DOCKETED

Docket Number:	15-AFC-02
Project Title:	Mission Rock Energy Center
TN #:	207160-12
Document Title:	Appendix 5.1D Health Risk Assessment Support Data
Description:	Application for Certification (Vol. 2)
Filer:	Sabrina Savala
Organization:	Mission Rock Energy Center, LLLC
Submitter Role:	Applicant
Submission Date:	12/31/2015 11:45:17 AM
Docketed Date:	12/31/2015

Appendix 5.1D
HRA Support Data

Health Risk Assessment Support Data

Health Risk Assessment Process, Goals, Assumptions, and Uses

“In recent years, the public has become increasingly aware of the presence of harmful chemicals in our environment. Many people express concerns about pesticides and other foreign substances in food, contaminants in drinking water, and toxic pollutants in the air. Others believe these concerns are exaggerated or unwarranted. How can we determine which of these potential hazards really deserve attention? How do we, as a society, decide where to focus our efforts and resources to control these hazards? When we hear about toxic threats that affect us personally, such as the discovery of industrial waste buried in our neighborhood or near our children’s school, how concerned should we be?

Health risk assessment is a scientific tool designed to help answer these questions. Government agencies rely on risk assessments to help them determine which potential hazards are the most significant. Risk assessments can also guide regulators in abating environmental hazards. Members of the public who learn the basics of risk assessment can improve their understanding of both real and perceived environmental hazards, and they can work more effectively with decision makers on solutions to environmental problems.

Chemicals can be either beneficial or harmful, depending on a number of factors, such as the amounts to which we are exposed. Low levels of some substances may be necessary for good health, but higher levels may be harmful. Health risk assessments are used to determine if a particular chemical poses a significant risk to human health and, if so, under what circumstances. Could exposure to a specific chemical cause significant health problems? How much of the chemical would someone have to be exposed to before it would be dangerous? How serious could the health risks be? What activities might put people at increased risk?

If it were possible to prevent all human exposure to all hazardous chemicals, there would be no need for risk assessment. However, the total removal of harmful pollutants from the environment is often infeasible or impossible, and many naturally occurring substances also pose health risks. Risk assessment helps scientists and regulators identify serious health hazards and determine realistic goals for reducing exposure to toxics so that there is no significant health threat to the public.

Estimating the hazards posed by toxic chemicals in the environment involves the compilation and evaluation of complex sets of data. Government regulators, therefore, turn to specialists to perform or assist with risk assessments. These specialists include scientists with degrees in toxicology (the study of the toxic effects of chemicals) and epidemiology (the study of disease or illness in populations) as well as physicians, biologists, chemists, and engineers.

The term “health risk assessment” is often misinterpreted. People sometimes think that a risk assessment will tell them whether a current health problem or symptom was caused by exposure to a chemical. This is not the case. Scientists who are searching for links between chemical exposures and health problems in a community may conduct an epidemiologic study. These studies typically include a survey of health problems in a community and a comparison of health problems in that community with those in other cities, communities, or the population as a whole.

Although they are both important, health risk assessments and epidemiologic studies have different objectives. Most epidemiologic studies evaluate whether past chemical exposures may be responsible for documented health problems in a specific group of people. In contrast, health risk assessments are

used to estimate whether current or future chemical exposures will pose health risks to a broad population, such as a city or a community. Scientific methods used in health risk assessment cannot be used to link individual illnesses to past chemical exposures, nor can health risk assessments and epidemiologic studies prove that a specific toxic substance caused an individual's illness.

The U.S. Environmental Protection Agency (U.S. EPA) is a leading risk assessment agency at the federal level. In California, the Office of Environmental Health Hazard Assessment (OEHHA) in the California Environmental Protection Agency (Cal/EPA) has the primary responsibility for developing procedures and practices for performing health risk assessments. Other agencies within Cal/EPA, such as the Department of Pesticide Regulation and the Department of Toxic Substances Control, have extensive risk assessment programs of their own but work closely with OEHHA.

The Department of Pesticide Regulation uses risk assessments to make regulatory decisions concerning safe pesticide uses. The Department of Toxic Substances Control uses risk assessments to determine requirements for the management and cleanup of hazardous wastes. OEHHA's health risk assessments are used by the Air Resources Board to develop regulations governing toxic air contaminants, and by the Department of Health Services to develop California's drinking water standards. These agencies' decisions take into account the seriousness of potential health effects along with the economic and technical feasibility of measures that can reduce the health risks.

Health risk assessment requires both sound science and professional judgment and is a constantly developing process. Cal/EPA is nationally recognized for developing new procedures that improve the accuracy of risk assessments. Cal/EPA also works closely with U.S. EPA in all phases of risk assessment.

The risk assessment process is typically described as consisting of four basic steps: hazard identification, exposure assessment, dose-response assessment, and risk characterization. Each of these steps will be explained in the following text.

Hazard Identification

In the first step, hazard identification, scientists determine the types of health problems a chemical could cause by reviewing studies of its effects in humans and laboratory animals. Depending on the chemical, these health effects may include short-term ailments, such as headaches; nausea; and eye, nose, and throat irritation; or chronic diseases, such as cancer. Effects on sensitive populations, such as pregnant women and their developing fetuses, the elderly, or those with health problems (including those with weakened immune systems), must also be considered. Responses to toxic chemicals will vary depending on the amount and length of exposure. For example, short-term exposure to low concentrations of chemicals may produce no noticeable effect, but continued exposure to the same levels of chemicals over a long period of time may eventually cause harm.

An important step in hazard identification is the selection of key research studies that can provide accurate, timely information on the hazards posed to humans by a particular chemical. The selection of a study is based upon factors such as whether the study has been peer reviewed by qualified scientists, whether the study's findings have been verified by other studies, and the species tested (human studies provide the best evidence). Some studies may involve humans that have been exposed to the chemical, while others may involve studies with laboratory animals.

Human data frequently are useful in evaluating human health risks associated with chemical exposures. Human epidemiologic studies typically examine the effects of chemical exposure on a large number of people, such as employees exposed to varying concentrations of chemicals in the workplace. In many cases, these exposures took place prior to the introduction of modern worker-safety measures.

One weakness of occupational studies is that they generally measure the effects of chemicals on healthy workers and do not consider children, the elderly, those with pre-existing medical conditions, or other sensitive groups. Since occupational studies are not controlled experiments, there may be uncertainties

about the amount and duration of exposure or the influence of lifestyle choices, such as smoking or alcohol use, on the health of workers in the studies. Exposure of workers to other chemicals at the same time may also influence and complicate the results.

Laboratory studies using human volunteers are better able to gauge some health effects because chemical exposures can then be measured with precision. But these studies usually involve small numbers of people and, in conformance with ethical and legal requirements, use only adults who agree to participate in the studies. Moreover, laboratory studies often use simple measurements that identify immediate responses to the chemical but might miss significant, longer-term health effects. Scientists can also use physicians' case reports of an industrial or transportation accident in which individuals were unintentionally exposed to a chemical. However, these reports may involve very small numbers of people, and the level of exposure to the chemical could be greater than exposures to the same chemical in the environment. Nevertheless, human studies are preferred for risk assessment, so OEHHA makes every effort to use them when they are available.

Because the effects of the vast majority of chemicals have not been studied in humans, scientists must often rely on animal studies to evaluate a chemical's health effects. Animal studies have the advantage of being performed under controlled laboratory conditions that reduce much of the uncertainty related to human studies. If animal studies are used, scientists must determine whether a chemical's health effects in humans are likely to be similar to those in the animals tested. Although effects seen in animals can also occur in humans, there may be subtle or even significant differences in the ways humans and experimental animals react to a chemical. Comparison of human and animal metabolism may be useful in selecting the animal species that should be studied, but it is often not possible to determine which species is most like humans in its response to a chemical exposure. However, if similar effects were found in more than one species, the results would strengthen the evidence that humans may also be at risk.

Exposure Assessment

In exposure assessment, scientists attempt to determine how long people were exposed to a chemical; how much of the chemical they were exposed to; whether the exposure was continuous or intermittent; and how people were exposed—through eating, drinking water and other liquids, breathing, or skin contact. All of this information is combined with factors such as breathing rates, water consumption, and daily activity patterns to estimate how much of the chemical was taken into the bodies of those exposed.

People can be exposed to toxic chemicals in various ways. These substances can be present in the air we breathe, the food we eat, or the water we drink. Some chemicals, due to their particular characteristics, may be both inhaled and ingested. For example, airborne chemicals can settle on the surface of water, soil, leaves, fruits, vegetables, and forage crops used as animal feed. Cows, chickens, or other livestock can become contaminated when eating, drinking, or breathing the chemicals present in the air, water, feed, and soil. Fish can absorb the chemicals as they swim in contaminated water or ingest contaminated food. Chemicals can be absorbed through the skin, so infants and children can be exposed simply by crawling or playing in contaminated dirt. They can also ingest chemicals if they put their fingers or toys in their mouths after playing in contaminated dirt. Chemicals can also be passed on from nursing mothers to their children through breast milk.

To estimate exposure levels, scientists rely on air, water, and soil monitoring; human blood and urine samples; or computer modeling. Although monitoring of a pollutant provides excellent data, it is time consuming, costly, and typically limited to only a few locations. For those reasons, scientists often rely on computer modeling, which uses mathematical equations to describe how a chemical is released and to estimate the speed and direction of its movement through the surrounding environment. Modeling

has the advantage of being relatively inexpensive and less time consuming, provided all necessary information is available and the accuracy of the model can be verified through testing.

Computer modeling is often used to assess chemical releases from industrial facilities. Such models require information on the type of chemicals released, facilities' hours of operation, industrial processes that release the chemicals, smokestack height and temperature, any pollution-control equipment that is used, surrounding land type (urban or rural), local topography and meteorology, and census data regarding the exposed population.

In all health risk assessments, scientists must make assumptions in order to estimate human exposure to a chemical. For example, scientists assessing the effects of air pollution may need to make assumptions about the time people spend outdoors, where they are more directly exposed to pollutants in the ambient air, or the time they spend in an area where the pollution is greatest. An assessment of soil contamination may require scientists to make assumptions about people's consumption of fruits and vegetables that may absorb soil contaminants.

To avoid underestimating actual human exposure to a chemical, scientists often look at the range of possible exposures. For example, people who jog in the afternoon, when urban air pollution levels are highest, would have much higher exposures to air pollutants than people who come home after work and relax indoors. Basing an exposure estimate on a value near the higher end of a range of exposure levels (closer to the levels experienced by the jogger than by the person remaining indoors) provides a realistic worst-case estimate of exposure. These kinds of conservative assumptions, which presume that people are exposed to the highest amounts of a chemical that can be considered credible, are referred to as "health-protective" assumptions.

The exposure estimates for the project analysis were conducted using HARP2. HARP2 (version 2.0.3) is currently the approved model for use in assessing health risks from facilities such as the MREC project.

Dose-Response Assessment

In dose-response assessment, scientists evaluate the information obtained during the hazard identification step to estimate the amount of a chemical that is likely to result in a particular health effect in humans.

An established principle in toxicology is that "the dose makes the poison." For example, a commonplace chemical like table salt is harmless in small quantities, but it can cause illness in large doses. Similarly, hydrochloric acid, a hazardous chemical, is produced naturally in our stomachs but can be quite harmful if taken in large doses.

Scientists perform a dose-response assessment to estimate how different levels of exposure to a chemical can impact the likelihood and severity of health effects. The dose-response relationship is often different for many chemicals that cause cancer than it is for those that cause other kinds of health problems.

The dose-response estimates for the project analysis were conducted using HARP2 (version 2.0.3).

Cancer Effects

For chemicals that cause cancer, the general assumption in risk assessment has been that there are no exposures that have "zero risk" unless there is clear evidence otherwise. In other words, even a very low exposure to a cancer-causing chemical may result in cancer if the chemical happens to alter cellular functions in a way that causes cancer to develop. Thus, even very low exposures to carcinogens might increase the risk of cancer, if only by a very small amount.

Several factors make it difficult to estimate the risk of cancer. Cancer appears to be a progressive disease because a series of cellular transformations is thought to occur before cancer develops. In addition, cancer in humans often develops many years after exposure to a chemical. Also, the best information available on the ability of chemicals to cause cancer often comes from studies in which a limited number of laboratory animals are exposed to levels of chemicals that are much higher than the levels humans would normally be exposed to in the environment. As a result, scientists use mathematical models based on studies of animals exposed to high levels of a chemical to estimate the probability of cancer developing in a diverse population of humans exposed to much lower levels. The uncertainty in these estimates may be rather large. To reduce these uncertainties, risk assessors must stay informed of new scientific research. Data from new studies can be used to improve estimates of cancer risks.

Non-cancer Effects

Non-cancer health effects (such as asthma, nervous system disorders, birth defects, and developmental problems in children) typically become more severe as exposure to a chemical increases. One goal of dose-response assessment is to estimate levels of exposure that pose only a low or negligible risk for non-cancer health effects. Scientists analyze studies of the health effects of a chemical to develop this estimate. They take into account such factors as the quality of the scientific studies, whether humans or laboratory animals were studied, and the degree to which some people may be more sensitive to the chemical than others. The estimated level of exposure that poses no significant health risks can be reduced to reflect these factors.

Risk Characterization

The last step in risk assessment brings together the information developed in the previous three steps to estimate the risk of health effects in an exposed population. In the risk characterization step, scientists analyze the information developed during the exposure and dose-response assessments to describe the resulting health risks that are expected to occur in the exposed population. This information is presented in different ways for cancer and non-cancer health effects, as explained below.

Cancer Risk

Cancer risk is often expressed as the maximum number of new cases of cancer projected to occur in a population of one million people due to exposure to the cancer-causing substance over a 70-year lifetime. For example, a cancer risk of one in one million means that in a population of one million people, not more than one additional person would be expected to develop cancer as the result of the exposure to the substance causing that risk.

An individual's actual risk of contracting cancer from exposure to a chemical is often less than the theoretical risk to the entire population calculated in the risk assessment. For example, the risk estimate for a drinking-water contaminant may be based on the health-protective assumption that the individual drinks two liters of water from a contaminated source daily over a 70-year lifetime. However, an individual's actual exposure to that contaminant would likely be lower due to a shorter time of residence in the area. Moreover, an individual's risk not only depends on the individual's exposure to a specific chemical but also on his or her genetic background (i.e., a family history of certain types of cancer); health; diet; and lifestyle choices, such as smoking or alcohol consumption.

Cancer risks presented in risk assessments are often compared to the overall risk of cancer in the general U.S. population (about 250,000 cases for every one million people) or to the risk posed by all harmful chemicals in a particular medium, such as the air. The cancer risk from breathing current levels of pollutants in California's ambient air over a 70-year lifetime is estimated to be ~760 in one million.

Non-cancer Risk

Non-cancer risk is usually determined by comparing the actual level of exposure to a chemical to the level of exposure that is not expected to cause any adverse effects, even in the most susceptible people. Levels of exposure at which no adverse health effects are expected are called "health reference levels," and they generally are based on the results of animal studies. However, scientists usually set health reference levels much lower than the levels of exposure that were found to have no adverse effects in the animals tested. This approach helps to ensure that real health risks are not underestimated by adjusting for possible differences in a chemical's effects on laboratory animals and humans; the possibility that some humans, such as children and the elderly, may be particularly sensitive to a chemical; and possible deficiencies in data from the animal studies.

Depending on the amount of uncertainty in the data, scientists may set a health reference level 100 to 10,000 times lower than the levels of exposure observed to have no adverse effects in animal studies. Exposures above the health reference level are not necessarily hazardous, but the risk of toxic effects increases as the dose increases. If an assessment determines that human exposure to a chemical exceeds the health reference level, further investigation is warranted.

Risk managers rely on risk assessments when making regulatory decisions, such as setting drinking water standards, or developing plans to clean up hazardous waste sites. Risk managers are responsible for protecting human health, but they must also consider public acceptance, as well as technological, economic, social, and political factors, when arriving at their decisions. For example, they may need to consider how much it would cost to remove a contaminant from drinking water supplies or how seriously the loss of jobs would affect a community if a factory were to close due to the challenge of meeting regulatory requirements that are set at the most stringent level.

Health risk assessments can help risk managers weigh the benefits and costs of various alternatives for reducing exposure to chemicals. For example, a health risk assessment of a hazardous waste site could help determine whether placing a clay cap over the waste to prevent exposure would offer the same health protection as the more costly option of removing the waste from the site.

One of the most difficult questions of risk management is: How much risk is acceptable? While it would be ideal to completely eliminate all exposure to hazardous chemicals, it is usually not possible or feasible to remove all traces of a chemical once it has been released into the environment. The goal of most regulators is to reduce the health risks associated with exposure to hazardous pollutants to a negligibly low level.

Regulators generally presume that a one-in-one million risk of cancer from life-long exposure to a hazardous chemical is an "acceptable risk" level because the risk is extremely low compared to the overall cancer rate. If a drinking water standard for a cancer-causing chemical were set at the level posing a "one-in-one million" risk, it would mean that not more than one additional cancer case (beyond what would normally occur in the population) would potentially occur in a population of one million people drinking water meeting that standard over a 70-year lifetime.

Actual regulatory standards for chemicals or hazardous waste cleanups may be set at less stringent risk levels, such as one in 100,000 (not more than one additional cancer case per 100,000 people) or one in 10,000 (not more than one additional cancer case per 10,000 people). These less stringent risk levels are often due to economic or technological considerations. Regulatory agencies generally view these higher risk levels to be acceptable if there is no feasible way to reduce the risks further."[1]

[1] A Guide to Health Risk Assessment, CalEPA-Office of Environmental Health Hazard Assessment, 1001 I Street, Sacramento, Ca. 95812, (est. 2001).

The following tables summarize the results of the HRA performed by the proposed MREC facility.

Table 5.1D-1 Criteria and Air Toxic Pollutants Emitted from MREC Facility

NOx	1-3 Butadiene
CO	Ethylbenzene
VOC*	Formaldehyde
SOx	Hexane (n-Hexane)
PM10/PM2.5	Naphthalene
Ammonia	Propylene
PAHs	Propylene Oxide
Acetaldehyde	Toluene
Acrolein	Xylene
Benzene	Diesel PM

Table 5.1D-2 Health Effects Significant Threshold Levels

	Significance Thresholds	
Agency	VCAPCD	State of California
Cancer Risk per million	<= 10.0	<= 1.0 without T-BACT <= 10.0 with T-BACT
Acute HI	1.0	1.0
Chronic HI	1.0	1.0
Cancer Burden	n/a	1.0

The other assumptions used in running the HARP program were as follows:

- Emission rates for non-criteria pollutants are taken from AFC Section 5.1, and from Appendix 5.1A.
- Number of residents affected is based upon the updated 2010 population data for those census tracts or portions of census tracts which lie within the maximum impact receptor radius of the proposed facility.
- All receptors were treated as residential receptors, which allows for the assumption that the MIR, if assumed residential, will represent the highest risk and no other receptor will show risks higher than the MIR. This deletes the need for running worker risks. Worker values were scaled directly from the 70-year cancer risk values based on the OEHHA recommended 25 year exposure period.
- Deposition velocity is taken to be 0.02 m/s, as recommended by ARB for controlled emission sources.
- Fraction of residents with home/gardens is the HARP2 default value which is likely conservatively high for the semi-rural area near the project site.

The HARP2 program is a tool that assists with the programmatic requirements of the Air Toxics Hot Spots Program, and it can be used for preparing health risk assessments for other related programs such as air toxic control measure development or facility permitting applications. HARP2 is a computer based risk assessment program which combines the tools of emission inventory database, facility prioritization, air dispersion modeling, and risk assessment analysis. Use of HARP2 promotes statewide consistency in the area of risk assessment, increases the efficiency of evaluating potential health impacts, and provides

a cost effective tool for developing facility health risk assessments. HARP2 may be used on single sources, facilities with multiple sources, or multiple facilities in close proximity to each other.

The receptor grid used in HARP2 was a combination of the following:

1. All identified grid receptors as input from the AERMOD analysis,
1. All identified sensitive receptors within the primary impact area as defined by the AERMOD analysis.

The HARP2 program results for acute and chronic inhalation and chronic non-inhalation exposures, cancer burden and individual cancer risk (workplace and residential) for the combustion sources are included in the CD with this Appendix. The results of the HARP2 calculations are summarized below.

The modeling results show that the maximum modeled cancer risk from MREC operations is expected to be 5.24 x E^{-6}. This risk is well below the VCAPCD significance value of 10 per million. T-BACT for simple cycle combustion turbines is the use of clean fuels (natural gas) and the operation of a CO catalyst. These T-BACT technologies are proposed for MREC, and as such, the significant risk threshold for MREC is 10 in a million. The chronic and acute non-cancer hazard indices are 0.00102 and 0.00179, respectively at the cancer MIR. Both are well below the significant impact level of 1.0. Detailed calculations and results for each significant receptor are included in the modeling results, which are being submitted electronically.

Table 5.1D-3 Health Risk Assessment Summary-operations

Turbines and Fire Pump Engine		
Risk Category	Facility Values	Applicable Significance Thresholds*
Cancer Risk at MIR	5.24^{-06}	See Table 5.1D-2 above.
Chronic Hazard Index at Cancer MIR	0.00102	
Chronic Hazard Index at Max Chronic Receptor	0.00228	
Acute Hazard Index at Cancer MIR	0.00179	
Acute Hazard Index at Max Acute Receptor	0.0566	

Cancer MIR – (Receptor #27, 306273.8, 3798390)

Max Acute non-MIR (Receptor #6093, 306200, 3797000)

Max Chronic non-MIR (Receptor #6011, 305800, 3796600)

Table 5.1D-4 presents a summary of risk and health data for the nearest residential, worker, and sensitive receptors.

Table 5.1D Summary of Risk and Health Data

Recp Type*	Recp #	UTM E	UTM N	Cancer Risk	Chronic HI	Acute HI
MIR	27	306273.8	3798390	5.24E-6	0.00102	0.00179
MEIR-North	8898	306264	3799566	1.30E-8	0.000038	0.0047
MEIR-South	8899	306144	3795267	3.98E-8	0.00022	0.0054
MEIR-East	8900	306531	3798541	7.10E-7	0.00055	0.0052
MEIR-West	8901	304929	3797623	1.69E-8	0.000035	0.0047
MEIR-R1a	8905	306551	3798554	6.55E-7	0.00056	0.0050
MEIR-R1b	8904	306529	3798630	3.61E-7	0.00072	0.0054
MEIR-R2	8903	306325	3798714	1.04E-7	0.00028	0.0036

Table 5.1D Summary of Risk and Health Data

Recp Type*	Recp #	UTM E	UTM N	Cancer Risk	Chronic HI	Acute HI
MEIW	8902	306257	3798462	3.79E-7	0.00018	0.0019
Nearest School	8884	306381	3800656	1.38E-8	0.000036	0.0037
Nearest Health Facility	8847	297887	3789325	4.42E-9	0.000013	0.0016
Nearest Daycare	None Identified	-	-	-	-	-
Nearest Convalescent Home	8844	295842	3793169	4.40E-9	0.000014	0.0014

MEIW risk is simply the 70 year risk adjusted for an exposure period of 25 years per OEHHA (2015).

The impact area cancer burden remains 0.0012.

*UTM coordinates for some receptors adjusted in final modeling file versus AFC Table 4.5-1.

The calculated health effects as summarized above do not exceed the district significance threshold values, therefore the health effects would be considered “not significant” and may even be “zero”.

Risk Assessment input and output files are included on the modeling CD. Due to the length of the HRA input and output files, hard copies are not provided in this appendix.

Construction HRA

A construction screening HRA was performed using the following assumptions as follows:

- The first three highest impacted receptors were chosen to represent the potential risks posed by construction related DPM emissions.
- Cancer risk and chronic hazard indices were computed using HARP2.
- A cancer inhalation unit risk value of 0.0003 $(ug/m^3)^{-1}$ was used.
- A cancer chronic inhalation REL of 5.0 $(ug/m^3)^{-1}$ was used.
- No acute inhalation REL exists for diesel PM.

The adjustment factor applied to the final 70-yr risk and hazard index values was based upon a construction work schedule of 1.92 years (a value of 2 years was used) to adjust the risk values to the construction period (OEHHA, 2015. Air Toxics Hot Spots Program Risk Assessment Guidelines, Chapter 8, Section 8.2.10).

The following table presents the results of the screening level assessment of health risks from the construction phase for the three (3) highest values evaluated on the construction receptor grid as well as the nearest residential, worker, and sensitive receptor locations.

Table 5.1D-5 Construction Screening HRA Summary

Receptor Type*	Receptor #	UTM E	UTM N	Cancer Risk**	Chronic HI
1	2469	306240	3798460	4.97E-6	0.0331
2	2599	306280	3798440	4.94E-6	0.0330
3	2533	306260	3798440	4.91E-6	0.0328

Table 5.1D-5 Construction Screening HRA Summary

Receptor Type*	Receptor #	UTM E	UTM N	Cancer Risk**	Chronic HI
MEIR-North	4854	306264	3799566	2.94E-8	0.000196
MEIR-South	4855	306144	3795267	2.10E-8	0.000134
MEIR-East	4856	306531	3798541	1.79E-6	0.0119
MEIR-West	4857	304929	3797623	1.52E-8	0.00010
MEIR-R1a	4861	306551	3798554	1.67E-6	0.0111
MEIR-R1b	4860	306529	3798630	1.67E-6	0.0111
MEIR-R2	4859	306325	3798714	8.23E-7	0.00548
MEIW	4858	306257	3798462	4.91E-6	0.0328
Nearest School	4840	306381	3800656	1.38E-8	0.000093
Nearest Health Facility	4803	297887	3789325	1.60E-9	0.000011
Nearest Daycare	None Identified	-	-	-	-
Nearest Convalescent Home	4800	295842	3793169	1.40E-9	0.0000095

*UTM coordinates for some receptors adjusted in final modeling file versus AFC Table 4.5-1.

**70 year risk values adjusted for the actual length of the construction period.

With respect to emissions from diesel fueled engines, use of the diesel PM exposure factors noted above are approved by CARB for the characterization of diesel engine exhaust and subsequent risk exposures. The diesel PM factor includes the range of fuel bound, and potentially emitted metals, PAHs, and a wide variety of other semi-volatile substances.

CARB notes the following in the diesel exhaust risk identification documents:

- The surrogate for whole diesel exhaust is diesel PM. PM10 is the basis for the potential risk calculations.
- When conducting an HRA, the potential cancer risk from inhalation exposure to diesel PM will outweigh the potential non-cancer health effects.
- When comparing whole diesel exhaust to speciated diesel exhaust, potential cancer risk from inhalation exposure to whole diesel exhaust will outweigh the multi-pathway cancer risk from the speciated compounds. For this reason, there will be few situations where an analysis of multi-pathway risk is necessary.

With respect to diesel particulate related risk values, the following should be noted:

The following comments were derived from http://www3.epa.gov/region1/eco/airtox/diesel.html, EPA Region 1 New England (2015).

EPA's National Scale Assessment uses several types of health hazard information to provide a quantitative "threshold of concern" or a health benchmark concentration at which it is expected that no adverse health effects occur at exposures to that level. Health effects information on carcinogenic, short and long term noncarcinogenic end points are used to establish selective protective health levels to compare to the modeled exposures levels. Unfortunately the exposure response data for diesel exhaust in human studies are considered too uncertain to develop a carcinogenic unit risk for EPA's use. There is

a Reference Concentration (RFC) that is used as a health benchmark protective of chronic noncarcinogenic health effects but it is for diesel exhaust and not specifically set for diesel particulate matter which is what was modeled in NATA. The RFC for diesel exhaust, which includes diesel particulate matter is 5 ug/m3. This value is similar to the National Ambient Air Quality Standard established for fine particulate matter which is 15ug/m3.

The EPA agrees that diesel exhaust is "likely to be carcinogenic to humans by inhalation." In their risk assessment, however, the EPA did not give a quantitative estimate of risk of lung cancer due to diesel exhaust exposures. There is some uncertainty "to definitively conclude that diesel exhaust is carcinogenic to humans." Although rat and mice studies demonstrate mutagenic and chromosomal effects, these studies do not reflect normal human exposure, as previously explained. The EPA decided that the human data from epidemiological studies are too uncertain to derive a quantitative estimate of cancer risk.

The following comments were derived from the EPA Health Risk Assessment for Diesel Engine Exhaust (EPA 600/8-90/057F, May 2002.

Acute (Short-Term Exposure) Effects

Information is limited for characterizing the potential health effects associated with acute or short-term exposure. However, on the basis of available human and animal evidence, it is concluded that acute or short-term (e.g., episodic) exposure to DE can cause acute irritation (e.g., eye, throat, bronchial), neurophysiological symptoms (e.g., lightheadedness, nausea), and respiratory symptoms (cough, phlegm).

There also is evidence for an immunologic effect–the exacerbation of allergenic responses to known allergens and asthma-like symptoms. The lack of adequate exposure-response information in the acute health effect studies precludes the development of recommendations about levels of exposure that would be presumed safe for these effects.

Chronic (Long-Term Exposure) Noncancer Respiratory Effects

Information from the available human studies is inadequate for a definitive evaluation of possible noncancer health effects from chronic exposure to DE. However, on the basis of extensive animal evidence, DE is judged to pose a chronic respiratory hazard to humans. Chronic-exposure, animal inhalation studies show a spectrum of dose-dependent inflammation and histopathological changes in the lung in several animal species including rats, mice, hamsters, and monkeys.

This assessment provides an estimate of inhalation exposure of DE (as measured by DPM) to which humans may be exposed throughout their lifetime without being likely to experience adverse noncancer respiratory effects. This exposure level, known as the reference concentration (RfC) for DE of 5 :g/m3 of DPM was derived on the basis of dose-response data on inflammatory and histopathological changes in the lung from rat inhalation studies. In recognition of the presence of DPM in ambient PM2.5, it also is appropriate to consider the wealth of PM2.5 human health effects data. In this regard, the 1997 National Ambient Air Quality Standard for PM2.5 of 15 :g/m3 (annual average concentration) also would be expected to provide a measure of protection from DPM, reflecting DPM's current approximate proportion to PM2.5.

Chronic (Long-Term Exposure) Carcinogenic Effects

This assessment concludes that DE is "likely to be carcinogenic to humans by inhalation" and that this hazard applies to environmental exposures. This conclusion is based on the totality of evidence from human, animal, and other supporting studies. There is considerable evidence demonstrating an association between DE exposure and increased lung cancer risk among workers in varied occupations where diesel engines historically have been used. The human evidence from occupational studies is

considered strongly supportive of a finding that DE exposure is causally associated with lung cancer, though the evidence is less than that needed to definitively conclude that DE is carcinogenic to humans.

There is some uncertainty about the degree to which confounders are having an influence on the observed cancer risk in the occupational studies, and there is uncertainty evolving from the lack of actual DE exposure data for the workers. In addition to the human evidence, there is supporting evidence of DPM's carcinogenicity and associated DPM organic compound extracts in rats and mice by noninhalation routes of exposure. Other supporting evidence includes the demonstrated mutagenic and chromosomal effects of DE and its organic constituents, and the suggestive evidence for bioavailability of the DPM organics in humans and animals. Although high exposure chronic rat inhalation studies show a significant lung cancer response, this is not thought predictive of a human hazard at lower environmental exposures. The rat response is considered to result from an overload of particles in the lung resulting from the high exposure, and such an overload is not expected to occur in humans at environmental exposures. Although the available human evidence shows a lung cancer hazard to be present at occupational exposures that are generally higher than environmental levels, it is reasonable to presume that the hazard extends to environmental exposure levels. While there is an incomplete understanding of the mode of action for DE-induced lung cancer that may occur in humans, there is the potential for a nonthreshold mutagenic mode of action stemming from the organics in the DE mixture. A case for an environmental hazard also is shown by the simple observation that the estimated higher environmental exposure levels are close to, if not overlapping, the lower range of occupational exposures for which lung cancer increases are reported.

These considerations taken together support the prudent public health choice of presuming a cancer hazard for DE at environmental levels of exposure. Overall, the evidence for a potential cancer hazard to humans resulting from chronic inhalation exposure to DE is persuasive, even though assumptions and uncertainties are involved. While the hazard evidence is persuasive, this does not lead to similar confidence in understanding the exposure/dose-response relationship. Given a carcinogenicity hazard, EPA typically performs a dose-response assessment of the human or animal data to develop a cancer unit risk estimate that can be used with exposure information to characterize the potential cancer disease impact on an exposed population. The DE human exposure-response data are considered too uncertain to derive a confident quantitative estimate of cancer unit risk, and with the chronic rat inhalation studies not being predictive for environmental levels of exposure, EPA has not developed a quantitative estimate of cancer unit risk.

In the absence of a cancer unit risk, simple exploratory analyses were used to provide a perspective of the range of possible lung cancer risk from environmental exposure to DE. The analyses make use of reported lung cancer risk increases in occupational epidemiologic studies and the differences between occupational and environmental exposure. The purpose of having a risk perspective is to illustrate and have a sense of the possible significance of the lung cancer hazard from environmental exposure. The risk perspective cannot be viewed as a definitive quantitative characterization of cancer risk nor is it suitable for estimation of exposure-specific population risks.

It is concluded that environmental exposure to DE may present a lung cancer hazard to humans. The particulate phase appears to have the greatest contribution to the carcinogenic effect, both the particle core and the associated organic compounds have demonstrated carcinogenic properties, although a role for the DE gas-phase components cannot be ruled out.

Using either EPA's 1986 Guidelines for Carcinogen Risk Assessment (U.S. EPA, 1986) or the proposed revisions (U.S. EPA, 1996b, 1999), DE is judged to be a probable human carcinogen, or likely to be carcinogenic to humans by inhalation, respectively. The weight of evidence for potential human carcinogenicity for DE is considered strong, even though inferences are involved in the overall assessment.

Even though available evidence supports a conclusion that DE is likely to be a human lung carcinogen, the conclusion of the dose-response evaluation is that the available data are not sufficient to confidently estimate a cancer unit risk or unit risk range. The absence of such a cancer unit risk for DE limits the ability to quantify, with confidence, the potential impact of the hazard on exposed populations.

In Summary….

Although OEHHA and the State of California have identified diesel exhaust (and diesel particulate matter) as carcinogens, and DPM as the risk surrogate for whole diesel exhaust, and has established a unit risk factor for DPM, it should be remembered that there is an entire body of scientific data and individuals that at this time who cannot conclude that a unit risk value for DPM can be established. The Applicant believes that this "other conclusion" should be considered when viewing and interpreting risk assessment values for DPM.

The following tables and figures are presented at the end of this appendix:

- Table 5.1D-6 Census Tract Numbers, Areas, and Population Data
- Table 5.1D-7 Sensitive Receptor Listing for the Primary Impact Radius
- Table 5.1D-8 OEHHA/CARB Risk Assessment Health Values
- Figure 5.1D-1 Sensitive Receptor Map
- Figure 5.1D-2 Census Tracts in the Immediate Impact Area
- Figure 5.1D-3 Operations MIR-1, -2, -3 Location Map

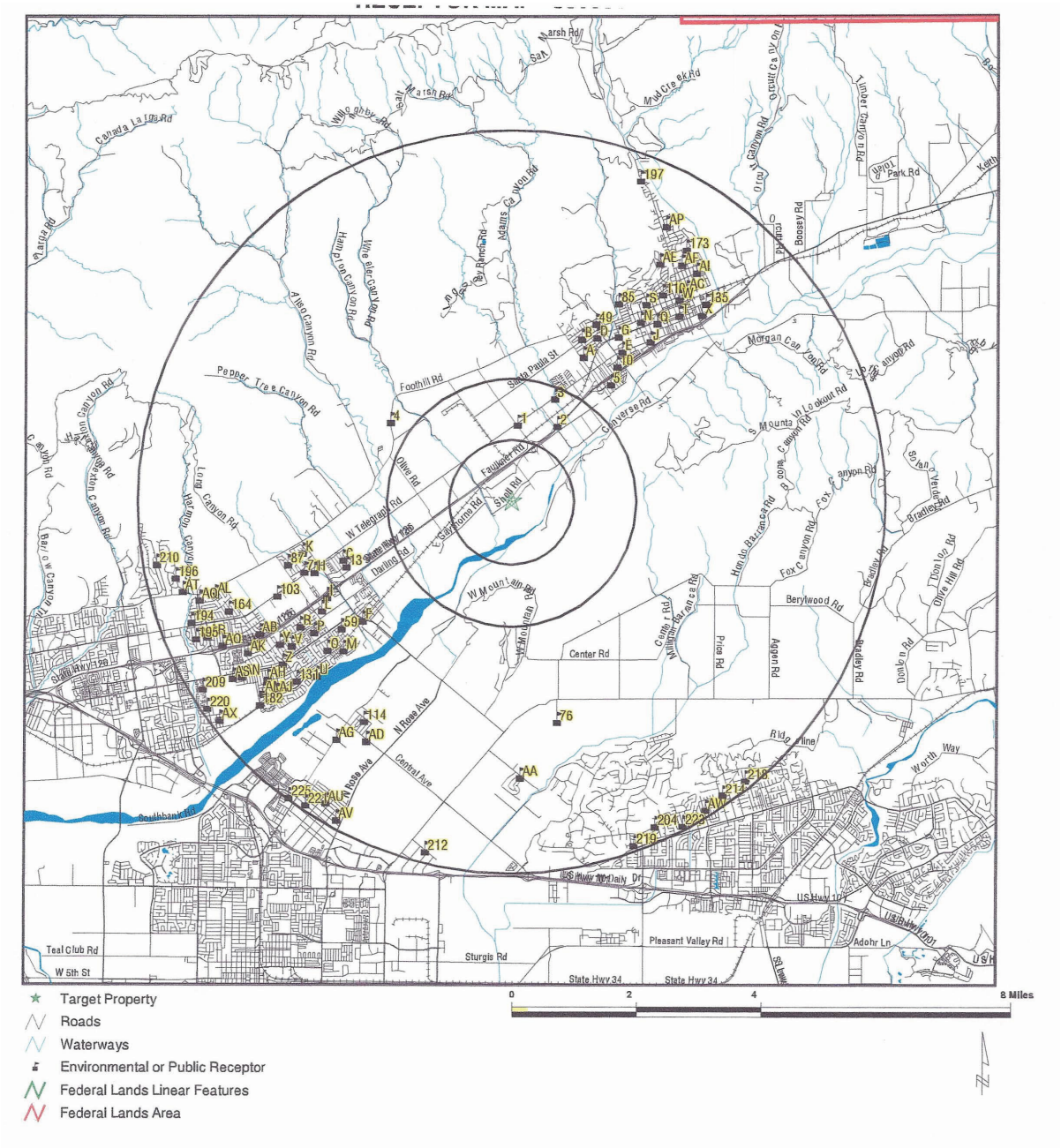

Figure 5.1D-1 Sensitive Receptor Map

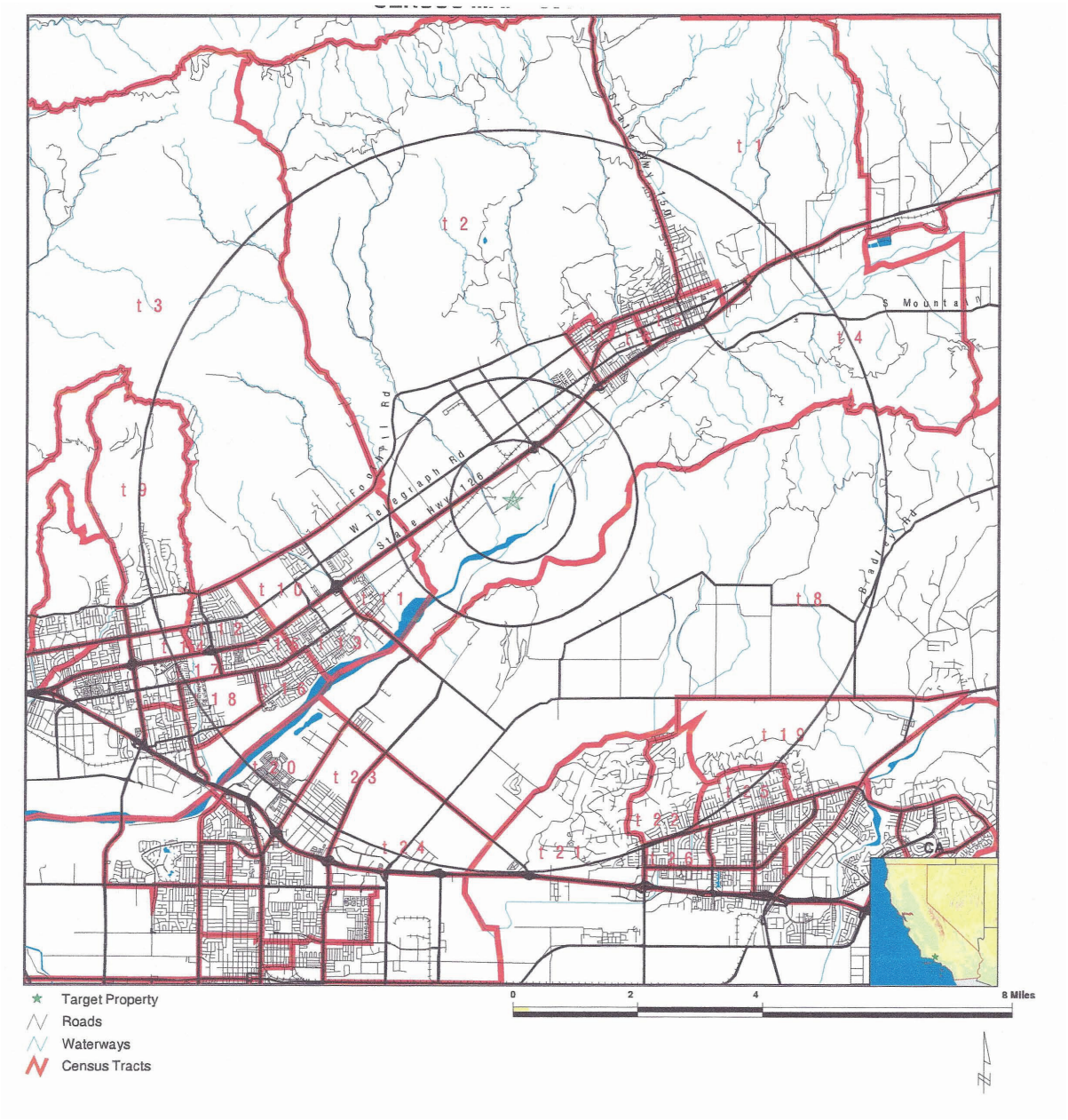

Figure 5.1D-2 Census Tracts in the Immediate Impact Area

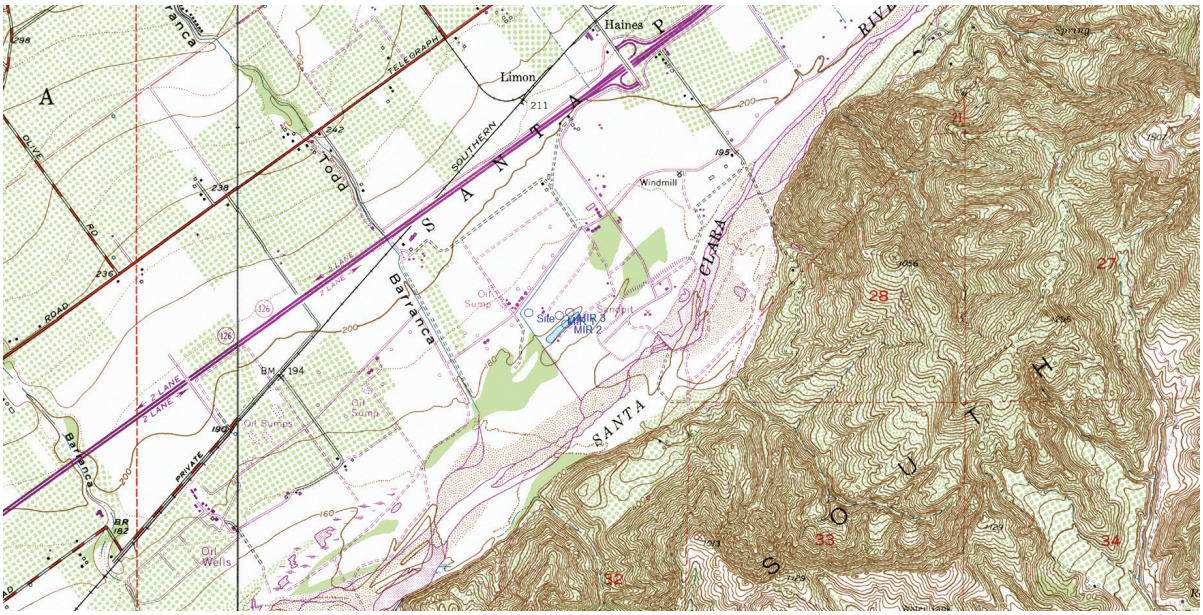

Figure 5.1D-3 Operations MIR 1, 2, and 3 Location Map

Table 5.1D-6 Census Tract Data

CENSUS FINDINGS

Map ID	Tract Number	Total Population	Population in Radius	Total Area(sq.mi.)	Area in Radius(sq.mi.)
T1	0004.00	6758	1222.3	15.72	2.84
T2	0008.00	7788	5544.3	37.21	26.49
T3	0012.04	2473	626.9	46.77	11.85
T4	0005.00	1867	1359.6	20.69	15.06
T5	0006.00	6141	6141.0	0.74	0.74
T6	0007.01	6441	6441.0	0.55	0.55
T7	0007.02	3065	3065.0	0.31	0.31
T8	0051.00	3768	2104.6	53.72	30.01
T9	0017.00	3531	1667.4	4.98	2.35
T10	0012.01	4235	4235.0	2.56	2.56
T11	0013.02	1778	1778.0	1.36	1.36
T12	0012.02	7191	7191.0	1.15	1.15
T13	0013.01	7724	7724.0	1.41	1.41
T14	0016.01	1099	376.8	0.43	0.15
T15	0014.01	3950	3950.0	0.52	0.52
T16	0014.02	5677	4328.1	1.63	1.24
T17	0015.03	5898	1956.5	0.80	0.26
T18	0015.06	5027	3477.2	1.07	0.74
T19	0052.02	2694	1233.2	4.83	2.21
T20	0050.04	5419	3822.2	1.95	1.38
T21	0052.05	5767	4919.4	4.45	3.80
T22	0052.04	3367	3312.0	1.49	1.46
T23	0050.03	8366	5683.6	1.70	1.15
T24	0050.02	3003	2339.5	3.14	2.45
T25	0052.03	5226	2323.3	1.05	0.47
T26	0055.03	3336	41.0	0.56	0.01

Table 5.1D-7 Ventura-Mission Rock Sensitive Receptor Listing

All coordinates and elevations from Google Earth UTM zone 11

Receptor Type	UTME (m)	UTMN (m)	Elev, ft.	Elev, m
school	309172	3811788	1075	328
school	316067	3805669	399	122
school	310597	3803225	272	83
school	310034	3802970	259	79
school	309499	3803778	379	116
school	308740	3802523	260	79
school	308021	3802177	250	76
college	308041	3801357	233	71
school	306408	3800540	238	73
school	302893	3800723	407	124
jail	305532	3798464	189	58
school	301110	3796911	234	71
school	298560	3795520	315	96
school	297844	3796671	566	173
school	298469	3795543	323	98
school	300075	3794435	196	60
school	299855	3794093	187	57
school	296760	3794956	280	85
school	297223	3793945	239	73
school	296845	3793933	230	70
school	295901	3795025	258	79
school	295273	3795195	268	82
school	294798	3795401	305	93
school	294643	3795146	251	77
school	294978	3793049	137	42
school	296718	3792859	171	52
school	296242	3794035	221	67
care fac	295842	3793169	165	50
med fac	296423	3793029	174	53
med fac	298140	3790207	62	19
hosp	297887	3789325	61	19
school	299125	3788312	66	20
school	300112	3788523	76	23
school	300120	3787817	58	18
school	300335	3787128	63	19
hosp	301372	3788395	74	23
hosp	302289	3788326	70	21
school	301852	3787698	65	20
school	301263	3786555	52	16
school	302106	3788936	77	23

Table 5.1D-7 Ventura-Mission Rock Sensitive Receptor Listing

All coordinates and elevations from Google Earth UTM zone 11

Receptor Type	UTME (m)	UTMN (m)	Elev, ft.	Elev, m
school	300981	3789763	85	26
school	301246	3790891	93	28
school	300258	3791370	83	25
school	299015	3791361	74	23
school	302505	3792298	108	33
school	302201	3793228	107	33
hosp	302274	3788326	70	21
airport	307021	3787961	66	20
school	308169	3787195	69	21
college	309046	3787388	81	25
school	309409	3789326	118	36
school	310240	3789203	122	37
school	310776	3789277	131	40
school	311471	3789486	146	44
health cntr	312132	3788614	154	47
school	311421	3790986	288	88
school	312217	3790123	180	55
school	313125	3788862	166	51
school	313941	3790412	197	60
school	313698	3787600	138	42
school	315003	3788317	180	55
school	315885	3789729	257	78
school	315091	3790149	205	62
school	315410	3790852	261	80
school	316316	3793134	319	97
hosp	313992	3791066	208	63
school	307044	3793414	203	62
school	306381	3800656	244	74
school	307989	3801311	234	71
school	308032	3802249	251	77
school	308726	3802593	262	80
health cntr	308948	3802356	247	75
airport	310431	3802616	238	73
school	310028	3803062	263	80
school	309573	3803754	359	109
school	310613	3803179	269	82
hosp	311152	3803579	292	89
hosp	310139	3804718	579	176
health cntr	301421	3796919	197	60
hosp	296826	3794256	249	76
school	297867	3789323	60	18
Residence-north	306264	3799566	203	62
Residence-south	306144	3795267	421	128
Residence-east	306531	3798541	189	58
Residence-west	304929	3797623	175	53
Residenc-R-1a	306551	3798554	189	58

Table 5.1D-7 Ventura-Mission Rock Sensitive Receptor Listing

All coordinates and elevations from Google Earth UTM zone 11

Receptor Type	UTME (m)	UTMN (m)	Elev, ft.	Elev, m
Residence-R-1b	306529	3798630	190	58
Residence-R2	306325	3798714	186	57
worker	306257	3798462	185	56

TABLE 5.1D-8 15 Pages

Table 1
CONSOLIDATED TABLE OF OEHHA/ARB APPROVED RISK ASSESSMENT HEALTH VALUES[a]

Substance	Chemical[b] Abstract Number	Noncancer Effects								Cancer Risk					
		Acute Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	8-Hour Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Oral (mg/kg-d)	Date[c] Value Reviewed [Added]	Inhalation[d] Unit Risk $(\mu g/m^3)^{-1}$	Inhalation[d] Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	M W A F[e]
ACETALDEHYDE	75-07-0	4.7E+02	12/08	3.0E+02	12/08	1.4E+02	12/08			2.7E-06	1.0E-02	4/99 [5/93]			1
ACETAMIDE	60-35-5									2.0E-05	7.0E-02	4/99			1
ACROLEIN	107-02-8	2.5E+00	12/08	7.0E-01	12/08	3.5E-01	12/08								1
ACRYLAMIDE	79-06-1									1.3E-03	4.5E+00	4/99 [7/90]			1
ACRYLIC ACID	79-10-7	6.0E+03	4/99												1
ACRYLONITRILE	107-13-1					5.0E+00	12/01			2.9E-04	1.0E+00	4/99 [1/91]			1
ALLYL CHLORIDE	107-05-1									6.0E-06	2.1E-02	4/99			1
2-AMINOANTHRAQUINONE	117-79-3									9.4E-06	3.3E-02	4/99			1
AMMONIA	7664-41-7	3.2E+03	4/99			2.0E+02	2/00								1
ANILINE	62-53-3									1.6E-06	5.7E-03	4/99			1
ARSENIC AND COMPOUNDS (INORGANIC)[TAC]	7440-38-2 1016 [1015]	2.0E-01	12/08	1.5E-02	12/08	1.5E-02	12/08	3.5E-06	12/08	3.3E-03 [TAC]	1.2E+01	7/90	1.5E+00	10/00	1
ARSINE	7784-42-1	2.0E-01	12/08	1.5E-02	12/08	1.5E-02	12/08								1
ASBESTOS[TAC, f]	1332-21-4									1.9E-04 [TACf]	2.2E+02	3/86			333.33
BENZENE[TAC]	71-43-2	2.7E+01	6/14	3.0E+00	6/14	3.0E+00	6/14			2.9E-05[TAC]	1.0E-01	1/85			1
BENZIDINE (AND ITS SALTS) *values also apply to:*	92-87-5									1.4E-01	5.0E+02	4/99 [1/91]			1
Benzidine based dyes	*1020*									*1.4E-01*	*5.0E+02*	*4/99 [1/91]*			*1*
Direct Black 38	*1937-37-7*									*1.4E-01*	*5.0E+02*	*4/99 [1/91]*			*1*
Direct Blue 6	*2602-46-2*									*1.4E-01*	*5.0E+02*	*4/99 [1/91]*			*1*
Direct Brown 95 (technical grade)	*16071-86-6*									*1.4E-01*	*5.0E+02*	*4/99 [1/91]*			*1*
BENZYL CHLORIDE	100-44-7	2.4E+02	4/99							4.9E-05	1.7E-01	4/99			1
BERYLLIUM AND COMPOUNDS	7440-41-7 [1021]					7.0E-03	12/01	2.0E-03	12/01	2.4E-03	8.4E+00	4/99 [7/90]			1
BIS(2-CHLOROETHYL)ETHER (Dichloroethyl ether)	111-44-4									7.1E-04	2.5E+00	4/99			1
BIS(CHLOROMETHYL)ETHER	542-88-1									1.3E-02	4.6E+01	4/99 [1/91]			1
BROMINE AND COMPOUNDS	7726-95-6 [1040]														1
POTASSIUM BROMATE	7758-01-2									1.4E-04	4.9E-01	4/99 [10/93]			1

Table last updated: May 13, 2015

Table 1
CONSOLIDATED TABLE OF OEHHA/ARB APPROVED RISK ASSESSMENT HEALTH VALUES[a]

Substance	Chemical[b] Abstract Number	Noncancer Effects								Cancer Risk					
		Acute Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	8-Hour Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Oral (mg/kg-d)	Date[c] Value Reviewed [Added]	Inhalation[d] Unit Risk $(\mu g/m^3)^{-1}$	Inhalation[d] Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	MWAF[e]
1,3-BUTADIENE[TAC]	106-99-0	6.6E+02	7/13	9.0E+00	7/13	2.0E+00	7/13			1.7E-04 TAC	6.0E-01	7/92			1
CADMIUM AND COMPOUNDS[TAC]	7440-43-9 [1045]					2.0E-02	1/01	5.0E-04	10/00	4.2E-03 TAC	1.5E+01	1/87			1
CAPROLACTAM	105-60-2	5.0E+01	10/13	7.0E+00	10/13	2.2E+00	10/13								
CARBON DISULFIDE	75-15-0	6.2E+03	4/99			8.0E+02	5/02								1
CARBON MONOXIDE	630-08-0	2.3E+04	4/99												1
CARBON TETRACHLORIDE[TAC] (Tetrachloromethane)	56-23-5	1.9E+03	4/99			4.0E+01	1/01			4.2E-05 TAC	1.5E-01	9/87			1
CHLORINATED PARAFFINS	108171-26-2									2.5E-05	8.9E-02	4/99			1
CHLORINE	7782-50-5	2.1E+02	4/99			2.0E-01	2/00								1
CHLORINE DIOXIDE	10049-04-4					6.0E-01	1/01								1
4-CHLORO-O-PHENYLENEDIAMINE	95-83-0									4.6E-06	1.6E-02	4/99			1
CHLOROBENZENE	108-90-7					1.0E+03	1/01								1
CHLOROFORM[TAC]	67-66-3	1.5E+02	4/99			3.0E+02	4/00			5.3E-06 TAC	1.9E-02	12/90			1
Chlorophenols	*1060*														*1*
PENTACHLOROPHENOL	87-86-5									5.1E-06	1.8E-02	4/99			1
2,4,6-TRICHLOROPHENOL	88-06-2									2.0E-05	7.0E-02	4/99 [1/91]			1
CHLOROPICRIN	76-06-2	2.9E+01	4/99			4.0E-01	12/01								1
p-CHLORO-o-TOLUIDINE	95-69-2									7.7E-05	2.7E-01	4/99			1
CHROMIUM 6+[TAC] *values also apply to:*[g]	18540-29-9					2.0E-01	1/01	2.0E-02	10/00	1.5E-01 TAC	5.1E+02	1/86	5.0E-01	*1/14*	1
Barium chromate	*10294-40-3*					*2.0E-01*	*1/01*	*2.0E-02*	*10/00*	*1.5E-01 TAC*	*5.1E+02*	*1/86*	*5.0E-01*	*1/14*	*0.2053*
Calcium chromate	*13765-19-0*					*2.0E-01*	*1/01*	*2.0E-02*	*10/00*	*1.5E-01 TAC*	*5.1E+02*	*1/86*	*5.0E-01*	*1/14*	*0.3332*
Lead chromate	*7758-97-6*					*2.0E-01*	*1/01*	*2.0E-02*	*10/00*	*1.5E-01 TAC*	*5.1E+02*	*1/86*	*5.0E-01*	*1/14*	*0.1609*
Sodium dichromate	*10588-01-9*					*2.0E-01*	*1/01*	*2.0E-02*	*10/00*	*1.5E-01 TAC*	*5.1E+02*	*1/86*	*5.0E-01*	*1/14*	*0.397*
Strontium chromate	*7789-06-2*					*2.0E-01*	*1/01*	*2.0E-02*	*10/00*	*1.5E-01 TAC*	*5.1E+02*	*1/86*	*5.0E-01*	*1/14*	*0.2554*
CHROMIUM TRIOXIDE (as chromic acid mist)	1333-82-0					2.0E-03	1/01	2.0E-02	10/00	1.5E-01 TAC	5.1E+02	1/86	5.0E-01	*1/14*	0.52
COPPER AND COMPOUNDS	7440-50-8 [1067]	1.0E+02	4/99												1
p-CRESIDINE	120-71-8									4.3E-05	1.5E-01	4/99			1
CRESOLS (mixtures of)	1319-77-3					6.0E+02	1/01								1
m-CRESOL	108-39-4					6.0E+02	1/01								1

Table last updated: May 13, 2015

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		Acute Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	8-Hour Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Oral (mg/kg-d)	Date[c] Value Reviewed [Added]	Inhalation[d] Unit Risk $(\mu g/m^3)^{-1}$	Inhalation[d] Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	M[e] W A F
o-CRESOL	95-48-7					6.0E+02	1/01								1
p-CRESOL	106-44-5					6.0E+02	1/01								1
CUPFERRON	135-20-6									6.3E-05	2.2E-01	4/99			1
Cyanide Compounds (inorganic)	*57-12-5 1073*	*3.4E+02*	*4/99*			*9.0E+00*	*4/00*								*1*
HYDROGEN CYANIDE (Hydrocyanic acid)	74-90-8	3.4E+02	4/99			9.0E+00	4/00								1
2,4-DIAMINOANISOLE	615-05-4									6.6E-06	2.3E-02	4/99			1
2,4-DIAMINOTOLUENE	95-80-7									1.1E-03	4.0E+00	4/99			1
1,2-DIBROMO-3-CHLOROPROPANE (DBCP)	96-12-8									2.0E-03	7.0E+00	4/99 [1/92]			1
p-DICHLOROBENZENE	106-46-7					8.0E+02	1/01			1.1E-05	4.0E-02	4/99 [1/91]			1
3,3-DICHLOROBENZIDINE	91-94-1									3.4E-04	1.2E+00	4/99 [1/91]			1
1,1,-DICHLOROETHANE (Ethylidene dichloride)	75-34-3									1.6E-06	5.7E-03	4/99			1
1,1-DICHLOROETHYLENE … (see Vinylidene Chloride)															
DI(2-ETHYLHEXYL)PHTHALATE (DEHP)	117-81-7									2.4E-06	8.4E-03	4/99 [1/92]	8.4E-03	10/00	1
DIESEL EXHAUST … (see Particulate Emissions from Diesel-Fueled Engines)															
DIETHANOLAMINE	111-42-2					3.0E+00	12/01								
p-DIMETHYLAMINOAZOBENZENE	60-11-7									1.3E-03	4.6E+00	4/99			1
N,N-DIMETHYL FORMAMIDE	68-12-2					8.0E+01	1/01								1
2,4-DINITROTOLUENE	121-14-2									8.9E-05	3.1E-01	4/99			1
1,4-DIOXANE* (1,4-Diethylene dioxide)	123-91-1	3.0E+03	4/99			3.0E+03	4/00			7.7E-06	2.7E-02	4/99 [1/91]			1
EPICHLOROHYDRIN (1-Chloro-2,3-epoxypropane)	106-89-8	1.3E+03	4/99			3.0E+00	1/01			2.3E-05	8.0E-02	4/99 [1/92]			1
1,2-EPOXYBUTANE	106-88-7					2.0E+01	1/01								1
ETHYL BENZENE	100-41-4					2.0E+03	2/00			2.5E-06	8.7E-3	11/07			1
ETHYL CHLORIDE (Chloroethane)	75-00-3					3.0E+04	4/00								1
ETHYLENE DIBROMIDE[TAC] (1,2-Dibromoethane)	106-93-4					8.0E-01	12/01			7.1E-05 TAC	2.5E-01	7/85			1
ETHYLENE DICHLORIDE[TAC] (1,2-Dichloroethane)	107-06-2					4.0E+02	1/01			2.1E-05 TAC	7.2E-02	9/85			1
ETHYLENE GLYCOL	107-21-1					4.0E+02	4/00								1

Table last updated: May 13, 2015

Table 1
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Substance	Chemical[b] Abstract Number	Noncancer Effects								Cancer Risk					
		Acute Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	8-Hour Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Oral (mg/kg-d)	Date[c] Value Reviewed [Added]	Inhalation[d] Unit Risk $(\mu g/m^3)^{-1}$	Inhalation[d] Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	MWAF[e]
ETHYLENE GLYCOL BUTYL ETHER … (see Glycol ethers)															
ETHYLENE OXIDE[TAC] (1,2-Epoxyethane)	75-21-8					3.0E+01	1/01			8.8E-05 TAC	3.1E-01	11/87			1
ETHYLENE THIOUREA	96-45-7									1.3E-05	4.5E-02	4/99			1
Fluorides	1101	*2.4E+02*	*4/99*			1.3E+01	8/03	4.0E-02	8/03						*1*
HYDROGEN FLUORIDE (Hydrofluoric acid)	7664-39-3	2.4E+02	4/99			1.4E+01	8/03	4.0E-02	8/03						1
FORMALDEHYDE[TAC]	50-00-0	5.5E+01	12/08	9.0E+00	12/08	9.0E+00	12/08			6.0E-06 TAC	2.1E-02	3/92			1
GLUTARALDEHYDE	111-30-8					8.0E-02	1/01								1
GLYCOL ETHERS	1115														1
ETHYLENE GLYCOL BUTYL ETHER – EGBE	111-76-2	1.4E+04	4/99												1
ETHYLENE GLYCOL ETHYL ETHER – EGEE	110-80-5	3.7E+02	4/99[1/92]			7.0E+01	2/00								1
ETHYLENE GLYCOL ETHYL ETHER ACETATE – EGEEA	111-15-9	1.4E+02	4/99			3.0E+02	2/00								1
ETHYLENE GLYCOL METHYL ETHER – EGME	109-86-4	9.3E+01	4/99			6.0E+01	2/00								1
ETHYLENE GLYCOL METHYL ETHER ACETATE – EGMEA	110-49-6					9.0E+01	2/00								1
HEXACHLOROBENZENE	118-74-1									5.1E-04	1.8E+00	4/99 [1/91]			1
HEXACHLOROCYCLOHEXANES (mixed or technical grade)	608-73-1									1.1E-03	4.0E+00	4/99 [1/91]	4.0E+00	10/00 [1/92]	1
alpha-HEXACHLOROCYCLOHEXANE	319-84-6									1.1E-03	4.0E+00	4/99 [1/91]	4.0E+00	10/00 [1/92]	1
beta-HEXACHLOROCYCLOHEXANE	319-85-7									1.1E-03	4.0E+00	4/99 [1/91]	4.0E+00	10/00 [1/92]	1
gamma-HEXACHLOROCYCLOHEXANE (Lindane)	58-89-9									3.1E-04	1.1E+00	4/99	1.1E+00	10/00	1
n-HEXANE	110-54-3					7.0E+03	4/00								1
HYDRAZINE	302-01-2					2.0E-01	1/01			4.9E-03	1.7E+01	4/99 [7/90]			1
HYDROCHLORIC ACID (Hydrogen chloride)	7647-01-0	2.1E+03	4/99			9.0E+00	2/00								1
HYDROGEN BROMIDE … (see Bromine & Compounds)															
HYDROGEN CYANIDE … (see Cyanide & Compounds)															

Table 1
CONSOLIDATED TABLE OF OEHHA/ARB APPROVED RISK ASSESSMENT HEALTH VALUES[a]

Substance	Chemical[b] Abstract Number	Noncancer Effects								Cancer Risk					
		Acute Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	8-Hour Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Oral (mg/kg-d)	Date[c] Value Reviewed [Added]	Inhalation[d] Unit Risk $(\mu g/m^3)^{-1}$	Inhalation[d] Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	MWAF[e]
HYDROGEN FLUORIDE … (see Fluorides & Compounds)															
HYDROGEN SELENIDE … (see Selenium & Compounds)															
HYDROGEN SULFIDE	7783-06-4	4.2E+01	4/99[7/90]			1.0E+01	4/00								1
ISOPHORONE	78-59-1					2.0E+03	12/01								
ISOPROPYL ALCOHOL (Isopropanol)	67-63-0	3.2E+03	4/99			7.0E+03	2/00								1
LEAD AND COMPOUNDS[TAC, h] (inorganic) *values also apply to:*	7439-92-1 1128 [1130]									1.2E-05 TAC	4.2E-02	4/97	8.5E-03	10/00	1
Lead acetate	*301-04-2*									*1.2E-05 TAC*	*4.2E-02*	*4/97*	*8.5E-03*	*10/00*	*0.637*
Lead phosphate	*7446-27-7*									*1.2E-05 TAC*	*4.2E-02*	*4/97*	*8.5E-03*	*10/00*	*0.7659*
Lead subacetate	*1335-32-6*									*1.2E-05 TAC*	*4.2E-02*	*4/97*	*8.5E-03*	*10/00*	*0.7696*
LINDANE ... (see gamma-Hexachlorocyclohexane)															
MALEIC ANHYDRIDE	108-31-6					7.0E-01	12/01								1
MANGANESE AND COMPOUNDS	7439-96-5 [1132]			1.7E-01	12/08	9.0E-02	12/08								1
MERCURY AND COMPOUNDS (INORGANIC)	7439-97-6 [1133]	6.0E-01	12/08	6.0E-02	12/08	3.0E-02	12/08	1.6E-04	12/08						1
Mercuric chloride	*7487-94-7*	*6.0E-01*	*12/08*	*6.0E-02*	*12/08*	*3.0E-02*	*12/08*	*1.6E-04*	*12/08*						*1*
METHANOL	67-56-1	2.8E+04	4/99			4.0E+03	4/00								1
METHYL BROMIDE (Bromomethane)	74-83-9	3.9E+03	4/99			5.0E+00	2/00								1
METHYL tertiary-BUTYL ETHER	1634-04-4					8.0E+03	2/00			2.6E-07	1.8E-03	11/99			1
METHYL CHLOROFORM (1,1,1-Trichloroethane)	71-55-6	6.8E+04	4/99			1.0E+03	2/00								1
METHYL ETHYL KETONE (2-Butanone)	78-93-3	1.3E+04	4/99												1
METHYL ISOCYANATE	624-83-9					1.0E+00	12/01								1
4,4'-METHYLENE BIS (2-CHLOROANILINE) (MOCA)	101-14-4									4.3E-04	1.5E+00	4/99			1
METHYLENE CHLORIDE[TAC] (Dichloromethane)	75-09-2	1.4E+04	4/99			4.0E+02	2/00			1.0E-06 TAC	3.5E-03	7/89			1
4,4'-METHYLENE DIANILINE (AND ITS DICHLORIDE)	101-77-9					2.0E+01	12/01			4.6E-04	1.6E+00	4/99	1.6E+00	10/00	1
METHYLENE DIPHENYL ISOCYANATE	101-68-8					7.0E-01	1/01								1
MICHLER'S KETONE (4,4'-Bis(dimethylamino)benzophenone)	90-94-8									2.5E-04	8.6E-01	4/99			1

Table 1
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Substance	Chemical[b] Abstract Number	Noncancer Effects								Cancer Risk					
		Acute Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	8-Hour Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Oral (mg/kg-d)	Date[c] Value Reviewed [Added]	Inhalation[d] Unit Risk $(\mu g/m^3)^{-1}$	Inhalation[d] Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	MWAF[e]
N-NITROSODI-n-BUTYLAMINE	924-16-3									3.1E-03	1.1E+01	4/99 [1/92]			1
N-NITROSODI-n-PROPYLAMINE	621-64-7									2.0E-03	7.0E+00	4/99 [1/91]			1
N-NITROSODIETHYLAMINE	55-18-5									1.0E-02	3.6E+01	4/99 [1/91]			1
N-NITROSODIMETHYLAMINE	62-75-9									4.6E-03	1.6E+01	4/99 [1/91]			1
N-NITROSODIPHENYLAMINE	86-30-6									2.6E-06	9.0E-03	4/99			1
N-NITROSO-N-METHYLETHYLAMINE	10595-95-6									6.3E-03	2.2E+01	4/99 [7/90]			1
N-NITROSOMORPHOLINE	59-89-2									1.9E-03	6.7E+00	4/99 [7/92]			1
N-NITROSOPIPERIDINE	100-75-4									2.7E-03	9.4E+00	4/99 [7/92]			1
N-NITROSOPYRROLIDINE	930-55-2									6.0E-04	2.1E+00	4/99 [7/90]			1
NAPHTHALENE ... (see Polycyclic aromatic hydrocarbons)															
NICKEL AND COMPOUNDS[TAC] *values also apply to:*	7440-02-0 [1145]	2.0E-01	3/12	6.0E-02	3/12	1.4E-02	3/12	1.1E-02	3/12	2.6E-04 TAC	9.1E-01	8/91			1
Nickel acetate	*373-02-4*	*2.0E-01*	*3/12*	*6.0E-02*	*3/12*	*1.4E-02*	*3/12*	*1.1E-02*	*3/12*	*2.6E-04 TAC*	*9.1E-01*	*8/91*			*0.3321*
Nickel carbonate	*3333-67-3*	*2.0E-01*	*3/12*	*6.0E-02*	*3/12*	*1.4E-02*	*3/12*	*1.1E-02*	*3/12*	*2.6E-04 TAC*	*9.1E-01*	*8/91*			*0.4945*
Nickel carbonyl	*13463-39-3*	*2.0E-01*	*3/12*	*6.0E-02*	*3/12*	*1.4E-02*	*3/12*	*1.1E-02*	*3/12*	*2.6E-04 TAC*	*9.1E-01*	*8/91*			*0.3438*
Nickel hydroxide	*12054-48-7*	*2.0E-01*	*3/12*	*6.0E-02*	*3/12*	*1.4E-02*	*3/12*	*1.1E-02*	*3/12*	*2.6E-04 TAC*	*9.1E-01*	*8/91*			*0.6332*
Nickelocene	*1271-28-9*	*2.0E-01*	*3/12*	*6.0E-02*	*3/12*	*1.4E-02*	*3/12*	*1.1E-02*	*3/12*	*2.6E-04 TAC*	*9.1E-01*	*8/91*			*0.4937*
NICKEL OXIDE	1313-99-1	*2.0E-01*	*3/12*	*6.0E-02*	*3/12*	2.0E-02	3/12	*1.1E-02*	*3/12*	*2.6E-04 TAC*	*9.1E-01*	*8/91*			0.7859
Nickel refinery dust from the pyrometallurgical process	*1146*	*2.0E-01*	*3/12*	*6.0E-02*	*3/12*	*1.4E-02*	*3/12*	*1.1E-02*	*3/12*	*2.6E-04 TAC*	*9.1E-01*	*8/91*			*1*
Nickel subsulfide	*12035-72-2*	*2.0E-01*	*3/12*	*6.0E-02*	*3/12*	*1.4E-02*	*3/12*	*1.1E-02*	*3/12*	*2.6E-04 TAC*	*9.1E-01*	*8/91*			*0.2443*
NITRIC ACID	7697-37-2	8.6E+01	4/99												1
NITROGEN DIOXIDE	10102-44-0	4.7E+02	4/99[1/92]												1
p-NITROSODIPHENYLAMINE	156-10-5									6.3E-06	2.2E-02	4/99			1
OZONE	10028-15-6	1.8E+02	4/99[1/92]												1
PARTICULATE EMISSIONS FROM DIESEL-FUELED ENGINES[TAC, i]	9901					5.0E+00 TAC	8/98			3.0E-04 TAC	1.1E+00	8/98			1

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PENTACHLOROPHENOL ... (see Chlorophenols)															
PERCHLOROETHYLENE[TAC] (Tetrachloroethylene)	127-18-4	2.0E+04	4/99			3.5E+01 TAC	10/91			5.9E-06 TAC	2.1E-02	10/91			1
PHENOL	108-95-2	5.8E+03	4/99			2.0E+02	4/00								1
PHOSGENE	75-44-5	4.0E+00	4/99												1
PHOSPHINE	7803-51-2					8.0E-01	9/02								1
PHOSPHORIC ACID	7664-38-2					7.0E+00	2/00								1
PHTHALIC ANHYDRIDE	85-44-9					2.0E+01	1/01								1
PCB (POLYCHLORINATED BIPHENYLS) (unspeciated mixture)[j]	1336-36-3									2.0E-05 [lowest risk] 1.1E-04 [low risk] 5.7E-04 [high risk]	7.0E-02 [lowest risk] 4.0E-01 [low risk] 2.0E+00 [high risk]	4/99	7.0E-02 [lowest risk] 4.0E-01 [low risk] 2.0E+00 [high risk]	10/00	1
PCB (POLYCHLORINATED BIPHENYLS (speciated)[k]															
3,3',4,4'-TETRACHLOROBIPHENYL (PCB 77)	32598-13-3					4.0E-01	8/03	1.0E-04	8/03	3.8E-03	1.3E+01	8/03	1.3E+01	8/03	1
3,4,4',5-TETRACHLOROBIPHENYL (PCB 81)	70362-50-4					1.3E-01	1/11	3.3E-05	1/11	1.1E-02	3.9E+01	1/11	3.9E+01	1/11	1
2,3,3',4,4'-PENTACHLOROBIPHENYL (PCB 105)	32598-14-4					1.3E+00	1/11	3.3E-04	1/11	1.1E-03	3.9E+00	1/11	3.9E+00	1/11	1
2,3,4,4',5-PENTACHLOROBIPHENYL (PCB 114)	74472-37-0					1.3E+00	1/11	3.3E-04	1/11	1.1E-03	3.9E+00	1/11	3.9E+00	1/11	1
2,3',4,4',5-PENTACHLOROBIPHENYL (PCB 118)	31508-00-6					1.3E+00	1/11	3.3E-04	1/11	1.1E-03	3.9E+00	1/11	3.9E+00	1/11	1
2,3',4,4',5'-PENTACHLOROBIPHENYL (PCB 123)	65510-44-3					1.3E+00	1/11	3.3E-04	1/11	1.1E-03	3.9E+00	1/11	3.9E+00	1/11	1
3,3',4,4',5-PENTACHLOROBIPHENYL (PCB 126)	57465-28-8					4.0E-04	8/03	1.0E-07	8/03	3.8E+00	1.3E+04	8/03	1.3E+04	8/03	1
2,3,3',4,4',5-HEXACHLOROBIPHENYL (PCB 156)	38380-08-4					1.3E+00	1/11	3.3E-04	1/11	1.1E-03	3.9E+00	1/11	3.9E+00	1/11	1
2,3,3',4,4',5'-HEXACHLOROBIPHENYL (PCB 157)	69782-90-7					1.3E+00	1/11	3.3E-04	1/11	1.1E-03	3.9E+00	1/11	3.9E+00	1/11	1

Table last updated: May 13, 2015

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		Acute Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	8-Hour Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Oral (mg/kg-d)	Date[c] Value Reviewed [Added]	Inhalation[d] Unit Risk $(\mu g/m^3)^{-1}$	Inhalation[d] Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	M[e] W A F
2,3',4,4',5,5'-HEXACHLOROBIPHENYL (PCB 167)	52663-72-6					1.3E+00	1/11	3.3E-04	1/11	1.1E-03	3.9E+00	1/11	3.9E+00	1/11	1
3,3',4,4',5,5'-HEXACHLOROBIPHENYL (PCB 169)	32774-16-6					1.3E-03	1/11	3.3E-07	1/11	1.1E+00	3.9E+03	1/11	3.9E+03	1/11	1
2,3,3',4,4',5,5'-HEPTACHLOROBIPHENYL (PCB 189)	39635-31-9					1.3E+00	1/11	3.3E-04	1/11	1.1E-03	3.9E+00	1/11	3.9E+00	1/11	1
POLYCHLORINATED DIBENZO-*P*-DIOXINS (PCDD) (Treated as 2,3,7,8-TCDD for HRA)[TAC, k]	1085 1086					4.0E-05	2/00	1.0E-08	10/00	3.8E+01 TAC	1.3E+05	8/86	1.3E+05 TAC	8/86	1
2,3,7,8-TETRACHLORODIBENZO-*P*-DIOXIN[TAC]	1746-01-6					4.0E-05	2/00	1.0E-08	10/00	3.8E+01 TAC	1.3E+05	8/86	1.3E+05 TAC	8/86	1
1,2,3,7,8-PENTACHLORODIBENZO-*P*-DIOXIN	40321-76-4					4.0E-05	8/03	1.0E-08	8/03	3.8E+01	1.3E+05	8/03	1.3E+05	8/03	1
1,2,3,4,7,8-HEXACHLORODIBENZO-*P*-DIOXIN	39227-28-6					4.0E-04	2/00	1.0E-07	10/00	3.8E+00	1.3E+04	4/99	1.3E+04	10/00	1
1,2,3,6,7,8-HEXACHLORODIBENZO-*P*-DIOXIN	57653-85-7					4.0E-04	2/00	1.0E-07	10/00	3.8E+00	1.3E+04	4/99	1.3E+04	10/00	1
1,2,3,7,8,9-HEXACHLORODIBENZO-*P*-DIOXIN	19408-74-3					4.0E-04	2/00	1.0E-07	10/00	3.8E+00	1.3E+04	4/99	1.3E+04	10/00	1
1,2,3,4,6,7,8-HEPTACHLORODIBENZO-*P*-DIOXIN	35822-46-9					4.0E-03	2/00	1.0E-06	10/00	3.8E-01	1.3E+03	4/99	1.3E+03	10/00	1
1,2,3,4,6,7,8,9-OCTACHLORODIBENZO-*P*-DIOXIN	3268-87-9					1.3E-01	1/11	3.3E-05	1/11	1.1E-02	3.9E+01	1/11	3.9E+01	1/11	1
POLYCHLORINATED DIBENZOFURANS (PCDF)[TAC, k] (Treated as 2,3,7,8-TCDD for HRA)	1080					4.0E-05	2/00	1.0E-08	10/00	3.8E+01 TAC	1.3E+05	8/86	1.3E+05 TAC	8/86	1
2,3,7,8-TETRACHLORODIBENZOFURAN	5120-73-19					4.0E-04	2/00	1.0E-07	10/00	3.8E+00	1.3E+04	4/99	1.3E+04	10/00	1
1,2,3,7,8-PENTACHLORODIBENZOFURAN	57117-41-6					1.3E-03	1/11	3.3E-07	1/11	1.1E+00	3.9E +03	1/11	3.9E +03	1/11	1
2,3,4,7,8-PENTACHLORODIBENZOFURAN	57117-31-4					1.3E-04	1/11	3.3E-08	1/11	1.1E+01	3.9E +04	1/11	3.9E +04	1/11	1
1,2,3,4,7,8-HEXACHLORODIBENZOFURAN	70648-26-9					4.0E-04	2/00	1.0E-07	10/00	3.8E+00	1.3E+04	4/99	1.3E+04	10/00	1
1,2,3,6,7,8-HEXACHLORODIBENZOFURAN	57117-44-9					4.0E-04	2/00	1.0E-07	10/00	3.8E+00	1.3E+04	4/99	1.3E+04	10/00	1

Table 1
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Substance	Chemical[b] Abstract Number	Noncancer Effects								Cancer Risk					
		Acute Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	8-Hour Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Oral (mg/kg-d)	Date[c] Value Reviewed [Added]	Inhalation[d] Unit Risk $(\mu g/m^3)^{-1}$	Inhalation[d] Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	M[e] W A F
1,2,3,7,8,9-HEXACHLORODIBENZOFURAN	72918-21-9					4.0E-04	2/00	1.0E-07	10/00	3.8E+00	1.3E+04	4/99	1.3E+04	10/00	1
2,3,4,6,7,8-HEXACHLORODIBENZOFURAN	60851-34-5					4.0E-04	2/00	1.0E-07	10/00	3.8E+00	1.3E+04	4/99	1.3E+04	10/00	1
1,2,3,4,6,7,8-HEPTACHLORODIBENZOFURAN	67562-39-4					4.0E-03	2/00	1.0E-06	10/00	3.8E-01	1.3E+03	4/99	1.3E+03	10/00	1
1,2,3,4,7,8,9-HEPTACHLORODIBENZOFURAN	55673-89-7					4.0E-03	2/00	1.0E-06	10/00	3.8E-01	1.3E+03	4/99	1.3E+03	10/00	1
1,2,3,4,6,7,8,9-OCTACHLORODIBENZOFURAN	39001-02-0					1.3E-01	1/11	3.3E-05	1/11	1.1E-02	3.9E +01	1/11	3.9E +01	1/11	1
POLYCYCLIC AROMATIC HYDROCARBON (PAH)[l] [Treated as B(a)P for HRA][l]	1150 1151									1.1E-03	3.9E+00	4/99 [4/94]	1.2E+01	10/00 [4/94]	1
BENZ(A)ANTHRACENE[l]	56-55-3									1.1E-04	3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
BENZO(A)PYRENE[l]	50-32-8									1.1E-03	3.9E+00	4/99 [4/94]	1.2E+01	10/00 [4/94]	1
BENZO(B)FLUORANTHENE[l]	205-99-2									1.1E-04	3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
BENZO(J)FLUORANTHENE[l]	205-82-3									1.1E-04	3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
BENZO(K)FLUORANTHENE[l]	207-08-9									1.1E-04	3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
CHRYSENE[l]	218-01-9									1.1E-05	3.9E-02	4/99 [4/94]	1.2E-01	10/00 [4/94]	1
DIBENZ(A,H)ACRIDINE[l]	226-36-8									1.1E-04	3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
DIBENZ(A,H)ANTHRACENE[l]	53-70-3									1.2E-03	4.1E+00	4/99 [4/94]	4.1E+00	10/00 [4/94]	1
DIBENZ(A,J)ACRIDINE[l]	224-42-0									1.1E-04	3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
DIBENZO(A,E)PYRENE[l]	192-65-4									1.1E-03	3.9E+00	4/99 [4/94]	1.2E+01	10/00 [4/94]	1
DIBENZO(A,H)PYRENE[l]	189-64-0									1.1E-02	3.9E+01	4/99 [4/94]	1.2E+02	10/00 [4/94]	1

Table 1
CONSOLIDATED TABLE OF OEHHA/ARB APPROVED RISK ASSESSMENT HEALTH VALUES[a]

Substance	Chemical[b] Abstract Number	Noncancer Effects								Cancer Risk					
		Acute Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	8-Hour Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Oral (mg/kg-d)	Date[c] Value Reviewed [Added]	Inhalation[d] Unit Risk $(\mu g/m^3)^{-1}$	Inhalation[d] Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	M[e] W A F
DIBENZO(A,I)PYRENE[l]	189-55-9									1.1E-02	3.9E+01	4/99 [4/94]	1.2E+02	10/00 [4/94]	1
DIBENZO(A,L)PYRENE[l]	191-30-0									1.1E-02	3.9E+01	4/99 [4/94]	1.2E+02	10/00 [4/94]	1
7H-DIBENZO(C,G)CARBAZOLE[l]	194-59-2									1.1E-03	3.9E+00	4/99 [4/94]	1.2E+01	10/00 [4/94]	1
7,12-DIMETHYLBENZ(A)ANTHRACENE[l]	57-97-6									7.1E-02	2.5E+02	4/99 [4/94]	2.5E+02	10/00 [4/94]	1
1,6-DINITROPYRENE[l]	42397-64-8									1.1E-02	3.9E+01	4/99 [4/94]	1.2E+02	10/00 [4/94]	1
1,8-DINITROPYRENE[l]	42397-65-9									1.1E-03	3.9E+00	4/99 [4/94]	1.2E+01	10/00 [4/94]	1
INDENO(1,2,3-C,D)PYRENE[l]	193-39-5									1.1E-04	3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
3-METHYLCHOLANTHRENE[l]	56-49-5									6.3E-03	2.2E+01	4/99 [4/94]	2.2E+01	10/00 [4/94]	1
5-METHYLCHRYSENE[l]	3697-24-3									1.1E-03	3.9E+00	4/99 [4/94]	1.2E+01	10/00 [4/94]	1
NAPHTHALENE	91-20-3					9.0E+00	4/00			3.4E-05	1.2E-01	8/04			1
5-NITROACENAPHTHENE[l]	602-87-9									3.7E-05	1.3E-01	4/99 [4/94]	1.3E-01	10/00 [4/94]	1
6-NITROCHRYSENE[l]	7496-02-8									1.1E-02	3.9E+01	4/99 [4/94]	1.2E+02	10/00 [4/94]	1
2-NITROFLUORENE[l]	607-57-8									1.1E-05	3.9E-02	4/99 [4/94]	1.2E-01	10/00 [4/94]	1
1-NITROPYRENE[l]	5522-43-0									1.1E-04	3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
4-NITROPYRENE[l]	57835-92-4									1.1E-04	3.9E-01	4/99 [4/94]	1.2E+00	10/00 [4/94]	1
POTASSIUM BROMATE.... ... (see Bromine & Compounds)															
1,3-PROPANE SULTONE	1120-71-4									6.9E-04	2.4E+00	4/99			1
PROPYLENE (PROPENE)	115-07-1					3.0E+03	4/00								1
PROPYLENE GLYCOL MONOMETHYL ETHER	107-98-2					7.0E+03	2/00								1
PROPYLENE OXIDE	75-56-9	3.1E+03	4/99			3.0E+01	2/00			3.7E-06	1.3E-02	4/99 [7/90]			1
SELENIUM AND COMPOUNDS[m]	7782-49-2 [1170]					2.0E+01	12/01	5.0E-03	12/01						1
HYDROGEN SELENIDE	7783-07-5	5.0E+00	4/99												1
Selenium sulfide	*7446-34-6*					*2.0E+01*	*12/01*	*5.0E-03*	*12/01*						*1*

Table last updated: May 13, 2015

Table 1
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Substance	Chemical[b] Abstract Number	Noncancer Effects								Cancer Risk					
		Acute Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	8-Hour Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Inhalation ($\mu g/m^3$)	Date[c] Value Reviewed [Added]	Chronic Oral (mg/kg-d)	Date[c] Value Reviewed [Added]	Inhalation[d] Unit Risk $(\mu g/m^3)^{-1}$	Inhalation[d] Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Date[c] Value Reviewed [Added]	MWAF[e]
SILICA [CRYSTALLINE, RESPIRABLE]	1175					3.0E+00	2/05								1
SODIUM HYDROXIDE	1310-73-2	8.0E+00	4/99												1
STYRENE	100-42-5	2.1E+04	4/99			9.0E+02	4/00								1
SULFATES	9960	1.2E+02	4/99												1
SULFUR DIOXIDE	7446-09-5	6.6E+02	4/99[1/92]												1
SULFURIC ACID	7664-93-9	1.2E+02	4/99			1.0E+00	12/01								1
SULFUR TRIOXIDE	*7446-71-9*	*1.2E+02*	*4/99*			*1.0E+00*	*12/01*								*1*
OLEUM	8014-95-7	1.2E+02	4/99												1
1,1,2,2-TETRACHLOROETHANE	79-34-5									5.8E-05	2.0E-01	4/99			1
TETRACHLOROPHENOLS ... (see Chlorophenols)															
2,4,5-TRICHLOROPHENOL ... (see Chlorophenols)															
2,4,6-TRICHLOROPHENOL ... (see Chlorophenols)															
THIOACETAMIDE	62-55-5									1.7E-03	6.1E+00	4/99			1
TOLUENE	108-88-3	3.7E+04	4/99			3.0E+02	4/00								1
Toluene diisocyantates	*26471-62-5*					*7.0E-02*	*1/01*			*1.1E-05*	*3.9E-02*	*4/99*			*1*
TOLUENE-2,4-DIISOCYANATE	584-84-9					7.0E-02	1/01			1.1E-05	3.9E-02	4/99			1
TOLUENE-2,6-DIISOCYANATE	91-08-7					7.0E-02	1/01			1.1E-05	3.9E-02	4/99			1
1,1,2-TRICHLOROETHANE (Vinyl trichloride)	79-00-5									1.6E-05	5.7E-02	4/99			1
TRICHLOROETHYLENE[TAC]	79-01-6					6.0E+02	4/00			2.0E-06 TAC	7.0E-03	10/90			1
TRIETHYLAMINE	121-44-8	2.8E+03	4/99			2.0E+02	9/02								1
URETHANE (Ethyl carbamate)	51-79-6									2.9E-04	1.0E+00	4/99 [7/90]			1
Vanadium Compounds	*N/A*														*1*
Vanadium (fume or dust)	*7440-62-2*	*3.0E+01*	*4/99*												*1*
VANADIUM PENTOXIDE	1314-62-1	3.0E+01	4/99												1
VINYL ACETATE	108-05-4					2.0E+02	12/01								1
VINYL CHLORIDE[TAC] (Chloroethylene)	75-01-4	1.8E+05	4/99							7.8E-05 TAC	2.7E-01	12/90			1
VINYLIDENE CHLORIDE (1,1-Dichloroethylene)	75-35-4					7.0E+01	1/01								1

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XYLENES (mixed isomers)	1330-20-7	2.2E+04	4/99			7.0E+02	4/00								1
m-XYLENE	108-38-3	2.2E+04	4/99			7.0E+02	4/00								1
o-XYLENE	95-47-6	2.2E+04	4/99			7.0E+02	4/00								1
p-XYLENE	106-42-3	2.2E+04	4/99			7.0E+02	4/00								1

Table 1
CONSOLIDATED TABLE OF OEHHA/ARB APPROVED RISK ASSESSMENT HEALTH VALUES[a]

Purpose: The purpose of this reference table is to provide a quick list of all health values that have been approved by the Office of Environmental Health Hazard Assessment (OEHHA) and the Air Resources Board (ARB) for use in facility health risk assessments conducted for the AB 2588 Air Toxics Hot Spots Program. The OEHHA has developed and adopted new risk assessment guidelines that update and replace the California Air Pollution Control Officers Association's (CAPCOA) *Air Toxics "Hot Spots" Program Revised 1992 Risk Assessment Guidelines, October 1993*. The OEHHA has adopted three technical support documents for these guidelines, which can be found on their website (http://www.oehha.ca.gov/air/hot_spots/index.html). This table lists the OEHHA adopted inhalation and oral cancer slope factors, noncancer acute Reference Exposure Levels (RELs), and inhalation and oral noncancer chronic RELs. OEHHA is still in the process of adopting new health values. Therefore, new health values will periodically be added to, or deleted from, this table. Users of this table are advised to monitor the OEHHA website (*www.oehha.ca.gov*) for any updates to the health values.

May 2008 update: The Air Resources Board adopted amendments to the AB 2588 Air Toxics "Hot Spots" Emission Inventory Criteria and Guidelines Regulation (Title 17, California Code of Regulations, Section 93300.5) on November 16, 2006. The amendments became effective on September 26, 2007, after approval from the Office of Administrative Law. Under the new amendments, the substances previously listed in Appendix A-I *(Substances For Which Emissions Must Be Quantified*) and Appendix F (*Criteria For Inputs For Risk Assessment Using Screening Air Dispersion Modeling*) of the ARB's *Air Toxics "Hot Spots" Emission Inventory Criteria and Guidelines (EICG) (July 1997)* have been removed from this table.

a The *italic* font used in this table clarify applicability of OEHHA adopted health effects values to individual or grouped substances listed in the *Air Toxics "Hot Spots" Emission Inventory Criteria and Guidelines*, Appendix A-I list of *"Substances For Which Emissions Must Be Quantified"*.

b Chemical Abstract Service Number (CAS): For chemical groupings and mixtures where a CAS number is not applicable, the 4-digit code used in the *Air Toxics "Hot Spots" Emission Inventory Criteria and Guidelines (EICG) Report* is listed. The 4-digit codes enclosed in brackets [ ] are codes that have been phased out, but may still appear on previously reported Hot Spots emissions. For information on the origin and use of the 4-digit code, see the EICG report.

c Date Value Reviewed [Added]: These columns list the date that the health value was last reviewed by OEHHA, and/or the Scientific Review Panel, and/or approved for use in the AB 2588 Air Toxics Hot Spots Program. If the health value is unchanged since it was first approved for use in the Hot Spots Program, then the date that the value was first approved for use by CAPCOA is listed within the brackets [ ].

- April 1999 is listed for the cancer potency values and noncancer acute RELs, which have been adopted by the OEHHA as part of the AB 2588 Hot Spot Risk Assessment Guidelines.
- February 2000, April 2000, January 2001, and December 2001 are listed for the first set of 22, the second set of 16, the third set of 22, and the fourth set of 12 noncancer chronic RELs, respectively. The chronic REL for carbon disulfide was adopted in May 2002. Chronic RELs for phosphine and triethylamine were adopted in September 2002. Chronic RELs for fluorides including hydrogen fluoride were adopted August 2003. Chronic REL for silica [crystalline respirable] was adopted February 2005.
- October 2000 is listed for the oral chronic RELs and oral cancer slope factors.
- Cancer potency value adopted for naphthalene in August 2004. The inhalation and oral cancer potency values for ethyl benzene were adopted in November 2007.
- For the substances identified as Toxic Air Contaminants, the Air Resources Board hearing date is listed. The dates for acetaldehyde, benzo[a]pyrene, and methyl tertiary-butyl ether represent the dates the values were approved by the Scientific Review Panel.
- On December 19, 2008, OEHHA adopted new acute, 8-hour, and chronic RELs for acetaldehyde, acrolein, arsenic, formaldehyde, manganese, and mercury. The most current health values can be found at: http://www.oehha.ca.gov/air/allrels.html.

  Note: 1. We present the new oral RELs only in milligrams (mg/kg-d), although OEHHA has presented them in other tables in either micrograms (μg/kg-d) or milligrams.

  2. All acute RELs use a 1-hour averaging period (OEHHA, 2008). RELs which were developed using earlier guidelines and specified a different averaging time are unchanged in concentration value, but now refer to the 1-hour averaging period. As of 8/1/2013, the affected chemicals are: benzene, carbon disulfide, carbon tetrachloride, chloroform, ethylene glycol monoethyl ether, ethylene glycol monoethyl ether acetate, and ethylene glycol monomethyl ether: These may be replaced by updated RELs following the OEHHA (2008) guidelines in due course.

  3. At OEHHA's direction, the chronic oral REL for arsenic does not apply to arsine because arsine is a gas and not particle associated.
- OEHHA's adoption of the World Health Organization's 2005 Toxicity Equivalency Factors for polychlorinated dibenzo-p-dioxins (PCDDs), dibenzofurans (PCDFs), and dioxin-like polychlorinated biphenyls (PCBs) occurred in January 2011. See Appendix C of OEHHA's *Air Toxics Hot Spots Program Technical Support Document for Cancer Potencies* at http://www.oehha.ca.gov/air/hot_spots/pdf/AppCdioxinTEFs013111.pdf for more information.
- On March 23, 2012, OEHHA adopted revised acute, 8-hour and chronic RELs for nickel and nickel compounds. The values of the RELs are listed in the table at: http://www.oehha.ca.gov/air/chronic_rels/032312CREL.html.
- On July 29, 2013, OEHHA adopted an acute and 8-hour REL, and a revised chronic REL for 1,3-butadiene. The REL values and summary can be found online at: http://www.oehha.ca.gov/air/hot_spots/index.html.
- On October 18, 2013 (February 2014 table update), OEHHA adopted acute, 8-hour, and chronic RELs for caprolactam. The REL values and summary can be found at: http://www.oehha.ca.gov/air/chronic_rels/pdf/Caprolactam2013.pdf. Changes have been made to target organs to the following substances with no change to health factors: Chloroform, Diethanolamine, Fluorides and Hydrogen Fluoride, Methylene Chloride, Styrene, Xylenes. The "date added" in this table reflects the date of the health factor only.
- On June 27, 2014, OEHHA adopted a new 8-hour REL and revised acute and chronic RELs for benzene. The REL values and summary can be found at: http://www.oehha.ca.gov/air/chronic_rels/BenzeneJune2014.html.

d Inhalation cancer potency factor: The "unit risk factor" has been replaced in the new risk assessment algorithms by a factor called the "inhalation cancer potency factor". Inhalation cancer potency factors are expressed as units of inverse dose [i.e., $(mg/kg\text{-}day)^{-1}$]. They were derived from unit risk factors [units = $(ug/m^3)^{-1}$] by assuming that a receptor weighs 70 kilograms and breathes 20 cubic meters of air per day. The inhalation potency factor is used to calculate a potential inhalation cancer risk using the new risk assessment algorithms defined in the OEHHA, *Air Toxics Hot Spots Program; Technical Support Document for Exposure Assessment and Stochastic Analysis (August 2012)*.

e	Molecular Weight Adjustment Factor: For most of the Hot Spots toxic metals, the OEHHA cancer potency factors and noncancer RELs apply to the weight of the toxic metal atom contained in the overall compound. Some of the Hot Spots compounds contain various elements along with the toxic metal atom (e.g., "Nickel hydroxide", CAS number 12054-48-7, has a formula of H2NiO2). Therefore, an adjustment to the reported pounds of the overall compound is needed before applying the OEHHA cancer potency factor and noncancer RELs for "Nickel and compounds" to such a compound. This ensures that the cancer potency factor and noncancer RELs are applied only to the fraction of the overall weight of the emissions that are associated with health effects of the metal. In other cases, the Hot Spots metals are already reported as the metal atom equivalent (e.g., CAS 7440-02-0, "Nickel"), and these cases do not use any further molecular weight adjustment. (Refer to Note [7] in Appendix A, List of Substances in the EICG Report for further information on how the emissions of various Hot Spots metal compounds are reported.) The appropriate molecular weight adjustment factors (MWAF) to be used along with the OEHHA cancer potency factors and noncancer RELs for Hot Spots metals can be found in the MWAF column of this table. So, for example, assume that 100 pounds of "Nickel hydroxide" emissions are reported under CAS number 12054-48-7. To get the Nickel atom equivalent of these emissions, multiply by the listed MWAF (0.6332) for Nickel hydroxide: • 100 pounds x 0.6332 = 63.32 pounds of Nickel atom equivalent. This step should be completed prior to applying the OEHHA cancer potency factor and noncancer RELs for "Nickel and compounds" in a calculation for a prioritization score or risk assessment calculation. (Note -The HARP software automatically applies the appropriate MWAF for each Hot Spots chemical (by CAS number), so the emissions should not be manually adjusted when using HARP. Therefore, if using HARP, you would use 100 pounds for Nickel hydroxide and HARP will make the MWAF adjustment for you. If not using HARP, you would use 63.32 pounds.) For more information on MWAF please refer to Section 4.2.1.1.1 of OEHHA's document The Air Toxics Hot Spots Program Guidance Manual for the Preparation of Risk Assessments (Guidance Manual) (February 2015). Note: The value listed in the MWAF column for Asbestos is not a molecular weight adjustment. This is a conversion factor for adjusting mass and fibers or structures. See Appendix C of OEHHA's Guidance Manual (February 2015) for more information on Asbestos reporting and risk assessment information or see the EICG report for reporting guidance. Also see the Asbestos footnote (designated by the letter f).
TAC	Toxic Air Contaminant: The Air Resources Board has identified this substance as a Toxic Air Contaminant.
f	Asbestos: The units for the Inhalation Cancer Potency factor for asbestos are (100 PCM fibers/m^3)$^{-1}$. A conversion factor of 100 fibers/0.003 µg can be multiplied by a receptor concentration of asbestos expressed in µg/m^3. Unless other information necessary to estimate the concentration (fibers/m^3) of asbestos at receptors of interest is available. A unit risk factor of 1.9 E 10^{-4} (µg/m^3)$^{-1}$ and an inhalation cancer potency factor of 2.2 E 10^{+2} (mg/kg BW * day)$^{-1}$ are available. For more information on asbestos quantity conversion factors, see Appendix F of OEHHA's *The Air Toxics Hot Spots Program Risk Assessment Guidelines; Part II; Technical Support Document for Cancer Potency Factors (May 2009)*, and Appendix C of OEHHA's Guidance Manual (February 2015)
g	Hexavalent Chromium: In July 2011, OEHHA developed the oral cancer slope factor for chromium 6+ and compounds for the California Public Health Goal in drinking water. As of February 2014, OEHHA states it should also be used for the Hot Spots program.
h	Inorganic Lead: Inorganic Lead was identified by the Air Resources Board as a Toxic Air Contaminant in April 1997. Since information on noncancer health effects show no identified threshold, no Reference Exposure Level has been developed. The document, *Risk Management Guidelines for New, Modified, and Existing Sources of Lead, March 2001*, has been developed by ARB and OEHHA staff for assessing noncancer health impacts from sources of lead. See Appendix F of OEHHA's document *The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments (2003)* for an overview of how to evaluate noncancer impacts from exposure to lead using these risk management guidelines.
i	Particulate Emissions from Diesel-Fueled Engines: The inhalation cancer potency factor was derived from whole diesel exhaust and should be used only for impacts from the inhalation pathway (based on diesel PM measurements). The inhalation impacts from speciated emissions from diesel-fueled engines are already accounted for in the inhalation cancer potency factor. However, at the discretion of the risk assessor, speciated emissions from diesel-fueled engines may be used to estimate acute noncancer health impacts or the contribution to cancer risk or chronic noncancer health impacts for the non-inhalation exposure pathway. See Appendix D of OEHHA's document *The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments* (2003) for more information. The noncancer chronic REL for diesel exhaust is based on assumptions of contributions of diesel PM to ambient PM. It should be used with diesel PM measurement.
j	Cancer Potency Factors (CPFs) for unspeciated mixtures of Polychlorinated Biphenyls: High Risk: For use in cases where congeners with more than four chlorines comprise more than one-half percent of total polychlorinated biphenyls. Use as default CPF for Tier 1 assessments. Low Risk: This number would not ordinarily be used in the Hot Spots program. Lowest Risk: For use in cases where congeners with more than four chlorines comprise less than one-half percent of total polychlorinated biphenyls. As of February, 2014, there is no approved method that can be used to assess the noncancer hazard of an unspeciated PCB mixture. Persons preparing HRAs for the Hot Spots Program should consult with OEHHA and the local Air Pollution Control or Air Quality Management District if an assessment of the noncancer hazard for unspeciated PCB mixtures is needed.
k	Polychlorinated Dibenzo-p-dioxins and Polychlorinated Dibenzofurans (also referred to as chlorinated dioxins and dibenzofurans) and dioxin-like PCB congeners: The OEHHA has adopted the World Health Organization 2005 (WHO-05) Toxicity Equivalency Factor scheme for evaluating the risk due to exposure to samples containing mixtures of polychlorinated dibenzo-p-dioxins (PCDD) and polychlorinated dibenzofurans (PCDF) and a number of dioxin-like PCB congeners. See Appendix A of OEHHA's Technical Support Document For Describing Available Cancer Potency Factors for more information about the scheme. See Appendix C (revised 01/20/11) of OEHHA's Technical Support Document: Methodologies for Derivation, Listing of Available Values, and Adjustments to Allow for Early Life Exposures (2009) online at http://oehha.ca.gov/air/hot_spots/tsd052909.html for more information about the scheme. The two numbers (i.e., 1085 and 1086) in the column listing Chemical Abstracts Numbers are used for reporting and risk assessment purposes. Be sure to input emissions under the proper code when using the HARP software. ID code 1085 has no health values associated with it in the HARP software; therefore, no health impacts will be calculated when using ID 1085. See the Emissions Inventory Criteria and Guidelines for more information on reporting emissions.

Table 1
CONSOLIDATED TABLE OF OEHHA/ARB APPROVED RISK ASSESSMENT HEALTH VALUES[a]

l	Polycyclic Aromatic Hydrocarbons (PAHs): These substances are PAH or PAH-derivatives that have OEHHA-developed Potency Equivalency Factors (PEFs) which were approved by the Scientific Review Panel in April 1994 (see ARB document entitled *Benzo[a]pyrene as a Toxic Air Contaminant*). PAH inhalation slope factors listed here have been adjusted by the PEFs. See OEHHA's Technical Support Document: Methodologies for Derivation, Listing of Available Values, and Adjustments to Allow for Early Life Exposures (2009) for more information about the scheme. Section 8.2.3 and Appendix G of OEHHA's *The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments (2003)* also contains information on PAHs. The two numbers (i.e., 1150 and 1151) in the column listing Chemical Abstracts Numbers are used for reporting and risk assessment purposes. Be sure to input emissions under the proper code when using the HARP software. ID code 1150 has no health values associated with it in the HARP software; therefore, no health impacts will be calculated when using ID 1150. See the Emissions Inventory Criteria and Guidelines for more information on reporting emissions.
m	SELENIUM AND COMPOUNDS: In February 2014, an oral REL was added to the consolidated table. The REL was adopted in Dec 2001, but could not be used by the Hot Spots Program (or HARP software) until transfer factors for the oral and dermal routes were adopted. Transfer factors are included in the OEHHA's Technical Support Document for Exposure Assessment and Stochastic Analysis (August 2012) and are added to the HARP software in March 2015.
N/A	Not Applicable.
Other Changes: • 10/18/2010, removed CHLORODIFLUOROMETHANE, which should have been removed in May 2008. February 2014: • Removed applicability of oleum to the sulfuric acid chronic inhalation REL because oleum represents only an acute health hazard. • Removed "METHYL MERCURY (see Mercury & Compounds)" entry because methyl mercury has different chemical properties, potency, and toxicity compared to elemental mercury and mercury salts, and it is not emitted directly from any California facilities.	