Biomass-Fired Cogeneration Project Health Risk Assessment
Anderson, California

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Date:
February 2010

Project Number:
29-23586A

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1 Introduction

On behalf of Sierra Pacific Industries (SPI), ENVIRON International Corp. (ENVIRON), has prepared this health risk assessment (HRA) for emission increases attributable to a new biomass-fired cogeneration unit (the Project) proposed for installation and operation at the existing SPI facility located at 19794 Riverside Avenue in Anderson, California (see Figure 1-1). This HRA was undertaken at the request of the Shasta County Department of Resource Management Planning Division (Planning Division) and Shasta County Air Quality Management District (AQMD).

The purpose of this evaluation is to quantify potential human health risks associated with toxic air contaminant (TAC) emission increases attributable to the Project. The analysis has been conducted in accordance with the Office of Environmental Health Hazard Assessment's (OEHHA's) Air Toxics Hot Spots Program Risk Assessment Guidelines: The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments (Air Toxics Guidance, August 2003).

Projected emissions are used by the air districts to categorize the various facilities as high, intermediate, and low priority. A project is prioritized based on the following: (1) toxicity of the substances emitted, (2) the quantity of each emitted substance, (3) the proximity of the emission sources to potential receptors, and (4) any other factors that indicate the potential for a significant health risk to the surrounding community. Based on this process, emissions attributable to the Project were prioritized as part of the initial permit application submitted to the AQMD, which designated the project as "high priority" and required that an HRA be prepared. According to the California Air Resources Board (CARB) and the California Air Pollution Control Officers Association (CAPCOA), the designation of a facility as high priority does not necessarily mean that it is emitting substances at a level that will significantly impact the surrounding community (CAPCOA, 1990). Only after an HRA is conducted can the possible health hazards resulting from project emissions be properly evaluated.

The objectives of this HRA are to: (1) estimate off-site air concentrations of the substances emitted by the proposed cogeneration unit and increased fuel delivery truck traffic, (2) evaluate potential exposures to the surrounding community, and (3) characterize the potential health risks to individuals associated with those levels of exposure. This assessment presents the results of this analysis based on refined air dispersion modeling, following the guidance provided by OEHHA (2003). A brief description of the Project and an outline of the report are presented below.

This HRA is a revision and update of an HRA developed for a similar project involving a smaller version of the same boiler design (200,000 pounds steam per hour instead of 250,000 pounds of steam per hour) that was submitted to the AQMD in November 2007. In addition to revising the emission rate calculations and regulatory analysis to reflect the currently proposed boiler, the air quality modeling was updated to reflect the most current versions and guidance, and the impact of diesel particulate matter (DPM) from increased fuel delivery truck traffic was included.

1.1   Project Description

SPI is a family-owned wood products company based in Redding, California. SPI currently operates an existing lumber manufacturing facility in Anderson, California. The existing facility is bordered on the northeast by the Sacramento River, on the northwest by a private parcel, on the southwest by Union Pacific Railroad tracks and SR273, and on the southeast by private parcels. SPI proposes to construct a new cogeneration unit at the facility that would burn biomass fuels in a boiler to produce steam that would be used to generate electricity and to heat existing lumber dry kilns at the facility. The installation of the cogeneration unit will not increase emissions from any existing emission units at the facility.

The cogeneration unit will consist of a biomass-fired water-wall boiler with a vibrating grate, a steam turbine, and a generator. The boiler will burn biomass fuels to produce high-pressure steam for the steam turbine. The steam turbine generator will generate up to 23 megawatts (MW) of electricity. Approximately 7 MW will be used to power on-site equipment; the remainder will be sold to a public utility. Low-pressure steam will be extracted from the steam turbine through a controlled extraction and used to heat the existing dry kilns.

1.2   Report Organization

The remainder of this document is organized as follows:

Section 2   Hazard Identification - Identifies all of the substances evaluated in this HRA for the Project. The substances evaluated for both cancer and noncancer end points are identified.

Section 3   Exposure Assessment - Describes the compounds included in the assessment , the air dispersion modeling for determining airborne concentrations, the exposure pathways evaluated, and the off-site receptors evaluated.

Section 4   Toxicity Assessment - Presents the toxicity criteria used to evaluate potential acute and chronic non-carcinogenic health effects and theoretical carcinogenic risk.

Section 5   Risk Characterization - Presents the results of the risk assessment for the exposure scenarios evaluated.

Section 6   Conclusions - Summarizes the results of the risk assessment.

Section 7   References – Presents the references used in this risk assessment.

2 Hazard Identification

Emission rates of 114 compounds expected to be emitted by the cogeneration unit are presented in Table 2-1. In addition to the compounds shown in Table 2-1, the HRA included DPM emitted by increased fuel haul truck traffic to and from the facility.

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3 Exposure Assessment

This section of the risk assessment describes environmental transport modeling and exposure parameters used to estimate the potential for human exposure to toxic air pollutant emissions attributable to the new cogeneration unit. The following sections (1) summarize and describe the source information and emission estimates used in the environmental transport models; (2) describe potentially exposed populations and exposure pathways; (3) describe the assumptions used in the air dispersion and exposure models; and (4) describe how the modeled concentrations were used in the risk analysis.

3.1 Source Identification/Emission Estimates

This section summarizes the emissions source associated with the Project and the estimated emissions of toxic air pollutants.

3.1.1 Source Identification

The final design of the biomass-fired boiler has not been determined, but it will be similar to a unit designed by the McBurney Corp. of Norcross, Georgia. It will have a maximum annual average heat input of approximately 425.4 million British thermal units per hour (MMBtu/hr) and a maximum steam generation rate of 250,000 pounds per hour (lb/hr). Over short-term periods, the boiler may be fired at heat input rates that exceed the annual average rate: an hourly maximum of 468.0 MMBtu/hr (10 percent greater than the annual average), and a maximum 24-hour average of 446.7 MMBtu/hr (5 percent greater than the annual average).

The boiler will be equipped with two natural gas burners, each with a maximum rated heat input of 62.5 MMBtu/hr, for start up and flame stabilization. The cogeneration unit design will incorporate a selective non-catalytic reduction (SNCR) system to reduce emissions of oxides of nitrogen (NO_X), as well as a multiclone and electrostatic precipitator (ESP) to control emissions of particulate matter (PM/PM_{10}). The exhaust system will be designed to accommodate an oxidation catalyst, which can be installed, if necessary, following startup to reduce carbon monoxide (CO) emissions to the level proposed in the air permit application. A closed-loop two-cell cooling tower will be used to dispose of waste heat from the steam turbine.

The proposed cogeneration unit will be located near the existing biomass-fired boiler at the facility. The location of the new unit was specified in the modeling in Universal Transverse Mercator (UTM) coordinates measured in meters. A facility plot plan showing the location of the cogeneration unit stack and significant structures is presented in Figure 3-1.

The proposed project is expected to require a maximum of 23 additional truck trips per day to either remove excess fuel for sale when the facility is producing more than the boiler can consume (and economic conditions are favorable for fuel sales), or to deliver fuel when the facility is producing less fuel than the cogeneration requires to maintain the contracted level of electricity generation. The trucks were assumed to arrive from or depart to north- or south-bound Interstate Highway 5.

3.1.2 Emission Estimates

The cogeneration unit emission rates used in this HRA are based on those reported by SPI in the air permit application submitted to the AQMD in February 2010. The emission rates are presented in Table 2-1. Background air concentrations for all emitted TACs were assumed to be zero.

For the increased fuel truck traffic, the motor vehicle emission factor model EMFAC2007 (Version 2.3) was used to calculate annual average DPM emission rates from 2010 until 2040, after which emission rates were assumed to be static. Because the trucks used to haul fuel will not be from a specific fleet, the model default fleet was used for each year.

The emission rates for the truck sources vary depending on the period calculation in HARP. For example, 70-year residential chronic and cancer calculations used 70-year average emission rates for trucks (2010-2080), and the 40-year worker chronic and cancer calculations used 40-year average emission rates for trucks (2010-2050). Because EMFAC2007 does not estimate emission factors past the year 2040, emission factors were assumed to remain the same after 2040. The 40- and 70-year average emission factors for each vehicle speed are presented in Table 3-1.

Truck traffic emission rates were varied based on the speed limit of each section of road along the route that will actually be travelled by the trucks. Figure 3-2 shows the routes taken by the trucks as they travel from Interstate 5 to the facility, and vice-versa. Table 3-2 shows the distance, number of one-way trips per day, and the average speed traveled used by EMFAC2007 to calculate the emission factor for each road segment. Because there are currently no signalized intersections in the routes taken by trucks in the analysis, potential stops were not given additional emissions. Instead, the speed decrease caused by un-signalized stops was taken into account in the estimation of average speed traveled for each segment. This is consistent with intersection modeling guidelines.

3.2 Description Of Potentially Exposed Populations

According to OEHHA guidance, risk assessments that utilize refined air dispersion modeling must provide a detailed analysis of the potentially exposed population to the air emissions from the facility. This analysis includes identification of maximum exposed individuals in residential (MEIR) and commercial/industrial areas (MEIW) and identification of sensitive receptors within the Zone of Impact (ZOI).

3.2.1 Identification of Residential and Occupational MEIs

The location of maximum potential hazard indexes or carcinogenic risk is referred to as the point of maximum impact (PMI). Designation of the PMI, as well as residential, and commercial/industrial receptors, was determined using an aerial photo (Figure 3-3). The nearest residential receptors are approximately 100 to 200 meters south of the facility, across SR273, and approximately 200 meters northeast of the facility property boundary, across the Sacramento River. Commercial/industrial receptors are located adjacent to the facility to the

south, southeast, and southwest. Receptor grids with 25- and 50-meter spacing were placed in the surrounding residential and commercial areas, and with 10-meter spacing on the property boundary. As described below, the grid increased in spacing as distance from the facility increased. The receptors were identified as being in industrial or residential areas to identify maximum exposed individuals for chronic and acute non-carcinogenic, and carcinogenic effects for the residential population (MEIR) and worker population (MEIW). Property boundary receptors were also used to identify the theoretical maximum exposed off-site individuals (PMI).

3.2.2 Sensitive Receptors

In accordance with CAPCOA guidance, potential risks at locations of sensitive receptors within the ZOI (discussed further in Section 5.2.1) such as schools and hospitals, should be identified. United States Geological Survey (USGS) maps were used to locate sensitive receptors. No sensitive receptors were identified in the ZOI.

3.3 Environmental Transport And Exposure Modeling

The HARP model (version 1.4a) developed by CARB (2008) was used to conduct the risk analysis for this HRA. Air dispersion modeling was used to estimate off-site concentrations of toxic air pollutants associated with emission rate increases attributable to the Project. The HARP model uses the output from the air dispersion model to predict TAC exposure and risk to the surrounding community. The assumptions used in HARP are discussed in more detail below.

3.3.1 Air Dispersion Model

Refined air dispersion modeling was performed using the U.S. EPA's preferred air dispersion model, AERMOD (Version 09292). Source release parameters used in the model for the cogeneration unit stack and the 620 volume sources used to represent the haul trucks are provided in Table 3-3. A unit emission rate, 1 gram per second (g/s), was used in the model for the cogeneration unit, so the predicted air concentrations in micrograms per cubic meter ($\mu g/m^3$) are actually normalized concentrations ($\mu g/m^3$ per g/s) that were scaled by HARP using emission rates (in g/s) to obtain the ground-level concentration of each TAC.

Fuel haul truck DPM emissions were also modeled using unit emission rates, but with a different approach. Each road segment was given an emission rate equal to one g/s, which was divided by the number of volume sources in the segment. AERMOD calculated normalized concentrations attributable to each road-segment, and HARP was used to combine those normalized concentrations with road-segment-specific emission rates to obtain concentrations of each toxic at each receptor.

The ground-level air concentrations were estimated for receptors located in four grids: one with 25-meter spacing that extended from the property boundary to 625 meters from the facility, a second with 50-meter spacing that extended from 625 m to 1.25 kilometers (km) from the facility, a third with 200-meter spacing that extended from 1.25 km to 2.5 km from the facility,

and a final grid with 500-meter spacing that extened from 2.5 km to 5 km from the facility. Boundary receptors were set up on the fenceline of the property at 25-meter spacing. The final receptor locations are shown in Figure 3-4.

Five years (2004 through 2008) of local hourly meteorological data, processed using AERMOD's meteorological preprocessor, AERMET (Version 06341), were used in this analysis. The meteorological data consisted of data from a surface station at Redding Municipal Airport (Station #24257) and regional upper-air conditions from Medford, Oregon (Station #24225). The annual average and maximum 1-hour air concentrations at each receptor were predicted for each year of meteorological data used. Input files for the dispersion modeling are presented in Appendix A.

3.3.2 HARP Exposure and Risk Model

HARP incorporates the algorithms and exposure assumptions provided in OEHHA's guidance (2003) for estimating exposures and calculating risks for HRAs. HARP typically incorporates the dispersion coefficients and ground-level concentrations predicted for each receptor using a dispersion model not used in this analysis, the Industrial Source Complex Short Term (ISCST3) model. Since the ISCST3 model is no longer the preferred U.S. EPA dispersion model, the CARB has developed a program called the HARP On-Ramp that converts AERMOD outputs into a format that can be used in the HARP exposure model. Version 1 of the HARP On-Ramp (CARB, 2009) was used to import AERMOD input files and ground-level concentration plot files into HARP, so that the environmental fate, exposure parameters, and dose calculation algorithms recommended by OEHHA and incorporated into HARP could be utilized.

Potential health effects attributable to the expected TAC emissions were evaluated for inhalation exposure only because most compounds will exist only as gases. However, other TACs were also evaluated for non-inhalation exposure, including dermal absorption, soil ingestion, ingestion of homegrown produce, and ingestion of mother's milk because they will exist as particulates and are presumed to settle into soil. For TACs evaluated for non-inhalation exposure, the HARP model predicts an oral dose at each receptor location for these non-inhalation pathways. Default parameters, including a 0.02 meter per second (m/s) deposition rate (for controlled sources) and a 0.15 fraction of ingested produce presumed to be homegrown (for non-urban areas), were used to evaluate exposure via these non-inhalation pathways (OEHHA, 2003). Estimates of worker exposure were calculated in HARP using default exposure parameters including an exposure frequency of 49 weeks per year, 5 days per week, 8 hours per day, and a 40-year exposure duration. Site-specific and exposure parameters used in the modeling are presented in Table 3-4.

3.4 Air Dispersion Modeling Results

For each modeled year, 26 sets of normalized concentrations were produced: 1-hour average maximum and annual concentrations for the cogeneration unit, and 1-hour average maximum and annual concentrations for each of the 12 modeled road segments. To facilitate the risk analysis process, the 5 years of predicted concentrations were reduced to obtain 5-year

average concentrations and maximum 1-hour concentrations at each receptor, which were used in the HARP risk assessment. The AERMOD input and concentration output files, as well as the HARP modeling files are provided in Appendix A. Individual receptors were identified for the PMI (receptor #4300), the MEIR (receptor #2804), and the MEIW (receptor #2968) for acute non-carcinogenic effects. The individual receptors identified for chronic non-carcinogenic effects were the PMI (receptor #4297), the MEIR (receptor #2890), and the MEIW (receptor #2892). For carcinogenic effects, the following individual receptors were identified for the PMI (receptor #4310), the MEIR (receptor #2890), and the MEIW (receptor #3105). The locations of these receptors are shown in Figure 3-3.

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4 Toxicity Assessment

This section describes the toxicity criteria developed for TACs evaluated in this HRA. Of the TACs evaluated, 24 are considered to pose potential acute non-carcinogenic hazards, 36 may cause potential chronic non-carcinogenic health effects, and 35 are identified as carcinogens. A summary of toxicity criteria for all TACs anticipated to be emitted from the new boiler is presented in Table 4-1 (OEHHA, 2003).

4.1 Non-Carcinogens

For chronic and acute non-carcinogenic effects, observable biological effects occur only after a threshold dose is reached. To establish health criteria, this threshold dose usually is estimated from the no-observed adverse effect level (NOAEL) or the lowest-observed adverse effect level (LOAEL) determined in studies of chronic exposure in animals by applying a series of uncertainty (safety) factors. For TACs identified for evaluation at the facility, OEHHA and CARB provide "reference exposure levels" (RELs) that represent levels of exposure below which adverse effects are not expected to occur with a substantial margin of safety. These RELs typically include uncertainty factors ranging from 10 to 1,000 to account for limitations in the quality or quantity of available data used to develop the RELs. RELs were published for inhalation exposure based on an acceptable air concentration (in $\mu g/m^3$) and for chronic, non-inhalation exposure based on an acceptable oral dose (in mg/kg-day).

For the purpose of evaluating cumulative effects of TAC exposure, OEHHA has categorized end points for adverse health effects for acute and chronic exposure. Only effects of TACs on the same end point are considered additive. Potential end points for chronic toxicological effects have been classified into thirteen categories in the OEHHA guidelines: alimentary, bone, cardiovascular, developmental, endocrine, eyes, hematologic, immune system, kidney, central/peripheral nervous system, reproductive, respiratory, and skin. However, in this analysis all effects of TACs were summed regardless of end point to maintain a conservative health risk assessment.

The potential end points for acute effects have been classified into ten categories: alimentary, cardiovascular, developmental, eyes, hematologic, immune system, nervous system, reproductive, respiratory, and skin. Several of the substances evaluated have an acute REL based on an averaging period other than one hour (4-, 6-, or 7-hour averaging time): arsenic, benzene, carbon tetrachloride and chloroform. Maximum 1-hour concentrations were conservatively used to evaluate these TACs. The RELs for potential acute and chronic health effects for the TACs evaluated in this assessment are presented in Table 4-1.

4.2 Carcinogens

Regulatory guidance assumes that TACs classified as carcinogens should be treated as if they have no threshold (U.S. EPA, 1989). This approach means that only a zero dose is assumed to result in zero risk (i.e., for all doses, some risk is assumed to be present, increasing linearly with increasing dose). Various mathematical models are used to estimate theoretically plausible

responses at these low doses. For TACs identified for evaluation, the OEHHA guidelines present unit risk values (URVs) that conservatively quantify (i.e., purposely over-predict) the likelihood of a carcinogenic response in an individual receiving a given dose of a specific TAC. URVs were published for inhalation exposure as the inverse of a concentration in air (in units of $(\mu g/m^3)^{-1}$) (OEHHA, 2005). For chronic, non-inhalation exposure oral potency values (OPVs) were published as the inverse of grams of TAC intake per kilogram of body weight per day (in units of $(mg/kg/day)^{-1}$) (OEHHA, 2005). Unlike non-carcinogenic effects, carcinogenic effects are considered additive for all TACs. The URVs and OPVs for TACs included in this assessment are presented in Table 4-1.

4.3 Lead

The detailed approach outlined in the Lead Guidance (CARB, 2001) was used to evaluate potential non-cancer health effects from exposure to lead. This approach consists of three steps: estimate maximum off-site concentrations at potential receptors, identify whether the area should be considered to be average or high exposure from other sources, and assess non-cancer risks (Section 5.3).

The California State Lead Guidance requires the estimation of the 30-day maximum off-site concentration in order to evaluate potential lead exposure. New Federal regulations require that lead concentrations be averaged on a rolling 3-month average basis to determine compliance with the ambient standard. However, AERMOD can not calculate 30-day average or 3-month rolling average concentrations. Instead, the maximum 1-hour concentration at the PMI was used and adjusted by a factor of 0.3 suggested by CARB guidance (2001) to estimate a 30-day average (e.g., the concentration of lead in air averaged over a 30-day period is presumed to be approximately a third of the predicted maximum one-hour concentration).

U.S. Census data from 2000 was evaluated to identify whether the Maximum Exposure Area (MEA), a 1-kilometer square area around the PMI overlapped with a census tract with "high potential for existing exposure" to lead. This is defined in the Lead Guidance as a census tract where the median year of construction for housing is 1960 or earlier and more than 30 percent of the population has an income less than 1.25 times the poverty level. The MEA fell within three census tracts (115, 121, and 123.01) of Shasta County. In these census tracts, the median year of construction ranged from 1974 to 1978, and between 12 and 30 percent of the population has an income less than 1.25 the poverty level (see Table 4-2), indicating that, according to guidance, local residents are considered to have an average existing exposure to lead from other sources (CARB, 2001). This conclusion is incorporated into the manner in which the risk of lead exposure is evaluated.

5 Risk Characterization

This final step of the risk assessment integrates the exposure estimates developed for the TAC emissions (Section 3.0) and the health effects data from which toxicity criteria are established (Section 4.0). The risk characterization section addresses both non-carcinogenic and carcinogenic health effects based on inhalation and non-inhalation exposure. Definition of the ZOI and identification of the MEIR and MEIW were based on the 50-meter receptor grid. In addition, the estimates of health risk that typically warrant regulatory action are discussed.

5.1 Non-Carcinogenic Health Effects

Potential chronic and acute non-carcinogenic health effects associated with exposure to TAC emissions attributable to the Project have been evaluated using the HARP model. For acute inhalation exposure, the HARP model divides the predicted maximum hourly concentration by the appropriate acute REL provided by OEHHA (Table 4-1). Non-inhalation pathways are not applicable to acute exposures (OEHHA, 2003). For chronic inhalation exposures, the predicted annual average air concentration for each TAC is divided by the chronic inhalation REL. For chronic non-inhalation exposure, the predicted oral dose is divided by the chronic, oral REL as appropriate.

These ratios are TAC-specific chronic or acute hazard quotients. The total hazard quotient reported for TACs with non-inhalation effects is the sum of the individual hazard quotients for inhalation and non-inhalation exposure.

Chronic and acute non-carcinogenic health effects were also evaluated in terms of their assumed potential additive effect on target organs or systems (e.g., central nervous system). For acute and chronic exposures, up to thirteen target organs or systems were evaluated using the HARP model (described in Section 4.1). The TACs that may affect the same target organ or system were evaluated by summing the individual hazard quotients to calculate a target organ-specific hazard index (HI). The following sections present the results of the chronic and acute non-carcinogenic evaluations. Chronic and acute hazard indexes less than or equal to 1.0 (i.e., the exposure is less than the health criteria are considered to be without significant public health impact with a substantial margin of safety due to the manner in which the REL is developed.. Additionally, hazard indexes greater than 1.0 do not necessarily mean that adverse health effects would be expected. Rather, on a TAC-specific basis, as the hazard index increases above 1 to 10 or higher, the level of regulatory concern and need for control increases.

The chronic and acute hazard quotients for inhalation exposure can be described by the equation below:

$$\text{Hazard Quotient}_{inh} = GCL / REL_{inh}$$

Where:

   $\text{Hazard Quotient}_{inh}$ =   TAC-specific hazard quotient for inhalation exposure pathways
   GLC   =   Ground-level air concentration at a receptor location ($\mu g/m^3$)

$$REL_{inh} \quad = \quad \text{Inhalation reference exposure level } (\mu g/m^3)$$

5.1.1 Acute Non-carcinogenic Results

The PMI for acute non-carcinogenic effects occurred at the southeastern fenceline at a property boundary receptor (#4300); the highest target organ-specific hazard index at that location was estimated to be 0.064. The highest target organ-specific hazard index in a residential area occurred south of the facility at a grid receptor (#2804) representing the MEIR; the hazard index at that location was estimated to be 0.050. The highest target organ-specific hazard index in a commercial/industrial area occurred south of the facility at a grid receptor (#2968) representing the MEIW; the hazard index at that location was estimated to be 0.051. The hazard indexes for the MEIW and MEIR are substantially below the regulatory significance level of 1.0.

The target organ/system with the highest hazard index is the respiratory system. Hazard indexes for all other target organs/systems (blood, bone, cardiovascular, central nervous system, developmental, endocrine, eye, gastrointestinal/liver, immune system, reproductive, and skin) were also less than 1.0 (Appendix B). Acrolein emissions contribute to approximately 77 percent of the acute target organ-specific hazard index for the key off-site receptors. Other TACs that contribute significantly to the hazard index include formaldehyde (approximately 10 percent) and sulfuric acid (approximately 8 percent). Table 4-3 presents the TAC-specific hazard quotients for acute health effects for the PMI, the MEIR, and the MEIW, and the locations of the key off-site receptors are shown in Figure 3-3.

5.1.2 Chronic Non-carcinogenic Results

The PMI for chronic non-carcinogenic health effects occurred at the southeastern fenceline at a property boundary receptor (#4297); the highest target organ-specific hazard index at that location was estimated to be 0.086. The highest target organ-specific hazard index in a residential area near the facility (MEIR) was at receptor #2890 with a hazard index at that location estimated to be 0.061. The highest target organ-specific hazard index in a commercial area near the facility (MEIW) was at receptor #2892 with a hazard index at that location estimated to be 0.065. The hazard indexes for the MEIW and MEIR are well below the regulatory significance level of 1.0.

The chronic non-carcinogenic hazard indexes were based on the effects to the respiratory system. Hazard indexes for all other target organs/systems (blood, bone, cardiovascular, central nervous system, developmental, endocrine, eye, gastrointestinal/liver, immune system, reproductive, and skin) were also less than 1.0 at the MEIW and MEIR (Appendix B). The compounds that contribute to the hazard indexes for the key off-site receptors are chlorine (48 percent), sulfuric acid (26 percent), formaldehyde (8 percent), manganese (7 percent), acrolein (6 percent) and hydrochloric acid (5 percent). Table 4-4 presents the TAC-specific hazard quotients for the PMI, the MEIR, and the MEIW, and the locations of the key off-site receptors are shown in Figure 3-3.

5.2   Carcinogenic Health Effects

In accordance with the OEHHA and CAPCOA guidance, cancer risk estimates based on the theoretical upper-bound excess cancer risk should be evaluated for the maximum exposed individuals, and a peak cancer receptor, if different.

For inhalation exposures, the theoretical upper-bound excess cancer risk is estimated assuming that an individual is exposed continuously to the annual average air concentrations over a 70 year lifetime. Once these annual average air concentrations are estimated for each of the receptors of interest, then the cancer risk is calculated for the carcinogenic AB 2588 TACs using the following equation:

$$\text{Cancer Risk}_{inh} = \text{GLC} \times \text{URV}$$

Where:

Cancer Risk$_{inh}$	=	Theoretical upper bound lifetime cancer risk
GLC	=	Ground-level concentration (µg/m3)
URV	=	Unit Risk Value (TAC-specific cancer potency factor) for inhalation $(µg/m^3)^{-1}$

For non-inhalation exposures, the theoretical upper-bound excess cancer risk is also estimated assuming that an individual is exposed continuously to the TACs over a 70-year lifetime. Once the lifetime oral dose from non-inhalation pathways is estimated, then the cancer risk is calculated for the carcinogenic TACs using the following equation:

$$\text{Cancer Risk}_{non\text{-}inh} = \text{OD} \times \text{OPV}$$

Where:

Cancer Risk$_{non\text{-}inh}$	=	Theoretical upper bound lifetime cancer risk
OD	=	Oral Dose (mg/kg/day)
OPV	=	Oral Potency Value (mg/kg/day)-1

Pursuant to the California Safe Drinking Water & Toxic Enforcement Act of 1986, the Office of Environmental Health Hazard Assessment has established a no significant risk level at 1x10 5 (California Code of Regulations Division 21.5, Title 22, §12703). In fact, many air management districts consider 1x10 5 to be an acceptable risk level for managing air emissions under the Toxics Hot Spots program.

5.2.1   Identification of the Zone Of Impact

The ZOI, as defined by CAPCOA, is the area within which there is a theoretical increased cancer risk of one-in-one million or greater based on a continuous, 70-year lifetime exposure to carcinogenic air emissions from the facility. The results from the HARP model for the evaluated receptor grids provides the information necessary to identify the ZOI by generating the isopleths (i.e., a geographical presentation of areas of equal risk) for the one-in-one million theoretical excess cancer risk. The modeling results indicated that the main ZOI extended approximately

1.1 kilometers north and south and approximately 0.5 kilometers east to west. There were three small additional ZOIs along Riverside Ave to the east of the facility. The ZOIs are presented along with the key off-site receptors (i.e., PMI, MEIR, MEIW) in Figure 3.

5.2.2 Estimated Theoretical Cancer Risks at Maximum Exposure Locations

The maximum off-site receptors for carcinogenic health effects occurred along the fenceline east of the facility at property boundary receptor #4310; the potential cancer risk at that location was estimated to be 2.1×10^{-6}. The maximum potential cancer risk predicted in a residential area south of the facility (MEIR) was at grid receptor #2890; the potential cancer risk at that location was estimated to be 1.3×10^{-6}. The maximum potential cancer risk predicted in a commercial/industrial area east of the facility (MEIW) was at grid receptor #3105; the potential cancer risk at that location was estimated to be 2.8×10^{-7}.

TACs that contribute significantly to the total cancer risks vary between the key off-site receptors, buy include arsenic, benzene, cadmium, diesel particulate matter, dioxin (2,3,7,8-TCDD), formaldehyde, and hexavalent chromium. Table 4-5 presents the TAC-specific risk for the maximum off-site receptor, the MEIR, and the MEIW. Based on this evaluation, the total excess cancer risk at the MEIR does not exceed the commonly applied level of significance (1×10^{-5}).

5.3 Lead Health Effects

The maximum estimated 30-day average concentrations of lead in air were 0.00051, 0.00040, and 0.00043 µg/m^3 for the PMI (property boundary receptor #4301), the MEIR (receptor #2804), and the MEIW (receptor #2758), respectively (see Table 4-6). All of the air concentrations were below the lead risk management level presented in the lead guidance (CARB, 2001) for neurodevelopmental effects in children assuming an average background exposure (0.3 µg/m^3). This is a conservative comparison for other potential health endpoints since the guidance value was calculated for the most sensitive target population (children). The results indicate that receptors at these locations would not experience adverse non-cancer health effects related to exposure to lead. Carcinogenic effects related to lead emissions are included in the total risks presented in Section 5.2.

6 Conclusions

The results of the risk evaluation are based on the operating conditions proposed in the combined ATC/PSD permit application and the TAC emissions calculated for the new boiler. Should boiler emissions, conditions, or toxicity criteria change, the information and conclusions in this report may no longer apply. The conclusions presented in this report are professional opinions based solely upon the data described in this report. They are intended exclusively for the purpose outlined herein and the site location and project indicated.

A summary of the acute and chronic target organ-specific non-carcinogenic, and carcinogenic health risks estimated for key off-site receptors is presented in Table 4-7. Based on the information provided for this HRA, the following conclusions can be made regarding the TAC emissions from the new boiler.

6.1 Acute Non-carcinogenic Health Hazards

The hazard indexes for acute target organ-specific non-carcinogenic effects were 0.064 for the PMI, 0.050 for the MEIR, and 0.051 for the MEIW. These values are all below 1.0, indicating that off-site impacts from boiler emissions should not result in unacceptable acute non-carcinogenic health effects under the conditions evaluated.

6.2 Chronic Non-carcinogenic Health Hazards

The hazard indexes for chronic target organ-specific non-carcinogenic effects were 0.086 for the PMI, 0.061 for the MEIR, and 0.065 for the MEIW. These values are all below 1.0, indicating that off-site impacts from boiler emissions should not result in unacceptable chronic non-carcinogenic health effects under the conditions evaluated.

6.3 Potential Carcinogenic Risks

The potential carcinogenic risks were 2.1×10^{-6} for the PMI, 1.3×10^{-6} for the MEIR, and 2.8×10^{-7} for the MEIW. These values do not exceed the commonly applied level of significance (1×10^{-5}, or ten-in-one million), indicating that off-site impacts from boiler emissions should not result in unacceptable carcinogenic health effects under the conditions evaluated.

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7 References

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Office of Environmental Health Hazard Assessment (OEHHA), 2003, The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments, August.

OEHHA/ARB, 2005, Consolidated Table of OEHHA/ARB Approved Risk Assessment Health Values, April.

ENVIRON

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Tables

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Table 2-1
Cogeneration Unit Air Toxic Contaminant Emission Rates

CAS No.	Chemical Name[1]	Annual Emissions[1,2] (lb/yr)	Maximum Hourly Emissions[1,2] (lb/hr)
83329	Acenaphthene	2.70E-02	3.39E-06
208968	Acenaphthylene	5.73E+00	7.19E-04
75070	Acetaldehyde	7.41E+02	9.30E-02
67641	Acetone	6.04E+02	7.58E-02
98862	Acetophenone	1.20E-02	1.51E-06
107028	Acrolein	1.18E+02	1.48E-02
7664417	Ammonia	7.53E+04	9.46E+00
120127	Anthracene	1.85E-01	2.32E-05
7440360	Antimony	1.72E+00	2.15E-04
7440382	Arsenic	1.84E+00	2.31E-04
7440393	Barium	5.67E+02	7.11E-02
100527	Benzaldehyde	3.15E+00	3.95E-04
71432	Benzene	3.21E+03	4.03E-01
56553	Benzo(a)anthracene	9.38E-03	1.18E-06
50328	Benzo(a)pyrene	1.22E-02	1.53E-06
205992	Benzo(b)fluoranthene	8.76E-03	1.10E-06
192972	Benzo(e)pyrene	9.66E-03	1.21E-06
191242	Benzo(g,h,i)perylene	1.72E-02	2.16E-06
205823	Benzo(j)fluoranthene	5.80E-01	7.28E-05
207089	Benzo(k)fluoranthene	8.88E-03	1.11E-06
65850	Benzoic Acid	1.74E-01	2.19E-05
7440417	Beryllium	5.78E+00	7.26E-04
117817	Bis(2-ethylhexyl)phthalate	1.73E-01	2.18E-05
74839	Bromomethane	1.04E+02	1.31E-02
78933	2-Butanone (MEK)	2.01E+01	2.52E-03
7440439	Cadmium	9.65E+00	1.21E-03
86748	Carbazole	6.67E+00	8.38E-04
37210165	Carbon Dioxide (CO_2)	7.71E+08	9.68E+04
56235	Carbon Tetrachloride	1.69E+02	2.12E-02
7782505	Chlorine	2.95E+03	3.71E-01
108907	Chlorobenzene	1.24E+02	1.55E-02
67663	Chloroform	1.03E+02	1.29E-02
74873	Chloromethane	8.61E+01	1.08E-02
91587	2-Chloronaphthalene	8.96E-03	1.13E-06
108430	2-Chlorophenol	1.26E-01	1.58E-05
18540299	Chromium, hexavalent	6.53E-01	8.20E-05
7440473	Chromium, trivalent	4.63E+00	5.82E-04
218019	Chrysene	1.03E-02	1.29E-06
7440484	Cobalt	3.33E+01	4.18E-03
7440508	Copper	1.53E+01	1.92E-03
4170303	Crotonaldehyde	3.69E+01	4.64E-03

ENVIRON

CAS No.	Chemical Name[1]	Annual Emissions[1,2] (lb/yr)	Maximum Hourly Emissions[1,2] (lb/hr)
2051243	Decachlorobiphenyl	9.88E-04	1.24E-07
53703	Dibenzo(a,h)anthracene	8.75E-03	1.10E-06
106934	1,2-Dibromoethene	2.04E+02	2.56E-02
2050682	Dichlorobiphenyl	1.41E-03	1.77E-07
107062	1,2-Dichloroethane	1.09E+02	1.37E-02
75092	Dichloromethane	1.07E+03	1.34E-01
78875	1,2-Dichloropropane	1.24E+02	1.56E-02
51285	2,4-Dinitrophenol	3.48E-01	4.36E-05
100414	Ethylbenzene	1.17E+02	1.46E-02
206440	Fluoranthene	1.93E+00	2.42E-04
86737	Fluorene	1.98E-01	2.48E-05
50000	Formaldehyde	7.30E+03	9.17E-01
28655712	Heptachlorobiphenyl	2.45E-04	3.07E-08
26601649	Hexachlorobiphenyl	1.08E-03	1.35E-07
37871004	HpCDD-Total	1.15E-04	1.44E-08
38998753	HpCDF-Total	2.38E-05	2.99E-09
34465468	HxCDD-Total	3.19E-04	4.00E-08
55684941	HxCDF-Total	5.71E-05	7.18E-09
66251	Hexanal	2.59E+01	3.26E-03
7647010	Hydrogen chloride	1.31E+04	1.65E+00
193395	Indeno(1,2,3-c,d)pyrene	8.85E-03	1.11E-06
7439896	Iron	3.70E+03	4.65E-01
78842	Isobutyraldehyde	4.29E+01	5.38E-03
7439921	Lead	4.45E+01	5.59E-03
7439965	Manganese	4.31E+02	5.41E-02
7439976	Mercury	1.55E+00	1.95E-04
74828	Methane	2.63E+05	3.30E+01
67561	Methanol	3.09E+03	3.88E-01
91576	2-Methylnaphthalene	1.02E+00	1.29E-04
7439987	Molybdenum	4.19E+00	5.26E-04
2051607	Monochlorobiphenyl	8.12E-04	1.02E-07
91203	Naphthalene	3.17E+02	3.98E-02
7440020	Nickel	1.06E+01	1.33E-03
10102439	Nitric Oxide (NO)	4.84E+05	6.08E+01
88755	2-Nitrophenol	3.96E-01	4.97E-05
100027	4-Nitrophenol	6.38E-01	8.01E-05
10024972	Nitrous Oxide (N$_2$O)	3.45E+04	4.33E+00
3268879	OCDD	8.71E-04	1.09E-07
39001020	OCDF	5.31E-05	6.67E-09
36088229	PeCDD-Total	6.39E-04	8.03E-08
30402154	PeCDF-Total	1.56E-04	1.96E-08
25429292	Pentachlorobiphenyl	2.42E-03	3.04E-07
87865	Pentachlorophenol	8.46E-02	1.06E-05

CAS No.	Chemical Name[1]	Annual Emissions[1,2] (lb/yr)	Maximum Hourly Emissions[1,2] (lb/hr)
198550	Perylene	1.93E-03	2.42E-07
86018	Phenanthrene	6.32E+00	7.93E-04
108952	Phenol	4.67E+01	5.87E-03
7723140	Phosphorus	1.32E+02	1.66E-02
7440097	Potassium	1.45E+05	1.82E+01
123386	Propionaldehyde	1.17E+01	1.47E-03
129000	Pyrene	1.11E+00	1.40E-04
7782492	Selenium	1.26E+01	1.58E-03
7440235	Sodium	1.35E+03	1.70E-01
7440246	Strontium	3.75E+01	4.71E-03
7664939	Sulfuric Acid	7.86E+03	9.86E-01
1746016	TCDD-Total	7.62E-04	9.57E-08
30402143	TCDF-Total	6.06E-04	7.60E-08
26914330	Tetrachlorobiphenyl	5.96E-03	7.49E-07
127184	Tetrachloroethene	1.42E+02	1.79E-02
7440315	Tin	1.46E+02	1.83E-02
7440326	Titanium	7.49E+01	9.41E-03
529204	o-Tolualdehyde	2.66E+01	3.35E-03
104870	p-Tolualdehyde	4.21E+01	5.29E-03
108883	Toluene	7.92E+01	9.94E-03
15862074	Trichlorobiphenyl	6.63E-03	8.33E-07
71556	1,1,1-Trichloroethane	1.15E+02	1.44E-02
79016	Trichloroethene	1.13E+02	1.42E-02
75694	Trichlorofluoromethane	1.51E+02	1.90E-02
88062	2,4,6-Trichlorophenol	4.23E-02	5.31E-06
1314621	Vanadium	2.21E+00	2.78E-04
75014	Vinyl Chloride	6.86E+01	8.61E-03
1330207	Xylene	9.12E+01	1.15E-02
7440655	Yttrium	1.12E+00	1.41E-04
7440666	Zinc	6.48E+02	8.14E-02

1. Abbreviations:

        HpCDD-Total = Total Heptachlorodibenzo-p-dioxin

        HpCDF-Total = Total Heptachlorodibenzofuran

        HxCDD-Total = Total Hexachlorodibenzo-p-dioxin

        HxCDF-Total = Total Hexachlorodibenzofuran

        OCDD = 1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin

        OCDF = 1,2,3,4,6,7,8,9-Octachlorodibenzofuran

        PeCDD-Total = Total Pentachlorodibenzo-p-dioxin

        PeCDF-Total = Total Pentachlorodibenzofuran

        TCDD-Total = 2,3,7,8-Tetrachlorodibenzo-p-dioxin

        TCDF-Total = Total Tetrachlorodibenzofuran

2. Chemical emission rates reported for the cogeneration unit in the Authority to Construct permit application submitted to Shasta County Air Quality Management District in February 2010.

Table 3-1
Fuel Haul Truck Emission Factors by Vehicle Speed

Speed Limit (miles per hour)	40-Year Emission Factor	70-Year Emission Factor	Emission Factor Unit
0	0.432	0.350	grams/idle-hour
15	0.190	0.152	grams/mile
25	0.129	0.107	grams/mile
35	0.113	0.096	grams/mile
45	0.120	0.104	grams/mile
65	0.201	0.173	grams/mile

Table 3-2
Modeled Road Segment Parameters and Calculated Emission Rates

Road Segment ID	Average Speed (mph)	Segment Length (meters)	One-way Trips Per Day	40-Year Emission Rate (lb/hr)	70-Year Emission Rate (lb/hr)
1	15	738	46	3.68E-04	2.94E-04
2	25	125	23	2.12E-05	1.76E-05
3	25	129	23	2.18E-05	1.81E-05
4	25	260	46	8.80E-05	7.30E-05
5	45	141	23	2.22E-05	1.92E-05
6	25	162	23	2.74E-05	2.28E-05
7	40	547	46	1.67E-04	1.43E-04
8	30	345	11.5	2.74E-05	2.30E-05
9	30	462	11.5	3.66E-05	3.08E-05
10	40	223	23	3.40E-05	2.92E-05
11	45	302	11.5	2.37E-05	2.06E-05
12	30	551	11.5	4.20E-05	3.61E-05

Table 3-3.
Source Emission Release Parameters

Emission Source	Source ID	UTM[1] Easting (m)	UTM[1] Northing (m)	Elevation (ft)	Stack Height (m)	Stack Temp (K)	Exit Velocity (m/s)	Stack Diameter (m)
New Boiler	1	557408.90	4480193.40	129.5	32.0	476	17.7	2.44

Emission Source	Source ID	UTM[1] Easting (m)	UTM[1] Northing (m)	Elevation (ft)	Release Height (m)	Sigma y (m)	Sigma z (m)
Fuel Haul Trucks	2-621	See Appendix A			2.0	3.721	4.233

1 Universal Transverse Mercator Coordinate System, Zone 10.

Table 3-4
Exposure Parameters

Parameter	Value	Rationale
Deposition Rate	0.02 m/s	Default for controlled sources
Fraction ingested – Homegrown produce	0.15	Conservative Default - Non-Urban
Receptor Grid Spacing	25 m	0 to 0.625 km from facility
	50 m	0.625 to 1.25 km from facility
	200 m	1.25 to 2.5 km from facility
	500 m	2.5 to 5 km from facility
Boundary Receptor Spacing	10 m	Protective of a large property line.

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Table 4-1
Toxic Air Contaminant Toxicity Criteria

CAS No.	Chemical	Acute Noncarcinogenic Inhalation Acute REL[1] (μg/m^3)	Chronic Noncarcinogenic Inhalation Chronic REL[1] (μg/m^3)	Oral Chronic REL[1] (mg/kg-day)	Carcinogenic Inhalation Unit Risk Value (μg/m^3)$^{-1}$	Inhalation Slope Factor (mg/kg-day)$^{-1}$	Oral Potency Value (mg/kg-day)$^{-1}$
83329	Acenaphthene	N/A	N/A	N/A	N/A	N/A	N/A
208968	Acenaphthylene	N/A	N/A	N/A	N/A	N/A	N/A
75070	Acetaldehyde	N/A	9	N/A	0.0000027	0.01	N/A
67641	Acetone	N/A	N/A	N/A	N/A	N/A	N/A
98862	Acetophenone	N/A	N/A	N/A	N/A	N/A	N/A
107028	Acrolein	0.19	0.06	N/A	N/A	N/A	N/A
7664417	Ammonia	3,200	200	N/A	N/A	N/A	N/A
120127	Anthracene	N/A	N/A	N/A	N/A	N/A	N/A
7440360	Antimony	N/A	N/A	N/A	N/A	N/A	N/A
7440382	Arsenic	0.19	0.03	0.0003	0.0033	12	1.5
7440393	Barium	N/A	N/A	N/A	N/A	N/A	N/A
100527	Benzaldehyde	N/A	N/A	N/A	N/A	N/A	N/A
71432	Benzene	1,300	60	N/A	0.000029	0.1	N/A
56553	Benzo(a)anthracene	N/A	N/A	N/A	0.00011	0.39	1.2
50328	Benzo(a)pyrene	N/A	N/A	N/A	0.0011	3.9	12
205992	Benzo(b)fluoranthene	N/A	N/A	N/A	0.00011	0.39	1.2
192972	Benzo(e)pyrene	N/A	N/A	N/A	N/A	N/A	N/A
191242	Benzo(g,h,i)perylene	N/A	N/A	N/A	N/A	N/A	N/A
205823	Benzo(j)fluoranthene	N/A	N/A	N/A	0.00011	0.39	1.2
207089	Benzo(k)fluoranthene	N/A	N/A	N/A	0.00011	0.39	1.2
65850	Benzoic Acid	N/A	N/A	N/A	N/A	N/A	N/A
7440417	Beryllium	N/A	0.007	0.002	0.0024	8.4	N/A

CAS No.	Chemical	Acute Noncarcinogenic	Chronic Noncarcinogenic		Carcinogenic		
		Inhalation Acute REL[1] (µg/m³)	Inhalation Chronic REL[1] (µg/m³)	Oral Chronic REL[1] (mg/kg-day)	Inhalation Unit Risk Value (µg/m³)⁻¹	Inhalation Slope Factor (mg/kg-day)⁻¹	Oral Potency Value (mg/kg-day)⁻¹
117817	Bis(2-ethylhexyl)phthalate	N/A	N/A	N/A	0.0000024	0.0084	0.0084
74839	Bromomethane	3,900	5	N/A	N/A	N/A	N/A
78933	2-Butanone (MEK)	13,000	N/A	N/A	N/A	N/A	N/A
7440439	Cadmium	N/A	0.02	0.0005	0.0042	15	N/A
86748	Carbazole	N/A	N/A	N/A	N/A	N/A	N/A
37210165	Carbon Dioxide (CO2)	N/A	N/A	N/A	N/A	N/A	N/A
56235	Carbon Tetrachloride	1,900	40	N/A	0.000042	0.15	N/A
7782505	Chlorine	210	0.2	N/A	N/A	N/A	N/A
108907	Chlorobenzene	N/A	1000	N/A	N/A	N/A	N/A
67663	Chloroform	150	300	N/A	0.0000053	0.019	N/A
74873	Chloromethane	N/A	N/A	N/A	N/A	N/A	N/A
91587	2-Chloronaphthalene	N/A	N/A	N/A	N/A	N/A	N/A
108430	2-Chlorophenol	N/A	N/A	N/A	N/A	N/A	N/A
18540299	Chromium, hexavalent	N/A	0.2	0.02	0.15	510	N/A
7440473	Chromium, trivalent	N/A	N/A	N/A	N/A	N/A	N/A
218019	Chrysene	N/A	N/A	N/A	0.000011	0.039	0.12
7440484	Cobalt	N/A	N/A	N/A	N/A	N/A	N/A
7440508	Copper	100	N/A	N/A	N/A	N/A	N/A
4170303	Crotonaldehyde	N/A	N/A	N/A	N/A	N/A	N/A
2051243	Decachlorobiphenyl	N/A	N/A	N/A	N/A	N/A	N/A
53703	Dibenzo(a,h)anthracene	N/A	N/A	N/A	0.0012	4.1	4.1
106934	1,2-Dibromoethene	N/A	0.8	N/A	0.000071	0.25	N/A

CAS No.	Chemical	Acute Noncarcinogenic	Chronic Noncarcinogenic		Carcinogenic		
		Inhalation Acute REL[1] (μg/m^3)	Inhalation Chronic REL[1] (μg/m^3)	Oral Chronic REL[1] (mg/kg-day)	Inhalation Unit Risk Value (μg/m^3)$^{-1}$	Inhalation Slope Factor (mg/kg-day)$^{-1}$	Oral Potency Value (mg/kg-day)$^{-1}$
2050682	Dichlorobiphenyl	N/A	N/A	N/A	N/A	N/A	N/A
107062	1,2-Dichloroethane	N/A	400	N/A	0.000021	0.072	N/A
75092	Dichloromethane	14,000	400	N/A	0.000001	0.0035	N/A
78875	1,2-Dichloropropane	N/A	N/A	N/A	0.000018	0.063	N/A
9901	Diesel Particulate Matter	N/A	5	N/A	0.0003	1.1	N/A
51285	2,4-Dinitrophenol	N/A	N/A	N/A	N/A	N/A	N/A
100414	Ethylbenzene	N/A	2000	N/A	0.0000025	0.0087	N/A
206440	Fluoranthene	N/A	N/A	N/A	N/A	N/A	N/A
86737	Fluorene	N/A	N/A	N/A	N/A	N/A	N/A
50000	Formaldehyde	94	3	N/A	0.000006	0.021	N/A
28655712	Heptachlorobiphenyl	N/A	N/A	N/A	N/A	N/A	N/A
26601649	Hexachlorobiphenyl	N/A	N/A	N/A	N/A	N/A	N/A
37871004	HpCDD-Total	N/A	N/A	N/A	N/A	N/A	N/A
38998753	HpCDF-Total	N/A	N/A	N/A	N/A	N/A	N/A
34465468	HxCDD-Total	N/A	N/A	N/A	N/A	N/A	N/A
55684941	HxCDF-Total	N/A	N/A	N/A	N/A	N/A	N/A
66251	Hexanal	N/A	N/A	N/A	N/A	N/A	N/A
7647010	Hydrogen chloride	2,100	9	N/A	N/A	N/A	N/A
193395	Indeno(1,2,3-c,d)pyrene	N/A	N/A	N/A	0.00011	0.39	1.2

CAS No.	Chemical	Acute Noncarcinogenic	Chronic Noncarcinogenic		Carcinogenic		
		Inhalation Acute REL[1] (µg/m³)	Inhalation Chronic REL[1] (µg/m³)	Oral Chronic REL[1] (mg/kg-day)	Inhalation Unit Risk Value (µg/m³)⁻¹	Inhalation Slope Factor (mg/kg-day)⁻¹	Oral Potency Value (mg/kg-day)⁻¹
7439896	Iron	N/A	N/A	N/A	N/A	N/A	N/A
78842	Isobutyraldehyde	N/A	N/A	N/A	N/A	N/A	N/A
7439921	Lead	N/A	N/A	N/A	0.000012	0.042	0.0085
7439965	Manganese	N/A	0.2	N/A	N/A	N/A	N/A
7439976	Mercury	1.8	0.09	0.0003	N/A	N/A	N/A
74828	Methane	N/A	N/A	N/A	N/A	N/A	N/A
67561	Methanol	28,000	4000	N/A	N/A	N/A	N/A
91576	2-Methylnaphthalene	N/A	N/A	N/A	N/A	N/A	N/A
7439987	Molybdenum	N/A	N/A	N/A	N/A	N/A	N/A
2051607	Monochlorobiphenyl	N/A	N/A	N/A	N/A	N/A	N/A
91203	Naphthalene	N/A	9	N/A	0.000034	0.12	N/A
7440020	Nickel	6	0.05	0.05	0.00026	0.91	N/A
10102439	Nitric Oxide (NO)	N/A	N/A	N/A	N/A	N/A	N/A
88755	2-Nitrophenol	N/A	N/A	N/A	N/A	N/A	N/A
100027	4-Nitrophenol	N/A	N/A	N/A	N/A	N/A	N/A
10024972	Nitrous Oxide (N2O)	N/A	N/A	N/A	N/A	N/A	N/A
3268879	OCDD	N/A	0.4	0.0001	0.0038	13	13
39001020	OCDF	N/A	0.4	0.0001	0.0038	13	13
36088229	PeCDD-Total	N/A	N/A	N/A	N/A	N/A	N/A
30402154	PeCDF-Total	N/A	N/A	N/A	N/A	N/A	N/A
25429292	Pentachlorobiphenyl	N/A	N/A	N/A	N/A	N/A	N/A

CAS No.	Chemical	Acute Noncarcinogenic	Chronic Noncarcinogenic		Carcinogenic		
		Inhalation Acute REL[1] (µg/m^3)	Inhalation Chronic REL[1] (µg/m^3)	Oral Chronic REL[1] (mg/kg-day)	Inhalation Unit Risk Value (µg/m^3)$^{-1}$	Inhalation Slope Factor (mg/kg-day)$^{-1}$	Oral Potency Value (mg/kg-day)$^{-1}$
87865	Pentachlorophenol	N/A	N/A	N/A	0.0000051	0.018	N/A
198550	Perylene	N/A	N/A	N/A	N/A	N/A	N/A
86018	Phenanthrene	N/A	N/A	N/A	N/A	N/A	N/A
108952	Phenol	5,800	200	N/A	N/A	N/A	N/A
7723140	Phosphorus	N/A	N/A	N/A	N/A	N/A	N/A
7440097	Potassium	N/A	N/A	N/A	N/A	N/A	N/A
123386	Propionaldehyde	N/A	N/A	N/A	N/A	N/A	N/A
129000	Pyrene	N/A	N/A	N/A	N/A	N/A	N/A
7782492	Selenium	N/A	20	N/A	N/A	N/A	N/A
7440235	Sodium	N/A	N/A	N/A	N/A	N/A	N/A
7440246	Strontium	N/A	N/A	N/A	N/A	N/A	N/A
7664939	Sulfuric Acid	120	1	N/A	N/A	N/A	N/A
1746016	TCDD-Total	N/A	0.00004	1E-08	38	130,000	130,000
30402143	TCDF-Total	N/A	N/A	N/A	N/A	N/A	N/A
26914330	Tetrachlorobiphenyl	N/A	N/A	N/A	N/A	N/A	N/A
127184	Tetrachloroethene	20,000	35	N/A	0.0000059	0.021	N/A
7440315	Tin	N/A	N/A	N/A	N/A	N/A	N/A
7440326	Titanium	N/A	N/A	N/A	N/A	N/A	N/A
529204	o-Tolualdehyde	N/A	N/A	N/A	N/A	N/A	N/A
104870	p-Tolualdehyde	N/A	N/A	N/A	N/A	N/A	N/A
108883	Toluene	37,000	300	N/A	N/A	N/A	N/A
15862074	Trichlorobiphenyl	N/A	N/A	N/A	N/A	N/A	N/A

CAS No.	Chemical	Acute Noncarcinogenic	Chronic Noncarcinogenic		Carcinogenic		
		Inhalation Acute REL[1] (µg/m^3)	Inhalation Chronic REL[1] (µg/m^3)	Oral Chronic REL[1] (mg/kg-day)	Inhalation Unit Risk Value (µg/m^3)$^{-1}$	Inhalation Slope Factor (mg/kg-day)$^{-1}$	Oral Potency Value (mg/kg-day)$^{-1}$
71556	1,1,1-Trichloroethane	68,000	1,000	N/A	N/A	N/A	N/A
79016	Trichloroethene	N/A	600	N/A	0.000002	0.007	N/A
75694	Trichlorofluoromethane	N/A	N/A	N/A	N/A	N/A	N/A
88062	2,4,6-Trichlorophenol	N/A	N/A	N/A	0.00002	0.07	N/A
1314621	Vanadium	30	N/A	N/A	N/A	N/A	N/A
75014	Vinyl Chloride	180,000	N/A	N/A	0.000078	0.27	N/A
1330207	Xylene	22,000	700	N/A	N/A	N/A	N/A
7440655	Yttrium	N/A	N/A	N/A	N/A	N/A	N/A
7440666	Zinc	N/A	N/A	N/A	N/A	N/A	N/A

1 REL = Reference Exposure Level

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Table 4-2
Census Data Summary for Lead Exposure

Ratio of Income to 1999 Poverty Level[1]	Number of Persons		
	Census Tract 115[2]	Census Tract 121[2]	Census Tract 123.02[2]
Under 0.50	210	548	248
0.50 to 0.74	11	414	119
0.75 to 0.99	139	401	209
1.00 to 1.24	264	239	285
1.25 to 1.49	317	419	267
1.50 to 1.74	334	335	255
1.75 to 1.84	79	276	83
1.85 to 1.99	112	136	135
2.00 and over	3,627	2,626	3,112
Number less than 1.25 times the poverty level	624	1,602	861
Total	5,093	5,394	4,713
Percentage[3]	12%	30%	18%
Median Year Structure Built[2]	1976	1974	1978

1 U.S. Census Bureau; Census 2000 Summary File 3 (SF 3) - Sample Data - http://factfinder.census.gov

2 Census tracts 115, 121, and 123.02 in Shasta County

3 Percentage of persons with an income less than 1.25 times the poverty level.

Table 4-3
Summary of Maximum Potential Acute Noncarcinogenic
Hazard Indices for Key Receptors

CAS No.	Chemical	PMI (#4300)	MEIR (#2804)	MEIW (#2968)
107028	Acrolein	2.39E-02	1.88E-02	1.88E-02
7664417	Ammonia	9.07E-04	7.14E-04	7.15E-04
7440382	Arsenic	3.73E-04	2.94E-04	2.94E-04
71432	Benzene	9.52E-05	7.49E-05	7.50E-05
74839	Bromomethane	1.03E-06	8.11E-07	8.13E-07
78933	2-Butanone (MEK)	5.95E-08	4.68E-08	4.69E-08
56235	Carbon Tetrachloride	3.43E-06	2.69E-06	2.70E-06
7782505	Chlorine	5.42E-04	4.27E-04	4.27E-04
67663	Chloroform	2.64E-05	2.08E-05	2.08E-05
7440508	Copper	5.89E-06	4.64E-06	4.65E-06
75092	Dichloromethane	2.94E-06	2.31E-06	2.32E-06
50000	Formaldehyde	2.99E-03	2.36E-03	2.36E-03
7647010	Hydrogen chloride	2.41E-04	1.90E-04	1.90E-04
7439976	Mercury	3.33E-05	2.62E-05	2.62E-05
67561	Methanol	4.25E-06	3.35E-06	3.35E-06
7440020	Nickel	6.80E-05	5.35E-05	5.36E-05
108952	Phenol	3.11E-07	2.44E-07	2.45E-07
7664939	Sulfuric Acid	2.52E-03	1.98E-03	1.99E-03
127184	Tetrachloroethene	2.75E-07	2.16E-07	2.17E-07
108883	Toluene	8.25E-08	6.49E-08	6.50E-08
71556	1,1,1-Trichloroethane	6.50E-08	5.11E-08	5.12E-08
1314621	Vanadium	2.84E-06	2.24E-06	2.24E-06
75014	Vinyl Chloride	1.47E-08	1.16E-08	1.16E-08
1330207	Xylene	1.60E-07	1.26E-07	1.26E-07
	Cumulative Total	3.12E-02	2.45E-02	2.46E-02

1. The maximum acute hazard index was highest for the respiratory system. Acute hazard indexes for the other target systems/organs were lower and are presented in Appendix A.

Table 4-4
Summary of Maximum Potential Chronic Noncarcinogenic
Hazard Indices for Key Receptors

CAS No.	Chemical	PMI (#4297)	MEIR (#2890)	MEIW[2] (#2892)	Maximum Target
75070	Acetaldehyde	1.67E-04	1.20E-04	1.33E-04	RESP
107028	Acrolein	3.99E-03	2.86E-03	3.18E-03	EYE
7664417	Ammonia	7.64E-04	5.48E-04	6.09E-04	RESP
7440382	Arsenic	2.98E-04	2.13E-04	1.77E-04	CV
71432	Benzene	1.09E-04	7.78E-05	8.65E-05	CNS
7440417	Beryllium	1.68E-03	1.20E-03	1.33E-03	IMMUN
74839	Bromomethane	4.22E-05	3.03E-05	3.36E-05	CNS
7440439	Cadmium	2.16E-03	1.55E-03	8.57E-04	KIDN
56235	Carbon Tetrachloride	8.58E-06	6.14E-06	6.83E-06	CNS
7782505	Chlorine	2.99E-02	2.15E-02	2.38E-02	RESP
108907	Chlorobenzene	2.52E-07	1.80E-07	2.00E-07	GILV
67663	Chloroform	6.97E-07	4.99E-07	5.55E-07	DEVEL
18540299	Chromium, hexavalent	6.63E-06	4.75E-06	5.28E-06	RESP
106934	1,2-Dibromoethene	5.18E-04	3.71E-04	4.12E-04	REPRO
107062	1,2-Dichloroethane	5.53E-07	3.96E-07	4.41E-07	GILV
75092	Dichloromethane	5.43E-06	3.89E-06	4.32E-06	CV
9901	Diesel Particulate Matter	1.33E-04	3.88E-05	6.00E-05	RESP
100414	Ethylbenzene	1.19E-07	8.51E-08	9.46E-08	DEVEL
50000	Formaldehyde	4.94E-03	3.54E-03	3.93E-03	EYE
7647010	Hydrogen chloride	2.95E-03	2.12E-03	2.35E-03	RESP
7439965	Manganese	4.37E-03	3.13E-03	3.48E-03	CNS
7439976	Mercury	5.71E-04	4.09E-04	1.34E-04	IMMUN
67561	Methanol	1.57E-06	1.12E-06	1.25E-06	DEVEL
91203	Naphthalene	7.15E-05	5.12E-05	5.69E-05	RESP
7440020	Nickel	4.30E-04	3.08E-04	3.43E-04	RESP
3268879	OCDD	5.87E-08	4.20E-08	2.43E-08	DEVEL
39001020	OCDF	3.58E-09	2.56E-09	1.48E-09	DEVEL
108952	Phenol	4.74E-07	3.40E-07	3.77E-07	CV
7782492	Selenium	1.28E-06	9.16E-07	1.02E-06	CV
7664939	Sulfuric Acid	1.60E-02	1.14E-02	1.27E-02	RESP
1746016	TCDD-Total	5.13E-04	3.68E-04	2.13E-04	DEVEL
127184	Tetrachloroethene	8.24E-06	5.90E-06	6.56E-06	GILV
108883	Toluene	5.36E-07	3.84E-07	4.27E-07	CNS
71556	1,1,1-Trichloroethane	2.33E-07	1.67E-07	1.86E-07	CNS
79016	Trichloroethene	3.82E-07	2.74E-07	3.04E-07	CNS
1330207	Xylene	2.64E-07	1.89E-07	2.11E-07	CNS
	Cumulative Total	8.57E-02	6.13E-02	6.53E-02	--

1. The maximum chronic hazard index was highest for the respiratory system. Chronic hazard indexes for the other target systems/organs were lower and are presented in Appendix A.

2. Exposure adjusted in the HARP model for a standard work schedule (49 wk/yr, 5 dy/wk, 8 hr/dy, 40 yr).

Table 4-5
Summary of Maximum Potential Carcinogenic Risk for Key Receptors

CAS No.	Chemical	PMI (#4310)	MEIR (#2890)	MEIW[1] (#3105)
75070	Acetaldehyde	4.96E-09	4.06E-09	1.69E-10
7440382	Arsenic	7.56E-08	6.19E-08	3.81E-09
71432	Benzene	2.15E-07	1.76E-07	7.33E-09
56553	Benzo(a)anthracene	1.1E-10	8.98E-11	2.25E-12
50328	Benzo(a)pyrene	1.43E-09	1.17E-09	2.93E-11
205992	Benzo(b)fluoranthene	1.02E-10	8.39E-11	2.1E-12
205823	Benzo(j)fluoranthene	6.79E-09	5.55E-09	1.39E-10
207089	Benzo(k)fluoranthene	1.04E-10	8.5E-11	2.13E-12
7440417	Beryllium	3.25E-08	2.66E-08	1.11E-09
117817	Bis(2-ethylhexyl)phthalate	1.18E-11	9.66E-12	4.25E-14
7440439	Cadmium	9.7E-08	7.93E-08	3.31E-09
56235	Carbon Tetrachloride	1.7E-08	1.39E-08	5.79E-10
67663	Chloroform	1.31E-09	1.07E-09	4.47E-11
18540299	Chromium, hexavalent	2.23E-07	1.83E-07	7.61E-09
218019	Chrysene	1.21E-11	9.86E-12	2.47E-13
53703	Dibenzo(a,h)anthracene	3.61E-10	2.95E-10	7.73E-12
106934	1,2-Dibromoethene	3.42E-08	2.8E-08	1.17E-09
107062	1,2-Dichloroethane	5.26E-09	4.3E-09	1.79E-10
75092	Dichloromethane	2.51E-09	2.05E-09	8.55E-11
78875	1,2-Dichloropropane	5.23E-09	4.28E-09	1.78E-10
9901	Diesel Particulate Matter	6.14E-07	8.05E-08	0.000000219
100414	Ethylbenzene	6.82E-10	5.58E-10	2.33E-11
50000	Formaldehyde	1.03E-07	8.4E-08	3.5E-09
193395	Indeno(1,2,3-c,d)pyrene	1.04E-10	8.47E-11	2.12E-12
7439921	Lead	6.47E-09	5.29E-09	2.57E-10
91203	Naphthalene	2.55E-08	2.08E-08	8.69E-10
7440020	Nickel	6.46E-09	5.29E-09	2.2E-10
3268879	OCDD	6.55E-11	5.36E-11	2.82E-12
39001020	OCDF	3.99E-12	3.27E-12	1.72E-13
87865	Pentachlorophenol	1.02E-12	8.35E-13	3.48E-14
1746016	TCDD-Total	5.73E-07	4.69E-07	2.47E-08
127184	Tetrachloroethene	2E-09	1.63E-09	6.81E-11
79016	Trichloroethene	5.3E-10	4.34E-10	1.81E-11
88062	2,4,6-Trichlorophenol	1.98E-12	1.62E-12	6.76E-14
75014	Vinyl Chloride	1.24E-08	1.02E-08	4.23E-10
	Cumulative Total	2.07E-06	1.27E-06	2.75E-07

1. Exposure adjusted in the HARP model for a standard work schedule (49 wk/yr, 5 dy/wk, 8 hr/dy, 40 yr).

Table 4-6
Predicted Lead Air Concentrations

Receptor Type	Receptor #	Dispersion Factor[1] (x/Q)	Lead Emission Rate (ER)	Maximum Lead Concentration[2] (Ca)	30-Day Average Correction Factor[3]	30-Day Average Concentration[4]	Lead Risk Management Level[5] ($\mu g/m^3$)
Fenceline	4301	2.44	0.00070	0.0017	0.3	0.00051	0.3
Resident	2804	1.92	0.00070	0.0013	0.3	0.00040	0.3
Commercial/ Industrial Worker	2758	2.05	0.00070	0.0014	0.3	0.00043	0.3

1. The maximum predicted 1-hour dispersion factor (normalized concentration) for key receptors ($\mu g/m^3/g/s$)

2. The maximum predicted concentration of lead in air is based on the maximum one-hour dispersion factors and is calculated as: Ca ($\mu g/m^3$) = x/Q ($\mu g/m^3$)/(g/s) x ER (g/s)

3. A factor of 0.3 is used to adjust the one-hour maximum data to a 30-day average for lead (ARB, 2001)

4. Modeled 1-hour average air concentration ($\mu g/m^3$) multiplied by the correction factor to obtain a 30-day average concentration. Average potential existing lead exposure is applicable because the median age of housing is later than 1960 (1974), and the percentage of persons with an income less than 1.25 times the poverty level is at 30 percent; (ARB, 2001)

5. Lead risk management level for neurodevelopmental effects in children assuming an average background exposure of 0.3 $\mu g/m^3$ (CARB, 2001)

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Table 4-7
Summary of Hazard Indices and Risks for Key Receptors

| Endpoint | Receptor Type | Receptor # | Coordinates | | Maximum Risk/HI |
			UTM[1] Easting	UTM[1] Northing	
Acute Noncarcinogenic	Fenceline	4300	557402.2	4479760.9	0.064
	Residential	2804	557200	4479600	0.050
	Commercial/Industrial Worker	2968	557550	4479600	0.051
Chronic Noncarcinogenic	Fenceline	4297	557402.4	4479737.4	0.086
	Residential	2890	557400	4479350	0.061
	Commercial/Industrial Worker	2892	557400	4479450	0.065
Carcinogenic	Fenceline	4310	557472.9	4479824.9	2.1E-06
	Residential	2890	557400	4479350	1.3E-06
	Commercial/Industrial Worker	3105	557800	4479950	2.8E-07

1 Universal Transverse Mercator Coordinate System, Zone 10.

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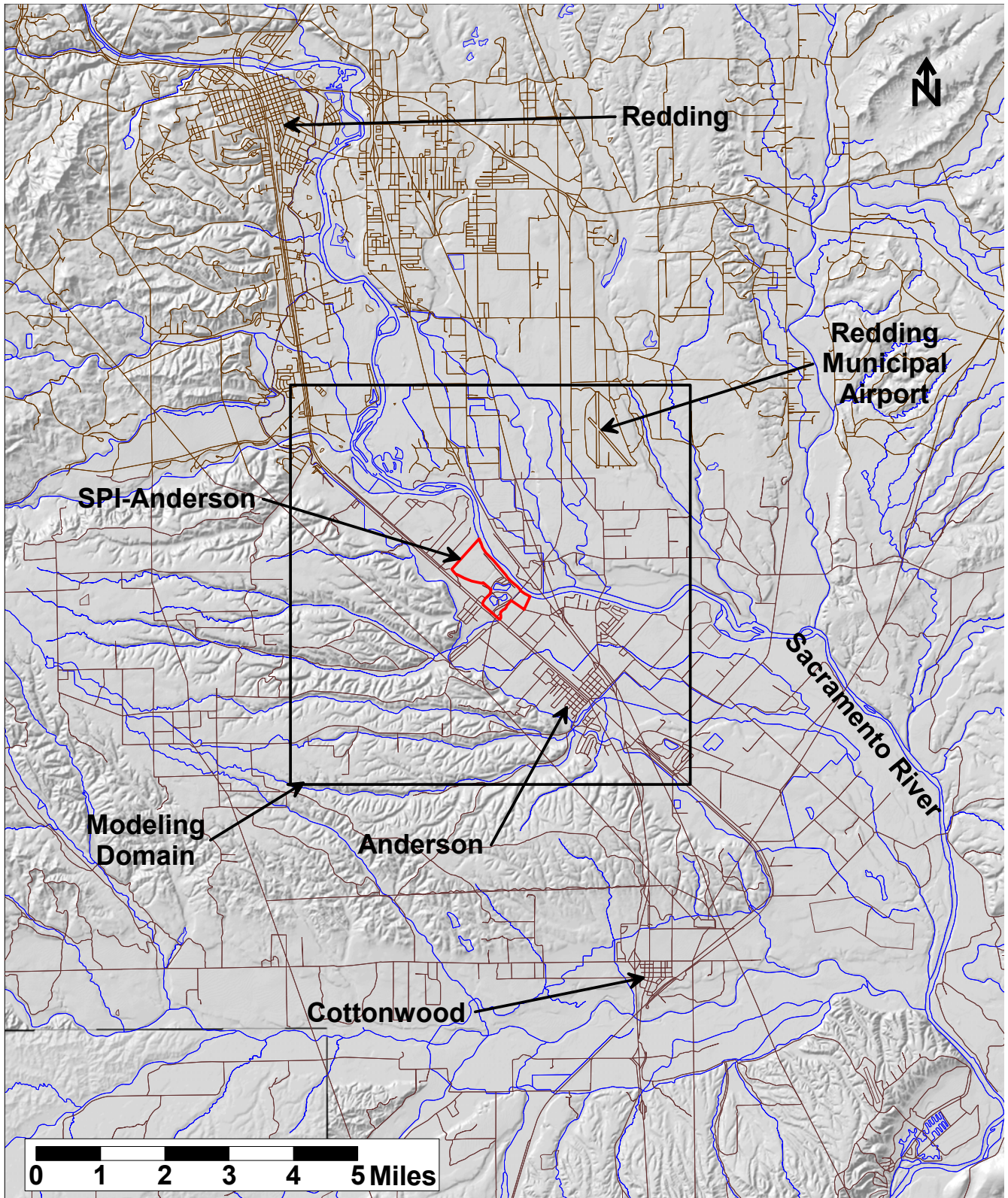
Figures

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Figure 1-1. Vicinity of Facility and Modeling Domain

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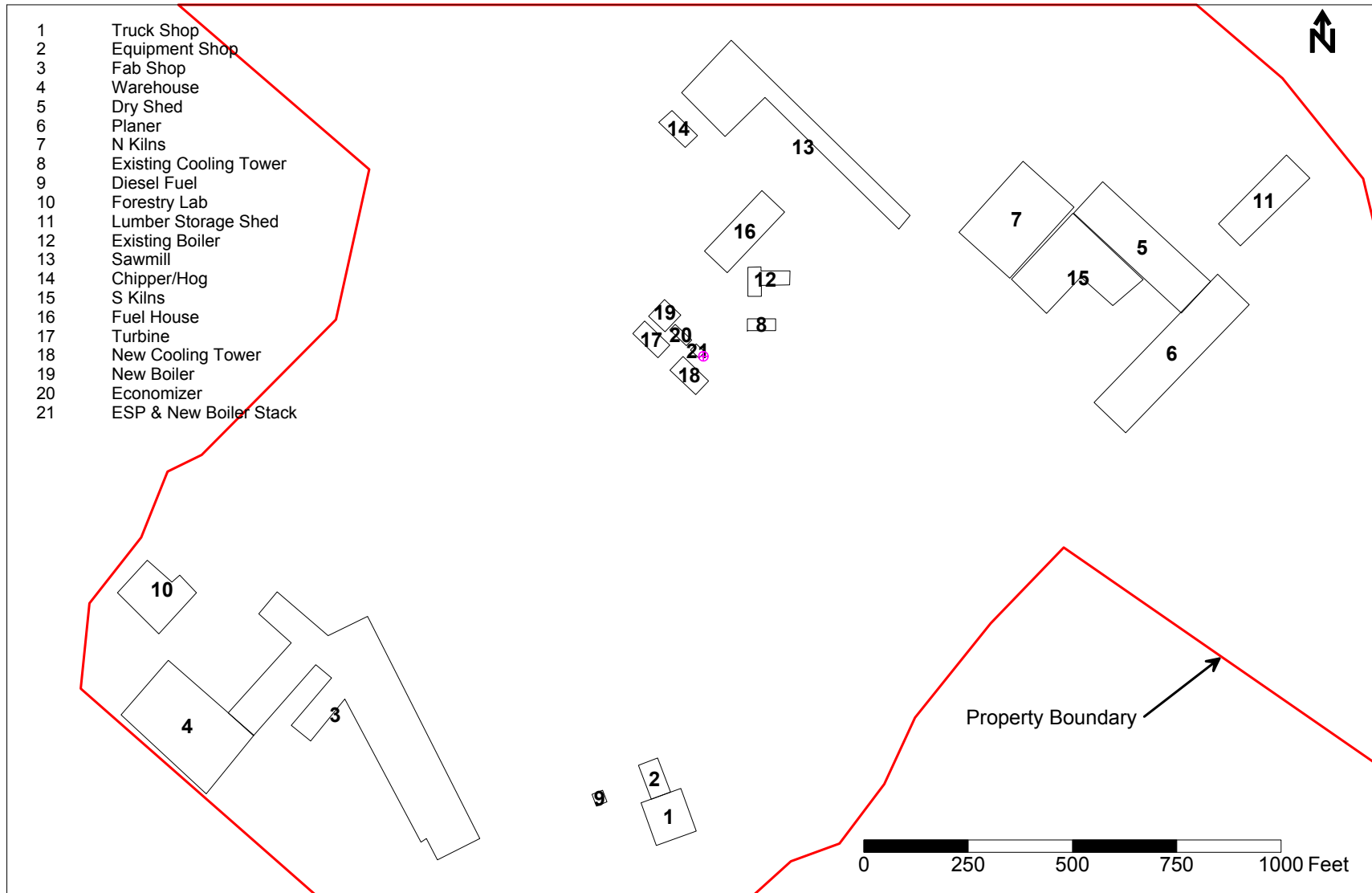(This page intentionally left blank.)

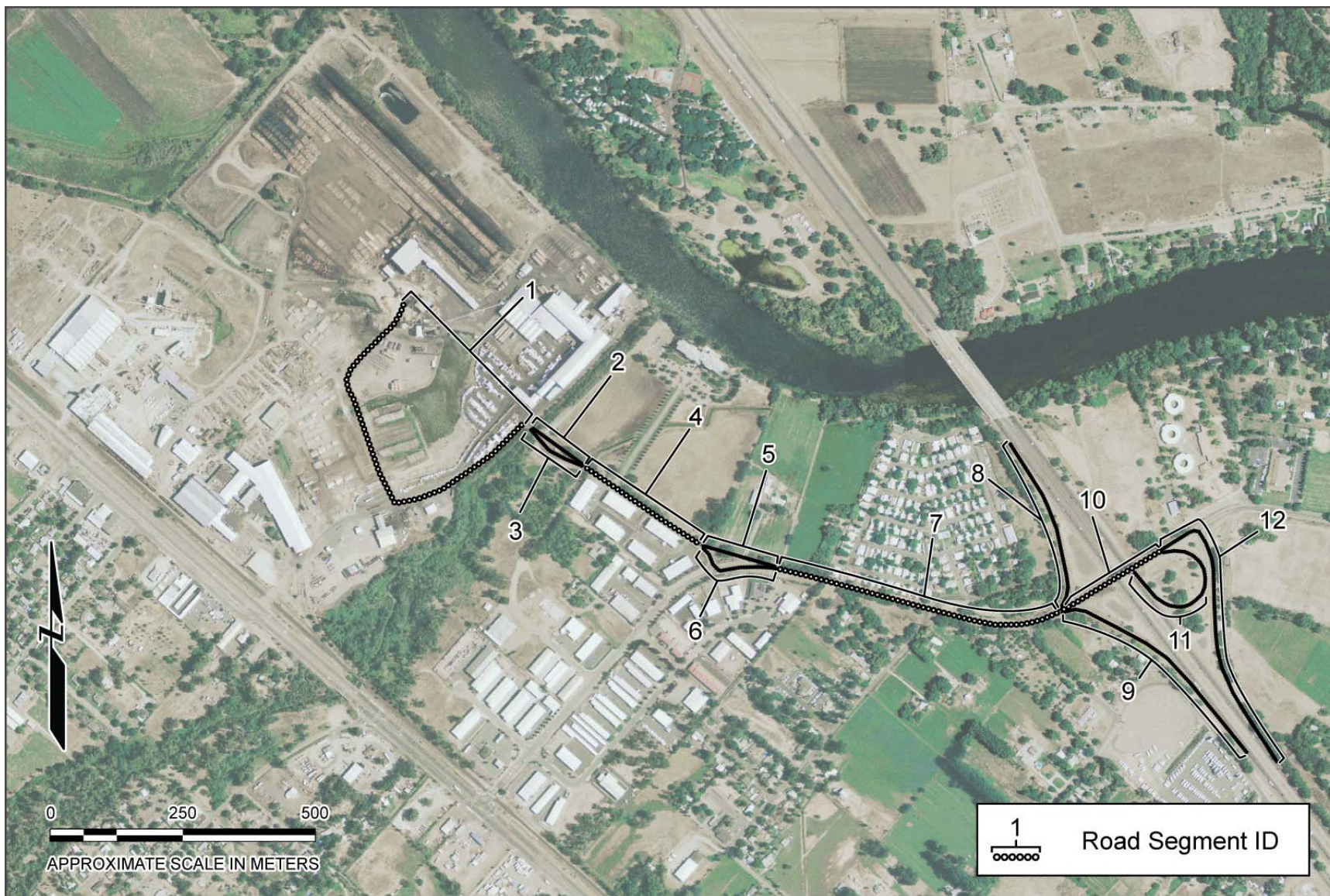**Figure 3-1. Facility Plot Plan and Cogeneration Unit Stack Location**
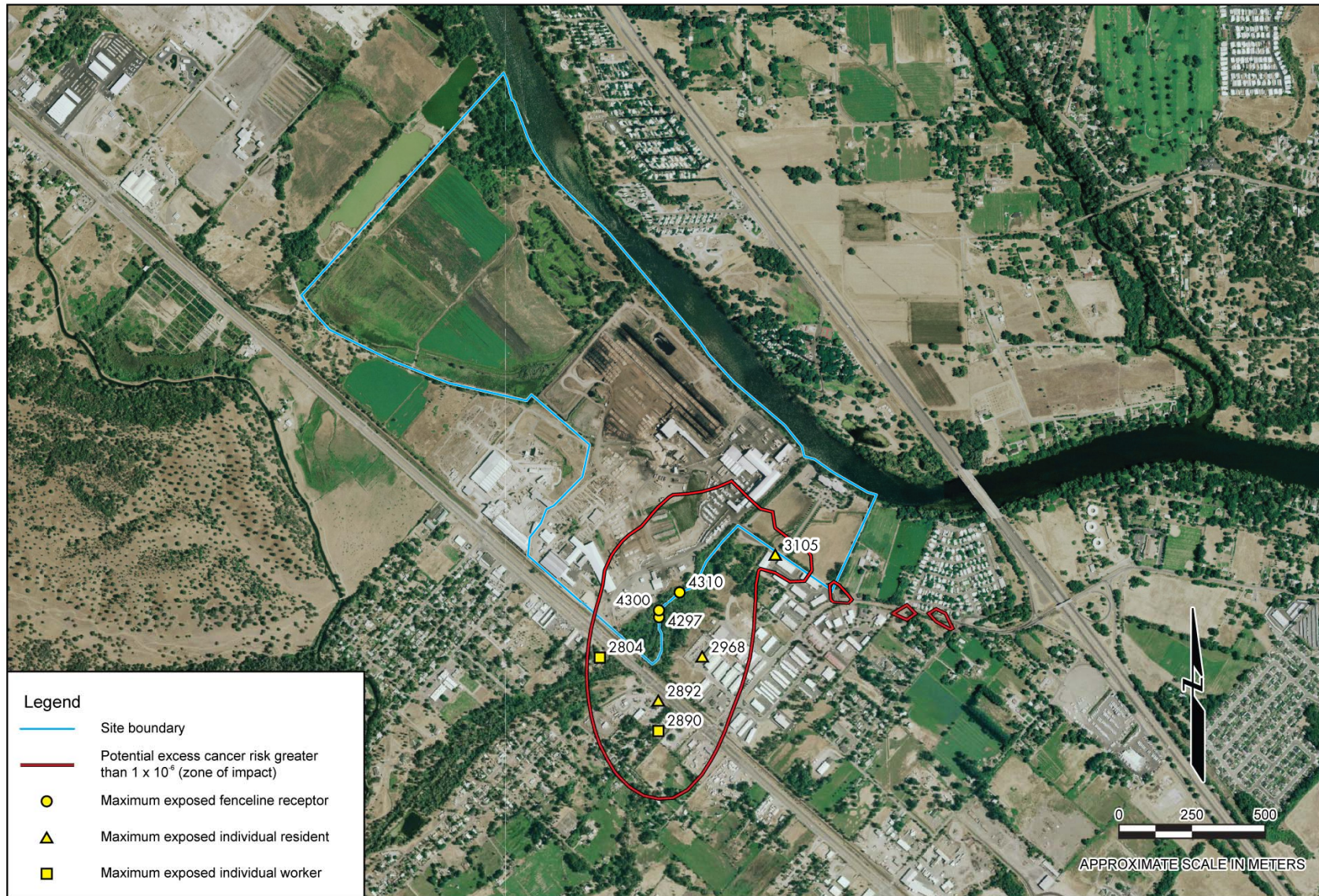
Figure 3-2. Fuel Haul Truck Route

Figure 3-3. Locations of Key Off-Site Receptors and Excess Carcinogenic Risk

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Appendix A:
CD-ROM of AERMOD and HARP
Input and Output Files

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February 2010

Project No.
29-23586A

Sierra Pacific Industries
Biomass-Fired Cogeneration
Project HRA
Anderson, CA
AERMOD and HARP
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