-04
Benzo(b)fluoranthene	6.67E-06	1.16E-04
Benzo(e)pyrene ²	1.40E-05	2.43E-04
Benzo(g,h,i)perylene ²	8.50E-06	1.47E-04
Benzo(k)fluoranthene	8.31E-05	1.44E-03
Chrysene	1.28E-05	2.22E-04
Dibenz(a,h)anthracene	6.49E-06	1.12E-04
Ethylbenzene	1.49E-03	2.58E-02
Fluoranthene ²	3.32E-05	5.75E-04
Fluorene ²	1.17E-04	2.03E-03
Formaldehyde	3.49E-01	6.05E+00
Hexane	1.21E-03	2.10E-02
Indeno(1,2,3-cd)pyrene	6.64E-06	1.15E-04
Naphthalene	3.67E-01	6.36E+00
Perylene ²	2.71E-05	4.70E-04
Phenanthrene ²	3.72E-04	6.45E-03
Propylene	1.71E-03	2.96E-02
Pyrene ²	4.08E-05	7.07E-04
Toluene	1.50E-03	2.60E-02
Xylene (Total)	1.49E-03	2.58E-02
Benzo(g,h,i)perylene ²	8.50E-06	1.47E-04

¹ Emission factors were obtained from the CARB CATEF database.

² There are no toxicity factors for this chemical. Therefore, no further analysis has been completed.

Sample Calculation:

Emissions (lbs/yr) = EF (lb/Mgal) * Diesel Throughput (Mgal/yr).

**Toxic Air Contaminant Emissions from CHCP Boiler #4 - Diesel
Combustion - Hourly**

Substance	Emissions Factor (lbs/Mgal) ¹	Hourly Emissions (lb/hr)
Max Diesel Heat Rating (MMBtu/hr)		180
2-Chloronaphthalene ²	1.84E-05	2.39E-05
2-Methylnaphthalene ²	1.40E-04	1.82E-04
Acenaphthene ²	2.11E-04	2.74E-04
Acenaphthylene ²	6.50E-05	8.45E-05
Anthracene ²	2.39E-05	3.11E-05
Benzene	2.54E-03	3.30E-03
Benzo(a)anthracene	1.35E-05	1.75E-05
Benzo(a)pyrene	7.55E-06	9.81E-06
Benzo(b)fluoranthene	6.67E-06	8.67E-06
Benzo(e)pyrene ²	1.40E-05	1.82E-05
Benzo(g,h,i)perylene ²	8.50E-06	1.10E-05
Benzo(k)fluoranthene	8.31E-05	1.08E-04
Chrysene	1.28E-05	1.66E-05
Dibenz(a,h)anthracene	6.49E-06	8.44E-06
Ethylbenzene	1.49E-03	1.94E-03
Fluoranthene ²	3.32E-05	4.32E-05
Fluorene ²	1.17E-04	1.52E-04
Formaldehyde	3.49E-01	4.54E-01
Hexane	1.21E-03	1.57E-03
Indeno(1,2,3-cd)pyrene	6.64E-06	8.63E-06
Naphthalene	3.67E-01	4.77E-01
Perylene ²	2.71E-05	3.52E-05
Phenanthrene ²	3.72E-04	4.84E-04
Propylene	1.71E-03	2.22E-03
Pyrene ²	4.08E-05	5.30E-05
Toluene	1.50E-03	1.95E-03
Xylene (Total)	1.49E-03	1.94E-03
Benzo(g,h,i)perylene ²	8.50E-06	1.10E-05

Notes:

¹ Emission factors were obtained from the CARB CATEF database.

² There are no toxicity factors for this chemical. Therefore, no further analysis has been completed.

Sample Calculation:

Emissions (lbs/hr) = EF (lb/Mgal) * Max Natural Gas Throughput (MMBtu/hr) / Diesel Heating Value (Btu/gal) * 1000.

Low Sulfur Diesel Heating Value = 138,490 Btu/gal

Low Sulfur Diesel Heating Value from

http://www.transportation.anl.gov/modeling_simulation/GREET/index.html.

Incinerator Emissions

Annual Emissions for Incinerator Firing							
Substance	Firing of Non-Infectious Waste with Natural Gas		Firing of Infectious Waste with Natural Gas		Firing Natural Gas		Total
	Emissions Factor (lbs/ton) ³	Emissions (lb/yr)	Emissions Factor (lbs/ton) ³	Emissions (lb/yr)	Emissions Factor (lbs/MMscf) ³	Emissions (lb/yr)	Emissions (lb/yr)
Throughput (ton/yr or MMscf/yr)	31.535		1.1		4.462		
Acenaphthene ⁴	-	-	1.07E-02	1.18E-02	-	-	1.18E-02
Acenaphthylene ⁴	-	-	2.78E-01	3.06E-01	-	-	3.06E-01
Acetaldehyde	-	-	-	-	8.87E-03	3.96E-02	3.96E-02
Anthracene ⁴	-	-	9.74E-02	1.07E-01	-	-	1.07E-01
Benzaldehyde ⁴	-	-	-	-	1.64E-02	7.32E-02	7.32E-02
Benzene	-	-	-	-	4.31E-03	1.92E-02	1.92E-02
Benzo(a)anthracene	-	-	3.56E-02	3.92E-02	-	-	3.92E-02
Benzo(a)pyrene	-	-	6.26E-03	6.89E-03	-	-	6.89E-03
Benzo(b)fluoranthene	-	-	1.90E-02	2.09E-02	-	-	2.09E-02
Benzo(g,h,i)perylene ⁴	-	-	6.91E-03	7.60E-03	-	-	7.60E-03
Benzo(k)fluoranthene	-	-	2.61E-02	2.87E-02	-	-	2.87E-02
Chromium (Hex)	-	-	1.29E-04	1.42E-04	-	-	1.42E-04
Chrysene	-	-	4.99E-02	5.49E-02	-	-	5.49E-02
Dibenz(a,h)anthracene	-	-	3.00E-03	3.30E-03	-	-	3.30E-03
Dioxin:4D 2378	1.16E-09	3.66E-08	1.50E-06	1.65E-06	-	-	1.69E-06
Dioxin:5D 12378	4.64E-09	1.46E-07	8.53E-06	9.38E-06	-	-	9.53E-06
Dioxin:6D 123478	2.48E-09	7.82E-08	1.36E-05	1.50E-05	-	-	1.50E-05
Dioxin:6D 123678	4.17E-09	1.32E-07	3.19E-05	3.51E-05	-	-	3.52E-05
Dioxin:6D 123789	3.47E-09	1.09E-07	2.20E-05	2.42E-05	-	-	2.43E-05
Dioxin:7D 1234678	1.17E-08	3.69E-07	5.69E-05	6.26E-05	-	-	6.30E-05
Dioxin:8D	1.72E-08	5.42E-07	3.25E-05	3.58E-05	-	-	3.63E-05
Fluoranthene ⁴	-	-	1.77E-01	1.95E-01	-	-	1.95E-01
Fluorene ⁴	-	-	1.37E-01	1.51E-01	-	-	1.51E-01
Formaldehyde	-	-	-	-	2.21E-01	9.86E-01	9.86E-01
Furan:4F 2378	3.56E-09	1.12E-07	2.21E-05	2.43E-05	-	-	2.44E-05
Furan:5F 12378	8.01E-09	2.53E-07	2.39E-05	2.63E-05	-	-	2.65E-05
Furan:5F 23478	1.33E-08	4.19E-07	4.25E-05	4.68E-05	-	-	4.72E-05
Furan:6F 123478	7.32E-09	2.31E-07	6.48E-05	7.13E-05	-	-	7.15E-05
Furan:6F 123678	7.62E-09	2.40E-07	2.45E-05	2.70E-05	-	-	2.72E-05
Furan:6F 123789	3.38E-09	1.07E-07	2.38E-07	2.62E-07	-	-	3.68E-07
Furan:6F 234678	9.81E-09	3.09E-07	2.38E-07	2.62E-07	-	-	5.71E-07
Furan:7F 1234678	1.52E-08	4.79E-07	1.98E-04	2.18E-04	-	-	2.18E-04
Furan:7F 1234789	4.41E-09	1.39E-07	1.53E-05	1.68E-05	-	-	1.70E-05
Furan:8F	7.84E-09	2.47E-07	3.60E-05	3.96E-05	-	-	3.98E-05
HCl	-	-	4.29E+01	4.72E+01	-	-	4.72E+01
Indeno(1,2,3-cd)pyrene	-	-	5.69E-03	6.26E-03	-	-	6.26E-03
Naphthalene	-	-	3.84E-01	4.22E-01	-	-	4.22E-01
Phenanthrene ⁴	-	-	3.29E-01	3.62E-01	-	-	3.62E-01
Pyrene ⁴	-	-	1.61E-01	1.77E-01	-	-	1.77E-01

Maximum Hourly Emissions for Incinerator Firing							
Substance	Firing of Non-Infectious Waste with Natural Gas ¹		Firing of Infectious Waste with Natural Gas ¹		Firing Natural Gas ^{2,3}		Total
	Emissions Factor (lbs/ton) ³	Emissions (lb/hr)	Emissions Factor (lbs/ton) ³	Emissions (lb/hr)	Emissions Factor (lbs/MMscf) ³	Emissions (lb/hr)	Emissions (lb/hr)
Max Throughput (tons/hr or MMscf/hr)	0.0337		0.0012		0.0060		
Acenaphthene	-	-	1.07E-02	1.26E-05	-	-	1.26E-05
Acenaphthylene	-	-	2.78E-01	3.27E-04	-	-	3.27E-04
Acetaldehyde	-	-	-	-	8.87E-03	5.22E-08	5.22E-08
Anthracene	-	-	9.74E-02	1.14E-04	-	-	1.14E-04
Benzaldehyde	-	-	-	-	1.64E-02	9.65E-08	9.65E-08
Benzene	-	-	-	-	4.31E-03	2.54E-08	2.54E-08
Benzo(a)anthracene	-	-	3.56E-02	4.18E-05	-	-	4.18E-05
Benzo(a)pyrene	-	-	6.26E-03	7.36E-06	-	-	7.36E-06
Benzo(b)fluoranthene	-	-	1.90E-02	2.23E-05	-	-	2.23E-05
Benzo(g,h,i)perylene	-	-	6.91E-03	8.12E-06	-	-	8.12E-06
Benzo(k)fluoranthene	-	-	2.61E-02	3.07E-05	-	-	3.07E-05
Chromium (Hex)	-	-	1.29E-04	1.52E-07	-	-	1.52E-07
Chrysene	-	-	4.99E-02	5.86E-05	-	-	5.86E-05
Dibenz(a,h)anthracene	-	-	3.00E-03	3.53E-06	-	-	3.53E-06
Dioxin:4D 2378	1.16E-09	3.91E-11	1.50E-06	1.76E-09	-	-	1.80E-09
Dioxin:5D 12378	4.64E-09	1.56E-10	8.53E-06	1.00E-08	-	-	1.02E-08
Dioxin:6D 123478	2.48E-09	8.36E-11	1.36E-05	1.60E-08	-	-	1.61E-08
Dioxin:6D 123678	4.17E-09	1.40E-10	3.19E-05	3.75E-08	-	-	3.76E-08
Dioxin:6D 123789	3.47E-09	1.17E-10	2.20E-05	2.59E-08	-	-	2.60E-08
Dioxin:7D 1234678	1.17E-08	3.94E-10	5.69E-05	6.69E-08	-	-	6.73E-08
Dioxin:8D	1.72E-08	5.42E-07	3.25E-05	3.82E-08	-	-	5.81E-07
Fluoranthene	-	-	1.77E-01	2.08E-04	-	-	2.08E-04
Fluorene	-	-	1.37E-01	1.61E-04	-	-	1.61E-04
Formaldehyde	-	-	-	-	2.21E-01	1.30E-06	1.30E-06
Furan:4F 2378	3.56E-09	1.20E-10	2.21E-05	2.60E-08	-	-	2.61E-08
Furan:5F 12378	8.01E-09	2.70E-10	2.39E-05	2.81E-08	-	-	2.84E-08
Furan:5F 23478	1.33E-08	4.48E-10	4.25E-05	4.99E-08	-	-	5.04E-08
Furan:6F 123478	7.32E-09	2.47E-10	6.48E-05	7.62E-08	-	-	7.64E-08
Furan:6F 123678	7.62E-09	2.57E-10	2.45E-05	2.88E-08	-	-	2.90E-08
Furan:6F 123789	3.38E-09	1.14E-10	2.38E-07	2.80E-10	-	-	3.94E-10
Furan:6F 234678	9.81E-09	3.31E-10	2.38E-07	2.80E-10	-	-	6.10E-10
Furan:7F 1234678	1.52E-08	5.12E-10	1.98E-04	2.33E-07	-	-	2.33E-07
Furan:7F 1234789	4.41E-09	1.49E-10	1.53E-05	1.80E-08	-	-	1.81E-08
Furan:8F	7.84E-09	2.64E-10	3.60E-05	4.23E-08	-	-	4.26E-08
HCl	-	-	4.29E+01	5.04E-02	-	-	5.04E-02
Indeno(1,2,3-cd)pyrene	-	-	5.69E-03	6.69E-06	-	-	6.69E-06
Naphthalene	-	-	3.84E-01	4.51E-04	-	-	4.51E-04
Phenanthrene	-	-	3.29E-01	3.87E-04	-	-	3.87E-04
Pyrene	-	-	1.61E-01	1.89E-04	-	-	1.89E-04

¹ Max material throughput based on 9 hours of operation, 2 days a week, 52 weeks per year.

² Max natural gas usage based on the incinerator maximum burn rate (0.006 MMscf/hr).

³ Emission factors were obtained from the CARB CATEF database.

⁴ There are no toxicity values for this chemical. Therefore, no further analysis has been performed.

- Sample Calculation:

Emissions = EF * Throughput

Emissions (lbs/hr) = EF (lb/MMscf) * Max Natural Gas Throughput (MMBtu/hr) / NG Heating Value (Btu/scf)

NG Heating Value = 1020 Btu/scf

Small Boiler Emissions Rates

ISC Modeling ID --> P62_96 P67_00 P3_00 P48_96 P56_96A and P56 P54_00 P65_03 P64_03 P44_96 P45_96 P53_00A P52_00A P55_00 P28_03 P5_00 PP49_96 P06_80 P9091_02 P9091_02 P101_03 P47_96 P39_99																							
Annual Emissions from Natural Gas Fired Boilers																							
Chemical	ARS J-1 Boiler (HW)	ARS J-1 Seven Boilers (S)	ARS K-2 Ten Boilers (S)	ARS P-1 Boiler (HW)	Castilian Dining Commons - Two Boilers (HW)	Comparative Medicine (Primate Lab - Boiler (S)	Contained Research - Two Boilers (HW)	Contained Research - Four Boilers (S)	Environmental and Horticulture K-1 Boiler (HW)	Environmental and Horticulture K-2 Boiler (HW)	Environmental Services Facility Five Boilers (S)	Environmental Services Facility Two Boilers (HW)	Equine Analytical Chemistry Laboratory Two Boilers (S)	Genome Launch Facility Boiler (HW)	Institute of Ecology, West Campus Boiler (HW)	ITEH - Geriatrics 1 Cagewash Three Boilers (S)	ITEH - Geriatrics 1 Cagewash Outside - Three Boilers (S)	Mondavi Center for the Performing Arts Boiler #1 (S)	Mondavi Center for the Performing Arts Boiler #2 (S)	Mondavi Center for the Performing Arts two boilers (HW)	Recreation Pool Boiler (HW)	Thoreau Hall Two Boilers (HW)	Total
Natural Gas Throughput (MMscf/yr)	19.4	24.41	32.076	10.48	0.0072	23.65	9.986	13.946	27.955	12.01	17.432	10.92	13.4	3.937	13.1	0.854	10.95	4.83	2.21	0.4	32.76	0.012	284.73
Substance	Emissions (lb/yr)																						
Acetaldehyde	1.72E-01	2.17E-01	2.85E-01	9.30E-02	6.39E-05	2.10E-01	8.86E-02	1.24E-01	2.48E-01	1.07E-01	1.55E-01	9.69E-02	1.19E-01	3.49E-02	1.16E-01	7.57E-03	9.71E-02	4.28E-02	1.96E-02	3.55E-03	2.91E-01	1.06E-04	2.53E+00
Benzaldehyde	3.18E-01	4.00E-01	5.26E-01	1.72E-01	1.18E-04	3.88E-01	1.64E-01	2.29E-01	4.58E-01	1.97E-01	2.86E-01	1.79E-01	2.20E-01	6.46E-02	2.15E-01	1.40E-02	1.80E-01	7.92E-02	3.62E-02	6.56E-03	5.37E-01	1.97E-04	4.67E+00
Benzene	8.36E-02	1.05E-01	1.38E-01	4.52E-02	3.10E-05	1.02E-01	4.30E-02	6.01E-02	1.20E-01	5.18E-02	7.51E-02	4.71E-02	5.78E-02	1.70E-02	5.65E-02	3.68E-03	4.72E-02	2.08E-02	9.53E-03	1.72E-03	1.41E-01	5.17E-05	1.23E+00
Formaldehyde	4.29E+00	5.39E+00	7.09E+00	2.32E+00	1.59E-03	5.23E+00	2.21E+00	3.08E+00	6.18E+00	2.65E+00	3.85E+00	2.41E+00	2.96E+00	8.70E-01	2.90E+00	1.89E-01	2.42E+00	1.07E+00	4.88E-01	8.84E-02	7.24E+00	2.65E-03	6.29E+01
																							7.13E+01

Maximum Hourly Emissions from Natural Gas Fired Boilers																								
	ARS J-1 Boiler (HW)	ARS J-1 Seven Boilers (S)	ARS K-2 Ten Boilers (S)	ARS P-1 Boiler (HW)	Castilian Dining Commons - Two Boilers (HW)	Comparative Medicine (Primate Lab - Boiler (S)	Contained Research - Two Boilers (HW)	Contained Research - Four Boilers (S)	Environmental and Horticulture K-1 Boiler (HW)	Environmental and Horticulture K-2 Boiler (HW)	Environmental Services Facility Five Boilers (S)	Environmental Services Facility Two Boilers (HW)	Equine Analytical Chemistry Laboratory Two Boilers (S)	Genome Launch Facility Boiler (HW)	Institute of Ecology, West Campus Boiler (HW)	ITEH - Geriatrics 1 Cagewash Three Boilers (S)	ITEH - Geriatrics 1 Cagewash Outside - Three Boilers (S)	Mondavi Center for the Performing Arts Boiler #1 (S)	Mondavi Center for the Performing Arts Boiler #2 (S)	Mondavi Center for the Performing Arts two boilers (HW)	Recreation Pool Boiler (HW)	Thoreau Hall Two Boilers (HW)	Total	
Max Natural Gas Throughput (MMBtu/hr)	2.22	2.8	3.98	1.2	1.05	2.7	2.28	1.6	3.2	1.375	2	1.25	1.53	3.9	1.5	1.1	1.2	2	2	2.15	3.75	1	45.79	
Substance	Emissions (lb/hr)																							
Acetaldehyde	1.93E-05	2.43E-05	3.46E-05	1.04E-05	9.13E-06	2.35E-05	1.98E-05	1.39E-05	2.78E-05	1.20E-05	1.74E-05	1.09E-05	1.33E-05	3.39E-05	1.30E-05	9.57E-06	1.04E-05	1.74E-05	1.74E-05	1.87E-05	3.26E-05	8.70E-06	3.98E-04	
Benzaldehyde ²	3.57E-05	4.50E-05	6.40E-05	1.93E-05	1.69E-05	4.34E-05	3.67E-05	2.57E-05	5.15E-05	2.21E-05	3.22E-05	2.01E-05	2.46E-05	6.27E-05	2.41E-05	1.77E-05	1.93E-05	3.22E-05	3.22E-05	3.46E-05	6.03E-05	1.61E-05	7.36E-04	
Benzene	4.31E-03	9.38E-06	1.18E-05	1.68E-05	5.07E-06	4.44E-06	1.14E-05	9.63E-06	6.76E-06	1.35E-05	5.81E-06	8.45E-06	5.28E-06	6.47E-06	1.65E-05	6.34E-06	4.65E-06	5.07E-06	8.45E-06	8.45E-06	9.08E-06	1.58E-05	4.23E-06	1.93E-04
Formaldehyde	4.81E-04	6.07E-04	8.62E-04	2.60E-04	2.28E-04	5.85E-04	4.94E-04	3.47E-04	6.93E-04	2.98E-04	4.33E-04	2.71E-04	3.32E-04	8.45E-04	3.25E-04	2.38E-04	2.60E-04	4.33E-04	4.33E-04	4.66E-04	8.13E-04	2.17E-04	9.92E-03	
																							1.12E-02	

Notes:

¹ Emission factors were obtained from the CARB CATEF database.

² There are no toxicity values for this chemical. Therefore, no further analysis has been performed.

Sample Calculation:

Emissions (lbs/yr) = EF (lb/MMscf) * Natural Gas Throughput (MMscf/yr)

Emissions (lbs/hr) = EF (lb/MMscf) * Max Natural Gas Throughput (MMBtu/hr) / NG Heating Value (Btu/scf)

NG Heating Value = 1020 Btu/scf

Heater and Kiln Emission Rates - Annual

SOURCE MODELING ID	WALNUTD	LGKILN	RAKUKILN	FDRIYKILN	ARTKILNS	
Annual Emissions from Natural Gas Fired Heaters						
Substance	Emissions Factor (lbs/MMscf) ¹	Walnut Dryer	Large Gas Kiln	Raku Kiln	Foundry Kiln	Art Department - 3 Kilns
Natural Gas Throughput (MMscf/yr)		12.096	0.142688	0.006408	0.00324	0.18504
Emissions (lb/yr)						
Acenaphthene ²	1.39E-06	1.68E-05	1.98E-07	8.91E-09	4.50E-09	2.57E-07
Acenaphthylene ²	1.21E-05	1.46E-04	1.73E-06	7.75E-08	3.92E-08	2.24E-06
Acetaldehyde	1.40E-02	1.69E-01	2.00E-03	8.97E-05	4.54E-05	2.59E-03
Acrolein	4.84E-03	5.85E-02	6.91E-04	3.10E-05	1.57E-05	8.96E-04
Anthracene ²	1.61E-06	1.95E-05	2.30E-07	1.03E-08	5.22E-09	2.98E-07
Benzene	1.12E-02	1.35E-01	1.60E-03	7.18E-05	3.63E-05	2.07E-03
Benzo(a)anthracene	1.96E-06	2.37E-05	2.80E-07	1.26E-08	6.35E-09	3.63E-07
Benzo(a)pyrene	9.80E-07	1.19E-05	1.40E-07	6.28E-09	3.18E-09	1.81E-07
Benzo(b)fluoranthene	1.14E-06	1.38E-05	1.63E-07	7.31E-09	3.69E-09	2.11E-07
Benzo(g,h,i)perylene ²	1.25E-06	1.51E-05	1.78E-07	8.01E-09	4.05E-09	2.31E-07
Benzo(k)fluoranthene	9.90E-07	1.20E-05	1.41E-07	6.34E-09	3.21E-09	1.83E-07
Chromium (VI) ³	-	-	5.92E-04	2.66E-05	1.34E-05	7.68E-04
Chrysene	1.39E-06	1.68E-05	1.98E-07	8.91E-09	4.50E-09	2.57E-07
Dibenz(a,h)anthracene	9.17E-07	1.11E-05	1.31E-07	5.88E-09	2.97E-09	1.70E-07
Ethylbenzene	2.25E-03	2.72E-02	3.21E-04	1.44E-05	7.29E-06	4.16E-04
Fluoranthene ²	1.19E-05	1.44E-04	1.70E-06	7.63E-08	3.86E-08	2.20E-06
Flourene ²	4.59E-06	5.55E-05	6.55E-07	2.94E-08	1.49E-08	8.49E-07
Formaldehyde	7.40E-02	8.95E-01	1.06E-02	4.74E-04	2.40E-04	1.37E-02
Indeno(1,2,3-cd)pyrene	1.17E-06	1.42E-05	1.67E-07	7.50E-09	3.79E-09	2.16E-07
Lead ³	-	-	2.50E-02	1.12E-03	5.67E-04	3.24E-02
Naphthalene	1.12E-03	1.35E-02	1.60E-04	7.18E-06	3.63E-06	2.07E-04
Phenanthrene	3.37E-05	4.08E-04	4.81E-06	2.16E-07	1.09E-07	6.24E-06
Propylene	2.35E-01	2.84E+00	3.35E-02	1.51E-03	7.61E-04	4.35E-02
Pyrene ²	5.60E-06	6.77E-05	7.99E-07	3.59E-08	1.81E-08	1.04E-06
Toluene	2.95E-02	3.57E-01	4.21E-03	1.89E-04	9.56E-05	5.46E-03
Xylene(Total)	1.43E-02	1.73E-01	2.04E-03	9.16E-05	4.63E-05	2.65E-03

Heater and Kiln Emission Rates - Maximum Hourly

Maximum Hourly Emissions from Natural Gas Fired Heaters						
Substance	Emissions Factor (lbs/MMscf)	Walnut Dryer	Large Gas Kiln	Raku Kiln	Foundry Kiln	Art Department - 3 Kilns
Max Natural Gas Throughput (MMBtu/hr)		4	0.36	0.28	0.425	1.08
Emissions (lb/hr)						
Acenaphthene ²	1.39E-06	5.45E-09	4.91E-10	3.82E-10	5.79E-10	1.47E-09
Acenaphthylene ²	1.21E-05	4.75E-08	4.27E-09	3.32E-09	5.04E-09	1.28E-08
Acetaldehyde	1.40E-02	5.49E-05	4.94E-06	3.84E-06	5.83E-06	1.48E-05
Acrolein	4.84E-03	1.90E-05	1.71E-06	1.33E-06	2.02E-06	5.12E-06
Anthracene ²	1.61E-06	6.31E-09	5.68E-10	4.42E-10	6.71E-10	1.70E-09
Benzene	1.12E-02	4.39E-05	3.95E-06	3.07E-06	4.67E-06	1.19E-05
Benzo(a)anthracene	1.96E-06	7.69E-09	6.92E-10	5.38E-10	8.17E-10	2.08E-09
Benzo(a)pyrene	9.80E-07	3.84E-09	3.46E-10	2.69E-10	4.08E-10	1.04E-09
Benzo(b)fluoranthene	1.14E-06	4.47E-09	4.02E-10	3.13E-10	4.75E-10	1.21E-09
Benzo(g,h,i)perylene ²	1.25E-06	4.90E-09	4.41E-10	3.43E-10	5.21E-10	1.32E-09
Benzo(k)fluoranthene	9.90E-07	3.88E-09	3.49E-10	2.72E-10	4.13E-10	1.05E-09
Chromium (VI) ³	-	-	1.46E-06	1.14E-06	1.73E-06	4.39E-06
Chrysene	1.39E-06	5.45E-09	4.91E-10	3.82E-10	5.79E-10	1.47E-09
Dibenz(a,h)anthracene	9.17E-07	3.60E-09	3.24E-10	2.52E-10	3.82E-10	9.71E-10
Ethylbenzene	2.25E-03	8.82E-06	7.94E-07	6.18E-07	9.38E-07	2.38E-06
Fluoranthene ²	1.19E-05	4.67E-08	4.20E-09	3.27E-09	4.96E-09	1.26E-08
Flourene ²	4.59E-06	1.80E-08	1.62E-09	1.26E-09	1.91E-09	4.86E-09
Formaldehyde	7.40E-02	2.90E-04	2.61E-05	2.03E-05	3.08E-05	7.84E-05
Indeno(1,2,3-cd)pyrene	1.17E-06	4.59E-09	4.13E-10	3.21E-10	4.88E-10	1.24E-09
Lead ³	-	-	6.17E-05	4.80E-05	7.29E-05	1.85E-04
Naphthalene	1.12E-03	4.39E-06	3.95E-07	3.07E-07	4.67E-07	1.19E-06
Phenanthrene	3.37E-05	1.32E-07	1.19E-08	9.25E-09	1.40E-08	3.57E-08
Propylene	2.35E-01	9.22E-04	8.29E-05	6.45E-05	9.79E-05	2.49E-04
Pyrene ²	5.60E-06	2.20E-08	1.98E-09	1.54E-09	2.33E-09	5.93E-09
Toluene	2.95E-02	1.16E-04	1.04E-05	8.10E-06	1.23E-05	3.12E-05
Xylene(Total)	1.43E-02	5.61E-05	5.05E-06	3.93E-06	5.96E-06	1.51E-05

Notes:

¹ Emission factors were obtained from the CARB CATEF database.

Sample Calculation:

Emissions (lbs/yr) = EF (lb/MMscf) * Natural Gas Throughput (MMscf/yr)

Emissions (lbs/hr) = EF (lb/MMscf) * Max Natural Gas Throughput (MMBtu/hr) / NG Heating Value (Btu/scf)

NG Heating Value = 1020 Btu/scf

² There are no toxicity factors fro this chemical. Therefore no further analysis has been performed.

³ Chromium and lead emissions based on assumed volatilization loss from ceramic glazing. UC Davis reported chromium content in Fe₂CrO₄ metallic oxide glaze, lead content in Frit 3403, and the annual usage of these materials in the 2005 AB 2588 EIUR. All this material would not volatilize, as assumed in the 2005 AB 2588 EIUR, as most pigment fuses with the ceramic. AP-42 Section 11.7 provides an emission factor of 3.0 lb of glaze per ton glaze used, based on an emission test with a glaze containing 24% by weight lead oxide. Applying this emission factor:

Fe₂CrO₄ metallic oxide glaze use in 2005: 0.66 lb.

Fe₂CrO₄ metallic oxide glaze has 0.34 lb Cr(VI) per lb Fe₂CrO₄:

Adjustment of Pb emission factor for Cr(VI) = (0.34/0.24)

Total annual emissions = (Emission Factor) x (Material Use) = (3 lb/2000 lb) x [(0.34/0.24) x (0.66 lb)] = 0.0014 lb/year

Frit 3403 use in 2005: 15 lb

Frit 3403 has 0.63 lb Pb per lb Frit 3403

Adjustment of Pb emission factor for Frit 3403 = (0.63/0.24)

Total annual emissions = (Emission Factor) x (Material Use) = (3 lb/2000 lb) x [(0.63/0.24) x (15 lb)] = 0.059 lb/year

These total emissions were apportioned between the kilns by firing use per kiln.

Primate Center Boiler # 2 (Landfill Gas Combustion Emissions)

PC Boiler #2 inlet LFG flowrate =	7,095,000 [scf/yr]		
Hourly Emissions calculated from assuming 100% landfill gas at boiler rating of:		12.6	MMBtu/hr
Landfill gas heating value (approx. range 400-600 Btu/scf):		500	Btu/scf
Maximum landfill gas flow per hour for modeling purposes:		25200	scf/hr

Compound	Molecular Weight, MW (g/mol)	Median ppmv ¹	Control Efficiencies (%)	Emissions (lb/yr)	
				Boiler	Boiler
Abated TAC Emissions from Flare:					
1,1,1-Trichloroethane (methyl chloroform)	133.42	0.48	99.6%	4.64E-03	1.65E-05
1,1,2,2-Tetrachloroethane	167.85	1.11	99.6%	1.35E-02	4.79E-05
1,1-Dichloroethane (ethylidene dichloride)	98.95	2.35	99.6%	1.68E-02	5.98E-05
1,1-Dichloroethene (vinylidene chloride)	96.94	0.2	99.6%	1.40E-03	4.99E-06
1,2-Dichloroethane (ethylene dichloride)	98.96	0.41	99.6%	2.94E-03	1.04E-05
1,2-Dichloropropane (propylene dichloride)	112.98	0.18	99.6%	1.47E-03	5.23E-06
2-Propanol (isopropyl alcohol)	60.11	50.1	99.8%	1.09E-01	3.87E-04
Acetone ²	58.08	7.01	99.8%	1.47E-02	5.24E-05
Acrylonitrile	53.06	6.33	99.8%	1.22E-02	4.32E-05
Bromodichloromethane ²	163.87	3.13	99.6%	3.71E-02	1.32E-04
Butane ²	58.12	5.03	99.8%	1.06E-02	3.76E-05
Carbon disulfide	76.13	0.58	99.8%	1.60E-03	5.68E-06
Carbon tetrachloride	153.84	0.004	99.6%	4.46E-05	1.58E-07
Carbonyl sulfide ²	60.07	0.49	99.8%	1.07E-03	3.79E-06
Chlorobenzene	112.56	0.25	99.6%	2.04E-03	7.24E-06
Chlorodifluoromethane (Freon 22) ²	67.47	1.3	99.6%	6.35E-03	2.26E-05
Chloroethane (ethyl chloride)	64.52	1.25	99.6%	5.84E-03	2.07E-05
Chloroform	119.39	0.03	99.6%	2.59E-04	9.21E-07
Chloromethane ²	50.49	1.21	99.6%	4.42E-03	1.57E-05
Dichlorobenzene (1,4- assumed)	147	0.21	99.6%	2.24E-03	7.94E-06
Dichlorodifluoromethane (Freon 12)	120.91	15.70	99.6%	1.37E-01	4.88E-04
Dichlorofluoromethane (Freon 21)	102.92	2.62	99.6%	1.95E-02	6.94E-05
Dichloromethane (methylene chloride)	84.94	14.3	99.6%	8.80E-02	3.12E-04
Dimethyl sulfide (methyl sulfide)	62.13	7.82	99.8%	1.76E-02	6.25E-05
Ethane ²	30.07	889	99.8%	9.68E-01	3.44E-03
Ethanol ²	46.08	27.2	99.8%	4.54E-02	1.61E-04
Ethyl mercaptan (ethanethiol) ²	62.13	2.28	99.8%	5.13E-03	1.82E-05
Ethyl benzene	106.16	4.61	99.8%	1.77E-02	6.29E-05
Ethylene dibromide (EDB)	187.88	0.001	99.6%	1.36E-05	4.83E-08
Fluorotrichloromethane (Freon 11) ²	137.38	0.76	99.6%	7.56E-03	2.69E-05
Hexane	86.17	6.57	99.8%	2.05E-02	7.28E-05
Hydrogen sulfide	34.08	35.5	99.8%	4.38E-02	1.56E-04
Mercury (total)	200.61	2.92E-04	0.0%	1.06E-03	3.77E-06
Methyl ethyl ketone	72.1	7.09	99.8%	1.85E-02	6.57E-05
Methyl isobutyl ketone ²	100.16	1.87	99.8%	6.78E-03	2.41E-05
Methyl mercaptan ²	48.1	2.49	99.8%	4.34E-03	1.54E-05
Pentane ²	72.15	3.29	99.8%	8.59E-03	3.05E-05
Perchloroethylene (tetrachloroethylene)	165.83	3.73	99.6%	4.48E-02	1.59E-04
Propane ²	44.09	11.1	99.8%	1.77E-02	6.29E-05
Trichloroethylene	131.4	2.82	99.6%	2.68E-02	9.53E-05
t-1,2-Dichloroethene ²	96.94	2.84	99.6%	1.99E-02	7.08E-05
Vinyl chloride	62.5	7.34	99.6%	3.32E-02	1.18E-04
Xylene	106.16	12.10	99.8%	4.65E-02	1.65E-04
Benzene	78.11	1.91	99.8%	5.40E-03	1.92E-05
Toluene	92.13	39.3	99.8%	1.31E-01	4.66E-04
Secondary TACs from Flare Combustion:					
Hydrochloric acid (HCl) ³	36.45	42.0	99.6%	2.76E+01	9.80E-02
Hydrofluoric acid (HF) ³	20.01	37.4	99.6%	1.35E+01	4.79E-02
Hydrogen bromide (HBr) ³	80.91	3.1	99.6%	4.57E+00	1.62E-02
Acetaldehyde ⁴	2.58E-01	lb/MMscf	--	1.83E+00	6.50E-03
Formaldehyde ⁴	2.95E+01	lb/MMscf	--	2.09E+02	7.43E-01
Acrolein ⁴	8.44E-02	lb/MMscf	--	5.99E-01	2.13E-03
Benzo(a)anthracene ³	2.12E-02	lb/MMscf	--	1.50E-01	5.34E-04
Benzo(a)pyrene ⁴	2.11E-02	lb/MMscf	--	1.50E-01	5.32E-04
Benzo(b)fluoranthene ⁴	2.11E-02	lb/MMscf	--	1.50E-01	5.32E-04
Benzo(k)fluoranthene ⁴	2.11E-02	lb/MMscf	--	1.50E-01	5.32E-04
Dibenzo(a,h)anthracene ³	2.11E-02	lb/MMscf	--	1.50E-01	5.32E-04
Indeno(1,2,3-cd)pyrene ⁴	2.11E-02	lb/MMscf	--	1.50E-01	5.32E-04
Naphthalene ³	1.30E+01	lb/MMscf	--	9.22E+01	3.28E-01
2,3,7,8-Tetrachlorodibenzo-p-dioxin ⁴	6.68E-07	lb/MMscf	--	4.74E-06	1.68E-08
1,2,3,7,8-Pentachlorodibenzo-p-dioxin ⁴	6.68E-07	lb/MMscf	--	4.74E-06	1.68E-08
1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin ⁴	6.68E-07	lb/MMscf	--	4.74E-06	1.68E-08
1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin ⁴	1.34E-06	lb/MMscf	--	9.51E-06	3.38E-08
1,2,3,4,5,6,7,8-Octachlorodibenzo-p-dioxin ⁴	1.34E-06	lb/MMscf	--	9.51E-06	3.38E-08
2,3,7,8-Tetrachlorodibenzofuran ⁴	6.68E-07	lb/MMscf	--	4.74E-06	1.68E-08
1,2,3,7,8-Pentachlorodibenzofuran ⁴	6.68E-07	lb/MMscf	--	4.74E-06	1.68E-08
1,2,3,6,7,8-Hexachlorodibenzofuran ⁴	6.68E-07	lb/MMscf	--	4.74E-06	1.68E-08
1,2,3,4,6,7,8-Heptachlorodibenzofuran ⁴	1.34E-06	lb/MMscf	--	9.51E-06	3.38E-08
1,2,3,4,5,6,7,8-Octachlorodibenzofuran ⁴	1.34E-06	lb/MMscf	--	9.51E-06	3.38E-08

Notes:

¹ Default concentrations, EPA AP-42, Section 2.4, September 1998.

² There are no toxicity factors for this chemical. Therefore no further analysis has been performed.

³ Calculated from assumed constituent default concentrations and acids created from 99.6% destruction efficiency.

⁴ These are products of incomplete combustion. There are no AP-42 emission factors; thus calculated from the CATEF emission factors for a landfill gas flare since produced chiefly from volatile organics in the landfill gas.

Calculations:

PC Boiler #2 inlet LFG flowrate =	7,433,640 scf/yr		
Hourly Emissions calculated from assuming 100% landfill gas at boiler ra		12.6	MMBtu/hr
Landfill gas heating value (approx. range 400-600 Btu/scf):		500	Btu/scf
Maximum landfill gas flow per hour for modeling purposes:		19841.26984	scf/hr

Landfill Gas Fugitive Emissions (from Landfill Surface)

Flare inlet LFG flowrate =	4,000 [scf/yr]
PC Boiler #2 inlet LFG flowrate =	7,095,000 [scf/yr]
Total collected LFG flowrate =	7,099,000 [scf/yr]
Estimated Collection Efficiency =	20%
Estimated LFG generation rate =	34,689,600 [scf/yr]
Estimated Fugitive Gas Flowrate =	27,590,600 [scf/yr]

Compound	Molecular Weight, MW (g/mol)	Median ppmv ¹	Emissions (lb/yr) ²	Emissions (lb/hr) ²
			Fugitive	Fugitive
1,1,1-Trichloroethane (methyl chloroform)	133.42	0.48	4.51E+00	5.15E-04
1,1,2,2-Tetrachloroethane	167.85	1.11	1.31E+01	1.50E-03
1,1-Dichloroethane (ethylidene dichloride)	98.95	2.35	1.64E+01	1.87E-03
1,1-Dichloroethene (vinylidene chloride)	96.94	0.2	1.36E+00	1.56E-04
1,2-Dichloroethane (ethylene dichloride)	98.96	0.41	2.86E+00	3.26E-04
1,2-Dichloropropane (propylene dichloride)	112.98	0.18	1.43E+00	1.63E-04
2-Propanol (isopropyl alcohol)	60.11	50.1	2.12E+02	2.42E-02
Acetone ³	58.08	7.01	2.87E+01	3.27E-03
Acrylonitrile	53.06	6.33	2.36E+01	2.70E-03
Bromodichloromethane	163.87	3.13	3.61E+01	4.12E-03
Butane ³	58.12	5.03	2.06E+01	2.35E-03
Carbon disulfide	76.13	0.58	3.11E+00	3.55E-04
Carbon tetrachloride	153.84	0.004	4.33E-02	4.95E-06
Carbonyl sulfide ³	60.07	0.49	2.07E+00	2.37E-04
Chlorobenzene	112.56	0.25	1.98E+00	2.26E-04
Chlorodifluoromethane (Freon 22) ²	67.47	1.3	6.18E+00	7.05E-04
Chloroethane (ethyl chloride)	64.52	1.25	5.68E+00	6.48E-04
Chloroform	119.39	0.03	2.52E-01	2.88E-05
Chloromethane ³	50.49	1.21	4.30E+00	4.91E-04
Dichlorobenzene (1,4- assumed)	147	0.21	2.17E+00	2.48E-04
Dichlorodifluoromethane (Freon 12) ³	120.91	15.70	1.34E+02	1.53E-02
Dichlorofluoromethane (Freon 21)	102.92	2.62	1.90E+01	2.17E-03
Dichloromethane (methylene chloride)	84.94	14.3	8.55E+01	9.76E-03
Dimethyl sulfide (methyl sulfide)	62.13	7.82	3.42E+01	3.90E-03
Ethane ³	30.07	889	1.88E+03	2.15E-01
Ethanol ³	46.08	27.2	8.82E+01	1.01E-02
Ethyl mercaptan (ethanethiol) ³	62.13	2.28	9.97E+00	1.14E-03
Ethyl benzene	106.16	4.61	3.45E+01	3.93E-03
Ethylene dibromide (EDB)	187.88	0.001	1.32E-02	1.51E-06
Fluorotrichloromethane (Freon 11) ³	137.38	0.76	7.35E+00	8.39E-04
Hexane	86.17	6.57	3.99E+01	4.55E-03
Hydrogen sulfide	34.08	35.5	8.52E+01	9.72E-03
Mercury (total)	200.61	2.92E-04	4.12E-03	4.71E-07
Methyl ethyl ketone	72.1	7.09	3.60E+01	4.11E-03
Methyl isobutyl ketone ³	100.16	1.87	1.32E+01	1.51E-03
Methyl mercaptan ³	48.1	2.49	8.43E+00	9.63E-04
Pentane ³	72.15	3.29	1.67E+01	1.91E-03
Perchloroethylene (tetrachloroethylene)	165.83	3.73	4.35E+01	4.97E-03
Propane ³	44.09	11.1	3.45E+01	3.93E-03
Trichloroethylene	131.4	2.82	2.61E+01	2.98E-03
t-1,2-Dichloroethene ³	96.94	2.84	1.94E+01	2.21E-03
Vinyl chloride	62.5	7.34	3.23E+01	3.69E-03
Xylene	106.16	12.10	9.04E+01	1.03E-02
Benzene	78.11	1.91	1.05E+01	1.20E-03
Toluene	92.13	39.3	2.55E+02	2.91E-02

Notes:

¹ Default concentrations, EPA AP-42, Section 2.4, September 1998

² For modeling purposes, emissions were divided by four, the number of sources used to represent the landfill

³ There are no toxicity factors for this chemical. Therefore, no further analysis was performed.

Calculations:

Flare inlet LFG flowrate =	4,000	[scf/yr]
PC Boiler #2 inlet LFG flowrate =	7,095,000	[scf/yr]
Total collected LFG flowrate =	7,099,000	[scf/yr]
Estimated Collection Efficiency =	21%	
Estimated LFG generation rate =	34,164,600	[scf/yr]
Estimated Fugitive Gas Flowrate =	27,065,600	[scf/yr]

Landfill Gas Flare Emissions

Flare inlet LFG flowrate = 4,000 [scf/yr]¹
 Hourly Emissions calculated from annual divided 28 hours (continuous flare operation)²

Compound	Molecular Weight, MW (g/mol)	Median ppmv ³	Control Efficiencies (%)	Emissions (lb/yr)	
				Flare	Flare
Abated TAC Emissions from Flare:					
1,1,1-Trichloroethane (methyl chloroform)	133.42	0.48	98.0%	1.31E-05	4.67E-07
1,1,2,2-Tetrachloroethane	167.85	1.11	98.0%	3.80E-05	1.36E-06
1,1-Dichloroethane (ethylidene dichloride)	98.95	2.35	98.0%	4.75E-05	1.70E-06
1,1-Dichloroethene (vinylidene chloride)	96.94	0.2	98.0%	3.96E-06	1.41E-07
1,2-Dichloroethane (ethylene dichloride)	98.96	0.41	98.0%	8.28E-06	2.96E-07
1,2-Dichloropropane (propylene dichloride)	112.98	0.18	98.0%	4.15E-06	1.48E-07
2-Propanol (isopropyl alcohol)	60.11	50.1	99.7%	9.22E-05	3.29E-06
Acetone ⁴	58.08	7.01	99.7%	1.25E-05	4.45E-07
Acrylonitrile	53.06	6.33	99.7%	1.03E-05	3.67E-07
Bromodichloromethane ⁴	163.87	3.13	98.0%	1.05E-04	3.74E-06
Butane ⁴	58.12	5.03	99.7%	8.95E-06	3.20E-07
Carbon disulfide	76.13	0.58	99.7%	1.35E-06	4.83E-08
Carbon tetrachloride	153.84	0.004	98.0%	1.26E-07	4.49E-09
Carbonyl sulfide ⁴	60.07	0.49	99.7%	9.01E-07	3.22E-08
Chlorobenzene	112.56	0.25	98.0%	5.74E-06	2.05E-07
Chlorodifluoromethane (Freon 22) ⁴	67.47	1.3	98.0%	1.79E-05	6.39E-07
Chloroethane (ethyl chloride)	64.52	1.25	98.0%	1.65E-05	5.88E-07
Chloroform	119.39	0.03	98.0%	7.31E-07	2.61E-08
Chloromethane ⁴	50.49	1.21	98.0%	1.25E-05	4.45E-07
Dichlorobenzene (1,4- assumed)	147	0.21	98.0%	6.30E-06	2.25E-07
Dichlorodifluoromethane (Freon 12)	120.91	15.70	98.0%	3.88E-04	1.38E-05
Dichlorofluoromethane (Freon 21)	102.92	2.62	98.0%	5.50E-05	1.97E-06
Dichloromethane (methylene chloride)	84.94	14.3	98.0%	2.48E-04	8.86E-06
Dimethyl sulfide (methyl sulfide)	62.13	7.82	99.7%	1.49E-05	5.31E-07
Ethane ⁴	30.07	889	99.7%	8.19E-04	2.92E-05
Ethanol ⁴	46.08	27.2	99.7%	3.84E-05	1.37E-06
Ethyl mercaptan (ethanethiol) ⁴	62.13	2.28	99.7%	4.34E-06	1.55E-07
Ethyl benzene	106.16	4.61	99.7%	1.50E-05	5.35E-07
Ethylene dibromide (EDB)	187.88	0.001	98.0%	3.84E-08	1.37E-09
Fluorotrichloromethane (Freon 11) ⁴	137.38	0.76	98.0%	2.13E-05	7.61E-07
Hexane	86.17	6.57	99.7%	1.73E-05	6.19E-07
Hydrogen sulfide	34.08	35.5	99.7%	3.70E-05	1.32E-06
Mercury (total)	200.61	2.92E-04	0.0%	5.98E-07	2.14E-08
Methyl ethyl ketone	72.1	7.09	99.7%	1.57E-05	5.59E-07
Methyl isobutyl ketone ⁴	100.16	1.87	99.7%	5.74E-06	2.05E-07
Methyl mercaptan ⁴	48.1	2.49	99.7%	3.67E-06	1.31E-07
Pentane ⁴	72.15	3.29	99.7%	7.27E-06	2.60E-07
Perchloroethylene (tetrachloroethylene)	165.83	3.73	98.0%	1.26E-04	4.51E-06
Propane ⁴	44.09	11.1	99.7%	1.50E-05	5.35E-07
Trichloroethylene	131.4	2.82	98.0%	7.56E-05	2.70E-06
t-1,2-Dichloroethene ⁴	96.94	2.84	98.0%	5.62E-05	2.01E-06
Vinyl chloride	62.5	7.34	98.0%	9.36E-05	3.34E-06
Xylene	106.16	12.10	99.7%	3.93E-05	1.40E-06
Benzene	78.11	1.91	99.7%	4.57E-06	1.63E-07
Toluene	92.13	39.3	99.7%	1.11E-04	3.96E-06
Secondary TACs from Flare Combustion:					
Hydrochloric acid (HCl) ⁵	36.45	42.0	98.0%	1.53E-02	5.47E-04
Hydrofluoric acid (HF) ⁵	20.01	37.4	98.0%	7.48E-03	2.67E-04
Hydrogen bromide (HBr) ⁵	80.91	3.1	98.0%	2.53E-03	9.05E-05
Acetaldehyde ⁶	2.58E-01	lb/MMscf	--	1.03E-03	3.69E-05
Formaldehyde ⁶	2.95E+01	lb/MMscf	--	1.18E-01	4.21E-03
Acrolein ⁶	8.44E-02	lb/MMscf	--	3.38E-04	1.21E-05
Benzo(a)anthracene ⁶	2.12E-02	lb/MMscf	--	8.48E-05	3.03E-06
Benzo(a)pyrene ⁶	2.11E-02	lb/MMscf	--	8.44E-05	3.01E-06
Benzo(b)fluoranthene ⁶	2.11E-02	lb/MMscf	--	8.44E-05	3.01E-06
Benzo(k)fluoranthene ⁶	2.11E-02	lb/MMscf	--	8.44E-05	3.01E-06
Dibenzo(a,h)anthracene ⁶	2.11E-02	lb/MMscf	--	8.44E-05	3.01E-06
Indeno(1,2,3-cd)pyrene ⁶	2.11E-02	lb/MMscf	--	8.44E-05	3.01E-06
Naphthalene ⁶	1.30E+01	lb/MMscf	--	5.20E-02	1.86E-03

Notes:

- ¹ Flare inlet LFG flowrate = 4,000 scf/yr for 2009 operations.
- ² Hourly Emissions calculated from annual divided 28 hours for 2009 operations.
- ³ Default concentrations, EPA AP-42, Section 2.4, September 1998.
- ⁴ There are no toxicity values for this chemical. Therefore, no further analysis has been performed.
- ⁵ Calculated from assumed constituent default concentrations and acids created from 98% destruction efficiency.
- ⁶ These are products of incomplete combustion. There are no AP-42 emission factors; thus calculated from the CATEF emission factors for a landfill gas flare since produced chiefly from volatile organics in the landfill gas.

Wastewater Treatment Plant Emissions

Substance	Annual Average Emissions (lbs/yr)	Maximum Hourly Emissions (lbs/hr)
Hydrogen Sulfide	8.14	9.29E-04
1,1,1-Trichloroethane	16.79	0.002
Chloroform	22.67	0.003
Methylene Chloride	42.42	0.005
Tetrachloroethylene	28.98	3.31E-03
Methyl Ethyl Ketone	14.63	1.67E-03
Methyl Isobutyl Ketone ¹	13.31	1.52E-03
Benzene	0.05	5.71E-06
Ethyl Benzene	0.04	4.57E-06
Toluene	921.83	0.105
Xylene	1113.92	0.127

¹ No toxicity factors exist for this chemical. Therefore, no further analysis has been performed.

- Referenced from the report " 2005 Emission Inventory Update Report for the University of California, Davis"

Storehouse/Receiving Bulk Solvent Storage

Solvent	Amount Stored (gal/yr)	Density (lb/gal)	Average Annual Emissions (lbs/yr)¹	Maximum Hourly Emissions (lbs/hr)²
Methanol	6	6.59	39.54	4.51E-03

¹ Annual Average (lb/yr) = Amount Stored (gal/yr) x Density (lb/gal)

² Maximum Hourly (lb/hr) = Annual Average (lb/yr) / 8760 (hr/yr)

Tank Emissions

Annual VOC Emission (lb/yr)				
	Splash and Fill loss		Fueling Loss	
	EF (lbs/1000)	VOCs	EF (lbs/1000)	VOCs
Agricultural Services ¹	11.5	8.85	11	84.62
Primate Center ^{1,2}	11.5	1.56	11	1.50
Plant Pathology ^{1,2}	11.5	0.17	11	0.16
Grounds Division ^{1,2}	11.5	4.25	11	4.07
Pomology ^{1,2}	11.5	1.38	11	1.32
University Airport ²	11.5	83.21	11	795.95
UST 73 Fleet Services ^{1,2}	11.5	344.51	11	329.53
		443.93		1217.14

Maximum Hourly Emissions (lb/hr)			
	Splash and Fill Los	Fueling Loss	Total
Agricultural Services ¹	1.01E-03	9.66E-03	1.07E-02
Primate Center ^{1,2}	1.79E-04	1.71E-04	3.49E-04
Plant Pathology ^{1,2}	1.94E-05	1.86E-05	3.80E-05
Grounds Division ^{1,2}	4.85E-04	4.64E-04	9.49E-04
Pomology ^{1,2}	1.58E-04	1.51E-04	3.08E-04
University Airport ²	9.50E-03	9.09E-02	1.00E-01
UST 73 Fleet Services ^{1,2}	3.93E-02	3.76E-02	7.69E-02
	0.05068		

Source Location	Isomers of Hexane ⁴	N-hexane	Cyclohexane ⁴	2,2,4-Trimethylpentane	Isomers of Xylene	Benzene	Toluene
Weight Percent³	0.0484	0.0185	0.0021	0.0041	0.001	0.0072	0.0062
Annual Emissions (lb/yr)							
Agricultural Services	4.52E+00	1.73E+00	1.96E-01	3.83E-01	9.35E-02	6.73E-01	5.79E-01
Primate Center	1.48E-01	5.66E-02	6.43E-03	1.25E-02	3.06E-03	2.20E-02	1.90E-02
Plant Pathology	1.61E-02	6.16E-03	6.99E-04	1.36E-03	3.33E-04	2.40E-03	2.06E-03
Grounds Division	4.02E-01	1.54E-01	1.75E-02	3.41E-02	8.32E-03	5.99E-02	5.16E-02
Pomology	1.31E-01	5.00E-02	5.67E-03	1.11E-02	2.70E-03	1.94E-02	1.67E-02
University Airport	4.26E+01	1.63E+01	1.85E+00	3.60E+00	8.79E-01	6.33E+00	5.45E+00
UST 73 Fleet Services	3.26E+01	1.25E+01	1.42E+00	2.76E+00	6.74E-01	4.85E+00	4.18E+00
Total	80.40	30.73	3.49	6.81	1.66	11.96	10.30
Maximum Hourly Emissions (lb/hr)							
Agricultural Services	5.16E-04	1.97E-04	2.24E-05	4.37E-05	1.07E-05	7.68E-05	6.62E-05
Primate Center	1.69E-05	6.46E-06	7.34E-07	1.43E-06	3.49E-07	2.52E-06	2.17E-06
Plant Pathology	1.84E-06	7.03E-07	7.98E-08	1.56E-07	3.80E-08	2.74E-07	2.36E-07
Grounds Division	4.59E-05	1.76E-05	1.99E-06	3.89E-06	9.49E-07	6.84E-06	5.89E-06
Pomology	1.49E-05	5.70E-06	6.47E-07	1.26E-06	3.08E-07	2.22E-06	1.91E-06
University Airport	4.86E-03	1.86E-03	2.11E-04	4.11E-04	1.00E-04	7.23E-04	6.22E-04
UST 73 Fleet Services	3.72E-03	1.42E-03	1.62E-04	3.15E-04	7.69E-05	5.54E-04	4.77E-04
Total	5.45E-03	2.08E-03	2.37E-04	4.62E-04	1.13E-04	8.11E-04	6.99E-04

Throughput	
Agricultural Services	7692.53 gallons
Primate Center	1360 gallons
Plant Pathology	147.9 gallons
Grounds Division	3696 gallons
Pomology	1200 gallons
University Airport	72359 gallons
UST 73 Fleet Services	299572 gallons

¹ 90% Phase I collection assumed for splash/fill loss.

² 90% Phase I collection assumed for refueling loss.

³ Weight percents from ARB Speciation Manual - Profile Number 708 Gasoline Vapors- 1985

⁴ No toxicity factors exist for this chemical exist. Therefore, no further analysis has been performed.

- Sample Calculation:

Emissions(lbs/yr) = EF (lb/lb vapor) * Gasoline Vapor Throughput (lb vapor/yr)

Emissions (lbs/hr) = Emissions (lbs/yr) / 8760

Chloroform Remediation Systems Emissions

Source ID	Source of Chloroform	Average Annual Emissions (lbs/yr)	Maximum Hourly Emissions (lbs/hr)
GW_TREAT	Groundwater Remediation System ¹	15.35	0.00175
AIRSTRIP	Groundwater Stripper ²	13.16	0.0015
INWSTRIP	In-well Stripper for the Dual Density Convection System ²	12.3	0.0014
Total		40.81	4.65E-03

Note:

¹ Located at the south campus disposal site (SCDS)

² Located by the campus landfill

Source: UC Davis Emission Inventory Update Report for the University of California, Davis.

Appendix D
Modeling Parameters

UC Davis Point Source Modeling Parameters

ISCST3 Source ID	Source Description	Easting (X) (m)	Northing (Y) (m)	Base Elevation (m)	Stack Height (m)	Temperature (K)	Exit Velocity (m/s)	Stack Diameter (m)
<u>Central Heating and Cooling Plant</u>								
CHCPBLR1	Central heating and cooling plant Boiler 1	608320.1	4266020.5	15.3	13.72	429.84	8.132	1.719
CHCPBLR2	Central heating and cooling plant Boiler 2	608319.6	4266009.5	15.4	13.72	429.84	8.068	1.719
CHCPBLR3	Central heating and cooling plant Boiler 3	608302.6	4266020.5	15.4	13.72	423.18	8.297	1.332
NEWBLRNG	Natural gas New CISCO Boiler Installed during 2009	608287.1	4266037.1		13.87	550	7.06	1.3716
NEWBLRDS	Diesel New CISCO Boiler Installed during 2009	608287.1	4266037.1		13.87	550	6.87	1.3716
<u>Primate Center Boilers</u>								
PRIMBLR1	Primate Center Boiler No 1 NG fired	604130.2	4266184.0	19.8	10.97	427.62	18.288	1.07
PCBLR2NG	Primate Center Boiler No 2 Natural Gas	604139.1	4266184.5	19.8	10.97	427.62	18.288	1.07
PCBLR2LG	Primate Center Boiler No 2 Landfill Gas	604139.1	4266184.5	19.8	10.97	427.62	18.288	1.07
<u>Landfill Gas Flare</u>								
LF_FLARE	Landfill gas flare	604166.5	4265537	20.7	5.49	1172.04	1.67	1.2192
<u>Veterinary Incinerator</u>								
INCIN	Veterinary Incinerator	607620.0	4265445.0	16.8	7.32	891	19.1	0.2042
<u>Small Boilers > 5MMBtu/hr</u>								
P62_96	ARS J-1 (H001)	608494.2	4264929.0	16.1	12.19	293.15	1.04	0.508
P67_00	ARS J-1 CAAN 3840 - 4 boilers	608496.9	4264929.0	16.1	4.27	338.71	1.32	0.3556
P3_00	ARS K-2 Co-located 2 stacks	608553.5	4264807.5	15.5	4.27	338.71	2.2	0.3556
P48_96	ARS P-1 (H040)	608613.9	4264742.0	15.2	9.14	293.15	0.88	0.4064
P54_00	Comparative Medicine (Primate Center)	604219.1	4266131.0	19.8	18.29	293.15	3.52	0.3048
P65_03	Contained Research	605497.2	4265587.5	19.2	6.10	299.82	1.52	0.3048
P64_03	Contained Research	605494.7	4265584.0	19.2	6.10	299.82	1.2	0.203
P44_96	Environmental Horticulture K-1	609259.4	4265844.5	16.5	7.32	348.71	1.24	0.6096
P45_96	Environmental Horticulture K-2	609269.9	4265856.0	16.5	4.57	320.93	1.44	0.3556
P53_00A	Environmental Services Facility A	606709.7	4265390.0	17.4	10.67	293.15	1.47	0.203
P52_00A	Environmental Services Facility (3 per stack)	606709.3	4265386.0	17.4	10.67	293.15	2.34	0.203
P55_00	Equine Analytical Chemistry Lab	607592.2	4265518.5	16.8	18.29	293.15	2.25	0.203
P28_03	Genome Launch Facility (plant reproduction)	607841.8	4266122.5	16.8	15.24	348.71	1.51	0.6096
P56_96A	Housing-Castillian DC	607527.7	4267286.0	16.5	4.57	348.71	2.05	0.1524
P56_96B	Housing - Castillian DC	607525.6	4267266.5	16.5	4.57	348.71	4.5	0.1524
P5_00	Institute of Ecology - West Campus	606180.4	4265026.5	19.8	4.57	348.71	3.35	0.253
P49_96	ITEH Geriatrics - cage wash inside co-located 3 stacks	608627.7	4263963.5	14.6	3.66	348.71	1.05	0.203
P06_80	ITEH Geriatrics Cagewash - outside - co-locate 3 stacks	608626.3	4263954.5	14.6	3.66	348.71	1.39	0.203
P9091_02	Mondavi Ctr for Performing Arts - 2 boilers	609123.4	4265661.0	16	36.58	293.15	20.88	0.1524
P101_03	Mondavi Ctr for Performing Arts	609111.2	4265658.0	16.2	36.58	293.15	11.22	0.1524
P47_96	Rec Pool	607974.4	4266216.0	16.5	3.66	348.71	2.1	0.5078
P39_99	Thoriau Hall - 2 stacks co-located	607809.1	4267040.0	16.1	15.24	348.71	1.75	0.203
<u>Chloroform Remediation</u>								
AIRSTRIP	Air Stripper	604445.0	4265820.0	20.1	4.57	293.15	0.582	0.2032
INWSTRIP	In-well Stripper for the Dual Density Convection System	608704.9	4263972.5	14.4	6.10	293.15	13.97	0.0508

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GW_TREAT	Ground Water Treatment	608918.3	4264003.0	13	7.62	293.15	15.52	0.3
Heaters and Kilns								
LGKILN	Large Kiln	608764.8	4266086.0	15.2	1.83	348.71	1.26	0.2032
RAKUKILN	Raku Kiln	608774.5	4266085.5	15.2	0.91	348.71	0.98	0.2032
FDRYKILN	Foundry Kiln	608784.6	4266081.0	15.2	0.91	348.71	1.48	0.2032
ARTKILNS	Three Art Dept Kilns to roof vent	609360.6	4266265.0	16.3	3.66	348.71	1.26	0.2032
STRHOUSE	Storehouse/Bulk Receiving and Storage	608915.5	4265732.0	16.8	3.66	293.15	15.24	0.9144
WALNUTD	Walnut Drier	605420.81	4265847	18.9	3.05	316.48	5.64	0.3048
Laboratory Fume Hoods								
TB187	Temporary Building 187	608592.8	4266624.5	14.8	5.49	293.15	15.24	0.9144
TB188	Temporary Building 188	608593.3	4266614.5	14.9	5.49	293.15	15.24	0.9144
VEI_1	Veihmeyer	608830.4	4266601.0	14.6	9.14	293.15	15.24	0.9144
ENOL_1	Enology	608826.3	4266519.5	14.6	9.14	293.15	15.24	0.9144
WICK_1	Wickson Hall	608893.1	4266503.0	14.6	12.80	293.15	15.24	0.9144
HOLG_1	Hoagland	608639.2	4266498.0	14.6	9.14	293.15	15.24	0.9144
MANN_1	Mann Hall	608534.8	4266434.0	14.9	5.49	293.15	15.24	0.9144
STORER_1	Storer Hall	608630.8	4266363.5	14.7	27.43	293.15	15.24	0.9144
HUTCH_1	Hutchison Hall/Biological Sci Unit 2	608721.7	4266374.5	14.6	23.77	293.15	15.24	0.9144
ASMUND_1	Asmundson Hall	608752.0	4266477.5	14.6	9.14	293.15	15.24	0.9144
ROBBINS1	Robbins Hall	608855.3	4266330.0	14.6	12.80	293.15	15.24	0.9144
TB202	Temporary Building 202	608673.4	4266146.0	14.9	5.49	293.15	15.24	0.9144
BRIGGS1	Briggs Hall and Life Sciences	608473.6	4266256.0	15.1	16.46	293.15	15.24	0.9144
TB194	Temporary Building 194	608428.3	4266073.5	15.2	5.49	293.15	15.24	0.9144
TB163	Temporary Building 163	608491.2	4266078.0	15.2	5.49	293.15	15.24	0.9144
TB193	Temporary Building 193	608441.3	4266043.0	15.2	5.49	293.15	15.24	0.9144
TB191	Temporary Building 191	608467.1	4266036.5	15.2	5.49	293.15	15.24	0.9144
TB166	Temporary Building 1664	608495.0	4266046.5	15.2	5.49	293.15	15.24	0.9144
TB167	Temporary Building 167	608499.7	4266037.5	15.2	5.49	293.15	15.24	0.9144
TB138	Temporary Building 138	608554.5	4266037.5	15.2	5.49	293.15	15.24	0.9144
TB155	Temporary Building 1554	608563.8	4265999.0	15.2	5.49	293.15	15.24	0.9144
TB156	Temporary Building 156	608576.8	4265993.5	15.2	5.49	293.15	15.24	0.9144
TB157	Temporary Building 157	608588.6	4265993.0	15.2	5.49	293.15	15.24	0.9144
TB151	Temporary Building 151	608579.3	4266011.5	15.2	5.49	293.15	15.24	0.9144
TB149	Temporary Building 149	608594.9	4266023.0	15.2	5.49	293.15	15.24	0.9144
TB153	Temporary Building 153	608607.8	4266007.0	15.2	5.49	293.15	15.24	0.9144
TB158	Temporary Building 158	608606.8	4265996.0	15.2	5.49	293.15	15.24	0.9144
ENGIN2_1	Engineering II	608611.7	4265947.0	15.5	12.80	293.15	15.24	0.9144
WALKER1	Walker Hall	608971.5	4266237.5	15	9.14	293.15	15.24	0.9144
CHEM1	Chemistry	608930.5	4266085.5	15.5	16.46	293.15	15.24	0.9144
CHEMANX1	Chemistry Annex	608997.3	4266024.0	16.4	20.12	293.15	15.24	0.9144
BAINER1	Bainer Hall	608768.9	4265965.0	15.6	16.46	293.15	15.24	0.9144

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CROCKER	Crocker Hall	608811.1	4265878.0	16.2	5.49	293.15	15.24	0.9144
AC_SURG1	Academic Surge	608794.1	4265753.5	16.8	9.14	293.15	15.24	0.9144
MEYER1	Meyer Hall	608648.8	4265704.0	16.5	23.16	293.15	15.24	0.9144
PHYSGEO1	Physics/Geology/Physics Unit 1	608925.4	4265900.5	16.4	23.16	293.15	15.24	0.9144
ENVHORT	Environmental Horticulture	609283.5	4265887.0	16.5	5.49	293.15	15.24	0.9144
THURMAN1	Thurman Hall	607610.6	4265461.0	16.8	5.49	293.15	15.24	0.9144
MADDY1	Maddy Hall	607586.4	4265517.0	16.8	7.92	293.15	15.24	0.9144
TUPPER	Tupper Hall	607756.0	4265591.5	16.5	16.46	293.15	15.24	0.9144
VETMED2	VET MED 2	607852.5	4265300.0	16.2	5.49	293.15	15.24	0.9144
ASMNDANX	Asmundson Annex	608720.9	4266474.0	14.6	5.49	293.15	15.24	0.9144
YOUNG	Young Hall	609234.3	4266550.0	15.3	9.45	293.15	15.24	0.9144
TB9	Temporary Building 9	609372.3	4266262.5	15.9	5.49	293.15	15.24	0.9144
ARSH1	ARS H-1 (Vet Meta Res)	608482.2	4264700.0	15.6	5.49	293.15	15.24	0.9144
SEROLOGY	Serology4	608553.4	4264629.0	15.2	5.49	293.15	15.24	0.9144
ARSR1	ARS R-1	608493.8	4264490.0	15.2	5.49	293.15	15.24	0.9144
ARSR2	ARS R-2	608494.3	4264463.0	15.2	5.49	293.15	15.24	0.9144
COMP_MED	Center For Comparative Medicine	604236.5	4266132.0	19.8	17.07	293.15	15.24	0.9144
PRIMCNTR	Primate Center	604176.2	4266185.5	19.8	10.97	293.15	15.24	0.9144
TB184	Temporary Building 184	604052.1	4266203.0	19.2	5.49	293.15	15.24	0.9144
TB160	Temporary Building 160	605953.0	4266118.5	18	5.49	293.15	15.24	0.9144
APCARU2	APCARU	606071.2	4266125.5	18	5.49	293.15	15.24	0.9144
ECOL_LAB	Ecology Lab (Aquadic Bio in bldg DB)	606196.6	4265041.5	19.8	5.49	293.15	15.24	0.9144
TB1	Temporary Building 1	606541.9	4265262.5	17.2	5.49	293.15	15.24	0.9144
CELL_BIO	ITEH Cellular Biology	608560.9	4264005.0	14.9	9.14	293.15	15.24	0.9144
ITEHPATH	ITEH Pathology Clinic	608507.1	4263932.5	14.6	5.49	293.15	15.24	0.9144
ARS_DL1	ARS DL-10/Field Shelter 5; Bovine Shed (Luekemia Lab)	608676.8	4264119.5	15.2	5.49	293.15	15.24	0.9144
COLEA	Cole Fac A	608497.9	4265593.0	16.8	5.49	293.15	15.24	0.9144
COLEB	Cole Fac B	608499.1	4265490.5	16.9	5.49	293.15	15.24	0.9144
COLEC	Cole Fac C	608588.6	4265605.0	16.8	5.49	293.15	15.24	0.9144
TB_31	TB 31	608781.0	4265310.0	16.2	5.49	293.15	15.24	0.9144
TB_33	TB 33	608731.0	4265601.0	17	5.49	293.15	15.24	0.9144
TB_164	TB 164	608491.0	4266068.5	15.2	5.49	293.15	15.24	0.9144
TB_165	TB 165	608491.6	4266056.0	15.2	5.49	293.15	15.24	0.9144
TB_205	TB 205	608403.5	4266045.0	15.2	5.49	293.15	15.24	0.9144
HH1	HH1	607840.6	4266637.1	16.4	5.49	293.15	15.24	0.9144
HH2	HH2	607833.1	4266591.1	16.5	5.49	293.15	15.24	0.9144
HH3	HH3	607844.2	4266544.3	16.5	5.49	293.15	15.24	0.9144
HH6	HH6	607911.6	4266543.9	16.2	5.49	293.15	15.24	0.9144
VMTH	Vet Med Teaching Hospital (VMTH)	607800.8	4265387.0	16.2	12.80	293.15	15.24	0.9144
ARSJ_BRN	ARS Iso Barn J bldg	608500.1	4264891.5	15.9	5.49	293.15	15.24	0.9144
ITEH_AH2	ITEH Animal Housing-2	608501.0	4263952.0	14.6	5.49	293.15	15.24	0.9144

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LEHRLAB	LEHR Lab and Office	608498.9	4264013.0	14.9	5.49	293.15	15.24	0.9144
TOXLAB	ITEH Toxic Pollutant Lab	608558.7	4264031.5	14.9	9.14	293.15	15.24	0.9144
AQUAWEED	Aqua weed lab/ Aq Tox Shelter 5	606024.8	4264512.0	17.2	5.49	293.15	15.24	0.9144
BEE_BIO	Bee Biology	605638.9	4265864.0	18.6	5.49	293.15	15.24	0.9144
IEHR_MED	LEHR CLN MED/Medical Clinic	608490.0	4263920.0	14.6	5.49	293.15	15.24	0.9144
ENGIN_3	Engineering 3 (EU3)	608729.4	4265842.5	16.2	12.80	293.15	15.24	0.9144
TB_196	TB 196 (Primate Center)	603983.4	4266140.0	19.8	5.49	293.15	15.24	0.9144
CRUESS	Cruess Replacement	608666.1	4266639.0	14.6	9.14	293.15	15.24	0.9144
HARHALL	Haring Hall Alteration	608673.4	4266295.0	14.6	9.14	293.15	15.24	0.9144
SCILAB	Science Laboratory Building	608613.5	4266272.5	14.9	15.54	293.15	15.24	0.9144
EVERSON	Everson Hall	609024.1	4266127.5	15.9	9.14	293.15	15.24	0.9144
COMPANML	Center for Companion Animal Health	607915.6	4265268.0	15.9	7.92	293.15	15.24	0.9144
EQNPERF	Equine Performance Laboratory	607683.4	4265268.0	16.7	7.92	293.15	15.24	0.9144
GENOMELS	Genome Launch Space	607885.3	4266136.0	16.8	7.32	293.15	15.24	0.9144
FPMS	FPMS	605455.9	4265657.5	19.2	5.49	293.15	15.24	0.9144
GERMPLSM	Germ Plasm	605337.9	4265742.0	19.2	5.49	293.15	15.24	0.9144
HC2	HC-2	606502.7	4265098.5	19.7	5.49	293.15	15.24	0.9144
MEDSCID	Med Sci D	607675.9	4265545.5	16.5	5.49	293.15	15.24	0.9144
COWELL	Cowell Student Health Center	608664.5	4266779.5	14.6	5.49	293.15	15.24	0.9144
PLANTSCI	Plant and Environmental Sciences	608841.0	4266663.5	14.3	12.80	293.15	15.24	0.9144
HUNTHALL	Hunt Hall	608970.6	4266640.5	14.9	9.14	293.15	15.24	0.9144
FOODSCI	Food Science	608602.1	4266590.0	14.9	9.14	293.15	15.24	0.9144
SURGE3	Surge III	608544.2	4266140.0	14.9	5.49	293.15	15.24	0.9144
TB_147	Temporary Buildling 147	608548.3	4266020.5	15.2	5.49	293.15	15.24	0.9144
TB_161	Temporary Building 161	608472.5	4266065.5	15.2	5.49	293.15	15.24	0.9144
TB_127	Temporary Building 127	608519.1	4264081.0	14.9	5.49	293.15	15.24	0.9144
TB_2	Temporary Building 2	606540.5	4265240.5	17	5.49	293.15	15.24	0.9144
GENOMBIO	Genome & Biomedical Science	607732.5	4265730.5	16.8	16.46	293.15	15.24	0.9144
TB_162	Temporary Building 162	608472.4	4266055.5	15.2	5.49	293.15	15.24	0.9144
<u>Diesel IC Engines</u>								
ICE60SUB	P-17-98 60 Sub (115KV)	608902.1	4265077.0	15.0	2.13	783.18	26.45	0.083
ICEASURG	No Permit Academic Surg	608839.4	4265721.8	16.8	2.11	763.47	13.16	0.064
ICEAMAT	No Permit Advanced Materials	608333.4	4265405.3	16.8	2.06	763.47	16.24	0.057
ICEAQTRT	P-90-94(a) Aquaculture Trout	603786.1	4264809.3	18.8	2.13	783.18	58.87	0.114
ICEAQWL	P-107-95(a) Aquaculture II Well	606056.2	4265063.6	19.5	2.13	783.18	69.86	0.102
ICEARCH	P-54-09 ARCH (rec hall)	608189.0	4266518.5	15.8	3.66	807.79	22.60	0.201
ICEBOWLY	P-94-94(a) Bowley G.H	607720.9	4266038.1	16.8	2.13	783.18	47.06	0.089
ICECCAH	P-118-03 CCAH	607867.6	4265245.8	16.0	2.90	800.40	17.98	0.204
ICECTNEU	No Permit Center for Neurosci	610467.8	4266184.6	14.6	1.83	763.47	13.16	0.064
ICEARTS	P-82-02 Center For the Arts	609057.7	4265657.6	16.7	1.40	688.74	94.26	0.127
ICECHILD	P-2-09 Child Health & Disease	603968.0	4266134.0	19.8	2.97	763.47	12.32	0.080

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ICECOLEB	P-09-01 Cole B	608522.3	4265486.0	16.6	1.45	797.07	74.51	0.102
ICECRSR	P-102-03 Contained Research	605452.3	4265690.9	19.2	4.34	749.85	30.09	0.204
ICECROCK	No Permit Crocker	608843.2	4265905.1	16.2	4.06	763.47	13.16	0.762
ICEDATA	P-08-01 Data Center	608687.3	4265833.6	16.1	2.03	838.74	62.65	0.203
ICETANK1	P-83-02 Dom Grd Water Tank 1	605570.9	4265275.1	20.1	1.42	819.29	71.08	0.102
ICEDMWL2	P-117-03 Dom Well # 2	609178.7	4265778.6	14.9	4.34	749.85	30.09	0.204
ICEDMWL3	P-119-03 Dom Well # 3	608437.9	4265293.4	16.7	1.50	783.18	29.58	0.076
ICEDMWL4	P-103-94(a) Dom Well # 4	608487.7	4264140.6	14.9	2.13	783.18	69.86	0.102
ICEDMWL6	P-95-94(a) Dom Well # 6A	606854.5	4265151.6	18.9	2.13	783.18	69.86	0.102
ICEDMWL7	P-42-97 Dom Well # 7a	604435.2	4264888.8	17.6	2.13	783.18	69.86	0.102
ICEENG2	P-101-94(a) Engineering II	608613.9	4265896.0	15.6	2.03	838.74	62.65	0.203
ICEENG3	P-01-00 Engineering III	608766.1	4265864.8	16.2	2.13	838.74	75.25	0.114
ICEEQUIN	P-02-00 Equine Lab	607638.4	4265530.0	16.6	2.97	660.96	22.89	0.152
ICEESF	P-32-99 ESF	606717.6	4265421.8	17.6	4.57	783.18	72.77	0.203
ICEFIRE	P-89-94(a) Fire/Police	608337.1	4266318.7	15.5	1.45	797.07	74.51	0.102
ICEFOOD	P-51-07 Food Science	608582.8	4266604.7	14.9	1.98	695.96	11.34	0.064
ICEFPMS	P-84-02 FPMS	605460.9	4265744.7	19.2	1.28	740.40	130.02	0.127
ICEGBSF	P-120-03 GBSF	607691.6	4265619.8	16.5	5.69	713.74	19.09	0.405
ICEGENOM	P-114-02 Genome Launch	607895.1	4266085.8	16.5	4.57	844.29	26.35	0.076
ICEHICKY	No Permit Hickey Gym	609103.6	4266654.2	15.0	1.42	763.47	52.63	0.032
ICESWLFT	P-210-95(a) Hutch Sew Lift Sta	607015.9	4266160.2	17.1	2.13	783.18	26.45	0.083
ICEHUTCH	No Permit Hutchison	608755.1	4266379.2	14.6	1.73	763.47	9.14	0.076
ICEECOL	P-115-03 Inst of ecology lab	606179.7	4265070.9	19.8	3.15	730.40	11.92	0.204
ICEITEH	No Permit ITEH (WR Lab)	608507.3	4263997.6	14.8	6.71	763.47	36.55	0.038
ICELIFE	P-54-97 Life Sciences	608469.1	4266315.0	15.1	5.18	783.18	58.21	0.203
ICEMOND	P-50-07 Mondavi RMI	609006.4	4265432.8	16.1	4.27	708.74	18.86	0.253
ICESTAD	P-59-07 Multi use stadium	607841.9	4265963.8	16.6	2.80	769.85	12.21	0.128
ICENEURO	No Permit Neurosci - off campus	610535.7	4266027.3	15.2	7.62	763.47	9.14	0.076
ICERES	P-16-09 New UG RES (Cat)	608999.2	4265601.6	17.5	3.66	788.74	23.33	0.201
ICEFIRST	No Permit Old Fire Station	609339.8	4266227.6	14.2	1.91	763.47	9.14	0.076
ICEPLANT	P-29-96(a0) Physical Plant	608947.7	4265839.1	16.8	2.74	755.40	118.21	0.114
ICEENVIR	P-120-01 Plant Envir Sci	608903.7	4266685.4	14.3	1.27	755.40	52.39	0.203
ICEGEN1	P-50-99(a) Port Gen # 1	609084.5	4266495.1	15.2	2.13	537.85	42.65	0.089
ICEGEN4	No Permit Port Gen # 14	608786.3	4266013.2	15.4	1.91	763.47	13.16	0.064
ICEGEN2	P-51-99(a) Port Gen # 2	606464.6	4265648.5	18.0	2.13	537.85	42.65	0.089
ICEGEN3	P-52-99(a) Port Gen # 3	603627.4	4265120.5	21.3	2.13	537.85	42.65	0.089
ICEGEN7	P-86-01 Port Gen # 7	608683.2	4265831.1	16.1	2.13	855.40	75.68	0.102
ICEGEN8	P-87-01 Port Gen # 8	604804.9	4265787.9	19.5	2.13	855.40	75.68	0.102
ICEANIM1	P-49-07 Pri Animal Hous # 1	604071.5	4266251.4	19.8	3.05	807.79	21.93	0.204
ICEANIML	P-31-98 Primate Animal	604111.4	4266247.3	19.8	2.44	755.40	118.21	0.114
ICEPRCCM	P-32-98 Primate CCM	604195.4	4266075.3	19.8	3.05	755.40	118.21	0.114

UC Davis Point Source Modeling Parameters

ISCST3 Source ID	Source Description	Easting (X) (m)	Northing (Y) (m)	Base Elevation (m)	Stack Height (m)	Temperature (K)	Exit Velocity (m/s)	Stack Diameter (m)
ICEFRZR	P-69-96(a) Primate Freezers	604139.2	4266169.0	19.8	2.44	844.29	46.57	0.102
ICEPLAB	P-102-94(a) Primate Lab	604157.1	4266156.0	19.8	3.66	783.18	43.66	0.203
ICEPQUAR	P-15-98 Primate Quarantine	604237.8	4265923.4	19.8	2.74	1013.74	83.94	0.114
ICEPSEW	No Permit Primate Sew Life Sta	604394.2	4265979.6	19.8	2.46	763.47	25.26	0.064
ICETB184	P-16-98 Primate TB 184	604036.5	4266203.3	18.7	2.74	977.62	82.79	0.114
ICETBN5	P-108-01 Primate TB North # 5	604029.9	4266079.4	19.7	2.36	844.29	23.81	0.083
ICETBS6	P-109-01 Primate TB South # 6	604025.1	4266084.3	19.6	2.36	844.29	23.81	0.083
ICEPARK	P-99-94(a) Quad Parking	609021.0	4266747.8	14.6	2.13	783.18	69.86	0.102
ICERHALL	P-93-94(a) Rec Hall	608194.2	4266518.5	15.8	2.13	783.18	44.09	0.083
ICENEUR1	P-111-95(a) Schl of Med Neurosci	610379.1	4266241.0	14.6	2.13	783.18	26.45	0.083
ICENEUR2	P-123-01 Schl of Med Neurosci	610367.6	4266151.9	14.9	2.36	769.29	44.53	0.083
ICESLAB	P-15-04 Science Lab	608652.5	4266287.5	14.6	3.35	722.07	19.04	0.253
ICESEGDN	P-74-05 Segundo Dinning	608417.9	4266674.4	15.2	2.80	738.74	22.18	0.128
ICESOCSC	P-126-95(a) Social Sci	609275.9	4266599.2	15.5	2.44	1013.74	106.24	0.102
ICESPARK	P-17-02 South Parking	609173.2	4265498.0	15.3	2.44	844.29	82.79	0.076
ICELIFT4	P-92-94(a) Storm Lift # 4	607744.5	4264937.7	15.9	1.52	783.18	77.62	0.152
ICELFT4N	C-09-129 Storm Lift #4 (new)	607749.7	4264932.1	16.3	2.74	966.51	30.46	0.128
ICETARGT	P-71-00 Targeted genomics	608556.2	4264787.3	15.5	2.51	755.40	122.81	0.114
ICETLCOM	P-111-01 Tele Comm.	608410.4	4265996.8	15.2	4.57	770.96	132.30	0.178
ICETHURM	P-91-94(a) Thurman Lab	607645.7	4265421.8	16.8	2.44	755.40	118.21	0.114
ICETXPOL	P-100-94(a) Toxic Pollutant	608636.9	4264019.6	14.9	2.13	783.18	64.68	0.152
ICETURF	P-17-09 TURF	604168.5	4266305.1	19.8	2.29	758.18	28.51	0.101
ICETUPPR	P-121-03 Tupper Load Dock	607704.4	4265619.8	16.5	4.33	763.74	15.49	0.280
ICEUNWL6	P-209-95(a) Util Well 6A	607854.8	4266159.1	16.8	1.42	819.29	71.08	0.102
ICEVEGA	P-07-01 Vega Crops	606290.9	4266161.4	17.7	2.44	755.40	118.21	0.114
ICEVTLAB	P-63-03 Vet Lab	607579.7	4265363.1	16.8	3.81	422.07	21.30	0.128
ICEVMD3	P-52-07 Vet Med 3A	607686.1	4265489.6	16.5	7.92	765.96	14.69	0.305
ICEVMD3A	P-53-07 Vet Med 3A	607686.1	4265464.0	16.5	7.92	765.96	14.69	0.305
ICEWTR	P-59-05 Watershed Sic	608843.2	4265721.8	16.8	3.05	844.29	16.07	0.101
ICEWSTPK	P-38-05 West Entry Park	608322.4	4266348.0	15.5	3.66	842.07	8.60	0.201
ICEWESPI	P-96-94(a) WEPT Influent	608390.2	4265612.5	16.7	2.13	783.18	62.58	0.203
ICEWESPS	P-88-99 WEPT South	608592.9	4264338.7	15.2	3.66	680.40	94.26	0.254

UC Davis Area Source Modeling Parameters

Source ID	Source Description	Easting (X) (m)	Northing (Y) (m)	Base Elevation (m)	Release Height (m)	Easterly Length (m)	Northerly Length (m)	Angle from North
CLOSED_1	Landfill	604241.9	4265353.7	20.7	0.0	160.0	157.6	0
CLOSED_2	Landfill	604241.9	4265196.2	20.7	0.0	160.0	157.6	0
CLOSED_3	Landfill	604241.9	4265039.0	21.0	0.0	160.0	157.6	0
CLOSED_4	Landfill	604241.9	4264881.8	18.5	0.0	160.0	157.6	0
WWTP	Waste Water Treatment Plant	608616.1	4264292.0	15.2	0.0	70.0	90.0	0
GDS_AST	Grounds Above-ground Storage Tank	609154.2	4266061.0	17.6	0.0	10.0	10.0	0
FLEETUST	Fleet Services Underground Storage Tank	608267.4	4265590.0	16.6	0.0	3.0	3.0	0
PRIM_AST	Primate Center Gasoline AST	604018.0	4266187.0	18.5	0.0	3.0	3.0	0
AG_AST	Agricultural Services AST	606664.8	4265265.5	17.1	0.0	3.0	3.0	0
PLT_PATH	Plant Pathology Storage Tank	608396.8	4264314.0	15.2	0.0	3.0	3.0	0
POM_AST	Pomology Above Ground Storage Tank	605416.1	4265858.5	18.9	0.0	3.0	3.0	0
AIRPTAST	Airport Above Ground Storage Tank	605634.0	4265385.5	19.8	0.0	3.0	3.0	0

Appendix E
Environmental Data Resources
Sensitive Receptor Report

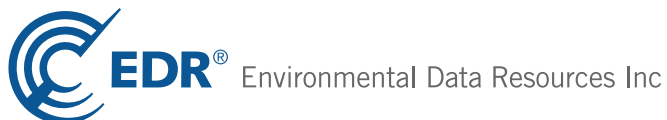
University of California Davis

One Shields Ave
Davis, CA 95616

Inquiry Number: 2790492.1s

June 11, 2010

EDR Offsite Receptor Report



440 Wheelers Farms Road
Milford, CT 06461
Toll Free: 800.352.0050
www.edrnet.com

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Records Searched/Data Currency Tracking Addendum	108

Thank you for your business
Please contact EDR at 1-800-352-0050
with any questions or comments.

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EXECUTIVE SUMMARY

A search of available records was conducted by Environmental Data Resources, Inc. (EDR). The EDR Offsite Receptor Report provides information which may be used to comply with the Clean Air Act Risk Management Program 112-R. "The rule requires that you estimate in the RMP residential populations within the circle defined by the endpoint for your worst-case and alternative release scenarios (i.e., the center of the circle is the point of release and the radius is the distance to the endpoint). In addition, you must report in the RMP whether certain types of public receptors and environmental receptors are within the circles."

The address of the subject property, for which the search was intended, is:

UNIVERSITY OF CALIFORNIA DAVIS
ONE SHIELDS AVE
DAVIS, CA 95616

Distance Searched: 5.000 miles from subject property

RECEPTOR SUMMARY

An X indicates the presence of the receptor within the search radius.

Residential Population

Estimated population within search radius: 64601 persons.

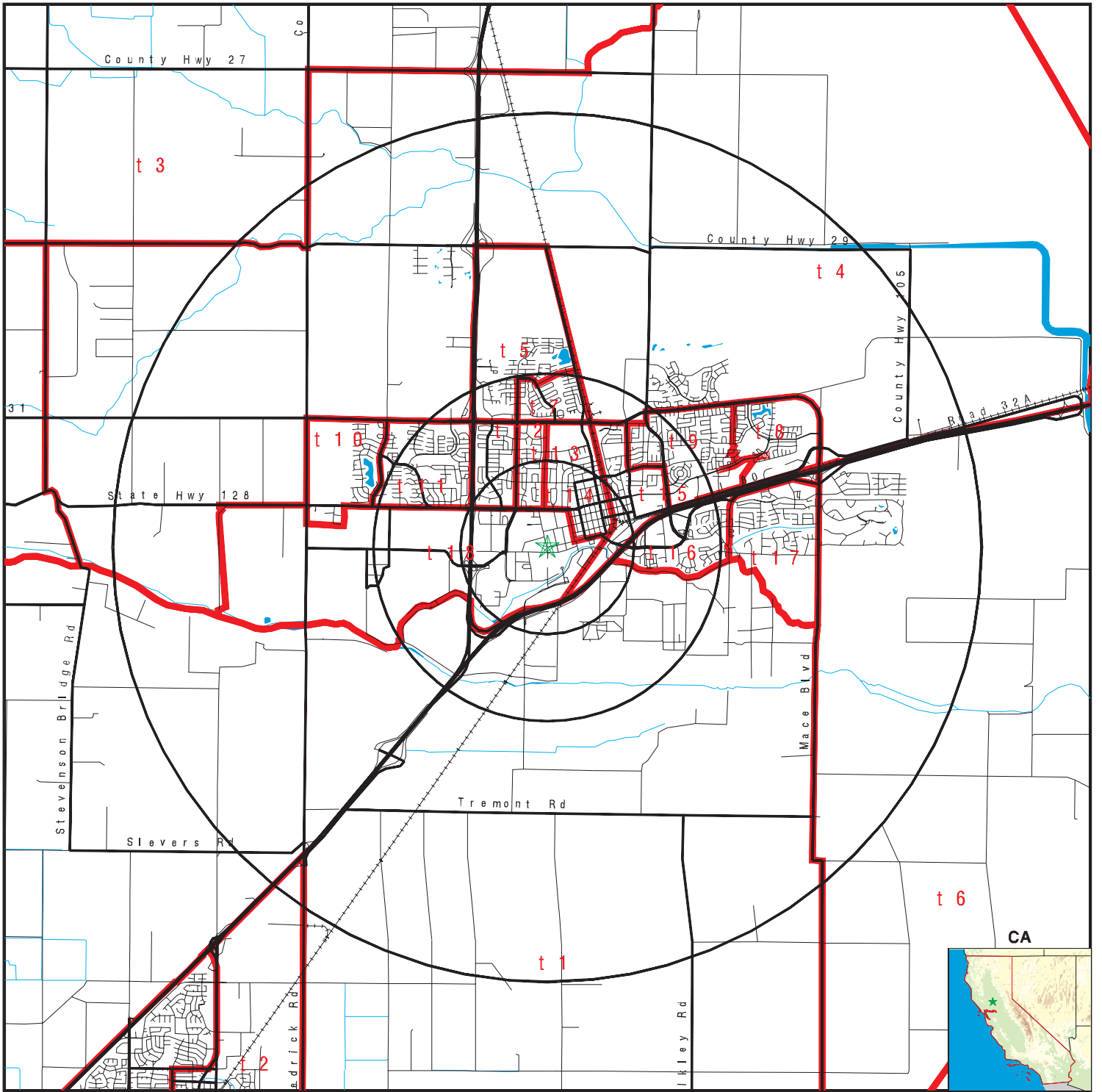
Other Public Receptors

Type	Within Search Radius	Sites Total
Day Care Centers:	<input checked="" type="checkbox"/>	96
Medical Centers:	<input type="checkbox"/>	
Nursing Homes:	<input checked="" type="checkbox"/>	3
Schools:	<input checked="" type="checkbox"/>	21
Hospitals:	<input checked="" type="checkbox"/>	31
Colleges:	<input type="checkbox"/>	
Arena:	<input type="checkbox"/>	
Prison:	<input type="checkbox"/>	

Environmental Receptors

Type	Within Search Radius	Sites Total
Federal Land:	<input type="checkbox"/>	

CENSUS MAP - 2790492.1s



- ★ Target Property
- ⚡ Roads
- 🌊 Waterways
- 📊 Census Tracts

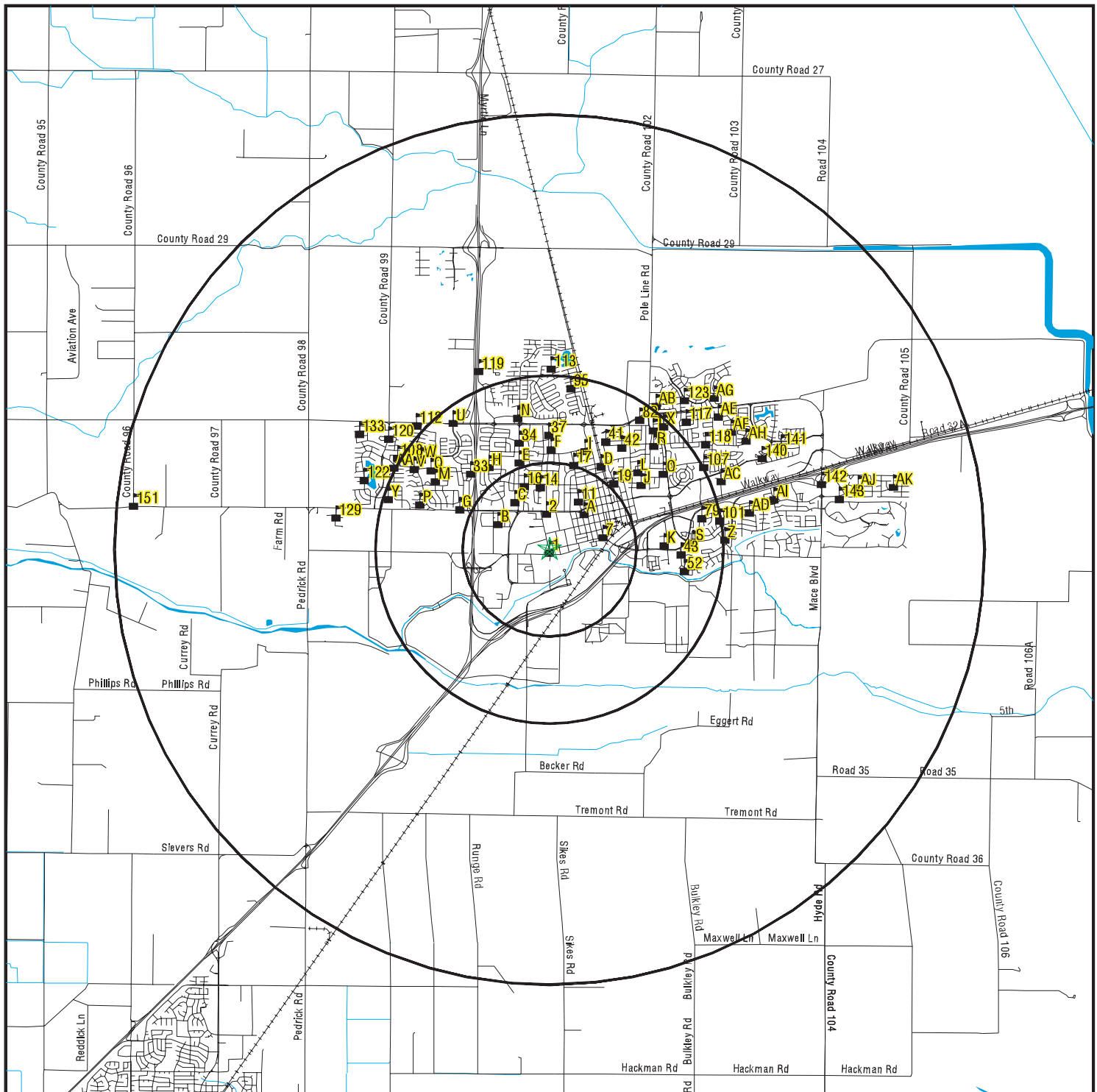


TARGET PROPERTY:	University of California Davis	CUSTOMER:	ERM - West, Inc.
ADDRESS:	One Shields Ave	CONTACT:	Vicki Hoffman
CITY/STATE/ZIP:	Davis CA 95616	INQUIRY #:	2790492.1s
LAT/LONG:	38.5399 / 121.7521	DATE:	June 11, 2010 11:37 am

CENSUS FINDINGS

Map ID	Tract Number	Total Population	Population in Radius	Total Area(sq.mi.)	Area in Radius(sq.mi.)
T1	2533.00	2834	473.3	171.28	28.61
T2	2534.02	5561	85.3	6.46	0.10
T3	0112.06	2929	8.1	105.02	0.29
T4	0105.05	2961	1442.8	52.96	25.81
T5	0105.10	5413	5413.0	1.78	1.78
T6	0105.06	2877	490.6	42.64	7.27
T7	0105.09	3529	3529.0	0.41	0.41
T8	0106.05	2836	2836.0	0.86	0.86
T9	0106.06	7678	7678.0	1.12	1.12
T10	0105.08	2520	2520.0	0.95	0.95
T11	0105.07	8683	8683.0	1.05	1.05
T12	0107.03	6619	6619.0	0.51	0.51
T13	0107.04	2165	2165.0	0.33	0.33
T14	0107.01	4549	4549.0	0.80	0.80
T15	0106.02	5177	5177.0	0.80	0.80
T16	0106.08	4864	4864.0	0.92	0.92
T17	0106.07	3650	3650.0	1.31	1.31
T18	0105.01	4418	4418.0	5.22	5.22

RECEPTOR MAP - 2790492.1s



- ★ Target Property
- Roads
- Waterways
- Environmental or Public Receptor
- Federal Lands Linear Features
- Federal Lands Area



TARGET PROPERTY: University of California Davis
ADDRESS: One Shields Ave
CITY/STATE/ZIP: Davis CA 95616
LAT/LONG: 38.5399 / 121.7521

CUSTOMER: ERM - West, Inc.
CONTACT: Vicki Hoffman
INQUIRY #: 2790492.1s
DATE: June 11, 2010 11:37 am

MAP FINDINGS

Map ID Direction Distance Distance (ft.) Elevation	Site	EDR ID Database
1 SW 0-1/8 mi 29 Higher	Hospital type: 01 Num of times COO: 00 Owner date: Not Reported City: DAVIS Has plan of corr: Not Reported Compliance status: A SSA county code: 670 Cross ref number: Not Reported FMS survey date: Not Reported Current survey date: 20030910 Medicare/Medicaid: 1 Facility name: COWELL STUDENT HEALTH CENTER Intermediary/Carrier: Not Reported Medicaid number: Not Reported Participation date: 19920901 Prior COO date: Not Reported Prior carrier: Not Reported Provider ID: 05D0862297 Record Status: A Region code: 09 Is Partial Record: Not Reported state abbrev: CA ssa state: 05 state region cd: M2 street address: 1 SHIELDS AVENUE Phone num: 5307522300 Termination reason: 00 Term Date: 20080322 Purpose of action: 2 Provider control: 07 Zip: 95616 Fips state: 06 Fips cnty: 113 SSA MSA: 499 SSA MSA size code: B Date accredited: Not Reported Accred expire date: Not Reported Accred Org: Not Reported Num beds: 0000 Num cert beds: 0000 Source: US_HOSPITAL_POSCLIA Edr id: SRHO20070143709	SRHO20070143709 AHA Hospitals
2 North 1/4-1/2 mi 2350 Higher	Hospital type: 01 Num of times COO: 00 Owner date: Not Reported City: DAVIS Has plan of corr: 1 Compliance status: A SSA county code: 670 Cross ref number: Not Reported FMS survey date: Not Reported Current survey date: 19940927	SRHO20070135397 AHA Hospitals

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Medicare/Medicaid: 1
 Facility name: WOODLAND CLINIC MEDICAL GROUP
 Intermediary/Carrier: Not Reported
 Medicaid number: Not Reported
 Participation date: 19920901
 Prior COO date: Not Reported
 Prior carrier: Not Reported
 Provider ID: 05D0613760
 Record Status: A
 Region code: 09
 Is Partial Record: Not Reported
 state abbrev: CA
 ssa state: 05
 state region cd: M2
 street address: 501 OAK AVE
 Phone num: 9167586666
 Termination reason: 12
 Term Date: 19941028
 Purpose of action: 1
 Provider control: 04
 Zip: 95616
 Fips state: 06
 Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0000
 Num cert beds: 0000
 Source: US_HOSPITAL_POSCLIA
 Edr id: SRHO20070135397

A3
 NE
 1/2-1 mi
 3112
 Higher

EDR ID: SRDCCA200745173
 Facility number: 570300152
 Facility name: COMMUNITY CHURCH NURSERY SCHOOL
 Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 850
 Facility status code: 03
 Address: 412 C STREET
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 412 C STREET
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: DAVIS COMMUNITY CHURCH
 Licensee type: C
 License effective date: 950517
 License expiration date: Not Reported
 License issue date: Not Reported

SRDCCA200745173
 Daycare

MAP FINDINGS

Map ID
Direction
Distance
Distance (ft.)
Elevation

Site

EDR ID
Database

Program type: "MAXIMUM CAPACITY: 24 AMBULATORY CHILDREN, AGES 2-6 YEARS. NO MORE THAN 12 SCHOOL AGE CHILDREN IN ATTENDANCE AT ANY TIME."
 "

Original app. received date: Not Reported
 Facility closed date: Not Reported
 Mailing address: 412 C ST
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "VAN KESSEL, ELISABETH"
 Facility capacity: 24
 Type of clients served: 950
 Facility phone: 5307582940

A4 NE 1/2-1 mi 3153 Higher	Ncessch: Schname05: Mstreet05: Mcity05: Mstate05: Mzip05: Mzip405: Member05: Phone05: Locale05: Type05: Level05: Gslo05: Gshi05: Edr id:	061062010344 DAVIS SCHOOL FOR INDEPENDENT STUDY 526 B ST. DAVIS CA 95616 3811 198 (530) 757-5333 3 4 4 KG 12 SRPU20071014155	SRPU20071014155 Public Schools
--	--	--	-----------------------------------

B5 WNW 1/2-1 mi 3226 Higher	EDR ID: Facility number: Facility name: Facility eval. code: Facility office number: Facility county number: Facility type code: Facility status code: Address: City: State: Zip: Alt. address: City: State: Zip: Facility investor: Licensee type: License effective date: License expiration date: License issue date: Program type:	SRDCCA200742381 573604200 LA RUE PARK CHILD DEVELOPMENT CENTER 0303 03 57 830 03 50 ATRIUM WAY DAVIS CA 95616 50 ATRIUM WAY DAVIS CA 95616 CAMPUS CHILD CARE INC. A 981106 Not Reported 981106 LICENSED TO SERVE INFANTS AGE 0 TO 18 MONTHS OLD.	SRDCCA200742381 Daycare
---	---	---	----------------------------

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Original app. received date: 981015
 Facility closed date: Not Reported
 Mailing address: 50 ATRIUM WAY
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "STAPLETON, TARA"
 Facility capacity: 15
 Type of clients served: 955
 Facility phone: 5307538716

B6
 WNW
 1/2-1 mi
 3226
 Higher

EDR ID: SRDCCA200750612
 Facility number: 573604201
 Facility name: LA RUE PARK CHILD DEVELOPMENT CENTER
 Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 850
 Facility status code: 03
 Address: 50 ATRIUM WAY
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 50 ATRIUM WAY
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: CAMPUS CHILD CARE INC.
 Licensee type: A
 License effective date: 981106
 License expiration date: Not Reported
 License issue date: 981106
 Program type: LICENSED TO SERVE 60 CHILDREN AGE 2 YEARS TO ENTRY INTO FIRST GRADE. TODDLER OPTION PROGRAM CAP. IS 20 FOR CHILDREN AGE 18 TO 30 MONTHS. WAIVER ON FILE FOR SHARED OUTDOOR SPACE.

SRDCCA200750612
 Daycare

Original app. received date: 981015
 Facility closed date: Not Reported
 Mailing address: 50 ATRIUM WAY
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: TARA STAPLETON
 Facility capacity: 80
 Type of clients served: 950
 Facility phone: 5307538716

7
 ENE
 1/2-1 mi
 3364
 Higher

Hospital type: 01
 Num of times COO: 00
 Owner date: Not Reported
 City: DAVIS
 Has plan of corr: Not Reported
 Compliance status: Not Reported

SRHO20070153692
 AHA Hospitals

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

SSA county code: 670
 Cross ref number: Not Reported
 FMS survey date: Not Reported
 Current survey date: Not Reported
 Medicare/Medicaid: Not Reported
 Facility name: YOLO HOSPICE
 Intermediary/Carrier: Not Reported
 Medicaid number: Not Reported
 Participation date: 19990726
 Prior COO date: Not Reported
 Prior carrier: Not Reported
 Provider ID: 05D0963217
 Record Status: A
 Region code: 09
 Is Partial Record: Y
 state abbrev: CA
 ssa state: 05
 state region cd: LAB
 street address: 132 E STREET
 Phone num: 5307585566
 Termination reason: 00
 Term Date: 20070725
 Purpose of action: Not Reported
 Provider control: 02
 Zip: 95616
 Fips state: 06
 Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0000
 Num cert beds: 0000
 Source: US_HOSPITAL_POSCLIA
 Edr id: SRHO20070153692

C8
 NNW
 1/2-1 mi
 3521
 Higher

Hospital type: 01
 Num of times COO: 01
 Owner date: 19980326
 City: DAVIS
 Has plan of corr: Not Reported
 Compliance status: A
 SSA county code: 670
 Cross ref number: Not Reported
 FMS survey date: Not Reported
 Current survey date: 19941219
 Medicare/Medicaid: 1
 Facility name: DAVIS PHYSICAL THERAPY/MATRIX
 Intermediary/Carrier: 52280
 Medicaid number: Not Reported
 Participation date: 19951219
 Prior COO date: Not Reported
 Prior carrier: Not Reported

SRHO20070011363
 AHA Hospitals

MAP FINDINGS

Map ID
Direction
Distance
Distance (ft.)
Elevation

Site

EDR ID
Database

Provider ID: 056744
 Record Status: A
 Region code: 09
 Is Partial Record: Not Reported
 state abbrev: CA
 ssa state: 05
 state region cd: S1
 street address: 635 ANDERSON RD #11
 Phone num: 9167561010
 Termination reason: 01
 Term Date: 19981130
 Purpose of action: 1
 Provider control: 06
 Zip: 95616
 Fips state: 06
 Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0000
 Num cert beds: 0000
 Source: US_HOSPITAL_POSOTHER
 Edr id: SRHO20070011363

C9
 NNW
 1/2-1 mi
 3521
 Higher

Hospital type: 01
 Num of times COO: 00
 Owner date: Not Reported
 City: DAVIS
 Has plan of corr: 1
 Compliance status: A
 SSA county code: 670
 Cross ref number: Not Reported
 FMS survey date: Not Reported
 Current survey date: 19950418
 Medicare/Medicaid: 1
 Facility name: RANDELL LABORATORIES
 Intermediary/Carrier: 00542
 Medicaid number: Not Reported
 Participation date: 19920901
 Prior COO date: Not Reported
 Prior carrier: Not Reported
 Provider ID: 05D0613702
 Record Status: A
 Region code: 09
 Is Partial Record: Not Reported
 state abbrev: CA
 ssa state: 05
 state region cd: M1
 street address: 635 ANDERSON RD #2
 Phone num: Not Reported
 Termination reason: 04
 Term Date: 19950413

SRHO20070135384
 AHA Hospitals

MAP FINDINGS

Map ID
Direction
Distance
Distance (ft.)
Elevation

Site

EDR ID
Database

Purpose of action: 2
 Provider control: 04
 Zip: 95616
 Fips state: 06
 Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0000
 Num cert beds: 0000
 Source: US_HOSPITAL_POSCLIA
 Edr id: SRHO20070135384

C10
 NNW
 1/2-1 mi
 3521
 Higher

Hospital type: 01
 Num of times COO: 00
 Owner date: Not Reported
 City: DAVIS
 Has plan of corr: Not Reported
 Compliance status: Not Reported
 SSA county code: 670
 Cross ref number: Not Reported
 FMS survey date: Not Reported
 Current survey date: Not Reported
 Medicare/Medicaid: Not Reported
 Facility name: JAMES A KENNEDY MD
 Intermediary/Carrier: Not Reported
 Medicaid number: Not Reported
 Participation date: 19921228
 Prior COO date: Not Reported
 Prior carrier: Not Reported
 Provider ID: 05D0613699
 Record Status: A
 Region code: 09
 Is Partial Record: Not Reported
 state abbrev: CA
 ssa state: 05
 state region cd: LAB
 street address: 635 ANDERSON RD #5
 Phone num: 9167532841
 Termination reason: 01
 Term Date: 19951231
 Purpose of action: Not Reported
 Provider control: 04
 Zip: 95616
 Fips state: 06
 Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0000

SRHO20070137446
 AHA Hospitals

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Num cert beds: 0000
 Source: US_HOSPITAL_POSCLIA
 Edr id: SRHO20070137446

11		SRPU20071014147
NNE	Ncessch: 061062001179	Public Schools
1/2-1 mi	Schname05: KING (MARTIN LUTHER) HIGH (CONTINUATION)	
3553	Mstreet05: 635 B ST.	
Higher	Mcity05: DAVIS	
	Mstate05: CA	
	Mzip05: 95616	
	Mzip405: Not Reported	
	Member05: 69	
	Phone05: (530) 757-5425	
	Locale05: 3	
	Type05: 4	
	Level05: 3	
	Gslo05: 11	
	Gshi05: 12	
	Edr id: SRPU20071014147	

B12		SRDCCA200742416
WNW	EDR ID: SRDCCA200742416	Daycare
1/2-1 mi	Facility number: 570311582	
3916	Facility name: RUSSELL PARK CHILD DEVELOPMENT CENTER (INFANTS)	
Higher	Facility eval. code: 0303	
	Facility office number: 03	
	Facility county number: 57	
	Facility type code: 830	
	Facility status code: 03	
	Address: 400 RUSSELL PARK	
	City: DAVIS	
	State: CA	
	Zip: 95616	
	Alt. address: 400 RUSSELL PARK	
	City: DAVIS	
	State: CA	
	Zip: 95616	
	Facility investor: CAMPUS CHILD CARE INC.	
	Licensee type: A	
	License effective date: 940904	
	License expiration date: Not Reported	
	License issue date: 880904	
	Program type: "MAXIMUM CAPACITY: 12 INFANTS (0-2 YEARS). THIS IS A COMBINATION CENTER, OVERALL CAPACITY IS NOT TO EXCEED 92 CHILDREN AT ONE TIME."	
	Original app. received date: 880817	
	Facility closed date: Not Reported	
	Mailing address: 400 RUSSELL PARK	
	Mailing city: DAVIS	
	Mailing state: CA	
	Mailing zip: 95616	
	Contact person: "CORRY, FRAN"	
	Facility capacity: 12	

MAP FINDINGS

Map ID
Direction
Distance
Distance (ft.)
Elevation

Site

EDR ID
Database

Type of clients served: 955
Facility phone: 5307532487

B13		SRDCCA200748387
WNW	EDR ID:	SRDCCA200748387
1/2-1 mi	Facility number:	570308535
3916	Facility name:	RUSSELL PARK CHILD DEVELOPMENT CENTER
Higher	Facility eval. code:	0303
	Facility office number:	03
	Facility county number:	57
	Facility type code:	850
	Facility status code:	03
	Address:	400 RUSSELL PARK
	City:	DAVIS
	State:	CA
	Zip:	95616
	Alt. address:	400 RUSSELL PARK
	City:	DAVIS
	State:	CA
	Zip:	95616
	Facility investor:	CAMPUS CHILD CARE INC.
	Licensee type:	A
	License effective date:	940904
	License expiration date:	Not Reported
	License issue date:	Not Reported
	Program type:	"MAXIMUM CAPACITY: 80 AMBULATORY PRESCHOOL CHILDREN. CAPACITY INCLUDES A TODDLER OPTION PROGRAM FOR A CAPACITY OF 20. THIS IS A COMBINATION CENTER, OVERALL CAPACITY NOT TO EXCEED 92 CHILDREN "AT ONE TIME.
	Original app. received date:	850612
	Facility closed date:	Not Reported
	Mailing address:	400 RUSSELL PARK
	Mailing city:	DAVIS
	Mailing state:	CA
	Mailing zip:	95616
	Contact person:	"CHORDAS, TONYA "
	Facility capacity:	80
	Type of clients served:	950
	Facility phone:	5307532487

14		SRDCCA200748388
North	EDR ID:	SRDCCA200748388
1/2-1 mi	Facility number:	570310219
3989	Facility name:	DAVIS PARENT NURSERY SCHOOL #2
Higher	Facility eval. code:	0303
	Facility office number:	03
	Facility county number:	57
	Facility type code:	850
	Facility status code:	03
	Address:	426 W 8TH STREET
	City:	DAVIS
	State:	CA
	Zip:	95616
	Alt. address:	426 W 8TH STREET

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: DAVIS PARENT NURSERY SCHOOL ASSOCIATION
 Licensee type: A
 License effective date: 930601
 License expiration date: Not Reported
 License issue date: 870901
 Program type: "SCHOOL AGE WAIVER ON FILE. 5 DAY MORNING SESSION M THRU F 8:45 A.M.-
 12:15 P.M. 3 DAY AFTERNOON SESSION T, W, TH 12:30 P.M.- 4:00 P.M.
 (M & F AFTERNOON PROGRAM 11:45 A.M.- 3:15 P.M.)
 "

Original app. received date: 870312
 Facility closed date: Not Reported
 Mailing address: 426 W 8TH STREET
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "DOUGLAS, KATHY "
 Facility capacity: 36
 Type of clients served: 950
 Facility phone: 5307575377

C15		SRDCCA200752884
NW	EDR ID: SRDCCA200752884	Daycare
1/2-1 mi	Facility number: 573609244	
4241	Facility name: INTERNATIONAL PARENT-CHILD LEARNING CENTER	
Higher	Facility eval. code: 0303	
	Facility office number: 03	
	Facility county number: 57	
	Facility type code: 850	
	Facility status code: 03	
	Address: 640 HAWTHORNE LANE	
	City: DAVIS	
	State: CA	
	Zip: 95616	
	Alt. address: 640 HAWTHORNE LANE	
	City: DAVIS	
	State: CA	
	Zip: 95616	
	Facility investor: INTERNATIONAL HOUSE - DAVIS	
	Licensee type: C	
	License effective date: 30902	
	License expiration date: Not Reported	
	License issue date: 030902	
	Program type: LICENSED TO SERVE CHILDREN FROM AGE 2 YEARS TO ENTRY INTO FIRST GRADE IN ROOMS 2 AND 3.	
	Original app. received date: 030529	
	Facility closed date: Not Reported	
	Mailing address: 10 COLLEGE PARK AVENUE	
	Mailing city: DAVIS	
	Mailing state: CA	
	Mailing zip: 95616	
	Contact person: "BARRATT, ELAINE "	
	Facility capacity: 21	

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Type of clients served: 950
 Facility phone: 5307560444

16 NNW EDR ID: SRDCCA200726294 SRDCCA200726294
 1/2-1 mi Facility number: 573610348 Daycare
 4312 Facility name: "MOORE, JANET "

Higher Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 810
 Facility status code: 03
 Address: 811 NORTH CAMPUS WY
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 811 NORTH CAMPUS WY
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "MOORE, JANET "
 Licensee type: A
 License effective date: 40903
 License expiration date: Not Reported
 License issue date: 040903
 Program type: "MAXIMUM CAPACITY: 6 CHILDREN WITH NO MORE THAN 3 INFANTS, OR 4 INFANTSONLY, OR CAPACITY 8 CHILDREN WHEN 2 ARE AT LEAST 6 YEARS OF AGE WITH AMAXIMUM OF 2 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED. ""OFF LIMITS: GARAGE, MASTER BEDROOM, SIDEYARDS."
 "

Original app. received date: 040810
 Facility closed date: Not Reported
 Mailing address: 811 NORTH CAMPUS WY
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "MOORE, JANET "
 Facility capacity: 8
 Type of clients served: 960
 Facility phone: 5307560719

17 NNE Pss school id: 00076116 SRPR20051023218
 1-2 mi Pss inst: ST JAMES ELEMENTARY SCHOOL Private Schools
 5487 Lograde: K

Higher Higrade: 8
 Pss address: 1215 B STREET
 Pss city: DAVIS
 Pss county no: 113
 Pss county fips: 06113
 Pss stabb: CA
 Pss fips: 06
 Pss zip5: 95616
 Pss phone: 5307563946

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Pss sch days:	185
Pss stu day hrs:	6.5
Pss library:	Yes
Pss enroll ug:	Not Reported
Pss enroll pk:	Not Reported
Pss enroll k:	30
Pss enroll 1:	32
Pss enroll 2:	33
Pss enroll 3:	34
Pss enroll 4:	34
Pss enroll 5:	34
Pss enroll 6:	31
Pss enroll 7:	34
Pss enroll 8:	32
Pss enroll 9:	Not Reported
Pss enroll 10:	Not Reported
Pss enroll 11:	Not Reported
Pss enroll 12:	Not Reported
Pss enroll t:	294
Pss enroll tk12:	294
Pss race ai:	0
Pss race as:	20
Pss race h:	50
Pss race b:	5
Pss race w:	219
Pss fte teach:	Not Reported
Pss locale:	3
Pss coed:	1
Pss type:	1
Pss level:	1
Pss relig:	1
Pss comm type:	2
Pss indian pct:	0
Pss asian pct:	6.8
Pss hisp pct:	17.01
Pss black pct:	1.7
Pss white pct:	74.49
Pss stdtch rt:	Not Reported
Pss orient:	1
Pss county name:	YOLO
Pss assoc 1:	National Catholic Educational Association (NCEA)
Pss assoc 2:	Not Reported
Pss assoc 3:	Not Reported
Pss assoc 4:	Not Reported
Pss assoc 5:	Not Reported
Pss assoc 6:	Not Reported
Pss assoc 7:	Not Reported
Source:	NCESDATA_E72D09B4
Edr id:	SRPR20051023218

D18			
NNE	EDR ID:	SRDCCA200745675	
1-2 mi	Facility number:	573603995	
5694	Facility name:	DISCOVERY PRESCHOOL	
Higher	Facility eval. code:	0303	

SRDCCA200745675
 Daycare

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Facility office number: 03
 Facility county number: 57
 Facility type code: 850
 Facility status code: 03
 Address: 1020 F ST.
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 1020 F ST.
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "SCHUSTER, SUSAN"
 Licensee type: A
 License effective date: 980928
 License expiration date: Not Reported
 License issue date: 980928
 Program type: 50 PRESCHOOL CHILDREN
 Original app. received date: 980729
 Facility closed date: Not Reported
 Mailing address: 1020 F ST.
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "SCHUSTER, SUSAN"
 Facility capacity: 50
 Type of clients served: 950
 Facility phone: 5307562231

19
 NE
 1-2 mi
 5708
 Higher

EDR ID: SRDCCA200723458
 Facility number: 573609348
 Facility name: "MURRAY-CLARK, JAMIE"
 Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 810
 Facility status code: 03
 Address: 731 J STREET
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 731 J STREET
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "MURRAY-CLARK, JAMIE"
 Licensee type: A
 License effective date: 40224
 License expiration date: Not Reported
 License issue date: 040224
 Program type: INACTIVE LICENSE 3/1/04--12/31/06
 Original app. received date: 030705
 Facility closed date: Not Reported
 Mailing address: 731 J STREET

SRDCCA200723458
 Daycare

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "MURRAY-CLARK, JAMIE "
 Facility capacity: 14
 Type of clients served: 960
 Facility phone: 5307534033

E20		SRDCCA200754391
NNW	EDR ID: SRDCCA200754391	Daycare
1-2 mi	Facility number: 573605861	
5752	Facility name: CESAR CHAVEZ STATE PRESCHOOL	
Higher	Facility eval. code: 0303	
	Facility office number: 03	
	Facility county number: 57	
	Facility type code: 850	
	Facility status code: 03	
	Address: 1221 ANDERSON ROAD	
	City: DAVIS	
	State: CA	
	Zip: 95616	
	Alt. address: "851 E, HAMILTON AVE. STE. 200 "	
	City: CAMPBELL	
	State: CA	
	Zip: 95008	
	Facility investor: CHILD DEVELOPMENT CENTERS	
	Licensee type: C	
	License effective date: 919	
	License expiration date: Not Reported	
	License issue date: 000919	
	Program type: LICENSE TO SERVE CHILDREN 3 YEARS THRU KINDERGARTEN M-F 8:30-11:30AM & 12:30-3:30PM IN THE STUDIO ROOM OR PLAY HOUSE ROOM 30.	
	Original app. received date: 000810	
	Facility closed date: Not Reported	
	Mailing address: "851 E, HAMILTON AVE. STE. 200 "	
	Mailing city: CAMPBELL	
	Mailing state: CA	
	Mailing zip: 95008	
	Contact person: "HAZLETT, JANE "	
	Facility capacity: 31	
	Type of clients served: 950	
	Facility phone: 5307533808	

E21		SRDCCA200743917
NNW	EDR ID: SRDCCA200743917	Daycare
1-2 mi	Facility number: 570310386	
5752	Facility name: CESAR CHAVEZ SCHOOL AGE CDC	
Higher	Facility eval. code: 0303	
	Facility office number: 03	
	Facility county number: 57	
	Facility type code: 840	
	Facility status code: 03	
	Address: 1221 ANDERSON ROAD	
	City: DAVIS	

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

State: CA
 Zip: 95616
 Alt. address: 1221 ANDERSON ROAD
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: CHILD DEVELOPMENT CENTERS
 Licensee type: C
 License effective date: 950228
 License expiration date: Not Reported
 License issue date: Not Reported
 Program type: "MAXIMUM CAPACITY: 124 SCHOOL-AGE CHILDREN. NO MORE THAN 66 CHILDREN IN THE MAIN PORTABLE, AND NO MORE THAN 31 CHILDREN IN RM.30&31 PORTABLE/STUDIO. AN ADDITIONAL 27 CHILDREN OR COMBINATIONS NOT TO EXCEED POSTED CAPACITY IN THE MULTI-PURPOSE ROOM.
 Original app. received date: 870604
 Facility closed date: Not Reported
 Mailing address: "851 E. HAMILTON AVE., STE 200 "
 Mailing city: CAMPBELL
 Mailing state: CA
 Mailing zip: 95008
 Contact person: "MONROE, MARY ALLISON "
 Facility capacity: 124
 Type of clients served: 950
 Facility phone: 5307533808

E22 NNW 1-2 mi 5752 Higher	Ncessch: Schname05: Mstreet05: Mcity05: Mstate05: Mzip05: Mzip405: Member05: Phone05: Locale05: Type05: Level05: G slo05: Gshi05: Edr id:	061062001183 CESAR CHAVEZ ELEMENTARY 1221 ANDERSON RD. DAVIS CA 95616 1899 568 (530) 757-5490 3 1 1 KG 06 SRPU20071014151	SRPU20071014151 Public Schools
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F23 North 1-2 mi 5812 Higher	Ncessch: Schname05: Mstreet05: Mcity05: Mstate05: Mzip05: Mzip405: Member05: Phone05: Locale05:	061062001176 DAVIS SENIOR HIGH 315 WEST 14TH ST. DAVIS CA 95616 1914 1743 (530) 757-5400 3	SRPU20071014144 Public Schools
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MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Type05: 1
 Level05: 3
 Gslo05: 10
 Gshi05: 12
 Edr id: SRPU20071014144

G24		SRDCCA200747358
WNW	EDR ID:	SRDCCA200747358
1-2 mi	Facility number:	573604596
5846	Facility name:	APPLEGATE NURSERY
Higher	Facility eval. code:	0303
	Facility office number:	03
	Facility county number:	57
	Facility type code:	850
	Facility status code:	03
	Address:	1701 RUSSELL BOULEVARD
	City:	DAVIS
	State:	CA
	Zip:	95616
	Alt. address:	2787 BELMONT DRIVE
	City:	DAVIS
	State:	CA
	Zip:	95616
	Facility investor:	"APPLEGATE NURSERY SCHOOL, INC. "
	Licensee type:	C
	License effective date:	990608
	License expiration date:	Not Reported
	License issue date:	990608
	Program type:	"LICENSED TO SERVE CHILDREN AGES 2 YEARS TO 5 YEARS (OPEN 8-5 DURING PUBLIC SCHOOL YEAR). 8:00-12:00 IN JUNE AND JULY, CLOSED IN AUGUST. "
	Original app. received date:	990416
	Facility closed date:	Not Reported
	Mailing address:	2787 BELMONT DRIVE
	Mailing city:	DAVIS
	Mailing state:	CA
	Mailing zip:	95616
	Contact person:	"HODGES, NANCY "
	Facility capacity:	30
	Type of clients served:	950
	Facility phone:	5307584850

G25		SRDCCA200735270
WNW	EDR ID:	SRDCCA200735270
1-2 mi	Facility number:	573613039
6261	Facility name:	"COOK, NOAH "
Higher	Facility eval. code:	0303
	Facility office number:	03
	Facility county number:	57
	Facility type code:	810
	Facility status code:	03
	Address:	1824 ALAMEDA AVE
	City:	DAVIS
	State:	CA

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Zip: 95616
 Alt. address: 1824 ALAMEDA AVE
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "COOK, NOAH"
 Licensee type: A
 License effective date: 60331
 License expiration date: Not Reported
 License issue date: 060331
 Program type: MAX. CAP(WHEN THERE IS AN ASSISTANT PRESENT): 12 - NO MORE THAN 4
 INFANTS. CAP 14 - NO MORE THAN 3 INFANTS. 1 CHILD IN
 KINDERGARTEN OR ELEMENTARY SCHOOL AND 1 CHILD AT LEAST AGE 6."OFF
 LIMITS:MSTR BDRM& CHILD RM, GARAGE AND SIDE YARD."
 Original app. received date: 060313
 Facility closed date: Not Reported
 Mailing address: 1824 ALAMEDA AVE
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "COOK, NOAH"
 Facility capacity: 14
 Type of clients served: 960
 Facility phone: 5307566235

H26			SRDCCA200743657
NW	EDR ID:	SRDCCA200743657	Daycare
1-2 mi	Facility number:	570312670	
6339	Facility name:	ROBERT E. WILLETT SCHOOL AGE CDC	
Higher	Facility eval. code:	0303	
	Facility office number:	03	
	Facility county number:	57	
	Facility type code:	840	
	Facility status code:	03	
	Address:	1207 SYCAMORE	
	City:	DAVIS	
	State:	CA	
	Zip:	95616	
	Alt. address:	1207 SYCAMORE	
	City:	DAVIS	
	State:	CA	
	Zip:	95616	
	Facility investor:	CHILD DEVELOPMENT CENTERS	
	Licensee type:	C	
	License effective date:	951201	
	License expiration date:	Not Reported	
	License issue date:	891201	
	Program type:	LICENSED TO SERVE UP TO 56 CHILDREN ENROLLED IN KINDERGARTEN AND ABOVE IN PORTABLE CLASSROOMS AND UP TO 28 SCHOOL-AGE CHILDREN IN CLASSROOM G-32.	
	Original app. received date:	890901	
	Facility closed date:	Not Reported	
	Mailing address:	"851 E. HAMILTON AVE., STE 200"	
	Mailing city:	CAMPBELL	
	Mailing state:	CA	

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Mailing zip: 95008
 Contact person: "ANGELO, MARISSA"
 Facility capacity: 84
 Type of clients served: 950
 Facility phone: 5307588342

H27			SRPU20071014152
NW	Ncessch:	061062001184	Public Schools
1-2 mi	Schname05:	ROBERT E. WILLETT ELEMENTARY	
6339	Mstreet05:	1207 SYCAMORE LN.	
Higher	Mcity05:	DAVIS	
	Mstate05:	CA	
	Mzip05:	95616	
	Mzip405:	1799	
	Member05:	518	
	Phone05:	(530) 757-5460	
	Locale05:	3	
	Type05:	1	
	Level05:	1	
	Gslo05:	KG	
	Gshi05:	06	
	Edr id:	SRPU20071014152	

D28			SRDCCA200731497
NNE	EDR ID:	SRDCCA200731497	Daycare
1-2 mi	Facility number:	573611174	
6446	Facility name:	"MALHOTRA, SURINDER"	
Higher	Facility eval. code:	0303	
	Facility office number:	03	
	Facility county number:	57	
	Facility type code:	810	
	Facility status code:	06	
	Address:	1018 J STREET	
	City:	DAVIS	
	State:	CA	
	Zip:	95616	
	Alt. address:	1018 J STREET	
	City:	DAVIS	
	State:	CA	
	Zip:	95616	
	Facility investor:	"MALHOTRA, SURINDER"	
	Licensee type:	A	
	License effective date:	50913	
	License expiration date:	Not Reported	
	License issue date:	050913	
	Program type:	"MAX. CAP: 6 - NO MORE THAN 3 INFANTS OR 4 INFANTS ONLY. CAP 8 - NO MORE THAN 2 INFANTS, 1 CHILD IN KINDERGARTEN OR ELEMENTARY SCHOOL AND 1 CHILD AT LEAST AGE 6."	
	Original app. received date:	050816	
	Facility closed date:	Not Reported	
	Mailing address:	1018 J STREET	
	Mailing city:	DAVIS	
	Mailing state:	CA	
	Mailing zip:	95616	

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Contact person: "MALHOTRA, SURINDER "
 Facility capacity: 8
 Type of clients served: 960
 Facility phone: 5307579728

I29		SRPU20071014148
NNE	Ncessch: 061062001180	Public Schools
1-2 mi	Schname05: NORTH DAVIS ELEMENTARY	
6457	Mstreet05: 555 EAST 14TH ST.	
Higher	Mcity05: DAVIS	
	Mstate05: CA	
	Mzip05: 95616	
	Mzip405: 2097	
	Member05: 461	
	Phone05: (530) 757-5475	
	Locale05: 3	
	Type05: 1	
	Level05: 1	
	Gslo05: KG	
	Gshi05: 06	
	Edr id: SRPU20071014148	

J30		SRDCCA200733411
NE	EDR ID: SRDCCA200733411	Daycare
1-2 mi	Facility number: 573611229	
6484	Facility name: "WONG-XOQUIC, HEATHER "	
Higher	Facility eval. code: 0303	
	Facility office number: 03	
	Facility county number: 57	
	Facility type code: 810	
	Facility status code: 03	
	Address: 1412 DUKE DRIVE	
	City: DAVIS	
	State: CA	
	Zip: 95616	
	Alt. address: 1412 DUKE DRIVE	
	City: DAVIS	
	State: CA	
	Zip: 95616	
	Facility investor: "WONG-XOQUIC, HEATHER "	
	Licensee type: A	
	License effective date: 60105	
	License expiration date: Not Reported	
	License issue date: 060105	
	Program type: MAX. CAP (WHEN THERE IS AN ASSISTANT PRESENT): 12 - NO MORE THAN 4 INFANTS. CAP 14 - NO MORE THAN 3 INFANTS. 1 CHILD IN KINDERGARTEN OR ELEMENTARY SCHOOL AND 1 CHILD AT LEAST AGE 6."HALLWAY, AND THE BACKYARD. "	
	Original app. received date: 051020	
	Facility closed date: Not Reported	
	Mailing address: 1412 DUKE DRIVE	
	Mailing city: DAVIS	
	Mailing state: CA	
	Mailing zip: 95616	

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Contact person: "WONG-XOQUIC, HEATHER "
 Facility capacity: 14
 Type of clients served: 960
 Facility phone: 5307530242

I31		SRDCCA200743600
NNE	EDR ID:	SRDCCA200743600
1-2 mi	Facility number:	570312615
6541	Facility name:	NORTH DAVIS SCHOOL AGE CHILD DEVELOPMENT CENTER
Higher	Facility eval. code:	0303
	Facility office number:	03
	Facility county number:	57
	Facility type code:	840
	Facility status code:	03
	Address:	607 EAST 14TH STREET
	City:	DAVIS
	State:	CA
	Zip:	95616
	Alt. address:	607 EAST 14TH STREET
	City:	DAVIS
	State:	CA
	Zip:	95616
	Facility investor:	CHILD DEVELOPMENT CENTERS
	Licensee type:	C
	License effective date:	951201
	License expiration date:	Not Reported
	License issue date:	891201
	Program type:	MAXIMUM CAPACITY: 112 LICENSED TO SERVE CHILDREN KINDERGARTEN THROUGH 12 YEARS OLD.
	Original app. received date:	890822
	Facility closed date:	Not Reported
	Mailing address:	"851 E. HAMILTON AVE., STE 200 "
	Mailing city:	CAMPBELL
	Mailing state:	CA
	Mailing zip:	95008
	Contact person:	CAMERON SCOTT
	Facility capacity:	112
	Type of clients served:	950
	Facility phone:	5307564350

F32		SRPU20071014158
North	Ncessch:	061062011542
1-2 mi	Schname05:	LEONARDO DAVINCI HIGH
6677	Mstreet05:	1602 OAK AVE.
Higher	Mcity05:	DAVIS
	Mstate05:	CA
	Mzip05:	95616
	Mzip405:	Not Reported
	Member05:	217
	Phone05:	(530) 757-7154
	Locale05:	3
	Type05:	1
	Level05:	3
	Gslo05:	10

MAP FINDINGS

Map ID Direction Distance Distance (ft.) Elevation	Site	EDR ID Database
Gshi05: Edr id:	12 SRPU20071014158	
33 NW 1-2 mi 6747 Higher	EDR ID: SRDCCA200716384 Facility number: 573607417 Facility name: "EBERLE, LISA" Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 810 Facility status code: 03 Address: 1646 COLUSA AVE City: DAVIS State: CA Zip: 95616 Alt. address: 1646 COLUSA AVE City: DAVIS State: CA Zip: 95616 Facility investor: "EBERLE, LISA" Licensee type: A License effective date: 119 License expiration date: Not Reported License issue date: 000119 Program type: "MAXIMUM CAPACITY: 12 CHILDREN, WITH NO MORE THAN 4 INFANTS, OR CAPACITY 14 CHILDREN WHEN 2 CHILDREN ARE AT LEAST 6 YEARS OF AGE WITH A MAXIMUM OF 3 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED" Original app. received date: 000101 Facility closed date: Not Reported Mailing address: 1646 COLUSA AVE Mailing city: DAVIS Mailing state: CA Mailing zip: 95616 Contact person: "EBERLE, LISA" Facility capacity: 14 Type of clients served: 960 Facility phone: 5307530906	SRDCCA200716384 Daycare
34 NNW 1-2 mi 6894 Higher	EDR ID: SRDCCA200755521 Facility number: 573610796 Facility name: GAN HAVERIM PRESCHOOL Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 850 Facility status code: 03 Address: 1715 ANDERSON ROAD City: DAVIS State: CA Zip: 95616 Alt. address: 1715 ANDERSON ROAD City: DAVIS	SRDCCA200755521 Daycare

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

State: CA
 Zip: 95616
 Facility investor: CONGREGATION BET HAVERIM
 Licensee type: C
 License effective date: 50901
 License expiration date: Not Reported
 License issue date: 050901
 Program type: LICENSED TO SERVE CHILDREN AGES 2 YEARS TO ENTRY INTO KINDERGARTEN.
 Original app. received date: 050531
 Facility closed date: Not Reported
 Mailing address: 1715 ANDERSON ROAD
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "LUKENBILL, JULIA"
 Facility capacity: 28
 Type of clients served: 950
 Facility phone: 5307580842

K35
 East
 1-2 mi
 6926
 Higher

Hospital type: 01
 Num of times COO: 00
 Owner date: Not Reported
 City: DAVIS
 Has plan of corr: Not Reported
 Compliance status: Not Reported
 SSA county code: 670
 Cross ref number: 05D0890900
 FMS survey date: Not Reported
 Current survey date: Not Reported
 Medicare/Medicaid: Not Reported
 Facility name: SUTTER DAVIS VISITING NURSE ASSOC
 Intermediary/Carrier: Not Reported
 Medicaid number: Not Reported
 Participation date: 19940919
 Prior COO date: Not Reported
 Prior carrier: Not Reported
 Provider ID: 05D0891838
 Record Status: A
 Region code: 09
 Is Partial Record: Y
 state abbrev: CA
 ssa state: 05
 state region cd: LAB
 street address: 1947 GALILEO COURT #102
 Phone num: 9167563892
 Termination reason: 01
 Term Date: 19960109
 Purpose of action: Not Reported
 Provider control: 02
 Zip: 95616
 Fips state: 06
 Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B

SRHO20070145308
 AHA Hospitals

MAP FINDINGS

Map ID
Direction
Distance
Distance (ft.)
Elevation

Site

EDR ID
Database

Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0000
 Num cert beds: 0000
 Source: US_HOSPITAL_POSCLIA
 Edr id: SRHO20070145308

K36
 East
 1-2 mi
 7023
 Higher

Hospital type: 01
 Num of times COO: 00
 Owner date: Not Reported
 City: DAVIS
 Has plan of corr: 1
 Compliance status: A
 SSA county code: 670
 Cross ref number: Not Reported
 FMS survey date: Not Reported
 Current survey date: 19991013
 Medicare/Medicaid: 1
 Facility name: TPMG - DAVIS MOB
 Intermediary/Carrier: Not Reported
 Medicaid number: Not Reported
 Participation date: 19920901
 Prior COO date: Not Reported
 Prior carrier: Not Reported
 Provider ID: 05D0680927
 Record Status: A
 Region code: 09
 Is Partial Record: Not Reported
 state abbrev: CA
 ssa state: 05
 state region cd: M2
 street address: 1955 COWELL BLVD
 Phone num: 9167577100
 Termination reason: 00
 Term Date: 20070215
 Purpose of action: 2
 Provider control: 02
 Zip: 95616
 Fips state: 06
 Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0000
 Num cert beds: 0000
 Source: US_HOSPITAL_POSCLIA
 Edr id: SRHO20070137560

SRHO20070137560
 AHA Hospitals

MAP FINDINGS

Map ID Direction Distance Distance (ft.) Elevation	Site	EDR ID Database
37 North 1-2 mi 7124 Higher	Hospital type: 01 Num of times COO: 01 Owner date: 19960531 City: DAVIS Has plan of corr: 1 Compliance status: A SSA county code: 670 Cross ref number: Not Reported FMS survey date: Not Reported Current survey date: 19950621 Medicare/Medicaid: 1 Facility name: SUTTER VISITING NURSE ASSOC - DAVIS Intermediary/Carrier: 52280 Medicaid number: Not Reported Participation date: 19930527 Prior COO date: Not Reported Prior carrier: 00040 Provider ID: 557294 Record Status: A Region code: 09 Is Partial Record: Not Reported state abbrev: CA ssa state: 05 state region cd: S1 street address: 1777 OAK AVENUE Phone num: 9167563892 Termination reason: 01 Term Date: 19980701 Purpose of action: 2 Provider control: 02 Zip: 95616 Fips state: 06 Fips cnty: 113 SSA MSA: 499 SSA MSA size code: B Date accredited: Not Reported Accred expire date: Not Reported Accred Org: 0 Num beds: 0000 Num cert beds: 0000 Source: US_HOSPITAL_POSOTHER Edr id: SRHO20070109026	SRHO20070109026 AHA Hospitals
L38 NE 1-2 mi 7230 Higher	EDR ID: SRDCCA200754393 Facility number: 573605859 Facility name: VALLEY OAK STATE PRESCHOOL Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 850 Facility status code: 03 Address: 1450 EAST 8TH STREET City: DAVIS	SRDCCA200754393 Daycare

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

State: CA
 Zip: 95616
 Alt. address: "851 E. HAMILTON AVE., STE. 200"
 City: CAMPBELL
 State: CA
 Zip: 95008
 Facility investor: CHILD DEVELOPMENT CENTERS
 Licensee type: C
 License effective date: 914
 License expiration date: Not Reported
 License issue date: 000914
 Program type: "LICENSED TO SERVE CHILDREN AGE 2.9 YEARS TO ENTRY INTO KINDERGARTEN. 19 CHILDREN TOTAL IN THE WEST PORTABLE ROOM, AND 24 CHILDREN TOTAL WHEN THE EAST PORTABLE ROOM IS NOT OCCUPIED BY SCHOOL AGE CHILDREN."
 "Original app. received date: 000810
 Facility closed date: Not Reported
 Mailing address: "851 E. HAMILTON AVE., STE. 200"
 Mailing city: CAMPBELL
 Mailing state: CA
 Mailing zip: 95008
 Contact person: "SCOTT, CAMERON"
 Facility capacity: 24
 Type of clients served: 950
 Facility phone: 5307539223

L39 NE 1-2 mi 7230 Higher	EDR ID: Facility number: Facility name: Facility eval. code: Facility office number: Facility county number: Facility type code: Facility status code: Address: City: State: Zip: Alt. address: City: State: Zip: Facility investor: Licensee type: License effective date: License expiration date: License issue date: Program type: Original app. received date: Facility closed date: Mailing address:	SRDCCA200743598 Daycare SRDCCA200743598 570312614 VALLEY OAK SCHOOL AGE CHILD DEVELOPMENT CENTER 0303 03 57 840 03 1450 EAST 8TH STREET DAVIS CA 95616 1450 EAST 8TH STREET DAVIS CA 95616 CHILD DEVELOPMENT CENTERS C 951018 Not Reported 891018 "LICENSED TO SERVE 66 CHILDREN ENROLLED IN KINDERGARTEN AND ABOVE IN CDC PORTABLE WEST-EAST ROOMS FROM 7:00AM-8:30AM & 11:30AM-6:00PM, AND 30 TOTAL CAPACITY IN EAST ROOM 8:30AM-11:30AM. 26 ADDITIONAL CHILDREN CAN BE ACCOMADATED IN THE SCHOOL LIBRARY & MPR ROOM WHEN AVAILABLE." 890822 Not Reported "851 E. HAMILTON AVE., STE 200"	SRDCCA200743598 Daycare
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MAP FINDINGS

Map ID Direction Distance Distance (ft.) Elevation	Site	EDR ID Database
	Mailing city: CAMPBELL Mailing state: CA Mailing zip: 85008 Contact person: "WILLIAMS, SHANIKA" Facility capacity: 92 Type of clients served: 950 Facility phone: 5307539223	
J40 NE 1-2 mi 7290 Higher	EDR ID: SRDCCA200716267 Facility number: 573607380 Facility name: "BOWERS, LEANN" Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 810 Facility status code: 03 Address: 743 M STREET City: DAVIS State: CA Zip: 95616 Alt. address: 743 M STREET City: DAVIS State: CA Zip: 95616 Facility investor: "BOWERS, LEANN" Licensee type: C License effective date: 930901 License expiration date: Not Reported License issue date: 930901 Program type: "MAXIMUM CAPACITY: 6 CHILDREN WITH NO MORE THAN 3 INFANTS, OR 4 INFANTSONLY, OR CAPACITY 8 CHILDREN WHEN 2 ARE AT LEAST 6 YEARS OF AGE WITH AMAXIMUM OF 2 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED" Original app. received date: 930101 Facility closed date: Not Reported Mailing address: 743 M STREET Mailing city: DAVIS Mailing state: CA Mailing zip: 95616 Contact person: "BOWERS, LEANN" Facility capacity: 8 Type of clients served: 960 Facility phone: 5307583284	SRDCCA200716267 Daycare
41 NNE 1-2 mi 7546 Higher	EDR ID: SRDCCA200719068 Facility number: 573608715 Facility name: "STONE, ELISA" Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 810 Facility status code: 03	SRDCCA200719068 Daycare

MAP FINDINGS

Map ID	Direction	Distance	Distance (ft.)	Elevation	Site	EDR ID	Database
					Address: 1318 J STREET City: DAVIS State: CA Zip: 95616 Alt. address: 1318 J STREET City: DAVIS State: CA Zip: 95616 Facility investor: "STONE, ELISA" Licensee type: A License effective date: 30106 License expiration date: Not Reported License issue date: 030106 Program type: "MAXIMUM CAPACITY: 12 CHILDREN, WITH NO MORE THAN 4 INFANTS, OR CAPACITY 14 CHILDREN WHEN 2 CHILDREN ARE AT LEAST 6 YEARS OF AGE WITH A MAXIMUM OF 3 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED. "OFF LIMITS: MASTER BEDROOM. Original app. received date: 021030 Facility closed date: Not Reported Mailing address: 1318 J STREET Mailing city: DAVIS Mailing state: CA Mailing zip: 95616 Contact person: "STONE, ELISA" Facility capacity: 14 Type of clients served: 960 Facility phone: 5307571150		
42	NE	1-2 mi	7709	Higher	Ncessch: 061062001178 Scname05: OLIVER WENDELL HOLMES JUNIOR HIGH Mstreet05: 1220 DREXEL DR. Mcity05: DAVIS Mstate05: CA Mzip05: 95616 Mzip405: 2123 Member05: 741 Phone05: (530) 757-5445 Locale05: 3 Type05: 1 Level05: 2 Gslo05: 07 Gshi05: 09 Edr id: SRPU20071014146	SRPU20071014146	Public Schools
43	East	1-2 mi	7976	Higher	EDR ID: SRDCCA200732086 Facility number: 573611359 Facility name: "THOMAS, DIANA" Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 810 Facility status code: 03	SRDCCA200732086	Daycare

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Address: 1540 VALDORA STREET #107
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 1540 VALDORA STREET #107
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "THOMAS, DIANA"
 Licensee type: A
 License effective date: 50829
 License expiration date: Not Reported
 License issue date: 050829
 Program type: "MAX. CAP: 6 - NO MORE THAN 3 INFANTS OR 4 INFANTS ONLY.
 CAP 8 - NO MORE THAN 2 INFANTS, 1 CHILD IN KINDERGARTEN OR ELEMENTARY
 SCHOOL AND 1 CHILD AT LEAST AGE 6."
 Original app. received date: 050725
 Facility closed date: Not Reported
 Mailing address: 1540 VALDORA STREET #107
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "THOMAS, DIANA"
 Facility capacity: 8
 Type of clients served: 960
 Facility phone: 5307562774

M44 WNW 1-2 mi 8089 Higher	Ncessch: Schname05: Mstreet05: Mcity05: Mstate05: Mzip05: Mzip405: Member05: Phone05: Locale05: Type05: Level05: Gslo05: Gshi05: Edr id:	061062001177 RALPH WALDO EMERSON JUNIOR HIGH 2121 CALAVERAS AVE. DAVIS CA 95616 3022 603 (530) 757-5430 3 1 2 07 09 SRPU20071014145	SRPU20071014145 Public Schools
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N45 NNW 1-2 mi 8092 Higher	Hospital type: Num of times COO: Owner date: City: Has plan of corr: Compliance status: SSA county code: Cross ref number: FMS survey date:	01 00 Not Reported DAVIS Not Reported Not Reported 670 05D0591916 Not Reported	SRHO20070144142 AHA Hospitals
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MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Current survey date: Not Reported
 Medicare/Medicaid: Not Reported
 Facility name: PHYSICIANS CLINICAL LABORATORY
 Intermediary/Carrier: Not Reported
 Medicaid number: Not Reported
 Participation date: 19950221
 Prior COO date: Not Reported
 Prior carrier: Not Reported
 Provider ID: 05D0898018
 Record Status: A
 Region code: 09
 Is Partial Record: Not Reported
 state abbrev: CA
 ssa state: 05
 state region cd: LAB
 street address: 2043 ANDERSON ROAD #D
 Phone num: 9167572522
 Termination reason: 01
 Term Date: 19950907
 Purpose of action: Not Reported
 Provider control: 04
 Zip: 95616
 Fips state: 06
 Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0000
 Num cert beds: 0000
 Source: US_HOSPITAL_POSCLIA
 Edr id: SRHO20070144142

N46
 NNW
 1-2 mi
 8092
 Higher

Hospital type: 01
 Num of times COO: 00
 Owner date: Not Reported
 City: DAVIS
 Has plan of corr: Not Reported
 Compliance status: Not Reported
 SSA county code: 670
 Cross ref number: Not Reported
 FMS survey date: Not Reported
 Current survey date: Not Reported
 Medicare/Medicaid: Not Reported
 Facility name: STEPHEN H FOSTER MD
 Intermediary/Carrier: Not Reported
 Medicaid number: Not Reported
 Participation date: 19930115
 Prior COO date: Not Reported
 Prior carrier: Not Reported
 Provider ID: 05D0613701
 Record Status: A
 Region code: 09

SRHO20070137459
 AHA Hospitals

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Is Partial Record: Not Reported
 state abbrev: CA
 ssa state: 05
 state region cd: LAB
 street address: 2043 ANDERSON RD
 Phone num: 9167536116
 Termination reason: 15
 Term Date: 19940831
 Purpose of action: Not Reported
 Provider control: 04
 Zip: 95616
 Fips state: 06
 Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0000
 Num cert beds: 0000
 Source: US_HOSPITAL_POSCLIA
 Edr id: SRHO20070137459

O47
 NE
 1-2 mi
 8205
 Higher

Hospital type: 03
 Num of times COO: 04
 Owner date: Not Reported
 City: DAVIS
 Has plan of corr: 1
 Compliance status: A
 SSA county code: 670
 Cross ref number: Not Reported
 FMS survey date: 20050330
 Current survey date: 20060126
 Medicare/Medicaid: 1
 Facility name: COURTYARD HEALTHCARE CENTER
 Intermediary/Carrier: 52280
 Medicaid number: 570390854
 Participation date: 19720101
 Prior COO date: 19950601
 Prior carrier: Not Reported
 Provider ID: 055922
 Record Status: A
 Region code: 09
 Is Partial Record: Not Reported
 state abbrev: CA
 ssa state: 05
 state region cd: S1
 street address: 1850 EAST 8TH STREET
 Phone num: 5307561800
 Termination reason: 00
 Term Date: Not Reported
 Purpose of action: 2
 Provider control: 03
 Zip: 95616

SRHO20070011729
 AHA Hospitals

MAP FINDINGS

Map ID
Direction
Distance
Distance (ft.)
Elevation

Site

EDR ID
Database

Fips state: 06
 Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0112
 Num cert beds: 0112
 Source: US_HOSPITAL_POSOTHER
 Edr id: SRHO20070011729

O48
 NE
 1-2 mi
 8205
 Higher

Hospital type: 01
 Num of times COO: 00
 Owner date: Not Reported
 City: DAVIS
 Has plan of corr: Not Reported
 Compliance status: Not Reported
 SSA county code: 670
 Cross ref number: Not Reported
 FMS survey date: Not Reported
 Current survey date: Not Reported
 Medicare/Medicaid: Not Reported
 Facility name: COURTYARD HEALTHCARE CENTER
 Intermediary/Carrier: Not Reported
 Medicaid number: Not Reported
 Participation date: 19930506
 Prior COO date: Not Reported
 Prior carrier: Not Reported
 Provider ID: 05D0613758
 Record Status: A
 Region code: 09
 Is Partial Record: Y
 state abbrev: CA
 ssa state: 05
 state region cd: LAB
 street address: 1850 E 8TH ST
 Phone num: 9167561800
 Termination reason: 00
 Term Date: 20080831
 Purpose of action: Not Reported
 Provider control: 04
 Zip: 95616
 Fips state: 06
 Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0000
 Num cert beds: 0000
 Source: US_HOSPITAL_POSCLIA
 Edr id: SRHO20070135396

SRHO20070135396
 AHA Hospitals

MAP FINDINGS

Map ID	Direction	Distance	Distance (ft.)	Elevation	Site	EDR ID	Database
O49	NE	1-2 mi	8205	Higher	Provnum: 055922 Nursinghomename: COURTYARD HEALTHCARE CENTER Street: 1850 EAST 8TH STREET City: DAVIS State: CA Zipcode: 95616 Phonenumber: 5307561800 Dateoflastinspection: 20060120 Certifiednumberofbeds: 112 Totalnumberofresidents: 83 Percofoccupiedbeds: 74 Categorydescription: Participating in Medicare and Medicaid Typeofownership: For profit - Corporation Locatedwithinahospital: NO Multinursinghomeownership: NO Residentandfamilycouncils: RESIDENT Edr id: SRNH20060900838	SRNH20060900838	Nursing Homes
P50	WNW	1-2 mi	8223	Higher	EDR ID: SRDCCA200719967 Facility number: 573608589 Facility name: "BENNETT, MELE" Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 810 Facility status code: 03 Address: 2307 SHIRE LN City: DAVIS State: CA Zip: 95616 Alt. address: 2307 SHIRE LN City: DAVIS State: CA Zip: 95616 Facility investor: "BENNETT, MELE" Licensee type: A License effective date: 21106 License expiration date: Not Reported License issue date: 021106 Program type: "MAXIMUM CAPACITY: 6 CHILDREN WITH NO MORE THAN 3 INFANTS, OR 4 INFANTSONLY, OR CAPACITY 8 CHILDREN WHEN 2 ARE AT LEAST 6 YEARS OF AGE WITH AMAXIMUM OF 2 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED" Original app. received date: 020910 Facility closed date: Not Reported Mailing address: 2307 SHIRE LN Mailing city: DAVIS Mailing state: CA Mailing zip: 95616 Contact person: "BENNETT, MELE" Facility capacity: 8 Type of clients served: 960 Facility phone: 5307581813	SRDCCA200719967	Daycare

MAP FINDINGS

Map ID Direction Distance Distance (ft.) Elevation	Site	EDR ID Database
O51 NE 1-2 mi 8267 Higher	Hospital type: 01 Num of times COO: 00 Owner date: Not Reported City: DAVIS Has plan of corr: Not Reported Compliance status: Not Reported SSA county code: 670 Cross ref number: Not Reported FMS survey date: Not Reported Current survey date: Not Reported Medicare/Medicaid: Not Reported Facility name: RON A BERRYHILL Intermediary/Carrier: Not Reported Medicaid number: Not Reported Participation date: 19930201 Prior COO date: Not Reported Prior carrier: Not Reported Provider ID: 05D0858906 Record Status: A Region code: 09 Is Partial Record: Not Reported state abbrev: CA ssa state: 05 state region cd: LAB street address: 914 CRAIG PLACE Phone num: 9167537532 Termination reason: 01 Term Date: 19950310 Purpose of action: Not Reported Provider control: 04 Zip: 95616 Fips state: 06 Fips cnty: 113 SSA MSA: 499 SSA MSA size code: B Date accredited: Not Reported Accred expire date: Not Reported Accred Org: Not Reported Num beds: 0000 Num cert beds: 0000 Source: US_HOSPITAL_POSCLIA Edr id: SRHO20070142179	SRHO20070142179 AHA Hospitals
52 East 1-2 mi 8269 Higher	EDR ID: SRDCCA200715734 Facility number: 573607575 Facility name: "SAH, B. DEVI Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 810 Facility status code: 03 Address: 1721 SAPPHIRE CIRCLE City: DAVIS	SRDCCA200715734 Daycare

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

State: CA
 Zip: 95616
 Alt. address: 1721 SAPPHIRE CIRCLE
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "SAH, B. DEVI "
 Licensee type: A
 License effective date: 960102
 License expiration date: Not Reported
 License issue date: 960102
 Program type: "MAXIMUM CAPACITY: 12 CHILDREN, WITH NO MORE THAN 4 INFANTS, OR CAPACITY 14 CHILDREN WHEN 2 CHILDREN ARE AT LEAST 6 YEARS OF AGE WITH A MAXIMUM OF 3 INFANTS. OFFLIMITS:GARAGE,UPSTAIRS,GARDEN,SIDE YARD. "
 Original app. received date: 960101
 Facility closed date: Not Reported
 Mailing address: 1721 SAPPHIRE CIRCLE
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "SAH, B. DEVI "
 Facility capacity: 12
 Type of clients served: 960
 Facility phone: 5307580405

<p>M53 NW 1-2 mi 8271 Higher</p>	<p>EDR ID: SRDCCA200738888 Facility number: 573613139 Facility name: "CHAVEZ, JOSEFINA " Facility eval. code: S305 Facility office number: 03 Facility county number: 57 Facility type code: 810 Facility status code: 03 Address: 2027 HUMBOLT AVENUE City: DAVIS State: CA Zip: 95616 Alt. address: 2027 HUMBOLT AVENUE City: DAVIS State: CA Zip: 95616 Facility investor: "CHAVEZ, JOSEFINA " Licensee type: A License effective date: 70315 License expiration date: Not Reported License issue date: 070315 Program type: "MAX. CAP: 6 - NO MORE THAN 3 INFANTS OR 4 INFANTS ONLY. CAP 8 - NO MORE THAN 2 INFANTS, 1 CHILD IN KINDERGARTEN OR ELEMENTARY SCHOOL AND 1 CHILD AT LEAST AGE 6. OFF-LIMIT AREAS: GARAGE AND ENTIRE UPSTAIRS. " Original app. received date: 061130 Facility closed date: Not Reported Mailing address: 2027 HUMBOLT AVENUE Mailing city: DAVIS Mailing state: CA</p>	<p>SRDCCA200738888 Daycare</p>
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MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Mailing zip: 95616
 Contact person: "CHAVEZ, JOSEFINA"
 Facility capacity: 8
 Type of clients served: 960
 Facility phone: 5307567853

O54 ENE 1-2 mi 8333 Higher	Hospital type: 02 Num of times COO: 00 Owner date: Not Reported City: DAVIS Has plan of corr: Not Reported Compliance status: A SSA county code: 670 Cross ref number: Not Reported FMS survey date: Not Reported Current survey date: 19831117 Medicare/Medicaid: 1 Facility name: SIERRA HEALTH CARE CONVALESCENT HOSP Intermediary/Carrier: Not Reported Medicaid number: Not Reported Participation date: 19751001 Prior COO date: Not Reported Prior carrier: Not Reported Provider ID: 05E150 Record Status: A Region code: 09 Is Partial Record: Not Reported state abbrev: CA ssa state: 05 state region cd: S1 street address: 715 POLE LINE ROAD Phone num: 9167564900 Termination reason: 07 Term Date: 19850201 Purpose of action: 2 Provider control: 03 Zip: 95616 Fips state: 06 Fips cnty: 113 SSA MSA: 499 SSA MSA size code: B Date accredited: Not Reported Accred expire date: Not Reported Accred Org: Not Reported Num beds: 0132 Num cert beds: 0132 Source: US_HOSPITAL_POSOTHER Edr id: SRHO20070007638	SRHO20070007638 AHA Hospitals
--	---	----------------------------------

O55 ENE 1-2 mi 8333 Higher	Hospital type: 03 Num of times COO: 00 Owner date: Not Reported	SRHO20070009834 AHA Hospitals
--	---	----------------------------------

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

City: DAVIS
 Has plan of corr: 1
 Compliance status: A
 SSA county code: 670
 Cross ref number: Not Reported
 FMS survey date: 20060307
 Current survey date: 20060214
 Medicare/Medicaid: 1
 Facility name: SIERRA HEALTH CARE CENTER
 Intermediary/Carrier: 00040
 Medicaid number: Not Reported
 Participation date: 19760801
 Prior COO date: Not Reported
 Prior carrier: 52280
 Provider ID: 055681
 Record Status: A
 Region code: 09
 Is Partial Record: Not Reported
 state abbrev: CA
 ssa state: 05
 state region cd: S1
 street address: 715 POLE LINE ROAD
 Phone num: 5307564900
 Termination reason: 00
 Term Date: Not Reported
 Purpose of action: 2
 Provider control: 03
 Zip: 95616
 Fips state: 06
 Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0132
 Num cert beds: 0132
 Source: US_HOSPITAL_POSOTHER
 Edr id: SRHO20070009834

O56
 ENE
 1-2 mi
 8333
 Higher

Hospital type: 01
 Num of times COO: 00
 Owner date: Not Reported
 City: DAVIS
 Has plan of corr: Not Reported
 Compliance status: Not Reported
 SSA county code: 670
 Cross ref number: Not Reported
 FMS survey date: Not Reported
 Current survey date: Not Reported
 Medicare/Medicaid: Not Reported
 Facility name: SIERRA HEALTHCARE CONVALESCENT
 Intermediary/Carrier: Not Reported
 Medicaid number: Not Reported

SRHO20070137787
 AHA Hospitals

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Participation date: 19931008
 Prior COO date: Not Reported
 Prior carrier: Not Reported
 Provider ID: 05D0701144
 Record Status: A
 Region code: 09
 Is Partial Record: Y
 state abbrev: CA
 ssa state: 05
 state region cd: LAB
 street address: 715 POLE LINE RD
 Phone num: 9167564900
 Termination reason: 00
 Term Date: 20080831
 Purpose of action: Not Reported
 Provider control: 04
 Zip: 95616
 Fips state: 06
 Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0000
 Num cert beds: 0000
 Source: US_HOSPITAL_POSCLIA
 Edr id: SRHO20070137787

O57
 ENE
 1-2 mi
 8333
 Higher

Provnum: 055681
 Nursinghomename: SIERRA HEALTH CARE CENTER
 Street: 715 POLE LINE ROAD
 City: DAVIS
 State: CA
 Zipcode: 95616
 Phonenumber: 5307564900
 Dateoflastinspection: 20060210
 Certifiednumberofbeds: 132
 Totalnumberofresidents: 122
 Percofoccupiedbeds: 92
 Categorydescription: Participating in Medicare and Medicaid
 Typeofownership: For profit - Corporation
 Locatedwithinhospital: NO
 Multinursinghomeownership: YES
 Residentandfamilycouncils: BOTH
 Edr id: SRNH20060900966

SRNH20060900966
 Nursing Homes

Q58
 NW
 1-2 mi
 8442
 Higher

EDR ID: SRDCCA200715713
 Facility number: 573607535
 Facility name: "NAVARRO, ESPERANZA"
 Facility eval. code: S305
 Facility office number: 03

SRDCCA200715713
 Daycare

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Facility county number: 57
 Facility type code: 810
 Facility status code: 03
 Address: 2031 IMPERIAL AVE
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 2031 IMPERIAL AVE
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "NAVARRO, ESPERANZA"
 Licensee type: A
 License effective date: 980727
 License expiration date: Not Reported
 License issue date: 980727
 Program type: "MAXIMUM CAPACITY: 6 CHILDREN WITH NO MORE THAN 3 INFANTS, OR 4
 INFANTSONLY, OR CAPACITY 8 CHILDREN WHEN 2 ARE AT LEAST 6 YEARS OF AGE
 WITH AMAXIMUM OF 2 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED
 "
 Original app. received date: 980101
 Facility closed date: Not Reported
 Mailing address: 2031 IMPERIAL AVE
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "NAVARRO, ESPERANZA"
 Facility capacity: 8
 Type of clients served: 960
 Facility phone: 5307587759

O59
 NE EDR ID: SRDCCA200716377
 1-2 mi SRDCCA200716377
 8503 Facility number: 573607405
 Higher Facility name: "CONNOLLY, COLLEEN"
 Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 810
 Facility status code: 03
 Address: 1930 HAUSSLER DRIVE
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 1930 HAUSSLER DRIVE
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "CONNOLLY, COLLEEN"
 Licensee type: A
 License effective date: 950801
 License expiration date: Not Reported
 License issue date: 950801
 Program type: "MAXIMUM CAPACITY: 12 CHILDREN, WITH NO MORE THAN 4 INFANTS, OR
 CAPACITY 14 CHILDREN WHEN 2 CHILDREN ARE AT LEAST 6 YEARS OF AGE WITH A
 MAXIMUM OF 3 INFANTS; PROPERTY/LANDLORD CONSENT IS REQUIRED. "

MAP FINDINGS

Map ID Direction Distance Distance (ft.) Elevation	Site	EDR ID Database
	Original app. received date: 950101 Facility closed date: Not Reported Mailing address: 1930 HAUSSLER DRIVE Mailing city: DAVIS Mailing state: CA Mailing zip: 95616 Contact person: "CONNOLLY, COLLEEN" Facility capacity: 14 Type of clients served: 960 Facility phone: 5307531138	
P60 WNW 1-2 mi 8536 Higher	EDR ID: SRDCCA200716231 Facility number: 573607490 Facility name: "LAMBERT, PATRICIA" Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 810 Facility status code: 03 Address: 2443 ELENDIL LANE City: DAVIS State: CA Zip: 95616 Alt. address: 2443 ELENDIL LANE City: DAVIS State: CA Zip: 95616 Facility investor: "LAMBERT, PATRICIA" Licensee type: A License effective date: 1016 License expiration date: Not Reported License issue date: 001016 Program type: "MAXIMUM CAPACITY: 12 CHILDREN, WITH NO MORE THAN 4 INFANTS, OR CAPACITY 14 CHILDREN WHEN 2 CHILDREN ARE AT LEAST 6 YEARS OF AGE WITH A MAXIMUM OF 3 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED." Original app. received date: 000101 Facility closed date: Not Reported Mailing address: 2443 ELENDIL LANE Mailing city: DAVIS Mailing state: CA Mailing zip: 95616 Contact person: "LAMBERT, PATRICIA" Facility capacity: 14 Type of clients served: 960 Facility phone: 5307532535	SRDCCA200716231 Daycare
R61 NE 1-2 mi 8652 Higher	EDR ID: SRDCCA200715670 Facility number: 573607371 Facility name: "BAKAY, DAVID AND SKOG, LESLYN" Facility eval. code: 0303 Facility office number: 03 Facility county number: 57	SRDCCA200715670 Daycare

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Facility type code: 810
 Facility status code: 03
 Address: 1109 CHESTNUT LANE
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 1109 CHESTNUT LANE
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "BAKAY, DAVID AND SKOG, LESLYN"
 Licensee type: A
 License effective date: 960903
 License expiration date: Not Reported
 License issue date: 960903
 Program type: "MAXIMUM CAPACITY: 12 CHILDREN, WITH NO MORE THAN 4 INFANTS, OR
 CAPACITY 14 CHILDREN WHEN 2 CHILDREN ARE AT LEAST 6 YEARS OF AGE WITH A
 MAXIMUM OF 3 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED."
 Original app. received date: 960101
 Facility closed date: Not Reported
 Mailing address: 1109 CHESTNUT LANE
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "BAKAY, DAVID"
 Facility capacity: 14
 Type of clients served: 960
 Facility phone: 5307583097

O62
 NE
 1-2 mi
 8659
 Higher

EDR ID: SRDCCA200716379
 Facility number: 573607409
 Facility name: "CUETARA, JULIE"
 Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 810
 Facility status code: 03
 Address: 903 SNYDER DRIVE
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 903 SNYDER DRIVE
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "CUETARA, JULIE"
 Licensee type: A
 License effective date: 940721
 License expiration date: Not Reported
 License issue date: 940721
 Program type: "MAXIMUM CAPACITY: 12 CHILDREN, WITH NO MORE THAN 4 INFANTS, OR CAPACITY
 14 CHILDREN WHEN 2 CHILDREN ARE AT LEAST 6 YEARS OF AGE WITH A MAXIMUM
 OF 3 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED."
 SRDCCA200716379
 Daycare

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Original app. received date: 940101
 Facility closed date: Not Reported
 Mailing address: 903 SNYDER DRIVE
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "CUETARA, JULIE"
 Facility capacity: 14
 Type of clients served: 960
 Facility phone: 5307588238

S63 East SRDCCA200715728 Daycare
 1-2 mi 8672 Higher

EDR ID: SRDCCA200715728
 Facility number: 573607561
 Facility name: "PYTEL, JEANNIE"
 Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 810
 Facility status code: 03
 Address: 1206 FARRAGUT CIRCLE
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 1206 FARRAGUT CIRCLE
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "PYTEL, JEANNIE"
 Licensee type: A
 License effective date: 941003
 License expiration date: Not Reported
 License issue date: 941003
 Program type: "MAXIMUM CAPACITY:12 CHILDREN WITH NO MORE THAN 4 INFANTS, OR CAPACITY 14 CHILDREN WHEN 2 CHILDREN ARE AT LEAST 6 YEARS OF AGE WITH A MAXIMUM OF 3 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED"

Original app. received date: 940101
 Facility closed date: Not Reported
 Mailing address: 1206 FARRAGUT CIRCLE
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "PYTEL, JEANNIE"
 Facility capacity: 14
 Type of clients served: 960
 Facility phone: 5307588498

S64 East SRDCCA200752180 Daycare
 1-2 mi 8748 Higher

EDR ID: SRDCCA200752180
 Facility number: 573603973
 Facility name: MERRYHILL SCHOOL
 Facility eval. code: 0303
 Facility office number: 03

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Facility county number: 57
 Facility type code: 850
 Facility status code: 03
 Address: 2650 LILLARD DRIVE
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 2565 MILLCREEK DRIVE
 City: SACRAMENTO
 State: CA
 Zip: 95833
 Facility investor: "NOBEL LEARNING COMMUNITIES, INC. "
 Licensee type: D
 License effective date: 980909
 License expiration date: Not Reported
 License issue date: 980831
 Program type: MAXIMUM CAPACITY: 60 PRESCHOOL CHILDREN. WAIVER ON FILE FOR OUTDOOR PLAYGROUND AND CHILDREN'S BATHROOM.
 Original app. received date: 980617
 Facility closed date: Not Reported
 Mailing address: 2565 MILLCREEK DR
 Mailing city: SACRAMENTO
 Mailing state: CA
 Mailing zip: 95833
 Contact person: "DUNBAR,LISA "
 Facility capacity: 60
 Type of clients served: 950
 Facility phone: 5302975100

S65
 East
 1-2 mi
 8748
 Higher

EDR ID: SRDCCA200746565
 Facility number: 573604042
 Facility name: MERRYHILL
 Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 840
 Facility status code: 03
 Address: 2650 LILLIARD DRIVE
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 1451 RIVER PARK DRIVE #141
 City: SACRAMENTO
 State: CA
 Zip: 95815
 Facility investor: "NOBEL LEARNING COMMUNITIES, INC. "
 Licensee type: D
 License effective date: 980909
 License expiration date: Not Reported
 License issue date: 980909
 Program type: MAXIMUM CAPACITY: 105 SCHOOL-AGE CHILDREN IN CLASSROOMS #5 THROUGH #14. COMBINATION CENTER.WAIVER ON FILE FOR OUTDOOR PLAYGROUND.
 Original app. received date: 980811
 Facility closed date: Not Reported

SRDCCA200746565
 Daycare

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Mailing address: 1451 RIVER PARK DRIVE #141
 Mailing city: SACRAMENTO
 Mailing state: CA
 Mailing zip: 95815
 Contact person: "DUNBAR, LISA"
 Facility capacity: 105
 Type of clients served: 950
 Facility phone: 5302975100

S66
 East
 1-2 mi
 8748
 Higher

Pss school id: A0300415
 Pss inst: MERRYHILL SCHOOL #1036
 Lograde: PK
 Higrade: 6
 Pss address: 2650 LILLARD DRIVE
 Pss city: DAVIS ROSA
 Pss county no: 113
 Pss county fips: 06113
 Pss stabb: CA
 Pss fips: 06
 Pss zip5: 95616
 Pss phone: 5302975100
 Pss sch days: 185
 Pss stu day hrs: 8
 Pss library: Yes
 Pss enroll ug: Not Reported
 Pss enroll pk: 14
 Pss enroll k: 129
 Pss enroll 1: 28
 Pss enroll 2: 24
 Pss enroll 3: 6
 Pss enroll 4: 4
 Pss enroll 5: 3
 Pss enroll 6: 4
 Pss enroll 7: Not Reported
 Pss enroll 8: Not Reported
 Pss enroll 9: Not Reported
 Pss enroll 10: Not Reported
 Pss enroll 11: Not Reported
 Pss enroll 12: Not Reported
 Pss enroll t: 212
 Pss enroll tk12: 198
 Pss race ai: Not Reported
 Pss race as: Not Reported
 Pss race h: Not Reported
 Pss race b: Not Reported
 Pss race w: Not Reported
 Pss fte teach: 14.1
 Pss locale: 3
 Pss coed: 1
 Pss type: 3
 Pss level: 1
 Pss relig: 3
 Pss comm type: 2
 Pss indian pct: Not Reported

SRPR20051023072
 Private Schools

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Pss asian pct: Not Reported
 Pss hisp pct: Not Reported
 Pss black pct: Not Reported
 Pss white pct: Not Reported
 Pss stdtch rt: 14.04
 Pss orient: 29
 Pss county name: YOLO
 Pss assoc 1: National Independent Private School Association (NIPSA)
 Pss assoc 2: Not Reported
 Pss assoc 3: Not Reported
 Pss assoc 4: Not Reported
 Pss assoc 5: Not Reported
 Pss assoc 6: Not Reported
 Pss assoc 7: Not Reported
 Source: NCESDATA_E72D09B4
 Edr id: SRPR20051023072

R67 NE EDR ID: SRDCCA200715692 SRDCCA200715692
 1-2 mi Facility number: 573607464 Daycare
 8874 Facility name: "HERNANDEZ, KAY "

Higher Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 810
 Facility status code: 03
 Address: 1205 CHESTNUT LANE
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 1205 CHESTNUT LANE
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "HERNANDEZ, KAY "
 Licensee type: A
 License effective date: 940302
 License expiration date: Not Reported
 License issue date: 940302
 Program type: "MAXIMUM CAPACITY: 6 CHILDREN WITH NO MORE THAN 3 INFANTS, OR 4
 INFANTSONLY, OR CAPACITY 8 CHILDREN WHEN 2 ARE AT LEAST 6 YEARS OF AGE
 WITH AMAXIMUM OF 2 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED
 "
 Original app. received date: 940301
 Facility closed date: Not Reported
 Mailing address: 1205 CHESTNUT LANE
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "HERNANDEZ, KAY "
 Facility capacity: 8
 Type of clients served: 960
 Facility phone: 5307535450

MAP FINDINGS

Map ID	Direction	Distance	Distance (ft.)	Elevation	Site	EDR ID	Database
N68						SRDCCA200738016	SRDCCA200738016
NNW						573613171	Daycare
1-2 mi						"	
8941						"VALCARENGHI, MICHELLE	
Higher						0303	
						03	
						57	
						810	
						03	
						765 BIANCO COURT	
						DAVIS	
						CA	
						95616	
						765 BIANCO COURT	
						DAVIS	
						CA	
						95616	
						"VALCARENGHI, MICHELLE	"
						A	
						61204	
						Not Reported	
						061204	
						"MAX. CAP: 6 - NO MORE THAN 3 INFANTS OR 4 INFANTS ONLY. CAP 8 - NO MORE THAN 2 INFANTS, 1 CHILD IN KINDERGARTEN OR ELEMENTARY SCHOOL AND 1 CHILD AT LEAST AGE 6. OFF LIMITS: UPSTAIRS AND GARAGE. "	
						061109	
						Not Reported	
						765 BIANCO COURT	
						DAVIS	
						CA	
						95616	
						"VALCARENGHI, MICHELLE	"
						8	
						960	
						5307562949	
<hr/>							
Q69						SRDCCA200716392	SRDCCA200716392
WNW						573607522	Daycare
1-2 mi							
9014						MEDINA, ELIZABETH	
Higher						0303	
						03	
						57	
						810	
						03	
						2124 HUMBOLT AVE	
						DAVIS	
						CA	
						95616	
						2124 HUMBOLT AVE	
						DAVIS	
						CA	
						95616	
						"MEDINA, ELIZABETH	"
						A	

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

License effective date: 940912
 License expiration date: Not Reported
 License issue date: 940912
 Program type: "MAXIMUM CAPACITY: 6 CHILDREN WITH NO MORE THAN 3 INFANTS, OR 4 INFANTSONLY, OR CAPACITY 8 CHILDREN WHEN 2 ARE AT LEAST 6 YEARS OF AGE WITH AMAXIMUM OF 2 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED"
 Original app. received date: 940101
 Facility closed date: Not Reported
 Mailing address: 2124 HUMBOLT AVE
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "MEDINA, ELIZABETH"
 Facility capacity: 8
 Type of clients served: 960
 Facility phone: 5307538271

O70		SRDCCA200723669
ENE	EDR ID:	SRDCCA200723669
1-2 mi	Facility number:	573609481
9066	Facility name:	"TROSTEL, TAMI"
Higher	Facility eval. code:	0303
	Facility office number:	03
	Facility county number:	57
	Facility type code:	810
	Facility status code:	03
	Address:	2109 E. 8TH STREET
	City:	DAVIS
	State:	CA
	Zip:	95616
	Alt. address:	2109 E. 8TH STREET
	City:	DAVIS
	State:	CA
	Zip:	95616
	Facility investor:	"TROSTEL, TAMI"
	Licensee type:	A
	License effective date:	30827
	License expiration date:	Not Reported
	License issue date:	030827
	Program type:	MAX. CAP (WHEN THERE IS AN ASSISTANT PRESENT): 12 - NO MORE THAN 4 INFANTS. CAP 14 - NO MORE THAN 3 INFANTS. 1 CHILD IN KINDERGARTEN OR ELEMENTARY SCHOOL AND 1 CHILD AT LEAST AGE 6.OFFLIMITS: UPSTAIRS.
	Original app. received date:	030815
	Facility closed date:	Not Reported
	Mailing address:	2109 E. 8TH STREET
	Mailing city:	DAVIS
	Mailing state:	CA
	Mailing zip:	95616
	Contact person:	"TROSTEL, TAMI"
	Facility capacity:	14
	Type of clients served:	960
	Facility phone:	5307533468

MAP FINDINGS

Map ID Direction Distance Distance (ft.) Elevation	Site	EDR ID Database
T71 NE 1-2 mi 9207 Higher	EDR ID: SRDCCA200733503 Facility number: 573611463 Facility name: "ALARCON-SOTO, SANDRA& SOTO, JUAN" Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 810 Facility status code: 03 Address: 1504 CYPRESS LANE City: DAVIS State: CA Zip: 95616 Alt. address: 1504 CYPRESS LANE City: DAVIS State: CA Zip: 95616 Facility investor: "ALARCON-SOTO, SANDRA& SOTO, JUAN" Licensee type: A License effective date: 60607 License expiration date: Not Reported License issue date: 060607 Program type: "MAXIMUM CAPACITY: 6 - NO MORE THAN 3 INFANTS OR 4 INFANTS ONLY. CAPACITY 8 - NO MORE THAN 2 INFANTS, 1 CHILD IN KINDERGARTEN OR ELEMENTARY SCHOOL AND 1 CHILD AT LEAST AGE 6. OFF LIMITS: SIDE YARDS ""GARAGE, MASTER BEDROOM AND DAUGHTHER'S ROOM." " Original app. received date: 051102 Facility closed date: Not Reported Mailing address: 1504 CYPRESS LANE Mailing city: DAVIS Mailing state: CA Mailing zip: 95616 Contact person: "ALARCON-SOTO, SANDRA" Facility capacity: 8 Type of clients served: 960 Facility phone: 5307532575	SRDCCA200733503 Daycare
R72 NE 1-2 mi 9210 Higher	EDR ID: SRDCCA200726508 Facility number: 573610205 Facility name: "SAH, VINA" Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 810 Facility status code: 03 Address: 1200 SNYDER DRIVE City: DAVIS State: CA Zip: 95616 Alt. address: 1200 SNYDER DRIVE City: DAVIS State: CA Zip: 95616	SRDCCA200726508 Daycare

MAP FINDINGS

Map ID Direction Distance Distance (ft.) Elevation	Site	EDR ID Database
	Facility investor: "SAH, VINA" Licensee type: A License effective date: 40916 License expiration date: Not Reported License issue date: 040916 Program type: "MAXIMUM CAPACITY: 6 CHILDREN WITH NO MORE THAN 3 INFANTS, OR 4 INFANTSONLY, OR CAPACITY 8 CHILDREN WHEN 2 ARE AT LEAST 6 YEARS OF AGE WITH AMAXIMUM OF 2 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED. ""OFF LIMITS: GARAGE, MASTER BEDROOM, GUEST BEDROOM." Original app. received date: 040720 Facility closed date: Not Reported Mailing address: 1200 SNYDER DRIVE Mailing city: DAVIS Mailing state: CA Mailing zip: 95616 Contact person: "SAH, VINA" Facility capacity: 8 Type of clients served: 960 Facility phone: 5307588589	
R73 NE 1-2 mi 9223 Higher	EDR ID: SRDCCA200717749 Facility number: 573608448 Facility name: "HASSAN, MABEL" Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 810 Facility status code: 03 Address: 1318 CHESTNUT LANE City: DAVIS State: CA Zip: 95616 Alt. address: 1318 CHESTNUT LANE City: DAVIS State: CA Zip: 95616 Facility investor: "HASSAN, MABEL" Licensee type: A License effective date: 30424 License expiration date: Not Reported License issue date: 030424 Program type: "MAXIMUM CAPACITY: 12 CHILDREN, WITH NO MORE THAN 4 INFANTS, OR CAPACITY 14 CHILDREN WHEN 2 CHILDREN ARE AT LEAST 6 YEARS OF AGE WITH A MAXIMUM OF 3 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED. "OFFLIMITS: GARAGE. Original app. received date: 020730 Facility closed date: Not Reported Mailing address: 1318 CHESTNUT LANE Mailing city: DAVIS Mailing state: CA Mailing zip: 95616 Contact person: "HASSAN, MABEL" Facility capacity: 14	SRDCCA200717749 Daycare

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Type of clients served: 960
 Facility phone: 5307562258

R74		SRDCCA200715729
NE	EDR ID:	SRDCCA200715729
1-2 mi	Facility number:	573607563
9292	Facility name:	"RAJBHANDARI, VIDYA "
Higher	Facility eval. code:	0303
	Facility office number:	03
	Facility county number:	57
	Facility type code:	810
	Facility status code:	03
	Address:	1320 MADRONE PLACE
	City:	DAVIS
	State:	CA
	Zip:	95616
	Alt. address:	1320 MADRONE PLACE
	City:	DAVIS
	State:	CA
	Zip:	95616
	Facility investor:	"RAJBHANDARI, VIDYA "
	Licensee type:	A
	License effective date:	970613
	License expiration date:	Not Reported
	License issue date:	970613
	Program type:	LICENSE IS INACTIVE FROM 7/1/05 UNTIL 6/30/06.
	Original app. received date:	970101
	Facility closed date:	Not Reported
	Mailing address:	1320 MADRONE PLACE
	Mailing city:	DAVIS
	Mailing state:	CA
	Mailing zip:	95616
	Contact person:	"RAJBHANDARI, VIDYA "
	Facility capacity:	8
	Type of clients served:	960
	Facility phone:	5307561733

U75		SRHO20070158046
NW	Hospital type:	01
1-2 mi	Num of times COO:	00
9422	Owner date:	Not Reported
Higher	City:	DAVIS
	Has plan of corr:	Not Reported
	Compliance status:	Not Reported
	SSA county code:	670
	Cross ref number:	Not Reported
	FMS survey date:	Not Reported
	Current survey date:	Not Reported
	Medicare/Medicaid:	Not Reported
	Facility name:	UNIVERSITY RETIREMENT COMM AT DAVIS
	Intermediary/Carrier:	Not Reported
	Medicaid number:	Not Reported
	Participation date:	20000324
	Prior COO date:	Not Reported

MAP FINDINGS

Map ID
Direction
Distance
Distance (ft.)
Elevation

Site

EDR ID
Database

Prior carrier: Not Reported
 Provider ID: 05D0971910
 Record Status: A
 Region code: 09
 Is Partial Record: Y
 state abbrev: CA
 ssa state: 05
 state region cd: LAB
 street address: 1515 SHASTA DR
 Phone num: 5307477030
 Termination reason: 00
 Term Date: 20080323
 Purpose of action: Not Reported
 Provider control: 02
 Zip: 95616
 Fips state: 06
 Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0000
 Num cert beds: 0000
 Source: US_HOSPITAL_POSCLIA
 Edr id: SRHO20070158046

U76
 NW
 1-2 mi
 9422
 Higher

Hospital type: 01
 Num of times COO: 00
 Owner date: Not Reported
 City: DAVIS
 Has plan of corr: 1
 Compliance status: A
 SSA county code: 670
 Cross ref number: Not Reported
 FMS survey date: Not Reported
 Current survey date: 20060425
 Medicare/Medicaid: 1
 Facility name: UNIVERSITY RETIREMENT
 Intermediary/Carrier: 52280
 Medicaid number: Not Reported
 Participation date: 20010416
 Prior COO date: Not Reported
 Prior carrier: Not Reported
 Provider ID: 555769
 Record Status: A
 Region code: 09
 Is Partial Record: Not Reported
 state abbrev: CA
 ssa state: 05
 state region cd: S1
 street address: 1515 SHASTA DRIVE
 Phone num: 5307477000
 Termination reason: 00

SRHO20070108901
 AHA Hospitals

MAP FINDINGS

Map ID
Direction
Distance
Distance (ft.)
Elevation

Site

EDR ID
Database

Term Date: Not Reported
 Purpose of action: 2
 Provider control: 05
 Zip: 95616
 Fips state: 06
 Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0037
 Num cert beds: 0037
 Source: US_HOSPITAL_POSOTHER
 Edr id: SRHO20070108901

U77
 NW
 1-2 mi
 9422
 Higher

Provnum: 555769
 Nursinghomename: UNIVERSITY RETIREMENT
 Street: 1515 SHASTA DRIVE
 City: DAVIS
 State: CA
 Zipcode: 95616
 Phonenummer: 5307477000
 Dateoflastinspection: 20060421
 Certifiednumberofbeds: 37
 Totalnumberofresidents: 30
 Percofoccupiedbeds: 81
 Categorydescription: Participating in Medicare Only
 Typeofownership: Non profit - Corporation
 Locatedwithinahospital: NO
 Multinursinghomeownership: YES
 Residentandfamilycouncils: RESIDENT
 Edr id: SRNH20060915164

SRNH20060915164
 Nursing Homes

V78
 WNW
 1-2 mi
 9472
 Higher

EDR ID: SRDCCA200746632
 Facility number: 573605645
 Facility name: A WORLD OF LEARNING
 Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 840
 Facility status code: 03
 Address: 2417 OAKENSHIELD
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 610 7TH STREET
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "WALLIN, NANCY
 Licensee type: A

SRDCCA200746632
 Daycare

MAP FINDINGS

Map ID
Direction
Distance
Distance (ft.)
Elevation

Site

EDR ID
Database

License effective date: 908
 License expiration date: Not Reported
 License issue date: 000908
 Program type: Not Reported
 Original app. received date: 000623
 Facility closed date: Not Reported
 Mailing address: 610 7TH STREET
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "WALLIN, NANCY"
 Facility capacity: 15
 Type of clients served: 950
 Facility phone: 5307503727

79
 ENE
 1-2 mi
 9472
 Higher

EDR ID: SRDCCA200716373
 Facility number: 573607396
 Facility name: "CHANG, DEE"
 Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 810
 Facility status code: 03
 Address: 2634 ALBANY AVE
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 2634 ALBANY AVE
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "CHANG, DEE"
 Licensee type: A
 License effective date: 1011
 License expiration date: Not Reported
 License issue date: 001011
 Program type: "MAXIMUM CAPACITY: 12 CHILDREN WITH NO MORE THAN 4 INFANTS, OR

SRDCCA200716373
 Daycare

CAPACITY14 CHILDREN WHEN 2 CHILDREN ARE AT LEAST 6 YEARS OF AGE WITH A
 MAXIMUMOF 3 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED
 "

Original app. received date: 000101
 Facility closed date: Not Reported
 Mailing address: 2634 ALBANY AVE
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "CHANG, DEE"
 Facility capacity: 14
 Type of clients served: 960
 Facility phone: 5307583619

MAP FINDINGS

Map ID	Direction	Distance	Distance (ft.)	Elevation	Site	EDR ID	Database
W80	NW	1-2 mi	9604	Higher	EDR ID: SRDCCA200742904 Facility number: 570319082 Facility name: PATWIN SCHOOL AGE CHILD DEVELOPMENT CENTER Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 840 Facility status code: 03 Address: 2222 SHASTA DRIVE City: DAVIS State: CA Zip: 95616 Alt. address: 2222 SHASTA DRIVE City: DAVIS State: CA Zip: 95616 Facility investor: CHILD DEVELOPMENT CENTERS Licensee type: C License effective date: 951009 License expiration date: Not Reported License issue date: 921009 Program type: LICENSED TO SERVE CHILDREN ENROLLED IN KINDERGARTEN AND ABOVE IN CDC PORTABLE CLASSROOMS 1 AND 2. Original app. received date: 920623 Facility closed date: Not Reported Mailing address: "851 E HAMILTON AVE., STE 200 " Mailing city: CAMPBELL Mailing state: CA Mailing zip: 95008 Contact person: "FADE, LISA " Facility capacity: 53 Type of clients served: 950 Facility phone: 5307561369	SRDCCA200742904	Daycare
W81	NW	1-2 mi	9604	Higher	Ncessch: 061062001905 Schname05: PATWIN ELEMENTARY Mstreet05: 2222 SHASTA DR. Mcity05: DAVIS Mstate05: CA Mzip05: 95616 Mzip405: 6634 Member05: 442 Phone05: (530) 757-5383 Locale05: 3 Type05: 1 Level05: 1 Gslo05: KG Gshi05: 06 Edr id: SRPU20071014153	SRPU20071014153	Public Schools

MAP FINDINGS

Map ID Direction Distance Distance (ft.) Elevation	Site	EDR ID Database
82 NE 1-2 mi 9724 Higher	Hospital type: 01 Num of times COO: 00 Owner date: Not Reported City: DAVIS Has plan of corr: Not Reported Compliance status: Not Reported SSA county code: 670 Cross ref number: Not Reported FMS survey date: Not Reported Current survey date: Not Reported Medicare/Medicaid: Not Reported Facility name: WELLNESS EXPRESS CLINIC Intermediary/Carrier: Not Reported Medicaid number: Not Reported Participation date: 20050420 Prior COO date: Not Reported Prior carrier: Not Reported Provider ID: 05D1039739 Record Status: A Region code: 09 Is Partial Record: Y state abbrev: CA ssa state: 05 state region cd: M2 street address: 1550 E COVELL BLVD Phone num: 9258553222 Termination reason: 00 Term Date: 20070419 Purpose of action: Not Reported Provider control: 04 Zip: 95616 Fips state: 06 Fips cnty: 113 SSA MSA: 499 SSA MSA size code: B Date accredited: Not Reported Accred expire date: Not Reported Accred Org: Not Reported Num beds: 0000 Num cert beds: 0000 Source: US_HOSPITAL_POSCLIA Edr id: SRHO20070159477	SRHO20070159477 AHA Hospitals
V83 NW 1-2 mi 9810 Higher	EDR ID: SRDCCA200718757 Facility number: 573607656 Facility name: "NOORISTANI, TAIBA" Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 810 Facility status code: 03 Address: 2324 SHASTA DR. #22 City: DAVIS	SRDCCA200718757 Daycare

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

State: CA
 Zip: 95616
 Alt. address: 2324 SHASTA DR. #22
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "NOORISTANI, TAIBA"
 Licensee type: A
 License effective date: 10829
 License expiration date: Not Reported
 License issue date: 010829
 Program type: "MAXIMUM CAPACITY: 6 CHILDREN WITH NO MORE THAN 3 INFANTS, OR 4 INFANTSONLY, OR CAPACITY 8 CHILDREN WHEN 2 ARE AT LEAST 6 YEARS OF AGE WITH AMAXIMUM OF 2 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED"
 Original app. received date: 010301
 Facility closed date: Not Reported
 Mailing address: 2324 SHASTA DR. #22
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "NOORISTANI, TAIBA"
 Facility capacity: 8
 Type of clients served: 960
 Facility phone: 5307561507

T84 NE 1-2 mi 9810 Higher	EDR ID: Facility number: Facility name: Facility eval. code: Facility office number: Facility county number: Facility type code: Facility status code: Address: City: State: Zip: Alt. address: City: State: Zip: Facility investor: Licensee type: License effective date: License expiration date: License issue date: Program type: Original app. received date: Facility closed date:	SRDCCA200718702 Daycare 573608207 "HARZULA, RUTH" 0303 03 57 810 03 1512 MADRONE LANE DAVIS CA 95616 1512 MADRONE LANE DAVIS CA 95616 "HARZULA, RUTH" A 20730 Not Reported 020730 "MAXIMUM CAPACITY: 12 CHILDREN, WITH NO MORE THAN 4 INFANTS, OR CAPACITY 14 CHILDREN WHEN 2 CHILDREN ARE AT LEAST 6 YEARS OF AGE WITH A MAXIMUM OF 3 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED" "OFFLIMITS: ALL BEDROOMS, GARAGE." " 020516 Not Reported	SRDCCA200718702 Daycare
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MAP FINDINGS

Map ID
Direction
Distance
Distance (ft.)
Elevation

Site

EDR ID
Database

Mailing address: 1512 MADRONE LANE
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "HARZULA, RUTH"
 Facility capacity: 14
 Type of clients served: 960
 Facility phone: 5307532203

U85
 NW
 1-2 mi
 9817
 Higher

Hospital type: 01
 Num of times COO: 00
 Owner date: Not Reported
 City: DAVIS
 Has plan of corr: Not Reported
 Compliance status: Not Reported
 SSA county code: 670
 Cross ref number: Not Reported
 FMS survey date: Not Reported
 Current survey date: Not Reported
 Medicare/Medicaid: Not Reported
 Facility name: SUTTER DAVIS HOSPITAL PULMONARY LAB
 Intermediary/Carrier: Not Reported
 Medicaid number: Not Reported
 Participation date: 19950929
 Prior COO date: Not Reported
 Prior carrier: Not Reported
 Provider ID: 05D0906201
 Record Status: A
 Region code: 09
 Is Partial Record: Y
 state abbrev: CA
 ssa state: 05
 state region cd: LAB
 street address: 2000 SUTTER PLACE
 Phone num: 9167575129
 Termination reason: 33
 Term Date: 19970928
 Purpose of action: Not Reported
 Provider control: 02
 Zip: 95617
 Fips state: 06
 Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0000
 Num cert beds: 0000
 Source: US_HOSPITAL_POSCLIA
 Edr id: SRHO20070150097

SRHO20070150097
 AHA Hospitals

MAP FINDINGS

Map ID	Direction	Distance	Distance (ft.)	Elevation	Site	EDR ID Database
U86	NW	1-2 mi	9817	Higher	Hospital type: 01 Num of times COO: 01 Owner date: 19960531 City: DAVIS Has plan of corr: 1 Compliance status: A SSA county code: 670 Cross ref number: Not Reported FMS survey date: Not Reported Current survey date: 19860829 Medicare/Medicaid: 1 Facility name: SUTTER DAVIS HOSPITAL Intermediary/Carrier: 00040 Medicaid number: Not Reported Participation date: 19680212 Prior COO date: Not Reported Prior carrier: 51051 Provider ID: 050537 Record Status: A Region code: 09 Is Partial Record: Not Reported state abbrev: CA ssa state: 05 state region cd: S1 street address: 2000 SUTTER PLACE Phone num: 5307566440 Termination reason: 00 Term Date: Not Reported Purpose of action: 2 Provider control: 04 Zip: 95616 Fips state: 06 Fips cnty: 113 SSA MSA: 499 SSA MSA size code: B Date accredited: 19830930 Accred expire date: 19860930 Accred Org: 1 Num beds: 0048 Num cert beds: 0048 Source: US_HOSPITAL_POSOTHER Edr id: SRHO20070007485	SRHO20070007485 AHA Hospitals
U87	NW	1-2 mi	9911	Higher	Hospital type: 01 Num of times COO: 00 Owner date: Not Reported City: DAVIS Has plan of corr: Not Reported Compliance status: Not Reported SSA county code: 670 Cross ref number: Not Reported FMS survey date: Not Reported Current survey date: Not Reported	SRHO20070148275 AHA Hospitals

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Medicare/Medicaid: Not Reported
 Facility name: ALICE VAN ALSTINE, MD
 Intermediary/Carrier: Not Reported
 Medicaid number: Not Reported
 Participation date: 19960509
 Prior COO date: Not Reported
 Prior carrier: Not Reported
 Provider ID: 05D0914772
 Record Status: A
 Region code: 09
 Is Partial Record: Y
 state abbrev: CA
 ssa state: 05
 state region cd: LAB
 street address: 2020 SUTTER PLACE #101
 Phone num: 9167505800
 Termination reason: 08
 Term Date: 20020508
 Purpose of action: Not Reported
 Provider control: 04
 Zip: 95616
 Fips state: 06
 Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0000
 Num cert beds: 0000
 Source: US_HOSPITAL_POSCLIA
 Edr id: SRHO20070148275

U88
 NW
 1-2 mi
 9911
 Higher

Hospital type: 01
 Num of times COO: 00
 Owner date: Not Reported
 City: DAVIS
 Has plan of corr: Not Reported
 Compliance status: Not Reported
 SSA county code: 670
 Cross ref number: Not Reported
 FMS survey date: Not Reported
 Current survey date: Not Reported
 Medicare/Medicaid: Not Reported
 Facility name: WILLIAM HOCH MD
 Intermediary/Carrier: Not Reported
 Medicaid number: Not Reported
 Participation date: 19930107
 Prior COO date: Not Reported
 Prior carrier: Not Reported
 Provider ID: 05D0856966
 Record Status: A
 Region code: 09
 Is Partial Record: Not Reported

SRHO20070142588
 AHA Hospitals

MAP FINDINGS

Map ID
Direction
Distance
Distance (ft.)
Elevation

Site

EDR ID
Database

state abbrev: CA
 ssa state: 05
 state region cd: LAB
 street address: 2020 SUTTER PLACE #102
 Phone num: 9167505858
 Termination reason: 08
 Term Date: 19980831
 Purpose of action: Not Reported
 Provider control: 04
 Zip: 95616
 Fips state: 06
 Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0000
 Num cert beds: 0000
 Source: US_HOSPITAL_POSCLIA
 Edr id: SRHO20070142588

U89
 NW
 1-2 mi
 9911
 Higher

Hospital type: 01
 Num of times COO: 00
 Owner date: Not Reported
 City: DAVIS
 Has plan of corr: Not Reported
 Compliance status: Not Reported
 SSA county code: 670
 Cross ref number: Not Reported
 FMS survey date: Not Reported
 Current survey date: Not Reported
 Medicare/Medicaid: Not Reported
 Facility name: JOHN D HERNRIED, MD
 Intermediary/Carrier: Not Reported
 Medicaid number: Not Reported
 Participation date: 19940602
 Prior COO date: Not Reported
 Prior carrier: Not Reported
 Provider ID: 05D0887024
 Record Status: A
 Region code: 09
 Is Partial Record: Y
 state abbrev: CA
 ssa state: 05
 state region cd: LAB
 street address: 2020 SUTTER PLACE
 Phone num: 9167505904
 Termination reason: 08
 Term Date: 20000601
 Purpose of action: Not Reported
 Provider control: 04
 Zip: 95616
 Fips state: 06

SRHO20070144499
 AHA Hospitals

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0000
 Num cert beds: 0000
 Source: US_HOSPITAL_POSCLIA
 Edr id: SRHO20070144499

U90
 NW
 1-2 mi
 9911
 Higher

Hospital type: 01
 Num of times COO: 00
 Owner date: Not Reported
 City: DAVIS
 Has plan of corr: Not Reported
 Compliance status: Not Reported
 SSA county code: 670
 Cross ref number: Not Reported
 FMS survey date: Not Reported
 Current survey date: Not Reported
 Medicare/Medicaid: Not Reported
 Facility name: PETER E DROUBAY MD
 Intermediary/Carrier: Not Reported
 Medicaid number: Not Reported
 Participation date: 19930521
 Prior COO date: Not Reported
 Prior carrier: Not Reported
 Provider ID: 05D0613718
 Record Status: A
 Region code: 09
 Is Partial Record: Not Reported
 state abbrev: CA
 ssa state: 05
 state region cd: LAB
 street address: 2020 SUTTER PL #202
 Phone num: 9167588751
 Termination reason: 07
 Term Date: 19940831
 Purpose of action: Not Reported
 Provider control: 04
 Zip: 95616
 Fips state: 06
 Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0000
 Num cert beds: 0000
 Source: US_HOSPITAL_POSCLIA
 Edr id: SRHO20070135395

SRHO20070135395
 AHA Hospitals

MAP FINDINGS

Map ID Direction Distance Distance (ft.) Elevation	Site	EDR ID Database
U91 NW 1-2 mi 9911 Higher	Hospital type: 01 Num of times COO: 00 Owner date: Not Reported City: DAVIS Has plan of corr: Not Reported Compliance status: Not Reported SSA county code: 670 Cross ref number: Not Reported FMS survey date: Not Reported Current survey date: Not Reported Medicare/Medicaid: Not Reported Facility name: CHARLES A DERBY MD Intermediary/Carrier: Not Reported Medicaid number: Not Reported Participation date: 19921222 Prior COO date: Not Reported Prior carrier: Not Reported Provider ID: 05D0717027 Record Status: A Region code: 09 Is Partial Record: Y state abbrev: CA ssa state: 05 state region cd: LAB street address: 2020 SUTTER PLACE #101 Phone num: 9167532804 Termination reason: 17 Term Date: 19960902 Purpose of action: Not Reported Provider control: 04 Zip: 95616 Fips state: 06 Fips cnty: 113 SSA MSA: 499 SSA MSA size code: B Date accredited: Not Reported Accred expire date: Not Reported Accred Org: Not Reported Num beds: 0000 Num cert beds: 0000 Source: US_HOSPITAL_POSCLIA Edr id: SRHO20070141220	SRHO20070141220 AHA Hospitals
U92 NW 1-2 mi 9911 Higher	Hospital type: 01 Num of times COO: 00 Owner date: Not Reported City: DAVIS Has plan of corr: Not Reported Compliance status: Not Reported SSA county code: 670 Cross ref number: Not Reported FMS survey date: Not Reported Current survey date: Not Reported	SRHO20070144443 AHA Hospitals

MAP FINDINGS

Map ID
Direction
Distance
Distance (ft.)
Elevation

Site

EDR ID
Database

Medicare/Medicaid: Not Reported
 Facility name: INTERNAL MEDICINE CONSULTANTS
 Intermediary/Carrier: Not Reported
 Medicaid number: Not Reported
 Participation date: 19940416
 Prior COO date: Not Reported
 Prior carrier: Not Reported
 Provider ID: 05D0885159
 Record Status: A
 Region code: 09
 Is Partial Record: Y
 state abbrev: CA
 ssa state: 05
 state region cd: LAB
 street address: 2020 SUTTER PL #202
 Phone num: Not Reported
 Termination reason: 08
 Term Date: 20020415
 Purpose of action: Not Reported
 Provider control: 02
 Zip: 95616
 Fips state: 06
 Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0000
 Num cert beds: 0000
 Source: US_HOSPITAL_POSCLIA
 Edr id: SRHO20070144443

U93
 NW
 1-2 mi
 10006
 Higher

Hospital type: 01
 Num of times COO: 00
 Owner date: Not Reported
 City: DAVIS
 Has plan of corr: Not Reported
 Compliance status: A
 SSA county code: 670
 Cross ref number: Not Reported
 FMS survey date: Not Reported
 Current survey date: 20020411
 Medicare/Medicaid: 1
 Facility name: DAVIS COMMUNITY CLINIC,THE
 Intermediary/Carrier: Not Reported
 Medicaid number: Not Reported
 Participation date: 19920901
 Prior COO date: Not Reported
 Prior carrier: Not Reported
 Provider ID: 05D0697113
 Record Status: A
 Region code: 09
 Is Partial Record: Not Reported

SRHO20070139307
 AHA Hospitals

MAP FINDINGS

Map ID
Direction
Distance
Distance (ft.)
Elevation

Site

EDR ID
Database

state abbrev: CA
 ssa state: 05
 state region cd: M2
 street address: 2040 SUTTER PLACE
 Phone num: 5307582060
 Termination reason: 00
 Term Date: 20081218
 Purpose of action: 2
 Provider control: 02
 Zip: 95616
 Fips state: 06
 Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0000
 Num cert beds: 0000
 Source: US_HOSPITAL_POSCLIA
 Edr id: SRHO20070139307

U94
 NW
 1-2 mi
 10006
 Higher

Hospital type: 01
 Num of times COO: 00
 Owner date: Not Reported
 City: DAVIS
 Has plan of corr: Not Reported
 Compliance status: Not Reported
 SSA county code: 670
 Cross ref number: Not Reported
 FMS survey date: Not Reported
 Current survey date: Not Reported
 Medicare/Medicaid: Not Reported
 Facility name: DAVIS COMMUNITY CLINIC
 Intermediary/Carrier: 00450
 Medicaid number: Not Reported
 Participation date: 20050309
 Prior COO date: Not Reported
 Prior carrier: Not Reported
 Provider ID: 051003
 Record Status: A
 Region code: 09
 Is Partial Record: Y
 state abbrev: CA
 ssa state: 05
 state region cd: S1
 street address: 2040 SUTTER PLACE
 Phone num: 5307582060
 Termination reason: 00
 Term Date: Not Reported
 Purpose of action: 1
 Provider control: 02
 Zip: 95616
 Fips state: 06

SRHO20070009629
 AHA Hospitals

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0000
 Num cert beds: 0000
 Source: US_HOSPITAL_POSOTHER
 Edr id: SRHO20070009629

95 North EDR ID: SRDCCA200716483 SRDCCA200716483
 1-2 mi Facility number: 573607443 Daycare
 10035 Facility name: "GREENAMYER, ALICIA "

Higher Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 810
 Facility status code: 03
 Address: 215 HUERTA PLACE
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 215 HUERTA PLACE
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "GREENAMYER, ALICIA & GERALD "
 Licensee type: A
 License effective date: 931101
 License expiration date: Not Reported
 License issue date: 931101
 Program type: "MAXIMUM CAPACITY: 12 CHILDREN, WITH NO MORE THAN 4 INFANTS, OR
 CAPACITY 14 CHILDREN WHEN 2 CHILDREN ARE AT LEAST 6 YEARS OF AGE WITH A
 MAXIMUM OF 3 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED "
 Original app. received date: 930101
 Facility closed date: Not Reported
 Mailing address: 215 HUERTA PLACE
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "GREENAMYER, ALICIA "
 Facility capacity: 14
 Type of clients served: 960
 Facility phone: 5307562478

T96 NE EDR ID: SRDCCA200723781 SRDCCA200723781
 1-2 mi Facility number: 573609453 Daycare
 10110 Facility name: "BOUGHTON, KRISTEN "
 Higher Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57

MAP FINDINGS

Map ID
Direction
Distance
Distance (ft.)
Elevation

Site

EDR ID
Database

Facility type code: 810
 Facility status code: 03
 Address: 1711 CHAPMAN PLACE
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 1711 CHAPMAN PLACE
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "BOUGHTON, KRISTEN"
 Licensee type: A
 License effective date: 31103
 License expiration date: Not Reported
 License issue date: 031103
 Program type: "MAXIMUM CAPACITY: 6 CHILDREN WITH NO MORE THAN 3 INFANTS, OR 4
 INFANTSONLY, OR CAPACITY 8 CHILDREN WHEN 2 ARE AT LEAST 6 YEARS OF AGE
 WITH AMAXIMUM OF 2 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS
 REQUIRED. "OFFLIMITS: MASTER BEDROOM.
 Original app. received date: 030811
 Facility closed date: Not Reported
 Mailing address: 1711 CHAPMAN PLACE
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "BOUGHTON, KRISTEN"
 Facility capacity: 8
 Type of clients served: 960
 Facility phone: 5307588186

X97 NE 1-2 mi 10230 Higher	Ncessch: Scname05: Mstreet05: Mcity05: Mstate05: Mzip05: Mzip405: Member05: Phone05: Locale05: Type05: Level05: Gslo05: Gshi05: Edr id:	061062001175 BIRCH LANE ELEMENTARY 1600 BIRCH LN. DAVIS CA 95616 1499 614 (530) 757-5395 3 1 1 KG 06 SRPU20071014143	SRPU20071014143 Public Schools
--	---	--	-----------------------------------

Y98 WNW 1-2 mi 10314 Higher	EDR ID: Facility number: Facility name: Facility eval. code: Facility office number: Facility county number:	SRDCCA200754334 573608094 PARKSIDE CHILDREN'S HOUSE 0303 03 57	SRDCCA200754334 Daycare
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MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Facility type code: 850
 Facility status code: 03
 Address: 2907 PORTAGE BAY WEST
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 2907 PORTAGE BAY WEST
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "HERNANDEZ, KARRIE"
 Licensee type: A
 License effective date: 20812
 License expiration date: Not Reported
 License issue date: 020812
 Program type: LICENSED TO SERVE CHILDREN FROM AGE 2 YEARS UNTIL ENTRY INTO FIRST GRADE.
 Original app. received date: 020416
 Facility closed date: Not Reported
 Mailing address: 2902 ROCKWELL CT.
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "HERNANDEZ, KARRIE"
 Facility capacity: 84
 Type of clients served: 950
 Facility phone: 5307532097

Y99
 WNW
 1-2 mi
 10314
 Higher

Pss school id: A9100758
 Pss inst: PARKSIDE CHILDREN'S HOUSE
 Lograde: PK
 Higrade: K
 Pss address: 2907 PORTAGE BAY WEST
 Pss city: DAVIS
 Pss county no: 113
 Pss county fips: 06113
 Pss stabb: CA
 Pss fips: 06
 Pss zip5: 95616
 Pss phone: 5307532030
 Pss sch days: 180
 Pss stu day hrs: 6
 Pss library: Yes
 Pss enroll ug: Not Reported
 Pss enroll pk: 52
 Pss enroll k: 12
 Pss enroll 1: Not Reported
 Pss enroll 2: Not Reported
 Pss enroll 3: Not Reported
 Pss enroll 4: Not Reported
 Pss enroll 5: Not Reported
 Pss enroll 6: Not Reported
 Pss enroll 7: Not Reported
 Pss enroll 8: Not Reported

SRPR20051022112
 Private Schools

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Pss enroll 9: Not Reported
 Pss enroll 10: Not Reported
 Pss enroll 11: Not Reported
 Pss enroll 12: Not Reported
 Pss enroll t: 64
 Pss enroll tk12: 12
 Pss race ai: 0
 Pss race as: 2
 Pss race h: 1
 Pss race b: 0
 Pss race w: 9
 Pss fte teach: 8.5
 Pss locale: 3
 Pss coed: 1
 Pss type: 2
 Pss level: 1
 Pss relig: 3
 Pss comm type: 2
 Pss indian pct: 0
 Pss asian pct: 16.67
 Pss hisp pct: 8.33
 Pss black pct: 0
 Pss white pct: 75
 Pss stdtch rt: 1.41
 Pss orient: 29
 Pss county name: YOLO
 Pss assoc 1: American Montessori Society (AMS)
 Pss assoc 2: Not Reported
 Pss assoc 3: Not Reported
 Pss assoc 4: Not Reported
 Pss assoc 5: Not Reported
 Pss assoc 6: Not Reported
 Pss assoc 7: Not Reported
 Source: NCESDATA_E72D09B4
 Edr id: SRPR20051022112

X100
 NE
 1-2 mi
 10408
 Higher

EDR ID: SRDCCA200742816
 Facility number: 570313700
 Facility name: BIRCH LANE SCHOOL-AGE CHILD DEVELOPMENT CENTER
 Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 840
 Facility status code: 03
 Address: 1700 BIRCH LANE
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: "851 E. HAMILTON AVE., STE 200 "
 City: CAMPBELL
 State: CA
 Zip: 95008
 Facility investor: CHILD DEVELOPMENT CENTERS
 Licensee type: C

SRDCCA200742816
 Daycare

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

License effective date: 930904
 License expiration date: Not Reported
 License issue date: 900904
 Program type: LICENSED TO SERVE CHILDREN ENROLLED IN KINDERGARTEN AND ABOVE IN THE CDC TWO ROOM PORTABLE ONLY.
 Original app. received date: 900813
 Facility closed date: Not Reported
 Mailing address: "851 E. HAMILTON AVE., STE 200 "
 Mailing city: CAMPBELL
 Mailing state: CA
 Mailing zip: 95008
 Contact person: "RUSSELL, RITA "
 Facility capacity: 56
 Type of clients served: 950
 Facility phone: 5307587251

101 East SRDCCA200718756 Daycare

EDR ID: SRDCCA200718756
 Facility number: 573607655
 Facility name: "LETELIER, EDUARDO "
 Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 810
 Facility status code: 03
 Address: 816 BRADDOCK CT.
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 816 BRADDOCK CT.
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "LETELIER, EDUARDO "
 Licensee type: A
 License effective date: 11203
 License expiration date: Not Reported
 License issue date: 011203
 Program type: "MAXIMUM CAPACITY: 6 CHILDREN WITH NO MORE THAN 3 INFANTS, OR 4 INFANTSONLY, OR CAPACITY 8 CHILDREN WHEN 2 ARE AT LEAST 6 YEARS OF AGE WITH AMAXIMUM OF 2 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED "
 Original app. received date: 010901
 Facility closed date: Not Reported
 Mailing address: 816 BRADDOCK CT.
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "LETELIER, EDURADO "
 Facility capacity: 8
 Type of clients served: 960
 Facility phone: 5307537886

MAP FINDINGS

Map ID Direction Distance Distance (ft.) Elevation	Site	EDR ID Database
Z102 East 1-2 mi 10528 Higher	<p>EDR ID: SRDCCA200753449</p> <p>Facility number: 573606907</p> <p>Facility name: DAVIS PARENT NURSERY SCHOOL</p> <p>Facility eval. code: 0303</p> <p>Facility office number: 03</p> <p>Facility county number: 57</p> <p>Facility type code: 850</p> <p>Facility status code: 03</p> <p>Address: 1441 DANBURY</p> <p>City: DAVIS</p> <p>State: CA</p> <p>Zip: 95616</p> <p>Alt. address: 426 W. 8TH ST.</p> <p>City: DAVIS</p> <p>State: CA</p> <p>Zip: 95616</p> <p>Facility investor: DAVIS PARENT NURSERY SCHOOL ASSOCIATION</p> <p>Licensee type: F</p> <p>License effective date: 11024</p> <p>License expiration date: Not Reported</p> <p>License issue date: 011024</p> <p>Program type: LICENSED TO SERVE CHILDREN FROM AGE 2 YEARS TO ENTRY INTO KINDERGARTENFROM 8:00 TO 4:30.</p> <p>Original app. received date: 010710</p> <p>Facility closed date: Not Reported</p> <p>Mailing address: 426 W. 8TH ST.</p> <p>Mailing city: DAVIS</p> <p>Mailing state: CA</p> <p>Mailing zip: 95616</p> <p>Contact person: "DOUGLAS, KATHERINE "</p> <p>Facility capacity: 30</p> <p>Type of clients served: 950</p> <p>Facility phone: 5307575375</p>	SRDCCA200753449 Daycare
Z103 East 1-2 mi 10528 Higher	<p>EDR ID: SRDCCA200753585</p> <p>Facility number: 573608506</p> <p>Facility name: MARGUERITE MONTGOMERY CHILD DEVELOPMENT CENTER</p> <p>Facility eval. code: 0303</p> <p>Facility office number: 03</p> <p>Facility county number: 57</p> <p>Facility type code: 850</p> <p>Facility status code: 03</p> <p>Address: 1441 DANBURY STREET</p> <p>City: DAVIS</p> <p>State: CA</p> <p>Zip: 95616</p> <p>Alt. address: 851 EAST HAMILTON AVE.</p> <p>City: CAMPBELL</p> <p>State: CA</p> <p>Zip: 95008</p> <p>Facility investor: CHILD DEVELPMENT CENTERS</p> <p>Licensee type: C</p> <p>License effective date: 20830</p>	SRDCCA200753585 Daycare

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

License expiration date: Not Reported
 License issue date: 020830
 Program type: LICENSED TO SERVE CHILDREN FROM AGE 2 YEARS TO ENTRY INTO KINDERGARTEN IN PORTABLE ROOM C-1 FROM 8:30 TO 11:30 A.M. WAIVER ON FILE FOR SHARED USE OF KINDERGARTEN PLAYGROUND.
 Original app. received date: 020821
 Facility closed date: Not Reported
 Mailing address: 851 EAST HAMILTON AVE.
 Mailing city: CAMPBELL
 Mailing state: CA
 Mailing zip: 95008
 Contact person: "WANDERER, MARY"
 Facility capacity: 30
 Type of clients served: 950
 Facility phone: 5302975014

Z104			SRDCCA200745945
East	EDR ID:	SRDCCA200745945	Daycare
1-2 mi	Facility number:	573607080	
10528	Facility name:	MARGUERITE MONTGOMERY CHILD DEVELOPMENT CENTER	
Higher	Facility eval. code:	0303	
	Facility office number:	03	
	Facility county number:	57	
	Facility type code:	840	
	Facility status code:	03	
	Address:	1441 DANBURY ST.	
	City:	DAVIS	
	State:	CA	
	Zip:	95616	
	Alt. address:	"851 E. HAMILTON AVE., STE. 200"	
	City:	CAMPBELL	
	State:	CA	
	Zip:	95008	
	Facility investor:	CHILD DEVELOPMENT CENTERS	
	Licensee type:	C	
	License effective date:	10829	
	License expiration date:	Not Reported	
	License issue date:	010829	
	Program type:	LICENSED TO SERVE CHILDREN ENROLLED IN KINDERGARTEN AND ABOVE IN PORTABLE ROOMS C-1 AND C-2 FROM 7:00-8:30 A.M. AND 11:30 A.M. TO 6:00 P.M. PROGRAM MAY SERVE A MAXIMUM OF 18 CHILDREN EXCLUSIVELY IN PORTABLE CLASS C-2 BETWEEN THE HOURS OF 8:30-11:30 A.M.	
	Original app. received date:	010824	
	Facility closed date:	Not Reported	
	Mailing address:	"851 E. HAMILTON AVE., STE 200 "	
	Mailing city:	CAMPBELL	
	Mailing state:	CA	
	Mailing zip:	95008	
	Contact person:	"WANDERER, MARY"	
	Facility capacity:	70	
	Type of clients served:	950	
	Facility phone:	5305574401	

MAP FINDINGS

Map ID	Direction	Distance	Distance (ft.)	Elevation	Site	EDR ID	Database
Z105	East	1-2 mi	10528	Higher	Ncessch: 061062010456 Schname05: MARGUERITE MONTGOMERY ELEMENTARY Mstreet05: 1441 DANBURY DR. Mcity05: DAVIS Mstate05: CA Mzip05: 95616 Mzip405: Not Reported Member05: 500 Phone05: (530) 759-2100 Locale05: 3 Type05: 1 Level05: 1 Gslo05: KG Gshi05: 06 Edr id: SRPU20071014156	SRPU20071014156	Public Schools
AA106	WNW	2-4 mi	10563	Higher	EDR ID: SRDCCA200715770 Facility number: 573607626 Facility name: "WILLIAMS, GINA" Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 810 Facility status code: 03 Address: 1111 CABOT STREET City: DAVIS State: CA Zip: 95616 Alt. address: 1111 CABOT STREET City: DAVIS State: CA Zip: 95616 Facility investor: "WILLIAMS, GINA" Licensee type: A License effective date: 981124 License expiration date: Not Reported License issue date: 981124 Program type: "MAXIMUM CAPACITY: 6 CHILDREN WITH NO MORE THAN 3 INFANTS, OR 4 INFANTSONLY, OR CAPACITY 8 CHILDREN WHEN 2 ARE AT LEAST 6 YEARS OF AGE WITH AMAXIMUM OF 2 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED." Original app. received date: 980101 Facility closed date: Not Reported Mailing address: 1111 CABOT STREET Mailing city: DAVIS Mailing state: CA Mailing zip: 95616 Contact person: "WILLIAMS, GINA" Facility capacity: 8 Type of clients served: 960 Facility phone: 5307599753	SRDCCA200715770	Daycare

MAP FINDINGS

Map ID Direction Distance Distance (ft.) Elevation	Site	EDR ID Database
107 ENE 2-4 mi 10683 Higher	EDR ID: SRDCCA200715732 Facility number: 573607570 Facility name: "REYNOSO, GUADALUPE" Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 810 Facility status code: 03 Address: 843 MESQUITE DRIVE City: DAVIS State: CA Zip: 95616 Alt. address: 843 MESQUITE DRIVE City: DAVIS State: CA Zip: 95616 Facility investor: "REYNOSO, GUADALUPE" Licensee type: A License effective date: 990601 License expiration date: Not Reported License issue date: 990601 Program type: "MAXIMUM CAPACITY: 6 CHILDREN WITH NO MORE THAN 3 INFANTS, OR 4 INFANTSONLY, OR CAPACITY 8 CHILDREN WHEN 2 ARE AT LEAST 6 YEARS OF AGE WITH AMAXIMUM OF 2 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED." Original app. received date: 990101 Facility closed date: Not Reported Mailing address: 843 MESQUITE DRIVE Mailing city: DAVIS Mailing state: CA Mailing zip: 95616 Contact person: "REYNOSO, GUADALUPE" Facility capacity: 8 Type of clients served: 960 Facility phone: 5307503859	SRDCCA200715732 Daycare
108 WNW 2-4 mi 10869 Higher	EDR ID: SRDCCA200716435 Facility number: 573607421 Facility name: "FARMER, KATIE" Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 810 Facility status code: 03 Address: 2742 SEINE AVE City: DAVIS State: CA Zip: 95616 Alt. address: 2742 SEINE AVE City: DAVIS State: CA Zip: 95616 Facility investor: "FARMER, KATIE"	SRDCCA200716435 Daycare

MAP FINDINGS

Map ID
Direction
Distance
Distance (ft.)
Elevation

Site

EDR ID
Database

Licensee type: A
 License effective date: 951207
 License expiration date: Not Reported
 License issue date: 951207
 Program type: "MAXIMUM CAPACITY: 12 CHILDREN, WITH NO MORE THAN 4 INFANTS,OR
 CAPACITY14 CHILDREN WHEN 2 CHILDREN ARE AT LEAST 6 YEARS OF AGE WITH A
 MAXIMUMOF 3 INFANTS; PROPERTY OWNER/LANDLORD CONSENT REQUIRED.
 "

Original app. received date: 950101
 Facility closed date: Not Reported
 Mailing address: 2742 SEINE AVE
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "FARMER, KATIE" "
 Facility capacity: 14
 Type of clients served: 960
 Facility phone: 5307584488

AA109
 WNW
 2-4 mi
 10942
 Higher

EDR ID: SRDCCA200715864
 Facility number: 573607732
 Facility name: "HILLMAN, ANNE" "
 Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 810
 Facility status code: 03
 Address: 2826 OTTOWA AVE.
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 2826 OTTOWA AVE.
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "HILLMAN, ANNE" "
 Licensee type: A
 License effective date: 20425
 License expiration date: Not Reported
 License issue date: 020425
 Program type: "LIC 9211 ON FILE, INACTIVE LICENSE FROM 2/1/04 TO 8/1/09
 "

Original app. received date: 020115
 Facility closed date: Not Reported
 Mailing address: 2826 OTTOWA AVE.
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "HILLMAN, ANNE" "
 Facility capacity: 8
 Type of clients served: 960
 Facility phone: 5307579276

SRDCCA200715864
 Daycare

MAP FINDINGS

Map ID Direction Distance Distance (ft.) Elevation	Site	EDR ID Database
AB110 NE 2-4 mi 10989 Higher	EDR ID: SRDCCA200748380 Facility number: 570306190 Facility name: MONTESSORI COUNTRY DAY Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 850 Facility status code: 03 Address: 1811 RENOIR City: DAVIS State: CA Zip: 95616 Alt. address: 1811 RENOIR City: DAVIS State: CA Zip: 95616 Facility investor: CAMPUS CHILD CARER INC. Licensee type: A License effective date: 930913 License expiration date: Not Reported License issue date: Not Reported Program type: MAXIMUM CAPACITY: 72 AMBULATORY PRESCHOOL CHILDREN (AGES 2-6 YEARS). Original app. received date: 830809 Facility closed date: Not Reported Mailing address: 2029 FAIRWAY DRIVE Mailing city: DAVIS Mailing state: CA Mailing zip: 95616 Contact person: "ROBERTSON, DEBBIE" Facility capacity: 72 Type of clients served: 950 Facility phone: 5307538373	SRDCCA200748380 Daycare
AB111 NE 2-4 mi 10989 Higher	Pss school id: K9500122 Pss inst: MONTESSORI COUNTRY DAY Lograde: PK Higrade: K Pss address: 1811 RENOIR AVENUE Pss city: DAVIS Pss county no: 113 Pss county fips: 06113 Pss stabb: CA Pss fips: 06 Pss zip5: 95616 Pss phone: 5307538373 Pss sch days: 185 Pss stu day hrs: 6 Pss library: Yes Pss enroll ug: Not Reported Pss enroll pk: 72 Pss enroll k: 20 Pss enroll 1: Not Reported Pss enroll 2: Not Reported	SRPR20051021879 Private Schools

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Pss enroll 3:	Not Reported
Pss enroll 4:	Not Reported
Pss enroll 5:	Not Reported
Pss enroll 6:	Not Reported
Pss enroll 7:	Not Reported
Pss enroll 8:	Not Reported
Pss enroll 9:	Not Reported
Pss enroll 10:	Not Reported
Pss enroll 11:	Not Reported
Pss enroll 12:	Not Reported
Pss enroll t:	92
Pss enroll tk12:	20
Pss race ai:	0
Pss race as:	5
Pss race h:	4
Pss race b:	1
Pss race w:	10
Pss fte teach:	2
Pss locale:	3
Pss coed:	1
Pss type:	2
Pss level:	1
Pss relig:	3
Pss comm type:	2
Pss indian pct:	0
Pss asian pct:	25
Pss hisp pct:	20
Pss black pct:	5
Pss white pct:	50
Pss stdtch rt:	10
Pss orient:	29
Pss county name:	YOLO
Pss assoc 1:	National Association for the Education of Young Children (NAEYC)
Pss assoc 2:	Not Reported
Pss assoc 3:	Not Reported
Pss assoc 4:	Not Reported
Pss assoc 5:	Not Reported
Pss assoc 6:	Not Reported
Pss assoc 7:	Not Reported
Source:	NCESDATA_E72D09B4
Edr id:	SRPR20051021879

112
 NW
 2-4 mi
 11142
 Higher

Hospital type:	01
Num of times COO:	00
Owner date:	Not Reported
City:	DAVIS
Has plan of corr:	Not Reported
Compliance status:	Not Reported
SSA county code:	670
Cross ref number:	Not Reported
FMS survey date:	Not Reported
Current survey date:	Not Reported
Medicare/Medicaid:	Not Reported
Facility name:	WOODLAND HEALTHCARE-DAVIS MEDICAL GRP

SRHO20070153517
 AHA Hospitals

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Intermediary/Carrier: Not Reported
 Medicaid number: Not Reported
 Participation date: 20010326
 Prior COO date: Not Reported
 Prior carrier: Not Reported
 Provider ID: 05D0984604
 Record Status: A
 Region code: 09
 Is Partial Record: Y
 state abbrev: CA
 ssa state: 05
 state region cd: M2
 street address: 2330 WEST COVELL
 Phone num: 5306623961
 Termination reason: 00
 Term Date: 20070325
 Purpose of action: Not Reported
 Provider control: 01
 Zip: 95616
 Fips state: 06
 Fips cnty: 113
 SSA MSA: 499
 SSA MSA size code: B
 Date accredited: Not Reported
 Accred expire date: Not Reported
 Accred Org: Not Reported
 Num beds: 0000
 Num cert beds: 0000
 Source: US_HOSPITAL_POSCLIA
 Edr id: SRHO20070153517

113
 North
 2-4 mi
 11142
 Higher

EDR ID: SRDCCA200723029
 Facility number: 573609500
 Facility name: "CHAVEZ, IRMA"
 Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 810
 Facility status code: 03
 Address: 330 NORTE AVENUE
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 330 NORTE AVENUE
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "CHAVEZ, IRMA"
 Licensee type: A
 License effective date: 31007
 License expiration date: Not Reported
 License issue date: 031007
 Program type: "MAXIMUM CAPACITY: 6 CHILDREN WITH NO MORE THAN 3 INFANTS, OR 4
 INFANTSONLY. PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED. OFFLIMITS:
 GARAGE, LAUNDRY ROOM, MASTER BATHROOM, SISTER'S BEDROOMS.
 "

SRDCCA200723029
 Daycare

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Original app. received date: 030820
 Facility closed date: Not Reported
 Mailing address: 330 NORTE AVENUE
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "CHAVEZ, IRMA "
 Facility capacity: 6
 Type of clients served: 960
 Facility phone: 5307580266

AC114
 ENE EDR ID: SRDCCA200751511 SRDCCA200751511
 2-4 mi Facility number: 573603061 Daycare
 11273 Facility name: MONTESSORI COUNTRY DAY II
 Lower Facility eval. code: 0303

Facility office number: 03
 Facility county number: 57
 Facility type code: 850
 Facility status code: 03
 Address: 2802 SPAFFORD
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 2802 SPAFFORD
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: CAMPUS CHILD CARE INC.
 Licensee type: A
 License effective date: 971014
 License expiration date: Not Reported
 License issue date: 971014
 Program type: TOTAL CAPACITY 82 WHICH INCLUDES THE TODDLER OPTION PROGRAM FOR 12.
 Original app. received date: 970602
 Facility closed date: Not Reported
 Mailing address: 2802 SPAFFORD
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "HANNAGAN, LORI "
 Facility capacity: 82
 Type of clients served: 950
 Facility phone: 5307535225

AC115
 ENE EDR ID: SRDCCA200741887 SRDCCA200741887
 2-4 mi Facility number: 573603060 Daycare
 11273 Facility name: MONTESSORI COUNTRY DAY II
 Lower Facility eval. code: 0303

Facility office number: 03
 Facility county number: 57
 Facility type code: 830
 Facility status code: 03

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Address: 2802 SPAFFORD
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 2802 SPAFFORD
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: CAMPUS CHILD CARE INC.
 Licensee type: A
 License effective date: 971016
 License expiration date: Not Reported
 License issue date: 971016
 Program type: 8 INFANTS. COMBINATION CENTER. TOTAL CAPACITY WITH PRESCHOOL AND TODDLER OPTION 90.
 Original app. received date: 970602
 Facility closed date: Not Reported
 Mailing address: 400 RUSSELL PARK
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: LORI HANNAGAN
 Facility capacity: 8
 Type of clients served: 955
 Facility phone: 5307535225

Z116			SRDCCA200734554
East	EDR ID:	SRDCCA200734554	Daycare
2-4 mi	Facility number:	573613068	
11309	Facility name:	"BARAJAS, MARIA	"
Higher	Facility eval. code:	0303	
	Facility office number:	03	
	Facility county number:	57	
	Facility type code:	810	
	Facility status code:	03	
	Address:	3133 NANTUCKET	
	City:	DAVIS	
	State:	CA	
	Zip:	95616	
	Alt. address:	3133 NANTUCKET	
	City:	DAVIS	
	State:	CA	
	Zip:	95616	
	Facility investor:	"BARAJAS, MARIA	"
	Licensee type:	A	
	License effective date:	60720	
	License expiration date:	Not Reported	
	License issue date:	060720	
	Program type:	"MAX. CAP: 6 - NO MORE THAN 3 INFANTS OR 4 INFANTS ONLY. CAP 8 - NO MORE THAN 2 INFANTS, 1 CHILD IN KINDERGARTEN OR ELEMENTARY SCHOOL AND 1 CHILD AT LEAST AGE 6. STANDAR, OFF-LIMIT AREAS INCLUDE BACK YARD, ""GARAGE, AND SECOND STORY. "	
	Original app. received date:	060519	
	Facility closed date:	Not Reported	

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Mailing address: 3133 NANTUCKET
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "BARAJAS, MARIA"
 Facility capacity: 8
 Type of clients served: 960
 Facility phone: 5307539434

117 NE EDR ID: SRDCCA200716230 SRDCCA200716230
 2-4 mi Facility number: 573607488 Daycare
 11469 Facility name: "KUSS, LESLIE"

Higher Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 810
 Facility status code: 03
 Address: 2422 BATES DRIVE
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 2422 BATES DRIVE
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "KUSS, LESLIE & CAMERON"
 Licensee type: A
 License effective date: 940702
 License expiration date: Not Reported
 License issue date: 940702
 Program type: "MAXIMUM CAPACITY: 12 CHILDREN, WITH NO MORE THAN 4 INFANTS, OR
 CAPACITY 14 CHILDREN WHEN 2 CHILDREN ARE AT LEAST 6 YEARS OF AGE WITH A
 MAXIMUM OF 3 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED"
 Original app. received date: 940701
 Facility closed date: Not Reported
 Mailing address: 2422 BATES DRIVE
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "KUSS, LESLIE"
 Facility capacity: 14
 Type of clients served: 960
 Facility phone: 5307585438

118 NE EDR ID: SRDCCA200700046 SRDCCA200700046
 2-4 mi Facility number: 570304441 Daycare
 11526 Facility name: PROGRESS RANCH - THE GROVE

Higher Facility eval. code: 0705
 Facility office number: 23
 Facility county number: 57
 Facility type code: 730
 Facility status code: 03

MAP FINDINGS

Map ID
Direction
Distance
Distance (ft.)
Elevation

Site

EDR ID
Database

Address: 2725 LOYOLA DRIVE
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: P.O. BOX 1287
 City: DAVIS
 State: CA
 Zip: 95617
 Facility investor: "PROGRESS RANCH, INC. "
 Licensee type: C
 License effective date: 930426
 License expiration date: Not Reported
 License issue date: Not Reported
 Program type: "AMBULATORY CHILDREN, AGES 5 THRU 17 YEARS."
 "

Original app. received date: 810330
 Facility closed date: Not Reported
 Mailing address: P.O.BOX 1287
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95617
 Contact person: "KUSAMA, RUSSELL K. "
 Facility capacity: 6
 Type of clients served: 950
 Facility phone: 5307532566

119
 NNW
 2-4 mi
 11803
 Higher

Pss school id: A9100759
 Pss inst: DAVIS WALDORF SCHOOL
 Lograde: K
 Higrade: 7
 Pss address: 3100 SYCAMORE LANE
 Pss city: DAVIS
 Pss county no: 113
 Pss county fips: 06113
 Pss stabb: CA
 Pss fips: 06
 Pss zip5: 95616
 Pss phone: 5307531651
 Pss sch days: 166
 Pss stu day hrs: 6.75
 Pss library: No
 Pss enroll ug: Not Reported
 Pss enroll pk: Not Reported
 Pss enroll k: 24
 Pss enroll 1: 11
 Pss enroll 2: 12
 Pss enroll 3: 14
 Pss enroll 4: 14
 Pss enroll 5: 6
 Pss enroll 6: 6
 Pss enroll 7: 17
 Pss enroll 8: Not Reported
 Pss enroll 9: Not Reported
 Pss enroll 10: Not Reported

SRPR20051023860
 Private Schools

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Pss enroll 11: Not Reported
 Pss enroll 12: Not Reported
 Pss enroll t: 104
 Pss enroll tk12: 104
 Pss race ai: 0
 Pss race as: 2
 Pss race h: 4
 Pss race b: 3
 Pss race w: 95
 Pss fte teach: 10.3
 Pss locale: 3
 Pss coed: 1
 Pss type: 6
 Pss level: 1
 Pss relig: 3
 Pss comm type: 2
 Pss indian pct: 0
 Pss asian pct: 1.92
 Pss hisp pct: 3.85
 Pss black pct: 2.88
 Pss white pct: 91.35
 Pss stdtch rt: 10.1
 Pss orient: 29
 Pss county name: YOLO
 Pss assoc 1: Association of Waldorf Schools of North America (AWSNA)
 Pss assoc 2: Not Reported
 Pss assoc 3: Not Reported
 Pss assoc 4: Not Reported
 Pss assoc 5: Not Reported
 Pss assoc 6: Not Reported
 Pss assoc 7: Not Reported
 Source: NCESDATA_E72D09B4
 Edr id: SRPR20051023860

120
 NW
 2-4 mi
 11952
 Higher

Pss school id: A9705085
 Pss inst: TENDER LEARNING CARE
 Lograde: PK
 Higrade: K
 Pss address: 1818 LAKE BLVD
 Pss city: DAVIS
 Pss county no: 113
 Pss county fips: 06113
 Pss stabb: CA
 Pss fips: 06
 Pss zip5: 95616
 Pss phone: 5307565351
 Pss sch days: 200
 Pss stu day hrs: 4
 Pss library: Yes
 Pss enroll ug: Not Reported
 Pss enroll pk: 64
 Pss enroll k: 10
 Pss enroll 1: Not Reported
 Pss enroll 2: Not Reported

SRPR20051027805
 Private Schools

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Pss enroll 3:	Not Reported
Pss enroll 4:	Not Reported
Pss enroll 5:	Not Reported
Pss enroll 6:	Not Reported
Pss enroll 7:	Not Reported
Pss enroll 8:	Not Reported
Pss enroll 9:	Not Reported
Pss enroll 10:	Not Reported
Pss enroll 11:	Not Reported
Pss enroll 12:	Not Reported
Pss enroll t:	74
Pss enroll tk12:	10
Pss race ai:	0
Pss race as:	1
Pss race h:	0
Pss race b:	1
Pss race w:	8
Pss fte teach:	1
Pss locale:	3
Pss coed:	1
Pss type:	7
Pss level:	1
Pss relig:	3
Pss comm type:	2
Pss indian pct:	0
Pss asian pct:	10
Pss hisp pct:	0
Pss black pct:	10
Pss white pct:	80
Pss stdtch rt:	10
Pss orient:	29
Pss county name:	YOLO
Pss assoc 1:	No Membership Association
Pss assoc 2:	Not Reported
Pss assoc 3:	Not Reported
Pss assoc 4:	Not Reported
Pss assoc 5:	Not Reported
Pss assoc 6:	Not Reported
Pss assoc 7:	Not Reported
Source:	NCESDATA_E72D09B4
Edr id:	SRPR20051027805

AD121
 East
 2-4 mi
 12001
 Lower

EDR ID: SRDCCA200727847
 Facility number: 573610708
 Facility name: "ALEMI, NAJ"
 Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 810
 Facility status code: 03
 Address: 3333 LAGUNA AVE
 City: DAVIS
 State: CA
 Zip: 95616

SRDCCA200727847
 Daycare

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Alt. address: 3333 LAGUNA AVE
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "ALEMI, NAJ"
 Licensee type: A
 License effective date: 50214
 License expiration date: Not Reported
 License issue date: 050214
 Program type: "MAX. CAP: 6 - NO MORE THAN 3 INFANTS OR 4 INFANTS ONLY.
 CAP 8 - NO MORE THAN 2 INFANTS, 1 CHILD IN KINDERGARTEN OR ELEMENTARY
 SCHOOL AND 1 CHILD AT LEAST AGE 6. OFFLIMITS: GARAGE & MASTER
 "BEDROOM.
 Original app. received date: 050113
 Facility closed date: Not Reported
 Mailing address: 3333 LAGUNA AVE
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "ALEMI, NAJ"
 Facility capacity: 8
 Type of clients served: 960
 Facility phone: 5307584462

122 WNW EDR ID: SRDCCA200715739 SRDCCA200715739
 2-4 mi Facility number: 573607582 Daycare
 12079 Facility name: "ROMERO, LUCY"
 Higher Facility eval. code: 0303

Facility office number: 03
 Facility county number: 57
 Facility type code: 810
 Facility status code: 03
 Address: 3207 CUTTER PLACE
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 3207 CUTTER PLACE
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "ROMERO, LUCY"
 Licensee type: C
 License effective date: 931101
 License expiration date: Not Reported
 License issue date: 931101
 Program type: "MAXIMUM CAPACITY: 6 CHILDREN WITH NO MORE THAN 3 INFANTS, OR 4
 INFANTSONLY, OR CAPACITY 8 CHILDREN WHEN 2 ARE AT LEAST 6 YEARS OF AGE
 WITH AMAXIMUM OF 2 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED
 "
 Original app. received date: 930101
 Facility closed date: Not Reported
 Mailing address: 3207 CUTTER PLACE
 Mailing city: DAVIS
 Mailing state: CA

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Mailing zip: 95616
 Contact person: "ROMERO, LUCY"
 Facility capacity: 8
 Type of clients served: 960
 Facility phone: 5307562751

123			SRDCCA200735141
NE	EDR ID:	SRDCCA200735141	Daycare
2-4 mi	Facility number:	573613003	
12337	Facility name:	"SALAMATI, ROSHAN"	
Lower	Facility eval. code:	0303	
	Facility office number:	03	
	Facility county number:	57	
	Facility type code:	810	
	Facility status code:	03	
	Address:	2444 MOORE BLVD. #152	
	City:	DAVIS	
	State:	CA	
	Zip:	95616	
	Alt. address:	PO BOX 1435	
	City:	DAVIS	
	State:	CA	
	Zip:	95617	
	Facility investor:	"SALAMATI, ROSHAN"	
	Licensee type:	A	
	License effective date:	60314	
	License expiration date:	Not Reported	
	License issue date:	060314	
	Program type:	"MAX.CAP:6-NO MORE THAN 3 INFANTS OR 4 INFANTS ONLY.CAP 8-NO MORE THAN 2 INFANTS,1 CHILD IN KINDER OR ELEM. SCHOOL & 1 CHILD AT LEAST 6. PROPERTY OWNER/LANDLORD CONSENT REQUIRED.OFF LIMIT AREAS: 2ND STORY"	
	Original app. received date:	060307	
	Facility closed date:	Not Reported	
	Mailing address:	PO BOX 1435	
	Mailing city:	DAVIS	
	Mailing state:	CA	
	Mailing zip:	95617	
	Contact person:	"SALAMATI, ROSHAN"	
	Facility capacity:	8	
	Type of clients served:	960	
	Facility phone:	5307576730	

AD124			SRDCCA200722127
ENE	EDR ID:	SRDCCA200722127	Daycare
2-4 mi	Facility number:	573609003	
12745	Facility name:	"SAH, RANJANA"	
Lower	Facility eval. code:	0303	
	Facility office number:	03	
	Facility county number:	57	
	Facility type code:	810	
	Facility status code:	03	
	Address:	3605 KOSO STREET	
	City:	DAVIS	

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

State: CA
 Zip: 95616
 Alt. address: 3605 KOSO STREET
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "SAH, RANJANA"
 Licensee type: A
 License effective date: 30312
 License expiration date: Not Reported
 License issue date: 030312
 Program type: MAX. CAP (WHEN THERE IS AN ASSISTANT PRESENT): 12 - NO MORE THAN 4 INFANTS. CAP 14 - NO MORE THAN 3 INFANTS. 1 CHILD IN KINDERGARTEN OR ELEMENTARY SCHOOL AND 1 CHILD AT LEAST AGE 6."OFFLIMITS: ALL OF UPSTAIRS, AND GARAGE."
 Original app. received date: 030218
 Facility closed date: Not Reported
 Mailing address: 3605 KOSO STREET
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "SAH, RANJANA"
 Facility capacity: 14
 Type of clients served: 960
 Facility phone: 5307534770

AE125
 NE
 2-4 mi
 12839
 Higher

EDR ID: SRDCCA200753311
 Facility number: 573609766
 Facility name: YOLO CRISIS NURSERY-FAMILIES FIRST INC.
 Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 850
 Facility status code: 03
 Address: 1701 BALSAM PLACE
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 2100 FIFTH STREET
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: FAMILIES FIRST INC.
 Licensee type: C
 License effective date: 40729
 License expiration date: Not Reported
 License issue date: 040729
 Program type: DUALY LICENSED AS A GROUP HOME AND CHILD CARE CENTER TO SERVE CHILDREN AGES 2 TO 5 YEARS. SEE LETTER FOR SPECIAL CONDITIONS.
 Original app. received date: 040102
 Facility closed date: Not Reported
 Mailing address: 2100 FIFTH STREET
 Mailing city: DAVIS

SRDCCA200753311
 Daycare

MAP FINDINGS

Map ID Direction Distance Distance (ft.) Elevation	Site	EDR ID Database
	Mailing state: CA Mailing zip: 95616 Contact person: "HEINTZ, LAURA" Facility capacity: 6 Type of clients served: 950 Facility phone: 5307586680	
AE126 NE 2-4 mi 12839 Higher	EDR ID: SRDCCA200744433 Facility number: 573609767 Facility name: YOLO CRISIS NURSERY-FAMILIES FIRST INC. Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 830 Facility status code: 03 Address: 1701 BALSAM PLACE City: DAVIS State: CA Zip: 95616 Alt. address: 2100 FIFTH STREET City: DAVIS State: CA Zip: 95616 Facility investor: FAMILIES FIRST INC. Licensee type: C License effective date: 40729 License expiration date: Not Reported License issue date: 040729 Program type: DUALY LICENSED AS A GROUP HOME AND CHILD CARE CENTER TO SERVE CHILDREN AGES 0 TO 24 MONTHS. SEE LETTER FOR SPECIAL CONDITIONS. Original app. received date: 040102 Facility closed date: Not Reported Mailing address: 2100 FIFTH STREET Mailing city: DAVIS Mailing state: CA Mailing zip: 95616 Contact person: "HEINTZ, LAURA" Facility capacity: 6 Type of clients served: 955 Facility phone: 5307586680	SRDCCA200744433 Daycare
AE127 NE 2-4 mi 12839 Higher	EDR ID: SRDCCA200700838 Facility number: 577001738 Facility name: "FAMILIES FIRST, INC." Facility eval. code: 0807 Facility office number: 23 Facility county number: 57 Facility type code: 730 Facility status code: 03 Address: 1701 BALSAM PLACE City: DAVIS State: CA	SRDCCA200700838 Daycare

MAP FINDINGS

Map ID
Direction
Distance
Distance (ft.)
Elevation

Site

EDR ID
Database

Zip: 95616
 Alt. address: 2100 FIFTH STREET
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "FAMILIES FIRST, INC. "
 Licensee type: C
 License effective date: 10328
 License expiration date: Not Reported
 License issue date: 010328
 Program type: "FACILITY IS A CRISIS NURSERY SERVING CHILDREN AGES 0-5, AMBULATORY OR NON-AMBULATORY. CAPACITY REDUCED TO 4 EFFECTIVE 7/28/04"
 "

Original app. received date: 010123
 Facility closed date: Not Reported
 Mailing address: 2100 FIFTH STREET
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "HEINTZ, LAURA "
 Facility capacity: 4
 Type of clients served: 950
 Facility phone: 5307586680

AF128
 NE
 2-4 mi
 13062
 Higher

EDR ID: SRDCCA200716389
 Facility number: 573607509
 Facility name: "MAROTTO, JOANNE "
 Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 810
 Facility status code: 03
 Address: 2964 LAYTON DRIVE
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 2964 LAYTON DRIVE
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "MAROTTO, JOANNE & SAMUEL "
 Licensee type: A
 License effective date: 941001
 License expiration date: Not Reported
 License issue date: 941001
 Program type: "MAXIMUM CAPACITY: 6 CHILDREN WITH NO MORE THAN 3 INFANTS, OR 4 INFANTSONLY, OR CAPACITY 8 CHILDREN WHEN 2 ARE AT LEAST 6 YEARS OF AGE WITH AMAXIMUM OF 2 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED"
 "

Original app. received date: 940101
 Facility closed date: Not Reported
 Mailing address: 2964 LAYTON DRIVE
 Mailing city: DAVIS

SRDCCA200716389
 Daycare

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Mailing state: CA
 Mailing zip: 95616
 Contact person: "MAROTTO, JOANNE"
 Facility capacity: 8
 Type of clients served: 960
 Facility phone: 5307584419

129 West EDR ID: SRDCCA200748386 SRDCCA200748386
 2-4 mi Facility number: 570307832 Daycare
 13134 Facility name: REDBUD MONTESSORI
 Higher Facility eval. code: 0303

Facility office number: 03
 Facility county number: 57
 Facility type code: 850
 Facility status code: 03
 Address: 27082 PATWIN ROAD
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: P.O. BOX 1562
 City: DAVIS
 State: CA
 Zip: 95617
 Facility investor: REDBUD MONTESSORI INC.
 Licensee type: A
 License effective date: 931016
 License expiration date: Not Reported
 License issue date: Not Reported
 Program type: "36 AMBULATORY CHILDREN ONLY, AGES 2-6 YEARS. STAFF-CHILD RATIO
 MUST BE MAINTAINED WHEN STAFF IS OFF PREMISES."
 "

Original app. received date: 840705
 Facility closed date: Not Reported
 Mailing address: P.O. BOX 1562
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95617
 Contact person: "GILL, KAREN"
 Facility capacity: 36
 Type of clients served: 950
 Facility phone: 5307532623

AF130 ENE EDR ID: SRDCCA200715694 SRDCCA200715694
 2-4 mi Facility number: 573607469 Daycare
 13361 Facility name: "HOLM, CHRISTINA"
 Lower Facility eval. code: 0303

Facility office number: 03
 Facility county number: 57
 Facility type code: 810
 Facility status code: 03
 Address: 1440 MONARCH LANE
 City: DAVIS

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

State: CA
 Zip: 95616
 Alt. address: 1440 MONARCH LANE
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "HOLM, CHRISTINA"
 Licensee type: A
 License effective date: 991105
 License expiration date: Not Reported
 License issue date: 991105
 Program type: "MAXIMUM CAPACITY: 12 CHILDREN WITH NO MORE THAN 4 INFANTS, OR CAPACITY 14 CHILDREN WHEN 2 CHILDREN ARE AT LEAST 6 YEARS OF AGE WITH A MAXIMUM OF 3 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED."
 Original app. received date: 990101
 Facility closed date: Not Reported
 Mailing address: 1440 MONARCH LANE
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "HOLM, CHRISTINA"
 Facility capacity: 14
 Type of clients served: 960
 Facility phone: 5307583746

AG131		SRDCCA200719291
NE	EDR ID: SRDCCA200719291	Daycare
2-4 mi	Facility number: 573608950	
13493	Facility name: "POUDYAL, SHANTI"	
Lower	Facility eval. code: 0303	
	Facility office number: 03	
	Facility county number: 57	
	Facility type code: 810	
	Facility status code: 03	
	Address: 2210 BEARDEN STREET	
	City: DAVIS	
	State: CA	
	Zip: 95616	
	Alt. address: 2210 BEARDEN STREET	
	City: DAVIS	
	State: CA	
	Zip: 95616	
	Facility investor: "POUDYAL, SHANTI"	
	Licensee type: A	
	License effective date: 30213	
	License expiration date: Not Reported	
	License issue date: 030213	
	Program type: "MAXIMUM CAPACITY: 6 CHILDREN WITH NO MORE THAN 3 INFANTS, OR 4 INFANTS ONLY, OR CAPACITY 8 CHILDREN WHEN 2 ARE AT LEAST 6 YEARS OF AGE WITH A MAXIMUM OF 2 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED. "OFF LIMITS: ALL OF UPSTAIRS."	
	Original app. received date: 030122	
	Facility closed date: Not Reported	
	Mailing address: 2210 BEARDEN STREET	

MAP FINDINGS

Map ID Direction Distance Distance (ft.) Elevation	Site	EDR ID Database
	Mailing city: DAVIS Mailing state: CA Mailing zip: 95616 Contact person: "POUDYAL, SHANTI" Facility capacity: 8 Type of clients served: 960 Facility phone: 5302975803	
AE132 NE 2-4 mi 13582 Higher	EDR ID: SRDCCA200715746 Facility number: 573607603 Facility name: "THORESON, NITA" Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 810 Facility status code: 03 Address: 2043 BEARDEN STREET City: DAVIS State: CA Zip: 95616 Alt. address: 2043 BEARDEN STREET City: DAVIS State: CA Zip: 95616 Facility investor: "THORESON, NITA" Licensee type: A License effective date: 1220 License expiration date: Not Reported License issue date: 001220 Program type: "MAXIMUM CAPACITY: 12 CHILDREN, WITH NO MORE THAN 4 INFANTS, OR CAPACITY 14 CHILDREN WHEN 2 CHILDREN ARE AT LEAST 6 YEARS OF AGE WITH A MAXIMUM OF 3 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED "REQUIRED Original app. received date: 990101 Facility closed date: Not Reported Mailing address: 2043 BEARDEN STREET Mailing city: DAVIS Mailing state: CA Mailing zip: 95616 Contact person: "THORESON, NITA" Facility capacity: 14 Type of clients served: 960 Facility phone: 5307921336	SRDCCA200715746 Daycare
133 WNW 2-4 mi 13584 Higher	EDR ID: SRDCCA200730984 Facility number: 573611199 Facility name: "STEPHENS, MARGARET" Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 810 Facility status code: 03	SRDCCA200730984 Daycare

MAP FINDINGS

Map ID	Direction	Distance	Distance (ft.)	Elevation	Site	EDR ID	Database
					Address: 3408 SEABRIGHT City: DAVIS State: CA Zip: 95616 Alt. address: 3408 SEABRIGHT City: DAVIS State: CA Zip: 95616 Facility investor: "STEPHENS, MARGARET" Licensee type: A License effective date: 51220 License expiration date: Not Reported License issue date: 051220 Program type: "MAX. CAP: 6 - NO MORE THAN 3 INFANTS OR 4 INFANTS ONLY. CAP 8 - NO MORE THAN 2 INFANTS, 1 CHILD IN KINDERGARTEN OR ELEMENTARY SCHOOL AND 1 CHILD AT LEAST AGE 6. OFF LIMITS: MASTER BEDROOM & "KITCHEN. Original app. received date: 050902 Facility closed date: Not Reported Mailing address: 3408 SEABRIGHT Mailing city: DAVIS Mailing state: CA Mailing zip: 95616 Contact person: "STEPHENS, MARGARET" Facility capacity: 8 Type of clients served: 960 Facility phone: 5307536351		
AE134	NE	2-4 mi	13583	Higher	EDR ID: SRDCCA200731664 Facility number: 573611389 Facility name: "HONEYCUTT, BEOLA" Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 810 Facility status code: 03 Address: 1719 MONARCH LANE City: DAVIS State: CA Zip: 95616 Alt. address: 1719 MONARCH LANE City: DAVIS State: CA Zip: 95616 Facility investor: "HONEYCUTT, BEOLA" Licensee type: A License effective date: 50913 License expiration date: Not Reported License issue date: 050913 Program type: MAX. CAP(WHEN THERE IS AN ASSISTANT PRESENT): 12 - NO MORE THAN 4 INFANTS. CAP 14 - NO MORE THAN 3 INFANTS. 1 CHILD IN KINDERGARTEN OR ELEMENTARY SCHOOL AND 1 CHILD AT LEAST AGE 6.OFF LIMITS: MASTER BEDROOM. Original app. received date: 050815	SRDCCA200731664	Daycare

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Facility closed date: Not Reported
 Mailing address: 1719 MONARCH LANE
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "HONEYCUTT, BEOLA"
 Facility capacity: 14
 Type of clients served: 960
 Facility phone: 5307502333

AH135
 ENE
 2-4 mi
 13724
 Higher

EDR ID: SRDCCA200755031
 Facility number: 573614065
 Facility name: DAVIS CHILDREN'S CENTER
 Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 850
 Facility status code: 03
 Address: 3100 LOYOLA DRIVE
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 526 B STREET
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: DAVIS JOINT UNIFIED SCHOOL DISTRICT
 Licensee type: F
 License effective date: 60724
 License expiration date: Not Reported
 License issue date: 060724
 Program type: "LICENSED TO SERVE CHILDREN FROM AGE 2 YEARS THROUGH KINDERGARTEN IN PORTABLE ROOMS C-14, C-16, C-18 AND C-19. WAIVERS ON FILE."
 "

SRDCCA200755031
 Daycare

AH136
 ENE
 2-4 mi
 13724
 Higher

Ncessch: 061062011834
 Scname05: FRED T. KOREMATSU ELEMENTARY SCHOOL AT MACE RANCH
 Mstreet05: 3100 LOYOLA DR.
 Mcity05: DAVIS
 Mstate05: CA
 Mzip05: 95616
 Mzip405: Not Reported

SRPU20071014159
 Public Schools

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Member05: -2
 Phone05: M
 Locale05: 3
 Type05: 1
 Level05: 4
 Gslo05: N
 Gshi05: N
 Edr id: SRPU20071014159

AG137		SRDCCA200721724
NE	EDR ID: SRDCCA200721724	Daycare
2-4 mi	Facility number: 573609090	
13865	Facility name: "DAHAL, SHAKUNTALA"	
Lower	Facility eval. code: 0303	
	Facility office number: 03	
	Facility county number: 57	
	Facility type code: 810	
	Facility status code: 03	
	Address: 2317 ROUALT ST.	
	City: DAVIS	
	State: CA	
	Zip: 95616	
	Alt. address: 2317 ROUALT ST.	
	City: DAVIS	
	State: CA	
	Zip: 95616	
	Facility investor: "DAHAL, SHAKUNTALA"	
	Licensee type: A	
	License effective date: 30514	
	License expiration date: Not Reported	
	License issue date: 030514	
	Program type: "MAXIMUM CAPACITY: 6 CHILDREN WITH NO MORE THAN 3 INFANTS, OR 4 INFANTSONLY, OR CAPACITY 8 CHILDREN WHEN 2 ARE AT LEAST 6 YEARS OF AGE WITH AMAXIMUM OF 2 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED. "OFF LIMITS: SON AND DAUGHTERS BEDROOMS.	
	Original app. received date: 030319	
	Facility closed date: Not Reported	
	Mailing address: 2317 ROUALT ST.	
	Mailing city: DAVIS	
	Mailing state: CA	
	Mailing zip: 95616	
	Contact person: "DAHAL, SHAKUNTALA"	
	Facility capacity: 8	
	Type of clients served: 960	
	Facility phone: 5307582595	

A1138		SRDCCA200747044
ENE	EDR ID: SRDCCA200747044	Daycare
2-4 mi	Facility number: 570311280	
13998	Facility name: MERRYHILL COUNTRY SCHOOL	
Lower	Facility eval. code: 0303	
	Facility office number: 03	
	Facility county number: 57	
	Facility type code: 850	

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Facility status code: 03
 Address: 222 LA VIDA WAY
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: "1451 RIVER PARK DR., STE. 141 "
 City: SACRAMENTO
 State: CA
 Zip: 95815
 Facility investor: "NOBEL LEARNING COMMUNITIES, INC. "
 Licensee type: D
 License effective date: 940604
 License expiration date: Not Reported
 License issue date: 880604
 Program type: EFFECTIVE 2/10/99 MAXIMUM CAPACITY: 88 PRESCHOOL CHILDREN WHICH INCLUDES NO MORE THAN 12 CHILDREN ENROLLED IN THEIR TODDLER-OPTION PROGRAM. COMBINATION CENTER. TOTAL CAPACITY NOT TO EXCEED 104CHILDREN AT ANY TIME. WAIVER FOR OUTDOOR PLAY SPACE ON FILE.
 Original app. received date: 880601
 Facility closed date: Not Reported
 Mailing address: "1451 RIVER PARK DR., STE. 141 "
 Mailing city: SACRAMENTO
 Mailing state: CA
 Mailing zip: 95815
 Contact person: "VALENZUELA, YVONNE "
 Facility capacity: 88
 Type of clients served: 950
 Facility phone: 5307539210

A1139
 ENE
 2-4 mi
 13998
 Lower

EDR ID: SRDCCA200742367
 Facility number: 570311281
 Facility name: MERRYHILL COUNTRY SCHOOL - LAVIDA
 Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 830
 Facility status code: 03
 Address: 222 LA VIDA WAY
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: "1451 RIVER PARK DR., STE. 141 "
 City: SACRAMENTO
 State: CA
 Zip: 95815
 Facility investor: "NOBEL LEARNING COMMUNITIES, INC. "
 Licensee type: D
 License effective date: 940604
 License expiration date: Not Reported
 License issue date: 880604
 Program type: "CAPACITY 16, AGES BIRTH - 2 YEARS. COMBINATION CENTER - TOTAL CAPACITY WITH DAY CARE PROGRAM NOT TO EXCEED 120 AT ANY TIME."
 Original app. received date: 880601

SRDCCA200742367
 Daycare

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Facility closed date: Not Reported
 Mailing address: "1451 RIVER PARK DR., STE. 141 "
 Mailing city: SACRAMENTO
 Mailing state: CA
 Mailing zip: 95815
 Contact person: "VALENZUELA, YVONNE "
 Facility capacity: 16
 Type of clients served: 955
 Facility phone: 5307539210

140 ENE SRDCCA200715731
 2-4 mi Facility number: 573607566 Daycare
 14092 Facility name: "RAMOS, OLGA "
 Higher Facility eval. code: 0303

Facility office number: 03
 Facility county number: 57
 Facility type code: 810
 Facility status code: 03
 Address: 1002 SAN GALLO TERRACE
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 1002 SAN GALLO TERRACE
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "RAMOS, OLGA "
 Licensee type: A
 License effective date: 607
 License expiration date: Not Reported
 License issue date: 000607
 Program type: "ON INACTIVE STATUS FROM APRIL 25, 2006 TO YDECEMBER 31, 2006. TB.
 "

Original app. received date: 000101
 Facility closed date: Not Reported
 Mailing address: 1002 SAN GALLO TERRACE
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "RAMOS, OLGA "
 Facility capacity: 8
 Type of clients served: 960
 Facility phone: 5307564370

141 ENE SRDCCA200726312
 2-4 mi Facility number: 573610334 Daycare
 15471 Facility name: "MOHAMED, SAYDA "
 Lower Facility eval. code: 0303

Facility office number: 03
 Facility county number: 57
 Facility type code: 810
 Facility status code: 03

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Address: 4019 VISTOSA ST.
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 4019 VISTOSA ST.
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "MOHAMED, SAYDA"
 Licensee type: A
 License effective date: 40916
 License expiration date: Not Reported
 License issue date: 040916
 Program type: "MAXIMUM CAPACITY: 6 CHILDREN WITH NO MORE THAN 3 INFANTS, OR 4
 INFANTSONLY, OR CAPACITY 8 CHILDREN WHEN 2 ARE AT LEAST 6 YEARS OF AGE
 WITH AMAXIMUM OF 2 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS
 REQUIRED. ""OFF LIMITS: UPSTAIRS, GARAGE, LAUNDRY ROOM."
 "

Original app. received date: 040805
 Facility closed date: Not Reported
 Mailing address: 4019 VISTOSA ST.
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "MOHAMED, SAYDA"
 Facility capacity: 8
 Type of clients served: 960
 Facility phone: 5307530589

142
 ENE
 2-4 mi
 16997
 Lower

EDR ID: SRDCCA200753171
 Facility number: 573610075
 Facility name: UNIVERSITY COVENANT NURSERY SCHOOL
 Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 850
 Facility status code: 03
 Address: 315 MACE BLVD.
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 315 MACE BLVD.
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: UNIVERSITY COVENANT CHURCH
 Licensee type: C
 License effective date: 40820
 License expiration date: Not Reported
 License issue date: 040820
 Program type: LICENSED TO SERVE CHILDREN FROM AGE 2 YEARS UNTIL ENTRY INTO FIRST
 GRADE.
 Original app. received date: 040624
 Facility closed date: Not Reported

SRDCCA200753171
 Daycare

MAP FINDINGS

Map ID	Direction	Distance	Distance (ft.)	Elevation	Site	EDR ID	Database
					Mailing address: 315 MACE BLVD. Mailing city: DAVIS Mailing state: CA Mailing zip: 95616 Contact person: "DUFFY, AMY" Facility capacity: 60 Type of clients served: 950 Facility phone: 5302190295		
143	East	2-4 mi	17870	Lower	EDR ID: SRDCCA200729329 Facility number: 573611117 Facility name: "CARSON, JESSICA" Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 810 Facility status code: 03 Address: 4913 COWELL BLVD. #A City: DAVIS State: CA Zip: 95616 Alt. address: 4913 COWELL BLVD. #A City: DAVIS State: CA Zip: 95616 Facility investor: "CARSON, JESSICA" Licensee type: A License effective date: 50603 License expiration date: Not Reported License issue date: 050603 Program type: "MAX. CAP: 6-NO MORE THAN 3 INFANTS OR 4 INFANTS ONLY. CAP. 8 NO MORE THAN 2 INFANTS, 1 CHILD IN KINDERGARTEN OR ELEMENTARY SCHOOL AND 1 CHILD AT LEAST AGE 6." Original app. received date: 050513 Facility closed date: Not Reported Mailing address: 4913 COWELL BLVD. #A Mailing city: DAVIS Mailing state: CA Mailing zip: 95616 Contact person: "CARSON, JESSICA" Facility capacity: 8 Type of clients served: 960 Facility phone: 5307583743	SRDCCA200729329	Daycare
AJ144	ENE	2-4 mi	18947	Lower	EDR ID: SRDCCA200743599 Facility number: 570312613 Facility name: PIONEER SCHOOL AGE CHILD DEVELOPMENT CENTER Facility eval. code: 0303 Facility office number: 03 Facility county number: 57 Facility type code: 840	SRDCCA200743599	Daycare

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Facility status code: 03
 Address: 5131 HAMEL STREET
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 5131 HAMEL STREET
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: CHILD DEVELOPMENT CENTERS
 Licensee type: C
 License effective date: 941118
 License expiration date: Not Reported
 License issue date: 900108
 Program type: LICENSED TO SERVE SCHOOL AGE CHILDREN IN KINDERGARTEN AND ABOVE IN PORTABLE ROOMS A & B.
 Original app. received date: 890822
 Facility closed date: Not Reported
 Mailing address: "851 E. HAMILTON AVE., STE 200 "
 Mailing city: CAMPBELL
 Mailing state: CA
 Mailing zip: 95008
 Contact person: R
 Facility capacity: 64
 Type of clients served: 950
 Facility phone: 5307580611

AJ145
 ENE
 2-4 mi
 18956
 Lower

EDR ID: SRDCCA200729021
 Facility number: 573610483
 Facility name: "BIRYUKOVA, TATYANA"
 Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 810
 Facility status code: 03
 Address: 5146 GLIDE DR.
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 5146 GLIDE DR.
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "BIRYUKOVA, TATYANA"
 Licensee type: A
 License effective date: 41117
 License expiration date: Not Reported
 License issue date: 041117
 Program type: "MAX. CAP: 6 - NO MORE THAN 3 INFANTS OR 4 INFANTS ONLY.
 CAP 8 - NO MORE THAN 2 INFANTS, 1 CHILD IN KINDERGARTEN OR ELEMENTARY
 SCHOOL AND 1 CHILD AT LEAST AGE 6.OFF LIMITS: MASTER BEDROOM,
 ""GARAGE, AND LAUNDRY ROOM."
 "

Original app. received date: 040927

SRDCCA200729021
 Daycare

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

Facility closed date: Not Reported
 Mailing address: 5146 GLIDE DR.
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "BIRYUKOVA, TATYANA "
 Facility capacity: 8
 Type of clients served: 960
 Facility phone: 5307470437

AJ146		SRDCCA200753096
ENE	EDR ID: SRDCCA200753096	Daycare
2-4 mi	Facility number: 573609452	
19181	Facility name: DJUSD CHILDREN'S CENTER	
Lower	Facility eval. code: 0303	
	Facility office number: 03	
	Facility county number: 57	
	Facility type code: 850	
	Facility status code: 03	
	Address: 5215 HAMEL STREET	
	City: DAVIS	
	State: CA	
	Zip: 95616	
	Alt. address: 530 B STREET	
	City: DAVIS	
	State: CA	
	Zip: 95616	
	Facility investor: DAVIS JOINT UNIFIED SCHOOL DISTRICT CHILDREN'S CTR	
	Licensee type: A	
	License effective date: 40312	
	License expiration date: Not Reported	
	License issue date: 040312	
	Program type: LICENSED TO SERVE CHILDREN FROM 2 YEARS TO ENTRY INTO KINDERGARTEN.	
	Original app. received date: 030909	
	Facility closed date: Not Reported	
	Mailing address: 530 B STREET	
	Mailing city: DAVIS	
	Mailing state: CA	
	Mailing zip: 95616	
	Contact person: "YUEN-FURTADO, MARIA "	
	Facility capacity: 22	
	Type of clients served: 950	
	Facility phone: 5307575340	

AJ147		SRPU20071014149
ENE	Ncessch: 061062001181	Public Schools
2-4 mi	Schname05: PIONEER ELEMENTARY	
19181	Mstreet05: 5215 HAMEL ST.	
Lower	Mcity05: DAVIS	
	Mstate05: CA	
	Mzip05: 95616	
	Mzip405: 4426	
	Member05: 579	
	Phone05: (530) 757-5480	

MAP FINDINGS

Map ID
Direction
Distance
Distance (ft.)
Elevation

Site

EDR ID
Database

Locale05: 3
 Type05: 1
 Level05: 1
 Gslo05: KG
 Gshi05: 06
 Edr id: SRPU20071014149

AJ148	East	EDR ID: SRDCCA200716381	SRDCCA200716381
2-4 mi		Facility number: 573607414	Daycare
19482		Facility name: "DEO, GODAWARI	"
Lower		Facility eval. code: 0303	
		Facility office number: 03	
		Facility county number: 57	
		Facility type code: 810	
		Facility status code: 03	
		Address: 5225 COWELL BLVD.	
		City: DAVIS	
		State: CA	
		Zip: 95616	
		Alt. address: 5225 COWELL BLVD.	
		City: DAVIS	
		State: CA	
		Zip: 95616	
		Facility investor: "DEO, GODAWARI	"
		Licensee type: A	
		License effective date: 727	
		License expiration date: Not Reported	
		License issue date: 000727	
		Program type: "MAXIMUM CAPACITY: 12 CHILDREN, WITH NO MORE THAN 4 INFANTS, OR CAPACITY 14 CHILDREN WHEN 2 CHILDREN ARE AT LEAST 6 YEARS OF AGE WITH A MAXIMUM OF 3 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED.	"
		Original app. received date: 000612	
		Facility closed date: Not Reported	
		Mailing address: 5225 COWELL BLVD.	
		Mailing city: DAVIS	
		Mailing state: CA	
		Mailing zip: 95616	
		Contact person: "DEO, GODAWARI	"
		Facility capacity: 14	
		Type of clients served: 960	
		Facility phone: 5307565818	

AK149	East	EDR ID: SRDCCA200732208	SRDCCA200732208
2-4 mi		Facility number: 573611364	Daycare
21062		Facility name: "YANCHER, LYNDIA & ROSS	"
Lower		Facility eval. code: 0303	
		Facility office number: 03	
		Facility county number: 57	
		Facility type code: 810	
		Facility status code: 03	
		Address: 5501 COWELL BLVD.	

MAP FINDINGS

Map ID
 Direction
 Distance
 Distance (ft.)
 Elevation

Site

EDR ID
 Database

City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 5501 COWELL BLVD.
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "YANCHER, LYNDA & ROSS"
 Licensee type: A
 License effective date: 50902
 License expiration date: Not Reported
 License issue date: 050902
 Program type: MAX. CAP(WHEN THERE IS AN ASSISTANT PRESENT): 12 - NO MORE THAN 4 INFANTS. CAP 14 - NO MORE THAN 3 INFANTS. 1 CHILD IN KINDERGARTEN OR ELEMENTARY SCHOOL AND 1 CHILD AT LEAST AGE 6.OFF LIMITS: SECOND FLOOR OF THE HOME.
 Original app. received date: 050727
 Facility closed date: Not Reported
 Mailing address: 5501 COWELL BLVD.
 Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "YANCHER, LYNDA & ROSS"
 Facility capacity: 14
 Type of clients served: 960
 Facility phone: 5307536920

AK150
 ENE
 4-6 mi
 21406
 Lower

EDR ID: SRDCCA200715735
 Facility number: 573607576
 Facility name: "SAH, NORMA"
 Facility eval. code: 0303
 Facility office number: 03
 Facility county number: 57
 Facility type code: 810
 Facility status code: 03
 Address: 5614 HOAG PLACE
 City: DAVIS
 State: CA
 Zip: 95616
 Alt. address: 5614 HOAG PLACE
 City: DAVIS
 State: CA
 Zip: 95616
 Facility investor: "SAH, NORMA & RAM"
 Licensee type: C
 License effective date: 10220
 License expiration date: Not Reported
 License issue date: 010220
 Program type: "MAXIMUM CAPACITY: 12 CHILDREN, WITH NO MORE THAN 4 INFANTS, OR CAPACITY 14 CHILDREN WHEN 2 CHILDREN ARE AT LEAST 6 YEARS OF AGE WITH A MAXIMUM OF 3 INFANTS; PROPERTY OWNER/LANDLORD CONSENT IS REQUIRED"
 Original app. received date: 000101
 Facility closed date: Not Reported
 Mailing address: 5614 HOAG PLACE

SRDCCA200715735
 Daycare

MAP FINDINGS

Map ID
Direction
Distance
Distance (ft.)
Elevation

Site

EDR ID
Database

Mailing city: DAVIS
 Mailing state: CA
 Mailing zip: 95616
 Contact person: "SAH, NORMA" "
 Facility capacity: 14
 Type of clients served: 960
 Facility phone: 5307585639

151
 West
 4-6 mi
 25390
 Higher

Ncessch: 061062005547
 Schname05: FAIRFIELD ELEMENTARY
 Mstreet05: 26960 COUNTY ROAD 96
 Mcity05: DAVIS
 Mstate05: CA
 Mzip05: 95616
 Mzip405: 9433
 Member05: 61
 Phone05: (530) 757-5370
 Locale05: 8
 Type05: 1
 Level05: 1
 Gslo05: KG
 Gshi05: 03
 Edr id: SRPU20071014154

SRPU20071014154
 Public Schools

RECORDS SEARCHED/DATA CURRENCY TRACKING

Census

Source: U.S. Census Bureau
Telephone: 301-457-4100

2000 U.S. Census data was used to estimate residential population following these EPA guidelines:
"Census data are presented by Census tract. If your circle covers only a portion of the tract, you should develop an estimate for that portion...Determine the population density per square mile (total population of the Census tract divided by the number of square miles in the tract) and apply that density figure to the number of square miles within your circle."

FED_LAND: Federal Lands

Source: USGS
Telephone: 888-275-8747

Federal lands data. Includes data from several Federal land management agencies, including Fish and Wildlife Service, Bureau of Land Management, National Park Service, and Forest Service. Includes National Parks, Forests, Monuments; Wildlife Sanctuaries, Preserves, Refuges; Federal Wilderness Areas.

AHA Hospitals:

Source: American Hospital Association, Inc.
Telephone: 312-280-5991

The database includes a listing of hospitals based on the American Hospital Association's annual survey of hospitals.

Medical Centers: Provider of Services Listing

Source: Centers for Medicare & Medicaid Services
Telephone: 410-786-3000

A listing of hospitals with Medicare provider number, produced by Centers of Medicare & Medicaid Services, a federal agency within the U.S. Department of Health and Human Services.

Nursing Homes

Source: National Institutes of Health
Telephone: 301-594-6248

Information on Medicare and Medicaid certified nursing homes in the United States.

Public Schools

Source: National Center for Education Statistics
Telephone: 202-502-7300

The National Center for Education Statistics' primary database on elementary and secondary public education in the United States. It is a comprehensive, annual, national statistical database of all public elementary and secondary schools and school districts, which contains data that are comparable across all states.

Private Schools

Source: National Center for Education Statistics
Telephone: 202-502-7300

The National Center for Education Statistics' primary database on private school locations in the United States.

Colleges - Integrated Postsecondary Education Data

Source: National Center for Education Statistics
Telephone: 202-502-7300

The National Center for Education Statistics' primary database on integrated postsecondary education in the United States.

Arenas

Source: Dunhill International

EDR indicates the location of buildings and facilities - arenas - where individuals who are public receptors are likely to be located.

Prisons: Bureau of Prisons Facilities

Source: Federal Bureau of Prisons
Telephone: 202-307-3198

List of facilities operated by the Federal Bureau of Prisons.

Daycare Centers: Licensed Facilities

Source: Department of Social Services
Telephone: 916-657-4041

STREET AND ADDRESS INFORMATION

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Sensitive Receptor Locations

Name	UTM Coordinates	
	East (m)	North (m)
COWELL STUDENT HEALTH CENTER ¹	608671.2	4266696.2
WOODLAND CLINIC MEDICAL GROUP	608783.3	4266976.2
COMMUNITY CHURCH NURSERY SCHOOL	609548.6	4266907.9
DAVIS SCHOOL FOR INDEPENDENT STUDY	609416.0	4267041.4
LA RUE PARK CHILD DEVELOPMENT CENTER ¹	607971.2	4266700.0
LA RUE PARK CHILD DEVELOPMENT CENTER ¹	607971.2	4266700.0
YOLO HOSPICE	609835.9	4266554.4
DAVIS PHYSICAL THERAPY/MATRIX	608264.8	4267161.2
RANDELL LABORATORIES	608264.8	4267161.2
JAMES A KENNEDY MD	608264.8	4267161.2
KING (MARTIN LUTHER) HIGH (CONTINUATION)	609377.1	4267210.7
RUSSELL PARK CHILD DEVELOPMENT CENTER (INFANTS) ¹	607807.1	4266839.8
RUSSELL PARK CHILD DEVELOPMENT CENTER ¹	607807.1	4266839.8
DAVIS PARENT NURSERY SCHOOL #2	608670.3	4267465.3
INTERNATIONAL PARENT-CHILD LEARNING CENTER	607994.5	4267229.7
MOORE, JANET	608365.9	4267484.4
ST JAMES ELEMENTARY SCHOOL	609274.7	4267881.9
DISCOVERY PRESCHOOL	609676.0	4267790.8
MURRAY-CLARK, JAMIE	610019.2	4267553.6
CESAR CHAVEZ STATE PRESCHOOL	608264.2	4267914.8
CESAR CHAVEZ SCHOOL AGE CDC	608264.2	4267914.8
CESAR CHAVEZ ELEMENTARY	608264.2	4267914.8
DAVIS SENIOR HIGH	608904.9	4268034.5
APPLEGATE NURSERY	607217.0	4266970.7
COOK, NOAH	607140.3	4267105.0
ROBERT E. WILLETT SCHOOL AGE CDC	607710.3	4267821.9
ROBERT E. WILLETT ELEMENTARY	607710.3	4267821.9
MALHOTRA, SURINDER	609887.6	4267933.6
NORTH DAVIS ELEMENTARY	609459.5	4268135.3
WONG-XOQUIC, HEATHER	610449.7	4267427.4
NORTH DAVIS SCHOOL AGE CHILD DEVELOPMENT CENTER	609504.7	4268147.0
LEONARDO DAVINCI HIGH	608810.7	4268297.4
EBERLE, LISA	607378.2	4267696.5
GAN HAVERIM PRESCHOOL	608264.5	4268281.1
SUTTER DAVIS VISITING NURSE ASSOC	610945.7	4266539.7
TPMG - DAVIS MOB	610992.6	4266299.5
SUTTER VISITING NURSE ASSOC - DAVIS	608808.8	4268433.8
VALLEY OAK STATE PRESCHOOL	610467.7	4267762.9
VALLEY OAK SCHOOL AGE CHILD DEVELOPMENT CENTER	610467.7	4267762.9
BOWERS, LEANN	610604.7	4267630.5
STONE, ELISA	609868.2	4268327.3
OLIVER WENDELL HOLMES JUNIOR HIGH	610160.8	4268214.8
THOMAS, DIANA	611283.5	4266255.8
RALPH WALDO EMERSON JUNIOR HIGH	606677.6	4267424.1
PHYSICIANS CLINICAL LABORATORY	608259.4	4268657.3
STEPHEN H FOSTER MD	608259.4	4268657.3
COURTYARD HEALTHCARE CENTER	610837.9	4267784.6

Sensitive Receptor Locations

Name	UTM Coordinates	
	East (m)	North (m)
COURTYARD HEALTHCARE CENTER	610837.9	4267784.6
COURTYARD HEALTHCARE CENTER	610837.9	4267784.6
BENNETT, MELE	606510.3	4267154.3
RON A BERRYHILL	610770.0	4267898.0
SAH, B. DEVI	611353.1	4265951.6
CHAVEZ, JOSEFINA	606764.9	4267676.0
SIERRA HEALTH CARE CONVALESCENT HOSP	611022.8	4267583.0
SIERRA HEALTH CARE CENTER	611022.8	4267583.0
SIERRA HEALTHCARE CONVALESCENT	611022.8	4267583.0
SIERRA HEALTH CARE CENTER	611022.8	4267583.0
NAVARRO, ESPERANZA	606755.1	4267753.6
CONNOLLY, COLLEEN	610879.3	4267878.5
LAMBERT, PATRICIA	606379.0	4267068.3
BAKAY, DAVID AND SKOG, LESLYN	610691.6	4268153.3
CUETARA, JULIE	610930.5	4267890.3
PYTEL, JEANNIE	611476.0	4266584.8
MERRYHILL SCHOOL	611512.7	4266448.8
MERRYHILL	611512.7	4266448.8
MERRYHILL SCHOOL #1036	611512.7	4266448.8
HERNANDEZ, KAY	610692.9	4268245.5
VALCARENGHI, MICHELLE	608166.3	4268900.2
MEDINA. ELIZABETH	606536.5	4267741.8
TROSTEL, TAMI	611131.9	4267825.3
ALARCON-SOTO, SANDRA& SOTO, JUAN	610613.4	4268448.6
SAH, VINA	610914.5	4268168.6
HASSAN, MABEL	610701.5	4268381.0
RAJBHANDARI, VIDYA	610783.2	4268335.5
UNIVERSITY RETIREMENT COMM AT DAVIS	607032.1	4268484.3
UNIVERSITY RETIREMENT	607032.1	4268484.3
UNIVERSITY RETIREMENT	607032.1	4268484.3
A WORLD OF LEARNING	606316.5	4267642.3
CHANG, DEE	611661.1	4266932.6
PATWIN SCHOOL AGE CHILD DEVELOPMENT CENTER	606458.0	4267946.1
PATWIN ELEMENTARY	606458.0	4267946.1
WELLNESS EXPRESS CLINIC	610485.7	4268736.5
NOORISTANI, TAIBA	606348.8	4267896.9
HARZULA, RUTH	610864.0	4268475.4
SUTTER DAVIS HOSPITAL PULMONARY LAB	607037.0	4268641.9
SUTTER DAVIS HOSPITAL	607037.0	4268641.9
ALICE VAN ALSTINE, MD	607036.5	4268677.4
WILLIAM HOCH MD	607036.5	4268677.4
JOHN D HERNRIED, MD	607036.5	4268677.4
PETER E DROUBAY MD	607036.5	4268677.4
CHARLES A DERBY MD	607036.5	4268677.4
INTERNAL MEDICINE CONSULTANTS	607036.5	4268677.4
DAVIS COMMUNITY CLINIC,THE	607036.0	4268713.0
DAVIS COMMUNITY CLINIC	607036.0	4268713.0

Sensitive Receptor Locations

Name	UTM Coordinates	
	East (m)	North (m)
GREENAMYER, ALICIA	609204.8	4269301.6
BOUGHTON, KRISTEN	610738.9	4268700.1
BIRCH LANE ELEMENTARY	610937.5	4268581.8
PARKSIDE CHILDREN'S HOUSE	605854.2	4267206.7
PARKSIDE CHILDREN'S HOUSE	605854.2	4267206.7
BIRCH LANE SCHOOL-AGE CHILD DEVELOPMENT CENTER	610930.3	4268660.5
LETELIER, EDUARDO	611990.3	4266895.0
DAVIS PARENT NURSERY SCHOOL	612051.3	4266517.4
MARGUERITE MONTGOMERY CHILD DEVELOPMENT CENTER	612051.3	4266517.4
MARGUERITE MONTGOMERY CHILD DEVELOPMENT CENTER	612051.3	4266517.4
MARGUERITE MONTGOMERY ELEMENTARY	612051.3	4266517.4
WILLIAMS, GINA	605954.5	4267665.3
REYNOSO, GUADALUPE	611681.1	4267875.2
FARMER, KATIE	606016.6	4267975.8
HILLMAN, ANNE	605958.1	4267919.5
MONTESSORI COUNTRY DAY	610790.6	4268993.8
MONTESSORI COUNTRY DAY	610790.6	4268993.8
WOODLAND HEALTHCARE-DAVIS MEDICAL GRP	606363.4	4268573.0
CHAVEZ, IRMA	608836.6	4269658.4
MONTESSORI COUNTRY DAY II	612008.0	4267622.2
MONTESSORI COUNTRY DAY II	612008.0	4267622.2
BARAJAS, MARIA	612278.9	4266640.4
KUSS, LESLIE	611346.1	4268712.9
PROGRESS RANCH - THE GROVE	611714.4	4268299.6
DAVIS WALDORF SCHOOL	607494.8	4269593.7
TENDER LEARNING CARE	605849.9	4268325.4
ALEMI, NAJ	612443.5	4266958.0
ROMERO, LUCY	605404.3	4267552.6
SALAMATI, ROSHAN	611311.0	4269108.7
SAH, RANJANA	612637.1	4267139.4
YOLO CRISIS NURSERY-FAMILIES FIRST INC.	611877.3	4268745.8
YOLO CRISIS NURSERY-FAMILIES FIRST INC.	611877.3	4268745.8
FAMILIES FIRST, INC.	611877.3	4268745.8
MAROTTO, JOANNE	612126.9	4268527.4
REDBUD MONTESSORI	604893.2	4266851.1
HOLM, CHRISTINA	612224.3	4268546.5
POUDYAL, SHANTI	611842.0	4269087.2
THORESON, NITA	611952.1	4269006.6
STEPHENS, MARGARET	605308.6	4268402.6
HONEYCUTT, BEOLA	612100.9	4268828.9
DAVIS CHILDREN'S CENTER	612462.0	4268376.7
FRED T. KOREMATSU ELEMENTARY SCHOOL AT MACE RANCH	612462.0	4268376.7
DAHAL, SHAKUNTALA	611860.9	4269230.6
MERRYHILL COUNTRY SCHOOL	612992.2	4267297.5
MERRYHILL COUNTRY SCHOOL - LAVIDA	612992.2	4267297.5
RAMOS, OLGA	612755.8	4268055.6
MOHAMED, SAYDA	613117.0	4268276.0

Sensitive Receptor Locations

Name	UTM Coordinates	
	East (m)	North (m)
UNIVERSITY COVENANT NURSERY SCHOOL	613859.6	4267594.0
CARSON, JESSICA	614194.7	4267323.5
PIONEER SCHOOL AGE CHILD DEVELOPMENT CENTER	614489.2	4267516.4
BIRYUKOVA, TATYANA	614442.6	4267723.3
DJUSD CHILDREN'S CENTER	614562.3	4267518.5
PIONEER ELEMENTARY	614562.3	4267518.5
DEO, GODAWARI	614676.1	4267424.7
YANCHER, LYNDA & ROSS	615152.7	4267495.9
SAH, NORMA	615232.8	4267625.8
FAIRFIELD ELEMENTARY	601151.8	4267022.8
HUTCHISON CHILD DEVELOPMENT CENTER ^{1,2}	607957.5	4266308.2

¹ This sensitive receptor is located onsite.

² This daycare center is new and is not included as part of the EDR Sensitive Receptor Report.

Appendix F
Modeling Files ISCST3 and
HARP Electronic Files

Appendix G
Chemical Profiles for Main
Contributors to Estimated Health
Risks

CHRONIC TOXICITY SUMMARY

GLUTARALDEHYDE

(1,5-pentanedial; 1,5-pentanedione; glutaric dialdehyde; Aldesen; Cidex; Sonacide)

CAS Registry Number: 111-30-8

I. Chronic Toxicity Summary

<i>Inhalation reference exposure level</i>	0.08 µg/m³ (0.02 ppb)
<i>Critical effect(s)</i>	Squamous metaplasia of the respiratory epithelium in the nose of male and female mice
<i>Hazard index target(s)</i>	Respiratory system

II. Chemical Property Summary (HSDB, 1996; CRC, 1994; Chemfinder, 2000)

<i>Description</i>	Colorless liquid/oil
<i>Molecular formula</i>	C ₅ H ₈ O ₂
<i>Molecular weight</i>	100.12 g/mol
<i>Boiling point</i>	188°C (decomposes) (CRC, 1994)
<i>Melting point</i>	-6°C (Chemfinder, 2000)
<i>Solubility</i>	Soluble in water, alcohol, benzene
<i>Conversion factor</i>	4.1 µg/m ³ per ppb at 25°C

III. Major Uses and Sources

Glutaraldehyde is a chemical frequently used as a disinfectant and sterilizing agent against bacteria and viruses (2% solution), an embalming fluid and tissue fixative, a component of leather tanning solutions, and an intermediate in the production of certain sealants, resins, dyes, and electrical products (HSDB, 1996). For commercial purposes, solutions of 99%, 50%, and 20% are available. Glutaraldehyde is also an atmospheric reaction product of cyclohexene. The annual statewide industrial emissions from facilities reporting under the Air Toxics Hot Spots Act in California based on the most recent inventory were estimated to be 29,603 pounds of glutaraldehyde (CARB, 2000).

IV. Effects of Human Exposure

Evidence of the toxicity of glutaraldehyde to humans is limited to reports of occupational exposure from its use as a disinfectant and sterilizing agent. Frequently observed effects from exposure include skin sensitivity resulting in dermatitis, and irritation of the eyes and nose with accompanying rhinitis (Jordan *et al.*, 1972; Corrado *et al.*, 1986; Hansen, 1983; Wiggins *et al.*,

1989). Occupational asthma has also been reported among workers repeatedly exposed to glutaraldehyde, particularly respiratory technologists who use glutaraldehyde as a sterilizing agent for endoscopes (Chan-Yeung *et al.*, 1993; Stenton *et al.*, 1994; Gannon *et al.*, 1995). Quantitation of the exposure levels that led to glutaraldehyde sensitization was not available from the studies.

V. Effects of Animal Exposure

The histopathology of the respiratory tract in rats and mice exposed to glutaraldehyde by inhalation was examined (Gross *et al.*, 1994). F344 rats and B6C3F1 mice (20 animals of each sex and of each species at each exposure level for a total of 480 rodents) were continuously exposed to glutaraldehyde in recirculating exposure chambers at concentrations of 0, 62.5, 125, 250, 500, or 1000 ppb glutaraldehyde for one day, 4 days, 6 weeks, or 13 weeks. At termination, respiratory tract tissue as well as duodenum and any gross lesions were collected and formalin fixed. Animals were treated with tritiated thymidine two hours before termination to evaluate cell replication in certain respiratory tract tissues. Respiratory tract tissue sections were made as follows: transverse sections of the nose and trachea, frontal section of the carina, and longitudinal section of the lung. Ten male and 10 female mice exposed to 1000 ppb and one female mouse exposed to 500 ppb group died during the course of the study. Two male and 3 female rats exposed to 1000 ppb died during the course of the study. Histopathological examination of animals surviving to the end of the study entailed scoring the severity of the finding from “no response” to “very severe” response on a 0 to 5 scale. Unit length labeling index, the indicator of cell proliferation, was evaluated by autoradiography at two sites: the nasal vestibule and the dorsal atrioturbinate.

Lesions in animals treated with glutaraldehyde appeared primarily in the anterior third of the nose. Lesions were apparently more increased in mice compared to rats due to some level of “background” non-suppurative lesions in the rats. Mice were considered devoid of background lesions. In the 13-week study, female mice were the most sensitive, with lesions averaging a score of 2 (mild and clear, but of limited extent and/or severity). The lesions were characterized as neutrophilic infiltration primarily in the squamous epithelium of the vestibule, with thickening of the epithelium leading to loss of the characteristic surface grooves. Both cell size and number were reported to be increased. Lesions were generally found to increase in nature and severity with increased time and level of exposure. Obstruction of the nasal vestibule was thought to account for the mortality of animals in the higher dose groups. In female mice at 13 weeks, all glutaraldehyde dose groups showed the accumulation of eosinophilic proteinaceous deposits in the respiratory epithelium of the maxilloturbinate margin. Examination of unit length labeling indices as a measure of growth showed significant increases in all treated groups of female mice. No evidence of exposure related lesions was found in the respiratory tract in the trachea, carina, bronchi, or lungs.

Mean Subjective Pathology Scores for Nasal Lesions in Female Mice at 13 Weeks

	<i>Glutaraldehyde</i>	<i>Intraepithelial neutrophils</i>	<i>Subepithelial neutrophils</i>	<i>Squamous metaplasia</i>
0 ppb		0	0.4	0
62.5 ppb		2.0	2.0	0
125 ppb		2.4	2.8	0
250 ppb		3.2	3.2	0
500 ppb		2.8	2.8	0.5
1000 ppb*		--	--	--

*Animals exposed to 1000 ppb died early in the experiment.

Greenspan *et al.* (1985) exposed male and female F-344 rats to 0, 0.3, 1.1 and 3.1 ppm glutaraldehyde and 0, 0.2, 0.63, and 2.1 ppm glutaraldehyde, respectively, in a 9-day study, and both sexes to 0, 21, 49, and 194 ppb glutaraldehyde in a 14 week study. Animal numbers were not specified. Exposures were conducted for 6 hours per day, 5 days per week. In the 9-day study, observations in the high and intermediate dose level groups included reduced body weight gain, inflammation of the nasal and olfactory mucosa, and sensory irritation. In the two highest doses of the 14-week study, statistically significant differences in body weight gain were observed as well as perinasal wetness. No histopathological indication of inflammation in olfactory or nasal mucosa was observed.

Mice were exposed to 0, 0.3, 1.0, and 2.6 ppm glutaraldehyde vapors for 6 hours/day for 4, 9, or 14 days (Zissu *et al.*, 1994). These mice were killed immediately after the exposure period. Other groups exposed to 1.0 ppm for 14 days were killed after recovery periods of 1, 2, and 4 weeks. After 4 days of exposure to the lowest dose, mice showed lesions in the respiratory epithelium of the septum, and the naso- and maxilloturbinates. After exposure to 1.0 ppm glutaraldehyde, lesions were still judged as severe after 2 weeks of recovery.

A study comparing the effects of intra-nasally instilled glutaraldehyde and formaldehyde on rat nasal epithelium found inflammation, epithelial degeneration, respiratory epithelial hypertrophy, and squamous metaplasia in treated animals (St. Clair *et al.*, 1990). Acute inhalation exposure to formaldehyde produced identical lesions. Ten-fold higher concentrations of instilled formaldehyde were required to produce the same effect as instilled glutaraldehyde.

In a chronic study, NTP (1998, 1999) exposed groups of 50 male and 50 female F344/N rats to 0, 250, 500, or 750 ppb glutaraldehyde vapor by inhalation for 6 h/day, 5 days/week, for 104 weeks. Survival of 500 and 750 ppb female rats was less than that of the chamber controls. Mean body weights of all exposed groups of male rats and 500 and 750 ppb female rats were generally less than those of the chamber controls. Increased incidences of nonneoplastic nasal lesions occurred primarily within the anterior section of the nose in 500 and 750 ppb rats and to a lesser extent in 250 ppb rats. The more significant lesions included hyperplasia and inflammation of the squamous and respiratory epithelia and squamous metaplasia of the respiratory epithelium. Thus 250 ppb (1000 $\mu\text{g}/\text{m}^3$) is a chronic LOAEL for rats.

In the same study NTP (1998, 1999) exposed groups of 50 male and 50 female B6C3F1 mice to 0, 62.5, 125, or 250 ppb glutaraldehyde vapor by inhalation for 6 h/day, 5 days/week, for 104

weeks. Survival of exposed mice was similar to that of the chamber controls. Mean body weights of female mice exposed to 250 ppb were generally less than those of the controls. The incidence of inflammation of the nose was marginally increased in 250 ppb females. Incidences of squamous metaplasia of the respiratory epithelium were increased in 250 ppb males and females and 125 ppb females. Incidences of hyaline degeneration of the respiratory epithelium were increased in all exposed groups of females. Thus 62.5 ppb was a chronic LOAEL for female mice.

Incidence of Nasal Lesions in Female Mice exposed for 104 weeks

	<i>Glutaraldehyde</i>	<i>Inflammation</i>	<i>Respiratory epithelium hyaline degeneration</i>	<i>Respiratory epithelium squamous metaplasia</i>
	0 ppb	6/50	16/50	7/50
	62.5 ppb	7/49	35/49	11/49
	125 ppb	13/50	32/50	16/50
	250 ppb	14/50	30/50	21/50

VI. Derivation of Chronic Reference Exposure Level (REL)

<i>Study</i>	NTP 1998, 1999
<i>Study population</i>	Male and female F344 rats and B6C3F1 mice (50/sex/group)
<i>Exposure method</i>	Continuous inhalation exposure (0, 62.5, 125, and 250 ppb in mice; 0, 250, 500, or 750 ppb in rats)
<i>Critical effects</i>	Respiratory epithelium squamous metaplasia
<i>LOAEL</i>	62.5 ppb (female mice)
<i>NOAEL</i>	Not observed
<i>BMC₀₅</i>	20.5 ppb
<i>Exposure continuity</i>	6 hr/day, 5 days/week
<i>Exposure duration</i>	104 weeks
<i>Equivalent continuous exposure</i>	3.7 ppb (20.5 x 6/24 x 5/7)
<i>Human equivalent concentration</i>	0.62 ppb (gas with extrathoracic respiratory effects, RGDR = 0.17, BW = 28 g, MV = 0.032 L/min, SA = 3 cm ²)
<i>LOAEL uncertainty factor</i>	not needed in BMC approach
<i>Subchronic uncertainty factor</i>	1
<i>Interspecies uncertainty factor</i>	3
<i>Intraspecies uncertainty factor</i>	10
<i>Cumulative uncertainty factor</i>	30
<i>Inhalation reference exposure level</i>	0.02 ppb (0.08 µg/m ³)

Several studies indicate that the upper respiratory tract is a target for the toxicity of glutaraldehyde from inhalation exposure. Reports of toxicity to humans show that exposure can

lead to occupational asthma as well as cause irritation of the eyes and nose with accompanying rhinitis. Likewise, animals exposed to glutaraldehyde by the inhalation route show evidence of respiratory irritation with the induction of lesions of the anterior nasal cavities upon long-term exposure (Gross *et al.*, 1994; Greenspan *et al.*, 1985; NTP, 1998, 1999). The NTP (1998, 1999) study yielded a chronic LOAEL for female mice of 62.5 ppb. Gross *et al.* (1994) showed neutrophilic infiltration in the olfactory epithelium in the lowest dose exposure group. (Female mice exposed to 62.5 ppb also showed subepithelial neutrophilic infiltration.) This level was taken to be the subchronic LOAEL. This effect on the nasal epithelium was demonstrated to be both concentration- and exposure duration-dependent.

A benchmark concentration was determined using EPA's version 1.20 BMC software and the dose-response data on respiratory epithelium squamous metaplasia in female mice. The quantal-linear model gave an MLE₀₅ of 31.24 ppb, a BMC₀₅ of 20.51 ppb, and a p value of 0.9471. With the benchmark approach no LOAEL UF is needed. The study was a lifetime study so the subchronic UF is 1. An interspecies UF of 3 rather than 10 was used since an RGDR adjustment had been made. The default intraspecies UF of 10 was used so that the total UF was 30. The resulting chronic REL for glutaraldehyde is 0.02 ppb (0.08 µg/m³).

For comparison with the proposed REL, the study of Gross *et al.* (1994) used 62.5 ppb continuous exposure. Multiplying by the RGDR of 0.17 and dividing by a cumulative uncertainty factor of 300 (3 for a LOAEL, 3 for subchronic, 3 for interspecies, and 10 for intraspecies) results in a REL of 0.035 ppb (0.1 µg/m³).

VII. Data Strengths and Limitations for Development of the REL

The major strength of the inhalation REL for glutaraldehyde is the availability of controlled exposure inhalation studies in multiple species at multiple exposure concentrations and with adequate histopathological analysis. Major areas of uncertainty are the lack of human data, the lack of reproductive and developmental toxicity studies, the lack of dermal sensitization studies, and the lack of observation of a NOAEL.

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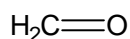
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Formaldehyde Reference Exposure Levels

(Methanal, oxomethane, methylene oxide)

CAS 50-00-0



1. Summary

The non-cancer adverse health effects of formaldehyde are largely a manifestation of its ability to irritate mucous membranes. As a result of its solubility in water and high reactivity, formaldehyde is efficiently absorbed into the mucus layers protecting the eyes and respiratory tract where it rapidly reacts, leading primarily to localized irritation. Acute high exposure may lead to eye, nose and throat irritation, and in the respiratory tract, nasal obstruction, pulmonary edema and dyspnea. Prolonged or repeated exposures have been associated with allergic sensitization, respiratory symptoms (coughing, wheezing, shortness of breath), histopathological changes in respiratory epithelium, and decrements in lung function. Children, especially those with diagnosed asthma, may be more likely to show impaired pulmonary function and symptoms than are adults following chronic exposure to formaldehyde. The studies reviewed for this document include those published through the Spring of 2008.

1.1 Formaldehyde Acute REL

<i>Reference Exposure Level</i>	55 $\mu\text{g}/\text{m}^3$ (44 ppb)
<i>Critical effect(s)</i>	Mild and moderate eye irritation
<i>Hazard Index target(s)</i>	Eye irritation

1.2 Formaldehyde 8-Hour REL

<i>Reference Exposure Level</i>	9 $\mu\text{g}/\text{m}^3$ (7 ppb)
<i>Critical effect(s)</i>	Nasal obstruction and discomfort, lower airway discomfort, and eye irritation
<i>Hazard Index target(s)</i>	Respiratory

1.3 Formaldehyde Chronic REL

<i>Reference Exposure Level</i>	9 $\mu\text{g}/\text{m}^3$ (7 ppb)
<i>Critical effect(s)</i>	Nasal obstruction and discomfort, lower airway discomfort, and eye irritation
<i>Hazard Index target(s)</i>	Respiratory

2. Physical & Chemical Properties (ATSDR, 1999)

<i>Description</i>	Colorless gas
<i>Molecular formula</i>	CH ₂ O
<i>Molecular weight</i>	30.03 g/mol
<i>Density</i>	0.815 g/L @ -20° C
<i>Boiling point</i>	-19.5° C
<i>Melting point</i>	-92° C
<i>Vapor pressure</i>	3883 mm Hg @ 25° C
<i>Flashpoint</i>	300° C
<i>Explosive limits</i>	7% - 73%
<i>Solubility</i>	soluble in water, alcohol, ether and other polar solvents
<i>Odor threshold</i>	0.05-0.5 ppm
<i>Metabolites</i>	formic acid
<i>Conversion factor</i>	1 ppm in air = 1.24 mg/m ³ @ 25° C

3. Occurrence and Major Uses

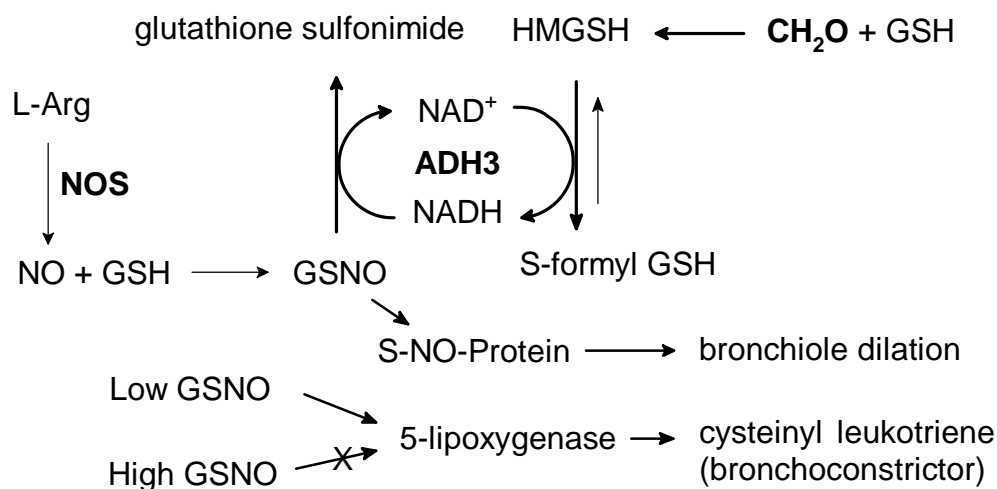
Formaldehyde has four major applications: as an intermediate in the manufacture of melamine, polyacetal, and phenolic resins; as an intermediate in the production of industrial chemicals; as a bactericide or fungicide; and as a component in the manufacture of end-use consumer products. Phenol-formaldehyde resins are used in the production of plywood, particleboard, foam insulation, and a wide variety of molded or extruded plastic items. Formaldehyde is also used as a preservative, a hardening and reducing agent, a corrosion inhibitor, a sterilizing agent, and in embalming fluids. Indoor sources include upholstery, permanent press fabrics, carpets, pesticide formulations, urea-formaldehyde foam insulation, and cardboard and paper products. Outdoor sources include emissions from fuel combustion (motor vehicles), industrial fuel combustion (power generators), oil refining processes, and other uses (copper plating, incinerators, etc.). The largest portion of outdoor ambient formaldehyde results from photochemical oxidation of a number of reactive organic gases in the atmosphere (CARB, 2006). According to the California Toxics Inventory (CARB, 2005a), the mean statewide ambient level of formaldehyde in 2004 was 2.69 ppb, with the highest levels (3.76 ppb) reported for the South Coast Air Basin. The California Air Resources Board (CARB) reported statewide emissions of 20,251 tons from stationary and mobile sources (CARB, 2005b).

4. Metabolism

Inhaled formaldehyde reacts rapidly at the site of contact and is efficiently absorbed in the respiratory tract. A portion of the formaldehyde entering the fluid layer covering the respiratory epithelium, the respiratory tract lining fluid (RTLFL), is reversibly hydrated to methylene glycol. Among other components, the RTLFL is rich in antioxidants including glutathione (Cross et al., 1994) with which formaldehyde may reversibly react to form *S*-hydroxymethylglutathione. Both the hydrated and unreacted formaldehyde may be absorbed into the epithelial layer where there is further opportunity for formaldehyde to bind to glutathione. This glutathione conjugate in turn is oxidized to *S*-formylglutathione by formaldehyde dehydrogenase. Hydrolysis of *S*-formylglutathione yields formate and glutathione. Formic acid may be eliminated in urine and

feces, or dehydrogenated to CO₂ and exhaled. The presence of glutathione and formaldehyde dehydrogenase in epithelial cells of the respiratory tract varies with location and influences the amount of formaldehyde reaching the blood. While glutathione-bound formaldehyde is rapidly metabolized, free formaldehyde in cells can form DNA-protein cross-links (Franks, 2005).

Formaldehyde dehydrogenase (ADH3), although central to the metabolism of formaldehyde, has a broad specificity that includes the structurally related compound, S-nitrosoglutathione (GSNO), an endogenous bronchodilator and reservoir of nitric oxide (NO) activity (Jensen et al., 1998). In cultured cells, formaldehyde appears to trigger ADH3-mediated GSNO reduction by enzyme-bound cofactor recycling (Staab et al., 2008). As shown in Figure 1, the S-hydroxymethylglutathione (HMGS) formed spontaneously from formaldehyde and glutathione is oxidized by ADH3 with the formation of NADH that may then participate in the ADH3-mediated reduction of GSNO (Thompson and Grafstrom, 2008). (Because of its participation in this reaction, ADH3 is also known as GSNO reductase.) This reductive pathway results in low levels of GSNO that in turn stimulate the production and activity of 5-lipoxygenase, the rate-limiting enzyme in the synthesis of powerful bronchoconstrictors, the cysteinyl leukotrienes. On the other hand, high levels of GSNO inhibit this enzyme and thus the synthesis of the bronchoconstrictors (Zaman et al., 2006). Up-regulation of the degradation of GSNO has been demonstrated in mouse lung following inhalation of formaldehyde (Yi et al., 2007), while low levels of GSNO in the lungs have been associated with severe asthma attacks in children (Gaston et al., 1998) and airway hyperactivity in mice (Que et al., 2005). These results suggest that the potential association of formaldehyde exposure with asthma-like respiratory symptoms is in part due to its effects on NO via the enhanced degradation of GSNO. Nitric oxide has multiple functions in the lungs, from its participation in the regulation of airway and vascular tone to mucin secretion and mucociliary clearance (Reynaert et al., 2005). The dysregulation of NO by formaldehyde helps to explain the variety and variability in the toxic manifestations following formaldehyde inhalation.

FIGURE 3 FORMALDEHYDE DRIVEN REDUCTION OF GSNO

Oxidation of the glutathione conjugate of formaldehyde, HMGSH, by ADH3 generates NADH that drives the reduction of GSNO, also by ADH3, thereby reducing the nitric oxide available for bronchiole dilation. Low GSNO levels stimulate, but high GSNO levels inhibit 5-lipoxygenase production of cysteinyl leukotriene.

5. Acute Toxicity of Formaldehyde

The acute effects of formaldehyde exposure appear to be largely a result of its irritant properties. However, some individuals experience symptoms following acute exposures that are a result of previous sensitization following acute high formaldehyde exposure, or long term low level exposures. For this reason, some of the studies included in this section describe manifestations of toxicity in which acute exposure was the precipitating event but in which the contribution of previous exposures or sensitization is unknown. Sensitization manifests as heightened responsiveness and may be of an immunological nature with the development of formaldehyde-specific IgE or IgG (e.g. Thrasher et al. 1987). Alternatively, heightened responsiveness may be neurologically mediated with involvement of the hypothalamic/pituitary/adrenal axis (Sorg et al., 2001a,b). In addition, genetic variation among individuals in the alcohol dehydrogenases mentioned above affects individual responses to formaldehyde. This is especially germane to studies in which the effects include symptoms such as bronchoconstriction and airway hyperreactivity, and in which there is unexpected individual variation.

Many of the studies described in this document have evaluated the relationship between formaldehyde inhalation and clinically-diagnosed asthma or asthma-like symptoms. Asthma is a chronic disease of airway obstruction resulting in variable airflow that has classically been considered to involve both airway inflammation and airway hyperresponsiveness. Asthma manifests as a characteristic cough, wheeze, and shortness of breath due to spasmodic contractions of the bronchi and mucus hypersecretion. These symptoms may or may not reflect an underlying allergic response. As shown in the study by Que et al. (2005), the hyperresponsiveness and the inflammation are not necessarily coupled. Although the RELs presented in this document are not based on studies that used asthma as the critical endpoint, uncertainty factors were applied in the REL estimates to explicitly consider the potential of

formaldehyde to cause or exacerbate asthma-like wheeze and cough symptoms, especially in asthmatic children. We have therefore included discussion of recent work that provides a biochemical mechanism by which formaldehyde exposure is linked to at least one symptom of asthma, bronchoconstriction. The bronchoconstrictive effects of formaldehyde exposure may be partially responsible for the lower airway discomfort reported in the study upon which the 8-hour and chronic RELs are based.

5.1 Acute Toxicity to Adult Humans

In small human studies, exposure to formaldehyde (1-3 ppm) has resulted in eye and upper respiratory tract irritation (Weber-Tschopp et al., 1977; Kulle et al., 1987). Most people cannot tolerate exposures to more than 5 ppm formaldehyde in air; above 10-20 ppm symptoms become severe and shortness of breath occurs (Feinman, 1988). High concentrations of formaldehyde may result in nasal obstruction, pulmonary edema, choking, dyspnea, and chest tightness (Porter, 1975; Solomons and Cochrane, 1984).

A few human case studies report severe pulmonary symptoms. A medical intern with known atopy and exposure to reportedly high (but unspecified) levels of formaldehyde over a period of 1 week developed dyspnea, chest tightness, and edema, following a subsequent 2 hour exposure to formaldehyde (Porter, 1975). Five workers exposed to formaldehyde from newly installed urea-formaldehyde chipboard in a poorly ventilated basement experienced intolerable eye and upper respiratory tract irritation, choking, marked dyspnea, and nasal obstruction (Solomons and Cochrane, 1984). However, the concentration of formaldehyde and the contribution of other airborne chemicals were unknown in both reports.

Numerous acute controlled and occupational human exposure studies have been conducted with both asthmatic and normal subjects to investigate formaldehyde's irritative and pulmonary effects (Frigas et al., 1984; Sheppard et al., 1984; Sauder et al., 1986; Schachter et al., 1986; Kulle et al., 1987; Sauder et al., 1987; Schachter et al., 1987; Witek et al., 1987; Uba et al., 1989; Harving et al., 1990; Akbar-Khanzadeh et al., 1994). Short exercise sessions during exposure on a bicycle ergometer were included in some of the studies. Concentrations of formaldehyde in the human exposure studies ranged as high as 3 ppm for up to 3 hours. The major findings in these studies were mild to moderate eye and upper respiratory tract irritation typical of mild discomfort from formaldehyde exposure.

Chemosensory irritation and subjective symptoms following exposure to formaldehyde at concentrations relevant to the workplace were examined by Lang et al. (2008) in 11 male and 10 female volunteers. Each subject was exposed for 4 hours to a randomized sequence of ten exposure conditions. These included exposures at concentrations of 0, 0.15, 0.3 and 0.5 ppm, exposures at 0.3 and 0.5 ppm that included four transient peak exposures at 0.6 and 1.0 ppm, respectively, and exposures in the presence of 10 ppm ethyl acetate of 0, 0.3, 0.5, and 0.5 ppm with 1.0 ppm peaks. Objective measures of irritation included conjunctival redness, blinking frequency, nasal flow resistance, pulmonary function, and reaction times. The participant's subjective evaluation of physical and mental wellbeing was assessed by questionnaire before, during and after each day's exposure. To assess the potential influence of personality traits on subjective responses, each subject's positive or negative affectivity was evaluated with PANAS (Positive and Negative Affectivity Schedule) that consists of 10 positive affects (interested,

excited, strong, enthusiastic, proud, alert, inspired, determined, attentive, and active) and 10 negative affects (distressed, upset, guilty, scared, hostile, irritable, ashamed, nervous, jittery, and afraid). Participants are asked to rate items on a scale from 1 to 5, based on the strength of emotion where 1 = "very slightly or not at all," and 5 = "extremely". Subjective ratings of eye irritation and olfactory symptoms were significantly higher than control at 0.3 ppm. However, when negative affectivity (anxiety) was included as a covariate, eye and olfactory irritation at this exposure level were no longer significant. Conjunctival irritation and blinking frequency, objective measures of irritation, were significantly elevated only with exposure to 0.5 ppm with peaks of 1.0 ppm ($p < 0.05$). The authors considered this level to be a LOAEL. However, at 0.5 ppm without 1.0 ppm peaks, conjunctival irritation and blinking were not significantly increased so this was considered a NOAEL for these effects. There were no statistically significant changes in nasal resistance, pulmonary function or reaction time. While there were large inter-individual differences in complaints or reports of wellbeing, there were no significant treatment effects. This study identified eye irritation as the most sensitive endpoint, with personality traits, such as negative affectivity, as a modifying factor.

In a human irritation study by Weber-Tschopp et al. (1977), 33 subjects were exposed to formaldehyde at concentrations ranging from 0.03-3.2 ppm (0.04-4.0 mg/m³) for 35 minutes. Thresholds were 1.2 ppm (1.5 mg/m³) for eye and nose irritation, 1.7 ppm (2.1 mg/m³) for eye blinking, and 2.1 ppm (2.6 mg/m³) for throat irritation.

Kulle et al. (1987) exposed nonasthmatic humans to up to 3.0 ppm (3.7 mg/m³) formaldehyde in a controlled environmental chamber for 3 hours. Significant dose-response relationships were seen with odor and eye irritation (Table 5.1) as ranked on symptom questionnaires as none, mild, moderate or severe. Irritation was assessed in this manner prior to exposure, at the end of exposure, and again 24 hour after exposure.

TABLE 5.1 MEAN SYMPTOM DIFFERENCE ($T_{180}-T_0$) \pm SE WITH FORMALDEHYDE* (FROM KULLE ET AL., 1987)

	Formaldehyde conc. (ppm)				P value
	0.0	1.0	2.0	3.0	
Odor sensation	0.00 \pm 0.00	0.22 \pm 0.15	0.44 \pm 0.18	1.00 \pm 0.29	<0.0001
Nose/throat irritation	0.00 \pm 0.00	0.11 \pm 0.11	0.33 \pm 0.17	0.22 \pm 0.15	0.054
Eye irritation	0.00 \pm 0.00	0.44 \pm 0.24	0.89 \pm 0.26	1.44 \pm 0.18	<0.0001
Chest discomfort	0.00 \pm 0.00	0.00 \pm 0.00	0.11 \pm 0.11	0.00 \pm 0.00	0.62
Cough	0.00 \pm 0.00	0.11 \pm 0.11	0.00 \pm 0.00	0.00 \pm 0.00	0.11
Headache	0.00 \pm 0.00	0.00 \pm 0.00	0.00 \pm 0.00	0.11 \pm 0.11	0.33

*Presence and severity of symptoms scored as: 0 = none; 1 = mild (present but not annoying); 2 = moderate (annoying); 3 = severe (debilitating). Data from Table II.

At 0.5 ppm for 3 hours, none of 9 subjects had eye irritation. At 1.0 ppm, 3 of 19 subjects reported mild eye irritation and one experienced moderate irritation. At 2.0 ppm, 6 subjects reported mild and 4 reported moderate eye irritation. Measured nasal flow resistance was increased at 3.0 ppm but not at 2.0 ppm (2.5 mg/m³). With respect to the lower respiratory tract, there were no significant decrements in pulmonary function nor increases in methacholine

induced bronchial reactivity as a result of 3-hour exposures to 0.5-3.0 ppm (0.6-3.7 mg/m³) formaldehyde at rest or during exercise, including 24 hours post exposure.

Eleven healthy subjects and nine patients with formalin skin sensitization were exposed to 0.5 mg/m³ (0.4 ppm) formaldehyde for 2 hours (Pazdrak et al., 1993). Nasal lavage was performed prior to and 5 to 10 minutes, 4 hours, and 18 hours after exposure. Rhinitis was reported and increases in the number and proportion of eosinophils, elevated albumin and increased protein levels were noted in nasal lavage fluid 4 and 18 hours after exposure. No differences were found between patients with skin sensitization and healthy subjects.

In a study by Green et al. (1987), volunteer asthmatic and normal subjects exposed to formaldehyde displayed decrements in pulmonary function. Exposure to 3 ppm formaldehyde for 1 hour resulted in clinically significant reductions of forced expiratory volume in one second (FEV₁) (defined as > 20% or more) and FEV₁/forced vital capacity (FVC) (ratio 70% or less) in 5 individuals in the study (2 of 16 asthmatics, 2 of 22 normal subjects, and one clinically normal subject with hyperactive airways). Of these individuals, 3 had reductions of FEV₁ of 20% or more during exposure. One of 22 asthmatics had a greater than 20% reduction in FEV₁ (-25.8%) at 17 minutes into exposure following a 15 minute moderate exercise session (minute ventilation [V_E] = 30-40 l/min), which, according to the authors, was low enough to prevent exercise-induced bronchospasm. One of 22 normal subjects also exhibited a greater than 20% clinically significant reduction in FEV₁ (-24.4%) and in FEV₁/FVC, which occurred at 47 minutes into exposure to 3 ppm formaldehyde. These reductions occurred following a second 15-minute heavy-exercise session (V_E = 60-70 l/min) near the end of the 1 hour exposure period. A third asymptomatic "normal" subject with hyperactive airways had a clinically significant reduction of FEV₁ (-20.5%) at 17 minutes, following the first heavy exercise session. This subject exhibited occult airway hyperactivity and was excluded from analysis with the other exposure groups due to his respiratory condition. Subjects exhibiting reductions in FEV₁ of greater than 20% following exposure also exhibited FEV₁/FVC ratios of less than 70%. However, none of the subjects in the study exhibited a clinically significant reduction of 50% or greater in airway conductance (SG_{aw}) during exposure to 3 ppm formaldehyde.

Kriebel et al. (2001) conducted a subchronic epidemiological study of 38 anatomy class students who, on average, were exposed to a geometric mean of 0.70 ± 2.13 ppm for 2 hours per week over 14 weeks. After class, eye, nose and throat irritation was significantly elevated compared with pre-laboratory session exposures, with a one unit increase in symptom intensity/ppm of formaldehyde. Peak expiratory flow (PEF) was found to decrease by 1%/ppm formaldehyde during the most recent exposure. Changes in PEF and symptom intensity following formaldehyde exposure were most pronounced during the first weeks of the semester but attenuated with time, suggesting partial acclimatization.

Rhinitis and a wide range of respiratory symptoms can result from exposure to formaldehyde. Some studies have reported that workers exposed to low concentrations may develop severe prolonged asthma attacks after prior exposure; this suggests that they may have become sensitized (Feinman, 1988). However, in adults, an association between formaldehyde exposure and allergic sensitization through IgE- and IgG-mediated mechanisms has been observed only inconsistently (Thrasher et al., 1987; Krakowiak et al., 1998; Wantke et al., 2000; Kim et al., 2001).

Formaldehyde provocation of human subjects, occupationally exposed to formaldehyde and suffering from respiratory symptoms such as wheezing, shortness of breath, or rhinitis, occasionally resulted in pulmonary function decrements (2 to 33% response rate) consistent with immediate, delayed, or both immediate and delayed bronchoconstriction (Hendrick and Lane, 1977; Wallenstein et al., 1978; Burge et al., 1985; Nordman et al., 1985). While some of the concentrations of formaldehyde that elicited a positive response following provocation tests (6 to 20.7 ppm) were quite high, the authors of these studies suggested that formaldehyde-induced bronchial hyperreactivity is due to specific sensitization to the gas. However, none of these studies was able to detect antibodies to formaldehyde which would support that sensitization to formaldehyde occurs through an immunologic pathway. Alternatively, the wheezing and shortness of breath may be related to the formaldehyde-stimulated depletion of the bronchodilator, GSNO, in the airways.

In controlled studies with asthmatics from urea-formaldehyde insulated homes, formaldehyde concentrations equal to or greater than those found in indoor environments have not resulted in hematologic or immunologic abnormalities. These tests include: blood count and differential, erythrocyte sedimentation rate; lymphocyte subpopulations (E-rosetting, T3, T4, T8, B73.1, Fc receptor positive lymphocytes and large granular lymphocytes); lymphocyte response to phytohemagglutinin and formalin-treated red blood cells; serum antibody against the Thomsen-Friedenrich RBC antigen and against formalin-RBC; and natural killer, interferon-boosted natural killer, and antibody-dependent cell-mediated cytotoxicity (Pross et al., 1987). While six of the studies cited above reported decrements in lung function associated with short-term formaldehyde exposure among at least some of the asthmatic subjects, a number of other exposure studies of patients with asthma have failed to demonstrate that exposure to formaldehyde results in onset or aggravation of the patients' asthmatic symptoms (Sheppard et al., 1984; Sauder et al., 1987; Harving et al., 1990; Krakowiak et al., 1998).

The effects of formaldehyde on asthmatics may be dependent on previous, repeated exposure to formaldehyde. Burge et al. (1985) found that 3 out of 15 occupationally exposed workers challenged with formaldehyde vapors at concentrations from 1.5 ppm to 20.6 ppm for brief durations exhibited late asthmatic reactions. Six other subjects had immediate asthmatic reactions likely due to irritant effects. Asthmatic responses (decreased PEF, FVC, and FEV₁) were observed in 12 occupationally-exposed workers challenged with 2.0 ppm (2.5 mg/m³) formaldehyde (Nordman et al., 1985). Similarly, asthmatic responses were observed in 5 of 28 hemodialysis workers occupationally exposed to formalin and challenged with formaldehyde vapors (concentration not measured) (Hendrick and Lane, 1977). In asthmatics not occupationally exposed to formaldehyde, Sheppard et al. (1984) found that a 10-minute challenge with 3 ppm formaldehyde coupled with moderate exercise did not induce significant changes in airway resistance or thoracic gas volume.

Gorski et al. (1992) evaluated the production of active oxygen species by neutrophils in 18 persons exposed to 0.5 mg/m³ formaldehyde for 2 hours. All 13 subjects who had allergic contact dermatitis (tested positive to formaldehyde in skin patch) exhibited significantly higher chemiluminescence of granulocytes isolated from whole blood 30 minutes and 24 hours post-exposure than the individuals who were not formaldehyde sensitive. Thus, the immune cellular response of skin-sensitized individuals to an inhalation exposure to formaldehyde indicates increased production of active oxygen species. This is consistent with increasing evidence that

endogenous or exogenous reactive oxygen and reactive nitrogen species are responsible for the airway inflammation of asthma (Sugiura and Ichinose, 2008).

In addition to its effects on the respiratory tract, the irritant properties of formaldehyde also manifest as ocular irritation. In an anatomy dissecting laboratory, formaldehyde levels were found to peak at 0.62 ppm, with a gradual decrease to 0.11 ppm. Formaldehyde-related irritation of the eyes, nose, throat, airways and skin was reported by 59% of the students. These effects were significantly ($p < 0.001$) higher among wearers of contact lenses compared with students without glasses or wearing glasses (Tanaka et al., 2003). The ability of contact lenses to trap and concentrate volatile compounds, and to extend the exposure time by limiting the eye's normal self-cleansing, may make contact lens wearers more susceptible to ocular exposure and irritation by formaldehyde.

5.2 Acute Toxicity to Infants and Children

No studies of the effects of acute exposure to formaldehyde in children or young experimental animals were located. However, as noted above for adults, there is evidence that following acute exposure to formaldehyde, asthmatics and others previously sensitized to formaldehyde may be more likely to show respiratory symptoms such as wheezing, shortness of breath, rhinitis, and/or decrements in pulmonary function consistent with immediate and/or delayed bronchoconstriction (Nordman et al., 1985; Burge et al., 1985; Hendrick and Lane, 1977; Wallenstein et al., 1978). Furthermore, some asthmatics may respond with significant reductions in lung function due to the irritant effects on asthma, sensitized or not. Additionally, the depletion of the endogenous bronchodilator, GSNO, following formaldehyde exposure may be particularly important in children. Gaston et al. (1998) compared concentrations of tracheal S-nitrosothiol concentrations in eight asthmatic children in respiratory failure with those of 21 non-asthmatic children undergoing elective surgery. In asthmatic children, the metabolism of GSNO was accelerated and the mean S-nitrosothiol concentrations significantly lower compared to normal children (65 ± 45 vs 502 ± 429 nmol/l). Thus asthmatic children, with low levels of GSNO, are expected to be unusually vulnerable to any further depletion of GSNO caused by formaldehyde.

The potential association between formaldehyde exposure and asthma is of special concern for children since, as noted in OEHHA (2001): "*OEHHA considers asthma to impact children more than adults. Children have higher prevalence rates of asthma than do adults (Mannino et al., 1998). In addition, asthma episodes can be more severe due to the smaller airways of children, and result in more hospitalizations in children, particularly from the ages of 0 to 4 years, than in adults (Mannino et al., 1998).*" Thus children, particularly asthmatic children, may be at greater risk from acute exposure to formaldehyde.

5.3 Acute Toxicity to Experimental Animals

Acute exposures of experimental animals to formaldehyde are associated with changes in pulmonary function (decreased respiratory rate, increased airway reactivity and resistance) at low concentrations, while pulmonary edema and death have been reported at high concentrations. Neurochemical and neurobehavioral changes have also been observed.

In 72 rats exposed to approximately 600-1,700 mg/m³ (500-1,400 ppm) formaldehyde vapor for 30 minutes, the LC₅₀ was found to be 1,000 mg/m³ (800 ppm) (Skog, 1950). The first deaths did not occur until 6 hours after cessation of exposure. Respiratory difficulty lasted several days after exposure and the last of 49 rats died after 15 days of purulent bronchitis and diffuse bronchopneumonia. Three weeks following exposure, histological examinations of the 23 surviving animals revealed bronchitis, pulmonary microhemorrhages, and edema. No changes were seen in other organs.

A multispecies study by Salem and Cullumbine (1960) showed that a 10-hour exposure to 15.4 ppm (19 mg/m³) formaldehyde vapor killed 3 out of 5 rabbits, 8 of 20 guinea pigs, and 17 of 50 mice. The report stated that formaldehyde exposure resulted in delayed lethality.

Alarie (1981) determined the 10 minute LC₅₀ for formaldehyde in mice to be 2,162 ppm (95% confidence interval, 1,687-2,770 ppm). The post-exposure observation period was 3 hours. From the concentration mortality graph provided in the report, an MLE₀₅ and BC₀₅ of 1,440 ppm and 778 ppm, respectively, could be estimated for a 10-minute formaldehyde exposure. However, as indicated in the previous reports, delayed deaths occur with formaldehyde which suggests that the 3-hour post-exposure observation period used in this study may not have been long enough.

In other lethality studies, Nagorny et al. (1979) determined a 4-hour formaldehyde LC₅₀ in rats and mice to be 588 mg/m³ (474 ppm) and 505 mg/m³ (407 ppm), respectively. However, the raw data for this study were not included in the report. Horton et al. (1963) observed that a 2-hour exposure of mice to 0.9 mg/l (900 mg/m³) formaldehyde resulted in deaths from massive pulmonary hemorrhage and edema, but a 2 hour exposure to 0.14 mg/l (140 mg/m³) did not produce signs of "substantial distress."

Swiecichowski et al., (1993) exposed groups of five to seven guinea pigs to 0.86, 3.4, 9.4, 31.1 ppm (1.1, 4.2, 11.6, 38.6 mg/m³) formaldehyde for 2 hours, or to 0.11, 0.31, 0.59, 1.05 ppm (0.14, 0.38, 0.73, 1.30 mg/m³) formaldehyde for 8 hours. An 8-hour exposure to levels greater than or equal to 0.3 ppm (≥ 0.4 mg/m³) formaldehyde was sufficient to produce a significant increase in airway reactivity. Similar effects occurred after greater than 9 ppm (> 11 mg/m³) formaldehyde for the 2-hour exposure group. Formaldehyde exposure also heightened airway smooth muscle responsiveness to acetylcholine (or carbachol) *ex vivo*. No inflammation or epithelial damage was seen up to 4 days after exposure. The researchers suggest that duration of exposure is important to the induction of airway hyperreactivity and that prolonged (8-hour), low-level exposures may generate abnormal physiologic responses in the airways not detectable after acute (2-hour) exposures.

Male F-344 rats, 7-9 weeks old, were exposed to 0.5, 2, 6 or 15 ppm formaldehyde for 6 hours per day for 1 to 4 days (Monteiro-Riviere and Popp, 1986). Effects noted in the rat nasal respiratory epithelium with 0.5 or 2 ppm were limited to altered cilia with occasional wing-like projections on the ends of the ciliary shafts. Effects noted at 6 ppm for 1 day were autophagic vacuoles in some basal cells, neutrophils in the basal and suprabasal layers, and hypertrophy of goblet and ciliated cells. Loss of microvilli in ciliated cells was noted at all exposure concentrations.

Rats were exposed to 0, 5, 10 or 20 ppm formaldehyde for 3 hours per day on 2 consecutive days (Boja et al., 1985). Decreased motor activity and neurochemical changes in dopamine and 5-hydroxytryptamine neurons were reported.

The effects of formaldehyde inhalation on open-field behavior in mice were examined by Malek et al. (2004) 2 and 24 hours after a single 2-hour exposure to 0, 1.1, 2.3 or 5.2 ppm. Two hours after exposure there were significant decreases in rearing and in several measures of exploratory behavior, with evidence of dose-dependence in all dose groups compared with controls. At 24 hours, there were still significant differences between dosed and control mice but the dose-dependence was no longer evident.

Nielson et al. (1999) analyzed the breathing patterns of Balb/c mice exposed to 0.2-13 ppm formaldehyde and found a concentration-dependent decrease in respiratory rate of 32.9%/log concentration. In the range of 0.3-4.0 ppm, the decrease in respiratory rates was attributable to sensory irritation. Above 4.0 ppm, bronchoconstriction also contributed to the decreased breathing rate. The authors suggest a NOEL of 0.3 ppm for these effects in mice.

Amdur (1960) exposed groups of 4 to 18 guinea pigs to formaldehyde at 0.05, 0.31, 0.58, 1.22, 3.6, 11.0, or 49 ppm formaldehyde for one hour. Resistance to flow and lung compliance were calculated from measures of intrapleural pressure, tidal volume, and rate of flow to the lungs at the end of exposure and one hour later. Resistance and compliance were significantly different from the control level for the 0.31 ppm exposure ($p < 0.05$) and increasingly significant at higher concentrations. One hour later, only the 49 ppm exposure remained significant ($p < 0.01$). In addition, the tracheas of groups of 6 to 10 guinea pigs were cannulated and exposed for one hour to 0.90, 5.2, 20, or 50 ppm formaldehyde, and 1.14 or 3.6 ppm formaldehyde with 10 mg/m³ sodium chloride. With the protective effect of the trachea bypassed, the resistance and compliance changed substantially. The addition of sodium chloride further enhanced the effect, including a significant effect after one hour for the 1.14 ppm formaldehyde exposure. These results show that formaldehyde that reaches the lungs has a marked effect on airways resistance and compliance in addition to an effect on the upper airways.

Riedel et al. (1996) studied the influence of formaldehyde exposure on allergic sensitization in guinea pigs. Three groups of guinea pigs (12/group) were exposed to clean air or two different formaldehyde concentrations (0.13 and 0.25 ppm) over five consecutive days. Following exposure, the animals were sensitized to allergen by inhalation of 0.5% ovalbumin (OA). Three weeks later the animals were subjected to bronchial provocation with OA and specific anti-OA-IgG1 (reaginic) antibodies in serum were measured. In another group of six animals, the respiratory tract was examined histologically for signs of inflammation directly after the end of formaldehyde or clean air exposure. In the group exposed to 0.25 ppm formaldehyde, 10/12

animals were found to be sensitized to OA (positive reaction on specific provocation) vs. 3/12 animals in the control group ($P < 0.01$). Furthermore, compressed air measurements of specific bronchial provocation and serum anti-OA-antibodies were significantly higher in the 0.25 ppm formaldehyde group than in controls. The median for compressed air measurement was 0.35 ml for the formaldehyde-exposed group vs. 0.09 ml for the controls ($p < 0.01$), indicating increased bronchial obstruction. The median for the anti-OA-IgG1 measured in the formaldehyde-exposed group was 13 vs. less than 10 EU in the controls, ($p < 0.05$), indicating enhanced sensitization. In the group exposed to 0.13 ppm formaldehyde, no significant difference was found compared to the control group. Histological examination found edema of the bronchial mucosa, but there was no sign of inflammation of the lower airways in formaldehyde-exposed guinea pigs. The investigators concluded that short-term exposure to a low concentration of formaldehyde (0.25 ppm) can significantly enhance sensitization to inhaled allergens in the guinea pig.

As described in Section 5, the main formaldehyde-metabolizing enzyme, ADH3, also reduces the endogenous bronchodilator GSNO. To examine the role of GSNO and ADH3 (known in this study as GSNO reductase, GSNOR) in airway tone and asthma, Que et al. (2005) used wild type mice and mice with a targeted deletion of GSNOR (GSNOR^{-/-}). Following a challenge with allergen (ovalbumin), GSNOR activity in bronchoalveolar lavage fluid from wild type mice increased significantly ($p < 0.05$) compared to buffer (PBS) controls, while as expected, no GSNOR activity was detected in the GSNOR^{-/-} mice with either treatment. Levels of S-nitrosothiols (SNO) were assayed in homogenates of lung tissues from both types of mouse and found to be barely detectable with PBS treatment. However, after ovalbumin challenge, SNO levels were significantly higher ($p < 0.02$) in GSNOR^{-/-} mice compared to wild type, indicating metabolism of SNOs by GSNOR under “asthmatic-like” conditions in wild type mice. Metabolism of GSNO results in a loss of bronchodilation capacity. Deletion of GSNOR had no effect on NO generation by NO synthase as there were no differences between wild type and GSNOR^{-/-} mice in nitrate or nitrite levels regardless of treatment. To investigate the effects of deletion of GSNOR on airway hyper-responsiveness, pulmonary resistance was measured at baseline (PBS) and after methacholine challenge, with and without ovalbumin treatment. At baseline, there was no difference among mouse types and treatments, while at higher methacholine levels (100-1000 $\mu\text{g}/\text{kg}$), pulmonary resistance was found to be significantly lower ($p < 0.001$) in GSNOR^{-/-} mice than in wild type, presumably due to higher GSNO levels that enhance bronchodilation. Importantly, ovalbumin caused a marked increase in airway responsiveness in wild type mice but had little effect in GSNOR^{-/-} mice. This indicates that GSNOR regulates basal airway tone as well as hyper-responsiveness to both allergen challenge and bronchoconstrictor agonists. It is also noteworthy that the total number and composition of leukocytes, levels of interleukin-13 and total serum IgE were comparable between wild type and GSNOR^{-/-} mice. This indicates that protection from asthma in the GSNOR^{-/-} mice is not a result of a suppressed response to allergen, and that SNOs, especially GSNO, can preserve airway patency in the presence of inflammation. Thus the inflammatory response is not linked to hyper-responsiveness as long as adequate levels of GSNO are maintained.

A connection between formaldehyde and the activity of GSNOR described in the study above by Que et al., was outlined by Thompson and Grafstrom (2008) and supported by Yi et al. (2007) and Staab et al. (2008). In the study by Yi and associates, groups of 6 mice were exposed to formaldehyde at 0, 1, or 3 mg/m^3 continuously for 72 hours. Following exposure, lungs were isolated to allow measurement of GSNOR mRNA levels by RT-PCR, and enzymatic activity

with GSNO. Formaldehyde at 3 mg/m³ significantly increased the numbers of GSNOR transcripts compared to control (0.58 vs 0.4 GSNOR/ β actin; $p < 0.05$), while GSNOR reduction of GSNO showed a significant dose-dependent increase with formaldehyde concentration ($p < 0.01$). The stimulation of GSNO reduction by formaldehyde was also observed by Staab et al. (2008) in an in vitro study using recombinant human GSNOR. In this study, GSNO levels in buccal carcinoma cells were reduced in a dose-dependent fashion following a 1 hour exposure to formaldehyde in the 1-5 mM range with significance at 5 mM ($p < 0.05$). The results from this study support a model in which formaldehyde (as the glutathione conjugate, HMGSH) is oxidized by GSNOR (ADH3) in the presence of high levels of NAD⁺, producing NADH. This process was found to be accelerated by high levels of GSNO. GSNO is in turn reduced with the oxidation of NADH to form glutathione sulfonimide. Formaldehyde thus depletes cellular SNO (in the form of GSNO) which results in dysregulation of NO signaling pathways.

6. Chronic Toxicity of Formaldehyde

6.1 Chronic Toxicity to Adult Humans

Formaldehyde primarily affects the mucous membranes of the upper airways and eyes. Exposed populations that have been studied include embalmers, residents in houses insulated with urea-formaldehyde foam, anatomy class students, histology technicians, wood and pulpmill workers, and asthmatics. A number of studies describing these effects have been briefly summarized below. For the sake of brevity, only the studies that best represent the given effects are presented. Formaldehyde is also a recognized carcinogen (IARC, 2006), however, this document will address only its non-carcinogenic properties.

In the study chosen for determination of the 8-hour and chronic RELs, nasal obstruction and discharge, and frequency of cough, wheezing, and symptoms of bronchitis were reported in 66 workers in a formaldehyde production plant exposed for 1-36 years (mean = 10 years) to a mean concentration of 0.21 ppm (0.26 mg/m³) formaldehyde (Wilhelmsson and Holmstrom, 1992). All workers were exposed almost exclusively to formaldehyde, the concentrations of which were measured in the ambient air of the worksite with personal sampling equipment. Referents consisted of 36 office workers in a government office with exposure to a mean concentration of 0.07 ppm (0.09 mg/m³) formaldehyde, and no industrial solvent or dust exposure. Symptom data, collected by questionnaire, were separated into general and work-related, and allowed identification of individuals with atopy and mucosal hyperreactivity. The critical effects from chronic exposure to formaldehyde in this study included nasal obstruction, lower airway discomfort, and eczema or itching. The frequency of reported lower airway discomfort (intermittent cough, wheezing, or symptoms of chronic bronchitis) was significantly higher among formaldehyde-exposed vs non-exposed workers (44 vs 14%; $p < 0.01$) (Table 6.1). Work-related nasal discomfort also was significantly higher in the formaldehyde group (53%) compared with the referent group (3%; $p < 0.001$). Similarly, work-related eye discomfort was 20% in the formaldehyde group but nonexistent among referents. The significant increase in symptoms of nasal discomfort in exposed workers did not correlate with total serum IgE antibody levels. However, two exposed workers, who complained of nasal discomfort, had elevated IgE levels. The investigators concluded that formaldehyde can induce nonspecific nasal hypersensitivity.

TABLE 6.1.1 SYMPTOMS OF FORMALDEHYDE EXPOSURE VS REFERENCE GROUP**(FROM WILHELMSSON AND HOLMSTROM, 1992)**

	Formaldehyde	Reference	Rate difference	
	% (n=66)	% (n=36)	%	95% CI
General nasal discomfort	67	25	42	24-60
Workplace nasal discomfort	53	3	50	37-63
General lower airway discomfort	44	14	30	14-47
Workplace lower airway discomfort	33	3	28	15-40
General eye discomfort	24	6	18	6-36
General skin discomfort	36	11	25	10-41

In a cross-sectional study supportive of these results, Edling et al. (1988) reported histopathological changes in nasal mucosa of workers (n=75) occupationally exposed to formaldehyde (one wood laminating plant) or formaldehyde plus wood dust (two particle board plants). Ambient formaldehyde measurements in these three composite wood processing plants between 1975 and 1983 gave a time-weighted average (TWA) of 0.1-1.1 mg/m³ (0.08- 0.89 ppm) with peaks of up to 5 mg/m³ (4 ppm). The exposed workers were compared on the basis of medical and work histories, clinical examinations and nasal biopsies to 25 workers selected with regard to age and smoking habits but without occupational formaldehyde exposure.

Based on the histories, there was a high frequency of eye and upper airway symptoms among workers. Nasal symptoms (running nose and crusting) associated with formaldehyde exposure were reported in 60% of the workers, while 75% complained of lacrimation. Clinical examinations revealed grossly normal nasal mucosa in 75% of the cases while 25% had swollen or dry changes, or both, to the nasal mucosa. Histological examination (Table 6.2) revealed that only 3 of the 75 formaldehyde-exposed workers had normal, ciliated pseudostratified epithelium. Squamous metaplasia was reportedly observed in 59, while 6 showed mild dysplasia, and in 8 there was loss of ciliated cells and goblet cell hyperplasia. The histological grading showed a significantly higher score for nasal lesions among workers with formaldehyde exposure when compared with the referents (2.9 versus 1.8; p < 0.05). Exposed smokers had a higher, but non-significant, score than ex-smokers and non-smokers.

While the mean exposure time was 10.5 years (range 1-39 yr), there was no discernable difference among histology scores as a function of years of employment. The histology scores were also not different between workers in the particle board plants, exposed to both formaldehyde and wood dust, and workers in the laminate plant with exposure only to formaldehyde. The authors thus attribute the pathological changes in the nasal mucosa and the other adverse effects to formaldehyde alone in the 0.1-1.1 mg/m³ range.

TABLE 6.1.2 DISTRIBUTION OF HISTOLOGICAL CHARACTERISTICS ASSOCIATED WITH FORMALDEHYDE EXPOSURE (FROM EDLING ET AL., 1988)

Histological characteristic	Grading score	Point score	Workers	%
Normal respiratory epithelium	0	0	3	4
Loss of ciliated cells	1	1	8	11
Mixed cuboidal/squamous epithelium, metaplasia	2	2	24	32
Stratified squamous epithelium	3	3	18	24
Keratosi	4	4	16	21
Budding of epithelium	add 1	5	0	0
Mild or moderate dysplasia	6	6	6	8
Severe dysplasia	7	7	0	0
Carcinoma	8	8	0	0

Histological changes in the nasal mucosa of formaldehyde-exposed workers were also reported by Boysen et al. (1990). In this study, nasal biopses were collected from 37 workers with 5 or more years of occupational formaldehyde exposure (0.5 - > 2 ppm) and compared with age-matched, unexposed controls who otherwise had similar environmental exposures and smoking habits. Histological changes in the nasal epithelium were scored as indicated in Table 6.1.3.

TABLE 6.1.3 TYPES OF NASAL EPITHELIA AND SCORING (FROM BOYSEN ET AL., 1990)

Types of epithelia	Histological score
Pseudostratified columnar	0
Stratified cuboidal	1
Mixed stratified cuboidal/stratified squamous	2
Stratified squamous, non-keratinizing	3
Stratified squamous, keratinizing	4
Dysplasia	5

As shown by the histological scoring in Table 6.1.4 below, metaplastic changes in the nasal epithelium were more pronounced in the formaldehyde-exposed workers although this difference did not reach statistical significance.

TABLE 6.1.4 HISTOLOGICAL SCORES OF NASAL EPITHELIA

	No	Histological score						Mean
		0	1	2	3	4	5	
Exposed	37	3	16	5	9	1	3	1.9
Controls	37	5	17	10	5	0	0	1.4

Rhinoscopic examination revealed hyperplastic nasal mucosa in 9 of 37 formaldehyde-exposed workers but in only 4 of the controls. In addition, the incidence of subjective nasal complaints was significantly ($p < 0.01$) higher in the exposed group. While the small size of this study, and the small amount of the nasal mucosa accessible to biopsy limited its ability to detect formaldehyde-related histopathology, the results are consistent with the histopathologies reported by Edling et al. above.

In another occupational health study (Grammer et al., 1990), 37 workers, who were exposed for an unspecified duration to formaldehyde concentrations in the range of 0.003 to 0.073 ppm, reported ocular irritation. However, no significant serum levels of IgE or IgG antibodies to formaldehyde-human serum albumin were detected.

Kerfoot and Mooney (1975) reported that estimated formaldehyde exposures of 0.25-1.39 ppm evoked numerous complaints of upper respiratory tract and eye irritation among seven embalmers at six different funeral homes. Three of the seven embalmers in this study reportedly had asthma. Levine et al. (1984) examined the death certificates of 1477 Ontario undertakers. Exposure measurements taken from a group of West Virginia embalmers were used as exposure estimates for the embalming process, ranging from 0.3-0.9 ppm (average 1-hour exposure) and 0.4-2.1 ppm (peak 30-minute exposure). Mortality due to non-malignant diseases was significantly elevated due to a two-fold excess of deaths related to the digestive system. The authors suggest increased alcoholism could have contributed to this increase.

Ritchie and Lehnen (1987) reported a dose-dependent increase in health complaints (eye and throat irritation, and headaches) in 2000 residents living in 397 mobile and 494 conventional homes. Complaints of symptoms of irritation were noted at concentrations of 0.1 ppm formaldehyde or above. Similarly, Liu et al. (1991) found that exposure to 0.09 ppm (0.135 mg/m³) formaldehyde exacerbated chronic respiratory and allergy problems in residents living in mobile homes.

Employees of mobile day-care centers (66 subjects) reported increased incidence of eye, nose and throat irritation, unnatural thirst, headaches, abnormal tiredness, menstrual disorders, and increased use of analgesics as compared to control workers (Olsen and Dossing, 1982). The mean formaldehyde concentration in these mobile units was 0.29 ppm (0.43 mg/m³) (range = 0.24 - 0.55 mg/m³). The exposed workers were exposed in these units for a minimum of 3 months. A control group of 26 subjects in different institutions was exposed to a mean concentration of 0.05 ppm (0.08 mg/m³) formaldehyde.

Occupants of houses insulated with urea-formaldehyde foam insulation (UFFI) (1726 subjects) were compared with control subjects (720 subjects) for subjective measures of irritation, measures of pulmonary function (FVC, FEV₁, FEF₂₅₋₇₅, FEF₅₀), nasal airway resistance, odor threshold for pyridine, nasal cytology, and hypersensitivity skin-patch testing (Broder et al., 1988). The mean length of time of exposure to UFFI was 4.6 years. The mean concentration of formaldehyde in the UFFI-exposed group was 0.043 ppm, compared with 0.035 ppm for the controls. A significant increase in symptoms of eye, nose and throat irritation was observed in subjects from UFFI homes, compared with controls. No other differences from control measurements were observed.

Alexandersson and Hedenstierna (1989) evaluated symptoms of irritation, spirometry, and immunoglobulin levels in 34 wood workers exposed to formaldehyde over a four-year period. Exposure to 0.4 - 0.5 ppm formaldehyde resulted in significant decreases in FVC, FEV₁, and FEF₂₅₋₇₅. Removal from exposure for four weeks allowed for normalization of lung function in the non-smokers.

Kriebel et al. (2001) conducted a subchronic epidemiological study of 38 anatomy class students who, on average, were exposed to a geometric mean of 0.70 ± 2.13 ppm formaldehyde for two hours per week over fourteen weeks. After class, eye, nose and throat irritation was significantly elevated compared with pre-laboratory session exposures, with a one unit increase in symptom intensity/ppm formaldehyde. Peak respiratory flow (PEF) was found to decrease by 1%/ppm formaldehyde during the most recent exposure. Changes in PEF and symptom intensity following formaldehyde exposure were most pronounced during the first week of the semester but attenuated with time, suggesting partial acclimatization.

Histology technicians (280 subjects) were shown to have reduced pulmonary function, as measured by FVC, FEV₁, FEF₂₅₋₇₅, and FEF₇₅₋₈₅, compared with 486 controls (Kilburn et al., 1989). The range of formaldehyde concentrations was 0.2 - 1.9 ppm, volatilized from formalin preservative solution.

Malaka and Kodama (1990) investigated the effects of formaldehyde exposure in plywood workers (93 exposed, 93 controls) exposed for 26.6 years, on average, to 1.13 ppm (range = 0.28 - 3.48 ppm). Fifty-three smokers were present in both exposed and control groups. Exposure assessment was divided into three categories: high (> 5 ppm), low (< 5 ppm), and none (reference group). Subjective irritation and pulmonary function tests were performed on each subject, and chest x-rays were taken of ten randomly selected volunteers from each group. Respiratory symptoms of irritation were found to be significantly increased in exposed individuals, compared with controls. In addition, exposed individuals exhibited significantly reduced FEV₁, FEV₁/FVC, and forced expiratory flow rate at 25% through 75% of FVC (FEF₂₅₋₇₅), compared with controls. Forced vital capacity was not significantly reduced. Pulmonary function was not found to be different after a work shift, compared to the same measurement taken before the shift. No differences in chest x-rays were observed between exposed and control workers.

Occupational exposure to formaldehyde concentrations estimated to be 0.025 ppm (0.038 mg/m³) for greater than six years resulted in complaints by 22 exposed workers of respiratory, gastrointestinal, musculoskeletal, and cardiovascular problems, and in elevated formic acid excretion in the urine (Srivastava et al., 1992). A control group of twenty seven workers unexposed to formaldehyde was used for comparison. A significantly higher incidence of abnormal chest x-rays was also observed in formaldehyde-exposed workers compared with controls.

Chemical plant workers (70 subjects) were exposed to a mean of 0.17 ppm (0.26 mg/m³) formaldehyde for an unspecified duration (Holmstrom and Wilhelmsson, 1988). Compared with 36 control workers not exposed to formaldehyde, the exposed subjects exhibited a higher frequency of eye, nose, and deep airway discomfort. In addition, the exposed subjects had diminished olfactory ability, delayed mucociliary clearance, and decreased FVC.

Alexandersson et al. (1982) compared the irritant symptoms and pulmonary function of 47 carpentry workers exposed to a mean concentration of formaldehyde of 0.36 ppm (range = 0.04 - 1.25 ppm) with 20 unexposed controls. The average length of employment for the exposed workers was 5.9 years. Symptoms of eye and throat irritation as well as airway obstruction were more common in exposed workers. In addition, a significant reduction in FEV₁, FEV₁/FVC, and MMF was observed in exposed workers compared with controls.

Horvath et al. (1988) compared subjective irritation and pulmonary function in 109 workers exposed to formaldehyde with similar measures in a control group of 254 subjects. The formaldehyde concentrations for the exposed and control groups were 0.69 ppm (1.04 mg/m³) and 0.05 ppm (0.08 mg/m³), respectively. Mean formaldehyde concentration in the pre-shift testing facility and the state (Wisconsin) ambient outdoor - formaldehyde level were both 0.04 ppm (0.06 mg/m³). Duration of formaldehyde exposure was not stated. Subjects were evaluated pre- and post work-shift and compared with control subjects. Significant differences in symptoms of irritation, FEV₁, FEV₁/FVC ratio, FEF₅₀, FEF₂₅, and FEF₇₅ were found when comparing exposed subjects' pre- and post work-shift values. However, the pre-workshift values were not different from controls.

The binding of formaldehyde to endogenous proteins creates haptens that can elicit an immune response. Chronic exposure to formaldehyde has been associated with immunological hypersensitivity as measured by elevated circulating IgG and IgE autoantibodies to human serum albumin (Thrasher et al., 1987). In addition, a decrease in the proportion of T-cells was observed, indicating altered immunity. Thrasher et al. (1990) later found that long-term exposure to formaldehyde was associated with autoantibodies, immune activation, and formaldehyde-albumin adducts in patients occupationally exposed, or residents of mobile homes or of homes containing particleboard sub-flooring. The authors suggest that the hypersensitivity induced by formaldehyde may account for a mechanism for asthma and other health complaints associated with formaldehyde exposure.

An epidemiological study of the effects of formaldehyde on 367 textile and shoe manufacturing workers employed for a mean duration of 12 years showed no significant association between formaldehyde exposure, pulmonary function (FVC, FEV₁, and PEF) in normal or asthmatic workers, and occurrence of specific IgE antibodies to formaldehyde (Gorski and Krakowiak, 1991). The concentrations of formaldehyde did not exceed 0.5 ppm (0.75 mg/m³).

Workers (38 total) exposed for a mean duration of 7.8 years to 0.11 - 2.12 ppm (mean = 0.33 ppm) formaldehyde were studied for their symptomatology, lung function, and total IgG and IgE levels in the serum (Alexandersson and Hedenstierna, 1988). The control group consisted of 18 unexposed individuals. Significant decrements in pulmonary function, FVC (p < 0.01) and FEV₁ (p < 0.05) were observed, compared with the controls. Eye, nose, and throat irritation was also reported more frequently by the exposed group. No correlation was found between duration of exposure, or formaldehyde concentration, and the presence of IgE and IgG antibodies.

As described in section 5.1, chronic or repeated exposure to formaldehyde may influence the response of asthmatics to acute or short-term challenges. In the study by Burge et al. (1985) late asthmatic reactions were noted in 3 out of 15 occupationally exposed workers after short-duration exposure to 1.5 – 20.6 ppm formaldehyde. Similarly, among workers with occupational

exposure to formaldehyde, asthmatic responses (decreased PEF, FVC, and FEV₁) were reported in 12 workers challenged with 1.67 ppm (2.5 mg/m³) formaldehyde (Nordman et al., 1985) and in 5 of 28 hemodialysis workers following challenge with formaldehyde vapors (concentration not measured) (Hendrick and Lane, 1977). In contrast, Sheppard et al. (1984) found that in asthmatics not occupationally exposed to formaldehyde, a 10-minute challenge with 3 ppm formaldehyde coupled with moderate exercise did not induce significant changes in airway resistance or thoracic gas volume. Thus individuals with chronic formaldehyde exposure may be at greater risk for adverse responses to acute exposures. These individuals may have been sensitized immunologically, as in the cases of elevated circulating antibodies, or rendered neurologically hyperresponsive, following repeated or chronic exposures to formaldehyde (Sorg et al., 2001a,b).

6.2 Chronic Toxicity to Infants and Children

There are few studies that compare the effects of chronic formaldehyde exposure on children versus adults. Among those that do there is evidence that children are more susceptible to the adverse effects of chronic exposure. Krzyzanowski et al. (1990) assessed chronic pulmonary symptoms and function in 298 children (6-15 years of age) and 613 adults (> 15 years of age) in relation to measured formaldehyde levels in their homes. Information on pulmonary symptoms and doctor-diagnosed asthma and chronic bronchitis was collected by questionnaire. Pulmonary function was assessed as peak expiratory flow rates (PEFR) measured up to four times a day. The prevalence of chronic respiratory symptoms in children was not related to formaldehyde levels measured in tertiles (< 40, 41-60, > 60 ppb). However, doctor-diagnosed asthma and chronic bronchitis were more prevalent in houses with elevated formaldehyde (p for trend < 0.02). This effect was driven by the high disease prevalence observed in homes with kitchen formaldehyde levels >60 ppb, and was especially pronounced among children with concomitant exposure to environmental tobacco smoke (Table 6.2.1). By comparison, in adults, while the prevalence rates of chronic cough and wheeze were somewhat higher in houses with higher formaldehyde, none of the respiratory symptoms or diseases was significantly related to formaldehyde levels.

TABLE 6.2.1 PREVALENCE RATE (PER 100) OF DIAGNOSED BRONCHITIS AND ASTHMA IN CHILDREN WITH FORMALDEHYDE (FROM KRZYZANOWSKI ET AL., 1990)

	Formaldehyde (ppb)			P value X^2 trend
	≤ 40 (N)	41-60 (N)	>60 (N)	
Bronchitis				
Household mean	3.5 (258)	17.2 (29)	9.1 (11)	<0.02
Main room mean	3.2 (253)	15.6 (32)	9.1 (11)	<0.01
Bedroom mean	3.8 (262)	16.0 (25)	9.1 (11)	<0.04
Subject's bedroom	4.7 (256)	6.7 (30)	11.1 (9)	>0.35
Kitchen	3.5 (255)	0 (22)	28.6 (21)	<0.001
No ETS	4.3 (141)	0 (12)	10.0 (10)	>0.40
ETS	1.9 (106)	0 (10)	45.5 (11)	<0.001
Asthma				
All children	11.7 (256)	4.2 (24)	23.8 (21)	<0.03
No ETS	8.5 (142)	8.3 (12)	0 (10)	>0.50
ETS	15.1 (106)	0 (12)	45.5 (11)	<0.05

In a random effects model, Krzyzanowski et al. (1990) reported that lung function (PEFR) in children, but not adults, was significantly decreased by formaldehyde (coefficient \pm SE: -1.28 ± 0.46 vs 0.09 ± 0.27). Measurements of PEFR in the morning suggested that children with asthma ($n = 4$) were more severely affected than healthy children (coefficient \pm SE: -1.45 ± 0.53 vs 0.09 ± 0.15) (Table 6.2.2). Compared to children, the effects of formaldehyde on pulmonary function in adults were smaller, transient, limited to morning measurements, and generally most pronounced among smokers exposed to the higher levels of formaldehyde. These studies suggest that children may be more susceptible to the effects of chronic formaldehyde exposure on lung function than are adults.

TABLE 6.2.2 RELATION OF PEFR (L/MIN) TO INDOOR FORMALDEHYDE

(from Krzyzanowski et al., 1990)

Factor	Child coefficient \pm SE	Adult coefficient \pm SE
HCHO house mean	-1.28 ± 0.46	0.09 ± 0.27
Morning vs bedtime	-6.10 ± 3.0	-5.90 ± 1.10
HCHO bdrm mean/morning	0.09 ± 0.15	-0.07 ± 0.04
HCHO bdrm mean/morning/asthma	-1.45 ± 0.53	

Among studies of children only, a case-control study by Rumchev et al. (2002) examined risk factors for asthma among young children (6 mo- 3 yr). Cases included children with clinically-diagnosed asthma, and controls were children of the same age group without such a diagnosis. Formaldehyde levels were measured in the homes, once in summer and once in winter. Questionnaires were used to assess potential risk factors for asthma and to collect parental reports of respiratory symptoms characteristic of asthma (cough, shortness of breath, wheeze, runny nose, trouble breathing, and hay fever) in their children. Formaldehyde levels were higher

in the homes of children exhibiting respiratory symptoms. Estimates of the relative risk for clinically-diagnosed asthma (odds ratios) were adjusted for measured indoor air pollutants, relative humidity, temperature, atopy, family history of asthma, age, gender, socioeconomic status, pets, smoke exposure, air conditioning, and gas appliances. Compared with children exposed to < 8 ppb, children in homes with formaldehyde levels > 49 ppb had a 39% higher risk of asthma ($p < 0.05$) after adjusting for common asthma risk factors.

Franklin et al. (2000) measured exhaled nitric oxide (eNO) levels in 224 children 6-13 years of age as an indicator of inflammation of the lower airways following chronic low-level formaldehyde exposure in the home. While there was no effect of formaldehyde on lung function measured by spirometry, eNO was significantly higher in children from homes with average formaldehyde levels ≥ 50 ppb compared with those from homes with levels ≤ 50 ppb (15.5 ppb eNO vs 8.7; $p = 0.02$).

Garrett et al. (1999) examined the association between formaldehyde levels at home (median $15.8 \mu\text{g}/\text{m}^3$; maximum $139 \mu\text{g}/\text{m}^3$) and atopy and allergic sensitization in 148 children, 7-14 years of age. The risk of atopy increased by 40% with each $10 \mu\text{g}/\text{m}^3$ increase in bedroom formaldehyde. Two measures of allergic sensitization to twelve common environmental allergens, the number of positive skin prick tests and maximum wheal size, both showed linear associations with increasing maximum formaldehyde exposure levels. After adjusting for parental asthma and allergy, there was no evidence of an association between asthma in the children and formaldehyde levels. However, these data do suggest that formaldehyde levels commonly found in homes can enhance sensitization of children to common aeroallergens.

Of the numerous, primarily occupational, studies in adults, the NOAEL and LOAEL are $17 \mu\text{g}/\text{m}^3$ (14 ppb) and $101 \mu\text{g}/\text{m}^3$ (81 ppb), respectively, after adjustment for exposure continuity. These values are based on data on nasal and eye irritation as observed in Wilhelmsson and Holstrom (1992), and histological lesions in the nasal cavity as documented in Edling et al. (1988). However, studies in children, including the Krzyzanowski study above, indicate adverse health impacts in children at concentrations as low as 30 ppb. Wantke et al. (1996) reported that formaldehyde-specific IgE and respiratory symptoms were reduced when children transferred from schools with formaldehyde concentrations of 43 to 75 ppb to schools with concentrations of 23 to 29 ppb. While these human studies are not entirely consistent with each other, and there is potential for confounding in each, nevertheless, taken together, they suggest that children may be more sensitive to formaldehyde toxicity than adults.

A potential role for formaldehyde, GSNOR and its metabolizing enzyme, GSNOR, in asthma is described in Section 5 above. The activity of GSNOR tends to be higher, and the levels of GSNOR lower, in the lungs of asthmatics compared to non-asthmatics. This connection prompted Wu et al. (2007) to investigate whether genetic variation in GSNOR is associated with childhood asthma and atopy. The study group included 532 children, aged 4 to 17 with clinically diagnosed asthma, and their parents. Seven single nucleotide polymorphisms (SNPs) in GSNOR were genotyped in DNA extracted from lymphocytes to examine the relationship between common haplotypes and asthma. Atopy was determined with skin prick tests using a collection of 25 aeroallergens. Two of the GSNOR SNPs were associated with increased risk of asthma, but none was associated with atopy. Whereas a lower risk for asthma was associated with one (RR 0.77; 95% CI 0.61-0.97) or two (RR 0.66; 95% CI 0.44-0.99) copies of the minor A allele of

SNP rs1154404, homozygosity for the major T allele of this SNP carried an increased risk of asthma. Homozygosity for the minor allele of SNP rs28730619 also carried an increased risk of asthma (RR 1.60; 95% CI 1.13-2.26; $p = 0.0077$). In the haplotype analysis, children with the most common GSNOR haplotype (GTCCG), that contained the major T allele of rs1154404 and the minor G allele of rs28730619, were at increased risk of childhood asthma. These results thus suggest that variants in GSNOR genotype influence childhood asthma susceptibility.

It should be noted that while term neonates have high levels of reduced glutathione in the fluid lining the lungs, these levels drop rapidly after birth. However, among premature infants, glutathione levels are typically substantially below those of term infants (Grigg et al., 1993) and adults (Reise et al., 1997). As a result of low levels of a critical component of formaldehyde metabolism, glutathione, these infants may be at increased risk from formaldehyde exposure.

6.3 Chronic Toxicity to Experimental Animals

Studies of the effects of chronic formaldehyde exposure in experimental animals tend to focus on lesions in the upper respiratory tract and the hyperplastic or metaplastic changes observed in the respiratory epithelium. Systemic effects, such as changes in body or organ weight, or blood chemistry, appear to be secondary to the effects of the olfactory irritation on feeding behavior. There is also evidence that repeated or long-term exposure to formaldehyde may cause neurologically-based hyperresponsiveness to formaldehyde (Sorg et al., 2001a) and altered expression of stress hormones (Sorg et al., 2001b).

In studies examining respiratory effects, Fischer-344 rats and B6C3F1 mice (120 animals/sex) were exposed to concentrations of 0, 2.0, 5.6, or 14.3 ppm formaldehyde vapor for 6 hours/day, 5 days/week for 24 months (Kerns et al., 1983). The exposure period was followed by up to six months of non-exposure. Interim sacrifices were conducted at 6, 12, 18, 24, 27, and 30 months. Both male and female rats in the 5.6 and 14.3 ppm groups demonstrated decreased body weights over the two-year period. At the 6 month sacrifice, the rats exposed to 14.3 ppm formaldehyde had non-neoplastic lesions of epithelial dysplasia in the nasal septum and turbinates. As the study progressed, epithelial dysplasia, squamous dysplasia, and mucopurulent rhinitis increased in severity and distribution in all exposure groups. In mice, cumulative survival decreased in males from 6 months to the end of the study. Serous rhinitis was detected at 6 months in the 14.3 ppm group of mice. Metaplastic and dysplastic changes were noted at 18 months in most rats in the 14.3 ppm group and in a few mice in the 5.6 ppm exposure group. By 24 months, the majority of mice in the 14.3 ppm group had metaplastic and dysplastic changes associated with serous rhinitis, in contrast to a few mice in the 5.6 ppm group and a few in the 2 ppm group (exact number not given).

Woutersen et al. (1989) exposed male Wistar rats (60 animals/group) 6 hours/day for 5 days/week to 0, 0.1, 1.0 and 10 ppm formaldehyde vapor for 28 months. Compound-related nasal lesions of the respiratory and olfactory epithelium were observed only in the 10 ppm group. In the respiratory epithelium, the lesions consisted of rhinitis, squamous metaplasia and basal cell/pseudoepithelial hyperplasia. In the olfactory region, the lesions included epithelial degeneration and rhinitis. No differences in behavior or mortality were noted among the various groups. However, growth retardation was observed in the 10 ppm group from day 14 onwards. In a parallel study, male Wistar rats were exposed to 0, 0.1, 1.0 and 10 ppm formaldehyde for 3

months followed by a 25-month observation period. Compound-related histopathological changes were found only in the noses of the 10 ppm group and comprised increased incidence of squamous metaplasia of the respiratory epithelium, and rhinitis.

In a chronic exposure study that primarily investigated aspects of nasal tumor development, Monticello et al. (1996) examined nasal cavities of male F-344 rats (0-10 ppm, 90 animals/group; 15 ppm, 147 animals) following exposure to 0, 0.7, 2, 6, 10, and 15 ppm formaldehyde for 6 hours/day, 5 days/week for 24 months. Treatment-related decreases in survival were apparent only in the 15 ppm group. Nasal lesions at the two highest doses included epithelial hypertrophy and hyperplasia, squamous metaplasia, and a mixed inflammatory cell infiltrate. Lesions in the 6 ppm group were minimal to absent and limited to focal squamous metaplasia in the anterior regions of the nasal cavity. No formaldehyde-induced lesions were observed in the 0.7 or 2 ppm groups.

Kamata et al. (1997) exposed 32 male F-344 rats/group to gaseous formaldehyde at 0, 0.3, 2, and 15 ppm 6 hours/day, 5 days/week for up to 28 weeks. A room control, non-exposed group was also included in the study. Five animals per group were randomly selected at the end of the 12, 18, and 24 months, and surviving animals at 28 months were sacrificed for full pathological evaluation. Behavioral effects related to sensory irritation were evident in the 15 ppm group. Significant decreases in food consumption, body weight and survival were also evident in this group. No exposure-related hematological findings were observed. Biochemical and organ weight examination revealed decreased triglyceride levels and absolute liver weights at the highest exposure, but was likely related to reduced food consumption. Abnormal histopathological findings were confined to the nasal cavity. Inflammatory cell infiltration, erosion or edema of the nasal cavity was evident in all groups, including controls. Significantly increased incidence of non-proliferative (squamous cell metaplasia without epithelial cell hyperplasia) and proliferative lesions (epithelial cell hyperplasia with squamous cell metaplasia) were observed in the nasal cavities beginning at 2 ppm. In the 0.3 ppm group, a non-significant increase in proliferative nasal lesions (4/20 animals) were observed in rats that were either sacrificed or died following the 18th month of exposure.

Rusch et al. (1983) exposed groups of 6 male cynomolgus monkeys, 20 male or female rats, and 10 male or female hamsters to 0, 0.2, 1.0, or 3.0 ppm (0, 0.24, 1.2, or 3.7 mg/m³) formaldehyde vapor for 22 hours/day, 7 days/week for 26 weeks. There was no treatment-related mortality during the study. In monkeys, the most significant findings were hoarseness, congestion and squamous metaplasia of the nasal turbinates in 6/6 monkeys exposed to 2.95 ppm. There were no signs of toxicity in the lower exposure groups. In the rat, squamous metaplasia and basal cell hyperplasia of the nasal epithelia were significantly increased in rats exposed to 2.95 ppm. The same group exhibited decreased body weights and decreased liver weights. In contrast to monkeys and rats, hamsters did not show any signs of response to exposure, even at 2.95 ppm.

Kimbell et al. (1997) exposed male F-344 rats (≤ 6 /group) to 0, 0.7, 2, 6, 10, and 15 ppm 6 hr/day, 5 days/week for 6 months. Squamous metaplasia was not observed in any regions of the nasal cavity in any of the control, 0.7, or 2 ppm groups. However, the extent and incidence of squamous metaplasia in the nasal cavity increased with increasing dose beginning at 6 ppm.

In subchronic studies, Wilmer et al. (1989) found that intermittent (8 hours/day, 5 days/week) exposures of rats to 4 ppm formaldehyde for 13 weeks resulted in significant histological changes in the nasal septum and turbinates. In contrast, continuous exposure of rats for 13 weeks to 2 ppm formaldehyde did not produce significant lesions. This study revealed the concentration dependent nature of the nasal lesions caused by formaldehyde exposure. Zwart et al. (1988) exposed male and female Wistar rats (50 animals/group/sex) to 0, 0.3, 1, and 3 ppm formaldehyde vapor for 6 hr/day, 5 days/week for 13 weeks. Compound related histopathological nasal changes varying from epithelial disarrangement to epithelial hyperplasia and squamous metaplasia were found in the 3 ppm group, and were restricted to a small area of the anterior respiratory epithelium. These changes were confirmed by electron microscopy and were not observed in other groups.

Woutersen et al. (1989) exposed rats (20 per group) to 0, 1, 10, or 20 ppm formaldehyde 6 hours/day, 5 days/week for 13 weeks. Rats exposed to 20 ppm displayed retarded growth, yellowing of the fur, and significant histological lesions in the respiratory epithelium. Exposure to 10 ppm did not affect growth, but resulted in significant histological lesions in the respiratory tract. No effects on specific organ weights, blood chemistries, liver glutathione levels, or urinalysis were detected at any level. No significant adverse effects were seen at the 1.0 ppm exposure level.

Appelman et al. (1988) found significant nasal lesions in rats (20 per group; 0, 0.1, 1.0, or 10.0 ppm) exposed to 10 ppm formaldehyde 6 hours/day, 5 days/week for 52 weeks, but exposure to 1.0 ppm or less for this period did not result in nasal histological lesions. However, the rats exposed to formaldehyde displayed decreased body weight in all groups compared with controls.

Apfelbach and Weiler (1991) determined that rats (5 exposed, 10 controls) exposed to 0.25 ppm (0.38 mg/m³) formaldehyde for 130 days lost the olfactory ability to detect ethyl acetate odor.

Maronpot et al. (1986) exposed groups of 20 mice to 0, 2, 4, 10, 20, or 40 ppm formaldehyde 6 hours/day, 5 days/week, for 13 weeks. Histological lesions in the upper respiratory epithelium were seen in animals exposed to 10 ppm or greater. Exposure to 40 ppm was lethal to the mice.

A six-month exposure of rats to 0, 0.5, 3, and 15 ppm formaldehyde (3 rats per group) resulted in significantly elevated total lung cytochrome P450 in all formaldehyde-exposed groups (Dallas et al., 1989). The degree of P450 induction was highest after 4 days exposure and decreased slightly over the course of the experiment.

A series of studies have addressed the effects of long-term repeated exposures to formaldehyde on altered functioning of the hypothalamic-pituitary-adrenal (HPA) axis (Sorg et al., 2001b) and on neurobehavioral changes in rats (Sorg et al., 2001a). To study formaldehyde's effects on the HPA, Sorg et al. (2001b) measured corticosterone levels in the trunk blood of male Sprague-Dawley rats 20 or 60 min following acute chamber exposures to air or formaldehyde (0.7 or 2.4 ppm). All groups showed increased corticosterone levels above naive basal levels at 20 min followed by a return to baseline by 60 min, with no differences between treatment groups. A second experiment assessed the effects of repeated formaldehyde exposure (1 h/day, 5 days/week for 2 or 4 weeks) on basal corticosterone levels and those after a final challenge. Basal corticosterone levels were increased above naive values after 2 week exposure to air or 0.7 ppm

formaldehyde. By 4 weeks, corticosterone levels in the air group returned to naive values, but remained elevated in the 0.7 ppm formaldehyde group. There were no differences in basal corticosterone levels among either 2.4 ppm exposed groups. After a final air or formaldehyde challenge, the 2 and 4 week air and 0.7 ppm formaldehyde groups had elevated corticosterone levels similar to their acute response, while in the 2 and 4 week 2.4 ppm formaldehyde groups, corticosterone levels were higher than their acute response levels, indicating enhanced reactivity of the HPA axis to subsequent formaldehyde. It thus appears that repeated low-level formaldehyde exposure alters HPA axis functioning and the release of stress hormones. Since glucocorticoids may stimulate or inhibit the synthesis of surfactant-associated proteins in the lung (Liley et al., 1988), the alteration of HPA function may represent another pathway by which formaldehyde affects pulmonary function. For example, the pulmonary surfactants that regulate surface tension in the lungs are in turn regulated by surfactant-associated proteins. Reports of lower airway discomfort associated with chronic formaldehyde exposure may be related to the altered release or activity of these surfactant-associated proteins in the lung.

In another study of the effects of formaldehyde and the hypothalamus-pituitary-adrenal (HPA) axis, Sari et al. (2004) exposed female C3H/He mice to formaldehyde (0, 80, 400, 2000 ppb) by inhalation for 16 h/day, 5 days/week, for 12 weeks. Immunocytochemistry was used to examine corticotropin releasing hormone (CRH)-immunoreactive (ir) neurons in the hypothalamus, and adrenocorticotropin hormone (ACTH)-ir cells in the pituitary. RT-PCR was used to quantify ACTH mRNA in the pituitary. Two groups of female mice were exposed, one of which comprised control mice with no allergen exposure. The other group was made allergic by injection of ovalbumin and alum prior to exposure to formaldehyde. Animals in the second group were further exposed to aerosolized ovalbumin as a booster four times during the exposure period. In the non-allergic group, formaldehyde caused a dose-dependent increase in the number of CRH-ir neurons with a similar pattern of increases in ACTHir cells and ACTH mRNA. The allergic mice showed an increase in basal levels of all these markers of HPA activity, and were responsive to the lowest concentration of formaldehyde. Thus at low levels of exposure, allergen and formaldehyde exposure exacerbate each other's effects on the stress response of the HPA.

7. Developmental and Reproductive Toxicity

In humans there are few data on the association of teratogenicity or adverse reproductive effects with formaldehyde exposure. Existing data do not suggest that formaldehyde, by inhalation or oral routes, produces significant teratogenic or reproductive effects (ATSDR, 1999).

A developmental toxicity study on formaldehyde was conducted by Martin (1990). Pregnant rats (25 per group) were exposed to 0, 2, 5, or 10 ppm formaldehyde for 6 hours/day, during days 6-15 of gestation. Although exposure to 10 ppm formaldehyde resulted in reduced food consumption and body weight gain in the maternal rats, no effects on the number, viability or normal development of the fetuses were seen. In addition, Saillenfait et al. (1989) exposed pregnant rats (25 per group) to 0, 5, 10, 20, or 40 ppm formaldehyde from days 6 - 20 of gestation. Maternal weight gain and fetal weight were significantly reduced in the 40 ppm exposure group. No significant fetotoxicity or teratogenic defects were observed at formaldehyde levels that were not also maternally toxic.

Evidence of embryotoxicity was reported by Kitaeva et al. (1990) in embryos of rats that had been exposed to formaldehyde by inhalation 4 h/d, 5 d/wk for 4 months. At 1.5 mg/m³, but not at 0.5 mg/m³, there was a significant increase in the proportion of degenerate embryos. By comparison, the bone marrow cells of the mothers appeared to be more sensitive to formaldehyde as shown by significant increases in the numbers of cells with aberrations, and the numbers of chromosomes with aberrations and aneuploidy at both dose levels.

In the context of developmental susceptibility to formaldehyde exposure, as noted above, the respiratory tract lining fluid (RTLFL) protecting the lungs is often lower in glutathione levels than is the RTLFL of adult lungs (Reise et al., 1997). This is especially true in premature infants who later develop chronic lung disease (Grigg et al., 1993). As glutathione is central to the lungs' antioxidant defenses, and is involved in the metabolism of inhaled formaldehyde, this relative deficiency may make the neonate's and infant's developing lungs more susceptible to toxic insult. It should be noted that ascorbate is also an important component of the lung's antioxidant defense, especially when glutathione levels are depressed (Jain et al., 1992). In healthy lungs, ascorbate helps to maintain glutathione levels. However, as is the case for glutathione, ascorbate levels in RTLFL drop during the first week following birth (Vyas et al., 2001), potentially adding to the neonate's susceptibility to glutathione-reactive substances. Indeed, alterations in lung development following early life air toxicant exposure has been shown for environmental tobacco smoke (Wang and Pinkerton, 2007) and ozone (Plopper et al., 2007). Whether early life exposure to formaldehyde has similar effects on lung development remains to be demonstrated. However, there is concern that allergen exposure can modulate trophic interactions of conducting airway epithelial and interstitial wall components (Finkelstein and Johnston, 2004) and alter postnatal development of the airways (Plopper et al., 2007). This, coupled with the ability of formaldehyde to enhance the immune response to proteins/allergens with which it binds (Thrasher et al., 1987, 1990), may render developing lungs more susceptible to formaldehyde exposure. If evidence of such developmental effects associated with formaldehyde exposure becomes available, a re-evaluation of the REL for formaldehyde may be necessary.

8. Derivation of Reference Exposure Levels

8.1 Formaldehyde Acute Reference Exposure Level

<i>Study</i>	Kulle et al., 1987
<i>Study population</i>	19 nonasthmatic, nonsmoking humans
<i>Exposure method</i>	Whole body to 0.5-3.0 ppm
<i>Exposure continuity</i>	Single exposure per concentration
<i>Exposure duration</i>	3 hr
<i>Critical effects</i>	mild and moderate eye irritation
<i>LOAEL</i>	1 ppm
<i>NOAEL</i>	0.5 ppm
<i>Benchmark concentration</i>	0.44 ppm
<i>Time-adjusted exposure</i>	not applied
<i>Human Equivalent Concentration</i>	not applied
<i>LOAEL uncertainty factor (UF_L)</i>	not applied
<i>Subchronic uncertainty factor (UF_s)</i>	not applied

<i>Interspecies uncertainty factor</i>	
<i>Toxicokinetic (UF_{A-k})</i>	1 (default, human study)
<i>Toxicodynamic (UF_{A-d})</i>	1 (default, human study)
<i>Intraspecies uncertainty factor</i>	
<i>Toxicokinetic (UF_{H-k})</i>	1 (site of contact; no systemic effects)
<i>Toxicodynamic (UF_{H-d})</i>	10 (asthma exacerbation in children)
<i>Cumulative uncertainty factor</i>	10
<i>Reference Exposure Level</i>	55 µg/m³ (44 ppb)

Acute Reference Exposure Levels are levels at which intermittent one-hour exposures are not expected to result in adverse health effects (see Section 5 of the Technical Support Document).

Kulle et al (1987) was chosen as the critical study for the determination of the acute REL as it used a sensitive endpoint, eye irritation. It featured human subjects showing significant ($p < 0.05$) responses with short-term exposures to a range of formaldehyde concentrations, and the data permitted the use of a benchmark concentration (BMC) approach. As described in the technical support document, OEHHA recommends the use of the BMC approach whenever the available data support it as the BMC method provides a more statistically sound estimate of the point of departure in the REL determination.

The proposed acute REL was based on a $BMCL_{05}$ for eye irritation, estimated using log-probit analysis (Crump, 1984). The $BMCL_{05}$ is defined as the 95% lower confidence limit of the concentration expected to produce a response rate of 5%. The resulting $BMCL_{05}$ from this analysis was 0.44 ppm (0.53 mg/m³) formaldehyde. The endpoint of eye irritancy appears to be more a function of formaldehyde concentration rather than duration of exposure (Yang et al., 2001), so no time correction factor was applied. An uncertainty factor (UF_{H-k}) of 1 was used since sensory irritation is not expected to involve large toxicokinetic differences among individuals. Although the toxicological endpoint is eye irritation, the REL should protect against all possible adverse effects. The respiratory irritant effect, with documented potential to exacerbate asthma, is clearly an effect with the potential to differentially impact infants and children. In addition, the ability of formaldehyde to exacerbate the immune response to aeroallergens is of especial concern during development of the lungs. The toxicodynamic component of the intraspecies uncertainty factor UF_{H-d} is therefore assigned an increased value of 10 to account for potential asthma exacerbation. These considerations are applied equally to the acute, 8-hour and chronic REL.

As noted in Section 5.1, contact lens wearers appear to be at greater risk for ocular irritation with formaldehyde exposure. However, since contact lens users, and infants and children are generally mutually exclusive groups, it is expected that with the ten-fold toxicodynamic UF_{H-d} described above, the acute REL should be adequately protective of these individuals as well.

8.2 Formaldehyde 8-Hour Reference Exposure Level

<i>Study</i>	Wilhelmsson and Holmstrom, 1992
<i>Study population</i>	66 chemical plant workers
<i>Exposure method</i>	Discontinuous occupational exposure
<i>Exposure continuity</i>	8 hr/day, 5 days/week (assumed)
<i>Exposure duration</i>	10 years (average); range 1-36 years
<i>Critical effects</i>	Nasal obstruction and discomfort, lower airway discomfort, and eye irritation.
<i>LOAEL</i>	Mean 0.26 mg/m ³ (range 0.05 – 0.6 mg/m ³) (described as exposed group)
<i>NOAEL</i>	Mean of 0.09 mg/m ³ (described as control group of office workers)
<i>Benchmark concentration</i>	not derived
<i>Time-adjusted exposure</i>	0.09 mg/m ³ (time adjustment not applied)
<i>Human Equivalent Concentration</i>	not applied
<i>LOAEL uncertainty factor (UF_L)</i>	1 (NOAEL observed)
<i>Subchronic uncertainty factor (UFs)</i>	not applied
<i>Interspecies Uncertainty Factor</i>	
<i>Toxicokinetic (UF_{A-k})</i>	1 (default, human study)
<i>Toxicodynamic (UF_{A-d})</i>	1 (default, human study)
<i>Intraspecies Uncertainty Factor</i>	
<i>Toxicokinetic (UF_{H-k})</i>	1 (site of contact; no systemic effects)
<i>Toxicodynamic (UF_{H-d})</i>	10 (asthma exacerbation in children)
<i>Cumulative uncertainty factor</i>	10
<i>Reference Exposure Level</i>	9 µg/m³ (7 ppb)

The 8-hour Reference Exposure Level is a concentration at or below which adverse noncancer health effects would not be anticipated for repeated 8-hour exposures (see Section 6 in the Technical Support Document).

The 8-hour REL is based on the occupational study by Wilhelmsson and Holmstrom (1992). This study evaluated the effects of formaldehyde on the upper airways of adult human subjects exposed to a mean formaldehyde concentration of 0.26 mg/m³ during the work day compared with a referent group exposed to 0.09 mg/m³. The critical effects in this study included nasal obstruction and discomfort, lower airway discomfort, and eye irritation. A NOAEL and a LOAEL may be derived from these data but no other dose-response information was provided. This study included only adults, but there is evidence that children may be more susceptible to long term exposures to formaldehyde than are adults. Thus, in the absence of child-specific data, an intraspecies uncertainty factor of 10 for toxicodynamic variability and developmental susceptibility was applied.

For comparison, the 8-hour REL of 9 $\mu\text{g}/\text{m}^3$ is similar to the value of 10 $\mu\text{g}/\text{m}^3$ based on increased pulmonary resistance in guinea pigs following an 8 hr exposure to 0.11 – 1.05 ppm formaldehyde (Swiecichowski et al., 1993). The NOAEL of 0.59 ppm in guinea pigs was adjusted to a Human Equivalent Concentration (HEC) of 0.49 ppm with a regional gas dose ratio (RGDR) of 0.826. Use of the HEC adjustment entails an interspecies uncertainty factor of 6, while an intraspecies uncertainty factor of 10 addresses toxicodynamic variability.

<i>Study</i>	Swiecichowski et al., 1993
<i>Study population</i>	25-35 adult male guinea pigs
<i>Exposure method</i>	Whole body exposure
<i>Exposure continuity</i>	
<i>Exposure duration</i>	8 hr
<i>Critical effects</i>	Increased specific pulmonary resistance
<i>LOAEL</i>	1.0 ppm
<i>NOAEL</i>	0.59 ppm
<i>Benchmark concentration</i>	not derived
<i>Time-adjusted exposure</i>	not applied
<i>Human Equivalent Concentration</i>	0.49 ppm (610 $\mu\text{g}/\text{m}^3$) (0.59 * RGDR 0.826 for pulmonary effects)
<i>LOAEL uncertainty factor (UF_L)</i>	1 (default: NOAEL observed)
<i>Subchronic uncertainty factor (UF_s)</i>	not applied
<i>Interspecies Uncertainty Factor</i>	
<i>Toxicokinetic (UF_{A-k})</i>	6 (with HEC adjustment)
<i>Toxicodynamic (UF_{A-d})</i>	1 (with HEC adjustment)
<i>Intraspecies Uncertainty Factor</i>	
<i>Toxicokinetic (UF_{H-k})</i>	1 (no systemic effect)
<i>Toxicodynamic (UF_{H-d})</i>	10 (potential asthma exacerbation in children)
<i>Cumulative uncertainty factor</i>	60
<i>Reference Exposure Level</i>	10 $\mu\text{g}/\text{m}^3$ (8 ppb)

8.3 Formaldehyde Chronic Reference Exposure Level

<i>Study</i>	Wilhelmsson and Holmstrom, 1992 supported by Edling et al., 1988
<i>Study population</i>	66 human chemical plant workers
<i>Exposure method</i>	Discontinuous occupational exposure
<i>Exposure continuity</i>	8 hr/day, 5 days/week (assumed)
<i>Exposure duration</i>	10 years (average); range 1-36 years
<i>Critical effects</i>	Nasal obstruction and discomfort, lower airway discomfort.
<i>LOAEL</i>	Mean 0.26 mg/m ³ (range 0.05 – 0.6 mg/m ³) (described as exposed group)
<i>NOAEL</i>	Mean of 0.09 mg/m ³ (described as control group of office workers)
<i>Benchmark concentration</i>	not derived
<i>Time-adjusted exposure</i>	0.09 mg/m ³ for NOAEL group
<i>Human Equivalent Concentration</i>	not applied
<i>LOAEL uncertainty factor (UF_L)</i>	not applied
<i>Subchronic uncertainty factor (UFs)</i>	not applied
<i>Interspecies uncertainty factor</i>	
<i>Toxicokinetic (UF_{A-k})</i>	1 (default, human study)
<i>Toxicodynamic (UF_{A-d})</i>	1 (default, human study)
<i>Intraspecies uncertainty factor</i>	
<i>Toxicokinetic (UF_{H-k})</i>	1 (no systemic effects)
<i>Toxicodynamic (UF_{H-d})</i>	10 (potential asthma exacerbation in children)
<i>Cumulative uncertainty factor</i>	10
<i>Reference Exposure Level</i>	9 µg/m³ (7 ppb)

The chronic Reference Exposure Level is a concentration at which adverse noncancer health effects would not be expected from chronic exposures (see Section 7 in the Technical Support Document).

The study by Wilhelmsson and Holmstrom (1992) was selected for development of the chronic REL as it investigated long-term exposure to formaldehyde relatively free of other confounding exposures. From this study it was possible to determine both a NOAEL and a LOAEL. Since this study included only adults, a combined intraspecies uncertainty factor of 10 for toxicodynamic variability was applied to account for the possibly greater susceptibility of children with long term exposures to formaldehyde.

The susceptibility of young children was examined in a study by Rumchev et al. (2002) that compared children (mean age 25 mo) with a clinical diagnosis of asthma to children without this diagnosis. The LOAEL used (60 µg/m³) represents the formaldehyde level at which the authors found a statistically elevated risk for asthma-related respiratory symptoms. For this comparison, the NOAEL was taken to be 30 µg/m³, the lower end of the NOAEL range. Intraspecies uncertainty factors of 3.16 for potential toxicodynamic variability and 1 for toxicokinetic

differences give a cumulative uncertainty factor of 3.16 for an inhalation chronic REL of 10 $\mu\text{g}/\text{m}^3$ (8 ppb), similar to the chronic REL calculated from the critical study.

<i>Study</i>	Rumchev et al., 2002
<i>Study population</i>	88 asthmatic children (mean age 25 mo); 104 nonasthmatic controls (mean age 20 mo)
<i>Exposure method</i>	Ambient in home
<i>Exposure continuity</i>	Continuous assumed
<i>Exposure duration</i>	range 0.5-3 years
<i>Critical effects</i>	Parent-reported respiratory symptoms (cough, shortness of breath, wheeze, trouble breathing)
<i>LOAEL</i>	60 $\mu\text{g}/\text{m}^3$
<i>NOAEL</i>	30 $\mu\text{g}/\text{m}^3$ (lower limit of NOAEL range)
<i>Benchmark concentration</i>	not derived
<i>Time-adjusted exposure</i>	not applied
<i>Human Equivalent Concentration</i>	30 $\mu\text{g}/\text{m}^3$
<i>LOAEL uncertainty factor (UF_L)</i>	1
<i>Subchronic uncertainty factor (UF_s)</i>	not applied
<i>Interspecies uncertainty factor</i>	
<i>Toxicokinetic (UF_{A-k})</i>	1 (default, human study)
<i>Toxicodynamic (UF_{A-d})</i>	1 (default, human study)
<i>Intraspecies uncertainty factor</i>	
<i>Toxicokinetic (UF_{H-k})</i>	1 (study performed in children)
<i>Toxicodynamic (UF_{H-d})</i>	$\sqrt{10}$ (inter-individual variation)
<i>Cumulative uncertainty factor</i>	$\sqrt{10}$
<i>Reference Exposure Level</i>	10 $\mu\text{g}/\text{m}^3$ (8 ppb)

The Rumchev study supports an association with exposure to formaldehyde and the observation of asthma symptoms (cough, shortness of breath, wheeze, trouble breathing) in children. However, it was not selected for REL development due to the difficulties in distinguishing asthma from other wheezing conditions in the clinical diagnoses in such a young population. There are additional uncertainties associated with the exposure continuity, and the possibility of observational and/or recall bias in the parental reports of respiratory symptoms characteristic of asthma.

For comparison with the chronic REL of 9 $\mu\text{g}/\text{m}^3$ (7 ppb) presented above, Table 8.3.1 below presents a summary of potential formaldehyde RELs based on chronic and subchronic animal studies originally presented in OEHHA (2000). The toxicological endpoint was nasal lesions, consisting principally of rhinitis, squamous metaplasia, and dysplasia of the respiratory epithelium.

The most striking observation is the similarity of potential RELs among the rat chronic studies (exposures ≥ 26 weeks) that contain a NOAEL. The range of RELs from these animal studies, 1.5 – 24.9 ppb, includes the proposed REL (7 ppb) based on a human study. Another related observation is that the NOAEL and LOAEL are similar among all the studies, regardless of exposure duration. The NOAEL and LOAEL are generally in the range of 1 - 4 ppm and 1 – 10 ppm, respectively, with the exception of the study by Kamata et al. (1997) that may be due to the

absence of a dose level between 2 and 0.3 ppm. It is also of interest that the studies of Rusch et al (1983) indicate that monkeys and rats are of about the same sensitivity. In addition, the results of the Rusch studies suggest that, at least for the endpoint of squamous metaplasia, formaldehyde concentration is more important than the total dose since these animals, receiving more continuous exposure, exhibited the same adverse effects seen in studies using more intermittent exposures.

ATSDR has estimated minimum risk levels (MRLs), defined as “an estimate of daily human exposure to a substance that is likely to be without an appreciable risk of adverse effects (noncarcinogenic) over a specified duration of exposure” (ATSDR, 1999). For formaldehyde inhalation exposures they describe as “acute” (≤ 14 days), the MRL is 40 ppb based on a LOAEL of 0.4 ppm from a study by Pazdrak et al. (1993), and a 9-fold uncertainty factor (3 for use of a LOAEL; 3 for intraspecies variability). This exposure period is much longer than the acute REL of one hour, but the acute REL represents possibly repeated exposures. The MRL for an “intermediate” exposure period of 15-364 days is 30 ppb based on a NOAEL of 0.98 ppm for clinical signs of nasopharyngeal irritation and lesions in the nasal epithelium in monkeys (Rusch et al., 1983). A chronic MRL (≥ 365 d) of 8 ppb was developed based on damage to nasal epithelium in chemical factory workers (Holmstrom et al., 1989). This number is similar to the chronic REL of 7 ppb reported here. The MRLs are more similar to the chronic RELs developed by OEHHA in that they assume continuous exposure over the specified time period rather than regular but periodic exposures, as assumed for the 8-hour RELs considered above. For 8-hr exposures, NIOSH (1988) suggested a TWA 8-hr REL of 16 ppb based on sensory irritation.

8.4 Formaldehyde as a Toxic Air Contaminant

Formaldehyde was identified by the ARB as a toxic air contaminant (TAC) in accordance with sections 39660-39662 of the California Health and Safety Code on March 12, 1992 (Title 17, California Code of Regulations, section 93001)(CCR, 2007). In view of the differential impacts on infants and children identified in Section 6.2, OEHHA recommends that formaldehyde be listed as a toxic air contaminant which may disproportionately impact children pursuant to Health and Safety Code, Section 39669.5(c).

Table 8.3.1. Summary of Chronic and Subchronic Formaldehyde Studies in Experimental Animals

Study	Animal	Duration	Exposure	LOAE		Time adj	DAF	LOAEL						Cum UF	REL ppb	REL µg/m ³
				L ppm	NOAEL ppm			UF	UFak	UFad	UFhk	UFhd	UFsc			
Woutersen 89	rat	28 mo	6 h 5 d	9.8	1	0.179	0.148	1	1	3.16	1	10	1	30	4.9	6.1
Kerns 83	rat	24 mo	6 h 5 d	2	n/a	0.357	0.296	6	1	3.16	1	10	1	200	1.5	1.8
Monticello 96	rat	24 mo	6 h 5 d	6.01	2.05	0.366	0.304	1	1	3.16	1	10	1	30	10.1	12.6
Kamata 97	rat	24-28 mo	6 h 5 d	2	0.3	0.054	0.044	1	1	3.16	1	10	1	30	1.5	1.8
Appelman 88	rat	52 wk	6 h 5 d	9.4	1	0.179	0.148	1	1	3.16	1	10	1	30	4.9	6.1
Rusch 83	rat	26 wk	22 h 7d	2.95	0.98	0.898	0.746	1	1	3.16	1	10	1	30	24.9	30.8
Kimbell 97	rat	26 wk	6 h 5 d	6	2	0.357	0.296	1	1	3.16	1	10	1	30	9.9	12.3
Wilmer 89	rat	13 wk	8 h 5 d	4	2	0.238	0.198	1	1	3.16	1	10	1	30	6.6	8.2
Woutersen 87	rat	13 wk	6 h 5 d	9.7	1	0.179	0.148	1	1	3.16	1	10	1	30	4.9	6.1
Zwart 88	rat	13 wk	6 h 5 d	2.98	1.01	0.180	0.15	1	1	3.16	1	10	1	30	5.0	6.2
Kerns 83	mouse	24 mo	6 h 5 d	5.6	2	0.357	0.296	1	2	3.16	1	10	1	60	4.9	6.1
Maronpot 86	mouse	13 wk	6 h 5 d	10.1	4.08	0.729	0.605	1	2	3.16	1	10	1	60	10.1	12.5
Rusch 83	monkey	26 wk	22 h 7d	2.95	0.98	0.898	not used	1	2	2	1	10	1	40	22.5	27.8

9. References

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FORMALDEHYDE

CAS No: 50-00-0

I. PHYSICAL AND CHEMICAL PROPERTIES (HSDB, 1998)

Molecular weight	30.03
Boiling point	-19.5°C
Melting point	-92°C
Vapor pressure	1.08 torr @ 26.1°C
Air concentration conversion	1 ppm = 1.24 mg/m ³ @ 25°C

II. HEALTH ASSESSMENT VALUES

Unit Risk Factor:	6.0 E-6 (µg/m ³) ⁻¹
Slope Factor:	2.1 E-2 (mg/kg-day) ⁻¹

[Rat nasal squamous carcinoma incidence data (Kerns *et al.*, 1983; U.S. EPA 1987), linearized multistage procedure (OEHHA, 1992), with pharmacokinetic interpolation of molecular dosimetry data to the tumor incidence data.]

III. CARCINOGENIC EFFECTS

Human Studies

Epidemiological studies have shown formaldehyde exposure to be significantly associated with cancer at sites in the respiratory tract in workers and in the general population. Studies of embalmers, who have used formaldehyde, have shown increased rates of brain cancer and of leukemia.

Many studies in the epidemiological literature support a link between formaldehyde and elevated risk of cancers of the upper respiratory tract. Among the industrial cohort studies, Stayner (1988) reported a relative risk of 3.4 (90% CI: 1.2-7.9) for buccal cancer, and Blair *et al.* (1986) reported a relative risk of 3.00 (90% CI: 1.30-5.92) for nasopharyngeal cancer. Among industrial proportional mortality studies, Liebling *et al.* (1984) reported a relative risk of 8.70 (90% CI: 1.50-27.33) for buccal/pharyngeal cancer and Stayner *et al.* (1985) reported a relative risk of 7.5 (90% CI: 2.0-19) for buccal cancer. In all of these studies the elevated risk was statistically significant. The population-based case control studies reported statistically significant relationships between formaldehyde exposure and upper respiratory cancers in three studies (Vaughan *et al.*, 1986a, b; Hayes *et al.*, 1986; Olsen *et al.*, 1984), although these cancers can appear in any of several sites.

In a subsequent report Blair *et al.* (1987) presented a summary of a further analysis resulting in a significant association between nasopharyngeal cancer and simultaneous exposure to formaldehyde and to particulate, indicating that such exposure may be a risk factor. Collins *et al.* (1988) have critiqued this finding and have added data.

The three largest - and therefore potentially most sensitive - industrial cohort studies reported elevated rates of lung cancer. The largest, Blair *et al.* (1986) with 26,561 U.S. workers, reported a statistically elevated death rate due to lung cancer, equivalent to 35% above the national average. The other two studies reporting elevated death rates due to lung cancer were Acheson *et al.* (1984a, b) with 7,680 British male workers, mostly young, and Stayner *et al.* (1988) with 11,030 U.S. workers, predominantly female. Some of the categories in the Acheson study showed statistically significant increases of lung cancer. The Stayner study found lung cancer to be elevated 14% overall, which was not statistically significant, but the exposures were well below those of the other two studies.

In the Blair *et al.* (1986) study the investigators concluded that a causal relationship between formaldehyde exposure and lung cancer was unlikely because of a lack of dose gradient for those tumors. Sterling and Weinkam (1988, 1989a, b) performed a reanalysis on the basis that Blair *et al.* (1986) failed to account for a “healthy-worker” effect in the original report. These corrected results showed that lung cancer was related to formaldehyde exposure in a dose-dependent manner, which was statistically significant. In a subsequent analysis of the same workers Blair *et al.* (1990) concluded that exposure to phenol, melamine, urea, and wood dust and other substances might account for some or all of the excess lung cancer observed.

Table 1: Cohort study on industrial exposure to formaldehyde (Blair *et al.*, 1986).

Exposure	Cancer Site	Number Observed	Number Exposed	SMR	90% Confidence Interval	
					<i>Lower</i>	<i>Upper</i>
0.1 - > 2.0 ppm time weighted average	brain	17	21	0.81	0.52	1.21
	leukemia	19	24	0.80	0.52	1.16
	buccal/pharynx	18	19	0.96	0.61	1.41
	lung	201	182	1.11	0.98	1.24
	larynx	12	8	1.42	0.87	2.43
	nasal	2	2.2	0.91	0.16	2.86
> 0 - 5.5 ppm-yr	lung, 20 yr latency	146	108	1.35	1.17	1.55
	hypopharynx	1	1.7	0.59	0.02	2.78
	nasopharynx	6	2.0	3.00	1.30	5.92
	oropharynx	5	2.6	1.92	0.76	4.04

Source: OEHHA (1992)

Recent epidemiological studies contribute to the conclusions only marginally. Gerin *et al.* (1989) presented the results of a large case control study with 3,726 cancer patients. The odds ratio for the highest exposure group with adenocarcinoma of the lung was nearly significant at the 95% confidence level, and there was an apparent trend of incidence of this cancer with exposure. Nevertheless, the authors concluded that there was no persuasive evidence of an increased risk of any type of cancer among men exposed to the exposure levels of formaldehyde cited by Blair *et al.* (1986) (Table 1). The study did not consider cancers of the nasal cavity, of the brain, or of leukemia. Bertazzi *et al.* (1989) presented an extension of a previous study (Bertazzi *et al.*, 1986) which had detected elevated lung cancer among 1,332 workers in a resin

manufacturing plant subject to formaldehyde exposure. In the extended study with more accurate estimates of exposure, the lung cancer rate was not elevated above expected for those exposed to formaldehyde (Bertazzi *et al.*, 1989). Linos *et al.* (1990) reported elevated rates of follicular non-Hodgkin's lymphoma and of acute myeloid leukemia among embalmers and funeral directors in a population-based case control study. The investigators did not attribute these tumors to formaldehyde exposure. Malker *et al.* (1990) found significantly elevated rates of incidence of nasopharyngeal cancer among workers in fiberboard plants and among book binders, both being subject to formaldehyde exposure.

Four recent occupational studies have investigated the relationship of formaldehyde exposure to histological changes, some of which are potentially precancerous lesions, in the nasal mucosa. Holmstrom *et al.* (1989) found that workers exposed to well-defined levels of formaldehyde developed significant changes in the middle turbinate, while those exposed to both formaldehyde and wood dust did not. Boysen *et al.* (1990) found in nasal biopses that workers exposed to formaldehyde showed a significantly higher degree of metaplastic alterations. Edling *et al.* (1988) found significant histological differences in the nasal mucosa of formaldehyde workers compared to unexposed workers but found no histological differences between those exposed to formaldehyde and those exposed to formaldehyde and wood dust. Berke (1987) found no statistical relationship between exfoliated nasal cells in formaldehyde-exposed workers and control groups. Thus, these studies provide some indication of possible histologic change due to formaldehyde exposure in humans, consistent with results in animals.

Animal Studies

A study sponsored by the Chemical Industry Institute for Toxicology (CIIT) has provided the most quantitatively useful evidence for the carcinogenicity of formaldehyde (Swenberg *et al.*, 1980a, b; Kerns *et al.*, 1983). This study used 120 male and 120 female Fischer-344 rats in each dose group, including a clean air group. The adjusted tumor incidences (adjusted for competing causes of death, including scheduled interim sacrifices) for squamous cell carcinomas in the nasal passages of males and females combined, when exposed to 0, 2.0, 5.6, or 14.3 ppm formaldehyde for 6 hours/day, 5 days/week for up to 24 months, were 0/156, 0/159, 2/153 and 94/140 (U.S. EPA, 1987). In an analogous study on mice, two mice in the high dose group (14.3 ppm) developed squamous cell carcinomas, a finding that was not statistically significant but was thought to be biologically significant due to the absence of this tumor in control animals and to concurrence with rat studies. Kerns *et al.* (1983) also reported benign tumors, including polypoid adenomas and squamous cell papillomas. Swenberg *et al.* (1980a, b) described a number of additional lesions in the nasal turbinates of rats exposed to formaldehyde for 18 months, including rhinitis, epithelial dysplasia and hyperplasia, squamous hyperplasia, and cellular atypia that occurred in a dose-related manner. Other inhalation studies (Albert *et al.*, 1982; Tobe *et al.*, 1985) have provided positive evidence for the carcinogenicity of formaldehyde.

Recent investigations of chronic toxicity have shown formaldehyde administered orally for 24 months to be carcinogenic in Sprague-Dawley rats but not in Wistar rats. Soffritti *et al.* (1989), using six exposure groups each of 50 male and 50 female Sprague-Dawley rats, with drinking water concentration of 10 to 1500 mg/L formaldehyde, reported increases in the percent of

animals bearing leukemias and gastrointestinal neoplasias at the higher exposures. Til *et al.* (1989), using three exposure groups, each of 70 male and 70 female Wistar rats, with drinking water concentrations of 20 to 1900 mg/L, reported numerous pathological changes at the highest exposure level, but no evidence of carcinogenicity at any level. Tobe *et al.* (1989), using three exposure groups, each of 20 male and 20 female Wistar rats, with drinking water concentrations of 200 to 5000 mg/L, also reported pathological changes at the highest exposures level but no significant increases in the incidence of any tumor in these small treatment groups. In a letter to the editor, Feron *et al.* (1990) questioned the conclusions and some methods of Soffritti *et al.* (1989).

Other types of exposures have produced a spectrum of results. Watanabe *et al.* (1954) presented a brief preliminary report of experimentally inducing sarcomas by repeated injections of an aqueous solution of formaldehyde in rats. Muller *et al.* (1978) induced a preneoplastic lesion of the oral mucosa by repeated exposure to formalin solution in rabbits. Homma *et al.* (1986) found that formalin solution repeatedly administered in transplanted rat bladders did not promote formation of tumors. Takahashi *et al.* (1986) found that formalin solution in diet did promote stomach tumors in Wistar rats. Iversen *et al.* (1988) found that topical skin application of formaldehyde solution in mice did not promote the formation of skin tumors.

IV. DERIVATION OF CANCER POTENCY

Basis for Cancer Potency

The International Agency for Research of Cancer (1987) has reviewed the evidence for carcinogenicity and found it to be limited in humans and sufficient in animals. U.S. EPA (1987) has classified formaldehyde in Group B1, probable human carcinogen. The U.S. Occupational Safety and Health Administration (U.S. OSHA, 1987) has concluded that “formaldehyde should be regarded as an occupational carcinogen,” based upon animal and human studies. Considering these previous determinations, along with the evidence of carcinogenicity, OEHHA staff (OEHHA, 1992) concluded that formaldehyde is a probable carcinogen and meets the definition of a “toxic air contaminant”: an air pollutant which may cause or contribute to an increase in mortality or an increase in serious illness, or which may pose a present or potential hazard to human health.

Formaldehyde is carcinogenic in rodents, as described above, producing squamous cell carcinomas in the nasal passages of male and female rats and male mice. Several different types of potentially precancerous abnormalities, including polypoid adenomas and squamous cell papillomas, have also been observed. The epidemiological evidence, while suggestive of a risk of human cancer due to formaldehyde exposure, was considered insufficient for risk assessment purposes on its own. OEHHA (1992) found the tumor incidence data in rats reported by Kerns *et al.* (1983) and used by U.S. EPA (1987) to be the most appropriate for use in developing a quantitative risk assessment.

Methodology

In developing a spectrum of predictions of cancer risk to humans, the OEHHA (1992) assessment applied a pharmacokinetic interpolation of the molecular dosimetry data to the animal cancer bioassay data of Kerns *et al.* (1983). The analysis used the linearized multistage procedure (GLOBAL86), and the procedure developed by Moolgavkar and others, which takes into account the proliferation of premalignant cells due to the formaldehyde exposure. Both models derive upper confidence limits (UCL) for excess cancer risk and extrapolate the risk to humans by means of three different scaling factors. Two scaling factors take into account the contact mechanism of carcinogenesis. However, they do so in different ways. One uses only a generic calculation in terms of body mass. The other takes specific account of comparative data on DNA binding in rats and monkeys to adjust the metabolic rate for humans; it assumes humans respond as do monkeys and uses the data of Casanova *et al.* (1989; 1991). The third scaling factor follows the default option of the California carcinogen guidelines (CDHS, 1985), which calculates the adjustment for rat exposures to obtain the equivalent human exposure on the basis of intake rate divided by body surface area.

Table 2: Formaldehyde inhalation bioassay data used to estimate cancer risk to rats

Exposure (ppm HCHO) ^a	Rate of DNA Binding ^b (pmol/mg-hr)	Lifetime Equivalent Metabolic Exposure ^b (ppm)	Incidence of Nasal Squamous Carcinomas ^c
0	0	0	0/156 (0%)
2	2.5	0.54	0/159 (0%)
5.6	15.9	3.4	2/153 (1.3%)
14.3	74.8	16.	94/140 (67.5%)

Source: adapted from OEHHA (1992)

^aFischer 344 rats inhaled indicated concentrations of formaldehyde gas 6 hours per day, 5 days per week for 24 months.

^bDetails on how these estimates were obtained are presented in OEHHA (1992)

^cBased on data partially reported in Kerns *et al.* (1983). Numerator and denominator are those used by U.S. EPA (1987).

For the best value of UCL on unit risk for a lifetime of exposure, the OEHHA staff selected $7 \times 10^{-3} \text{ ppm}^{-1}$ ($6.0 \times 10^{-6} (\mu\text{g}/\text{m}^3)^{-1}$), based on molecular dosimetry data in a three-stage model and using the standard surface-area scaling factor, 1.2. The range of calculated values of UCL on unit risks is $0.3 \times 10^{-3} \text{ ppm}^{-1}$ to $40 \times 10^{-3} \text{ ppm}^{-1}$ (0.25×10^{-6} to $33 \times 10^{-6} (\mu\text{g}/\text{m}^3)^{-1}$).

In a review of epidemiological studies for workers exposed to formaldehyde the study by Blair *et al.* (1986) was selected as the most reliable for quantitative comparisons. That study, the largest and best documented study available, evaluated mortality in a cohort of more than 26,000 workers. The observed risk of death by lung cancer in exposed workers was 15×10^{-3} over their career. Based on extrapolation of rat cancer risk predictions to humans for a 40-hour work week for 20 years and an exposure level of 1.0 ppm, the prediction of 95% upper confidence limits on respiratory tract cancer was 32×10^{-3} for the three-stage tissue-dose model with generic contact

scaling factor. Thus, the upper range of human cancer risk predictions from the rat bioassay data (Kerns *et al.*, 1983) was consistent with the occupational exposure cancer risk data.

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CHRONIC TOXICITY SUMMARY

CHLORINATED DIBENZO-*p*-DIOXINS and CHLORINATED DIBENZOFURANS (INCLUDING 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN)

(Polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) including 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) which is the principal congener of concern based on toxicity)

CAS Registry Number: 1746-01-6 (TCDD); 5120-73-19 (TCDF)

I. Chronic Toxicity Summary

<i>Inhalation reference exposure level</i>	0.00004 µg/m³ (40 pg/m³)
<i>Oral reference exposure level</i>	1 x 10⁻⁸ mg/kg/day (10 pg/kg/day)
<i>Critical effect(s)</i>	Increased mortality, decreased weight gain, depression of erythroid parameters, increased urinary excretion of porphyrins and delta-aminolevulinic acid, increased serum activities of alkaline phosphatase, gamma-glutamyl transferase and glutamic-pyruvic transaminase, gross and histopathological changes in the liver, lymphoid tissue, lung and vascular tissues in rats.
<i>Hazard index target(s)</i>	Alimentary system (liver); reproductive system; development; endocrine system; respiratory system; hematopoietic system

II. Physical and Chemical Properties (HSDB, 1995; 1999)

<i>Description</i>	All are white crystalline powders at 25° C.
<i>Molecular Formula</i>	C ₁₂ H ₄ C ₁₄ O ₂ (TCDD)
<i>Molecular Weight</i>	321.97 g/mol (TCDD)
<i>Density</i>	1.827 g/ml (estimated for TCDD)
<i>Boiling Point</i>	412.2°C (estimated for TCDD)
<i>Melting Point</i>	305-306°C (TCDD)
<i>Vapor Pressure</i>	1.52 x 10 ⁻⁹ torr at 25°C (TCDD)
<i>Solubility</i>	In water: 19.3 ng/L at 22°C (TCDD)
<i>Log K_{ow}</i>	6.15-7.28 (6.8 for TCDD)
<i>(octanol/water partition coefficient)</i>	
<i>Log K_{oc}</i>	6.0-7.39
<i>(organic-carbon distribution coefficient)</i>	
<i>Henry's Law Constant</i>	8.1 x 10 ⁻⁵ ATM-m ³ /mol

III. Major Uses and Sources

The chlorinated dioxins and furans are generated as by-products from various combustion and chemical processes. PCDDs are produced during incomplete combustion of chlorine containing wastes like municipal solid waste, sewage sludge, and hospital and hazardous wastes. Various metallurgical processes involving heat, and burning of coal, wood, petroleum products and used tires for energy generation also generate PCDDs. Chemical manufacturing of chlorinated phenols (e.g., pentachlorophenol), polychlorinated biphenyls (PCBs), the phenoxy herbicides (e.g., 2,4,5 T), chlorinated benzenes, chlorinated aliphatic compounds, chlorinated catalysts and halogenated diphenyl ethers are known to generate PCDDs as a by-product under certain conditions. While manufacture of many of these compounds and formulations has been discontinued in the United States, continued manufacture elsewhere in the world combined with use and disposal of products containing PCDD by-products results in the inadvertent release of PCDDs into the environment. Industrial and municipal processes in which naturally occurring phenolic compounds are chlorinated can produce PCDDs; the best example is chlorine bleaching of wood pulp in the manufacture of paper products. Additionally, municipal sewage sludge has been documented to occasionally contain PCDDs and PCDFs. Annual statewide industrial emissions from facilities reporting under the Air Toxics Hot Spots Act in California based on the most recent inventory were estimated to be 0.123 pounds of 2,3,7,8-TCDD, 0.244 pounds of 1,2,3,4,7,8-hexachlorodibenzodioxin and lesser amounts of other polychlorinated dibenzodioxins and dibenzofurans (CARB, 1999).

IIIa. 2,3,7,8 Tetrachlorodibenzo-p-dioxin Toxic Equivalent

2,3,7,8-Tetrachlorodibenzo-p-dioxin is considered the most potent congener of the polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) families of compounds. Potency of PCDD and PCDF congeners correlates with the binding affinity to the cytosolic Ah receptor. Structure activity studies have demonstrated that optimal biological activity and Ah-receptor binding requires congeners with a planar conformation and chlorines at the corners of the molecule at the 2,3,7,8 positions (Poland and Knutson, 1982; Safe, 1986). Chlorines at both ortho positions in these molecules (i.e., positions 1 and 9) sterically hinder a planar conformation that lessens the congeners' biological activity. Thus only 15 of 210 different PCDDs and PCDFs congeners possess significant biological activity based on chlorines in the 2,3,7,8 positions and some degree of planar conformation (Safe, 1986; U.S. EPA 1989). These include two tetrachloro-congeners: 2,3,7,8-tetrachlorodibenzo-p-dioxin and 2,3,7,8-tetrachlorodibenzofuran; three pentachloro congeners: 1,2,3,7,8-pentachlorodibenzo-p-dioxin, 1,2,3,7,8-pentachlorodibenzofuran, and 2,3,4,7,8-pentachlorodibenzofuran; seven hexachloro congeners: 1,2,3,4,7,8 or 1,2,3,6,7,8 or 1,2,3,7,8,9-hexachlorodibenzo-p-dioxins and hexachlorodibenzofurans and 2,3,4,6,7,8-hexachlorodibenzofuran; and three heptachloro congeners: 1,2,3,4,6,7,8-heptachlorodibenzo-p-dioxin, 1,2,3,4,6,7,8-heptachlorodibenzofuran and 1,2,3,4,7,8,9-heptachlorodibenzofuran (U.S. EPA, 1989). The structures of the dibenzo-p-dioxins and dibenzofurans along with their numbering schemes are shown in Figure 1. Toxic equivalents are calculated relative to the most potent congener, 2,3,7,8-tetrachlorodibenzo-p-dioxin, and are determined based on structure activity studies examining relative affinity for the

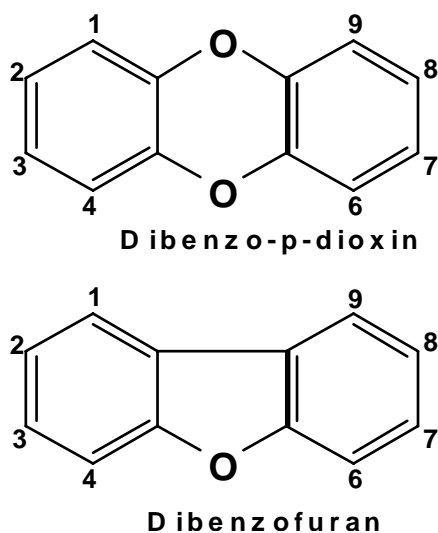
Ah receptor as well as on relative toxicity of different congeners. Values for the international system of toxic equivalents are provided in Table 1 (U.S. EPA, 1989).

Table 1. International Toxic Equivalency Factors (I-TEFs) for PCDDs and PCDFs Chlorinated in the 2,3,7, and 8 Positions. (U.S. EPA 1989.)

Compound ^{1,2}	I-TEF
Mono-, Di-, and Tri-CDDs and CDFs	0
<u>TetraCDD</u>	
2,3,7,8-substituted	1.0
Others	0
<u>PentaCDD</u>	
2,3,7,8-substituted	0.5
Others	0
<u>HexaCDD</u>	
2,3,7,8-substituted	0.1
Others	0
<u>HeptaCDD</u>	
2,3,7,8-substituted	0.01
Others	0
<u>OctaCDD</u>	
	0.001
<u>TetraCDF</u>	
<u>2,3,7,8</u>	0.1
<u>Others</u>	0
<u>PentaCDF</u>	
1,2,3,7,8-PentaCDF	0.05
2,3,4,7,8-PentaCDF	0.5
others	0
<u>HexaCDF</u>	
2,3,7,8-substituted	0.1
Others	0
<u>HeptaCDF</u>	
2,3,7,8-substituted	0.01
Others	0
<u>OctaCDF</u>	
	0.001

¹ CDD designates chlorinated dibenzo-p-dioxin

² CDF designates chlorinated dibenzofuran

Figure 1. Structures of the Dibenzop-dioxins and Dibenzofurans

IV. Effects of Human Exposure

The information available on possible chronic toxic effects in humans is complicated by the relative insensitivity of epidemiological studies, the limited ability of case studies of exposed individuals to establish cause and effect relationships, the heterogeneous nature of human populations, the broad spectrum of exposures to other toxic agents in the human environment, and the episodic exposure of many of the exposed human populations which have been studied (e.g., Seveso, Italy). As a result, a limited number of effects have been associated with exposure to dioxins in humans. The meaning of these effects in terms of toxicity in most cases remains to be clarified. The majority of information comes from cross-sectional medical studies.

Chloracne is the most widely recognized effect of exposure to 2,3,7,8-TCDD and TCDD-like PCDDs and PCDFs. Chloracne is a persistent condition, which is characterized by comedones, keratin cysts and inflamed papules and is seen after acute and chronic exposure to various chlorinated aromatic compounds (Moses and Prioleau, 1985). Other dermal effects include hyperpigmentation and hirsutism or hypertrichosis (Jirasek *et al.*, 1974; Goldman, 1972; Suskind *et al.*, 1953; Ashe and Suskind, 1950); both appear to resolve themselves more quickly over time than chloracne, making them more of an acute response rather than a chronic response (U.S. EPA, 1994a). Epidemiological data available for 2,3,7,8-TCDD have not allowed a determination of the threshold dose required for production of chloracne (U.S. EPA, 1994b). Case studies suggest that there may be a relationship between 2,3,7,8-TCDD exposure and hepatomegaly (Reggiani, 1980; Jirasek *et al.*, 1974; Suskind *et al.*, 1953; Ashe and Suskind, 1950) and hepatic enzyme changes (Mocarelli *et al.*, 1986; May, 1982; Martin 1984; Moses *et al.*, 1984). Nevertheless, cross sectional epidemiological studies of trichlorophenol (TCP) production workers (Suskind and Hertzberg., 1984; Bond *et al.*, 1983; Moses *et al.*, 1984; Calvert *et al.* 1992), Vietnam veterans (Centers for Disease Control Vietnam Experience Study, 1988; Roegner *et al.*, 1991) and Missouri residents (Webb *et al.*, 1989; Hoffman *et al.*, 1986)

found little evidence for an association between exposure and hepatomegaly suggesting that this is not a chronic response. There is a consistent pattern of increased levels of serum gamma glutamyl transferase in populations exposed to 2,3,7,8-TCDD which is presumably of hepatic origin (Mocarelli, 1986; Caramaschi *et al.*, 1981, May, 1982; Martin, 1984; Moses *et al.*, 1984; Calvert *et al.*, 1992; Centers For Disease Control Vietnam Experience Study, 1988). Two cross sectional studies have associated diabetes and elevated fasting serum glucose levels with relatively high serum 2,3,7,8-TCDD levels (Sweeney *et al.*, 1992; Roegner *et al.*, 1991). However other studies provided mixed results (Moses *et al.*, 1984; Centers for Disease Control Vietnam Experience Study, 1988; Ott *et al.*, 1993). TCDD has been associated with effects on reproductive hormonal status in males. The likelihood of abnormally low testosterone levels was 2 to 4 times greater in individuals with serum 2,3,7,8-TCDD levels above 20 pg/ml (Egeland *et al.* 1994) and increased serum levels of luteinizing hormone and follicle stimulating hormone have been documented (Egeland *et al.*, 1994). A number of other effects have been reported that were either not seen as chronic effects or effects seen long term in only one population of exposed persons. These include elevated liver enzymes (aspartate aminotransferase and alanine aminotransferase), pulmonary disorders, neurologic disorders, and changes in porphyrin metabolism and kidney disorders (U.S. EPA, 1994c). Areas in which there is presently insufficient information to draw solid conclusions include effects on the circulatory system, reproductive effects, immunological effects, effects on metabolism and handling of lipids, and on thyroid function (U.S. EPA, 1994c). Recent findings in Rhesus monkeys have shown 2,3,7,8-TCDD to cause endometriosis (Reier *et al.*, 1993) and epidemiological studies are currently underway to determine if there is an association between TCDD exposure and endometriosis in human populations exposed by the Seveso accident.

Potential effects of a toxicant on normal fetal development include fetal death, growth retardation, structural malformations and organ system dysfunction. Evidence for all four of these responses has been seen in human populations exposed to dioxin-like compounds. In these poisoning episodes populations were exposed to a complex mixture of halogenated aromatic hydrocarbons contained within PCBs, PCDFs and PCDDs mixtures thus limiting the conclusions that could be drawn from the data. In the Yusho and Yu-Cheng poisoning episodes, human populations consumed rice oil contaminated with PCBs, PCDFs and PCDDs. Yu-Cheng women experienced high perinatal mortality in hyperpigmented infants born to affected mothers (Hsu *et al.* 1985). This occurred in women with overt signs of toxicity (chloracne) (Rogan, 1982) and Rogan notes that, when there is no sign of toxicity in the mother, the likelihood of fetotoxicity appears to lessen considerably in the infants. Signs of toxicity from dioxin like compounds were absent in infants born to mothers apparently not affected in the Seveso, Italy and Times Beach, Missouri, incidents (Reggiani, 1989; Hoffman and Stehr-Green, 1989), which supports Rogan's conclusion. There was an increased incidence of decreased birth weight in infants born to affected mothers in the Yusho and Yu-Cheng incidents suggesting fetal growth retardation (Wong and Huang, 1981; Law *et al.*, 1981; Lan *et al.*, 1989; Rogan *et al.*, 1988). The structural malformation, rocker bottom heel, was observed in Yusho infants (Yamashita and Hayashi, 1985) making this malformation a possible result of exposure to dioxin-like compounds. Nevertheless, it is unknown if these compounds produce malformations in humans. Evidence for possible organ system dysfunction in humans comes from a study of Yu-Cheng children which found that children exposed in utero experienced delays in attaining developmental milestones, and exhibited neurobehavioral abnormalities (Rogan *et al.*, 1988)

suggesting involvement of CNS function. Dysfunction of dermal tissues is noted in exposed infants of the Yusho and Yu-Cheng incidents and is characterized by hyperpigmentation of the skin, fingernails, and toenails, hypersecretion of the meibomian glands, and premature tooth eruption (Taki *et al.*, 1969; Yamaguchi *et al.*, 1971; Funatsu *et al.*, 1971; Wong and Huang, 1981; Hsu *et al.*, 1985; Yamashita and Hayashi, 1985; Rogan *et al.*, 1988; Rogan, 1989; Lan *et al.*, 1989).

V. Effects of Animal Exposure

The toxicity to laboratory animals encompasses a number of areas including changes in energy metabolism manifested as wasting syndrome, hepatotoxicity, effects on tissue of epithelial origin, various endocrine effects, effects on vitamin A storage and use, immune system effects and reproductive and developmental toxicity. The limited number of chronic studies available do not examine all these endpoints. Therefore subchronic exposures are included here in order to provide a more complete coverage of potential chronic toxic effects of these compounds.

Wasting syndrome is one of the most broadly occurring toxic effects. The wasting syndrome is characterized by loss of adipose tissue and lean muscle mass and is produced in all species and strains tested, but there are difference in sensitivity (U.S. EPA 1994d; Peterson *et al.*, 1984; Max and Silbergeld, 1987). Numerous studies have not yet established the mechanism of wasting syndrome (U.S. EPA, 1994e). Hepatotoxicity is also seen in all species tested, but there is considerable variation in species sensitivity (U.S. EPA, 1994d). TCDD induces hyperplasia and hypertrophy of liver parenchymal cells. Morphological and biochemical changes in the liver include increased SGOT and SGPT, induction of microsomal monooxygenases and proliferation of the smooth endoplasmic reticulum, porphyria, increased regenerative DNA synthesis, hyperlipidemia, hyperbilirubinemia, hypercholesterolemia, hyperproteinemia, degenerative and necrotic changes, mononuclear cell infiltration, multinucleated giant hepatocytes, increased numbers of mitotic figures, and parenchymal cell necrosis (U.S. EPA, 1994d; WHO/IPCS, 1989). Epithelial effects seen include chloracne (rabbit ear and the hairless mouse) (Jones and Krizek, 1962; Schwetz *et al.*, 1973) and hyperplasia and/or metaplasia of gastric mucosa, intestinal mucosa, the urinary tract, the bile duct and the gall bladder (U.S. EPA 1994f). TCDD exposure results in endocrine like effects including epidermal growth factor like effects such as early eye opening and incisor eruption in the mouse neonate (Madhukar *et al.*, 1984), glucocorticoid like effects such as involution of lymphoid tissues (U.S. EPA, 1994g; Sunahara *et al.*, 1989), alteration in thyroid hormone levels and in some cases thyroid hormone like effects (WHO/IPCS, 1989; Rozman *et al.*, 1984), decreases in serum testosterone and dihydrotestosterone (Mittler *et al.*, 1984; Keys *et al.*, 1985; Moore and Peterson, 1985), and changes in arachidonic acid metabolism and prostaglandin synthesis (Quilley and Rifkind, 1986; Rifkind *et al.*, 1990). TCDD is known to decrease hepatic vitamin A storage (Thunberg *et al.*, 1979). TCDD and other dioxin like PCDDs and PCDFs are potent suppressors of both cellular and humoral immune system function, characteristically producing thymic involution at low doses and involution of other lymphoid tissues at higher doses (U.S. EPA 1994h).

In animal studies there is a large body of information available documenting both developmental and reproductive toxicity of 2,3,7,8-TCDD and other PCDDs and PCDFs. These compounds are

acutely toxic to early life stages of fish and birds with fish being most sensitive (LD₅₀ of 0.4 µg/kg for rainbow trout sac fry eggs and LD₅₀ of 34 ng/kg for lake trout eggs); some species of birds are also relatively sensitive (LD₅₀ of 0.25 µg/kg for chicken eggs) (Peterson *et al.*, 1993). 2,3,7,8-TCDD has been documented to increase the incidence of prenatal mortality in a number of species of laboratory animals including the Rhesus monkey, Guinea pig, rabbit, rat, hamster, and mouse (Peterson *et al.*, 1993). Exposure to 2,3,7,8-TCDD during gestation produces a characteristic set of fetotoxic responses in most laboratory animals which includes: thymic hypoplasia, subcutaneous edema, and decreased growth (Peterson *et al.*, 1993). More species specific responses include cleft palate formation in the mouse at doses below maternal toxicity (Moore *et al.*, 1973; Smith *et al.*, 1976; Couture *et al.*, 1990), intestinal hemorrhage in the rat (Sparschu *et al.*, 1971), hydronephrosis in the mouse and hamster (Moore *et al.*, 1973; Smith *et al.*, 1976; Couture *et al.*, 1990; Birnbaum *et al.*, 1989; Olson *et al.*, 1990), and extra ribs in the rabbit (Giavini *et al.*, 1982). Female rats have also been found to be affected by perinatal exposure to 2,3,7,8-TCDD with clefting of the clitoris, incomplete or absent vaginal opening and a smaller vaginal orifice after a dose of 1 µg/kg to the mother on day 15 of gestation (Gray *et al.*, 1993).

A number of effects on adult reproductive function are seen in male animals exposed in utero to 2,3,7,8-TCDD. TCDD reduces plasma androgen levels in the adult male rat and perinatal exposure decreases spermatogenesis, spermatogenic function and reproductive capability, feminizes male sexual behavior, and feminizes male gonadotrophic function (LH secretion) (Mably *et al.*, 1991; Mably *et al.*, 1992a,b,c). Evidence suggests that these effects are the result of impaired sexual differentiation of the CNS, which in male rats is dependent on exposure of the developing brain to testosterone.

There are numerous studies detailing the effects of the PCDDs, PCDFs and other dioxin like compounds, however a large number of these studies were conducted as either acute or subchronic exposures, studies in which it is unlikely that body burdens had reached steady state levels. Detailed below are three chronic studies that were considered in the setting of a chronic toxicity exposure level.

The most definitive study of chronic toxicity in rats is that of Kociba *et al.* (1978). This study involved the administration of 2,3,7,8-TCDD in the diet at doses of 1 ng/kg/day, 10 ng/kg/day, and 100 ng/kg/day to groups of 50 male and 50 female Sprague Dawley rats for two years. A group of 86 male and 86 female rats received diet with solvent vehicle alone and served as controls. The following observations (excluding carcinogenic effects) were seen at the 100 ng/kg/day dose: increased mortality, decreased weight gain, depressed erythroid values, increased urinary excretion of porphyrins and delta-aminolevulinic acid, and increased serum activities of alkaline phosphatase, gamma-glutamyl transferase, and glutamic-pyruvic transaminase. Histopathologic changes were noted in the liver, lymphoid tissue, respiratory and vascular tissues. The primary ultrastructural change in the liver was proliferation of the rough endoplasmic reticulum. At the 10 ng/kg/day dose the severity of toxic symptoms was less than that of the 100 ng/kg/day dose and included increased urinary excretion of porphyrins in females as well as liver and lung lesions. The 1 ng/kg/day dose produced no discernible significant toxic effects. Interpretation of this study by the authors was that the 1 ng/kg/day dose was a NOAEL.

Two chronic toxicity studies are available in the mouse. The first is a one year study conducted by Toth *et al.* (1979) using male Swiss mice administered weekly oral doses of 7, 700, and 7000 ng/kg/day. In this study 2,3,7,8-TCDD administration resulted in amyloidosis and dermatitis in 0 of 38 control animals, 5 of 44 animals receiving 7 ng/kg/day, 10 of 44 animals receiving 700 ng/kg/day and 17 of 43 animals receiving 7,000 ng/kg/day. The other study was from the NTP 1982 gavage study (NTP, 1982) in B6C3F1 mice. This study employed groups of 50 male and 50 female mice. The males received doses of 0, 10, 50, and 500 ng/kg/week by gavage for two years while female mice received doses of 0, 40, 200, and 2000 ng/kg/week by gavage for two years. No adverse effects were seen at the lowest doses tested in each sex, which correspond to NOAELs of approximately 1.4 and 6 ng/kg/day for males and females, respectively. Neither chronic toxicity study in mice reported data on enzyme activity.

VI. Derivation of Chronic Reference Exposure Level (REL)

<i>Study</i>	Kociba <i>et al.</i> (1978)
<i>Study population</i>	Sprague-Dawley rats of both sexes (50/treatment group/sex)
<i>Exposure method</i>	Continuous dietary exposure starting at seven weeks of age for 2 years
<i>Critical effects</i>	Increased mortality, decreased weight gain, depression of hematologic measures, increased urinary excretion of porphyrins and delta-aminolevulinic acid, increased serum activities of alkaline phosphatase, gamma-glutamyl transferase and glutamic-pyruvic transaminase, gross and histopathological changes in the liver, lymphoid tissue, lung and vascular tissues
<i>Observed LOAEL</i>	210 ppt in diet (0.01 µg/kg/day)
<i>Observed NOAEL</i>	22 ppt in diet (0.001 µg/kg/day)
<i>Exposure continuity</i>	Continuous exposure via the diet
<i>Exposure duration</i>	2 years
<i>Subchronic uncertainty factor</i>	1
<i>LOAEL uncertainty factor</i>	1
<i>Interspecies uncertainty factor</i>	10
<i>Intraspecies uncertainty factor</i>	10
<i>Cumulative uncertainty factor</i>	100
<i>Oral reference exposure level</i>	10 pg/kg/day
<i>Route-to-route extrapolation</i>	3,500 µg/m ³ per mg/kg/day
<i>Inhalation reference exposure level</i>	40 pg/m ³ (0.00004 µg/m ³)

The data available for chronic toxic effects in humans have a number of limitations. Some studies did not determine the body burden of compounds necessary to estimate dose.; The Yusho and Yu-Cheng poisoning episodes have uncertainty because exposure was to complex mixtures of halogenated aromatic hydrocarbons rather than to individual congeners. And epidemiological

studies and case studies have limitations in determining cause and effect relationships. Therefore, an animal study was chosen for determination of a NOAEL/LOAEL. The study chosen for use was that of Kociba *et al.* (1978), based on the duration of the study (2 years), the number of animals employed (50 per treatment group per sex), testing of both sexes, a dose range, which spanned from an apparent NOAEL to severe hepatic effects including carcinogenic effects, a complete histopathological examination of all organ systems, examination of urinary excretion of porphyrins and delta-aminolevulinic acid, and determination of serum activities of alkaline phosphatase, gamma-glutamyl transferase, and glutamic-pyruvic transaminase. The elevation of human serum values for gamma-glutamyl transferase is one of the consistently seen chronic responses in exposed human populations and reflects changes in liver biochemistry. Thus the examination of markers of liver toxicity also altered in animal models of chronic toxicity make the Kociba study an appropriate choice for detecting potential chronic toxic effects of 2,3,7,8-TCDD in humans. The NOAEL in the Kociba *et al.* (1978) study was determined to be 1 ng/kg body weight/day. For the purposes of determining the REL the 1 ng/kg/day dose was considered to be a NOAEL based upon the observations of Kociba *et al.* (1978).

VII. Data Strengths and Limitations for Development of the REL

NOAELs from a number of other studies compare favorably with the 1 ng/kg/day NOAEL. These include the NOAEL from the NTP (1982) study in B6C3F1 mice and the NOEL for enzyme induction in rats and marmosets calculated by Neubert (1991) of 1 ng/kg. Furthermore the 1 ng/kg/day NOAEL is lower than the LOAELs observed by Toth *et al.* (1979) of 7 ng/kg/day in mice and by Schantz *et al.* (1978) of 2.3 ng/kg/day in rhesus monkeys. Current exposure assessments for 2,3,7,8-TCDD and other dioxin-like compounds including the PCBs, PCDDs, and PCDFs estimate that the average daily background dose in the U.S. is 3-6 pg TEQ/kg/day (U.S. EPA 1994i) also placing the REL close to background exposures. The REL of 10 pg/kg/day should be protective of chronic effects on liver function and avoid significant increases in exposure over the background level of human exposure.

The strengths of the inhalation REL include the availability of chronic exposure data from a well-conducted study with histopathological analysis, the observation of a NOAEL, and the demonstration of a dose-response relationship. Major areas of uncertainty are the lack of adequate human exposure data and the lack of chronic inhalation exposure studies.

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CHLORINATED DIBENZO-*p*-DIOXINS

CAS No: 1746-01-6

CHLORINATED DIBENZOFURANS

CAS No: 5120-73-19

I. PHYSICAL AND CHEMICAL PROPERTIES (From HSDB (1998) except as noted)

2,3,7,8-Tetrachlorodibenzo-*p*-dioxin

Molecular weight	322
Boiling point	decomposes (NIOSH, 1994)
Melting point	305-306 °C
Vapor pressure	7.4×10^{-10} mm Hg at 25 °C
Air concentration conversion	not available

2,3,7,8-Tetrachlorodibenzofuran

Molecular weight	305.99
Boiling point	not available
Melting point	not available
Vapor pressure	not available
Air concentration conversion	not available

II. HEALTH ASSESSMENT VALUES

Congener	Unit Risk ($\mu\text{g}/\text{m}^3$) ⁻¹	Slope Factor ($\text{mg}/\text{kg}/\text{day}$) ⁻¹
PCDDs		
2,3,7,8-Tetrachlorodibenzo- <i>p</i> -dioxin	3.8 E+1	1.3 E+5
1,2,3,7,8-Pentachlorodibenzo- <i>p</i> -dioxin	3.8 E+1	1.3 E+5
1,2,3,4,7,8-Hexachlorodibenzo- <i>p</i> -dioxin	3.8 E+0	1.3 E+4
1,2,3,6,7,8-Hexachlorodibenzo- <i>p</i> -dioxin	3.8 E+0	1.3 E+4
1,2,3,7,8,9-Hexachlorodibenzo- <i>p</i> -dioxin	3.8 E+0	1.3 E+4
1,2,3,4,6,7,8-Heptachlorodibenzo- <i>p</i> -dioxin	3.8 E-1	1.3 E+3
1,2,3,4,5,6,7,8-Octachlorodibenzo- <i>p</i> -dioxin	3.8 E-3	1.3 E+1
PCDFs		
2,3,7,8-Tetrachlorodibenzofuran	3.8 E+0	1.3 E+4
1,2,3,7,8-Pentachlorodibenzofuran	1.9 E+0	6.5 E+3
2,3,4,7,8-Pentachlorodibenzofuran	1.9 E+1	6.5 E+4
1,2,3,4,7,8-Hexachlorodibenzofuran	3.8 E+0	1.3 E+4
1,2,3,6,7,8-Hexachlorodibenzofuran	3.8 E+0	1.3 E+4
1,2,3,7,8,9-Hexachlorodibenzofuran	3.8 E+0	1.3 E+4
2,3,4,6,7,8-Hexachlorodibenzofuran	3.8 E+0	1.3 E+4
1,2,3,4,6,7,8-Heptachlorodibenzofuran	3.8 E-1	1.3 E+3
1,2,3,4,7,8,9-Heptachlorodibenzofuran	3.8 E-1	1.3 E+3
1,2,3,4,5,6,7,8-Octachlorodibenzofuran	3.8 E-3	1.3 E+1

Congener	Unit Risk ($\mu\text{g}/\text{m}^3$) ⁻¹	Slope Factor ($\text{mg}/\text{kg}/\text{day}$) ⁻¹
PCBs (IUPAC #, structure)		
77 3,3',4,4'-Tetrachlorobiphenyl	3.8 E-3	1.3 E+1
81 3,4,4',5- Tetrachlorobiphenyl	3.8 E-3	1.3 E+1
105 2,3,3',4,4'- Pentachlorobiphenyl	3.8 E-3	1.3 E+1
114 2,3,4,4',5- Pentachlorobiphenyl	1.9 E-2	6.5 E+1
118 2,3',4,4',5- Pentachlorobiphenyl	3.8 E-3	1.3 E+1
123 2',3,4,4',5- Pentachlorobiphenyl	3.8 E-3	1.3 E+1
126 3,3',4,4',5- Pentachlorobiphenyl	3.8 E+0	1.3 E+4
156 2,3,3',4,4',5- Hexachlorobiphenyl	1.9 E-2	6.5 E+1
157 2,3,3',4,4',5'- Hexachlorobiphenyl	1.9 E-2	6.5 E+1
167 2,3',4,4',5,5'- Hexachlorobiphenyl	3.8 E-4	1.3 E+0
169 3,3',4,4',5,5'- Hexachlorobiphenyl	3.8 E-1	1.3 E+3
189 2,3,3',4,4',5,5'- Heptachlorobiphenyl	3.8 E-3	1.3 E+1

PCDDs = polychlorinated dibenzo-*p*-dioxins. PCDFs = polychlorinated dibenzofurans. PCBs = polychlorinated biphenyls. IUPAC = International Union for Pure and Applied Chemistry.

[Linearized multistage procedure (GLOBAL79), fitted to male mouse hepatic adenoma and carcinoma data (NTP, 1982), body weight scaling, cross-route extrapolation (CDHS, 1986).]

III. CARCINOGENIC EFFECTS

Human Studies

Comprehensive reviews of the human studies of dioxin exposure and cancer risk available at the time the document entitled *Health Effects of Chlorinated Dioxins and Dibenzofurans* was written for the Toxic Air Contaminant (TAC) program (CDHS, 1986) are found in US EPA (1984) and Veterans Administration (VA) (1981, 1984). A more recent review of human dioxin exposure and cancer risk studies can be found in ATSDR (1999).

Dioxins have never been intentional products. In human exposure studies, PCDDs (polychlorinated dibenzo-*p*-dioxins) and PCDFs (polychlorinated dibenzofurans) have only been present as contaminants of other toxic chemicals, such as herbicides. Hence all studies of human PCDD/PCDF exposures have been studies of exposure to chemical mixtures that may have contained PCDD and PCDF.

VA (1981, 1984) summarized what is known about the presence of PCDD and PCDF in commercially-used chemicals. In general, PCDDs and PCDFs may be present as contaminants in the herbicide 2,4,5-trichlorophenoxyacetic acid (2,4,5T). Levels of 2,3,7,8-TCDD in 2,4,5-T have been found as high as six parts per million (Rappe *et al.* 1982). Another widely used herbicide, 2,4-dichlorophenoxyacetic acid (2,4-D) is

generally regarded as uncontaminated with 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD). Cochran *et al.* (1982) did detect traces of di-, tri-, and TetraCDD as high as one part per billion in technical grade 2,4-D from Canada. However, the TetraCDD isomer found in these samples was the 1,3,6,8-TCDD isomer, not the more toxic 2,3,7,8-TCDD.

Agent Orange, which was a mixture of 2,4,5-T and 2,4-D, has been shown to contain 2,3,7,8-TCDD concentrations as high as 15-47 parts per million with an average of about 2 ppm (VA 1981). PCDDs and/or PCDFs have also been found in the parts per million range in commercially used polychlorinated biphenyls (PCB), trichlorophenol (TCP), tetrachlorophenol, and pentachlorophenol (PCP) (Rappe *et al.* 1982, Hardell 1983).

Several case/control studies have been conducted in Sweden and in New Zealand. In these countries, phenoxyacetic acids and chlorophenols were used extensively for agriculture and forestry. After clinical observations of several patients with soft-tissue sarcomas (STS) and a history of heavy exposure to phenoxyacetic acids, Hardell and Sandstrom (1979) conducted a case/control study of STS and herbicide exposure. Cases were drawn from a university hospital in Northern Sweden, and consisted of 52 adult males with STS diagnosed between 1970 and 1977. Controls were drawn from general population registries, at a 4:1 matching ratio, and matched to cases on sex, age, place of residence, and vital status (whether alive or deceased). The investigators considered only non-malignant deaths for deceased controls. Study subjects (or their next of kin) provided exposure histories by a mailed questionnaire with a telephone follow-up. The odds ratio (OR) for exposure to phenoxyacetic acids only (excluding subjects exposed to chlorophenols) was 5.3 (95% confidence interval (95% CI) 2.4-11.5). For exposure to chlorophenols only (excluding those exposed to phenoxyacetic acids) the OR was 6.6 (95% CI 2.1-20.9).

To confirm these findings, Ericksson *et al.* (1981) replicated this study in Southern Sweden, using cases from a cancer registry. Similar study methods were used, including matching controls from a population registry (at a 2:1 ratio), and determining exposure by mail and telephone questionnaires. The investigators calculated separate odds ratios for exposure to phenoxy acids known to be contaminated with PCDD and PCDF (OR-17.0; 95% CI 2.1-140.0) and for exposure to phenoxy acids thought to be free of PCDD and PCDF (OR-4.2; 95% CI 1.2-14.9). When exposure was dichotomized into categories of 30 days or less, or more than 30 days, the ORs were 5.7 and 8.5, respectively, possibly indicating a dose-response trend.

One of the drawbacks of this study is that, exposure histories were provided by the study subjects; therefore, the results may be influenced by recall bias. Cases (or their next of kin) may be more likely to recall an exposure than a healthy person. In order to investigate this possible bias, Hardell (1981) duplicated the study methods using cases of colon cancer. Here there was no significant association with exposure to herbicides. Therefore, Hardell concluded that the association with STS was not due to reporting differences between diseased cases and healthy controls.

Smith *et al.* (1984) reported a similar case/control study in New Zealand. Here, male cases of STS were gathered from a national cancer registry, with controls also being

selected from the same registry. This method of control selection was designed to avoid differential recall. Unlike the Swedish studies, however, the New Zealand study showed no significant associations with reported phenoxy herbicide spraying. The authors suggested that if dioxin were the necessary agent, that Swedish herbicides may have been more contaminated than New Zealand herbicides. However, Smith *et al.* (1984) note that the Swedish investigators also found a significant association between STS and non-dioxin-contaminated herbicides, indicating that if the association were true, dioxin would not be the sole agent.

Another case/control study reported in brief by Olsen and Jensen (1984) of cases from the Danish Cancer Registry failed to show an association between nasal cancer and chlorophenol exposure, although nasal cancer was associated with occupational exposure to wood dust.

In a letter to Lancet, Milham (1982) reported proportionate mortality data from Washington state indicating that farmers suffered a significantly larger proportion of deaths due to STS. No other group occupationally exposed (foresters, orchardists, tree farmers) showed an excess of STS; however, the exposure assessment was based on occupations taken from death certificates. Furthermore, Milham indicated that 2,4-D was the predominant herbicide used, and 2,4-D is not generally contaminated with 2,3,7,8-TCDD.

A cohort study of phenoxy acid herbicide applicators in Finland was reported by Riihimaki *et al.* (1983). A historical cohort of 1926 herbicide applicators was assembled from the records of four large employers, including the Finnish Highway Authority and State Railways. These male workers had used chlorinated phenoxyacids for at least two weeks between 1955 and 1971. Their mortality between 1972 and 1980 was studied by comparing their names against population registers. National mortality figures provided expected age-standardized numbers of deaths. Deaths from all causes, and for all cancers, were less than expected. The power of this study to detect an increase in STS was poor, however, as only 0.1 case of STS was expected based on general population rates. Furthermore, as deaths in the cohort were studied only after 1972, 45 deaths that occurred in this group before 1972 were not tallied. (Even for post-1971 deaths, however, the follow-up period may also have been too short for a sufficient tumor latency period to have elapsed.)

There have been four potentially exposed occupational cohorts studied in the United States. Zack and Suskind (1980) reported the follow-up of Monsanto employees in Nitro, West Virginia, who were involved in a 1949 accident during the processing of trichlorophenol. A sudden violent reaction released fumes and residues into a building interior. Apparently, the released chemical mixture was not analyzed, but the authors assumed that it contained TCDD, as exposed workers developed chloracne. A historical cohort of 121 white male employees was assembled from company records on the basis of their having exhibited skin disorders "attributed to the 1949 TCP process accident." Their vital status was traced through 1978, providing a maximum of 29 years of follow-up per person. The standardized mortality ratio (SMR) for all causes of death in

this cohort (relative to US white males) was significantly decreased (32 observed deaths vs. 46.4 expected). One cancer site showed an excess: lung cancer (5 observed vs. 2.85 expected), although this SMR of 1.75 was not statistically significant. Interestingly, there occurred one STS, a fibrous histiocytoma. However, the authors calculated SMRs (and expected numbers of deaths) only for causes with five or more observed deaths.

Zack and Gaffey (1983) described another cohort from this plant, composed of 884 male workers employed for at least one year between 1955 and 1977. It is not clear whether workers exposed in the 1949 accident were included. The same methods were used to calculate SMRs. Only 25 malignancies occurred, compared to 30.9 expected. However, two specific sites were notably elevated: lung cancer, with 14 observed vs. 9.9 expected (SMR 1.4; 95% CI 0.8-2.4), and bladder cancer, with 9 observed vs. 0.9 expected (SMR 9.9; 95% CI 4.5-18.8). One STS occurred in a worker judged to have been exposed to TCDD. One drawback to this study is that exposure histories were only constructed for the 163 decedents - and only 36% of these were judged to have had potential exposure to 2,4,5-T (and therefore TCDD). Therefore, the true exposed cohort may only have been one-third the size of the entire study group.

Cook *et al.* (1980) presented a similar historical cohort study of Dow chemical employees. In 1964, chloracne occurred in workers in a trichlorophenol manufacturing area. Industrial hygiene investigations concluded that TCDD was responsible and changes were made in the operations to decrease exposure. Levels of TCDD during this period were unknown because concentrations fell below the limit of detection at that time, 0.02 µg/ml of air (Cook 1981a); however, wipe samples were positive for TCDD. Cook *et al.* (1980) assembled a cohort of 39 workers thought to have high exposure potential, and 22 workers thought to have lower exposure. Among the high-exposure group, 87% had a history of chloracne, compared to 68% of the low-exposure group. Their vital status was determined through 1978. There were only four deaths (vs 7.8 expected based on US white males), although three of these deaths were due to neoplasms (vs 1.6 expected). One neoplasm was a fibrosarcoma.

Another Dow cohort was investigated by Ott *et al.* (1980). This cohort contained 204 white males involved in 2,4,5-T production between 1951 and 1971. The authors determined each worker's vital status through 1976, resulting in a median length of time since first exposure of about 20 years. Only one malignancy (a respiratory cancer) was recorded vs. 3.6 expected from US population rates. This cancer death occurred among the employees with 20 or more years of latency; in this group 0.9 deaths were expected.

Besides the small sample size, there are other problems with using this study for risk assessment. The exposure to TCDD may have been minimal. Environmental sampling of the breathing zone in 1969 revealed 2,4,5-T concentrations between 0.2 and 0.8 mg/m³. Product specifications at that time called for a maximum TCDD concentration of 1 ppm. Assuming the maximum level of both 2,4,5-T in the breathing zone, and TCDD in the 2,4,5-T, the concentration of TCDD in the breathing zone would have been 10⁻⁶ of the concentration of 2,4,5-T, or 0.8 ng/m³. Ott *et al.* also noted that 157 of the 204 workers (77%) were exposed for less than one year. Furthermore, a review of medical records of the cohort uncovered no cases of chloracne.

A further analysis of Dow employees was presented by Bond *et al.* (1983), who reported a morbidity survey on the combined cohorts previously described by Cook *et al.* (1980) and Ott *et al.* (1980). Bond *et al.* found few differences between the morbidity of these workers and a matched control group of workers from other locations in the plant. There were, however, more ulcers and diseases of the digestive system (excluding liver) in the 2,4,5-T cohort, at roughly twice the prevalence in the controls. However, because the investigators only studied cohort members who participated in company medical programs between 1976 and 1978, only 69% of the original cohort was included. The study did not include workers who had died, retired, or left the company, raising the possibility that the most affected workers might have been missed.

Following the publication of the four US mortality studies, reports began to appear in *Lancet* of four additional cases of STS among these cohorts, bringing the apparent total to seven (Honchar and Halperin 1981, Cook 1981b, Moses and Selikoff 1981, Johnson *et al.* 1981). The proportion of deaths in these merged cohorts due to STS appeared to be far greater than would be expected (Fingerhut and Halperin 1983), although there is great difficulty in estimating expected rates of STS using general population statistics (Cook and Cartmill 1984). Fingerhut (cited in VA 1984) had the diagnoses of the seven cases reviewed by two pathologists. The pathologists could only agree on a diagnosis of STS for three of the seven, another three being reclassified, and the last diagnosis being disputed. Of the three definite cases, only two had frank chloracne to corroborate exposure. The VA review (1984) concluded that the occurrence of even two cases of STS among these relatively small cohorts warranted continued surveillance.

Other cohort studies of occupational exposures have come from Great Britain, West Germany, and the Netherlands. May (1973 and 1982) only briefly described the aftermath of a 1968 accidental release of TCP with a "higher than normal" concentration of TCDD. A total of 79 cases of chloracne were recorded, but May did not specify how many workers were exposed, so that an attack rate cannot be calculated. A survey of 46 of these workers, who were still with the company 10 years later, revealed that roughly half still had some chloracne (May, 1982). There were no other clinical problems reported, and no cases of cancer (although clearly few if any would be expected in a group this small).

Thiess *et al.* (1982) published a carefully-reported study of 74 workers exposed to dioxins during a 1953 reactor accident in a German 2,4,5-T plant. After a 23-year follow-up, this cohort exhibited seven deaths due to malignancies (vs. 4.09 expected from West German population rates), including three deaths due to stomach cancer (vs. 0.7 expected). The latter was statistically significant at a one-sided 95% level. No cases of STS occurred, although less than 0.1 would have been expected.

A mortality study of workers present at an explosion in an herbicide factory in Amsterdam was summarized by Dalderup and Zellenrath (1983). Between 200 and 500 g of TCDD were thought to have been liberated. The investigation traced 141 of 145 workers potentially exposed, and 69 (49%) had developed chloracne. After 20 years of follow-up, 8 of the workers had died with cancer (vs. 6.9 expected), yielding an SMR of

1.2 (95% CI 0.5-2.3). No STS deaths were seen. Unfortunately, the authors did not calculate SMRs separately for the group with frank chloracne (an indicator of stronger exposure), as the crude mortality for this chloracne group was 20%, and for the non-chloracne group 15%.

At the time the dioxin TAC document was prepared (CDHS, 1986), reports were starting to appear in the literature on the effects of Agent Orange herbicide exposure in Vietnam. However, most of those reports were at the time primarily anecdotal, or interim results. Agent Orange was composed of equal parts 2,4-D and 2,4,5-T, and about 90,000 tons of herbicides were sprayed in Vietnam between 1962 and 1971. Hay (1983) mentioned evidence from Vietnamese studies that "suggests a link" between herbicide exposure and liver cancer, but provided no details. Sarma and Jacobs (1982) reported three patients with STS who claimed Agent Orange exposure while serving in Vietnam.

The US Air Force's Ranch Hands study (summarized by VA, 1984) had released some initial results at the time the dioxin TAC document was prepared. This was a cohort study of some 1200 military personnel who worked on Operation Ranch Hand, the herbicide spraying operation. These subjects were matched (in a 5:1 ratio) with personnel who flew only cargo missions in Vietnam. As of 1983, the total mortality rates were nearly identical between the two groups. Only four cases of cancer had occurred among the exposed, and none were STS. The investigators stressed the preliminary nature of the data, the relatively low power of a study of this size to detect rare tumors such as STS, and the relatively short latency period up to that time (12-21 years).

A report by Greenwald *et al.* (1984) gave the results of a case/control study of STS in New York State. Cases of STS (n = 281) diagnosed between 1962 and 1980, who were between the ages of 18 to 29 during the war in Vietnam, were selected from the state cancer registry. Cases were individually age matched to living controls drawn from drivers' license files. The investigators gathered exposure information from subjects or next of kin by a telephone questionnaire. The questions focused on Vietnam service (and Agent Orange exposure in particular), but included other exposures such as chemical manufacturing and herbicide spraying in general. Only 3% of the cases and 4% of the controls had a history of Agent Orange, dioxin, or 2,4,5-T exposure. None of the various exposures proved statistically significant.

The power of this study can be criticized, with exposures as rare as they were. Also, the inclusion of cancer cases from the early 1960s can be questioned. These cases would not have had sufficient latency to have been caused by an exposure in Vietnam.

In 1983, an Australian Royal Commission began investigating the effects of Agent Orange exposure to Australian Vietnam veterans. However, their report, released in 1985, does not supply much information on the effects of PCDDs. The executive summary concluded that "only a very limited number of Australian servicemen were ever directly exposed," and further, that the dose received by the majority of Australian veterans was "so minute that it may, without doubt, be ignored," (e.g., it noted that no Australians developed chloracne). Not surprisingly, the Commission found no evidence of any cancer excess among the "exposed" servicemen (Royal Commission, 1985).

There are only a few cases where dioxin exposure of the general population has been documented; the Seveso incident in Italy, is one of them. In 1976, a chemical plant producing 2,4,5-trichlorophenol, exploded and released into the air several chemicals including TCDD in the vicinity of Seveso. The Seveso incident represents a unique event in the sense that exposure to the toxic chemical was not limited to occupational exposure by workers but the whole population was affected by the TCDD release in the area surrounding a pentachlorophenol manufacturing facility that experienced an explosion and fire releasing dioxins into the atmosphere. Children, woman and men of various age were exposed to different degrees depending on the distance and direction from the origin of the plume.

Abate *et al.* (1982) summarized the series of studies following the 1976 accidental release of TCDD from a TCP-producing plant in Seveso, Italy. The investigators looked at mortality rates for 11 municipalities for four years after the accident and reported no increase in cancer mortality. These studies served mainly to provide baseline rates for future studies, because clearly not enough time had elapsed to provide the minimum 10 to 20 years required for an increased cancer risk to become manifest (Bruzzi, 1983).

Fifteen years after the industrial accident, Bertazzi *et al.* (1997) examined the cancer mortality among residents (20 to 74 years old) of Seveso by comparing populations living in dioxin contaminated areas (divided into three zones: highest, lower and lowest zone of exposure to dioxin, zone A, B, and R, respectively) with population from neighboring noncontaminated areas (zone nonABR). No increase for all-cancer mortality, or major specific sites like respiratory cancer among males and breast cancer among females, was found. However, other specific cancer mortality was observed and could be associated with dioxin exposure. Table 1 represents cancer mortality for men and women living in zone B.

Increased mortality from stomach cancer (RR = 2.4; 95% CI = 0.8-5.7) was reported 10 years after the accident in women living in zone B. In men, increased mortality from rectal cancer (RR = 6.2; 95% CI = 1.7-15.9) was observed. Leukemia in men represented one of the highest risks seen in zone B for hematologic neoplasms and was statistically significant (RR = 3.1; 95% CI = 1.3-6.4). Multiple myeloma in women (RR = 6.6; 95% CI = 1.8-16.8), and Hodgkin's disease in both genders (RR = 3.3; 95% CI = 0.4-11.9 in men; and RR = 6.5; 95% CI = 0.7-23.5 in women) were also noted in that zone. In the young population (20,000 subjects aged 0 to 19 years old), some cases of cancer were also found (Pesatori *et al.*, 1993). Cancer cases noted included two ovarian cancers and Hodgkin's lymphoma; myeloid leukemia represented the most evident increase although not statistically significant (RR = 2.7; 95% CI = 0.7-11.4). Two cases of thyroid cancer were also reported (RR = 4.6; 95% CI = 0.6-32.7). This observation represents an important result because of its magnitude and its correlation with experimental observations. None of the elevated cancer incidences in zone A, the area with the highest exposure, were statistically significant; however, this area also had the smallest population. Additionally, it should be noted that the Seveso population was exposed to 2-3 orders of magnitude times the level of dioxin normally experienced by the general population of industrialized countries. In 1997, individuals living in the contaminated

area at the time of the accident still experienced high level of plasma TCDD 20 years after the industrial accident in Seveso. Geometric means for plasma TCDD concentration for individuals who lived in zone A, B and nonABR (control zone) in 1976 were 53.2, 11.0 and 4.9 ppt, respectively. Women in these three groups represented the gender with the highest plasma TCDD contamination (Landi et al., 1997). The authors concluded that the results indicate a positive association between dioxin exposure and certain cancers, but further study is needed to clarify this association.

Table 1. Female and male deaths in zone B for selected causes, 1976-1991, ten years or more since first exposure (latency) and duration of exposure (length of stay in contaminated area) (Adapted from Bertazzi et al., 1997).

		Latency > 10 years		Length of stay > 10 years	
		Female	Male	Female	Male
All cancers	OBS	23	31	20	29
	RR	1.4	1.0	1.4	1.1
	(95% CI)	(0.9 – 2.1)	(0.7 – 1.4)	(0.8 – 2.1)	(0.7 – 1.6)
Digestive cancer	OBS	10	12	9	12
	RR	1.5	1.0	1.6	1.2
	(95% CI)	(0.7 – 2.7)	(0.5 – 1.8)	(0.7 – 2.9)	(0.6 – 2.1)
Stomach cancer	OBS	5	X	4	
	RR	2.4	X	2.3	
	(95% CI)	(0.8 – 5.7)		(0.6 – 6.0)	
Lymphatic and hemopoietic	OBS	4	4	3	4
	RR	2.8	2.5	2.4	2.5
	(95% CI)	(0.7 – 7.1)	(0.7 – 6.4)	(0.5 – 7.1)	(0.7 – 6.4)
Multiple myeloma	OBS	3		2	
	RR	15.9		11.0	
	(95% CI)	(3.2 – 46.5)		(1.2 – 39.6)	
Rectal cancer	OBS		4		4
	RR		6.2		7.2
	(95% CI)		(1.7 – 15.9)		(1.9 – 18.4)
Leukemia	OBS		2		2
	RR		3.4		3.9
	(95% CI)		(0.4 – 12.3)		(0.4 – 14.1)

OBS = observed deaths RR = relative risk CI = confidence interval

Animal Studies

Van Miller *et al.* (1977a,b) reported the results of a study in which rats were fed diets containing from 1 ppt to 1 ppm of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) for 78 weeks. Surviving rats were killed after 95 weeks. Laparotomies were performed on all surviving rats at 65 weeks and all tumors were biopsied. Rats in the three highest dose groups, receiving 50 ppb or more, died early. A variety of tumors were found in rats receiving 5 ppt to 5 ppb while no-neoplasms were found in the control or low-dose groups. The absence of tumors in these two groups is unusual in this strain of rats. In

addition, because of the small number of animals in each group (10) the study was inadequate to determine the carcinogenic potential of TCDD.

Toth *et al.* (1979) administered TCDD to male Swiss/H/Riop strain mice by gavage once a week for a year, then followed them for their lifetime. The weekly doses were 0.007, 0.7, and 7.0 µg/kg. Analysis of the results from this study focused on the incidence of liver tumors. A significant increase in the incidence of liver tumors was observed in the intermediate-dose group compared to the four separate control groups. The high-dose group, however, had an incidence of liver tumors that was similar to the control group. This finding may be explained by the early mortality in the high-dose group. The average life span was 424 days for this group, compared to average life spans of between 577 and 651 days for the control groups. If the treated animals had lived it is possible that more tumors may have formed.

Kociba *et al.* (1978) conducted a two-year feeding study in male and female Sprague-Dawley rats given diets containing 2200, 210, or 22 parts per trillion (w/w) TCDD for two years. Consumption of these diets resulted in daily doses of 0.1, 0.01, and 0.001 µg/kg body weight, respectively. There were 50 male and 50 female rats in each treatment group and 86 animals of each sex in the control group. There was a statistically significant ($p < 0.05$) increase in cumulative mortality for the high-dose female group in the latter half of the study. Body weights of the male and female high-dose groups were significantly ($p < 0.05$) reduced for the last three quarters of the study; however, food intake was not altered. The combined incidence of hepatocellular carcinomas and hepatocellular neoplastic nodules in the intermediate and high-dose groups of female rats was increased above the control group. Statistically significant increased incidences of stratified squamous cell carcinomas of the hard palate and/or nasal turbinates were observed in both male and female high-dose groups. The male group also had an increased incidence of squamous cell carcinoma of the tongue, while the female group had an increased incidence of keratinizing squamous cell carcinoma of the lung.

US EPA (1981) reviewed this study and had an independent pathologist, Robert Squire, review the tissue pathology. The incidences of significant tumors reported by Kociba *et al.* (1978) and by Squire (US EPA, 1981) are given in Table 2 for male and female rats. The results of Squire's review did not differ greatly from those reported by Kociba *et al.* (1978).

CDHS staff members concurred with earlier reviewers (IARC 1982, EPA 1984) that the study reported by Kociba *et al.* (1978) was an adequately conducted chronic carcinogenicity bioassay of TCDD, with significant effects observed at the two higher dose levels.

Table 2: Tumor incidences in Osborne-Mendel rats receiving 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD) in the diet for two years (US EPA, 1984)

Tumor type, sex	Dose level (µg/kg-day)			
	0	0.001	0.01	0.1
	Tumor incidence ^a			
Tongue, stratified squamous cell carcinoma male	0/76 (0/77)	1/49 (1/44)	1/49 (1/49)	4/42 (<i>p</i> = 0.015) (3/44) (<i>p</i> = 0.046)
Nasal turbinates/hard palate, squamous cell carcinoma male	0/51 (0/55)	1/34 (1/34)	0/27 (0/26)	4/30 (<i>p</i> = 0.017) (6/30) (<i>p</i> = 0.002)
female	1/54 (0/54)	0/30 (0/30)	1/27 (1/27)	5/24 (<i>p</i> = 0.009) (5/22) (<i>p</i> = 0.001)
lung, keratinizing squamous cell carcinoma female	0/86 (0/86)	0/50 (0/50)	0/49 (0/49)	7/49 (<i>p</i> < 0.001) (8/47) (<i>p</i> < 0.001)
Liver, hepatocellular hyperplastic nodules, carcinomas female	9/86 (16/86)	3/50 (8/50)	18/50 (<i>p</i> < 0.001) (27/50) (<i>p</i> < 0.001)	34/48 (<i>p</i> < 0.001) (33/47) (<i>p</i> < 0.001)

P values determined using Fisher's exact test.

^a Number of animals with tumor over number of animals examined (incidence reported by Kociba *et al.*, 1978). Numbers in parentheses give the incidence reported by Squire (US EPA, 1984).

The National Toxicology Program (NTP 1982a) conducted an oncogenicity bioassay of TCDD in male and female Osborne-Mendel rats. They were administered TCDD in a 9:1 corn oil:acetone vehicle by gavage at dose levels of 0.005, 0.025, or 0.25 µg/kg twice a week for 104 weeks. The treatment groups consisted of 50 rats of each sex and a vehicle control group that was made up of three subgroups of 25 rats of each sex. An untreated control group, also made up of three subgroups of 25 rats of each sex, was included in the study, but not in the statistical analysis of the results by NTP. At the dose levels used, TCDD did not have a significant effect on survival of any treatment group. The high-dose group of male rats did have a statistically-significant increased incidence of subcutaneous tissue fibromas, but it was not considered biologically significant because of the variability found. All male treatment groups had significantly (*p* < 0.05) increased incidences of thyroid follicular cell adenomas or adenomas and carcinomas, although the low- and intermediate-dose level group incidences were not significant when compared to the untreated control group by CDHS staff. The female high-dose group had significantly (*p* < 0.05) increased incidences of several tumor types, including subcutaneous tissue fibrosarcomas, liver neoplastic nodules or hepatocellular carcinomas, and adrenal cortical adenomas. Of these 3 tumors, NTP considered only the liver tumors

to be related to TCDD administration. The incidences of these tumors are given in Table 3. Toxic hepatitis was found in 14 male and 32 female high-dose level rats.

Table 3: Tumor incidences in male and female Osborne-Mendel rats given 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD) by gavage for two years (NTP, 1982a)

Sex, tumor type	Dose level (µg/kg-week)			
	0	0.01	0.05	0.5
Males	Tumor incidence ^a			
Thyroid				
Follicular cell adenoma	1/69	5/48 (<i>p</i> = 0.042)	6/50 (<i>p</i> = 0.021)	10/50 (<i>p</i> = 0.001)
Follicular cell adenoma/carcinoma	1/69	5/48 (<i>p</i> = 0.042)	8/50 (<i>p</i> = 0.004)	11/50 (<i>p</i> < 0.001)
Females				
Subcutaneous tissue, fibrosarcoma	0/75	2/50	3/50	4/49 (<i>p</i> = 0.023) [3] ^b
Liver				
Neoplastic nodules/ hepatocellular carcinoma	5/75	1/49	3/50	14/49 (<i>p</i> = 0.001)
Adrenal				
Cortical adenoma or adenoma NOS	11/73	8/49	4/49	14/46 (<i>p</i> = 0.039)

^a Number of animals with tumor over number of animals examined.

^b Number of animals with hepatocellular carcinoma.

NOS = Not otherwise specified. *P* values determined using Fisher's exact test.

NTP (1982a) also conducted a carcinogenicity bioassay with TCDD in male and female B6C3F₁ hybrid strain mice. The protocol was similar to that used in the rat study with male mice receiving the same doses of TCDD. Female rats, however, received larger doses of 0.02, 0.1 or 1.0 µg/kg twice a week. These dose levels did not have a statistically significant effect on survival of any treatment group. Male mice in the highest dose group had a significantly increased incidence of hepatocellular carcinomas. The high-dose female group had significantly increased incidences of subcutaneous tissue fibrosarcomas, hepatocellular adenomas or carcinomas, and thyroid follicular-cell adenomas. NTP considered only liver tumors and thyroid tumors to be related to TCDD administration. NTP also considered histiocytic lymphomas to have been increased in the high-dose female group; however, the staff of DHS did not consider that these lymphomas were increased when the incidences in all control subgroups were considered. The observed tumor incidences in both male and female mice are given in Table 4. Toxic hepatitis was observed in 44 male and 34 female high-dose group animals. It was also observed in several animals of the other treatment groups.

Table 4: Tumor incidences in male and female B6C3F₁ mice given 2,3,7,8-Tetrachloro-dibenzo-*p*-dioxin (TCDD) by gavage for two years (NTP, 1982a).

Sex, tumor type	Dose level (µg/kg-week) ^a			
	0	0.01 (0.04)	0.05 (0.2)	0.5 (2.0)
	Tumor incidence ^b			
males				
liver (hepatocellular carcinoma)	8/73	9/49	8/49	17/50 (<i>p</i> = 0.002)
Hepatocellular adenoma or carcinoma	15/73	12/49	13/49	27/50 (<i>p</i> < 0.001)
females				
Subcutaneous tissue, fibrosarcoma	1/74	1/50	1/48	5/47 (<i>p</i> = 0.032)
liver, hepatocellular carcinoma	1/73	2/50	2/48	6/47 (<i>p</i> = 0.014)
hepatocellular adenoma or carcinoma	3/73	6/50	6/48	11/47 (<i>p</i> = 0.002)
thyroid, follicular cell adenoma	0/69	3/50	1/47	5/46 (<i>p</i> = 0.009)

P values determined using Fisher's exact test.

^a Dose administered to male mice; dose administered to female mice in parentheses.

^b Number of animals with tumor over number of animals examined.

Both rat and mouse carcinogenicity bioassays conducted by NTP appear to have been done in an adequate manner. The number of treatment groups and the large dose range used in the studies are not typical of NTP bioassays, although it was similar to that used by Kociba *et al.* (1978). However, it may not have been large enough to include a dose level which produced no effect. Most significantly increased tumor incidences only occurred in the high-dose level groups, but a statistically significant dose-related trend was found in all groups.

NTP (1982b) also conducted a dermal oncogenicity bioassay on TCDD in male and female Swiss-Webster mice. TCDD in an acetone suspension was applied to the skin three days per week for 104 weeks. The male rats received 0.001 µg per application and the females received 0.005 µg per application. Separate groups of male and female mice were treated with one application of 50 µg 7,12-dimethylbenz(*a*)anthracene (DMBA) one week prior to the start of TCDD treatments. The only significantly (*p* = 0.01) increased incidences of tumors observed were among female mice. Both the TCDD- and DMBA/TCDD-treated groups had a similar incidences of fibrosarcoma in the integumentary system (8/27 and 8/29, respectively), compared to the vehicle control of 2/41. In NTP's judgment, the results of this experiment indicated that TCDD was carcinogenic.

HexaCDDs have been tested for carcinogenicity by NTP (1980a) in both Osborne-Mendel rats and B6C3F₁ mice. The bioassay tested a mixture of HexaCDDs containing 31 percent 1,2,3,6,7,8-HexaCDD and 67 percent 1,2,3,7,8,9-HexaCDD. Lower chlorinated PCDDs made up the remaining 2% of the mixture, including 0.04 percent TetraCDDs. Male and female rats and male mice received weekly doses of 1.25,

2.5 or 5 µg/kg, administered by gavage twice a week. The female mice were administered doses of 2.5, 5.0, or 10 µg/kg/week.

A dose-related "toxic hepatitis", which was noninflammatory and consisted of degenerative changes in the liver, was observed in treated rats. The treated groups of female rats had significantly increased incidences of liver neoplastic nodules. Four high-dose animals were diagnosed as having hepatocellular carcinoma. The mice also had a dose-related incidence of "toxic hepatitis" and the high-dose male and female mouse groups had statistically significant increased incidences of hepatocellular adenomas and combined incidences of hepatocellular adenomas and carcinomas. The incidences of these tumors are given in Table 5.

Several pathologists have independently evaluated the slides made from the female rat livers in this bioassay. The re-evaluations found fewer neoplastic nodules and carcinomas than did the original evaluation. Although the incidences of neoplastic nodules and carcinomas are probably lower than originally reported, the incidence is still significant in the high-dose group. The results of four separate evaluations of the liver pathology of the female rats are given in Table 6.

A dermal application carcinogenicity bioassay of the same mixture of HexaCDD in male and female Swiss-Webster mice was also conducted by NTP (1980b). This study was similar to the TCDD dermal oncogenicity bioassay in its protocol. Thirty mice of each sex were treated with 0.005 µg of the dioxin mixture three times per week for the first 16 weeks, which was increased to 0.01 µg thereafter. A similar group was initially treated once with 50 µg DMBA before being treated with the HexaCDD mixture. Thirty untreated and 45 vehicle-treated mice of each sex were used as controls. Although there was a slight increase in fibrosarcomas of the integumentary system, this was not considered by NTP to be a significant carcinogenic response. DMBA pretreatment had no additional effect.

DHS staff members agreed with IARC (1982) that there is adequate evidence to support a conclusion that TCDD is carcinogenic to rats and mice and that TCDD should be considered a potential carcinogen to humans. The NTP bioassays (NTP 1980a) of HexaCDDs also indicated that the mixture used was tumorigenic.

Table 5: Tumor incidences in female Osborne-Mendel rats and male and female B6C3F₁ mice given HexaCDD by gavage for two years (NTP, 1980a)

Sex, species, tumor type	Dose level (µg/kg-week)			
	0	1.25 (2.5)	2.5 (5.0)	5.0 (10)
	Tumor incidence			
female rat liver, neoplastic nodule or hepatocellular carcinoma	5/75	10/50 (<i>p</i> = 0.026)	12/50 (<i>p</i> = 0.007)	30/50 (<i>p</i> < 0.001)
male mice				
liver, hepatocellular adenoma	7/73	5/50	9/49	15/4 (<i>p</i> = 0.003)
liver, hepatocellular adenoma or carcinoma	15/73	14/50	14/49	24/48 (<i>p</i> = 0.001)
female mice				
liver, hepatocellular adenoma	2/73	4/48	4/47	9/47 (<i>p</i> = 0.003)
liver, hepatocellular adenoma or carcinoma	3/73	4/48	6/47	10/47 (<i>p</i> = 0.004)

P values determined using Fisher's exact test.

^a Dose administered to male mice; dose administered to female mice in parentheses.

^b Number of animals with tumor over number of animals examined.

Table 6: Incidence of liver tumors based on four separate pathological evaluations of female rats given HexaCDD by gavage for two years^a (CDHS, 1986)

Pathologist and Diagnosis	dose level (µg/kg-week)			
	0	1.25	2.5	5
	Tumor incidence ^b			
NTP (1980) Neoplastic nodules or hepatocellular carcinoma	5/75	10/50 <i>p</i> = 0.026	12/50 <i>p</i> = 0.007	30/50 (4) ^c <i>p</i> < 0.001
Squire (1983) Neoplastic nodules	1/75	4/50	7/50 <i>p</i> = 0.007	7/50 <i>p</i> = 0.007
Haberman and Schueler (Schueler 1983) Neoplastic nodules or hepatocellular carcinoma	NA	NA	NA	17/50 (3) ^d
Hildebrandt (1983) Neoplastic nodules or hepatocellular carcinoma	1/75	5/50 <i>p</i> = 0.037	7/50 <i>p</i> = 0.007	18/50(2) <i>p</i> < 0.001

^a Chi-square test for trend in proportions for NTP, Squire, and Hildebrandt studies significant at $\alpha = 0.05$ level.

^b Number of animals with tumor over number of animals examined.

^c Number of animals diagnosed with hepatocellular carcinoma is shown in parentheses.

^d The diagnosis for nine of the animals with neoplastic nodules was considered a matter of judgment by the pathologist.

NA = Not available.

IV. DERIVATION OF CANCER POTENCY

Basis for Cancer Potency

Several human epidemiological studies of PCDD exposure reviewed in the dioxin TAC document (CDHS, 1986) reported results which suggested an increase in cancer incidence or mortality associated with PCDD exposure (Hardell and Sandstrom, 1979; Ericksson *et al.*, 1981; Zack and Gaffey, 1983). However, these and the other studies described in the dioxin TAC document suffer from a number of limitations. The characterization of exposure to PCDD/PCDF were at best, uncertain. Usually the exposure occurred at a time when there were no sensitive measures of exposure levels. Exposure was often based on job title, self-reported use of substances which may have had PCDD contamination, or exposure to an event thought to have liberated PCDDs. Additionally, none of the human exposures described have been solely to PCDDs or PCDFs, but rather to a mixture of chemicals. PCDDs were only trace contaminants of other toxic chemicals. Many of the occupationally exposed subjects were exposed only briefly (e.g., during an accidental release), or worked in a possibly contaminated environment for a short time. For example, more than 75% of the workers studied by Ott *et al.* (1980) had been exposed for less than one year. Finally, many of the discussed studies, including the four US cohorts, have been hampered by small samples. Studies of only a few hundred subjects lack sufficient power to detect small increases in the risk of rare tumors. For these reasons, DHS staff members concluded that the epidemiologic data available at the time the dioxin TAC document was written provided insufficient information to conclude whether or not PCDDs or PCDFs are human carcinogens.

CDHS (1986) found that the most sensitive species, sex, and site for the induction of cancer by TCDD is the male mouse with hepatocellular adenomas or carcinomas (NTP, 1982a). This response is an order of magnitude greater than the least sensitive species, sex, and site examined, the female mouse subcutaneous fibromas. It is interesting to note that there is less than a four-fold difference in the unit risk between animal species for liver tumors. CDHS therefore developed an inhalation cancer unit risk value for TCDD based on the NTP (1982a) male mouse hepatocellular adenoma/carcinoma tumor data. CDHS also developed an inhalation cancer unit risk value for HexaCDD based on the most sensitive species, sex, and site for the induction of cancer. The data set chosen was the NTP (1980b) female rat liver neoplastic nodule or hepatocellular carcinoma incidence data as evaluated by Hildebrandt (1983).

Methodology

GLOBAL79 was used to fit a linearized multistage procedure to the NTP (1982a) male mouse hepatocellular adenoma/carcinoma tumor data for TCDD, and the NTP (1980b) female rat neoplastic nodule/hepatocellular carcinoma data for HexaCDD as evaluated by Hildebrandt (1983). This procedure provided point estimates of the extra risk for both the maximum likelihood estimate (MLE) and the linearized 95% upper confidence value (UCL). The UCL is calculated by maximizing the linear term of the procedure, or forcing a best fitting linear term if one is not present. This method of calculating the

UCL is consistent both with the expected low-dose linearity and the linear nonthreshold theory of carcinogenesis. The slope of the 95% UCL, q_1^* , is taken as a plausible upper bound of cancer potency of TCDD at low doses.

The animal exposure data (NTP 1980a, 1982a) was converted into equivalent human exposures by applying appropriate scaling factors. The following assumptions were made: Oral and inhalation routes are equivalent, the concentration of TCDD in the air was assumed to be the daily oral dose, the route of exposure does not affect absorption, and there is no difference in metabolism and pharmacokinetics between animals and humans. The total weekly dose levels were averaged over the entire week to get the daily dose level. This procedure assumes that daily dosing of the animals in the NTP studies would have given the same results as did the actual twice weekly dosing schedule. Since the half-life of TCDD is relatively long, both dosing schedules should produce similar concentrations of TCDD in the animal tissues, and therefore would be expected to give similar results. The calculated daily doses are given in Table 7. Human equivalent exposures are listed in Table 8.

Because the animal dose levels for TCDD were converted to human equivalent exposure from inhalation, the 95% UCL, q_1^* , is a measure of the greatest potential excess cancer risk for humans. If the lifetime daily exposure is expressed in $\mu\text{g}/\text{m}^3$, then q_1^* is the excess risk associated with this exposure. Since q_1^* for humans is a unit measure of excess lifetime cancer risk associated with exposure to TCDD, it is termed the unit risk. With the unit risk, the 95% UCL of excess risk may be calculated for any low-level exposure to TCDD by the equation $R = \text{unit risk} \times \text{dose}$, where R is the 95% UCL of excess lifetime cancer risk. The cancer unit risks calculated by CDHS using the above procedure for TCDD and HexaCDD were $38 (\mu\text{g}/\text{m}^3)^{-1}$ and $1 (\mu\text{g}/\text{m}^3)^{-1}$, respectively.

Table 7: Calculated daily dose levels for NTP (1980a, 1982a) TCDD and HexaCDD chronic studies in rats and mice (CDHS, 1986)

Chemical	Animal	Reported Dose Level ($\mu\text{g}/\text{kg}\text{-week}$)	Calculated Dose Level ($\mu\text{g}/\text{kg}\text{-day}$)
TCDD	male and female rats, male mice	0.01	0.0014
		0.05	0.0071
		0.5	0.071
	female mice	0.04	0.0057
		0.2	0.029
		2.0	0.29
HexaCDD	female rats	1.25	0.18
		2.5	0.36
		5.0	0.71
	female mice	2.5	0.36
		5.0	0.71
		10	1.40

Table 8: Calculated equivalent human exposure to TCDD and HexaCDD based on daily animal dose levels from NTP (1980a, 1984a) carcinogenicity studies (CDHS, 1986)

Chemical	Animal	Daily Dose Level ($\mu\text{g}/\text{kg}\text{-day}$)	Airborne Concentration for Equivalent Human Exposure (ng/m^3)
TCDD	female rat (0.45) ^a	0.0014	0.93
		0.0071	4.6
		0.071	46
	male mice (0.048)	0.0014	0.44
		0.0071	2.2
		0.071	22
	female mice (0.04)	0.0057	1.7
		0.029	8.4
		0.29	84
HexaCDD	female rats (0.45) a	0.18	120
		0.36	230
		0.71	460
	female mice (0.04)	0.36	100
		0.71	210
		1.43	420

^a Number in parentheses is animal body weight in kilograms.

CDHS recognized that total PCDD/PCDF in the air is composed of dozens of PCDD and PCDF homologues and isomers. The chemicals in such a mixture are difficult to - quantitate analytically. As a result, usually only total PCDD and total PCDF are measured. In the Air Toxics Hot Spots program, certain dioxin sources are required to perform stack testing and speciate the 2,3,7,8-congeners. Thus, more data are becoming available to adequately characterize the risk from dioxin sources in California.

To estimate cancer risks from such mixtures requires information about: (1) the proportion of each PCDD and PCDF in the mixture, and (2) the carcinogenic potency of each. However, these data are not generally available. The proportion of isomers differs depending on the emission source, and only three isomers had been tested for carcinogenic potency (2,3,7,8-TCDD and a mixture of 1,2,3,6,7,8- and 1,2,3,7,8,9-HexaCDD). It was also recognized that not all 2,3,7,8-isomer PCDDs and PCDFs are equally carcinogenic. The results of the bioassays on TCDD and HexaCDD suggested that carcinogenic potency may decline in homologues more chlorinated than TCDD. It was therefore assumed that PCDDs and PCDFs that are not chlorinated on the 2,3,7,8 positions or do not have at least one ring position open are noncarcinogenic. Additionally, it was also considered that the 2,3,7,8-isomer PentaCDD has a carcinogenic potency equivalent to TCDD, and that 2,3,7,8-isomer HeptaCDD is equivalent in carcinogenic potency to 2,3,7,8-isomer HexaCDD. The potencies for the homologous PCDDs were also used for the PCDFs. Using this approach, the potency of a given concentration of PCDDs would be 2% of the potency of TCDD. The potency of a mixture of PCDFs would be 3% of the potency of TCDD.

Another toxicity equivalency factor (TEF) scheme was developed after 1986 during an international symposium (NATO/CCMS, 1988a,b), and it was adopted by US EPA (US EPA, 1989) and the Department of Toxic Substances Control (DTSC) (DTSC, 1992). The international scheme, referred to as ITEFs, is based on experimental cancer and noncancer data for many 2,3,7,8-PCDDs and 2,3,7,8-PCDFs and on the assumption that the mechanism of all PCDD/PCDF-related biologic effects are based on initial binding to a specific protein, the *Ah* receptor. Because the ITEF scheme incorporated more experimental data from cancer and noncancer studies for more PCDDs/PCDFs than does the CTEF scheme, the replacement of the CTEFs by the ITEFs was considered appropriate for use in risk assessment. This approach also increases uniformity among Cal/EPA guidelines. The TEFs contained in the dioxin TAC (CDHS, 1986) document and the ITEFs are listed in Table 8. The cancer unit risks and potency factors for chlorinated dibenzo-*p*-dioxins and dibenzofurans listed in the 1999 chemical summary and Hot Spots Unit Risk and Cancer Potency Values table (OEHHA, 1999) were generated by applying the appropriate ITEFs to the cancer unit risk and potency factor for 2,3,7,8-TCDD calculated in the dioxin TAC document.

As TEFs for PCDDs and PCDFs were developed, considerable efforts went into the study of quantitative structure activity relationships (QSAR) for polychlorinated biphenyls (PCBs). PCB congeners substituted in the para and at least 2 of the meta positions but not at any of the ortho positions can adopt structural conformations most resembling that of 2,3,7,8-TCDD, therefore have the greatest potency and exert their toxicity through the *Ah* receptor pathway. These coplanar PCB congeners are structurally similar to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin and therefore are termed dioxin-like PCBs. Introduction of one chlorine in the ortho position results in a decrease in toxic potency and PCBs with more than one chlorine in the ortho positions lack some effects exerted by non- and mono-ortho PCBs. These PCB congeners show a different spectrum of toxic effects (Safe, 1994).

In 1991, U.S. EPA considered using the TEF methodology for PCBs. They noted that only a small subset of the 209 PCB congeners elicits dioxin-like activity and meet the criteria for inclusion in the TEF methodology. In an attempt to harmonize TEF schemes for dioxin-like compounds, the World Health Organization - European Center for Environmental Health (WHO-ECEH) and the International Program on Chemical Safety (IPCS) generated a database consisting of almost 1,200 peer-reviewed publications, representing all the available toxicological data for PCBs up to the end of 1993. From a selected number of these publications and based on four inclusion criteria, the WHO-ECEH and the IPCS proposed TEF values for 13 dioxin-like PCBs (Ahlborg *et al.*, 1994). The inclusion criteria are:

1. The compound should show structural similarity to PCDDs and PCDFs.
2. It should bind to the *Ah* receptor.
3. It should induce dioxin-specific biochemical and toxic responses.
4. It should be persistent and accumulate in the food chain.

In addition, the first WHO PCB TEF consultation (Ahlborg *et al.*, 1994) recommended expanding the current database to include all relevant information on PCDDs, PCDFs and other dioxin-like compounds that satisfied the four inclusion criteria.

Some terminologies and definitions applicable to TEFs were reviewed prior to the second WHO-ECEH consultation (van Leeuwen, 1997). The term TEF, used in the past to describe any experimental end point to be compared with TCDD was reconsidered since not all end points are “toxic” end points. For example, end points such as binding to the *Ah* receptor and induction of ethoxyresorufin-O-deethylase (EROD) are mostly considered biological/biochemical responses. Therefore, experimental end points, for which numerical values are compared to the response to TCDD, should be termed “Relative Potency” values (REPs). These REPs could be the result of a single laboratory experiment looking at a single end point. REPs are derived from the available data either used as reported in each publication, or calculated by comparing dose-response curves or ratios of medium effective doses (ED₅₀), median lethal dose (LD₅₀), median effective concentration (EC₅₀) etc. A chemical’s TEF is then derived from all available REPs examined for that compound. Thus, the term TEF is restricted to describe an overall estimate of the order-of-magnitude of the toxicity of a compound relative to the toxicity of TCDD. This estimate is derived by consensus, using careful scientific judgment of all available data (van Leeuwen, 1997; van den Berg *et al.*, 1998). The derivation of TEF consensus using *Ah* receptor-specific end points gives more weight to toxic responses than to biochemical (e.g., enzyme induction) responses and it puts more weight on *in vivo* data than on *in vitro* results. In fact, the weighting order of contributing *in vivo* data was: chronic > subchronic > subacute > acute.

In its most recent consultation in 1997, the WHO-ECEH proposed amendments to the previous NATO/WHO I-TEF scheme (NATO/CCMS, 1989). For revision of the existing mammalian TEFs, the WHO-ECEH committee agreed that if the available information was considered insufficient to warrant a change, the existing value would remain. The suggested WHO₉₇ TEFs for humans and mammals along with the CTEFs and ITEFs are presented in Table 9. Taking advantage of new data and understanding of the underlying mechanisms of toxicity of dioxin-like compounds, the WHO-ECEH’s re-evaluation and extension of the TEF concept lead to the following amendments:

1) For 1,2,3,7,8-PeCDD, an increase in TEF value from 0.5 to 1.0 was recommended, based on new *in vivo* tumor promotion data and CYP 1A1/A2 induction potencies from subchronic studies.

2) For OCDD, the TEF value was reduced from 0.001 to 0.0001 based on a recalculation of the old data in which exposure versus tissue concentrations were compared (administered dose); originally the TEF was based on body burdens of the chemical following subchronic exposures.

3) For OCDF, the TEF value was changed from 0.001 to 0.0001 based on new *in vivo* EROD induction potency values (81) and an expected structural similarity with OCDD; thus, for the *in vivo* situation, a change in analogy with OCDD is recommended.

The Scientific Review Panel on Toxic Air Contaminants (SRP) reviewed and endorsed the use of the WHO₉₇ TEFs in Hot Spots risk assessments at its June 20, 2003 meeting. The cancer unit risks and potency factors for chlorinated dibenzo-*p*-dioxins and dibenzofurans and polychlorinated biphenyls listed in this chemical summary and the Hot Spots Unit Risk and Cancer Potency Values table were generated by applying the appropriate WHO₉₇ TEFs to the cancer unit risk and potency factor for 2,3,7,8-TCDD calculated in the dioxin TAC document.

Table 9: Toxicity equivalency factors for chlorinated dibenzo-*p*-dioxins and dibenzofurans (relative to 2,3,7,8-TCDD)

Congener	California TEF ^a	I-TEF ^b	TEF _{WHO/97} ^c
PCDDs			
2,3,7,8-TCDD	1	1	1
1,2,3,7,8-PeCDD	1	0.5	1
1,2,3,4,7,8-HxCDD	0.03	0.1	0.1
1,2,3,6,7,8-HxCDD	0.03	0.1	0.1
1,2,3,7,8,9-HxCDD	0.03	0.1	0.1
1,2,3,4,6,7,8-HpCDD	0.03	0.01	0.01
1,2,3,4,6,7,8,9-OCDD		0.001	0.0001
PCDFs			
2,3,7,8-TCDF	1	0.1	0.1
1,2,3,7,8-PeCDF	1	0.05	0.05
2,3,4,7,8-PeCDF	1	0.5	0.5
1,2,3,4,7,8-HxCDF	0.03	0.1	0.1
1,2,3,6,7,8-HxCDF	0.03	0.1	0.1
1,2,3,7,8,9-HxCDF	0.03	0.1	0.1
2,3,4,6,7,8-HxCDF	0.03	0.1	0.1
1,2,3,4,6,7,8-HpCDF	0.03	0.01	0.01
1,2,3,4,7,8,9-HpCDF	0.03	0.01	0.01
1,2,3,4,6,7,8,9-OCDF		0.001	0.0001
PCBs (IUPAC #, Structure)			
77	3,3',4,4'-TCB		0.0001
81	3,4,4',5-TCB		0.0001
105	2,3,3',4,4'-PeCB		0.0001
114	2,3,4,4',5-PeCB		0.0005
118	2,3',4,4',5-PeCB		0.0001
123	2',3,4,4',5-PeCB		0.0001
126	3,3',4,4',5-PeCB		0.1
156	2,3,3',4,4',5-HxCB		0.0005
157	2,3,3',4,4',5'-HxCB		0.0005
167	2,3',4,4',5,5'-HxCB		0.00001
169	3,3',4,4',5,5'-HxCB		0.01
189	2,3,3',4,4',5,5'-HpCB		0.0001

Value introduced or changed

^a CDHS, 1986

^b NATO/CCMS, 1989.

^c van Leeuwen, 1997.

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PARTICULATE MATTER FROM DIESEL-FUELED ENGINES

CAS No: not available

I. PHYSICAL AND CHEMICAL PROPERTIES (From HSDB, 1998)

Molecular weight	not applicable
Boiling point	not applicable
Melting point	not applicable
Vapor pressure	not applicable
Air concentration conversion	not applicable

II. HEALTH ASSESSMENT VALUES

Unit Risk Factor: $1.3 \text{ E-4} - 1.5 \text{ E-3} (\mu\text{g}/\text{m}^3)^{-1}$ (measured as particulate matter)[Scientific Review Panel unit risk “reasonable estimate” = $3.0 \text{ E-4} (\mu\text{g}/\text{m}^3)^{-1}$.]

Slope Factor: $1.1 \text{ E+0} (\text{mg}/\text{kg}\text{-day})^{-1}$

[Human occupational exposure lung tumor incidence (Garshick *et al.* (1987a, 1988), estimated exposure concentrations (Woskie *et al.*, 1988a,b), relative risk model (OEHHA, 1998); human occupational exposure lung tumor incidence, meta-analysis (OEHHA, 1998).]

III. CARCINOGENIC EFFECTS

Human Studies

The epidemiological evidence concerning the carcinogenicity of diesel exhaust primarily involves cancers of the lung and bladder. The review of human diesel exhaust-exposure cancer studies in the document entitled *Health Risk Assessment For Diesel Exhaust* written for the Toxic Air Contaminant (TAC) program (OEHHA, 1998) focuses first on studies of lung cancer (Sections 6.2.1 and 6.2.2) and then turns to those of bladder cancer (Section 6.2.3). The evidence for causation of lung cancer was then assessed using criteria for causal inference from epidemiological studies (Section 6.2.4). The evidence linking diesel exposure and bladder cancer was not as extensive or compelling, and is discussed in the diesel exhaust TAC document but not in this summary. Because there are no epidemiological studies involving industrial hygiene measurements concurrent with the exposures of the study populations, exposure has typically been defined by the surrogate measures of usual occupation or job classification within an industry.

Review Of Lung Cancer Studies

The question of whether diesel exhaust causes lung cancer has been addressed by both industry-based cohort and case-control studies as well as population-based studies of lung cancer. In Section 6 of the diesel exhaust TAC document (OEHHA, 1998), the review of the lung cancer studies was divided into five parts focusing on studies of: (1) truck drivers, (2) transport and equipment workers, (3) dock workers, (4) railway workers, and (5) other miscellaneous

occupations involving diesel exhaust exposure. This summary will focus on the railway workers studies, which were used to derive the range of human cancer risks associated with diesel exhaust exposure. A summary of all occupational studies evaluating the relationship between diesel exhaust exposure and lung cancer is provided in Table 1.

Studies Of Lung Cancer Among Railway Workers

In 1959, Kaplan studied lung cancer mortality among employees of the Baltimore and Ohio Railroad. This railroad initiated locomotive dieselization in 1935, completing this process by 1958. Workers employed at any time between 1953 and 1958 were eligible for entry into the cohort; 154 deaths from primary cancers of the lung or bronchus were identified. Exposure was categorized into three groups by job type. The lung cancer SMR for the most exposed group, relative to the general population, was 0.875. The limited number of years of exposure to diesel exhaust for some members of the cohort and the abbreviated follow-up time do not allow for sufficient latency to be informative regarding the relationship of diesel exhaust exposure to lung cancer. In addition, no data on smoking were available.

In the Third National Cancer Survey discussed above, Williams *et al.* (1977) found a nonsignificant increased risk for railroad workers among lung cancer patients, OR = 1.40, based on 12 cases (no confidence intervals reported).

Howe *et al.* (1983) carried out a mortality study of 43,826 male pensioners of the Canadian National Railroad. The cohort consisted of all male pensioners who were alive at the beginning of 1965. Subjects were followed until 1977, by which time 933 deaths from respiratory cancer (trachea, bronchus and lung) had been recorded. Occupations at the time of retirement were classified as “nonexposed”, “possibly exposed” or “probably exposed”. Analysis restricted to individuals retiring after 1950 (n = 897 cases) yielded relative risks of 1.00, 1.20 ($p = 0.013$), and 1.35 ($p < 0.001$) for the three exposure groups, respectively (test for trend: $p < 0.001$). There was little change in these effect estimates when individuals involved in locomotive maintenance (and who therefore may have been exposed to asbestos) were excluded from the analysis (n = 69).

This study also found coal dust to be associated with lung cancer, with a similar increasing trend with degree of exposure. Because of a high degree of overlap between exposures to coal dust and to diesel exhaust, the authors could not separate the effects of the two. However, since there is evidence from animal and human studies for the carcinogenicity of diesel exhaust, but such evidence does not exist for coal dust, the apparent effect of coal dust was more likely to have been due to confounding by diesel exhaust, rather than vice versa. No smoking information was available for this study, although there were increasing trends with degree of diesel exposure for mortality from emphysema (SMRs = 1.00, 1.35, and 1.44) and other smoking-related cancers combined (SMRs = 1.00, 1.08, and 1.16). The authors suggested that since the results were based on internal comparisons little variation in smoking would be expected among the different diesel exposure groups.

Garshick *et al.* (1987a) carried out a case-control study of lung cancer in U.S. railroad workers. Cases comprised 1,256 lung cancer deaths occurring between 1981 and 1982 in the population of

active or retired railroad workers who had had 10 years or more of railroad service and were born in 1900 or later. Two controls who had died of causes other than cancer, suicide or accident were matched to each case by dates of birth and death. Next of kin were interviewed to obtain information about the decedents, including their smoking habits. Job codes were obtained from the Railroad Retirement Board, and an industrial hygiene survey was used to classify the degree of diesel exposure for each job type. Jobs were dichotomously categorized as exposed or not exposed to diesel exhaust.

Garshick *et al.* considered exposure to diesel exhaust to have begun in 1959, since the transition from steam to diesel-powered locomotives took place mainly in the 1950s, and was nearly complete in 1959. Years of diesel exhaust exposure to death or retirement were totaled for each worker. The analysis separated those workers who died at age 65 (retirement age) or older (921 cases and 1,748 controls) from those workers <64 years at death (335 cases and 637 controls). Analysis by logistic regression showed no effect of diesel exhaust in the workers in the older age category, who had substantially less diesel exposure than those in the younger category. For example, 36% of cases and 43% of controls had no exposure in the younger group, while 52% of cases and 53% of controls had no exposure in the older group. Furthermore, 35% of cases and 26% of controls had more than 19 years of diesel exposure in the younger group, while only 3% of cases and controls had more than 19 years of diesel exposure in the older group.

In the group whose members were younger than 64 years old at time of death, the analysis by Garshick *et al.* showed evidence of an exposure-response relationship with an OR of 1.41 (95% C.I. = 1.06-1.88) for 20 or more years of exposure (diesel-years) after adjusting for smoking and asbestos exposure. Excluding exposure occurring within five years of death, the OR for 15 or more years of cumulative diesel exposure was 1.43 (95% C.I. = 1.06-1.94). For workers with 5 to 14 years of cumulative exposure, the OR was 1.07 (95% C.I. = 0.69-1.66) relative to a reference category of 0 to 4 diesel exposure years.

Garshick *et al.* (1988) also conducted a retrospective cohort study of U.S. railroad workers. Eligible for inclusion in the cohort were white males aged 40 to 64 years, who started work between 1939 and 1949 and were still employed in 1959 in designated jobs. Follow-up extended through 1980. Jobs with recognized asbestos exposure were not included in the job codes selected for study, although some of the selected occupations had at least some potential for asbestos exposure. The cohort consisted of 55,407 men, among whom there were 19,396 deaths, including 1,694 attributable to lung cancer. Diesel exhaust exposure was characterized based on their 1959 job group. Career paths were found to be very stable in the railways, such that a worker aged 40-44 with a diesel-exposed job in 1959 was likely to have a diesel-exposed job 20 years later; similarly a nonexposed person in 1959 was likely to have a nonexposed job 20 years later.

The youngest workers in 1959 had the longest potential duration of diesel exposure in the cohort. In a proportional-hazards model these workers had the highest estimated relative risks for lung cancer associated with diesel exhaust exposure: the relative risk for the group aged 40-44 in 1959 was 1.45 (95% C.I. = 1.11-1.89); for the group aged 45-49 the relative risk was 1.33 (95% C.I. = 1.03-1.73); for the group aged 50-54, 1.12 (95% C.I. = 0.88-1.42); for the group aged 55-59, 1.18 (95% C.I. = 0.94-1.50); and for the group aged 60-64, 0.99 (95% C.I. = 0.74-1.33).

Though the results were statistically significant only for the two youngest groups, there was a decreasing trend with increasing age in 1959 (except for the 55-59 year age group), implying declining risk with decreasing duration of exposure.

When exposure to diesel over the last five years before death was excluded, a relationship was apparent between lung cancer risk and duration of exposure. The group with greater than 15 years of cumulative exposure had a RR for lung cancer of 1.72 (95% C.I. = 1.27-2.33); for those with 10 to 14 years of exposure the RR was 1.32 (95% C.I. = 1.13-1.56); for 5 to 9 years, 1.24 (95% C.I. = 1.06-1.44); and for 1-4 years, 1.20 (95% C.I. = 1.01-1.44). All of these results are statistically significant.

Although no smoking information was available for the cohort, the previous case-control study of railway workers by the same group (Garshick *et al.*, 1987a) reported that little change occurred in the estimates of diesel exhaust effect due to adjustment for smoking habits and asbestos exposure (unadjusted OR = 1.39, 95% C.I. = 1.05-1.83; adjusted OR = 1.41, 95% C.I. = 1.06-1.88). In this analysis, the larger percentage of workers whose pack-year history was unknown (23% of cases and 22% of controls) was treated as a separate category of smoking. In additional analyses using only those workers for whom the investigators had detailed smoking data (n = 758), the ORs for 20 years of diesel exposure ranged from 1.50-1.53, adjusted for asbestos exposure and several specifications of cigarette smoking history. These models included pack-years as a single continuous variable, as two independent variables (cigarettes per day and years of smoking), or as a categorical variable classified in terms of the number of years the study subject had stopped smoking prior to death. These analyses suggested that the diesel exhaust-lung cancer odds ratios were not confounded by cigarette smoking in this population. Moreover, in a group of railroad workers previously surveyed for asbestos exposure (Garshick *et al.*, 1987b) there was no difference in smoking prevalence between workers with and without diesel exhaust exposure (data not presented).

It should be noted that the case-control and the cohort studies by Garshick *et al.* involved different study populations: The case-control study (Garshick *et al.* 1987a) contained cases and controls who had died in 1981 and 1982, whereas the cohort study (Garshick *et al.*, 1988) involved deaths occurring up to 1980. Thus, they may be considered different tests of the hypothesis of an association between lung cancer and diesel exhaust exposure, although this does not exclude the possibility of a common bias shared by the two studies, such as exposure to chemicals transported by rail or to suspended dusts and debris.

In the American Cancer Society prospective mortality study mentioned above (see Section 6.2.1.1, OEHHA, 1998), Boffetta *et al.* (1988) found an age- and smoking-adjusted RR of 1.59 (95% C.I. = 0.94-2.69) for lung cancer mortality in railroad workers. This estimate was based on only 14 lung cancer deaths.

Swanson *et al.* (1993) also examined the industrial category of railroad workers in their case-control study of lung cancer. The smoking-adjusted odds ratios for white males (67 cases) were 1.2 (95% C.I. = 0.5-2.7) for 1-9 years of employment and 2.4 (95% C.I. = 1.1-5.1) for more than 10 years of employment (χ^2 test for trend: $p < 0.05$). Elevated, but nonsignificant, smoking-adjusted ORs were also associated with the 31 lung cancer cases occurring in African-American

railroad workers, OR = 2.6 (95% C.I. = 0.8-7.9) for 1-9 years and OR = 2.7 for ≥ 10 years of employment (95% C.I. = 0.6-12.1).

Nokso-Koivisto and Pukkala (1994) compared the incidence of lung cancer among locomotive drivers to the total Finnish population. The retrospective cohort consisted of the 8,391 members of the Finnish Locomotive Drivers' Association from 1953 until 1991 (retired drivers remain members until death). After excluding 302 members for lack of personal identification information, an overall standardized incidence ratio (SIR) of 0.86 (95% C.I. = 0.75-0.97) was found (236 cases). The overall incidence for all cancer sites was also lower than expected, SIR 0.95 (95% C.I. = 0.89-1.01) but the incidence of mesothelioma (SIR 4.05, 95% C.I. = 1.75-7.97) and oral cavity/pharyngeal cancers (SIR 1.75, 95% C.I. = 1.02-2.80) were significantly increased. Prior to the 1970s Finnish drivers trained for 2 years in railroad workshops, where significant exposure to asbestos occurred routinely during steam engine maintenance, with little, if any, diesel exposure. Only drivers working after this period had the potential for substantial exposure to diesel exhaust, and the electrification of the railroad in the 1970s and 1980s may also have reduced the proportion of the cohort's person-years that truly reflect exposure to diesel exhaust. No data on smoking within the cohort were available, though a cross-sectional study of locomotive drivers in 1976 showed that the prevalences of current smokers (40%), ex-smokers (34%), and never-smokers (26%) were similar to those in the Finnish population as a whole.

All three population-based case-control studies found elevated risks for lung cancer in railroad workers (Williams *et al.*, 1977; Boffetta *et al.*, 1988; Swanson *et al.*, 1993); however, only the study by Swanson *et al.* (1993) found a statistically significant increase, with a smoking-adjusted OR of 2.4 (95% C.I. = 1.1-5.1) for workers with ten or more years of employment. This study also found evidence of a significant exposure-response relationship for the 67 cases observed in white railroad workers. Williams *et al.* (1977) and Boffetta *et al.* (1988) had relatively fewer railroad workers (12 and 14 cases respectively) and no information on duration of exposure.

In the railroad industry-based studies, three of the larger studies identified statistically significant increases in relative risk (Howe *et al.*, 1983; Garshick *et al.*, 1987a; Garshick *et al.*, 1988). The large cohort reported on by Howe *et al.* (1983) found elevated risks for individuals categorized as "probably" and "possibly" exposed to diesel exhaust, but without adjustment for smoking or duration of employment, the underlying risk is uncertain. In both the case-control and cohort studies by Garshick *et al.*, 1987a, 1988), significantly increased risks were associated with long-term employment in diesel-related railroad jobs. Both studies had substantial exposure assessment, sufficient latency, and duration of employment data, and the case-control investigation also controlled for potential confounding by smoking and by asbestos exposure. In contrast, the study by Nokso-Koivisto *et al.* (1994), found no increase in lung cancer risk among Finnish locomotive engineers, though the description of the cohort indicates the earlier cases were unlikely to have experienced any diesel exposure.

Studies Of Lung Cancer Among Truck Drivers

The studies that have examined the lung cancer risk to truck drivers are summarized in Table 1. These studies have consistently reported small increases in lung cancer relative risk. However, the studies suffer from various deficiencies, including small numbers of subjects, inadequate adjustment for confounding, and crude exposure assessments, usually based on occupational classification. Most of the earlier studies did not adjust for smoking. Because of evidence that truck drivers have a higher smoking prevalence (Wynder and Higgins, 1986), individual studies that do not account for smoking generally provide limited evidence regarding carcinogenicity. Before 1988, the two studies that took smoking into account, Williams *et al.* (1977) and Hall & Wynder (1984), had ORs of 1.4 - 1.5, which were not statistically significant. The third study that accounted for smoking (Damber and Larsson, 1985, 1987), only found significantly elevated risks in truck drivers who smoked after stratifying on age (i.e., only for those > 70 years old at diagnosis). However, in the follow-up study, after analyzing for duration of employment (20 or more years), elevated but nonsignificant risks were observed for all professional drivers combined (Damber and Larsson, 1987).

By comparison, the majority of studies published since 1988 have adjusted for smoking to varying degrees. Of the smoking-adjusted population based studies, two of four found statistically significant increases in the relative risk for lung cancer associated with occupation as a truck driver, especially in individuals employed for 10 or more years (Hayes *et al.* 1989; Swanson *et al.* 1993). In addition, both studies reported some evidence of a positive trend between increased duration of employment and risk for lung cancer. Although both found statistically significant trends ($p < 0.05$), the only stratum with statistically significant relative risk estimates was that including 20 or more years' employment as a truck driver, with ORs of 1.5 (95% C.I. = 1.0-2.3) and 2.5 (95% C.I. = 1.1-4.4), reported by Hayes *et al.* (1989) and Swanson *et al.* (1993), respectively.

Three of the six more recent industry-specific studies adjusted for smoking, at either the individual (Benhamou *et al.* (1988) and Steenland *et al.* (1990)) or group level (Pfluger and Minder 1994). The two studies of professional drivers, a portion of which included truck drivers, found significantly elevated estimates of relative risk with smoking-adjusted ORs of 1.42 (95% C.I. = 1.07-1.89) and 1.48 (95% C.I. = 1.30-1.68) (Benhamou *et al.*, 1988 and Pfluger and Minder, 1994, respectively). The one smoking-adjusted study focusing on trucking, Steenland *et al.* (1990), found elevated relative risk estimates for several occupational and duration of employment categories; however, the only statistically significant risk estimate found was for diesel truck drivers with greater than 34 years of exposure, (OR = 1.89; 95% C.I. = 1.04-3.42).

While several population-based studies enrolled a large number of subjects overall (Williams *et al.* 1977; Milne *et al.*, 1983; Hall and Wynder, 1984; Damber and Larsson, 1987; Boffetta *et al.* 1988), the actual numbers of subjects occupationally exposed to diesel exhaust (considered here as truck drivers) were small. Of the larger, general population studies (Hayes *et al.*, 1989; Benhamou *et al.*, 1988; Boffetta *et al.*, 1990; Swanson *et al.*, 1993) and industry- or occupation-specific studies (Ahlberg *et al.*, 1981; Rafnsson and Gunnarsdottir, 1991; Guberan *et al.*, 1992; Hansen *et al.*, 1993; Pfluger and Minder, 1994; Steenland *et al.*, 1990) with greater numbers of truck drivers, significantly elevated smoking-adjusted risk estimates were limited mainly to the

case-control studies described above (Hayes *et al.*, 1989; Benhamou *et al.*, 1988; Steenland *et al.*, 1990; Swanson *et al.*, 1993; Pfluger and Minder, 1994). Although several industry-specific cohort studies found significantly elevated risks associated with truck or professional driving, with SMRs ranging between 1.33 and 2.14, all lacked smoking data.

Studies Of Lung Cancer Among Transport Workers

Table 1 summarizes the studies that have examined the lung cancer risk to truck drivers. Most studies of transportation workers are limited by small sample size, lack of smoking data, or limited follow-up. None of the three studies of London transportation workers, drivers or garage workers, (Raffle, 1957; Waller, 1981; Rushton *et al.*, 1983) obtained information on smoking. In addition, two lacked sufficient follow-up (Raffle, 1957; Rushton *et al.*, 1983), excluded retirees, or suffered from small sample size (Raffle, 1957; Waller, 1981). Of the other European studies focusing on bus company employees (Edling *et al.*, 1987; Netterström, 1988; Gustavsson *et al.*, 1990), only Gustavsson *et al.* (1990) found an elevated risk for lung cancer, with an overall SMR of 1.22 (95% C.I. = 0.71-1.96). However, in the more detailed nested case-control analysis using conditional logistic regression, estimated RRs increased with the cumulative diesel-exhaust exposure index, as noted above.

Of the three studies reporting increased risks for heavy equipment operators (Wong *et al.*, 1985; Boffetta *et al.*, 1988; Hayes *et al.*, 1989), only the RR reported by Boffetta *et al.* (1988) was statistically significant (RR = 2.6; 95% C.I. = 1.12-6.06). However, this estimate was based on only five lung cancer deaths. The large industry-specific cohort study of Wong *et al.* (1985) did not find an elevated risk for lung cancer among unionized heavy equipment operators (SMR = 0.99; 95% C.I. = 0.88-1.10). A subset of individuals retiring at age 65 did have a significantly elevated risk, but a group excess in emphysema deaths (SMR = 2.75; 95% C.I. = 2.09-3.55) and the absence of smoking data suggest that the increased risk may have been related more to tobacco use than to diesel exhaust exposure.

Table 1: Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Truck Drivers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval ^a or P-Value	Comments
Menck and Henderson, 1976 USA	Cohort Truck drivers	109	SMR 1.65	$p < 0.01$	Included 2,161 lung cancer cases identified from death certificates in white males, aged 20 to 64, from 1968 through 1970, and 1777 incident cases of lung cancer reported to LA County Cancer Surveillance Program for 1972 - 73. Occupational information obtained from death certificates or hospital admission sheets/medical records represented the last occupation and industry of employment. No data on smoking.
Decoufle <i>et al.</i> 1977 USA	Case-control Truck or tractor driver	56	OR 1.07	N.S.	Hospital-based study of 6,434 cancers cases admitted to Roswell Park Memorial Institute between 1956 and 1965. Controls were patients admitted with non-neoplastic disease. Occupation and smoking data obtained by questionnaire. Crude adjustment for smoking. Inadequate latency.
	≥ 5 years as truck, bus or taxi driver	50	0.89	N.S.	
Williams <i>et al.</i> 1977 USA	Case-control Transportation Industry	38	RR 1.17	N.S.	Study examined cancer incidence and its relation to occupation and industry based on the U.S. 3rd National Cancer Survey. The number of cases of cancer at various sites were compared with that of cases at all other sites combined. Occupational history (main and recent employment) and data on smoking were obtained by interview (n = 7,518). IARC noted the potential bias in this study due to the relatively low level of response to the questionnaire (57%). Results were controlled for tobacco use, alcohol consumption, race, education and geographic location.
	Truck drivers	22	1.52	N.S.	
	Railroad workers	12	1.40	N.S.	
	Truck Industry	13	1.34	N.S.	
Leupker and Smith, 1978 USA	Cohort Total cohort	34	SMR 1.21	N.S.	Death certificates for a 3-month period in 1976 in the Central States Teamster population were examined. Comparison group was the US male population and was not adjusted for race. No data on smoking. Authors noted the follow-up was short. Retirees and members with lapsed benefits were excluded. 48,358 members were eligible in the 50-59 age group.
	Age 50-59	not given	1.37	$p < 0.001$	
Ahlberg <i>et al.</i> 1981 Sweden	Cohort All truck drivers*	161	RR 1.33	1.13-1.56	Cohort consisted of 34,027 Swedish drivers considered to be exposed to diesel exhaust identified from the 1960 national census. Reference population consisted of blue-collar workers from the same census thought to have had no exposure to petroleum products or chemicals (n=686,708). No data on smoking; however, a study of 470 professional drivers in Stockholm found that 78% of fuel truck drivers and 31% of other truck drivers smoked compared to 40% in the Swedish population (citing unpublished study). # Subset of all non-fuel tank drivers. *Does not include fuel tank drivers.
	Stockholm truck drivers#		1.62	1.15-2.28	

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or p -values reported in original study. DE = Diesel Exhaust. OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Truck Drivers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval ^a or P-Value	Comments
Milne <i>et al.</i> 1983 USA	Case-control		OR		Study compared lung cancer deaths with mortality from all other cancers in Alameda County between 1958 and 1962 to investigate possible associations between lung cancer and occupation. Data on cause of death and occupation were obtained from death certificates. No data on smoking or the types of vehicle engines. Results reported are for males. *Results in parentheses are ORs with potential occupationally related cancer removed from the control population. Significant risk estimates only observed when compared with control group before such cancers removed.
	Occupational groups:				
	All transport operatives	36	1.3 (1.1)*	N.S.	
	Bus drivers	4	3.5 (2.8)*	$p < 0.05^*$	
	Truck drivers	23	1.6 (1.3)*	$p < 0.05^*$	
	Other transport	7	0.7 (0.6)*	N.S.	
Hall and Wynder, 1984 USA	Industry groups:				Study consisted of 502 men with histologically confirmed primary lung cancer (20 to 80 years old) and matched control patients in 18 hospitals in six cities. Controls with tobacco-related diseases were excluded. Patients were interviewed between December 1980 and November 1982. Smoking data were obtained. Occupations were grouped either dichotomously as exposed to diesel exhaust (warehousemen, bus drivers, truck drivers, railroad workers, heavy equipment operators) or unexposed. Exposure categorization also conducted by NIOSH-based occupational classifications with job title classified as having "probable" exposure to diesel exhaust as either "high" (10 cases), "moderate" (16 cases) or "little or none" (476 cases). No significantly elevated risks were reported in this latter analysis (data not shown here). See also Boffetta <i>et al.</i> , 1990. *Compared DE exposed to unexposed within each smoking category.
	Railroad	34	0.8 (0.8)*	N.S.	
	Case-control		OR		
	<u>Usual employment:</u>				
	Total diesel-exposed - adjusted for smoking	45	2.0 1.4	1.2-3.2 0.8-2.4	
	<u>Selected occupations:</u>				
Truck drivers					
Railroad workers					
Heavy equipment repairmen & operators	22	1.4	0.7-2.6		
- adjusted for smoking	5	2.6	0.5-12.8		
	10	3.5	1.0-11.8		
		1.9	0.6-5.5		
<u>Smoking & DE exposure:</u>					
Non & ex-smokers	10	1.46*	0.9-2.3		
≤ 20 cigarettes/day	10	0.82*	0.5-1.4		
> 20 cigarettes/day	7	1.30*	0.8-2.1		

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or p -values reported in original study. DE = Diesel Exhaust. OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Truck Drivers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval ^a or P-Value	Comments
Boffetta <i>et al.</i> , 1990 USA	<u>Exposure by occupation:</u>		OR		Study consisted of 2584 histologically confirmed lung cancer cases and 5009 controls derived from 18 hospitals in six cities. Controls were patients with current non-tobacco-related diseases matched by age, hospital and year of interview. Exposure was assessed by occupational titles and self-reported exposure to diesel exhaust. Results were adjusted for smoking, education and asbestos exposure by logistic regression. Occupations were classified as having probable, possible or no diesel exhaust exposure. Exposure prevalence was low. Only 15.6% of the controls were ever in an exposed job and 6.4% were considered probably exposed. Self-reported exposure to diesel exhaust had consistently higher point estimates of risk than those based on occupational classification, suggesting the possibility of recall bias. See also Hall and Wynder, 1984. *Duration of employment data only available for 23 cases and 27 controls of all patients classified as truck drivers (114 cases and 176 controls).
	“Possible” exposure	240	0.92	0.76-1.10	
	“Probable” exposure	210	0.95	0.78-1.16	
	<u>By duration:</u>				
	“Probable” DE				
	1-15 years	4	0.52	0.15-1.86	
	16-30 years	15	0.70	0.34-1.44	
	31+ years	17	1.49	0.72-3.11	
	Truck driver*				
	1-15 years	4	1.83	0.31-10.73	
16-30 years	12	0.94	0.41-2.15		
30+ years	7	1.17	0.40-3.41		
<u>Self-reported exposure:</u>			1.21	0.73-2.02	
<u>By duration</u>					
1-15 years	11	0.90	0.40-1.99		
16-30 years	12	1.04	0.44-2.48		
31+ years	12	2.39	0.87-6.57		
Damber and Larsson, 1985 Sweden	Case-control		OR		Study included 604 male patients with lung cancer from the 3 most northern counties in Sweden (all new cases reported to the Swedish Cancer Registry in 1972 to 77 who had died at least one year before the start of the study in 1979). Matched controls were drawn from the national registry for causes of death. Living controls were also used. Data on occupational and smoking habits were obtained by questionnaire. Study focused on professional drivers, most of whose vehicles had diesel engines. Investigators noted that drivers had considerably higher average tobacco consumption than nondrivers. Authors stated that the study suggests a synergistic interaction between smoking and occupational exposure. See also Damber and Larsson 1987. Risk estimates presented for portion of cohort with date of birth after 1900. # Subset of all drivers. * Compared to nondrivers. ** Compared to nondrivers/nonsmokers, where “nonsmokers” included ex-smokers who had quit for at least 10 years.
	<u>By age of diagnosis:</u>				
	Professional drivers				
	<70 years	40	1.00*	0.66-1.50	
	≥70 years	23	3.15*	1.66-6.00	
	Truck drivers [#]				
	<70 years	22	0.83*	0.50-1.40	
	≥70 years	13	5.70*	2.22-14.67	
	<u>By age & smoking status:</u>				
	Drivers/				
Nonsmokers**					
<70 years	NG	1.9	0.5-5.5		
≥70 years	NG	4.5	1.1-16.4		
Drivers/Smokers**					
<70 years	NG	6.0	3.5-10.3		
≥70 years	NG	20.8	9.4-46.0		

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or *p*-values reported in original study. DE = Diesel Exhaust. OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Truck Drivers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval ^a or P-Value	Comments
Damber and Larsson, 1987 Sweden	Case-control Professional drivers Years worked ≥1 ≥20 Adjusted for smoking ≥1 ≥20	72 37 72 37	OR 1.3 1.5 1.0 1.2	0.9-1.9 0.9-2.6 0.7-1.5 0.6-2.2	Study consisted of 600 men with lung cancer in northern Sweden reported to the Swedish Cancer Registry from 1972 through 1977 and dead before the start of the study (1979). Cases were matched with both dead and living controls. Results reported here are for comparisons with dead controls. Results with living controls were in good agreement. See Damber and Larsson (1985) for study focused on professional drivers only.
Boffetta <i>et al.</i> 1988 USA	Prospective Cohort <u>Self-reported as DE:</u> All DE exposed By duration exposure: 1-15 years 16+ years DE & smoking status*: nonsmokers ex-smokers current smokers <u>Occupation:</u> Railroad worker Truck driver Heavy equipment By occupation & DE: Truck/exposed Truck/nonexposed	174 7 85 78 14 48 5 18** 18**	RR 1.18 1.05 1.21 1.73 11.06 19.82 1.59 1.24 2.60 1.22 1.19	0.97-1.44 0.80-1.39 0.94-1.56 [#] 0.60-4.95 6.27-19.53 11.20-35.07 0.94-2.69 0.93-1.66 1.12-6.06 0.77-1.95 0.74-1.89	Included 461,981 males, aged 40 to 79, participating in the American Cancer Society's Prospective Mortality Study in 1982. Follow-up for two years. Exposure assessment was based on self-reported (questionnaire) occupation and diesel exhaust exposure. Investigators stated that, although the sample was large, it was comprised of volunteers, who were healthier and were less frequently exposed to important risk factors such as smoking and alcohol. Reference population included men with no reported exposure or likely occupational exposure to diesel exhaust. Results were adjusted for smoking and other occupational exposures (asbestos, coal and stone dust, coal tar pitch, and gas exhaust). See Hall and Wynder, 1984. *Smoking data not available for all subjects. **Diesel exhaust exposure data not available for all truck drivers. [#] Test for trend reported by investigators as 0.05 < p < 0.10.
Benhamou <i>et al.</i> 1988 France	Case-control Motor vehicle mechanic Transport equipment operators Professional drivers	65 157 128	RR 1.06 1.35 1.42	0.73-1.54 1.05-1.75 1.07-1.89	Study consisted of 1,334 histologically confirmed lung cancer cases and 2,409 controls matched on sex, age, hospital admission and interviewer. Study was conducted between 1976 and 1980. Results were adjusted for smoking and are limited to males. Occupation was determined by questionnaire (interview). The types of motor vehicle engines worked with were not specified. No evidence of increased risk with increased duration of exposure (years employed).

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or p-values reported in original study. DE = Diesel Exhaust. OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Truck Drivers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval ^a or P-Value	Comments
Hayes <i>et al.</i> 1989 USA	Case-control Pooled Analysis Truck Drivers < 10 yrs employed ≥ 10 yrs employed Heavy Equipment < 10 yrs employed ≥ 10 yrs employed Bus Drivers < 10 yrs employed ≥ 10 yrs employed	161 112 7 10 23 24	OR 1.0 1.5 1.5 2.1 1.1 1.7	0.8-1.3 1.1-2.0 0.4-5.3 0.6-7.1 0.6-2.1 0.8-3.4	The study is a pooled analysis of three case-control studies conducted between 1976 and 1983 in Florida, New Jersey, and Louisiana. Total eligible cases = 2,291 and controls = 2,570. All occupational data were recoded from original interviews. No specific information regarding diesel exposure or engine type. ORs were adjusted for birth cohort (<1910, 1910-19, 1920-29, 1930+), usual daily cigarette use, and state.
Steenland <i>et al.</i> 1990 USA	Case-control <u>Occupation data:</u> 1) Teamster records data Long-haul driver Short-haul driver 2) Next-of-kin data Truck driver, diesel Truck driver, gasoline Truck driver, both <u>Duration employment after 1959*:</u> 1) Teamster records data Long-haul driver 1-11 years 12-17 years ≥18 years 2) Next-of-kin data Diesel truck driver 1-24 years 25-34 years ≥35 years	162 228 213 48 72 56	OR 1.27 1.31 1.42 1.22 1.25 1.08 1.41 1.55 1.27 1.26 1.89	0.83-1.93 0.81-2.11 0.89-2.26 0.79-1.88 0.81-1.95 0.68-1.70 0.90-2.21 0.97-2.47 0.70-2.27 0.74-2.16 1.04-3.42	Study consisted of 1,086 lung cancer cases and 1,085 controls among truck drivers in the Central States Teamsters Union. Information on work history was obtained from next of kin and union records. Subjects died in 1982-83 after applying for pensions, which required at least 20 years of union membership. Subjects were classified according to the job category in which they worked the longest. Union data provided no information on the type of truck driven. 90% of union long-haul drivers were also identified as diesel truck drivers by next of kin. Results were adjusted for smoking and asbestos exposure. Smoking data obtained by next-of-kin interview used in both types of exposure classification. Steenland <i>et al.</i> (1992) summarized results from a recent industrial hygiene survey of exposure to diesel exhaust in the trucking industry, and found that elemental carbon measurements were generally consistent with the results; i.e., mechanics had the highest exposure and the highest risks, followed by long-haul and local drivers. Authors noted that exposure to asbestos may account for some of the observed effects in mechanics, but its confounding effect was probably small. Study results for truck mechanics and dock workers were elevated but not significant. *Study also presented risk estimates for duration of employment inclusive of the pre-1959 work era for both job ascertainment categories and for majority of job classifications.

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or *p*-values reported in original study. DE = Diesel Exhaust. OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Truck Drivers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval ^a or P-Value	Comments
Burns and Swanson 1991 USA	Case-control		OR		Occupational and smoking histories were obtained by telephone interview for 5,935 incident lung cancer cases and 3,956 incident colon and rectal cancer controls diagnosed between 1984 and 1987 and reported to the Detroit cancer registry. The smoking- and race-adjusted OR for all drivers (238 cases, 86 controls) was 1.88 (95% C.I. = 1.37-2.58), while drivers of "heavy trucks" (166 cases, 48 controls), maintained a higher risk even after adjustment for smoking, OR = 2.31 (95% C.I. = 1.56-3.42). Mechanics also had a significantly elevated OR for lung cancer (OR = 1.72, 95% C.I. = 1.15-2.59). The types of the vehicle engines were not specified. Results were adjusted for smoking. See Swanson <i>et al.</i> 1993.
	Drivers (white)	187	2.40	1.65-3.48	
	All drivers (race adj.)	238	1.88	1.37-2.58	
	Railroad workers	14	1.27	0.45-3.53	
Swanson <i>et al.</i> 1993 USA	Case-control		OR		Cases and controls were from OCISS (see Burns and Swanson, 1991 for description of subjects). Incident lung cancer cases among black and white males, aged 40 to 84, from 1984 through 1987 are included in this report. Controls were colon and rectal cancer cases. Information on occupation, smoking, medical history were obtained by telephone interview. Results were adjusted for age at diagnosis, race and smoking. *Test for trend $p \leq 0.05$.
	Occupation and duration:				
	1) White males				
	Heavy truck drivers				
	0 years	88	1.0	Reference	
	1-9 years	78	1.4	0.8-2.4*	
	10-19 years	38	1.6	0.8-3.5*	
	20+ years	121	2.5	1.1-4.4*	
	Light truck drivers				
	0 years	88	1.0	Reference	
	1-9 years	46	1.7	0.9-3.3	
	10+ years	36	2.1	0.9-4.6	
	Railroad workers				
	0 years	73	1.0	Reference	
	1-9 years	27	1.2	0.5-2.7	
	10+ years	40	2.4	1.1-5.1	
2) Black males					
Heavy truck drivers					
0 years	12	1.0	Reference		
1-9 years	27	2.7	0.8-9.2		
10-19 years	16	1.9	0.5-7.2		
20+ years	16	2.1	0.5-9.2		
Railroad workers					
0 years	15	1.0	Reference		
1-9 years	22	2.6	0.8-7.9		
10+ years	9	2.7	0.6-12.1		

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or *p*-values reported in original study. DE = Diesel Exhaust. OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Truck Drivers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval ^a or P-Value	Comments
Rafnsson and Gunnarsdottir 1991 Iceland	Cohort Truck drivers Duration employment: <2 years 2-10 years 11-30 years >30 years	24	SMR 2.14 2.70 2.46 0.68 2.32	1.37-3.18 0.74-6.92 0.99-5.08 0.01-3.76 0.85-5.04	Cohort consisted of truck and taxi drivers in Reykjavik followed from 1951 to 1988. National mortality rates were used as for comparison. Information on truck drivers was obtained from their union. No data on smoking or type of vehicle engines used. No trend of increased risk with increased follow-up time was observed.
Guberan <i>et al.</i> 1992 Switzerland	Cohort Professional drivers	77	SMR 1.50	1.23-1.81	Cohort identified from vehicle license records of professional drivers required to obtain special license during the period from 1949 to 1961. Excluding individuals born prior to 1900, 1,726 drivers were eligible. Lung cancer cases identified from death and tumor registries through 1986. No smoking data obtained. Approximately 1/3 to 1/4 of professional drivers were reported to be long-haul truck drivers. Death rates compared to regional mortality rates. A significant ($p < 0.02$) upward trend in lung cancer mortality with time from first exposure was also observed: SMRs = 0.67, 1.18, 1.30, 1.35, and 2.59 for 0-14, 15-24, 25-34, 35-44, and ≥ 45 years, respectively (no confidence intervals reported).
Hansen 1993 Denmark	Cohort Age on Nov. 9, 1970 15-29 30-39 40-44 45-49 50-54 55-59 60-64 65-74 Total	0 3 3 11 12 19 22 6 76	SMR 1.96 0.56 1.17 1.10 2.29 2.27 2.60 1.60	 0.40-5.73 0.12-1.64 0.58-2.09 0.57-1.93 1.38-3.58 1.42-3.44 0.95-5.65 1.26-2.00	Cohort consisted of 14,225 truck drivers followed for a 10-year period. Comparisons were made with another cohort of unskilled laborers. Members of the cohort were identified from the file of a nationwide census conducted in 1970. Self-reported occupation, trade, industry and employment on the day of the census were recorded. The study was comprised of unskilled male laborers 15 to 74 years old who were occupationally active on the day of the census. 627 truck drivers and 3,811 members of the control cohort died within the 10 years. No data on smoking. Diesel engines have comprised most of Danish fleet of trucks since the late 1940s.
Pfluger and Minder, 1994 Switzerland	Case-control Professional drivers - smoking adjusted	284	OR 2.27 1.48	1.99-2.58 1.30-1.68	Mortality of Swiss professional drivers (truck, bus and taxi drivers) was determined from death certificates and compared to census data to obtain occupation and age-specific death rates. No individual smoking data were available, but an indirect adjustment was conducted based on occupation specific mortality rates.

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or p -values reported in original study. DE = Diesel Exhaust. OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Transport (i.e., bus) and Equipment Workers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval ^a or P-Value	Comments
Raffle 1957 England	Cohort Overall Bus & trolley drivers Age 55-64	96 30	SMR 1.4	 N.S.	Cohort consisted of deaths, retirements and transfers due to lung cancer in London transport employees (bus and trolley workers, bus engineers), aged 45 to 64 years, in jobs with presumably different exposures to exhaust fumes in 1950 to 1954. Only cases arising during exposure employment were considered. Rates were compared to lung cancer mortality in other company employees. Diesel buses had been gradually introduced since the 1930s. At the end of WWII only 15% of the buses still used petrol. All had been replaced by 1950. Consequently, the duration of exposure of some workers to DE might have been short. No data on smoking. See also Waller 1981.
Waller 1981 England	Cohort All workers Bus drivers Bus conductors Engineers, garages Engineers, central works Motormen and guards	667 259 130 177 42 59	SMR 0.79 0.75 0.75 0.90 0.66 0.87	 NP NP NP NP NP	Cohort consisted of lung cancer deaths and retirements or transfers due to lung cancer in men, aged 45 to 64, employed within five categories of London Transport employees. Mortality was compared to men in Greater London. The study covered 25 years ending in 1974, thus including some of the data described by Raffle (1957). No data on smoking. Those who retired at age 65 or left earlier were not followed up, thus limiting the extent of case ascertainment.
Rushton <i>et al.</i> 1983 England	Cohort	102	SMR 1.01	 $p = 0.94$	Cohort consisted of 8,684 men employed as maintenance workers in 71 bus garages in London for at least one year from 1967 to 1975. Follow-up through 1975. No data on smoking. Authors noted short follow-up period (average of 6 years). Lung cancer mortality was compared with the male population of England and Wales. The all-cause mortality was significantly lower than expected based on London residence.
Buiatti <i>et al.</i> 1985 Italy	Case-control Transportation Taxi driving Train conductors	45 20 7	OR 1.1 1.8 1.4	 0.7-1.6 1.0-3.4 0.5-3.9	Study consisted of 340 confirmed cases in males (and 817 controls) in Florence, diagnosed from 1981 through 1983 in the regional general hospital and a referral center for lung cancer. Controls were matched on sex, age, date of admission and smoking, and were from the same hospital. Diesel exhaust exposure was assessed by questionnaire for all jobs held for more than one year.

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or *p*-values reported in original study. DE = Diesel Exhaust, OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio, NP = not presented.

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Transport (i.e. bus) and Equipment Workers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval ^a or P-Value	Comments
Wong <i>et al.</i> 1985 USA	Cohort Total <u>By Duration</u> <5 years 5-9 years 10-14 years 15-19 years ≥20 years All retired members Normal retired members	309 10 25 53 58 163 155 86	SMR 0.99 0.45 0.75 1.08 1.02 1.07 1.64* 1.30**	0.88-1.10 N.S. N.S. N.S. N.S. $p = 0.05$ $p < 0.01$ $p < 0.05$	Cohort consisted of 34,156 male members of a heavy construction equipment operators union for at least one year from 1964 through 1978. Mortality experience was compared with that of the US white male population. Partial work history was available for some cohort members through the union. A random sample of union members was surveyed to determine smoking habits, and no significant difference between members and the general population was found. Work groups evaluated were considered to have high exposure to diesel exhaust (scraper operator, bulldozer operator, backhoe operator and loader operator) or low exposure (mechanical maintenance workers and engineers). Overall mortality in the cohort was less than that in the U.S. male population (SMR 0.81, 95% C.I. 0.79-0.84). Workers were also categorized by job title and potential exposure, but no significant risks were observed. Analysis of retirees found an excess risk for lung cancer* and emphysema. *Includes also retirements due to ill health. *Normal retirees are those workers retired at or over 65 and early retirees who reached 65.
Edling <i>et al.</i> 1987 Sweden	Cohort Bus company employees Bus drivers Bus garage workers Clerks	6 5 1 0	SMR 0.67 0.69	Not presented	Cohort consisted of 694 bus garage employees followed from 1951 through 1983. Men were divided into three exposure categories (clerks, bus drivers and bus garage workers). Clerks were assumed to have had the lowest exposure to diesel exhaust and bus garage workers the highest. Authors stated that the power of the study to detect specific cancers was limited. No data on smoking.
Netterstrom 1988 Denmark	Cohort Bus drivers	15	SMR 0.87	0.48-1.43	Cohort of 2,465 Danish bus drivers from three companies during the period 1978 to 1984. Cases were identified through death and cancer registries. Death rates were compared with national rates. No data on smoking were available. Mean value for employment duration among the lung cancer cases was 30 years

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or p -values reported in original study. DE = Diesel Exhaust, OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Transport (i.e. bus) and Equipment Workers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval ^a or P-Value	Comments
Gustavsson <i>et al.</i> 1990 Sweden	Cohort Total (deaths) DE exposure index: 0-10* 10-30 >30 Nested case-control (20 incident cases) 0-10* 10-20 20-30 >30	17 5 5 7 5 2 3 10	SMR 1.22 0.97 1.52 1.27 RR 1.0 1.34 1.81 2.43	0.71-1.96 Reference 1.09-1.64 1.20-2.71 1.32-4.47	Cohort consisted of 695 bus garage workers employed as mechanics, servicemen or hostlers for at least six months in five bus garages in Stockholm between 1945 and 1970. A nested case-control study was performed within the cohort. Follow-up was through 1986. No data on smoking although no large variation in smoking habits was expected within the cohort. Exposure to diesel exhaust and asbestos were assessed based on time period-specific data on job tasks. Lung cancer cases were identified through tumor and death registries. In the cohort analysis regional rates were used for comparison. *Cumulative exposure index values (unitless).
Gustafsson <i>et al.</i> 1986 Sweden	Cohort Deaths Incident cases	 71 89	SMR 1.29 SIR 1.53	 1.02-1.63 1.24-1.80	Cohort consisted of 6,071 Swedish dockworkers first employed before 1974 for at least six months. The group was followed from January 1961 through January 1981. Cancer morbidity was determined among 6,071 dockworkers who had been alive and without cancer in January 1961. Comparison group was Swedish male population. Diesel trucks were introduced into Swedish ports in the late 1950s and became prevalent during the 1960s. No data on smoking. See Emmelin <i>et al.</i> (1993) for results from the follow-up study. Employment as a dockworker was the only information on diesel exhaust exposure used in the analysis.

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or *p*-values reported in original study. DE = Diesel Exhaust, OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Transport (i.e. bus) and Equipment Workers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval ^a or P-Value	Comments
Emmelin <i>et al.</i> 1993 Sweden	Case-control		OR		Study was a nested case-control of lung cancer among Swedish male dockworkers in the cohort studied by Gustafsson <i>et al.</i> (1986). 154 referents were matched to 50 cases on port and date of birth. Indices of exposure to diesel exposure were derived from employment records and records of annual fuel consumption by diesel vehicles. Three different exposure classifications were created: "machine time", "fuel consumption" and "exposed time". Information on smoking was obtained from questionnaires and interviews with foremen or workers who had worked with subjects. Response rate for mailed questionnaires was low (67%) but information from the interviews was available for 95% of the subjects. Some ex-smokers were classified as never smokers. No exposure level ("low", "medium", or "high") was significant for any DE exposure scheme (only "high" strata reported here). Comparisons based on exposure and smoking tended to find more elevated risks. Investigators noted that the increase in the OR for both smoking and exhaust exposure indicate that smoking does not explain the results from the exposure-only models, and that there may be an interaction between smoking and exhaust exposure. No information on asbestos exposure, which was said to have decreased by the 1970s. See also Gustafsson <i>et al.</i> (1986). * "Low" exposure category used for reference comparison. **Note: authors reported confidence intervals at 90% level.
	<u>Exposure variable:</u> Machine time high*	14	1.3	0.3-5.6**	
	Fuel consumption high*	15	1.7	0.5-5.9**	
	Exposed time high*	19	2.9	0.8-10.7**	
	<u>Exposure & Smoking:</u> Machine time medium		1.8	0.5-6.6**	
	high		2.9	0.6-14.4**	
	smoker		5.7	2.4-13.3**	
	Fuel consumption medium		1.5	0.5-4.8**	
	high		2.9	0.7-11.5**	
	smoker		5.5	2.4-12.7**	
	Exposed time medium		2.7	0.6-11.3**	
	high		6.8	1.3-34.9**	
	smoker		6.2	2.6-14.6**	
Kaplan 1959 USA	Cohort		SMR		Cohort consisted of 6,506 deaths among railroad workers from the Baltimore and Ohio Railroad Relief Department between 1953 and 1958. Subjects were categorized into 3 groups by exposed to diesel exhaust and compared with national lung cancer mortality rates. IARC noted that since the changeover to diesel engines began in 1935 and was 95% completed by 1959 (Garshick <i>et al.</i> 1988), few, if any, of the lung cancer deaths could have occurred in workers with more than 10 years of exposure to diesel exhaust. No data on smoking.
	Total	154	0.80	0.68-0.94	
	Most likely exposed	49	0.875	N.S.	

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or *p*-values reported in original study. DE = Diesel Exhaust, OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Railroad Workers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence ^a Interval or P-Value	Comments
Garshick <i>et al.</i> 1988 USA	Cohort	1694	RR		Cohort consisted of 55,407 white male railroad workers aged 40-64 exposed to little or no asbestos who had started work between 1939 and 1949 and had worked 10 to 20 years after 1959. Follow-up through 1980. Industrial hygiene data were used to categorize jobs as exposed or unexposed. No data on smoking; however, authors noted that there was no difference in smoking habits by job title in comparison studies of current workers (see Garshick <i>et al.</i> 1987). Diesel exhaust exposure in the US railroad industry occurred after WWII. The approximate midpoint of dieselization was in 1952 and by 1959, 95% of the locomotives were diesel-powered. Workers aged 40 to 44 in 1959 were the group with the longest possible duration of exposure. Most workers with potential asbestos exposure were excluded, though some did have potential exposure to asbestos (shopworkers and hostlers). Analyses were done with and without these groups. Exposure was assessed from samples of respirable dust taken in 1980s (Woskie <i>et al.</i> 1988a). Mean exposure levels suggested a five-fold range of exposure between clerks and shopworkers (Woskie <i>et al.</i> 1988b). These values confirmed the assignment of categories of diesel exhaust exposure in the present study and Garshick <i>et al.</i> 1987. * Excluding exposure to diesel exhaust over the 4 years preceding the year of death
	<u>By Age in 1959 w/ DE:</u>				
	40-44		1.45	1.11-1.89	
	45-49		1.33	1.03-1.73	
	50-54		1.12	0.88-1.42	
	55-59		1.18	0.94-1.50	
	60-64		0.99	0.74-1.33	
	Minus those w/ asbestos exposure				
	40-44		1.57	1.19-2.06	
	45-49		1.34	1.02-1.76	
	<u>By Years DE Exposure:*</u>				
	1-4 years		1.20	1.01-1.44	
5-9 years	1.24	1.06-1.44			
10-14 years	1.32	1.13-1.56			
≥ 15 years	1.72	1.27-2.33			
Minus those w/ asbestos exposure					
1-4 years	1.34	1.08-1.65			
5-9 years	1.33	1.12-1.58			
10-14 years	1.33	1.10-1.60			
≥ 15 years	1.82	1.30-2.55			
Nokso-Koivisto and Pukkula, 1994 Finland	Cohort Total	236	SIR 0.86	0.75 – 0.97	Cohort consisted of 8,391 members of the Finnish Locomotive Drivers' Association from 1953 to 1991 (including retirees). Information was not available for 302 members. No smoking data were available. The overall incidence for all cancer sites was lower than expected when compared to national rates (SIR = 0.95).

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or *p*-values reported in original study. DE = Diesel Exhaust, OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer (Additional Studies Other Than Those Listed In Above Categories)

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval or P-Value	Comments
Wegman and Peters, 1978 USA	Case-control Total study Transportation equipment operatives	91	OR		Tumor registry-based study of oat cell carcinoma during 1965 to 1972. Cancer controls identified from same registry. Smoking data collected but not used in analysis (94% cases and 78% controls smoked). Two methods used to classify occupation, registry-derived or combination of registry and next-of-kin questionnaire data. Number of cases classified as transportation equipment operatives decreased from 8 to 5 between two methods.
	- Registry derived - Combination w/ registry data	8 5	8.67 1.26	NP NP	
Coggon <i>et al.</i> 1984 England	Case-control Total DE exposed High DE exposure	 172 32	RR 1.3 1.1	 1.0-1.6 0.7-1.8	Study included all men 40 years of age in England and Wales who had died of tracheobronchial cancer from 1975 through 1979. A job exposure matrix was constructed in which occupations were grouped according to likely exposure to each of nine known or putative carcinogens. Occupational information abstracted from the death certificates. No information on smoking. IARC noted the limitations of information on death certificates, the young age of the subjects, short exposure and latency times, and the lack of data on smoking and other potential confounders.
Lerchen <i>et al.</i> 1987 USA	Case-control Diesel exhaust fumes - adjusted for smoking Diesel engine mechanics - adjusted for smoking	 7 5	OR 0.6 1.0	 0.2 – 1.6 0.2 – 2.0	Population-based case-control study of 506 patients diagnosed between January 1980 and December 31, 1982, and reported to the New Mexico tumor registry (333 males and 173 females). Data on lifetime occupation and smoking were obtained by personal interview and self-reported history of exposure to specific agents. Matched controls were selected randomly from the telephone directory or for persons over 65 from the roster of participants in a health insurance plan. Only seven males reported exposure to diesel exhaust.
Magnani <i>et al.</i> 1988 England	Cohort All DE exposure	 NP	SMR 1.07	 1.04 – 1.10	General population-based cohort analysis of death certificate and census survey information on 31,925 men with lung cancer between 1970-72. No smoking data were available. A job-exposure matrix was developed for several potential carcinogens, including diesel exhaust.

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or *p*-values reported in original study. NP = not presented. DE = Diesel Exhaust, OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer (Additional Studies Other Than Those Listed In Above Categories).

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence ^a Interval or P-Value	Comments
Siemiatycki <i>et al.</i> 1988 Canada	Case-control Lung cell types among DE exposed: Oat cell Squamous cell Adenocarcinoma Other Total DE-exposed occupations minus mining:	34 81 28 34 177 70	OR 1.1 1.2 0.9 1.0 1.1	 0.8-1.5** 1.0-1.5** 0.6-1.2** 0.8-1.4** 0.8-1.5**	This population-based case-control study provided information on the association between several cancer types and 10 types of exhaust and combustion products. Interviews were carried out for 3,726 cancer patients, aged 35 to 70, diagnosed in any of 19 participating Montreal area hospitals. Each type of cancer was a case series; reference groups were selected from among the other cancer patients interviewed. Results reported are adjusted for smoking, socioeconomic status, ethnic group and several other potential confounders. Authors noted that the excess lung cancers were concentrated among mine and quarry workers. **Authors reported 90% confidence intervals.
Bender <i>et al.</i> 1989 USA	Cohort State highway workers	NP	SMR 0.69	 0.52 – 0.90	Cohort consisted of Minnesota highway workers employed for a minimum of one year and working at least one day after January 1, 1945. Mortality was compared to state rates. No data were available on smoking. Overall mortality was significantly lower than the expected, SMR = 0.83 (95% C.I. = 0.73-0.94).
Kauppinen <i>et al.</i> , 1993 Finland	Case-control Engine exhaust exposure: Any exposure ≥ 1 month 1 month - 5 years > 5 years	8 5 3	OR 1.7 0.39 2.21	 0.55-5.20** 0.05-2.94** 0.65-7.48**	Nested case-control study of woodworkers in Finland consisted of 136 lung cancer cases diagnosed between 1957 to 1982 and 408 matched controls. Original cohort consisted of 7,307 workers from 35 factories. Multiple chemical exposures were analyzed for, including engine exhaust (combination of diesel and gasoline engines). Smoking, age, and other chemical exposures were adjusted for; however, only a small number of individuals were categorized as having been exposed to engine exhaust. **Authors reported 90% confidence intervals.

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or *p*-values reported in original study. NP = not presented. DE = Diesel Exhaust, OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Animal Studies

Section 6.1 (Animal Studies) of the diesel exhaust TAC document (OEHHA, 1998) describes the results of diesel exhaust inhalation carcinogenicity bioassays performed using mice, rats, hamsters and monkeys. The studies in rats provided the only clear and unequivocal evidence of diesel exhaust-induced carcinogenicity in animals.

The results of eleven animal cancer bioassays of inhalation of diesel exhaust alone were available at the time the document entitled *Health Risk Assessment For Diesel Exhaust* was written for the Toxic Air Contaminant (TAC) program (OEHHA, 1998). None of the four studies with either (a) exposure periods of less than 7 hours/day, 5 days/week for 24 months or (b) particulate exposure concentrations of less than 2.2 mg/m³ (Karagianes *et al.*, 1981; White *et al.*, 1983; Lewis *et al.*, 1986, 1989; Takemoto *et al.*, 1986) gave positive results for carcinogenesis of diesel exhaust. The seven studies that presented positive results are as follows: Brightwell *et al.*, 1986, 1989; Heinrich *et al.*, 1986; Ishinishi *et al.*, 1986a; Iwai 1986; Mauderly *et al.*, 1987a; Heinrich *et al.*, 1995; Nikula *et al.*, 1995. Results of these studies are described in detail in the diesel exhaust TAC document (OEHHA, 1998).

IV. DERIVATION OF CANCER POTENCY

Basis for Cancer Potency

The diesel exhaust TAC document (OEHHA, 1998) stated that the results of the epidemiological analyses described above are consistent with a positive association between occupational exposure to diesel exhaust and an increased risk of developing lung cancer. The diesel exhaust TAC document reviewed the evidence for causality in the association between diesel exhaust and cancer of the lung. The following criteria for causal inference were considered: (1) the consistency of the findings; (2) the strength of the associations; (3) the possibility that findings are due to bias; (4) the likelihood that findings are due to chance; (5) evidence for exposure-response relationships; (6) temporality of the associations; and (7) biological plausibility of a causal association.

Chapter 6 of the diesel exhaust TAC document provided evidence consistent with a causal relationship between occupational diesel exhaust exposure and lung cancer. A lengthy discussion of causal inference, including the strengths and limitations of the underlying data, can be found in Section 6.2.4 of that document. The key findings relating lung cancer and occupational exposure to diesel exhaust are as follows: the majority of studies examining the diesel exhaust-lung cancer association have reported elevated estimates of relative risk, many of which are statistically significant. The consistency of these findings is unlikely to be due to chance. Moreover, with the possible exception of some studies that did not take smoking into account, the results are unlikely to be explained by confounding or bias. This is reinforced by the results of a meta-analysis undertaken by OEHHA staff (summarized below, and presented in detail in Appendix C of the diesel TAC document (OEHHA, 1998)), in which statistically significant pooled estimates of relative risk persisted through numerous subset and sensitivity analyses. The most important potential confounder is cigarette smoking, which was measured

and controlled for in multiple studies: in the meta-analysis the pooled relative risk estimate for studies that adjusted for smoking was 1.43 (95% C.I. = 1.31-1.57). In addition, several studies provide evidence of exposure-response relationships. The strength of the associations reported is typically within the range considered “weak” in epidemiology (i.e., estimates of relative risk between 1 and 2); nonetheless, this is not a bar to causal inference as long as other criteria are met, as discussed in Section 6.2.4 of the diesel exhaust TAC document. The temporal relationship between exposures and lung cancer is consistent with a causal relationship.

Additionally, the basic hypothesis -- that occupational exposure to diesel exhaust causes human lung cancer -- is highly plausible biologically. The evidence can be briefly summarized as follows: (1) Diesel exhaust has been shown to induce lung and other cancers in laboratory animal studies (Brightwell *et al.* 1989; Heinrich *et al.* 1986a; Iwai *et al.* 1986; Mauderly *et al.* 1987a); (2) Diesel exhaust has been shown to contain highly mutagenic substances, including polycyclic aromatic hydrocarbons and nitroaromatic compounds (Ball *et al.*, 1990; Gallagher *et al.*, 1993; Nielsen *et al.*, 1996; Sera *et al.*, 1994); (3) Diesel exhaust contains many substances which occur in recognized complex mixtures of human respiratory carcinogens, including cigarette smoke and coke oven emissions (IARC, 1989); and (4) Diesel exhaust contains known and probable human carcinogens.

Therefore, a reasonable and very likely explanation for the increased risks of lung cancer observed in the occupational epidemiological studies is a causal association between diesel exhaust exposure and lung cancer.

Results based on the human data and those based on the animal data are both subject to uncertainty. The principal uncertainties in using the rat data are their application to humans in terms of response, the choice of dose-response model to extrapolate the risk to environmental concentrations, the presence or absence of a threshold for response, and the range of dose extrapolation involved. While there are issues surrounding the quantitation of worker exposure to diesel exhaust, the uncertainty of extrapolating from one species (rat) to another (human) is avoided by using the epidemiological data to estimate risk to humans from diesel exhaust exposure. OEHHA preferred, on balance, to use the epidemiological data in order to estimate risk to humans from diesel exhaust exposure. Therefore, only the unit risk estimates based on human data were included in the final range of cancer unit risks associated with exposure to particulate matter from diesel-fueled engines in the diesel exhaust TAC document (OEHHA, 1998). OEHHA included quantitative risk assessment data based on rat studies in Appendix G of the diesel exhaust TAC document (OEHHA, 1998) for informational purposes.

Quantitative Meta-Analysis on the Relationship of Occupational Exposure to Diesel Exhaust and Lung Cancer

A meta-analysis was conducted to summarize and help interpret the published reports examining the relationship of lung cancer and exposure to diesel exhaust (OEHHA, 1998). A meta-analysis systematically combines the results of previous studies in order to generate a quantitative summary of a body of research and to examine the sources of variability among studies (for review see Petitti, 1994). The variability, or heterogeneity, of results among studies may exist due to numerous factors, including differences in study design, exposures experienced

by study subjects, methods and accuracy of exposure ascertainment, length of follow-up, and control of confounders (such as smoking).

As described in OEHHA (1998), 30 studies, contributing a total of 39 effect estimates, were utilized in the meta-analysis. The pooled relative risks for lung cancer from all 39 risk estimates combined varied with the statistical model used, 1.04 (95% C.I. = 1.02-1.06) under the fixed-effects model and 1.33 (95% C.I. = 1.21-1.46) with the random-effects model. However, significant evidence of heterogeneity was found (DerSimonian and Laird Q-statistic = 214.59, 38 d.f., $p < 0.001$). Heterogeneity in this context refers to large between-study variability. The presence of heterogeneity undermines the validity of the pooled estimates, and suggests the need for additional analysis to identify the sources of heterogeneity. As discussed in detail in Appendix C of OEHHA (1998), this involved deriving pooled estimates for a variety of subsets of the reports.

Through subset analysis, several factors were identified which strongly influenced both the magnitude and the degree of heterogeneity of the pooled risk estimates: (1) whether or not a study adjusted for smoking, (2) study design (3) the exposure assessment, as developed from occupational categories, (4) the presence of selection bias, as manifested by an observed “healthy worker effect”, and other study characteristics (See Appendix C of OEHHA (1998)). By stratifying the meta-analysis on whether the risk estimates accounted for smoking, the effect of failure to control for this exposure on the pooled estimate became readily apparent. Not only did the positive association between diesel-exhaust exposure and lung cancer persist, but the pooled risk estimate increased to 1.43 (95% C.I. = 1.31-1.57, random-effects model) with little evidence of heterogeneity among the 12 studies controlling for smoking.

The case-control studies (15 included in the meta-analysis) gave a summary estimate of 1.44 (95% C.I. = 1.33-1.56), again with little evidence of heterogeneity, while the estimate based on the results of the cohort studies remained heterogeneous. The lower pooled RR estimate and substantial heterogeneity obtained from the cohort subanalysis was probably due at least in part to failure to adjust for smoking, as only one of sixteen cohort studies controlled for this confounder, while most case-control studies did (11 of 14 studies, accounting for 17 of the 20 case-control risk estimates).

The “healthy worker effect” (HWE - here based on significantly lower than expected all-cause mortality) is a manifestation of selection bias related to hiring and retention of workers who are typically healthier than the general population, resulting in spuriously lower risk estimates for a variety of illnesses, including those potentially related to occupational exposures. Subsetting the cohort studies into those with and those without an obvious healthy worker effect markedly reduced the degree of heterogeneity in the group without the HWE (Q-statistic = 11.190, 9 d.f., $p = 0.27$), and produced an increase in the magnitude of the pooled relative risk (RR = 1.52, 95% C.I. = 1.36-1.71-1.78, random-effects model). In contrast, those studies whose results were characterized by the presence of a HWE continued to show substantial heterogeneity, and the pooled risk estimates declined. Thus, selection bias is likely to have played a role in the heterogeneity observed among the cohort studies. Selection bias results from choosing a study sample that is not representative of the entire population that could have been studied, and can distort the measure of effect (e.g., relative risk or odds ratio) (Rothman, 1986). With respect to

exposure assessment, statistically significant pooled estimates of elevated risk lacking evidence of heterogeneity were identified in several occupational subgroup analyses, both with and without additional stratification for smoking. Prior to stratifying by adjustment for smoking, the occupational subgroups involving trucking (pooled RR = 1.47, 95% C.I. = 1.33-1.63), the railroad industry (random-effects pooled RR = 1.45, 95% C.I. = 1.08-1.93), mechanics and garage workers (random-effects pooled RR = 1.35 (95% C.I. = 1.03-1.78), general transportation and professional drivers (random-effects pooled RR = 1.45, 95% C.I. = 1.31-1.60) gave risk estimates greater than the overall pooled risk estimate. The pooled RR estimates for trucking and general transportation and professional drivers showed little to no evidence of heterogeneity; however, estimates for the railroad industry demonstrated considerable heterogeneity (Q statistic = 30.90, $p < 0.001$).

Further stratification of the occupational subgroup analysis by adjustment for smoking produced a large impact on the pooled risk estimates, with all smoking-adjusted subgroup estimates displaying little evidence of heterogeneity and leading to increased risk estimates in all but one of the occupational categories. Pooled risk estimates by occupation in smoking-adjusted studies showed little evidence of heterogeneity for several occupations under both models, including truck drivers (random-effects pooled RR = 1.53, 95% C.I. = 1.20-1.94), railroad workers (random-effects pooled RR = 1.68, 95% C.I. = 1.28-2.19), and diesel mechanics and garage workers (random-effects pooled RR = 1.25, 95% C.I. = 0.87-1.80). The pooled estimates for the heavy equipment operators and dock workers and for the railroad industry studies adjusting for smoking displayed the most dramatic changes relative to the occupational analysis without smoking stratification. Among the former subgroup, the pooled risk estimate changed from 1.28 (random-effects model, 95% C.I. = 0.99-1.66) to 2.43 (95% C.I. = 1.21-4.88). Among the railroad industry studies, the pooled risk estimate also increased substantially (from 1.45 to 1.68, 95% C.I. = 1.28-2.19). In both subgroups, the pooled smoking-adjusted estimates showed little evidence of heterogeneity, though these estimates were based on two studies in the former instance and three in the latter. However, the other two heavy equipment operator and dock worker studies and the other three railroad industry studies that were not adjusted for smoking still displayed evidence of heterogeneity (Q-statistics = 2.933, 1 d.f., $p = 0.09$, and 21.517, 2 d.f., $p < 0.001$, respectively).

The meta-analysis also identified evidence of exposure-response relationships in the subgroup analyses based on duration of employment. However, as noted in OEHHA (1998), this analysis was hampered by the absence of duration-specific risk estimates in approximately one-half the studies. While the initial analysis conducted on all the included studies resulted in elevated pooled risk estimates for strata in which exposure durations were greater than 10 years relative to those with less than 10 years of exposure or for which the exposure durations were not clear from the published reports, there was still significant evidence of heterogeneity for several of the duration strata. In contrast, the analysis utilizing only estimates from the smoking-adjusted studies showed some evidence of an exposure-response gradient without evidence of statistical heterogeneity. The summary risks for all three exposure-duration strata were: RR = 1.39 (95% C.I. 1.19-1.63) for < 10 years (based on three estimates from two studies), RR = 1.64 (95% C.I. = 1.40-1.93) for $10 \leq$ to < 20 years (11 estimates from 6 studies), and RR = 1.64 (95% C.I. = 1.26-2.14) for ≥ 20 years (four estimates from four studies). The pooled risk estimate for those

studies for which the exposure duration was not clear in the published reports was 1.24 (95% C.I. = 1.00-1.54) (six estimates from four studies) (see Table C-4 in Appendix C of OEHHA (1998)).

These results were robust to a variety of sensitivity analyses. In an analysis of potential publication bias, however, there appeared to be a modest increase in the RR estimates with increasing sample size (reflected in a decreased standard error of the estimates). Publication bias, or the increased likelihood or preference for the publication of statistically significant results compared to nonsignificant or null results, may potentially distort pooled risk estimates. Publication bias is generally attributed to journal editorial policies that prefer “positive” results, so that small, statistically nonsignificant studies are less likely to be published than large, statistically nonsignificant studies (Greenland, 1994). However, it should be noted that the studies with the smallest standard errors were almost exclusively cohort studies that did not adjust for smoking and which also had a clear HWE, suggesting that other significant biases are likely to have played a role in creating an appearance of publication bias. Therefore, although publication bias cannot be ruled out, the inclusion of numerous studies of varying sample sizes and statistically insignificant findings, as well as the uncontrolled confounding and likely selection bias affecting many of the larger cohort studies, make it unlikely that the result of this meta-analysis can be completely explained by publication bias.

In summary, the meta-analysis indicated a consistent positive association between occupations involving diesel exhaust exposure and the development of lung cancer. Although substantial heterogeneity existed in the initial pooled analysis, stratification on several factors identified a persistent positive relationship. The major sources of heterogeneity included: (1) whether or not a study adjusted for smoking, (2) study design (3) the exposure assessment, as developed from occupational categories, (4) and the presence of selection bias, as manifested by an observed healthy worker effect. Taking these factors into account tended to increase the estimates of relative risks of lung cancer from occupational exposure to diesel exhaust.

Another independently conducted meta-analysis of diesel exhaust exposure and lung cancer produced remarkably similar results, with an overall pooled relative risk estimate of 1.33 (95% C.I. = 1.24-1.44) (Bhatia *et al.*, 1998). In that analysis, the study inclusion and exclusion criteria were somewhat different than those used by OEHHA staff, so that 23 studies were included. Consequently, the results of some of their subset analyses differed from those described in OEHHA (1998). In addition, those authors used only a fixed-effects model to derive pooled risk estimates, and did not focus on explorations of sources of heterogeneity. Nevertheless, Bhatia and co-workers also found a persistent positive relationship between diesel exhaust exposure and lung cancer that could not be attributed to potential confounding by cigarette smoking. Moreover, in the narrower group of studies in their report, they identified a positive exposure-response relationship in studies stratified by exposure duration.

Table 2. Studies Included in Meta-analysis of Diesel Exhaust Exposure and Lung Cancer

Study (year)	Design (Location)*	Occupation or Exposure Group	Smoking Adjusted	RR	C.I.
Ahlberg <i>et al.</i> (1981)	Cohort (†)	Truck drivers	no	1.33	1.13-1.56
Balarajan & McDowall (1988)	Cohort (†)	Truck drivers	no	1.59	1.00-2.53 ^a
Bender <i>et al.</i> (1989)	Cohort (‡)	Highway maintenance	no	0.69	0.52-0.90
Benhamou <i>et al.</i> (1988)	Case-control (†)	Professional drivers	yes	1.42	1.07-1.89
Buiatti <i>et al.</i> (1985)	Case-control (†)	Transportation general	yes	1.1	0.7-1.6
Benhamou <i>et al.</i> (1988)	Case-control (†)	Mechanics	yes	1.06	0.73-1.54
Boffetta <i>et al.</i> (1988)	Cohort (‡)	Truck drivers	yes	1.24	0.93-1.66
	Cohort (‡)	Railroad workers	yes	1.59	0.94-2.69
	Cohort (‡)	Heavy equipment operators	yes	2.60	1.12-6.06
Boffetta <i>et al.</i> (1990)	Case-control (‡)	Probable DE ≥ 30 yr	yes	1.49	0.72-3.11
Coggon <i>et al.</i> (1984)	Case-control (†)	Diesel exhaust exposed group	no	1.1	0.7-1.8
Damber & Larsson (1987)	Case-control (†)	Professional drivers	yes	1.2	0.6-2.2
Edling <i>et al.</i> (1987)	Cohort (†)	Bus drivers	no	0.69 ^b	0.2-1.6 ^b
Garshick <i>et al.</i> (1987)	Case-control (‡)	Railroad workers ≥ 20 yrs ^c	yes	1.55	1.09-2.21
Garshick <i>et al.</i> (1988)	Cohort (‡)	Railroad workers ≥ 15 yrs ^c	no	1.82	1.30-2.55
Guberman <i>et al.</i> (1992)	Cohort (†)	Professional drivers	no	1.50	1.23-1.81 ^c
Gustafsson <i>et al.</i> (1986)	Cohort (†)	Dock workers	no	1.32	1.05-1.66
Gustavsson <i>et al.</i> (1990)	Nested case-control (†)	Bus garage workers > 20 yr ^d	no	1.49 ^d	1.25-1.77 ^d
Hansen (1993)	Cohort (†)	Truck drivers	no	1.6	1.26-2.0
Hayes <i>et al.</i> (1989)	Case-control (‡)	Truck drivers ≥ 10 yr	yes	1.5	1.1-2.0
	Case-control (‡)	Bus drivers ≥ 10 yr	yes	1.7	0.8-3.4
	Case-control (‡)	Mechanic (excl auto) ≥ 10 yr	yes	2.1	0.9-5.2
	Case-control (‡)	Heavy equip. operators ≥ 10 yr	yes	2.1	0.6-7.1
Howe <i>et al.</i> (1983)	Cohort (‡)	Railroad workers probably exposed	no	1.35	1.13-1.61 ^a
Lerchen <i>et al.</i> (1987)	Case-control (‡)	Diesel exhaust grouped	yes	0.6	0.2-1.6
Magnani <i>et al.</i> (1988)	Death certificate study (†)	Diesel exhaust grouped	no	0.97	0.95-1.00
Menck & Henderson (1976)	Cohort (‡)	Truck drivers	no	1.65	1.13-2.40 ^a
	Cohort (‡)	Mechanic (excl auto)	no	3.32	1.35-8.18 ^a
Nokso-Koivisto & Pukkala (1994)	Cohort (†)	Railroad workers	no	0.90 ^d	0.79-1.04 ^d
Pfluger & Minder (1994)	Case-control (†)	Professional drivers	yes	1.48	1.30-1.68
Rafnsson & Gunnarsdottir (1991)	Cohort (†)	Truck drivers ≥ 30 yr	no	2.32	0.85-5.04
Rushton <i>et al.</i> (1983)	Cohort (†)	Bus garage workers/mechanics	no	1.01	0.82-1.22
Siemiatycki <i>et al.</i> (1988)	Case-control (‡)	Diesel exhaust grouped	yes	1.1	0.8-1.5 ^c
Steenland <i>et al.</i> (1990)	Case-control (‡)	Truck drivers ≥ 18 yr	yes	1.55	0.97-2.47
	Case-control (‡)	Truck mechanic ≥ 18 yr	yes	1.50	0.59-3.40
	Case-control (‡)	Heavy truck drivers ≥ 20 yr	yes	2.44 ^d	1.43-4.16 ^d
Swanson <i>et al.</i> (1993)	Case-control (‡)	Railroad workers ≥ 10 yr	yes	2.46 ^d	1.24-4.89 ^a
	Case-control (‡)	Transportation equip. operators	no	2.39 ^b	0.70-8.05 ^b
Wegman & Peters (1978)	Case-control (‡)	Transportation equip. operators	no	2.39 ^b	0.70-8.05 ^b
Wong <i>et al.</i> (1985)	Cohort (‡)	Heavy equip. operators ≥ 20 yr	no	1.07	1.00-1.15 ^a

^a Calculated from p-value.

^b Calculated from data presented in publication.

^c Risk estimates excluding shop workers.

^d Pooled risk estimates from two racial or duration categories.

^e 90% confidence intervals originally presented within study.

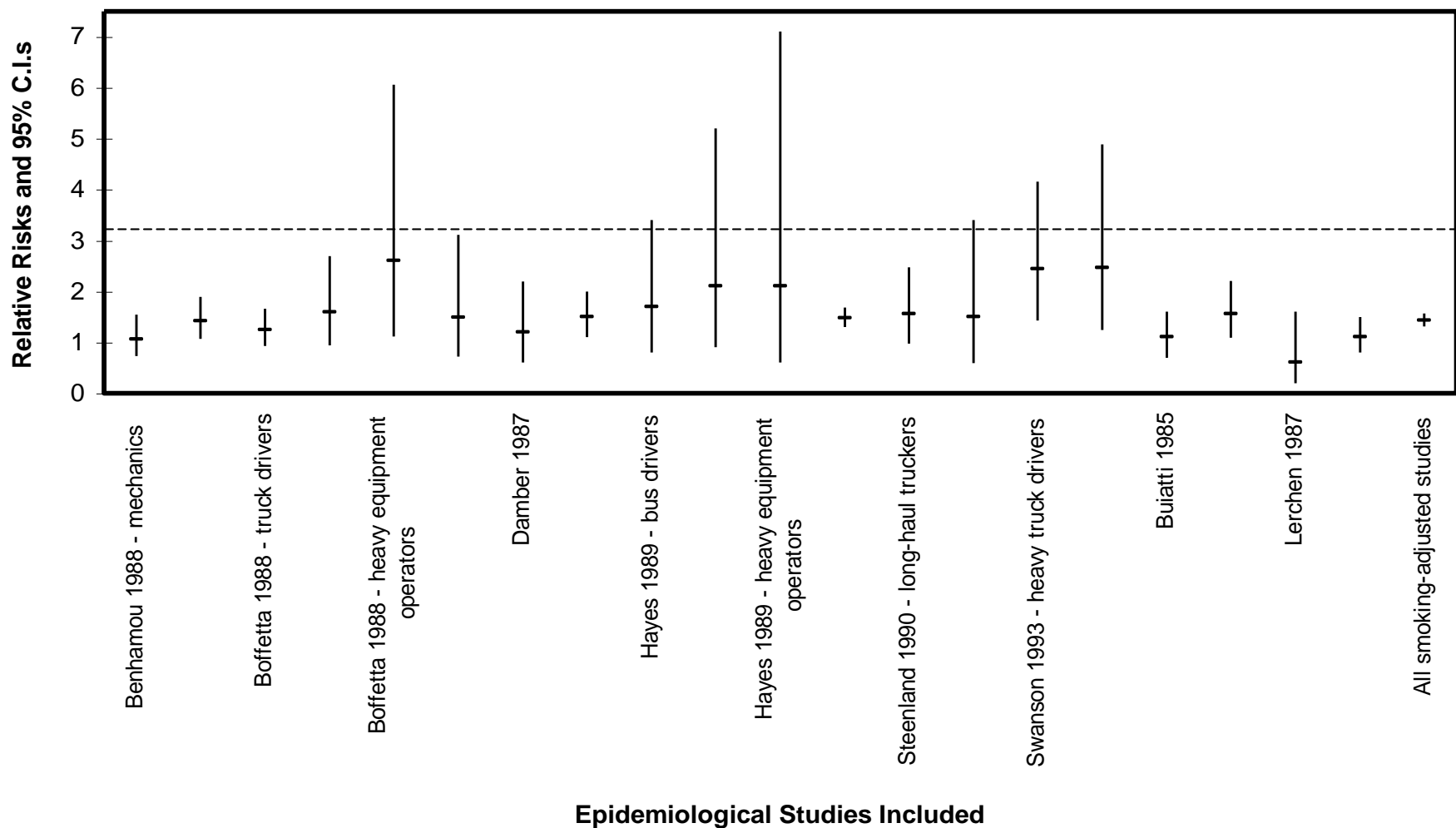
DE = diesel exhaust

RR = risk ratio

C.I. = 95% confidence interval.

* Location: (†)Europe, (‡)North America

Figure 1: Estimates of Relative Risks for Smoking-Adjusted Studies of Diesel Exhaust Exposure and Lung Cancer



Methodology

The complex and potentially variable mix of chemical species in the condensed phase and the vapor phase of diesel exhaust, required the measure of exposure related to carcinogenic risk to be specified. The most commonly used measure of exposure is atmospheric concentration of particles in $\mu\text{g}/\text{m}^3$. That measure is obtained from the mass of particles collected on a filter per volume of the air that flowed through the filter. On the basis of its relation to health studies and its general practicality, that measure was used in the diesel exhaust TAC document cancer risk assessment (OEHHA, 1998).

OEHHA used two approaches to employing epidemiological studies for diesel exhaust quantitative risk assessment. The first approach used the overall relative risks derived from the meta-analysis along with an overall range of exposure for all the studies. The second approach focused upon the railroad worker studies in developing the range of unit cancer risks.

Meta-analysis-Derived Cancer Unit Risks

The results of the meta-analysis provide information useful in bracketing the broadest likely range of plausible carcinogenic potencies for diesel exhaust. The pooled relative risk values derived from the 12 epidemiological studies in the meta-analysis which adjusted for smoking were 1.44 (95% C.I. 1.32 -1.56) for the fixed effects model and 1.43 (95% C.I. 1.31 -1.57) for the random effects model. The magnitude of these relative risks provide information on the potential magnitude of the cancer risk associated with diesel exhaust exposure. For the random effects model the upper 95% confidence limit on excess relative risk is 0.57.

None of the studies in the meta-analysis provide direct measurements of exposure concentration over the time of their follow up. Therefore, to the extent that the meta-analysis can be used to bracket the carcinogenic potency of diesel exhaust, the exposures of the various study populations need to be reconstructed. Hammond (1998) has reviewed the available industrial hygiene survey literature on the occupations considered in the meta-analysis (bus garage workers, mechanics, truck drivers, heavy equipment operators, railroad workers) and provided estimates of the plausible possible ranges of workplace exposures of diesel exhaust respirable particulate matter for those occupations. Because of the overall limitations in the data, the estimated ranges for each occupational subgroup of interest are particularly broad. The lowest plausible estimate of occupational exposure for any such subgroup is $5 \mu\text{g}/\text{m}^3$ (heavy equipment operators). The highest plausible estimate of any occupational subgroup is $500 \mu\text{g}/\text{m}^3$ (bus garage workers, railroad workers, mechanics). The total range of plausible exposures for the different populations therefore varies 100-fold. Using these air concentrations and the assumption that workers inhaling 10m^3 of air per work shift were exposed to them for over 45 year period for a 70 year lifetime, it is possible to characterize a bracket of risks compatible with the results of the meta-analysis:

$$\begin{aligned}
q_1^* &= \text{Excess relative risk} \times \text{CA lifetime lung cancer risk.} \\
&\quad \text{Air concentration} \times \text{exposure factor} \times \text{intermittency factors} \times \text{duration of} \\
&\quad \text{exposure/lifetime} \\
&= 0.57 \times 0.025 \\
&\quad (5 \text{ or } 500 \mu\text{g}/\text{m}^3) \times 10 \text{ m}^3/\text{shift}/20\text{m}^3/\text{d} \times 5\text{d}/7\text{d} \times 48\text{wk}/52\text{wk} \times 45 \text{ yrs}/70\text{yrs}
\end{aligned}$$

Therefore, the results of the meta-analysis bracket lung cancer risks up to approximately $1.3 \times 10^{-4} (\mu\text{g}/\text{m}^3)^{-1}$ (assuming all the worker populations in the meta-analysis were exposed to $5 \mu\text{g}/\text{m}^3$) to $1.3 \times 10^{-2} (\mu\text{g}/\text{m}^3)^{-1}$ (assuming all the workers populations in the meta-analysis were exposed to $500 \mu\text{g}/\text{m}^3$). As these assumptions establish the extreme bounds of probable exposures, and such calculations based upon a meta-analysis are novel and subject to further possible refinements, these results are not incorporated into the range of risks. However, these results do bracket the carcinogenic potencies which would be consistent with the results of the meta-analysis and the broadest range of exposure estimates.

A more plausible range can be estimated by determining the 90% confidence interval (CI) of the range of risks. For the meta-analysis the range of concentrations thought to be plausible by Hammond (personal communication) was 5 to $500 \mu\text{g}/\text{m}^3$ with a mean of about $200 \mu\text{g}/\text{m}^3$, which corresponds to a unit risk of $3.3 \times 10^{-4} (\mu\text{g}/\text{m}^3)^{-1}$. Using that concentration range as the 98% CI for a shifted lognormal distribution fixes the geometric standard deviation at 1.22 with a shift of the origin of the distribution by $330 \mu\text{g}/\text{m}^3$. The 90% CI for this distribution of concentration is [52.5 to $356.5 \mu\text{g}/\text{m}^3$], corresponding to a 90% CI for the distribution of unit risk of [1.6×10^{-4} to $1.2 \times 10^{-3} (\mu\text{g}/\text{m}^3)^{-1}$].

Railroad Worker Study-Derived Cancer Unit Risks

Quantitative relationships were also developed between lung cancer risk and exposure to diesel exhaust for two nation-wide studies of lung cancer rates in U. S. railroad workers. These relationships provided additional values for the range of risk to the general California population. The first, Garshick *et al.* (1987a), is a case-control study. Using a logistic regression, that study determined the coefficient of the logistic relationship of the odds of lung cancer for duration of the workers' exposure to diesel exhaust. The coefficient determined in that study was used to estimate lifetime unit risks for exposure of the general population. The second study, Garshick *et al.* (1988), is a cohort study. Using a proportional hazards model, that study calculated the relative hazard of lung cancer for increasing duration of worker exposure. However, those numerical results have not been supported by Garshick (1991); so instead of using them to derive lifetime unit risks for the general population, new analyses were performed with the individual data, upon which that study is based, to determine a linear relationship of lung cancer hazard for worker exposure to diesel exhaust.

The term hazard was used for a prediction of incidence (cancers per year per population) resulting from a model. Relative hazard is generally called relative risk in epidemiological model work, and the term, relative risk, was used in the context of the epidemiology results. The lifetime inhalation unit risk, often simply called unit risk, is defined as the probability of contracting lung cancer from a 70-year exposure to a unit concentration ($1 \mu\text{g}/\text{m}^3$) of diesel exhaust.

The unit risks ultimately derived for the general population assume that the mass concentration of particles governs the risk of diesel exhaust, regardless of the particular type of diesel engine or fuel. The resulting estimate of risk entails uncertainties due primarily to the limited exposure information available and to the choice of models and data used in the analysis.

These two studies are among a number of studies establishing excess relative risk of lung cancer among workers exposed to diesel exhaust. These two studies were specifically selected for the quantitative risk assessment because of their general excellence, their apparent finding of a relationship of cancer rate to duration of exposure and because of the availability of measurements of diesel exhaust among such railroad workers from the early 1980's in other studies. The case-control study appears to have an advantage in obtaining direct information on smoking rates, while the cohort study has an advantage of smaller confidence intervals of the risk estimates.

Estimating Cumulative Exposure

The risk relationships developed for the case-control study and the initial analyses for the cohort study used cumulative atmospheric exposure to diesel exhaust particles as the effective dose. The use of cumulative exposure, defined as the area under the curve (AUC) of concentration versus time, required a specification of the temporal pattern of exposure concentration. However, direct measurements of exposure concentration over the time of the follow up were not available.

Therefore, the calculations required reconstruction of the exposure history in order to determine cumulative exposure. The reconstruction was undertaken using (1) personal exposure measurements on railroad workers just after the end of the follow-up period in that study, (2) historical data on the dieselization of locomotives in the United States, and (3) descriptive information. The analysis included workers on trains and excluded shop workers from the original cohort because of mixed exposures, including no exposure to an unknown number in this group.

Exposure Measurements In The Early 1980s

Woskie *et al.* (1988b) estimated national average concentrations of respirable particulate matter (RSP) for 13 job-groups. These concentrations were obtained by temperature correction of measurements of respirable particulate matter (RSP) made in 1982-1983 in the northern region of the United States, as reported in Woskie *et al.* (1988a). The investigators adjusted these concentrations to remove the portion of RSP attributable to environmental tobacco smoke (ETS). The average values of the ETS-adjusted RSP for the principal categories of workers are listed in Table 3 for exposed and unexposed workers.

Table 3: Number of Workers in the Exposure Categories and the Cohort Averages of the Worker Exposure Concentration Following the Garshick *et al.* (1988) Cohort Study.

Exposure status	Career group	Number of workers	Subsequent exposure concentration ^a ($\mu\text{g}/\text{m}^3$)
Uncertain	Shopworkers	12,092	141(those exposed)
Exposed ^b	Engineers, firemen	11,005	71
	Brakemen, conductors, hostlers	18,285	89
Unexposed ^c	Clerks	10,475	33
	Signalmen	3548	58

^a Exposures reported by Woskie *et al* (1988b) for these career groups, based on measurements of ETS-adjusted RSP, circa 1982-3.

^b For all exposed workers in the table, except for those shopworkers who were exposed, the temporal exposure patterns are assumed to be the same, and the concentrations are close to each other; so a simple population-weighted average for the two career groups characterizes the average concentration for the exposed group, train workers, circa 1982-83:

$$(11,005 \times 71 + 18285 \times 89) / (11005 + 18285) = 82 \mu\text{g}/\text{m}^3$$

^c For all unexposed workers (background) in the table except for those shopworkers who were unexposed, the concentrations are close to each other; so a simple population-weighted average for the two groups characterizes the average background concentration, circa 1982-83:

$$(10475 \times 33 + 3548 \times 58) / (10475 + 3548) = 39 \mu\text{g}/\text{m}^3.$$

Reconstruction Of The Time Course Of Concentration

In order to estimate the time course of the exposure factors for the cohort, it was necessary to make assumptions about time trends of nationwide average concentration breathed by the workers. The exposure measurements made just after the follow-up period constitute a baseline for the reconstruction. The reconstruction of the time course of concentration proceeds by developing an exposure factor to multiply these baseline values. The analyses below explore the effect of alternative patterns of exposure concentration and baseline values.

Dieselization of the U.S. railroads began after the Second World War ended in 1945. The exposure of the railroad workers up until 1981 can be divided into two periods: (1) an initial period of increasing dieselization of U.S. locomotives from 1945 until mostly completed in 1959 and (2) a subsequent period of a moderate rate of addition of locomotives that were less smoky.

Woskie *et al.* (1988b) reported data showing a linear rise of percent dieselization with time in the first period from 1945 to 1959. They reported data from the Bureau of Labor Statistics showing that by 1947 fourteen percent of locomotives were diesel, by 1952 fifty-five percent were diesel, and by 1959 ninety-five percent were diesel. This linear rise of dieselization may be expected to have produced a linear rise of the national average exposure concentration around the trains. This linear rise is used in all the more realistic exposure patterns.

The exposure of workers on trains would then generally have declined as the newer, less smoky locomotives replaced the older, smokier locomotives on the main lines. To quantify the anecdotal information of greater smokiness of locomotives in the period before 1960, the national average exposure concentration was assumed to decline linearly in the second period, 1960-1980, to the baseline measured in 1982-3. The decline assumed from 1959 to 1980 is consistent with the report of sharp decreases of emissions of new engines between the 1970's and the 1980's. Emissions from naturally aspirated four-stroke engines declined from 2.1-3.0 g/kW-hr in the 1970's to 0.25 -0.6 g/kW-hr in the 1980's (Sawyer and Johnson, 1995).

In order to bracket the exposure of the railroad workers to diesel exhaust a variety of patterns of exposure are considered. The patterns are characterized by two components: a) the extent of change from 1959 to 1980 in diesel exhaust exposure, expressed as a ratio, and b) the average exposure concentration for the workers on trains measured in the Woskie *et al.* (1988a) study (i.e., the baseline). The alternate ratios are as follows: a) a ratio of 1 suggested and used in Crump *et al.* (1991) as more realistic than the Garshick *et al.* (1987a, 1988) assumption of constant concentration from 1959-1980 and none before that; b) a ratio of 2 suggested by K. Hammond to allow for a modest peak in 1959; c) a ratio of 3 allowing for more peak, a scaled down version of the exposure factor of 10 that Woskie *et al.* (1988b) reported for exposure concentration of shopworkers to nitrogen dioxide in enclosures including engine test sheds; and d) a ratio of 10, peak of the magnitude of values for the engine test sheds. The alternate baselines of exposure concentrations are as follows: 1) 40 $\mu\text{g}/\text{m}^3$, obtained by subtracting the background measurement of the unexposed workers from the measurement of the train workers, rounded down; 2) 50 $\mu\text{g}/\text{m}^3$, which also subtracted background from the train worker measurements but rounded up to allow somewhat for measurements of workers on trains not having as much exposure to non-diesel exhaust background particulate as the clerks; and 3) 80 $\mu\text{g}/\text{m}^3$, obtained by assuming that the entire ETS-adjusted RSP of the train workers is diesel exhaust while the clerks are considered unexposed to diesel exhaust (0 concentration).

The specific alternative patterns of linear decline (if any) of concentration from 1959 through 1980 are:

1. no decline, constant at the baseline values of 50, a ramp (1,50) pattern suggested and used in Crump *et al.* (1991).
2. declining 3-fold from a peak of 150 to a baseline of 50, a roof (3,50) pattern, the preferred pattern in this report;
3. declining 10-fold from a peak of 500 to a baseline of 50, a roof (10,50) pattern, suggested in information submitted by the Engine Manufacturers Association;
4. declining, 2-fold from a peak of 80 to a baseline of 40, a roof (2,40) pattern suggested by K. Hammond, one of the investigators in the Woskie *et al.* study; and
5. declining 3-fold from a peak of 240 to a baseline of 80, a roof (3,80) pattern, a variant on Pattern 3 for not subtracting background ETS-adjusted RSP in the exposed group while still maintaining
6. unexposed workers at zero concentration.

Calculation Of Cumulative Exposure

The estimate of the time course permits calculation of the overall average cumulative exposure for the cohort for each year of the follow-up period, 1959-1980. The cumulative exposure factor was calculated as the area under the curve (AUC) of the exposure factor (EF, ratio of concentration to baseline concentration) for successive years. Cumulative exposure is the cumulative exposure factor times the baseline value.

Intermittency Correction

The equivalent exposure duration for non-continuous exposure was scaled on the basis of volume of air breathed. Exposure durations are calculated to have the same cumulative yearly intake of the substance as produced by continuous inhalation of 20 m³/day at the concentration of the substance breathed in. Assuming that the average exposed member of the cohort inhales 10 m³ during an 8-hour working day implies an adjustment factor of 10/20 to multiply the exposure concentration to account for ventilation rate not equaling the standard human daily inhalation of 20 m³/day. Adjusting for the discontinuous work week and work year yields additional adjustment factors of 5/7 for exposure days per week and 48/52 for weeks per year, all to multiply the exposure measure. In order to take account of the non-continuous work exposure, the resulting overall multiplicative factor on exposure duration is

$$(10/20)(5/7)(48/52) = 0.33.$$

Determining Lifetime Unit Risk From The Relative-Risk Slope

The analyses below calculate the relationship between relative risk (relative hazard) and duration of exposure. The relative risk is the prediction of the ratio: incidence (yearly death rate per population) of lung cancer due to diesel exhaust divided by the background incidence of lung cancer. In the principal modeling of both sets of epidemiological data, reported below in this chapter, relative risks are fitted linearly to duration of exposure. From that slope, an estimate of the slope with respect to cumulative exposure for the specific alternative patterns of occupational exposure considered is obtained by modifying the duration scale for the slope. The approximation for this modification is simply to multiply the duration scale by the overall area under the curve (AUC) of the desired pattern and to divide by the total duration of exposure in the analysis.

Approximations may often be used to determine lifetime unit risk from this slope, but the present work will, for consistency and accuracy, use life-table calculations for that determination. This calculation starts with a background life table for lung cancer in California. For each unit risk to be calculated, a modification of that table is constructed in a way that includes the predicted effect of a lifetime exposure to 1 unit of concentration, 1 µg/m³ in the present calculations. The predicted effect is incorporated by multiplying the background lung cancer incidence for each age interval in the table by the relative risk (relative hazard) for that age interval. The relative risk is (1+ excess relative risk due to exposure). The excess relative risk due to exposure for unit concentration is the slope of relative risk with concentration, obtained from the epidemiological analyses. Using the general model based on cumulative exposure, as in the present calculations,

the excess relative risk requires the slope coefficient per concentration-year to be multiplied by the age in years for each age group in the table and to be divided by the intermittency factor. Any ages that fall within the number of years of detection lag prior to the target age have zero excess relative hazard. The modified table is completed in the manner of the original table. The lifetime unit risk is then the following difference: the probability of lung cancer at the target age in the table modified by exposure less the probability at the same age in the original table.

Use of the Garshick *et al.* (1987a) Case-Control Study to Estimate Unit Risk

The first study used to estimate lung cancer risk due to diesel exhaust exposure is the case-control study of U.S. railroad workers by Garshick *et al.* (1987a). For this case-control study Garshick *et al.* (1987a) collected 15,059 US railroad worker death records for 1981. They matched each of 1256 lung-cancer cases with 2 other deaths, each of those having nearly the same date of birth and death. For each of the controls, death was due to a specified natural cause with no mention of cancer on the death certificate. For each subject, Garshick *et al.* (1987a) determined years in a job with diesel exposure, asbestos exposure and smoking history. Taking into account the effect of age, their analysis used multivariate conditional logistic regression to determine the relationship between lung cancer and duration of exposure to diesel exhaust. For workers with more than 20 years exposure and for exclusion of shopworkers, they calculated the odds ratio was 1.55 (95% CI = 1.09, 2.21) with a referent category of 0 to 4 years work in a job exposed to diesel exhaust.

From the odds ratio for a 20 year duration of exposure, the coefficient of increase with duration of exposure was estimated by assuming a linear rise over the 20 years. Using a calculation similar to that used by Garshick *et al.* with shopworkers included, the slope coefficient for the odds ratio is 0.022 (90% C.I. = 0.0071, 0.037) year⁻¹. Because the odds ratio approximates relative risk (Breslow and Day, 1980, pp. 69-73), this value is approximately the rate of increase of relative risk (relative hazard) and is used in a life table to obtain the lifetime unit risk. The modified life table calculation for unit concentration (1 µg/m³) for 5-yr. lag from carcinogenesis to death is in Table 7-1 of the diesel exhaust TAC document (OEHHA, 1998). The resulting unit risks are presented in Point I in Table 7-3 of the diesel exhaust TAC document. The highest values in that set are for the assumption that workers on trains have a ramp (1,50) pattern of exposure. The 95% UCL for lifetime unit risk is 2.4×10^{-3} (µg/m³)⁻¹, with an MLE of 1.4×10^{-3} (µg/m³)⁻¹. For the roof (3,50) pattern of exposure, the procedure is similar, but the exposure scale is increased by the ratio 65/22, representing the ratio of area under the EF of the roof to the area under the EF of the block. The resulting 95% UCL for lifetime unit risk is 1.0×10^{-3} (µg/m³)⁻¹, with an MLE of 6.2×10^{-4} (µg/m³)⁻¹. The lowest values in the set are for the roof (10,50) pattern of exposure. Using a similar approach, multiplying the exposure scale by the AUC ratio of 191/22, the 95% UCL for lifetime unit risk is 3.6×10^{-4} (µg/m³)⁻¹, with an MLE of 2.1×10^{-4} (µg/m³)⁻¹.

Using the slope coefficient for the analysis including shopworkers, reported in Garshick *et al.* (1987a), McClellan *et al.* (1989) previously calculated the expected increase in U.S. lung cancer deaths per year for each µg/m³ of diesel exhaust exposure for two alternative exposure concentrations, 125 µg/m³ and 500 µg/m³, constant from 1959-1980. Mauderly (1992a) used these death rates to estimate unit risks, finding expected values of 1.2×10^{-3} (lifetime-µg/m³)⁻¹

and 2.9×10^{-4} (lifetime $\mu\text{g}/\text{m}^3$)⁻¹, respectively. These values are close to the higher MLE values just given. Even though the higher concentrations assumed by McClellan *et al.* would tend to produce lower unit risks, the effect of using the more accurate life table method has a counteracting effect.

Use of the Garshick *et al.* (1988) Cohort Study to Estimate Unit Risk

The second study selected to estimate lung cancer risk due to diesel exhaust exposure was the retrospective cohort study of U. S. railroad workers by Garshick *et al.* (1988). The present analysis uses the individual data collected for that study in new calculations to determine slopes for the relationship of incidence to cumulative exposure. The analysis uses reconstructions of exposure, the ramp and the roof exposure patterns, to adjust the slope obtained from the analysis that is implemented with duration of exposure as the measure of exposure.

Further material on the cohort is developed in Appendices D, E, F of the diesel exhaust TAC document (OEHHA, 1998). Appendix E contains references to correspondence cited in this chapter. (The original unpublished documents referred to in Appendix E are available on request from the California Air Resources Board, Stationary Source Division or from the U.S. EPA docket for the Health Assessment Document for Diesel Emissions at the National Center for Environmental Assessment, Washington, DC. 20460 (1997)).

Description of the Original Study

The cohort consisted of 55,407 railroad workers, who were aged 40-64 in 1959 and who had started railroad service 10-20 years earlier; 1694 lung cancers were identified. The unexposed group in the cohort, the clerks and signal tenders, constituted 25.3% of the whole cohort. To develop the original data set, Garshick *et al.* (1988) obtained the following information for each individual in their cohort of railroad workers for the follow-up years of 1959-1980: cause of death by death certificate, the primary job classification for each year, and months worked in that classification in each year. In addition, the investigation obtained the age at the start of follow-up in 1959, total service months and, for those workers who began work after 1946, the date of starting work. From these data Garshick *et al.* calculated the elapsed time of exposure for each individual from 1959 up to each follow-up year or up to the four years before each follow-up year.

Relative Risk Analysis

Because of much uncertainty about the proportion of shop workers exposed to diesel exhaust, OEHHA decided to exclude them from the analysis, as suggested by the study authors and other participants at the Diesel Exhaust Workshop, January, 1996. Garshick (1991) had previously called attention to dilution of the effect of diesel exhaust on the shop workers because of the inclusion of shopworkers in that cohort who had no true exposure. The original study obtained risk estimates both with and without the shop workers, and found the results changed very little. The exclusion of shop workers simplifies the analysis in that lung burden calculations are not needed because the exposures of other exposed workers, namely train workers, are sufficiently low that lung burden may be assumed essentially proportional to atmospheric exposures.

Exposure measurements for 1982-83 (Woskie *et al.* 1988a), just after the end of the follow-up period, show that train workers considered here all experienced approximately the same average concentration of diesel exhaust (for example, $50 \mu\text{g}/\text{m}^3$, rounded, for use in determining unit risk in this work). The present work uses years with any month of exposure time, excluding the four years previous to each year of observation as the average lag time from carcinogenesis to death. This calculation of exposure time starts in 1952 and continues yearly through 1980, the end of follow-up. It extends 7 years back from 1959, the start of follow-up, to account on the average for the assumed linear rise of exposure from 1945 to 1959. The unexposed workers are assigned zero exposure time throughout.

The OEHHA analysis uses two programs in the EPICURE software package, which is designed for several standard kinds of epidemiological analysis. The first program, DATAB, reduces the individual data to cells with each desired variable having a single value for the cell. The cells are designated by a set of numbers, one for each categorical variable to determine the category number of that variable. The second program, AMFIT, determines parameters of a model to provide a best fit of the data using Poisson regression, a maximum likelihood procedure (Breslow and Day, 1987). The calculation approach is described in more detail for the closely related calculations using general models, in Appendix D of the diesel exhaust TAC document (OEHHA, 1998).

The assumptions not otherwise specified here are essentially those of Garshick *et al.* (1988). For example, all years of the study are included, and their rather irregular boundary points on years of exposure are used.

The OEHHA analysis explored the fit and other characteristics of a number of forms of a general model. The model that appeared to be most satisfactory is the one with linear and quadratic continuous covariates, age and calendar year. The slope calculated for relative risk (relative hazard) per year of exposure is 0.015 (95% CI: 0.0086 to 0.022) year^{-1} . The slope divided by the intermittency correction (0.33) and the assumed constant concentration (e.g., $50 \mu\text{g}/\text{m}^3$ for 29 years) and multiplied by attained age provides the excess relative hazard to determine the increase of lung cancer rates for the lifetable calculation of the unit risk. The resulting unit risks are presented in Point II in Table 4, and closely parallel the results for the case-control study (Point I). The highest values in that set are for the assumption that workers on trains have a ramp (1,50) pattern of exposure. For the ramp pattern the result is a 95% UCL of $1.8 \times 10^{-3} (\mu\text{g}/\text{m}^3)^{-1}$ and a MLE of $1.3 \times 10^{-3} (\mu\text{g}/\text{m}^3)^{-1}$. For the roof (3,50) pattern of exposure, the procedure is similar, but the exposure scale is increased by the ratio $65/29$, representing the ratio of area under the EF of the roof to the area under the EF of the ramp. The result is a 95% UCL of $8.2 \times 10^{-4} (\mu\text{g}/\text{m}^3)^{-1}$ and a MLE of $5.7 \times 10^{-4} (\mu\text{g}/\text{m}^3)^{-1}$. The lowest values in the set are for the roof (10,50) pattern of exposure. Using a similar approach, multiplying the exposure scale by the AUC ratio of $191/29$, the 95% UCL for lifetime unit risk is $2.8 \times 10^{-4} (\mu\text{g}/\text{m}^3)^{-1}$, with an MLE of, $1.9 \times 10^{-4} (\mu\text{g}/\text{m}^3)^{-1}$.

Table 4: Values from Unit Risk for Diesel Exhaust from Using Hazard Slope on Exposure Measure in California Life-Table. Garshick *et al.* (1987a, 1988) Studies of U.S. Railroad Workers.

	q_1 ($\mu\text{g}/\text{m}^3$) ⁻¹	
	MLE	95% UCL
<u>I. Case-Control study (1987a) using published slope coefficient for hazard on years of exposure to diesel exhaust (Section 7.3.3)</u>		
A. Adapted to ramp (1,50) pattern of exposure	1.4×10^{-3}	2.4×10^{-3}
B. Adapted to roof (2,40) pattern of exposure	1.1×10^{-3}	1.8×10^{-3}
C. Adapted to roof (3,50) pattern of exposure	6.2×10^{-4}	1.0×10^{-3}
D. Adapted to roof (3,80) pattern of exposure	3.9×10^{-4}	6.6×10^{-4}
E. Adapted to roof (10,50) pattern of exposure	2.1×10^{-4}	3.6×10^{-4}
<u>II. Cohort study (1988) using individual data to obtain a slope for hazard on years of exposure to diesel exhaust (Section 7.3.4)</u> <u>Continuous covariates: (attained age and calendar year)</u> <u>or (age-at-start-of study and calendar year)</u>		
A. Adapted to ramp (1,50) pattern of exposure	1.3×10^{-3}	1.8×10^{-3}
B. Adapted to roof (2,40) pattern of exposure	9.9×10^{-4}	1.4×10^{-3}
B. Adapted to roof (3,50) pattern of exposure	5.7×10^{-4}	8.2×10^{-4}
D. Adapted to roof (3,80) pattern of exposure	3.6×10^{-4}	5.1×10^{-4}
E. Adapted to roof (10,50) pattern of exposure	1.9×10^{-4}	2.8×10^{-4}
<u>III. Cohort study (1988) applying time varying concentrations to individual data to obtain a slope of hazard on exposure (from Appendix D)</u>		
A. Ramp (1,50) pattern of exposure		
1. General multiplicative model with age-at-start-of-study and U.S. rates as categorical covariates	1.2×10^{-3}	1.9×10^{-3}
2. 6th/7-stage model with age-at-start-of study as categorical covariate	2.4×10^{-4}	3.8×10^{-4}
B. Roof (3,50) pattern of exposure		
1. General multiplicative model with age-at-start-of-study and U.S. rates as categorical covariates	5.1×10^{-4}	7.2×10^{-4}
2. 6th/7-stage model with age-at-start-of-study as categorical covariate	8.1×10^{-5}	1.3×10^{-4}
3. 7th/7-stage model with age-at-start-of-study as categorical covariate	1.0×10^{-4}	1.5×10^{-4}

Discussion of Results

The investigation of the forms of the model using Poisson regression explored the use of categorical covariates, calendar year and age-at-start-of-follow-up that verified the categorical trend with exposure that Garshick *et al.* (1988) had obtained for relative hazard by using a Cox regression with calendar year as the principal time scale and age-at-start-of-follow-up as a covariate. This result was an elevated relative risk (relative hazard) for the middle durations of exposure and an apparent rise at the highest exposure, albeit with large error bars. Crump (1997) found by direct comparison a close correspondence of results for this Poisson regression and a Cox regression that replicated Garshick *et al.*

The investigation also explored the use of a general model with the categorical covariates, calendar year and attained age, that verified the categorical results for relative risk in Crump *et al.* (1991) and Crump (1997). This result showed a rise and then an apparent fall of relative risk for increasing exposure. Age and calendar year are important determinants of lung cancer rate, and Crump (1997) has argued that this choice should be used for covariates because it is the most accurate in characterizing background rates and, further, that a fall of relative risk at the higher exposure, obtained for this choice of covariates, is not consistent with an exposure response.

It should be kept in mind that the categorical trends of the relative risk with duration of exposure are all used to represent a large cloud of observed points of incidence as a function of duration of exposure. Appendix F of the diesel exhaust TAC document (OEHHA, 1998) indicates that the discrepancy between the results of Garshick *et al.* and of Crump *et al.* may be more apparent than real. The slopes for the relative risk are significant for both these choices of covariate, but the slope for the use of calendar year and age-at-start is about twice that for the use of calendar year and attained age. The latter slope is larger, though less significant, than the identical slope obtained in the present analysis using continuous forms of either pair of covariates. The use of the continuous form of the covariates appears to have a salutary effect on reducing the variance of the slope estimate. This choice allows some flexibility, but not a lot, in describing time trends.

Conclusion

Based on the human data, the principal finding of the diesel exhaust TAC document quantitative risk assessment is a range of lifetime unit risk (95% UCL) as shown in the right-hand column of Table 4 above. The lowest value in the range is 1.3×10^{-4} , and the highest value is 2.4×10^{-3} . The geometric mean unit risk obtained from these end points of the range of values is 6×10^{-4} (lifetime- $\mu\text{g}/\text{m}^3$)⁻¹. The geometric mean provides information on the central tendency of the range and is not to be confused with a best estimate identified from the available calculations. The lower end of the range is the rounded value for both forms of multistage model using the roof exposure pattern for the data of the Garshick *et al.* (1988) cohort study of U.S. railroad workers. OEHHA concluded that incorporation of the roof exposure pattern and biologically-based analyses improved the unit risk estimates. Consequently, unit risk values incorporating this information, those at the lower end of the range, provide more scientifically defensible values. The upper end of the range is obtained using the published results of the Garshick *et al.*

(1987a) case-control study for US railroad workers. The Scientific Review Panel concluded in their findings that a reasonable estimate of the cancer unit risk is $3 \times 10^{-4} (\mu\text{g}/\text{m}^3)^{-1}$.

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CHLOROFORM

CAS No.: 67-66-3

I. PHYSICAL AND CHEMICAL PROPERTIES (From HSDB, 1998)

Molecular weight	119.49
Boiling point	61° C
Melting point	-63.5° C
Vapor pressure	200 mm Hg 25° C
Air concentration conversion	1 ppm = 4.9 mg/m ³ at 25° C

II. HEALTH ASSESSMENT VALUES

Unit Risk Factor:	5.3 E-6 (µg/m ³) ⁻¹
Slope Factor:	1.9 E-2 (mg/kg-day) ⁻¹

[Calculated by CDHS (1990) using a nonthreshold linear procedure. This unit risk is the arithmetic average of unit risks generated by CDHS and Bogen *et al.* (1989) for renal tumors observed in rats and mice reported by Jorgenson *et al.* (1985) and NCI (1976), and the geometric mean for supporting data sets (Roe *et al.*, 1979; Tumasonis *et al.*, 1985).]

III. CARCINOGENIC EFFECTS

Human Studies

There is no information currently available in the open literature which examines the potential relationship between exposure to chloroform in an occupational setting and human cancer. However, several studies are available which examine the relationship between trihalomethanes (THM) in drinking water and human cancer.

Many studies have concentrated on chlorination of water and concomitant production of halogenated carcinogens as a causative factor in human cancers. Cantor *et al.* (1978) compared age-adjusted cancer mortality rates by site and sex for whites in the years 1968-71 to measures of THM and the drinking water. A weighed linear regression model was used to predict cancer rates in 923 U.S. counties which were over 50% urban in 1970. Reasonably strong associations between bladder cancer and THM levels in drinking water were found after controlling for confounding by urbanization, ethnicity, social class, and county industrialization. The association was not changed by controlling for occupation in certain high-risk (for bladder cancer) industries nor by lung cancer rates used as a surrogate measure for cigarette smoking. The measure of THM most associated with bladder cancer in both white males and females was that of bromine-containing trihalomethanes (BTHM). Chloroform and total trihalomethanes (TTHM) were not as well associated. There were inconsistent associations between other cancer sites and THM levels. However, there was some evidence of an association of chloroform in

drinking water with kidney cancer in males, which Cantor *et al.* believed warrants further study.

Hogan *et al.* (1979) examined the potential association between chloroform levels in finished drinking water supplies and various site-specific cancer mortality rates. The most consistent associations were between chloroform “exposure” and cancers of the bladder, rectum and large intestine. Hogan *et al.* stated that the results of this ecological study must be interpreted with caution and the association between chloroform levels in drinking water and certain types of cancer (e.g., bladder, large intestine and rectum) warrant further study.

Carlo and Mettlin (1980) analyzed 4,255 cases of cancer reported in Erie County, NY, between 1973 and 1976 for any relationship between cancer and type of water source, THM levels, and a variety of socioeconomic variables. No significant association between THM and cancers were noted in the regression analyses for the total population. When regression analyses were conducted for population stratified by race-sex, a significant association was found between THM levels in drinking water and pancreatic cancer in white males ($p < 0.05$). The investigators caution that the lack of association between THM and pancreatic cancer in other sex-race groups and absence of association between THM and other cancer raises doubts as to the validity of this finding.

Brenniman *et al.* (1980) conducted a case-control study in Illinois to determine whether an association exists between chlorination of drinking water and gastrointestinal and urinary tract cancers. Cases (3,208) and controls (43,666) were classified according to residence in chlorinated and unchlorinated groundwater communities. Elevated risk was found for cancers of the gallbladder, large intestine, total gastrointestinal, and urinary tract for women. However, the investigators considered the results tenuous because, when the data were subclassified according to several control variables, the associations were not strengthened. Many confounding factors were not controlled including smoking, diet, ethnicity, and occupation.

Alavanja *et al.* (1980) conducted a case-control study on all gastrointestinal and urinary tract cancer deaths occurring from January 1, 1968 through December 31, 1970 in seven counties in New York. There was a statistically significant excess risk of stomach cancer in females, and of stomach, esophagus, large intestine, rectum, liver and kidney, pancreas, and urinary bladder in males residing in chlorinated water areas in the seven counties studied. The investigators concluded that the excess risk was associated with living in chlorinated areas of certain counties and was not due to a disparity in the age, race, or ethnic distribution, or to urban/rural classification, hazardous occupation, or a surface vs. ground water difference. Several confounding factors were not controlled including cigarette smoking and diet.

The association between site-specific cancer mortality and THM exposure, as estimated by chlorine dose, was investigated by Young *et al.* (1981). Cases were obtained from death certificates provided by the Wisconsin Bureau of Health Statistics and consisted of all white female deaths that occurred 1972-77 within 28 counties due to malignant

neoplasms of esophagus, stomach, colon, rectum, liver, bile ducts, pancreas, urinary bladder, kidney, lung, breast, and brain. Only death from colon cancer was associated with chlorine dose ($p < 0.05$). The risk of colon cancer, calculated as odds ratios, was over twice as great when the water source was affected by rural runoff. This variable was tested because of the assumption that rural runoff increased the organic precursors to THMs. While the association of colon cancer with chlorination and rural runoff factors is provocative, the findings of this study must be considered inconclusive due to the possible underestimation of risk associated with misclassification error and spurious contribution from unknown colon cancer risk factors (Young *et al.*, 1981).

Wilkins and Comstock (1981) conducted a nonconcurrent prospective study to investigate possible relationships between products of water chlorination and human cancer. Site and sex-specific incidence rates for malignant neoplasm of liver, biliary passages, kidney, and bladder were constructed from hospital records, a cancer registry, and death certificates. Incidence rates for cancer of the bladder among men and cancer of the liver among women were not significant relative to the other exposure groups among persons using water from the chlorinated surface supply. While the results were only weakly suggestive, Wilkins and Comstock noted that bladder cancer has been suggestively linked with chloroform and other indices of THM in drinking water in other studies.

Gottlieb and Carr (1982) studied the potential relationship between chlorination of drinking water and cancer in 20 south Louisiana parishes. Chlorinated surface water was associated with a significant risk for rectal cancer ($p = 0.012$). The odds ratio for rectal cancer in groups receiving high chlorination level (> 1.09 ppm chlorine) to groups with no chlorinations is 1.53 (95% CI=1.15-2.04) in surface water supplied areas. Gottlieb and Carr concluded that there appears to be some cancer risk associated with water chlorination, but definitive studies are needed with respect to the role of industrial confounders and the importance of co-contaminants.

Lawrence *et al.* (1984) used a case-control approach to study the association of chloroform exposure via drinking water to colorectal cancer in white female teachers in upstate New York. Analysis was based on 395 cases of colon and rectal cancer and 395 control noncancer deaths matched with respect to age and year of death. No effect of cumulative chloroform exposure on incidence of colorectal cancer deaths was observed.

Cantor *et al.* (1987) examined the association between use of chlorinated drinking water and bladder cancer by a case-control study design. The investigators interviewed 2,982 cases and 5,782 controls in 10 geographic areas of the U.S. Risk of bladder cancer was primarily associated with use of tap water rather than nontap beverages. Among white males, the coefficients for tap and nontap beverages were 0.176 ($p < 0.001$) and 0.037 ($p = 0.42$), and among white females, the coefficients were 0.123 ($p = 0.09$) and 0.089 ($p = 0.39$), respectively. It was suggested that nonvolatile components of tap water may be associated with risk of bladder cancer since both heated and nonheated tap water beverages were significantly associated with bladder cancer risk among males. The relative risk increased with increasing tap water intake. While this investigation was

quite thorough in many respects, there is a need for confirmation of these findings. The contribution of chloroform in the etiology of human bladder cancer in men may be overshadowed by other nonvolatile chemicals present in the drinking water.

Overall, the present epidemiological evidence suggests an association between chlorinated drinking water consumption and human cancer, particularly bladder and gastrointestinal cancers. However, these relationships cannot be directly correlated to chloroform exposure because many other carcinogens are found in drinking water including other chlorinated halomethanes, brominated halomethanes, industrial pollutants, and nonvolatile halogenated compounds.

Animal Studies

The National Cancer Institute conducted carcinogenesis bioassays of chloroform in both sexes of Osborne-Mendel rats and B6C3F₁ mice (NCI, 1976). Mice and rats were given either corn oil or chloroform in corn oil by gavage, 5 days/week for 78 weeks. Time-weighted average doses for female rats were 100 and 200 mg/kg, and for male and female mice were 138 and 277 mg/kg, and 238 and 477 mg/kg, respectively. Tumor incidences are listed in Table 1.

A statistically significant increase ($p < 0.05$) in epithelial tumors of renal tubular origin was noted in the treated males. Ten carcinomas, two of which had metastasized, and three adenomas of renal tubular origin were found in 12 high dose male rats. In the low dose males, two carcinomas and two adenomas of tubular origin were observed in four out of 50 animals. Among the 48 high dose female rats, one tubular epithelial carcinoma and one renal squamous cell carcinoma were observed. No renal epithelial tumors were noted in matched or colony controls. The NCI reported that these type of tumors rarely occur spontaneously in Osborne-Mendel rats.

The incidence of thyroid tumors in female rats was statistically higher than controls in both treated groups ($p = 0.05$, Fisher exact test) but not in treated male rats. The incidence of hepatocellular carcinoma or neoplastic nodules was not increased in the chloroform-treated rats. Although inflammatory pulmonary lesions occurred in all test groups, the lesions were more severe and occurred more frequently in the chloroform-treated rats.

The incidence of hepatocellular carcinomas in mice was significantly elevated in all treatment groups ($p < 0.001$, Fisher exact test). The NCI reported that in their experience the spontaneous incidence of hepatocellular carcinomas in B6C3F₁ mice is about 5-10% in males and 1% in females. The NCI concluded that chloroform treatment was associated with increased incidences of hepatocellular carcinomas in male and female mice and renal epithelial tumors in male rats.

In addition, Reuber (1979), based on his examination of the histological sections from the NCI study, concluded that chloroform treatment was also associated with cancer of the

liver in rats and an increased incidence of malignant lymphomas in mice. However, the NCI did not agree with his findings.

The carcinogenicity of chloroform given in drinking water was evaluated in male Osborne-Mendel rats and female B6C3F₁ mice (Jorgenson *et al.*, 1985). The chloroform used (technical grade), was found to contain 100 ppb diethylcarbonate, and trace amounts of 1,1-dichloroethane, dichloroethylene, carbon tetrachloride, and an unidentified C₅H₁₀ hydrocarbon. Time-weighted average doses of chloroform calculated based on water consumption rates and body weight, ranged up to 160 and 263 mg/kg-day for rats and mice, respectively. Two control groups were used, an untreated control, and a control group of animals with restricted access to water.

Jorgenson *et al.* observed a dose-related significant increase in renal tubular cell adenomas and adenocarcinomas in male rats, but found no treatment-related increases in tumor incidence in the female mice (Table 1). The lack of liver tumors in female B6C3F₁ mice is in sharp contrast to the results of the NCI study. A major difference between the NCI study and the Jorgenson study is the mode of administration. Administration of chloroform to rats in a corn oil vehicle slowed the gastrointestinal absorption of chloroform relative to the absorption rate observed after administration as a bolus in water (Withey *et al.*, 1983). In the Jorgenson *et al.* study, the rats received small doses of chloroform each time they drank water. The corn oil vehicle effect (Withey *et al.*, 1983) may have diminished the differences in absorption kinetics expected with the two different methods of administration. Therefore, any differences in peak blood concentrations between the NCI study and the Jorgenson study may not have been sufficient to account for the difference in liver tumor incidence. Physiologic or metabolic changes produced by corn oil consumption might interact with chemical carcinogens altering the production of liver tumors (Bull *et al.*, 1986; Newberne *et al.*, 1979).

A series of experiments was conducted by the Huntingdon Research Center to determine the effects of chronic ingestion of chloroform in a toothpaste base in mice, rats, and beagle dogs. In the first set of experiments (Roe *et al.*, 1979), doses of 17 and 60 mg chloroform/kg were administered by gavage in toothpaste to male and female ICI mice, 6 days/week for 80 weeks followed by a 16 week observation period (Experiment I). Controls (N=104) were treated with 1 ml chloroform-free toothpaste/kg-day. Aside from increased nonneoplastic liver lesions (moderate fatty degeneration), the only significant difference in pathology reported was an increase in the incidence of kidney tumors in high dose male mice, three were hypernephromas (tubular adenocarcinoma) and the remainder were adenomas (tumor incidences listed in Table 1). The incidence of renal tumors in high-dose male ICI mice was significantly greater than control mice ($p = 0.00012$, Fisher exact test). None of the female ICI mice examined developed renal tumors (Roe *et al.*, 1979). Roe *et al.* (1979) also investigated other components of the toothpaste base for carcinogenicity using male ICI mice. No lesion in this part of the study could be correlated with treatment.

In a third mouse experiment (Experiment III), Roe *et al.* (1979) compared the effects of toothpaste containing 3.5% chloroform on male mice of four different strains (C57BL,

CBA, CF/1, and ICI). Treatment with chloroform was not associated with any increase in liver or lung neoplasms relative to vehicle-treated controls in any of the four strains tested but was associated with significantly higher incidences of moderate to severe kidney pathology in CBA and CF/1 mice relative to the controls ($p < 0.0001$, chi-square test).

Palmer *et al.* (1979) gave groups of 50 Sprague-Dawley rats (both sexes) 0 or 60 mg chloroform/kg-day, 6 days/week by gavage in a toothpaste base for 80 weeks, followed by a 15 week observation period. There were no differences in the incidences of tumors of any site examined, including brain, lung, liver, kidney, and mammary gland, between treated and control animals. Heywood *et al.* (1979) investigated the carcinogenicity of chloroform in a toothpaste base in beagle dogs. Groups of male and female dogs received toothpaste base with 0, 15 or 30 mg chloroform/kg-day, 6 days/week for 7.5 years (8-16 dogs/sex), followed by a 20-24 week recovery period. Treatment with chloroform at the high dose was associated with significant elevations in SGPT levels but no treatment-related tumors were observed.

Chloroform treatment of rats via drinking water was associated with hepatic neoplastic nodules and hepatic adenofibrosis (Tumasonis *et al.*, 1985). Chloroform was administered to male and female Wistar rats in the drinking water at about 220 mg/kg/day and 160 mg/kg/day for the female and male rats, respectively. The incidence of hepatic neoplastic nodules was significantly elevated in treated females compared to controls ($p < 0.03$, Fisher exact test). In males, the incidence of hepatic neoplastic nodules did not differ in control and chloroform-treated groups. Increased incidences of hepatic adenofibrosis were observed in chloroform-treated males and females relative to controls. In contrast to the NCI and the Jorgenson *et al.* studies, renal tumors were not associated with chloroform treatment. However, Tumasonis *et al.* indicated that kidneys were only examined when grossly observable lesions were evident. Hence, kidney tumors may have been missed by this protocol. Tumasonis *et al.* concluded that chloroform is a hepatocarcinogen in Wistar rats.

IV. DERIVATION OF CANCER POTENCY

Basis for Cancer Potency

Chloroform is carcinogenic to rats and mice (NCI, 1976; Roe *et al.*, 1979; Jorgenson *et al.*, 1985). The International Agency for Research on Cancer (IARC) has classified chloroform as a possible human carcinogen (Group 2B). Similarly, the U.S. EPA has placed chloroform in Group B2 in their classification scheme, based on sufficient evidence of carcinogenicity in animals, but inadequate epidemiologic evidence. Current evidence and understanding of the carcinogenic process is insufficient to classify chloroform as either a genotoxic or epigenetic carcinogen, and it is possible that both types of effects are involved.

Table 1: Chloroform carcinogenicity bioassay tumor incidence data used to estimate cancer potency (CDHS, 1990)

Study	Strain/Species	Sex	Tumor Site	Lifetime Daily Dose (mg/kg-day)	Tumor Incidence
NCI (1976)	B6C3F ₁ mouse	M	hepatocellular carcinoma	control	1/18
				83	18/50
				167	44/45
	B6C3F ₁ mouse	F	hepatocellular carcinoma	control	0/20
				143	36/45
				287	39/41
	Osborne-Mendel rat	M	renal tubular adenoma or adenocarcinoma	control	0/19
				45	4/38
				90	12/27
Jorgenson <i>et al.</i> (1985)	Osborne-Mendel rat	M	renal tubular adenoma or adenocarcinoma	control	4/301
				18	4/313
				38	4/148
				79	3/48
Roe <i>et al.</i> (1979)	ICI mouse (Experiment I)	M	renal tubular adenoma or adenocarcinoma	control	0/72
				12	0/37
				43	8/37
	ICI mouse (Experiment II)	M	renal tubular adenoma or adenocarcinoma	control	6/237
				40	9/49
	ICI mouse (Experiment III) ^a	M	renal tubular adenoma or adenocarcinoma	control	1/49
				42	5/47
	ICI mouse (Experiment III) ^b	M	renal tubular adenoma or adenocarcinoma	control	1/50
42				12/48	
Tumasonis <i>et al.</i> (1985)	Wistar rat	F	cholangiocarcinoma	control	0/18
				220	34/40
	Wistar rat	M	cholangiocarcinoma	control	0/22
				160	17/28
Reuber <i>et al.</i> (1979) using NCI (1976)	Osborne-Mendel rat	F	cholangiocarcinoma and cholangiofibroma	control	0/20
				50	3/39
				100	11/39

^a toothpaste base was used as the vehicle; ^b arachis oil was used as the vehicle

The estimation of cancer risk to humans from exposure to chloroform by CDHS (1990) is based on animal studies. Data were chosen based primarily on statistical significance, as discussed below.

Methodology

The following data sets were evaluated to estimate chloroform cancer potency: 1) Liver tumor data in male and female B6C3F₁ mice, and renal tubular cell tumors in male Osborne-Mendel rats from the NCI (1976) study were chosen because statistically significant increases in these tumor types were observed in chloroform treated animals relative to controls; 2) Renal tubular cell tumor data in male Osborne-Mendel rats from the Jorgenson *et al.* (1985) study and in male ICI mice in the Roe *et al.* (1979) study were used for risk estimation based on a statistically significant increase in kidney tumors in

chloroform treated animals relative to controls; 3) Liver cholangiocarcinoma ("adenofibrosis") data in female rats from Tumasonis *et al.* (1985), and from Reuber's reanalysis of the NCI (1976) slides (Reuber, 1979) were also analyzed with the linearized multistage model (GLOBAL86). Administered doses were transformed to lifetime doses by adjusting for the number of days exposed per week and the ratio of the length of exposure to the length of the experiment (exposure plus observation period).

Calculated q_1^* values from the above studies ranged from 8.1×10^{-4} to 1.9×10^{-2} (mg/kg-day)⁻¹. These represent cancer potency estimates for rats and mice and must be converted to theoretical equivalent potency values for humans. This conversion is based on equivalency of dose per unit surface area according to Anderson *et al.* (1983). These "human" cancer potencies range from 4.2×10^{-3} to 2.6×10^{-1} (mg/kg-day)⁻¹. Scaling factors ranged from 5.19 to 13.57.

The NCI (1976) and Jorgenson *et al.* (1985) studies were the most thorough studies in terms of the number of doses tested, sample size, histological examination of the animals, and other procedural and statistical methods presented. As such, CDHS placed more confidence in the potency slopes from these studies than in the other studies. The potency slopes derived from Roe *et al.* (1979) and Tumasonis *et al.* (1985) fall within the range of those from the NCI and Jorgenson studies.

Bogen *et al.* (1989) used a physiologically based pharmacokinetic (PBPK) model to estimate metabolized dose for chloroform to use in the analysis of cancer potency with the linearized multistage model to carcinogenicity bioassay data from NCI (1976), Jorgenson *et al.* (1985), Roe *et al.* (1979), and Tumasonis *et al.* (1985). In the application of the model, the liver was considered to metabolize chloroform through a saturable enzyme system following Michaelis-Menten kinetics. This approach is consistent with the evidence that chloroform metabolites are responsible for toxicity and probably for the carcinogenicity of chloroform. The potency estimates made from these studies ranged from 4.8×10^{-3} to 5.0×10^{-1} (mg/kg-day)⁻¹. These corresponded to unit risks of 4.5×10^{-6} to 4.7×10^{-4} (ppb)⁻¹. These potency estimates are incorporated into DHS staff's best estimate of cancer potency for chloroform.

There are no studies on the carcinogenicity of chloroform by the inhalation route. Therefore, estimation of the cancer risk from exposure to chloroform in the ambient air required extrapolation from the oral route. In so doing, it is assumed that chloroform is also carcinogenic by the inhalation route, and that the risk posed by an absorbed inhaled dose of chloroform is equivalent to that posed by the same dose absorbed after oral administration. In the final risk range, the DHS staff included tumor sites that did not appear to be vehicle-dependent. Therefore, the liver tumors were not included in the range of risks or the best estimate of risk, due to the possible potentiation of liver tumors by the corn oil vehicle.

The best estimate of unit risk was considered by CDHS (1990) to be the arithmetic average of unit risks generated by CDHS (1990) and Bogen *et al.* (1989) for rat renal tumors in Jorgenson *et al.* (1985) and NCI (1976) and of the geometric mean for

supporting data sets (Roe *et al.*, 1979; Tumasonis *et al.*, 1985). This unit risk, $5.3 \text{ E-6 } (\mu\text{g}/\text{m}^3)^{-1}$, represents the best estimate using a nonthreshold linear procedure and using most of the data on the carcinogenicity of chloroform. It included analysis by PBPK modeling of metabolized dose, as well as analysis of potency based on applied dose.

V. REFERENCES

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ACUTE TOXICITY SUMMARY

CHLOROFORM

(trichloromethane, formyl trichloride, methenyl trichloride, methyl trichloride)

CAS Registry Number: 67-66-3

I. Acute Toxicity Summary (for a 7-hour exposure)

<i>Inhalation reference exposure level</i>	150 µg/m³
<i>Critical effect(s)</i>	histological changes in the nasal epithelium
<i>Hazard Index target(s)</i>	Respiratory System; Nervous System; Reproductive/developmental

II. Physical and Chemical Properties (HSDB, 1993 except as noted)

<i>Description</i>	colorless liquid
<i>Molecular formula</i>	CHCl ₃
<i>Molecular weight</i>	119.49
<i>Density</i>	1.483 g/cm ³ @ 20°C
<i>Boiling point</i>	61°C
<i>Melting point</i>	-63.5°C
<i>Vapor pressure</i>	200 mm Hg @ 25°C
<i>Flashpoint</i>	not applicable; non-flammable liquid, vapor will burn at high temperatures
<i>Explosive limits</i>	not applicable
<i>Solubility</i>	soluble in water, carbon tetrachloride, carbon disulfide, alcohols, benzene, ethers, oils
<i>Odor threshold</i>	192 ppm (geometric mean) (AIHA, 1989)
<i>Odor description</i>	sweet, suffocating (AIHA, 1989)
<i>Metabolites</i>	carbon dioxide, phosgene
<i>Conversion factor</i>	1 ppm = 4.88 mg/m ³ @ 25°C

III. Major Uses or Sources

Chloroform (CHCl₃) is used in industry and laboratory settings as a solvent for adhesives, pesticides, fats, oils, and rubbers. It is also used as a chemical intermediate for fluorocarbon 22, dyes, pesticides, and tribromomethane. It is produced as a byproduct of water and sewage chlorination. Chloroform is also produced in large quantities as a byproduct of wood pulp chlorination in the production of paper products.

IV. Acute Toxicity to Humans

In humans, pulmonary excretion was found to be the major means of elimination following a single oral dose of 0.5 or 1.0 g CHCl₃ (Fry *et al.*, 1972). Up to 68% of the unchanged CHCl₃ and up to 50.6% of the metabolite carbon dioxide were found in the expired air within eight hours of administration. Chloroform in the urine accounted for less than 1% of the oral dose.

Signs of acute CHCl₃ toxicity include fainting, vomiting, dizziness, salivation, fatigue, headache, respiratory depression, and coma (IRIS, 1993). Few reports were found in the literature on the acute toxicity of CHCl₃ to humans in chamber studies. However, a number of case reports exist stemming from its use as an anesthetic.

Cardiac arrhythmia, bradycardia, and cardiac arrest resulting in death have been reported following the use of CHCl₃ as an anesthetic in concentrations of approximately 8,000 to 22,500 ppm (39,000 to 110,000 mg/m³) (Payne, 1981). Severe liver and kidney damage were noted in an adult male following fatal suicidal ingestion of approximately 6 ounces of CHCl₃ (Piersol *et al.*, 1933).

The incidence of liver enlargement and jaundice was increased in workers exposed to 2-204 ppm (10-995 mg/m³) CHCl₃ for at least one year (Bomski *et al.*, 1967). Jaundice was reported in 31 workers occupationally exposed to 14-400 ppm (68-1,952 mg/m³) CHCl₃ for 6 months or less (Phoon *et al.*, 1983).

Predisposing Conditions for Chloroform Toxicity

Medical: Persons with skin, eye, respiratory, liver, kidney or neurological conditions may be more sensitive to the effects of chloroform (Reprotext, 1999).

Chemical: Epinephrine (e.g., in bronchodilators) may potentiate the cardiac effects of chloroform exposure (Reprotext, 1999). Concurrent exposure to barbiturates has been shown to increase chloroform toxicity by induction of liver cytochrome P-450 activity (Cornish *et al.*, 1973). The potentiation of chloroform-induced hepatotoxicity and nephrotoxicity by various alcohols and ketones is well documented (Cowlen *et al.*, 1984; Iijima *et al.*, 1983; Brown and Hewitt, 1984.)

V. Acute Toxicity to Laboratory Animals

Beagle dogs exposed to 14,500 ppm (70,800 mg/m³) CHCl₃ survived an average of 202 minutes (Von Oettingen *et al.*, 1949). The oral LD₅₀ in male and female adult Sprague-Dawley rats is reported as 908 mg CHCl₃/kg and 1,117 mg CHCl₃/kg, respectively (Chu *et al.*, 1980).

Hepatocellular necrosis was observed in adult female mice following a single 4-hour exposure to 200 ppm (976 mg/m³) CHCl₃ (Kylin *et al.*, 1963). Hepatic fatty infiltration was noted following a single 4-hour exposure to 100 ppm (488 mg/m³) CHCl₃. Some studies report that chloroform renal toxicity is gender-dependent, while hepatotoxicity is similar in both sexes (Smith *et al.*, 1983 and 1984; Hill *et al.*, 1975; Pohl *et al.*, 1984; Taylor *et al.*, 1974).

Cytochrome P-450-mediated metabolism of CHCl_3 in the liver and kidneys has been demonstrated to produce phosgene in rats (Pohl *et al.*, 1979). Hepatotoxicity following chloroform exposure is thought to be due largely to phosgene and other reactive CHCl_3 metabolites. Metabolism of CHCl_3 to phosgene is also responsible for the nephrotoxicity of CHCl_3 (Bailie *et al.*, 1984).

Male rats were exposed to 1, 3, 10, 30, 100, or 300 ppm CHCl_3 6 hours per day for 7 days (Mery *et al.*, 1994). Statistically significant, concentration-dependent, bony proliferation was observed in the ethmoid turbinates of rats exposed to 10 ppm CHCl_3 or greater. Cellular hypertrophy and proliferation in the nasal pharyngeal and olfactory mucosal regions were also increased in a concentration dependent manner in rats exposed to 10 ppm CHCl_3 or greater. No adverse effects were observed following exposure to 3 ppm (15 mg/m³) CHCl_3 .

VI. Reproductive or Developmental Toxicity

Pregnant rats were exposed to 30, 100, or 300 ppm (150, 500, or 1,500 mg/m³) CHCl_3 for 7 hours per day on days 6-15 of gestation (Schwetz *et al.*, 1974). A significant increase in the number of fetal resorptions and a decrease in fetal body weights and crown-rump lengths were observed in those animals exposed to 300 ppm CHCl_3 . Following maternal exposure to 100 ppm CHCl_3 , fetuses exhibited a significant increase in malformations including acaudia, imperforate anus, missing ribs and delayed sternal ossification. An increase in the incidence of wavy ribs and delayed skull ossification, as well as reduced fetal crown-rump length, were observed following maternal exposure to 30 ppm CHCl_3 . Maternal toxicity was observed in all three exposure groups.

The incidence of abnormal sperm was significantly increased in male mice exposed to 400 ppm (1,952 mg/m³) CHCl_3 for 4 hours/day for 5 days (Land *et al.*, 1981).

Chloroform has not been listed as a developmental or reproductive toxicant under Proposition 65.

VII. Derivation of Acute Reference Exposure Level and Other Severity Levels

Reference Exposure Level (level protective against severe adverse effects; estimated for 7 hour exposure): **0.03 ppm (150 µg/m³)**

<i>Study</i>	Schwetz et al (1974)
<i>Study population</i>	pregnant rats
<i>Exposure method</i>	inhalation exposures to 30, 100, 300 ppm for 7 h/d, days 6-15 of gestation
<i>Critical Effect</i>	fetotoxicity
<i>LOAEL</i>	30 ppm
<i>NOAEL</i>	not determined
<i>Exposure duration</i>	7 hours/day
<i>LOAEL uncertainty factor</i>	10
<i>Interspecies uncertainty factor</i>	10
<i>Intraspecies uncertainty factor</i>	10
<i>Cumulative uncertainty factor</i>	1000
<i>Reference Exposure Level (7 h)</i>	0.03 ppm (0.15 mg/m ³ ; 150 µg/m ³)

The study by Schwetz *et al.* (1974) is the only published developmental toxicity study of chloroform. Exposure of pregnant rats to 30 ppm (150 mg/m³) CHCl₃ for 7 hours per day on days 6-15 of gestation resulted in fetotoxicity as indicated by decreased crown-rump length and increased incidences of wavy ribs and skeletal ossification defects. Maternal toxicity was also observed. An abstract by Dilley *et al.* (1977) indicates an absence of teratological effects in rats exposed to 20,000 mg/m³ CHCl₃ on days 7-14 of gestation. The data from this study were not available for review, therefore, the Schwetz *et al.* study is used in developing the severe adverse effect level for chloroform. A NOAEL was estimated from the reported LOAEL using an uncertainty factor of 10. An additional uncertainty factor of 100 was applied to account for inter- and intraspecies differences. The level protective against severe adverse effects for a 7 hour exposure is estimated as 0.03 ppm (0.15 mg/m³).

Level Protective Against Life-threatening Effects

No recommendation is made due to the limitations of the database. NIOSH (1995) lists a (revised) IDLH of 500 ppm based on acute inhalation toxicity in humans but the selection of the level is somewhat arbitrary and the IDLH does not make allowance for sensitive individuals.

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ACRYLAMIDE

CAS No: 79-06-1

I. PHYSICAL AND CHEMICAL PROPERTIES (From HSDB, 1994)

Molecular weight	71.08
Boiling point	125°C at 25 mm Hg
Melting point	84.5
Vapor pressure	0.007 mm Hg at 25°C
Air concentration conversion	1 ppm = 2.91 mg/m ³

II. HEALTH ASSESSMENT VALUES

Unit Risk Factor: 1.3 E-3 (µg/m³)⁻¹

Slope Factor: 4.5 E+0 (mg/kg-day)⁻¹

[Calculated by US EPA/IRIS (1988, 1993) from female Fischer 344 rat tumor data (central nervous system, mammary and thyroid glands, uterus, oral cavity) (Johnson *et al.*, 1986) using a linearized multistage procedure, extra risk; adopted by CDHS/RCHAS (1990).]

III. CARCINOGENIC EFFECTS

Human Studies

US EPA (1993) reviewed a study of cancer mortality in workers exposed to acrylamide by Collins (1984). Data from a long duration exposure group (10 individuals) and a short duration/intermittent exposure group (52 individuals) was analyzed using a standardized proportional mortality ratio (SPMR) procedure. No excess mortality for all types of cancer combined was noted in either group. Mortality from lung and central nervous system cancer appeared to be slightly elevated. However, the SPMRs were not significantly different from expected values, due to small group size. US EPA (1993) also noted additional study limitations including underrepresentation of the potential at-risk worker population, incomplete cause of death ascertainment, and incomplete exposure data.

Sobel *et al.* (1986) studied the mortality experience of 371 workers (365 white males, 6 white females) employed in acrylamide monomer production and polymerization operations at the Michigan Division of the Dow Chemical Company from 1955 through 1979. Vital status followup was performed from the date of the first potential exposure to December 31, 1982. Mortality comparisons were made between the cohort and United States white male mortality rates; comparisons were made with a subcohort of workers previously exposed to organic dyes both included and excluded. Slight excesses of mortality from all cancers (11 observed/7.9 expected), digestive tract cancer (4 observed/1.9 expected) and respiratory tract cancer (4 observed/2.9 expected) were observed in the total cohort; these excesses were not observed when the organic dye exposure subcohort was excluded. The authors concluded that the study did not support a relationship between acrylamide exposure and general or specific cancer mortality.

However, US EPA (1988) considers this study insufficient to assess the carcinogenicity of acrylamide in humans because of small cohort size, multiple chemical exposures, limited followup, and short exposure duration (167 cohort members had < 1 year of employment; 109 had 1-4 years of employment).

Animal Studies

Bull *et al.* (1984a) exposed female Sencar mice and male and female A/J mice to acrylamide. Female Sencar mice (40/treatment group) were exposed to 0, 12.5, 25.0 or 50.0 mg/kg body weight acrylamide by gavage, intraperitoneal injection or dermal application. Doses were administered 6 times over a 2 week period; total doses were 0, 75, 150 and 300 mg/kg. Acrylamide was dissolved in distilled water for gavage and intraperitoneal injection administration, and in ethanol for dermal application. Two weeks after the cessation of acrylamide exposure, 1.0 µg 12-*O*-tetradecanoyl-phenol-13-acetate (TPA) dissolved in 0.2 ml acetone was applied to the shaved back of each animal 3 times/week for 20 weeks. A promotion control group was included which received 300 mg/kg acrylamide followed by dermal applications of 0.2 ml acetone on the same treatment schedule and duration as the animals receiving TPA. All animals were sacrificed at 52 weeks, and were evaluated for the presence of skin tumors. Male and female A/J mouse (40/sex/treatment group) acrylamide exposures were conducted at laboratories of the US EPA (Cincinnati, OH) and the Medical College of Ohio (Toledo, OH) (MCO). Animals exposed at US EPA received acrylamide dissolved in distilled water by gavage 3 times/week for 8 weeks at doses of 0, 6.25, 12.5 or 25 mg/kg. Animals exposed at MCO initially received acrylamide by intraperitoneal injection 3 times/week for 8 weeks at doses of 0, 1, 3, 10, 30 or 60 mg/kg; however, peripheral neuropathy and decreased survival forced treatment termination on the 60 mg/kg group after the 11th injection. An untreated control group was also included. Animals were sacrificed after either 7 months (US EPA) or 6 months (MCO) and examined for lung adenomas. Acrylamide induced skin tumors (squamous cell papillomas and carcinomas) in TPA-promoted female Sencar mice in a dose-dependent manner when administered by gavage, intraperitoneal injection or dermal application. Acrylamide did not induce skin tumors by any route of administration in animals not receiving TPA. Tumor incidence data from female Sencar mice exposed to acrylamide are listed in Table 1.

The incidence of lung adenomas in both male and female A/J mice exposed to acrylamide by either gavage or intraperitoneal injection was significantly increased in a dose-related manner (Bull *et al.*, 1984a). Tumor incidence data for animals treated by intraperitoneal injection is listed in Table 2; numerical tumor incidence data for animals exposed to acrylamide by gavage was not listed.

Acrylamide dissolved in water was administered by gavage (0, 75, 150 or 200 mg/kg body weight, divided into 6 equal portions) to female ICR-Swiss mice (40 animals/treatment group) over a 2 week period (Bull *et al.*, 1984b). Two weeks after the last acrylamide exposure, the animals were exposed 3 times/week to dermal applications of 2.5 µg TPA for 20 weeks. Another group of 20 animals were exposed to a total dose of 300 mg/kg acrylamide, but received dermal applications of acetone alone. All animals were sacrificed after 52 weeks. Acrylamide caused a significant dose-related increase in the incidence of skin tumors (papillomas and

carcinomas combined). The incidence in animals also receiving TPA was 0/35, 4/34, 4/32 and 13/32 (number of animals with tumors/number of animals examined) for the control, low, mid and high dose groups, respectively; the skin tumor incidence in animals receiving 300 mg/kg acrylamide but not TPA was 10/36. Acrylamide-treated animals also demonstrated a significant dose-related increase in the incidence of lung tumors (alveolar and bronchiolar adenomas and carcinomas). The incidence in animals also receiving TPA was 4/36, 8/34, 6/36 and 11/34 for the control, low, mid and high dose groups, respectively; the lung tumor incidence in animals receiving 300 mg/kg acrylamide but not TPA was 14/36.

Table 1. Skin tumor (squamous cell papillomas and carcinomas) incidence in female Sencar mice exposed to acrylamide (Bull *et al.*, 1984a)

Total administered dose ¹ (mg/kg body weight)	Route of administration	TPA ²	Tumor incidence
0	gavage	+	2/40
75		+	12/40
100		+	23/40
300		+	30/40
300		-	0/20
0	intraperitoneal injection	+	0/40
75		+	10/40
100		+	13/40
300		+	21/40
300		-	0/20
0	dermal	+	7/40
75		+	4/40
100		+	11/40
300		+	18/40
300		-	0/20

1. The exposure duration was less than lifetime (2 weeks); the total administered dose listed was not adjusted to reflect a less-than-lifetime exposure.
2. TPA = 12-*O*-tetradecanoyl-phenol-13-acetate

Table 2. Lung adenoma incidence in male and female A/J mice exposed to acrylamide by intraperitoneal injection (Bull *et al.*, 1984a)

Dose level ¹ (mg/kg body weight)	Percent of animals with tumors	
	males	females
0	13	8
1	50	35
3	38	53
10	59	79
30	93	93

1. The exposure duration was less than lifetime (8 weeks); the dose level listed was not adjusted to reflect a less-than-lifetime exposure.

Robinson *et al.* (1986) exposed female SENCAR, BALB/c, A/J and ICR-Swiss mice (60 mice/strain/treatment group) to a single 50 mg/kg body weight dose of acrylamide by intraperitoneal injection; 2 days later 40 of the 60 mice in each treatment group received 1.0 µg (SENCAR), 2.5 µg (A/J and ICR-Swiss) or 5.0 µg (BALB/c) TPA in 0.2 ml acetone applied dermally 3 times/week for 20 weeks. The remaining 20 mice/strain/treatment group received acetone alone for the same treatment schedule and duration. All animals were sacrificed at 40 weeks, and were only examined for the number of skin papillomas and lung adenomas/animal. Acrylamide induced a significant increase in the number of skin papillomas and lung adenomas per animal in SENCAR mice receiving TPA treatment. The total number of animals bearing tumors was not listed. No significant increase in either tumor type was noted in the other mouse strains tested; tumor data for the animals receiving acrylamide but not TPA was not reported.

Male and female Fischer 344 rats (90/sex/treatment group) were exposed to acrylamide in drinking water for 2 years (Johnson *et al.*, 1986). Acrylamide water concentrations were adjusted to provide dosages of 0, 0.01, 0.1, 0.5 or 2 mg/kg body weight/day. Interim sacrifices (10 animals/sex/treatment group) were performed at 6, 12 and 18 months. A maximum tolerated dose (MTD) was achieved based on decreased weight gain, increased mortality during the last 4 months of the study and the appearance of several toxic effects (including peripheral nerve degeneration) in the 2 mg/kg/day group. Increases in the incidences of a number of tumor types were observed in the 2.0 mg/kg/day exposure group animals. An increased incidence of thyroid gland-follicular epithelium tumors was observed in both males and females. In females, increased tumor incidences were noted in the mammary glands, central nervous system, oral tissues, uterus and clitoral gland. An increased incidence of scrotal mesothelioma was noted in males, in both the 2.0 and 0.5 mg/kg/day exposure group; additionally, although not statistically significant, the incidence of scrotal mesothelioma in the 0.1 mg/kg/day group was greater than either the control group or historical control incidences. Male rats in the 2.0 mg/kg/day exposure group also had a significant increase in adrenal pheochromocytomas, and an increased incidence of central nervous system tumors when compared to historical controls but not when compared to concurrent controls. Tumor incidence data is listed in Table 3.

Table 3. Acrylamide-induced tumor incidences in male and female Fischer 344 rats (Johnson *et al.*, 1986)

Administered dose (mg/kg/day)	Human equivalent dose ¹ (mg/kg/day)	Tumor type	Tumor incidence	
			males	females
0	0	combined central nervous system (CNS), mammary gland, oral cavity, thyroid gland, uterus ²	NA	13/60
0.01	0.001		NA	18/60
0.1	0.015		NA	14/60
0.5	0.076		NA	21/60
2.0	0.305		NA	46/60
0	0		adrenal pheochromacytomas ³	3/60
0.01	0.001	7/60		NA
0.1	0.015	7/60		NA
0.5	0.076	5/60		NA
2.0	0.305	10/60		NA
0	0	central nervous system ⁴		5/60
0.01	0.001		2/60	2/60
0.1	0.015		0/60	1/60
0.5	0.076		3/60	1/60
2.0	0.305		8/60	9/60
0	0		oral cavity ⁵	6/60
0.01	0.001	7/60		3/60
0.1	0.015	1/60		2/60
0.5	0.076	5/60		3/60
2.0	0.305	6/60		8/60
0	0	mammary gland ⁶		NA
0.01	0.001		NA	2/60
0.1	0.015		NA	1/60
0.5	0.076		NA	5/58
2.0	0.305		NA	8/61
0	0		scrotal mesothelioma	3/60
0.01	0.001	0/60		NA
0.1	0.015	7/60		NA
0.5	0.076	11/60		NA
2.0	0.305	10/60		NA
0	0	thyroid ⁷		1/60
0.01	0.001		0/58	0/59
0.1	0.015		2/59	1/59
0.5	0.076		1/59	1/58
2.0	0.305		7/59	5/60
0	0		uterine adenocarcinomas	NA
0.01	0.001	NA		2/60
0.1	0.015	NA		1/60
0.5	0.076	NA		0/59
2.0	0.305	NA		5/60

Table 3 (continued). Acrylamide-induced tumor incidences in male and female Fischer 344 rats (Johnson *et al.*, 1986)

- 1, 2. As calculated by US EPA (1988).
 3. Benign and malignant.
 4. Tumors of glial origin or glial proliferation suggestive of early tumor.
 5. Squamous cell papillomas and carcinomas.
 6. Adenomas and adenocarcinomas.
 7. Males: follicular adenomas; females: follicular adenomas and adenocarcinomas.
- NA not available

IV. DERIVATION OF CANCER POTENCY

Basis for Cancer Potency

The studies by Bull *et al.* (1984a, 1984b), Robinson *et al.* (1986) and Johnson *et al.* (1986) indicate that acrylamide is capable of acting as both an initiator and a complete carcinogen in animals. However, only the Johnson *et al.* (1986) study contained a data set suitable for generating a cancer potency factor. Female Sencar mice developing tumors after exposure to acrylamide in the study by Bull *et al.* (1984a) were also additionally exposed to TPA; animals not exposed to TPA did not develop skin tumors. Female A/J mice exposed in that study to acrylamide by either gavage or intraperitoneal injection developed an increased incidence of lung adenomas without requiring TPA exposure. However, the animals were not evaluated for tumor types other than lung adenomas, and numerical tumor incidence data for animals exposed to acrylamide by gavage was not listed. Also, the exposure and observation durations for animals exposed by gavage (8 weeks and 7 months, respectively) and by intraperitoneal injection (8 weeks and 6 months, respectively) were short. Female ICR-Swiss mice exposed to acrylamide by gavage in the study by Bull *et al.* (1984b) were generally also exposed to TPA; only one exposure group was included which received acrylamide (300 mg/kg) but not TPA. Additionally, the exposure duration was only 2 weeks and the exposure duration was less than lifetime (52 weeks). In the study by Robinson *et al.* (1986), all animals for which tumor incidence data was reported were exposed to TPA as well as acrylamide. Animals in the Johnson *et al.* (1986) study were exposed to acrylamide alone for the lifetime of the animals, and were comprehensively examined for tumors. For these reasons, tumor incidence data from the Johnson *et al.* (1986) study was used to derive a cancer potency factor for acrylamide.

Methodology

As recommended in the US EPA Guidelines for Carcinogen Risk Assessment (1986), US EPA (1988) pooled tumor incidence data from different tumor sites, under the consideration that risk numbers derived from site-specific tumor incidence data potentially may not be predictive of, and may in fact underestimate, “whole-body” risks that are determined using the pooled individual animal data. The dose-response curves for each sex based on the pooled tumor incidence (benign and malignant) constituted the data sets of choice for risk assessment. Tumors at a particular site were added into the pool only when the tumor site had statistically significantly increased incidence at least at the high dose level (treated vs. control). The female

rat was considered to be the more sensitive sex, as there were significantly increased tumor incidences at a greater number of sites than in the males; the female rat tumor data was therefore used as the basis of a risk estimate. A linearized multistage procedure (GLOBAL 83) was used to calculate a cancer potency factor (q_1^*) from the female rat tumor incidence data. Surface area scaling was employed to transform animal cancer potency factors to human cancer potency factors, using the relationship ($q_{\text{human}} = q_{\text{animal}} * (bw_h / bw_a)^{1/3}$), where q_{human} is the human potency, q_{animal} is the animal potency, and bw_h and bw_a are the human and animal body weights, respectively. Body weight values used for humans and rats were 70 kg and 0.2 kg, respectively. No exposure route adjustment was made to the risk estimates because data exists which indicates that the pharmacokinetics and tissue distribution of acrylamide were not significantly affected by the dose administered or the route of administration (Dearfield *et al.*, 1988). US EPA calculated a cancer potency value (q_{human}) of $4.5 \text{ E}+0 (\text{mg/kg-day})^{-1}$. A unit risk factor was then calculated from the cancer potency factor by OEHHA/ATES using a reference human body weight of 70 kg and an inspiration rate of $20 \text{ m}^3/\text{day}$. The unit risk should not be used if the air concentration exceeds $8 \mu\text{g}/\text{m}^3$, as above this concentration the unit risk may not be appropriate.

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ACUTE TOXICITY SUMMARY

CHLOROFORM

(trichloromethane, formyl trichloride, methenyl trichloride, methyl trichloride)

CAS Registry Number: 67-66-3

I. Acute Toxicity Summary (for a 7-hour exposure)

<i>Inhalation reference exposure level</i>	150 µg/m³
<i>Critical effect(s)</i>	histological changes in the nasal epithelium
<i>Hazard Index target(s)</i>	Respiratory System; Nervous System; Reproductive/developmental

II. Physical and Chemical Properties (HSDB, 1993 except as noted)

<i>Description</i>	colorless liquid
<i>Molecular formula</i>	CHCl ₃
<i>Molecular weight</i>	119.49
<i>Density</i>	1.483 g/cm ³ @ 20°C
<i>Boiling point</i>	61°C
<i>Melting point</i>	-63.5°C
<i>Vapor pressure</i>	200 mm Hg @ 25°C
<i>Flashpoint</i>	not applicable; non-flammable liquid, vapor will burn at high temperatures
<i>Explosive limits</i>	not applicable
<i>Solubility</i>	soluble in water, carbon tetrachloride, carbon disulfide, alcohols, benzene, ethers, oils
<i>Odor threshold</i>	192 ppm (geometric mean) (AIHA, 1989)
<i>Odor description</i>	sweet, suffocating (AIHA, 1989)
<i>Metabolites</i>	carbon dioxide, phosgene
<i>Conversion factor</i>	1 ppm = 4.88 mg/m ³ @ 25°C

III. Major Uses or Sources

Chloroform (CHCl₃) is used in industry and laboratory settings as a solvent for adhesives, pesticides, fats, oils, and rubbers. It is also used as a chemical intermediate for fluorocarbon 22, dyes, pesticides, and tribromomethane. It is produced as a byproduct of water and sewage chlorination. Chloroform is also produced in large quantities as a byproduct of wood pulp chlorination in the production of paper products.

IV. Acute Toxicity to Humans

In humans, pulmonary excretion was found to be the major means of elimination following a single oral dose of 0.5 or 1.0 g CHCl₃ (Fry *et al.*, 1972). Up to 68% of the unchanged CHCl₃ and up to 50.6% of the metabolite carbon dioxide were found in the expired air within eight hours of administration. Chloroform in the urine accounted for less than 1% of the oral dose.

Signs of acute CHCl₃ toxicity include fainting, vomiting, dizziness, salivation, fatigue, headache, respiratory depression, and coma (IRIS, 1993). Few reports were found in the literature on the acute toxicity of CHCl₃ to humans in chamber studies. However, a number of case reports exist stemming from its use as an anesthetic.

Cardiac arrhythmia, bradycardia, and cardiac arrest resulting in death have been reported following the use of CHCl₃ as an anesthetic in concentrations of approximately 8,000 to 22,500 ppm (39,000 to 110,000 mg/m³) (Payne, 1981). Severe liver and kidney damage were noted in an adult male following fatal suicidal ingestion of approximately 6 ounces of CHCl₃ (Piersol *et al.*, 1933).

The incidence of liver enlargement and jaundice was increased in workers exposed to 2-204 ppm (10-995 mg/m³) CHCl₃ for at least one year (Bomski *et al.*, 1967). Jaundice was reported in 31 workers occupationally exposed to 14-400 ppm (68-1,952 mg/m³) CHCl₃ for 6 months or less (Phoon *et al.*, 1983).

Predisposing Conditions for Chloroform Toxicity

Medical: Persons with skin, eye, respiratory, liver, kidney or neurological conditions may be more sensitive to the effects of chloroform (Reprotext, 1999).

Chemical: Epinephrine (e.g., in bronchodilators) may potentiate the cardiac effects of chloroform exposure (Reprotext, 1999). Concurrent exposure to barbiturates has been shown to increase chloroform toxicity by induction of liver cytochrome P-450 activity (Cornish *et al.*, 1973). The potentiation of chloroform-induced hepatotoxicity and nephrotoxicity by various alcohols and ketones is well documented (Cowlen *et al.*, 1984; Iijima *et al.*, 1983; Brown and Hewitt, 1984.)

V. Acute Toxicity to Laboratory Animals

Beagle dogs exposed to 14,500 ppm (70,800 mg/m³) CHCl₃ survived an average of 202 minutes (Von Oettingen *et al.*, 1949). The oral LD₅₀ in male and female adult Sprague-Dawley rats is reported as 908 mg CHCl₃/kg and 1,117 mg CHCl₃/kg, respectively (Chu *et al.*, 1980).

Hepatocellular necrosis was observed in adult female mice following a single 4-hour exposure to 200 ppm (976 mg/m³) CHCl₃ (Kylin *et al.*, 1963). Hepatic fatty infiltration was noted following a single 4-hour exposure to 100 ppm (488 mg/m³) CHCl₃. Some studies report that chloroform renal toxicity is gender-dependent, while hepatotoxicity is similar in both sexes (Smith *et al.*, 1983 and 1984; Hill *et al.*, 1975; Pohl *et al.*, 1984; Taylor *et al.*, 1974).

Cytochrome P-450-mediated metabolism of CHCl_3 in the liver and kidneys has been demonstrated to produce phosgene in rats (Pohl *et al.*, 1979). Hepatotoxicity following chloroform exposure is thought to be due largely to phosgene and other reactive CHCl_3 metabolites. Metabolism of CHCl_3 to phosgene is also responsible for the nephrotoxicity of CHCl_3 (Bailie *et al.*, 1984).

Male rats were exposed to 1, 3, 10, 30, 100, or 300 ppm CHCl_3 6 hours per day for 7 days (Mery *et al.*, 1994). Statistically significant, concentration-dependent, bony proliferation was observed in the ethmoid turbinates of rats exposed to 10 ppm CHCl_3 or greater. Cellular hypertrophy and proliferation in the nasal pharyngeal and olfactory mucosal regions were also increased in a concentration dependent manner in rats exposed to 10 ppm CHCl_3 or greater. No adverse effects were observed following exposure to 3 ppm (15 mg/m³) CHCl_3 .

VI. Reproductive or Developmental Toxicity

Pregnant rats were exposed to 30, 100, or 300 ppm (150, 500, or 1,500 mg/m³) CHCl_3 for 7 hours per day on days 6-15 of gestation (Schwetz *et al.*, 1974). A significant increase in the number of fetal resorptions and a decrease in fetal body weights and crown-rump lengths were observed in those animals exposed to 300 ppm CHCl_3 . Following maternal exposure to 100 ppm CHCl_3 , fetuses exhibited a significant increase in malformations including acaudia, imperforate anus, missing ribs and delayed sternal ossification. An increase in the incidence of wavy ribs and delayed skull ossification, as well as reduced fetal crown-rump length, were observed following maternal exposure to 30 ppm CHCl_3 . Maternal toxicity was observed in all three exposure groups.

The incidence of abnormal sperm was significantly increased in male mice exposed to 400 ppm (1,952 mg/m³) CHCl_3 for 4 hours/day for 5 days (Land *et al.*, 1981).

Chloroform has not been listed as a developmental or reproductive toxicant under Proposition 65.

VII. Derivation of Acute Reference Exposure Level and Other Severity Levels

Reference Exposure Level (level protective against severe adverse effects; estimated for 7 hour exposure): **0.03 ppm (150 µg/m³)**

<i>Study</i>	Schwetz et al (1974)
<i>Study population</i>	pregnant rats
<i>Exposure method</i>	inhalation exposures to 30, 100, 300 ppm for 7 h/d, days 6-15 of gestation
<i>Critical Effect</i>	fetotoxicity
<i>LOAEL</i>	30 ppm
<i>NOAEL</i>	not determined
<i>Exposure duration</i>	7 hours/day
<i>LOAEL uncertainty factor</i>	10
<i>Interspecies uncertainty factor</i>	10
<i>Intraspecies uncertainty factor</i>	10
<i>Cumulative uncertainty factor</i>	1000
<i>Reference Exposure Level (7 h)</i>	0.03 ppm (0.15 mg/m ³ ; 150 µg/m ³)

The study by Schwetz *et al.* (1974) is the only published developmental toxicity study of chloroform. Exposure of pregnant rats to 30 ppm (150 mg/m³) CHCl₃ for 7 hours per day on days 6-15 of gestation resulted in fetotoxicity as indicated by decreased crown-rump length and increased incidences of wavy ribs and skeletal ossification defects. Maternal toxicity was also observed. An abstract by Dilley *et al.* (1977) indicates an absence of teratological effects in rats exposed to 20,000 mg/m³ CHCl₃ on days 7-14 of gestation. The data from this study were not available for review, therefore, the Schwetz *et al.* study is used in developing the severe adverse effect level for chloroform. A NOAEL was estimated from the reported LOAEL using an uncertainty factor of 10. An additional uncertainty factor of 100 was applied to account for inter- and intraspecies differences. The level protective against severe adverse effects for a 7 hour exposure is estimated as 0.03 ppm (0.15 mg/m³).

Level Protective Against Life-threatening Effects

No recommendation is made due to the limitations of the database. NIOSH (1995) lists a (revised) IDLH of 500 ppm based on acute inhalation toxicity in humans but the selection of the level is somewhat arbitrary and the IDLH does not make allowance for sensitive individuals.

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CHLOROFORM

CAS No.: 67-66-3

I. PHYSICAL AND CHEMICAL PROPERTIES (From HSDB, 1998)

Molecular weight	119.49
Boiling point	61° C
Melting point	-63.5° C
Vapor pressure	200 mm Hg 25° C
Air concentration conversion	1 ppm = 4.9 mg/m ³ at 25° C

II. HEALTH ASSESSMENT VALUES

Unit Risk Factor:	5.3 E-6 (µg/m ³) ⁻¹
Slope Factor:	1.9 E-2 (mg/kg-day) ⁻¹

[Calculated by CDHS (1990) using a nonthreshold linear procedure. This unit risk is the arithmetic average of unit risks generated by CDHS and Bogen *et al.* (1989) for renal tumors observed in rats and mice reported by Jorgenson *et al.* (1985) and NCI (1976), and the geometric mean for supporting data sets (Roe *et al.*, 1979; Tumasonis *et al.*, 1985).]

III. CARCINOGENIC EFFECTS

Human Studies

There is no information currently available in the open literature which examines the potential relationship between exposure to chloroform in an occupational setting and human cancer. However, several studies are available which examine the relationship between trihalomethanes (THM) in drinking water and human cancer.

Many studies have concentrated on chlorination of water and concomitant production of halogenated carcinogens as a causative factor in human cancers. Cantor *et al.* (1978) compared age-adjusted cancer mortality rates by site and sex for whites in the years 1968-71 to measures of THM and the drinking water. A weighed linear regression model was used to predict cancer rates in 923 U.S. counties which were over 50% urban in 1970. Reasonably strong associations between bladder cancer and THM levels in drinking water were found after controlling for confounding by urbanization, ethnicity, social class, and county industrialization. The association was not changed by controlling for occupation in certain high-risk (for bladder cancer) industries nor by lung cancer rates used as a surrogate measure for cigarette smoking. The measure of THM most associated with bladder cancer in both white males and females was that of bromine-containing trihalomethanes (BTHM). Chloroform and total trihalomethanes (TTHM) were not as well associated. There were inconsistent associations between other cancer sites and THM levels. However, there was some evidence of an association of chloroform in

drinking water with kidney cancer in males, which Cantor *et al.* believed warrants further study.

Hogan *et al.* (1979) examined the potential association between chloroform levels in finished drinking water supplies and various site-specific cancer mortality rates. The most consistent associations were between chloroform “exposure” and cancers of the bladder, rectum and large intestine. Hogan *et al.* stated that the results of this ecological study must be interpreted with caution and the association between chloroform levels in drinking water and certain types of cancer (e.g., bladder, large intestine and rectum) warrant further study.

Carlo and Mettlin (1980) analyzed 4,255 cases of cancer reported in Erie County, NY, between 1973 and 1976 for any relationship between cancer and type of water source, THM levels, and a variety of socioeconomic variables. No significant association between THM and cancers were noted in the regression analyses for the total population. When regression analyses were conducted for population stratified by race-sex, a significant association was found between THM levels in drinking water and pancreatic cancer in white males ($p < 0.05$). The investigators caution that the lack of association between THM and pancreatic cancer in other sex-race groups and absence of association between THM and other cancer raises doubts as to the validity of this finding.

Brenniman *et al.* (1980) conducted a case-control study in Illinois to determine whether an association exists between chlorination of drinking water and gastrointestinal and urinary tract cancers. Cases (3,208) and controls (43,666) were classified according to residence in chlorinated and unchlorinated groundwater communities. Elevated risk was found for cancers of the gallbladder, large intestine, total gastrointestinal, and urinary tract for women. However, the investigators considered the results tenuous because, when the data were subclassified according to several control variables, the associations were not strengthened. Many confounding factors were not controlled including smoking, diet, ethnicity, and occupation.

Alavanja *et al.* (1980) conducted a case-control study on all gastrointestinal and urinary tract cancer deaths occurring from January 1, 1968 through December 31, 1970 in seven counties in New York. There was a statistically significant excess risk of stomach cancer in females, and of stomach, esophagus, large intestine, rectum, liver and kidney, pancreas, and urinary bladder in males residing in chlorinated water areas in the seven counties studied. The investigators concluded that the excess risk was associated with living in chlorinated areas of certain counties and was not due to a disparity in the age, race, or ethnic distribution, or to urban/rural classification, hazardous occupation, or a surface vs. ground water difference. Several confounding factors were not controlled including cigarette smoking and diet.

The association between site-specific cancer mortality and THM exposure, as estimated by chlorine dose, was investigated by Young *et al.* (1981). Cases were obtained from death certificates provided by the Wisconsin Bureau of Health Statistics and consisted of all white female deaths that occurred 1972-77 within 28 counties due to malignant

neoplasms of esophagus, stomach, colon, rectum, liver, bile ducts, pancreas, urinary bladder, kidney, lung, breast, and brain. Only death from colon cancer was associated with chlorine dose ($p < 0.05$). The risk of colon cancer, calculated as odds ratios, was over twice as great when the water source was affected by rural runoff. This variable was tested because of the assumption that rural runoff increased the organic precursors to THMs. While the association of colon cancer with chlorination and rural runoff factors is provocative, the findings of this study must be considered inconclusive due to the possible underestimation of risk associated with misclassification error and spurious contribution from unknown colon cancer risk factors (Young *et al.*, 1981).

Wilkins and Comstock (1981) conducted a nonconcurrent prospective study to investigate possible relationships between products of water chlorination and human cancer. Site and sex-specific incidence rates for malignant neoplasm of liver, biliary passages, kidney, and bladder were constructed from hospital records, a cancer registry, and death certificates. Incidence rates for cancer of the bladder among men and cancer of the liver among women were not significant relative to the other exposure groups among persons using water from the chlorinated surface supply. While the results were only weakly suggestive, Wilkins and Comstock noted that bladder cancer has been suggestively linked with chloroform and other indices of THM in drinking water in other studies.

Gottlieb and Carr (1982) studied the potential relationship between chlorination of drinking water and cancer in 20 south Louisiana parishes. Chlorinated surface water was associated with a significant risk for rectal cancer ($p = 0.012$). The odds ratio for rectal cancer in groups receiving high chlorination level (> 1.09 ppm chlorine) to groups with no chlorinations is 1.53 (95% CI=1.15-2.04) in surface water supplied areas. Gottlieb and Carr concluded that there appears to be some cancer risk associated with water chlorination, but definitive studies are needed with respect to the role of industrial confounders and the importance of co-contaminants.

Lawrence *et al.* (1984) used a case-control approach to study the association of chloroform exposure via drinking water to colorectal cancer in white female teachers in upstate New York. Analysis was based on 395 cases of colon and rectal cancer and 395 control noncancer deaths matched with respect to age and year of death. No effect of cumulative chloroform exposure on incidence of colorectal cancer deaths was observed.

Cantor *et al.* (1987) examined the association between use of chlorinated drinking water and bladder cancer by a case-control study design. The investigators interviewed 2,982 cases and 5,782 controls in 10 geographic areas of the U.S. Risk of bladder cancer was primarily associated with use of tap water rather than nontap beverages. Among white males, the coefficients for tap and nontap beverages were 0.176 ($p < 0.001$) and 0.037 ($p = 0.42$), and among white females, the coefficients were 0.123 ($p = 0.09$) and 0.089 ($p = 0.39$), respectively. It was suggested that nonvolatile components of tap water may be associated with risk of bladder cancer since both heated and nonheated tap water beverages were significantly associated with bladder cancer risk among males. The relative risk increased with increasing tap water intake. While this investigation was

quite thorough in many respects, there is a need for confirmation of these findings. The contribution of chloroform in the etiology of human bladder cancer in men may be overshadowed by other nonvolatile chemicals present in the drinking water.

Overall, the present epidemiological evidence suggests an association between chlorinated drinking water consumption and human cancer, particularly bladder and gastrointestinal cancers. However, these relationships cannot be directly correlated to chloroform exposure because many other carcinogens are found in drinking water including other chlorinated halomethanes, brominated halomethanes, industrial pollutants, and nonvolatile halogenated compounds.

Animal Studies

The National Cancer Institute conducted carcinogenesis bioassays of chloroform in both sexes of Osborne-Mendel rats and B6C3F₁ mice (NCI, 1976). Mice and rats were given either corn oil or chloroform in corn oil by gavage, 5 days/week for 78 weeks. Time-weighted average doses for female rats were 100 and 200 mg/kg, and for male and female mice were 138 and 277 mg/kg, and 238 and 477 mg/kg, respectively. Tumor incidences are listed in Table 1.

A statistically significant increase ($p < 0.05$) in epithelial tumors of renal tubular origin was noted in the treated males. Ten carcinomas, two of which had metastasized, and three adenomas of renal tubular origin were found in 12 high dose male rats. In the low dose males, two carcinomas and two adenomas of tubular origin were observed in four out of 50 animals. Among the 48 high dose female rats, one tubular epithelial carcinoma and one renal squamous cell carcinoma were observed. No renal epithelial tumors were noted in matched or colony controls. The NCI reported that these type of tumors rarely occur spontaneously in Osborne-Mendel rats.

The incidence of thyroid tumors in female rats was statistically higher than controls in both treated groups ($p = 0.05$, Fisher exact test) but not in treated male rats. The incidence of hepatocellular carcinoma or neoplastic nodules was not increased in the chloroform-treated rats. Although inflammatory pulmonary lesions occurred in all test groups, the lesions were more severe and occurred more frequently in the chloroform-treated rats.

The incidence of hepatocellular carcinomas in mice was significantly elevated in all treatment groups ($p < 0.001$, Fisher exact test). The NCI reported that in their experience the spontaneous incidence of hepatocellular carcinomas in B6C3F₁ mice is about 5-10% in males and 1% in females. The NCI concluded that chloroform treatment was associated with increased incidences of hepatocellular carcinomas in male and female mice and renal epithelial tumors in male rats.

In addition, Reuber (1979), based on his examination of the histological sections from the NCI study, concluded that chloroform treatment was also associated with cancer of the

liver in rats and an increased incidence of malignant lymphomas in mice. However, the NCI did not agree with his findings.

The carcinogenicity of chloroform given in drinking water was evaluated in male Osborne-Mendel rats and female B6C3F₁ mice (Jorgenson *et al.*, 1985). The chloroform used (technical grade), was found to contain 100 ppb diethylcarbonate, and trace amounts of 1,1-dichloroethane, dichloroethylene, carbon tetrachloride, and an unidentified C₅H₁₀ hydrocarbon. Time-weighted average doses of chloroform calculated based on water consumption rates and body weight, ranged up to 160 and 263 mg/kg-day for rats and mice, respectively. Two control groups were used, an untreated control, and a control group of animals with restricted access to water.

Jorgenson *et al.* observed a dose-related significant increase in renal tubular cell adenomas and adenocarcinomas in male rats, but found no treatment-related increases in tumor incidence in the female mice (Table 1). The lack of liver tumors in female B6C3F₁ mice is in sharp contrast to the results of the NCI study. A major difference between the NCI study and the Jorgenson study is the mode of administration. Administration of chloroform to rats in a corn oil vehicle slowed the gastrointestinal absorption of chloroform relative to the absorption rate observed after administration as a bolus in water (Withey *et al.*, 1983). In the Jorgenson *et al.* study, the rats received small doses of chloroform each time they drank water. The corn oil vehicle effect (Withey *et al.*, 1983) may have diminished the differences in absorption kinetics expected with the two different methods of administration. Therefore, any differences in peak blood concentrations between the NCI study and the Jorgenson study may not have been sufficient to account for the difference in liver tumor incidence. Physiologic or metabolic changes produced by corn oil consumption might interact with chemical carcinogens altering the production of liver tumors (Bull *et al.*, 1986; Newberne *et al.*, 1979).

A series of experiments was conducted by the Huntingdon Research Center to determine the effects of chronic ingestion of chloroform in a toothpaste base in mice, rats, and beagle dogs. In the first set of experiments (Roe *et al.*, 1979), doses of 17 and 60 mg chloroform/kg were administered by gavage in toothpaste to male and female ICI mice, 6 days/week for 80 weeks followed by a 16 week observation period (Experiment I). Controls (N=104) were treated with 1 ml chloroform-free toothpaste/kg-day. Aside from increased nonneoplastic liver lesions (moderate fatty degeneration), the only significant difference in pathology reported was an increase in the incidence of kidney tumors in high dose male mice, three were hypernephromas (tubular adenocarcinoma) and the remainder were adenomas (tumor incidences listed in Table 1). The incidence of renal tumors in high-dose male ICI mice was significantly greater than control mice ($p = 0.00012$, Fisher exact test). None of the female ICI mice examined developed renal tumors (Roe *et al.*, 1979). Roe *et al.* (1979) also investigated other components of the toothpaste base for carcinogenicity using male ICI mice. No lesion in this part of the study could be correlated with treatment.

In a third mouse experiment (Experiment III), Roe *et al.* (1979) compared the effects of toothpaste containing 3.5% chloroform on male mice of four different strains (C57BL,

CBA, CF/1, and ICI). Treatment with chloroform was not associated with any increase in liver or lung neoplasms relative to vehicle-treated controls in any of the four strains tested but was associated with significantly higher incidences of moderate to severe kidney pathology in CBA and CF/1 mice relative to the controls ($p < 0.0001$, chi-square test).

Palmer *et al.* (1979) gave groups of 50 Sprague-Dawley rats (both sexes) 0 or 60 mg chloroform/kg-day, 6 days/week by gavage in a toothpaste base for 80 weeks, followed by a 15 week observation period. There were no differences in the incidences of tumors of any site examined, including brain, lung, liver, kidney, and mammary gland, between treated and control animals. Heywood *et al.* (1979) investigated the carcinogenicity of chloroform in a toothpaste base in beagle dogs. Groups of male and female dogs received toothpaste base with 0, 15 or 30 mg chloroform/kg-day, 6 days/week for 7.5 years (8-16 dogs/sex), followed by a 20-24 week recovery period. Treatment with chloroform at the high dose was associated with significant elevations in SGPT levels but no treatment-related tumors were observed.

Chloroform treatment of rats via drinking water was associated with hepatic neoplastic nodules and hepatic adenofibrosis (Tumasonis *et al.*, 1985). Chloroform was administered to male and female Wistar rats in the drinking water at about 220 mg/kg/day and 160 mg/kg/day for the female and male rats, respectively. The incidence of hepatic neoplastic nodules was significantly elevated in treated females compared to controls ($p < 0.03$, Fisher exact test). In males, the incidence of hepatic neoplastic nodules did not differ in control and chloroform-treated groups. Increased incidences of hepatic adenofibrosis were observed in chloroform-treated males and females relative to controls. In contrast to the NCI and the Jorgenson *et al.* studies, renal tumors were not associated with chloroform treatment. However, Tumasonis *et al.* indicated that kidneys were only examined when grossly observable lesions were evident. Hence, kidney tumors may have been missed by this protocol. Tumasonis *et al.* concluded that chloroform is a hepatocarcinogen in Wistar rats.

IV. DERIVATION OF CANCER POTENCY

Basis for Cancer Potency

Chloroform is carcinogenic to rats and mice (NCI, 1976; Roe *et al.*, 1979; Jorgenson *et al.*, 1985). The International Agency for Research on Cancer (IARC) has classified chloroform as a possible human carcinogen (Group 2B). Similarly, the U.S. EPA has placed chloroform in Group B2 in their classification scheme, based on sufficient evidence of carcinogenicity in animals, but inadequate epidemiologic evidence. Current evidence and understanding of the carcinogenic process is insufficient to classify chloroform as either a genotoxic or epigenetic carcinogen, and it is possible that both types of effects are involved.

Table 1: Chloroform carcinogenicity bioassay tumor incidence data used to estimate cancer potency (CDHS, 1990)

Study	Strain/Species	Sex	Tumor Site	Lifetime Daily Dose (mg/kg-day)	Tumor Incidence
NCI (1976)	B6C3F ₁ mouse	M	hepatocellular carcinoma	control	1/18
				83	18/50
				167	44/45
	B6C3F ₁ mouse	F	hepatocellular carcinoma	control	0/20
				143	36/45
				287	39/41
	Osborne-Mendel rat	M	renal tubular adenoma or adenocarcinoma	control	0/19
				45	4/38
				90	12/27
Jorgenson <i>et al.</i> (1985)	Osborne-Mendel rat	M	renal tubular adenoma or adenocarcinoma	control	4/301
				18	4/313
				38	4/148
				79	3/48
Roe <i>et al.</i> (1979)	ICI mouse (Experiment I)	M	renal tubular adenoma or adenocarcinoma	control	0/72
				12	0/37
				43	8/37
	ICI mouse (Experiment II)	M	renal tubular adenoma or adenocarcinoma	control	6/237
				40	9/49
	ICI mouse (Experiment III) ^a	M	renal tubular adenoma or adenocarcinoma	control	1/49
				42	5/47
	ICI mouse (Experiment III) ^b	M	renal tubular adenoma or adenocarcinoma	control	1/50
42				12/48	
Tumasonis <i>et al.</i> (1985)	Wistar rat	F	cholangiocarcinoma	control	0/18
				220	34/40
	Wistar rat	M	cholangiocarcinoma	control	0/22
				160	17/28
Reuber <i>et al.</i> (1979) using NCI (1976)	Osborne-Mendel rat	F	cholangiocarcinoma and cholangiofibroma	control	0/20
				50	3/39
				100	11/39

^a toothpaste base was used as the vehicle; ^b arachis oil was used as the vehicle

The estimation of cancer risk to humans from exposure to chloroform by CDHS (1990) is based on animal studies. Data were chosen based primarily on statistical significance, as discussed below.

Methodology

The following data sets were evaluated to estimate chloroform cancer potency: 1) Liver tumor data in male and female B6C3F₁ mice, and renal tubular cell tumors in male Osborne-Mendel rats from the NCI (1976) study were chosen because statistically significant increases in these tumor types were observed in chloroform treated animals relative to controls; 2) Renal tubular cell tumor data in male Osborne-Mendel rats from the Jorgenson *et al.* (1985) study and in male ICI mice in the Roe *et al.* (1979) study were used for risk estimation based on a statistically significant increase in kidney tumors in

chloroform treated animals relative to controls; 3) Liver cholangiocarcinoma ("adenofibrosis") data in female rats from Tumasonis *et al.* (1985), and from Reuber's reanalysis of the NCI (1976) slides (Reuber, 1979) were also analyzed with the linearized multistage model (GLOBAL86). Administered doses were transformed to lifetime doses by adjusting for the number of days exposed per week and the ratio of the length of exposure to the length of the experiment (exposure plus observation period).

Calculated q_1^* values from the above studies ranged from 8.1×10^{-4} to 1.9×10^{-2} (mg/kg-day)⁻¹. These represent cancer potency estimates for rats and mice and must be converted to theoretical equivalent potency values for humans. This conversion is based on equivalency of dose per unit surface area according to Anderson *et al.* (1983). These "human" cancer potencies range from 4.2×10^{-3} to 2.6×10^{-1} (mg/kg-day)⁻¹. Scaling factors ranged from 5.19 to 13.57.

The NCI (1976) and Jorgenson *et al.* (1985) studies were the most thorough studies in terms of the number of doses tested, sample size, histological examination of the animals, and other procedural and statistical methods presented. As such, CDHS placed more confidence in the potency slopes from these studies than in the other studies. The potency slopes derived from Roe *et al.* (1979) and Tumasonis *et al.* (1985) fall within the range of those from the NCI and Jorgenson studies.

Bogen *et al.* (1989) used a physiologically based pharmacokinetic (PBPK) model to estimate metabolized dose for chloroform to use in the analysis of cancer potency with the linearized multistage model to carcinogenicity bioassay data from NCI (1976), Jorgenson *et al.* (1985), Roe *et al.* (1979), and Tumasonis *et al.* (1985). In the application of the model, the liver was considered to metabolize chloroform through a saturable enzyme system following Michaelis-Menten kinetics. This approach is consistent with the evidence that chloroform metabolites are responsible for toxicity and probably for the carcinogenicity of chloroform. The potency estimates made from these studies ranged from 4.8×10^{-3} to 5.0×10^{-1} (mg/kg-day)⁻¹. These corresponded to unit risks of 4.5×10^{-6} to 4.7×10^{-4} (ppb)⁻¹. These potency estimates are incorporated into DHS staff's best estimate of cancer potency for chloroform.

There are no studies on the carcinogenicity of chloroform by the inhalation route. Therefore, estimation of the cancer risk from exposure to chloroform in the ambient air required extrapolation from the oral route. In so doing, it is assumed that chloroform is also carcinogenic by the inhalation route, and that the risk posed by an absorbed inhaled dose of chloroform is equivalent to that posed by the same dose absorbed after oral administration. In the final risk range, the DHS staff included tumor sites that did not appear to be vehicle-dependent. Therefore, the liver tumors were not included in the range of risks or the best estimate of risk, due to the possible potentiation of liver tumors by the corn oil vehicle.

The best estimate of unit risk was considered by CDHS (1990) to be the arithmetic average of unit risks generated by CDHS (1990) and Bogen *et al.* (1989) for rat renal tumors in Jorgenson *et al.* (1985) and NCI (1976) and of the geometric mean for

supporting data sets (Roe *et al.*, 1979; Tumasonis *et al.*, 1985). This unit risk, $5.3 \text{ E-6 } (\mu\text{g}/\text{m}^3)^{-1}$, represents the best estimate using a nonthreshold linear procedure and using most of the data on the carcinogenicity of chloroform. It included analysis by PBPK modeling of metabolized dose, as well as analysis of potency based on applied dose.

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PARTICULATE MATTER FROM DIESEL-FUELED ENGINES

CAS No: not available

I. PHYSICAL AND CHEMICAL PROPERTIES (From HSDB, 1998)

Molecular weight	not applicable
Boiling point	not applicable
Melting point	not applicable
Vapor pressure	not applicable
Air concentration conversion	not applicable

II. HEALTH ASSESSMENT VALUES

Unit Risk Factor: $1.3 \text{ E-4} - 1.5 \text{ E-3} (\mu\text{g}/\text{m}^3)^{-1}$ (measured as particulate matter)[Scientific Review Panel unit risk “reasonable estimate” = $3.0 \text{ E-4} (\mu\text{g}/\text{m}^3)^{-1}$.]

Slope Factor: $1.1 \text{ E+0} (\text{mg}/\text{kg}\text{-day})^{-1}$

[Human occupational exposure lung tumor incidence (Garshick *et al.* (1987a, 1988), estimated exposure concentrations (Woskie *et al.*, 1988a,b), relative risk model (OEHHA, 1998); human occupational exposure lung tumor incidence, meta-analysis (OEHHA, 1998).]

III. CARCINOGENIC EFFECTS

Human Studies

The epidemiological evidence concerning the carcinogenicity of diesel exhaust primarily involves cancers of the lung and bladder. The review of human diesel exhaust-exposure cancer studies in the document entitled *Health Risk Assessment For Diesel Exhaust* written for the Toxic Air Contaminant (TAC) program (OEHHA, 1998) focuses first on studies of lung cancer (Sections 6.2.1 and 6.2.2) and then turns to those of bladder cancer (Section 6.2.3). The evidence for causation of lung cancer was then assessed using criteria for causal inference from epidemiological studies (Section 6.2.4). The evidence linking diesel exposure and bladder cancer was not as extensive or compelling, and is discussed in the diesel exhaust TAC document but not in this summary. Because there are no epidemiological studies involving industrial hygiene measurements concurrent with the exposures of the study populations, exposure has typically been defined by the surrogate measures of usual occupation or job classification within an industry.

Review Of Lung Cancer Studies

The question of whether diesel exhaust causes lung cancer has been addressed by both industry-based cohort and case-control studies as well as population-based studies of lung cancer. In Section 6 of the diesel exhaust TAC document (OEHHA, 1998), the review of the lung cancer studies was divided into five parts focusing on studies of: (1) truck drivers, (2) transport and equipment workers, (3) dock workers, (4) railway workers, and (5) other miscellaneous

occupations involving diesel exhaust exposure. This summary will focus on the railway workers studies, which were used to derive the range of human cancer risks associated with diesel exhaust exposure. A summary of all occupational studies evaluating the relationship between diesel exhaust exposure and lung cancer is provided in Table 1.

Studies Of Lung Cancer Among Railway Workers

In 1959, Kaplan studied lung cancer mortality among employees of the Baltimore and Ohio Railroad. This railroad initiated locomotive dieselization in 1935, completing this process by 1958. Workers employed at any time between 1953 and 1958 were eligible for entry into the cohort; 154 deaths from primary cancers of the lung or bronchus were identified. Exposure was categorized into three groups by job type. The lung cancer SMR for the most exposed group, relative to the general population, was 0.875. The limited number of years of exposure to diesel exhaust for some members of the cohort and the abbreviated follow-up time do not allow for sufficient latency to be informative regarding the relationship of diesel exhaust exposure to lung cancer. In addition, no data on smoking were available.

In the Third National Cancer Survey discussed above, Williams *et al.* (1977) found a nonsignificant increased risk for railroad workers among lung cancer patients, OR = 1.40, based on 12 cases (no confidence intervals reported).

Howe *et al.* (1983) carried out a mortality study of 43,826 male pensioners of the Canadian National Railroad. The cohort consisted of all male pensioners who were alive at the beginning of 1965. Subjects were followed until 1977, by which time 933 deaths from respiratory cancer (trachea, bronchus and lung) had been recorded. Occupations at the time of retirement were classified as “nonexposed”, “possibly exposed” or “probably exposed”. Analysis restricted to individuals retiring after 1950 (n = 897 cases) yielded relative risks of 1.00, 1.20 ($p = 0.013$), and 1.35 ($p < 0.001$) for the three exposure groups, respectively (test for trend: $p < 0.001$). There was little change in these effect estimates when individuals involved in locomotive maintenance (and who therefore may have been exposed to asbestos) were excluded from the analysis (n = 69).

This study also found coal dust to be associated with lung cancer, with a similar increasing trend with degree of exposure. Because of a high degree of overlap between exposures to coal dust and to diesel exhaust, the authors could not separate the effects of the two. However, since there is evidence from animal and human studies for the carcinogenicity of diesel exhaust, but such evidence does not exist for coal dust, the apparent effect of coal dust was more likely to have been due to confounding by diesel exhaust, rather than vice versa. No smoking information was available for this study, although there were increasing trends with degree of diesel exposure for mortality from emphysema (SMRs = 1.00, 1.35, and 1.44) and other smoking-related cancers combined (SMRs = 1.00, 1.08, and 1.16). The authors suggested that since the results were based on internal comparisons little variation in smoking would be expected among the different diesel exposure groups.

Garshick *et al.* (1987a) carried out a case-control study of lung cancer in U.S. railroad workers. Cases comprised 1,256 lung cancer deaths occurring between 1981 and 1982 in the population of

active or retired railroad workers who had had 10 years or more of railroad service and were born in 1900 or later. Two controls who had died of causes other than cancer, suicide or accident were matched to each case by dates of birth and death. Next of kin were interviewed to obtain information about the decedents, including their smoking habits. Job codes were obtained from the Railroad Retirement Board, and an industrial hygiene survey was used to classify the degree of diesel exposure for each job type. Jobs were dichotomously categorized as exposed or not exposed to diesel exhaust.

Garshick *et al.* considered exposure to diesel exhaust to have begun in 1959, since the transition from steam to diesel-powered locomotives took place mainly in the 1950s, and was nearly complete in 1959. Years of diesel exhaust exposure to death or retirement were totaled for each worker. The analysis separated those workers who died at age 65 (retirement age) or older (921 cases and 1,748 controls) from those workers <64 years at death (335 cases and 637 controls). Analysis by logistic regression showed no effect of diesel exhaust in the workers in the older age category, who had substantially less diesel exposure than those in the younger category. For example, 36% of cases and 43% of controls had no exposure in the younger group, while 52% of cases and 53% of controls had no exposure in the older group. Furthermore, 35% of cases and 26% of controls had more than 19 years of diesel exposure in the younger group, while only 3% of cases and controls had more than 19 years of diesel exposure in the older group.

In the group whose members were younger than 64 years old at time of death, the analysis by Garshick *et al.* showed evidence of an exposure-response relationship with an OR of 1.41 (95% C.I. = 1.06-1.88) for 20 or more years of exposure (diesel-years) after adjusting for smoking and asbestos exposure. Excluding exposure occurring within five years of death, the OR for 15 or more years of cumulative diesel exposure was 1.43 (95% C.I. = 1.06-1.94). For workers with 5 to 14 years of cumulative exposure, the OR was 1.07 (95% C.I. = 0.69-1.66) relative to a reference category of 0 to 4 diesel exposure years.

Garshick *et al.* (1988) also conducted a retrospective cohort study of U.S. railroad workers. Eligible for inclusion in the cohort were white males aged 40 to 64 years, who started work between 1939 and 1949 and were still employed in 1959 in designated jobs. Follow-up extended through 1980. Jobs with recognized asbestos exposure were not included in the job codes selected for study, although some of the selected occupations had at least some potential for asbestos exposure. The cohort consisted of 55,407 men, among whom there were 19,396 deaths, including 1,694 attributable to lung cancer. Diesel exhaust exposure was characterized based on their 1959 job group. Career paths were found to be very stable in the railways, such that a worker aged 40-44 with a diesel-exposed job in 1959 was likely to have a diesel-exposed job 20 years later; similarly a nonexposed person in 1959 was likely to have a nonexposed job 20 years later.

The youngest workers in 1959 had the longest potential duration of diesel exposure in the cohort. In a proportional-hazards model these workers had the highest estimated relative risks for lung cancer associated with diesel exhaust exposure: the relative risk for the group aged 40-44 in 1959 was 1.45 (95% C.I. = 1.11-1.89); for the group aged 45-49 the relative risk was 1.33 (95% C.I. = 1.03-1.73); for the group aged 50-54, 1.12 (95% C.I. = 0.88-1.42); for the group aged 55-59, 1.18 (95% C.I. = 0.94-1.50); and for the group aged 60-64, 0.99 (95% C.I. = 0.74-1.33).

Though the results were statistically significant only for the two youngest groups, there was a decreasing trend with increasing age in 1959 (except for the 55-59 year age group), implying declining risk with decreasing duration of exposure.

When exposure to diesel over the last five years before death was excluded, a relationship was apparent between lung cancer risk and duration of exposure. The group with greater than 15 years of cumulative exposure had a RR for lung cancer of 1.72 (95% C.I. = 1.27-2.33); for those with 10 to 14 years of exposure the RR was 1.32 (95% C.I. = 1.13-1.56); for 5 to 9 years, 1.24 (95% C.I. = 1.06-1.44); and for 1-4 years, 1.20 (95% C.I. = 1.01-1.44). All of these results are statistically significant.

Although no smoking information was available for the cohort, the previous case-control study of railway workers by the same group (Garshick *et al.*, 1987a) reported that little change occurred in the estimates of diesel exhaust effect due to adjustment for smoking habits and asbestos exposure (unadjusted OR = 1.39, 95% C.I. = 1.05-1.83; adjusted OR = 1.41, 95% C.I. = 1.06-1.88). In this analysis, the larger percentage of workers whose pack-year history was unknown (23% of cases and 22% of controls) was treated as a separate category of smoking. In additional analyses using only those workers for whom the investigators had detailed smoking data (n = 758), the ORs for 20 years of diesel exposure ranged from 1.50-1.53, adjusted for asbestos exposure and several specifications of cigarette smoking history. These models included pack-years as a single continuous variable, as two independent variables (cigarettes per day and years of smoking), or as a categorical variable classified in terms of the number of years the study subject had stopped smoking prior to death. These analyses suggested that the diesel exhaust-lung cancer odds ratios were not confounded by cigarette smoking in this population. Moreover, in a group of railroad workers previously surveyed for asbestos exposure (Garshick *et al.*, 1987b) there was no difference in smoking prevalence between workers with and without diesel exhaust exposure (data not presented).

It should be noted that the case-control and the cohort studies by Garshick *et al.* involved different study populations: The case-control study (Garshick *et al.* 1987a) contained cases and controls who had died in 1981 and 1982, whereas the cohort study (Garshick *et al.*, 1988) involved deaths occurring up to 1980. Thus, they may be considered different tests of the hypothesis of an association between lung cancer and diesel exhaust exposure, although this does not exclude the possibility of a common bias shared by the two studies, such as exposure to chemicals transported by rail or to suspended dusts and debris.

In the American Cancer Society prospective mortality study mentioned above (see Section 6.2.1.1, OEHHA, 1998), Boffetta *et al.* (1988) found an age- and smoking-adjusted RR of 1.59 (95% C.I. = 0.94-2.69) for lung cancer mortality in railroad workers. This estimate was based on only 14 lung cancer deaths.

Swanson *et al.* (1993) also examined the industrial category of railroad workers in their case-control study of lung cancer. The smoking-adjusted odds ratios for white males (67 cases) were 1.2 (95% C.I. = 0.5-2.7) for 1-9 years of employment and 2.4 (95% C.I. = 1.1-5.1) for more than 10 years of employment (χ^2 test for trend: $p < 0.05$). Elevated, but nonsignificant, smoking-adjusted ORs were also associated with the 31 lung cancer cases occurring in African-American

railroad workers, OR = 2.6 (95% C.I. = 0.8-7.9) for 1-9 years and OR = 2.7 for ≥ 10 years of employment (95% C.I. = 0.6-12.1).

Nokso-Koivisto and Pukkala (1994) compared the incidence of lung cancer among locomotive drivers to the total Finnish population. The retrospective cohort consisted of the 8,391 members of the Finnish Locomotive Drivers' Association from 1953 until 1991 (retired drivers remain members until death). After excluding 302 members for lack of personal identification information, an overall standardized incidence ratio (SIR) of 0.86 (95% C.I. = 0.75-0.97) was found (236 cases). The overall incidence for all cancer sites was also lower than expected, SIR 0.95 (95% C.I. = 0.89-1.01) but the incidence of mesothelioma (SIR 4.05, 95% C.I. = 1.75-7.97) and oral cavity/pharyngeal cancers (SIR 1.75, 95% C.I. = 1.02-2.80) were significantly increased. Prior to the 1970s Finnish drivers trained for 2 years in railroad workshops, where significant exposure to asbestos occurred routinely during steam engine maintenance, with little, if any, diesel exposure. Only drivers working after this period had the potential for substantial exposure to diesel exhaust, and the electrification of the railroad in the 1970s and 1980s may also have reduced the proportion of the cohort's person-years that truly reflect exposure to diesel exhaust. No data on smoking within the cohort were available, though a cross-sectional study of locomotive drivers in 1976 showed that the prevalences of current smokers (40%), ex-smokers (34%), and never-smokers (26%) were similar to those in the Finnish population as a whole.

All three population-based case-control studies found elevated risks for lung cancer in railroad workers (Williams *et al.*, 1977; Boffetta *et al.*, 1988; Swanson *et al.*, 1993); however, only the study by Swanson *et al.* (1993) found a statistically significant increase, with a smoking-adjusted OR of 2.4 (95% C.I. = 1.1-5.1) for workers with ten or more years of employment. This study also found evidence of a significant exposure-response relationship for the 67 cases observed in white railroad workers. Williams *et al.* (1977) and Boffetta *et al.* (1988) had relatively fewer railroad workers (12 and 14 cases respectively) and no information on duration of exposure.

In the railroad industry-based studies, three of the larger studies identified statistically significant increases in relative risk (Howe *et al.*, 1983; Garshick *et al.*, 1987a; Garshick *et al.*, 1988). The large cohort reported on by Howe *et al.* (1983) found elevated risks for individuals categorized as "probably" and "possibly" exposed to diesel exhaust, but without adjustment for smoking or duration of employment, the underlying risk is uncertain. In both the case-control and cohort studies by Garshick *et al.*, 1987a, 1988), significantly increased risks were associated with long-term employment in diesel-related railroad jobs. Both studies had substantial exposure assessment, sufficient latency, and duration of employment data, and the case-control investigation also controlled for potential confounding by smoking and by asbestos exposure. In contrast, the study by Nokso-Koivisto *et al.* (1994), found no increase in lung cancer risk among Finnish locomotive engineers, though the description of the cohort indicates the earlier cases were unlikely to have experienced any diesel exposure.

Studies Of Lung Cancer Among Truck Drivers

The studies that have examined the lung cancer risk to truck drivers are summarized in Table 1. These studies have consistently reported small increases in lung cancer relative risk. However, the studies suffer from various deficiencies, including small numbers of subjects, inadequate adjustment for confounding, and crude exposure assessments, usually based on occupational classification. Most of the earlier studies did not adjust for smoking. Because of evidence that truck drivers have a higher smoking prevalence (Wynder and Higgins, 1986), individual studies that do not account for smoking generally provide limited evidence regarding carcinogenicity. Before 1988, the two studies that took smoking into account, Williams *et al.* (1977) and Hall & Wynder (1984), had ORs of 1.4 - 1.5, which were not statistically significant. The third study that accounted for smoking (Damber and Larsson, 1985, 1987), only found significantly elevated risks in truck drivers who smoked after stratifying on age (i.e., only for those > 70 years old at diagnosis). However, in the follow-up study, after analyzing for duration of employment (20 or more years), elevated but nonsignificant risks were observed for all professional drivers combined (Damber and Larsson, 1987).

By comparison, the majority of studies published since 1988 have adjusted for smoking to varying degrees. Of the smoking-adjusted population based studies, two of four found statistically significant increases in the relative risk for lung cancer associated with occupation as a truck driver, especially in individuals employed for 10 or more years (Hayes *et al.* 1989; Swanson *et al.* 1993). In addition, both studies reported some evidence of a positive trend between increased duration of employment and risk for lung cancer. Although both found statistically significant trends ($p < 0.05$), the only stratum with statistically significant relative risk estimates was that including 20 or more years' employment as a truck driver, with ORs of 1.5 (95% C.I. = 1.0-2.3) and 2.5 (95% C.I. = 1.1-4.4), reported by Hayes *et al.* (1989) and Swanson *et al.* (1993), respectively.

Three of the six more recent industry-specific studies adjusted for smoking, at either the individual (Benhamou *et al.* (1988) and Steenland *et al.* (1990)) or group level (Pfluger and Minder 1994). The two studies of professional drivers, a portion of which included truck drivers, found significantly elevated estimates of relative risk with smoking-adjusted ORs of 1.42 (95% C.I. = 1.07-1.89) and 1.48 (95% C.I. = 1.30-1.68) (Benhamou *et al.*, 1988 and Pfluger and Minder, 1994, respectively). The one smoking-adjusted study focusing on trucking, Steenland *et al.* (1990), found elevated relative risk estimates for several occupational and duration of employment categories; however, the only statistically significant risk estimate found was for diesel truck drivers with greater than 34 years of exposure, (OR = 1.89; 95% C.I. = 1.04-3.42).

While several population-based studies enrolled a large number of subjects overall (Williams *et al.* 1977; Milne *et al.*, 1983; Hall and Wynder, 1984; Damber and Larsson, 1987; Boffetta *et al.* 1988), the actual numbers of subjects occupationally exposed to diesel exhaust (considered here as truck drivers) were small. Of the larger, general population studies (Hayes *et al.*, 1989; Benhamou *et al.*, 1988; Boffetta *et al.*, 1990; Swanson *et al.*, 1993) and industry- or occupation-specific studies (Ahlberg *et al.*, 1981; Rafnsson and Gunnarsdottir, 1991; Guberan *et al.*, 1992; Hansen *et al.*, 1993; Pfluger and Minder, 1994; Steenland *et al.*, 1990) with greater numbers of truck drivers, significantly elevated smoking-adjusted risk estimates were limited mainly to the

case-control studies described above (Hayes *et al.*, 1989; Benhamou *et al.*, 1988; Steenland *et al.*, 1990; Swanson *et al.*, 1993; Pfluger and Minder, 1994). Although several industry-specific cohort studies found significantly elevated risks associated with truck or professional driving, with SMRs ranging between 1.33 and 2.14, all lacked smoking data.

Studies Of Lung Cancer Among Transport Workers

Table 1 summarizes the studies that have examined the lung cancer risk to truck drivers. Most studies of transportation workers are limited by small sample size, lack of smoking data, or limited follow-up. None of the three studies of London transportation workers, drivers or garage workers, (Raffle, 1957; Waller, 1981; Rushton *et al.*, 1983) obtained information on smoking. In addition, two lacked sufficient follow-up (Raffle, 1957; Rushton *et al.*, 1983), excluded retirees, or suffered from small sample size (Raffle, 1957; Waller, 1981). Of the other European studies focusing on bus company employees (Edling *et al.*, 1987; Netterström, 1988; Gustavsson *et al.*, 1990), only Gustavsson *et al.* (1990) found an elevated risk for lung cancer, with an overall SMR of 1.22 (95% C.I. = 0.71-1.96). However, in the more detailed nested case-control analysis using conditional logistic regression, estimated RRs increased with the cumulative diesel-exhaust exposure index, as noted above.

Of the three studies reporting increased risks for heavy equipment operators (Wong *et al.*, 1985; Boffetta *et al.*, 1988; Hayes *et al.*, 1989), only the RR reported by Boffetta *et al.* (1988) was statistically significant (RR = 2.6; 95% C.I. = 1.12-6.06). However, this estimate was based on only five lung cancer deaths. The large industry-specific cohort study of Wong *et al.* (1985) did not find an elevated risk for lung cancer among unionized heavy equipment operators (SMR = 0.99; 95% C.I. = 0.88-1.10). A subset of individuals retiring at age 65 did have a significantly elevated risk, but a group excess in emphysema deaths (SMR = 2.75; 95% C.I. = 2.09-3.55) and the absence of smoking data suggest that the increased risk may have been related more to tobacco use than to diesel exhaust exposure.

Table 1: Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Truck Drivers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval ^a or P-Value	Comments
Menck and Henderson, 1976 USA	Cohort Truck drivers	109	SMR 1.65	$p < 0.01$	Included 2,161 lung cancer cases identified from death certificates in white males, aged 20 to 64, from 1968 through 1970, and 1777 incident cases of lung cancer reported to LA County Cancer Surveillance Program for 1972 - 73. Occupational information obtained from death certificates or hospital admission sheets/medical records represented the last occupation and industry of employment. No data on smoking.
Decoufle <i>et al.</i> 1977 USA	Case-control Truck or tractor driver	56	OR 1.07	N.S.	Hospital-based study of 6,434 cancers cases admitted to Roswell Park Memorial Institute between 1956 and 1965. Controls were patients admitted with non-neoplastic disease. Occupation and smoking data obtained by questionnaire. Crude adjustment for smoking. Inadequate latency.
	≥ 5 years as truck, bus or taxi driver	50	0.89	N.S.	
Williams <i>et al.</i> 1977 USA	Case-control Transportation Industry	38	RR 1.17	N.S.	Study examined cancer incidence and its relation to occupation and industry based on the U.S. 3rd National Cancer Survey. The number of cases of cancer at various sites were compared with that of cases at all other sites combined. Occupational history (main and recent employment) and data on smoking were obtained by interview (n = 7,518). IARC noted the potential bias in this study due to the relatively low level of response to the questionnaire (57%). Results were controlled for tobacco use, alcohol consumption, race, education and geographic location.
	Truck drivers	22	1.52	N.S.	
	Railroad workers	12	1.40	N.S.	
	Truck Industry	13	1.34	N.S.	
Leupker and Smith, 1978 USA	Cohort Total cohort	34	SMR 1.21	N.S.	Death certificates for a 3-month period in 1976 in the Central States Teamster population were examined. Comparison group was the US male population and was not adjusted for race. No data on smoking. Authors noted the follow-up was short. Retirees and members with lapsed benefits were excluded. 48,358 members were eligible in the 50-59 age group.
	Age 50-59	not given	1.37	$p < 0.001$	
Ahlberg <i>et al.</i> 1981 Sweden	Cohort All truck drivers*	161	RR 1.33	1.13-1.56	Cohort consisted of 34,027 Swedish drivers considered to be exposed to diesel exhaust identified from the 1960 national census. Reference population consisted of blue-collar workers from the same census thought to have had no exposure to petroleum products or chemicals (n=686,708). No data on smoking; however, a study of 470 professional drivers in Stockholm found that 78% of fuel truck drivers and 31% of other truck drivers smoked compared to 40% in the Swedish population (citing unpublished study). # Subset of all non-fuel tank drivers. *Does not include fuel tank drivers.
	Stockholm truck drivers#		1.62	1.15-2.28	

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or p -values reported in original study. DE = Diesel Exhaust. OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Truck Drivers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval ^a or P-Value	Comments
Milne <i>et al.</i> 1983 USA	Case-control		OR		Study compared lung cancer deaths with mortality from all other cancers in Alameda County between 1958 and 1962 to investigate possible associations between lung cancer and occupation. Data on cause of death and occupation were obtained from death certificates. No data on smoking or the types of vehicle engines. Results reported are for males. *Results in parentheses are ORs with potential occupationally related cancer removed from the control population. Significant risk estimates only observed when compared with control group before such cancers removed.
	Occupational groups:				
	All transport operatives	36	1.3 (1.1)*	N.S.	
	Bus drivers	4	3.5	$p < 0.05^*$	
	Truck drivers	23	(2.8)*	$p < 0.05^*$	
	Other transport	7	1.6 (1.3)*	N.S.	
Hall and Wynder, 1984 USA	Industry groups:				Study consisted of 502 men with histologically confirmed primary lung cancer (20 to 80 years old) and matched control patients in 18 hospitals in six cities. Controls with tobacco-related diseases were excluded. Patients were interviewed between December 1980 and November 1982. Smoking data were obtained. Occupations were grouped either dichotomously as exposed to diesel exhaust (warehousemen, bus drivers, truck drivers, railroad workers, heavy equipment operators) or unexposed. Exposure categorization also conducted by NIOSH-based occupational classifications with job title classified as having "probable" exposure to diesel exhaust as either "high" (10 cases), "moderate" (16 cases) or "little or none" (476 cases). No significantly elevated risks were reported in this latter analysis (data not shown here). See also Boffetta <i>et al.</i> , 1990. *Compared DE exposed to unexposed within each smoking category.
	Railroad	34	0.7 (0.6)*	N.S.	
			0.8 (0.8)*		
	Case-control		OR		
	<u>Usual employment:</u>				
	Total diesel-exposed - adjusted for smoking	45	2.0 1.4	1.2-3.2 0.8-2.4	
<u>Selected occupations:</u>					
Truck drivers					
Railroad workers					
Heavy equipment repairmen & operators	22	1.4	0.7-2.6		
- adjusted for smoking	5	2.6	0.5-12.8		
	10	3.5	1.0-11.8		
		1.9	0.6-5.5		
<u>Smoking & DE exposure:</u>					
Non & ex-smokers	10	1.46*	0.9-2.3		
≤ 20 cigarettes/day	10	0.82*	0.5-1.4		
> 20 cigarettes/day	7	1.30*	0.8-2.1		

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or p -values reported in original study. DE = Diesel Exhaust. OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Truck Drivers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval ^a or P-Value	Comments
Boffetta <i>et al.</i> , 1990 USA	<u>Exposure by occupation:</u>		OR		Study consisted of 2584 histologically confirmed lung cancer cases and 5009 controls derived from 18 hospitals in six cities. Controls were patients with current non-tobacco-related diseases matched by age, hospital and year of interview. Exposure was assessed by occupational titles and self-reported exposure to diesel exhaust. Results were adjusted for smoking, education and asbestos exposure by logistic regression. Occupations were classified as having probable, possible or no diesel exhaust exposure. Exposure prevalence was low. Only 15.6% of the controls were ever in an exposed job and 6.4% were considered probably exposed. Self-reported exposure to diesel exhaust had consistently higher point estimates of risk than those based on occupational classification, suggesting the possibility of recall bias. See also Hall and Wynder, 1984. *Duration of employment data only available for 23 cases and 27 controls of all patients classified as truck drivers (114 cases and 176 controls).
	“Possible” exposure	240	0.92	0.76-1.10	
	“Probable” exposure	210	0.95	0.78-1.16	
	<u>By duration:</u>				
	“Probable” DE				
	1-15 years	4	0.52	0.15-1.86	
	16-30 years	15	0.70	0.34-1.44	
	31+ years	17	1.49	0.72-3.11	
	<u>Truck driver*</u>				
	1-15 years	4	1.83	0.31-10.73	
16-30 years	12	0.94	0.41-2.15		
30+ years	7	1.17	0.40-3.41		
<u>Self-reported exposure:</u>			1.21	0.73-2.02	
<u>By duration</u>					
1-15 years	11	0.90	0.40-1.99		
16-30 years	12	1.04	0.44-2.48		
31+ years	12	2.39	0.87-6.57		
Damber and Larsson, 1985 Sweden	Case-control		OR		Study included 604 male patients with lung cancer from the 3 most northern counties in Sweden (all new cases reported to the Swedish Cancer Registry in 1972 to 77 who had died at least one year before the start of the study in 1979). Matched controls were drawn from the national registry for causes of death. Living controls were also used. Data on occupational and smoking habits were obtained by questionnaire. Study focused on professional drivers, most of whose vehicles had diesel engines. Investigators noted that drivers had considerably higher average tobacco consumption than nondrivers. Authors stated that the study suggests a synergistic interaction between smoking and occupational exposure. See also Damber and Larsson 1987. Risk estimates presented for portion of cohort with date of birth after 1900. # Subset of all drivers. * Compared to nondrivers. ** Compared to nondrivers/nonsmokers, where “nonsmokers” included ex-smokers who had quit for at least 10 years.
	<u>By age of diagnosis:</u>				
	Professional drivers				
	<70 years	40	1.00*	0.66-1.50	
	≥70 years	23	3.15*	1.66-6.00	
	<u>Truck drivers[#]</u>				
	<70 years	22	0.83*	0.50-1.40	
	≥70 years	13	5.70*	2.22-14.67	
	<u>By age & smoking status:</u>				
	<u>Drivers/</u>				
<u>Nonsmokers**</u>					
<70 years	NG	1.9	0.5-5.5		
≥70 years	NG	4.5	1.1-16.4		
<u>Drivers/Smokers**</u>					
<70 years	NG	6.0	3.5-10.3		
≥70 years	NG	20.8	9.4-46.0		

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or *p*-values reported in original study. DE = Diesel Exhaust. OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Truck Drivers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval ^a or P-Value	Comments
Damber and Larsson, 1987 Sweden	Case-control Professional drivers Years worked ≥1 ≥20 Adjusted for smoking ≥1 ≥20	72 37 72 37	OR 1.3 1.5 1.0 1.2	0.9-1.9 0.9-2.6 0.7-1.5 0.6-2.2	Study consisted of 600 men with lung cancer in northern Sweden reported to the Swedish Cancer Registry from 1972 through 1977 and dead before the start of the study (1979). Cases were matched with both dead and living controls. Results reported here are for comparisons with dead controls. Results with living controls were in good agreement. See Damber and Larsson (1985) for study focused on professional drivers only.
Boffetta <i>et al.</i> 1988 USA	Prospective Cohort <u>Self-reported as DE:</u> All DE exposed By duration exposure: 1-15 years 16+ years DE & smoking status*: nonsmokers ex-smokers current smokers <u>Occupation:</u> Railroad worker Truck driver Heavy equipment By occupation & DE: Truck/exposed Truck/nonexposed	174 7 85 78 14 48 5 18** 18**	RR 1.18 1.05 1.21 1.73 11.06 19.82 1.59 1.24 2.60 1.22 1.19	0.97-1.44 0.80-1.39 0.94-1.56 [#] 0.60-4.95 6.27-19.53 11.20-35.07 0.94-2.69 0.93-1.66 1.12-6.06 0.77-1.95 0.74-1.89	Included 461,981 males, aged 40 to 79, participating in the American Cancer Society's Prospective Mortality Study in 1982. Follow-up for two years. Exposure assessment was based on self-reported (questionnaire) occupation and diesel exhaust exposure. Investigators stated that, although the sample was large, it was comprised of volunteers, who were healthier and were less frequently exposed to important risk factors such as smoking and alcohol. Reference population included men with no reported exposure or likely occupational exposure to diesel exhaust. Results were adjusted for smoking and other occupational exposures (asbestos, coal and stone dust, coal tar pitch, and gas exhaust). See Hall and Wynder, 1984. *Smoking data not available for all subjects. **Diesel exhaust exposure data not available for all truck drivers. [#] Test for trend reported by investigators as 0.05 < p < 0.10.
Benhamou <i>et al.</i> 1988 France	Case-control Motor vehicle mechanic Transport equipment operators Professional drivers	65 157 128	RR 1.06 1.35 1.42	0.73-1.54 1.05-1.75 1.07-1.89	Study consisted of 1,334 histologically confirmed lung cancer cases and 2,409 controls matched on sex, age, hospital admission and interviewer. Study was conducted between 1976 and 1980. Results were adjusted for smoking and are limited to males. Occupation was determined by questionnaire (interview). The types of motor vehicle engines worked with were not specified. No evidence of increased risk with increased duration of exposure (years employed).

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or p-values reported in original study. DE = Diesel Exhaust. OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Truck Drivers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval ^a or P-Value	Comments
Hayes <i>et al.</i> 1989 USA	Case-control Pooled Analysis Truck Drivers < 10 yrs employed ≥ 10 yrs employed Heavy Equipment < 10 yrs employed ≥ 10 yrs employed Bus Drivers < 10 yrs employed ≥ 10 yrs employed	161 112 7 10 23 24	OR 1.0 1.5 1.5 2.1 1.1 1.7	0.8-1.3 1.1-2.0 0.4-5.3 0.6-7.1 0.6-2.1 0.8-3.4	The study is a pooled analysis of three case-control studies conducted between 1976 and 1983 in Florida, New Jersey, and Louisiana. Total eligible cases = 2,291 and controls = 2,570. All occupational data were recoded from original interviews. No specific information regarding diesel exposure or engine type. ORs were adjusted for birth cohort (<1910, 1910-19, 1920-29, 1930+), usual daily cigarette use, and state.
Steenland <i>et al.</i> 1990 USA	Case-control <u>Occupation data:</u> 1) Teamster records data Long-haul driver Short-haul driver 2) Next-of-kin data Truck driver, diesel Truck driver, gasoline Truck driver, both <u>Duration employment after 1959*:</u> 1) Teamster records data Long-haul driver 1-11 years 12-17 years ≥18 years 2) Next-of-kin data Diesel truck driver 1-24 years 25-34 years ≥35 years	162 228 213 48 72 56	OR 1.27 1.31 1.42 1.22 1.25 1.08 1.41 1.55 1.27 1.26 1.89	0.83-1.93 0.81-2.11 0.89-2.26 0.79-1.88 0.81-1.95 0.68-1.70 0.90-2.21 0.97-2.47 0.70-2.27 0.74-2.16 1.04-3.42	Study consisted of 1,086 lung cancer cases and 1,085 controls among truck drivers in the Central States Teamsters Union. Information on work history was obtained from next of kin and union records. Subjects died in 1982-83 after applying for pensions, which required at least 20 years of union membership. Subjects were classified according to the job category in which they worked the longest. Union data provided no information on the type of truck driven. 90% of union long-haul drivers were also identified as diesel truck drivers by next of kin. Results were adjusted for smoking and asbestos exposure. Smoking data obtained by next-of-kin interview used in both types of exposure classification. Steenland <i>et al.</i> (1992) summarized results from a recent industrial hygiene survey of exposure to diesel exhaust in the trucking industry, and found that elemental carbon measurements were generally consistent with the results; i.e., mechanics had the highest exposure and the highest risks, followed by long-haul and local drivers. Authors noted that exposure to asbestos may account for some of the observed effects in mechanics, but its confounding effect was probably small. Study results for truck mechanics and dock workers were elevated but not significant. *Study also presented risk estimates for duration of employment inclusive of the pre-1959 work era for both job ascertainment categories and for majority of job classifications.

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or *p*-values reported in original study. DE = Diesel Exhaust. OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Truck Drivers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval ^a or P-Value	Comments
Burns and Swanson 1991 USA	Case-control		OR		Occupational and smoking histories were obtained by telephone interview for 5,935 incident lung cancer cases and 3,956 incident colon and rectal cancer controls diagnosed between 1984 and 1987 and reported to the Detroit cancer registry. The smoking- and race-adjusted OR for all drivers (238 cases, 86 controls) was 1.88 (95% C.I. = 1.37-2.58), while drivers of "heavy trucks" (166 cases, 48 controls), maintained a higher risk even after adjustment for smoking, OR = 2.31 (95% C.I. = 1.56-3.42). Mechanics also had a significantly elevated OR for lung cancer (OR = 1.72, 95% C.I. = 1.15-2.59). The types of the vehicle engines were not specified. Results were adjusted for smoking. See Swanson <i>et al.</i> 1993.
	Drivers (white)	187	2.40	1.65-3.48	
	All drivers (race adj.)	238	1.88	1.37-2.58	
	Railroad workers	14	1.27	0.45-3.53	
Swanson <i>et al.</i> 1993 USA	Case-control		OR		Cases and controls were from OCISS (see Burns and Swanson, 1991 for description of subjects). Incident lung cancer cases among black and white males, aged 40 to 84, from 1984 through 1987 are included in this report. Controls were colon and rectal cancer cases. Information on occupation, smoking, medical history were obtained by telephone interview. Results were adjusted for age at diagnosis, race and smoking. *Test for trend $p \leq 0.05$.
	Occupation and duration:				
	1) White males				
	Heavy truck drivers				
	0 years	88	1.0	Reference	
	1-9 years	78	1.4	0.8-2.4*	
	10-19 years	38	1.6	0.8-3.5*	
	20+ years	121	2.5	1.1-4.4*	
	Light truck drivers				
	0 years	88	1.0	Reference	
	1-9 years	46	1.7	0.9-3.3	
	10+ years	36	2.1	0.9-4.6	
	Railroad workers				
	0 years	73	1.0	Reference	
	1-9 years	27	1.2	0.5-2.7	
	10+ years	40	2.4	1.1-5.1	
	2) Black males				
Heavy truck drivers					
0 years	12	1.0	Reference		
1-9 years	27	2.7	0.8-9.2		
10-19 years	16	1.9	0.5-7.2		
20+ years	16	2.1	0.5-9.2		
Railroad workers					
0 years	15	1.0	Reference		
1-9 years	22	2.6	0.8-7.9		
10+ years	9	2.7	0.6-12.1		

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or *p*-values reported in original study. DE = Diesel Exhaust. OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Truck Drivers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval ^a or P-Value	Comments
Rafnsson and Gunnarsdottir 1991 Iceland	Cohort Truck drivers Duration employment: <2 years 2-10 years 11-30 years >30 years	24	SMR 2.14 2.70 2.46 0.68 2.32	1.37-3.18 0.74-6.92 0.99-5.08 0.01-3.76 0.85-5.04	Cohort consisted of truck and taxi drivers in Reykjavik followed from 1951 to 1988. National mortality rates were used as for comparison. Information on truck drivers was obtained from their union. No data on smoking or type of vehicle engines used. No trend of increased risk with increased follow-up time was observed.
Guberan <i>et al.</i> 1992 Switzerland	Cohort Professional drivers	77	SMR 1.50	1.23-1.81	Cohort identified from vehicle license records of professional drivers required to obtain special license during the period from 1949 to 1961. Excluding individuals born prior to 1900, 1,726 drivers were eligible. Lung cancer cases identified from death and tumor registries through 1986. No smoking data obtained. Approximately 1/3 to 1/4 of professional drivers were reported to be long-haul truck drivers. Death rates compared to regional mortality rates. A significant ($p < 0.02$) upward trend in lung cancer mortality with time from first exposure was also observed: SMRs = 0.67, 1.18, 1.30, 1.35, and 2.59 for 0-14, 15-24, 25-34, 35-44, and ≥ 45 years, respectively (no confidence intervals reported).
Hansen 1993 Denmark	Cohort Age on Nov. 9, 1970 15-29 30-39 40-44 45-49 50-54 55-59 60-64 65-74 Total	0 3 3 11 12 19 22 6 76	SMR 1.96 0.56 1.17 1.10 2.29 2.27 2.60 1.60	 0.40-5.73 0.12-1.64 0.58-2.09 0.57-1.93 1.38-3.58 1.42-3.44 0.95-5.65 1.26-2.00	Cohort consisted of 14,225 truck drivers followed for a 10-year period. Comparisons were made with another cohort of unskilled laborers. Members of the cohort were identified from the file of a nationwide census conducted in 1970. Self-reported occupation, trade, industry and employment on the day of the census were recorded. The study was comprised of unskilled male laborers 15 to 74 years old who were occupationally active on the day of the census. 627 truck drivers and 3,811 members of the control cohort died within the 10 years. No data on smoking. Diesel engines have comprised most of Danish fleet of trucks since the late 1940s.
Pfluger and Minder, 1994 Switzerland	Case-control Professional drivers - smoking adjusted	284	OR 2.27 1.48	1.99-2.58 1.30-1.68	Mortality of Swiss professional drivers (truck, bus and taxi drivers) was determined from death certificates and compared to census data to obtain occupation and age-specific death rates. No individual smoking data were available, but an indirect adjustment was conducted based on occupation specific mortality rates.

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or p -values reported in original study. DE = Diesel Exhaust. OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Transport (i.e., bus) and Equipment Workers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval ^a or P-Value	Comments
Raffle 1957 England	Cohort Overall Bus & trolley drivers Age 55-64	96 30	SMR 1.4	 N.S.	Cohort consisted of deaths, retirements and transfers due to lung cancer in London transport employees (bus and trolley workers, bus engineers), aged 45 to 64 years, in jobs with presumably different exposures to exhaust fumes in 1950 to 1954. Only cases arising during exposure employment were considered. Rates were compared to lung cancer mortality in other company employees. Diesel buses had been gradually introduced since the 1930s. At the end of WWII only 15% of the buses still used petrol. All had been replaced by 1950. Consequently, the duration of exposure of some workers to DE might have been short. No data on smoking. See also Waller 1981.
Waller 1981 England	Cohort All workers Bus drivers Bus conductors Engineers, garages Engineers, central works Motormen and guards	667 259 130 177 42 59	SMR 0.79 0.75 0.75 0.90 0.66 0.87	 NP NP NP NP NP	Cohort consisted of lung cancer deaths and retirements or transfers due to lung cancer in men, aged 45 to 64, employed within five categories of London Transport employees. Mortality was compared to men in Greater London. The study covered 25 years ending in 1974, thus including some of the data described by Raffle (1957). No data on smoking. Those who retired at age 65 or left earlier were not followed up, thus limiting the extent of case ascertainment.
Rushton <i>et al.</i> 1983 England	Cohort	102	SMR 1.01	 $p = 0.94$	Cohort consisted of 8,684 men employed as maintenance workers in 71 bus garages in London for at least one year from 1967 to 1975. Follow-up through 1975. No data on smoking. Authors noted short follow-up period (average of 6 years). Lung cancer mortality was compared with the male population of England and Wales. The all-cause mortality was significantly lower than expected based on London residence.
Buiatti <i>et al.</i> 1985 Italy	Case-control Transportation Taxi driving Train conductors	45 20 7	OR 1.1 1.8 1.4	 0.7-1.6 1.0-3.4 0.5-3.9	Study consisted of 340 confirmed cases in males (and 817 controls) in Florence, diagnosed from 1981 through 1983 in the regional general hospital and a referral center for lung cancer. Controls were matched on sex, age, date of admission and smoking, and were from the same hospital. Diesel exhaust exposure was assessed by questionnaire for all jobs held for more than one year.

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or *p*-values reported in original study. DE = Diesel Exhaust, OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio, NP = not presented.

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Transport (i.e. bus) and Equipment Workers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval ^a or P-Value	Comments
Wong <i>et al.</i> 1985 USA	Cohort Total <u>By Duration</u> <5 years 5-9 years 10-14 years 15-19 years ≥20 years All retired members Normal retired members	309 10 25 53 58 163 155 86	SMR 0.99 0.45 0.75 1.08 1.02 1.07 1.64* 1.30**	0.88-1.10 N.S. N.S. N.S. N.S. $p = 0.05$ $p < 0.01$ $p < 0.05$	Cohort consisted of 34,156 male members of a heavy construction equipment operators union for at least one year from 1964 through 1978. Mortality experience was compared with that of the US white male population. Partial work history was available for some cohort members through the union. A random sample of union members was surveyed to determine smoking habits, and no significant difference between members and the general population was found. Work groups evaluated were considered to have high exposure to diesel exhaust (scraper operator, bulldozer operator, backhoe operator and loader operator) or low exposure (mechanical maintenance workers and engineers). Overall mortality in the cohort was less than that in the U.S. male population (SMR 0.81, 95% C.I. 0.79-0.84). Workers were also categorized by job title and potential exposure, but no significant risks were observed. Analysis of retirees found an excess risk for lung cancer* and emphysema. *Includes also retirements due to ill health. *Normal retirees are those workers retired at or over 65 and early retirees who reached 65.
Edling <i>et al.</i> 1987 Sweden	Cohort Bus company employees Bus drivers Bus garage workers Clerks	6 5 1 0	SMR 0.67 0.69	Not presented	Cohort consisted of 694 bus garage employees followed from 1951 through 1983. Men were divided into three exposure categories (clerks, bus drivers and bus garage workers). Clerks were assumed to have had the lowest exposure to diesel exhaust and bus garage workers the highest. Authors stated that the power of the study to detect specific cancers was limited. No data on smoking.
Netterstrom 1988 Denmark	Cohort Bus drivers	15	SMR 0.87	0.48-1.43	Cohort of 2,465 Danish bus drivers from three companies during the period 1978 to 1984. Cases were identified through death and cancer registries. Death rates were compared with national rates. No data on smoking were available. Mean value for employment duration among the lung cancer cases was 30 years

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or p -values reported in original study. DE = Diesel Exhaust, OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Transport (i.e. bus) and Equipment Workers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval ^a or P-Value	Comments
Gustavsson <i>et al.</i> 1990 Sweden	Cohort Total (deaths) DE exposure index: 0-10* 10-30 >30 Nested case-control (20 incident cases) 0-10* 10-20 20-30 >30	17 5 5 7 5 2 3 10	SMR 1.22 0.97 1.52 1.27 RR 1.0 1.34 1.81 2.43	0.71-1.96 Reference 1.09-1.64 1.20-2.71 1.32-4.47	Cohort consisted of 695 bus garage workers employed as mechanics, servicemen or hostlers for at least six months in five bus garages in Stockholm between 1945 and 1970. A nested case-control study was performed within the cohort. Follow-up was through 1986. No data on smoking although no large variation in smoking habits was expected within the cohort. Exposure to diesel exhaust and asbestos were assessed based on time period-specific data on job tasks. Lung cancer cases were identified through tumor and death registries. In the cohort analysis regional rates were used for comparison. *Cumulative exposure index values (unitless).
Gustafsson <i>et al.</i> 1986 Sweden	Cohort Deaths Incident cases	 71 89	SMR 1.29 SIR 1.53	 1.02-1.63 1.24-1.80	Cohort consisted of 6,071 Swedish dockworkers first employed before 1974 for at least six months. The group was followed from January 1961 through January 1981. Cancer morbidity was determined among 6,071 dockworkers who had been alive and without cancer in January 1961. Comparison group was Swedish male population. Diesel trucks were introduced into Swedish ports in the late 1950s and became prevalent during the 1960s. No data on smoking. See Emmelin <i>et al.</i> (1993) for results from the follow-up study. Employment as a dockworker was the only information on diesel exhaust exposure used in the analysis.

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or *p*-values reported in original study. DE = Diesel Exhaust, OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Transport (i.e. bus) and Equipment Workers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval ^a or P-Value	Comments
Emmelin <i>et al.</i> 1993 Sweden	Case-control		OR		Study was a nested case-control of lung cancer among Swedish male dockworkers in the cohort studied by Gustafsson <i>et al.</i> (1986). 154 referents were matched to 50 cases on port and date of birth. Indices of exposure to diesel exposure were derived from employment records and records of annual fuel consumption by diesel vehicles. Three different exposure classifications were created: "machine time", "fuel consumption" and "exposed time". Information on smoking was obtained from questionnaires and interviews with foremen or workers who had worked with subjects. Response rate for mailed questionnaires was low (67%) but information from the interviews was available for 95% of the subjects. Some ex-smokers were classified as never smokers. No exposure level ("low", "medium", or "high") was significant for any DE exposure scheme (only "high" strata reported here). Comparisons based on exposure and smoking tended to find more elevated risks. Investigators noted that the increase in the OR for both smoking and exhaust exposure indicate that smoking does not explain the results from the exposure-only models, and that there may be an interaction between smoking and exhaust exposure. No information on asbestos exposure, which was said to have decreased by the 1970s. See also Gustafsson <i>et al.</i> (1986). * "Low" exposure category used for reference comparison. **Note: authors reported confidence intervals at 90% level.
	<u>Exposure variable:</u> Machine time high*	14	1.3	0.3-5.6**	
	Fuel consumption high*	15	1.7	0.5-5.9**	
	Exposed time high*	19	2.9	0.8-10.7**	
	<u>Exposure & Smoking:</u> Machine time medium		1.8	0.5-6.6**	
	high		2.9	0.6-14.4**	
	smoker		5.7	2.4-13.3**	
	Fuel consumption medium		1.5	0.5-4.8**	
	high		2.9	0.7-11.5**	
	smoker		5.5	2.4-12.7**	
	Exposed time medium		2.7	0.6-11.3**	
	high		6.8	1.3-34.9**	
	smoker		6.2	2.6-14.6**	
Kaplan 1959 USA	Cohort		SMR		Cohort consisted of 6,506 deaths among railroad workers from the Baltimore and Ohio Railroad Relief Department between 1953 and 1958. Subjects were categorized into 3 groups by exposed to diesel exhaust and compared with national lung cancer mortality rates. IARC noted that since the changeover to diesel engines began in 1935 and was 95% completed by 1959 (Garshick <i>et al.</i> 1988), few, if any, of the lung cancer deaths could have occurred in workers with more than 10 years of exposure to diesel exhaust. No data on smoking.
	Total	154	0.80	0.68-0.94	
	Most likely exposed	49	0.875	N.S.	

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or *p*-values reported in original study. DE = Diesel Exhaust, OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer Studies Among Railroad Workers

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence ^a Interval or P-Value	Comments
Garshick <i>et al.</i> 1988 USA	Cohort	1694	RR		Cohort consisted of 55,407 white male railroad workers aged 40-64 exposed to little or no asbestos who had started work between 1939 and 1949 and had worked 10 to 20 years after 1959. Follow-up through 1980. Industrial hygiene data were used to categorize jobs as exposed or unexposed. No data on smoking; however, authors noted that there was no difference in smoking habits by job title in comparison studies of current workers (see Garshick <i>et al.</i> 1987). Diesel exhaust exposure in the US railroad industry occurred after WWII. The approximate midpoint of dieselization was in 1952 and by 1959, 95% of the locomotives were diesel-powered. Workers aged 40 to 44 in 1959 were the group with the longest possible duration of exposure. Most workers with potential asbestos exposure were excluded, though some did have potential exposure to asbestos (shopworkers and hostlers). Analyses were done with and without these groups. Exposure was assessed from samples of respirable dust taken in 1980s (Woskie <i>et al.</i> 1988a). Mean exposure levels suggested a five-fold range of exposure between clerks and shopworkers (Woskie <i>et al.</i> 1988b). These values confirmed the assignment of categories of diesel exhaust exposure in the present study and Garshick <i>et al.</i> 1987. * Excluding exposure to diesel exhaust over the 4 years preceding the year of death
	<u>By Age in 1959 w/ DE:</u>				
	40-44		1.45	1.11-1.89	
	45-49		1.33	1.03-1.73	
	50-54		1.12	0.88-1.42	
	55-59		1.18	0.94-1.50	
	60-64		0.99	0.74-1.33	
	Minus those w/ asbestos exposure				
	40-44		1.57	1.19-2.06	
	45-49		1.34	1.02-1.76	
	<u>By Years DE Exposure:*</u>				
	1-4 years		1.20	1.01-1.44	
5-9 years	1.24	1.06-1.44			
10-14 years	1.32	1.13-1.56			
≥ 15 years	1.72	1.27-2.33			
Minus those w/ asbestos exposure					
1-4 years	1.34	1.08-1.65			
5-9 years	1.33	1.12-1.58			
10-14 years	1.33	1.10-1.60			
≥ 15 years	1.82	1.30-2.55			
Nokso-Koivisto and Pukkula, 1994 Finland	Cohort Total	236	SIR 0.86	0.75 – 0.97	Cohort consisted of 8,391 members of the Finnish Locomotive Drivers' Association from 1953 to 1991 (including retirees). Information was not available for 302 members. No smoking data were available. The overall incidence for all cancer sites was lower than expected when compared to national rates (SIR = 0.95).

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or *p*-values reported in original study. DE = Diesel Exhaust, OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer
(Additional Studies Other Than Those Listed In Above Categories)

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence Interval or P-Value	Comments
Wegman and Peters, 1978 USA	Case-control Total study Transportation equipment operatives	91	OR		Tumor registry-based study of oat cell carcinoma during 1965 to 1972. Cancer controls identified from same registry. Smoking data collected but not used in analysis (94% cases and 78% controls smoked). Two methods used to classify occupation, registry-derived or combination of registry and next-of-kin questionnaire data. Number of cases classified as transportation equipment operatives decreased from 8 to 5 between two methods.
	- Registry derived - Combination w/ registry data	8 5	8.67 1.26	NP NP	
Coggon <i>et al.</i> 1984 England	Case-control Total DE exposed High DE exposure	 172 32	RR 1.3 1.1	 1.0-1.6 0.7-1.8	Study included all men 40 years of age in England and Wales who had died of tracheobronchial cancer from 1975 through 1979. A job exposure matrix was constructed in which occupations were grouped according to likely exposure to each of nine known or putative carcinogens. Occupational information abstracted from the death certificates. No information on smoking. IARC noted the limitations of information on death certificates, the young age of the subjects, short exposure and latency times, and the lack of data on smoking and other potential confounders.
Lerchen <i>et al.</i> 1987 USA	Case-control Diesel exhaust fumes - adjusted for smoking Diesel engine mechanics - adjusted for smoking	 7 5	OR 0.6 1.0	 0.2 – 1.6 0.2 – 2.0	Population-based case-control study of 506 patients diagnosed between January 1980 and December 31, 1982, and reported to the New Mexico tumor registry (333 males and 173 females). Data on lifetime occupation and smoking were obtained by personal interview and self-reported history of exposure to specific agents. Matched controls were selected randomly from the telephone directory or for persons over 65 from the roster of participants in a health insurance plan. Only seven males reported exposure to diesel exhaust.
Magnani <i>et al.</i> 1988 England	Cohort All DE exposure	 NP	SMR 1.07	 1.04 – 1.10	General population-based cohort analysis of death certificate and census survey information on 31,925 men with lung cancer between 1970-72. No smoking data were available. A job-exposure matrix was developed for several potential carcinogens, including diesel exhaust.

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or *p*-values reported in original study. NP = not presented. DE = Diesel Exhaust, OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Table 1 (continued): Epidemiological Studies of Exposure to Diesel Exhaust and Lung Cancer (Additional Studies Other Than Those Listed In Above Categories).

Reference	Study Design, Population, and Exposures	Cases or deaths	Effect Measure	Confidence ^a Interval or P-Value	Comments
Siemiatycki <i>et al.</i> 1988 Canada	Case-control Lung cell types among DE exposed: Oat cell Squamous cell Adenocarcinoma Other Total DE-exposed occupations minus mining:	34 81 28 34 177 70	OR 1.1 1.2 0.9 1.0 1.1	 0.8-1.5** 1.0-1.5** 0.6-1.2** 0.8-1.4** 0.8-1.5**	This population-based case-control study provided information on the association between several cancer types and 10 types of exhaust and combustion products. Interviews were carried out for 3,726 cancer patients, aged 35 to 70, diagnosed in any of 19 participating Montreal area hospitals. Each type of cancer was a case series; reference groups were selected from among the other cancer patients interviewed. Results reported are adjusted for smoking, socioeconomic status, ethnic group and several other potential confounders. Authors noted that the excess lung cancers were concentrated among mine and quarry workers. **Authors reported 90% confidence intervals.
Bender <i>et al.</i> 1989 USA	Cohort State highway workers	NP	SMR 0.69	 0.52 – 0.90	Cohort consisted of Minnesota highway workers employed for a minimum of one year and working at least one day after January 1, 1945. Mortality was compared to state rates. No data were available on smoking. Overall mortality was significantly lower than the expected, SMR = 0.83 (95% C.I. = 0.73-0.94).
Kauppinen <i>et al.</i> , 1993 Finland	Case-control Engine exhaust exposure: Any exposure ≥ 1 month 1 month - 5 years > 5 years	8 5 3	OR 1.7 0.39 2.21	 0.55-5.20** 0.05-2.94** 0.65-7.48**	Nested case-control study of woodworkers in Finland consisted of 136 lung cancer cases diagnosed between 1957 to 1982 and 408 matched controls. Original cohort consisted of 7,307 workers from 35 factories. Multiple chemical exposures were analyzed for, including engine exhaust (combination of diesel and gasoline engines). Smoking, age, and other chemical exposures were adjusted for; however, only a small number of individuals were categorized as having been exposed to engine exhaust. **Authors reported 90% confidence intervals.

^a 95% Confidence intervals unless noted. N.S.= Not significant. No confidence intervals or *p*-values reported in original study. NP = not presented. DE = Diesel Exhaust, OR = Odds Ratio, RR = Relative Risk, SIR = Standardized Incidence Ratio, SMR = Standardized Mortality Ratio

Animal Studies

Section 6.1 (Animal Studies) of the diesel exhaust TAC document (OEHHA, 1998) describes the results of diesel exhaust inhalation carcinogenicity bioassays performed using mice, rats, hamsters and monkeys. The studies in rats provided the only clear and unequivocal evidence of diesel exhaust-induced carcinogenicity in animals.

The results of eleven animal cancer bioassays of inhalation of diesel exhaust alone were available at the time the document entitled *Health Risk Assessment For Diesel Exhaust* was written for the Toxic Air Contaminant (TAC) program (OEHHA, 1998). None of the four studies with either (a) exposure periods of less than 7 hours/day, 5 days/week for 24 months or (b) particulate exposure concentrations of less than 2.2 mg/m³ (Karagianes *et al.*, 1981; White *et al.*, 1983; Lewis *et al.*, 1986, 1989; Takemoto *et al.*, 1986) gave positive results for carcinogenesis of diesel exhaust. The seven studies that presented positive results are as follows: Brightwell *et al.*, 1986, 1989; Heinrich *et al.*, 1986; Ishinishi *et al.*, 1986a; Iwai 1986; Mauderly *et al.*, 1987a; Heinrich *et al.*, 1995; Nikula *et al.*, 1995. Results of these studies are described in detail in the diesel exhaust TAC document (OEHHA, 1998).

IV. DERIVATION OF CANCER POTENCY

Basis for Cancer Potency

The diesel exhaust TAC document (OEHHA, 1998) stated that the results of the epidemiological analyses described above are consistent with a positive association between occupational exposure to diesel exhaust and an increased risk of developing lung cancer. The diesel exhaust TAC document reviewed the evidence for causality in the association between diesel exhaust and cancer of the lung. The following criteria for causal inference were considered: (1) the consistency of the findings; (2) the strength of the associations; (3) the possibility that findings are due to bias; (4) the likelihood that findings are due to chance; (5) evidence for exposure-response relationships; (6) temporality of the associations; and (7) biological plausibility of a causal association.

Chapter 6 of the diesel exhaust TAC document provided evidence consistent with a causal relationship between occupational diesel exhaust exposure and lung cancer. A lengthy discussion of causal inference, including the strengths and limitations of the underlying data, can be found in Section 6.2.4 of that document. The key findings relating lung cancer and occupational exposure to diesel exhaust are as follows: the majority of studies examining the diesel exhaust-lung cancer association have reported elevated estimates of relative risk, many of which are statistically significant. The consistency of these findings is unlikely to be due to chance. Moreover, with the possible exception of some studies that did not take smoking into account, the results are unlikely to be explained by confounding or bias. This is reinforced by the results of a meta-analysis undertaken by OEHHA staff (summarized below, and presented in detail in Appendix C of the diesel TAC document (OEHHA, 1998)), in which statistically significant pooled estimates of relative risk persisted through numerous subset and sensitivity analyses. The most important potential confounder is cigarette smoking, which was measured

and controlled for in multiple studies: in the meta-analysis the pooled relative risk estimate for studies that adjusted for smoking was 1.43 (95% C.I. = 1.31-1.57). In addition, several studies provide evidence of exposure-response relationships. The strength of the associations reported is typically within the range considered “weak” in epidemiology (i.e., estimates of relative risk between 1 and 2); nonetheless, this is not a bar to causal inference as long as other criteria are met, as discussed in Section 6.2.4 of the diesel exhaust TAC document. The temporal relationship between exposures and lung cancer is consistent with a causal relationship.

Additionally, the basic hypothesis -- that occupational exposure to diesel exhaust causes human lung cancer -- is highly plausible biologically. The evidence can be briefly summarized as follows: (1) Diesel exhaust has been shown to induce lung and other cancers in laboratory animal studies (Brightwell *et al.* 1989; Heinrich *et al.* 1986a; Iwai *et al.* 1986; Mauderly *et al.* 1987a); (2) Diesel exhaust has been shown to contain highly mutagenic substances, including polycyclic aromatic hydrocarbons and nitroaromatic compounds (Ball *et al.*, 1990; Gallagher *et al.*, 1993; Nielsen *et al.*, 1996; Sera *et al.*, 1994); (3) Diesel exhaust contains many substances which occur in recognized complex mixtures of human respiratory carcinogens, including cigarette smoke and coke oven emissions (IARC, 1989); and (4) Diesel exhaust contains known and probable human carcinogens.

Therefore, a reasonable and very likely explanation for the increased risks of lung cancer observed in the occupational epidemiological studies is a causal association between diesel exhaust exposure and lung cancer.

Results based on the human data and those based on the animal data are both subject to uncertainty. The principal uncertainties in using the rat data are their application to humans in terms of response, the choice of dose-response model to extrapolate the risk to environmental concentrations, the presence or absence of a threshold for response, and the range of dose extrapolation involved. While there are issues surrounding the quantitation of worker exposure to diesel exhaust, the uncertainty of extrapolating from one species (rat) to another (human) is avoided by using the epidemiological data to estimate risk to humans from diesel exhaust exposure. OEHHA preferred, on balance, to use the epidemiological data in order to estimate risk to humans from diesel exhaust exposure. Therefore, only the unit risk estimates based on human data were included in the final range of cancer unit risks associated with exposure to particulate matter from diesel-fueled engines in the diesel exhaust TAC document (OEHHA, 1998). OEHHA included quantitative risk assessment data based on rat studies in Appendix G of the diesel exhaust TAC document (OEHHA, 1998) for informational purposes.

Quantitative Meta-Analysis on the Relationship of Occupational Exposure to Diesel Exhaust and Lung Cancer

A meta-analysis was conducted to summarize and help interpret the published reports examining the relationship of lung cancer and exposure to diesel exhaust (OEHHA, 1998). A meta-analysis systematically combines the results of previous studies in order to generate a quantitative summary of a body of research and to examine the sources of variability among studies (for review see Petitti, 1994). The variability, or heterogeneity, of results among studies may exist due to numerous factors, including differences in study design, exposures experienced

by study subjects, methods and accuracy of exposure ascertainment, length of follow-up, and control of confounders (such as smoking).

As described in OEHHA (1998), 30 studies, contributing a total of 39 effect estimates, were utilized in the meta-analysis. The pooled relative risks for lung cancer from all 39 risk estimates combined varied with the statistical model used, 1.04 (95% C.I. = 1.02-1.06) under the fixed-effects model and 1.33 (95% C.I. = 1.21-1.46) with the random-effects model. However, significant evidence of heterogeneity was found (DerSimonian and Laird Q-statistic = 214.59, 38 d.f., $p < 0.001$). Heterogeneity in this context refers to large between-study variability. The presence of heterogeneity undermines the validity of the pooled estimates, and suggests the need for additional analysis to identify the sources of heterogeneity. As discussed in detail in Appendix C of OEHHA (1998), this involved deriving pooled estimates for a variety of subsets of the reports.

Through subset analysis, several factors were identified which strongly influenced both the magnitude and the degree of heterogeneity of the pooled risk estimates: (1) whether or not a study adjusted for smoking, (2) study design (3) the exposure assessment, as developed from occupational categories, (4) the presence of selection bias, as manifested by an observed “healthy worker effect”, and other study characteristics (See Appendix C of OEHHA (1998)). By stratifying the meta-analysis on whether the risk estimates accounted for smoking, the effect of failure to control for this exposure on the pooled estimate became readily apparent. Not only did the positive association between diesel-exhaust exposure and lung cancer persist, but the pooled risk estimate increased to 1.43 (95% C.I. = 1.31-1.57, random-effects model) with little evidence of heterogeneity among the 12 studies controlling for smoking.

The case-control studies (15 included in the meta-analysis) gave a summary estimate of 1.44 (95% C.I. = 1.33-1.56), again with little evidence of heterogeneity, while the estimate based on the results of the cohort studies remained heterogeneous. The lower pooled RR estimate and substantial heterogeneity obtained from the cohort subanalysis was probably due at least in part to failure to adjust for smoking, as only one of sixteen cohort studies controlled for this confounder, while most case-control studies did (11 of 14 studies, accounting for 17 of the 20 case-control risk estimates).

The “healthy worker effect” (HWE - here based on significantly lower than expected all-cause mortality) is a manifestation of selection bias related to hiring and retention of workers who are typically healthier than the general population, resulting in spuriously lower risk estimates for a variety of illnesses, including those potentially related to occupational exposures. Subsetting the cohort studies into those with and those without an obvious healthy worker effect markedly reduced the degree of heterogeneity in the group without the HWE (Q-statistic = 11.190, 9 d.f., $p = 0.27$), and produced an increase in the magnitude of the pooled relative risk (RR = 1.52, 95% C.I. = 1.36-1.71-1.78, random-effects model). In contrast, those studies whose results were characterized by the presence of a HWE continued to show substantial heterogeneity, and the pooled risk estimates declined. Thus, selection bias is likely to have played a role in the heterogeneity observed among the cohort studies. Selection bias results from choosing a study sample that is not representative of the entire population that could have been studied, and can distort the measure of effect (e.g., relative risk or odds ratio) (Rothman, 1986). With respect to

exposure assessment, statistically significant pooled estimates of elevated risk lacking evidence of heterogeneity were identified in several occupational subgroup analyses, both with and without additional stratification for smoking. Prior to stratifying by adjustment for smoking, the occupational subgroups involving trucking (pooled RR = 1.47, 95% C.I. = 1.33-1.63), the railroad industry (random-effects pooled RR = 1.45, 95% C.I. = 1.08-1.93), mechanics and garage workers (random-effects pooled RR = 1.35 (95% C.I. = 1.03-1.78), general transportation and professional drivers (random-effects pooled RR = 1.45, 95% C.I. = 1.31-1.60) gave risk estimates greater than the overall pooled risk estimate. The pooled RR estimates for trucking and general transportation and professional drivers showed little to no evidence of heterogeneity; however, estimates for the railroad industry demonstrated considerable heterogeneity (Q statistic = 30.90, $p < 0.001$).

Further stratification of the occupational subgroup analysis by adjustment for smoking produced a large impact on the pooled risk estimates, with all smoking-adjusted subgroup estimates displaying little evidence of heterogeneity and leading to increased risk estimates in all but one of the occupational categories. Pooled risk estimates by occupation in smoking-adjusted studies showed little evidence of heterogeneity for several occupations under both models, including truck drivers (random-effects pooled RR = 1.53, 95% C.I. = 1.20-1.94), railroad workers (random-effects pooled RR = 1.68, 95% C.I. = 1.28-2.19), and diesel mechanics and garage workers (random-effects pooled RR = 1.25, 95% C.I. = 0.87-1.80). The pooled estimates for the heavy equipment operators and dock workers and for the railroad industry studies adjusting for smoking displayed the most dramatic changes relative to the occupational analysis without smoking stratification. Among the former subgroup, the pooled risk estimate changed from 1.28 (random-effects model, 95% C.I. = 0.99-1.66) to 2.43 (95% C.I. = 1.21-4.88). Among the railroad industry studies, the pooled risk estimate also increased substantially (from 1.45 to 1.68, 95% C.I. = 1.28-2.19). In both subgroups, the pooled smoking-adjusted estimates showed little evidence of heterogeneity, though these estimates were based on two studies in the former instance and three in the latter. However, the other two heavy equipment operator and dock worker studies and the other three railroad industry studies that were not adjusted for smoking still displayed evidence of heterogeneity (Q-statistics = 2.933, 1 d.f., $p = 0.09$, and 21.517, 2 d.f., $p < 0.001$, respectively).

The meta-analysis also identified evidence of exposure-response relationships in the subgroup analyses based on duration of employment. However, as noted in OEHHA (1998), this analysis was hampered by the absence of duration-specific risk estimates in approximately one-half the studies. While the initial analysis conducted on all the included studies resulted in elevated pooled risk estimates for strata in which exposure durations were greater than 10 years relative to those with less than 10 years of exposure or for which the exposure durations were not clear from the published reports, there was still significant evidence of heterogeneity for several of the duration strata. In contrast, the analysis utilizing only estimates from the smoking-adjusted studies showed some evidence of an exposure-response gradient without evidence of statistical heterogeneity. The summary risks for all three exposure-duration strata were: RR = 1.39 (95% C.I. 1.19-1.63) for < 10 years (based on three estimates from two studies), RR = 1.64 (95% C.I. = 1.40-1.93) for $10 \leq$ to < 20 years (11 estimates from 6 studies), and RR = 1.64 (95% C.I. = 1.26-2.14) for ≥ 20 years (four estimates from four studies). The pooled risk estimate for those

studies for which the exposure duration was not clear in the published reports was 1.24 (95% C.I. = 1.00-1.54) (six estimates from four studies) (see Table C-4 in Appendix C of OEHHA (1998)).

These results were robust to a variety of sensitivity analyses. In an analysis of potential publication bias, however, there appeared to be a modest increase in the RR estimates with increasing sample size (reflected in a decreased standard error of the estimates). Publication bias, or the increased likelihood or preference for the publication of statistically significant results compared to nonsignificant or null results, may potentially distort pooled risk estimates. Publication bias is generally attributed to journal editorial policies that prefer “positive” results, so that small, statistically nonsignificant studies are less likely to be published than large, statistically nonsignificant studies (Greenland, 1994). However, it should be noted that the studies with the smallest standard errors were almost exclusively cohort studies that did not adjust for smoking and which also had a clear HWE, suggesting that other significant biases are likely to have played a role in creating an appearance of publication bias. Therefore, although publication bias cannot be ruled out, the inclusion of numerous studies of varying sample sizes and statistically insignificant findings, as well as the uncontrolled confounding and likely selection bias affecting many of the larger cohort studies, make it unlikely that the result of this meta-analysis can be completely explained by publication bias.

In summary, the meta-analysis indicated a consistent positive association between occupations involving diesel exhaust exposure and the development of lung cancer. Although substantial heterogeneity existed in the initial pooled analysis, stratification on several factors identified a persistent positive relationship. The major sources of heterogeneity included: (1) whether or not a study adjusted for smoking, (2) study design (3) the exposure assessment, as developed from occupational categories, (4) and the presence of selection bias, as manifested by an observed healthy worker effect. Taking these factors into account tended to increase the estimates of relative risks of lung cancer from occupational exposure to diesel exhaust.

Another independently conducted meta-analysis of diesel exhaust exposure and lung cancer produced remarkably similar results, with an overall pooled relative risk estimate of 1.33 (95% C.I. = 1.24-1.44) (Bhatia *et al.*, 1998). In that analysis, the study inclusion and exclusion criteria were somewhat different than those used by OEHHA staff, so that 23 studies were included. Consequently, the results of some of their subset analyses differed from those described in OEHHA (1998). In addition, those authors used only a fixed-effects model to derive pooled risk estimates, and did not focus on explorations of sources of heterogeneity. Nevertheless, Bhatia and co-workers also found a persistent positive relationship between diesel exhaust exposure and lung cancer that could not be attributed to potential confounding by cigarette smoking. Moreover, in the narrower group of studies in their report, they identified a positive exposure-response relationship in studies stratified by exposure duration.

Table 2. Studies Included in Meta-analysis of Diesel Exhaust Exposure and Lung Cancer

Study (year)	Design (Location)*	Occupation or Exposure Group	Smoking Adjusted	RR	C.I.
Ahlberg <i>et al.</i> (1981)	Cohort (†)	Truck drivers	no	1.33	1.13-1.56
Balarajan & McDowall (1988)	Cohort (†)	Truck drivers	no	1.59	1.00-2.53 ^a
Bender <i>et al.</i> (1989)	Cohort (‡)	Highway maintenance	no	0.69	0.52-0.90
Benhamou <i>et al.</i> (1988)	Case-control (†)	Professional drivers	yes	1.42	1.07-1.89
Buiatti <i>et al.</i> (1985)	Case-control (†)	Transportation general	yes	1.1	0.7-1.6
Benhamou <i>et al.</i> (1988)	Case-control (†)	Mechanics	yes	1.06	0.73-1.54
Boffetta <i>et al.</i> (1988)	Cohort (‡)	Truck drivers	yes	1.24	0.93-1.66
	Cohort (‡)	Railroad workers	yes	1.59	0.94-2.69
	Cohort (‡)	Heavy equipment operators	yes	2.60	1.12-6.06
Boffetta <i>et al.</i> (1990)	Case-control (‡)	Probable DE ≥ 30 yr	yes	1.49	0.72-3.11
Coggon <i>et al.</i> (1984)	Case-control (†)	Diesel exhaust exposed group	no	1.1	0.7-1.8
Damber & Larsson (1987)	Case-control (†)	Professional drivers	yes	1.2	0.6-2.2
Edling <i>et al.</i> (1987)	Cohort (†)	Bus drivers	no	0.69 ^b	0.2-1.6 ^b
Garshick <i>et al.</i> (1987)	Case-control (‡)	Railroad workers ≥ 20 yrs ^c	yes	1.55	1.09-2.21
Garshick <i>et al.</i> (1988)	Cohort (‡)	Railroad workers ≥ 15 yrs ^c	no	1.82	1.30-2.55
Guberman <i>et al.</i> (1992)	Cohort (†)	Professional drivers	no	1.50	1.23-1.81 ^c
Gustafsson <i>et al.</i> (1986)	Cohort (†)	Dock workers	no	1.32	1.05-1.66
Gustavsson <i>et al.</i> (1990)	Nested case-control (†)	Bus garage workers > 20 yr ^d	no	1.49 ^d	1.25-1.77 ^d
Hansen (1993)	Cohort (†)	Truck drivers	no	1.6	1.26-2.0
Hayes <i>et al.</i> (1989)	Case-control (‡)	Truck drivers ≥ 10 yr	yes	1.5	1.1-2.0
	Case-control (‡)	Bus drivers ≥ 10 yr	yes	1.7	0.8-3.4
	Case-control (‡)	Mechanic (excl auto) ≥ 10 yr	yes	2.1	0.9-5.2
	Case-control (‡)	Heavy equip. operators ≥ 10 yr	yes	2.1	0.6-7.1
Howe <i>et al.</i> (1983)	Cohort (‡)	Railroad workers probably exposed	no	1.35	1.13-1.61 ^a
Lerchen <i>et al.</i> (1987)	Case-control (‡)	Diesel exhaust grouped	yes	0.6	0.2-1.6
Magnani <i>et al.</i> (1988)	Death certificate study (†)	Diesel exhaust grouped	no	0.97	0.95-1.00
Menck & Henderson (1976)	Cohort (‡)	Truck drivers	no	1.65	1.13-2.40 ^a
	Cohort (‡)	Mechanic (excl auto)	no	3.32	1.35-8.18 ^a
Nokso-Koivisto & Pukkala (1994)	Cohort (†)	Railroad workers	no	0.90 ^d	0.79-1.04 ^d
Pfluger & Minder (1994)	Case-control (†)	Professional drivers	yes	1.48	1.30-1.68
Rafnsson & Gunnarsdottir (1991)	Cohort (†)	Truck drivers ≥ 30 yr	no	2.32	0.85-5.04
Rushton <i>et al.</i> (1983)	Cohort (†)	Bus garage workers/mechanics	no	1.01	0.82-1.22
Siemiatycki <i>et al.</i> (1988)	Case-control (‡)	Diesel exhaust grouped	yes	1.1	0.8-1.5 ^c
Steenland <i>et al.</i> (1990)	Case-control (‡)	Truck drivers ≥ 18 yr	yes	1.55	0.97-2.47
	Case-control (‡)	Truck mechanic ≥ 18 yr	yes	1.50	0.59-3.40
	Case-control (‡)	Heavy truck drivers ≥ 20 yr	yes	2.44 ^d	1.43-4.16 ^d
Swanson <i>et al.</i> (1993)	Case-control (‡)	Railroad workers ≥ 10 yr	yes	2.46 ^d	1.24-4.89 ^a
	Case-control (‡)	Transportation equip. operators	no	2.39 ^b	0.70-8.05 ^b
Wegman & Peters (1978)	Case-control (‡)	Transportation equip. operators	no	2.39 ^b	0.70-8.05 ^b
Wong <i>et al.</i> (1985)	Cohort (‡)	Heavy equip. operators ≥ 20 yr	no	1.07	1.00-1.15 ^a

^a Calculated from p-value.

^b Calculated from data presented in publication.

^c Risk estimates excluding shop workers.

^d Pooled risk estimates from two racial or duration categories.

^e 90% confidence intervals originally presented within study.

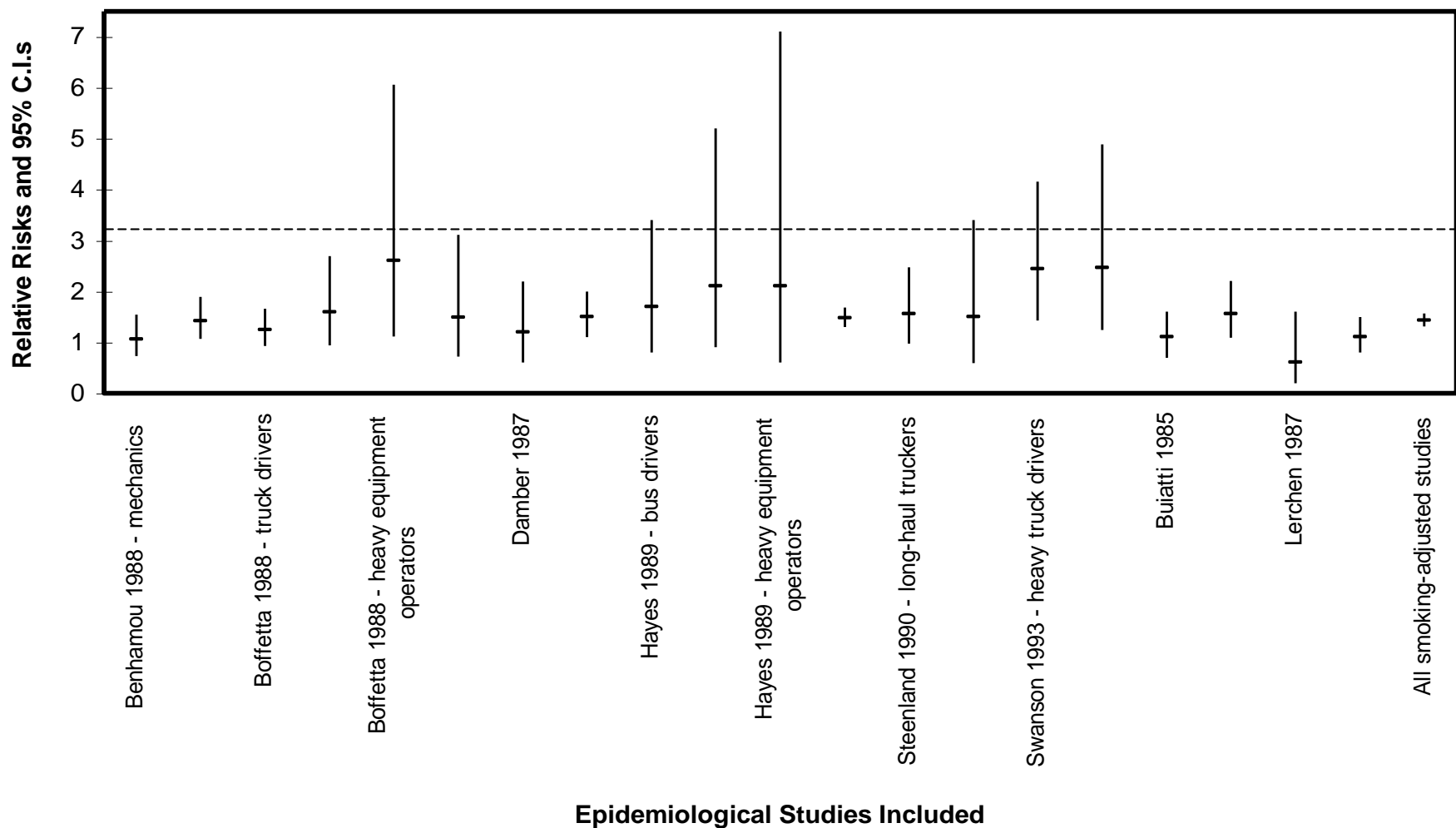
DE = diesel exhaust

RR = risk ratio

C.I. = 95% confidence interval.

* Location: (†)Europe, (‡)North America

Figure 1: Estimates of Relative Risks for Smoking-Adjusted Studies of Diesel Exhaust Exposure and Lung Cancer



Methodology

The complex and potentially variable mix of chemical species in the condensed phase and the vapor phase of diesel exhaust, required the measure of exposure related to carcinogenic risk to be specified. The most commonly used measure of exposure is atmospheric concentration of particles in $\mu\text{g}/\text{m}^3$. That measure is obtained from the mass of particles collected on a filter per volume of the air that flowed through the filter. On the basis of its relation to health studies and its general practicality, that measure was used in the diesel exhaust TAC document cancer risk assessment (OEHHA, 1998).

OEHHA used two approaches to employing epidemiological studies for diesel exhaust quantitative risk assessment. The first approach used the overall relative risks derived from the meta-analysis along with an overall range of exposure for all the studies. The second approach focused upon the railroad worker studies in developing the range of unit cancer risks.

Meta-analysis-Derived Cancer Unit Risks

The results of the meta-analysis provide information useful in bracketing the broadest likely range of plausible carcinogenic potencies for diesel exhaust. The pooled relative risk values derived from the 12 epidemiological studies in the meta-analysis which adjusted for smoking were 1.44 (95% C.I. 1.32 -1.56) for the fixed effects model and 1.43 (95% C.I. 1.31 -1.57) for the random effects model. The magnitude of these relative risks provide information on the potential magnitude of the cancer risk associated with diesel exhaust exposure. For the random effects model the upper 95% confidence limit on excess relative risk is 0.57.

None of the studies in the meta-analysis provide direct measurements of exposure concentration over the time of their follow up. Therefore, to the extent that the meta-analysis can be used to bracket the carcinogenic potency of diesel exhaust, the exposures of the various study populations need to be reconstructed. Hammond (1998) has reviewed the available industrial hygiene survey literature on the occupations considered in the meta-analysis (bus garage workers, mechanics, truck drivers, heavy equipment operators, railroad workers) and provided estimates of the plausible possible ranges of workplace exposures of diesel exhaust respirable particulate matter for those occupations. Because of the overall limitations in the data, the estimated ranges for each occupational subgroup of interest are particularly broad. The lowest plausible estimate of occupational exposure for any such subgroup is $5 \mu\text{g}/\text{m}^3$ (heavy equipment operators). The highest plausible estimate of any occupational subgroup is $500 \mu\text{g}/\text{m}^3$ (bus garage workers, railroad workers, mechanics). The total range of plausible exposures for the different populations therefore varies 100-fold. Using these air concentrations and the assumption that workers inhaling 10m^3 of air per work shift were exposed to them for over 45 year period for a 70 year lifetime, it is possible to characterize a bracket of risks compatible with the results of the meta-analysis:

$$\begin{aligned}
q_1^* &= \text{Excess relative risk} \times \text{CA lifetime lung cancer risk.} \\
&\quad \text{Air concentration} \times \text{exposure factor} \times \text{intermittency factors} \times \text{duration of} \\
&\quad \text{exposure/lifetime} \\
&= 0.57 \times 0.025 \\
&\quad (5 \text{ or } 500 \mu\text{g}/\text{m}^3) \times 10 \text{ m}^3/\text{shift}/20\text{m}^3/\text{d} \times 5\text{d}/7\text{d} \times 48\text{wk}/52\text{wk} \times 45 \text{ yrs}/70\text{yrs}
\end{aligned}$$

Therefore, the results of the meta-analysis bracket lung cancer risks up to approximately $1.3 \times 10^{-4} (\mu\text{g}/\text{m}^3)^{-1}$ (assuming all the worker populations in the meta-analysis were exposed to $5 \mu\text{g}/\text{m}^3$) to $1.3 \times 10^{-2} (\mu\text{g}/\text{m}^3)^{-1}$ (assuming all the workers populations in the meta-analysis were exposed to $500 \mu\text{g}/\text{m}^3$). As these assumptions establish the extreme bounds of probable exposures, and such calculations based upon a meta-analysis are novel and subject to further possible refinements, these results are not incorporated into the range of risks. However, these results do bracket the carcinogenic potencies which would be consistent with the results of the meta-analysis and the broadest range of exposure estimates.

A more plausible range can be estimated by determining the 90% confidence interval (CI) of the range of risks. For the meta-analysis the range of concentrations thought to be plausible by Hammond (personal communication) was 5 to $500 \mu\text{g}/\text{m}^3$ with a mean of about $200 \mu\text{g}/\text{m}^3$, which corresponds to a unit risk of $3.3 \times 10^{-4} (\mu\text{g}/\text{m}^3)^{-1}$. Using that concentration range as the 98% CI for a shifted lognormal distribution fixes the geometric standard deviation at 1.22 with a shift of the origin of the distribution by $330 \mu\text{g}/\text{m}^3$. The 90% CI for this distribution of concentration is [52.5 to $356.5 \mu\text{g}/\text{m}^3$], corresponding to a 90% CI for the distribution of unit risk of [1.6×10^{-4} to $1.2 \times 10^{-3} (\mu\text{g}/\text{m}^3)^{-1}$].

Railroad Worker Study-Derived Cancer Unit Risks

Quantitative relationships were also developed between lung cancer risk and exposure to diesel exhaust for two nation-wide studies of lung cancer rates in U. S. railroad workers. These relationships provided additional values for the range of risk to the general California population. The first, Garshick *et al.* (1987a), is a case-control study. Using a logistic regression, that study determined the coefficient of the logistic relationship of the odds of lung cancer for duration of the workers' exposure to diesel exhaust. The coefficient determined in that study was used to estimate lifetime unit risks for exposure of the general population. The second study, Garshick *et al.* (1988), is a cohort study. Using a proportional hazards model, that study calculated the relative hazard of lung cancer for increasing duration of worker exposure. However, those numerical results have not been supported by Garshick (1991); so instead of using them to derive lifetime unit risks for the general population, new analyses were performed with the individual data, upon which that study is based, to determine a linear relationship of lung cancer hazard for worker exposure to diesel exhaust.

The term hazard was used for a prediction of incidence (cancers per year per population) resulting from a model. Relative hazard is generally called relative risk in epidemiological model work, and the term, relative risk, was used in the context of the epidemiology results. The lifetime inhalation unit risk, often simply called unit risk, is defined as the probability of contracting lung cancer from a 70-year exposure to a unit concentration ($1 \mu\text{g}/\text{m}^3$) of diesel exhaust.

The unit risks ultimately derived for the general population assume that the mass concentration of particles governs the risk of diesel exhaust, regardless of the particular type of diesel engine or fuel. The resulting estimate of risk entails uncertainties due primarily to the limited exposure information available and to the choice of models and data used in the analysis.

These two studies are among a number of studies establishing excess relative risk of lung cancer among workers exposed to diesel exhaust. These two studies were specifically selected for the quantitative risk assessment because of their general excellence, their apparent finding of a relationship of cancer rate to duration of exposure and because of the availability of measurements of diesel exhaust among such railroad workers from the early 1980's in other studies. The case-control study appears to have an advantage in obtaining direct information on smoking rates, while the cohort study has an advantage of smaller confidence intervals of the risk estimates.

Estimating Cumulative Exposure

The risk relationships developed for the case-control study and the initial analyses for the cohort study used cumulative atmospheric exposure to diesel exhaust particles as the effective dose. The use of cumulative exposure, defined as the area under the curve (AUC) of concentration versus time, required a specification of the temporal pattern of exposure concentration. However, direct measurements of exposure concentration over the time of the follow up were not available.

Therefore, the calculations required reconstruction of the exposure history in order to determine cumulative exposure. The reconstruction was undertaken using (1) personal exposure measurements on railroad workers just after the end of the follow-up period in that study, (2) historical data on the dieselization of locomotives in the United States, and (3) descriptive information. The analysis included workers on trains and excluded shop workers from the original cohort because of mixed exposures, including no exposure to an unknown number in this group.

Exposure Measurements In The Early 1980s

Woskie *et al.* (1988b) estimated national average concentrations of respirable particulate matter (RSP) for 13 job-groups. These concentrations were obtained by temperature correction of measurements of respirable particulate matter (RSP) made in 1982-1983 in the northern region of the United States, as reported in Woskie *et al.* (1988a). The investigators adjusted these concentrations to remove the portion of RSP attributable to environmental tobacco smoke (ETS). The average values of the ETS-adjusted RSP for the principal categories of workers are listed in Table 3 for exposed and unexposed workers.

Table 3: Number of Workers in the Exposure Categories and the Cohort Averages of the Worker Exposure Concentration Following the Garshick *et al.* (1988) Cohort Study.

Exposure status	Career group	Number of workers	Subsequent exposure concentration ^a ($\mu\text{g}/\text{m}^3$)
Uncertain	Shopworkers	12,092	141(those exposed)
Exposed ^b	Engineers, firemen	11,005	71
	Brakemen, conductors, hostlers	18,285	89
Unexposed ^c	Clerks	10,475	33
	Signalmen	3548	58

^a Exposures reported by Woskie *et al* (1988b) for these career groups, based on measurements of ETS-adjusted RSP, circa 1982-3.

^b For all exposed workers in the table, except for those shopworkers who were exposed, the temporal exposure patterns are assumed to be the same, and the concentrations are close to each other; so a simple population-weighted average for the two career groups characterizes the average concentration for the exposed group, train workers, circa 1982-83:

$$(11,005 \times 71 + 18285 \times 89) / (11005 + 18285) = 82 \mu\text{g}/\text{m}^3$$

^c For all unexposed workers (background) in the table except for those shopworkers who were unexposed, the concentrations are close to each other; so a simple population-weighted average for the two groups characterizes the average background concentration, circa 1982-83:

$$(10475 \times 33 + 3548 \times 58) / (10475 + 3548) = 39 \mu\text{g}/\text{m}^3.$$

Reconstruction Of The Time Course Of Concentration

In order to estimate the time course of the exposure factors for the cohort, it was necessary to make assumptions about time trends of nationwide average concentration breathed by the workers. The exposure measurements made just after the follow-up period constitute a baseline for the reconstruction. The reconstruction of the time course of concentration proceeds by developing an exposure factor to multiply these baseline values. The analyses below explore the effect of alternative patterns of exposure concentration and baseline values.

Dieselization of the U.S. railroads began after the Second World War ended in 1945. The exposure of the railroad workers up until 1981 can be divided into two periods: (1) an initial period of increasing dieselization of U.S. locomotives from 1945 until mostly completed in 1959 and (2) a subsequent period of a moderate rate of addition of locomotives that were less smoky.

Woskie *et al.* (1988b) reported data showing a linear rise of percent dieselization with time in the first period from 1945 to 1959. They reported data from the Bureau of Labor Statistics showing that by 1947 fourteen percent of locomotives were diesel, by 1952 fifty-five percent were diesel, and by 1959 ninety-five percent were diesel. This linear rise of dieselization may be expected to have produced a linear rise of the national average exposure concentration around the trains. This linear rise is used in all the more realistic exposure patterns.

The exposure of workers on trains would then generally have declined as the newer, less smoky locomotives replaced the older, smokier locomotives on the main lines. To quantify the anecdotal information of greater smokiness of locomotives in the period before 1960, the national average exposure concentration was assumed to decline linearly in the second period, 1960-1980, to the baseline measured in 1982-3. The decline assumed from 1959 to 1980 is consistent with the report of sharp decreases of emissions of new engines between the 1970's and the 1980's. Emissions from naturally aspirated four-stroke engines declined from 2.1-3.0 g/kW-hr in the 1970's to 0.25 -0.6 g/kW-hr in the 1980's (Sawyer and Johnson, 1995).

In order to bracket the exposure of the railroad workers to diesel exhaust a variety of patterns of exposure are considered. The patterns are characterized by two components: a) the extent of change from 1959 to 1980 in diesel exhaust exposure, expressed as a ratio, and b) the average exposure concentration for the workers on trains measured in the Woskie *et al.* (1988a) study (i.e., the baseline). The alternate ratios are as follows: a) a ratio of 1 suggested and used in Crump *et al.* (1991) as more realistic than the Garshick *et al.* (1987a, 1988) assumption of constant concentration from 1959-1980 and none before that; b) a ratio of 2 suggested by K. Hammond to allow for a modest peak in 1959; c) a ratio of 3 allowing for more peak, a scaled down version of the exposure factor of 10 that Woskie *et al.* (1988b) reported for exposure concentration of shopworkers to nitrogen dioxide in enclosures including engine test sheds; and d) a ratio of 10, peak of the magnitude of values for the engine test sheds. The alternate baselines of exposure concentrations are as follows: 1) 40 $\mu\text{g}/\text{m}^3$, obtained by subtracting the background measurement of the unexposed workers from the measurement of the train workers, rounded down; 2) 50 $\mu\text{g}/\text{m}^3$, which also subtracted background from the train worker measurements but rounded up to allow somewhat for measurements of workers on trains not having as much exposure to non-diesel exhaust background particulate as the clerks; and 3) 80 $\mu\text{g}/\text{m}^3$, obtained by assuming that the entire ETS-adjusted RSP of the train workers is diesel exhaust while the clerks are considered unexposed to diesel exhaust (0 concentration).

The specific alternative patterns of linear decline (if any) of concentration from 1959 through 1980 are:

1. no decline, constant at the baseline values of 50, a ramp (1,50) pattern suggested and used in Crump *et al.* (1991).
2. declining 3-fold from a peak of 150 to a baseline of 50, a roof (3,50) pattern, the preferred pattern in this report;
3. declining 10-fold from a peak of 500 to a baseline of 50, a roof (10,50) pattern, suggested in information submitted by the Engine Manufacturers Association;
4. declining, 2-fold from a peak of 80 to a baseline of 40, a roof (2,40) pattern suggested by K. Hammond, one of the investigators in the Woskie *et al.* study; and
5. declining 3-fold from a peak of 240 to a baseline of 80, a roof (3,80) pattern, a variant on Pattern 3 for not subtracting background ETS-adjusted RSP in the exposed group while still maintaining
6. unexposed workers at zero concentration.

Calculation Of Cumulative Exposure

The estimate of the time course permits calculation of the overall average cumulative exposure for the cohort for each year of the follow-up period, 1959-1980. The cumulative exposure factor was calculated as the area under the curve (AUC) of the exposure factor (EF, ratio of concentration to baseline concentration) for successive years. Cumulative exposure is the cumulative exposure factor times the baseline value.

Intermittency Correction

The equivalent exposure duration for non-continuous exposure was scaled on the basis of volume of air breathed. Exposure durations are calculated to have the same cumulative yearly intake of the substance as produced by continuous inhalation of 20 m³/day at the concentration of the substance breathed in. Assuming that the average exposed member of the cohort inhales 10 m³ during an 8-hour working day implies an adjustment factor of 10/20 to multiply the exposure concentration to account for ventilation rate not equaling the standard human daily inhalation of 20 m³/day. Adjusting for the discontinuous work week and work year yields additional adjustment factors of 5/7 for exposure days per week and 48/52 for weeks per year, all to multiply the exposure measure. In order to take account of the non-continuous work exposure, the resulting overall multiplicative factor on exposure duration is

$$(10/20)(5/7)(48/52) = 0.33.$$

Determining Lifetime Unit Risk From The Relative-Risk Slope

The analyses below calculate the relationship between relative risk (relative hazard) and duration of exposure. The relative risk is the prediction of the ratio: incidence (yearly death rate per population) of lung cancer due to diesel exhaust divided by the background incidence of lung cancer. In the principal modeling of both sets of epidemiological data, reported below in this chapter, relative risks are fitted linearly to duration of exposure. From that slope, an estimate of the slope with respect to cumulative exposure for the specific alternative patterns of occupational exposure considered is obtained by modifying the duration scale for the slope. The approximation for this modification is simply to multiply the duration scale by the overall area under the curve (AUC) of the desired pattern and to divide by the total duration of exposure in the analysis.

Approximations may often be used to determine lifetime unit risk from this slope, but the present work will, for consistency and accuracy, use life-table calculations for that determination. This calculation starts with a background life table for lung cancer in California. For each unit risk to be calculated, a modification of that table is constructed in a way that includes the predicted effect of a lifetime exposure to 1 unit of concentration, 1 µg/m³ in the present calculations. The predicted effect is incorporated by multiplying the background lung cancer incidence for each age interval in the table by the relative risk (relative hazard) for that age interval. The relative risk is (1+ excess relative risk due to exposure). The excess relative risk due to exposure for unit concentration is the slope of relative risk with concentration, obtained from the epidemiological analyses. Using the general model based on cumulative exposure, as in the present calculations,

the excess relative risk requires the slope coefficient per concentration-year to be multiplied by the age in years for each age group in the table and to be divided by the intermittency factor. Any ages that fall within the number of years of detection lag prior to the target age have zero excess relative hazard. The modified table is completed in the manner of the original table. The lifetime unit risk is then the following difference: the probability of lung cancer at the target age in the table modified by exposure less the probability at the same age in the original table.

Use of the Garshick *et al.* (1987a) Case-Control Study to Estimate Unit Risk

The first study used to estimate lung cancer risk due to diesel exhaust exposure is the case-control study of U.S. railroad workers by Garshick *et al.* (1987a). For this case-control study Garshick *et al.* (1987a) collected 15,059 US railroad worker death records for 1981. They matched each of 1256 lung-cancer cases with 2 other deaths, each of those having nearly the same date of birth and death. For each of the controls, death was due to a specified natural cause with no mention of cancer on the death certificate. For each subject, Garshick *et al.* (1987a) determined years in a job with diesel exposure, asbestos exposure and smoking history. Taking into account the effect of age, their analysis used multivariate conditional logistic regression to determine the relationship between lung cancer and duration of exposure to diesel exhaust. For workers with more than 20 years exposure and for exclusion of shopworkers, they calculated the odds ratio was 1.55 (95% CI = 1.09, 2.21) with a referent category of 0 to 4 years work in a job exposed to diesel exhaust.

From the odds ratio for a 20 year duration of exposure, the coefficient of increase with duration of exposure was estimated by assuming a linear rise over the 20 years. Using a calculation similar to that used by Garshick *et al.* with shopworkers included, the slope coefficient for the odds ratio is 0.022 (90% C.I. = 0.0071, 0.037) year⁻¹. Because the odds ratio approximates relative risk (Breslow and Day, 1980, pp. 69-73), this value is approximately the rate of increase of relative risk (relative hazard) and is used in a life table to obtain the lifetime unit risk. The modified life table calculation for unit concentration (1 µg/m³) for 5-yr. lag from carcinogenesis to death is in Table 7-1 of the diesel exhaust TAC document (OEHHA, 1998). The resulting unit risks are presented in Point I in Table 7-3 of the diesel exhaust TAC document. The highest values in that set are for the assumption that workers on trains have a ramp (1,50) pattern of exposure. The 95% UCL for lifetime unit risk is 2.4×10^{-3} (µg/m³)⁻¹, with an MLE of 1.4×10^{-3} (µg/m³)⁻¹. For the roof (3,50) pattern of exposure, the procedure is similar, but the exposure scale is increased by the ratio 65/22, representing the ratio of area under the EF of the roof to the area under the EF of the block. The resulting 95% UCL for lifetime unit risk is 1.0×10^{-3} (µg/m³)⁻¹, with an MLE of 6.2×10^{-4} (µg/m³)⁻¹. The lowest values in the set are for the roof (10,50) pattern of exposure. Using a similar approach, multiplying the exposure scale by the AUC ratio of 191/22, the 95% UCL for lifetime unit risk is 3.6×10^{-4} (µg/m³)⁻¹, with an MLE of 2.1×10^{-4} (µg/m³)⁻¹.

Using the slope coefficient for the analysis including shopworkers, reported in Garshick *et al.* (1987a), McClellan *et al.* (1989) previously calculated the expected increase in U.S. lung cancer deaths per year for each µg/m³ of diesel exhaust exposure for two alternative exposure concentrations, 125 µg/m³ and 500 µg/m³, constant from 1959-1980. Mauderly (1992a) used these death rates to estimate unit risks, finding expected values of 1.2×10^{-3} (lifetime-µg/m³)⁻¹

and 2.9×10^{-4} (lifetime $\mu\text{g}/\text{m}^3$)⁻¹, respectively. These values are close to the higher MLE values just given. Even though the higher concentrations assumed by McClellan *et al.* would tend to produce lower unit risks, the effect of using the more accurate life table method has a counteracting effect.

Use of the Garshick *et al.* (1988) Cohort Study to Estimate Unit Risk

The second study selected to estimate lung cancer risk due to diesel exhaust exposure was the retrospective cohort study of U. S. railroad workers by Garshick *et al.* (1988). The present analysis uses the individual data collected for that study in new calculations to determine slopes for the relationship of incidence to cumulative exposure. The analysis uses reconstructions of exposure, the ramp and the roof exposure patterns, to adjust the slope obtained from the analysis that is implemented with duration of exposure as the measure of exposure.

Further material on the cohort is developed in Appendices D, E, F of the diesel exhaust TAC document (OEHHA, 1998). Appendix E contains references to correspondence cited in this chapter. (The original unpublished documents referred to in Appendix E are available on request from the California Air Resources Board, Stationary Source Division or from the U.S. EPA docket for the Health Assessment Document for Diesel Emissions at the National Center for Environmental Assessment, Washington, DC. 20460 (1997)).

Description of the Original Study

The cohort consisted of 55,407 railroad workers, who were aged 40-64 in 1959 and who had started railroad service 10-20 years earlier; 1694 lung cancers were identified. The unexposed group in the cohort, the clerks and signal tenders, constituted 25.3% of the whole cohort. To develop the original data set, Garshick *et al.* (1988) obtained the following information for each individual in their cohort of railroad workers for the follow-up years of 1959-1980: cause of death by death certificate, the primary job classification for each year, and months worked in that classification in each year. In addition, the investigation obtained the age at the start of follow-up in 1959, total service months and, for those workers who began work after 1946, the date of starting work. From these data Garshick *et al.* calculated the elapsed time of exposure for each individual from 1959 up to each follow-up year or up to the four years before each follow-up year.

Relative Risk Analysis

Because of much uncertainty about the proportion of shop workers exposed to diesel exhaust, OEHHA decided to exclude them from the analysis, as suggested by the study authors and other participants at the Diesel Exhaust Workshop, January, 1996. Garshick (1991) had previously called attention to dilution of the effect of diesel exhaust on the shop workers because of the inclusion of shopworkers in that cohort who had no true exposure. The original study obtained risk estimates both with and without the shop workers, and found the results changed very little. The exclusion of shop workers simplifies the analysis in that lung burden calculations are not needed because the exposures of other exposed workers, namely train workers, are sufficiently low that lung burden may be assumed essentially proportional to atmospheric exposures.

Exposure measurements for 1982-83 (Woskie *et al.* 1988a), just after the end of the follow-up period, show that train workers considered here all experienced approximately the same average concentration of diesel exhaust (for example, $50 \mu\text{g}/\text{m}^3$, rounded, for use in determining unit risk in this work). The present work uses years with any month of exposure time, excluding the four years previous to each year of observation as the average lag time from carcinogenesis to death. This calculation of exposure time starts in 1952 and continues yearly through 1980, the end of follow-up. It extends 7 years back from 1959, the start of follow-up, to account on the average for the assumed linear rise of exposure from 1945 to 1959. The unexposed workers are assigned zero exposure time throughout.

The OEHHA analysis uses two programs in the EPICURE software package, which is designed for several standard kinds of epidemiological analysis. The first program, DATAB, reduces the individual data to cells with each desired variable having a single value for the cell. The cells are designated by a set of numbers, one for each categorical variable to determine the category number of that variable. The second program, AMFIT, determines parameters of a model to provide a best fit of the data using Poisson regression, a maximum likelihood procedure (Breslow and Day, 1987). The calculation approach is described in more detail for the closely related calculations using general models, in Appendix D of the diesel exhaust TAC document (OEHHA, 1998).

The assumptions not otherwise specified here are essentially those of Garshick *et al.* (1988). For example, all years of the study are included, and their rather irregular boundary points on years of exposure are used.

The OEHHA analysis explored the fit and other characteristics of a number of forms of a general model. The model that appeared to be most satisfactory is the one with linear and quadratic continuous covariates, age and calendar year. The slope calculated for relative risk (relative hazard) per year of exposure is 0.015 (95% CI: 0.0086 to 0.022) year^{-1} . The slope divided by the intermittency correction (0.33) and the assumed constant concentration (e.g., $50 \mu\text{g}/\text{m}^3$ for 29 years) and multiplied by attained age provides the excess relative hazard to determine the increase of lung cancer rates for the lifetable calculation of the unit risk. The resulting unit risks are presented in Point II in Table 4, and closely parallel the results for the case-control study (Point I). The highest values in that set are for the assumption that workers on trains have a ramp (1,50) pattern of exposure. For the ramp pattern the result is a 95% UCL of $1.8 \times 10^{-3} (\mu\text{g}/\text{m}^3)^{-1}$ and a MLE of $1.3 \times 10^{-3} (\mu\text{g}/\text{m}^3)^{-1}$. For the roof (3,50) pattern of exposure, the procedure is similar, but the exposure scale is increased by the ratio $65/29$, representing the ratio of area under the EF of the roof to the area under the EF of the ramp. The result is a 95% UCL of $8.2 \times 10^{-4} (\mu\text{g}/\text{m}^3)^{-1}$ and a MLE of $5.7 \times 10^{-4} (\mu\text{g}/\text{m}^3)^{-1}$. The lowest values in the set are for the roof (10,50) pattern of exposure. Using a similar approach, multiplying the exposure scale by the AUC ratio of $191/29$, the 95% UCL for lifetime unit risk is $2.8 \times 10^{-4} (\mu\text{g}/\text{m}^3)^{-1}$, with an MLE of, $1.9 \times 10^{-4} (\mu\text{g}/\text{m}^3)^{-1}$.

Table 4: Values from Unit Risk for Diesel Exhaust from Using Hazard Slope on Exposure Measure in California Life-Table. Garshick *et al.* (1987a, 1988) Studies of U.S. Railroad Workers.

	q1 ($\mu\text{g}/\text{m}^3$) ⁻¹	
	MLE	95% UCL
<u>I. Case-Control study (1987a) using published slope coefficient for hazard on years of exposure to diesel exhaust (Section 7.3.3)</u>		
A. Adapted to ramp (1,50) pattern of exposure	1.4×10^{-3}	2.4×10^{-3}
B. Adapted to roof (2,40) pattern of exposure	1.1×10^{-3}	1.8×10^{-3}
C. Adapted to roof (3,50) pattern of exposure	6.2×10^{-4}	1.0×10^{-3}
D. Adapted to roof (3,80) pattern of exposure	3.9×10^{-4}	6.6×10^{-4}
E. Adapted to roof (10,50) pattern of exposure	2.1×10^{-4}	3.6×10^{-4}
<u>II. Cohort study (1988) using individual data to obtain a slope for hazard on years of exposure to diesel exhaust (Section 7.3.4)</u> <u>Continuous covariates: (attained age and calendar year)</u> <u>or (age-at-start-of study and calendar year)</u>		
A. Adapted to ramp (1,50) pattern of exposure	1.3×10^{-3}	1.8×10^{-3}
B. Adapted to roof (2,40) pattern of exposure	9.9×10^{-4}	1.4×10^{-3}
B. Adapted to roof (3,50) pattern of exposure	5.7×10^{-4}	8.2×10^{-4}
D. Adapted to roof (3,80) pattern of exposure	3.6×10^{-4}	5.1×10^{-4}
E. Adapted to roof (10,50) pattern of exposure	1.9×10^{-4}	2.8×10^{-4}
<u>III. Cohort study (1988) applying time varying concentrations to individual data to obtain a slope of hazard on exposure (from Appendix D)</u>		
A. Ramp (1,50) pattern of exposure		
1. General multiplicative model with age-at-start-of-study and U.S. rates as categorical covariates	1.2×10^{-3}	1.9×10^{-3}
2. 6th/7-stage model with age-at-start-of study as categorical covariate	2.4×10^{-4}	3.8×10^{-4}
B. Roof (3,50) pattern of exposure		
1. General multiplicative model with age-at-start-of-study and U.S. rates as categorical covariates	5.1×10^{-4}	7.2×10^{-4}
2. 6th/7-stage model with age-at-start-of-study as categorical covariate	8.1×10^{-5}	1.3×10^{-4}
3. 7th/7-stage model with age-at-start-of-study as categorical covariate	1.0×10^{-4}	1.5×10^{-4}

Discussion of Results

The investigation of the forms of the model using Poisson regression explored the use of categorical covariates, calendar year and age-at-start-of-follow-up that verified the categorical trend with exposure that Garshick *et al.* (1988) had obtained for relative hazard by using a Cox regression with calendar year as the principal time scale and age-at-start-of-follow-up as a covariate. This result was an elevated relative risk (relative hazard) for the middle durations of exposure and an apparent rise at the highest exposure, albeit with large error bars. Crump (1997) found by direct comparison a close correspondence of results for this Poisson regression and a Cox regression that replicated Garshick *et al.*

The investigation also explored the use of a general model with the categorical covariates, calendar year and attained age, that verified the categorical results for relative risk in Crump *et al.* (1991) and Crump (1997). This result showed a rise and then an apparent fall of relative risk for increasing exposure. Age and calendar year are important determinants of lung cancer rate, and Crump (1997) has argued that this choice should be used for covariates because it is the most accurate in characterizing background rates and, further, that a fall of relative risk at the higher exposure, obtained for this choice of covariates, is not consistent with an exposure response.

It should be kept in mind that the categorical trends of the relative risk with duration of exposure are all used to represent a large cloud of observed points of incidence as a function of duration of exposure. Appendix F of the diesel exhaust TAC document (OEHHA, 1998) indicates that the discrepancy between the results of Garshick *et al.* and of Crump *et al.* may be more apparent than real. The slopes for the relative risk are significant for both these choices of covariate, but the slope for the use of calendar year and age-at-start is about twice that for the use of calendar year and attained age. The latter slope is larger, though less significant, than the identical slope obtained in the present analysis using continuous forms of either pair of covariates. The use of the continuous form of the covariates appears to have a salutary effect on reducing the variance of the slope estimate. This choice allows some flexibility, but not a lot, in describing time trends.

Conclusion

Based on the human data, the principal finding of the diesel exhaust TAC document quantitative risk assessment is a range of lifetime unit risk (95% UCL) as shown in the right-hand column of Table 4 above. The lowest value in the range is 1.3×10^{-4} , and the highest value is 2.4×10^{-3} . The geometric mean unit risk obtained from these end points of the range of values is 6×10^{-4} (lifetime- $\mu\text{g}/\text{m}^3$)⁻¹. The geometric mean provides information on the central tendency of the range and is not to be confused with a best estimate identified from the available calculations. The lower end of the range is the rounded value for both forms of multistage model using the roof exposure pattern for the data of the Garshick *et al.* (1988) cohort study of U.S. railroad workers. OEHHA concluded that incorporation of the roof exposure pattern and biologically-based analyses improved the unit risk estimates. Consequently, unit risk values incorporating this information, those at the lower end of the range, provide more scientifically defensible values. The upper end of the range is obtained using the published results of the Garshick *et al.*

(1987a) case-control study for US railroad workers. The Scientific Review Panel concluded in their findings that a reasonable estimate of the cancer unit risk is $3 \times 10^{-4} (\mu\text{g}/\text{m}^3)^{-1}$.

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CHLORINATED DIBENZO-*p*-DIOXINS

CAS No: 1746-01-6

CHLORINATED DIBENZOFURANS

CAS No: 5120-73-19

I. PHYSICAL AND CHEMICAL PROPERTIES (From HSDB (1998) except as noted)

2,3,7,8-Tetrachlorodibenzo-*p*-dioxin

Molecular weight	322
Boiling point	decomposes (NIOSH, 1994)
Melting point	305-306 °C
Vapor pressure	7.4×10^{-10} mm Hg at 25 °C
Air concentration conversion	not available

2,3,7,8-Tetrachlorodibenzofuran

Molecular weight	305.99
Boiling point	not available
Melting point	not available
Vapor pressure	not available
Air concentration conversion	not available

II. HEALTH ASSESSMENT VALUES

Congener	Unit Risk ($\mu\text{g}/\text{m}^3$) ⁻¹	Slope Factor ($\text{mg}/\text{kg}/\text{day}$) ⁻¹
PCDDs		
2,3,7,8-Tetrachlorodibenzo- <i>p</i> -dioxin	3.8 E+1	1.3 E+5
1,2,3,7,8-Pentachlorodibenzo- <i>p</i> -dioxin	3.8 E+1	1.3 E+5
1,2,3,4,7,8-Hexachlorodibenzo- <i>p</i> -dioxin	3.8 E+0	1.3 E+4
1,2,3,6,7,8-Hexachlorodibenzo- <i>p</i> -dioxin	3.8 E+0	1.3 E+4
1,2,3,7,8,9-Hexachlorodibenzo- <i>p</i> -dioxin	3.8 E+0	1.3 E+4
1,2,3,4,6,7,8-Heptachlorodibenzo- <i>p</i> -dioxin	3.8 E-1	1.3 E+3
1,2,3,4,5,6,7,8-Octachlorodibenzo- <i>p</i> -dioxin	3.8 E-3	1.3 E+1
PCDFs		
2,3,7,8-Tetrachlorodibenzofuran	3.8 E+0	1.3 E+4
1,2,3,7,8-Pentachlorodibenzofuran	1.9 E+0	6.5 E+3
2,3,4,7,8-Pentachlorodibenzofuran	1.9 E+1	6.5 E+4
1,2,3,4,7,8-Hexachlorodibenzofuran	3.8 E+0	1.3 E+4
1,2,3,6,7,8-Hexachlorodibenzofuran	3.8 E+0	1.3 E+4
1,2,3,7,8,9-Hexachlorodibenzofuran	3.8 E+0	1.3 E+4
2,3,4,6,7,8-Hexachlorodibenzofuran	3.8 E+0	1.3 E+4
1,2,3,4,6,7,8-Heptachlorodibenzofuran	3.8 E-1	1.3 E+3
1,2,3,4,7,8,9-Heptachlorodibenzofuran	3.8 E-1	1.3 E+3
1,2,3,4,5,6,7,8-Octachlorodibenzofuran	3.8 E-3	1.3 E+1

Congener	Unit Risk ($\mu\text{g}/\text{m}^3$) ⁻¹	Slope Factor ($\text{mg}/\text{kg}/\text{day}$) ⁻¹
PCBs (IUPAC #, structure)		
77 3,3',4,4'-Tetrachlorobiphenyl	3.8 E-3	1.3 E+1
81 3,4,4',5- Tetrachlorobiphenyl	3.8 E-3	1.3 E+1
105 2,3,3',4,4'- Pentachlorobiphenyl	3.8 E-3	1.3 E+1
114 2,3,4,4',5- Pentachlorobiphenyl	1.9 E-2	6.5 E+1
118 2,3',4,4',5- Pentachlorobiphenyl	3.8 E-3	1.3 E+1
123 2',3,4,4',5- Pentachlorobiphenyl	3.8 E-3	1.3 E+1
126 3,3',4,4',5- Pentachlorobiphenyl	3.8 E+0	1.3 E+4
156 2,3,3',4,4',5- Hexachlorobiphenyl	1.9 E-2	6.5 E+1
157 2,3,3',4,4',5'- Hexachlorobiphenyl	1.9 E-2	6.5 E+1
167 2,3',4,4',5,5'- Hexachlorobiphenyl	3.8 E-4	1.3 E+0
169 3,3',4,4',5,5'- Hexachlorobiphenyl	3.8 E-1	1.3 E+3
189 2,3,3',4,4',5,5'- Heptachlorobiphenyl	3.8 E-3	1.3 E+1

PCDDs = polychlorinated dibenzo-*p*-dioxins. PCDFs = polychlorinated dibenzofurans. PCBs = polychlorinated biphenyls. IUPAC = International Union for Pure and Applied Chemistry.

[Linearized multistage procedure (GLOBAL79), fitted to male mouse hepatic adenoma and carcinoma data (NTP, 1982), body weight scaling, cross-route extrapolation (CDHS, 1986).]

III. CARCINOGENIC EFFECTS

Human Studies

Comprehensive reviews of the human studies of dioxin exposure and cancer risk available at the time the document entitled *Health Effects of Chlorinated Dioxins and Dibenzofurans* was written for the Toxic Air Contaminant (TAC) program (CDHS, 1986) are found in US EPA (1984) and Veterans Administration (VA) (1981, 1984). A more recent review of human dioxin exposure and cancer risk studies can be found in ATSDR (1999).

Dioxins have never been intentional products. In human exposure studies, PCDDs (polychlorinated dibenzo-*p*-dioxins) and PCDFs (polychlorinated dibenzofurans) have only been present as contaminants of other toxic chemicals, such as herbicides. Hence all studies of human PCDD/PCDF exposures have been studies of exposure to chemical mixtures that may have contained PCDD and PCDF.

VA (1981, 1984) summarized what is known about the presence of PCDD and PCDF in commercially-used chemicals. In general, PCDDs and PCDFs may be present as contaminants in the herbicide 2,4,5-trichlorophenoxyacetic acid (2,4,5T). Levels of 2,3,7,8-TCDD in 2,4,5-T have been found as high as six parts per million (Rappe *et al.* 1982). Another widely used herbicide, 2,4-dichlorophenoxyacetic acid (2,4-D) is

generally regarded as uncontaminated with 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD). Cochran *et al.* (1982) did detect traces of di-, tri-, and TetraCDD as high as one part per billion in technical grade 2,4-D from Canada. However, the TetraCDD isomer found in these samples was the 1,3,6,8-TCDD isomer, not the more toxic 2,3,7,8-TCDD.

Agent Orange, which was a mixture of 2,4,5-T and 2,4-D, has been shown to contain 2,3,7,8-TCDD concentrations as high as 15-47 parts per million with an average of about 2 ppm (VA 1981). PCDDs and/or PCDFs have also been found in the parts per million range in commercially used polychlorinated biphenyls (PCB), trichlorophenol (TCP), tetrachlorophenol, and pentachlorophenol (PCP) (Rappe *et al.* 1982, Hardell 1983).

Several case/control studies have been conducted in Sweden and in New Zealand. In these countries, phenoxyacetic acids and chlorophenols were used extensively for agriculture and forestry. After clinical observations of several patients with soft-tissue sarcomas (STS) and a history of heavy exposure to phenoxyacetic acids, Hardell and Sandstrom (1979) conducted a case/control study of STS and herbicide exposure. Cases were drawn from a university hospital in Northern Sweden, and consisted of 52 adult males with STS diagnosed between 1970 and 1977. Controls were drawn from general population registries, at a 4:1 matching ratio, and matched to cases on sex, age, place of residence, and vital status (whether alive or deceased). The investigators considered only non-malignant deaths for deceased controls. Study subjects (or their next of kin) provided exposure histories by a mailed questionnaire with a telephone follow-up. The odds ratio (OR) for exposure to phenoxyacetic acids only (excluding subjects exposed to chlorophenols) was 5.3 (95% confidence interval (95% CI) 2.4-11.5). For exposure to chlorophenols only (excluding those exposed to phenoxyacetic acids) the OR was 6.6 (95% CI 2.1-20.9).

To confirm these findings, Ericksson *et al.* (1981) replicated this study in Southern Sweden, using cases from a cancer registry. Similar study methods were used, including matching controls from a population registry (at a 2:1 ratio), and determining exposure by mail and telephone questionnaires. The investigators calculated separate odds ratios for exposure to phenoxy acids known to be contaminated with PCDD and PCDF (OR-17.0; 95% CI 2.1-140.0) and for exposure to phenoxy acids thought to be free of PCDD and PCDF (OR-4.2; 95% CI 1.2-14.9). When exposure was dichotomized into categories of 30 days or less, or more than 30 days, the ORs were 5.7 and 8.5, respectively, possibly indicating a dose-response trend.

One of the drawbacks of this study is that, exposure histories were provided by the study subjects; therefore, the results may be influenced by recall bias. Cases (or their next of kin) may be more likely to recall an exposure than a healthy person. In order to investigate this possible bias, Hardell (1981) duplicated the study methods using cases of colon cancer. Here there was no significant association with exposure to herbicides. Therefore, Hardell concluded that the association with STS was not due to reporting differences between diseased cases and healthy controls.

Smith *et al.* (1984) reported a similar case/control study in New Zealand. Here, male cases of STS were gathered from a national cancer registry, with controls also being

selected from the same registry. This method of control selection was designed to avoid differential recall. Unlike the Swedish studies, however, the New Zealand study showed no significant associations with reported phenoxy herbicide spraying. The authors suggested that if dioxin were the necessary agent, that Swedish herbicides may have been more contaminated than New Zealand herbicides. However, Smith *et al.* (1984) note that the Swedish investigators also found a significant association between STS and non-dioxin-contaminated herbicides, indicating that if the association were true, dioxin would not be the sole agent.

Another case/control study reported in brief by Olsen and Jensen (1984) of cases from the Danish Cancer Registry failed to show an association between nasal cancer and chlorophenol exposure, although nasal cancer was associated with occupational exposure to wood dust.

In a letter to *Lancet*, Milham (1982) reported proportionate mortality data from Washington state indicating that farmers suffered a significantly larger proportion of deaths due to STS. No other group occupationally exposed (foresters, orchardists, tree farmers) showed an excess of STS; however, the exposure assessment was based on occupations taken from death certificates. Furthermore, Milham indicated that 2,4-D was the predominant herbicide used, and 2,4-D is not generally contaminated with 2,3,7,8-TCDD.

A cohort study of phenoxy acid herbicide applicators in Finland was reported by Riihimaki *et al.* (1983). A historical cohort of 1926 herbicide applicators was assembled from the records of four large employers, including the Finnish Highway Authority and State Railways. These male workers had used chlorinated phenoxyacids for at least two weeks between 1955 and 1971. Their mortality between 1972 and 1980 was studied by comparing their names against population registers. National mortality figures provided expected age-standardized numbers of deaths. Deaths from all causes, and for all cancers, were less than expected. The power of this study to detect an increase in STS was poor, however, as only 0.1 case of STS was expected based on general population rates. Furthermore, as deaths in the cohort were studied only after 1972, 45 deaths that occurred in this group before 1972 were not tallied. (Even for post-1971 deaths, however, the follow-up period may also have been too short for a sufficient tumor latency period to have elapsed.)

There have been four potentially exposed occupational cohorts studied in the United States. Zack and Suskind (1980) reported the follow-up of Monsanto employees in Nitro, West Virginia, who were involved in a 1949 accident during the processing of trichlorophenol. A sudden violent reaction released fumes and residues into a building interior. Apparently, the released chemical mixture was not analyzed, but the authors assumed that it contained TCDD, as exposed workers developed chloracne. A historical cohort of 121 white male employees was assembled from company records on the basis of their having exhibited skin disorders "attributed to the 1949 TCP process accident." Their vital status was traced through 1978, providing a maximum of 29 years of follow-up per person. The standardized mortality ratio (SMR) for all causes of death in

this cohort (relative to US white males) was significantly decreased (32 observed deaths vs. 46.4 expected). One cancer site showed an excess: lung cancer (5 observed vs. 2.85 expected), although this SMR of 1.75 was not statistically significant. Interestingly, there occurred one STS, a fibrous histiocytoma. However, the authors calculated SMRs (and expected numbers of deaths) only for causes with five or more observed deaths.

Zack and Gaffey (1983) described another cohort from this plant, composed of 884 male workers employed for at least one year between 1955 and 1977. It is not clear whether workers exposed in the 1949 accident were included. The same methods were used to calculate SMRs. Only 25 malignancies occurred, compared to 30.9 expected. However, two specific sites were notably elevated: lung cancer, with 14 observed vs. 9.9 expected (SMR 1.4; 95% CI 0.8-2.4), and bladder cancer, with 9 observed vs. 0.9 expected (SMR 9.9; 95% CI 4.5-18.8). One STS occurred in a worker judged to have been exposed to TCDD. One drawback to this study is that exposure histories were only constructed for the 163 decedents - and only 36% of these were judged to have had potential exposure to 2,4,5-T (and therefore TCDD). Therefore, the true exposed cohort may only have been one-third the size of the entire study group.

Cook *et al.* (1980) presented a similar historical cohort study of Dow chemical employees. In 1964, chloracne occurred in workers in a trichlorophenol manufacturing area. Industrial hygiene investigations concluded that TCDD was responsible and changes were made in the operations to decrease exposure. Levels of TCDD during this period were unknown because concentrations fell below the limit of detection at that time, 0.02 µg/ml of air (Cook 1981a); however, wipe samples were positive for TCDD. Cook *et al.* (1980) assembled a cohort of 39 workers thought to have high exposure potential, and 22 workers thought to have lower exposure. Among the high-exposure group, 87% had a history of chloracne, compared to 68% of the low-exposure group. Their vital status was determined through 1978. There were only four deaths (vs 7.8 expected based on US white males), although three of these deaths were due to neoplasms (vs 1.6 expected). One neoplasm was a fibrosarcoma.

Another Dow cohort was investigated by Ott *et al.* (1980). This cohort contained 204 white males involved in 2,4,5-T production between 1951 and 1971. The authors determined each worker's vital status through 1976, resulting in a median length of time since first exposure of about 20 years. Only one malignancy (a respiratory cancer) was recorded vs. 3.6 expected from US population rates. This cancer death occurred among the employees with 20 or more years of latency; in this group 0.9 deaths were expected.

Besides the small sample size, there are other problems with using this study for risk assessment. The exposure to TCDD may have been minimal. Environmental sampling of the breathing zone in 1969 revealed 2,4,5-T concentrations between 0.2 and 0.8 mg/m³. Product specifications at that time called for a maximum TCDD concentration of 1 ppm. Assuming the maximum level of both 2,4,5-T in the breathing zone, and TCDD in the 2,4,5-T, the concentration of TCDD in the breathing zone would have been 10⁻⁶ of the concentration of 2,4,5-T, or 0.8 ng/m³. Ott *et al.* also noted that 157 of the 204 workers (77%) were exposed for less than one year. Furthermore, a review of medical records of the cohort uncovered no cases of chloracne.

A further analysis of Dow employees was presented by Bond *et al.* (1983), who reported a morbidity survey on the combined cohorts previously described by Cook *et al.* (1980) and Ott *et al.* (1980). Bond *et al.* found few differences between the morbidity of these workers and a matched control group of workers from other locations in the plant. There were, however, more ulcers and diseases of the digestive system (excluding liver) in the 2,4,5-T cohort, at roughly twice the prevalence in the controls. However, because the investigators only studied cohort members who participated in company medical programs between 1976 and 1978, only 69% of the original cohort was included. The study did not include workers who had died, retired, or left the company, raising the possibility that the most affected workers might have been missed.

Following the publication of the four US mortality studies, reports began to appear in *Lancet* of four additional cases of STS among these cohorts, bringing the apparent total to seven (Honchar and Halperin 1981, Cook 1981b, Moses and Selikoff 1981, Johnson *et al.* 1981). The proportion of deaths in these merged cohorts due to STS appeared to be far greater than would be expected (Fingerhut and Halperin 1983), although there is great difficulty in estimating expected rates of STS using general population statistics (Cook and Cartmill 1984). Fingerhut (cited in VA 1984) had the diagnoses of the seven cases reviewed by two pathologists. The pathologists could only agree on a diagnosis of STS for three of the seven, another three being reclassified, and the last diagnosis being disputed. Of the three definite cases, only two had frank chloracne to corroborate exposure. The VA review (1984) concluded that the occurrence of even two cases of STS among these relatively small cohorts warranted continued surveillance.

Other cohort studies of occupational exposures have come from Great Britain, West Germany, and the Netherlands. May (1973 and 1982) only briefly described the aftermath of a 1968 accidental release of TCP with a "higher than normal" concentration of TCDD. A total of 79 cases of chloracne were recorded, but May did not specify how many workers were exposed, so that an attack rate cannot be calculated. A survey of 46 of these workers, who were still with the company 10 years later, revealed that roughly half still had some chloracne (May, 1982). There were no other clinical problems reported, and no cases of cancer (although clearly few if any would be expected in a group this small).

Thiess *et al.* (1982) published a carefully-reported study of 74 workers exposed to dioxins during a 1953 reactor accident in a German 2,4,5-T plant. After a 23-year follow-up, this cohort exhibited seven deaths due to malignancies (vs. 4.09 expected from West German population rates), including three deaths due to stomach cancer (vs. 0.7 expected). The latter was statistically significant at a one-sided 95% level. No cases of STS occurred, although less than 0.1 would have been expected.

A mortality study of workers present at an explosion in an herbicide factory in Amsterdam was summarized by Dalderup and Zellenrath (1983). Between 200 and 500 g of TCDD were thought to have been liberated. The investigation traced 141 of 145 workers potentially exposed, and 69 (49%) had developed chloracne. After 20 years of follow-up, 8 of the workers had died with cancer (vs. 6.9 expected), yielding an SMR of

1.2 (95% CI 0.5-2.3). No STS deaths were seen. Unfortunately, the authors did not calculate SMRs separately for the group with frank chloracne (an indicator of stronger exposure), as the crude mortality for this chloracne group was 20%, and for the non-chloracne group 15%.

At the time the dioxin TAC document was prepared (CDHS, 1986), reports were starting to appear in the literature on the effects of Agent Orange herbicide exposure in Vietnam. However, most of those reports were at the time primarily anecdotal, or interim results. Agent Orange was composed of equal parts 2,4-D and 2,4,5-T, and about 90,000 tons of herbicides were sprayed in Vietnam between 1962 and 1971. Hay (1983) mentioned evidence from Vietnamese studies that "suggests a link" between herbicide exposure and liver cancer, but provided no details. Sarma and Jacobs (1982) reported three patients with STS who claimed Agent Orange exposure while serving in Vietnam.

The US Air Force's Ranch Hands study (summarized by VA, 1984) had released some initial results at the time the dioxin TAC document was prepared. This was a cohort study of some 1200 military personnel who worked on Operation Ranch Hand, the herbicide spraying operation. These subjects were matched (in a 5:1 ratio) with personnel who flew only cargo missions in Vietnam. As of 1983, the total mortality rates were nearly identical between the two groups. Only four cases of cancer had occurred among the exposed, and none were STS. The investigators stressed the preliminary nature of the data, the relatively low power of a study of this size to detect rare tumors such as STS, and the relatively short latency period up to that time (12-21 years).

A report by Greenwald *et al.* (1984) gave the results of a case/control study of STS in New York State. Cases of STS (n = 281) diagnosed between 1962 and 1980, who were between the ages of 18 to 29 during the war in Vietnam, were selected from the state cancer registry. Cases were individually age matched to living controls drawn from drivers' license files. The investigators gathered exposure information from subjects or next of kin by a telephone questionnaire. The questions focused on Vietnam service (and Agent Orange exposure in particular), but included other exposures such as chemical manufacturing and herbicide spraying in general. Only 3% of the cases and 4% of the controls had a history of Agent Orange, dioxin, or 2,4,5-T exposure. None of the various exposures proved statistically significant.

The power of this study can be criticized, with exposures as rare as they were. Also, the inclusion of cancer cases from the early 1960s can be questioned. These cases would not have had sufficient latency to have been caused by an exposure in Vietnam.

In 1983, an Australian Royal Commission began investigating the effects of Agent Orange exposure to Australian Vietnam veterans. However, their report, released in 1985, does not supply much information on the effects of PCDDs. The executive summary concluded that "only a very limited number of Australian servicemen were ever directly exposed," and further, that the dose received by the majority of Australian veterans was "so minute that it may, without doubt, be ignored," (e.g., it noted that no Australians developed chloracne). Not surprisingly, the Commission found no evidence of any cancer excess among the "exposed" servicemen (Royal Commission, 1985).

There are only a few cases where dioxin exposure of the general population has been documented; the Seveso incident in Italy, is one of them. In 1976, a chemical plant producing 2,4,5-trichlorophenol, exploded and released into the air several chemicals including TCDD in the vicinity of Seveso. The Seveso incident represents a unique event in the sense that exposure to the toxic chemical was not limited to occupational exposure by workers but the whole population was affected by the TCDD release in the area surrounding a pentachlorophenol manufacturing facility that experienced an explosion and fire releasing dioxins into the atmosphere. Children, woman and men of various age were exposed to different degrees depending on the distance and direction from the origin of the plume.

Abate *et al.* (1982) summarized the series of studies following the 1976 accidental release of TCDD from a TCP-producing plant in Seveso, Italy. The investigators looked at mortality rates for 11 municipalities for four years after the accident and reported no increase in cancer mortality. These studies served mainly to provide baseline rates for future studies, because clearly not enough time had elapsed to provide the minimum 10 to 20 years required for an increased cancer risk to become manifest (Bruzzi, 1983).

Fifteen years after the industrial accident, Bertazzi *et al.* (1997) examined the cancer mortality among residents (20 to 74 years old) of Seveso by comparing populations living in dioxin contaminated areas (divided into three zones: highest, lower and lowest zone of exposure to dioxin, zone A, B, and R, respectively) with population from neighboring noncontaminated areas (zone nonABR). No increase for all-cancer mortality, or major specific sites like respiratory cancer among males and breast cancer among females, was found. However, other specific cancer mortality was observed and could be associated with dioxin exposure. Table 1 represents cancer mortality for men and women living in zone B.

Increased mortality from stomach cancer (RR = 2.4; 95% CI = 0.8-5.7) was reported 10 years after the accident in women living in zone B. In men, increased mortality from rectal cancer (RR = 6.2; 95% CI = 1.7-15.9) was observed. Leukemia in men represented one of the highest risks seen in zone B for hematologic neoplasms and was statistically significant (RR = 3.1; 95% CI = 1.3-6.4). Multiple myeloma in women (RR = 6.6; 95% CI = 1.8-16.8), and Hodgkin's disease in both genders (RR = 3.3; 95% CI = 0.4-11.9 in men; and RR = 6.5; 95% CI = 0.7-23.5 in women) were also noted in that zone. In the young population (20,000 subjects aged 0 to 19 years old), some cases of cancer were also found (Pesatori *et al.*, 1993). Cancer cases noted included two ovarian cancers and Hodgkin's lymphoma; myeloid leukemia represented the most evident increase although not statistically significant (RR = 2.7; 95% CI = 0.7-11.4). Two cases of thyroid cancer were also reported (RR = 4.6; 95% CI = 0.6-32.7). This observation represents an important result because of its magnitude and its correlation with experimental observations. None of the elevated cancer incidences in zone A, the area with the highest exposure, were statistically significant; however, this area also had the smallest population. Additionally, it should be noted that the Seveso population was exposed to 2-3 orders of magnitude times the level of dioxin normally experienced by the general population of industrialized countries. In 1997, individuals living in the contaminated

area at the time of the accident still experienced high level of plasma TCDD 20 years after the industrial accident in Seveso. Geometric means for plasma TCDD concentration for individuals who lived in zone A, B and nonABR (control zone) in 1976 were 53.2, 11.0 and 4.9 ppt, respectively. Women in these three groups represented the gender with the highest plasma TCDD contamination (Landi et al., 1997). The authors concluded that the results indicate a positive association between dioxin exposure and certain cancers, but further study is needed to clarify this association.

Table 1. Female and male deaths in zone B for selected causes, 1976-1991, ten years or more since first exposure (latency) and duration of exposure (length of stay in contaminated area) (Adapted from Bertazzi et al., 1997).

		Latency > 10 years		Length of stay > 10 years	
		Female	Male	Female	Male
All cancers	OBS	23	31	20	29
	RR	1.4	1.0	1.4	1.1
	(95% CI)	(0.9 – 2.1)	(0.7 – 1.4)	(0.8 – 2.1)	(0.7 – 1.6)
Digestive cancer	OBS	10	12	9	12
	RR	1.5	1.0	1.6	1.2
	(95% CI)	(0.7 – 2.7)	(0.5 – 1.8)	(0.7 – 2.9)	(0.6 – 2.1)
Stomach cancer	OBS	5	X	4	
	RR	2.4	X	2.3	
	(95% CI)	(0.8 – 5.7)		(0.6 – 6.0)	
Lymphatic and hemopoietic	OBS	4	4	3	4
	RR	2.8	2.5	2.4	2.5
	(95% CI)	(0.7 – 7.1)	(0.7 – 6.4)	(0.5 – 7.1)	(0.7 – 6.4)
Multiple myeloma	OBS	3		2	
	RR	15.9		11.0	
	(95% CI)	(3.2 – 46.5)		(1.2 – 39.6)	
Rectal cancer	OBS		4		4
	RR		6.2		7.2
	(95% CI)		(1.7 – 15.9)		(1.9 – 18.4)
Leukemia	OBS		2		2
	RR		3.4		3.9
	(95% CI)		(0.4 – 12.3)		(0.4 – 14.1)

OBS = observed deaths RR = relative risk CI = confidence interval

Animal Studies

Van Miller *et al.* (1977a,b) reported the results of a study in which rats were fed diets containing from 1 ppt to 1 ppm of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) for 78 weeks. Surviving rats were killed after 95 weeks. Laparotomies were performed on all surviving rats at 65 weeks and all tumors were biopsied. Rats in the three highest dose groups, receiving 50 ppb or more, died early. A variety of tumors were found in rats receiving 5 ppt to 5 ppb while no-neoplasms were found in the control or low-dose groups. The absence of tumors in these two groups is unusual in this strain of rats. In

addition, because of the small number of animals in each group (10) the study was inadequate to determine the carcinogenic potential of TCDD.

Toth *et al.* (1979) administered TCDD to male Swiss/H/Riop strain mice by gavage once a week for a year, then followed them for their lifetime. The weekly doses were 0.007, 0.7, and 7.0 µg/kg. Analysis of the results from this study focused on the incidence of liver tumors. A significant increase in the incidence of liver tumors was observed in the intermediate-dose group compared to the four separate control groups. The high-dose group, however, had an incidence of liver tumors that was similar to the control group. This finding may be explained by the early mortality in the high-dose group. The average life span was 424 days for this group, compared to average life spans of between 577 and 651 days for the control groups. If the treated animals had lived it is possible that more tumors may have formed.

Kociba *et al.* (1978) conducted a two-year feeding study in male and female Sprague-Dawley rats given diets containing 2200, 210, or 22 parts per trillion (w/w) TCDD for two years. Consumption of these diets resulted in daily doses of 0.1, 0.01, and 0.001 µg/kg body weight, respectively. There were 50 male and 50 female rats in each treatment group and 86 animals of each sex in the control group. There was a statistically significant ($p < 0.05$) increase in cumulative mortality for the high-dose female group in the latter half of the study. Body weights of the male and female high-dose groups were significantly ($p < 0.05$) reduced for the last three quarters of the study; however, food intake was not altered. The combined incidence of hepatocellular carcinomas and hepatocellular neoplastic nodules in the intermediate and high-dose groups of female rats was increased above the control group. Statistically significant increased incidences of stratified squamous cell carcinomas of the hard palate and/or nasal turbinates were observed in both male and female high-dose groups. The male group also had an increased incidence of squamous cell carcinoma of the tongue, while the female group had an increased incidence of keratinizing squamous cell carcinoma of the lung.

US EPA (1981) reviewed this study and had an independent pathologist, Robert Squire, review the tissue pathology. The incidences of significant tumors reported by Kociba *et al.* (1978) and by Squire (US EPA, 1981) are given in Table 2 for male and female rats. The results of Squire's review did not differ greatly from those reported by Kociba *et al.* (1978).

CDHS staff members concurred with earlier reviewers (IARC 1982, EPA 1984) that the study reported by Kociba *et al.* (1978) was an adequately conducted chronic carcinogenicity bioassay of TCDD, with significant effects observed at the two higher dose levels.

Table 2: Tumor incidences in Osborne-Mendel rats receiving 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD) in the diet for two years (US EPA, 1984)

Tumor type, sex	Dose level (µg/kg-day)			
	0	0.001	0.01	0.1
	Tumor incidence ^a			
Tongue, stratified squamous cell carcinoma male	0/76 (0/77)	1/49 (1/44)	1/49 (1/49)	4/42 (<i>p</i> = 0.015) (3/44) (<i>p</i> = 0.046)
Nasal turbinates/hard palate, squamous cell carcinoma male	0/51 (0/55)	1/34 (1/34)	0/27 (0/26)	4/30 (<i>p</i> = 0.017) (6/30) (<i>p</i> = 0.002)
female	1/54 (0/54)	0/30 (0/30)	1/27 (1/27)	5/24 (<i>p</i> = 0.009) (5/22) (<i>p</i> = 0.001)
lung, keratinizing squamous cell carcinoma female	0/86 (0/86)	0/50 (0/50)	0/49 (0/49)	7/49 (<i>p</i> < 0.001) (8/47) (<i>p</i> < 0.001)
Liver, hepatocellular hyperplastic nodules, carcinomas female	9/86 (16/86)	3/50 (8/50)	18/50 (<i>p</i> < 0.001) (27/50) (<i>p</i> < 0.001)	34/48 (<i>p</i> < 0.001) (33/47) (<i>p</i> < 0.001)

P values determined using Fisher's exact test.

^a Number of animals with tumor over number of animals examined (incidence reported by Kociba *et al.*, 1978). Numbers in parentheses give the incidence reported by Squire (US EPA, 1984).

The National Toxicology Program (NTP 1982a) conducted an oncogenicity bioassay of TCDD in male and female Osborne-Mendel rats. They were administered TCDD in a 9:1 corn oil:acetone vehicle by gavage at dose levels of 0.005, 0.025, or 0.25 µg/kg twice a week for 104 weeks. The treatment groups consisted of 50 rats of each sex and a vehicle control group that was made up of three subgroups of 25 rats of each sex. An untreated control group, also made up of three subgroups of 25 rats of each sex, was included in the study, but not in the statistical analysis of the results by NTP. At the dose levels used, TCDD did not have a significant effect on survival of any treatment group. The high-dose group of male rats did have a statistically-significant increased incidence of subcutaneous tissue fibromas, but it was not considered biologically significant because of the variability found. All male treatment groups had significantly (*p* < 0.05) increased incidences of thyroid follicular cell adenomas or adenomas and carcinomas, although the low- and intermediate-dose level group incidences were not significant when compared to the untreated control group by CDHS staff. The female high-dose group had significantly (*p* < 0.05) increased incidences of several tumor types, including subcutaneous tissue fibrosarcomas, liver neoplastic nodules or hepatocellular carcinomas, and adrenal cortical adenomas. Of these 3 tumors, NTP considered only the liver tumors

to be related to TCDD administration. The incidences of these tumors are given in Table 3. Toxic hepatitis was found in 14 male and 32 female high-dose level rats.

Table 3: Tumor incidences in male and female Osborne-Mendel rats given 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD) by gavage for two years (NTP, 1982a)

Sex, tumor type	Dose level (µg/kg-week)			
	0	0.01	0.05	0.5
Males	Tumor incidence ^a			
Thyroid				
Follicular cell adenoma	1/69	5/48 (<i>p</i> = 0.042)	6/50 (<i>p</i> = 0.021)	10/50 (<i>p</i> = 0.001)
Follicular cell adenoma/carcinoma	1/69	5/48 (<i>p</i> = 0.042)	8/50 (<i>p</i> = 0.004)	11/50 (<i>p</i> < 0.001)
Females				
Subcutaneous tissue, fibrosarcoma	0/75	2/50	3/50	4/49 (<i>p</i> = 0.023) [3] ^b
Liver				
Neoplastic nodules/ hepatocellular carcinoma	5/75	1/49	3/50	14/49 (<i>p</i> = 0.001)
Adrenal				
Cortical adenoma or adenoma NOS	11/73	8/49	4/49	14/46 (<i>p</i> = 0.039)

^a Number of animals with tumor over number of animals examined.

^b Number of animals with hepatocellular carcinoma.

NOS = Not otherwise specified. *P* values determined using Fisher's exact test.

NTP (1982a) also conducted a carcinogenicity bioassay with TCDD in male and female B6C3F₁ hybrid strain mice. The protocol was similar to that used in the rat study with male mice receiving the same doses of TCDD. Female rats, however, received larger doses of 0.02, 0.1 or 1.0 µg/kg twice a week. These dose levels did not have a statistically significant effect on survival of any treatment group. Male mice in the highest dose group had a significantly increased incidence of hepatocellular carcinomas. The high-dose female group had significantly increased incidences of subcutaneous tissue fibrosarcomas, hepatocellular adenomas or carcinomas, and thyroid follicular-cell adenomas. NTP considered only liver tumors and thyroid tumors to be related to TCDD administration. NTP also considered histiocytic lymphomas to have been increased in the high-dose female group; however, the staff of DHS did not consider that these lymphomas were increased when the incidences in all control subgroups were considered. The observed tumor incidences in both male and female mice are given in Table 4. Toxic hepatitis was observed in 44 male and 34 female high-dose group animals. It was also observed in several animals of the other treatment groups.

Table 4: Tumor incidences in male and female B6C3F₁ mice given 2,3,7,8-Tetrachloro-dibenzo-*p*-dioxin (TCDD) by gavage for two years (NTP, 1982a).

Sex, tumor type	Dose level (µg/kg-week) ^a			
	0	0.01 (0.04)	0.05 (0.2)	0.5 (2.0)
	Tumor incidence ^b			
males				
liver (hepatocellular carcinoma)	8/73	9/49	8/49	17/50 (<i>p</i> = 0.002)
Hepatocellular adenoma or carcinoma	15/73	12/49	13/49	27/50 (<i>p</i> < 0.001)
females				
Subcutaneous tissue, fibrosarcoma	1/74	1/50	1/48	5/47 (<i>p</i> = 0.032)
liver, hepatocellular carcinoma	1/73	2/50	2/48	6/47 (<i>p</i> = 0.014)
hepatocellular adenoma or carcinoma	3/73	6/50	6/48	11/47 (<i>p</i> = 0.002)
thyroid, follicular cell adenoma	0/69	3/50	1/47	5/46 (<i>p</i> = 0.009)

P values determined using Fisher's exact test.

^a Dose administered to male mice; dose administered to female mice in parentheses.

^b Number of animals with tumor over number of animals examined.

Both rat and mouse carcinogenicity bioassays conducted by NTP appear to have been done in an adequate manner. The number of treatment groups and the large dose range used in the studies are not typical of NTP bioassays, although it was similar to that used by Kociba *et al.* (1978). However, it may not have been large enough to include a dose level which produced no effect. Most significantly increased tumor incidences only occurred in the high-dose level groups, but a statistically significant dose-related trend was found in all groups.

NTP (1982b) also conducted a dermal oncogenicity bioassay on TCDD in male and female Swiss-Webster mice. TCDD in an acetone suspension was applied to the skin three days per week for 104 weeks. The male rats received 0.001 µg per application and the females received 0.005 µg per application. Separate groups of male and female mice were treated with one application of 50 µg 7,12-dimethylbenz(*a*)anthracene (DMBA) one week prior to the start of TCDD treatments. The only significantly (*p* = 0.01) increased incidences of tumors observed were among female mice. Both the TCDD- and DMBA/TCDD-treated groups had a similar incidences of fibrosarcoma in the integumentary system (8/27 and 8/29, respectively), compared to the vehicle control of 2/41. In NTP's judgment, the results of this experiment indicated that TCDD was carcinogenic.

HexaCDDs have been tested for carcinogenicity by NTP (1980a) in both Osborne-Mendel rats and B6C3F₁ mice. The bioassay tested a mixture of HexaCDDs containing 31 percent 1,2,3,6,7,8-HexaCDD and 67 percent 1,2,3,7,8,9-HexaCDD. Lower chlorinated PCDDs made up the remaining 2% of the mixture, including 0.04 percent TetraCDDs. Male and female rats and male mice received weekly doses of 1.25,

2.5 or 5 µg/kg, administered by gavage twice a week. The female mice were administered doses of 2.5, 5.0, or 10 µg/kg/week.

A dose-related "toxic hepatitis", which was noninflammatory and consisted of degenerative changes in the liver, was observed in treated rats. The treated groups of female rats had significantly increased incidences of liver neoplastic nodules. Four high-dose animals were diagnosed as having hepatocellular carcinoma. The mice also had a dose-related incidence of "toxic hepatitis" and the high-dose male and female mouse groups had statistically significant increased incidences of hepatocellular adenomas and combined incidences of hepatocellular adenomas and carcinomas. The incidences of these tumors are given in Table 5.

Several pathologists have independently evaluated the slides made from the female rat livers in this bioassay. The re-evaluations found fewer neoplastic nodules and carcinomas than did the original evaluation. Although the incidences of neoplastic nodules and carcinomas are probably lower than originally reported, the incidence is still significant in the high-dose group. The results of four separate evaluations of the liver pathology of the female rats are given in Table 6.

A dermal application carcinogenicity bioassay of the same mixture of HexaCDD in male and female Swiss-Webster mice was also conducted by NTP (1980b). This study was similar to the TCDD dermal oncogenicity bioassay in its protocol. Thirty mice of each sex were treated with 0.005 µg of the dioxin mixture three times per week for the first 16 weeks, which was increased to 0.01 µg thereafter. A similar group was initially treated once with 50 µg DMBA before being treated with the HexaCDD mixture. Thirty untreated and 45 vehicle-treated mice of each sex were used as controls. Although there was a slight increase in fibrosarcomas of the integumentary system, this was not considered by NTP to be a significant carcinogenic response. DMBA pretreatment had no additional effect.

DHS staff members agreed with IARC (1982) that there is adequate evidence to support a conclusion that TCDD is carcinogenic to rats and mice and that TCDD should be considered a potential carcinogen to humans. The NTP bioassays (NTP 1980a) of HexaCDDs also indicated that the mixture used was tumorigenic.

Table 5: Tumor incidences in female Osborne-Mendel rats and male and female B6C3F₁ mice given HexaCDD by gavage for two years (NTP, 1980a)

Sex, species, tumor type	Dose level (µg/kg-week)			
	0	1.25 (2.5)	2.5 (5.0)	5.0 (10)
	Tumor incidence			
female rat liver, neoplastic nodule or hepatocellular carcinoma	5/75	10/50 (<i>p</i> = 0.026)	12/50 (<i>p</i> = 0.007)	30/50 (<i>p</i> < 0.001)
male mice				
liver, hepatocellular adenoma	7/73	5/50	9/49	15/4 (<i>p</i> = 0.003)
liver, hepatocellular adenoma or carcinoma	15/73	14/50	14/49	24/48 (<i>p</i> = 0.001)
female mice				
liver, hepatocellular adenoma	2/73	4/48	4/47	9/47 (<i>p</i> = 0.003)
liver, hepatocellular adenoma or carcinoma	3/73	4/48	6/47	10/47 (<i>p</i> = 0.004)

P values determined using Fisher's exact test.

^a Dose administered to male mice; dose administered to female mice in parentheses.

^b Number of animals with tumor over number of animals examined.

Table 6: Incidence of liver tumors based on four separate pathological evaluations of female rats given HexaCDD by gavage for two years^a (CDHS, 1986)

Pathologist and Diagnosis	dose level (µg/kg-week)			
	0	1.25	2.5	5
	Tumor incidence ^b			
NTP (1980) Neoplastic nodules or hepatocellular carcinoma	5/75	10/50 <i>p</i> = 0.026	12/50 <i>p</i> = 0.007	30/50 (4) ^c <i>p</i> < 0.001
Squire (1983) Neoplastic nodules	1/75	4/50	7/50 <i>p</i> = 0.007	7/50 <i>p</i> = 0.007
Haberman and Schueler (Schueler 1983) Neoplastic nodules or hepatocellular carcinoma	NA	NA	NA	17/50 (3) ^d
Hildebrandt (1983) Neoplastic nodules or hepatocellular carcinoma	1/75	5/50 <i>p</i> = 0.037	7/50 <i>p</i> = 0.007	18/50(2) <i>p</i> < 0.001

^a Chi-square test for trend in proportions for NTP, Squire, and Hildebrandt studies significant at $\alpha = 0.05$ level.

^b Number of animals with tumor over number of animals examined.

^c Number of animals diagnosed with hepatocellular carcinoma is shown in parentheses.

^d The diagnosis for nine of the animals with neoplastic nodules was considered a matter of judgment by the pathologist.

NA = Not available.

IV. DERIVATION OF CANCER POTENCY

Basis for Cancer Potency

Several human epidemiological studies of PCDD exposure reviewed in the dioxin TAC document (CDHS, 1986) reported results which suggested an increase in cancer incidence or mortality associated with PCDD exposure (Hardell and Sandstrom, 1979; Ericksson *et al.*, 1981; Zack and Gaffey, 1983). However, these and the other studies described in the dioxin TAC document suffer from a number of limitations. The characterization of exposure to PCDD/PCDF were at best, uncertain. Usually the exposure occurred at a time when there were no sensitive measures of exposure levels. Exposure was often based on job title, self-reported use of substances which may have had PCDD contamination, or exposure to an event thought to have liberated PCDDs. Additionally, none of the human exposures described have been solely to PCDDs or PCDFs, but rather to a mixture of chemicals. PCDDs were only trace contaminants of other toxic chemicals. Many of the occupationally exposed subjects were exposed only briefly (e.g., during an accidental release), or worked in a possibly contaminated environment for a short time. For example, more than 75% of the workers studied by Ott *et al.* (1980) had been exposed for less than one year. Finally, many of the discussed studies, including the four US cohorts, have been hampered by small samples. Studies of only a few hundred subjects lack sufficient power to detect small increases in the risk of rare tumors. For these reasons, DHS staff members concluded that the epidemiologic data available at the time the dioxin TAC document was written provided insufficient information to conclude whether or not PCDDs or PCDFs are human carcinogens.

CDHS (1986) found that the most sensitive species, sex, and site for the induction of cancer by TCDD is the male mouse with hepatocellular adenomas or carcinomas (NTP, 1982a). This response is an order of magnitude greater than the least sensitive species, sex, and site examined, the female mouse subcutaneous fibromas. It is interesting to note that there is less than a four-fold difference in the unit risk between animal species for liver tumors. CDHS therefore developed an inhalation cancer unit risk value for TCDD based on the NTP (1982a) male mouse hepatocellular adenoma/carcinoma tumor data. CDHS also developed an inhalation cancer unit risk value for HexaCDD based on the most sensitive species, sex, and site for the induction of cancer. The data set chosen was the NTP (1980b) female rat liver neoplastic nodule or hepatocellular carcinoma incidence data as evaluated by Hildebrandt (1983).

Methodology

GLOBAL79 was used to fit a linearized multistage procedure to the NTP (1982a) male mouse hepatocellular adenoma/carcinoma tumor data for TCDD, and the NTP (1980b) female rat neoplastic nodule/hepatocellular carcinoma data for HexaCDD as evaluated by Hildebrandt (1983). This procedure provided point estimates of the extra risk for both the maximum likelihood estimate (MLE) and the linearized 95% upper confidence value (UCL). The UCL is calculated by maximizing the linear term of the procedure, or forcing a best fitting linear term if one is not present. This method of calculating the

UCL is consistent both with the expected low-dose linearity and the linear nonthreshold theory of carcinogenesis. The slope of the 95% UCL, q_1^* , is taken as a plausible upper bound of cancer potency of TCDD at low doses.

The animal exposure data (NTP 1980a, 1982a) was converted into equivalent human exposures by applying appropriate scaling factors. The following assumptions were made: Oral and inhalation routes are equivalent, the concentration of TCDD in the air was assumed to be the daily oral dose, the route of exposure does not affect absorption, and there is no difference in metabolism and pharmacokinetics between animals and humans. The total weekly dose levels were averaged over the entire week to get the daily dose level. This procedure assumes that daily dosing of the animals in the NTP studies would have given the same results as did the actual twice weekly dosing schedule. Since the half-life of TCDD is relatively long, both dosing schedules should produce similar concentrations of TCDD in the animal tissues, and therefore would be expected to give similar results. The calculated daily doses are given in Table 7. Human equivalent exposures are listed in Table 8.

Because the animal dose levels for TCDD were converted to human equivalent exposure from inhalation, the 95% UCL, q_1^* , is a measure of the greatest potential excess cancer risk for humans. If the lifetime daily exposure is expressed in $\mu\text{g}/\text{m}^3$, then q_1^* is the excess risk associated with this exposure. Since q_1^* for humans is a unit measure of excess lifetime cancer risk associated with exposure to TCDD, it is termed the unit risk. With the unit risk, the 95% UCL of excess risk may be calculated for any low-level exposure to TCDD by the equation $R = \text{unit risk} \times \text{dose}$, where R is the 95% UCL of excess lifetime cancer risk. The cancer unit risks calculated by CDHS using the above procedure for TCDD and HexaCDD were $38 (\mu\text{g}/\text{m}^3)^{-1}$ and $1 (\mu\text{g}/\text{m}^3)^{-1}$, respectively.

Table 7: Calculated daily dose levels for NTP (1980a, 1982a) TCDD and HexaCDD chronic studies in rats and mice (CDHS, 1986)

Chemical	Animal	Reported Dose Level ($\mu\text{g}/\text{kg}\text{-week}$)	Calculated Dose Level ($\mu\text{g}/\text{kg}\text{-day}$)
TCDD	male and female rats, male mice	0.01	0.0014
		0.05	0.0071
		0.5	0.071
	female mice	0.04	0.0057
		0.2	0.029
		2.0	0.29
HexaCDD	female rats	1.25	0.18
		2.5	0.36
		5.0	0.71
	female mice	2.5	0.36
		5.0	0.71
		10	1.40

Table 8: Calculated equivalent human exposure to TCDD and HexaCDD based on daily animal dose levels from NTP (1980a, 1984a) carcinogenicity studies (CDHS, 1986)

Chemical	Animal	Daily Dose Level ($\mu\text{g}/\text{kg}\text{-day}$)	Airborne Concentration for Equivalent Human Exposure (ng/m^3)
TCDD	female rat (0.45) ^a	0.0014	0.93
		0.0071	4.6
		0.071	46
	male mice (0.048)	0.0014	0.44
		0.0071	2.2
		0.071	22
	female mice (0.04)	0.0057	1.7
		0.029	8.4
		0.29	84
HexaCDD	female rats (0.45) a	0.18	120
		0.36	230
		0.71	460
	female mice (0.04)	0.36	100
		0.71	210
		1.43	420

^a Number in parentheses is animal body weight in kilograms.

CDHS recognized that total PCDD/PCDF in the air is composed of dozens of PCDD and PCDF homologues and isomers. The chemicals in such a mixture are difficult to - quantitate analytically. As a result, usually only total PCDD and total PCDF are measured. In the Air Toxics Hot Spots program, certain dioxin sources are required to perform stack testing and speciate the 2,3,7,8-congeners. Thus, more data are becoming available to adequately characterize the risk from dioxin sources in California.

To estimate cancer risks from such mixtures requires information about: (1) the proportion of each PCDD and PCDF in the mixture, and (2) the carcinogenic potency of each. However, these data are not generally available. The proportion of isomers differs depending on the emission source, and only three isomers had been tested for carcinogenic potency (2,3,7,8-TCDD and a mixture of 1,2,3,6,7,8- and 1,2,3,7,8,9-HexaCDD). It was also recognized that not all 2,3,7,8-isomer PCDDs and PCDFs are equally carcinogenic. The results of the bioassays on TCDD and HexaCDD suggested that carcinogenic potency may decline in homologues more chlorinated than TCDD. It was therefore assumed that PCDDs and PCDFs that are not chlorinated on the 2,3,7,8 positions or do not have at least one ring position open are noncarcinogenic. Additionally, it was also considered that the 2,3,7,8-isomer PentaCDD has a carcinogenic potency equivalent to TCDD, and that 2,3,7,8-isomer HeptaCDD is equivalent in carcinogenic potency to 2,3,7,8-isomer HexaCDD. The potencies for the homologous PCDDs were also used for the PCDFs. Using this approach, the potency of a given concentration of PCDDs would be 2% of the potency of TCDD. The potency of a mixture of PCDFs would be 3% of the potency of TCDD.

Another toxicity equivalency factor (TEF) scheme was developed after 1986 during an international symposium (NATO/CCMS, 1988a,b), and it was adopted by US EPA (US EPA, 1989) and the Department of Toxic Substances Control (DTSC) (DTSC, 1992). The international scheme, referred to as ITEFs, is based on experimental cancer and noncancer data for many 2,3,7,8-PCDDs and 2,3,7,8-PCDFs and on the assumption that the mechanism of all PCDD/PCDF-related biologic effects are based on initial binding to a specific protein, the *Ah* receptor. Because the ITEF scheme incorporated more experimental data from cancer and noncancer studies for more PCDDs/PCDFs than does the CTEF scheme, the replacement of the CTEFs by the ITEFs was considered appropriate for use in risk assessment. This approach also increases uniformity among Cal/EPA guidelines. The TEFs contained in the dioxin TAC (CDHS, 1986) document and the ITEFs are listed in Table 8. The cancer unit risks and potency factors for chlorinated dibenzo-*p*-dioxins and dibenzofurans listed in the 1999 chemical summary and Hot Spots Unit Risk and Cancer Potency Values table (OEHHA, 1999) were generated by applying the appropriate ITEFs to the cancer unit risk and potency factor for 2,3,7,8-TCDD calculated in the dioxin TAC document.

As TEFs for PCDDs and PCDFs were developed, considerable efforts went into the study of quantitative structure activity relationships (QSAR) for polychlorinated biphenyls (PCBs). PCB congeners substituted in the para and at least 2 of the meta positions but not at any of the ortho positions can adopt structural conformations most resembling that of 2,3,7,8-TCDD, therefore have the greatest potency and exert their toxicity through the *Ah* receptor pathway. These coplanar PCB congeners are structurally similar to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin and therefore are termed dioxin-like PCBs. Introduction of one chlorine in the ortho position results in a decrease in toxic potency and PCBs with more than one chlorine in the ortho positions lack some effects exerted by non- and mono-ortho PCBs. These PCB congeners show a different spectrum of toxic effects (Safe, 1994).

In 1991, U.S. EPA considered using the TEF methodology for PCBs. They noted that only a small subset of the 209 PCB congeners elicits dioxin-like activity and meet the criteria for inclusion in the TEF methodology. In an attempt to harmonize TEF schemes for dioxin-like compounds, the World Health Organization - European Center for Environmental Health (WHO-ECEH) and the International Program on Chemical Safety (IPCS) generated a database consisting of almost 1,200 peer-reviewed publications, representing all the available toxicological data for PCBs up to the end of 1993. From a selected number of these publications and based on four inclusion criteria, the WHO-ECEH and the IPCS proposed TEF values for 13 dioxin-like PCBs (Ahlborg *et al.*, 1994). The inclusion criteria are:

1. The compound should show structural similarity to PCDDs and PCDFs.
2. It should bind to the *Ah* receptor.
3. It should induce dioxin-specific biochemical and toxic responses.
4. It should be persistent and accumulate in the food chain.

In addition, the first WHO PCB TEF consultation (Ahlborg *et al.*, 1994) recommended expanding the current database to include all relevant information on PCDDs, PCDFs and other dioxin-like compounds that satisfied the four inclusion criteria.

Some terminologies and definitions applicable to TEFs were reviewed prior to the second WHO-ECEH consultation (van Leeuwen, 1997). The term TEF, used in the past to describe any experimental end point to be compared with TCDD was reconsidered since not all end points are “toxic” end points. For example, end points such as binding to the *Ah* receptor and induction of ethoxyresorufin-O-deethylase (EROD) are mostly considered biological/biochemical responses. Therefore, experimental end points, for which numerical values are compared to the response to TCDD, should be termed “Relative Potency” values (REPs). These REPs could be the result of a single laboratory experiment looking at a single end point. REPs are derived from the available data either used as reported in each publication, or calculated by comparing dose-response curves or ratios of medium effective doses (ED₅₀), median lethal dose (LD₅₀), median effective concentration (EC₅₀) etc. A chemical’s TEF is then derived from all available REPs examined for that compound. Thus, the term TEF is restricted to describe an overall estimate of the order-of-magnitude of the toxicity of a compound relative to the toxicity of TCDD. This estimate is derived by consensus, using careful scientific judgment of all available data (van Leeuwen, 1997; van den Berg *et al.*, 1998). The derivation of TEF consensus using *Ah* receptor-specific end points gives more weight to toxic responses than to biochemical (e.g., enzyme induction) responses and it puts more weight on *in vivo* data than on *in vitro* results. In fact, the weighting order of contributing *in vivo* data was: chronic > subchronic > subacute > acute.

In its most recent consultation in 1997, the WHO-ECEH proposed amendments to the previous NATO/WHO I-TEF scheme (NATO/CCMS, 1989). For revision of the existing mammalian TEFs, the WHO-ECEH committee agreed that if the available information was considered insufficient to warrant a change, the existing value would remain. The suggested WHO₉₇ TEFs for humans and mammals along with the CTEFs and ITEFs are presented in Table 9. Taking advantage of new data and understanding of the underlying mechanisms of toxicity of dioxin-like compounds, the WHO-ECEH’s re-evaluation and extension of the TEF concept lead to the following amendments:

- 1) For 1,2,3,7,8-PeCDD, an increase in TEF value from 0.5 to 1.0 was recommended, based on new *in vivo* tumor promotion data and CYP 1A1/A2 induction potencies from subchronic studies.
- 2) For OCDD, the TEF value was reduced from 0.001 to 0.0001 based on a recalculation of the old data in which exposure versus tissue concentrations were compared (administered dose); originally the TEF was based on body burdens of the chemical following subchronic exposures.
- 3) For OCDF, the TEF value was changed from 0.001 to 0.0001 based on new *in vivo* EROD induction potency values (81) and an expected structural similarity with OCDD; thus, for the *in vivo* situation, a change in analogy with OCDD is recommended.

The Scientific Review Panel on Toxic Air Contaminants (SRP) reviewed and endorsed the use of the WHO₉₇ TEFs in Hot Spots risk assessments at its June 20, 2003 meeting. The cancer unit risks and potency factors for chlorinated dibenzo-*p*-dioxins and dibenzofurans and polychlorinated biphenyls listed in this chemical summary and the Hot Spots Unit Risk and Cancer Potency Values table were generated by applying the appropriate WHO₉₇ TEFs to the cancer unit risk and potency factor for 2,3,7,8-TCDD calculated in the dioxin TAC document.

Table 9: Toxicity equivalency factors for chlorinated dibenzo-*p*-dioxins and dibenzofurans (relative to 2,3,7,8-TCDD)

Congener	California TEF ^a	I-TEF ^b	TEF _{WHO/97} ^c
PCDDs			
2,3,7,8-TCDD	1	1	1
1,2,3,7,8-PeCDD	1	0.5	1
1,2,3,4,7,8-HxCDD	0.03	0.1	0.1
1,2,3,6,7,8-HxCDD	0.03	0.1	0.1
1,2,3,7,8,9-HxCDD	0.03	0.1	0.1
1,2,3,4,6,7,8-HpCDD	0.03	0.01	0.01
1,2,3,4,6,7,8,9-OCDD		0.001	0.0001
PCDFs			
2,3,7,8-TCDF	1	0.1	0.1
1,2,3,7,8-PeCDF	1	0.05	0.05
2,3,4,7,8-PeCDF	1	0.5	0.5
1,2,3,4,7,8-HxCDF	0.03	0.1	0.1
1,2,3,6,7,8-HxCDF	0.03	0.1	0.1
1,2,3,7,8,9-HxCDF	0.03	0.1	0.1
2,3,4,6,7,8-HxCDF	0.03	0.1	0.1
1,2,3,4,6,7,8-HpCDF	0.03	0.01	0.01
1,2,3,4,7,8,9-HpCDF	0.03	0.01	0.01
1,2,3,4,6,7,8,9-OCDF		0.001	0.0001
PCBs (IUPAC #, Structure)			
77	3,3',4,4'-TCB		0.0001
81	3,4,4',5-TCB		0.0001
105	2,3,3',4,4'-PeCB		0.0001
114	2,3,4,4',5-PeCB		0.0005
118	2,3',4,4',5-PeCB		0.0001
123	2',3,4,4',5-PeCB		0.0001
126	3,3',4,4',5-PeCB		0.1
156	2,3,3',4,4',5-HxCB		0.0005
157	2,3,3',4,4',5'-HxCB		0.0005
167	2,3',4,4',5,5'-HxCB		0.00001
169	3,3',4,4',5,5'-HxCB		0.01
189	2,3,3',4,4',5,5'-HpCB		0.0001

Value introduced or changed

^a CDHS, 1986

^b NATO/CCMS, 1989.

^c van Leeuwen, 1997.

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CHRONIC TOXICITY SUMMARY

CHLORINATED DIBENZO-*p*-DIOXINS and CHLORINATED DIBENZOFURANS (INCLUDING 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN)

(Polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) including 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) which is the principal congener of concern based on toxicity)

CAS Registry Number: 1746-01-6 (TCDD); 5120-73-19 (TCDF)

I. Chronic Toxicity Summary

<i>Inhalation reference exposure level</i>	0.00004 µg/m³ (40 pg/m³)
<i>Oral reference exposure level</i>	1 x 10⁻⁸ mg/kg/day (10 pg/kg/day)
<i>Critical effect(s)</i>	Increased mortality, decreased weight gain, depression of erythroid parameters, increased urinary excretion of porphyrins and delta-aminolevulinic acid, increased serum activities of alkaline phosphatase, gamma-glutamyl transferase and glutamic-pyruvic transaminase, gross and histopathological changes in the liver, lymphoid tissue, lung and vascular tissues in rats.
<i>Hazard index target(s)</i>	Alimentary system (liver); reproductive system; development; endocrine system; respiratory system; hematopoietic system

II. Physical and Chemical Properties (HSDB, 1995; 1999)

<i>Description</i>	All are white crystalline powders at 25° C.
<i>Molecular Formula</i>	C ₁₂ H ₄ C ₁₄ O ₂ (TCDD)
<i>Molecular Weight</i>	321.97 g/mol (TCDD)
<i>Density</i>	1.827 g/ml (estimated for TCDD)
<i>Boiling Point</i>	412.2°C (estimated for TCDD)
<i>Melting Point</i>	305-306°C (TCDD)
<i>Vapor Pressure</i>	1.52 x 10 ⁻⁹ torr at 25°C (TCDD)
<i>Solubility</i>	In water: 19.3 ng/L at 22°C (TCDD)
<i>Log K_{ow}</i>	6.15-7.28 (6.8 for TCDD)
<i>(octanol/water partition coefficient)</i>	
<i>Log K_{oc}</i>	6.0-7.39
<i>(organic-carbon distribution coefficient)</i>	
<i>Henry's Law Constant</i>	8.1 x 10 ⁻⁵ ATM-m ³ /mol

III. Major Uses and Sources

The chlorinated dioxins and furans are generated as by-products from various combustion and chemical processes. PCDDs are produced during incomplete combustion of chlorine containing wastes like municipal solid waste, sewage sludge, and hospital and hazardous wastes. Various metallurgical processes involving heat, and burning of coal, wood, petroleum products and used tires for energy generation also generate PCDDs. Chemical manufacturing of chlorinated phenols (e.g., pentachlorophenol), polychlorinated biphenyls (PCBs), the phenoxy herbicides (e.g., 2,4,5 T), chlorinated benzenes, chlorinated aliphatic compounds, chlorinated catalysts and halogenated diphenyl ethers are known to generate PCDDs as a by-product under certain conditions. While manufacture of many of these compounds and formulations has been discontinued in the United States, continued manufacture elsewhere in the world combined with use and disposal of products containing PCDD by-products results in the inadvertent release of PCDDs into the environment. Industrial and municipal processes in which naturally occurring phenolic compounds are chlorinated can produce PCDDs; the best example is chlorine bleaching of wood pulp in the manufacture of paper products. Additionally, municipal sewage sludge has been documented to occasionally contain PCDDs and PCDFs. Annual statewide industrial emissions from facilities reporting under the Air Toxics Hot Spots Act in California based on the most recent inventory were estimated to be 0.123 pounds of 2,3,7,8-TCDD, 0.244 pounds of 1,2,3,4,7,8-hexachlorodibenzodioxin and lesser amounts of other polychlorinated dibenzodioxins and dibenzofurans (CARB, 1999).

IIIa. 2,3,7,8 Tetrachlorodibenzo-p-dioxin Toxic Equivalent

2,3,7,8-Tetrachlorodibenzo-p-dioxin is considered the most potent congener of the polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) families of compounds. Potency of PCDD and PCDF congeners correlates with the binding affinity to the cytosolic Ah receptor. Structure activity studies have demonstrated that optimal biological activity and Ah-receptor binding requires congeners with a planar conformation and chlorines at the corners of the molecule at the 2,3,7,8 positions (Poland and Knutson, 1982; Safe, 1986). Chlorines at both ortho positions in these molecules (i.e., positions 1 and 9) sterically hinder a planar conformation that lessens the congeners' biological activity. Thus only 15 of 210 different PCDDs and PCDFs congeners possess significant biological activity based on chlorines in the 2,3,7,8 positions and some degree of planar conformation (Safe, 1986; U.S. EPA 1989). These include two tetrachloro-congeners: 2,3,7,8-tetrachlorodibenzo-p-dioxin and 2,3,7,8-tetrachlorodibenzofuran; three pentachloro congeners: 1,2,3,7,8-pentachlorodibenzo-p-dioxin, 1,2,3,7,8-pentachlorodibenzofuran, and 2,3,4,7,8-pentachlorodibenzofuran; seven hexachloro congeners: 1,2,3,4,7,8 or 1,2,3,6,7,8 or 1,2,3,7,8,9-hexachlorodibenzo-p-dioxins and hexachlorodibenzofurans and 2,3,4,6,7,8-hexachlorodibenzofuran; and three heptachloro congeners: 1,2,3,4,6,7,8-heptachlorodibenzo-p-dioxin, 1,2,3,4,6,7,8-heptachlorodibenzofuran and 1,2,3,4,7,8,9-heptachlorodibenzofuran (U.S. EPA, 1989). The structures of the dibenzo-p-dioxins and dibenzofurans along with their numbering schemes are shown in Figure 1. Toxic equivalents are calculated relative to the most potent congener, 2,3,7,8-tetrachlorodibenzo-p-dioxin, and are determined based on structure activity studies examining relative affinity for the

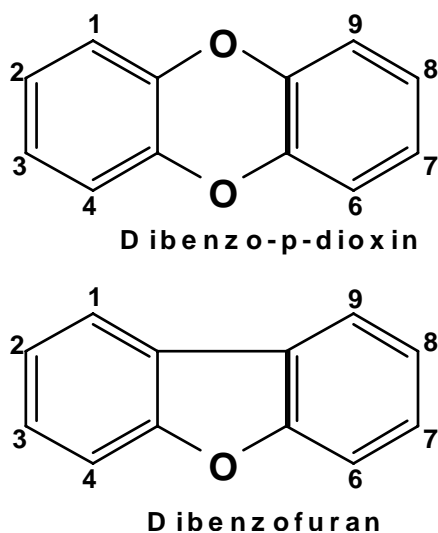
Ah receptor as well as on relative toxicity of different congeners. Values for the international system of toxic equivalents are provided in Table 1 (U.S. EPA, 1989).

Table 1. International Toxic Equivalency Factors (I-TEFs) for PCDDs and PCDFs Chlorinated in the 2,3,7, and 8 Positions. (U.S. EPA 1989.)

Compound ^{1,2}	I-TEF
Mono-, Di-, and Tri-CDDs and CDFs	0
<u>TetraCDD</u>	
2,3,7,8-substituted	1.0
Others	0
<u>PentaCDD</u>	
2,3,7,8-substituted	0.5
Others	0
<u>HexaCDD</u>	
2,3,7,8-substituted	0.1
Others	0
<u>HeptaCDD</u>	
2,3,7,8-substituted	0.01
Others	0
<u>OctaCDD</u>	0.001
<u>TetraCDF</u>	
<u>2,3,7,8</u>	0.1
<u>Others</u>	0
<u>PentaCDF</u>	
1,2,3,7,8-PentaCDF	0.05
2,3,4,7,8-PentaCDF	0.5
others	0
<u>HexaCDF</u>	
2,3,7,8-substituted	0.1
Others	0
<u>HeptaCDF</u>	
2,3,7,8-substituted	0.01
Others	0
<u>OctaCDF</u>	0.001

¹ CDD designates chlorinated dibenzo-p-dioxin

² CDF designates chlorinated dibenzofuran

Figure 1. Structures of the Dibenzo-p-dioxins and Dibenzofurans

IV. Effects of Human Exposure

The information available on possible chronic toxic effects in humans is complicated by the relative insensitivity of epidemiological studies, the limited ability of case studies of exposed individuals to establish cause and effect relationships, the heterogeneous nature of human populations, the broad spectrum of exposures to other toxic agents in the human environment, and the episodic exposure of many of the exposed human populations which have been studied (e.g., Seveso, Italy). As a result, a limited number of effects have been associated with exposure to dioxins in humans. The meaning of these effects in terms of toxicity in most cases remains to be clarified. The majority of information comes from cross-sectional medical studies.

Chloracne is the most widely recognized effect of exposure to 2,3,7,8-TCDD and TCDD-like PCDDs and PCDFs. Chloracne is a persistent condition, which is characterized by comedones, keratin cysts and inflamed papules and is seen after acute and chronic exposure to various chlorinated aromatic compounds (Moses and Prioleau, 1985). Other dermal effects include hyperpigmentation and hirsutism or hypertrichosis (Jirasek *et al.*, 1974; Goldman, 1972; Suskind *et al.*, 1953; Ashe and Suskind, 1950); both appear to resolve themselves more quickly over time than chloracne, making them more of an acute response rather than a chronic response (U.S. EPA, 1994a). Epidemiological data available for 2,3,7,8-TCDD have not allowed a determination of the threshold dose required for production of chloracne (U.S. EPA, 1994b). Case studies suggest that there may be a relationship between 2,3,7,8-TCDD exposure and hepatomegaly (Reggiani, 1980; Jirasek *et al.*, 1974; Suskind *et al.*, 1953; Ashe and Suskind, 1950) and hepatic enzyme changes (Mocarelli *et al.*, 1986; May, 1982; Martin 1984; Moses *et al.*, 1984). Nevertheless, cross sectional epidemiological studies of trichlorophenol (TCP) production workers (Suskind and Hertzberg., 1984; Bond *et al.*, 1983; Moses *et al.*, 1984; Calvert *et al.* 1992), Vietnam veterans (Centers for Disease Control Vietnam Experience Study, 1988; Roegner *et al.*, 1991) and Missouri residents (Webb *et al.*, 1989; Hoffman *et al.*, 1986)

found little evidence for an association between exposure and hepatomegaly suggesting that this is not a chronic response. There is a consistent pattern of increased levels of serum gamma glutamyl transferase in populations exposed to 2,3,7,8-TCDD which is presumably of hepatic origin (Mocarelli, 1986; Caramaschi *et al.*, 1981, May, 1982; Martin, 1984; Moses *et al.*, 1984; Calvert *et al.*, 1992; Centers For Disease Control Vietnam Experience Study, 1988). Two cross sectional studies have associated diabetes and elevated fasting serum glucose levels with relatively high serum 2,3,7,8-TCDD levels (Sweeney *et al.*, 1992; Roegner *et al.*, 1991). However other studies provided mixed results (Moses *et al.*, 1984; Centers for Disease Control Vietnam Experience Study, 1988; Ott *et al.*, 1993). TCDD has been associated with effects on reproductive hormonal status in males. The likelihood of abnormally low testosterone levels was 2 to 4 times greater in individuals with serum 2,3,7,8-TCDD levels above 20 pg/ml (Egeland *et al.* 1994) and increased serum levels of luteinizing hormone and follicle stimulating hormone have been documented (Egeland *et al.*, 1994). A number of other effects have been reported that were either not seen as chronic effects or effects seen long term in only one population of exposed persons. These include elevated liver enzymes (aspartate aminotransferase and alanine aminotransferase), pulmonary disorders, neurologic disorders, and changes in porphyrin metabolism and kidney disorders (U.S. EPA, 1994c). Areas in which there is presently insufficient information to draw solid conclusions include effects on the circulatory system, reproductive effects, immunological effects, effects on metabolism and handling of lipids, and on thyroid function (U.S. EPA, 1994c). Recent findings in Rhesus monkeys have shown 2,3,7,8-TCDD to cause endometriosis (Reier *et al.*, 1993) and epidemiological studies are currently underway to determine if there is an association between TCDD exposure and endometriosis in human populations exposed by the Seveso accident.

Potential effects of a toxicant on normal fetal development include fetal death, growth retardation, structural malformations and organ system dysfunction. Evidence for all four of these responses has been seen in human populations exposed to dioxin-like compounds. In these poisoning episodes populations were exposed to a complex mixture of halogenated aromatic hydrocarbons contained within PCBs, PCDFs and PCDDs mixtures thus limiting the conclusions that could be drawn from the data. In the Yusho and Yu-Cheng poisoning episodes, human populations consumed rice oil contaminated with PCBs, PCDFs and PCDDs. Yu-Cheng women experienced high perinatal mortality in hyperpigmented infants born to affected mothers (Hsu *et al.* 1985). This occurred in women with overt signs of toxicity (chloracne) (Rogan, 1982) and Rogan notes that, when there is no sign of toxicity in the mother, the likelihood of fetotoxicity appears to lessen considerably in the infants. Signs of toxicity from dioxin like compounds were absent in infants born to mothers apparently not affected in the Seveso, Italy and Times Beach, Missouri, incidents (Reggiani, 1989; Hoffman and Stehr-Green, 1989), which supports Rogan's conclusion. There was an increased incidence of decreased birth weight in infants born to affected mothers in the Yusho and Yu-Cheng incidents suggesting fetal growth retardation (Wong and Huang, 1981; Law *et al.*, 1981; Lan *et al.*, 1989; Rogan *et al.*, 1988). The structural malformation, rocker bottom heel, was observed in Yusho infants (Yamashita and Hayashi, 1985) making this malformation a possible result of exposure to dioxin-like compounds. Nevertheless, it is unknown if these compounds produce malformations in humans. Evidence for possible organ system dysfunction in humans comes from a study of Yu-Cheng children which found that children exposed in utero experienced delays in attaining developmental milestones, and exhibited neurobehavioral abnormalities (Rogan *et al.*, 1988)

suggesting involvement of CNS function. Dysfunction of dermal tissues is noted in exposed infants of the Yusho and Yu-Cheng incidents and is characterized by hyperpigmentation of the skin, fingernails, and toenails, hypersecretion of the meibomian glands, and premature tooth eruption (Taki *et al.*, 1969; Yamaguchi *et al.*, 1971; Funatsu *et al.*, 1971; Wong and Huang, 1981; Hsu *et al.*, 1985; Yamashita and Hayashi, 1985; Rogan *et al.*, 1988; Rogan, 1989; Lan *et al.*, 1989).

V. Effects of Animal Exposure

The toxicity to laboratory animals encompasses a number of areas including changes in energy metabolism manifested as wasting syndrome, hepatotoxicity, effects on tissue of epithelial origin, various endocrine effects, effects on vitamin A storage and use, immune system effects and reproductive and developmental toxicity. The limited number of chronic studies available do not examine all these endpoints. Therefore subchronic exposures are included here in order to provide a more complete coverage of potential chronic toxic effects of these compounds.

Wasting syndrome is one of the most broadly occurring toxic effects. The wasting syndrome is characterized by loss of adipose tissue and lean muscle mass and is produced in all species and strains tested, but there are difference in sensitivity (U.S. EPA 1994d; Peterson *et al.*, 1984; Max and Silbergeld, 1987). Numerous studies have not yet established the mechanism of wasting syndrome (U.S. EPA, 1994e). Hepatotoxicity is also seen in all species tested, but there is considerable variation in species sensitivity (U.S. EPA, 1994d). TCDD induces hyperplasia and hypertrophy of liver parenchymal cells. Morphological and biochemical changes in the liver include increased SGOT and SGPT, induction of microsomal monooxygenases and proliferation of the smooth endoplasmic reticulum, porphyria, increased regenerative DNA synthesis, hyperlipidemia, hyperbilirubinemia, hypercholesterolemia, hyperproteinemia, degenerative and necrotic changes, mononuclear cell infiltration, multinucleated giant hepatocytes, increased numbers of mitotic figures, and parenchymal cell necrosis (U.S. EPA, 1994d; WHO/IPCS, 1989). Epithelial effects seen include chloracne (rabbit ear and the hairless mouse) (Jones and Krizek, 1962; Schwetz *et al.*, 1973) and hyperplasia and/or metaplasia of gastric mucosa, intestinal mucosa, the urinary tract, the bile duct and the gall bladder (U.S. EPA 1994f). TCDD exposure results in endocrine like effects including epidermal growth factor like effects such as early eye opening and incisor eruption in the mouse neonate (Madhukar *et al.*, 1984), glucocorticoid like effects such as involution of lymphoid tissues (U.S. EPA, 1994g; Sunahara *et al.*, 1989), alteration in thyroid hormone levels and in some cases thyroid hormone like effects (WHO/IPCS, 1989; Rozman *et al.*, 1984), decreases in serum testosterone and dihydrotestosterone (Mittler *et al.*, 1984; Keys *et al.*, 1985; Moore and Peterson, 1985), and changes in arachidonic acid metabolism and prostaglandin synthesis (Quilley and Rifkind, 1986; Rifkind *et al.*, 1990). TCDD is known to decrease hepatic vitamin A storage (Thunberg *et al.*, 1979). TCDD and other dioxin like PCDDs and PCDFs are potent suppressors of both cellular and humoral immune system function, characteristically producing thymic involution at low doses and involution of other lymphoid tissues at higher doses (U.S. EPA 1994h).

In animal studies there is a large body of information available documenting both developmental and reproductive toxicity of 2,3,7,8-TCDD and other PCDDs and PCDFs. These compounds are

acutely toxic to early life stages of fish and birds with fish being most sensitive (LD₅₀ of 0.4 µg/kg for rainbow trout sac fry eggs and LD₅₀ of 34 ng/kg for lake trout eggs); some species of birds are also relatively sensitive (LD₅₀ of 0.25 µg/kg for chicken eggs) (Peterson *et al.*, 1993). 2,3,7,8-TCDD has been documented to increase the incidence of prenatal mortality in a number of species of laboratory animals including the Rhesus monkey, Guinea pig, rabbit, rat, hamster, and mouse (Peterson *et al.*, 1993). Exposure to 2,3,7,8-TCDD during gestation produces a characteristic set of fetotoxic responses in most laboratory animals which includes: thymic hypoplasia, subcutaneous edema, and decreased growth (Peterson *et al.*, 1993). More species specific responses include cleft palate formation in the mouse at doses below maternal toxicity (Moore *et al.*, 1973; Smith *et al.*, 1976; Couture *et al.*, 1990), intestinal hemorrhage in the rat (Sparschu *et al.*, 1971), hydronephrosis in the mouse and hamster (Moore *et al.*, 1973; Smith *et al.*, 1976; Couture *et al.*, 1990; Birnbaum *et al.*, 1989; Olson *et al.*, 1990), and extra ribs in the rabbit (Giavini *et al.*, 1982). Female rats have also been found to be affected by perinatal exposure to 2,3,7,8-TCDD with clefting of the clitoris, incomplete or absent vaginal opening and a smaller vaginal orifice after a dose of 1 µg/kg to the mother on day 15 of gestation (Gray *et al.*, 1993).

A number of effects on adult reproductive function are seen in male animals exposed in utero to 2,3,7,8-TCDD. TCDD reduces plasma androgen levels in the adult male rat and perinatal exposure decreases spermatogenesis, spermatogenic function and reproductive capability, feminizes male sexual behavior, and feminizes male gonadotrophic function (LH secretion) (Mably *et al.*, 1991; Mably *et al.*, 1992a,b,c). Evidence suggests that these effects are the result of impaired sexual differentiation of the CNS, which in male rats is dependent on exposure of the developing brain to testosterone.

There are numerous studies detailing the effects of the PCDDs, PCDFs and other dioxin like compounds, however a large number of these studies were conducted as either acute or subchronic exposures, studies in which it is unlikely that body burdens had reached steady state levels. Detailed below are three chronic studies that were considered in the setting of a chronic toxicity exposure level.

The most definitive study of chronic toxicity in rats is that of Kociba *et al.* (1978). This study involved the administration of 2,3,7,8-TCDD in the diet at doses of 1 ng/kg/day, 10 ng/kg/day, and 100 ng/kg/day to groups of 50 male and 50 female Sprague Dawley rats for two years. A group of 86 male and 86 female rats received diet with solvent vehicle alone and served as controls. The following observations (excluding carcinogenic effects) were seen at the 100 ng/kg/day dose: increased mortality, decreased weight gain, depressed erythroid values, increased urinary excretion of porphyrins and delta-aminolevulinic acid, and increased serum activities of alkaline phosphatase, gamma-glutamyl transferase, and glutamic-pyruvic transaminase. Histopathologic changes were noted in the liver, lymphoid tissue, respiratory and vascular tissues. The primary ultrastructural change in the liver was proliferation of the rough endoplasmic reticulum. At the 10 ng/kg/day dose the severity of toxic symptoms was less than that of the 100 ng/kg/day dose and included increased urinary excretion of porphyrins in females as well as liver and lung lesions. The 1 ng/kg/day dose produced no discernible significant toxic effects. Interpretation of this study by the authors was that the 1 ng/kg/day dose was a NOAEL.

Two chronic toxicity studies are available in the mouse. The first is a one year study conducted by Toth *et al.* (1979) using male Swiss mice administered weekly oral doses of 7, 700, and 7000 ng/kg/day. In this study 2,3,7,8-TCDD administration resulted in amyloidosis and dermatitis in 0 of 38 control animals, 5 of 44 animals receiving 7 ng/kg/day, 10 of 44 animals receiving 700 ng/kg/day and 17 of 43 animals receiving 7,000 ng/kg/day. The other study was from the NTP 1982 gavage study (NTP, 1982) in B6C3F1 mice. This study employed groups of 50 male and 50 female mice. The males received doses of 0, 10, 50, and 500 ng/kg/week by gavage for two years while female mice received doses of 0, 40, 200, and 2000 ng/kg/week by gavage for two years. No adverse effects were seen at the lowest doses tested in each sex, which correspond to NOAELs of approximately 1.4 and 6 ng/kg/day for males and females, respectively. Neither chronic toxicity study in mice reported data on enzyme activity.

VI. Derivation of Chronic Reference Exposure Level (REL)

<i>Study</i>	Kociba <i>et al.</i> (1978)
<i>Study population</i>	Sprague-Dawley rats of both sexes (50/treatment group/sex)
<i>Exposure method</i>	Continuous dietary exposure starting at seven weeks of age for 2 years
<i>Critical effects</i>	Increased mortality, decreased weight gain, depression of hematologic measures, increased urinary excretion of porphyrins and delta-aminolevulinic acid, increased serum activities of alkaline phosphatase, gamma-glutamyl transferase and glutamic-pyruvic transaminase, gross and histopathological changes in the liver, lymphoid tissue, lung and vascular tissues
<i>Observed LOAEL</i>	210 ppt in diet (0.01 µg/kg/day)
<i>Observed NOAEL</i>	22 ppt in diet (0.001 µg/kg/day)
<i>Exposure continuity</i>	Continuous exposure via the diet
<i>Exposure duration</i>	2 years
<i>Subchronic uncertainty factor</i>	1
<i>LOAEL uncertainty factor</i>	1
<i>Interspecies uncertainty factor</i>	10
<i>Intraspecies uncertainty factor</i>	10
<i>Cumulative uncertainty factor</i>	100
<i>Oral reference exposure level</i>	10 pg/kg/day
<i>Route-to-route extrapolation</i>	3,500 µg/m ³ per mg/kg/day
<i>Inhalation reference exposure level</i>	40 pg/m ³ (0.00004 µg/m ³)

The data available for chronic toxic effects in humans have a number of limitations. Some studies did not determine the body burden of compounds necessary to estimate dose.; The Yusho and Yu-Cheng poisoning episodes have uncertainty because exposure was to complex mixtures of halogenated aromatic hydrocarbons rather than to individual congeners. And epidemiological

studies and case studies have limitations in determining cause and effect relationships. Therefore, an animal study was chosen for determination of a NOAEL/LOAEL. The study chosen for use was that of Kociba *et al.* (1978), based on the duration of the study (2 years), the number of animals employed (50 per treatment group per sex), testing of both sexes, a dose range, which spanned from an apparent NOAEL to severe hepatic effects including carcinogenic effects, a complete histopathological examination of all organ systems, examination of urinary excretion of porphyrins and delta-aminolevulinic acid, and determination of serum activities of alkaline phosphatase, gamma-glutamyl transferase, and glutamic-pyruvic transaminase. The elevation of human serum values for gamma-glutamyl transferase is one of the consistently seen chronic responses in exposed human populations and reflects changes in liver biochemistry. Thus the examination of markers of liver toxicity also altered in animal models of chronic toxicity make the Kociba study an appropriate choice for detecting potential chronic toxic effects of 2,3,7,8-TCDD in humans. The NOAEL in the Kociba *et al.* (1978) study was determined to be 1 ng/kg body weight/day. For the purposes of determining the REL the 1 ng/kg/day dose was considered to be a NOAEL based upon the observations of Kociba *et al.* (1978).

VII. Data Strengths and Limitations for Development of the REL

NOAELs from a number of other studies compare favorably with the 1 ng/kg/day NOAEL. These include the NOAEL from the NTP (1982) study in B6C3F1 mice and the NOEL for enzyme induction in rats and marmosets calculated by Neubert (1991) of 1 ng/kg. Furthermore the 1 ng/kg/day NOAEL is lower than the LOAELs observed by Toth *et al.* (1979) of 7 ng/kg/day in mice and by Schantz *et al.* (1978) of 2.3 ng/kg/day in rhesus monkeys. Current exposure assessments for 2,3,7,8-TCDD and other dioxin-like compounds including the PCBs, PCDDs, and PCDFs estimate that the average daily background dose in the U.S. is 3-6 pg TEQ/kg/day (U.S. EPA 1994i) also placing the REL close to background exposures. The REL of 10 pg/kg/day should be protective of chronic effects on liver function and avoid significant increases in exposure over the background level of human exposure.

The strengths of the inhalation REL include the availability of chronic exposure data from a well-conducted study with histopathological analysis, the observation of a NOAEL, and the demonstration of a dose-response relationship. Major areas of uncertainty are the lack of adequate human exposure data and the lack of chronic inhalation exposure studies.

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FORMALDEHYDE

CAS No: 50-00-0

I. PHYSICAL AND CHEMICAL PROPERTIES (HSDB, 1998)

Molecular weight	30.03
Boiling point	-19.5°C
Melting point	-92°C
Vapor pressure	1.08 torr @ 26.1°C
Air concentration conversion	1 ppm = 1.24 mg/m ³ @ 25°C

II. HEALTH ASSESSMENT VALUES

Unit Risk Factor:	6.0 E-6 (µg/m ³) ⁻¹
Slope Factor:	2.1 E-2 (mg/kg-day) ⁻¹

[Rat nasal squamous carcinoma incidence data (Kerns *et al.*, 1983; U.S. EPA 1987), linearized multistage procedure (OEHHA, 1992), with pharmacokinetic interpolation of molecular dosimetry data to the tumor incidence data.]

III. CARCINOGENIC EFFECTS

Human Studies

Epidemiological studies have shown formaldehyde exposure to be significantly associated with cancer at sites in the respiratory tract in workers and in the general population. Studies of embalmers, who have used formaldehyde, have shown increased rates of brain cancer and of leukemia.

Many studies in the epidemiological literature support a link between formaldehyde and elevated risk of cancers of the upper respiratory tract. Among the industrial cohort studies, Stayner (1988) reported a relative risk of 3.4 (90% CI: 1.2-7.9) for buccal cancer, and Blair *et al.* (1986) reported a relative risk of 3.00 (90% CI: 1.30-5.92) for nasopharyngeal cancer. Among industrial proportional mortality studies, Liebling *et al.* (1984) reported a relative risk of 8.70 (90% CI: 1.50-27.33) for buccal/pharyngeal cancer and Stayner *et al.* (1985) reported a relative risk of 7.5 (90% CI: 2.0-19) for buccal cancer. In all of these studies the elevated risk was statistically significant. The population-based case control studies reported statistically significant relationships between formaldehyde exposure and upper respiratory cancers in three studies (Vaughan *et al.*, 1986a, b; Hayes *et al.*, 1986; Olsen *et al.*, 1984), although these cancers can appear in any of several sites.

In a subsequent report Blair *et al.* (1987) presented a summary of a further analysis resulting in a significant association between nasopharyngeal cancer and simultaneous exposure to formaldehyde and to particulate, indicating that such exposure may be a risk factor. Collins *et al.* (1988) have critiqued this finding and have added data.

The three largest - and therefore potentially most sensitive - industrial cohort studies reported elevated rates of lung cancer. The largest, Blair *et al.* (1986) with 26,561 U.S. workers, reported a statistically elevated death rate due to lung cancer, equivalent to 35% above the national average. The other two studies reporting elevated death rates due to lung cancer were Acheson *et al.* (1984a, b) with 7,680 British male workers, mostly young, and Stayner *et al.* (1988) with 11,030 U.S. workers, predominantly female. Some of the categories in the Acheson study showed statistically significant increases of lung cancer. The Stayner study found lung cancer to be elevated 14% overall, which was not statistically significant, but the exposures were well below those of the other two studies.

In the Blair *et al.* (1986) study the investigators concluded that a causal relationship between formaldehyde exposure and lung cancer was unlikely because of a lack of dose gradient for those tumors. Sterling and Weinkam (1988, 1989a, b) performed a reanalysis on the basis that Blair *et al.* (1986) failed to account for a “healthy-worker” effect in the original report. These corrected results showed that lung cancer was related to formaldehyde exposure in a dose-dependent manner, which was statistically significant. In a subsequent analysis of the same workers Blair *et al.* (1990) concluded that exposure to phenol, melamine, urea, and wood dust and other substances might account for some or all of the excess lung cancer observed.

Table 1: Cohort study on industrial exposure to formaldehyde (Blair *et al.*, 1986).

Exposure	Cancer Site	Number Observed	Number Exposed	SMR	90% Confidence Interval	
					<i>Lower</i>	<i>Upper</i>
0.1 - > 2.0 ppm time weighted average	brain	17	21	0.81	0.52	1.21
	leukemia	19	24	0.80	0.52	1.16
	buccal/pharynx	18	19	0.96	0.61	1.41
	lung	201	182	1.11	0.98	1.24
	larynx	12	8	1.42	0.87	2.43
	nasal	2	2.2	0.91	0.16	2.86
> 0 - 5.5 ppm-yr	lung, 20 yr latency	146	108	1.35	1.17	1.55
	hypopharynx	1	1.7	0.59	0.02	2.78
	nasopharynx	6	2.0	3.00	1.30	5.92
	oropharynx	5	2.6	1.92	0.76	4.04

Source: OEHHA (1992)

Recent epidemiological studies contribute to the conclusions only marginally. Gerin *et al.* (1989) presented the results of a large case control study with 3,726 cancer patients. The odds ratio for the highest exposure group with adenocarcinoma of the lung was nearly significant at the 95% confidence level, and there was an apparent trend of incidence of this cancer with exposure. Nevertheless, the authors concluded that there was no persuasive evidence of an increased risk of any type of cancer among men exposed to the exposure levels of formaldehyde cited by Blair *et al.* (1986) (Table 1). The study did not consider cancers of the nasal cavity, of the brain, or of leukemia. Bertazzi *et al.* (1989) presented an extension of a previous study (Bertazzi *et al.*, 1986) which had detected elevated lung cancer among 1,332 workers in a resin

manufacturing plant subject to formaldehyde exposure. In the extended study with more accurate estimates of exposure, the lung cancer rate was not elevated above expected for those exposed to formaldehyde (Bertazzi *et al.*, 1989). Linos *et al.* (1990) reported elevated rates of follicular non-Hodgkin's lymphoma and of acute myeloid leukemia among embalmers and funeral directors in a population-based case control study. The investigators did not attribute these tumors to formaldehyde exposure. Malker *et al.* (1990) found significantly elevated rates of incidence of nasopharyngeal cancer among workers in fiberboard plants and among book binders, both being subject to formaldehyde exposure.

Four recent occupational studies have investigated the relationship of formaldehyde exposure to histological changes, some of which are potentially precancerous lesions, in the nasal mucosa. Holmstrom *et al.* (1989) found that workers exposed to well-defined levels of formaldehyde developed significant changes in the middle turbinate, while those exposed to both formaldehyde and wood dust did not. Boysen *et al.* (1990) found in nasal biopses that workers exposed to formaldehyde showed a significantly higher degree of metaplastic alterations. Edling *et al.* (1988) found significant histological differences in the nasal mucosa of formaldehyde workers compared to unexposed workers but found no histological differences between those exposed to formaldehyde and those exposed to formaldehyde and wood dust. Berke (1987) found no statistical relationship between exfoliated nasal cells in formaldehyde-exposed workers and control groups. Thus, these studies provide some indication of possible histologic change due to formaldehyde exposure in humans, consistent with results in animals.

Animal Studies

A study sponsored by the Chemical Industry Institute for Toxicology (CIIT) has provided the most quantitatively useful evidence for the carcinogenicity of formaldehyde (Swenberg *et al.*, 1980a, b; Kerns *et al.*, 1983). This study used 120 male and 120 female Fischer-344 rats in each dose group, including a clean air group. The adjusted tumor incidences (adjusted for competing causes of death, including scheduled interim sacrifices) for squamous cell carcinomas in the nasal passages of males and females combined, when exposed to 0, 2.0, 5.6, or 14.3 ppm formaldehyde for 6 hours/day, 5 days/week for up to 24 months, were 0/156, 0/159, 2/153 and 94/140 (U.S. EPA, 1987). In an analogous study on mice, two mice in the high dose group (14.3 ppm) developed squamous cell carcinomas, a finding that was not statistically significant but was thought to be biologically significant due to the absence of this tumor in control animals and to concurrence with rat studies. Kerns *et al.* (1983) also reported benign tumors, including polypoid adenomas and squamous cell papillomas. Swenberg *et al.* (1980a, b) described a number of additional lesions in the nasal turbinates of rats exposed to formaldehyde for 18 months, including rhinitis, epithelial dysplasia and hyperplasia, squamous hyperplasia, and cellular atypia that occurred in a dose-related manner. Other inhalation studies (Albert *et al.*, 1982; Tobe *et al.*, 1985) have provided positive evidence for the carcinogenicity of formaldehyde.

Recent investigations of chronic toxicity have shown formaldehyde administered orally for 24 months to be carcinogenic in Sprague-Dawley rats but not in Wistar rats. Soffritti *et al.* (1989), using six exposure groups each of 50 male and 50 female Sprague-Dawley rats, with drinking water concentration of 10 to 1500 mg/L formaldehyde, reported increases in the percent of

animals bearing leukemias and gastrointestinal neoplasias at the higher exposures. Til *et al.* (1989), using three exposure groups, each of 70 male and 70 female Wistar rats, with drinking water concentrations of 20 to 1900 mg/L, reported numerous pathological changes at the highest exposure level, but no evidence of carcinogenicity at any level. Tobe *et al.* (1989), using three exposure groups, each of 20 male and 20 female Wistar rats, with drinking water concentrations of 200 to 5000 mg/L, also reported pathological changes at the highest exposures level but no significant increases in the incidence of any tumor in these small treatment groups. In a letter to the editor, Feron *et al.* (1990) questioned the conclusions and some methods of Soffritti *et al.* (1989).

Other types of exposures have produced a spectrum of results. Watanabe *et al.* (1954) presented a brief preliminary report of experimentally inducing sarcomas by repeated injections of an aqueous solution of formaldehyde in rats. Muller *et al.* (1978) induced a preneoplastic lesion of the oral mucosa by repeated exposure to formalin solution in rabbits. Homma *et al.* (1986) found that formalin solution repeatedly administered in transplanted rat bladders did not promote formation of tumors. Takahashi *et al.* (1986) found that formalin solution in diet did promote stomach tumors in Wistar rats. Iversen *et al.* (1988) found that topical skin application of formaldehyde solution in mice did not promote the formation of skin tumors.

IV. DERIVATION OF CANCER POTENCY

Basis for Cancer Potency

The International Agency for Research of Cancer (1987) has reviewed the evidence for carcinogenicity and found it to be limited in humans and sufficient in animals. U.S. EPA (1987) has classified formaldehyde in Group B1, probable human carcinogen. The U.S. Occupational Safety and Health Administration (U.S. OSHA, 1987) has concluded that “formaldehyde should be regarded as an occupational carcinogen,” based upon animal and human studies. Considering these previous determinations, along with the evidence of carcinogenicity, OEHHA staff (OEHHA, 1992) concluded that formaldehyde is a probable carcinogen and meets the definition of a “toxic air contaminant”: an air pollutant which may cause or contribute to an increase in mortality or an increase in serious illness, or which may pose a present or potential hazard to human health.

Formaldehyde is carcinogenic in rodents, as described above, producing squamous cell carcinomas in the nasal passages of male and female rats and male mice. Several different types of potentially precancerous abnormalities, including polypoid adenomas and squamous cell papillomas, have also been observed. The epidemiological evidence, while suggestive of a risk of human cancer due to formaldehyde exposure, was considered insufficient for risk assessment purposes on its own. OEHHA (1992) found the tumor incidence data in rats reported by Kerns *et al.* (1983) and used by U.S. EPA (1987) to be the most appropriate for use in developing a quantitative risk assessment.

Methodology

In developing a spectrum of predictions of cancer risk to humans, the OEHHA (1992) assessment applied a pharmacokinetic interpolation of the molecular dosimetry data to the animal cancer bioassay data of Kerns *et al.* (1983). The analysis used the linearized multistage procedure (GLOBAL86), and the procedure developed by Moolgavkar and others, which takes into account the proliferation of premalignant cells due to the formaldehyde exposure. Both models derive upper confidence limits (UCL) for excess cancer risk and extrapolate the risk to humans by means of three different scaling factors. Two scaling factors take into account the contact mechanism of carcinogenesis. However, they do so in different ways. One uses only a generic calculation in terms of body mass. The other takes specific account of comparative data on DNA binding in rats and monkeys to adjust the metabolic rate for humans; it assumes humans respond as do monkeys and uses the data of Casanova *et al.* (1989; 1991). The third scaling factor follows the default option of the California carcinogen guidelines (CDHS, 1985), which calculates the adjustment for rat exposures to obtain the equivalent human exposure on the basis of intake rate divided by body surface area.

Table 2: Formaldehyde inhalation bioassay data used to estimate cancer risk to rats

Exposure (ppm HCHO) ^a	Rate of DNA Binding ^b (pmol/mg-hr)	Lifetime Equivalent Metabolic Exposure ^b (ppm)	Incidence of Nasal Squamous Carcinomas ^c
0	0	0	0/156 (0%)
2	2.5	0.54	0/159 (0%)
5.6	15.9	3.4	2/153 (1.3%)
14.3	74.8	16.	94/140 (67.5%)

Source: adapted from OEHHA (1992)

^aFischer 344 rats inhaled indicated concentrations of formaldehyde gas 6 hours per day, 5 days per week for 24 months.

^bDetails on how these estimates were obtained are presented in OEHHA (1992)

^cBased on data partially reported in Kerns *et al.* (1983). Numerator and denominator are those used by U.S. EPA (1987).

For the best value of UCL on unit risk for a lifetime of exposure, the OEHHA staff selected $7 \times 10^{-3} \text{ ppm}^{-1}$ ($6.0 \times 10^{-6} (\mu\text{g}/\text{m}^3)^{-1}$), based on molecular dosimetry data in a three-stage model and using the standard surface-area scaling factor, 1.2. The range of calculated values of UCL on unit risks is $0.3 \times 10^{-3} \text{ ppm}^{-1}$ to $40 \times 10^{-3} \text{ ppm}^{-1}$ (0.25×10^{-6} to $33 \times 10^{-6} (\mu\text{g}/\text{m}^3)^{-1}$).

In a review of epidemiological studies for workers exposed to formaldehyde the study by Blair *et al.* (1986) was selected as the most reliable for quantitative comparisons. That study, the largest and best documented study available, evaluated mortality in a cohort of more than 26,000 workers. The observed risk of death by lung cancer in exposed workers was 15×10^{-3} over their career. Based on extrapolation of rat cancer risk predictions to humans for a 40-hour work week for 20 years and an exposure level of 1.0 ppm, the prediction of 95% upper confidence limits on respiratory tract cancer was 32×10^{-3} for the three-stage tissue-dose model with generic contact

scaling factor. Thus, the upper range of human cancer risk predictions from the rat bioassay data (Kerns *et al.*, 1983) was consistent with the occupational exposure cancer risk data.

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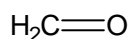
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Formaldehyde Reference Exposure Levels

(Methanal, oxomethane, methylene oxide)

CAS 50-00-0



1. Summary

The non-cancer adverse health effects of formaldehyde are largely a manifestation of its ability to irritate mucous membranes. As a result of its solubility in water and high reactivity, formaldehyde is efficiently absorbed into the mucus layers protecting the eyes and respiratory tract where it rapidly reacts, leading primarily to localized irritation. Acute high exposure may lead to eye, nose and throat irritation, and in the respiratory tract, nasal obstruction, pulmonary edema and dyspnea. Prolonged or repeated exposures have been associated with allergic sensitization, respiratory symptoms (coughing, wheezing, shortness of breath), histopathological changes in respiratory epithelium, and decrements in lung function. Children, especially those with diagnosed asthma, may be more likely to show impaired pulmonary function and symptoms than are adults following chronic exposure to formaldehyde. The studies reviewed for this document include those published through the Spring of 2008.

1.1 Formaldehyde Acute REL

<i>Reference Exposure Level</i>	55 $\mu\text{g}/\text{m}^3$ (44 ppb)
<i>Critical effect(s)</i>	Mild and moderate eye irritation
<i>Hazard Index target(s)</i>	Eye irritation

1.2 Formaldehyde 8-Hour REL

<i>Reference Exposure Level</i>	9 $\mu\text{g}/\text{m}^3$ (7 ppb)
<i>Critical effect(s)</i>	Nasal obstruction and discomfort, lower airway discomfort, and eye irritation
<i>Hazard Index target(s)</i>	Respiratory

1.3 Formaldehyde Chronic REL

<i>Reference Exposure Level</i>	9 $\mu\text{g}/\text{m}^3$ (7 ppb)
<i>Critical effect(s)</i>	Nasal obstruction and discomfort, lower airway discomfort, and eye irritation
<i>Hazard Index target(s)</i>	Respiratory

2. Physical & Chemical Properties (ATSDR, 1999)

<i>Description</i>	Colorless gas
<i>Molecular formula</i>	CH ₂ O
<i>Molecular weight</i>	30.03 g/mol
<i>Density</i>	0.815 g/L @ -20° C
<i>Boiling point</i>	-19.5° C
<i>Melting point</i>	-92° C
<i>Vapor pressure</i>	3883 mm Hg @ 25° C
<i>Flashpoint</i>	300° C
<i>Explosive limits</i>	7% - 73%
<i>Solubility</i>	soluble in water, alcohol, ether and other polar solvents
<i>Odor threshold</i>	0.05-0.5 ppm
<i>Metabolites</i>	formic acid
<i>Conversion factor</i>	1 ppm in air = 1.24 mg/m ³ @ 25° C

3. Occurrence and Major Uses

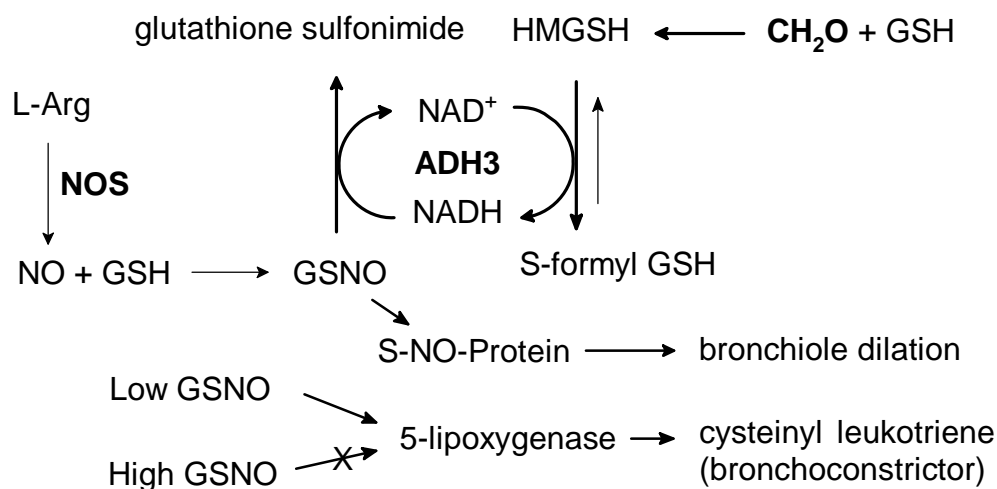
Formaldehyde has four major applications: as an intermediate in the manufacture of melamine, polyacetal, and phenolic resins; as an intermediate in the production of industrial chemicals; as a bactericide or fungicide; and as a component in the manufacture of end-use consumer products. Phenol-formaldehyde resins are used in the production of plywood, particleboard, foam insulation, and a wide variety of molded or extruded plastic items. Formaldehyde is also used as a preservative, a hardening and reducing agent, a corrosion inhibitor, a sterilizing agent, and in embalming fluids. Indoor sources include upholstery, permanent press fabrics, carpets, pesticide formulations, urea-formaldehyde foam insulation, and cardboard and paper products. Outdoor sources include emissions from fuel combustion (motor vehicles), industrial fuel combustion (power generators), oil refining processes, and other uses (copper plating, incinerators, etc.). The largest portion of outdoor ambient formaldehyde results from photochemical oxidation of a number of reactive organic gases in the atmosphere (CARB, 2006). According to the California Toxics Inventory (CARB, 2005a), the mean statewide ambient level of formaldehyde in 2004 was 2.69 ppb, with the highest levels (3.76 ppb) reported for the South Coast Air Basin. The California Air Resources Board (CARB) reported statewide emissions of 20,251 tons from stationary and mobile sources (CARB, 2005b).

4. Metabolism

Inhaled formaldehyde reacts rapidly at the site of contact and is efficiently absorbed in the respiratory tract. A portion of the formaldehyde entering the fluid layer covering the respiratory epithelium, the respiratory tract lining fluid (RTLFL), is reversibly hydrated to methylene glycol. Among other components, the RTLFL is rich in antioxidants including glutathione (Cross et al., 1994) with which formaldehyde may reversibly react to form *S*-hydroxymethylglutathione. Both the hydrated and unreacted formaldehyde may be absorbed into the epithelial layer where there is further opportunity for formaldehyde to bind to glutathione. This glutathione conjugate in turn is oxidized to *S*-formylglutathione by formaldehyde dehydrogenase. Hydrolysis of *S*-formylglutathione yields formate and glutathione. Formic acid may be eliminated in urine and

feces, or dehydrogenated to CO₂ and exhaled. The presence of glutathione and formaldehyde dehydrogenase in epithelial cells of the respiratory tract varies with location and influences the amount of formaldehyde reaching the blood. While glutathione-bound formaldehyde is rapidly metabolized, free formaldehyde in cells can form DNA-protein cross-links (Franks, 2005).

Formaldehyde dehydrogenase (ADH3), although central to the metabolism of formaldehyde, has a broad specificity that includes the structurally related compound, S-nitrosoglutathione (GSNO), an endogenous bronchodilator and reservoir of nitric oxide (NO) activity (Jensen et al., 1998). In cultured cells, formaldehyde appears to trigger ADH3-mediated GSNO reduction by enzyme-bound cofactor recycling (Staab et al., 2008). As shown in Figure 1, the S-hydroxymethylglutathione (HMGS) formed spontaneously from formaldehyde and glutathione is oxidized by ADH3 with the formation of NADH that may then participate in the ADH3-mediated reduction of GSNO (Thompson and Grafstrom, 2008). (Because of its participation in this reaction, ADH3 is also known as GSNO reductase.) This reductive pathway results in low levels of GSNO that in turn stimulate the production and activity of 5-lipoxygenase, the rate-limiting enzyme in the synthesis of powerful bronchoconstrictors, the cysteinyl leukotrienes. On the other hand, high levels of GSNO inhibit this enzyme and thus the synthesis of the bronchoconstrictors (Zaman et al., 2006). Up-regulation of the degradation of GSNO has been demonstrated in mouse lung following inhalation of formaldehyde (Yi et al., 2007), while low levels of GSNO in the lungs have been associated with severe asthma attacks in children (Gaston et al., 1998) and airway hyperactivity in mice (Que et al., 2005). These results suggest that the potential association of formaldehyde exposure with asthma-like respiratory symptoms is in part due to its effects on NO via the enhanced degradation of GSNO. Nitric oxide has multiple functions in the lungs, from its participation in the regulation of airway and vascular tone to mucin secretion and mucociliary clearance (Reynaert et al., 2005). The dysregulation of NO by formaldehyde helps to explain the variety and variability in the toxic manifestations following formaldehyde inhalation.

FIGURE 3 FORMALDEHYDE DRIVEN REDUCTION OF GSNO

Oxidation of the glutathione conjugate of formaldehyde, HMGSH, by ADH3 generates NADH that drives the reduction of GSNO, also by ADH3, thereby reducing the nitric oxide available for bronchiole dilation. Low GSNO levels stimulate, but high GSNO levels inhibit 5-lipoxygenase production of cysteinyl leukotriene.

5. Acute Toxicity of Formaldehyde

The acute effects of formaldehyde exposure appear to be largely a result of its irritant properties. However, some individuals experience symptoms following acute exposures that are a result of previous sensitization following acute high formaldehyde exposure, or long term low level exposures. For this reason, some of the studies included in this section describe manifestations of toxicity in which acute exposure was the precipitating event but in which the contribution of previous exposures or sensitization is unknown. Sensitization manifests as heightened responsiveness and may be of an immunological nature with the development of formaldehyde-specific IgE or IgG (e.g. Thrasher et al. 1987). Alternatively, heightened responsiveness may be neurologically mediated with involvement of the hypothalamic/pituitary/adrenal axis (Sorg et al., 2001a,b). In addition, genetic variation among individuals in the alcohol dehydrogenases mentioned above affects individual responses to formaldehyde. This is especially germane to studies in which the effects include symptoms such as bronchoconstriction and airway hyperreactivity, and in which there is unexpected individual variation.

Many of the studies described in this document have evaluated the relationship between formaldehyde inhalation and clinically-diagnosed asthma or asthma-like symptoms. Asthma is a chronic disease of airway obstruction resulting in variable airflow that has classically been considered to involve both airway inflammation and airway hyperresponsiveness. Asthma manifests as a characteristic cough, wheeze, and shortness of breath due to spasmodic contractions of the bronchi and mucus hypersecretion. These symptoms may or may not reflect an underlying allergic response. As shown in the study by Que et al. (2005), the hyperresponsiveness and the inflammation are not necessarily coupled. Although the RELs presented in this document are not based on studies that used asthma as the critical endpoint, uncertainty factors were applied in the REL estimates to explicitly consider the potential of

formaldehyde to cause or exacerbate asthma-like wheeze and cough symptoms, especially in asthmatic children. We have therefore included discussion of recent work that provides a biochemical mechanism by which formaldehyde exposure is linked to at least one symptom of asthma, bronchoconstriction. The bronchoconstrictive effects of formaldehyde exposure may be partially responsible for the lower airway discomfort reported in the study upon which the 8-hour and chronic RELs are based.

5.1 Acute Toxicity to Adult Humans

In small human studies, exposure to formaldehyde (1-3 ppm) has resulted in eye and upper respiratory tract irritation (Weber-Tschopp et al., 1977; Kulle et al., 1987). Most people cannot tolerate exposures to more than 5 ppm formaldehyde in air; above 10-20 ppm symptoms become severe and shortness of breath occurs (Feinman, 1988). High concentrations of formaldehyde may result in nasal obstruction, pulmonary edema, choking, dyspnea, and chest tightness (Porter, 1975; Solomons and Cochrane, 1984).

A few human case studies report severe pulmonary symptoms. A medical intern with known atopy and exposure to reportedly high (but unspecified) levels of formaldehyde over a period of 1 week developed dyspnea, chest tightness, and edema, following a subsequent 2 hour exposure to formaldehyde (Porter, 1975). Five workers exposed to formaldehyde from newly installed urea-formaldehyde chipboard in a poorly ventilated basement experienced intolerable eye and upper respiratory tract irritation, choking, marked dyspnea, and nasal obstruction (Solomons and Cochrane, 1984). However, the concentration of formaldehyde and the contribution of other airborne chemicals were unknown in both reports.

Numerous acute controlled and occupational human exposure studies have been conducted with both asthmatic and normal subjects to investigate formaldehyde's irritative and pulmonary effects (Frigas et al., 1984; Sheppard et al., 1984; Sauder et al., 1986; Schachter et al., 1986; Kulle et al., 1987; Sauder et al., 1987; Schachter et al., 1987; Witek et al., 1987; Uba et al., 1989; Harving et al., 1990; Akbar-Khanzadeh et al., 1994). Short exercise sessions during exposure on a bicycle ergometer were included in some of the studies. Concentrations of formaldehyde in the human exposure studies ranged as high as 3 ppm for up to 3 hours. The major findings in these studies were mild to moderate eye and upper respiratory tract irritation typical of mild discomfort from formaldehyde exposure.

Chemosensory irritation and subjective symptoms following exposure to formaldehyde at concentrations relevant to the workplace were examined by Lang et al. (2008) in 11 male and 10 female volunteers. Each subject was exposed for 4 hours to a randomized sequence of ten exposure conditions. These included exposures at concentrations of 0, 0.15, 0.3 and 0.5 ppm, exposures at 0.3 and 0.5 ppm that included four transient peak exposures at 0.6 and 1.0 ppm, respectively, and exposures in the presence of 10 ppm ethyl acetate of 0, 0.3, 0.5, and 0.5 ppm with 1.0 ppm peaks. Objective measures of irritation included conjunctival redness, blinking frequency, nasal flow resistance, pulmonary function, and reaction times. The participant's subjective evaluation of physical and mental wellbeing was assessed by questionnaire before, during and after each day's exposure. To assess the potential influence of personality traits on subjective responses, each subject's positive or negative affectivity was evaluated with PANAS (Positive and Negative Affectivity Schedule) that consists of 10 positive affects (interested,

excited, strong, enthusiastic, proud, alert, inspired, determined, attentive, and active) and 10 negative affects (distressed, upset, guilty, scared, hostile, irritable, ashamed, nervous, jittery, and afraid). Participants are asked to rate items on a scale from 1 to 5, based on the strength of emotion where 1 = "very slightly or not at all," and 5 = "extremely". Subjective ratings of eye irritation and olfactory symptoms were significantly higher than control at 0.3 ppm. However, when negative affectivity (anxiety) was included as a covariate, eye and olfactory irritation at this exposure level were no longer significant. Conjunctival irritation and blinking frequency, objective measures of irritation, were significantly elevated only with exposure to 0.5 ppm with peaks of 1.0 ppm ($p < 0.05$). The authors considered this level to be a LOAEL. However, at 0.5 ppm without 1.0 ppm peaks, conjunctival irritation and blinking were not significantly increased so this was considered a NOAEL for these effects. There were no statistically significant changes in nasal resistance, pulmonary function or reaction time. While there were large inter-individual differences in complaints or reports of wellbeing, there were no significant treatment effects. This study identified eye irritation as the most sensitive endpoint, with personality traits, such as negative affectivity, as a modifying factor.

In a human irritation study by Weber-Tschopp et al. (1977), 33 subjects were exposed to formaldehyde at concentrations ranging from 0.03-3.2 ppm (0.04-4.0 mg/m³) for 35 minutes. Thresholds were 1.2 ppm (1.5 mg/m³) for eye and nose irritation, 1.7 ppm (2.1 mg/m³) for eye blinking, and 2.1 ppm (2.6 mg/m³) for throat irritation.

Kulle et al. (1987) exposed nonasthmatic humans to up to 3.0 ppm (3.7 mg/m³) formaldehyde in a controlled environmental chamber for 3 hours. Significant dose-response relationships were seen with odor and eye irritation (Table 5.1) as ranked on symptom questionnaires as none, mild, moderate or severe. Irritation was assessed in this manner prior to exposure, at the end of exposure, and again 24 hour after exposure.

TABLE 5.1 MEAN SYMPTOM DIFFERENCE ($T_{180}-T_0$) \pm SE WITH FORMALDEHYDE* (FROM KULLE ET AL., 1987)

	Formaldehyde conc. (ppm)				P value
	0.0	1.0	2.0	3.0	
Odor sensation	0.00 \pm 0.00	0.22 \pm 0.15	0.44 \pm 0.18	1.00 \pm 0.29	<0.0001
Nose/throat irritation	0.00 \pm 0.00	0.11 \pm 0.11	0.33 \pm 0.17	0.22 \pm 0.15	0.054
Eye irritation	0.00 \pm 0.00	0.44 \pm 0.24	0.89 \pm 0.26	1.44 \pm 0.18	<0.0001
Chest discomfort	0.00 \pm 0.00	0.00 \pm 0.00	0.11 \pm 0.11	0.00 \pm 0.00	0.62
Cough	0.00 \pm 0.00	0.11 \pm 0.11	0.00 \pm 0.00	0.00 \pm 0.00	0.11
Headache	0.00 \pm 0.00	0.00 \pm 0.00	0.00 \pm 0.00	0.11 \pm 0.11	0.33

*Presence and severity of symptoms scored as: 0 = none; 1 = mild (present but not annoying); 2 = moderate (annoying); 3 = severe (debilitating). Data from Table II.

At 0.5 ppm for 3 hours, none of 9 subjects had eye irritation. At 1.0 ppm, 3 of 19 subjects reported mild eye irritation and one experienced moderate irritation. At 2.0 ppm, 6 subjects reported mild and 4 reported moderate eye irritation. Measured nasal flow resistance was increased at 3.0 ppm but not at 2.0 ppm (2.5 mg/m³). With respect to the lower respiratory tract, there were no significant decrements in pulmonary function nor increases in methacholine

induced bronchial reactivity as a result of 3-hour exposures to 0.5-3.0 ppm (0.6-3.7 mg/m³) formaldehyde at rest or during exercise, including 24 hours post exposure.

Eleven healthy subjects and nine patients with formalin skin sensitization were exposed to 0.5 mg/m³ (0.4 ppm) formaldehyde for 2 hours (Pazdrak et al., 1993). Nasal lavage was performed prior to and 5 to 10 minutes, 4 hours, and 18 hours after exposure. Rhinitis was reported and increases in the number and proportion of eosinophils, elevated albumin and increased protein levels were noted in nasal lavage fluid 4 and 18 hours after exposure. No differences were found between patients with skin sensitization and healthy subjects.

In a study by Green et al. (1987), volunteer asthmatic and normal subjects exposed to formaldehyde displayed decrements in pulmonary function. Exposure to 3 ppm formaldehyde for 1 hour resulted in clinically significant reductions of forced expiratory volume in one second (FEV₁) (defined as > 20% or more) and FEV₁/forced vital capacity (FVC) (ratio 70% or less) in 5 individuals in the study (2 of 16 asthmatics, 2 of 22 normal subjects, and one clinically normal subject with hyperactive airways). Of these individuals, 3 had reductions of FEV₁ of 20% or more during exposure. One of 22 asthmatics had a greater than 20% reduction in FEV₁ (-25.8%) at 17 minutes into exposure following a 15 minute moderate exercise session (minute ventilation [V_E] = 30-40 l/min), which, according to the authors, was low enough to prevent exercise-induced bronchospasm. One of 22 normal subjects also exhibited a greater than 20% clinically significant reduction in FEV₁ (-24.4%) and in FEV₁/FVC, which occurred at 47 minutes into exposure to 3 ppm formaldehyde. These reductions occurred following a second 15-minute heavy-exercise session (V_E = 60-70 l/min) near the end of the 1 hour exposure period. A third asymptomatic "normal" subject with hyperactive airways had a clinically significant reduction of FEV₁ (-20.5%) at 17 minutes, following the first heavy exercise session. This subject exhibited occult airway hyperactivity and was excluded from analysis with the other exposure groups due to his respiratory condition. Subjects exhibiting reductions in FEV₁ of greater than 20% following exposure also exhibited FEV₁/FVC ratios of less than 70%. However, none of the subjects in the study exhibited a clinically significant reduction of 50% or greater in airway conductance (SG_{aw}) during exposure to 3 ppm formaldehyde.

Kriebel et al. (2001) conducted a subchronic epidemiological study of 38 anatomy class students who, on average, were exposed to a geometric mean of 0.70 ± 2.13 ppm for 2 hours per week over 14 weeks. After class, eye, nose and throat irritation was significantly elevated compared with pre-laboratory session exposures, with a one unit increase in symptom intensity/ppm of formaldehyde. Peak expiratory flow (PEF) was found to decrease by 1%/ppm formaldehyde during the most recent exposure. Changes in PEF and symptom intensity following formaldehyde exposure were most pronounced during the first weeks of the semester but attenuated with time, suggesting partial acclimatization.

Rhinitis and a wide range of respiratory symptoms can result from exposure to formaldehyde. Some studies have reported that workers exposed to low concentrations may develop severe prolonged asthma attacks after prior exposure; this suggests that they may have become sensitized (Feinman, 1988). However, in adults, an association between formaldehyde exposure and allergic sensitization through IgE- and IgG-mediated mechanisms has been observed only inconsistently (Thrasher et al., 1987; Krakowiak et al., 1998; Wantke et al., 2000; Kim et al., 2001).

Formaldehyde provocation of human subjects, occupationally exposed to formaldehyde and suffering from respiratory symptoms such as wheezing, shortness of breath, or rhinitis, occasionally resulted in pulmonary function decrements (2 to 33% response rate) consistent with immediate, delayed, or both immediate and delayed bronchoconstriction (Hendrick and Lane, 1977; Wallenstein et al., 1978; Burge et al., 1985; Nordman et al., 1985). While some of the concentrations of formaldehyde that elicited a positive response following provocation tests (6 to 20.7 ppm) were quite high, the authors of these studies suggested that formaldehyde-induced bronchial hyperreactivity is due to specific sensitization to the gas. However, none of these studies was able to detect antibodies to formaldehyde which would support that sensitization to formaldehyde occurs through an immunologic pathway. Alternatively, the wheezing and shortness of breath may be related to the formaldehyde-stimulated depletion of the bronchodilator, GSNO, in the airways.

In controlled studies with asthmatics from urea-formaldehyde insulated homes, formaldehyde concentrations equal to or greater than those found in indoor environments have not resulted in hematologic or immunologic abnormalities. These tests include: blood count and differential, erythrocyte sedimentation rate; lymphocyte subpopulations (E-rosetting, T3, T4, T8, B73.1, Fc receptor positive lymphocytes and large granular lymphocytes); lymphocyte response to phytohemagglutinin and formalin-treated red blood cells; serum antibody against the Thomsen-Friedenrich RBC antigen and against formalin-RBC; and natural killer, interferon-boosted natural killer, and antibody-dependent cell-mediated cytotoxicity (Pross et al., 1987). While six of the studies cited above reported decrements in lung function associated with short-term formaldehyde exposure among at least some of the asthmatic subjects, a number of other exposure studies of patients with asthma have failed to demonstrate that exposure to formaldehyde results in onset or aggravation of the patients' asthmatic symptoms (Sheppard et al., 1984; Sauder et al., 1987; Harving et al., 1990; Krakowiak et al., 1998).

The effects of formaldehyde on asthmatics may be dependent on previous, repeated exposure to formaldehyde. Burge et al. (1985) found that 3 out of 15 occupationally exposed workers challenged with formaldehyde vapors at concentrations from 1.5 ppm to 20.6 ppm for brief durations exhibited late asthmatic reactions. Six other subjects had immediate asthmatic reactions likely due to irritant effects. Asthmatic responses (decreased PEF, FVC, and FEV₁) were observed in 12 occupationally-exposed workers challenged with 2.0 ppm (2.5 mg/m³) formaldehyde (Nordman et al., 1985). Similarly, asthmatic responses were observed in 5 of 28 hemodialysis workers occupationally exposed to formalin and challenged with formaldehyde vapors (concentration not measured) (Hendrick and Lane, 1977). In asthmatics not occupationally exposed to formaldehyde, Sheppard et al. (1984) found that a 10-minute challenge with 3 ppm formaldehyde coupled with moderate exercise did not induce significant changes in airway resistance or thoracic gas volume.

Gorski et al. (1992) evaluated the production of active oxygen species by neutrophils in 18 persons exposed to 0.5 mg/m³ formaldehyde for 2 hours. All 13 subjects who had allergic contact dermatitis (tested positive to formaldehyde in skin patch) exhibited significantly higher chemiluminescence of granulocytes isolated from whole blood 30 minutes and 24 hours post-exposure than the individuals who were not formaldehyde sensitive. Thus, the immune cellular response of skin-sensitized individuals to an inhalation exposure to formaldehyde indicates increased production of active oxygen species. This is consistent with increasing evidence that

endogenous or exogenous reactive oxygen and reactive nitrogen species are responsible for the airway inflammation of asthma (Sugiura and Ichinose, 2008).

In addition to its effects on the respiratory tract, the irritant properties of formaldehyde also manifest as ocular irritation. In an anatomy dissecting laboratory, formaldehyde levels were found to peak at 0.62 ppm, with a gradual decrease to 0.11 ppm. Formaldehyde-related irritation of the eyes, nose, throat, airways and skin was reported by 59% of the students. These effects were significantly ($p < 0.001$) higher among wearers of contact lenses compared with students without glasses or wearing glasses (Tanaka et al., 2003). The ability of contact lenses to trap and concentrate volatile compounds, and to extend the exposure time by limiting the eye's normal self-cleansing, may make contact lens wearers more susceptible to ocular exposure and irritation by formaldehyde.

5.2 Acute Toxicity to Infants and Children

No studies of the effects of acute exposure to formaldehyde in children or young experimental animals were located. However, as noted above for adults, there is evidence that following acute exposure to formaldehyde, asthmatics and others previously sensitized to formaldehyde may be more likely to show respiratory symptoms such as wheezing, shortness of breath, rhinitis, and/or decrements in pulmonary function consistent with immediate and/or delayed bronchoconstriction (Nordman et al., 1985; Burge et al., 1985; Hendrick and Lane, 1977; Wallenstein et al., 1978). Furthermore, some asthmatics may respond with significant reductions in lung function due to the irritant effects on asthma, sensitized or not. Additionally, the depletion of the endogenous bronchodilator, GSNO, following formaldehyde exposure may be particularly important in children. Gaston et al. (1998) compared concentrations of tracheal S-nitrosothiol concentrations in eight asthmatic children in respiratory failure with those of 21 non-asthmatic children undergoing elective surgery. In asthmatic children, the metabolism of GSNO was accelerated and the mean S-nitrosothiol concentrations significantly lower compared to normal children (65 ± 45 vs 502 ± 429 nmol/l). Thus asthmatic children, with low levels of GSNO, are expected to be unusually vulnerable to any further depletion of GSNO caused by formaldehyde.

The potential association between formaldehyde exposure and asthma is of special concern for children since, as noted in OEHHA (2001): "*OEHHA considers asthma to impact children more than adults. Children have higher prevalence rates of asthma than do adults (Mannino et al., 1998). In addition, asthma episodes can be more severe due to the smaller airways of children, and result in more hospitalizations in children, particularly from the ages of 0 to 4 years, than in adults (Mannino et al., 1998).*" Thus children, particularly asthmatic children, may be at greater risk from acute exposure to formaldehyde.

5.3 Acute Toxicity to Experimental Animals

Acute exposures of experimental animals to formaldehyde are associated with changes in pulmonary function (decreased respiratory rate, increased airway reactivity and resistance) at low concentrations, while pulmonary edema and death have been reported at high concentrations. Neurochemical and neurobehavioral changes have also been observed.

In 72 rats exposed to approximately 600-1,700 mg/m³ (500-1,400 ppm) formaldehyde vapor for 30 minutes, the LC₅₀ was found to be 1,000 mg/m³ (800 ppm) (Skog, 1950). The first deaths did not occur until 6 hours after cessation of exposure. Respiratory difficulty lasted several days after exposure and the last of 49 rats died after 15 days of purulent bronchitis and diffuse bronchopneumonia. Three weeks following exposure, histological examinations of the 23 surviving animals revealed bronchitis, pulmonary microhemorrhages, and edema. No changes were seen in other organs.

A multispecies study by Salem and Cullumbine (1960) showed that a 10-hour exposure to 15.4 ppm (19 mg/m³) formaldehyde vapor killed 3 out of 5 rabbits, 8 of 20 guinea pigs, and 17 of 50 mice. The report stated that formaldehyde exposure resulted in delayed lethality.

Alarie (1981) determined the 10 minute LC₅₀ for formaldehyde in mice to be 2,162 ppm (95% confidence interval, 1,687-2,770 ppm). The post-exposure observation period was 3 hours. From the concentration mortality graph provided in the report, an MLE₀₅ and BC₀₅ of 1,440 ppm and 778 ppm, respectively, could be estimated for a 10-minute formaldehyde exposure. However, as indicated in the previous reports, delayed deaths occur with formaldehyde which suggests that the 3-hour post-exposure observation period used in this study may not have been long enough.

In other lethality studies, Nagorny et al. (1979) determined a 4-hour formaldehyde LC₅₀ in rats and mice to be 588 mg/m³ (474 ppm) and 505 mg/m³ (407 ppm), respectively. However, the raw data for this study were not included in the report. Horton et al. (1963) observed that a 2-hour exposure of mice to 0.9 mg/l (900 mg/m³) formaldehyde resulted in deaths from massive pulmonary hemorrhage and edema, but a 2 hour exposure to 0.14 mg/l (140 mg/m³) did not produce signs of "substantial distress."

Swiecichowski et al., (1993) exposed groups of five to seven guinea pigs to 0.86, 3.4, 9.4, 31.1 ppm (1.1, 4.2, 11.6, 38.6 mg/m³) formaldehyde for 2 hours, or to 0.11, 0.31, 0.59, 1.05 ppm (0.14, 0.38, 0.73, 1.30 mg/m³) formaldehyde for 8 hours. An 8-hour exposure to levels greater than or equal to 0.3 ppm (≥ 0.4 mg/m³) formaldehyde was sufficient to produce a significant increase in airway reactivity. Similar effects occurred after greater than 9 ppm (> 11 mg/m³) formaldehyde for the 2-hour exposure group. Formaldehyde exposure also heightened airway smooth muscle responsiveness to acetylcholine (or carbachol) *ex vivo*. No inflammation or epithelial damage was seen up to 4 days after exposure. The researchers suggest that duration of exposure is important to the induction of airway hyperreactivity and that prolonged (8-hour), low-level exposures may generate abnormal physiologic responses in the airways not detectable after acute (2-hour) exposures.

Male F-344 rats, 7-9 weeks old, were exposed to 0.5, 2, 6 or 15 ppm formaldehyde for 6 hours per day for 1 to 4 days (Monteiro-Riviere and Popp, 1986). Effects noted in the rat nasal respiratory epithelium with 0.5 or 2 ppm were limited to altered cilia with occasional wing-like projections on the ends of the ciliary shafts. Effects noted at 6 ppm for 1 day were autophagic vacuoles in some basal cells, neutrophils in the basal and suprabasal layers, and hypertrophy of goblet and ciliated cells. Loss of microvilli in ciliated cells was noted at all exposure concentrations.

Rats were exposed to 0, 5, 10 or 20 ppm formaldehyde for 3 hours per day on 2 consecutive days (Boja et al., 1985). Decreased motor activity and neurochemical changes in dopamine and 5-hydroxytryptamine neurons were reported.

The effects of formaldehyde inhalation on open-field behavior in mice were examined by Malek et al. (2004) 2 and 24 hours after a single 2-hour exposure to 0, 1.1, 2.3 or 5.2 ppm. Two hours after exposure there were significant decreases in rearing and in several measures of exploratory behavior, with evidence of dose-dependence in all dose groups compared with controls. At 24 hours, there were still significant differences between dosed and control mice but the dose-dependence was no longer evident.

Nielson et al. (1999) analyzed the breathing patterns of Balb/c mice exposed to 0.2-13 ppm formaldehyde and found a concentration-dependent decrease in respiratory rate of 32.9%/log concentration. In the range of 0.3-4.0 ppm, the decrease in respiratory rates was attributable to sensory irritation. Above 4.0 ppm, bronchoconstriction also contributed to the decreased breathing rate. The authors suggest a NOEL of 0.3 ppm for these effects in mice.

Amdur (1960) exposed groups of 4 to 18 guinea pigs to formaldehyde at 0.05, 0.31, 0.58, 1.22, 3.6, 11.0, or 49 ppm formaldehyde for one hour. Resistance to flow and lung compliance were calculated from measures of intrapleural pressure, tidal volume, and rate of flow to the lungs at the end of exposure and one hour later. Resistance and compliance were significantly different from the control level for the 0.31 ppm exposure ($p < 0.05$) and increasingly significant at higher concentrations. One hour later, only the 49 ppm exposure remained significant ($p < 0.01$). In addition, the tracheas of groups of 6 to 10 guinea pigs were cannulated and exposed for one hour to 0.90, 5.2, 20, or 50 ppm formaldehyde, and 1.14 or 3.6 ppm formaldehyde with 10 mg/m³ sodium chloride. With the protective effect of the trachea bypassed, the resistance and compliance changed substantially. The addition of sodium chloride further enhanced the effect, including a significant effect after one hour for the 1.14 ppm formaldehyde exposure. These results show that formaldehyde that reaches the lungs has a marked effect on airways resistance and compliance in addition to an effect on the upper airways.

Riedel et al. (1996) studied the influence of formaldehyde exposure on allergic sensitization in guinea pigs. Three groups of guinea pigs (12/group) were exposed to clean air or two different formaldehyde concentrations (0.13 and 0.25 ppm) over five consecutive days. Following exposure, the animals were sensitized to allergen by inhalation of 0.5% ovalbumin (OA). Three weeks later the animals were subjected to bronchial provocation with OA and specific anti-OA-IgG1 (reaginic) antibodies in serum were measured. In another group of six animals, the respiratory tract was examined histologically for signs of inflammation directly after the end of formaldehyde or clean air exposure. In the group exposed to 0.25 ppm formaldehyde, 10/12

animals were found to be sensitized to OA (positive reaction on specific provocation) vs. 3/12 animals in the control group ($P < 0.01$). Furthermore, compressed air measurements of specific bronchial provocation and serum anti-OA-antibodies were significantly higher in the 0.25 ppm formaldehyde group than in controls. The median for compressed air measurement was 0.35 ml for the formaldehyde-exposed group vs. 0.09 ml for the controls ($p < 0.01$), indicating increased bronchial obstruction. The median for the anti-OA-IgG1 measured in the formaldehyde-exposed group was 13 vs. less than 10 EU in the controls, ($p < 0.05$), indicating enhanced sensitization. In the group exposed to 0.13 ppm formaldehyde, no significant difference was found compared to the control group. Histological examination found edema of the bronchial mucosa, but there was no sign of inflammation of the lower airways in formaldehyde-exposed guinea pigs. The investigators concluded that short-term exposure to a low concentration of formaldehyde (0.25 ppm) can significantly enhance sensitization to inhaled allergens in the guinea pig.

As described in Section 5, the main formaldehyde-metabolizing enzyme, ADH3, also reduces the endogenous bronchodilator GSNO. To examine the role of GSNO and ADH3 (known in this study as GSNO reductase, GSNOR) in airway tone and asthma, Que et al. (2005) used wild type mice and mice with a targeted deletion of GSNOR (GSNOR^{-/-}). Following a challenge with allergen (ovalbumin), GSNOR activity in bronchoalveolar lavage fluid from wild type mice increased significantly ($p < 0.05$) compared to buffer (PBS) controls, while as expected, no GSNOR activity was detected in the GSNOR^{-/-} mice with either treatment. Levels of S-nitrosothiols (SNO) were assayed in homogenates of lung tissues from both types of mouse and found to be barely detectable with PBS treatment. However, after ovalbumin challenge, SNO levels were significantly higher ($p < 0.02$) in GSNOR^{-/-} mice compared to wild type, indicating metabolism of SNOs by GSNOR under “asthmatic-like” conditions in wild type mice. Metabolism of GSNO results in a loss of bronchodilation capacity. Deletion of GSNOR had no effect on NO generation by NO synthase as there were no differences between wild type and GSNOR^{-/-} mice in nitrate or nitrite levels regardless of treatment. To investigate the effects of deletion of GSNOR on airway hyper-responsiveness, pulmonary resistance was measured at baseline (PBS) and after methacholine challenge, with and without ovalbumin treatment. At baseline, there was no difference among mouse types and treatments, while at higher methacholine levels (100-1000 $\mu\text{g}/\text{kg}$), pulmonary resistance was found to be significantly lower ($p < 0.001$) in GSNOR^{-/-} mice than in wild type, presumably due to higher GSNO levels that enhance bronchodilation. Importantly, ovalbumin caused a marked increase in airway responsiveness in wild type mice but had little effect in GSNOR^{-/-} mice. This indicates that GSNOR regulates basal airway tone as well as hyper-responsiveness to both allergen challenge and bronchoconstrictor agonists. It is also noteworthy that the total number and composition of leukocytes, levels of interleukin-13 and total serum IgE were comparable between wild type and GSNOR^{-/-} mice. This indicates that protection from asthma in the GSNOR^{-/-} mice is not a result of a suppressed response to allergen, and that SNOs, especially GSNO, can preserve airway patency in the presence of inflammation. Thus the inflammatory response is not linked to hyper-responsiveness as long as adequate levels of GSNO are maintained.

A connection between formaldehyde and the activity of GSNOR described in the study above by Que et al., was outlined by Thompson and Grafstrom (2008) and supported by Yi et al. (2007) and Staab et al. (2008). In the study by Yi and associates, groups of 6 mice were exposed to formaldehyde at 0, 1, or 3 mg/m^3 continuously for 72 hours. Following exposure, lungs were isolated to allow measurement of GSNOR mRNA levels by RT-PCR, and enzymatic activity

with GSNO. Formaldehyde at 3 mg/m³ significantly increased the numbers of GSNOR transcripts compared to control (0.58 vs 0.4 GSNOR/ β actin; $p < 0.05$), while GSNOR reduction of GSNO showed a significant dose-dependent increase with formaldehyde concentration ($p < 0.01$). The stimulation of GSNO reduction by formaldehyde was also observed by Staab et al. (2008) in an in vitro study using recombinant human GSNOR. In this study, GSNO levels in buccal carcinoma cells were reduced in a dose-dependent fashion following a 1 hour exposure to formaldehyde in the 1-5 mM range with significance at 5 mM ($p < 0.05$). The results from this study support a model in which formaldehyde (as the glutathione conjugate, HMGSH) is oxidized by GSNOR (ADH3) in the presence of high levels of NAD⁺, producing NADH. This process was found to be accelerated by high levels of GSNO. GSNO is in turn reduced with the oxidation of NADH to form glutathione sulfonimide. Formaldehyde thus depletes cellular SNO (in the form of GSNO) which results in dysregulation of NO signaling pathways.

6. Chronic Toxicity of Formaldehyde

6.1 Chronic Toxicity to Adult Humans

Formaldehyde primarily affects the mucous membranes of the upper airways and eyes. Exposed populations that have been studied include embalmers, residents in houses insulated with urea-formaldehyde foam, anatomy class students, histology technicians, wood and pulpmill workers, and asthmatics. A number of studies describing these effects have been briefly summarized below. For the sake of brevity, only the studies that best represent the given effects are presented. Formaldehyde is also a recognized carcinogen (IARC, 2006), however, this document will address only its non-carcinogenic properties.

In the study chosen for determination of the 8-hour and chronic RELs, nasal obstruction and discharge, and frequency of cough, wheezing, and symptoms of bronchitis were reported in 66 workers in a formaldehyde production plant exposed for 1-36 years (mean = 10 years) to a mean concentration of 0.21 ppm (0.26 mg/m³) formaldehyde (Wilhelmsson and Holmstrom, 1992). All workers were exposed almost exclusively to formaldehyde, the concentrations of which were measured in the ambient air of the worksite with personal sampling equipment. Referents consisted of 36 office workers in a government office with exposure to a mean concentration of 0.07 ppm (0.09 mg/m³) formaldehyde, and no industrial solvent or dust exposure. Symptom data, collected by questionnaire, were separated into general and work-related, and allowed identification of individuals with atopy and mucosal hyperreactivity. The critical effects from chronic exposure to formaldehyde in this study included nasal obstruction, lower airway discomfort, and eczema or itching. The frequency of reported lower airway discomfort (intermittent cough, wheezing, or symptoms of chronic bronchitis) was significantly higher among formaldehyde-exposed vs non-exposed workers (44 vs 14%; $p < 0.01$) (Table 6.1). Work-related nasal discomfort also was significantly higher in the formaldehyde group (53%) compared with the referent group (3%; $p < 0.001$). Similarly, work-related eye discomfort was 20% in the formaldehyde group but nonexistent among referents. The significant increase in symptoms of nasal discomfort in exposed workers did not correlate with total serum IgE antibody levels. However, two exposed workers, who complained of nasal discomfort, had elevated IgE levels. The investigators concluded that formaldehyde can induce nonspecific nasal hypersensitivity.

TABLE 6.1.1 SYMPTOMS OF FORMALDEHYDE EXPOSURE VS REFERENCE GROUP

(FROM WILHELMSSON AND HOLMSTROM, 1992)

	Formaldehyde	Reference	Rate difference	
	% (n=66)	% (n=36)	%	95% CI
General nasal discomfort	67	25	42	24-60
Workplace nasal discomfort	53	3	50	37-63
General lower airway discomfort	44	14	30	14-47
Workplace lower airway discomfort	33	3	28	15-40
General eye discomfort	24	6	18	6-36
General skin discomfort	36	11	25	10-41

In a cross-sectional study supportive of these results, Edling et al. (1988) reported histopathological changes in nasal mucosa of workers (n=75) occupationally exposed to formaldehyde (one wood laminating plant) or formaldehyde plus wood dust (two particle board plants). Ambient formaldehyde measurements in these three composite wood processing plants between 1975 and 1983 gave a time-weighted average (TWA) of 0.1-1.1 mg/m³ (0.08- 0.89 ppm) with peaks of up to 5 mg/m³ (4 ppm). The exposed workers were compared on the basis of medical and work histories, clinical examinations and nasal biopsies to 25 workers selected with regard to age and smoking habits but without occupational formaldehyde exposure.

Based on the histories, there was a high frequency of eye and upper airway symptoms among workers. Nasal symptoms (running nose and crusting) associated with formaldehyde exposure were reported in 60% of the workers, while 75% complained of lacrimation. Clinical examinations revealed grossly normal nasal mucosa in 75% of the cases while 25% had swollen or dry changes, or both, to the nasal mucosa. Histological examination (Table 6.2) revealed that only 3 of the 75 formaldehyde-exposed workers had normal, ciliated pseudostratified epithelium. Squamous metaplasia was reportedly observed in 59, while 6 showed mild dysplasia, and in 8 there was loss of ciliated cells and goblet cell hyperplasia. The histological grading showed a significantly higher score for nasal lesions among workers with formaldehyde exposure when compared with the referents (2.9 versus 1.8; p < 0.05). Exposed smokers had a higher, but non-significant, score than ex-smokers and non-smokers.

While the mean exposure time was 10.5 years (range 1-39 yr), there was no discernable difference among histology scores as a function of years of employment. The histology scores were also not different between workers in the particle board plants, exposed to both formaldehyde and wood dust, and workers in the laminate plant with exposure only to formaldehyde. The authors thus attribute the pathological changes in the nasal mucosa and the other adverse effects to formaldehyde alone in the 0.1-1.1 mg/m³ range.

TABLE 6.1.2 DISTRIBUTION OF HISTOLOGICAL CHARACTERISTICS ASSOCIATED WITH FORMALDEHYDE EXPOSURE (FROM EDLING ET AL., 1988)

Histological characteristic	Grading score	Point score	Workers	%
Normal respiratory epithelium	0	0	3	4
Loss of ciliated cells	1	1	8	11
Mixed cuboidal/squamous epithelium, metaplasia	2	2	24	32
Stratified squamous epithelium	3	3	18	24
Keratosi	4	4	16	21
Budding of epithelium	add 1	5	0	0
Mild or moderate dysplasia	6	6	6	8
Severe dysplasia	7	7	0	0
Carcinoma	8	8	0	0

Histological changes in the nasal mucosa of formaldehyde-exposed workers were also reported by Boysen et al. (1990). In this study, nasal biopses were collected from 37 workers with 5 or more years of occupational formaldehyde exposure (0.5 - > 2 ppm) and compared with age-matched, unexposed controls who otherwise had similar environmental exposures and smoking habits. Histological changes in the nasal epithelium were scored as indicated in Table 6.1.3.

TABLE 6.1.3 TYPES OF NASAL EPITHELIA AND SCORING (FROM BOYSEN ET AL., 1990)

Types of epithelia	Histological score
Pseudostratified columnar	0
Stratified cuboidal	1
Mixed stratified cuboidal/stratified squamous	2
Stratified squamous, non-keratinizing	3
Stratified squamous, keratinizing	4
Dysplasia	5

As shown by the histological scoring in Table 6.1.4 below, metaplastic changes in the nasal epithelium were more pronounced in the formaldehyde-exposed workers although this difference did not reach statistical significance.

TABLE 6.1.4 HISTOLOGICAL SCORES OF NASAL EPITHELIA

	No	Histological score						Mean
		0	1	2	3	4	5	
Exposed	37	3	16	5	9	1	3	1.9
Controls	37	5	17	10	5	0	0	1.4

Rhinoscopic examination revealed hyperplastic nasal mucosa in 9 of 37 formaldehyde-exposed workers but in only 4 of the controls. In addition, the incidence of subjective nasal complaints was significantly ($p < 0.01$) higher in the exposed group. While the small size of this study, and the small amount of the nasal mucosa accessible to biopsy limited its ability to detect formaldehyde-related histopathology, the results are consistent with the histopathologies reported by Edling et al. above.

In another occupational health study (Grammer et al., 1990), 37 workers, who were exposed for an unspecified duration to formaldehyde concentrations in the range of 0.003 to 0.073 ppm, reported ocular irritation. However, no significant serum levels of IgE or IgG antibodies to formaldehyde-human serum albumin were detected.

Kerfoot and Mooney (1975) reported that estimated formaldehyde exposures of 0.25-1.39 ppm evoked numerous complaints of upper respiratory tract and eye irritation among seven embalmers at six different funeral homes. Three of the seven embalmers in this study reportedly had asthma. Levine et al. (1984) examined the death certificates of 1477 Ontario undertakers. Exposure measurements taken from a group of West Virginia embalmers were used as exposure estimates for the embalming process, ranging from 0.3-0.9 ppm (average 1-hour exposure) and 0.4-2.1 ppm (peak 30-minute exposure). Mortality due to non-malignant diseases was significantly elevated due to a two-fold excess of deaths related to the digestive system. The authors suggest increased alcoholism could have contributed to this increase.

Ritchie and Lehnen (1987) reported a dose-dependent increase in health complaints (eye and throat irritation, and headaches) in 2000 residents living in 397 mobile and 494 conventional homes. Complaints of symptoms of irritation were noted at concentrations of 0.1 ppm formaldehyde or above. Similarly, Liu et al. (1991) found that exposure to 0.09 ppm (0.135 mg/m³) formaldehyde exacerbated chronic respiratory and allergy problems in residents living in mobile homes.

Employees of mobile day-care centers (66 subjects) reported increased incidence of eye, nose and throat irritation, unnatural thirst, headaches, abnormal tiredness, menstrual disorders, and increased use of analgesics as compared to control workers (Olsen and Dossing, 1982). The mean formaldehyde concentration in these mobile units was 0.29 ppm (0.43 mg/m³) (range = 0.24 - 0.55 mg/m³). The exposed workers were exposed in these units for a minimum of 3 months. A control group of 26 subjects in different institutions was exposed to a mean concentration of 0.05 ppm (0.08 mg/m³) formaldehyde.

Occupants of houses insulated with urea-formaldehyde foam insulation (UFFI) (1726 subjects) were compared with control subjects (720 subjects) for subjective measures of irritation, measures of pulmonary function (FVC, FEV₁, FEF₂₅₋₇₅, FEF₅₀), nasal airway resistance, odor threshold for pyridine, nasal cytology, and hypersensitivity skin-patch testing (Broder et al., 1988). The mean length of time of exposure to UFFI was 4.6 years. The mean concentration of formaldehyde in the UFFI-exposed group was 0.043 ppm, compared with 0.035 ppm for the controls. A significant increase in symptoms of eye, nose and throat irritation was observed in subjects from UFFI homes, compared with controls. No other differences from control measurements were observed.

Alexandersson and Hedenstierna (1989) evaluated symptoms of irritation, spirometry, and immunoglobulin levels in 34 wood workers exposed to formaldehyde over a four-year period. Exposure to 0.4 - 0.5 ppm formaldehyde resulted in significant decreases in FVC, FEV₁, and FEF₂₅₋₇₅. Removal from exposure for four weeks allowed for normalization of lung function in the non-smokers.

Kriebel et al. (2001) conducted a subchronic epidemiological study of 38 anatomy class students who, on average, were exposed to a geometric mean of 0.70 ± 2.13 ppm formaldehyde for two hours per week over fourteen weeks. After class, eye, nose and throat irritation was significantly elevated compared with pre-laboratory session exposures, with a one unit increase in symptom intensity/ppm formaldehyde. Peak respiratory flow (PEF) was found to decrease by 1%/ppm formaldehyde during the most recent exposure. Changes in PEF and symptom intensity following formaldehyde exposure were most pronounced during the first week of the semester but attenuated with time, suggesting partial acclimatization.

Histology technicians (280 subjects) were shown to have reduced pulmonary function, as measured by FVC, FEV₁, FEF₂₅₋₇₅, and FEF₇₅₋₈₅, compared with 486 controls (Kilburn et al., 1989). The range of formaldehyde concentrations was 0.2 - 1.9 ppm, volatilized from formalin preservative solution.

Malaka and Kodama (1990) investigated the effects of formaldehyde exposure in plywood workers (93 exposed, 93 controls) exposed for 26.6 years, on average, to 1.13 ppm (range = 0.28 - 3.48 ppm). Fifty-three smokers were present in both exposed and control groups. Exposure assessment was divided into three categories: high (> 5 ppm), low (< 5 ppm), and none (reference group). Subjective irritation and pulmonary function tests were performed on each subject, and chest x-rays were taken of ten randomly selected volunteers from each group. Respiratory symptoms of irritation were found to be significantly increased in exposed individuals, compared with controls. In addition, exposed individuals exhibited significantly reduced FEV₁, FEV₁/FVC, and forced expiratory flow rate at 25% through 75% of FVC (FEF₂₅₋₇₅), compared with controls. Forced vital capacity was not significantly reduced. Pulmonary function was not found to be different after a work shift, compared to the same measurement taken before the shift. No differences in chest x-rays were observed between exposed and control workers.

Occupational exposure to formaldehyde concentrations estimated to be 0.025 ppm (0.038 mg/m³) for greater than six years resulted in complaints by 22 exposed workers of respiratory, gastrointestinal, musculoskeletal, and cardiovascular problems, and in elevated formic acid excretion in the urine (Srivastava et al., 1992). A control group of twenty seven workers unexposed to formaldehyde was used for comparison. A significantly higher incidence of abnormal chest x-rays was also observed in formaldehyde-exposed workers compared with controls.

Chemical plant workers (70 subjects) were exposed to a mean of 0.17 ppm (0.26 mg/m³) formaldehyde for an unspecified duration (Holmstrom and Wilhelmsson, 1988). Compared with 36 control workers not exposed to formaldehyde, the exposed subjects exhibited a higher frequency of eye, nose, and deep airway discomfort. In addition, the exposed subjects had diminished olfactory ability, delayed mucociliary clearance, and decreased FVC.

Alexandersson et al. (1982) compared the irritant symptoms and pulmonary function of 47 carpentry workers exposed to a mean concentration of formaldehyde of 0.36 ppm (range = 0.04 - 1.25 ppm) with 20 unexposed controls. The average length of employment for the exposed workers was 5.9 years. Symptoms of eye and throat irritation as well as airway obstruction were more common in exposed workers. In addition, a significant reduction in FEV₁, FEV₁/FVC, and MMF was observed in exposed workers compared with controls.

Horvath et al. (1988) compared subjective irritation and pulmonary function in 109 workers exposed to formaldehyde with similar measures in a control group of 254 subjects. The formaldehyde concentrations for the exposed and control groups were 0.69 ppm (1.04 mg/m³) and 0.05 ppm (0.08 mg/m³), respectively. Mean formaldehyde concentration in the pre-shift testing facility and the state (Wisconsin) ambient outdoor - formaldehyde level were both 0.04 ppm (0.06 mg/m³). Duration of formaldehyde exposure was not stated. Subjects were evaluated pre- and post work-shift and compared with control subjects. Significant differences in symptoms of irritation, FEV₁, FEV₁/FVC ratio, FEF₅₀, FEF₂₅, and FEF₇₅ were found when comparing exposed subjects' pre- and post work-shift values. However, the pre-workshift values were not different from controls.

The binding of formaldehyde to endogenous proteins creates haptens that can elicit an immune response. Chronic exposure to formaldehyde has been associated with immunological hypersensitivity as measured by elevated circulating IgG and IgE autoantibodies to human serum albumin (Thrasher et al., 1987). In addition, a decrease in the proportion of T-cells was observed, indicating altered immunity. Thrasher et al. (1990) later found that long-term exposure to formaldehyde was associated with autoantibodies, immune activation, and formaldehyde-albumin adducts in patients occupationally exposed, or residents of mobile homes or of homes containing particleboard sub-flooring. The authors suggest that the hypersensitivity induced by formaldehyde may account for a mechanism for asthma and other health complaints associated with formaldehyde exposure.

An epidemiological study of the effects of formaldehyde on 367 textile and shoe manufacturing workers employed for a mean duration of 12 years showed no significant association between formaldehyde exposure, pulmonary function (FVC, FEV₁, and PEF) in normal or asthmatic workers, and occurrence of specific IgE antibodies to formaldehyde (Gorski and Krakowiak, 1991). The concentrations of formaldehyde did not exceed 0.5 ppm (0.75 mg/m³).

Workers (38 total) exposed for a mean duration of 7.8 years to 0.11 - 2.12 ppm (mean = 0.33 ppm) formaldehyde were studied for their symptomatology, lung function, and total IgG and IgE levels in the serum (Alexandersson and Hedenstierna, 1988). The control group consisted of 18 unexposed individuals. Significant decrements in pulmonary function, FVC ($p < 0.01$) and FEV₁ ($p < 0.05$) were observed, compared with the controls. Eye, nose, and throat irritation was also reported more frequently by the exposed group. No correlation was found between duration of exposure, or formaldehyde concentration, and the presence of IgE and IgG antibodies.

As described in section 5.1, chronic or repeated exposure to formaldehyde may influence the response of asthmatics to acute or short-term challenges. In the study by Burge et al. (1985) late asthmatic reactions were noted in 3 out of 15 occupationally exposed workers after short-duration exposure to 1.5 – 20.6 ppm formaldehyde. Similarly, among workers with occupational

exposure to formaldehyde, asthmatic responses (decreased PEF, FVC, and FEV₁) were reported in 12 workers challenged with 1.67 ppm (2.5 mg/m³) formaldehyde (Nordman et al., 1985) and in 5 of 28 hemodialysis workers following challenge with formaldehyde vapors (concentration not measured) (Hendrick and Lane, 1977). In contrast, Sheppard et al. (1984) found that in asthmatics not occupationally exposed to formaldehyde, a 10-minute challenge with 3 ppm formaldehyde coupled with moderate exercise did not induce significant changes in airway resistance or thoracic gas volume. Thus individuals with chronic formaldehyde exposure may be at greater risk for adverse responses to acute exposures. These individuals may have been sensitized immunologically, as in the cases of elevated circulating antibodies, or rendered neurologically hyperresponsive, following repeated or chronic exposures to formaldehyde (Sorg et al., 2001a,b).

6.2 Chronic Toxicity to Infants and Children

There are few studies that compare the effects of chronic formaldehyde exposure on children versus adults. Among those that do there is evidence that children are more susceptible to the adverse effects of chronic exposure. Krzyzanowski et al. (1990) assessed chronic pulmonary symptoms and function in 298 children (6-15 years of age) and 613 adults (> 15 years of age) in relation to measured formaldehyde levels in their homes. Information on pulmonary symptoms and doctor-diagnosed asthma and chronic bronchitis was collected by questionnaire. Pulmonary function was assessed as peak expiratory flow rates (PEFR) measured up to four times a day. The prevalence of chronic respiratory symptoms in children was not related to formaldehyde levels measured in tertiles (< 40, 41-60, > 60 ppb). However, doctor-diagnosed asthma and chronic bronchitis were more prevalent in houses with elevated formaldehyde (p for trend < 0.02). This effect was driven by the high disease prevalence observed in homes with kitchen formaldehyde levels >60 ppb, and was especially pronounced among children with concomitant exposure to environmental tobacco smoke (Table 6.2.1). By comparison, in adults, while the prevalence rates of chronic cough and wheeze were somewhat higher in houses with higher formaldehyde, none of the respiratory symptoms or diseases was significantly related to formaldehyde levels.

TABLE 6.2.1 PREVALENCE RATE (PER 100) OF DIAGNOSED BRONCHITIS AND ASTHMA IN CHILDREN WITH FORMALDEHYDE (FROM KRZYZANOWSKI ET AL., 1990)

	Formaldehyde (ppb)			P value X^2 trend
	≤ 40 (N)	41-60 (N)	>60 (N)	
Bronchitis				
Household mean	3.5 (258)	17.2 (29)	9.1 (11)	<0.02
Main room mean	3.2 (253)	15.6 (32)	9.1 (11)	<0.01
Bedroom mean	3.8 (262)	16.0 (25)	9.1 (11)	<0.04
Subject's bedroom	4.7 (256)	6.7 (30)	11.1 (9)	>0.35
Kitchen				
No ETS	4.3 (141)	0 (12)	10.0 (10)	>0.40
ETS	1.9 (106)	0 (10)	45.5 (11)	<0.001
Asthma				
All children	11.7 (256)	4.2 (24)	23.8 (21)	<0.03
No ETS	8.5 (142)	8.3 (12)	0 (10)	>0.50
ETS	15.1 (106)	0 (12)	45.5 (11)	<0.05

In a random effects model, Krzyzanowski et al. (1990) reported that lung function (PEFR) in children, but not adults, was significantly decreased by formaldehyde (coefficient \pm SE: -1.28 ± 0.46 vs 0.09 ± 0.27). Measurements of PEFR in the morning suggested that children with asthma ($n = 4$) were more severely affected than healthy children (coefficient \pm SE: -1.45 ± 0.53 vs 0.09 ± 0.15) (Table 6.2.2). Compared to children, the effects of formaldehyde on pulmonary function in adults were smaller, transient, limited to morning measurements, and generally most pronounced among smokers exposed to the higher levels of formaldehyde. These studies suggest that children may be more susceptible to the effects of chronic formaldehyde exposure on lung function than are adults.

TABLE 6.2.2 RELATION OF PEFR (L/MIN) TO INDOOR FORMALDEHYDE

(from Krzyzanowski et al., 1990)

Factor	Child coefficient \pm SE	Adult coefficient \pm SE
HCHO house mean	-1.28 ± 0.46	0.09 ± 0.27
Morning vs bedtime	-6.10 ± 3.0	-5.90 ± 1.10
HCHO bdrm mean/morning	0.09 ± 0.15	-0.07 ± 0.04
HCHO bdrm mean/morning/asthma	-1.45 ± 0.53	

Among studies of children only, a case-control study by Rumchev et al. (2002) examined risk factors for asthma among young children (6 mo- 3 yr). Cases included children with clinically-diagnosed asthma, and controls were children of the same age group without such a diagnosis. Formaldehyde levels were measured in the homes, once in summer and once in winter. Questionnaires were used to assess potential risk factors for asthma and to collect parental reports of respiratory symptoms characteristic of asthma (cough, shortness of breath, wheeze, runny nose, trouble breathing, and hay fever) in their children. Formaldehyde levels were higher

in the homes of children exhibiting respiratory symptoms. Estimates of the relative risk for clinically-diagnosed asthma (odds ratios) were adjusted for measured indoor air pollutants, relative humidity, temperature, atopy, family history of asthma, age, gender, socioeconomic status, pets, smoke exposure, air conditioning, and gas appliances. Compared with children exposed to < 8 ppb, children in homes with formaldehyde levels > 49 ppb had a 39% higher risk of asthma ($p < 0.05$) after adjusting for common asthma risk factors.

Franklin et al. (2000) measured exhaled nitric oxide (eNO) levels in 224 children 6-13 years of age as an indicator of inflammation of the lower airways following chronic low-level formaldehyde exposure in the home. While there was no effect of formaldehyde on lung function measured by spirometry, eNO was significantly higher in children from homes with average formaldehyde levels ≥ 50 ppb compared with those from homes with levels ≤ 50 ppb (15.5 ppb eNO vs 8.7; $p = 0.02$).

Garrett et al. (1999) examined the association between formaldehyde levels at home (median $15.8 \mu\text{g}/\text{m}^3$; maximum $139 \mu\text{g}/\text{m}^3$) and atopy and allergic sensitization in 148 children, 7-14 years of age. The risk of atopy increased by 40% with each $10 \mu\text{g}/\text{m}^3$ increase in bedroom formaldehyde. Two measures of allergic sensitization to twelve common environmental allergens, the number of positive skin prick tests and maximum wheal size, both showed linear associations with increasing maximum formaldehyde exposure levels. After adjusting for parental asthma and allergy, there was no evidence of an association between asthma in the children and formaldehyde levels. However, these data do suggest that formaldehyde levels commonly found in homes can enhance sensitization of children to common aeroallergens.

Of the numerous, primarily occupational, studies in adults, the NOAEL and LOAEL are $17 \mu\text{g}/\text{m}^3$ (14 ppb) and $101 \mu\text{g}/\text{m}^3$ (81 ppb), respectively, after adjustment for exposure continuity. These values are based on data on nasal and eye irritation as observed in Wilhelmsson and Holstrom (1992), and histological lesions in the nasal cavity as documented in Edling et al. (1988). However, studies in children, including the Krzyzanowski study above, indicate adverse health impacts in children at concentrations as low as 30 ppb. Wantke et al. (1996) reported that formaldehyde-specific IgE and respiratory symptoms were reduced when children transferred from schools with formaldehyde concentrations of 43 to 75 ppb to schools with concentrations of 23 to 29 ppb. While these human studies are not entirely consistent with each other, and there is potential for confounding in each, nevertheless, taken together, they suggest that children may be more sensitive to formaldehyde toxicity than adults.

A potential role for formaldehyde, GSNOR and its metabolizing enzyme, GSNOR, in asthma is described in Section 5 above. The activity of GSNOR tends to be higher, and the levels of GSNOR lower, in the lungs of asthmatics compared to non-asthmatics. This connection prompted Wu et al. (2007) to investigate whether genetic variation in GSNOR is associated with childhood asthma and atopy. The study group included 532 children, aged 4 to 17 with clinically diagnosed asthma, and their parents. Seven single nucleotide polymorphisms (SNPs) in GSNOR were genotyped in DNA extracted from lymphocytes to examine the relationship between common haplotypes and asthma. Atopy was determined with skin prick tests using a collection of 25 aeroallergens. Two of the GSNOR SNPs were associated with increased risk of asthma, but none was associated with atopy. Whereas a lower risk for asthma was associated with one (RR 0.77; 95% CI 0.61-0.97) or two (RR 0.66; 95% CI 0.44-0.99) copies of the minor A allele of

SNP rs1154404, homozygosity for the major T allele of this SNP carried an increased risk of asthma. Homozygosity for the minor allele of SNP rs28730619 also carried an increased risk of asthma (RR 1.60; 95% CI 1.13-2.26; $p = 0.0077$). In the haplotype analysis, children with the most common GSNOR haplotype (GTCCG), that contained the major T allele of rs1154404 and the minor G allele of rs28730619, were at increased risk of childhood asthma. These results thus suggest that variants in GSNOR genotype influence childhood asthma susceptibility.

It should be noted that while term neonates have high levels of reduced glutathione in the fluid lining the lungs, these levels drop rapidly after birth. However, among premature infants, glutathione levels are typically substantially below those of term infants (Grigg et al., 1993) and adults (Reise et al., 1997). As a result of low levels of a critical component of formaldehyde metabolism, glutathione, these infants may be at increased risk from formaldehyde exposure.

6.3 Chronic Toxicity to Experimental Animals

Studies of the effects of chronic formaldehyde exposure in experimental animals tend to focus on lesions in the upper respiratory tract and the hyperplastic or metaplastic changes observed in the respiratory epithelium. Systemic effects, such as changes in body or organ weight, or blood chemistry, appear to be secondary to the effects of the olfactory irritation on feeding behavior. There is also evidence that repeated or long-term exposure to formaldehyde may cause neurologically-based hyperresponsiveness to formaldehyde (Sorg et al., 2001a) and altered expression of stress hormones (Sorg et al., 2001b).

In studies examining respiratory effects, Fischer-344 rats and B6C3F1 mice (120 animals/sex) were exposed to concentrations of 0, 2.0, 5.6, or 14.3 ppm formaldehyde vapor for 6 hours/day, 5 days/week for 24 months (Kerns et al., 1983). The exposure period was followed by up to six months of non-exposure. Interim sacrifices were conducted at 6, 12, 18, 24, 27, and 30 months. Both male and female rats in the 5.6 and 14.3 ppm groups demonstrated decreased body weights over the two-year period. At the 6 month sacrifice, the rats exposed to 14.3 ppm formaldehyde had non-neoplastic lesions of epithelial dysplasia in the nasal septum and turbinates. As the study progressed, epithelial dysplasia, squamous dysplasia, and mucopurulent rhinitis increased in severity and distribution in all exposure groups. In mice, cumulative survival decreased in males from 6 months to the end of the study. Serous rhinitis was detected at 6 months in the 14.3 ppm group of mice. Metaplastic and dysplastic changes were noted at 18 months in most rats in the 14.3 ppm group and in a few mice in the 5.6 ppm exposure group. By 24 months, the majority of mice in the 14.3 ppm group had metaplastic and dysplastic changes associated with serous rhinitis, in contrast to a few mice in the 5.6 ppm group and a few in the 2 ppm group (exact number not given).

Woutersen et al. (1989) exposed male Wistar rats (60 animals/group) 6 hours/day for 5 days/week to 0, 0.1, 1.0 and 10 ppm formaldehyde vapor for 28 months. Compound-related nasal lesions of the respiratory and olfactory epithelium were observed only in the 10 ppm group. In the respiratory epithelium, the lesions consisted of rhinitis, squamous metaplasia and basal cell/pseudoepithelial hyperplasia. In the olfactory region, the lesions included epithelial degeneration and rhinitis. No differences in behavior or mortality were noted among the various groups. However, growth retardation was observed in the 10 ppm group from day 14 onwards. In a parallel study, male Wistar rats were exposed to 0, 0.1, 1.0 and 10 ppm formaldehyde for 3

months followed by a 25-month observation period. Compound-related histopathological changes were found only in the noses of the 10 ppm group and comprised increased incidence of squamous metaplasia of the respiratory epithelium, and rhinitis.

In a chronic exposure study that primarily investigated aspects of nasal tumor development, Monticello et al. (1996) examined nasal cavities of male F-344 rats (0-10 ppm, 90 animals/group; 15 ppm, 147 animals) following exposure to 0, 0.7, 2, 6, 10, and 15 ppm formaldehyde for 6 hours/day, 5 days/week for 24 months. Treatment-related decreases in survival were apparent only in the 15 ppm group. Nasal lesions at the two highest doses included epithelial hypertrophy and hyperplasia, squamous metaplasia, and a mixed inflammatory cell infiltrate. Lesions in the 6 ppm group were minimal to absent and limited to focal squamous metaplasia in the anterior regions of the nasal cavity. No formaldehyde-induced lesions were observed in the 0.7 or 2 ppm groups.

Kamata et al. (1997) exposed 32 male F-344 rats/group to gaseous formaldehyde at 0, 0.3, 2, and 15 ppm 6 hours/day, 5 days/week for up to 28 weeks. A room control, non-exposed group was also included in the study. Five animals per group were randomly selected at the end of the 12, 18, and 24 months, and surviving animals at 28 months were sacrificed for full pathological evaluation. Behavioral effects related to sensory irritation were evident in the 15 ppm group. Significant decreases in food consumption, body weight and survival were also evident in this group. No exposure-related hematological findings were observed. Biochemical and organ weight examination revealed decreased triglyceride levels and absolute liver weights at the highest exposure, but was likely related to reduced food consumption. Abnormal histopathological findings were confined to the nasal cavity. Inflammatory cell infiltration, erosion or edema of the nasal cavity was evident in all groups, including controls. Significantly increased incidence of non-proliferative (squamous cell metaplasia without epithelial cell hyperplasia) and proliferative lesions (epithelial cell hyperplasia with squamous cell metaplasia) were observed in the nasal cavities beginning at 2 ppm. In the 0.3 ppm group, a non-significant increase in proliferative nasal lesions (4/20 animals) were observed in rats that were either sacrificed or died following the 18th month of exposure.

Rusch et al. (1983) exposed groups of 6 male cynomolgus monkeys, 20 male or female rats, and 10 male or female hamsters to 0, 0.2, 1.0, or 3.0 ppm (0, 0.24, 1.2, or 3.7 mg/m³) formaldehyde vapor for 22 hours/day, 7 days/week for 26 weeks. There was no treatment-related mortality during the study. In monkeys, the most significant findings were hoarseness, congestion and squamous metaplasia of the nasal turbinates in 6/6 monkeys exposed to 2.95 ppm. There were no signs of toxicity in the lower exposure groups. In the rat, squamous metaplasia and basal cell hyperplasia of the nasal epithelia were significantly increased in rats exposed to 2.95 ppm. The same group exhibited decreased body weights and decreased liver weights. In contrast to monkeys and rats, hamsters did not show any signs of response to exposure, even at 2.95 ppm.

Kimbell et al. (1997) exposed male F-344 rats (≤ 6 /group) to 0, 0.7, 2, 6, 10, and 15 ppm 6 hr/day, 5 days/week for 6 months. Squamous metaplasia was not observed in any regions of the nasal cavity in any of the control, 0.7, or 2 ppm groups. However, the extent and incidence of squamous metaplasia in the nasal cavity increased with increasing dose beginning at 6 ppm.

In subchronic studies, Wilmer et al. (1989) found that intermittent (8 hours/day, 5 days/week) exposures of rats to 4 ppm formaldehyde for 13 weeks resulted in significant histological changes in the nasal septum and turbinates. In contrast, continuous exposure of rats for 13 weeks to 2 ppm formaldehyde did not produce significant lesions. This study revealed the concentration dependent nature of the nasal lesions caused by formaldehyde exposure. Zwart et al. (1988) exposed male and female Wistar rats (50 animals/group/sex) to 0, 0.3, 1, and 3 ppm formaldehyde vapor for 6 hr/day, 5 days/week for 13 weeks. Compound related histopathological nasal changes varying from epithelial disarrangement to epithelial hyperplasia and squamous metaplasia were found in the 3 ppm group, and were restricted to a small area of the anterior respiratory epithelium. These changes were confirmed by electron microscopy and were not observed in other groups.

Woutersen et al. (1989) exposed rats (20 per group) to 0, 1, 10, or 20 ppm formaldehyde 6 hours/day, 5 days/week for 13 weeks. Rats exposed to 20 ppm displayed retarded growth, yellowing of the fur, and significant histological lesions in the respiratory epithelium. Exposure to 10 ppm did not affect growth, but resulted in significant histological lesions in the respiratory tract. No effects on specific organ weights, blood chemistries, liver glutathione levels, or urinalysis were detected at any level. No significant adverse effects were seen at the 1.0 ppm exposure level.

Appelman et al. (1988) found significant nasal lesions in rats (20 per group; 0, 0.1, 1.0, or 10.0 ppm) exposed to 10 ppm formaldehyde 6 hours/day, 5 days/week for 52 weeks, but exposure to 1.0 ppm or less for this period did not result in nasal histological lesions. However, the rats exposed to formaldehyde displayed decreased body weight in all groups compared with controls.

Apfelbach and Weiler (1991) determined that rats (5 exposed, 10 controls) exposed to 0.25 ppm (0.38 mg/m³) formaldehyde for 130 days lost the olfactory ability to detect ethyl acetate odor.

Maronpot et al. (1986) exposed groups of 20 mice to 0, 2, 4, 10, 20, or 40 ppm formaldehyde 6 hours/day, 5 days/week, for 13 weeks. Histological lesions in the upper respiratory epithelium were seen in animals exposed to 10 ppm or greater. Exposure to 40 ppm was lethal to the mice.

A six-month exposure of rats to 0, 0.5, 3, and 15 ppm formaldehyde (3 rats per group) resulted in significantly elevated total lung cytochrome P450 in all formaldehyde-exposed groups (Dallas et al., 1989). The degree of P450 induction was highest after 4 days exposure and decreased slightly over the course of the experiment.

A series of studies have addressed the effects of long-term repeated exposures to formaldehyde on altered functioning of the hypothalamic-pituitary-adrenal (HPA) axis (Sorg et al., 2001b) and on neurobehavioral changes in rats (Sorg et al., 2001a). To study formaldehyde's effects on the HPA, Sorg et al. (2001b) measured corticosterone levels in the trunk blood of male Sprague-Dawley rats 20 or 60 min following acute chamber exposures to air or formaldehyde (0.7 or 2.4 ppm). All groups showed increased corticosterone levels above naive basal levels at 20 min followed by a return to baseline by 60 min, with no differences between treatment groups. A second experiment assessed the effects of repeated formaldehyde exposure (1 h/day, 5 days/week for 2 or 4 weeks) on basal corticosterone levels and those after a final challenge. Basal corticosterone levels were increased above naive values after 2 week exposure to air or 0.7 ppm

formaldehyde. By 4 weeks, corticosterone levels in the air group returned to naive values, but remained elevated in the 0.7 ppm formaldehyde group. There were no differences in basal corticosterone levels among either 2.4 ppm exposed groups. After a final air or formaldehyde challenge, the 2 and 4 week air and 0.7 ppm formaldehyde groups had elevated corticosterone levels similar to their acute response, while in the 2 and 4 week 2.4 ppm formaldehyde groups, corticosterone levels were higher than their acute response levels, indicating enhanced reactivity of the HPA axis to subsequent formaldehyde. It thus appears that repeated low-level formaldehyde exposure alters HPA axis functioning and the release of stress hormones. Since glucocorticoids may stimulate or inhibit the synthesis of surfactant-associated proteins in the lung (Liley et al., 1988), the alteration of HPA function may represent another pathway by which formaldehyde affects pulmonary function. For example, the pulmonary surfactants that regulate surface tension in the lungs are in turn regulated by surfactant-associated proteins. Reports of lower airway discomfort associated with chronic formaldehyde exposure may be related to the altered release or activity of these surfactant-associated proteins in the lung.

In another study of the effects of formaldehyde and the hypothalamus-pituitary-adrenal (HPA) axis, Sari et al. (2004) exposed female C3H/He mice to formaldehyde (0, 80, 400, 2000 ppb) by inhalation for 16 h/day, 5 days/week, for 12 weeks. Immunocytochemistry was used to examine corticotropin releasing hormone (CRH)-immunoreactive (ir) neurons in the hypothalamus, and adrenocorticotropin hormone (ACTH)-ir cells in the pituitary. RT-PCR was used to quantify ACTH mRNA in the pituitary. Two groups of female mice were exposed, one of which comprised control mice with no allergen exposure. The other group was made allergic by injection of ovalbumin and alum prior to exposure to formaldehyde. Animals in the second group were further exposed to aerosolized ovalbumin as a booster four times during the exposure period. In the non-allergic group, formaldehyde caused a dose-dependent increase in the number of CRH-ir neurons with a similar pattern of increases in ACTHir cells and ACTH mRNA. The allergic mice showed an increase in basal levels of all these markers of HPA activity, and were responsive to the lowest concentration of formaldehyde. Thus at low levels of exposure, allergen and formaldehyde exposure exacerbate each other's effects on the stress response of the HPA.

7. Developmental and Reproductive Toxicity

In humans there are few data on the association of teratogenicity or adverse reproductive effects with formaldehyde exposure. Existing data do not suggest that formaldehyde, by inhalation or oral routes, produces significant teratogenic or reproductive effects (ATSDR, 1999).

A developmental toxicity study on formaldehyde was conducted by Martin (1990). Pregnant rats (25 per group) were exposed to 0, 2, 5, or 10 ppm formaldehyde for 6 hours/day, during days 6-15 of gestation. Although exposure to 10 ppm formaldehyde resulted in reduced food consumption and body weight gain in the maternal rats, no effects on the number, viability or normal development of the fetuses were seen. In addition, Saillenfait et al. (1989) exposed pregnant rats (25 per group) to 0, 5, 10, 20, or 40 ppm formaldehyde from days 6 - 20 of gestation. Maternal weight gain and fetal weight were significantly reduced in the 40 ppm exposure group. No significant fetotoxicity or teratogenic defects were observed at formaldehyde levels that were not also maternally toxic.

Evidence of embryotoxicity was reported by Kitaeva et al. (1990) in embryos of rats that had been exposed to formaldehyde by inhalation 4 h/d, 5 d/wk for 4 months. At 1.5 mg/m³, but not at 0.5 mg/m³, there was a significant increase in the proportion of degenerate embryos. By comparison, the bone marrow cells of the mothers appeared to be more sensitive to formaldehyde as shown by significant increases in the numbers of cells with aberrations, and the numbers of chromosomes with aberrations and aneuploidy at both dose levels.

In the context of developmental susceptibility to formaldehyde exposure, as noted above, the respiratory tract lining fluid (RTLFL) protecting the lungs is often lower in glutathione levels than is the RTLFL of adult lungs (Reise et al., 1997). This is especially true in premature infants who later develop chronic lung disease (Grigg et al., 1993). As glutathione is central to the lungs' antioxidant defenses, and is involved in the metabolism of inhaled formaldehyde, this relative deficiency may make the neonate's and infant's developing lungs more susceptible to toxic insult. It should be noted that ascorbate is also an important component of the lung's antioxidant defense, especially when glutathione levels are depressed (Jain et al., 1992). In healthy lungs, ascorbate helps to maintain glutathione levels. However, as is the case for glutathione, ascorbate levels in RTLFL drop during the first week following birth (Vyas et al., 2001), potentially adding to the neonate's susceptibility to glutathione-reactive substances. Indeed, alterations in lung development following early life air toxicant exposure has been shown for environmental tobacco smoke (Wang and Pinkerton, 2007) and ozone (Plopper et al., 2007). Whether early life exposure to formaldehyde has similar effects on lung development remains to be demonstrated. However, there is concern that allergen exposure can modulate trophic interactions of conducting airway epithelial and interstitial wall components (Finkelstein and Johnston, 2004) and alter postnatal development of the airways (Plopper et al., 2007). This, coupled with the ability of formaldehyde to enhance the immune response to proteins/allergens with which it binds (Thrasher et al., 1987, 1990), may render developing lungs more susceptible to formaldehyde exposure. If evidence of such developmental effects associated with formaldehyde exposure becomes available, a re-evaluation of the REL for formaldehyde may be necessary.

8. Derivation of Reference Exposure Levels

8.1 Formaldehyde Acute Reference Exposure Level

<i>Study</i>	Kulle et al., 1987
<i>Study population</i>	19 nonasthmatic, nonsmoking humans
<i>Exposure method</i>	Whole body to 0.5-3.0 ppm
<i>Exposure continuity</i>	Single exposure per concentration
<i>Exposure duration</i>	3 hr
<i>Critical effects</i>	mild and moderate eye irritation
<i>LOAEL</i>	1 ppm
<i>NOAEL</i>	0.5 ppm
<i>Benchmark concentration</i>	0.44 ppm
<i>Time-adjusted exposure</i>	not applied
<i>Human Equivalent Concentration</i>	not applied
<i>LOAEL uncertainty factor (UF_L)</i>	not applied
<i>Subchronic uncertainty factor (UF_s)</i>	not applied

<i>Interspecies uncertainty factor</i>	
<i>Toxicokinetic (UF_{A-k})</i>	1 (default, human study)
<i>Toxicodynamic (UF_{A-d})</i>	1 (default, human study)
<i>Intraspecies uncertainty factor</i>	
<i>Toxicokinetic (UF_{H-k})</i>	1 (site of contact; no systemic effects)
<i>Toxicodynamic (UF_{H-d})</i>	10 (asthma exacerbation in children)
<i>Cumulative uncertainty factor</i>	10
<i>Reference Exposure Level</i>	55 µg/m³ (44 ppb)

Acute Reference Exposure Levels are levels at which intermittent one-hour exposures are not expected to result in adverse health effects (see Section 5 of the Technical Support Document).

Kulle et al (1987) was chosen as the critical study for the determination of the acute REL as it used a sensitive endpoint, eye irritation. It featured human subjects showing significant ($p < 0.05$) responses with short-term exposures to a range of formaldehyde concentrations, and the data permitted the use of a benchmark concentration (BMC) approach. As described in the technical support document, OEHHA recommends the use of the BMC approach whenever the available data support it as the BMC method provides a more statistically sound estimate of the point of departure in the REL determination.

The proposed acute REL was based on a $BMCL_{05}$ for eye irritation, estimated using log-probit analysis (Crump, 1984). The $BMCL_{05}$ is defined as the 95% lower confidence limit of the concentration expected to produce a response rate of 5%. The resulting $BMCL_{05}$ from this analysis was 0.44 ppm (0.53 mg/m³) formaldehyde. The endpoint of eye irritancy appears to be more a function of formaldehyde concentration rather than duration of exposure (Yang et al., 2001), so no time correction factor was applied. An uncertainty factor (UF_{H-k}) of 1 was used since sensory irritation is not expected to involve large toxicokinetic differences among individuals. Although the toxicological endpoint is eye irritation, the REL should protect against all possible adverse effects. The respiratory irritant effect, with documented potential to exacerbate asthma, is clearly an effect with the potential to differentially impact infants and children. In addition, the ability of formaldehyde to exacerbate the immune response to aeroallergens is of especial concern during development of the lungs. The toxicodynamic component of the intraspecies uncertainty factor UF_{H-d} is therefore assigned an increased value of 10 to account for potential asthma exacerbation. These considerations are applied equally to the acute, 8-hour and chronic REL.

As noted in Section 5.1, contact lens wearers appear to be at greater risk for ocular irritation with formaldehyde exposure. However, since contact lens users, and infants and children are generally mutually exclusive groups, it is expected that with the ten-fold toxicodynamic UF_{H-d} described above, the acute REL should be adequately protective of these individuals as well.

8.2 Formaldehyde 8-Hour Reference Exposure Level

<i>Study</i>	Wilhelmsson and Holmstrom, 1992
<i>Study population</i>	66 chemical plant workers
<i>Exposure method</i>	Discontinuous occupational exposure
<i>Exposure continuity</i>	8 hr/day, 5 days/week (assumed)
<i>Exposure duration</i>	10 years (average); range 1-36 years
<i>Critical effects</i>	Nasal obstruction and discomfort, lower airway discomfort, and eye irritation.
<i>LOAEL</i>	Mean 0.26 mg/m ³ (range 0.05 – 0.6 mg/m ³) (described as exposed group)
<i>NOAEL</i>	Mean of 0.09 mg/m ³ (described as control group of office workers)
<i>Benchmark concentration</i>	not derived
<i>Time-adjusted exposure</i>	0.09 mg/m ³ (time adjustment not applied)
<i>Human Equivalent Concentration</i>	not applied
<i>LOAEL uncertainty factor (UF_L)</i>	1 (NOAEL observed)
<i>Subchronic uncertainty factor (UFs)</i>	not applied
<i>Interspecies Uncertainty Factor</i>	
<i>Toxicokinetic (UF_{A-k})</i>	1 (default, human study)
<i>Toxicodynamic (UF_{A-d})</i>	1 (default, human study)
<i>Intraspecies Uncertainty Factor</i>	
<i>Toxicokinetic (UF_{H-k})</i>	1 (site of contact; no systemic effects)
<i>Toxicodynamic (UF_{H-d})</i>	10 (asthma exacerbation in children)
<i>Cumulative uncertainty factor</i>	10
<i>Reference Exposure Level</i>	9 µg/m³ (7 ppb)

The 8-hour Reference Exposure Level is a concentration at or below which adverse noncancer health effects would not be anticipated for repeated 8-hour exposures (see Section 6 in the Technical Support Document).

The 8-hour REL is based on the occupational study by Wilhelmsson and Holmstrom (1992). This study evaluated the effects of formaldehyde on the upper airways of adult human subjects exposed to a mean formaldehyde concentration of 0.26 mg/m³ during the work day compared with a referent group exposed to 0.09 mg/m³. The critical effects in this study included nasal obstruction and discomfort, lower airway discomfort, and eye irritation. A NOAEL and a LOAEL may be derived from these data but no other dose-response information was provided. This study included only adults, but there is evidence that children may be more susceptible to long term exposures to formaldehyde than are adults. Thus, in the absence of child-specific data, an intraspecies uncertainty factor of 10 for toxicodynamic variability and developmental susceptibility was applied.

For comparison, the 8-hour REL of 9 $\mu\text{g}/\text{m}^3$ is similar to the value of 10 $\mu\text{g}/\text{m}^3$ based on increased pulmonary resistance in guinea pigs following an 8 hr exposure to 0.11 – 1.05 ppm formaldehyde (Swiecichowski et al., 1993). The NOAEL of 0.59 ppm in guinea pigs was adjusted to a Human Equivalent Concentration (HEC) of 0.49 ppm with a regional gas dose ratio (RGDR) of 0.826. Use of the HEC adjustment entails an interspecies uncertainty factor of 6, while an intraspecies uncertainty factor of 10 addresses toxicodynamic variability.

<i>Study</i>	Swiecichowski et al., 1993
<i>Study population</i>	25-35 adult male guinea pigs
<i>Exposure method</i>	Whole body exposure
<i>Exposure continuity</i>	
<i>Exposure duration</i>	8 hr
<i>Critical effects</i>	Increased specific pulmonary resistance
<i>LOAEL</i>	1.0 ppm
<i>NOAEL</i>	0.59 ppm
<i>Benchmark concentration</i>	not derived
<i>Time-adjusted exposure</i>	not applied
<i>Human Equivalent Concentration</i>	0.49 ppm (610 $\mu\text{g}/\text{m}^3$) (0.59 * RGDR 0.826 for pulmonary effects)
<i>LOAEL uncertainty factor (UF_L)</i>	1 (default: NOAEL observed)
<i>Subchronic uncertainty factor (UF_s)</i>	not applied
<i>Interspecies Uncertainty Factor</i>	
<i>Toxicokinetic (UF_{A-k})</i>	6 (with HEC adjustment)
<i>Toxicodynamic (UF_{A-d})</i>	1 (with HEC adjustment)
<i>Intraspecies Uncertainty Factor</i>	
<i>Toxicokinetic (UF_{H-k})</i>	1 (no systemic effect)
<i>Toxicodynamic (UF_{H-d})</i>	10 (potential asthma exacerbation in children)
<i>Cumulative uncertainty factor</i>	60
<i>Reference Exposure Level</i>	10 $\mu\text{g}/\text{m}^3$ (8 ppb)

8.3 Formaldehyde Chronic Reference Exposure Level

<i>Study</i>	Wilhelmsson and Holmstrom, 1992 supported by Edling et al., 1988
<i>Study population</i>	66 human chemical plant workers
<i>Exposure method</i>	Discontinuous occupational exposure
<i>Exposure continuity</i>	8 hr/day, 5 days/week (assumed)
<i>Exposure duration</i>	10 years (average); range 1-36 years
<i>Critical effects</i>	Nasal obstruction and discomfort, lower airway discomfort.
<i>LOAEL</i>	Mean 0.26 mg/m ³ (range 0.05 – 0.6 mg/m ³) (described as exposed group)
<i>NOAEL</i>	Mean of 0.09 mg/m ³ (described as control group of office workers)
<i>Benchmark concentration</i>	not derived
<i>Time-adjusted exposure</i>	0.09 mg/m ³ for NOAEL group
<i>Human Equivalent Concentration</i>	not applied
<i>LOAEL uncertainty factor (UF_L)</i>	not applied
<i>Subchronic uncertainty factor (UF_s)</i>	not applied
<i>Interspecies uncertainty factor</i>	
<i>Toxicokinetic (UF_{A-k})</i>	1 (default, human study)
<i>Toxicodynamic (UF_{A-d})</i>	1 (default, human study)
<i>Intraspecies uncertainty factor</i>	
<i>Toxicokinetic (UF_{H-k})</i>	1 (no systemic effects)
<i>Toxicodynamic (UF_{H-d})</i>	10 (potential asthma exacerbation in children)
<i>Cumulative uncertainty factor</i>	10
<i>Reference Exposure Level</i>	9 µg/m³ (7 ppb)

The chronic Reference Exposure Level is a concentration at which adverse noncancer health effects would not be expected from chronic exposures (see Section 7 in the Technical Support Document).

The study by Wilhelmsson and Holmstrom (1992) was selected for development of the chronic REL as it investigated long-term exposure to formaldehyde relatively free of other confounding exposures. From this study it was possible to determine both a NOAEL and a LOAEL. Since this study included only adults, a combined intraspecies uncertainty factor of 10 for toxicodynamic variability was applied to account for the possibly greater susceptibility of children with long term exposures to formaldehyde.

The susceptibility of young children was examined in a study by Rumchev et al. (2002) that compared children (mean age 25 mo) with a clinical diagnosis of asthma to children without this diagnosis. The LOAEL used (60 µg/m³) represents the formaldehyde level at which the authors found a statistically elevated risk for asthma-related respiratory symptoms. For this comparison, the NOAEL was taken to be 30 µg/m³, the lower end of the NOAEL range. Intraspecies uncertainty factors of 3.16 for potential toxicodynamic variability and 1 for toxicokinetic

differences give a cumulative uncertainty factor of 3.16 for an inhalation chronic REL of $10 \mu\text{g}/\text{m}^3$ (8 ppb), similar to the chronic REL calculated from the critical study.

<i>Study</i>	Rumchev et al., 2002
<i>Study population</i>	88 asthmatic children (mean age 25 mo); 104 nonasthmatic controls (mean age 20 mo)
<i>Exposure method</i>	Ambient in home
<i>Exposure continuity</i>	Continuous assumed
<i>Exposure duration</i>	range 0.5-3 years
<i>Critical effects</i>	Parent-reported respiratory symptoms (cough, shortness of breath, wheeze, trouble breathing)
<i>LOAEL</i>	$60 \mu\text{g}/\text{m}^3$
<i>NOAEL</i>	$30 \mu\text{g}/\text{m}^3$ (lower limit of NOAEL range)
<i>Benchmark concentration</i>	not derived
<i>Time-adjusted exposure</i>	not applied
<i>Human Equivalent Concentration</i>	$30 \mu\text{g}/\text{m}^3$
<i>LOAEL uncertainty factor (UF_L)</i>	1
<i>Subchronic uncertainty factor (UF_s)</i>	not applied
<i>Interspecies uncertainty factor</i>	
<i>Toxicokinetic (UF_{A-k})</i>	1 (default, human study)
<i>Toxicodynamic (UF_{A-d})</i>	1 (default, human study)
<i>Intraspecies uncertainty factor</i>	
<i>Toxicokinetic (UF_{H-k})</i>	1 (study performed in children)
<i>Toxicodynamic (UF_{H-d})</i>	$\sqrt{10}$ (inter-individual variation)
<i>Cumulative uncertainty factor</i>	$\sqrt{10}$
<i>Reference Exposure Level</i>	$10 \mu\text{g}/\text{m}^3$ (8 ppb)

The Rumchev study supports an association with exposure to formaldehyde and the observation of asthma symptoms (cough, shortness of breath, wheeze, trouble breathing) in children. However, it was not selected for REL development due to the difficulties in distinguishing asthma from other wheezing conditions in the clinical diagnoses in such a young population. There are additional uncertainties associated with the exposure continuity, and the possibility of observational and/or recall bias in the parental reports of respiratory symptoms characteristic of asthma.

For comparison with the chronic REL of $9 \mu\text{g}/\text{m}^3$ (7 ppb) presented above, Table 8.3.1 below presents a summary of potential formaldehyde RELs based on chronic and subchronic animal studies originally presented in OEHHA (2000). The toxicological endpoint was nasal lesions, consisting principally of rhinitis, squamous metaplasia, and dysplasia of the respiratory epithelium.

The most striking observation is the similarity of potential RELs among the rat chronic studies (exposures ≥ 26 weeks) that contain a NOAEL. The range of RELs from these animal studies, 1.5 – 24.9 ppb, includes the proposed REL (7 ppb) based on a human study. Another related observation is that the NOAEL and LOAEL are similar among all the studies, regardless of exposure duration. The NOAEL and LOAEL are generally in the range of 1 - 4 ppm and 1 – 10 ppm, respectively, with the exception of the study by Kamata et al. (1997) that may be due to the

absence of a dose level between 2 and 0.3 ppm. It is also of interest that the studies of Rusch et al (1983) indicate that monkeys and rats are of about the same sensitivity. In addition, the results of the Rusch studies suggest that, at least for the endpoint of squamous metaplasia, formaldehyde concentration is more important than the total dose since these animals, receiving more continuous exposure, exhibited the same adverse effects seen in studies using more intermittent exposures.

ATSDR has estimated minimum risk levels (MRLs), defined as “an estimate of daily human exposure to a substance that is likely to be without an appreciable risk of adverse effects (noncarcinogenic) over a specified duration of exposure” (ATSDR, 1999). For formaldehyde inhalation exposures they describe as “acute” (≤ 14 days), the MRL is 40 ppb based on a LOAEL of 0.4 ppm from a study by Pazdrak et al. (1993), and a 9-fold uncertainty factor (3 for use of a LOAEL; 3 for intraspecies variability). This exposure period is much longer than the acute REL of one hour, but the acute REL represents possibly repeated exposures. The MRL for an “intermediate” exposure period of 15-364 days is 30 ppb based on a NOAEL of 0.98 ppm for clinical signs of nasopharyngeal irritation and lesions in the nasal epithelium in monkeys (Rusch et al., 1983). A chronic MRL (≥ 365 d) of 8 ppb was developed based on damage to nasal epithelium in chemical factory workers (Holmstrom et al., 1989). This number is similar to the chronic REL of 7 ppb reported here. The MRLs are more similar to the chronic RELs developed by OEHHA in that they assume continuous exposure over the specified time period rather than regular but periodic exposures, as assumed for the 8-hour RELs considered above. For 8-hr exposures, NIOSH (1988) suggested a TWA 8-hr REL of 16 ppb based on sensory irritation.

8.4 Formaldehyde as a Toxic Air Contaminant

Formaldehyde was identified by the ARB as a toxic air contaminant (TAC) in accordance with sections 39660-39662 of the California Health and Safety Code on March 12, 1992 (Title 17, California Code of Regulations, section 93001)(CCR, 2007). In view of the differential impacts on infants and children identified in Section 6.2, OEHHA recommends that formaldehyde be listed as a toxic air contaminant which may disproportionately impact children pursuant to Health and Safety Code, Section 39669.5(c).

Table 8.3.1. Summary of Chronic and Subchronic Formaldehyde Studies in Experimental Animals

Study	Animal	Duration	Exposure	LOAE		Time adj	DAF	LOAEL						Cum UF	REL ppb	REL µg/m ³
				L ppm	NOAEL ppm			UF	UFak	UFad	UFhk	UFhd	UFsc			
Woutersen 89	rat	28 mo	6 h 5 d	9.8	1	0.179	0.148	1	1	3.16	1	10	1	30	4.9	6.1
Kerns 83	rat	24 mo	6 h 5 d	2	n/a	0.357	0.296	6	1	3.16	1	10	1	200	1.5	1.8
Monticello 96	rat	24 mo	6 h 5 d	6.01	2.05	0.366	0.304	1	1	3.16	1	10	1	30	10.1	12.6
Kamata 97	rat	24-28 mo	6 h 5 d	2	0.3	0.054	0.044	1	1	3.16	1	10	1	30	1.5	1.8
Appelman 88	rat	52 wk	6 h 5 d	9.4	1	0.179	0.148	1	1	3.16	1	10	1	30	4.9	6.1
Rusch 83	rat	26 wk	22 h 7d	2.95	0.98	0.898	0.746	1	1	3.16	1	10	1	30	24.9	30.8
Kimbell 97	rat	26 wk	6 h 5 d	6	2	0.357	0.296	1	1	3.16	1	10	1	30	9.9	12.3
Wilmer 89	rat	13 wk	8 h 5 d	4	2	0.238	0.198	1	1	3.16	1	10	1	30	6.6	8.2
Woutersen 87	rat	13 wk	6 h 5 d	9.7	1	0.179	0.148	1	1	3.16	1	10	1	30	4.9	6.1
Zwart 88	rat	13 wk	6 h 5 d	2.98	1.01	0.180	0.15	1	1	3.16	1	10	1	30	5.0	6.2
Kerns 83	mouse	24 mo	6 h 5 d	5.6	2	0.357	0.296	1	2	3.16	1	10	1	60	4.9	6.1
Maronpot 86	mouse	13 wk	6 h 5 d	10.1	4.08	0.729	0.605	1	2	3.16	1	10	1	60	10.1	12.5
Rusch 83	monkey	26 wk	22 h 7d	2.95	0.98	0.898	not used	1	2	2	1	10	1	40	22.5	27.8

9. References

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CHRONIC TOXICITY SUMMARY

GLUTARALDEHYDE

(1,5-pentanedial; 1,5-pentanedione; glutaric dialdehyde; Aldesen; Cidex; Sonacide)

CAS Registry Number: 111-30-8

I. Chronic Toxicity Summary

<i>Inhalation reference exposure level</i>	0.08 µg/m³ (0.02 ppb)
<i>Critical effect(s)</i>	Squamous metaplasia of the respiratory epithelium in the nose of male and female mice
<i>Hazard index target(s)</i>	Respiratory system

II. Chemical Property Summary (HSDB, 1996; CRC, 1994; Chemfinder, 2000)

<i>Description</i>	Colorless liquid/oil
<i>Molecular formula</i>	C ₅ H ₈ O ₂
<i>Molecular weight</i>	100.12 g/mol
<i>Boiling point</i>	188°C (decomposes) (CRC, 1994)
<i>Melting point</i>	-6°C (Chemfinder, 2000)
<i>Solubility</i>	Soluble in water, alcohol, benzene
<i>Conversion factor</i>	4.1 µg/m ³ per ppb at 25°C

III. Major Uses and Sources

Glutaraldehyde is a chemical frequently used as a disinfectant and sterilizing agent against bacteria and viruses (2% solution), an embalming fluid and tissue fixative, a component of leather tanning solutions, and an intermediate in the production of certain sealants, resins, dyes, and electrical products (HSDB, 1996). For commercial purposes, solutions of 99%, 50%, and 20% are available. Glutaraldehyde is also an atmospheric reaction product of cyclohexene. The annual statewide industrial emissions from facilities reporting under the Air Toxics Hot Spots Act in California based on the most recent inventory were estimated to be 29,603 pounds of glutaraldehyde (CARB, 2000).

IV. Effects of Human Exposure

Evidence of the toxicity of glutaraldehyde to humans is limited to reports of occupational exposure from its use as a disinfectant and sterilizing agent. Frequently observed effects from exposure include skin sensitivity resulting in dermatitis, and irritation of the eyes and nose with accompanying rhinitis (Jordan *et al.*, 1972; Corrado *et al.*, 1986; Hansen, 1983; Wiggins *et al.*,

1989). Occupational asthma has also been reported among workers repeatedly exposed to glutaraldehyde, particularly respiratory technologists who use glutaraldehyde as a sterilizing agent for endoscopes (Chan-Yeung *et al.*, 1993; Stenton *et al.*, 1994; Gannon *et al.*, 1995). Quantitation of the exposure levels that led to glutaraldehyde sensitization was not available from the studies.

V. Effects of Animal Exposure

The histopathology of the respiratory tract in rats and mice exposed to glutaraldehyde by inhalation was examined (Gross *et al.*, 1994). F344 rats and B6C3F1 mice (20 animals of each sex and of each species at each exposure level for a total of 480 rodents) were continuously exposed to glutaraldehyde in recirculating exposure chambers at concentrations of 0, 62.5, 125, 250, 500, or 1000 ppb glutaraldehyde for one day, 4 days, 6 weeks, or 13 weeks. At termination, respiratory tract tissue as well as duodenum and any gross lesions were collected and formalin fixed. Animals were treated with tritiated thymidine two hours before termination to evaluate cell replication in certain respiratory tract tissues. Respiratory tract tissue sections were made as follows: transverse sections of the nose and trachea, frontal section of the carina, and longitudinal section of the lung. Ten male and 10 female mice exposed to 1000 ppb and one female mouse exposed to 500 ppb group died during the course of the study. Two male and 3 female rats exposed to 1000 ppb died during the course of the study. Histopathological examination of animals surviving to the end of the study entailed scoring the severity of the finding from “no response” to “very severe” response on a 0 to 5 scale. Unit length labeling index, the indicator of cell proliferation, was evaluated by autoradiography at two sites: the nasal vestibule and the dorsal atrioturbinate.

Lesions in animals treated with glutaraldehyde appeared primarily in the anterior third of the nose. Lesions were apparently more increased in mice compared to rats due to some level of “background” non-suppurative lesions in the rats. Mice were considered devoid of background lesions. In the 13-week study, female mice were the most sensitive, with lesions averaging a score of 2 (mild and clear, but of limited extent and/or severity). The lesions were characterized as neutrophilic infiltration primarily in the squamous epithelium of the vestibule, with thickening of the epithelium leading to loss of the characteristic surface grooves. Both cell size and number were reported to be increased. Lesions were generally found to increase in nature and severity with increased time and level of exposure. Obstruction of the nasal vestibule was thought to account for the mortality of animals in the higher dose groups. In female mice at 13 weeks, all glutaraldehyde dose groups showed the accumulation of eosinophilic proteinaceous deposits in the respiratory epithelium of the maxilloturbinate margin. Examination of unit length labeling indices as a measure of growth showed significant increases in all treated groups of female mice. No evidence of exposure related lesions was found in the respiratory tract in the trachea, carina, bronchi, or lungs.

Mean Subjective Pathology Scores for Nasal Lesions in Female Mice at 13 Weeks

	<i>Glutaraldehyde</i>	<i>Intraepithelial neutrophils</i>	<i>Subepithelial neutrophils</i>	<i>Squamous metaplasia</i>
0 ppb		0	0.4	0
62.5 ppb		2.0	2.0	0
125 ppb		2.4	2.8	0
250 ppb		3.2	3.2	0
500 ppb		2.8	2.8	0.5
1000 ppb*		--	--	--

*Animals exposed to 1000 ppb died early in the experiment.

Greenspan *et al.* (1985) exposed male and female F-344 rats to 0, 0.3, 1.1 and 3.1 ppm glutaraldehyde and 0, 0.2, 0.63, and 2.1 ppm glutaraldehyde, respectively, in a 9-day study, and both sexes to 0, 21, 49, and 194 ppb glutaraldehyde in a 14 week study. Animal numbers were not specified. Exposures were conducted for 6 hours per day, 5 days per week. In the 9-day study, observations in the high and intermediate dose level groups included reduced body weight gain, inflammation of the nasal and olfactory mucosa, and sensory irritation. In the two highest doses of the 14-week study, statistically significant differences in body weight gain were observed as well as perinasal wetness. No histopathological indication of inflammation in olfactory or nasal mucosa was observed.

Mice were exposed to 0, 0.3, 1.0, and 2.6 ppm glutaraldehyde vapors for 6 hours/day for 4, 9, or 14 days (Zissu *et al.*, 1994). These mice were killed immediately after the exposure period. Other groups exposed to 1.0 ppm for 14 days were killed after recovery periods of 1, 2, and 4 weeks. After 4 days of exposure to the lowest dose, mice showed lesions in the respiratory epithelium of the septum, and the naso- and maxilloturbinates. After exposure to 1.0 ppm glutaraldehyde, lesions were still judged as severe after 2 weeks of recovery.

A study comparing the effects of intra-nasally instilled glutaraldehyde and formaldehyde on rat nasal epithelium found inflammation, epithelial degeneration, respiratory epithelial hypertrophy, and squamous metaplasia in treated animals (St. Clair *et al.*, 1990). Acute inhalation exposure to formaldehyde produced identical lesions. Ten-fold higher concentrations of instilled formaldehyde were required to produce the same effect as instilled glutaraldehyde.

In a chronic study, NTP (1998, 1999) exposed groups of 50 male and 50 female F344/N rats to 0, 250, 500, or 750 ppb glutaraldehyde vapor by inhalation for 6 h/day, 5 days/week, for 104 weeks. Survival of 500 and 750 ppb female rats was less than that of the chamber controls. Mean body weights of all exposed groups of male rats and 500 and 750 ppb female rats were generally less than those of the chamber controls. Increased incidences of nonneoplastic nasal lesions occurred primarily within the anterior section of the nose in 500 and 750 ppb rats and to a lesser extent in 250 ppb rats. The more significant lesions included hyperplasia and inflammation of the squamous and respiratory epithelia and squamous metaplasia of the respiratory epithelium. Thus 250 ppb (1000 $\mu\text{g}/\text{m}^3$) is a chronic LOAEL for rats.

In the same study NTP (1998, 1999) exposed groups of 50 male and 50 female B6C3F1 mice to 0, 62.5, 125, or 250 ppb glutaraldehyde vapor by inhalation for 6 h/day, 5 days/week, for 104

weeks. Survival of exposed mice was similar to that of the chamber controls. Mean body weights of female mice exposed to 250 ppb were generally less than those of the controls. The incidence of inflammation of the nose was marginally increased in 250 ppb females. Incidences of squamous metaplasia of the respiratory epithelium were increased in 250 ppb males and females and 125 ppb females. Incidences of hyaline degeneration of the respiratory epithelium were increased in all exposed groups of females. Thus 62.5 ppb was a chronic LOAEL for female mice.

Incidence of Nasal Lesions in Female Mice exposed for 104 weeks

	<i>Glutaraldehyde</i>	<i>Inflammation</i>	<i>Respiratory epithelium hyaline degeneration</i>	<i>Respiratory epithelium squamous metaplasia</i>
	0 ppb	6/50	16/50	7/50
	62.5 ppb	7/49	35/49	11/49
	125 ppb	13/50	32/50	16/50
	250 ppb	14/50	30/50	21/50

VI. Derivation of Chronic Reference Exposure Level (REL)

<i>Study</i>	NTP 1998, 1999
<i>Study population</i>	Male and female F344 rats and B6C3F1 mice (50/sex/group)
<i>Exposure method</i>	Continuous inhalation exposure (0, 62.5, 125, and 250 ppb in mice; 0, 250, 500, or 750 ppb in rats)
<i>Critical effects</i>	Respiratory epithelium squamous metaplasia
<i>LOAEL</i>	62.5 ppb (female mice)
<i>NOAEL</i>	Not observed
<i>BMC₀₅</i>	20.5 ppb
<i>Exposure continuity</i>	6 hr/day, 5 days/week
<i>Exposure duration</i>	104 weeks
<i>Equivalent continuous exposure</i>	3.7 ppb (20.5 x 6/24 x 5/7)
<i>Human equivalent concentration</i>	0.62 ppb (gas with extrathoracic respiratory effects, RGDR = 0.17, BW = 28 g, MV = 0.032 L/min, SA = 3 cm ²)
<i>LOAEL uncertainty factor</i>	not needed in BMC approach
<i>Subchronic uncertainty factor</i>	1
<i>Interspecies uncertainty factor</i>	3
<i>Intraspecies uncertainty factor</i>	10
<i>Cumulative uncertainty factor</i>	30
<i>Inhalation reference exposure level</i>	0.02 ppb (0.08 µg/m ³)

Several studies indicate that the upper respiratory tract is a target for the toxicity of glutaraldehyde from inhalation exposure. Reports of toxicity to humans show that exposure can

lead to occupational asthma as well as cause irritation of the eyes and nose with accompanying rhinitis. Likewise, animals exposed to glutaraldehyde by the inhalation route show evidence of respiratory irritation with the induction of lesions of the anterior nasal cavities upon long-term exposure (Gross *et al.*, 1994; Greenspan *et al.*, 1985; NTP, 1998, 1999). The NTP (1998, 1999) study yielded a chronic LOAEL for female mice of 62.5 ppb. Gross *et al.* (1994) showed neutrophilic infiltration in the olfactory epithelium in the lowest dose exposure group. (Female mice exposed to 62.5 ppb also showed subepithelial neutrophilic infiltration.) This level was taken to be the subchronic LOAEL. This effect on the nasal epithelium was demonstrated to be both concentration- and exposure duration-dependent.

A benchmark concentration was determined using EPA's version 1.20 BMC software and the dose-response data on respiratory epithelium squamous metaplasia in female mice. The quantal-linear model gave an MLE₀₅ of 31.24 ppb, a BMC₀₅ of 20.51 ppb, and a p value of 0.9471. With the benchmark approach no LOAEL UF is needed. The study was a lifetime study so the subchronic UF is 1. An interspecies UF of 3 rather than 10 was used since an RGDR adjustment had been made. The default intraspecies UF of 10 was used so that the total UF was 30. The resulting chronic REL for glutaraldehyde is 0.02 ppb (0.08 µg/m³).

For comparison with the proposed REL, the study of Gross *et al.* (1994) used 62.5 ppb continuous exposure. Multiplying by the RGDR of 0.17 and dividing by a cumulative uncertainty factor of 300 (3 for a LOAEL, 3 for subchronic, 3 for interspecies, and 10 for intraspecies) results in a REL of 0.035 ppb (0.1 µg/m³).

VII. Data Strengths and Limitations for Development of the REL

The major strength of the inhalation REL for glutaraldehyde is the availability of controlled exposure inhalation studies in multiple species at multiple exposure concentrations and with adequate histopathological analysis. Major areas of uncertainty are the lack of human data, the lack of reproductive and developmental toxicity studies, the lack of dermal sensitization studies, and the lack of observation of a NOAEL.

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CHRONIC TOXICITY SUMMARY

HYDROGEN CHLORIDE*(Hydrochloric acid; anhydrous hydrogen chloride; muriatic acid)***CAS Registry Number: 7647-01-0****I. Chronic Reference Exposure Level**

<i>Inhalation reference exposure level</i>	9 µg/m³ (6 ppb)
<i>Critical effect(s)</i>	Hyperplasia of nasal mucosa, larynx, and trachea in rats
<i>Hazard index target(s)</i>	Respiratory system

II. Physical and Chemical Properties (HSDB, 1999)

<i>Description</i>	Colorless gas
<i>Molecular formula</i>	HCl
<i>Molecular weight</i>	36.46
<i>Density</i>	1.49 g/L @ 25° C
<i>Boiling point</i>	-84.9° C (HCl gas)
<i>Melting point</i>	-114.8° C (HCl gas)
<i>Solubility</i>	Soluble in water, alcohol, benzene, ether; insoluble in hydrocarbons
<i>Conversion factor</i>	1 ppm = 1.49 mg/m ³ at 25°C

III. Major Uses or Sources

Hydrogen chloride (HCl) is used in the manufacture of vinyl chloride, fertilizers, dyes, artificial silk, and pigments for paints. It is also used in electroplating, soap refining, and leather tanning. Other consumers of HCl include the photographic, textile and rubber industries (HSDB, 1999).

Hydrogen chloride is produced in large quantities during combustion of most materials and especially materials with a high chlorine content. Thus, HCl is a major product formed during the thermal decomposition of polyvinyl chloride, a commonly used plastic polymer (Burleigh-Flayer *et al.*, 1985). It is also released in large quantities during the test firing of some rocket and missile engines (Wohlslagel *et al.*, 1976). Since HCl is extremely hygroscopic, it generally exists as an aerosol in the ambient atmosphere. The annual statewide industrial emissions from facilities reporting under the Air Toxics Hot Spots Act in California based on the most recent inventory were estimated to be 2,570,888 pounds of HCl (CARB, 1999b).

IV. Effects of Human Exposure

Few reports are available on the effects of chronic HCl exposure on humans. Bleeding of the nose and gums and ulceration of the mucous membranes was observed following repeated occupational exposure to HCl mist at high but unquantified concentrations (Stokinger, 1981).

In another report, workers exposed to various mineral acids, including HCl, exhibited etching and erosion of the front teeth (Ten Bruggen Cate, 1968). Dental erosion was noted in 176 of 555 (32%) workers examined between 1962 and 1964, and progressive erosion was reported in 66 of 324 (20%) workers examined repeatedly. Rates of active erosion were highest (50%) in the most highly-exposed category (battery formation workers), intermediate (23%) in an intermediate-exposure category (picklers), and low (7%) in a low-exposure category (other processes). Grade 1 erosion (enamel loss) was noted in workers exposed for greater than 3 months; grade 2 erosion (loss of enamel and dentine) was noted after 2.5 to 5 years exposure; and grade 3 (loss of enamel and dentine with exposure of secondary dentine) was noted after six or more years of exposure.

V. Effects of Animal Exposure

Male Sprague-Dawley rats were exposed to 10 ppm HCl for 6 hours per day, 5 days per week over their lifetime (Sellakumar *et al.*, 1985). No differences in body weights or survival were observed between 99 exposed and 99 control animals. Increased incidences of hyperplasia of the nasal mucosa (62/99 vs. 51/99), larynx (22/99 vs. 2/99), and trachea (26/99 vs. 2/99) were observed in exposed rats compared to air-exposed controls.

A 90-day inhalation study using B6C3F1 mice and Sprague-Dawley and Fisher 344 rats exposed the animals (groups of 31 males and 31 females for each species and strain) to 10, 20, or 50 ppm HCl for 6 hours per day, 5 days per week over 90 days (Toxigenics, 1984). Several animals died during the study, though the deaths were not considered to be exposure related. A slight but significant decrease in body weight gain was reported in male and female mice and in male Fischer 344 rats in the high-exposure groups. No effect were noted in hematology, clinical chemistry, or urinalysis. Minimal or mild rhinitis was observed in both strains of rats. Concentration- and time-related lesions were noted in the anterior portion of the nasal cavity of exposed rats. Cheilitis, eosinophilic globules in the nasal epithelium and accumulation of macrophages in the peripheral tissues were observed in mice of all exposed groups. This study thus observed a LOAEL for both mice and rats of 10 ppm. The U.S. EPA considered this study supportive of the portal-of-entry effects observed at 10 ppm in the lifetime rat study (USEPA, 1999). Female rats (8-15/group) exposed to 302 ppm HCl for 1 hour either 12 days prior to mating or on day 9 of gestation exhibited severe dyspnea and cyanosis; the exposure was lethal to one-third of the exposed animals (Pavlova, 1976). Fetal mortality was significantly higher in rats exposed during pregnancy. Organ functional abnormalities observed in offspring exposed at 2-3 months of age were reported to be similar to those observed in the exposed dams.

Female rats were exposed to 302 ppm HCl for 1 hour prior to mating (GEOMET Technologies, 1981). Exposure killed 20 to 30% of the rats. In rats surviving 6 days after exposure, a decrease in blood oxygen saturation was reported, as were kidney, liver, and spleen effects. Estrus cycles

were also altered. In rats mated 12-16 days postexposure and killed on day 21 of pregnancy, a decrease in fetal weight, an increase in relative fetal lung weights, and reduced numbers of live fetuses were observed.

Derivation of Chronic Reference Exposure Level

<i>Study</i>	Sellakumar <i>et al.</i> , 1985
<i>Study population</i>	Sprague-Dawley rats (100 males)
<i>Exposure method</i>	Discontinuous whole-body inhalation (0 or 10 ppm)
<i>Critical effects</i>	Hyperplasia of the nasal mucosa, larynx and trachea
<i>LOAEL</i>	10 ppm
<i>NOAEL</i>	Not identified
<i>Exposure continuity</i>	6 hours per day, 5 days per week
<i>Average experimental exposure</i>	1.8 ppm for LOAEL group
<i>Human equivalent concentration</i>	0.57 ppm (gas with extrathoracic respiratory effects, RGDR = 0.32, based on rat MV _a = 0.33 L/min, MV _h = 13.8 L/min, SA _a (ET) = 15 cm ² ; Sa _h = 200 cm ³) (U.S. EPA, 1994)
<i>Exposure duration</i>	Lifetime
<i>LOAEL uncertainty factor</i>	3 (<30% incidence; mild effect)
<i>Subchronic uncertainty factor</i>	1
<i>Interspecies uncertainty factor</i>	3
<i>Intraspecies uncertainty factor</i>	10
<i>Cumulative uncertainty factor</i>	100
<i>Reference Concentration (RfC)</i>	0.006 ppm (6 ppb; 0.009 mg/m ³ ; 9 µg/m ³)

Both extrathoracic and tracheobronchial effects have been associated with exposures to hydrogen chloride. The REL was based on extrathoracic effects as humans are predicted to be relatively more susceptible to the effects of hydrogen chloride in that region. An intermediate LOAEL factor was used as the effects were both mild and occurring at a low incidence at the dose tested.

VII. Data Strengths and Limitations for Development of the REL

The USEPA based its RfC of 7 µg/m³ on the same study. U.S. EPA evaluated this RfC as a having a low level of confidence because of (1) the use of only one dose; (2) limited toxicity evaluation; (3) the lack of reproductive toxicity data; and (4) the lack of chronic exposure studies (U.S. EPA, 1994). OEHHA agrees with this assessment. The database for chronic exposure to this common chemical is limited.

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ACRYLAMIDE

CAS No: 79-06-1

I. PHYSICAL AND CHEMICAL PROPERTIES (From HSDB, 1994)

Molecular weight	71.08
Boiling point	125°C at 25 mm Hg
Melting point	84.5
Vapor pressure	0.007 mm Hg at 25°C
Air concentration conversion	1 ppm = 2.91 mg/m ³

II. HEALTH ASSESSMENT VALUES

Unit Risk Factor: 1.3 E-3 (µg/m³)⁻¹

Slope Factor: 4.5 E+0 (mg/kg-day)⁻¹

[Calculated by US EPA/IRIS (1988, 1993) from female Fischer 344 rat tumor data (central nervous system, mammary and thyroid glands, uterus, oral cavity) (Johnson *et al.*, 1986) using a linearized multistage procedure, extra risk; adopted by CDHS/RCHAS (1990).]

III. CARCINOGENIC EFFECTS

Human Studies

US EPA (1993) reviewed a study of cancer mortality in workers exposed to acrylamide by Collins (1984). Data from a long duration exposure group (10 individuals) and a short duration/intermittent exposure group (52 individuals) was analyzed using a standardized proportional mortality ratio (SPMR) procedure. No excess mortality for all types of cancer combined was noted in either group. Mortality from lung and central nervous system cancer appeared to be slightly elevated. However, the SPMRs were not significantly different from expected values, due to small group size. US EPA (1993) also noted additional study limitations including underrepresentation of the potential at-risk worker population, incomplete cause of death ascertainment, and incomplete exposure data.

Sobel *et al.* (1986) studied the mortality experience of 371 workers (365 white males, 6 white females) employed in acrylamide monomer production and polymerization operations at the Michigan Division of the Dow Chemical Company from 1955 through 1979. Vital status followup was performed from the date of the first potential exposure to December 31, 1982. Mortality comparisons were made between the cohort and United States white male mortality rates; comparisons were made with a subcohort of workers previously exposed to organic dyes both included and excluded. Slight excesses of mortality from all cancers (11 observed/7.9 expected), digestive tract cancer (4 observed/1.9 expected) and respiratory tract cancer (4 observed/2.9 expected) were observed in the total cohort; these excesses were not observed when the organic dye exposure subcohort was excluded. The authors concluded that the study did not support a relationship between acrylamide exposure and general or specific cancer mortality.

However, US EPA (1988) considers this study insufficient to assess the carcinogenicity of acrylamide in humans because of small cohort size, multiple chemical exposures, limited followup, and short exposure duration (167 cohort members had < 1 year of employment; 109 had 1-4 years of employment).

Animal Studies

Bull *et al.* (1984a) exposed female Sencar mice and male and female A/J mice to acrylamide. Female Sencar mice (40/treatment group) were exposed to 0, 12.5, 25.0 or 50.0 mg/kg body weight acrylamide by gavage, intraperitoneal injection or dermal application. Doses were administered 6 times over a 2 week period; total doses were 0, 75, 150 and 300 mg/kg. Acrylamide was dissolved in distilled water for gavage and intraperitoneal injection administration, and in ethanol for dermal application. Two weeks after the cessation of acrylamide exposure, 1.0 µg 12-*O*-tetradecanoyl-phenol-13-acetate (TPA) dissolved in 0.2 ml acetone was applied to the shaved back of each animal 3 times/week for 20 weeks. A promotion control group was included which received 300 mg/kg acrylamide followed by dermal applications of 0.2 ml acetone on the same treatment schedule and duration as the animals receiving TPA. All animals were sacrificed at 52 weeks, and were evaluated for the presence of skin tumors. Male and female A/J mouse (40/sex/treatment group) acrylamide exposures were conducted at laboratories of the US EPA (Cincinnati, OH) and the Medical College of Ohio (Toledo, OH) (MCO). Animals exposed at US EPA received acrylamide dissolved in distilled water by gavage 3 times/week for 8 weeks at doses of 0, 6.25, 12.5 or 25 mg/kg. Animals exposed at MCO initially received acrylamide by intraperitoneal injection 3 times/week for 8 weeks at doses of 0, 1, 3, 10, 30 or 60 mg/kg; however, peripheral neuropathy and decreased survival forced treatment termination on the 60 mg/kg group after the 11th injection. An untreated control group was also included. Animals were sacrificed after either 7 months (US EPA) or 6 months (MCO) and examined for lung adenomas. Acrylamide induced skin tumors (squamous cell papillomas and carcinomas) in TPA-promoted female Sencar mice in a dose-dependent manner when administered by gavage, intraperitoneal injection or dermal application. Acrylamide did not induce skin tumors by any route of administration in animals not receiving TPA. Tumor incidence data from female Sencar mice exposed to acrylamide are listed in Table 1.

The incidence of lung adenomas in both male and female A/J mice exposed to acrylamide by either gavage or intraperitoneal injection was significantly increased in a dose-related manner (Bull *et al.*, 1984a). Tumor incidence data for animals treated by intraperitoneal injection is listed in Table 2; numerical tumor incidence data for animals exposed to acrylamide by gavage was not listed.

Acrylamide dissolved in water was administered by gavage (0, 75, 150 or 200 mg/kg body weight, divided into 6 equal portions) to female ICR-Swiss mice (40 animals/treatment group) over a 2 week period (Bull *et al.*, 1984b). Two weeks after the last acrylamide exposure, the animals were exposed 3 times/week to dermal applications of 2.5 µg TPA for 20 weeks. Another group of 20 animals were exposed to a total dose of 300 mg/kg acrylamide, but received dermal applications of acetone alone. All animals were sacrificed after 52 weeks. Acrylamide caused a significant dose-related increase in the incidence of skin tumors (papillomas and

carcinomas combined). The incidence in animals also receiving TPA was 0/35, 4/34, 4/32 and 13/32 (number of animals with tumors/number of animals examined) for the control, low, mid and high dose groups, respectively; the skin tumor incidence in animals receiving 300 mg/kg acrylamide but not TPA was 10/36. Acrylamide-treated animals also demonstrated a significant dose-related increase in the incidence of lung tumors (alveolar and bronchiolar adenomas and carcinomas). The incidence in animals also receiving TPA was 4/36, 8/34, 6/36 and 11/34 for the control, low, mid and high dose groups, respectively; the lung tumor incidence in animals receiving 300 mg/kg acrylamide but not TPA was 14/36.

Table 1. Skin tumor (squamous cell papillomas and carcinomas) incidence in female Sencar mice exposed to acrylamide (Bull *et al.*, 1984a)

Total administered dose ¹ (mg/kg body weight)	Route of administration	TPA ²	Tumor incidence
0	gavage	+	2/40
75		+	12/40
100		+	23/40
300		+	30/40
300		-	0/20
0	intraperitoneal injection	+	0/40
75		+	10/40
100		+	13/40
300		+	21/40
300		-	0/20
0	dermal	+	7/40
75		+	4/40
100		+	11/40
300		+	18/40
300		-	0/20

1. The exposure duration was less than lifetime (2 weeks); the total administered dose listed was not adjusted to reflect a less-than-lifetime exposure.
2. TPA = 12-*O*-tetradecanoyl-phenol-13-acetate

Table 2. Lung adenoma incidence in male and female A/J mice exposed to acrylamide by intraperitoneal injection (Bull *et al.*, 1984a)

Dose level ¹ (mg/kg body weight)	Percent of animals with tumors	
	males	females
0	13	8
1	50	35
3	38	53
10	59	79
30	93	93

1. The exposure duration was less than lifetime (8 weeks); the dose level listed was not adjusted to reflect a less-than-lifetime exposure.

Robinson *et al.* (1986) exposed female SENCAR, BALB/c, A/J and ICR-Swiss mice (60 mice/strain/treatment group) to a single 50 mg/kg body weight dose of acrylamide by intraperitoneal injection; 2 days later 40 of the 60 mice in each treatment group received 1.0 µg (SENCAR), 2.5 µg (A/J and ICR-Swiss) or 5.0 µg (BALB/c) TPA in 0.2 ml acetone applied dermally 3 times/week for 20 weeks. The remaining 20 mice/strain/treatment group received acetone alone for the same treatment schedule and duration. All animals were sacrificed at 40 weeks, and were only examined for the number of skin papillomas and lung adenomas/animal. Acrylamide induced a significant increase in the number of skin papillomas and lung adenomas per animal in SENCAR mice receiving TPA treatment. The total number of animals bearing tumors was not listed. No significant increase in either tumor type was noted in the other mouse strains tested; tumor data for the animals receiving acrylamide but not TPA was not reported.

Male and female Fischer 344 rats (90/sex/treatment group) were exposed to acrylamide in drinking water for 2 years (Johnson *et al.*, 1986). Acrylamide water concentrations were adjusted to provide dosages of 0, 0.01, 0.1, 0.5 or 2 mg/kg body weight/day. Interim sacrifices (10 animals/sex/treatment group) were performed at 6, 12 and 18 months. A maximum tolerated dose (MTD) was achieved based on decreased weight gain, increased mortality during the last 4 months of the study and the appearance of several toxic effects (including peripheral nerve degeneration) in the 2 mg/kg/day group. Increases in the incidences of a number of tumor types were observed in the 2.0 mg/kg/day exposure group animals. An increased incidence of thyroid gland-follicular epithelium tumors was observed in both males and females. In females, increased tumor incidences were noted in the mammary glands, central nervous system, oral tissues, uterus and clitoral gland. An increased incidence of scrotal mesothelioma was noted in males, in both the 2.0 and 0.5 mg/kg/day exposure group; additionally, although not statistically significant, the incidence of scrotal mesothelioma in the 0.1 mg/kg/day group was greater than either the control group or historical control incidences. Male rats in the 2.0 mg/kg/day exposure group also had a significant increase in adrenal pheochromocytomas, and an increased incidence of central nervous system tumors when compared to historical controls but not when compared to concurrent controls. Tumor incidence data is listed in Table 3.

Table 3. Acrylamide-induced tumor incidences in male and female Fischer 344 rats (Johnson *et al.*, 1986)

Administered dose (mg/kg/day)	Human equivalent dose ¹ (mg/kg/day)	Tumor type	Tumor incidence	
			males	females
0	0	combined central nervous system (CNS), mammary gland, oral cavity, thyroid gland, uterus ²	NA	13/60
0.01	0.001		NA	18/60
0.1	0.015		NA	14/60
0.5	0.076		NA	21/60
2.0	0.305		NA	46/60
0	0		adrenal pheochromacytomas ³	3/60
0.01	0.001	7/60		NA
0.1	0.015	7/60		NA
0.5	0.076	5/60		NA
2.0	0.305	10/60		NA
0	0	central nervous system ⁴		5/60
0.01	0.001		2/60	2/60
0.1	0.015		0/60	1/60
0.5	0.076		3/60	1/60
2.0	0.305		8/60	9/60
0	0		oral cavity ⁵	6/60
0.01	0.001	7/60		3/60
0.1	0.015	1/60		2/60
0.5	0.076	5/60		3/60
2.0	0.305	6/60		8/60
0	0	mammary gland ⁶		NA
0.01	0.001		NA	2/60
0.1	0.015		NA	1/60
0.5	0.076		NA	5/58
2.0	0.305		NA	8/61
0	0		scrotal mesothelioma	3/60
0.01	0.001	0/60		NA
0.1	0.015	7/60		NA
0.5	0.076	11/60		NA
2.0	0.305	10/60		NA
0	0	thyroid ⁷		1/60
0.01	0.001		0/58	0/59
0.1	0.015		2/59	1/59
0.5	0.076		1/59	1/58
2.0	0.305		7/59	5/60
0	0		uterine adenocarcinomas	NA
0.01	0.001	NA		2/60
0.1	0.015	NA		1/60
0.5	0.076	NA		0/59
2.0	0.305	NA		5/60

Table 3 (continued). Acrylamide-induced tumor incidences in male and female Fischer 344 rats (Johnson *et al.*, 1986)

- 1, 2. As calculated by US EPA (1988).
 3. Benign and malignant.
 4. Tumors of glial origin or glial proliferation suggestive of early tumor.
 5. Squamous cell papillomas and carcinomas.
 6. Adenomas and adenocarcinomas.
 7. Males: follicular adenomas; females: follicular adenomas and adenocarcinomas.
- NA not available

IV. DERIVATION OF CANCER POTENCY

Basis for Cancer Potency

The studies by Bull *et al.* (1984a, 1984b), Robinson *et al.* (1986) and Johnson *et al.* (1986) indicate that acrylamide is capable of acting as both an initiator and a complete carcinogen in animals. However, only the Johnson *et al.* (1986) study contained a data set suitable for generating a cancer potency factor. Female Sencar mice developing tumors after exposure to acrylamide in the study by Bull *et al.* (1984a) were also additionally exposed to TPA; animals not exposed to TPA did not develop skin tumors. Female A/J mice exposed in that study to acrylamide by either gavage or intraperitoneal injection developed an increased incidence of lung adenomas without requiring TPA exposure. However, the animals were not evaluated for tumor types other than lung adenomas, and numerical tumor incidence data for animals exposed to acrylamide by gavage was not listed. Also, the exposure and observation durations for animals exposed by gavage (8 weeks and 7 months, respectively) and by intraperitoneal injection (8 weeks and 6 months, respectively) were short. Female ICR-Swiss mice exposed to acrylamide by gavage in the study by Bull *et al.* (1984b) were generally also exposed to TPA; only one exposure group was included which received acrylamide (300 mg/kg) but not TPA. Additionally, the exposure duration was only 2 weeks and the exposure duration was less than lifetime (52 weeks). In the study by Robinson *et al.* (1986), all animals for which tumor incidence data was reported were exposed to TPA as well as acrylamide. Animals in the Johnson *et al.* (1986) study were exposed to acrylamide alone for the lifetime of the animals, and were comprehensively examined for tumors. For these reasons, tumor incidence data from the Johnson *et al.* (1986) study was used to derive a cancer potency factor for acrylamide.

Methodology

As recommended in the US EPA Guidelines for Carcinogen Risk Assessment (1986), US EPA (1988) pooled tumor incidence data from different tumor sites, under the consideration that risk numbers derived from site-specific tumor incidence data potentially may not be predictive of, and may in fact underestimate, “whole-body” risks that are determined using the pooled individual animal data. The dose-response curves for each sex based on the pooled tumor incidence (benign and malignant) constituted the data sets of choice for risk assessment. Tumors at a particular site were added into the pool only when the tumor site had statistically significantly increased incidence at least at the high dose level (treated vs. control). The female

rat was considered to be the more sensitive sex, as there were significantly increased tumor incidences at a greater number of sites than in the males; the female rat tumor data was therefore used as the basis of a risk estimate. A linearized multistage procedure (GLOBAL 83) was used to calculate a cancer potency factor (q_1^*) from the female rat tumor incidence data. Surface area scaling was employed to transform animal cancer potency factors to human cancer potency factors, using the relationship ($q_{\text{human}} = q_{\text{animal}} * (bw_h / bw_a)^{1/3}$), where q_{human} is the human potency, q_{animal} is the animal potency, and bw_h and bw_a are the human and animal body weights, respectively. Body weight values used for humans and rats were 70 kg and 0.2 kg, respectively. No exposure route adjustment was made to the risk estimates because data exists which indicates that the pharmacokinetics and tissue distribution of acrylamide were not significantly affected by the dose administered or the route of administration (Dearfield *et al.*, 1988). US EPA calculated a cancer potency value (q_{human}) of $4.5 \text{ E}+0 \text{ (mg/kg-day)}^{-1}$. A unit risk factor was then calculated from the cancer potency factor by OEHHA/ATES using a reference human body weight of 70 kg and an inspiration rate of $20 \text{ m}^3/\text{day}$. The unit risk should not be used if the air concentration exceeds $8 \mu\text{g}/\text{m}^3$, as above this concentration the unit risk may not be appropriate.

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CHRONIC TOXICITY SUMMARY

HYDROGEN CHLORIDE*(Hydrochloric acid; anhydrous hydrogen chloride; muriatic acid)***CAS Registry Number: 7647-01-0****I. Chronic Reference Exposure Level**

<i>Inhalation reference exposure level</i>	9 µg/m³ (6 ppb)
<i>Critical effect(s)</i>	Hyperplasia of nasal mucosa, larynx, and trachea in rats
<i>Hazard index target(s)</i>	Respiratory system

II. Physical and Chemical Properties (HSDB, 1999)

<i>Description</i>	Colorless gas
<i>Molecular formula</i>	HCl
<i>Molecular weight</i>	36.46
<i>Density</i>	1.49 g/L @ 25° C
<i>Boiling point</i>	-84.9° C (HCl gas)
<i>Melting point</i>	-114.8° C (HCl gas)
<i>Solubility</i>	Soluble in water, alcohol, benzene, ether; insoluble in hydrocarbons
<i>Conversion factor</i>	1 ppm = 1.49 mg/m ³ at 25°C

III. Major Uses or Sources

Hydrogen chloride (HCl) is used in the manufacture of vinyl chloride, fertilizers, dyes, artificial silk, and pigments for paints. It is also used in electroplating, soap refining, and leather tanning. Other consumers of HCl include the photographic, textile and rubber industries (HSDB, 1999).

Hydrogen chloride is produced in large quantities during combustion of most materials and especially materials with a high chlorine content. Thus, HCl is a major product formed during the thermal decomposition of polyvinyl chloride, a commonly used plastic polymer (Burleigh-Flayer *et al.*, 1985). It is also released in large quantities during the test firing of some rocket and missile engines (Wohlslagel *et al.*, 1976). Since HCl is extremely hygroscopic, it generally exists as an aerosol in the ambient atmosphere. The annual statewide industrial emissions from facilities reporting under the Air Toxics Hot Spots Act in California based on the most recent inventory were estimated to be 2,570,888 pounds of HCl (CARB, 1999b).

IV. Effects of Human Exposure

Few reports are available on the effects of chronic HCl exposure on humans. Bleeding of the nose and gums and ulceration of the mucous membranes was observed following repeated occupational exposure to HCl mist at high but unquantified concentrations (Stokinger, 1981).

In another report, workers exposed to various mineral acids, including HCl, exhibited etching and erosion of the front teeth (Ten Bruggen Cate, 1968). Dental erosion was noted in 176 of 555 (32%) workers examined between 1962 and 1964, and progressive erosion was reported in 66 of 324 (20%) workers examined repeatedly. Rates of active erosion were highest (50%) in the most highly-exposed category (battery formation workers), intermediate (23%) in an intermediate-exposure category (picklers), and low (7%) in a low-exposure category (other processes). Grade 1 erosion (enamel loss) was noted in workers exposed for greater than 3 months; grade 2 erosion (loss of enamel and dentine) was noted after 2.5 to 5 years exposure; and grade 3 (loss of enamel and dentine with exposure of secondary dentine) was noted after six or more years of exposure.

V. Effects of Animal Exposure

Male Sprague-Dawley rats were exposed to 10 ppm HCl for 6 hours per day, 5 days per week over their lifetime (Sellakumar *et al.*, 1985). No differences in body weights or survival were observed between 99 exposed and 99 control animals. Increased incidences of hyperplasia of the nasal mucosa (62/99 vs. 51/99), larynx (22/99 vs. 2/99), and trachea (26/99 vs. 2/99) were observed in exposed rats compared to air-exposed controls.

A 90-day inhalation study using B6C3F1 mice and Sprague-Dawley and Fisher 344 rats exposed the animals (groups of 31 males and 31 females for each species and strain) to 10, 20, or 50 ppm HCl for 6 hours per day, 5 days per week over 90 days (Toxigenics, 1984). Several animals died during the study, though the deaths were not considered to be exposure related. A slight but significant decrease in body weight gain was reported in male and female mice and in male Fischer 344 rats in the high-exposure groups. No effect were noted in hematology, clinical chemistry, or urinalysis. Minimal or mild rhinitis was observed in both strains of rats. Concentration- and time-related lesions were noted in the anterior portion of the nasal cavity of exposed rats. Cheilitis, eosinophilic globules in the nasal epithelium and accumulation of macrophages in the peripheral tissues were observed in mice of all exposed groups. This study thus observed a LOAEL for both mice and rats of 10 ppm. The U.S. EPA considered this study supportive of the portal-of-entry effects observed at 10 ppm in the lifetime rat study (USEPA, 1999). Female rats (8-15/group) exposed to 302 ppm HCl for 1 hour either 12 days prior to mating or on day 9 of gestation exhibited severe dyspnea and cyanosis; the exposure was lethal to one-third of the exposed animals (Pavlova, 1976). Fetal mortality was significantly higher in rats exposed during pregnancy. Organ functional abnormalities observed in offspring exposed at 2-3 months of age were reported to be similar to those observed in the exposed dams.

Female rats were exposed to 302 ppm HCl for 1 hour prior to mating (GEOMET Technologies, 1981). Exposure killed 20 to 30% of the rats. In rats surviving 6 days after exposure, a decrease in blood oxygen saturation was reported, as were kidney, liver, and spleen effects. Estrus cycles

were also altered. In rats mated 12-16 days postexposure and killed on day 21 of pregnancy, a decrease in fetal weight, an increase in relative fetal lung weights, and reduced numbers of live fetuses were observed.

Derivation of Chronic Reference Exposure Level

<i>Study</i>	Sellakumar <i>et al.</i> , 1985
<i>Study population</i>	Sprague-Dawley rats (100 males)
<i>Exposure method</i>	Discontinuous whole-body inhalation (0 or 10 ppm)
<i>Critical effects</i>	Hyperplasia of the nasal mucosa, larynx and trachea
<i>LOAEL</i>	10 ppm
<i>NOAEL</i>	Not identified
<i>Exposure continuity</i>	6 hours per day, 5 days per week
<i>Average experimental exposure</i>	1.8 ppm for LOAEL group
<i>Human equivalent concentration</i>	0.57 ppm (gas with extrathoracic respiratory effects, RGDR = 0.32, based on rat MV _a = 0.33 L/min, MV _h = 13.8 L/min, SA _a (ET) = 15 cm ² ; Sa _h = 200 cm ³) (U.S. EPA, 1994)
<i>Exposure duration</i>	Lifetime
<i>LOAEL uncertainty factor</i>	3 (<30% incidence; mild effect)
<i>Subchronic uncertainty factor</i>	1
<i>Interspecies uncertainty factor</i>	3
<i>Intraspecies uncertainty factor</i>	10
<i>Cumulative uncertainty factor</i>	100
<i>Reference Concentration (RfC)</i>	0.006 ppm (6 ppb; 0.009 mg/m ³ ; 9 µg/m ³)

Both extrathoracic and tracheobronchial effects have been associated with exposures to hydrogen chloride. The REL was based on extrathoracic effects as humans are predicted to be relatively more susceptible to the effects of hydrogen chloride in that region. An intermediate LOAEL factor was used as the effects were both mild and occurring at a low incidence at the dose tested.

VII. Data Strengths and Limitations for Development of the REL

The USEPA based its RfC of 7 µg/m³ on the same study. U.S. EPA evaluated this RfC as a having a low level of confidence because of (1) the use of only one dose; (2) limited toxicity evaluation; (3) the lack of reproductive toxicity data; and (4) the lack of chronic exposure studies (U.S. EPA, 1994). OEHHA agrees with this assessment. The database for chronic exposure to this common chemical is limited.

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Appendix H
Risk Characterization

Cancer Risk for PMI at Grid Receptor # 248 by Chemical
 Located along Russell Boulevard, north of campus near the intersection of S. Campus Way

Chemical No.	Chemical	Inhalation	Dermal	Soil	Mother's Milk	Home grown Vegetables	Oral	Total	Percent of Total
1	Diesel engine exhaust, particulate matter (Diesel PM)	1.44E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.44E-06	66.6667%
2	Formaldehyde	5.81E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.81E-08	2.6898%
3	Benzo[a]pyrene	1.26E-10	1.67E-09	2.50E-10	0.00E+00	6.11E-09	8.03E-09	8.15E-09	0.3773%
4	Dibenz[a,h]anthracene	8.52E-11	3.68E-10	5.52E-11	0.00E+00	1.35E-09	1.77E-09	1.86E-09	0.0861%
5	Carbon tetrachloride	2.78E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.78E-08	1.2870%
6	Benz[a]anthracene	5.25E-11	6.98E-10	1.05E-10	0.00E+00	2.56E-09	3.36E-09	3.41E-09	0.1579%
7	Methanol	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
8	Isopropyl alcohol	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
9	Chloroform	7.47E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.47E-08	3.4583%
10	Dimethyl formamide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
11	Benzene	2.16E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.16E-08	1.0000%
12	Methyl chloroform [1,1,1-TCA]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
13	Ethyl chloride [Chloroethane]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
14	Vinyl chloride	2.31E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.31E-09	0.1069%
15	Acetaldehyde	3.89E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.89E-10	0.0180%
16	Methylene chloride [Dichloromethane]	2.25E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.25E-08	1.0417%
17	Carbon disulfide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
18	1,1-Dichloroethane	2.47E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.47E-11	0.0011%
19	Vinylidene chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
20	1,2-Dichloropropane	2.39E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.39E-11	0.0011%
21	Methyl ethyl ketone [2-Butanone]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
22	Trichloroethylene	1.96E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.96E-10	0.0091%
23	Acrylamide	7.99E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.99E-08	3.6991%
24	1,1,2,2-Tetrachloroethane	6.95E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.95E-10	0.0322%
25	Naphthalene	2.33E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.33E-09	0.1079%
26	Ethyl benzene	8.10E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.10E-11	0.0038%
27	p-Dichlorobenzene	2.30E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.30E-11	0.0011%
28	Ethylene dibromide [EDB]	8.76E-13	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.76E-13	0.0000%
29	1,3-Butadiene	2.41E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.41E-08	1.1157%
30	Acrolein	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
31	Ethylene dichloride [EDC]	7.58E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.58E-09	0.3509%
32	Acrylonitrile	8.16E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.16E-09	0.3778%
33	Toluene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
34	Chlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
35	Hexane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
36	Glutaraldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
37	Propylene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
38	Triethylamine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
39	1,4-Dioxane	5.37E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.37E-09	0.2486%
40	Perchloroethylene [Tetrachloroethene]	2.47E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.47E-09	0.1144%
41	Indeno[1,2,3-cd]pyrene	1.18E-11	1.56E-10	2.34E-11	0.00E+00	5.72E-10	7.52E-10	7.64E-10	0.0354%
42	Benzo[b]fluoranthene	2.98E-11	3.97E-10	5.95E-11	0.00E+00	1.45E-09	1.91E-09	1.94E-09	0.0898%
43	Benzo[k]fluoranthene	4.03E-11	5.36E-10	8.02E-11	0.00E+00	1.96E-09	2.58E-09	2.62E-09	0.1213%
44	Chrysene	6.79E-12	9.04E-11	1.35E-11	0.00E+00	3.31E-10	4.34E-10	4.41E-10	0.0204%
45	Hydrazine	4.28E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.28E-08	1.9815%
46	Xylenes (mixed)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
47	2,3,7,8-Tetrachlorodibenzo-p-dioxin	7.38E-10	3.10E-09	1.30E-09	3.48E-09	6.06E-10	8.49E-09	9.23E-09	0.4273%
48	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin	1.50E-12	6.32E-12	2.65E-12	7.10E-12	1.24E-12	1.73E-11	1.88E-11	0.0009%
49	Lead	2.00E-11	1.28E-12	4.20E-11	0.00E+00	8.65E-11	1.30E-10	1.50E-10	0.0069%
50	Mercury	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
51	Hydrochloric acid	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
52	Hydrogen fluoride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
53	Nitric acid	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
54	Hydrogen sulfide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
55	Phosphine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
56	Chromium, hexavalent (& compounds)	6.66E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.66E-09	0.3083%
57	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin	1.00E-09	4.21E-09	1.76E-09	4.73E-09	8.22E-10	1.15E-08	1.25E-08	0.5787%
58	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin	2.60E-10	1.09E-09	4.58E-10	1.23E-09	2.14E-10	3.00E-09	3.26E-09	0.1509%
59	1,2,3,4,6,7,8,9-Octachlorodibenzofuran	1.65E-12	6.93E-12	2.90E-12	7.78E-12	1.35E-12	1.90E-11	2.06E-11	0.0010%
60	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin	6.18E-10	2.60E-09	1.09E-09	2.92E-09	5.08E-10	7.11E-09	7.73E-09	0.3579%
61	1,2,3,7,8-Pentachlorodibenzo-p-dioxin	3.97E-09	1.67E-08	6.99E-09	1.87E-08	3.26E-09	4.57E-08	4.96E-08	2.2963%
62	2,3,7,8-Tetrachlorodibenzofuran	1.01E-09	4.24E-09	1.78E-09	4.77E-09	8.29E-10	1.16E-08	1.26E-08	0.5833%
63	1,2,3,4,7,8,9-Heptachlorodibenzofuran	7.00E-11	2.95E-10	1.23E-10	3.31E-10	5.75E-11	8.06E-10	8.76E-10	0.0406%
64	2,3,4,7,8-Pentachlorodibenzofuran	9.72E-09	4.09E-08	1.71E-08	4.59E-08	7.99E-09	1.12E-07	1.22E-07	5.6481%
65	1,2,3,7,8-Pentachlorodibenzofuran	5.48E-10	2.30E-09	9.65E-10	2.59E-09	4.50E-10	6.31E-09	6.85E-09	0.3171%
66	1,2,3,6,7,8-Hexachlorodibenzofuran	1.12E-09	4.73E-09	1.98E-09	5.31E-09	9.24E-10	1.29E-08	1.41E-08	0.6528%
67	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin	1.45E-09	6.12E-09	2.56E-09	6.87E-09	1.19E-09	1.67E-08	1.82E-08	0.8426%
68	2,3,4,6,7,8-Hexachlorodibenzofuran	2.35E-11	9.89E-11	4.14E-11	1.11E-10	1.93E-11	2.71E-10	2.94E-10	0.0136%
69	1,2,3,4,6,7,8-Heptachlorodibenzofuran	8.98E-10	3.78E-09	1.58E-09	4.24E-09	7.38E-10	1.03E-08	1.12E-08	0.5185%
70	1,2,3,4,7,8-Hexachlorodibenzofuran	2.94E-09	1.24E-08	5.19E-09	1.39E-08	2.42E-09	3.39E-08	3.68E-08	1.7037%
71	1,2,3,7,8,9-Hexachlorodibenzofuran	1.52E-11	6.38E-11	2.67E-11	7.16E-11	1.25E-11	1.74E-10	1.90E-10	0.0088%
SUM		1.86E-06	1.07E-07	4.36E-08	1.15E-07	3.45E-08	3.00E-07	2.16E-06	100.0000%

Cancer Risk for the PMI at Grid Receptor # 248 by Source
 Located along Russell Boulevard, north of campus near the intersection of S. Campus Way

Source No.	Source Identification	Inhalation	Dermal	Soil	Mother's Milk	Home grown Vegetables	Oral	Total	Percent of Total
201	P-108-01 Primate TB North # 5	2.20E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.20E-10	0.0102%
202	P-109-01 Primate TB South # 6	2.33E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.33E-10	0.0108%
203	P-99-94(a) Quad Parking	3.42E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.42E-09	0.1583%
204	P-93-94(a) Rec Hall	3.03E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.03E-08	1.4028%
205	P-111-95(a) Schl of Med Neurosci	1.44E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.44E-09	0.0667%
206	P-123-01 Schl of Med Neurosci	1.64E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.64E-09	0.0759%
207	P-15-04 Science Lab	6.56E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.56E-09	0.3037%
208	P-74-05 Segundo Dinning	2.21E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.21E-07	10.2315%
209	P-126-95(a) Social Sci	7.72E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.72E-09	0.3574%
210	P-17-02 South Parking	6.55E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.55E-09	0.3032%
211	P-92-94(a) Storm Lift # 4	2.38E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.38E-08	1.1019%
212	C-09-129 Storm Lift #4 (new)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
213	P-71-00 Targeted genomics	4.26E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.26E-09	0.1972%
214	P-111-01 Tele Comm.	1.39E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.39E-08	0.6435%
215	P-91-94(a) Thurman Lab	2.40E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.40E-08	1.1111%
216	P-100-94(a) Toxic Pollutant	1.20E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.20E-08	0.5556%
217	P-17-09 TURF	1.42E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.42E-11	0.0007%
218	P-121-03 Tupper Load Dock	1.06E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.06E-08	0.4907%
219	P-209-95(a) Util Well 6A	1.17E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.17E-08	0.5417%
220	P-07-01 Vega Crops	5.96E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.96E-09	0.2759%
221	P-63-03 Vet Lab	2.80E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.80E-09	0.1296%
222	P-52-07 Vet Med 3A	7.33E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.33E-09	0.3394%
223	P-53-07 Vet Med 3A	5.37E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.37E-09	0.2486%
224	P-59-05 Watershed Sic	3.98E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.98E-09	0.1843%
225	P-38-05 West Entry Park	1.32E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.32E-08	0.6111%
226	P-96-94(a) WEPT Influent	9.14E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.14E-08	4.2315%
227	P-88-99 WEPT South	1.14E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.14E-08	0.5278%
228	Landfill	1.90E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.90E-09	0.0880%
229	Landfill	2.16E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.16E-09	0.1000%
230	Landfill	2.73E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.73E-09	0.1264%
231	Landfill	3.33E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.33E-09	0.1542%
232	Waste Water Treatment Plant	4.06E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.06E-09	0.1880%
233	Grounds Above-ground Storage Tank	8.46E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.46E-11	0.0039%
234	Fleet Services Underground Storage Tank	4.05E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.05E-09	0.1875%
235	Primate Center Gasoline AST	4.42E-13	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.42E-13	0.0000%
236	Agricultural Services AST	1.45E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.45E-10	0.0067%
237	Plant Pathology Storage Tank	8.87E-13	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.87E-13	0.0000%
238	Pomology Above Ground Storage Tank	6.20E-13	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.20E-13	0.0000%
239	Airport Above Ground Storage Tank	4.84E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.84E-10	0.0224%
SUM		1.86E-06	1.07E-07	4.36E-08	1.15E-07	3.45E-08	3.00E-07	2.16E-06	100.0000%

Chronic HI for PMI at Grid Receptor # 257 by Chemical
 Located along Russell Boulevard, north of Russell Intramural Field

Chemical No.	Chemical	Cardiovascular system	Central Nervous System	Bone	Develop-mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
53	Nitric acid	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
54	Hydrogen sulfide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.47E-05	0.00E+00	0.00E+00	1.47E-05	0.1567%
55	Phosphine	0.00E+00	4.03E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.03E-07	0.00E+00	4.03E-07	0.00E+00	4.03E-07	0.00E+00	4.03E-07	4.03E-07	0.0043%
56	Chromium, hexavalent (& compounds)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.65E-07	0.00E+00	2.87E-08	3.65E-07	0.0039%
57	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	7.47E-06	7.47E-06	0.00E+00	7.47E-06	0.00E+00	0.00E+00	7.47E-06	7.47E-06	0.00E+00	7.47E-06	7.47E-06	0.0796%
58	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	1.94E-06	1.94E-06	0.00E+00	1.94E-06	0.00E+00	0.00E+00	1.94E-06	1.94E-06	0.00E+00	1.94E-06	1.94E-06	0.0207%
59	1,2,3,4,6,7,8,9-Octachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	1.23E-08	1.23E-08	0.00E+00	1.23E-08	0.00E+00	0.00E+00	1.23E-08	1.23E-08	0.00E+00	1.23E-08	1.23E-08	0.0001%
60	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	4.61E-06	4.61E-06	0.00E+00	4.61E-06	0.00E+00	0.00E+00	4.61E-06	4.61E-06	0.00E+00	4.61E-06	4.61E-06	0.0491%
61	1,2,3,7,8-Pentachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	2.97E-05	2.97E-05	0.00E+00	2.97E-05	0.00E+00	0.00E+00	2.97E-05	2.97E-05	0.00E+00	2.97E-05	2.97E-05	0.3166%
62	2,3,7,8-Tetrachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	7.54E-06	7.54E-06	0.00E+00	7.54E-06	0.00E+00	0.00E+00	7.54E-06	7.54E-06	0.00E+00	7.54E-06	7.54E-06	0.0804%
63	1,2,3,4,7,8,9-Heptachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	5.23E-07	5.23E-07	0.00E+00	5.23E-07	0.00E+00	0.00E+00	5.23E-07	5.23E-07	0.00E+00	5.23E-07	5.23E-07	0.0056%
64	2,3,4,7,8-Pentachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	7.26E-05	7.26E-05	0.00E+00	7.26E-05	0.00E+00	0.00E+00	7.26E-05	7.26E-05	0.00E+00	7.26E-05	7.26E-05	0.7740%
65	1,2,3,7,8-Pentachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	4.09E-06	4.09E-06	0.00E+00	4.09E-06	0.00E+00	0.00E+00	4.09E-06	4.09E-06	0.00E+00	4.09E-06	4.09E-06	0.0436%
66	1,2,3,6,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	8.40E-06	8.40E-06	0.00E+00	8.40E-06	0.00E+00	0.00E+00	8.40E-06	8.40E-06	0.00E+00	8.40E-06	8.40E-06	0.0896%
67	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	1.09E-05	1.09E-05	0.00E+00	1.09E-05	0.00E+00	0.00E+00	1.09E-05	1.09E-05	0.00E+00	1.09E-05	1.09E-05	0.1162%
68	2,3,4,6,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	1.76E-07	1.76E-07	0.00E+00	1.76E-07	0.00E+00	0.00E+00	1.76E-07	1.76E-07	0.00E+00	1.76E-07	1.76E-07	0.0019%
69	1,2,3,4,6,7,8-Heptachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	6.71E-06	6.71E-06	0.00E+00	6.71E-06	0.00E+00	0.00E+00	6.71E-06	6.71E-06	0.00E+00	6.71E-06	6.71E-06	0.0715%
70	1,2,3,4,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	2.20E-05	2.20E-05	0.00E+00	2.20E-05	0.00E+00	0.00E+00	2.20E-05	2.20E-05	0.00E+00	2.20E-05	2.20E-05	0.2345%
71	1,2,3,7,8,9-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	1.13E-07	1.13E-07	0.00E+00	1.13E-07	0.00E+00	0.00E+00	1.13E-07	1.13E-07	0.00E+00	1.13E-07	1.13E-07	0.0012%
SUM		7.04E-05	1.74E-04	2.23E-05	3.64E-04	2.39E-04	1.82E-06	3.39E-04	0.00E+00	7.65E-05	1.91E-04	9.38E-03	0.00E+00	1.97E-04	9.38E-03	100.0000%

Acute HI for PMI at Grid Receptor # 26 by Chemical
 Northeast of the Primate Center along the property boundary

Chemical No.	Chemical	Cardiovascular system	Central Nervous System	Bone	Develop-mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
53	Nitric acid	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.72E-02	0.00E+00	0.00E+00	1.72E-02	0.0000%
54	Hydrogen sulfide	0.00E+00	2.12E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.12E-02	0.0000%
55	Phosphine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
56	Chromium, hexavalent (& compounds)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
57	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
58	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
59	1,2,3,4,6,7,8,9-Octachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
60	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
61	1,2,3,7,8-Pentachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
62	2,3,7,8-Tetrachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
63	1,2,3,4,7,8,9-Heptachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
64	2,3,4,7,8-Pentachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
65	1,2,3,7,8-Pentachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
66	1,2,3,6,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
67	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
68	2,3,4,6,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
69	1,2,3,4,6,7,8-Heptachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
70	1,2,3,4,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
71	1,2,3,7,8,9-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
SUM		0.00E+00	3.47E-02	0.00E+00	1.28E-02	0.00E+00	1.12E-01	4.85E-05	1.62E-04	0.00E+00	1.27E-02	2.69E-02	0.00E+00	1.62E-04	1.12E-01	100.0000%

Cancer Risk for MEIR at Grid Receptor # 2046 by Chemical
Northeast of the Primate Center on Larue Way

Chemical No.	Chemical	Inhalation	Dermal	Soil	Mother's Milk	Home grown Vegetables	Oral	Total	Percent of Total
1	Diesel engine exhaust, particulate matter (Diesel PM)	1.34E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.34E-06	66.3366%
2	Formaldehyde	5.57E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.57E-08	2.7574%
3	Benzo[a]pyrene	1.24E-10	1.64E-09	2.46E-10	0.00E+00	6.01E-09	7.90E-09	8.02E-09	0.3970%
4	Dibenz[a,h]anthracene	8.46E-11	3.66E-10	5.48E-11	0.00E+00	1.34E-09	1.76E-09	1.84E-09	0.0911%
5	Carbon tetrachloride	2.65E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.65E-08	1.3119%
6	Benz[a]anthracene	5.11E-11	6.79E-10	1.02E-10	0.00E+00	2.48E-09	3.27E-09	3.32E-09	0.1644%
7	Methanol	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
8	Isopropyl alcohol	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
9	Chloroform	7.10E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.10E-08	3.5149%
10	Dimethyl formamide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
11	Benzene	2.09E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.09E-08	1.0347%
12	Methyl chloroform [1,1,1-TCA]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
13	Ethyl chloride [Chloroethane]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
14	Vinyl chloride	2.45E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.45E-09	0.1213%
15	Acetaldehyde	3.87E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.87E-10	0.0192%
16	Methylene chloride [Dichloromethane]	2.13E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.13E-08	1.0545%
17	Carbon disulfide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
18	1,1-Dichloroethane	2.63E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.63E-11	0.0013%
19	Vinylidene chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
20	1,2-Dichloropropane	2.54E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.54E-11	0.0013%
21	Methyl ethyl ketone [2-Butanone]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
22	Trichloroethylene	1.92E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.92E-10	0.0095%
23	Acrylamide	7.60E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.60E-08	3.7624%
24	1,1,2,2-Tetrachloroethane	7.39E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.39E-10	0.0366%
25	Naphthalene	2.30E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.30E-09	0.1139%
26	Ethyl benzene	8.60E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.60E-11	0.0043%
27	p-Dichlorobenzene	2.45E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.45E-11	0.0012%
28	Ethylene dibromide [EDB]	9.32E-13	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.32E-13	0.0000%
29	1,3-Butadiene	2.28E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.28E-08	1.1287%
30	Acrolein	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
31	Ethylene dichloride [EDC]	7.27E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.27E-09	0.3599%
32	Acrylonitrile	8.47E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.47E-09	0.4193%
33	Toluene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
34	Chlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
35	Hexane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
36	Glutaraldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
37	Propylene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
38	Triethylamine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
39	1,4-Dioxane	5.10E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.10E-09	0.2525%
40	Perchloroethylene [Tetrachloroethene]	2.40E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.40E-09	0.1188%
41	Indeno[1,2,3-cd]pyrene	1.16E-11	1.54E-10	2.31E-11	0.00E+00	5.64E-10	7.41E-10	7.53E-10	0.0373%
42	Benzo[b]fluoranthene	2.91E-11	3.87E-10	5.80E-11	0.00E+00	1.42E-09	1.86E-09	1.89E-09	0.0936%
43	Benzo[k]fluoranthene	3.92E-11	5.21E-10	7.81E-11	0.00E+00	1.91E-09	2.51E-09	2.55E-09	0.1262%
44	Chrysene	6.58E-12	8.75E-11	1.31E-11	0.00E+00	3.20E-10	4.21E-10	4.27E-10	0.0211%
45	Hydrazine	4.06E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.06E-08	2.0099%
46	Xylenes (mixed)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
47	2,3,7,8-Tetrachlorodibenzo-p-dioxin	7.16E-10	3.01E-09	1.26E-09	3.38E-09	5.89E-10	8.25E-09	8.97E-09	0.4441%
48	1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin	1.46E-12	6.13E-12	2.57E-12	6.88E-12	1.20E-12	1.68E-11	1.82E-11	0.0009%
49	Lead	1.88E-11	1.20E-12	3.95E-11	0.00E+00	8.12E-11	1.22E-10	1.41E-10	0.0070%
50	Mercury	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
51	Hydrochloric acid	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
52	Hydrogen fluoride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
53	Nitric acid	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
54	Hydrogen sulfide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
55	Phosphine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
56	Chromium, hexavalent (& compounds)	6.26E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.26E-09	0.3099%
57	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin	9.69E-10	4.08E-09	1.71E-09	4.58E-09	7.97E-10	1.12E-08	1.21E-08	0.5990%
58	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin	2.52E-10	1.06E-09	4.44E-10	1.19E-09	2.07E-10	2.90E-09	3.16E-09	0.1564%
59	1,2,3,4,6,7,8,9-Octachlorodibenzofuran	1.60E-12	6.71E-12	2.81E-12	7.54E-12	1.31E-12	1.84E-11	2.00E-11	0.0010%
60	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin	5.98E-10	2.52E-09	1.05E-09	2.83E-09	4.92E-10	6.89E-09	7.49E-09	0.3708%
61	1,2,3,7,8-Pentachlorodibenzo-p-dioxin	3.84E-09	1.62E-08	6.77E-09	1.82E-08	3.16E-09	4.43E-08	4.81E-08	2.3812%
62	2,3,7,8-Tetrachlorodibenzofuran	9.77E-10	4.11E-09	1.72E-09	4.62E-09	8.03E-10	1.13E-08	1.22E-08	0.6040%
63	1,2,3,4,7,8,9-Heptachlorodibenzofuran	6.78E-11	2.85E-10	1.19E-10	3.20E-10	5.57E-11	7.81E-10	8.49E-10	0.0420%
64	2,3,4,7,8-Pentachlorodibenzofuran	9.41E-09	3.96E-08	1.66E-08	4.45E-08	7.74E-09	1.08E-07	1.18E-07	5.8416%
65	1,2,3,7,8-Pentachlorodibenzofuran	5.31E-10	2.23E-09	9.35E-10	2.51E-09	4.36E-10	6.11E-09	6.64E-09	0.3287%
66	1,2,3,6,7,8-Hexachlorodibenzofuran	1.09E-09	4.58E-09	1.92E-09	5.14E-09	8.95E-10	1.25E-08	1.36E-08	0.6733%
67	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin	1.41E-09	5.92E-09	2.48E-09	6.65E-09	1.16E-09	1.62E-08	1.76E-08	0.8713%
68	2,3,4,6,7,8-Hexachlorodibenzofuran	2.28E-11	9.58E-11	4.01E-11	1.08E-10	1.87E-11	2.62E-10	2.85E-10	0.0141%
69	1,2,3,4,6,7,8-Heptachlorodibenzofuran	8.70E-10	3.66E-09	1.53E-09	4.11E-09	7.15E-10	1.00E-08	1.09E-08	0.5396%
70	1,2,3,4,7,8-Hexachlorodibenzofuran	2.85E-09	1.20E-08	5.02E-09	1.35E-08	2.34E-09	3.28E-08	3.57E-08	1.7673%
71	1,2,3,7,8,9-Hexachlorodibenzofuran	1.47E-11	6.18E-11	2.59E-11	6.93E-11	1.21E-11	1.69E-10	1.84E-10	0.0091%
SUM		1.73E-06	1.03E-07	4.22E-08	1.12E-07	3.35E-08	2.91E-07	2.02E-06	100.0000%

Cancer Risk for the MEIR at Grid Receptor # 2046 by Source
 Northeast of the Primate Center on Larue Way

Source No.	Source Identification	Inhalation	Dermal	Soil	Mother's Milk	Home grown Vegetables	Oral	Total	Percent of Total
201	P-108-01 Primate TB North # 5	2.23E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.23E-10	0.0110%
202	P-109-01 Primate TB South # 6	2.36E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.36E-10	0.0117%
203	P-99-94(a) Quad Parking	3.51E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.51E-09	0.1738%
204	P-93-94(a) Rec Hall	2.71E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.71E-08	1.3416%
205	P-111-95(a) Schl of Med Neurosci	1.36E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.36E-09	0.0673%
206	P-123-01 Schl of Med Neurosci	1.81E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.81E-09	0.0896%
207	P-15-04 Science Lab	6.04E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.04E-09	0.2990%
208	P-74-05 Segundo Dining	1.81E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.81E-07	8.9604%
209	P-126-95(a) Social Sci	8.94E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.94E-09	0.4426%
210	P-17-02 South Parking	6.30E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.30E-09	0.3119%
211	P-92-94(a) Storm Lift # 4	2.30E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.30E-08	1.1386%
212	C-09-129 Storm Lift #4 (new)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
213	P-71-00 Targeted genomics	4.11E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.11E-09	0.2035%
214	P-111-01 Tele Comm.	1.35E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.35E-08	0.6683%
215	P-91-94(a) Thurman Lab	2.38E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.38E-08	1.1782%
216	P-100-94(a) Toxic Pollutant	1.17E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.17E-08	0.5792%
217	P-17-09 TURF	1.42E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.42E-11	0.0007%
218	P-121-03 Tupper Load Dock	1.03E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.03E-08	0.5099%
219	P-209-95(a) Util Well 6A	1.13E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.13E-08	0.5594%
220	P-07-01 Vega Crops	6.50E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.50E-09	0.3218%
221	P-63-03 Vet Lab	2.70E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.70E-09	0.1337%
222	P-52-07 Vet Med 3A	7.12E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.12E-09	0.3525%
223	P-53-07 Vet Med 3A	5.22E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.22E-09	0.2584%
224	P-59-05 Watershed Sic	3.80E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.80E-09	0.1881%
225	P-38-05 West Entry Park	1.30E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.30E-08	0.6436%
226	P-96-94(a) WEPT Influent	8.98E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.98E-08	4.4455%
227	P-88-99 WEPT South	1.11E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.11E-08	0.5495%
228	Landfill	1.96E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.96E-09	0.0970%
229	Landfill	2.33E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.33E-09	0.1153%
230	Landfill	2.95E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.95E-09	0.1460%
231	Landfill	3.51E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.51E-09	0.1738%
232	Waste Water Treatment Plant	3.91E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.91E-09	0.1936%
233	Grounds Above-ground Storage Tank	7.79E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.79E-11	0.0039%
234	Fleet Services Underground Storage Tank	4.10E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.10E-09	0.2030%
235	Primate Center Gasoline AST	4.62E-13	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.62E-13	0.0000%
236	Agricultural Services AST	1.50E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.50E-10	0.0074%
237	Plant Pathology Storage Tank	8.68E-13	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.68E-13	0.0000%
238	Pomology Above Ground Storage Tank	6.43E-13	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.43E-13	0.0000%
239	Airport Above Ground Storage Tank	5.03E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.03E-10	0.0249%
SUM		1.73E-06	1.03E-07	4.22E-08	1.12E-07	3.35E-08	2.91E-07	2.02E-06	100.0000%

Chronic HI for MEIR at Grid Receptor # 2051 by Chemical
 Northeast of the Primate Center on Larue Way

Chemical No.	Chemical	Cardiovascular system	Central Nervous System	Bone	Developmental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
53	Nitric acid	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
54	Hydrogen sulfide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.42E-05	0.00E+00	0.00E+00	1.42E-05	0.1638%
55	Phosphine	0.00E+00	3.80E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.80E-07	0.00E+00	3.80E-07	0.00E+00	3.80E-07	0.00E+00	3.80E-07	3.80E-07	0.0044%
56	Chromium, hexavalent (& compounds)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.34E-07	0.00E+00	2.63E-08	3.34E-07	0.0039%
57	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	7.23E-06	7.23E-06	0.00E+00	7.23E-06	0.00E+00	0.00E+00	7.23E-06	7.23E-06	0.00E+00	7.23E-06	7.23E-06	0.0834%
58	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	1.88E-06	1.88E-06	0.00E+00	1.88E-06	0.00E+00	0.00E+00	1.88E-06	1.88E-06	0.00E+00	1.88E-06	1.88E-06	0.0217%
59	1,2,3,4,6,7,8,9-Octachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	1.19E-08	1.19E-08	0.00E+00	1.19E-08	0.00E+00	0.00E+00	1.19E-08	1.19E-08	0.00E+00	1.19E-08	1.19E-08	0.0001%
60	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	4.46E-06	4.46E-06	0.00E+00	4.46E-06	0.00E+00	0.00E+00	4.46E-06	4.46E-06	0.00E+00	4.46E-06	4.46E-06	0.0514%
61	1,2,3,7,8-Pentachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	2.87E-05	2.87E-05	0.00E+00	2.87E-05	0.00E+00	0.00E+00	2.87E-05	2.87E-05	0.00E+00	2.87E-05	2.87E-05	0.3310%
62	2,3,7,8-Tetrachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	7.29E-06	7.29E-06	0.00E+00	7.29E-06	0.00E+00	0.00E+00	7.29E-06	7.29E-06	0.00E+00	7.29E-06	7.29E-06	0.0841%
63	1,2,3,4,7,8,9-Heptachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	5.06E-07	5.06E-07	0.00E+00	5.06E-07	0.00E+00	0.00E+00	5.06E-07	5.06E-07	0.00E+00	5.06E-07	5.06E-07	0.0058%
64	2,3,4,7,8-Pentachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	7.02E-05	7.02E-05	0.00E+00	7.02E-05	0.00E+00	0.00E+00	7.02E-05	7.02E-05	0.00E+00	7.02E-05	7.02E-05	0.8097%
65	1,2,3,7,8-Pentachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	3.96E-06	3.96E-06	0.00E+00	3.96E-06	0.00E+00	0.00E+00	3.96E-06	3.96E-06	0.00E+00	3.96E-06	3.96E-06	0.0457%
66	1,2,3,6,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	8.12E-06	8.12E-06	0.00E+00	8.12E-06	0.00E+00	0.00E+00	8.12E-06	8.12E-06	0.00E+00	8.12E-06	8.12E-06	0.0937%
67	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	1.05E-05	1.05E-05	0.00E+00	1.05E-05	0.00E+00	0.00E+00	1.05E-05	1.05E-05	0.00E+00	1.05E-05	1.05E-05	0.1211%
68	2,3,4,6,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	1.70E-07	1.70E-07	0.00E+00	1.70E-07	0.00E+00	0.00E+00	1.70E-07	1.70E-07	0.00E+00	1.70E-07	1.70E-07	0.0020%
69	1,2,3,4,6,7,8-Heptachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	6.49E-06	6.49E-06	0.00E+00	6.49E-06	0.00E+00	0.00E+00	6.49E-06	6.49E-06	0.00E+00	6.49E-06	6.49E-06	0.0749%
70	1,2,3,4,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	2.13E-05	2.13E-05	0.00E+00	2.13E-05	0.00E+00	0.00E+00	2.13E-05	2.13E-05	0.00E+00	2.13E-05	2.13E-05	0.2457%
71	1,2,3,7,8,9-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	1.09E-07	1.09E-07	0.00E+00	1.09E-07	0.00E+00	0.00E+00	1.09E-07	1.09E-07	0.00E+00	1.09E-07	1.09E-07	0.0013%
SUM		6.87E-05	1.66E-04	2.03E-05	3.47E-04	2.30E-04	1.72E-06	3.24E-04	0.00E+00	7.22E-05	1.86E-04	8.67E-03	0.00E+00	1.91E-04	8.67E-03	100.0000%

Chronic HI for the MEIR at Grid Receptor # 2051 by Source
Northeast of the Primate Center on Larue Way

Source No.	Source Identification	Cardiovascular system	Central Nervous System	Bone	Develop- mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
1	Central Heating and Cooling Plant Boiler #1	0.00E+00	1.17E-07	0.00E+00	1.17E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.01E-05	0.00E+00	1.17E-07	4.01E-05	0.4625%
2	Central Heating and Cooling Plant Boiler #2	0.00E+00	1.84E-07	0.00E+00	1.84E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.31E-05	0.00E+00	1.84E-07	6.31E-05	0.7278%
3	Central Heating and Cooling Plant Boiler #3	0.00E+00	2.98E-08	0.00E+00	2.98E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.02E-05	0.00E+00	2.98E-08	1.02E-05	0.1176%
4	Central Heating and Cooling Plant Boiler #4, Natural Gas	0.00E+00	1.30E-07	0.00E+00	1.30E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.46E-05	0.00E+00	1.30E-07	4.46E-05	0.5144%
5	Central Heating and Cooling Plant Boiler #4, Diesel	0.00E+00	6.61E-09	0.00E+00	6.41E-09	9.92E-11	0.00E+00	9.92E-11	0.00E+00	9.92E-11	0.00E+00	1.06E-05	0.00E+00	5.64E-09	1.06E-05	0.1223%
6	Primate Center Boiler No 1 Natural Gas	0.00E+00	2.33E-10	0.00E+00	2.33E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.00E-08	0.00E+00	2.33E-10	8.00E-08	0.0009%
7	Primate Center Boiler No 2 Natural Gas	0.00E+00	2.37E-10	0.00E+00	2.37E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.12E-08	0.00E+00	2.37E-10	8.12E-08	0.0009%
8	Primate Center Boiler No 2 Landfill Gas	5.04E-11	9.11E-08	2.21E-07	9.53E-07	8.62E-07	1.02E-11	8.62E-07	0.00E+00	9.12E-08	8.62E-07	9.86E-06	0.00E+00	8.62E-07	9.86E-06	0.1137%
9	Landfill Flare	2.11E-13	7.64E-11	1.82E-10	7.61E-11	1.26E-15	4.28E-14	1.26E-12	0.00E+00	7.72E-11	1.88E-14	7.51E-09	0.00E+00	2.59E-14	7.51E-09	0.0001%
10	Incinerator	0.00E+00	2.87E-09	0.00E+00	1.75E-04	1.75E-04	0.00E+00	1.75E-04	0.00E+00	0.00E+00	1.75E-04	2.24E-04	0.00E+00	1.75E-04	2.24E-04	2.5836%
11	ARS J-1 (H001)	0.00E+00	1.29E-08	0.00E+00	1.29E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.41E-06	0.00E+00	1.29E-08	4.41E-06	0.0509%
12	ARS J-1 CAAN 3840 - 4 boilers	0.00E+00	1.89E-08	0.00E+00	1.89E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.50E-06	0.00E+00	1.89E-08	6.50E-06	0.0750%
13	ARS K-2 Co-located 2 stacks	0.00E+00	2.41E-08	0.00E+00	2.41E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.27E-06	0.00E+00	2.41E-08	8.27E-06	0.0954%
14	ARS K-2 (H040)	0.00E+00	7.98E-09	0.00E+00	7.98E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.74E-06	0.00E+00	7.98E-09	2.74E-06	0.0316%
15	Contained Research	0.00E+00	7.24E-10	0.00E+00	7.24E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.48E-07	0.00E+00	7.24E-10	2.48E-07	0.0029%
16	Environmental Horticulture K-1	0.00E+00	7.09E-10	0.00E+00	7.09E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.43E-07	0.00E+00	7.09E-10	2.43E-07	0.0028%
17	Environmental Horticulture K-2	0.00E+00	9.93E-10	0.00E+00	9.93E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.40E-07	0.00E+00	9.93E-10	3.40E-07	0.0039%
18	Environmental Services Facility A	0.00E+00	4.30E-08	0.00E+00	4.30E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.48E-05	0.00E+00	4.30E-08	1.48E-05	0.1707%
19	Environmenatl Services Facility (3 per stack)	0.00E+00	2.53E-08	0.00E+00	2.53E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.66E-06	0.00E+00	2.53E-08	8.66E-06	0.0999%
20	Genome Launch Facility (plant reproduction)	0.00E+00	4.66E-09	0.00E+00	4.66E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.60E-06	0.00E+00	4.66E-09	1.60E-06	0.0185%
21	Equine Analytical Chemistry Lab	0.00E+00	2.91E-09	0.00E+00	2.91E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.96E-07	0.00E+00	2.91E-09	9.96E-07	0.0115%
22	Housing - Castillian DC	0.00E+00	7.77E-09	0.00E+00	7.77E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.66E-06	0.00E+00	7.77E-09	2.66E-06	0.0307%
23	Housing - Castillian DC	0.00E+00	2.58E-09	0.00E+00	2.58E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.83E-07	0.00E+00	2.58E-09	8.83E-07	0.0102%
24	Comparative Medicine (Primate Center)	0.00E+00	1.02E-12	0.00E+00	1.02E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.49E-10	0.00E+00	1.02E-12	3.49E-10	0.0000%
25	Contained Research	0.00E+00	9.79E-13	0.00E+00	9.79E-13	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.36E-10	0.00E+00	9.79E-13	3.36E-10	0.0000%
26	Institute of Ecology - West Campus	0.00E+00	2.66E-09	0.00E+00	2.66E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.12E-07	0.00E+00	2.66E-09	9.12E-07	0.0105%
27	ITEH Geriatrics - cage wash inside co-located 3 stacks	0.00E+00	5.03E-10	0.00E+00	5.03E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.73E-07	0.00E+00	5.03E-10	1.73E-07	0.0020%
28	ITEH Geriatrics Cagewash - outside - co-locate 3 stacks	0.00E+00	6.41E-09	0.00E+00	6.41E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.20E-06	0.00E+00	6.41E-09	2.20E-06	0.0254%
29	Mondavi Ctr for Performing Arts - 2 boilers	0.00E+00	3.52E-09	0.00E+00	3.52E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.21E-06	0.00E+00	3.52E-09	1.21E-06	0.0140%
30	Mondavi Ctr for Performing Arts	0.00E+00	2.07E-10	0.00E+00	2.07E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.10E-08	0.00E+00	2.07E-10	7.10E-08	0.0008%
31	Rec Pool	0.00E+00	3.75E-08	0.00E+00	3.75E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.29E-05	0.00E+00	3.75E-08	1.29E-05	0.1488%
32	Thoreau Hall - 2 stacks co-located	0.00E+00	3.61E-12	0.00E+00	3.61E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.24E-09	0.00E+00	3.61E-12	1.24E-09	0.0000%
33	Air Stripper	0.00E+00	0.00E+00	0.00E+00	2.78E-08	0.00E+00	0.00E+00	2.78E-08	0.00E+00	2.78E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.78E-08	0.0000%
34	In-well Stripper	0.00E+00	0.00E+00	0.00E+00	3.67E-07	0.00E+00	0.00E+00	3.67E-07	0.00E+00	3.67E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.67E-07	0.0000%
35	Ground Water Treatment	0.00E+00	0.00E+00	0.00E+00	4.85E-07	0.00E+00	0.00E+00	4.85E-07	0.00E+00	4.85E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.85E-07	0.0000%
36	Large Kiln	0.00E+00	1.54E-09	0.00E+00	1.45E-09	5.68E-12	0.00E+00	5.68E-12	0.00E+00	5.68E-12	0.00E+00	2.19E-07	0.00E+00	9.18E-09	2.19E-07	0.0025%
37	Raku Kiln	0.00E+00	7.18E-11	0.00E+00	6.73E-11	0.00E+00	2.64E-13	0.00E+00	2.64E-13	0.00E+00	0.00E+00	1.02E-08	0.00E+00	4.27E-10	1.02E-08	0.0001%
38	Foundry Kiln	0.00E+00	3.72E-11	0.00E+00	3.49E-11	1.37E-13	0.00E+00	1.37E-13	0.00E+00	1.37E-13	0.00E+00	5.26E-09	0.00E+00	2.21E-10	5.26E-09	0.0001%
39	Three Art Dept Kilns to roof vent	0.00E+00	3.18E-09	0.00E+00	2.97E-09	1.17E-11	0.00E+00	1.17E-11	0.00E+00	1.17E-11	0.00E+00	4.50E-07	0.00E+00	1.89E-08	4.50E-07	0.0052%
40	Storehouse/Bulk Receiving and Storage	0.00E+00	0.00E+00	0.00E+00	2.51E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.51E-07	0.0000%
41	Walnut Dryer	0.00E+00	2.82E-09	0.00E+00	2.64E-09	1.04E-11	0.00E+00	1.04E-11	0.00E+00	1.04E-11	0.00E+00	2.08E-07	0.00E+00	1.72E-09	2.08E-07	0.0024%
42	Temporary Building 187	1.53E-08	1.23E-07	5.23E-08	3.56E-07	1.65E-07	3.70E-09	3.97E-07	0.00E+00	1.53E-07	0.00E+00	2.99E-05	0.00E+00	8.51E-09	2.99E-05	0.3449%
43	Temporary Building 188	1.24E-08	9.88E-08	4.22E-08	2.87E-07	1.32E-07	2.98E-09	3.20E-07	0.00E+00	1.23E-07	0.00E+00	2.41E-05	0.00E+00	6.85E-09	2.41E-05	0.2780%
44	Veihmeyer	2.03E-08	4.91E-07	3.63E-06	8.89E-07	8.34E-08	1.84E-08	7.38E-07	0.00E+00	2.03E-07	0.00E+00	6.70E-05	0.00E+00	3.46E-08	6.70E-05	0.7728%
45	Enology	1.39E-08	1.11E-07	4.72E-08	3.22E-07	1.49E-07	3.35E-09	3.59E-07	0.00E+00	1.38E-07	0.00E+00	2.71E-05	0.00E+00	7.70E-09	2.71E-05	0.3126%
46	Wickson Hall	3.55E-07	2.84E-06	1.21E-06	8.26E-06	3.81E-06	8.58E-08	9.20E-06	0.00E+00	3.54E-06	0.00E+00	6.93E-04	0.00E+00	1.97E-07	6.93E-04	7.9931%
47	Hoagland	1.67E-07	1.34E-06	5.69E-07	3.88E-06	1.79E-06	4.04E-08	4.32E-06	0.00E+00	1.66E-06	0.00E+00	3.26E-04	0.00E+00	9.27E-08	3.26E-04	3.7601%
48	Mann Hall	3.62E-08	2.89E-07	1.23E-07	8.39E-07	3.87E-07	8.72E-09	9.34E-07	0.00E+00	3.59E-07	0.00E+00	7.05E-05	0.00E+00	2.01E-08	7.05E-05	0.8131%
49	Storer Hall	7.75E-09	6.19E-08	2.64E-08	1.80E-07	8.31E-08	1.87E-09	2.00E-07	0.00E+00	7.71E-08	0.00E+00	1.51E-05	0.00E+00	4.30E-09	1.51E-05	0.1742%
50	Hutchison Hall/Biological Sci Unit 2	1.55E-07	1.24E-06	5.29E-07	3.60E-06	1.67E-06	3.74E-08	4.02E-06	0.00E+00	1.54E-06	0.00E+00	3.02E-04	0.00E+00	8.59E-08	3.02E-04	3.4833%
51	Asmundson Hall	9.42E-08	7.53E-07	3.21E-07	2.19E-06	1.01E-06	2.27E-08	2.44E-06	0.00E+00	9.36E-07	0.00E+00	1.84E-04	0.00E+00	5.22E-08	1.84E-04	2.1223%
52	Robbins Hall	9.28E-08	7.41E-07	3.17E-07	2.15E-06	9.95E-07	2.24E-08	2.40E-06	0.00E+00	9.22E-07	0.00E+00	1.81E-04	0.00E+00	5.15E-08	1.81E-04	2.0877%
53	Temporary Building 202	5.09E-09	4.07E-08	1.74E-08	1.18E-07	5.46E-08	1.23E-09	1.32E-07	0.00E+00	5.07E-08	0.00E+00	9.93E-06	0.00E+00	2.83E-09	9.93E-06	0.1145%
54	Briggs Hall and Life Sciences	4.53E-07	3.62E-06	1.55E-06	1.09E-07	4.85E-06	1.17E-05	1.17E-05	0.00E+00	4.51E-06	0.00E+00	8.82E-04	0.00E+00	2.51E-07	8.82E-04	10.1730%
55	Temporary Building 194	5.49E-09	4.39E-08	1.87E-08	1.27E-07	5.88E-08	1.32E-09	1.42E-07	0.00E+00	5.46E-08	0.00E+00	1.07E-05	0.00E+00	3.04E-09	1.07E-05	0.1234%
56	Food Science	5.48E-10	4.38E-09	1.87E-09	1.27E-08	5.88E-09	1.32E-10	1.42E-08	0.00E+00	5.45E-09	0.00E+00	1.07E-06	0.00E+00	3.04E-10	1.07E-06	0.0123%
57																

Chronic HI for the MEIR at Grid Receptor # 2051 by Source
 Northeast of the Primate Center on Larue Way

Source No.	Source Identification	Cardiovascular system	Central Nervous System	Bone	Develop- mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
181	P-59-07 Multi use stadium	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.15E-07	0.00E+00	0.00E+00	7.15E-07	0.0082%
182	No Permit Neurosci - off campus	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.08E-06	0.00E+00	0.00E+00	1.08E-06	0.0125%
183	P-16-09 New UG RES (Cat)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.54E-06	0.00E+00	0.00E+00	7.54E-06	0.0870%
184	No Permit Old Fire Station	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.30E-06	0.00E+00	0.00E+00	5.30E-06	0.0611%
185	P-29-96(a) Physical Plant	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.08E-05	0.00E+00	0.00E+00	1.08E-05	0.1246%
186	P-120-01 Plant Envir Sci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.08E-05	0.00E+00	0.00E+00	3.08E-05	0.3552%
187	P-50-99(a) Port Gen # 1	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.23E-05	0.00E+00	0.00E+00	2.23E-05	0.2572%
188	No Permit Port Gen # 14	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.04E-05	0.00E+00	0.00E+00	6.04E-05	0.6967%
189	P-51-99(a) Port Gen # 2	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.16E-06	0.00E+00	0.00E+00	5.16E-06	0.0595%
190	P-52-99(a) Port Gen # 3	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.98E-07	0.00E+00	0.00E+00	3.98E-07	0.0046%
191	P-86-01 Port Gen # 7	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.78E-06	0.00E+00	0.00E+00	1.78E-06	0.0205%
192	P-87-01 Port Gen # 8	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.32E-06	0.00E+00	0.00E+00	1.32E-06	0.0152%
193	P-49-07 Pri Animal Hous # 1	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.29E-07	0.00E+00	0.00E+00	1.29E-07	0.0015%
194	P-31-98 Primate Animal	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.61E-07	0.00E+00	0.00E+00	1.61E-07	0.0019%
195	P-32-98 Primate CCM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.29E-07	0.00E+00	0.00E+00	2.29E-07	0.0026%
196	P-69-96(a) Primate Freezers	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.96E-08	0.00E+00	0.00E+00	6.96E-08	0.0008%
197	P-102-94(a) Primate Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.18E-07	0.00E+00	0.00E+00	6.18E-07	0.0071%
198	P-15-98 Primate Quarantine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.87E-07	0.00E+00	0.00E+00	2.87E-07	0.0033%
199	No Permit Primate Sew Life Sta	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.28E-08	0.00E+00	0.00E+00	8.28E-08	0.0010%
200	P-16-98 Primate TB 184	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.24E-07	0.00E+00	0.00E+00	3.24E-07	0.0037%
201	P-108-01 Primate TB North # 5	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.17E-07	0.00E+00	0.00E+00	1.17E-07	0.0013%
202	P-109-01 Primate TB South # 6	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.23E-07	0.00E+00	0.00E+00	1.23E-07	0.0014%
203	P-99-94(a) Quad Parking	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.95E-05	0.00E+00	0.00E+00	1.95E-05	0.2249%
204	P-93-94(a) Rec Hall	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.05E-06	0.00E+00	0.00E+00	4.05E-06	0.0467%
205	P-111-95(a) Schl of Med Neurosci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.81E-06	0.00E+00	0.00E+00	2.81E-06	0.0324%
206	P-123-01 Schl of Med Neurosci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.28E-06	0.00E+00	0.00E+00	3.28E-06	0.0378%
207	P-15-04 Science Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.30E-06	0.00E+00	0.00E+00	3.30E-06	0.0381%
208	P-74-05 Segundo Dining	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.90E-05	0.00E+00	0.00E+00	2.90E-05	0.3345%
209	P-126-95(a) Social Sci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.23E-05	0.00E+00	0.00E+00	2.23E-05	0.2572%
210	P-17-02 South Parking	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.72E-06	0.00E+00	0.00E+00	4.72E-06	0.0544%
211	P-92-94(a) Storm Lift # 4	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.33E-05	0.00E+00	0.00E+00	1.33E-05	0.1534%
212	C-09-129 Storm Lift #4 (new)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
213	P-71-00 Targeted genomics	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.88E-06	0.00E+00	0.00E+00	1.88E-06	0.0217%
214	P-111-01 Tele Comm.	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.38E-06	0.00E+00	0.00E+00	8.38E-06	0.0967%
215	P-91-94(a) Thurman Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.17E-05	0.00E+00	0.00E+00	1.17E-05	0.1349%
216	P-100-94(a) Toxic Pollutant	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.35E-06	0.00E+00	0.00E+00	6.35E-06	0.0732%
217	P-17-09 TURF	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.66E-09	0.00E+00	0.00E+00	7.66E-09	0.0001%
218	P-121-03 Tupper Load Dock	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.40E-06	0.00E+00	0.00E+00	4.40E-06	0.0507%
219	P-209-95(a) Util Well 6A	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.81E-06	0.00E+00	0.00E+00	2.81E-06	0.0324%
220	P-07-01 Vega Crops	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.56E-06	0.00E+00	0.00E+00	2.56E-06	0.0295%
221	P-63-03 Vet Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.28E-06	0.00E+00	0.00E+00	1.28E-06	0.0148%
222	P-52-07 Vet Med 3A	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.38E-06	0.00E+00	0.00E+00	3.38E-06	0.0390%
223	P-53-07 Vet Med 3A	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.52E-06	0.00E+00	0.00E+00	2.52E-06	0.0291%
224	P-59-05 Watershed Sic	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.03E-06	0.00E+00	0.00E+00	3.03E-06	0.0349%
225	P-38-05 West Entry Park	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.10E-06	0.00E+00	0.00E+00	6.10E-06	0.0704%
226	P-96-94(a) WEPT Influent	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.69E-05	0.00E+00	0.00E+00	4.69E-05	0.5409%
227	P-88-99 WEPT South	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.83E-06	0.00E+00	0.00E+00	5.83E-06	0.0672%
228	Landfill	2.97E-08	4.12E-07	0.00E+00	3.62E-07	2.39E-09	6.03E-09	1.80E-07	0.00E+00	3.94E-07	3.11E-09	1.97E-06	0.00E+00	2.43E-08	1.97E-06	0.0227%
229	Landfill	3.11E-08	4.31E-07	0.00E+00	3.80E-07	2.50E-09	6.32E-09	1.88E-07	0.00E+00	4.13E-07	3.26E-09	2.07E-06	0.00E+00	2.55E-08	2.07E-06	0.0239%
230	Landfill	3.52E-08	4.89E-07	0.00E+00	4.31E-07	2.84E-09	7.16E-09	2.13E-07	0.00E+00	4.68E-07	3.69E-09	2.34E-06	0.00E+00	2.89E-08	2.34E-06	0.0270%
231	Landfill	4.35E-08	6.04E-07	0.00E+00	5.32E-07	3.50E-09	8.84E-09	2.64E-07	0.00E+00	5.77E-07	4.56E-09	2.89E-06	0.00E+00	3.57E-08	2.89E-06	0.0333%
232	Waste Water Treatment Plant	1.13E-06	5.08E-05	0.00E+00	3.34E-05	2.12E-10	0.00E+00	9.60E-06	0.00E+00	9.60E-06	0.00E+00	5.81E-05	0.00E+00	8.85E-09	5.81E-05	0.6701%
233	Grounds Above-ground Storage Tank	0.00E+00	5.86E-08	0.00E+00	5.69E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.95E-09	0.00E+00	4.86E-08	5.86E-08	0.0001%
234	Fleet Services Underground Storage Tank	0.00E+00	2.14E-06	0.00E+00	2.07E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.26E-07	0.00E+00	1.77E-06	2.14E-06	0.0038%
235	Primate Center Gasoline AST	0.00E+00	2.63E-10	0.00E+00	2.55E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.02E-11	0.00E+00	2.18E-10	2.63E	

Acute HI for MEIR at Grid Receptor # 1886 by Chemical
 Northeast of the Primate Center on Larue Way

Chemical No.	Chemical	Cardiovascular system	Central Nervous System	Bone	Develop- mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
1	Diesel engine exhaust, particulate matter (Diesel PM)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
2	Formaldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.82E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.82E-02	90.6915%
3	Benzo[a]pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
4	Dibenz[a,h]anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
5	Carbon tetrachloride	0.00E+00	5.45E-05	0.00E+00	5.45E-05	0.00E+00	0.00E+00	5.45E-05	0.00E+00	0.00E+00	5.45E-05	0.00E+00	0.00E+00	0.00E+00	5.45E-05	0.0000%
6	Benz[a]anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
7	Methanol	0.00E+00	6.03E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.03E-04	0.0000%
8	Isopropyl alcohol	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.82E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.82E-03	0.00E+00	0.00E+00	1.82E-03	2.4202%
9	Chloroform	0.00E+00	1.37E-02	0.00E+00	1.37E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.37E-02	0.00E+00	0.00E+00	0.00E+00	1.37E-02	0.0000%
10	Dimethyl formamide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
11	Benzene	0.00E+00	0.00E+00	0.00E+00	1.60E-04	0.00E+00	0.00E+00	0.00E+00	1.60E-04	0.00E+00	1.60E-04	0.00E+00	0.00E+00	1.60E-04	1.60E-04	0.0000%
12	Methyl chloroform [1,1,1-TCA]	0.00E+00	8.12E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.12E-07	0.0000%
13	Ethyl chloride [Chloroethane]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
14	Vinyl chloride	0.00E+00	1.46E-06	0.00E+00	0.00E+00	0.00E+00	1.46E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.46E-06	0.00E+00	0.00E+00	1.46E-06	0.0019%
15	Acetaldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.39E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.39E-05	0.00E+00	0.00E+00	7.39E-05	0.0983%
16	Methylene chloride [Dichloromethane]	0.00E+00	2.48E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.48E-04	0.0000%
17	Carbon disulfide	0.00E+00	4.09E-06	0.00E+00	4.09E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.09E-06	0.00E+00	0.00E+00	0.00E+00	4.09E-06	0.0000%
18	1,1-Dichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
19	Vinylidene chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
20	1,2-Dichloropropane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
21	Methyl ethyl ketone [2-Butanone]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.38E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.38E-05	0.00E+00	0.00E+00	2.38E-05	0.0316%
22	Trichloroethylene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
23	Acrylamide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
24	1,1,2,2-Tetrachloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
25	Naphthalene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
26	Ethyl benzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
27	p-Dichlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
28	Ethylene dibromide [EDB]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
29	1,3-Butadiene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
30	Acrolein	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.32E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.32E-03	0.00E+00	0.00E+00	2.32E-03	3.0851%
31	Ethylene dichloride [EDC]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
32	Acrylonitrile	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
33	Toluene	0.00E+00	9.16E-05	0.00E+00	9.16E-05	0.00E+00	9.16E-05	0.00E+00	0.00E+00	0.00E+00	9.16E-05	9.16E-05	0.00E+00	0.00E+00	9.16E-05	0.1218%
34	Chlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
35	Hexane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
36	Glutaraldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
37	Propylene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
38	Triethylamine	0.00E+00	1.28E-05	0.00E+00	0.00E+00	0.00E+00	1.28E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.28E-05	0.0170%
39	1,4-Dioxane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.10E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.10E-05	0.00E+00	0.00E+00	3.10E-05	0.0412%
40	Perchloroethylene [Tetrachloroethene]	0.00E+00	1.94E-05	0.00E+00	0.00E+00	0.00E+00	1.94E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.94E-05	0.00E+00	0.00E+00	1.94E-05	0.0258%
41	Indeno[1,2,3-cd]pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
42	Benzo[b]fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
43	Benzo[k]fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
44	Chrysene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
45	Hydrazine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
46	Xylenes (mixed)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.83E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.83E-05	0.00E+00	0.00E+00	9.83E-05	0.1307%
47	2,3,7,8-Tetrachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
48	1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
49	Lead	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
50	Mercury	0.00E+00	7.07E-05	0.00E+00	7.07E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.07E-05	0.0000%
51	Hydrochloric acid	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.86E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.86E-03	0.00E+00	0.00E+00	1.86E-03	2.4734%
52	Hydrogen fluoride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.04E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.04E-04	0.00E+00	0.00E+00	6.04E-04	0.8032%

Acute HI for MEIR at Grid Receptor # 1886 by Chemical
Northeast of the Primate Center on Larue Way

Chemical No.	Chemical	Cardiovascular system	Central Nervous System	Bone	Developmental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
53	Nitric acid	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.95E-02	0.00E+00	0.00E+00	1.95E-02	0.0000%
54	Hydrogen sulfide	0.00E+00	1.67E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.67E-02	0.0000%
55	Phosphine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
56	Chromium, hexavalent (& compounds)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
57	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
58	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
59	1,2,3,4,6,7,8,9-Octachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
60	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
61	1,2,3,7,8-Pentachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
62	2,3,7,8-Tetrachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
63	1,2,3,4,7,8,9-Heptachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
64	2,3,4,7,8-Pentachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
65	1,2,3,7,8-Pentachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
66	1,2,3,6,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
67	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
68	2,3,4,6,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
69	1,2,3,4,6,7,8-Heptachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
70	1,2,3,4,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
71	1,2,3,7,8,9-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
SUM		0.00E+00	3.16E-02	0.00E+00	1.41E-02	0.00E+00	7.52E-02	5.45E-05	1.60E-04	0.00E+00	1.41E-02	2.64E-02	0.00E+00	1.60E-04	7.52E-02	100.0000%

Acute HI for the MEIR at Grid Receptor # 1886 by Source
Northeast of the Primate Center on Larue Way

Source No.	Source Identification	Cardiovascular system	Central Nervous System	Bone	Develop-mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
1	Central Heating and Cooling Plant Boiler #1	0.00E+00	0.00E+00	0.00E+00	3.86E-07	0.00E+00	4.69E-04	0.00E+00	3.86E-07	0.00E+00	3.86E-07	2.20E-06	0.00E+00	3.86E-07	4.69E-04	0.6237%
2	Central Heating and Cooling Plant Boiler #2	0.00E+00	0.00E+00	0.00E+00	3.93E-07	0.00E+00	4.78E-04	0.00E+00	3.93E-07	0.00E+00	3.93E-07	2.24E-06	0.00E+00	3.93E-07	4.78E-04	0.6356%
3	Central Heating and Cooling Plant Boiler #3	0.00E+00	0.00E+00	0.00E+00	4.35E-07	0.00E+00	5.30E-04	0.00E+00	4.35E-07	0.00E+00	4.35E-07	2.47E-06	0.00E+00	4.35E-07	5.30E-04	0.7048%
4	Central Heating and Cooling Plant Boiler #4, Natural Gas	0.00E+00	0.00E+00	0.00E+00	6.83E-07	0.00E+00	8.31E-04	0.00E+00	6.83E-07	0.00E+00	6.83E-07	3.90E-06	0.00E+00	6.83E-07	8.31E-04	1.1051%
5	Central Heating and Cooling Plant Boiler #4, Diesel	0.00E+00	6.31E-08	0.00E+00	3.10E-06	0.00E+00	9.88E-03	0.00E+00	3.04E-06	0.00E+00	3.10E-06	1.69E-07	0.00E+00	3.04E-06	9.88E-03	13.1383%
6	Primate Center Boiler No 1 Natural Gas	0.00E+00	0.00E+00	0.00E+00	9.16E-08	0.00E+00	1.11E-04	0.00E+00	9.16E-08	0.00E+00	9.16E-08	5.22E-07	0.00E+00	9.16E-08	1.11E-04	0.1476%
7	Primate Center Boiler No 2 Natural Gas	0.00E+00	0.00E+00	0.00E+00	9.38E-08	0.00E+00	1.14E-04	0.00E+00	9.38E-08	0.00E+00	9.38E-08	5.36E-07	0.00E+00	9.38E-08	1.14E-04	0.1516%
8	Primate Center Boiler No 2 Landfill Gas	0.00E+00	2.30E-05	0.00E+00	1.45E-05	0.00E+00	3.35E-02	1.91E-10	3.38E-08	0.00E+00	7.90E-08	2.55E-03	0.00E+00	3.38E-08	3.35E-02	44.5479%
9	Landfill Flare	0.00E+00	4.16E-07	0.00E+00	2.20E-07	0.00E+00	5.05E-04	1.44E-11	7.64E-10	0.00E+00	2.54E-09	3.83E-05	0.00E+00	7.64E-10	5.05E-04	0.6715%
10	Incinerator	0.00E+00	0.00E+00	0.00E+00	1.96E-10	0.00E+00	2.41E-04	0.00E+00	1.96E-10	0.00E+00	1.96E-10	2.41E-04	0.00E+00	1.96E-10	2.41E-04	0.3205%
11	ARS J-1 (H001)	0.00E+00	0.00E+00	0.00E+00	6.96E-08	0.00E+00	8.48E-05	0.00E+00	6.96E-08	0.00E+00	6.96E-08	3.96E-07	0.00E+00	6.96E-08	8.48E-05	0.1128%
12	ARS J-1 CAAN 3840 - 4 boilers	0.00E+00	0.00E+00	0.00E+00	9.02E-08	0.00E+00	1.10E-04	0.00E+00	9.02E-08	0.00E+00	9.02E-08	5.14E-07	0.00E+00	9.02E-08	1.10E-04	0.1463%
13	ARS K-2 Co-located 2 stacks	0.00E+00	0.00E+00	0.00E+00	1.19E-07	0.00E+00	1.45E-04	0.00E+00	1.19E-07	0.00E+00	1.19E-07	6.77E-07	0.00E+00	1.19E-07	1.45E-04	0.1928%
14	ARS K-2 (H040)	0.00E+00	0.00E+00	0.00E+00	3.34E-08	0.00E+00	4.07E-05	0.00E+00	3.34E-08	0.00E+00	3.34E-08	1.90E-07	0.00E+00	3.34E-08	4.07E-05	0.0541%
15	Contained Research	0.00E+00	0.00E+00	0.00E+00	1.80E-07	0.00E+00	2.20E-04	0.00E+00	1.80E-07	0.00E+00	1.80E-07	1.03E-06	0.00E+00	1.80E-07	2.20E-04	0.2926%
16	Environmental Horticulture K-1	0.00E+00	0.00E+00	0.00E+00	3.14E-07	0.00E+00	3.82E-04	0.00E+00	3.14E-07	0.00E+00	3.14E-07	1.78E-06	0.00E+00	3.14E-07	3.82E-04	0.5080%
17	Environmental Horticulture K-2	0.00E+00	0.00E+00	0.00E+00	2.15E-07	0.00E+00	2.62E-04	0.00E+00	2.15E-07	0.00E+00	2.15E-07	1.22E-06	0.00E+00	2.15E-07	2.62E-04	0.3484%
18	Environmental Services Facility A	0.00E+00	0.00E+00	0.00E+00	7.14E-08	0.00E+00	8.70E-05	0.00E+00	7.14E-08	0.00E+00	7.14E-08	4.07E-07	0.00E+00	7.14E-08	8.70E-05	0.1157%
19	Environmenatl Services Facility (3 per stack)	0.00E+00	0.00E+00	0.00E+00	3.71E-08	0.00E+00	4.52E-05	0.00E+00	3.71E-08	0.00E+00	3.71E-08	2.12E-07	0.00E+00	3.71E-08	4.52E-05	0.0601%
20	Genome Launch Facility (plant reproduction)	0.00E+00	0.00E+00	0.00E+00	8.61E-08	0.00E+00	1.05E-04	0.00E+00	8.61E-08	0.00E+00	8.61E-08	4.90E-07	0.00E+00	8.61E-08	1.05E-04	0.1396%
21	Equine Analytical Chemistry Lab	0.00E+00	0.00E+00	0.00E+00	5.33E-08	0.00E+00	6.50E-05	0.00E+00	5.33E-08	0.00E+00	5.33E-08	3.04E-07	0.00E+00	5.33E-08	6.50E-05	0.0864%
22	Housing - Castillian DC	0.00E+00	0.00E+00	0.00E+00	1.99E-08	0.00E+00	2.42E-05	0.00E+00	1.99E-08	0.00E+00	1.99E-08	1.13E-07	0.00E+00	1.99E-08	2.42E-05	0.0322%
23	Housing - Castillian DC	0.00E+00	0.00E+00	0.00E+00	9.88E-08	0.00E+00	1.20E-04	0.00E+00	9.88E-08	0.00E+00	9.88E-08	5.62E-07	0.00E+00	9.88E-08	1.20E-04	0.1596%
24	Comparative Medicine (Primate Center)	0.00E+00	0.00E+00	0.00E+00	1.93E-08	0.00E+00	2.36E-05	0.00E+00	1.93E-08	0.00E+00	1.93E-08	1.10E-07	0.00E+00	1.93E-08	2.36E-05	0.0314%
25	Contained Research	0.00E+00	0.00E+00	0.00E+00	1.92E-08	0.00E+00	2.35E-05	0.00E+00	1.92E-08	0.00E+00	1.92E-08	1.10E-07	0.00E+00	1.92E-08	2.35E-05	0.0313%
26	Institute of Ecology - West Campus	0.00E+00	0.00E+00	0.00E+00	1.14E-07	0.00E+00	1.38E-04	0.00E+00	1.14E-07	0.00E+00	1.14E-07	6.45E-07	0.00E+00	1.14E-07	1.38E-04	0.1835%
27	ITEH Geriatrics - cage wash inside co-located 3 stacks	0.00E+00	0.00E+00	0.00E+00	2.33E-08	0.00E+00	2.83E-05	0.00E+00	2.33E-08	0.00E+00	2.33E-08	1.32E-07	0.00E+00	2.33E-08	2.83E-05	0.0376%
28	ITEH Geriatrics Cagewash - outside - co-locate 3 stacks	0.00E+00	0.00E+00	0.00E+00	2.53E-08	0.00E+00	3.09E-05	0.00E+00	2.53E-08	0.00E+00	2.53E-08	1.44E-07	0.00E+00	2.53E-08	3.09E-05	0.0411%
29	Mondavi Ctr for Performing Arts - 2 boilers	0.00E+00	0.00E+00	0.00E+00	2.44E-08	0.00E+00	2.97E-05	0.00E+00	2.44E-08	0.00E+00	2.44E-08	1.39E-07	0.00E+00	2.44E-08	2.97E-05	0.0395%
30	Mondavi Ctr for Performing Arts	0.00E+00	0.00E+00	0.00E+00	1.31E-08	0.00E+00	1.60E-05	0.00E+00	1.31E-08	0.00E+00	1.31E-08	7.46E-08	0.00E+00	1.31E-08	1.60E-05	0.0213%
31	Rec Pool	0.00E+00	0.00E+00	0.00E+00	1.07E-07	0.00E+00	1.31E-04	0.00E+00	1.07E-07	0.00E+00	1.07E-07	6.11E-07	0.00E+00	1.07E-07	1.31E-04	0.1742%
32	Thoreau Hall - 2 stacks co-located	0.00E+00	0.00E+00	0.00E+00	2.17E-08	0.00E+00	2.64E-05	0.00E+00	2.17E-08	0.00E+00	2.17E-08	1.23E-07	0.00E+00	2.17E-08	2.64E-05	0.0351%
33	Air Stripper	0.00E+00	5.49E-04	0.00E+00	5.49E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.49E-04	0.00E+00	0.00E+00	0.00E+00	5.49E-04	0.0000%
34	In-well Stripper	0.00E+00	5.25E-05	0.00E+00	5.25E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.25E-05	0.00E+00	0.00E+00	0.00E+00	5.25E-05	0.0000%
35	Ground Water Treatment	0.00E+00	6.12E-05	0.00E+00	6.12E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.12E-05	0.00E+00	0.00E+00	0.00E+00	6.12E-05	0.0000%
36	Large Kiln	0.00E+00	2.03E-09	0.00E+00	2.40E-08	0.00E+00	8.46E-06	0.00E+00	2.20E-08	0.00E+00	2.40E-08	5.03E-06	0.00E+00	2.20E-08	8.46E-06	0.0113%
37	Raku Kiln	0.00E+00	1.57E-09	0.00E+00	1.85E-08	0.00E+00	6.53E-06	0.00E+00	1.69E-08	0.00E+00	1.85E-08	3.88E-06	0.00E+00	1.69E-08	6.53E-06	0.0087%
38	Foundry Kiln	0.00E+00	2.42E-09	0.00E+00	2.86E-08	0.00E+00	1.01E-05	0.00E+00	2.62E-08	0.00E+00	2.86E-08	5.99E-06	0.00E+00	2.62E-08	1.01E-05	0.0134%
39	Three Art Dept Kilns to roof vent	0.00E+00	4.91E-09	0.00E+00	5.82E-08	0.00E+00	2.04E-05	0.00E+00	5.33E-08	0.00E+00	5.82E-08	1.21E-05	0.00E+00	5.33E-08	2.04E-05	0.0271%
40	Storehouse/Bulk Receiving and Storage	0.00E+00	7.98E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.98E-07	0.0000%
41	Walnut Dryer	0.00E+00	1.28E-07	0.00E+00	1.51E-06	0.00E+00	5.32E-04	0.00E+00	1.38E-06	0.00E+00	1.51E-06	3.16E-04	0.00E+00	1.38E-06	5.32E-04	0.7074%
42	Temporary Building 187	0.00E+00	1.30E-05	0.00E+00	1.23E-05	0.00E+00	3.52E-05	7.19E-08	1.79E-08	0.00E+00	1.23E-05	4.02E-06	0.00E+00	1.79E-08	3.52E-05	0.0468%
43	Temporary Building 188	0.00E+00	1.03E-05	0.00E+00	9.78E-06	0.00E+00	2.81E-05	5.73E-08	1.42E-08	0.00E+00	9.78E-06	3.20E-06	0.00E+00	1.42E-08	2.81E-05	0.0374%
44	Veihmeyer	0.00E+00	8.67E-06	0.00E+00	7.09E-06	0.00E+00	3.62E-05	2.62E-07	5.82E-08	0.00E+00	7.09E-06	1.68E-04	0.00E+00	5.82E-08	1.68E-04	0.0481%
45	Enology	0.00E+00	1.35E-05	0.00E+00	1.27E-05	0.00E+00	3.69E-05	7.43E-08	1.85E-08	0.00E+00	1.27E-05	5.40E-05	0.00E+00	1.85E-08	5.40E-05	0.0491%
46	Wickson Hall	0.00E+00	2.39E-04	0.00E+00	2.26E-04	0.00E+00	6.55E-04	1.32E-06	3.30E-07	0.00E+00	2.26E-04	2.17E-03	0.00E+00	3.30E-07	2.17E-03	0.8710%
47	Hoagland	0.00E+00	1.67E-04	0.00E+00	1.58E-04	0.00E+00	4.53E-04	9.22E-07	2.30E-07	0.00E+00	1.58E-04	1.51E-04	0.00E+00	2.30E-07	4.53E-04	0.6024%
48	Mann Hall	0.00E+00	5.75E-05	0.00E+00	5.43E-05	0.00E+00	3.17E-07	7.89E-08	0.00E+00	5.43E-05	5.43E-05	7.40E-05	0.00E+00	7.89E-08	1.55E-04	0.2061%
49	Storer Hall	0.00E+00	9.18E-06	0.00E+00	8.67E-06	0.00E+00	2.49E-05	5.07E-08	1.26E-08	0.00E+00	8.67E-06	4.47E-05	0.00E+00	1.26E-08	4.47E-05	0.0331%
50	Hutchison Hall/Biological Sci Unit 2	0.00E+00	1.82E-04	0.00E+00	1.72E-04	0.00E+00	4.95E-04	1.01E-06	2.52E-07	0.00E+00	1.72E-04	3.25E-04	0.00E+00	2.52E-07	4.95E-04	0.6582%
51	Asmundson Hall	0.00E+00	9.28E-05	0.00E+00	8.77E-05	0.00E+00	2.52E-04	5.12E-07	1.28E-07	0.00E+00	8.77E-05	2.66E-04	0.00E+00	1.28E-07	2.66E-04	0.3351%
52	Robbins Hall	0.00E+00	1.04E-04	0.00E+00	9.80E-05	0.00E+00	2.82E-04	5.74E-07	1.43E-07	0.00E+00	9.80E-05	1.26E-04	0.00E+00	1.43E-07	2.82E-04	0.3750%
53	Temporary Building 202	0.00E+00	1.24E-05	0.00E+00	1.17E-05	0.00E+00	3.35E-05	6.82E-08	1.70E-08	0.00E+00	1.17E-05	3.82E-06	0.00E+00	1.70E-08	3.35E-05	0.0445%
54	Briggs Hall and Life Sciences	0.00E+00	7.16E-04	0.00E+00	6.77E-04	0.00E+00	1.95E-03	3.96E-06	9.84E-07	0.00E+00	6.77E-04	1.97E-03	0.00E+00	9.84E-07	1.97E-03	2.5931%
55	Temporary Building 194	0.00E+00	1.98E-05	0.00E+00	1.87E-05	0.00E+00	5.39E-05	1.10E-07	2.73E-08	0.00E+00	1.87E-05	1.43E-04	0.00E+00	2.73E-08	1.43E-04	0.0717%
56	Food Science	0.00E+00	1.86E-06	0.00E+00	1.76E-06	0.00E+00	5.05E-06	1.03E-08	2.56E-09	0.00E+00	1.76E-06	5.75E-07	0.00E+00	2.56E-09	5.05E-06	0.0067%
57																

Acute HI for the MEIR at Grid Receptor # 1886 by Source
Northeast of the Primate Center on Larue Way

Source No.	Source Identification	Cardiovascular system	Central Nervous System	Bone	Develop- mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
61	Temporary Building 138	0.00E+00	1.41E-05	0.00E+00	1.33E-05	0.00E+00	3.81E-05	7.77E-08	1.94E-08	0.00E+00	1.33E-05	8.90E-05	0.00E+00	1.94E-08	8.90E-05	0.0507%
62	Temporary Building 155	0.00E+00	1.06E-05	0.00E+00	9.98E-06	0.00E+00	2.87E-05	5.84E-08	1.45E-08	0.00E+00	9.98E-06	3.27E-06	0.00E+00	1.45E-08	2.87E-05	0.0382%
63	Temporary Building 156	0.00E+00	9.58E-06	0.00E+00	9.05E-06	0.00E+00	2.60E-05	5.32E-08	1.32E-08	0.00E+00	9.05E-06	2.97E-06	0.00E+00	1.32E-08	2.60E-05	0.0346%
64	Temporary Building 157	0.00E+00	8.38E-06	0.00E+00	7.91E-06	0.00E+00	2.27E-05	4.64E-08	1.15E-08	0.00E+00	7.91E-06	2.60E-06	0.00E+00	1.15E-08	2.27E-05	0.0302%
65	Temporary Building 151	0.00E+00	1.22E-05	0.00E+00	1.15E-05	0.00E+00	3.31E-05	6.72E-08	1.68E-08	0.00E+00	1.15E-05	3.77E-06	0.00E+00	1.68E-08	3.31E-05	0.0440%
66	Temporary Building 149	0.00E+00	8.91E-06	0.00E+00	8.41E-06	0.00E+00	2.42E-05	4.93E-08	1.23E-08	0.00E+00	8.41E-06	2.76E-06	0.00E+00	1.23E-08	2.42E-05	0.0322%
67	Temporary Building 153	0.00E+00	5.93E-06	0.00E+00	5.60E-06	0.00E+00	1.61E-05	3.28E-08	8.15E-09	0.00E+00	5.60E-06	1.83E-06	0.00E+00	8.15E-09	1.61E-05	0.0214%
68	Temporary Building 158	0.00E+00	5.87E-06	0.00E+00	5.54E-06	0.00E+00	1.59E-05	3.24E-08	8.07E-09	0.00E+00	5.54E-06	1.81E-06	0.00E+00	8.07E-09	1.59E-05	0.0211%
69	Engineering II	0.00E+00	4.86E-04	0.00E+00	4.61E-04	0.00E+00	1.16E-05	2.63E-07	4.31E-06	0.00E+00	4.61E-04	4.35E-04	0.00E+00	4.31E-06	4.86E-04	0.0154%
70	Walker Hall	0.00E+00	4.62E-06	0.00E+00	3.77E-06	0.00E+00	1.92E-05	1.40E-07	3.10E-08	0.00E+00	3.77E-06	9.40E-06	0.00E+00	3.10E-08	1.92E-05	0.0255%
71	Chemistry	0.00E+00	1.59E-03	0.00E+00	1.51E-03	0.00E+00	3.84E-05	8.64E-07	1.41E-05	0.00E+00	1.51E-03	1.28E-03	0.00E+00	1.41E-05	1.59E-03	0.0511%
72	Chemistry Annex	0.00E+00	9.18E-04	0.00E+00	8.70E-04	0.00E+00	2.19E-05	4.98E-07	8.16E-06	0.00E+00	8.70E-04	4.07E-04	0.00E+00	8.16E-06	9.18E-04	0.0291%
73	Bainer Hall	0.00E+00	7.94E-04	0.00E+00	7.52E-04	0.00E+00	1.91E-05	4.31E-07	7.05E-06	0.00E+00	7.52E-04	4.99E-04	0.00E+00	7.05E-06	7.94E-04	0.0254%
74	Crocker Hall	0.00E+00	1.79E-06	0.00E+00	1.47E-06	0.00E+00	7.47E-06	5.42E-08	1.20E-08	0.00E+00	1.47E-06	2.92E-05	0.00E+00	1.20E-08	2.92E-05	0.0099%
75	Academic Surge	0.00E+00	5.49E-05	0.00E+00	5.19E-05	0.00E+00	1.49E-04	3.05E-07	7.56E-08	0.00E+00	5.19E-05	7.52E-05	0.00E+00	7.56E-08	1.49E-04	0.1981%
76	Meyer Hall	0.00E+00	3.57E-04	0.00E+00	3.38E-04	0.00E+00	9.67E-04	1.98E-06	4.92E-07	0.00E+00	3.38E-04	1.34E-03	0.00E+00	4.92E-07	1.34E-03	1.2859%
77	Physics/Geology/Physics Unit 1	0.00E+00	1.18E-05	0.00E+00	9.61E-06	0.00E+00	4.90E-05	3.55E-07	7.90E-08	0.00E+00	9.61E-06	1.18E-03	0.00E+00	7.90E-08	1.18E-03	0.0652%
78	Environmental Horticulture	0.00E+00	7.37E-05	0.00E+00	6.96E-05	0.00E+00	2.00E-04	4.06E-07	1.01E-07	0.00E+00	6.96E-05	3.61E-05	0.00E+00	1.01E-07	2.00E-04	0.2660%
79	Thurman Hall	0.00E+00	1.07E-04	0.00E+00	1.01E-04	0.00E+00	2.92E-04	5.92E-07	1.48E-07	0.00E+00	1.01E-04	1.53E-04	0.00E+00	1.48E-07	2.92E-04	0.3883%
80	Maddy Hall	0.00E+00	1.75E-04	0.00E+00	1.65E-04	0.00E+00	4.75E-04	9.70E-07	2.41E-07	0.00E+00	1.65E-04	3.95E-04	0.00E+00	2.41E-07	4.75E-04	0.6316%
81	Tupper Hall	0.00E+00	5.40E-04	0.00E+00	5.10E-04	0.00E+00	1.47E-03	2.99E-06	7.43E-07	0.00E+00	5.10E-04	1.01E-03	0.00E+00	7.43E-07	1.47E-03	1.9548%
82	VET MED 2	0.00E+00	5.91E-05	0.00E+00	5.58E-05	0.00E+00	1.61E-04	3.27E-07	8.13E-08	0.00E+00	5.58E-05	9.28E-05	0.00E+00	8.13E-08	1.61E-04	0.2141%
83	Asmundson Annex	0.00E+00	6.02E-06	0.00E+00	5.69E-06	0.00E+00	1.63E-05	3.32E-08	8.29E-09	0.00E+00	5.69E-06	1.86E-06	0.00E+00	8.29E-09	1.63E-05	0.0217%
84	Young Hall	0.00E+00	3.26E-05	0.00E+00	3.08E-05	0.00E+00	8.85E-05	1.80E-07	4.49E-08	0.00E+00	3.08E-05	5.48E-05	0.00E+00	4.49E-08	8.85E-05	0.1177%
85	Temporary Building 9	0.00E+00	7.43E-06	0.00E+00	7.02E-06	0.00E+00	2.01E-05	4.11E-08	1.02E-08	0.00E+00	7.02E-06	2.30E-06	0.00E+00	1.02E-08	2.01E-05	0.0267%
86	ARS H-1 (Vet Meta Res)	0.00E+00	1.50E-07	0.00E+00	1.22E-07	0.00E+00	6.24E-07	4.52E-09	1.01E-09	0.00E+00	1.22E-07	3.05E-07	0.00E+00	1.01E-09	6.24E-07	0.0008%
87	Serology4	0.00E+00	1.85E-05	0.00E+00	1.75E-05	0.00E+00	5.03E-05	1.02E-07	2.55E-08	0.00E+00	1.75E-05	5.72E-06	0.00E+00	2.55E-08	5.03E-05	0.0669%
88	ARS R-1	0.00E+00	9.10E-07	0.00E+00	8.60E-07	0.00E+00	2.46E-06	5.04E-09	1.25E-09	0.00E+00	8.60E-07	2.81E-07	0.00E+00	1.25E-09	2.46E-06	0.0033%
89	ARS R-2	0.00E+00	1.30E-05	0.00E+00	1.23E-05	0.00E+00	3.54E-05	7.20E-08	1.79E-08	0.00E+00	1.23E-05	4.03E-06	0.00E+00	1.79E-08	3.54E-05	0.0471%
90	Center For Comparative Medicine	0.00E+00	3.37E-04	0.00E+00	3.19E-04	0.00E+00	9.15E-04	1.86E-06	4.64E-07	0.00E+00	3.19E-04	3.37E-04	0.00E+00	4.64E-07	9.15E-04	1.2168%
91	Primate Center	0.00E+00	1.95E-04	0.00E+00	1.85E-04	0.00E+00	5.30E-04	1.08E-06	2.70E-07	0.00E+00	1.85E-04	6.05E-05	0.00E+00	2.70E-07	5.30E-04	0.7048%
92	Temporary Building 184	0.00E+00	9.53E-05	0.00E+00	9.00E-05	0.00E+00	2.58E-04	5.27E-07	1.31E-07	0.00E+00	9.00E-05	2.94E-05	0.00E+00	1.31E-07	2.58E-04	0.3431%
93	Temporary Building 160	0.00E+00	1.98E-05	0.00E+00	1.87E-05	0.00E+00	5.37E-05	1.09E-07	2.72E-08	0.00E+00	1.87E-05	6.11E-06	0.00E+00	2.72E-08	5.37E-05	0.0714%
94	APCARU	0.00E+00	2.19E-05	0.00E+00	2.07E-05	0.00E+00	5.93E-05	1.21E-07	3.00E-08	0.00E+00	2.07E-05	6.75E-06	0.00E+00	3.00E-08	5.93E-05	0.0789%
95	Ecology Lab (Aquadic Bio in bldg DB)	0.00E+00	2.55E-05	0.00E+00	2.41E-05	0.00E+00	6.91E-05	1.41E-07	3.50E-08	0.00E+00	2.41E-05	2.62E-04	0.00E+00	3.50E-08	2.62E-04	0.0919%
96	Temporary Building 1	0.00E+00	8.00E-06	0.00E+00	7.56E-06	0.00E+00	2.17E-05	4.43E-08	1.10E-08	0.00E+00	7.56E-06	2.48E-06	0.00E+00	1.10E-08	2.17E-05	0.0289%
97	ITEH Cellular Biology	0.00E+00	1.16E-05	0.00E+00	1.16E-05	0.00E+00	3.13E-05	6.39E-08	1.59E-08	0.00E+00	1.09E-05	3.56E-06	0.00E+00	1.59E-08	3.13E-05	0.0416%
98	ITEH Pathology Clinic	0.00E+00	1.13E-05	0.00E+00	1.07E-05	0.00E+00	3.07E-05	6.25E-08	1.56E-08	0.00E+00	1.07E-05	3.50E-06	0.00E+00	1.56E-08	3.07E-05	0.0408%
99	ARS DL-10/Field Shelter 5; Bovine Shed (Luekemia Lab)	0.00E+00	4.46E-06	0.00E+00	4.21E-06	0.00E+00	1.21E-05	2.47E-08	6.15E-09	0.00E+00	4.21E-06	1.38E-06	0.00E+00	6.15E-09	1.21E-05	0.0161%
100	Cole Fac A	0.00E+00	1.02E-05	0.00E+00	9.67E-06	0.00E+00	2.78E-05	5.66E-08	1.41E-08	0.00E+00	9.67E-06	3.17E-06	0.00E+00	1.41E-08	2.78E-05	0.0370%
101	Cole Fac B	0.00E+00	9.13E-06	0.00E+00	8.62E-06	0.00E+00	2.48E-05	5.06E-08	1.25E-08	0.00E+00	8.62E-06	2.83E-06	0.00E+00	1.25E-08	2.48E-05	0.0330%
102	Cole Fac C	0.00E+00	1.45E-05	0.00E+00	1.37E-05	0.00E+00	3.92E-05	8.01E-08	1.99E-08	0.00E+00	1.37E-05	4.47E-06	0.00E+00	1.99E-08	3.92E-05	0.0521%
103	TB 31	0.00E+00	1.68E-06	0.00E+00	1.59E-06	0.00E+00	4.58E-06	9.32E-09	2.32E-09	0.00E+00	1.59E-06	5.21E-07	0.00E+00	2.32E-09	4.58E-06	0.0061%
104	TB 33	0.00E+00	9.38E-06	0.00E+00	8.87E-06	0.00E+00	2.54E-05	5.19E-08	1.29E-08	0.00E+00	8.87E-06	2.90E-06	0.00E+00	1.29E-08	2.54E-05	0.0338%
105	TB 164	0.00E+00	1.31E-05	0.00E+00	1.23E-05	0.00E+00	3.55E-05	7.25E-08	1.80E-08	0.00E+00	1.23E-05	8.66E-05	0.00E+00	1.80E-08	3.55E-05	0.0472%
106	TB 165	0.00E+00	1.34E-05	0.00E+00	1.27E-05	0.00E+00	3.64E-05	7.42E-08	1.85E-08	0.00E+00	1.27E-05	4.15E-06	0.00E+00	1.85E-08	3.64E-05	0.0484%
107	TB 205	0.00E+00	1.39E-05	0.00E+00	1.31E-05	0.00E+00	3.78E-05	7.67E-08	1.91E-08	0.00E+00	1.31E-05	3.94E-05	0.00E+00	1.91E-08	3.94E-05	0.0503%
108	HH1	0.00E+00	2.30E-06	0.00E+00	2.17E-06	0.00E+00	6.24E-06	1.27E-08	3.16E-09	0.00E+00	2.17E-06	7.11E-07	0.00E+00	3.16E-09	6.24E-06	0.0083%
109	HH2	0.00E+00	8.06E-06	0.00E+00	7.61E-06	0.00E+00	2.19E-05	4.46E-08	1.11E-08	0.00E+00	7.61E-06	2.49E-06	0.00E+00	1.11E-08	2.19E-05	0.0291%
110	HH3	0.00E+00	1.51E-06	0.00E+00	1.43E-06	0.00E+00	4.09E-06	8.35E-09	2.08E-09	0.00E+00	1.43E-06	4.66E-07	0.00E+00	2.08E-09	4.09E-06	0.0054%
111	HH6	0.00E+00	1.32E-05	0.00E+00	1.25E-05	0.00E+00	3.58E-05	7.30E-08	1.81E-08	0.00E+00	1.25E-05	1.29E-04	0.00E+00	1.81E-08	1.30E-04	0.0476%
112	Vet Med Teaching Hospital (VMTH)	0.00E+00	2.56E-05	0.00E+00	2.42E-05	0.00E+00	6.93E-05	1.41E-07	3.51E-08	0.00E+00	2.42E-05	7.90E-06	0.00E+00	3.51E-08	6.93E-05	0.0922%
113	ARS Iso Barn J bldg	0.00E+00	6.92E-07	0.00E+00	6.54E-07	0.00E+00	1.88E-06	3.83E-09	9.50E-10	0.00E+00	6.54E-07	2.14E-07	0.00E+00	9.50E-10	1.88E-06	0.0025%
114	ITEH Animal Housing-2	0.00E+00	2.72E-06	0.00E+00	2.23E-06	0.00E+00	1.14E-05	8.23E-08	1.83E-08	0.00E+00	2.23E-06	1.53E-05	0.00E+00	1.83E-08	1.53E-05	0.0152%
115	LEHR Lab and Office	0.00E+00	3.11E-06	0.00E+00	2.54E-06	0.00E+00	1.30E-05	9.39E-08	2.09E-08	0.00E+00	2.54E-06	6.33E-06	0.00E+00	2.09E-08	1.30E-05	0.0173%
116	ITEH Toxic Pollutant Lab	0.00E+00	2.43E-06	0.00E+00	1.98E-06	0.00E+00	1.01E-05	7.33E-08	1.63E-08	0.00E+00	1.98E-06	1.51E-04	0.00E+00	1.63E-08	1.51E-04	0.0134%
117	Aqua weed lab/Aq Tox Shelter 5	0.00E+00	2.68E-05	0.00E+00	2.53E-05	0.0										

Acute HI for the MEIR at Grid Receptor # 1886 by Source
Northeast of the Primate Center on Larue Way

Source No.	Source Identification	Cardiovascular system	Central Nervous System	Bone	Develop-mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
181	P-59-07 Multi use stadium	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
182	No Permit Neurosci - off campus	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
183	P-16-09 New UG RES (Cat)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
184	No Permit Old Fire Station	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
185	P-29-96(a) Physical Plant	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
186	P-120-01 Plant Envir Sci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
187	P-50-99(a) Port Gen # 1	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
188	No Permit Port Gen # 14	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
189	P-51-99(a) Port Gen # 2	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
190	P-52-99(a) Port Gen # 3	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
191	P-86-01 Port Gen # 7	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
192	P-87-01 Port Gen # 8	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
193	P-49-07 Pri Animal Hous # 1	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
194	P-31-98 Primate Animal	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
195	P-32-98 Primate CCM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
196	P-69-96(a) Primate Freezers	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
197	P-102-94(a) Primate Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
198	P-15-98 Primate Quarantine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
199	No Permit Primate Sew Life Sta	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
200	P-16-98 Primate TB 184	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
201	P-108-01 Primate TB North # 5	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
202	P-109-01 Primate TB South # 6	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
203	P-99-94(a) Quad Parking	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
204	P-93-94(a) Rec Hall	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
205	P-111-95(a) Schl of Med Neurosci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
206	P-123-01 Schl of Med Neurosci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
207	P-15-04 Science Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
208	P-74-05 Segundo Dinning	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
209	P-126-95(a) Social Sci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
210	P-17-02 South Parking	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
211	P-92-94(a) Storm Lift # 4	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
212	C-09-129 Storm Lift #4 (new)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
213	P-71-00 Targeted genomics	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
214	P-111-01 Tele Comm.	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
215	P-91-94(a) Thurman Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
216	P-100-94(a) Toxic Pollutant	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
217	P-17-09 TURF	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
218	P-121-03 Tupper Load Dock	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
219	P-209-95(a) Util Well 6A	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
220	P-07-01 Vega Crops	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
221	P-63-03 Vet Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
222	P-52-07 Vet Med 3A	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
223	P-53-07 Vet Med 3A	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
224	P-59-05 Watershed Sic	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
225	P-38-05 West Entry Park	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
226	P-96-94(a) WEPT Influent	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
227	P-88-99 WEPT South	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
228	Landfill	0.00E+00	2.76E-03	0.00E+00	3.24E-05	0.00E+00	1.11E-04	3.08E-08	1.09E-05	0.00E+00	2.31E-05	1.11E-04	0.00E+00	1.09E-05	2.76E-03	0.1476%
229	Landfill	0.00E+00	4.52E-03	0.00E+00	5.30E-05	0.00E+00	1.82E-04	5.04E-08	1.78E-05	0.00E+00	3.79E-05	1.82E-04	0.00E+00	1.78E-05	4.52E-03	0.2420%
230	Landfill	0.00E+00	5.04E-03	0.00E+00	5.92E-05	0.00E+00	2.03E-04	5.62E-08	1.99E-05	0.00E+00	4.22E-05	2.03E-04	0.00E+00	1.99E-05	5.04E-03	0.2699%
231	Landfill	0.00E+00	4.38E-03	0.00E+00	5.14E-05	0.00E+00	1.76E-04	4.88E-08	1.73E-05	0.00E+00	3.67E-05	1.76E-04	0.00E+00	1.73E-05	4.38E-03	0.2340%
232	Waste Water Treatment Plant	0.00E+00	4.18E-04	0.00E+00	2.10E-04	0.00E+00	8.18E-05	0.00E+00	4.03E-08	0.00E+00	2.10E-04	8.18E-05	0.00E+00	4.03E-08	4.18E-04	0.1088%
233	Grounds Above-ground Storage Tank	0.00E+00	1.02E-09	0.00E+00	3.46E-08	0.00E+00	1.29E-09	0.00E+00	3.36E-08	0.00E+00	3.46E-08	1.29E-09	0.00E+00	3.36E-08	3.46E-08	0.0000%
234	Fleet Services Underground Storage Tank	0.00E+00	1.07E-07	0.00E+00	3.64E-06	0.00E+00	1.36E-07	0.00E+00	3.54E-06	0.00E+00	3.64E-06	1.36E-07	0.00E+00	3.54E-06	3.64E-06	0.0002%
235	Primate Center Gasoline AST	0.00E+00	3.37E-09	0.00E+00	1.15E-07	0.00E+00	4.28E-09	0.00E+00	1.11E-07	0.00E+00	1.15E-07	4.28E-09	0.00E+00	1.11E-07	1.15E-07	0.0

Cancer Risk for MEIW at Grid Receptor # 2045 by Chemical
 Corner of Russel Blvd. and Anderson Road - Rite Aid

Chemical No.	Chemical	Inhalation	Dermal	Soil	Mother's Milk	Home grown Vegetables	Oral	Total	Percent of Total
1	Diesel engine exhaust, particulate matter (Diesel PM)	2.39E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.39E-07	64.9457%
2	Formaldehyde	1.04E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.04E-08	2.8261%
3	Benzo[a]pyrene	2.76E-11	6.33E-10	8.22E-11	0.00E+00	0.00E+00	7.15E-10	7.42E-10	0.2016%
4	Dibenz[a,h]anthracene	1.90E-11	1.42E-10	1.84E-11	0.00E+00	0.00E+00	1.60E-10	1.79E-10	0.0486%
5	Carbon tetrachloride	4.88E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.88E-09	1.3261%
6	Benz[a]anthracene	1.13E-11	2.59E-10	3.37E-11	0.00E+00	0.00E+00	2.93E-10	3.04E-10	0.0826%
7	Methanol	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
8	Isopropyl alcohol	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
9	Chloroform	1.34E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.34E-08	3.6413%
10	Dimethyl formamide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
11	Benzene	4.22E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.22E-09	1.1467%
12	Methyl chloroform [1,1,1-TCA]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
13	Ethyl chloride [Chloroethane]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
14	Vinyl chloride	5.28E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.28E-10	0.1435%
15	Acetaldehyde	7.18E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.18E-11	0.0195%
16	Methylene chloride [Dichloromethane]	4.16E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.16E-09	1.1304%
17	Carbon disulfide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
18	1,1-Dichloroethane	5.65E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.65E-12	0.0015%
19	Vinylidene chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
20	1,2-Dichloropropane	5.47E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.47E-12	0.0015%
21	Methyl ethyl ketone [2-Butanone]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
22	Trichloroethylene	3.71E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.71E-11	0.0101%
23	Acrylamide	1.40E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.40E-08	3.8043%
24	1,1,2,2-Tetrachloroethane	1.59E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.59E-10	0.0432%
25	Naphthalene	4.67E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.67E-10	0.1269%
26	Ethyl benzene	1.85E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.85E-11	0.0050%
27	p-Dichlorobenzene	5.27E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.27E-12	0.0014%
28	Ethylene dibromide [EDB]	2.01E-13	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.01E-13	0.0001%
29	1,3-Butadiene	4.56E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.56E-09	1.2391%
30	Acrolein	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
31	Ethylene dichloride [EDC]	1.35E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.35E-09	0.3668%
32	Acrylonitrile	1.78E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.78E-09	0.4837%
33	Toluene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
34	Chlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
35	Hexane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
36	Glutaraldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
37	Propylene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
38	Triethylamine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
39	1,4-Dioxane	9.81E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.81E-10	0.2666%
40	Perchloroethylene [Tetrachloroethene]	4.38E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.38E-10	0.1190%
41	Indeno[1,2,3-cd]pyrene	2.59E-12	5.94E-11	7.72E-12	0.00E+00	0.00E+00	6.71E-11	6.97E-11	0.0189%
42	Benzo[b]fluoranthene	6.45E-12	1.48E-10	1.93E-11	0.00E+00	0.00E+00	1.67E-10	1.74E-10	0.0473%
43	Benzo[k]fluoranthene	8.69E-12	1.99E-10	2.59E-11	0.00E+00	0.00E+00	2.25E-10	2.34E-10	0.0636%
44	Chrysene	1.45E-12	3.33E-11	4.33E-12	0.00E+00	0.00E+00	3.77E-11	3.91E-11	0.0106%
45	Hydrazine	7.59E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.59E-09	2.0625%
46	Xylenes (mixed)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
47	2,3,7,8-Tetrachlorodibenzo-p-dioxin	1.58E-10	1.15E-09	4.18E-10	0.00E+00	0.00E+00	1.57E-09	1.73E-09	0.4701%
48	1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin	3.21E-13	2.33E-12	8.48E-13	0.00E+00	0.00E+00	3.18E-12	3.50E-12	0.0010%
49	Lead	3.70E-12	6.89E-12	1.16E-11	0.00E+00	0.00E+00	1.85E-11	2.22E-11	0.0060%
50	Mercury	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
51	Hydrochloric acid	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
52	Hydrogen fluoride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
53	Nitric acid	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
54	Hydrogen sulfide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
55	Phosphine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
56	Chromium, hexavalent (& compounds)	1.11E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.11E-09	0.3016%
57	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin	2.14E-10	1.55E-09	5.64E-10	0.00E+00	0.00E+00	2.12E-09	2.33E-09	0.6332%
58	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin	5.56E-11	4.04E-10	1.47E-10	0.00E+00	0.00E+00	5.51E-10	6.06E-10	0.1647%
59	1,2,3,4,6,7,8,9-Octachlorodibenzofuran	3.52E-13	2.56E-12	9.29E-13	0.00E+00	0.00E+00	3.49E-12	3.84E-12	0.0010%
60	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin	1.32E-10	9.59E-10	3.48E-10	0.00E+00	0.00E+00	1.31E-09	1.44E-09	0.3913%
61	1,2,3,7,8-Pentachlorodibenzo-p-dioxin	8.48E-10	6.16E-09	2.24E-09	0.00E+00	0.00E+00	8.40E-09	9.25E-09	2.5136%
62	2,3,7,8-Tetrachlorodibenzofuran	2.16E-10	1.57E-09	5.69E-10	0.00E+00	0.00E+00	2.14E-09	2.35E-09	0.6386%
63	1,2,3,4,7,8,9-Heptachlorodibenzofuran	1.50E-11	1.09E-10	3.95E-11	0.00E+00	0.00E+00	1.48E-10	1.63E-10	0.0443%
64	2,3,4,7,8-Pentachlorodibenzofuran	2.08E-09	1.51E-08	5.48E-09	0.00E+00	0.00E+00	2.06E-08	2.26E-08	6.1413%
65	1,2,3,7,8-Pentachlorodibenzofuran	1.17E-10	8.50E-10	3.09E-10	0.00E+00	0.00E+00	1.16E-09	1.28E-09	0.3478%
66	1,2,3,6,7,8-Hexachlorodibenzofuran	2.40E-10	1.75E-09	6.34E-10	0.00E+00	0.00E+00	2.38E-09	2.62E-09	0.7120%
67	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin	3.11E-10	2.26E-09	8.20E-10	0.00E+00	0.00E+00	3.08E-09	3.39E-09	0.9212%
68	2,3,4,6,7,8-Hexachlorodibenzofuran	5.02E-12	3.65E-11	1.33E-11	0.00E+00	0.00E+00	4.97E-11	5.48E-11	0.0149%
69	1,2,3,4,6,7,8-Heptachlorodibenzofuran	1.92E-10	1.39E-09	5.07E-10	0.00E+00	0.00E+00	1.90E-09	2.09E-09	0.5679%
70	1,2,3,4,7,8-Hexachlorodibenzofuran	6.29E-10	4.57E-09	1.66E-09	0.00E+00	0.00E+00	6.23E-09	6.86E-09	1.8641%
71	1,2,3,7,8,9-Hexachlorodibenzofuran	3.24E-12	2.35E-11	8.54E-12	0.00E+00	0.00E+00	3.21E-11	3.53E-11	0.0096%
SUM		3.14E-07	3.93E-08	1.40E-08	0.00E+00	0.00E+00	5.33E-08	3.68E-07	100.0000%

Cancer Risk for the MEIW at Grid Receptor # 2045 by Source
Corner of Russel Blvd. and Anderson Road - Rite Aid

Source No.	Source Identification	Inhalation	Dermal	Soil	Mother's Milk	Home grown Vegetables	Oral	Total	Percent of Total
1	Central Heating and Cooling Plant Boiler #1	3.51E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.51E-10	0.0954%
2	Central Heating and Cooling Plant Boiler #2	5.32E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.32E-10	0.1446%
3	Central Heating and Cooling Plant Boiler #3	1.03E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.03E-10	0.0280%
4	Central Heating and Cooling Plant Boiler #4, Natural Gas	4.09E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.09E-10	0.1111%
5	Central Heating and Cooling Plant Boiler #4, Diesel	3.04E-10	1.10E-11	1.43E-12	0.00E+00	0.00E+00	1.24E-11	3.16E-10	0.0859%
6	Primate Center Boiler No 1 Natural Gas	1.15E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.15E-12	0.0003%
7	Primate Center Boiler No 2 Natural Gas	1.17E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.17E-12	0.0003%
8	Primate Center Boiler No 2 Landfill Gas	2.89E-10	5.35E-10	1.09E-10	0.00E+00	0.00E+00	6.44E-10	9.33E-10	0.2535%
9	Landfill Flare	2.32E-13	3.19E-13	4.15E-14	0.00E+00	0.00E+00	3.61E-13	5.93E-13	0.0002%
10	Incinerator	5.34E-09	3.88E-08	1.38E-08	0.00E+00	0.00E+00	5.26E-08	5.80E-08	15.7609%
11	ARS J-1 (H001)	7.56E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.56E-11	0.0205%
12	ARS J-1 CAAN 3840 - 4 boilers	1.13E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.13E-10	0.0307%
13	ARS K-2 Co-located 2 stacks	1.28E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.28E-10	0.0348%
14	ARS K-2 (H040)	3.68E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.68E-11	0.0100%
15	Contained Research	3.72E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.72E-12	0.0010%
16	Environmental Horticulture K-1	6.25E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.25E-12	0.0017%
17	Environmental Horticulture K-2	8.61E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.61E-12	0.0023%
18	Environmental Services Facility A	1.78E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.78E-10	0.0484%
19	Environmental Services Facility (3 per stack)	1.01E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.01E-10	0.0274%
20	Genome Launch Facility (plant reproduction)	3.93E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.93E-11	0.0107%
21	Equine Analytical Chemistry Lab	2.45E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.45E-11	0.0067%
22	Housing - Castillian DC	4.91E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.91E-11	0.0133%
23	Housing - Castillian DC	2.67E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.67E-11	0.0073%
24	Comparative Medicine (Primate Center)	1.47E-14	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.47E-14	0.0000%
25	Contained Research	1.36E-14	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.36E-14	0.0000%
26	Institute of Ecology - West Campus	1.91E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.91E-11	0.0052%
27	ITEH Geriatrics - cage wash inside co-located 3 stacks	2.18E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.18E-12	0.0006%
28	ITEH Geriatrics Cagewash - outside - co-locate 3 stacks	2.79E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.79E-11	0.0076%
29	Mondavi Ctr for Performing Arts - 2 boilers	1.18E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.18E-11	0.0032%
30	Mondavi Ctr for Performing Arts	6.74E-13	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.74E-13	0.0002%
31	Rec Pool	4.20E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.20E-10	0.1141%
32	Thoreau Hall - 2 stacks co-located	4.11E-14	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.11E-14	0.0000%
33	Air Stripper	1.01E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.01E-11	0.0027%
34	In-well Stripper	1.07E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.07E-10	0.0291%
35	Ground Water Treatment	1.20E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.20E-10	0.0326%
36	Large Kiln	5.32E-10	3.47E-12	5.80E-12	0.00E+00	0.00E+00	9.28E-12	5.41E-10	0.1470%
37	Raku Kiln	2.42E-11	1.57E-13	2.63E-13	0.00E+00	0.00E+00	4.21E-13	2.46E-11	0.0067%
38	Foundry Kiln	1.21E-11	7.92E-14	1.32E-13	0.00E+00	0.00E+00	2.12E-13	1.23E-11	0.0033%
39	Three Art Dept Kilns to roof vent	5.01E-10	3.26E-12	5.46E-12	0.00E+00	0.00E+00	8.72E-12	5.09E-10	0.1383%
40	Storehouse/Bulk Receiving and Storage	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
41	Walnut Dryer	2.42E-12	1.34E-13	1.74E-14	0.00E+00	0.00E+00	1.52E-13	2.58E-12	0.0007%
42	Temporary Building 187	1.66E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.66E-10	0.0451%
43	Temporary Building 188	1.31E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.31E-10	0.0356%
44	Veihmeyer	1.63E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.63E-10	0.0443%
45	Enology	1.06E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.06E-10	0.0288%
46	Wickson Hall	1.27E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.27E-09	0.3451%
47	Hoagland	1.33E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.33E-09	0.3614%
48	Mann Hall	4.28E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.28E-10	0.1163%
49	Storer Hall	8.64E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.64E-11	0.0235%
50	Hutchison Hall/Biological Sci Unit 2	1.85E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.85E-09	0.5027%
51	Asmundson Hall	7.56E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.56E-10	0.2054%
52	Robbins Hall	9.00E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.00E-10	0.2446%
53	Temporary Building 202	6.66E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.66E-11	0.0181%
54	Briggs Hall and Life Sciences	6.45E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.45E-09	1.7527%
55	Temporary Building 194	8.62E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.62E-11	0.0234%
56	Food Science	7.86E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.86E-12	0.0021%
57	Temporary Building 193	3.26E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.26E-11	0.0089%
58	Temporary Building 191	2.61E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.61E-11	0.0071%
59	Temporary Building 166	2.59E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.59E-11	0.0070%
60	Temporary Building 167	5.10E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.10E-11	0.0139%
61	Temporary Building 138	5.19E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.19E-11	0.0141%
62	Temporary Building 155	4.89E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.89E-11	0.0133%
63	Temporary Building 156	4.42E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.42E-11	0.0120%
64	Temporary Building 157	3.83E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.83E-11	0.0104%
65	Temporary Building 151	5.59E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.59E-11	0.0152%
66	Temporary Building 149	3.09E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.09E-11	0.0084%
67	Temporary Building 153	2.75E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.75E-11	0.0075%
68	Temporary Building 158	2.72E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.72E-11	0.0074%
69	Engineering II	1.28E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.28E-09	0.3478%
70	Walker Hall	6.38E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.38E-11	0.0173%
71	Chemistry	8.89E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.89E-09	2.4158%
72	Chemistry Annex	4.68E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.68E-09	1.2717%
73	Bainer Hall	2.49E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.49E-09	0.6766%
74	Crocker Hall	2.45E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.45E-11	0.0067%
75	Academic Surge	2.85E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.85E-10	0.0774%
76	Meyer Hall	1.74E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.74E-09	0.4728%
77	Physics/Geology/Physics Unit 1	1.40E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.40E-10	0.0380%
78	Environmental Horticulture	2.48E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.48E-10	0.0674%
79	Thurman Hall	1.96E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.96E-10	0.0533%
80	Maddy Hall	3.78E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.78E-10	0.1027%
81	Tupper Hall	1.92E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.92E-09	0.5217%
82	VET MED 2	1.51E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.51E-10	0.0410%
83	Asmundson Annex	6.96E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.96E-11	0.0189%
84	Young Hall	1.56E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.56E-10	0.0424%
85	Temporary Building 9	2.83E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.83E-11	0.0077%
86	ARS H-1 (Vet Meta Res)	1.05E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.05E-12	0.0003%
87	Serology4	4.05E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.05E-11	0.0110%
88	ARS R-1	2.16E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.16E-12	0.0006%
89	ARS R-2	2.93E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.93E-11	0.0080%
90	Center For Comparative Medicine	1.89E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.89E-11	0.0051%
91	Primate Center	1.08E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.08E-11	0.0029%
92	Temporary Building 184	3.62E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.62E-12	0.0010%
93	Temporary Building 160	1.61E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.61E-12	0.0004%
94	APCARU	2.45E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.45E-12	0.0007%
95	Ecology Lab (Aquatic Bio in bldg DB)	1.31E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.31E-11	0.0036%
96	Temporary Building 1	5.70E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.70E-12	0.0015%
97	ITEH Cellular Biology	2.62E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.62E-11	0.0071%
98	ITEH Pathology Clinic	2.45E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.45E-11	0.0067%
99	ARS DL-10/Field Shelter 5; Bovine Shed (Luekemia Lab)	9.39E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.		

Cancer Risk for the MEIW at Grid Receptor # 2045 by Source
Corner of Russel Blvd. and Anderson Road - Rite Aid

Source No.	Source Identification	Inhalation	Dermal	Soil	Mother's Milk	Home grown Vegetables	Oral	Total	Percent of Total
101	Cole Fac B	3.13E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.13E-11	0.0085%
102	Cole Fac C	4.63E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.63E-11	0.0126%
103	TB 31	5.08E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.08E-12	0.0014%
104	TB 33	2.90E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.90E-11	0.0079%
105	TB 164	5.32E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.32E-11	0.0145%
106	TB 165	5.23E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.23E-11	0.0142%
107	TB 205	6.02E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.02E-11	0.0164%
108	HH1	1.31E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.31E-11	0.0036%
109	HH2	5.34E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.34E-11	0.0145%
110	HH3	1.17E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.17E-11	0.0032%
111	HH6	1.31E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.31E-10	0.0356%
112	Vet Med Teaching Hospital (VMTH)	9.50E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.50E-11	0.0258%
113	ARS Iso Barn J bldg	1.93E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.93E-12	0.0005%
114	ITEH Animal Housing-2	1.52E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.52E-11	0.0041%
115	LEHR Lab and Office	1.76E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.76E-11	0.0048%
116	ITEH Toxic Pollutant Lab	1.44E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.44E-11	0.0039%
117	Aqua weed lab/Aq Tox Shelter 5	1.27E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.27E-11	0.0035%
118	Bee Biology	1.85E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.85E-12	0.0005%
119	LEHR CLN MED/Medical Clinic	3.92E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.92E-11	0.0107%
120	Engineering 3 (EU3)	8.06E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.06E-10	0.2190%
121	TB 196 (Primate Center)	9.54E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.54E-12	0.0026%
122	Cruess Replacement	8.07E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.07E-10	0.2193%
123	Haring Hall Alteration	2.92E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.92E-09	0.7935%
124	Science Laboratory Building	2.65E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.65E-09	0.7201%
125	FPMS	7.24E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.24E-10	0.1967%
126	Everson Hall	2.86E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.86E-11	0.0078%
127	Center for Companion Animal Health	3.11E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.11E-10	0.0845%
128	Genome Launch Space	1.54E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.54E-09	0.4185%
129	Surge III	4.35E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.35E-12	0.0012%
130	Temporary Building 147	3.09E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.09E-12	0.0008%
131	Temporary Building 161	4.76E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.76E-11	0.0129%
132	Temporary Building 2	2.51E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.51E-12	0.0007%
133	Temporary Building 162	3.59E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.59E-11	0.0098%
134	Genome & Biomedical Science	2.47E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.47E-09	0.6712%
135	Temporary Building 127	6.43E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.43E-10	0.1747%
136	HC-2	9.44E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.44E-10	0.2565%
137	Germ Plasm	7.74E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.74E-10	0.2103%
138	Plant and Environmental Sciences	8.29E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.29E-11	0.0225%
139	Hunt Hall	5.42E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.42E-11	0.0147%
140	Cowell Student Health Center	1.54E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.54E-11	0.0042%
141	Med Sci D	3.27E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.27E-11	0.0089%
142	Equine Performance Laboratory	8.46E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.46E-09	2.2989%
143	Temporary Building 163	1.07E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.07E-10	0.0291%
144	P-17-98 60 Sub (115KV)	4.11E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.11E-10	0.1117%
145	No Permit Academic Surg	1.41E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.41E-09	0.3832%
146	No Permit Advanced Materials	9.36E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.36E-10	0.2543%
147	P-90-94(a) Aquaculture Trout	1.01E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.01E-09	0.2745%
148	P-107-95(a) Aquaculture II Well	3.18E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.18E-10	0.0864%
149	P-54-09 ARCH (rec hall)	1.46E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.46E-09	0.3967%
150	P-94-94(a) Bowley G.H	5.15E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.15E-09	1.3995%
151	P-118-03 CCAH	1.25E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.25E-10	0.0340%
152	No Permit Center for Neurosci	1.19E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.19E-10	0.0323%
153	P-82-02 Center For the Arts	1.71E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.71E-09	0.4647%
154	P-2-09 Child Health & Disease	4.60E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.60E-12	0.0013%
155	P-09-01 Cole B	4.62E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.62E-10	0.1255%
156	P-102-03 Contained Research	1.08E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.08E-10	0.0293%
157	No Permit Crocker	2.44E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.44E-10	0.0663%
158	P-08-01 Data Center	1.50E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.50E-09	0.4076%
159	P-83-02 Dom Grd Water Tank 1	1.77E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.77E-10	0.0481%
160	P-117-03 Dom Well # 2	1.29E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.29E-08	3.5054%
161	P-119-03 Dom Well # 3	4.17E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.17E-09	1.1332%
162	P-103-94(a) Dom Well # 4	7.75E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.75E-10	0.2106%
163	P-95-94(a) Dom Well # 6A	7.63E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.63E-10	0.2073%
164	P-42-97 Dom Well # 7a	6.72E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.72E-11	0.0183%
165	P-101-94(a) Engineering II	3.15E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.15E-09	0.8560%
166	P-01-00 Engineering III	3.99E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.99E-09	1.0842%
167	P-02-00 Equine Lab	9.25E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.25E-09	2.5136%
168	P-32-99 ESF	8.03E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.03E-10	0.2182%
169	P-89-94(a) Fire/Police	1.72E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.72E-08	4.6739%
170	P-51-07 Food Science	1.71E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.71E-09	0.4647%
171	P-84-02 FPMS	1.07E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.07E-10	0.0291%
172	P-120-03 GBSF	2.31E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.31E-09	0.6277%
173	P-114-02 Genome Launch	2.70E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.70E-09	0.7337%
174	No Permit Hickey Gym	4.42E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.42E-10	0.1201%
175	P-210-95(a) Hutch Sew Lift Sta	6.22E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.22E-10	0.1690%
176	No Permit Hutchison	1.14E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.14E-09	0.3098%
177	P-115-03 Inst of ecology lab	4.10E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.10E-10	0.1114%
178	No Permit ITEH (WR Lab)	1.34E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.34E-10	0.0364%
179	P-54-97 Life Sciences	4.98E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.98E-08	13.5326%
180	P-50-07 Mondavi RMI	7.63E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.63E-10	0.2073%
181	P-59-07 Multi use stadium	4.45E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.45E-10	0.1209%
182	No Permit Neurosci - off campus	1.24E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.24E-10	0.0337%
183	P-16-09 New UG RES (Cat)	1.57E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.57E-09	0.4266%
184	No Permit Old Fire Station	7.12E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.12E-10	0.1935%
185	P-29-96(a) Physical Plant	2.31E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.31E-09	0.6277%
186	P-120-01 Plant Envir Sci	1.90E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.90E-09	0.5163%
187	P-50-99(a) Port Gen # 1	2.64E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.64E-09	0.7174%
188	No Permit Port Gen # 14	1.53E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.53E-08	4.1576%
189	P-51-99(a) Port Gen # 2	2.94E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.94E-09	0.7989%
190	P-52-99(a) Port Gen # 3	1.70E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.70E-10	0.0462%
191	P-86-01 Port Gen # 7	5.72E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.72E-10	0.1554%
192	P-87-01 Port Gen # 8	5.18E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.18E-10	0.1408%
193	P-49-07 Pri Animal Hous # 1	4.94E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.94E-11	0.0134%
194	P-31-98 Primate Animal	6.22E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.22E-11	0.0169%
195	P-32-98 Primate CCM	8.81E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.81E-11	0.0239%
196	P-69-96(a) Primate Freezers	2.76E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.76E-11	0.0075%
197	P-102-94(a) Primate Lab	2.44E-10	0.00E+00	0.00E+00	0.00E				

Cancer Risk for the MEIW at Grid Receptor # 2045 by Source
 Corner of Russel Blvd. and Anderson Road - Rite Aid

Source No.	Source Identification	Inhalation	Dermal	Soil	Mother's Milk	Home grown Vegetables	Oral	Total	Percent of Total
201	P-108-01 Primate TB North # 5	4.58E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.58E-11	0.0124%
202	P-109-01 Primate TB South # 6	4.84E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.84E-11	0.0132%
203	P-99-94(a) Quad Parking	5.35E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.35E-10	0.1454%
204	P-93-94(a) Rec Hall	5.22E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.22E-09	1.4185%
205	P-111-95(a) Schl of Med Neurosci	2.62E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.62E-10	0.0712%
206	P-123-01 Schl of Med Neurosci	2.92E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.92E-10	0.0793%
207	P-15-04 Science Lab	1.03E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.03E-09	0.2799%
208	P-74-05 Segundo Dinning	2.42E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.42E-08	6.5761%
209	P-126-95(a) Social Sci	1.23E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.23E-09	0.3342%
210	P-17-02 South Parking	1.31E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.31E-09	0.3560%
211	P-92-94(a) Storm Lift # 4	4.28E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.28E-09	1.1630%
212	C-09-129 Storm Lift #4 (new)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
213	P-71-00 Targeted genomics	7.32E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.33E-10	0.1992%
214	P-111-01 Tele Comm.	2.29E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.29E-09	0.6223%
215	P-91-94(a) Thurman Lab	4.92E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.92E-09	1.3370%
216	P-100-94(a) Toxic Pollutant	2.17E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.17E-09	0.5897%
217	P-17-09 TURF	2.91E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.91E-12	0.0008%
218	P-121-03 Tupper Load Dock	2.05E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.05E-09	0.5571%
219	P-209-95(a) Util Well 6A	2.50E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.50E-09	0.6793%
220	P-07-01 Vega Crops	1.51E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.51E-09	0.4103%
221	P-63-03 Vet Lab	5.34E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.34E-10	0.1451%
222	P-52-07 Vet Med 3A	1.45E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.45E-09	0.3940%
223	P-53-07 Vet Med 3A	1.06E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.06E-09	0.2880%
224	P-59-05 Watershed Sic	7.42E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.42E-10	0.2016%
225	P-38-05 West Entry Park	3.14E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.14E-09	0.8533%
226	P-96-94(a) WEPT Influent	1.72E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.72E-08	4.6739%
227	P-88-99 WEPT South	2.01E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.01E-09	0.5462%
228	Landfill	4.15E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.15E-10	0.1128%
229	Landfill	5.11E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.11E-10	0.1389%
230	Landfill	6.43E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.43E-10	0.1747%
231	Landfill	7.48E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.48E-10	0.2033%
232	Waste Water Treatment Plant	6.93E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.93E-10	0.1883%
233	Grounds Above-ground Storage Tank	1.41E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.41E-11	0.0038%
234	Fleet Services Underground Storage Tank	9.51E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.51E-10	0.2584%
235	Primate Center Gasoline AST	9.31E-14	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.31E-14	0.0000%
236	Agricultural Services AST	3.28E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.28E-11	0.0089%
237	Plant Pathology Storage Tank	1.69E-13	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.69E-13	0.0000%
238	Pomology Above Ground Storage Tank	1.40E-13	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.40E-13	0.0000%
239	Airport Above Ground Storage Tank	1.09E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.09E-10	0.0296%
SUM		3.14E-07	3.93E-08	1.40E-08	0.00E+00	0.00E+00	5.33E-08	3.68E-07	100.0000%

Chronic HI for MEIW at Grid Receptor # 2045 by Chemical
 Corner of Russel Blvd. and Anderson Road - Rite Aid

Chemical No.	Chemical	Cardiovascular system	Central Nervous System	Bone	Develop-mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
1	Diesel engine exhaust, particulate matter (Diesel PM)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.60E-04	0.00E+00	0.00E+00	7.60E-04	11.4977%
2	Formaldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.63E-04	0.00E+00	0.00E+00	9.63E-04	14.5688%
3	Benzo[a]pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
4	Dibenz[a,h]anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
5	Carbon tetrachloride	0.00E+00	1.42E-05	0.00E+00	1.42E-05	0.00E+00	0.00E+00	1.42E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.42E-05	0.0000%
6	Benz[a]anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
7	Methanol	0.00E+00	0.00E+00	0.00E+00	2.44E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.44E-05	0.0000%
8	Isopropyl alcohol	0.00E+00	0.00E+00	0.00E+00	3.23E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.23E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.23E-06	0.0000%
9	Chloroform	0.00E+00	0.00E+00	0.00E+00	4.10E-05	0.00E+00	0.00E+00	4.10E-05	0.00E+00	4.10E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.10E-05	0.0000%
10	Dimethyl formamide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.49E-06	0.00E+00	0.00E+00	0.00E+00	4.49E-06	0.00E+00	0.00E+00	4.49E-06	0.0679%
11	Benzene	0.00E+00	1.23E-05	0.00E+00	1.23E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.23E-05	1.23E-05	0.0000%
12	Methyl chloroform [1,1,1-TCA]	0.00E+00	1.75E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.75E-07	0.0000%
13	Ethyl chloride [Chloroethane]	0.00E+00	0.00E+00	0.00E+00	2.01E-10	0.00E+00	0.00E+00	2.01E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.01E-10	0.0000%
14	Vinyl chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
15	Acetaldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.97E-07	0.00E+00	0.00E+00	8.97E-07	0.0136%
16	Methylene chloride [Dichloromethane]	5.20E-05	5.20E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.20E-05	0.0000%
17	Carbon disulfide	0.00E+00	4.12E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.12E-09	0.00E+00	0.00E+00	0.00E+00	4.12E-09	0.0000%
18	1,1-Dichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
19	Vinylidene chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.07E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.07E-08	0.0000%
20	1,2-Dichloropropane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
21	Methyl ethyl ketone [2-Butanone]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
22	Trichloroethylene	0.00E+00	1.55E-07	0.00E+00	0.00E+00	0.00E+00	1.55E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.55E-07	0.0000%
23	Acrylamide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
24	1,1,2,2-Tetrachloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
25	Naphthalene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.57E-06	0.00E+00	0.00E+00	7.57E-06	0.1145%
26	Ethyl benzene	0.00E+00	0.00E+00	0.00E+00	1.86E-08	1.86E-08	0.00E+00	1.86E-08	0.00E+00	1.86E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.86E-08	0.0000%
27	p-Dichlorobenzene	0.00E+00	2.88E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.88E-09	0.00E+00	2.88E-09	0.00E+00	2.88E-09	0.00E+00	0.00E+00	2.88E-09	0.0000%
28	Ethylene dibromide [EDB]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.76E-08	0.00E+00	0.00E+00	0.00E+00	1.76E-08	0.0000%
29	1,3-Butadiene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.65E-06	0.00E+00	0.00E+00	0.00E+00	6.65E-06	0.0000%
30	Acrolein	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.90E-07	0.00E+00	0.00E+00	7.90E-07	0.0120%
31	Ethylene dichloride [EDC]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.20E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.20E-07	0.0000%
32	Acrylonitrile	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.21E-06	0.00E+00	0.00E+00	6.21E-06	0.0939%
33	Toluene	0.00E+00	3.95E-05	0.00E+00	3.95E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.95E-05	0.00E+00	0.00E+00	3.95E-05	0.5976%
34	Chlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.10E-09	0.00E+00	2.10E-09	2.10E-09	0.00E+00	0.00E+00	0.00E+00	2.10E-09	0.0000%
35	Hexane	0.00E+00	1.36E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.36E-07	0.0000%
36	Glutaraldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.68E-03	0.00E+00	0.00E+00	2.68E-03	40.5446%
37	Propylene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.86E-09	0.00E+00	0.00E+00	1.86E-09	0.0000%
38	Triethylamine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.14E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.14E-06	0.0000%
39	1,4-Dioxane	2.12E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.12E-07	0.00E+00	2.12E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.12E-07	0.0000%
40	Perchloroethylene [Tetrachloroethene]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.04E-05	0.00E+00	1.04E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.04E-05	0.0000%
41	Indeno[1,2,3-cd]pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
42	Benzo[b]fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
43	Benzo[k]fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
44	Chrysene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
45	Hydrazine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.91E-05	0.00E+00	3.91E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.91E-05	0.0000%
46	Xylenes (mixed)	0.00E+00	1.82E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.82E-05	0.00E+00	0.00E+00	1.82E-05	0.2753%
47	2,3,7,8-Tetrachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	3.68E-06	3.68E-06	0.00E+00	3.68E-06	0.00E+00	0.00E+00	3.68E-06	3.68E-06	0.00E+00	3.68E-06	3.68E-06	0.0557%
48	1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	7.46E-09	7.46E-09	0.00E+00	7.46E-09	0.00E+00	0.00E+00	7.46E-09	7.46E-09	0.00E+00	7.46E-09	7.46E-09	0.0001%
49	Lead	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
50	Mercury	0.00E+00	6.23E-07	0.00E+00	6.23E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.23E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.23E-07	0.0000%
51	Hydrochloric acid	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.97E-03	0.00E+00	0.00E+00	1.97E-03	29.8033%
52	Hydrogen fluoride	0.00E+00	0.00E+00	1.35E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.35E-05	0.00E+00	0.00E+00	1.35E-05	0.2042%

Chronic HI for MEIW at Grid Receptor # 2045 by Chemical
 Corner of Russel Blvd. and Anderson Road - Rite Aid

Chemical No.	Chemical	Cardiovascular system	Central Nervous System	Bone	Develop-mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
53	Nitric acid	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
54	Hydrogen sulfide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.73E-05	0.00E+00	0.00E+00	1.73E-05	0.2617%
55	Phosphine	0.00E+00	3.83E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.83E-07	0.00E+00	3.83E-07	0.00E+00	3.83E-07	0.00E+00	3.83E-07	3.83E-07	0.0058%
56	Chromium, hexavalent (& compounds)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.91E-07	0.00E+00	7.05E-09	1.91E-07	0.0029%
57	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	4.97E-06	4.97E-06	0.00E+00	4.97E-06	0.00E+00	0.00E+00	4.97E-06	4.97E-06	0.00E+00	4.97E-06	4.97E-06	0.0752%
58	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	1.29E-06	1.29E-06	0.00E+00	1.29E-06	0.00E+00	0.00E+00	1.29E-06	1.29E-06	0.00E+00	1.29E-06	1.29E-06	0.0195%
59	1,2,3,4,6,7,8,9-Octachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	8.18E-09	8.18E-09	0.00E+00	8.18E-09	0.00E+00	0.00E+00	8.18E-09	8.18E-09	0.00E+00	8.18E-09	8.18E-09	0.0001%
60	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	3.07E-06	3.07E-06	0.00E+00	3.07E-06	0.00E+00	0.00E+00	3.07E-06	3.07E-06	0.00E+00	3.07E-06	3.07E-06	0.0464%
61	1,2,3,7,8-Pentachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	1.97E-05	1.97E-05	0.00E+00	1.97E-05	0.00E+00	0.00E+00	1.97E-05	1.97E-05	0.00E+00	1.97E-05	1.97E-05	0.2980%
62	2,3,7,8-Tetrachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	5.01E-06	5.01E-06	0.00E+00	5.01E-06	0.00E+00	0.00E+00	5.01E-06	5.01E-06	0.00E+00	5.01E-06	5.01E-06	0.0758%
63	1,2,3,4,7,8,9-Heptachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	3.47E-07	3.47E-07	0.00E+00	3.47E-07	0.00E+00	0.00E+00	3.47E-07	3.47E-07	0.00E+00	3.47E-07	3.47E-07	0.0052%
64	2,3,4,7,8-Pentachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	4.82E-05	4.82E-05	0.00E+00	4.82E-05	0.00E+00	0.00E+00	4.82E-05	4.82E-05	0.00E+00	4.82E-05	4.82E-05	0.7292%
65	1,2,3,7,8-Pentachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	2.72E-06	2.72E-06	0.00E+00	2.72E-06	0.00E+00	0.00E+00	2.72E-06	2.72E-06	0.00E+00	2.72E-06	2.72E-06	0.0411%
66	1,2,3,6,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	5.58E-06	5.58E-06	0.00E+00	5.58E-06	0.00E+00	0.00E+00	5.58E-06	5.58E-06	0.00E+00	5.58E-06	5.58E-06	0.0844%
67	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	7.22E-06	7.22E-06	0.00E+00	7.22E-06	0.00E+00	0.00E+00	7.22E-06	7.22E-06	0.00E+00	7.22E-06	7.22E-06	0.1092%
68	2,3,4,6,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	1.17E-07	1.17E-07	0.00E+00	1.17E-07	0.00E+00	0.00E+00	1.17E-07	1.17E-07	0.00E+00	1.17E-07	1.17E-07	0.0018%
69	1,2,3,4,6,7,8-Heptachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	4.46E-06	4.46E-06	0.00E+00	4.46E-06	0.00E+00	0.00E+00	4.46E-06	4.46E-06	0.00E+00	4.46E-06	4.46E-06	0.0675%
70	1,2,3,4,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	1.46E-05	1.46E-05	0.00E+00	1.46E-05	0.00E+00	0.00E+00	1.46E-05	1.46E-05	0.00E+00	1.46E-05	1.46E-05	0.2209%
71	1,2,3,7,8,9-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	7.52E-08	7.52E-08	0.00E+00	7.52E-08	0.00E+00	0.00E+00	7.52E-08	7.52E-08	0.00E+00	7.52E-08	7.52E-08	0.0011%
SUM		5.22E-05	1.38E-04	1.35E-05	2.56E-04	1.60E-04	1.29E-06	2.32E-04	0.00E+00	5.59E-05	1.28E-04	6.61E-03	0.00E+00	1.34E-04	6.61E-03	100.0000%

Chronic HI for the MEIW at Grid Receptor # 2045 by Source
 Corner of Russel Blvd. and Anderson Road - Rite Aid

Source No.	Source Identification	Cardiovascular system	Central Nervous System	Bone	Develop- mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
1	Central Heating and Cooling Plant Boiler #1	0.00E+00	8.55E-08	0.00E+00	8.55E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.93E-05	0.00E+00	8.55E-08	2.93E-05	0.4433%
2	Central Heating and Cooling Plant Boiler #2	0.00E+00	1.30E-07	0.00E+00	1.30E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.44E-05	0.00E+00	1.30E-07	4.44E-05	0.6717%
3	Central Heating and Cooling Plant Boiler #3	0.00E+00	2.51E-08	0.00E+00	2.51E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.56E-06	0.00E+00	2.51E-08	8.56E-06	0.1295%
4	Central Heating and Cooling Plant Boiler #4, Natural Gas	0.00E+00	9.92E-08	0.00E+00	9.92E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.41E-05	0.00E+00	9.92E-08	3.41E-05	0.5159%
5	Central Heating and Cooling Plant Boiler #4, Diesel	0.00E+00	5.10E-09	0.00E+00	4.94E-09	7.65E-11	0.00E+00	7.65E-11	0.00E+00	7.65E-11	0.00E+00	8.18E-06	0.00E+00	4.35E-09	8.18E-06	0.1238%
6	Primate Center Boiler No 1 Natural Gas	0.00E+00	2.80E-10	0.00E+00	2.80E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.59E-08	0.00E+00	2.80E-10	9.59E-08	0.0015%
7	Primate Center Boiler No 2 Natural Gas	0.00E+00	2.84E-10	0.00E+00	2.84E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.74E-08	0.00E+00	2.84E-10	9.74E-08	0.0015%
8	Primate Center Boiler No 2 Landfill Gas	6.05E-11	3.92E-08	2.65E-07	5.76E-07	5.37E-07	1.23E-11	5.38E-07	0.00E+00	3.93E-08	5.37E-07	1.13E-05	0.00E+00	5.37E-07	1.13E-05	0.1710%
9	Landfill Flare	2.63E-13	3.44E-11	2.27E-10	3.41E-11	3.18E-15	5.35E-14	1.57E-12	0.00E+00	3.54E-11	2.35E-14	9.38E-09	0.00E+00	3.23E-14	9.38E-09	0.0001%
10	Incinerator	0.00E+00	3.79E-09	0.00E+00	1.21E-04	1.21E-04	0.00E+00	1.21E-04	0.00E+00	0.00E+00	1.21E-04	1.85E-04	0.00E+00	1.21E-04	1.85E-04	2.7988%
11	ARS J-1 (H001)	0.00E+00	1.84E-08	0.00E+00	1.84E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.31E-06	0.00E+00	1.84E-08	6.31E-06	0.0955%
12	ARS J-1 CAAN 3840 - 4 boilers	0.00E+00	2.74E-08	0.00E+00	2.74E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.41E-06	0.00E+00	2.74E-08	9.41E-06	0.1424%
13	ARS K-2 Co-located 2 stacks	0.00E+00	3.10E-08	0.00E+00	3.10E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.07E-05	0.00E+00	3.10E-08	1.07E-05	0.1619%
14	ARS K-2 (H040)	0.00E+00	8.95E-09	0.00E+00	8.95E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.07E-06	0.00E+00	8.95E-09	3.07E-06	0.0464%
15	Contained Research	0.00E+00	9.06E-10	0.00E+00	9.06E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.10E-07	0.00E+00	9.06E-10	3.10E-07	0.0047%
16	Environmental Horticulture K-1	0.00E+00	1.52E-09	0.00E+00	1.52E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.22E-07	0.00E+00	1.52E-09	5.22E-07	0.0079%
17	Environmental Horticulture K-2	0.00E+00	2.10E-09	0.00E+00	2.10E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.18E-07	0.00E+00	2.10E-09	7.18E-07	0.0109%
18	Environmental Services Facility A	0.00E+00	4.31E-08	0.00E+00	4.31E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.48E-05	0.00E+00	4.31E-08	1.48E-05	0.2239%
19	Environmentatl Services Facility (3 per stack)	0.00E+00	2.47E-08	0.00E+00	2.47E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.44E-06	0.00E+00	2.47E-08	8.44E-06	0.1277%
20	Genome Launch Facility (plant reproduction)	0.00E+00	9.57E-09	0.00E+00	9.57E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.28E-06	0.00E+00	9.57E-09	3.28E-06	0.0496%
21	Equine Analytical Chemistry Lab	0.00E+00	5.97E-09	0.00E+00	5.97E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.04E-06	0.00E+00	5.97E-09	2.04E-06	0.0309%
22	Housing - Castillian DC	0.00E+00	1.20E-08	0.00E+00	1.20E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.09E-06	0.00E+00	1.20E-08	4.09E-06	0.0619%
23	Housing - Castillian DC	0.00E+00	6.52E-09	0.00E+00	6.52E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.23E-06	0.00E+00	6.52E-09	2.23E-06	0.0337%
24	Comparative Medicine (Primate Center)	0.00E+00	3.58E-12	0.00E+00	3.58E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.23E-09	0.00E+00	3.58E-12	1.23E-09	0.0000%
25	Contained Research	0.00E+00	3.31E-12	0.00E+00	3.31E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.14E-09	0.00E+00	3.31E-12	1.14E-09	0.0000%
26	Institute of Ecology - West Campus	0.00E+00	4.66E-09	0.00E+00	4.66E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.60E-06	0.00E+00	4.66E-09	1.60E-06	0.0242%
27	ITEH Geriatrics - cage wash inside co-located 3 stacks	0.00E+00	5.31E-10	0.00E+00	5.31E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.82E-07	0.00E+00	5.31E-10	1.82E-07	0.0028%
28	ITEH Geriatrics Cagewash - outside - co-locate 3 stacks	0.00E+00	6.79E-09	0.00E+00	6.79E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.33E-06	0.00E+00	6.79E-09	2.33E-06	0.0352%
29	Mondavi Ctr for Performing Arts - 2 boilers	0.00E+00	2.85E-09	0.00E+00	2.85E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.82E-07	0.00E+00	2.85E-09	9.82E-07	0.0149%
30	Mondavi Ctr for Performing Arts	0.00E+00	1.64E-10	0.00E+00	1.64E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.63E-08	0.00E+00	1.64E-10	5.63E-08	0.0009%
31	Rec Pool	0.00E+00	1.02E-07	0.00E+00	1.02E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.51E-05	0.00E+00	1.02E-07	3.51E-05	0.5310%
32	Thoreau Hall - 2 stacks co-located	0.00E+00	1.00E-11	0.00E+00	1.00E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.43E-09	0.00E+00	1.00E-11	3.43E-09	0.0001%
33	Air Stripper	0.00E+00	0.00E+00	0.00E+00	3.09E-08	0.00E+00	0.00E+00	3.09E-08	0.00E+00	3.09E-08	0.00E+00	0.00E+00	0.00E+00	3.09E-08	3.09E-08	0.0000%
34	In-well Stripper	0.00E+00	0.00E+00	0.00E+00	3.29E-07	0.00E+00	0.00E+00	3.29E-07	0.00E+00	3.29E-07	0.00E+00	0.00E+00	0.00E+00	3.29E-07	3.29E-07	0.0000%
35	Ground Water Treatment	0.00E+00	0.00E+00	0.00E+00	3.69E-07	0.00E+00	0.00E+00	3.69E-07	0.00E+00	3.69E-07	0.00E+00	0.00E+00	0.00E+00	3.69E-07	3.69E-07	0.0000%
36	Large Kiln	0.00E+00	1.34E-09	0.00E+00	1.25E-09	4.93E-12	0.00E+00	4.93E-12	0.00E+00	4.93E-12	0.00E+00	1.89E-07	0.00E+00	4.17E-09	1.89E-07	0.0029%
37	Raku Kiln	0.00E+00	6.08E-11	0.00E+00	5.69E-11	2.24E-13	0.00E+00	2.24E-13	0.00E+00	2.24E-13	0.00E+00	8.60E-09	0.00E+00	1.90E-10	8.60E-09	0.0001%
38	Foundry Kiln	0.00E+00	3.06E-11	0.00E+00	2.86E-11	1.13E-13	0.00E+00	1.13E-13	0.00E+00	1.13E-13	0.00E+00	4.32E-09	0.00E+00	9.50E-11	4.32E-09	0.0001%
39	Three Art Dept Kilns to roof vent	0.00E+00	1.26E-09	0.00E+00	1.18E-09	4.63E-12	0.00E+00	4.63E-12	0.00E+00	4.63E-12	0.00E+00	1.78E-07	0.00E+00	3.92E-09	1.78E-07	0.0027%
40	Storehouse/Bulk Receiving and Storage	0.00E+00	0.00E+00	0.00E+00	1.57E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.57E-07	1.57E-07	0.0000%
41	Walnut Dryer	0.00E+00	4.35E-09	0.00E+00	4.07E-09	1.60E-11	0.00E+00	1.60E-11	0.00E+00	1.60E-11	0.00E+00	3.21E-07	0.00E+00	2.65E-09	3.21E-07	0.0049%
42	Temporary Building 187	1.08E-08	8.67E-08	3.70E-08	2.52E-07	1.16E-07	2.61E-09	2.81E-07	0.00E+00	1.08E-07	0.00E+00	2.12E-05	0.00E+00	6.01E-09	2.12E-05	0.3207%
43	Temporary Building 188	8.58E-09	6.86E-08	2.93E-08	1.99E-07	9.20E-08	2.07E-09	2.22E-07	0.00E+00	8.54E-08	0.00E+00	1.67E-05	0.00E+00	4.76E-09	1.67E-05	0.2526%
44	Veihmeyer	6.45E-09	1.56E-07	1.15E-06	2.82E-07	2.65E-08	5.83E-09	2.34E-07	0.00E+00	6.45E-08	0.00E+00	2.13E-05	0.00E+00	1.10E-08	2.13E-05	0.3222%
45	Enology	6.87E-09	5.49E-08	2.33E-08	1.59E-07	7.35E-08	1.66E-09	1.77E-07	0.00E+00	6.83E-08	0.00E+00	1.34E-05	0.00E+00	3.81E-09	1.34E-05	0.2027%
46	Wickson Hall	8.21E-08	6.56E-07	2.80E-07	1.91E-06	8.80E-07	1.98E-08	2.13E-06	0.00E+00	8.19E-07	0.00E+00	1.60E-04	0.00E+00	4.56E-08	1.60E-04	2.4206%
47	Hoagland	8.71E-08	6.94E-07	2.96E-07	2.02E-06	9.32E-07	2.10E-08	2.25E-06	0.00E+00	8.65E-07	0.00E+00	1.70E-04	0.00E+00	4.82E-08	1.70E-04	2.5719%
48	Mann Hall	2.79E-08	2.23E-07	9.49E-08	6.47E-07	2.99E-07	6.72E-09	7.20E-07	0.00E+00	2.77E-07	0.00E+00	5.43E-05	0.00E+00	1.55E-08	5.43E-05	0.8215%
49	Storer Hall	5.64E-09	4.50E-08	1.92E-08	1.31E-07	6.05E-08	1.36E-09	1.46E-07	0.00E+00	5.61E-08	0.00E+00	1.10E-05	0.00E+00	3.13E-09	1.10E-05	0.1664%
50	Hutchison Hall/Biological Sci Unit 2	1.21E-07	9.64E-07	4.11E-07	2.80E-06	1.30E-06	2.91E-08	3.13E-06	0.00E+00	1.20E-06	0.00E+00	2.35E-04	0.00E+00	6.69E-08	2.35E-04	3.5552%
51	Asmundson Hall	4.94E-08	3.95E-07	1.68E-07	1.15E-06	5.30E-07	1.28E-06	1.28E-06	0.00E+00	4.90E-07	0.00E+00	9.62E-05	0.00E+00	2.74E-08	9.62E-05	1.4554%
52	Robbins Hall	5.87E-08	4.69E-07	2.00E-07	1.36E-06	6.30E-07	1.42E-08	1.52E-06	0.00E+00	5.84E-07	0.00E+00	1.14E-04	0.00E+00	3.26E-08	1.14E-04	1.7247%
53	Temporary Building 202	4.35E-09	3.48E-08	1.48E-08	1.01E-07	4.67E-08	1.05E-09	1.13E-07	0.00E+00	4.33E-08	0.00E+00	8.48E-06	0.00E+00	2.41E-09	8.48E-06	0.1283%
54	Briggs Hall and Life Sciences	4.20E-07	3.36E-06	1.43E-06	9.76E-06	4.50E-06	1.01E-07	1.09E-05	0.00E+00	4.18E-06	0.00E+00	8.18E-04	0.00E+00	2.33E-07	8.18E-04	12.3752%
55	Temporary Building 194	5.63E-09	4.50E-08	1.92E-08	1.31E-07	6.04E-08	1.36E-09	1.46E-07	0.00E+00	5.60E-08	0.00E+00	1.10E-05	0.00E+00	3.12E-09	1.10E-05	0.1664%
56	Food Science	5.13E-10	4.10E-09	1.75E-09	1.19E-08	5.51E-09	1.24E-10	1.33E-08	0.00E+00	5.11E-09	0.00E+00	1.00E-06	0.00E+00	2.85E-10	1.00E-06	0.0151%
57																

Chronic HI for the MEIW at Grid Receptor # 2045 by Source
 Corner of Russel Blvd. and Anderson Road - Rite Aid

Source No.	Source Identification	Cardiovascular system	Central Nervous System	Bone	Develop- mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
62	Temporary Building 155	3.20E-09	2.55E-08	1.09E-08	7.42E-08	3.43E-08	7.70E-10	8.27E-08	0.00E+00	3.18E-08	0.00E+00	6.22E-06	0.00E+00	1.77E-09	6.22E-06	0.0941%
63	Temporary Building 156	2.88E-09	2.30E-08	9.85E-09	6.70E-08	3.09E-08	6.97E-10	7.47E-08	0.00E+00	2.87E-08	0.00E+00	5.62E-06	0.00E+00	1.60E-09	5.62E-06	0.0850%
64	Temporary Building 157	2.50E-09	2.00E-08	8.53E-09	5.80E-08	2.69E-08	6.03E-10	6.47E-08	0.00E+00	2.48E-08	0.00E+00	4.88E-06	0.00E+00	1.39E-09	4.88E-06	0.0738%
65	Temporary Building 151	3.65E-09	2.92E-08	1.25E-08	8.48E-08	3.91E-08	8.81E-10	9.44E-08	0.00E+00	3.63E-08	0.00E+00	7.12E-06	0.00E+00	2.03E-09	7.12E-06	0.1077%
66	Temporary Building 149	2.02E-09	1.61E-08	6.89E-09	4.69E-08	2.16E-08	4.87E-10	5.22E-08	0.00E+00	2.01E-08	0.00E+00	3.94E-06	0.00E+00	1.12E-09	3.94E-06	0.0596%
67	Temporary Building 153	1.80E-09	1.44E-08	6.15E-09	4.17E-08	1.93E-08	4.34E-10	4.65E-08	0.00E+00	1.79E-08	0.00E+00	3.50E-06	0.00E+00	1.00E-09	3.50E-06	0.0530%
68	Temporary Building 158	1.78E-09	1.42E-08	6.06E-09	4.12E-08	1.91E-08	4.28E-10	4.59E-08	0.00E+00	1.77E-08	0.00E+00	3.46E-06	0.00E+00	9.86E-10	3.46E-06	0.0523%
69	Engineering II	4.03E-06	5.04E-06	3.41E-09	2.82E-06	8.86E-07	4.89E-08	2.48E-06	0.00E+00	1.42E-06	0.00E+00	9.70E-06	0.00E+00	5.60E-07	9.70E-06	0.1467%
70	Walker Hall	2.53E-09	6.10E-08	4.51E-07	1.11E-07	1.04E-08	2.30E-09	9.18E-08	0.00E+00	2.53E-08	0.00E+00	8.32E-06	0.00E+00	4.31E-09	8.32E-06	0.1259%
71	Chemistry	1.85E-05	2.32E-05	1.57E-08	1.30E-05	4.07E-06	2.24E-07	1.14E-05	0.00E+00	6.54E-06	4.38E-06	4.47E-05	0.00E+00	2.58E-06	4.47E-05	0.6762%
72	Chemistry Annex	9.64E-06	1.21E-05	8.15E-09	6.74E-06	2.12E-06	1.17E-07	5.93E-06	0.00E+00	3.40E-06	2.26E-06	2.34E-05	0.00E+00	1.34E-06	2.34E-05	0.3540%
73	Bainer Hall	7.16E-06	8.97E-06	6.07E-09	5.03E-06	1.58E-06	8.72E-08	4.42E-06	0.00E+00	2.54E-06	0.00E+00	1.80E-05	0.00E+00	9.99E-07	1.80E-05	0.2723%
74	Crocker Hall	9.75E-10	2.35E-08	1.73E-07	4.26E-08	3.99E-09	8.84E-10	3.53E-08	0.00E+00	9.73E-09	0.00E+00	3.21E-06	0.00E+00	1.66E-09	3.21E-06	0.0486%
75	Academic Surge	1.86E-08	1.49E-07	6.36E-08	4.33E-07	2.00E-07	4.50E-09	4.82E-07	0.00E+00	1.85E-07	0.00E+00	3.63E-05	0.00E+00	1.04E-08	3.63E-05	0.5492%
76	Meyer Hall	1.13E-07	1.29E-06	3.86E-07	2.63E-06	1.22E-06	2.73E-08	3.32E-06	0.00E+00	1.51E-06	0.00E+00	2.21E-04	0.00E+00	4.46E-07	2.21E-04	3.3434%
77	Physics/Geology/Physics Unit 1	5.57E-09	1.34E-07	9.95E-07	2.43E-07	2.28E-08	5.04E-09	2.02E-07	0.00E+00	5.56E-08	0.00E+00	1.83E-05	0.00E+00	9.47E-09	1.83E-05	0.2769%
78	Environmental Horticulture	1.61E-08	1.29E-07	5.51E-08	3.75E-07	1.73E-07	3.89E-09	4.18E-07	0.00E+00	1.61E-07	0.00E+00	3.15E-05	0.00E+00	8.95E-09	3.15E-05	0.4766%
79	Thurman Hall	1.28E-08	1.02E-07	4.36E-08	2.97E-07	1.37E-07	3.08E-09	3.31E-07	0.00E+00	1.27E-07	0.00E+00	2.49E-05	0.00E+00	7.09E-09	2.49E-05	0.3767%
80	Maddy Hall	2.47E-08	1.97E-07	8.40E-08	5.72E-07	2.64E-07	5.94E-09	6.38E-07	0.00E+00	2.45E-07	0.00E+00	4.80E-05	0.00E+00	1.37E-08	4.80E-05	0.7262%
81	Tupper Hall	1.25E-07	9.99E-07	4.26E-07	2.90E-06	1.34E-06	3.01E-08	3.23E-06	0.00E+00	1.24E-06	0.00E+00	2.44E-04	0.00E+00	6.93E-08	2.44E-04	3.6914%
82	VET MED 2	9.87E-09	7.87E-08	3.36E-08	2.29E-07	1.05E-07	2.38E-09	2.55E-07	0.00E+00	9.81E-08	0.00E+00	1.92E-05	0.00E+00	5.47E-09	1.92E-05	0.2905%
83	Asmundson Annex	4.54E-09	3.63E-08	1.55E-08	1.05E-07	4.87E-08	1.09E-09	1.17E-07	0.00E+00	4.52E-08	0.00E+00	8.84E-06	0.00E+00	2.51E-09	8.84E-06	0.1337%
84	Young Hall	1.02E-08	8.14E-08	3.48E-08	2.36E-07	1.09E-07	2.46E-09	2.64E-07	0.00E+00	1.01E-07	0.00E+00	1.99E-05	0.00E+00	5.65E-09	1.99E-05	0.3011%
85	Temporary Building 9	1.85E-09	1.48E-08	6.29E-09	4.29E-08	1.98E-08	4.45E-10	4.78E-08	0.00E+00	1.84E-08	0.00E+00	3.59E-06	0.00E+00	1.02E-09	3.59E-06	0.0543%
86	ARS H-1 (Vet Meta Res)	4.16E-11	1.00E-09	7.42E-09	1.82E-09	1.70E-10	3.79E-11	1.51E-09	0.00E+00	4.17E-10	0.00E+00	1.37E-07	0.00E+00	7.09E-11	1.37E-07	0.0021%
87	Serology4	2.65E-09	2.12E-08	9.05E-09	6.14E-08	2.84E-08	6.41E-10	6.85E-08	0.00E+00	2.63E-08	0.00E+00	5.15E-06	0.00E+00	1.47E-09	5.15E-06	0.0779%
88	ARS R-1	1.41E-10	1.13E-09	4.81E-10	3.28E-09	1.51E-09	3.40E-11	3.65E-09	0.00E+00	1.40E-09	0.00E+00	2.76E-07	0.00E+00	7.83E-11	2.76E-07	0.0042%
89	ARS R-2	1.91E-09	1.53E-08	6.53E-09	4.45E-08	2.05E-08	4.62E-10	4.95E-08	0.00E+00	1.90E-08	0.00E+00	3.73E-06	0.00E+00	1.06E-09	3.73E-06	0.0564%
90	Center For Comparative Medicine	1.24E-09	9.89E-09	4.21E-09	2.87E-08	1.33E-08	2.98E-10	3.20E-08	0.00E+00	1.23E-08	0.00E+00	2.41E-06	0.00E+00	6.86E-10	2.41E-06	0.0365%
91	Primate Center	7.05E-10	5.63E-09	2.40E-09	1.64E-08	7.57E-09	1.70E-10	1.82E-08	0.00E+00	7.00E-09	0.00E+00	1.37E-06	0.00E+00	3.91E-10	1.37E-06	0.0207%
92	Temporary Building 184	2.36E-10	1.89E-09	8.08E-10	5.48E-09	2.54E-09	5.71E-11	6.11E-09	0.00E+00	2.34E-09	0.00E+00	4.61E-07	0.00E+00	1.31E-10	4.61E-07	0.0070%
93	Temporary Building 160	1.05E-10	8.43E-10	3.59E-10	2.45E-09	1.13E-09	2.54E-11	2.72E-09	0.00E+00	1.05E-09	0.00E+00	2.05E-07	0.00E+00	5.84E-11	2.05E-07	0.0031%
94	APCARU	1.60E-10	1.28E-09	5.45E-10	3.71E-09	1.71E-09	3.85E-11	4.13E-09	0.00E+00	1.59E-09	0.00E+00	3.11E-07	0.00E+00	8.86E-11	3.11E-07	0.0047%
95	Ecology Lab (Aquadic Bio in bldg DB)	8.58E-10	6.86E-09	2.93E-09	1.99E-08	9.19E-09	2.07E-10	2.22E-08	0.00E+00	8.54E-09	0.00E+00	1.68E-06	0.00E+00	4.76E-10	1.68E-06	0.0254%
96	Temporary Building 1	3.72E-10	2.97E-09	1.27E-09	8.62E-09	3.97E-09	9.60E-11	9.60E-09	0.00E+00	3.97E-09	0.00E+00	7.25E-07	0.00E+00	2.06E-10	7.25E-07	0.0110%
97	ITEH Cellular Biology	1.71E-09	1.36E-08	5.84E-09	3.97E-08	1.83E-08	4.12E-10	4.42E-08	0.00E+00	1.70E-08	0.00E+00	3.33E-06	0.00E+00	9.47E-10	3.33E-06	0.0504%
98	ITEH Pathology Clinic	1.60E-09	1.28E-08	5.45E-09	3.71E-08	1.72E-08	3.86E-10	4.14E-08	0.00E+00	1.59E-08	0.00E+00	3.12E-06	0.00E+00	8.86E-10	3.12E-06	0.0472%
99	ARS DL-10/Field Shelter 5; Bovine Shed (Luekemia Lab)	6.12E-10	4.90E-09	2.09E-09	1.42E-08	6.57E-09	1.48E-10	1.58E-08	0.00E+00	6.09E-09	0.00E+00	1.19E-06	0.00E+00	3.39E-10	1.19E-06	0.0180%
100	Cole Fac A	2.41E-09	1.93E-08	8.22E-09	5.59E-08	2.59E-08	5.81E-10	6.23E-08	0.00E+00	2.39E-08	0.00E+00	4.68E-06	0.00E+00	1.34E-09	4.68E-06	0.0708%
101	Cole Fac B	2.05E-09	1.63E-08	6.97E-09	4.75E-08	2.19E-08	4.92E-10	5.28E-08	0.00E+00	2.03E-08	0.00E+00	3.98E-06	0.00E+00	1.13E-09	3.98E-06	0.0602%
102	Cole Fac C	3.02E-09	2.42E-08	1.03E-08	7.02E-08	3.24E-08	7.29E-10	7.82E-08	0.00E+00	3.00E-08	0.00E+00	5.89E-06	0.00E+00	1.68E-09	5.89E-06	0.0891%
103	TB 31	3.31E-10	2.65E-09	1.13E-09	7.70E-09	3.56E-09	7.99E-11	8.58E-09	0.00E+00	3.30E-09	0.00E+00	6.47E-07	0.00E+00	1.84E-10	6.47E-07	0.0098%
104	TB 33	1.89E-09	1.51E-08	6.44E-09	4.38E-08	2.03E-08	4.55E-10	4.89E-08	0.00E+00	1.88E-08	0.00E+00	3.69E-06	0.00E+00	1.05E-09	3.69E-06	0.0558%
105	TB 164	3.47E-09	2.77E-08	1.18E-08	8.06E-08	3.72E-08	8.36E-10	8.98E-08	0.00E+00	3.46E-08	0.00E+00	6.76E-06	0.00E+00	1.92E-09	6.76E-06	0.1023%
106	TB 165	3.42E-09	2.73E-08	1.17E-08	7.93E-08	3.66E-08	8.24E-10	8.83E-08	0.00E+00	3.40E-08	0.00E+00	6.67E-06	0.00E+00	1.89E-09	6.67E-06	0.1009%
107	TB 205	3.93E-09	3.14E-08	1.34E-08	9.12E-08	4.22E-08	9.48E-10	1.02E-07	0.00E+00	3.91E-08	0.00E+00	7.66E-06	0.00E+00	2.18E-09	7.66E-06	0.1159%
108	HH1	8.56E-10	6.84E-09	2.92E-09	1.99E-08	9.17E-09	2.07E-10	2.21E-08	0.00E+00	8.51E-09	0.00E+00	1.67E-06	0.00E+00	4.74E-10	1.67E-06	0.0253%
109	HH2	3.48E-09	2.79E-08	1.19E-08	8.09E-08	3.73E-08	8.41E-10	9.01E-08	0.00E+00	3.47E-08	0.00E+00	6.79E-06	0.00E+00	1.93E-09	6.79E-06	0.1027%
110	HH3	7.64E-10	6.10E-09	2.61E-09	1.77E-08	8.18E-09	1.84E-10	1.97E-08	0.00E+00	7.59E-09	0.00E+00	1.49E-06	0.00E+00	4.23E-10	1.49E-06	0.0225%
111	HH6	8.58E-09	6.86E-08	2.92E-08	1.99E-07	9.21E-08	2.07E-09	2.22E-07	0.00E+00	8.54E-08	0.00E+00	1.67E-05	0.00E+00	4.76E-09	1.67E-05	0.2526%
112	Vet Med Teaching Hospital (VMTH)	6.21E-09	4.96E-08	2.12E-08	1.44E-07	6.64E-08	1.49E-09	1.60E-07	0.00E+00	6.17E-08	0.00E+00	1.21E-05	0.00E+00	3.43E-09	1.21E-05	0.1831%
113	ARS Iso Barn J bldg	1.26E-10	1.00E-09	4.29E-10	2.92E-09	1.35E-09	3.03E-11	3.25E-09	0.00E+00	1.25E-09	0.00E+00	2.45E-07	0.00E+00	6.98E-11	2.45E-07	0.0037%
114	ITEH Animal Housing-2	6.03E-10	1.45E-08	1.08E-07	2.64E-08	2.47E-09	5.48E-10	2.19E-08	0.00E+00	6.03E-09	0.00E+00	1.99E-06	0.00E+00	1.03E-09	1.99E-06	0.0301%
115	LEHR Lab and Office	6.98E-10	1.68E-08	1.25E-07	3.05E-08	2.86E-09	6.33E-10	2.53E-08	0.00E+00	6.98E-09	0.00E+00	2.30E-06	0.00E+00	1.19E-09	2.30E-06	0.0348%
116	ITEH Toxic Pollutant Lab	5.70E-10	1.37E-08	1.02E-07	2.49E-08	2.33E-09	5.17E-10	2.06E-08	0.00E+00	5.69E-09	0.00E+00	1.88E-06	0.00E+00	9.68E-10	1.88E-06	0.0284%
117	Aqua weed lab/Aq Tox Shelter 5	8.28E-10	6.62E-09	2.83E-09	1.92E-08	8.89E-09	2.14E-08	1.92E-08	0.00E+00	8.24E-09	0.00E+00	1.61E-06	0.00E+00	4.60E-10	1.61E-06	0.0244%
118	Bee Biology	1.21E-10	9.64E-10	4.12E-10	2.81E-09	1.3										

Chronic HI for the MEIW at Grid Receptor # 2045 by Source
Corner of Russel Blvd. and Anderson Road - Rite Aid

Source No.	Source Identification	Cardiovascular system	Central Nervous System	Bone	Develop- mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
184	No Permit Old Fire Station	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.26E-06	0.00E+00	0.00E+00	2.26E-06	0.0342%
185	P-29-96(a) Physical Plant	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.35E-06	0.00E+00	0.00E+00	7.35E-06	0.1112%
186	P-120-01 Plant Envir Sci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.04E-06	0.00E+00	0.00E+00	6.04E-06	0.0914%
187	P-50-99(a) Port Gen # 1	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.39E-06	0.00E+00	0.00E+00	8.40E-06	0.1269%
188	No Permit Port Gen # 14	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.86E-05	0.00E+00	0.00E+00	4.86E-05	0.7352%
189	P-51-99(a) Port Gen # 2	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.36E-06	0.00E+00	0.00E+00	9.36E-06	0.1416%
190	P-52-99(a) Port Gen # 3	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.39E-07	0.00E+00	0.00E+00	5.39E-07	0.0082%
191	P-86-01 Port Gen # 7	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.82E-06	0.00E+00	0.00E+00	1.82E-06	0.0275%
192	P-87-01 Port Gen # 8	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.65E-06	0.00E+00	0.00E+00	1.65E-06	0.0250%
193	P-49-07 Pri Animal Hous # 1	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.57E-07	0.00E+00	0.00E+00	1.57E-07	0.0024%
194	P-31-98 Primate Animal	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.98E-07	0.00E+00	0.00E+00	1.98E-07	0.0030%
195	P-32-98 Primate CCM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.80E-07	0.00E+00	0.00E+00	2.80E-07	0.0042%
196	P-69-96(a) Primate Freezers	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.77E-08	0.00E+00	0.00E+00	8.77E-08	0.0013%
197	P-102-94(a) Primate Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.77E-07	0.00E+00	0.00E+00	7.77E-07	0.0118%
198	P-15-98 Primate Quarantine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.28E-07	0.00E+00	0.00E+00	3.28E-07	0.0050%
199	No Permit Primate Sew Life Sta	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.69E-08	0.00E+00	0.00E+00	9.69E-08	0.0015%
200	P-16-98 Primate TB 184	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.99E-07	0.00E+00	0.00E+00	3.99E-07	0.0060%
201	P-108-01 Primate TB North # 5	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.46E-07	0.00E+00	0.00E+00	1.46E-07	0.0022%
202	P-109-01 Primate TB South # 6	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.54E-07	0.00E+00	0.00E+00	1.54E-07	0.0023%
203	P-99-94(a) Quad Parking	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.70E-06	0.00E+00	0.00E+00	1.70E-06	0.0257%
204	P-93-94(a) Rec Hall	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.66E-05	0.00E+00	0.00E+00	1.66E-05	0.2511%
205	P-111-95(a) Schl of Med Neurosci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.33E-07	0.00E+00	0.00E+00	8.33E-07	0.0126%
206	P-123-01 Schl of Med Neurosci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.29E-07	0.00E+00	0.00E+00	9.29E-07	0.0141%
207	P-15-04 Science Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.27E-06	0.00E+00	0.00E+00	3.27E-06	0.0495%
208	P-74-05 Segundo Dinning	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.69E-05	0.00E+00	0.00E+00	7.69E-05	1.1634%
209	P-126-95(a) Social Sci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.91E-06	0.00E+00	0.00E+00	3.91E-06	0.0592%
210	P-17-02 South Parking	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.18E-06	0.00E+00	0.00E+00	4.18E-06	0.0632%
211	P-92-94(a) Storm Lift # 4	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.36E-05	0.00E+00	0.00E+00	1.36E-05	0.2057%
212	C-09-129 Storm Lift #4 (new)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
213	P-71-00 Targeted genomics	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.33E-06	0.00E+00	0.00E+00	2.33E-06	0.0352%
214	P-111-01 Tele Comm.	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.28E-06	0.00E+00	0.00E+00	7.28E-06	0.1101%
215	P-91-94(a) Thurman Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.57E-05	0.00E+00	0.00E+00	1.57E-05	0.2375%
216	P-100-94(a) Toxic Pollutant	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.91E-06	0.00E+00	0.00E+00	6.91E-06	0.1045%
217	P-17-09 TURF	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.25E-09	0.00E+00	0.00E+00	9.25E-09	0.0001%
218	P-121-03 Tupper Load Dock	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.53E-06	0.00E+00	0.00E+00	6.53E-06	0.0988%
219	P-209-95(a) Util Well 6A	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.95E-06	0.00E+00	0.00E+00	7.95E-06	0.1203%
220	P-07-01 Vega Crops	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.81E-06	0.00E+00	0.00E+00	4.81E-06	0.0728%
221	P-63-03 Vet Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.70E-06	0.00E+00	0.00E+00	1.70E-06	0.0257%
222	P-52-07 Vet Med 3A	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.61E-06	0.00E+00	0.00E+00	4.61E-06	0.0697%
223	P-53-07 Vet Med 3A	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.38E-06	0.00E+00	0.00E+00	3.38E-06	0.0511%
224	P-59-05 Watershed Sic	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.36E-06	0.00E+00	0.00E+00	2.36E-06	0.0357%
225	P-38-05 West Entry Park	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.98E-06	0.00E+00	0.00E+00	9.98E-06	0.1510%
226	P-96-94(a) WEPT Influent	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.47E-05	0.00E+00	0.00E+00	5.47E-05	0.8275%
227	P-88-99 WEPT South	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.39E-06	0.00E+00	0.00E+00	6.39E-06	0.0967%
228	Landfill	4.07E-08	3.76E-07	0.00E+00	3.09E-07	3.27E-09	8.26E-09	2.46E-07	0.00E+00	3.51E-07	4.26E-07	2.70E-06	0.00E+00	3.33E-08	2.70E-06	0.0408%
229	Landfill	5.00E-08	4.63E-07	0.00E+00	3.80E-07	4.03E-09	1.02E-08	3.03E-07	0.00E+00	4.32E-07	5.24E-09	3.33E-06	0.00E+00	4.10E-08	3.33E-06	0.0504%
230	Landfill	6.30E-08	5.83E-07	0.00E+00	4.78E-07	5.07E-09	1.28E-08	3.81E-07	0.00E+00	5.44E-07	6.60E-09	4.19E-06	0.00E+00	5.16E-08	4.19E-06	0.0634%
231	Landfill	7.33E-08	6.78E-07	0.00E+00	5.57E-07	5.90E-09	1.49E-08	4.44E-07	0.00E+00	6.34E-07	7.68E-09	4.88E-06	0.00E+00	6.01E-08	4.88E-06	0.0738%
232	Waste Water Treatment Plant	1.08E-06	4.85E-05	0.00E+00	3.20E-05	2.03E-10	0.00E+00	9.18E-06	0.00E+00	9.18E-06	0.00E+00	5.55E-05	0.00E+00	8.46E-09	5.55E-05	0.8396%
233	Grounds Above-ground Storage Tank	0.00E+00	4.96E-08	0.00E+00	4.82E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.57E-09	0.00E+00	4.11E-08	4.96E-08	0.0001%
234	Fleet Services Underground Storage Tank	0.00E+00	3.35E-06	0.00E+00	3.25E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.11E-07	0.00E+00	2.77E-06	3.35E-06	0.0077%
235	Primate Center Gasoline AST	0.00E+00	3.28E-10	0.00E+00	3.18E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.01E-11	0.00E+00	2.72E-10	3.28E-10	0.0000%
236	Agricultural Services AST	0.00E+00	1.15E-07	0.00E+00	1.12E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.76E-08	0.00E+00	9.56E-08	1.15E-07	0.0003%
237	Plant Pathology Storage Tank	0.00E+00	5.95E-10	0.00E+00	5.79E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.07E-11	0.00E+00	4.94E-10	5.95E-10	0.0000%
238	Pomology Above Ground Storage Tank	0.00E+00	4.92E-10	0.00E+00	4.78E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.50E-11	0.00E+00	4.08E-10	4.92E-10	0.0000%
239	Airport Above Ground Storage Tank	0.00E+00	3.84E-07	0.00E+00	3.73E-07	0.00E+00										

Acute HI for MEIW at Grid Receptor # 1910 by Chemical
Northeast of the Primate Center at the Grace Valley Christian Academy

Chemical No.	Chemical	Cardiovascular system	Central Nervous System	Bone	Develop-mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
1	Diesel engine exhaust, particulate matter (Diesel PM)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
2	Formaldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.40E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.40E-02	91.2621%
3	Benzo[a]pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
4	Dibenz[a,h]anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
5	Carbon tetrachloride	0.00E+00	4.91E-05	0.00E+00	4.91E-05	0.00E+00	0.00E+00	4.91E-05	0.00E+00	0.00E+00	4.91E-05	0.00E+00	0.00E+00	0.00E+00	4.91E-05	0.0000%
6	Benz[a]anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
7	Methanol	0.00E+00	5.46E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.46E-04	0.0000%
8	Isopropyl alcohol	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.66E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.66E-03	0.00E+00	0.00E+00	1.66E-03	1.6117%
9	Chloroform	0.00E+00	1.26E-02	0.00E+00	1.26E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.26E-02	0.00E+00	0.00E+00	0.00E+00	1.26E-02	0.0000%
10	Dimethyl formamide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
11	Benzene	0.00E+00	0.00E+00	0.00E+00	1.69E-04	0.00E+00	0.00E+00	0.00E+00	1.69E-04	0.00E+00	1.69E-04	0.00E+00	0.00E+00	1.69E-04	1.69E-04	0.0000%
12	Methyl chloroform [1,1,1-TCA]	0.00E+00	7.36E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.36E-07	0.0000%
13	Ethyl chloride [Chloroethane]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
14	Vinyl chloride	0.00E+00	1.38E-06	0.00E+00	0.00E+00	0.00E+00	1.38E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.38E-06	0.00E+00	0.00E+00	1.38E-06	0.0013%
15	Acetaldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.98E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.98E-05	0.00E+00	0.00E+00	9.98E-05	0.0969%
16	Methylene chloride [Dichloromethane]	0.00E+00	2.32E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.32E-04	0.0000%
17	Carbon disulfide	0.00E+00	3.86E-06	0.00E+00	3.86E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.86E-06	0.00E+00	0.00E+00	0.00E+00	3.86E-06	0.0000%
18	1,1-Dichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
19	Vinylidene chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
20	1,2-Dichloropropane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
21	Methyl ethyl ketone [2-Butanone]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.23E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.23E-05	0.00E+00	0.00E+00	2.23E-05	0.0217%
22	Trichloroethylene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
23	Acrylamide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
24	1,1,2,2-Tetrachloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
25	Naphthalene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
26	Ethyl benzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
27	p-Dichlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
28	Ethylene dibromide [EDB]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
29	1,3-Butadiene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
30	Acrolein	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.03E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.03E-03	0.00E+00	0.00E+00	4.03E-03	3.9126%
31	Ethylene dichloride [EDC]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
32	Acrylonitrile	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
33	Toluene	0.00E+00	8.38E-05	0.00E+00	8.38E-05	0.00E+00	8.38E-05	0.00E+00	0.00E+00	0.00E+00	8.38E-05	8.38E-05	0.00E+00	0.00E+00	8.38E-05	0.0814%
34	Chlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
35	Hexane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
36	Glutaraldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
37	Propylene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
38	Triethylamine	0.00E+00	1.17E-05	0.00E+00	0.00E+00	0.00E+00	1.17E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.17E-05	0.0114%
39	1,4-Dioxane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.86E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.86E-05	0.00E+00	0.00E+00	2.86E-05	0.0278%
40	Perchloroethylene [Tetrachloroethene]	0.00E+00	1.82E-05	0.00E+00	0.00E+00	0.00E+00	1.82E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.82E-05	0.00E+00	0.00E+00	1.82E-05	0.0177%
41	Indeno[1,2,3-cd]pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
42	Benzo[b]fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
43	Benzo[k]fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
44	Chrysene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
45	Hydrazine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
46	Xylenes (mixed)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.64E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.64E-05	0.00E+00	0.00E+00	8.64E-05	0.0839%
47	2,3,7,8-Tetrachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
48	1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
49	Lead	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
50	Mercury	0.00E+00	8.08E-05	0.00E+00	8.08E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.08E-05	0.0000%
51	Hydrochloric acid	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.75E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.75E-03	0.00E+00	0.00E+00	1.75E-03	1.6990%
52	Hydrogen fluoride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.01E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.01E-03	0.00E+00	0.00E+00	1.01E-03	0.9806%

Acute HI for MEIW at Grid Receptor # 1910 by Chemical
 Northeast of the Primate Center at the Grace Valley Christian Academy

Chemical No.	Chemical	Cardiovascular system	Central Nervous System	Bone	Develop-mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
53	Nitric acid	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.75E-02	0.00E+00	0.00E+00	1.75E-02	0.0000%
54	Hydrogen sulfide	0.00E+00	1.58E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.58E-02	0.0000%
55	Phosphine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
56	Chromium, hexavalent (& compounds)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
57	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
58	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
59	1,2,3,4,6,7,8,9-Octachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
60	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
61	1,2,3,7,8-Pentachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
62	2,3,7,8-Tetrachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
63	1,2,3,4,7,8,9-Heptachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
64	2,3,4,7,8-Pentachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
65	1,2,3,7,8-Pentachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
66	1,2,3,6,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
67	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
68	2,3,4,6,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
69	1,2,3,4,6,7,8-Heptachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
70	1,2,3,4,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
71	1,2,3,7,8,9-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
SUM		0.00E+00	2.94E-02	0.00E+00	1.30E-02	0.00E+00	1.03E-01	4.91E-05	1.69E-04	0.00E+00	1.29E-02	2.62E-02	0.00E+00	1.69E-04	1.03E-01	100.0000%

Acute HI for the MEIW at Grid Receptor # 1910 by Source
 Northeast of the Primate Center at the Grace Valley Christian Academy

Source No.	Source Identification	Cardiovascular system	Central Nervous System	Bone	Developmental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
1	Central Heating and Cooling Plant Boiler #1	0.00E+00	0.00E+00	0.00E+00	3.98E-07	0.00E+00	4.84E-04	0.00E+00	3.98E-07	0.00E+00	3.98E-07	2.27E-06	0.00E+00	3.98E-07	4.84E-04	0.4699%
2	Central Heating and Cooling Plant Boiler #2	0.00E+00	0.00E+00	0.00E+00	3.95E-07	0.00E+00	4.80E-04	0.00E+00	3.95E-07	0.00E+00	3.95E-07	2.25E-06	0.00E+00	3.95E-07	4.80E-04	0.4660%
3	Central Heating and Cooling Plant Boiler #3	0.00E+00	0.00E+00	0.00E+00	4.18E-07	0.00E+00	5.10E-04	0.00E+00	4.18E-07	0.00E+00	4.18E-07	2.38E-06	0.00E+00	4.18E-07	5.10E-04	0.4951%
4	Central Heating and Cooling Plant Boiler #4, Natural Gas	0.00E+00	0.00E+00	0.00E+00	6.66E-07	0.00E+00	8.11E-04	0.00E+00	6.66E-07	0.00E+00	6.66E-07	3.80E-06	0.00E+00	6.66E-07	8.11E-04	0.7874%
5	Central Heating and Cooling Plant Boiler #4, Diesel	0.00E+00	6.12E-08	0.00E+00	3.01E-06	0.00E+00	9.59E-03	0.00E+00	2.95E-06	0.00E+00	3.01E-06	1.64E-07	0.00E+00	2.95E-06	9.59E-03	9.3107%
6	Primate Center Boiler No 1 Natural Gas	0.00E+00	0.00E+00	0.00E+00	1.69E-07	0.00E+00	2.05E-04	0.00E+00	1.69E-07	0.00E+00	1.69E-07	9.62E-07	0.00E+00	1.69E-07	2.05E-04	0.1990%
7	Primate Center Boiler No 2 Natural Gas	0.00E+00	0.00E+00	0.00E+00	1.80E-07	0.00E+00	2.19E-04	0.00E+00	1.80E-07	0.00E+00	1.80E-07	1.03E-06	0.00E+00	1.80E-07	2.19E-04	0.2126%
8	Primate Center Boiler No 2 Landfill Gas	0.00E+00	4.41E-05	0.00E+00	2.77E-05	0.00E+00	6.42E-02	3.65E-10	6.48E-08	0.00E+00	1.51E-07	4.88E-03	0.00E+00	6.48E-08	6.42E-02	62.3301%
9	Landfill Flare	0.00E+00	5.05E-07	0.00E+00	2.67E-07	0.00E+00	6.13E-04	1.75E-11	9.28E-10	0.00E+00	3.08E-09	4.66E-05	0.00E+00	9.28E-10	6.13E-04	0.5951%
10	Incinerator	0.00E+00	0.00E+00	0.00E+00	1.47E-10	0.00E+00	1.81E-04	0.00E+00	1.47E-10	0.00E+00	1.47E-10	1.81E-04	0.00E+00	1.47E-10	1.81E-04	0.1757%
11	ARS J-1 (H001)	0.00E+00	0.00E+00	0.00E+00	6.06E-08	0.00E+00	7.38E-05	0.00E+00	6.06E-08	0.00E+00	6.06E-08	3.45E-07	0.00E+00	6.06E-08	7.38E-05	0.0717%
12	ARS J-1 CAAN 3840 - 4 boilers	0.00E+00	0.00E+00	0.00E+00	7.84E-08	0.00E+00	9.58E-05	0.00E+00	7.84E-08	0.00E+00	7.84E-08	4.47E-07	0.00E+00	7.84E-08	9.58E-05	0.0930%
13	ARS K-2 Co-located 2 stacks	0.00E+00	0.00E+00	0.00E+00	1.15E-07	0.00E+00	1.40E-04	0.00E+00	1.15E-07	0.00E+00	1.15E-07	6.57E-07	0.00E+00	1.15E-07	1.40E-04	0.1359%
14	ARS K-2 (H040)	0.00E+00	0.00E+00	0.00E+00	3.39E-08	0.00E+00	4.13E-05	0.00E+00	3.39E-08	0.00E+00	3.39E-08	1.92E-07	0.00E+00	3.39E-08	4.13E-05	0.0401%
15	Contained Research	0.00E+00	0.00E+00	0.00E+00	3.33E-07	0.00E+00	4.05E-04	0.00E+00	3.33E-07	0.00E+00	3.33E-07	1.90E-06	0.00E+00	3.33E-07	4.05E-04	0.3932%
16	Environmental Horticulture K-1	0.00E+00	0.00E+00	0.00E+00	1.60E-07	0.00E+00	1.95E-04	0.00E+00	1.60E-07	0.00E+00	1.60E-07	9.10E-07	0.00E+00	1.60E-07	1.95E-04	0.1893%
17	Environmental Horticulture K-2	0.00E+00	0.00E+00	0.00E+00	1.11E-07	0.00E+00	1.36E-04	0.00E+00	1.11E-07	0.00E+00	1.11E-07	6.33E-07	0.00E+00	1.11E-07	1.36E-04	0.1320%
18	Environmental Services Facility A	0.00E+00	0.00E+00	0.00E+00	5.62E-08	0.00E+00	6.85E-05	0.00E+00	5.62E-08	0.00E+00	5.62E-08	3.20E-07	0.00E+00	5.62E-08	6.85E-05	0.0665%
19	Environmentatl Services Facility (3 per stack)	0.00E+00	0.00E+00	0.00E+00	2.94E-08	0.00E+00	3.58E-05	0.00E+00	2.94E-08	0.00E+00	2.94E-08	1.68E-07	0.00E+00	2.94E-08	3.58E-05	0.0348%
20	Genome Launch Facility (plant reproduction)	0.00E+00	0.00E+00	0.00E+00	9.94E-08	0.00E+00	1.21E-04	0.00E+00	9.94E-08	0.00E+00	9.94E-08	5.66E-07	0.00E+00	9.94E-08	1.21E-04	0.1175%
21	Equine Analytical Chemistry Lab	0.00E+00	0.00E+00	0.00E+00	6.15E-08	0.00E+00	7.49E-05	0.00E+00	6.15E-08	0.00E+00	6.15E-08	3.51E-07	0.00E+00	6.15E-08	7.49E-05	0.0727%
22	Housing - Castillian DC	0.00E+00	0.00E+00	0.00E+00	2.22E-08	0.00E+00	2.71E-05	0.00E+00	2.22E-08	0.00E+00	2.22E-08	1.26E-07	0.00E+00	2.22E-08	2.71E-05	0.0263%
23	Housing - Castillian DC	0.00E+00	0.00E+00	0.00E+00	8.14E-08	0.00E+00	9.90E-05	0.00E+00	8.14E-08	0.00E+00	8.14E-08	4.63E-07	0.00E+00	8.14E-08	9.90E-05	0.0961%
24	Comparative Medicine (Primate Center)	0.00E+00	0.00E+00	0.00E+00	1.66E-08	0.00E+00	2.02E-05	0.00E+00	1.66E-08	0.00E+00	1.66E-08	9.44E-08	0.00E+00	1.66E-08	2.02E-05	0.0196%
25	Contained Research	0.00E+00	0.00E+00	0.00E+00	1.59E-08	0.00E+00	1.94E-05	0.00E+00	1.59E-08	0.00E+00	1.59E-08	9.04E-08	0.00E+00	1.59E-08	1.94E-05	0.0188%
26	Institute of Ecology - West Campus	0.00E+00	0.00E+00	0.00E+00	6.40E-08	0.00E+00	7.79E-05	0.00E+00	6.40E-08	0.00E+00	6.40E-08	3.63E-07	0.00E+00	6.40E-08	7.79E-05	0.0756%
27	ITEH Geriatrics - cage wash inside co-located 3 stacks	0.00E+00	0.00E+00	0.00E+00	1.78E-08	0.00E+00	2.16E-05	0.00E+00	1.78E-08	0.00E+00	1.78E-08	1.01E-07	0.00E+00	1.78E-08	2.16E-05	0.0210%
28	ITEH Geriatrics Cagewash - outside - co-locate 3 stacks	0.00E+00	0.00E+00	0.00E+00	1.97E-08	0.00E+00	2.39E-05	0.00E+00	1.97E-08	0.00E+00	1.97E-08	1.11E-07	0.00E+00	1.97E-08	2.39E-05	0.0232%
29	Mondavi Ctr for Performing Arts - 2 boilers	0.00E+00	0.00E+00	0.00E+00	2.23E-08	0.00E+00	2.72E-05	0.00E+00	2.23E-08	0.00E+00	2.23E-08	1.27E-07	0.00E+00	2.23E-08	2.72E-05	0.0264%
30	Mondavi Ctr for Performing Arts	0.00E+00	0.00E+00	0.00E+00	1.20E-08	0.00E+00	1.46E-05	0.00E+00	1.20E-08	0.00E+00	1.20E-08	6.82E-08	0.00E+00	1.20E-08	1.46E-05	0.0142%
31	Rec Pool	0.00E+00	0.00E+00	0.00E+00	1.09E-07	0.00E+00	1.34E-04	0.00E+00	1.09E-07	0.00E+00	1.09E-07	6.25E-07	0.00E+00	1.09E-07	1.34E-04	0.1301%
32	Thoreau Hall - 2 stacks co-located	0.00E+00	0.00E+00	0.00E+00	1.92E-08	0.00E+00	2.33E-05	0.00E+00	1.92E-08	0.00E+00	1.92E-08	1.09E-07	0.00E+00	1.92E-08	2.33E-05	0.0226%
33	Air Stripper	0.00E+00	5.64E-04	0.00E+00	5.64E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.64E-04	0.00E+00	0.00E+00	5.64E-04	5.64E-04	0.0000%
34	In-well Stripper	0.00E+00	4.82E-05	0.00E+00	4.82E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.82E-05	0.00E+00	0.00E+00	4.82E-05	4.82E-05	0.0000%
35	Ground Water Treatment	0.00E+00	7.65E-05	0.00E+00	7.65E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.65E-05	0.00E+00	0.00E+00	7.65E-05	7.65E-05	0.0000%
36	Large Kiln	0.00E+00	2.20E-09	0.00E+00	2.60E-08	0.00E+00	9.16E-06	0.00E+00	2.38E-08	0.00E+00	2.60E-08	5.45E-06	0.00E+00	2.38E-08	9.16E-06	0.0089%
37	Raku Kiln	0.00E+00	1.71E-09	0.00E+00	2.01E-08	0.00E+00	7.09E-06	0.00E+00	1.84E-08	0.00E+00	2.01E-08	4.21E-06	0.00E+00	1.84E-08	7.09E-06	0.0069%
38	Foundry Kiln	0.00E+00	2.61E-09	0.00E+00	3.08E-08	0.00E+00	1.08E-05	0.00E+00	2.82E-08	0.00E+00	3.08E-08	6.44E-06	0.00E+00	2.82E-08	1.08E-05	0.0105%
39	Three Art Dept Kilns to roof vent	0.00E+00	4.42E-09	0.00E+00	5.25E-08	0.00E+00	1.84E-05	0.00E+00	4.80E-08	0.00E+00	5.25E-08	1.09E-05	0.00E+00	4.80E-08	1.84E-05	0.0179%
40	Storehouse/Bulk Receiving and Storage	0.00E+00	7.07E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.07E-07	0.0000%
41	Walnut Dryer	0.00E+00	9.22E-08	0.00E+00	1.08E-06	0.00E+00	3.82E-04	0.00E+00	9.93E-07	0.00E+00	1.08E-06	2.27E-04	0.00E+00	9.93E-07	3.82E-04	0.3709%
42	Temporary Building 187	0.00E+00	1.14E-05	0.00E+00	1.08E-05	0.00E+00	3.09E-05	6.30E-08	1.56E-08	0.00E+00	1.08E-05	3.52E-06	0.00E+00	1.56E-08	3.09E-05	0.0300%
43	Temporary Building 188	0.00E+00	9.17E-06	0.00E+00	8.66E-06	0.00E+00	2.49E-05	5.07E-08	1.26E-08	0.00E+00	8.66E-06	2.83E-06	0.00E+00	1.26E-08	2.49E-05	0.0242%
44	Veihmeyer	0.00E+00	7.42E-06	0.00E+00	6.06E-06	0.00E+00	3.09E-05	2.24E-07	4.98E-08	0.00E+00	6.06E-06	1.44E-04	0.00E+00	4.98E-08	1.44E-04	0.0300%
45	Enology	0.00E+00	1.18E-05	0.00E+00	1.11E-05	0.00E+00	3.23E-05	6.51E-08	1.62E-08	0.00E+00	1.11E-05	4.73E-05	0.00E+00	1.62E-08	4.73E-05	0.0314%
46	Wickson Hall	0.00E+00	2.13E-04	0.00E+00	2.01E-04	0.00E+00	5.82E-04	1.18E-06	2.93E-07	0.00E+00	2.01E-04	1.93E-03	0.00E+00	2.93E-07	1.93E-03	0.5650%
47	Hoagland	0.00E+00	1.75E-04	0.00E+00	1.65E-04	0.00E+00	4.73E-04	9.65E-07	2.40E-07	0.00E+00	1.65E-04	1.58E-04	0.00E+00	2.40E-07	4.73E-04	0.4592%
48	Mann Hall	0.00E+00	4.67E-05	0.00E+00	4.41E-05	0.00E+00	1.26E-04	2.57E-07	6.41E-08	0.00E+00	4.41E-05	6.01E-05	0.00E+00	6.41E-08	1.26E-04	0.1223%
49	Storer Hall	0.00E+00	8.20E-06	0.00E+00	7.75E-06	0.00E+00	2.22E-05	4.54E-08	1.13E-08	0.00E+00	7.75E-06	4.00E-05	0.00E+00	1.13E-08	4.00E-05	0.0216%
50	Hutchison Hall/Biological Sci Unit 2	0.00E+00	1.59E-04	0.00E+00	1.50E-04	0.00E+00	4.32E-04	8.81E-07	2.20E-07	0.00E+00	1.50E-04	2.83E-04	0.00E+00	2.20E-07	4.32E-04	0.4194%
51	Asmundson Hall	0.00E+00	9.52E-05	0.00E+00	8.99E-05	0.00E+00	2.58E-04	5.25E-07	1.31E-07	0.00E+00	8.99E-05	2.73E-04	0.00E+00	1.31E-07	2.73E-04	0.2505%
52	Robbins Hall	0.00E+00	9.19E-05	0.00E+00	8.69E-05	0.00E+00	2.50E-04	5.09E-07	1.26E-07	0.00E+00	8.69E-05	1.12E-04	0.00E+00	1.26E-07	2.50E-04	0.2427%
53	Temporary Building 202	0.00E+00	1.04E-05	0.00E+00	9.83E-06	0.00E+00	2.82E-05	5.75E-08	1.43E-08	0.00E+00	9.83E-06	3.22E-06	0.00E+00	1.43E-08	2.82E-05	0.0274%
54	Briggs Hall and Life Sciences	0.00E+00	6.49E-04	0.00E+00	6.14E-04	0.00E+00	1.76E-03	3.59E-06	8.92E-07	0.00E+00	6.14E-04	1.78E-03	0.00E+00	8.92E-07	1.78E-03	1.7087%
55	Temporary Building 194	0.00E+00	1.83E-05	0.00E+00	1.73E-05	0.00E+00	4.97E-05	1.01E-07	2.52E-08	0.00E+00	1.73E-05	1.32E-04	0.00E+00	2.52E-08	1.32E-04	0.0483%
56	Food Science	0.00E+00	1.79E-06	0.00E+00	1.69E-06	0.00E+00	4.87E-06	9.93E-09	2.47E-09	0.00E+00	1.69E-06	5.54E-07	0.00E+00	2.47E-09	4.87E-06	0.0047%
57																

Acute HI for the MEIW at Grid Receptor # 1910 by Source
Northeast of the Primate Center at the Grace Valley Christian Academy

Source No.	Source Identification	Cardiovascular system	Central Nervous System	Bone	Developmental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
62	Temporary Building 155	0.00E+00	8.57E-06	0.00E+00	8.09E-06	0.00E+00	2.32E-05	4.74E-08	1.18E-08	0.00E+00	8.09E-06	2.65E-06	0.00E+00	1.18E-08	2.32E-05	0.0225%
63	Temporary Building 156	0.00E+00	7.72E-06	0.00E+00	7.29E-06	0.00E+00	2.10E-05	4.28E-08	1.07E-08	0.00E+00	7.29E-06	2.39E-06	0.00E+00	1.07E-08	2.10E-05	0.0204%
64	Temporary Building 157	0.00E+00	6.77E-06	0.00E+00	6.40E-06	0.00E+00	1.84E-05	3.75E-08	9.30E-09	0.00E+00	6.40E-06	2.10E-06	0.00E+00	9.30E-09	1.84E-05	0.0179%
65	Temporary Building 151	0.00E+00	1.02E-05	0.00E+00	9.64E-06	0.00E+00	2.77E-05	5.63E-08	1.41E-08	0.00E+00	9.64E-06	3.16E-06	0.00E+00	1.41E-08	2.77E-05	0.0269%
66	Temporary Building 149	0.00E+00	7.72E-06	0.00E+00	7.29E-06	0.00E+00	2.10E-05	4.27E-08	1.06E-08	0.00E+00	7.29E-06	2.39E-06	0.00E+00	1.06E-08	2.10E-05	0.0204%
67	Temporary Building 153	0.00E+00	4.98E-06	0.00E+00	4.71E-06	0.00E+00	1.35E-05	2.75E-08	6.85E-09	0.00E+00	4.71E-06	1.54E-06	0.00E+00	6.85E-09	1.35E-05	0.0131%
68	Temporary Building 158	0.00E+00	4.82E-06	0.00E+00	4.55E-06	0.00E+00	1.31E-05	2.66E-08	6.62E-09	0.00E+00	4.55E-06	1.49E-06	0.00E+00	6.62E-09	1.31E-05	0.0127%
69	Engineering II	0.00E+00	4.22E-04	0.00E+00	3.99E-04	0.00E+00	1.00E-05	2.28E-07	3.74E-06	0.00E+00	3.99E-04	3.78E-04	0.00E+00	3.74E-06	4.22E-04	0.0097%
70	Walker Hall	0.00E+00	4.14E-06	0.00E+00	3.38E-06	0.00E+00	1.72E-05	1.25E-07	2.78E-08	0.00E+00	3.38E-06	8.42E-06	0.00E+00	2.78E-08	1.72E-05	0.0167%
71	Chemistry	0.00E+00	1.51E-03	0.00E+00	1.43E-03	0.00E+00	3.64E-05	8.20E-07	1.34E-05	0.00E+00	1.43E-03	1.22E-03	0.00E+00	1.34E-05	1.51E-03	0.0353%
72	Chemistry Annex	0.00E+00	9.64E-04	0.00E+00	9.12E-04	0.00E+00	2.30E-05	5.22E-07	8.56E-06	0.00E+00	9.12E-04	4.27E-04	0.00E+00	8.56E-06	9.64E-04	0.0223%
73	Bainer Hall	0.00E+00	6.73E-04	0.00E+00	6.37E-04	0.00E+00	1.62E-05	3.65E-07	5.98E-06	0.00E+00	6.37E-04	4.23E-04	0.00E+00	5.98E-06	6.73E-04	0.0157%
74	Crocker Hall	0.00E+00	1.66E-06	0.00E+00	1.36E-06	0.00E+00	6.91E-06	5.01E-08	1.11E-08	0.00E+00	1.36E-06	2.70E-05	0.00E+00	1.11E-08	2.70E-05	0.0067%
75	Academic Surge	0.00E+00	5.02E-05	0.00E+00	4.74E-05	0.00E+00	4.74E-05	2.78E-07	6.90E-08	0.00E+00	4.74E-05	6.86E-05	0.00E+00	6.90E-08	1.36E-04	0.1320%
76	Meyer Hall	0.00E+00	3.20E-04	0.00E+00	3.02E-04	0.00E+00	8.65E-04	1.77E-06	4.40E-07	0.00E+00	3.02E-04	1.20E-03	0.00E+00	4.40E-07	1.20E-03	0.8398%
77	Physics/Geology/Physics Unit 1	0.00E+00	9.25E-06	0.00E+00	7.56E-06	0.00E+00	3.86E-05	2.79E-07	6.22E-08	0.00E+00	7.56E-06	9.25E-04	0.00E+00	6.22E-08	9.25E-04	0.0375%
78	Environmental Horticulture	0.00E+00	6.24E-05	0.00E+00	5.89E-05	0.00E+00	1.69E-04	3.44E-07	8.56E-08	0.00E+00	5.89E-05	3.06E-05	0.00E+00	8.56E-08	1.69E-04	0.1641%
79	Thurman Hall	0.00E+00	7.46E-05	0.00E+00	7.05E-05	0.00E+00	2.03E-04	4.11E-07	1.02E-07	0.00E+00	7.05E-05	1.07E-04	0.00E+00	1.02E-07	2.03E-04	0.1971%
80	Maddy Hall	0.00E+00	1.71E-04	0.00E+00	1.62E-04	0.00E+00	4.64E-04	9.47E-07	2.35E-07	0.00E+00	1.62E-04	3.85E-04	0.00E+00	2.35E-07	4.64E-04	0.4505%
81	Tupper Hall	0.00E+00	4.88E-04	0.00E+00	4.61E-04	0.00E+00	1.33E-03	2.70E-06	6.71E-07	0.00E+00	4.61E-04	9.13E-04	0.00E+00	6.71E-07	1.33E-03	1.2913%
82	VET MED 2	0.00E+00	4.67E-05	0.00E+00	4.41E-05	0.00E+00	1.27E-04	2.59E-07	6.43E-08	0.00E+00	4.41E-05	7.33E-05	0.00E+00	6.43E-08	1.27E-04	0.1233%
83	Asmundson Annex	0.00E+00	5.44E-06	0.00E+00	5.13E-06	0.00E+00	1.47E-05	3.00E-08	7.48E-09	0.00E+00	5.13E-06	1.68E-06	0.00E+00	7.48E-09	1.47E-05	0.0143%
84	Young Hall	0.00E+00	2.98E-05	0.00E+00	2.81E-05	0.00E+00	8.07E-05	1.64E-07	4.10E-08	0.00E+00	2.81E-05	5.00E-05	0.00E+00	4.10E-08	8.07E-05	0.0783%
85	Temporary Building 9	0.00E+00	7.23E-06	0.00E+00	6.83E-06	0.00E+00	1.96E-05	4.00E-08	9.96E-09	0.00E+00	6.83E-06	2.24E-06	0.00E+00	9.96E-09	1.96E-05	0.0190%
86	ARS H-1 (Vet Meta Res)	0.00E+00	1.36E-07	0.00E+00	1.11E-07	0.00E+00	5.66E-07	4.09E-09	9.14E-10	0.00E+00	1.11E-07	2.76E-07	0.00E+00	9.14E-10	5.66E-07	0.0005%
87	Serology4	0.00E+00	1.52E-05	0.00E+00	1.43E-05	0.00E+00	4.12E-05	8.39E-08	2.09E-08	0.00E+00	1.43E-05	4.69E-06	0.00E+00	2.09E-08	4.12E-05	0.0400%
88	ARS R-1	0.00E+00	9.66E-07	0.00E+00	9.13E-07	0.00E+00	2.61E-06	5.35E-09	1.33E-09	0.00E+00	9.13E-07	2.98E-07	0.00E+00	1.33E-09	2.61E-06	0.0025%
89	ARS R-2	0.00E+00	1.31E-05	0.00E+00	1.23E-05	0.00E+00	3.55E-05	7.21E-08	1.80E-08	0.00E+00	1.23E-05	4.04E-06	0.00E+00	1.80E-08	3.55E-05	0.0345%
90	Center For Comparative Medicine	0.00E+00	4.11E-04	0.00E+00	3.88E-04	0.00E+00	1.11E-03	2.27E-06	5.65E-07	0.00E+00	3.88E-04	4.11E-04	0.00E+00	5.65E-07	1.11E-03	1.0777%
91	Primate Center	0.00E+00	1.81E-04	0.00E+00	1.71E-04	0.00E+00	4.92E-04	1.01E-06	2.50E-07	0.00E+00	1.71E-04	5.62E-05	0.00E+00	2.50E-07	4.92E-04	0.4777%
92	Temporary Building 184	0.00E+00	9.49E-05	0.00E+00	8.96E-05	0.00E+00	2.57E-04	5.24E-07	1.30E-07	0.00E+00	8.96E-05	2.93E-05	0.00E+00	1.30E-07	2.57E-04	0.2495%
93	Temporary Building 160	0.00E+00	1.44E-05	0.00E+00	1.36E-05	0.00E+00	3.91E-05	7.94E-08	1.98E-08	0.00E+00	1.36E-05	4.44E-06	0.00E+00	1.98E-08	3.91E-05	0.0380%
94	APCARU	0.00E+00	2.00E-05	0.00E+00	1.89E-05	0.00E+00	5.41E-05	1.10E-07	2.74E-08	0.00E+00	1.89E-05	6.16E-06	0.00E+00	2.74E-08	5.41E-05	0.0525%
95	Ecology Lab (Aquatic Bio in bldg DB)	0.00E+00	1.87E-05	0.00E+00	1.76E-05	0.00E+00	5.05E-05	1.03E-07	2.56E-08	0.00E+00	1.76E-05	1.91E-04	0.00E+00	2.56E-08	1.91E-04	0.0490%
96	Temporary Building 1	0.00E+00	6.02E-06	0.00E+00	5.69E-06	0.00E+00	1.63E-05	3.33E-08	8.30E-09	0.00E+00	5.69E-06	1.86E-06	0.00E+00	8.30E-09	1.63E-05	0.0158%
97	ITEH Cellular Biology	0.00E+00	1.07E-05	0.00E+00	1.01E-05	0.00E+00	2.90E-05	5.91E-08	1.47E-08	0.00E+00	1.01E-05	3.30E-06	0.00E+00	1.47E-08	2.90E-05	0.0282%
98	ITEH Pathology Clinic	0.00E+00	9.89E-06	0.00E+00	9.34E-06	0.00E+00	2.68E-05	5.45E-08	1.36E-08	0.00E+00	9.34E-06	3.05E-06	0.00E+00	1.36E-08	2.68E-05	0.0260%
99	ARS DL-10/Field Shelter 5; Bovine Shed (Luekemia Lab)	0.00E+00	4.52E-06	0.00E+00	4.27E-06	0.00E+00	1.23E-05	2.50E-08	6.23E-09	0.00E+00	4.27E-06	1.40E-06	0.00E+00	6.23E-09	1.23E-05	0.0119%
100	Cole Fac A	0.00E+00	9.56E-06	0.00E+00	9.03E-06	0.00E+00	2.60E-05	5.28E-08	1.31E-08	0.00E+00	9.03E-06	2.96E-06	0.00E+00	1.31E-08	2.60E-05	0.0252%
101	Cole Fac B	0.00E+00	8.03E-06	0.00E+00	7.59E-06	0.00E+00	2.18E-05	4.46E-08	1.10E-08	0.00E+00	7.59E-06	2.49E-06	0.00E+00	1.10E-08	2.18E-05	0.0212%
102	Cole Fac C	0.00E+00	1.31E-05	0.00E+00	1.24E-05	0.00E+00	3.57E-05	7.28E-08	1.81E-08	0.00E+00	1.24E-05	4.07E-06	0.00E+00	1.81E-08	3.57E-05	0.0347%
103	TB 31	0.00E+00	1.54E-06	0.00E+00	1.46E-06	0.00E+00	4.19E-06	8.53E-09	2.12E-09	0.00E+00	1.46E-06	4.76E-07	0.00E+00	2.12E-09	4.19E-06	0.0041%
104	TB 33	0.00E+00	8.47E-06	0.00E+00	8.00E-06	0.00E+00	2.29E-05	4.69E-08	1.16E-08	0.00E+00	8.00E-06	2.62E-06	0.00E+00	1.16E-08	2.29E-05	0.0222%
105	TB 164	0.00E+00	1.22E-05	0.00E+00	1.15E-05	0.00E+00	3.32E-05	6.79E-08	1.68E-08	0.00E+00	1.15E-05	8.10E-05	0.00E+00	1.68E-08	8.11E-05	0.0322%
106	TB 165	0.00E+00	1.21E-05	0.00E+00	1.14E-05	0.00E+00	3.29E-05	6.71E-08	1.67E-08	0.00E+00	1.14E-05	3.75E-06	0.00E+00	1.67E-08	3.29E-05	0.0319%
107	TB 205	0.00E+00	1.17E-05	0.00E+00	1.11E-05	0.00E+00	3.18E-05	6.46E-08	1.61E-08	0.00E+00	1.11E-05	3.32E-05	0.00E+00	1.61E-08	3.32E-05	0.0309%
108	HH1	0.00E+00	1.77E-06	0.00E+00	1.67E-06	0.00E+00	4.81E-06	9.80E-09	2.43E-09	0.00E+00	1.67E-06	5.48E-07	0.00E+00	2.43E-09	4.81E-06	0.0047%
109	HH2	0.00E+00	7.33E-06	0.00E+00	6.92E-06	0.00E+00	1.99E-05	4.06E-08	1.01E-08	0.00E+00	6.92E-06	2.26E-06	0.00E+00	1.01E-08	1.99E-05	0.0193%
110	HH3	0.00E+00	1.63E-06	0.00E+00	1.54E-06	0.00E+00	4.43E-06	9.04E-09	2.25E-09	0.00E+00	1.54E-06	5.05E-07	0.00E+00	2.25E-09	4.43E-06	0.0043%
111	HH6	0.00E+00	1.41E-05	0.00E+00	1.33E-05	0.00E+00	3.83E-05	7.81E-08	1.94E-08	0.00E+00	1.33E-05	1.39E-04	0.00E+00	1.94E-08	1.39E-04	0.0372%
112	Vet Med Teaching Hospital (VMTH)	0.00E+00	2.39E-05	0.00E+00	2.26E-05	0.00E+00	6.48E-05	1.32E-07	3.28E-08	0.00E+00	2.26E-05	7.38E-06	0.00E+00	3.28E-08	6.48E-05	0.0629%
113	ARS Iso Barn J bldg	0.00E+00	5.82E-07	0.00E+00	5.50E-07	0.00E+00	1.58E-06	3.23E-09	8.00E-10	0.00E+00	5.50E-07	1.80E-07	0.00E+00	8.00E-10	1.58E-06	0.0015%
114	ITEH Animal Housing-2	0.00E+00	2.35E-06	0.00E+00	1.92E-06	0.00E+00	9.81E-06	7.11E-08	1.58E-08	0.00E+00	1.92E-06	1.32E-05	0.00E+00	1.58E-08	1.32E-05	0.0095%
115	LEHR Lab and Office	0.00E+00	2.78E-06	0.00E+00	2.27E-06	0.00E+00	1.16E-05	8.39E-08	1.87E-08	0.00E+00	2.27E-06	5.66E-06	0.00E+00	1.87E-08	1.16E-05	0.0113%
116	ITEH Toxic Pollutant Lab	0.00E+00	2.15E-06	0.00E+00	1.76E-06	0.00E+00	8.96E-06	6.48E-08	1.44E-08	0.00E+00	1.76E-06	1.33E-04	0.00E+00	1.44E-08	1.33E-04	0.0087%
117	Aqua weed lab/Aq Tox Shelter 5	0.00E+00	2.06E-05	0.00E+00	1.95E-05	0.00E+00	5.59E-05	1.14E-07	2.83E-08	0.00E+00	1.95E-05	9.63E-05	0.00E+00	2.83E-08	9.63E-05	0.0543%
118	Bee Biology	0.00E+00	1.72E-05	0.00E+00	1.62E-05	0.00E+										

Acute HI for the MEIW at Grid Receptor # 1910 by Source
 Northeast of the Primate Center at the Grace Valley Christian Academy

Source No.	Source Identification	Cardiovascular system	Central Nervous System	Bone	Developmental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
184	No Permit Old Fire Station	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
185	P-29-96(a) Physical Plant	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
186	P-120-01 Plant Envir Sci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
187	P-50-99(a) Port Gen # 1	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
188	No Permit Port Gen # 14	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
189	P-51-99(a) Port Gen # 2	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
190	P-52-99(a) Port Gen # 3	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
191	P-86-01 Port Gen # 7	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
192	P-87-01 Port Gen # 8	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
193	P-49-07 Pri Animal Hous # 1	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
194	P-31-98 Primate Animal	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
195	P-32-98 Primate CCM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
196	P-69-96(a) Primate Freezers	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
197	P-102-94(a) Primate Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
198	P-15-98 Primate Quarantine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
199	No Permit Primate Sew Life Sta	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
200	P-16-98 Primate TB 184	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
201	P-108-01 Primate TB North # 5	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
202	P-109-01 Primate TB South # 6	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
203	P-99-94(a) Quad Parking	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
204	P-93-94(a) Rec Hall	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
205	P-111-95(a) Schl of Med Neurosci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
206	P-123-01 Schl of Med Neurosci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
207	P-15-04 Science Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
208	P-74-05 Segundo Dinning	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
209	P-126-95(a) Social Sci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
210	P-17-02 South Parking	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
211	P-92-94(a) Storm Lift # 4	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
212	C-09-129 Storm Lift #4 (new)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
213	P-71-00 Targeted genomics	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
214	P-111-01 Tele Comm.	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
215	P-91-94(a) Thurman Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
216	P-100-94(a) Toxic Pollutant	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
217	P-17-09 TURF	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
218	P-121-03 Tupper Load Dock	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
219	P-209-95(a) Util Well 6A	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
220	P-07-01 Vega Crops	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
221	P-63-03 Vet Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
222	P-52-07 Vet Med 3A	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
223	P-53-07 Vet Med 3A	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
224	P-59-05 Watershed Sic	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
225	P-38-05 West Entry Park	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
226	P-96-94(a) WEPT Influent	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
227	P-88-99 WEPT South	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
228	Landfill	0.00E+00	3.73E-03	0.00E+00	4.38E-05	0.00E+00	1.50E-04	4.16E-08	1.47E-05	0.00E+00	3.12E-05	1.50E-04	0.00E+00	1.47E-05	3.73E-03	0.1456%
229	Landfill	0.00E+00	3.96E-03	0.00E+00	4.65E-05	0.00E+00	1.59E-04	4.42E-08	1.56E-05	0.00E+00	3.32E-05	1.59E-04	0.00E+00	1.56E-05	3.96E-03	0.1544%
230	Landfill	0.00E+00	4.05E-03	0.00E+00	4.75E-05	0.00E+00	1.63E-04	4.51E-08	1.60E-05	0.00E+00	3.39E-05	1.63E-04	0.00E+00	1.60E-05	4.05E-03	0.1583%
231	Landfill	0.00E+00	4.03E-03	0.00E+00	4.73E-05	0.00E+00	1.62E-04	4.50E-08	1.59E-05	0.00E+00	3.38E-05	1.62E-04	0.00E+00	1.59E-05	4.03E-03	0.1573%
232	Waste Water Treatment Plant	0.00E+00	3.47E-04	0.00E+00	1.74E-04	0.00E+00	6.78E-05	0.00E+00	3.35E-08	0.00E+00	1.74E-04	6.78E-05	0.00E+00	3.35E-08	3.47E-04	0.0658%
233	Grounds Above-ground Storage Tank	0.00E+00	1.02E-09	0.00E+00	3.48E-08	0.00E+00	1.30E-09	0.00E+00	3.38E-08	0.00E+00	3.48E-08	1.30E-09	0.00E+00	3.38E-08	3.48E-08	0.0000%
234	Fleet Services Underground Storage Tank	0.00E+00	9.79E-08	0.00E+00	3.33E-06	0.00E+00	1.24E-07	0.00E+00	3.24E-06	0.00E+00	3.33E-06	1.24E-07	0.00E+00	3.24E-06	3.33E-06	0.0001%
235	Primate Center Gasoline AST	0.00E+00	5.64E-09	0.00E+00	1.92E-07	0.00E+00	7.17E-09	0.00E+00	1.86E-07	0.00E+00	1.92E-07	7.17E-09	0.00E+00	1.86E-07	1.92E-07	0.0000%
236	Agricultural Services AST	0.00E+00	2.24E-08	0.00E+00	7.61E-07	0.00E+00	2.84E-08	0.00E+00	7.38E-07	0.00E+00	7.61E-07	2.84E-08	0.00E+00	7.38E-07	7.61E-07	0.0000%
237	Plant Pathology Storage Tank	0.00E+00	5.46E-11	0.00E+00	1.86E-09	0.00E+00	6.94E-11	0.00E+00	1.80E-09	0.00E+00	1.86E-09	6.94E-11	0.00E+00	1.80E-09	1.86E-09	0.0000%
238	Pomology Above Ground Storage Tank	0.00E+00	1.96E-09	0.00E+00	6.68E-08	0.00E+00	2.49E-09	0.00E+00	6.48E-08	0.00E+00	6.68E-08	2.49E-09	0.00E+00	6.48E-08	6.68E-08	0.0000%

Cancer Risk for Maximum Sensitive Receptor - by Chemical
 Located at the the Woodland Clinic Medical Group

Chemical No.	Chemical	Inhalation	Dermal	Soil	Mother's Milk	Home grown Vegetables	Oral	Total	Percent of Total
1	Diesel engine exhaust, particulate matter (Diesel PM)	1.22E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.22E-06	62.8866%
2	Formaldehyde	6.90E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.90E-08	3.5567%
3	Benzo[a]pyrene	1.06E-10	1.41E-09	2.12E-10	0.00E+00	5.17E-09	6.80E-09	6.91E-09	0.3562%
4	Dibenz[a,h]anthracene	7.36E-11	3.18E-10	4.76E-11	0.00E+00	1.16E-09	1.53E-09	1.60E-09	0.0825%
5	Carbon tetrachloride	3.42E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.42E-08	1.7629%
6	Benz[a]anthracene	4.33E-11	5.76E-10	8.63E-11	0.00E+00	2.11E-09	2.77E-09	2.81E-09	0.1448%
7	Methanol	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
8	Isopropyl alcohol	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
9	Chloroform	8.87E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.87E-08	4.5722%
10	Dimethyl formamide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
11	Benzene	2.32E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.32E-08	1.1959%
12	Methyl chloroform [1,1,1-TCA]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
13	Ethyl chloride [Chloroethane]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
14	Vinyl chloride	1.75E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.75E-09	0.0902%
15	Acetaldehyde	4.95E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.95E-10	0.0255%
16	Methylene chloride [Dichloromethane]	2.57E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.57E-08	1.3247%
17	Carbon disulfide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
18	1,1-Dichloroethane	1.87E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.87E-11	0.0010%
19	Vinylidene chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
20	1,2-Dichloropropane	1.81E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.81E-11	0.0009%
21	Methyl ethyl ketone [2-Butanone]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
22	Trichloroethylene	2.31E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.31E-10	0.0119%
23	Acrylamide	9.74E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.74E-08	5.0206%
24	1,1,2,2-Tetrachloroethane	5.28E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.28E-10	0.0272%
25	Naphthalene	2.66E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.66E-09	0.1371%
26	Ethyl benzene	6.21E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.21E-11	0.0032%
27	p-Dichlorobenzene	1.75E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.75E-11	0.0009%
28	Ethylene dibromide [EDB]	6.65E-13	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.65E-13	0.0000%
29	1,3-Butadiene	2.63E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.63E-08	1.3557%
30	Acrolein	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
31	Ethylene dichloride [EDC]	1.02E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.02E-08	0.5258%
32	Acrylonitrile	7.16E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.16E-09	0.3691%
33	Toluene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
34	Chlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
35	Hexane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
36	Glutaraldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
37	Propylene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
38	Triethylamine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
39	1,4-Dioxane	6.25E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.25E-09	0.3222%
40	Perchloroethylene [Tetrachloroethene]	2.53E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.53E-09	0.1304%
41	Indeno[1,2,3-cd]pyrene	9.99E-12	1.33E-10	1.99E-11	0.00E+00	4.86E-10	6.39E-10	6.49E-10	0.0335%
42	Benzo[b]fluoranthene	2.48E-11	3.29E-10	4.93E-11	0.00E+00	1.20E-09	1.58E-09	1.61E-09	0.0830%
43	Benzo[k]fluoranthene	3.37E-11	4.48E-10	6.71E-11	0.00E+00	1.64E-09	2.15E-09	2.19E-09	0.1129%
44	Chrysene	5.56E-12	7.39E-11	1.11E-11	0.00E+00	2.70E-10	3.55E-10	3.61E-10	0.0186%
45	Hydrazine	5.14E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.14E-08	2.6495%
46	Xylenes (mixed)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
47	2,3,7,8-Tetrachlorodibenzo-p-dioxin	6.06E-10	2.55E-09	1.07E-09	2.86E-09	4.98E-10	6.98E-09	7.59E-09	0.3912%
48	1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin	1.23E-12	5.17E-12	2.16E-12	5.80E-12	1.01E-12	1.41E-11	1.54E-11	0.0008%
49	Lead	3.49E-11	2.23E-12	7.35E-11	0.00E+00	1.51E-10	2.27E-10	2.62E-10	0.0135%
50	Mercury	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
51	Hydrochloric acid	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
52	Hydrogen fluoride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
53	Nitric acid	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
54	Hydrogen sulfide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
55	Phosphine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
56	Chromium, hexavalent (& compounds)	1.14E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.14E-08	0.5876%
57	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin	8.17E-10	3.44E-09	1.44E-09	3.86E-09	6.72E-10	9.41E-09	1.02E-08	0.5258%
58	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin	2.13E-10	8.95E-10	3.75E-10	1.00E-09	1.75E-10	2.45E-09	2.66E-09	0.1371%
59	1,2,3,4,6,7,8,9-Octachlorodibenzofuran	1.35E-12	5.66E-12	2.37E-12	6.36E-12	1.11E-12	1.55E-11	1.68E-11	0.0009%
60	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin	5.05E-10	2.12E-09	8.89E-10	2.38E-09	4.15E-10	5.81E-09	6.31E-09	0.3253%
61	1,2,3,7,8-Pentachlorodibenzo-p-dioxin	3.24E-09	1.36E-08	5.71E-09	1.53E-08	2.67E-09	3.73E-08	4.06E-08	2.0928%
62	2,3,7,8-Tetrachlorodibenzofuran	8.24E-10	3.47E-09	1.45E-09	3.90E-09	6.78E-10	9.49E-09	1.03E-08	0.5309%
63	1,2,3,4,7,8,9-Heptachlorodibenzofuran	5.72E-11	2.41E-10	1.01E-10	2.70E-10	4.70E-11	6.58E-10	7.16E-10	0.0369%
64	2,3,4,7,8-Pentachlorodibenzofuran	7.94E-09	3.34E-08	1.40E-08	3.75E-08	6.52E-09	9.14E-08	9.94E-08	5.1237%
65	1,2,3,7,8-Pentachlorodibenzofuran	4.48E-10	1.88E-09	7.88E-10	2.11E-09	3.68E-10	5.15E-09	5.60E-09	0.2887%
66	1,2,3,6,7,8-Hexachlorodibenzofuran	9.19E-10	3.87E-09	1.62E-09	4.34E-09	7.55E-10	1.06E-08	1.15E-08	0.5928%
67	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin	1.19E-09	5.00E-09	2.09E-09	5.61E-09	9.76E-10	1.37E-08	1.49E-08	0.7680%
68	2,3,4,6,7,8-Hexachlorodibenzofuran	1.92E-11	8.08E-11	3.38E-11	9.07E-11	1.58E-11	2.21E-10	2.40E-10	0.0124%
69	1,2,3,4,6,7,8-Heptachlorodibenzofuran	7.34E-10	3.09E-09	1.29E-09	3.47E-09	6.03E-10	8.45E-09	9.19E-09	0.4737%
70	1,2,3,4,7,8-Hexachlorodibenzofuran	2.40E-09	1.01E-08	4.24E-09	1.14E-08	1.98E-09	2.77E-08	3.01E-08	1.5515%
71	1,2,3,7,8,9-Hexachlorodibenzofuran	1.24E-11	5.21E-11	2.18E-11	5.85E-11	1.02E-11	1.43E-10	1.55E-10	0.0080%
SUM		1.69E-06	8.72E-08	3.57E-08	9.42E-08	2.86E-08	2.46E-07	1.94E-06	100.0000%

Cancer Risk for Maximum Sensitive Receptor - by Source
 Located at the the Woodland Clinic Medical Group

Source No.	Source Identification	Inhalation	Dermal	Soil	Mother's Milk	Home grown Vegetables	Oral	Total	Percent of Total
1	Central Heating and Cooling Plant Boiler #1	2.44E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.44E-09	0.1258%
2	Central Heating and Cooling Plant Boiler #2	3.82E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.82E-09	0.1969%
3	Central Heating and Cooling Plant Boiler #3	6.43E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.43E-10	0.0331%
4	Central Heating and Cooling Plant Boiler #4, Natural Gas	2.80E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.80E-09	0.1443%
5	Central Heating and Cooling Plant Boiler #4, Diesel	2.07E-09	3.89E-11	5.83E-12	0.00E+00	1.42E-10	1.87E-10	2.26E-09	0.1165%
6	Primate Center Boiler No 1 Natural Gas	4.98E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.98E-12	0.0003%
7	Primate Center Boiler No 2 Natural Gas	5.05E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.05E-12	0.0003%
8	Primate Center Boiler No 2 Landfill Gas	1.23E-09	1.21E-09	2.82E-10	4.25E-10	3.10E-09	5.01E-09	6.24E-09	0.3216%
9	Landfill Flare	9.63E-13	6.93E-13	1.04E-13	0.00E+00	2.54E-12	3.33E-12	4.30E-12	0.0002%
10	Incinerator	2.05E-08	8.59E-08	3.53E-08	9.37E-08	2.52E-08	2.40E-07	2.61E-07	13.4536%
11	ARS J-1 (H001)	2.98E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.98E-10	0.0154%
12	ARS J-1 CAAN 3840 - 4 boilers	4.44E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.44E-10	0.0229%
13	ARS K-2 Co-located 2 stacks	5.98E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.98E-10	0.0308%
14	ARS K-2 (H040)	2.04E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.04E-10	0.0105%
15	Contained Research	1.56E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.56E-11	0.0008%
16	Environmental Horticulture K-1	1.63E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.63E-11	0.0008%
17	Environmental Horticulture K-2	2.27E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.27E-11	0.0012%
18	Environmental Services Facility A	9.28E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.28E-10	0.0478%
19	Environmental Services Facility (3 per stack)	5.40E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.40E-10	0.0278%
20	Genome Launch Facility (plant reproduction)	1.08E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.08E-10	0.0056%
21	Equine Analytical Chemistry Lab	6.75E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.75E-11	0.0035%
22	Housing - Castillian DC	1.89E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.89E-10	0.0097%
23	Housing - Castillian DC	6.63E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.63E-11	0.0034%
24	Comparative Medicine (Primate Center)	2.67E-14	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.67E-14	0.0000%
25	Contained Research	2.44E-14	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.44E-14	0.0000%
26	Institute of Ecology - West Campus	5.97E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.97E-11	0.0031%
27	ITEH Geriatrics - cage wash inside co-located 3 stacks	1.20E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.20E-11	0.0006%
28	ITEH Geriatrics Cagewash - outside - co-located 3 stacks	1.52E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.52E-10	0.0078%
29	Mondavi Ctr for Performing Arts - 2 boilers	6.78E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.78E-11	0.0035%
30	Mondavi Ctr for Performing Arts	3.96E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.96E-12	0.0002%
31	Rec Pool	1.02E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.02E-09	0.0526%
32	Thoreau Hall - 2 stacks co-located	8.32E-14	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.32E-14	0.0000%
33	Air Stripper	4.75E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.75E-11	0.0024%
34	In-well Stripper	6.61E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.61E-10	0.0341%
35	Ground Water Treatment	8.01E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.01E-10	0.0413%
36	Large Kiln	4.35E-09	1.04E-12	2.84E-11	0.00E+00	5.91E-11	8.85E-11	4.44E-09	0.2289%
37	Raku Kiln	1.99E-10	4.72E-14	1.29E-12	0.00E+00	2.69E-12	4.03E-12	2.03E-10	0.0105%
38	Foundry Kiln	9.97E-11	2.38E-14	6.53E-13	0.00E+00	1.36E-12	2.03E-12	1.02E-10	0.0053%
39	Three Art Dept Kilns to roof vent	6.61E-09	1.57E-12	4.32E-11	0.00E+00	8.97E-11	1.34E-10	6.74E-09	0.3474%
40	Storehouse/Bulk Receiving and Storage	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
41	Walnut Dryer	8.40E-12	2.42E-13	3.62E-14	0.00E+00	8.85E-13	1.16E-12	9.56E-12	0.0005%
42	Temporary Building 187	1.68E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.68E-09	0.0866%
43	Temporary Building 188	1.31E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.31E-09	0.0675%
44	Veihmeyer	2.78E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.78E-09	0.1433%
45	Enology	1.39E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.39E-09	0.0716%
46	Wickson Hall	2.00E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.00E-08	1.0309%
47	Hoagland	1.31E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.31E-08	0.6753%
48	Mann Hall	3.18E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.18E-09	0.1639%
49	Storer Hall	5.98E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.98E-10	0.0308%
50	Hutchison Hall/Biological Sci Unit 2	1.35E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.35E-08	0.6959%
51	Asmundson Hall	9.11E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.11E-09	0.4696%
52	Robbins Hall	6.75E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.75E-09	0.3479%
53	Temporary Building 202	4.08E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.08E-10	0.0210%
54	Briggs Hall and Life Sciences	3.80E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.80E-08	1.9588%
55	Temporary Building 194	4.43E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.43E-10	0.0228%
56	Food Science	4.57E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.57E-11	0.0024%
57	Temporary Building 193	1.73E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.73E-10	0.0089%
58	Temporary Building 191	1.45E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.45E-10	0.0075%
59	Temporary Building 166	1.47E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.47E-10	0.0076%
60	Temporary Building 167	2.88E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.88E-10	0.0148%
61	Temporary Building 138	2.79E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.79E-10	0.0144%
62	Temporary Building 155	2.38E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.38E-10	0.0123%
63	Temporary Building 156	2.15E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.15E-10	0.0111%
64	Temporary Building 157	1.89E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.89E-10	0.0097%
65	Temporary Building 151	2.75E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.75E-10	0.0142%
66	Temporary Building 149	1.69E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.69E-10	0.0087%
67	Temporary Building 153	1.40E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.40E-10	0.0072%
68	Temporary Building 158	1.38E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.38E-10	0.0071%
69	Engineering II	7.17E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.17E-09	0.3696%
70	Walker Hall	3.68E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.68E-10	0.0190%
71	Chemistry	5.10E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.10E-08	2.6289%
72	Chemistry Annex	2.71E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.71E-08	1.3969%
73	Bainer Hall	1.97E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.97E-08	1.0155%
74	Crocker Hall	1.91E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.91E-10	0.0098%
75	Academic Surge	1.93E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.93E-09	0.0995%
76	Meyer Hall	1.05E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.05E-08	0.5412%
77	Physics/Geology/Physics Unit 1	9.54E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.54E-10	0.0492%
78	Environmental Horticulture	1.24E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.24E-09	0.0639%
79	Thurman Hall	8.08E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.08E-10	0.0416%
80	Maddy Hall	1.45E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.45E-09	0.0747%
81	Tupper Hall	7.89E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.89E-09	0.4067%
82	VET MED 2	7.64E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.64E-10	0.0394%
83	Asmundson Annex	5.86E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.86E-10	0.0302%
84	Young Hall	2.00E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.00E-09	0.1031%
85	Temporary Building 9	3.04E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.04E-10	0.0157%
86	ARS H-1 (Vet Meta Res)	4.11E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.11E-12	0.0002%
87	Serology4	1.91E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.91E-10	0.0098%
88	ARS R-1	8.81E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.81E-12	0.0005%
89	ARS R-2	1.20E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.20E-10	0.0062%
90	Center For Comparative Medicine	7.94E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.94E-11	0.0041%
91	Primate Center	4.54E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.54E-11	0.0023%
92	Temporary Building 184	1.54E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.54E-11	0.0008%
93	Temporary Building 160	5.87E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.87E-12	0.0003%
94	APCARU	8.26E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.26E-12	0.0004%
95	Ecology Lab (Aquadic Bio in bldg DB)	4.11E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.11E-11	0.0021%
96	Temporary Building 1	1.64E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.64E-11	0.0008%
97	ITEH Cellular Biology	1.26E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.26E-10	0.0065%
98	ITEH Pathology Clinic	1.07E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.07E-10	0.0

Cancer Risk for Maximum Sensitive Receptor - by Source
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Source No.	Source Identification	Inhalation	Dermal	Soil	Mother's Milk	Home grown Vegetables	Oral	Total	Percent of Total
101	Cole Fac B	1.33E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.33E-10	0.0069%
102	Cole Fac C	2.34E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.34E-10	0.0121%
103	TB 31	3.69E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.69E-11	0.0019%
104	TB 33	2.07E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.07E-10	0.0107%
105	TB 164	3.06E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.06E-10	0.0158%
106	TB 165	2.99E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.99E-10	0.0154%
107	TB 205	2.81E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.81E-10	0.0145%
108	HH1	8.90E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.90E-12	0.0005%
109	HH2	3.88E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.88E-11	0.0020%
110	HH3	9.93E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.93E-12	0.0005%
111	HH6	1.12E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.12E-10	0.0058%
112	Vet Med Teaching Hospital (VMTH)	4.15E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.15E-10	0.0214%
113	ARS Iso Barn J bldg	7.79E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.79E-12	0.0004%
114	ITEH Animal Housing-2	6.57E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.57E-11	0.0034%
115	LEHR Lab and Office	7.53E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.53E-11	0.0039%
116	ITEH Toxic Pollutant Lab	6.91E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.91E-11	0.0036%
117	Aqua weed lab/Aq Tox Shelter 5	4.39E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.39E-11	0.0023%
118	Bee Biology	5.67E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.67E-12	0.0003%
119	LEHR CLN MED/Medical Clinic	1.66E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.66E-10	0.0086%
120	Engineering 3 (EU3)	5.65E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.65E-09	0.2912%
121	TB 196 (Primate Center)	4.03E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.03E-11	0.0021%
122	Cruess Replacement	1.42E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.42E-08	0.7320%
123	Haring Hall Alteration	1.86E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.86E-08	0.9588%
124	Science Laboratory Building	1.62E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.62E-08	0.8351%
125	FPMS	3.83E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.83E-09	0.1974%
126	Everson Hall	1.55E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.55E-10	0.0080%
127	Center for Companion Animal Health	1.30E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.30E-09	0.0670%
128	Genome Launch Space	3.93E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.93E-09	0.2026%
129	Surge III	1.14E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.14E-11	0.0006%
130	Temporary Building 147	9.87E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.87E-12	0.0005%
131	Temporary Building 161	1.47E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.47E-10	0.0076%
132	Temporary Building 2	1.04E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.04E-11	0.0005%
133	Temporary Building 162	1.01E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.01E-09	0.0521%
134	Genome & Biomedical Science	3.82E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.82E-08	1.9691%
135	Temporary Building 127	7.75E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.75E-09	0.3995%
136	HC-2	1.06E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.06E-08	0.5464%
137	Germ Plasm	4.75E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.75E-09	0.2454%
138	Plant and Environmental Sciences	4.49E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.49E-10	0.0231%
139	Hunt Hall	3.07E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.07E-10	0.0158%
140	Cowell Student Health Center	6.83E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.83E-11	0.0035%
141	Med Sci D	9.48E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.48E-11	0.0049%
142	Equine Performance Laboratory	3.26E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.26E-08	1.6804%
143	Temporary Building 163	6.03E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.03E-10	0.0311%
144	P-17-98 60 Sub (115KV)	2.88E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.88E-09	0.1485%
145	No Permit Academic Surg	1.03E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.03E-08	0.5309%
146	No Permit Advanced Materials	3.26E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.26E-09	0.1680%
147	P-90-94(a) Aquaculture Trout	3.29E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.29E-09	0.1696%
148	P-107-95(a) Aquaculture II Well	1.05E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.05E-09	0.0541%
149	P-54-09 ARCH (rec hall)	2.51E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.51E-09	0.1294%
150	P-94-94(a) Bowley G.H	1.20E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.20E-08	0.6186%
151	P-118-03 CCAH	6.11E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.11E-10	0.0315%
152	No Permit Center for Neurosci	1.44E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.44E-09	0.0742%
153	P-82-02 Center For the Arts	1.01E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.01E-08	0.5206%
154	P-2-09 Child Health & Disease	1.92E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.92E-11	0.0010%
155	P-09-01 Cole B	2.02E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.02E-09	0.1041%
156	P-102-03 Contained Research	2.97E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.97E-10	0.0153%
157	No Permit Crocker	5.30E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.30E-09	0.2732%
158	P-08-01 Data Center	1.02E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.02E-08	0.5258%
159	P-83-02 Dom Grd Water Tank 1	5.80E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.80E-10	0.0299%
160	P-117-03 Dom Well # 2	7.09E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.09E-08	3.6546%
161	P-119-03 Dom Well # 3	1.46E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.46E-08	0.7526%
162	P-103-94(a) Dom Well # 4	3.21E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.21E-09	0.1655%
163	P-95-94(a) Dom Well # 6A	2.30E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.30E-09	0.1186%
164	P-42-97 Dom Well # 7a	2.37E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.37E-10	0.0122%
165	P-101-94(a) Engineering II	1.63E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.63E-08	0.8402%
166	P-01-00 Engineering III	1.92E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.92E-08	0.9897%
167	P-02-00 Equine Lab	3.70E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.70E-08	1.9072%
168	P-32-99 ESF	2.24E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.24E-09	0.1155%
169	P-89-94(a) Fire/Police	5.59E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.59E-08	2.8814%
170	P-51-07 Food Science	1.01E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.01E-08	0.5206%
171	P-84-02 FPMS	3.11E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.11E-10	0.0160%
172	P-120-03 GBSF	9.45E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.45E-09	0.4871%
173	P-114-02 Genome Launch	7.38E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.38E-09	0.3804%
174	No Permit Hickey Gym	1.11E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.11E-08	0.5722%
175	P-210-95(a) Hutch Sew Lift Sta	1.23E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.23E-09	0.0634%
176	No Permit Hutchison	8.60E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.60E-09	0.4433%
177	P-115-03 Inst of ecology lab	1.29E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.29E-09	0.0665%
178	No Permit ITEH (WR Lab)	5.86E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.86E-10	0.0302%
179	P-54-97 Life Sciences	2.65E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.65E-07	13.6598%
180	P-50-07 Mondavi RMI	4.61E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.61E-09	0.2376%
181	P-59-07 Multi use stadium	1.38E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.38E-09	0.0711%
182	No Permit Neurosci - off campus	1.62E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.62E-09	0.0835%
183	P-16-09 New UG RES (Cat)	1.00E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.00E-08	0.5155%
184	No Permit Old Fire Station	8.77E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.77E-09	0.4521%
185	P-29-96(a) Physical Plant	1.49E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.49E-08	0.7680%
186	P-120-01 Plant Envir Sci	3.62E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.62E-08	1.8660%
187	P-50-99(a) Port Gen # 1	3.47E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.47E-08	1.7887%
188	No Permit Port Gen # 14	1.25E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.25E-07	6.4433%
189	P-51-99(a) Port Gen # 2	9.10E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.10E-09	0.4691%
190	P-52-99(a) Port Gen # 3	6.58E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.58E-10	0.0339%
191	P-86-01 Port Gen # 7	3.61E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.61E-09	0.1861%
192	P-87-01 Port Gen # 8	2.18E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.18E-09	0.1124%
193	P-49-07 Pri Animal Hous # 1	2.13E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.13E-10	0.0110%
194	P-31-98 Primate Animal	2.66E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.66E-10	0.0137%
195	P-32-98 Primate CCM	3.77E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.77E-10	0.0194%
196	P-69-96(a) Primate Freezers	1.15E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0		

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Source No.	Source Identification	Inhalation	Dermal	Soil	Mother's Milk	Home grown Vegetables	Oral	Total	Percent of Total
201	P-108-01 Primate TB North # 5	1.92E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.92E-10	0.0099%
202	P-109-01 Primate TB South # 6	2.03E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.03E-10	0.0105%
203	P-99-94(a) Quad Parking	2.11E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.11E-08	1.0876%
204	P-93-94(a) Rec Hall	8.95E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.95E-09	0.4613%
205	P-111-95(a) Schl of Med Neurosci	2.87E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.87E-09	0.1479%
206	P-123-01 Schl of Med Neurosci	4.56E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.57E-09	0.2356%
207	P-15-04 Science Lab	5.88E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.88E-09	0.3031%
208	P-74-05 Segundo Dinning	7.94E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.94E-08	4.0928%
209	P-126-95(a) Social Sci	2.25E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.25E-08	1.1598%
210	P-17-02 South Parking	7.31E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.31E-09	0.3768%
211	P-92-94(a) Storm Lift # 4	2.19E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.19E-08	1.1289%
212	C-09-129 Storm Lift #4 (new)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
213	P-71-00 Targeted genomics	3.48E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.48E-09	0.1794%
214	P-111-01 Tele Comm.	1.35E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.35E-08	0.6959%
215	P-91-94(a) Thurman Lab	2.06E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.06E-08	1.0619%
216	P-100-94(a) Toxic Pollutant	1.14E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.14E-08	0.5876%
217	P-17-09 TURF	1.27E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.27E-11	0.0007%
218	P-121-03 Tupper Load Dock	8.21E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.21E-09	0.4232%
219	P-209-95(a) Util Well 6A	5.60E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.60E-09	0.2887%
220	P-07-01 Vega Crops	4.35E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.35E-09	0.2242%
221	P-63-03 Vet Lab	2.27E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.27E-09	0.1170%
222	P-52-07 Vet Med 3A	5.98E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.98E-09	0.3082%
223	P-53-07 Vet Med 3A	4.42E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.42E-09	0.2278%
224	P-59-05 Watershed Sic	5.10E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.10E-09	0.2629%
225	P-38-05 West Entry Park	1.24E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.24E-08	0.6392%
226	P-96-94(a) WEPT Influent	7.39E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.39E-08	3.8093%
227	P-88-99 WEPT South	1.06E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.06E-08	0.5464%
228	Landfill	1.60E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.60E-09	0.0825%
229	Landfill	1.69E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.69E-09	0.0871%
230	Landfill	1.95E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.95E-09	0.1005%
231	Landfill	2.43E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.43E-09	0.1253%
232	Waste Water Treatment Plant	4.23E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.23E-09	0.2180%
233	Grounds Above-ground Storage Tank	9.04E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.04E-11	0.0047%
234	Fleet Services Underground Storage Tank	3.43E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.43E-09	0.1768%
235	Primate Center Gasoline AST	3.91E-13	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.91E-13	0.0000%
236	Agricultural Services AST	9.48E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.48E-11	0.0049%
237	Plant Pathology Storage Tank	6.14E-13	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.14E-13	0.0000%
238	Pomology Above Ground Storage Tank	5.01E-13	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.01E-13	0.0000%
239	Airport Above Ground Storage Tank	3.50E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.50E-10	0.0180%
SUM		1.69E-06	8.72E-08	3.57E-08	9.42E-08	2.86E-08	2.46E-07	1.94E-06	100.0000%

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Chemical No.	Chemical	Cardiovascular system	Central Nervous System	Bone	Develop-mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
1	Diesel engine exhaust, particulate matter (Diesel PM)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.63E-04	0.00E+00	0.00E+00	7.63E-04	8.7100%
2	Formaldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.26E-03	0.00E+00	0.00E+00	1.26E-03	14.3836%
3	Benzo[a]pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
4	Dibenz[a,h]anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
5	Carbon tetrachloride	0.00E+00	1.97E-05	0.00E+00	1.97E-05	0.00E+00	0.00E+00	1.97E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.97E-05	0.0000%
6	Benz[a]anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
7	Methanol	0.00E+00	0.00E+00	0.00E+00	3.30E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.30E-05	0.0000%
8	Isopropyl alcohol	0.00E+00	0.00E+00	0.00E+00	4.46E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.46E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.46E-06	0.0000%
9	Chloroform	0.00E+00	0.00E+00	0.00E+00	5.37E-05	0.00E+00	0.00E+00	5.37E-05	0.00E+00	5.37E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.37E-05	0.0000%
10	Dimethyl formamide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.99E-06	0.00E+00	0.00E+00	0.00E+00	5.99E-06	0.00E+00	0.00E+00	5.99E-06	0.0684%
11	Benzene	0.00E+00	1.34E-05	0.00E+00	1.34E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.34E-05	1.34E-05	0.0000%
12	Methyl chloroform [1,1,1-TCA]	0.00E+00	2.09E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.09E-07	0.0000%
13	Ethyl chloride [Chloroethane]	0.00E+00	0.00E+00	0.00E+00	1.31E-10	0.00E+00	0.00E+00	1.31E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.31E-10	0.0000%
14	Vinyl chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
15	Acetaldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.22E-06	0.00E+00	0.00E+00	1.22E-06	0.0139%
16	Methylene chloride [Dichloromethane]	6.33E-05	6.33E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.33E-05	0.0000%
17	Carbon disulfide	0.00E+00	2.70E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.70E-09	0.00E+00	0.00E+00	0.00E+00	2.70E-09	0.0000%
18	1,1-Dichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
19	Vinylidene chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.35E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.35E-08	0.0000%
20	1,2-Dichloropropane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
21	Methyl ethyl ketone [2-Butanone]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
22	Trichloroethylene	0.00E+00	1.90E-07	0.00E+00	0.00E+00	0.00E+00	1.90E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.90E-07	0.0000%
23	Acrylamide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
24	1,1,2,2-Tetrachloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
25	Naphthalene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.51E-06	0.00E+00	0.00E+00	8.51E-06	0.0971%
26	Ethyl benzene	0.00E+00	0.00E+00	0.00E+00	1.23E-08	1.23E-08	0.00E+00	1.23E-08	0.00E+00	1.23E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.23E-08	0.0000%
27	p-Dichlorobenzene	0.00E+00	1.88E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.88E-09	0.00E+00	1.88E-09	0.00E+00	1.88E-09	0.00E+00	0.00E+00	1.88E-09	0.0000%
28	Ethylene dibromide [EDB]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.15E-08	0.00E+00	0.00E+00	0.00E+00	1.15E-08	0.0000%
29	1,3-Butadiene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.56E-06	0.00E+00	0.00E+00	0.00E+00	7.56E-06	0.0000%
30	Acrolein	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.90E-07	0.00E+00	0.00E+00	7.90E-07	0.0090%
31	Ethylene dichloride [EDC]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.22E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.22E-06	0.0000%
32	Acrylonitrile	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.95E-06	0.00E+00	0.00E+00	4.95E-06	0.0565%
33	Toluene	0.00E+00	4.73E-05	0.00E+00	4.73E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.73E-05	0.00E+00	0.00E+00	4.73E-05	0.5400%
34	Chlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.37E-09	0.00E+00	1.37E-09	1.37E-09	0.00E+00	0.00E+00	0.00E+00	1.37E-09	0.0000%
35	Hexane	0.00E+00	1.37E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.37E-07	0.0000%
36	Glutaraldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.71E-03	0.00E+00	0.00E+00	3.71E-03	42.3516%
37	Propylene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.27E-09	0.00E+00	0.00E+00	2.27E-09	0.0000%
38	Triethylamine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.47E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.47E-06	0.0000%
39	1,4-Dioxane	2.66E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.66E-07	0.00E+00	2.66E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.66E-07	0.0000%
40	Perchloroethylene [Tetrachloroethene]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.19E-05	0.00E+00	1.19E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.19E-05	0.0000%
41	Indeno[1,2,3-cd]pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
42	Benzo[b]fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
43	Benzo[k]fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
44	Chrysene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
45	Hydrazine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.22E-05	0.00E+00	5.22E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.22E-05	0.0000%
46	Xylenes (mixed)	0.00E+00	2.22E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.22E-05	0.00E+00	0.00E+00	2.22E-05	0.2534%
47	2,3,7,8-Tetrachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	5.95E-06	5.95E-06	0.00E+00	5.95E-06	0.00E+00	0.00E+00	5.95E-06	5.95E-06	0.00E+00	5.95E-06	5.95E-06	0.0679%
48	1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	1.21E-08	1.21E-08	0.00E+00	1.21E-08	0.00E+00	0.00E+00	1.21E-08	1.21E-08	0.00E+00	1.21E-08	1.21E-08	0.0001%
49	Lead	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
50	Mercury	0.00E+00	1.16E-06	0.00E+00	1.16E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.16E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.16E-06	0.0000%
51	Hydrochloric acid	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.70E-03	0.00E+00	0.00E+00	2.70E-03	30.8219%
52	Hydrogen fluoride	0.00E+00	0.00E+00	2.05E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.05E-05	0.00E+00	0.00E+00	2.05E-05	0.2340%

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53	Nitric acid	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
54	Hydrogen sulfide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.59E-05	0.00E+00	0.00E+00	1.59E-05	0.1815%
55	Phosphine	0.00E+00	4.56E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.56E-07	0.00E+00	4.56E-07	0.00E+00	4.56E-07	0.00E+00	4.56E-07	4.56E-07	0.0052%
56	Chromium, hexavalent (& compounds)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.86E-07	0.00E+00	3.04E-08	3.86E-07	0.0044%
57	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	8.03E-06	8.03E-06	0.00E+00	8.03E-06	0.00E+00	0.00E+00	8.03E-06	8.03E-06	0.00E+00	8.03E-06	8.03E-06	0.0917%
58	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	2.09E-06	2.09E-06	0.00E+00	2.09E-06	0.00E+00	0.00E+00	2.09E-06	2.09E-06	0.00E+00	2.09E-06	2.09E-06	0.0239%
59	1,2,3,4,6,7,8,9-Octachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	1.32E-08	1.32E-08	0.00E+00	1.32E-08	0.00E+00	0.00E+00	1.32E-08	1.32E-08	0.00E+00	1.32E-08	1.32E-08	0.0002%
60	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	4.96E-06	4.96E-06	0.00E+00	4.96E-06	0.00E+00	0.00E+00	4.96E-06	4.96E-06	0.00E+00	4.96E-06	4.96E-06	0.0566%
61	1,2,3,7,8-Pentachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	3.19E-05	3.19E-05	0.00E+00	3.19E-05	0.00E+00	0.00E+00	3.19E-05	3.19E-05	0.00E+00	3.19E-05	3.19E-05	0.3642%
62	2,3,7,8-Tetrachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	8.10E-06	8.10E-06	0.00E+00	8.10E-06	0.00E+00	0.00E+00	8.10E-06	8.10E-06	0.00E+00	8.10E-06	8.10E-06	0.0925%
63	1,2,3,4,7,8,9-Heptachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	5.62E-07	5.62E-07	0.00E+00	5.62E-07	0.00E+00	0.00E+00	5.62E-07	5.62E-07	0.00E+00	5.62E-07	5.62E-07	0.0064%
64	2,3,4,7,8-Pentachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	7.80E-05	7.80E-05	0.00E+00	7.80E-05	0.00E+00	0.00E+00	7.80E-05	7.80E-05	0.00E+00	7.80E-05	7.80E-05	0.8904%
65	1,2,3,7,8-Pentachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	4.40E-06	4.40E-06	0.00E+00	4.40E-06	0.00E+00	0.00E+00	4.40E-06	4.40E-06	0.00E+00	4.40E-06	4.40E-06	0.0502%
66	1,2,3,6,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	9.02E-06	9.02E-06	0.00E+00	9.02E-06	0.00E+00	0.00E+00	9.02E-06	9.02E-06	0.00E+00	9.02E-06	9.02E-06	0.1030%
67	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	1.17E-05	1.17E-05	0.00E+00	1.17E-05	0.00E+00	0.00E+00	1.17E-05	1.17E-05	0.00E+00	1.17E-05	1.17E-05	0.1336%
68	2,3,4,6,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	1.89E-07	1.89E-07	0.00E+00	1.89E-07	0.00E+00	0.00E+00	1.89E-07	1.89E-07	0.00E+00	1.89E-07	1.89E-07	0.0022%
69	1,2,3,4,6,7,8-Heptachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	7.21E-06	7.21E-06	0.00E+00	7.21E-06	0.00E+00	0.00E+00	7.21E-06	7.21E-06	0.00E+00	7.21E-06	7.21E-06	0.0823%
70	1,2,3,4,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	2.36E-05	2.36E-05	0.00E+00	2.36E-05	0.00E+00	0.00E+00	2.36E-05	2.36E-05	0.00E+00	2.36E-05	2.36E-05	0.2694%
71	1,2,3,7,8,9-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	1.22E-07	1.22E-07	0.00E+00	1.22E-07	0.00E+00	0.00E+00	1.22E-07	1.22E-07	0.00E+00	1.22E-07	1.22E-07	0.0014%
SUM		6.36E-05	1.68E-04	2.05E-05	3.68E-04	2.48E-04	1.66E-06	3.41E-04	0.00E+00	7.20E-05	2.03E-04	8.76E-03	0.00E+00	2.10E-04	8.76E-03	100.0000%

**Chronic HI for Maximum Sensitive Receptor - by Source
 Located at the the Woodland Clinic Medical Group**

Source No.	Source Identification	Cardiovascular system	Central Nervous System	Bone	Develop-mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
1	Central Heating and Cooling Plant Boiler #1	0.00E+00	1.17E-07	0.00E+00	1.17E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.01E-05	0.00E+00	1.17E-07	4.01E-05	0.4578%
2	Central Heating and Cooling Plant Boiler #2	0.00E+00	1.83E-07	0.00E+00	1.83E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.28E-05	0.00E+00	1.83E-07	6.28E-05	0.7169%
3	Central Heating and Cooling Plant Boiler #3	0.00E+00	3.11E-08	0.00E+00	3.11E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.06E-05	0.00E+00	3.11E-08	1.06E-05	0.1210%
4	Central Heating and Cooling Plant Boiler #4, Natural Gas	0.00E+00	1.34E-07	0.00E+00	1.34E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.62E-05	0.00E+00	1.34E-07	4.62E-05	0.5274%
5	Central Heating and Cooling Plant Boiler #4, Diesel	0.00E+00	6.86E-09	0.00E+00	6.64E-09	1.03E-10	0.00E+00	1.03E-10	0.00E+00	1.03E-10	0.00E+00	1.10E-05	0.00E+00	5.85E-09	1.10E-05	0.1256%
6	Primate Center Boiler No 1 Natural Gas	0.00E+00	2.39E-10	0.00E+00	2.39E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.20E-08	0.00E+00	2.39E-10	8.20E-08	0.0009%
7	Primate Center Boiler No 2 Natural Gas	0.00E+00	2.43E-10	0.00E+00	2.43E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.32E-08	0.00E+00	2.43E-10	8.32E-08	0.0009%
8	Primate Center Boiler No 2 Landfill Gas	5.17E-11	9.33E-08	2.26E-07	9.76E-07	8.83E-07	1.05E-11	8.83E-07	0.00E+00	9.34E-08	8.83E-07	1.01E-05	0.00E+00	8.83E-07	1.01E-05	0.1153%
9	Landfill Flare	2.17E-13	7.87E-11	1.87E-10	7.84E-11	2.62E-15	4.41E-14	1.30E-12	0.00E+00	7.95E-11	1.94E-14	7.73E-09	0.00E+00	2.66E-14	7.73E-09	0.0001%
10	Incinerator	0.00E+00	3.19E-09	0.00E+00	1.95E-04	1.95E-04	0.00E+00	1.95E-04	0.00E+00	0.00E+00	1.95E-04	2.49E-04	0.00E+00	1.95E-04	2.49E-04	2.8425%
11	ARS J-1 (H001)	0.00E+00	1.43E-08	0.00E+00	1.43E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.91E-06	0.00E+00	1.43E-08	4.91E-06	0.0561%
12	ARS J-1 CAAN 3840 - 4 boilers	0.00E+00	2.13E-08	0.00E+00	2.13E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.31E-06	0.00E+00	2.13E-08	7.31E-06	0.0834%
13	ARS K-2 Co-located 2 stacks	0.00E+00	2.87E-08	0.00E+00	2.87E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.85E-06	0.00E+00	2.87E-08	9.85E-06	0.1124%
14	ARS K-2 (H040)	0.00E+00	9.80E-09	0.00E+00	9.80E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.36E-06	0.00E+00	9.80E-09	3.36E-06	0.0384%
15	Contained Research	0.00E+00	7.49E-10	0.00E+00	7.49E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.57E-07	0.00E+00	7.49E-10	2.57E-07	0.0029%
16	Environmental Horticulture K-1	0.00E+00	7.80E-10	0.00E+00	7.80E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.68E-07	0.00E+00	7.80E-10	2.68E-07	0.0031%
17	Environmental Horticulture K-2	0.00E+00	1.09E-09	0.00E+00	1.09E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.74E-07	0.00E+00	1.09E-09	3.74E-07	0.0043%
18	Environmental Services Facility A	0.00E+00	4.44E-08	0.00E+00	4.44E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.53E-05	0.00E+00	4.44E-08	1.53E-05	0.1747%
19	Environmentatl Services Facility (3 per stack)	0.00E+00	2.60E-08	0.00E+00	2.60E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.90E-06	0.00E+00	2.60E-08	8.90E-06	0.1016%
20	Genome Launch Facility (plant reproduction)	0.00E+00	5.20E-09	0.00E+00	5.20E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.78E-06	0.00E+00	5.20E-09	1.78E-06	0.0203%
21	Equine Analytical Chemistry Lab	0.00E+00	3.25E-09	0.00E+00	3.25E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.11E-06	0.00E+00	3.25E-09	1.11E-06	0.0127%
22	Housing - Castillian DC	0.00E+00	9.10E-09	0.00E+00	9.10E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.12E-06	0.00E+00	9.10E-09	3.12E-06	0.0356%
23	Housing - Castillian DC	0.00E+00	3.19E-09	0.00E+00	3.19E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.09E-06	0.00E+00	3.19E-09	1.09E-06	0.0124%
24	Comparative Medicine (Primate Center)	0.00E+00	1.28E-12	0.00E+00	1.28E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.41E-10	0.00E+00	1.28E-12	4.41E-10	0.0000%
25	Contained Research	0.00E+00	1.17E-12	0.00E+00	1.17E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.02E-10	0.00E+00	1.17E-12	4.02E-10	0.0000%
26	Institute of Ecology - West Campus	0.00E+00	2.87E-09	0.00E+00	2.87E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.84E-07	0.00E+00	2.87E-09	9.84E-07	0.0112%
27	ITEH Geriatrics - cage wash inside co-located 3 stacks	0.00E+00	5.74E-10	0.00E+00	5.74E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.97E-07	0.00E+00	5.74E-10	1.97E-07	0.0022%
28	ITEH Geriatrics Cagewash - outside - co-locate 3 stacks	0.00E+00	7.32E-09	0.00E+00	7.32E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.51E-06	0.00E+00	7.32E-09	2.51E-06	0.0287%
29	Mondavi Ctr for Performing Arts - 2 boilers	0.00E+00	3.25E-09	0.00E+00	3.25E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.12E-06	0.00E+00	3.25E-09	1.12E-06	0.0128%
30	Mondavi Ctr for Performing Arts	0.00E+00	1.90E-10	0.00E+00	1.90E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.53E-08	0.00E+00	1.90E-10	6.53E-08	0.0007%
31	Rec Pool	0.00E+00	4.88E-08	0.00E+00	4.88E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.68E-05	0.00E+00	4.88E-08	1.68E-05	0.1918%
32	Thoreau Hall - 2 stacks co-located	0.00E+00	4.00E-12	0.00E+00	4.00E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.37E-09	0.00E+00	4.00E-12	1.37E-09	0.0000%
33	Air Stripper	0.00E+00	0.00E+00	0.00E+00	2.88E-08	0.00E+00	0.00E+00	2.88E-08	0.00E+00	2.88E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.88E-08	0.0000%
34	In-well Stripper	0.00E+00	0.00E+00	0.00E+00	4.01E-07	0.00E+00	0.00E+00	4.01E-07	0.00E+00	4.01E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.01E-07	0.0000%
35	Ground Water Treatment	0.00E+00	0.00E+00	0.00E+00	4.85E-07	0.00E+00	0.00E+00	4.85E-07	0.00E+00	4.85E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.85E-07	0.0000%
36	Large Kiln	0.00E+00	2.16E-09	0.00E+00	2.02E-09	7.94E-12	0.00E+00	7.94E-12	0.00E+00	7.94E-12	0.00E+00	3.06E-07	0.00E+00	1.28E-08	3.06E-07	0.0035%
37	Raku Kiln	0.00E+00	9.85E-11	0.00E+00	9.23E-11	3.62E-13	0.00E+00	3.62E-13	0.00E+00	3.62E-13	0.00E+00	1.39E-08	0.00E+00	5.86E-10	1.39E-08	0.0002%
38	Foundry Kiln	0.00E+00	4.97E-11	0.00E+00	4.65E-11	1.83E-13	0.00E+00	1.83E-13	0.00E+00	1.83E-13	0.00E+00	7.02E-09	0.00E+00	2.94E-10	7.02E-09	0.0001%
39	Three Art Dept Kilns to roof vent	0.00E+00	3.28E-09	0.00E+00	3.07E-09	1.21E-11	0.00E+00	1.21E-11	0.00E+00	1.21E-11	0.00E+00	4.64E-07	0.00E+00	1.95E-08	4.64E-07	0.0053%
40	Storehouse/Bulk Receiving and Storage	0.00E+00	0.00E+00	0.00E+00	2.21E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.19E-07	0.00E+00	0.00E+00	2.21E-07	0.0000%
41	Walnut Dryer	0.00E+00	2.98E-09	0.00E+00	2.79E-09	1.10E-11	0.00E+00	1.10E-11	0.00E+00	1.10E-11	0.00E+00	2.19E-07	0.00E+00	1.82E-09	2.19E-07	0.0025%
42	Temporary Building 187	2.16E-08	1.73E-07	7.37E-08	5.01E-07	2.32E-07	5.21E-09	5.59E-07	0.00E+00	2.15E-07	0.00E+00	4.21E-05	0.00E+00	1.20E-08	4.21E-05	0.4806%
43	Temporary Building 188	1.69E-08	1.35E-07	5.78E-08	3.93E-07	1.82E-07	4.08E-09	4.38E-07	0.00E+00	1.69E-07	0.00E+00	3.30E-05	0.00E+00	9.39E-09	3.30E-05	0.3767%
44	Veihmeyer	2.18E-08	5.26E-07	3.89E-06	9.53E-07	8.94E-08	1.97E-08	7.91E-07	0.00E+00	2.18E-07	0.00E+00	7.18E-05	0.00E+00	3.71E-08	7.18E-05	0.8196%
45	Enology	1.77E-08	1.41E-07	6.02E-08	4.11E-07	1.90E-07	4.27E-09	4.57E-07	0.00E+00	1.76E-07	0.00E+00	3.45E-05	0.00E+00	9.82E-09	3.45E-05	0.3938%
46	Wickson Hall	2.55E-07	2.04E-06	8.70E-07	5.93E-06	2.74E-06	6.17E-08	6.61E-06	0.00E+00	2.55E-06	0.00E+00	4.98E-04	0.00E+00	1.42E-07	4.98E-04	5.6849%
47	Hoagland	1.69E-07	1.34E-06	5.73E-07	3.91E-06	1.80E-06	4.06E-08	4.35E-06	0.00E+00	1.67E-06	0.00E+00	3.28E-04	0.00E+00	9.34E-08	3.28E-04	3.7443%
48	Mann Hall	4.10E-08	3.28E-07	1.39E-07	9.50E-07	4.38E-07	9.87E-09	1.06E-06	0.00E+00	4.06E-07	0.00E+00	7.98E-05	0.00E+00	2.27E-08	7.98E-05	0.9110%
49	Storer Hall	7.71E-09	6.15E-08	2.62E-08	1.79E-07	8.27E-08	1.85E-09	1.99E-07	0.00E+00	7.66E-08	0.00E+00	1.50E-05	0.00E+00	4.27E-09	1.50E-05	0.1712%
50	Hutchison Hall/Biological Sci Unit 2	1.74E-07	1.39E-06	5.94E-07	4.05E-06	1.87E-06	4.20E-08	4.51E-06	0.00E+00	1.73E-06	0.00E+00	3.40E-04	0.00E+00	9.65E-08	3.40E-04	3.8813%
51	Asmundson Hall	1.17E-07	9.39E-07	4.01E-07	2.73E-06	1.26E-06	2.83E-08	3.04E-06	0.00E+00	1.17E-06	0.00E+00	2.29E-04	0.00E+00	6.51E-08	2.29E-04	2.6142%
52	Robbins Hall	8.69E-08	6.94E-07	2.97E-07	2.02E-06	9.32E-07	2.10E-08	2.25E-06	0.00E+00	8.64E-07	0.00E+00	1.69E-04	0.00E+00	4.82E-08	1.69E-04	1.9292%
53	Temporary Building 202	5.25E-09	4.20E-08	1.79E-08	1.22E-07	5.63E-08	1.27E-09	1.36E-07	0.00E+00	5.22E-08	0.00E+00	1.02E-05	0.00E+00	2.91E-09	1.02E-05	0.1164%
54	Briggs Hall and Life Sciences	4.87E-07	3.89E-06	1.66E-06	1.13E-05	5.23E-06	1.18E-07	1.26E-05	0.00E+00	4.85E-06	0.00E+00	9.49E-04	0.00E+00	2.70E-07	9.49E-04	10.8333%
55	Temporary Building 194	5.71E-09	4.56E-08	1.95E-08	1.33E-07	6.12E-08	1.38E-09	1.48E-07	0.00E+00	5.68E-08	0.00E+00	1.11E-05	0.00E+00	3.17E-09	1.11E-05	0.1267%
56	Food Science	5.88E-10	4.70E-09	2.01E-09	1.37E-08	6.31E-09	1.42E-10	1.52E-08	0.00E+00	5.85E-09	0.00E+00	1.15E-06	0.00E+00	3.26E-10	1.15E-06	0.0131%
57	Temporary Building 1															

Chronic HI for Maximum Sensitive Receptor - by Source
 Located at the the Woodland Clinic Medical Group

Source No.	Source Identification	Cardiovascular system	Central Nervous System	Bone	Develop-mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
62	Temporary Building 155	3.06E-09	2.45E-08	1.04E-08	7.11E-08	3.28E-08	7.38E-10	7.92E-08	0.00E+00	3.05E-08	0.00E+00	5.96E-06	0.00E+00	1.70E-09	5.96E-06	0.0680%
63	Temporary Building 156	2.77E-09	2.21E-08	9.47E-09	6.44E-08	2.98E-08	6.70E-10	7.18E-08	0.00E+00	2.76E-08	0.00E+00	5.40E-06	0.00E+00	1.54E-09	5.41E-06	0.0616%
64	Temporary Building 157	2.43E-09	1.94E-08	8.30E-09	5.65E-08	2.62E-08	5.86E-10	6.30E-08	0.00E+00	2.42E-08	0.00E+00	4.75E-06	0.00E+00	1.35E-09	4.75E-06	0.0542%
65	Temporary Building 151	3.54E-09	2.83E-08	1.21E-08	8.22E-08	3.79E-08	8.54E-10	9.15E-08	0.00E+00	3.52E-08	0.00E+00	6.90E-06	0.00E+00	1.96E-09	6.90E-06	0.0788%
66	Temporary Building 149	2.18E-09	1.74E-08	7.42E-09	5.05E-08	2.33E-08	5.24E-10	5.62E-08	0.00E+00	2.16E-08	0.00E+00	4.24E-06	0.00E+00	1.21E-09	4.24E-06	0.0484%
67	Temporary Building 153	1.80E-09	1.44E-08	6.15E-09	4.17E-08	1.93E-08	4.34E-10	4.65E-08	0.00E+00	1.79E-08	0.00E+00	3.51E-06	0.00E+00	1.00E-09	3.51E-06	0.0401%
68	Temporary Building 158	1.78E-09	1.42E-08	6.06E-09	4.11E-08	1.90E-08	4.28E-10	4.59E-08	0.00E+00	1.76E-08	0.00E+00	3.45E-06	0.00E+00	9.85E-10	3.45E-06	0.0394%
69	Engineering II	4.45E-06	5.58E-06	3.77E-09	3.12E-06	9.80E-07	5.40E-08	2.74E-06	0.00E+00	1.57E-06	0.00E+00	1.07E-05	0.00E+00	6.19E-07	1.07E-05	0.1221%
70	Walker Hall	2.89E-09	6.96E-08	5.14E-07	1.26E-07	1.18E-08	2.62E-09	1.05E-07	0.00E+00	2.89E-08	0.00E+00	9.49E-06	0.00E+00	4.92E-09	9.49E-06	0.1083%
71	Chemistry	2.10E-05	2.62E-05	1.78E-08	1.47E-05	4.61E-06	2.54E-07	1.29E-05	0.00E+00	7.41E-06	4.96E-06	5.06E-05	0.00E+00	2.92E-06	5.06E-05	0.5776%
72	Chemistry Annex	1.10E-05	1.38E-05	9.29E-09	7.69E-06	2.41E-06	1.33E-07	6.76E-06	0.00E+00	3.88E-06	2.57E-06	2.67E-05	0.00E+00	1.53E-06	2.67E-05	0.3048%
73	Bainer Hall	1.12E-05	1.40E-05	9.48E-09	7.84E-06	2.46E-06	1.36E-07	6.90E-06	0.00E+00	3.96E-06	0.00E+00	2.81E-05	0.00E+00	1.56E-06	2.81E-05	0.3208%
74	Crocker Hall	1.50E-09	3.62E-08	2.66E-07	6.56E-08	6.15E-09	1.36E-09	5.43E-08	0.00E+00	1.50E-08	0.00E+00	4.94E-06	0.00E+00	2.55E-09	4.94E-06	0.0564%
75	Academic Surge	2.49E-08	1.99E-07	8.50E-08	5.78E-07	6.02E-09	6.45E-07	0.00E+00	2.67E-07	2.48E-07	4.86E-05	0.00E+00	0.00E+00	1.38E-08	4.86E-05	0.5548%
76	Meyer Hall	1.35E-07	1.53E-06	4.59E-07	3.13E-06	1.45E-06	3.25E-08	3.94E-06	0.00E+00	1.79E-06	0.00E+00	2.63E-04	0.00E+00	5.30E-07	2.63E-04	3.0023%
77	Physics/Geology/Physics Unit 1	7.47E-09	1.80E-07	1.34E-06	3.27E-07	3.06E-08	6.77E-09	2.71E-07	0.00E+00	7.47E-08	0.00E+00	2.46E-05	0.00E+00	1.27E-08	2.46E-05	0.2808%
78	Environmental Horticulture	1.60E-08	1.28E-07	5.46E-08	3.71E-07	1.72E-07	3.85E-09	4.14E-07	0.00E+00	1.59E-07	0.00E+00	3.11E-05	0.00E+00	8.86E-09	3.11E-05	0.3550%
79	Thurman Hall	1.04E-08	8.32E-08	3.55E-08	2.42E-07	1.11E-07	2.51E-09	2.69E-07	0.00E+00	1.04E-07	0.00E+00	2.03E-05	0.00E+00	5.77E-09	2.03E-05	0.2317%
80	Maddy Hall	1.87E-08	1.49E-07	6.36E-08	4.33E-07	4.50E-09	4.83E-07	0.00E+00	2.00E-07	1.86E-07	0.00E+00	3.63E-05	0.00E+00	1.03E-08	3.63E-05	0.4144%
81	Tupper Hall	1.02E-07	8.12E-07	3.46E-07	2.36E-06	1.09E-06	2.45E-08	2.63E-06	0.00E+00	1.01E-06	0.00E+00	1.98E-04	0.00E+00	5.63E-08	1.98E-04	2.2603%
82	VET MED 2	9.86E-09	7.86E-08	3.36E-08	2.29E-07	1.05E-07	2.38E-09	2.54E-07	0.00E+00	9.80E-08	0.00E+00	1.92E-05	0.00E+00	5.46E-09	1.92E-05	0.2192%
83	Asmundson Annex	7.54E-09	6.03E-08	2.57E-08	1.75E-07	8.09E-08	1.82E-09	1.95E-07	0.00E+00	7.51E-08	0.00E+00	1.47E-05	0.00E+00	4.18E-09	1.47E-05	0.1678%
84	Young Hall	2.59E-08	2.06E-07	8.82E-08	5.99E-07	2.77E-07	6.23E-09	6.68E-07	0.00E+00	2.57E-07	0.00E+00	5.03E-05	0.00E+00	1.43E-08	5.03E-05	0.5742%
85	Temporary Building 9	3.91E-09	3.13E-08	1.34E-08	9.09E-08	4.20E-08	9.44E-10	1.01E-07	0.00E+00	3.89E-08	0.00E+00	7.61E-06	0.00E+00	2.17E-09	7.61E-06	0.0869%
86	ARS H-1 (Vet Meta Res)	3.21E-11	7.76E-10	5.73E-09	1.41E-09	1.32E-10	2.92E-11	1.17E-09	0.00E+00	3.22E-10	0.00E+00	1.06E-07	0.00E+00	5.47E-11	1.06E-07	0.0012%
87	Serology4	2.46E-09	1.96E-08	8.41E-09	5.70E-08	2.63E-08	5.95E-10	6.36E-08	0.00E+00	2.44E-08	0.00E+00	4.78E-06	0.00E+00	1.36E-09	4.78E-06	0.0546%
88	ARS R-1	1.13E-10	9.07E-10	3.87E-10	2.63E-09	1.22E-09	2.74E-11	2.93E-09	0.00E+00	1.13E-09	0.00E+00	2.22E-07	0.00E+00	6.30E-11	2.22E-07	0.0025%
89	ARS R-2	1.54E-09	1.23E-08	5.27E-09	3.59E-08	1.66E-08	3.72E-10	3.99E-08	0.00E+00	1.54E-08	0.00E+00	3.00E-06	0.00E+00	8.56E-10	3.01E-06	0.0342%
90	Center For Comparative Medicine	1.02E-09	8.17E-09	3.48E-09	2.37E-08	1.10E-08	2.46E-10	2.64E-08	0.00E+00	1.02E-08	0.00E+00	1.99E-06	0.00E+00	5.67E-10	1.99E-06	0.0227%
91	Primate Center	5.85E-10	4.67E-09	2.00E-09	1.36E-08	6.28E-09	1.41E-10	1.51E-08	0.00E+00	5.82E-09	0.00E+00	1.14E-06	0.00E+00	3.25E-10	1.14E-06	0.0130%
92	Temporary Building 184	1.98E-10	1.58E-09	6.79E-10	4.60E-09	2.13E-09	4.80E-11	5.13E-09	0.00E+00	1.97E-09	0.00E+00	3.87E-07	0.00E+00	1.10E-10	3.87E-07	0.0044%
93	Temporary Building 160	7.56E-11	6.04E-10	2.58E-10	1.76E-09	8.10E-10	1.82E-11	1.95E-09	0.00E+00	7.51E-10	0.00E+00	1.47E-07	0.00E+00	4.19E-11	1.47E-07	0.0017%
94	APCARU	1.06E-10	8.50E-10	3.63E-10	2.47E-09	1.14E-09	2.56E-11	2.75E-09	0.00E+00	1.06E-09	0.00E+00	2.07E-07	0.00E+00	5.90E-11	2.07E-07	0.0024%
95	Ecology Lab (Aquadic Bio in bldg DB)	5.30E-10	4.23E-09	1.81E-09	1.23E-08	5.68E-09	1.28E-10	1.37E-08	0.00E+00	5.27E-09	0.00E+00	1.03E-06	0.00E+00	2.94E-10	1.03E-06	0.0118%
96	Temporary Building 1	2.11E-10	1.69E-09	7.21E-10	4.91E-09	5.10E-11	5.46E-09	0.00E+00	2.26E-09	2.10E-09	0.00E+00	4.12E-07	0.00E+00	1.17E-10	4.12E-07	0.0047%
97	ITEH Cellular Biology	1.63E-09	1.30E-08	5.57E-09	3.78E-08	1.75E-08	3.93E-10	4.22E-08	0.00E+00	1.62E-08	0.00E+00	3.18E-06	0.00E+00	9.03E-10	3.18E-06	0.0363%
98	ITEH Pathology Clinic	1.38E-09	1.10E-08	4.71E-09	3.20E-08	1.48E-08	3.33E-10	3.57E-08	0.00E+00	1.37E-08	0.00E+00	2.69E-06	0.00E+00	7.65E-10	2.69E-06	0.0307%
99	ARS DL-10/Field Shelter 5; Bovine Shed (Luekemia Lab)	7.21E-10	5.77E-09	2.46E-09	1.67E-08	7.74E-09	1.74E-10	1.87E-08	0.00E+00	7.17E-09	0.00E+00	1.40E-06	0.00E+00	4.00E-10	1.40E-06	0.0160%
100	Cole Fac A	2.07E-09	1.66E-08	7.07E-09	4.81E-08	2.22E-08	4.99E-10	5.36E-08	0.00E+00	2.06E-08	0.00E+00	4.03E-06	0.00E+00	1.15E-09	4.03E-06	0.0460%
101	Cole Fac B	1.72E-09	1.37E-08	5.85E-09	3.98E-08	1.84E-08	4.13E-10	4.43E-08	0.00E+00	1.71E-08	0.00E+00	3.34E-06	0.00E+00	9.50E-10	3.34E-06	0.0381%
102	Cole Fac C	3.02E-09	2.41E-08	1.03E-08	7.01E-08	3.24E-08	7.29E-10	7.81E-08	0.00E+00	3.00E-08	0.00E+00	5.89E-06	0.00E+00	1.68E-09	5.89E-06	0.0672%
103	TB 31	4.73E-10	3.79E-09	1.62E-09	1.10E-08	5.09E-09	1.14E-10	1.23E-08	0.00E+00	4.73E-09	0.00E+00	9.26E-07	0.00E+00	2.63E-10	9.26E-07	0.0106%
104	TB 33	2.67E-09	2.13E-08	9.09E-09	6.19E-08	2.86E-08	6.43E-10	6.90E-08	0.00E+00	2.65E-08	0.00E+00	5.20E-06	0.00E+00	1.48E-09	5.20E-06	0.0594%
105	TB 164	3.95E-09	3.15E-08	1.35E-08	9.17E-08	4.24E-08	9.51E-10	1.02E-07	0.00E+00	3.93E-08	0.00E+00	7.69E-06	0.00E+00	2.19E-09	7.69E-06	0.0878%
106	TB 165	3.85E-09	3.08E-08	1.31E-08	8.94E-08	4.12E-08	9.29E-10	9.96E-08	0.00E+00	3.83E-08	0.00E+00	7.52E-06	0.00E+00	2.14E-09	7.52E-06	0.0858%
107	TB 205	3.63E-09	2.90E-08	1.24E-08	8.42E-08	3.90E-08	8.75E-10	9.39E-08	0.00E+00	3.61E-08	0.00E+00	7.07E-06	0.00E+00	2.01E-09	7.07E-06	0.0807%
108	HH1	1.15E-10	9.17E-10	3.92E-10	2.66E-09	1.23E-09	2.78E-11	2.97E-09	0.00E+00	1.14E-09	0.00E+00	2.23E-07	0.00E+00	6.36E-11	2.23E-07	0.0025%
109	HH2	5.00E-10	4.00E-09	1.71E-09	1.16E-08	5.35E-09	1.21E-10	1.29E-08	0.00E+00	4.97E-09	0.00E+00	9.74E-07	0.00E+00	2.77E-10	9.74E-07	0.0111%
110	HH3	1.28E-10	1.02E-09	4.36E-10	2.97E-09	1.37E-09	3.08E-11	3.31E-09	0.00E+00	1.27E-09	0.00E+00	2.49E-07	0.00E+00	7.09E-11	2.49E-07	0.0028%
111	HH6	1.44E-09	1.15E-08	4.92E-09	3.35E-08	1.55E-08	3.48E-10	3.73E-08	0.00E+00	1.43E-08	0.00E+00	2.80E-06	0.00E+00	8.00E-10	2.80E-06	0.0320%
112	Vet Med Teaching Hospital (VMTH)	5.35E-09	4.27E-08	1.82E-08	1.29E-09	5.72E-08	1.29E-09	1.38E-07	0.00E+00	5.32E-08	0.00E+00	1.04E-05	0.00E+00	2.96E-09	1.04E-05	0.1187%
113	ARS Iso Barn J bldg	1.00E-10	8.02E-10	3.42E-10	2.33E-09	1.08E-09	2.42E-11	2.60E-09	0.00E+00	9.98E-10	0.00E+00	1.96E-07	0.00E+00	5.57E-11	1.96E-07	0.0022%
114	ITEH Animal Housing-2	5.14E-10	1.24E-08	9.18E-08	2.25E-08	2.10E-09	4.67E-10	1.86E-08	0.00E+00	5.14E-09	0.00E+00	1.69E-06	0.00E+00	8.74E-10	1.69E-06	0.0193%
115	LEHR Lab and Office	5.90E-10	1.42E-08	1.05E-07	2.58E-08	2.42E-09	5.35E-10	2.14E-08	0.00E+00	5.90E-09	0.00E+00	1.94E-06	0.00E+00	1.00E-09	1.94E-06	0.0221%
116	ITEH Toxic Pollutant Lab	5.42E-10	1.30E-08	9.65E-08	2.36E-08	2.21E-09	4.91E-10	1.96E-08	0.00E+00	5.40E-09	0.00E+00	1.78E-06	0.00E+00	9.19E-10	1.78E-06	0.0203%
117	Aqua weed lab/Aq Tox Shelter 5	5.65E-10	4.52E-09	1.93E-09	1.31E-08	6.07E-09	1.36E-10	1.46E-08	0.00E+00	5.62E-09	0.00E+00	1.10E-06	0.00E+00	3.14E-10	1.10E-06	0.0126%
118	Bee Biology	7.31E-11	5.83E-10	2.49E-10	1.70E-09	7.85E-10	1									

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Source No.	Source Identification	Cardiovascular system	Central Nervous System	Bone	Develop- mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
184	No Permit Old Fire Station	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.51E-06	0.00E+00	0.00E+00	5.51E-06	0.0629%
185	P-29-96(a) Physical Plant	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.36E-06	0.00E+00	0.00E+00	9.36E-06	0.1068%
186	P-120-01 Plant Envir Sci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.27E-05	0.00E+00	0.00E+00	2.27E-05	0.2591%
187	P-50-99(a) Port Gen # 1	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.18E-05	0.00E+00	0.00E+00	2.18E-05	0.2489%
188	No Permit Port Gen # 14	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.85E-05	0.00E+00	0.00E+00	7.85E-05	0.8961%
189	P-51-99(a) Port Gen # 2	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.71E-06	0.00E+00	0.00E+00	5.71E-06	0.0652%
190	P-52-99(a) Port Gen # 3	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.13E-07	0.00E+00	0.00E+00	4.13E-07	0.0047%
191	P-86-01 Port Gen # 7	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.26E-06	0.00E+00	0.00E+00	2.26E-06	0.0258%
192	P-87-01 Port Gen # 8	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.37E-06	0.00E+00	0.00E+00	1.37E-06	0.0156%
193	P-49-07 Pri Animal Hous # 1	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.34E-07	0.00E+00	0.00E+00	1.34E-07	0.0015%
194	P-31-98 Primate Animal	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.67E-07	0.00E+00	0.00E+00	1.67E-07	0.0019%
195	P-32-98 Primate CCM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.37E-07	0.00E+00	0.00E+00	2.37E-07	0.0027%
196	P-69-96(a) Primate Freezers	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.21E-08	0.00E+00	0.00E+00	7.21E-08	0.0008%
197	P-102-94(a) Primate Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.39E-07	0.00E+00	0.00E+00	6.39E-07	0.0073%
198	P-15-98 Primate Quarantine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.96E-07	0.00E+00	0.00E+00	2.96E-07	0.0034%
199	No Permit Primate Sew Life Sta	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.59E-08	0.00E+00	0.00E+00	8.59E-08	0.0010%
200	P-16-98 Primate TB 184	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.35E-07	0.00E+00	0.00E+00	3.35E-07	0.0038%
201	P-108-01 Primate TB North # 5	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.21E-07	0.00E+00	0.00E+00	1.21E-07	0.0014%
202	P-109-01 Primate TB South # 6	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.27E-07	0.00E+00	0.00E+00	1.27E-07	0.0014%
203	P-99-94(a) Quad Parking	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.33E-05	0.00E+00	0.00E+00	1.33E-05	0.1518%
204	P-93-94(a) Rec Hall	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.62E-06	0.00E+00	0.00E+00	5.62E-06	0.0642%
205	P-111-95(a) Schl of Med Neurosci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.80E-06	0.00E+00	0.00E+00	1.80E-06	0.0205%
206	P-123-01 Schl of Med Neurosci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.87E-06	0.00E+00	0.00E+00	2.87E-06	0.0328%
207	P-15-04 Science Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.69E-06	0.00E+00	0.00E+00	3.69E-06	0.0421%
208	P-74-05 Segundo Dinning	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.98E-05	0.00E+00	0.00E+00	4.98E-05	0.5685%
209	P-126-95(a) Social Sci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.41E-05	0.00E+00	0.00E+00	1.41E-05	0.1610%
210	P-17-02 South Parking	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.59E-06	0.00E+00	0.00E+00	4.59E-06	0.0524%
211	P-92-94(a) Storm Lift # 4	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.38E-05	0.00E+00	0.00E+00	1.38E-05	0.1575%
212	C-09-129 Storm Lift #4 (new)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
213	P-71-00 Targeted genomics	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.19E-06	0.00E+00	0.00E+00	2.19E-06	0.0250%
214	P-111-01 Tele Comm.	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.46E-06	0.00E+00	0.00E+00	8.46E-06	0.0966%
215	P-91-94(a) Thurman Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.29E-05	0.00E+00	0.00E+00	1.29E-05	0.1473%
216	P-100-94(a) Toxic Pollutant	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.17E-06	0.00E+00	0.00E+00	7.17E-06	0.0818%
217	P-17-09 TURF	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.95E-09	0.00E+00	0.00E+00	7.95E-09	0.0001%
218	P-121-03 Tupper Load Dock	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.15E-06	0.00E+00	0.00E+00	5.15E-06	0.0588%
219	P-209-95(a) Util Well 6A	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.52E-06	0.00E+00	0.00E+00	3.52E-06	0.0402%
220	P-07-01 Vega Crops	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.73E-06	0.00E+00	0.00E+00	2.73E-06	0.0312%
221	P-63-03 Vet Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.42E-06	0.00E+00	0.00E+00	1.42E-06	0.0162%
222	P-52-07 Vet Med 3A	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.75E-06	0.00E+00	0.00E+00	3.75E-06	0.0428%
223	P-53-07 Vet Med 3A	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.77E-06	0.00E+00	0.00E+00	2.77E-06	0.0316%
224	P-59-05 Watershed Sic	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.20E-06	0.00E+00	0.00E+00	3.20E-06	0.0365%
225	P-38-05 West Entry Park	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.81E-06	0.00E+00	0.00E+00	7.81E-06	0.0892%
226	P-96-94(a) WEPT Influent	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.64E-05	0.00E+00	0.00E+00	4.64E-05	0.5297%
227	P-88-99 WEPT South	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.66E-06	0.00E+00	0.00E+00	6.66E-06	0.0760%
228	Landfill	3.10E-08	4.30E-07	0.00E+00	3.79E-07	2.49E-09	6.29E-09	1.88E-07	0.00E+00	4.11E-07	3.24E-09	2.06E-06	0.00E+00	2.54E-08	2.06E-06	0.0235%
229	Landfill	3.27E-08	4.54E-07	0.00E+00	3.99E-07	2.63E-09	6.64E-09	1.98E-07	0.00E+00	4.34E-07	3.42E-09	2.17E-06	0.00E+00	2.68E-08	2.17E-06	0.0248%
230	Landfill	3.78E-08	5.24E-07	0.00E+00	4.62E-07	3.04E-09	7.67E-09	2.29E-07	0.00E+00	5.01E-07	3.96E-09	2.51E-06	0.00E+00	3.10E-08	2.51E-06	0.0287%
231	Landfill	4.71E-08	6.53E-07	0.00E+00	5.75E-07	3.79E-09	9.56E-09	2.85E-07	0.00E+00	6.24E-07	4.93E-09	3.13E-06	0.00E+00	3.86E-08	3.13E-06	0.0357%
232	Waste Water Treatment Plant	1.30E-06	5.85E-05	0.00E+00	3.85E-05	2.45E-10	0.00E+00	1.11E-05	0.00E+00	1.11E-05	0.00E+00	6.70E-05	0.00E+00	1.02E-08	6.70E-05	0.7648%
233	Grounds Above-ground Storage Tank	0.00E+00	6.27E-08	0.00E+00	6.10E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.58E-09	0.00E+00	5.20E-08	6.27E-08	0.0001%
234	Fleet Services Underground Storage Tank	0.00E+00	2.38E-06	0.00E+00	2.31E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.64E-07	0.00E+00	1.97E-06	2.38E-06	0.0042%
235	Primate Center Gasoline AST	0.00E+00	2.72E-10	0.00E+00	2.64E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.16E-11	0.00E+00	2.25E-10	2.72E-10	0.0000%
236	Agricultural Services AST	0.00E+00	6.58E-08	0.00E+00	6.39E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.00E-08	0.00E+00	5.45E-08	6.58E-08	0.0001%
237	Plant Pathology Storage Tank	0.00E+00	4.26E-10	0.00E+00	4.14E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.48E-11	0.00E+00	3.53E-10	4.26E-10	0.0000%
238	Pomology Above Ground Storage Tank	0.00E+00	3.48E-10	0.00E+00	3.38E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.31E-11	0.00E+00	2.88E-10	3.48E-10	0.0000%
239																

Acute HI for Maximum Sensitive Receptor - by Chemical
 Located at the the Woodland Clinic Medical Group

Chemical No.	Chemical	Cardiovascular system	Central Nervous System	Bone	Develop-mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
2	Formaldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.42E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.42E-02	89.9573%
3	Benzo[a]pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
4	Dibenz[a,h]anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
5	Carbon tetrachloride	0.00E+00	1.34E-04	0.00E+00	1.34E-04	0.00E+00	0.00E+00	1.34E-04	0.00E+00	0.00E+00	1.34E-04	0.00E+00	0.00E+00	0.00E+00	1.34E-04	0.0000%
6	Benz[a]anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
7	Methanol	0.00E+00	1.49E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.49E-03	0.0000%
8	Isopropyl alcohol	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.19E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.19E-03	0.00E+00	0.00E+00	3.19E-03	3.4081%
9	Chloroform	0.00E+00	3.34E-02	0.00E+00	3.34E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.34E-02	0.00E+00	0.00E+00	0.00E+00	3.34E-02	0.0000%
10	Dimethyl formamide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
11	Benzene	0.00E+00	0.00E+00	0.00E+00	2.03E-04	0.00E+00	0.00E+00	0.00E+00	2.03E-04	0.00E+00	2.03E-04	0.00E+00	0.00E+00	2.03E-04	2.03E-04	0.0000%
12	Methyl chloroform [1,1,1-TCA]	0.00E+00	1.63E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.63E-06	0.0000%
13	Ethyl chloride [Chloroethane]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
14	Vinyl chloride	0.00E+00	1.14E-07	0.00E+00	0.00E+00	0.00E+00	1.14E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.14E-07	0.00E+00	0.00E+00	1.14E-07	0.0001%
15	Acetaldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.20E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.20E-04	0.00E+00	0.00E+00	1.20E-04	0.1282%
16	Methylene chloride [Dichloromethane]	0.00E+00	5.27E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.27E-04	0.0000%
17	Carbon disulfide	0.00E+00	3.17E-07	0.00E+00	3.17E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.17E-07	0.00E+00	0.00E+00	0.00E+00	3.17E-07	0.0000%
18	1,1-Dichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
19	Vinylidene chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
20	1,2-Dichloropropane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
21	Methyl ethyl ketone [2-Butanone]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.68E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.68E-06	0.00E+00	0.00E+00	8.68E-06	0.0093%
22	Trichloroethylene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
23	Acrylamide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
24	1,1,2,2-Tetrachloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
25	Naphthalene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
26	Ethyl benzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
27	p-Dichlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
28	Ethylene dibromide [EDB]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
29	1,3-Butadiene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
30	Acrolein	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.03E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.03E-03	0.00E+00	0.00E+00	1.03E-03	1.1004%
31	Ethylene dichloride [EDC]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
32	Acrylonitrile	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
33	Toluene	0.00E+00	1.80E-04	0.00E+00	1.80E-04	0.00E+00	1.80E-04	0.00E+00	0.00E+00	0.00E+00	1.80E-04	1.80E-04	0.00E+00	0.00E+00	1.80E-04	0.1923%
34	Chlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
35	Hexane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
36	Glutaraldehyde	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
37	Propylene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
38	Triethylamine	0.00E+00	3.20E-05	0.00E+00	0.00E+00	0.00E+00	3.20E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.20E-05	0.0342%
39	1,4-Dioxane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.87E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.87E-05	0.00E+00	0.00E+00	7.87E-05	0.0841%
40	Perchloroethylene [Tetrachloroethene]	0.00E+00	1.07E-05	0.00E+00	0.00E+00	0.00E+00	1.07E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.07E-05	0.00E+00	0.00E+00	1.07E-05	0.0114%
41	Indeno[1,2,3-cd]pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
42	Benzo[b]fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
43	Benzo[k]fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
44	Chrysene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
45	Hydrazine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
46	Xylenes (mixed)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.42E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.42E-04	0.00E+00	0.00E+00	3.42E-04	0.3654%
47	2,3,7,8-Tetrachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
48	1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
49	Lead	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
50	Mercury	0.00E+00	9.33E-06	0.00E+00	9.33E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.33E-06	0.0000%
51	Hydrochloric acid	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.91E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.91E-03	0.00E+00	0.00E+00	3.91E-03	4.1774%
52	Hydrogen fluoride	0.00E+00	0.00E+00	0.00E+00	0.00E+00											

Acute HI for Maximum Sensitive Receptor - by Chemical
 Located at the the Woodland Clinic Medical Group

Chemical No.	Chemical	Cardiovascular system	Central Nervous System	Bone	Develop-mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
53	Nitric acid	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.46E-02	0.00E+00	0.00E+00	7.46E-02	0.0000%
54	Hydrogen sulfide	0.00E+00	2.47E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.47E-03	0.0000%
55	Phosphine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
56	Chromium, hexavalent (& compounds)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
57	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
58	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
59	1,2,3,4,6,7,8,9-Octachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
60	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
61	1,2,3,7,8-Pentachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
62	2,3,7,8-Tetrachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
63	1,2,3,4,7,8,9-Heptachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
64	2,3,4,7,8-Pentachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
65	1,2,3,7,8-Pentachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
66	1,2,3,6,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
67	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
68	2,3,4,6,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
69	1,2,3,4,6,7,8-Heptachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
70	1,2,3,4,7,8-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
71	1,2,3,7,8,9-Hexachlorodibenzofuran	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
SUM		0.00E+00	3.83E-02	0.00E+00	3.40E-02	0.00E+00	9.36E-02	1.34E-04	2.03E-04	0.00E+00	3.40E-02	8.40E-02	0.00E+00	2.03E-04	9.36E-02	100.0000%

Acute HI for Maximum Sensitive Receptor - by Source
 Located at the the Woodland Clinic Medical Group

Source No.	Source Identification	Cardiovascular system	Central Nervous System	Bone	Develop-mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
1	Central Heating and Cooling Plant Boiler #1	0.00E+00	0.00E+00	0.00E+00	5.55E-07	0.00E+00	6.76E-04	0.00E+00	5.55E-07	0.00E+00	5.55E-07	3.17E-06	0.00E+00	5.55E-07	6.76E-04	0.7222%
2	Central Heating and Cooling Plant Boiler #2	0.00E+00	0.00E+00	0.00E+00	5.40E-07	0.00E+00	6.57E-04	0.00E+00	5.40E-07	0.00E+00	5.40E-07	3.08E-06	0.00E+00	5.40E-07	6.57E-04	0.7019%
3	Central Heating and Cooling Plant Boiler #3	0.00E+00	0.00E+00	0.00E+00	6.41E-07	0.00E+00	7.81E-04	0.00E+00	6.41E-07	0.00E+00	6.41E-07	3.65E-06	0.00E+00	6.41E-07	7.81E-04	0.8344%
4	Central Heating and Cooling Plant Boiler #4, Natural Gas	0.00E+00	0.00E+00	0.00E+00	9.61E-07	0.00E+00	1.17E-03	0.00E+00	9.61E-07	0.00E+00	9.61E-07	5.48E-06	0.00E+00	9.61E-07	1.17E-03	1.2500%
5	Central Heating and Cooling Plant Boiler #4, Diesel	0.00E+00	8.71E-08	0.00E+00	4.28E-06	0.00E+00	1.36E-02	0.00E+00	4.20E-06	0.00E+00	4.28E-06	2.33E-07	0.00E+00	4.20E-06	1.36E-02	14.5299%
6	Primate Center Boiler No 1 Natural Gas	0.00E+00	0.00E+00	0.00E+00	3.17E-08	0.00E+00	3.86E-05	0.00E+00	3.17E-08	0.00E+00	3.17E-08	1.81E-07	0.00E+00	3.17E-08	3.86E-05	0.0412%
7	Primate Center Boiler No 2 Natural Gas	0.00E+00	0.00E+00	0.00E+00	3.20E-08	0.00E+00	3.90E-05	0.00E+00	3.20E-08	0.00E+00	3.20E-08	1.83E-07	0.00E+00	3.20E-08	3.90E-05	0.0417%
8	Primate Center Boiler No 2 Landfill Gas	0.00E+00	7.85E-06	0.00E+00	4.93E-06	0.00E+00	1.14E-02	6.50E-11	1.15E-08	0.00E+00	2.69E-08	8.69E-04	0.00E+00	1.15E-08	1.14E-02	12.1795%
9	Landfill Flare	0.00E+00	1.48E-07	0.00E+00	7.81E-08	0.00E+00	1.79E-04	5.11E-12	2.71E-10	0.00E+00	9.01E-10	1.36E-05	0.00E+00	2.71E-10	1.79E-04	0.1912%
10	Incinerator	0.00E+00	0.00E+00	0.00E+00	1.47E-10	0.00E+00	1.81E-04	0.00E+00	1.47E-10	0.00E+00	1.47E-10	1.81E-04	0.00E+00	1.47E-10	1.81E-04	0.1934%
11	ARS J-1 (H001)	0.00E+00	0.00E+00	0.00E+00	8.65E-08	0.00E+00	1.05E-04	0.00E+00	8.65E-08	0.00E+00	8.65E-08	4.92E-07	0.00E+00	8.65E-08	1.05E-04	0.1122%
12	ARS J-1 CAAN 3840 - 4 boilers	0.00E+00	0.00E+00	0.00E+00	1.43E-07	0.00E+00	1.74E-04	0.00E+00	1.43E-07	0.00E+00	1.43E-07	8.14E-07	0.00E+00	1.43E-07	1.74E-04	0.1859%
13	ARS K-2 Co-located 2 stacks	0.00E+00	0.00E+00	0.00E+00	2.70E-07	0.00E+00	3.29E-04	0.00E+00	2.70E-07	0.00E+00	2.70E-07	1.54E-06	0.00E+00	2.70E-07	3.29E-04	0.3515%
14	ARS K-2 (H040)	0.00E+00	0.00E+00	0.00E+00	5.28E-08	0.00E+00	6.43E-05	0.00E+00	5.28E-08	0.00E+00	5.28E-08	2.99E-07	0.00E+00	5.28E-08	6.43E-05	0.0687%
15	Contained Research	0.00E+00	0.00E+00	0.00E+00	2.65E-08	0.00E+00	3.23E-05	0.00E+00	2.65E-08	0.00E+00	2.65E-08	1.51E-07	0.00E+00	2.65E-08	3.23E-05	0.0345%
16	Environmental Horticulture K-1	0.00E+00	0.00E+00	0.00E+00	5.97E-08	0.00E+00	7.27E-05	0.00E+00	5.97E-08	0.00E+00	5.97E-08	3.40E-07	0.00E+00	5.97E-08	7.27E-05	0.0777%
17	Environmental Horticulture K-2	0.00E+00	0.00E+00	0.00E+00	4.20E-08	0.00E+00	5.12E-05	0.00E+00	4.20E-08	0.00E+00	4.20E-08	2.39E-07	0.00E+00	4.20E-08	5.12E-05	0.0547%
18	Environmental Services Facility A	0.00E+00	0.00E+00	0.00E+00	1.80E-07	0.00E+00	2.20E-04	0.00E+00	1.80E-07	0.00E+00	1.80E-07	1.03E-06	0.00E+00	1.80E-07	2.20E-04	0.2350%
19	Environmental Services Facility (3 per stack)	0.00E+00	0.00E+00	0.00E+00	1.82E-07	0.00E+00	2.22E-04	0.00E+00	1.82E-07	0.00E+00	1.82E-07	1.04E-06	0.00E+00	1.82E-07	2.22E-04	0.2372%
20	Genome Launch Facility (plant reproduction)	0.00E+00	0.00E+00	0.00E+00	8.62E-08	0.00E+00	1.05E-04	0.00E+00	8.62E-08	0.00E+00	8.62E-08	4.91E-07	0.00E+00	8.62E-08	1.05E-04	0.1122%
21	Equine Analytical Chemistry Lab	0.00E+00	0.00E+00	0.00E+00	5.36E-08	0.00E+00	6.53E-05	0.00E+00	5.36E-08	0.00E+00	5.36E-08	3.06E-07	0.00E+00	5.36E-08	6.53E-05	0.0698%
22	Housing - Castillian DC	0.00E+00	0.00E+00	0.00E+00	4.62E-08	0.00E+00	5.63E-05	0.00E+00	4.62E-08	0.00E+00	4.62E-08	2.63E-07	0.00E+00	4.62E-08	5.63E-05	0.0601%
23	Housing - Castillian DC	0.00E+00	0.00E+00	0.00E+00	1.10E-07	0.00E+00	1.34E-04	0.00E+00	1.10E-07	0.00E+00	1.10E-07	6.25E-07	0.00E+00	1.10E-07	1.34E-04	0.1432%
24	Comparative Medicine (Primate Center)	0.00E+00	0.00E+00	0.00E+00	4.62E-08	0.00E+00	5.64E-05	0.00E+00	4.62E-08	0.00E+00	4.62E-08	2.63E-07	0.00E+00	4.62E-08	5.64E-05	0.0603%
25	Contained Research	0.00E+00	0.00E+00	0.00E+00	4.23E-08	0.00E+00	5.15E-05	0.00E+00	4.23E-08	0.00E+00	4.23E-08	2.41E-07	0.00E+00	4.23E-08	5.15E-05	0.0550%
26	Institute of Ecology - West Campus	0.00E+00	0.00E+00	0.00E+00	4.47E-08	0.00E+00	5.44E-05	0.00E+00	4.47E-08	0.00E+00	4.47E-08	2.54E-07	0.00E+00	4.47E-08	5.44E-05	0.0581%
27	ITEH Geriatrics - cage wash inside co-located 3 stacks	0.00E+00	0.00E+00	0.00E+00	4.10E-08	0.00E+00	4.98E-05	0.00E+00	4.10E-08	0.00E+00	4.10E-08	2.33E-07	0.00E+00	4.10E-08	4.98E-05	0.0532%
28	ITEH Geriatrics Cagewash - outside - co-locate 3 stacks	0.00E+00	0.00E+00	0.00E+00	4.46E-08	0.00E+00	5.43E-05	0.00E+00	4.46E-08	0.00E+00	4.46E-08	2.53E-07	0.00E+00	4.46E-08	5.43E-05	0.0580%
29	Mondavi Ctr for Performing Arts - 2 boilers	0.00E+00	0.00E+00	0.00E+00	7.44E-08	0.00E+00	9.06E-05	0.00E+00	7.44E-08	0.00E+00	7.44E-08	4.24E-07	0.00E+00	7.44E-08	9.06E-05	0.0968%
30	Mondavi Ctr for Performing Arts	0.00E+00	0.00E+00	0.00E+00	4.04E-08	0.00E+00	4.92E-05	0.00E+00	4.04E-08	0.00E+00	4.04E-08	2.30E-07	0.00E+00	4.04E-08	4.92E-05	0.0526%
31	Rec Pool	0.00E+00	0.00E+00	0.00E+00	2.50E-07	0.00E+00	3.06E-04	0.00E+00	2.50E-07	0.00E+00	2.50E-07	1.43E-06	0.00E+00	2.50E-07	3.06E-04	0.3269%
32	Thoreau Hall - 2 stacks co-located	0.00E+00	0.00E+00	0.00E+00	3.80E-08	0.00E+00	4.64E-05	0.00E+00	3.80E-08	0.00E+00	3.80E-08	2.16E-07	0.00E+00	3.80E-08	4.64E-05	0.0496%
33	Air Stripper	0.00E+00	5.63E-05	0.00E+00	5.63E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.63E-05	0.00E+00	0.00E+00	0.00E+00	5.63E-05	0.0000%
34	In-well Stripper	0.00E+00	9.02E-05	0.00E+00	9.02E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.02E-05	0.00E+00	0.00E+00	0.00E+00	9.02E-05	0.0000%
35	Ground Water Treatment	0.00E+00	1.05E-04	0.00E+00	1.05E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.05E-04	0.00E+00	0.00E+00	0.00E+00	1.05E-04	0.0000%
36	Large Kiln	0.00E+00	1.27E-08	0.00E+00	1.50E-07	0.00E+00	5.29E-05	0.00E+00	1.37E-07	0.00E+00	1.50E-07	3.15E-05	0.00E+00	1.37E-07	5.29E-05	0.0565%
37	Raku Kiln	0.00E+00	2.29E-08	0.00E+00	2.70E-07	0.00E+00	9.52E-05	0.00E+00	2.47E-07	0.00E+00	2.70E-07	5.66E-05	0.00E+00	2.47E-07	9.52E-05	0.1017%
38	Foundry Kiln	0.00E+00	2.97E-08	0.00E+00	3.51E-07	0.00E+00	1.23E-04	0.00E+00	3.21E-07	0.00E+00	3.51E-07	7.33E-05	0.00E+00	3.21E-07	1.23E-04	0.1314%
39	Three Art Dept Kilns to roof vent	0.00E+00	5.91E-08	0.00E+00	7.01E-07	0.00E+00	2.46E-04	0.00E+00	6.42E-07	0.00E+00	7.01E-07	1.46E-04	0.00E+00	6.42E-07	2.46E-04	0.2628%
40	Storehouse/Bulk Receiving and Storage	0.00E+00	2.71E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.71E-06	0.0000%
41	Walnut Dryer	0.00E+00	2.07E-08	0.00E+00	2.44E-07	0.00E+00	8.59E-05	0.00E+00	2.23E-07	0.00E+00	2.44E-07	5.10E-05	0.00E+00	2.23E-07	8.59E-05	0.0918%
42	Temporary Building 187	0.00E+00	6.15E-05	0.00E+00	5.81E-05	0.00E+00	1.67E-04	3.40E-07	8.45E-08	0.00E+00	5.81E-05	1.90E-05	0.00E+00	8.45E-08	1.67E-04	0.1784%
43	Temporary Building 188	0.00E+00	4.98E-05	0.00E+00	4.70E-05	0.00E+00	1.35E-04	2.75E-07	6.85E-08	0.00E+00	4.70E-05	1.54E-05	0.00E+00	6.85E-08	1.35E-04	0.1442%
44	Veihmeyer	0.00E+00	6.37E-05	0.00E+00	5.21E-05	0.00E+00	2.66E-04	1.92E-06	4.27E-07	0.00E+00	5.21E-05	1.24E-03	0.00E+00	4.27E-07	1.24E-03	0.2842%
45	Enology	0.00E+00	1.28E-04	0.00E+00	1.21E-04	0.00E+00	3.50E-04	7.05E-07	1.76E-07	0.00E+00	1.21E-04	5.13E-04	0.00E+00	1.76E-07	5.13E-04	0.3739%
46	Wickson Hall	0.00E+00	2.38E-03	0.00E+00	2.25E-03	0.00E+00	6.52E-03	1.32E-05	3.28E-06	0.00E+00	2.25E-03	2.16E-02	0.00E+00	3.28E-06	2.16E-02	6.9658%
47	Hoagland	0.00E+00	1.47E-03	0.00E+00	1.39E-03	0.00E+00	3.98E-03	8.11E-06	2.02E-06	0.00E+00	1.39E-03	1.33E-03	0.00E+00	2.02E-06	3.98E-03	4.2521%
48	Mann Hall	0.00E+00	1.30E-04	0.00E+00	1.23E-04	0.00E+00	3.51E-04	7.17E-07	1.79E-07	0.00E+00	1.23E-04	1.67E-04	0.00E+00	1.79E-07	3.52E-04	0.3750%
49	Storer Hall	0.00E+00	4.09E-05	0.00E+00	3.86E-05	0.00E+00	1.11E-04	2.26E-07	5.62E-08	0.00E+00	3.86E-05	1.99E-04	0.00E+00	5.62E-08	1.99E-04	0.1186%
50	Hutchison Hall/Biological Sci Unit 2	0.00E+00	7.82E-04	0.00E+00	7.38E-04	0.00E+00	2.12E-03	4.32E-06	1.08E-06	0.00E+00	7.38E-04	1.39E-03	0.00E+00	1.08E-06	2.12E-03	2.2650%
51	Asmundson Hall	0.00E+00	9.00E-04	0.00E+00	8.51E-04	0.00E+00	2.44E-03	4.96E-06	1.24E-06	0.00E+00	8.51E-04	2.58E-03	0.00E+00	1.24E-06	2.58E-03	2.6068%
52	Robbins Hall	0.00E+00	3.73E-04	0.00E+00	3.52E-04	0.00E+00	1.01E-03	2.06E-06	5.13E-07	0.00E+00	3.52E-04	4.52E-04	0.00E+00	5.13E-07	1.01E-03	1.0791%
53	Temporary Building 202	0.00E+00	3.01E-05	0.00E+00	2.84E-05	0.00E+00	8.16E-05	1.66E-07	4.14E-08	0.00E+00	2.84E-05	9.31E-06	0.00E+00	4.14E-08	8.16E-05	0.0872%
54	Briggs Hall and Life Sciences	0.00E+00	2.27E-03	0.00E+00	2.14E-03	0.00E+00	6.16E-03	1.25E-05	3.12E-06	0.00E+00	2.14E-03	6.23E-03	0.00E+00	3.12E-06	6.23E-03	6.5812%
55	Temporary Building 194	0.00E+00	3.78E-05	0.00E+00	3.57E-05	0.00E+00	1.03E-04	2.10E-07	5.20E-08	0.00E+00	3.57E-05	2.72E-04	0.00E+00	5.20E-08	2.72E-04	0.1100%
56	Food Science	0.00E+00	3.97E-06	0.00E+00	3.75E-06	0.00E+00	1.08E-05	2.20E-08	5.46E-09	0.00E+00	3.75E-06	1.23E-06	0.00E+00	5.46E-09	1.08E-05	0.0115%
57	Temporary Building 193															

Acute HI for Maximum Sensitive Receptor - by Source
 Located at the the Woodland Clinic Medical Group

Source No.	Source Identification	Cardiovascular system	Central Nervous System	Bone	Develop- mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
62	Temporary Building 155	0.00E+00	2.36E-05	0.00E+00	2.23E-05	0.00E+00	6.40E-05	1.30E-07	3.24E-08	0.00E+00	2.23E-05	7.29E-06	0.00E+00	3.24E-08	6.40E-05	0.0684%
63	Temporary Building 156	0.00E+00	2.18E-05	0.00E+00	2.06E-05	0.00E+00	5.92E-05	1.21E-07	3.01E-08	0.00E+00	2.06E-05	6.76E-06	0.00E+00	3.01E-08	5.92E-05	0.0632%
64	Temporary Building 157	0.00E+00	1.91E-05	0.00E+00	1.80E-05	0.00E+00	5.17E-05	1.06E-07	2.62E-08	0.00E+00	1.80E-05	5.91E-06	0.00E+00	2.62E-08	5.17E-05	0.0552%
65	Temporary Building 151	0.00E+00	2.77E-05	0.00E+00	2.62E-05	0.00E+00	7.54E-05	1.53E-07	3.82E-08	0.00E+00	2.62E-05	8.59E-06	0.00E+00	3.82E-08	7.54E-05	0.0806%
66	Temporary Building 149	0.00E+00	1.79E-05	0.00E+00	1.69E-05	0.00E+00	4.86E-05	9.89E-08	2.46E-08	0.00E+00	1.69E-05	5.54E-06	0.00E+00	2.46E-08	4.86E-05	0.0519%
67	Temporary Building 153	0.00E+00	1.39E-05	0.00E+00	1.31E-05	0.00E+00	3.76E-05	7.67E-08	1.91E-08	0.00E+00	1.31E-05	4.29E-06	0.00E+00	1.91E-08	3.76E-05	0.0402%
68	Temporary Building 158	0.00E+00	1.37E-05	0.00E+00	1.30E-05	0.00E+00	3.73E-05	7.60E-08	1.89E-08	0.00E+00	1.30E-05	4.25E-06	0.00E+00	1.89E-08	3.73E-05	0.0399%
69	Engineering II	0.00E+00	1.04E-03	0.00E+00	9.84E-04	0.00E+00	2.47E-05	5.62E-07	9.21E-06	0.00E+00	9.84E-04	9.30E-04	0.00E+00	9.21E-06	1.04E-03	0.0264%
70	Walker Hall	0.00E+00	9.86E-06	0.00E+00	8.06E-06	0.00E+00	4.11E-05	2.98E-07	6.62E-08	0.00E+00	8.06E-06	2.01E-05	0.00E+00	6.62E-08	4.11E-05	0.0439%
71	Chemistry	0.00E+00	4.13E-03	0.00E+00	3.91E-03	0.00E+00	9.97E-05	2.24E-06	3.67E-05	0.00E+00	3.91E-03	3.33E-03	0.00E+00	3.67E-05	4.13E-03	0.1065%
72	Chemistry Annex	0.00E+00	2.56E-03	0.00E+00	2.42E-03	0.00E+00	6.11E-05	1.39E-06	2.27E-05	0.00E+00	2.42E-03	1.13E-03	0.00E+00	2.27E-05	2.56E-03	0.0653%
73	Bainer Hall	0.00E+00	2.12E-03	0.00E+00	2.01E-03	0.00E+00	5.09E-05	1.15E-06	1.88E-05	0.00E+00	2.01E-03	1.33E-03	0.00E+00	1.88E-05	2.12E-03	0.0544%
74	Crocker Hall	0.00E+00	7.22E-06	0.00E+00	5.90E-06	0.00E+00	3.01E-05	2.18E-07	4.84E-08	0.00E+00	5.90E-06	1.17E-04	0.00E+00	4.84E-08	1.17E-04	0.0322%
75	Academic Surge	0.00E+00	1.56E-04	0.00E+00	1.48E-04	0.00E+00	4.23E-04	8.67E-07	2.15E-07	0.00E+00	1.48E-04	2.14E-04	0.00E+00	2.15E-07	4.23E-04	0.4519%
76	Meyer Hall	0.00E+00	9.01E-04	0.00E+00	8.52E-04	0.00E+00	2.44E-03	4.98E-06	1.24E-06	0.00E+00	8.52E-04	3.38E-03	0.00E+00	1.24E-06	3.38E-03	2.6068%
77	Physics/Geology/Physics Unit 1	0.00E+00	2.79E-05	0.00E+00	2.28E-05	0.00E+00	1.17E-04	8.43E-07	1.88E-07	0.00E+00	2.28E-05	2.79E-03	0.00E+00	1.88E-07	2.79E-03	0.1250%
78	Environmental Horticulture	0.00E+00	1.48E-04	0.00E+00	1.40E-04	0.00E+00	4.02E-04	8.19E-07	2.04E-07	0.00E+00	1.40E-04	7.28E-05	0.00E+00	2.04E-07	4.02E-04	0.4295%
79	Thurman Hall	0.00E+00	1.11E-04	0.00E+00	1.05E-04	0.00E+00	3.03E-04	6.13E-07	1.53E-07	0.00E+00	1.05E-04	1.59E-04	0.00E+00	1.53E-07	3.03E-04	0.3237%
80	Maddy Hall	0.00E+00	1.80E-04	0.00E+00	1.70E-04	0.00E+00	4.87E-04	9.94E-07	2.47E-07	0.00E+00	1.70E-04	4.05E-04	0.00E+00	2.47E-07	4.88E-04	0.5203%
81	Tupper Hall	0.00E+00	9.78E-04	0.00E+00	9.23E-04	0.00E+00	2.66E-03	5.41E-06	1.34E-06	0.00E+00	9.23E-04	1.83E-03	0.00E+00	1.34E-06	2.66E-03	2.8419%
82	VET MED 2	0.00E+00	1.02E-04	0.00E+00	9.68E-05	0.00E+00	2.79E-04	5.67E-07	1.41E-07	0.00E+00	9.68E-05	1.61E-04	0.00E+00	1.41E-07	2.79E-04	0.2981%
83	Asmundson Annex	0.00E+00	8.10E-05	0.00E+00	7.65E-05	0.00E+00	2.20E-04	4.47E-07	1.12E-07	0.00E+00	7.65E-05	2.51E-05	0.00E+00	1.12E-07	2.20E-04	0.2350%
84	Young Hall	0.00E+00	2.57E-04	0.00E+00	2.43E-04	0.00E+00	6.98E-04	1.42E-06	3.54E-07	0.00E+00	2.43E-04	4.32E-04	0.00E+00	3.54E-07	6.98E-04	0.7457%
85	Temporary Building 9	0.00E+00	2.47E-05	0.00E+00	2.34E-05	0.00E+00	6.70E-05	1.37E-07	3.41E-08	0.00E+00	2.34E-05	7.65E-06	0.00E+00	3.41E-08	6.71E-05	0.0716%
86	ARS H-1 (Vet Meta Res)	0.00E+00	2.35E-07	0.00E+00	1.92E-07	0.00E+00	9.81E-07	7.09E-09	1.58E-09	0.00E+00	1.92E-07	4.79E-07	0.00E+00	1.58E-09	9.81E-07	0.0010%
87	Serology4	0.00E+00	3.50E-05	0.00E+00	3.30E-05	0.00E+00	9.50E-05	1.93E-07	4.82E-08	0.00E+00	3.30E-05	1.08E-05	0.00E+00	4.82E-08	9.50E-05	0.1015%
88	ARS R-1	0.00E+00	1.71E-06	0.00E+00	1.62E-06	0.00E+00	4.63E-06	9.48E-09	2.35E-09	0.00E+00	1.62E-06	5.29E-07	0.00E+00	2.35E-09	4.63E-06	0.0049%
89	ARS R-2	0.00E+00	2.36E-05	0.00E+00	2.23E-05	0.00E+00	6.41E-05	1.30E-07	3.25E-08	0.00E+00	2.23E-05	7.31E-06	0.00E+00	3.25E-08	6.41E-05	0.0685%
90	Center For Comparative Medicine	0.00E+00	6.68E-05	0.00E+00	6.31E-05	0.00E+00	1.81E-04	3.69E-07	9.18E-08	0.00E+00	6.31E-05	6.68E-05	0.00E+00	9.18E-08	1.81E-04	0.1934%
91	Primate Center	0.00E+00	4.92E-05	0.00E+00	4.64E-05	0.00E+00	1.33E-04	2.73E-07	6.78E-08	0.00E+00	4.64E-05	1.52E-05	0.00E+00	6.78E-08	1.33E-04	0.1421%
92	Temporary Building 184	0.00E+00	1.71E-05	0.00E+00	1.61E-05	0.00E+00	4.63E-05	9.44E-08	2.35E-08	0.00E+00	1.61E-05	5.28E-06	0.00E+00	2.35E-08	4.63E-05	0.0495%
93	Temporary Building 160	0.00E+00	5.78E-06	0.00E+00	5.46E-06	0.00E+00	1.57E-05	3.19E-08	7.94E-09	0.00E+00	5.46E-06	1.78E-06	0.00E+00	7.94E-09	1.57E-05	0.0168%
94	APCARU	0.00E+00	7.41E-06	0.00E+00	7.01E-06	0.00E+00	2.01E-05	4.09E-08	1.02E-08	0.00E+00	7.01E-06	2.29E-06	0.00E+00	1.02E-08	2.01E-05	0.0215%
95	Ecology Lab (Aquadic Bio in bldg DB)	0.00E+00	1.28E-05	0.00E+00	1.21E-05	0.00E+00	3.48E-05	7.08E-08	1.76E-08	0.00E+00	1.21E-05	1.32E-04	0.00E+00	1.76E-08	1.32E-04	0.0372%
96	Temporary Building 1	0.00E+00	5.86E-06	0.00E+00	5.53E-06	0.00E+00	1.59E-05	3.24E-08	8.07E-09	0.00E+00	5.53E-06	1.81E-06	0.00E+00	8.07E-09	1.59E-05	0.0170%
97	ITEH Cellular Biology	0.00E+00	1.62E-05	0.00E+00	1.53E-05	0.00E+00	4.38E-05	8.94E-08	2.22E-08	0.00E+00	1.53E-05	4.99E-06	0.00E+00	2.22E-08	4.38E-05	0.0468%
98	ITEH Pathology Clinic	0.00E+00	2.00E-05	0.00E+00	1.89E-05	0.00E+00	5.43E-05	1.10E-07	2.75E-08	0.00E+00	1.89E-05	6.18E-06	0.00E+00	2.75E-08	5.43E-05	0.0580%
99	ARS DL-10/Field Shelter 5; Bovine Shed (Luekemia Lab)	0.00E+00	7.50E-06	0.00E+00	7.08E-06	0.00E+00	2.03E-05	4.15E-08	1.03E-08	0.00E+00	7.08E-06	2.32E-06	0.00E+00	1.03E-08	2.03E-05	0.0217%
100	Cole Fac A	0.00E+00	2.13E-05	0.00E+00	2.01E-05	0.00E+00	5.77E-05	1.17E-07	2.92E-08	0.00E+00	2.01E-05	6.58E-06	0.00E+00	2.92E-08	5.77E-05	0.0616%
101	Cole Fac B	0.00E+00	1.87E-05	0.00E+00	1.76E-05	0.00E+00	5.07E-05	1.04E-07	2.56E-08	0.00E+00	1.76E-05	5.78E-06	0.00E+00	2.56E-08	5.07E-05	0.0542%
102	Cole Fac C	0.00E+00	2.43E-05	0.00E+00	2.30E-05	0.00E+00	6.60E-05	1.35E-07	3.35E-08	0.00E+00	2.30E-05	7.53E-06	0.00E+00	3.35E-08	6.60E-05	0.0705%
103	TB 31	0.00E+00	4.40E-06	0.00E+00	4.16E-06	0.00E+00	1.20E-05	2.43E-08	6.05E-09	0.00E+00	4.16E-06	1.36E-06	0.00E+00	6.05E-09	1.20E-05	0.0128%
104	TB 33	0.00E+00	2.01E-05	0.00E+00	1.90E-05	0.00E+00	5.44E-05	1.11E-07	2.76E-08	0.00E+00	1.90E-05	6.21E-06	0.00E+00	2.76E-08	5.44E-05	0.0581%
105	TB 164	0.00E+00	2.69E-05	0.00E+00	2.54E-05	0.00E+00	7.32E-05	1.49E-07	3.71E-08	0.00E+00	2.54E-05	1.78E-04	0.00E+00	3.71E-08	1.78E-04	0.0782%
106	TB 165	0.00E+00	2.62E-05	0.00E+00	2.48E-05	0.00E+00	7.13E-05	1.45E-07	3.62E-08	0.00E+00	2.48E-05	8.13E-06	0.00E+00	3.62E-08	7.13E-05	0.0762%
107	TB 205	0.00E+00	2.59E-05	0.00E+00	2.44E-05	0.00E+00	7.03E-05	1.43E-07	3.55E-08	0.00E+00	2.44E-05	7.33E-05	0.00E+00	3.55E-08	7.33E-05	0.0751%
108	HH1	0.00E+00	3.97E-06	0.00E+00	3.75E-06	0.00E+00	1.08E-05	2.19E-08	5.45E-09	0.00E+00	3.75E-06	1.23E-06	0.00E+00	5.45E-09	1.08E-05	0.0115%
109	HH2	0.00E+00	9.53E-06	0.00E+00	9.00E-06	0.00E+00	2.59E-05	5.28E-08	1.31E-08	0.00E+00	9.00E-06	2.95E-06	0.00E+00	1.31E-08	2.59E-05	0.0277%
110	HH3	0.00E+00	3.42E-06	0.00E+00	3.23E-06	0.00E+00	9.28E-06	1.89E-08	4.71E-09	0.00E+00	3.23E-06	1.06E-06	0.00E+00	4.71E-09	9.28E-06	0.0099%
111	HH6	0.00E+00	3.16E-05	0.00E+00	2.99E-05	0.00E+00	8.57E-05	1.75E-07	4.35E-08	0.00E+00	2.99E-05	3.10E-04	0.00E+00	4.35E-08	3.10E-04	0.0916%
112	Vet Med Teaching Hospital (VMTH)	0.00E+00	3.39E-05	0.00E+00	3.20E-05	0.00E+00	9.18E-05	1.87E-07	4.65E-08	0.00E+00	3.20E-05	9.18E-05	0.00E+00	4.65E-08	9.18E-05	0.0981%
113	ARS Iso Barn J bldg	0.00E+00	1.10E-06	0.00E+00	1.04E-06	0.00E+00	2.97E-06	6.07E-09	1.51E-09	0.00E+00	1.04E-06	3.39E-07	0.00E+00	1.51E-09	2.97E-06	0.0032%
114	ITEH Animal Housing-2	0.00E+00	4.89E-06	0.00E+00	3.99E-06	0.00E+00	2.04E-05	1.48E-07	3.28E-08	0.00E+00	3.99E-06	2.74E-05	0.00E+00	3.28E-08	2.74E-05	0.0218%
115	LEHR Lab and Office	0.00E+00	5.74E-06	0.00E+00	4.69E-06	0.00E+00	2.39E-05	1.73E-07	3.85E-08	0.00E+00	4.69E-06	1.17E-05	0.00E+00	3.85E-08	2.39E-05	0.0255%
116	ITEH Toxic Pollutant Lab	0.00E+00	3.43E-06	0.00E+00	2.80E-06	0.00E+00	1.43E-05	1.04E-07	2.31E-08	0.00E+00	2.80E-06	2.13E-04	0.00E+00	2.31E-08	2.13E-04	0.0153%
117	Aqua weed lab/Aq Tox Shelter 5	0.00E+00	1.23E-05	0.00E+00	1.16E-05	0.00E+00	3.33E-05	6.78E-08	1.68E-08	0.00E+00	1.16E-05	5.73E-05	0.00E+00	1.68E-08	5.73E-05	0.0356%
118	Bee Biology	0.00E+00	5.85E-06	0.00E+00	5.53E-06	0.00E+00	1									

Acute HI for Maximum Sensitive Receptor - by Source
 Located at the the Woodland Clinic Medical Group

Source No.	Source Identification	Cardiovascular system	Central Nervous System	Bone	Develop- mental	Endocrine System	Eyes	Alimentary System	Immune system	Kidneys	Reproductive System	Respiratory System	Skin	Blood	Maximum	Percent of Total
184	No Permit Old Fire Station	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
185	P-29-96(a) Physical Plant	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
186	P-120-01 Plant Envir Sci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
187	P-50-99(a) Port Gen # 1	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
188	No Permit Port Gen # 14	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
189	P-51-99(a) Port Gen # 2	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
190	P-52-99(a) Port Gen # 3	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
191	P-86-01 Port Gen # 7	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
192	P-87-01 Port Gen # 8	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
193	P-49-07 Pri Animal Hous # 1	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
194	P-31-98 Primate Animal	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
195	P-32-98 Primate CCM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
196	P-69-96(a) Primate Freezers	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
197	P-102-94(a) Primate Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
198	P-15-98 Primate Quarantine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
199	No Permit Primate Sew Life Sta	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
200	P-16-98 Primate TB 184	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
201	P-108-01 Primate TB North # 5	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
202	P-109-01 Primate TB South # 6	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
203	P-99-94(a) Quad Parking	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
204	P-93-94(a) Rec Hall	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
205	P-111-95(a) Schl of Med Neurosci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
206	P-123-01 Schl of Med Neurosci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
207	P-15-04 Science Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
208	P-74-05 Segundo Dinning	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
209	P-126-95(a) Social Sci	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
210	P-17-02 South Parking	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
211	P-92-94(a) Storm Lift # 4	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
212	C-09-129 Storm Lift #4 (new)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
213	P-71-00 Targeted genomics	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
214	P-111-01 Tele Comm.	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
215	P-91-94(a) Thurman Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
216	P-100-94(a) Toxic Pollutant	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
217	P-17-09 TURF	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
218	P-121-03 Tupper Load Dock	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
219	P-209-95(a) Util Well 6A	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
220	P-07-01 Vega Crops	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
221	P-63-03 Vet Lab	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
222	P-52-07 Vet Med 3A	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
223	P-53-07 Vet Med 3A	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
224	P-59-05 Watershed Sic	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
225	P-38-05 West Entry Park	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
226	P-96-94(a) WEPT Influent	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
227	P-88-99 WEPT South	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000%
228	Landfill	0.00E+00	3.12E-04	0.00E+00	3.67E-06	0.00E+00	1.25E-05	3.48E-09	1.23E-06	0.00E+00	2.62E-06	1.25E-05	0.00E+00	1.23E-06	3.12E-04	0.0134%
229	Landfill	0.00E+00	2.73E-04	0.00E+00	3.20E-06	0.00E+00	1.10E-05	3.04E-09	1.08E-06	0.00E+00	2.29E-06	1.10E-05	0.00E+00	1.08E-06	2.73E-04	0.0118%
230	Landfill	0.00E+00	3.43E-04	0.00E+00	4.02E-06	0.00E+00	1.38E-05	3.82E-09	1.35E-06	0.00E+00	2.87E-06	1.38E-05	0.00E+00	1.35E-06	3.43E-04	0.0147%
231	Landfill	0.00E+00	3.67E-04	0.00E+00	4.30E-06	0.00E+00	1.47E-05	4.09E-09	1.44E-06	0.00E+00	3.07E-06	1.47E-05	0.00E+00	1.44E-06	3.67E-04	0.0157%
232	Waste Water Treatment Plant	0.00E+00	2.45E-03	0.00E+00	1.23E-03	0.00E+00	4.80E-04	0.00E+00	2.37E-07	0.00E+00	1.23E-03	4.80E-04	0.00E+00	2.37E-07	2.45E-03	0.5128%
233	Grounds Above-ground Storage Tank	0.00E+00	1.02E-08	0.00E+00	3.47E-07	0.00E+00	1.29E-08	0.00E+00	3.36E-07	0.00E+00	3.47E-07	1.29E-08	0.00E+00	3.36E-07	3.47E-07	0.0000%
234	Fleet Services Underground Storage Tank	0.00E+00	1.30E-06	0.00E+00	4.42E-05	0.00E+00	1.65E-06	0.00E+00	4.29E-05	0.00E+00	4.42E-05	1.65E-06	0.00E+00	4.29E-05	4.42E-05	0.0018%
235	Primate Center Gasoline AST	0.00E+00	3.35E-10	0.00E+00	1.14E-08	0.00E+00	4.26E-10	0.00E+00	1.11E-08	0.00E+00	1.14E-08	4.26E-10	0.00E+00	1.11E-08	1.14E-08	0.0000%
236	Agricultural Services AST	0.00E+00	2.80E-08	0.00E+00	9.51E-07	0.00E+00	3.56E-08	0.00E+00	9.23E-07	0.00E+00	9.51E-07	3.56E-08	0.00E+00	9.23E-07	9.51E-07	0.0000%
237	Plant Pathology Storage Tank	0.00E+00	2.26E-10	0.00E+00	7.70E-09	0.00E+00	2.87E-10	0.00E+00	7.47E-09	0.00E+00	7.70E-09	2.87E-10	0.00E+00	7.47E-09	7.70E-09	0.0000%
238	Pomology Above Ground Storage Tank	0.00E+00	4.06E-10	0.00E+00	1.38E-08	0.00E+00	5.16E-10	0.00E+00	1.34E-08	0.00E+00	1.38E-08	5.16E-10	0.00E+00	1.34E-08	1.38E-08	0.0000%
239																