Appendix C3
Health Risk Assessment for the Southern California Intermodal Gateway (SCIG)

Appendix C3

Health Risk Assessment for the Southern California Intermodal Gateway (SCIG)

1.0 Introduction

This document describes the methods and results of a health risk assessment (HRA) that evaluates potential public health effects from toxic air contaminant (TAC) emissions generated by the construction and operation of the Port of Los Angeles SCIG Project (Project or proposed Project). TACs are compounds that are known or suspected to cause adverse health effects after short-term (acute) or long-term (chronic) exposure.

The HRA evaluated health effects associated with the following alternatives:

- Unmitigated Proposed Project with and without mitigation
- No Project
- Reduced Project, with and without mitigation

The HRA analyzed Project emissions and potential human exposure to the emissions during the 70-year period from 2013 to 2082; the Baseline is based on the 70-year period from 2005-2074.

This HRA was prepared in accordance with the *Health Risk Assessment Protocol for Port of Los Angeles Terminal Improvement Projects* (Protocol) (Port of Los Angeles, 2005a). The Protocol is a living document, developed by the Port in consultation with the South Coast Air Quality Management District (SCAQMD), California Air Resources Board (CARB), and Office of Environmental Health Hazard Assessment (OEHHA). In general, the Protocol follows the methodology for preparing Tier 1 risk assessments described in *The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments* (OEHHA, 2003), *Supplemental Guidelines for Preparing Risk Assessments for the Air Toxics "Hot Spots" Information and Assessment Act (AB2588)* (SCAQMD, 2005a), *Health Risk Assessment Guidance for Analyzing Cancer Risks from Mobile Source Diesel Emissions* (SCAQMD, 2002), and *ARB Health Risk Assessment Guidance for Rail Yard and Intermodal Facilities* (CARB, 2006a). Prior to development of the HRA, a project-specific Protocol was prepared based on methods in the above-cited documents and reviewed by the SCAQMD prior to implementation (POLA, 2008).

The HRA process requires the completion of four general steps to estimate health impact results: (1) quantify Project-generated emissions; (2) identify ground-level receptor locations that may be affected by the emissions (including both a regular grid of receptors and any additional sensitive receptor locations such as schools, hospitals, convalescent homes, and/or daycare centers); (3) perform dispersion modeling analyses to estimate ambient TAC concentrations at each receptor location; and (4) use established methods to

estimate potential health effects at each receptor location. The following sections describe in detail the methods used to complete each step of the HRA.

2.0 Development of Emission Scenarios Used in the HRA

2.1 Emission Sources

The following emission sources were included in the health risk assessment:

Locomotives break-down and build activities and idling within the SCIG facility, and off-site train travel between the SCIG facility and the Alameda Corridor, as far north as the intersection of the Alameda Corridor with CA-91. The northern boundary of the emission source domain for off-site train transit was set at CA-91 to be consistent with the truck source domain, described below.

Locomotive emissions in the Baseline only included minor switching activity associated with locomotives calling on certain Baseline tenant facilities. Locomotives were otherwise not included in the Baseline as the SCIG facility did not exist in the Baseline year.

Trucks traveling along designated truck routes to and from the SCIG facility, including the following major roadway segments:

- On-site driving and idling
- Pacific Coast Highway (PCH) from the facility to the Terminal Island (TI) Freeway Interchange
- TI Freeway to East I Street and Anaheim Street
- Anaheim Street to Alameda Street
- Alameda Street to Harry Bridges Boulevard
- Harry Bridges Boulevard to West Basin Terminals
- Anaheim Street to the I-710
- I-710 to Port of Long Beach Terminals
- TI Freeway to Terminal Island Terminals

On-site truck emissions include trucks waiting at the SCIG facility in-gate, driving from the in-gate to the on-site loading tracks, and driving and idling on-site to drop off and pick up their loads.

Refueling trucks visiting the SCIG facility were modeled as exiting the facility and using the PCH to the I-110 and I-710 freeways, and then north on these freeways to the interchanges with the I-405.

Relocated tenants drayage trucks conducting trips between the Port terminals and their facilities exit the facilities at either the TI Freeway or the Sepulveda Boulevard driveway at the north end of the SCIG facility. Trucks exiting at the Sepulveda Boulevard driveway primarily travel west on Sepulveda to Alameda Street and south on Alameda Street to various Port of Los Angeles and Port of Long Beach terminal destinations. Trucks exiting at the TI Freeway driveway travel south on the TI Freeway to various Port of Los Angeles and Port of Long Beach terminals. In addition to the relocated tenant drayage trucks traveling to and from the Ports, vendor trucks visit certain relocated tenant

sites. These trucks travel on Sepulveda Boulevard to Alameda Street or the I-110 and then north to destinations throughout the South Coast area (for relocated tenant sites at the Sepulveda driveway), or north on the TI Freeway to the PCH and east or west to the I-110 or I-710 and then north to destinations throughout the South Coast area (for relocated tenants sites at the TI Freeway driveways). Vendor trucks visiting the relocated tenant sites were tracked as far as the intersection of Alameda Street, the I-110 or the I-710 with the I-405 freeway. Beyond the intersection of these roadways with the I-405, the destinations of these trucks were unknown and a sensitivity analysis indicated that their contributions to the total risk from all Project sources at the maximum occupational and residential receptors were minimal.

In the analysis for the Reduced Project Alternative, container cargo not handled by the SCIG facility continued to be drayed to the Hobart Yard in downtown Los Angeles. Truck routes to the Hobart Yard are described in the Transportation analysis in Section 3.10. In the No Project Alternative, all drayage trucks are modeled as traveling to the Hobart Yard following the truck routes described in Section 3.10.

In the Baseline analysis, drayage trucks traveling between the Baseline tenant sites and the Port terminals were modeled. Trucks primarily exit at the PCH driveways and Sepulveda driveways, and use a variety of major roadways to travel to and from the site and the Port terminals, including:

- On-site driving and idling
- PCH from the site to the Terminal Island (TI) Freeway Interchange
- TI Freeway to Terminal Island
- PCH from the site to the I-710
- I-710 to Port of Long Beach Terminals
- PCH to Alameda Street
- Alameda Street to Harry Bridges Boulevard
- Harry Bridges Boulevard to West Basin Terminals
- Sepulveda Boulevard to the TI Freeway
- TI Freeway to Terminal Island Terminals
- TI Freeway to PCH
- PCH to the I-710
- I-710 to Port of Long Beach Terminals

The Baseline vendor trucks calling on tenant facilities were modeled as traveling east and west on the PCH to the I-710 and I-110 respectively, and north on these freeways to the interchanges with the I-405.

A sensitivity analysis was performed to examine potential impacts from trucks traveling on roadways farther from the facility than the links described above. The sensitivity analysis showed that each roadway segment at these distances contributes no greater than 0.2 percent to the total risks from all Project sources at the maximum residential and occupational receptors. Therefore, these roadway segments were not included in the emission source domain for truck travel.

Rail Yard and Cargo-Handling Equipment at the SCIG facility and tenant sites, including yard tractors, rail wheel change-out machines, forklifts, top picks and other equipment types.

These equipment types were also modeled in the Baseline for tenant facilities that make use of these equipment types.

Light-Duty Gasoline Vehicles, including service trucks on-site and employee commute vehicles for the SCIG facility and relocated tenant sites.

Construction Equipment, including off-road diesel equipment, on-road delivery and haul trucks, rail delivery, and general cargo ship delivery. In accordance with SCAQMD guidance, only onsite construction emissions were included in the HRA.

Construction equipment was not modeled for the Baseline and No Project Alternative because those scenarios would have no construction activities.

2.2 TAC Emission Calculation Approach

The determination of health risks in this HRA required the calculation of 70-year average, 40-year average, maximum annual, and maximum 1-hour emission rates. The 70-year-average emission rates were used to determine individual lifetime cancer risks for residents, recreational receptors, and sensitive receptors. Cancer risks for workers were calculated based on TAC emissions calculated over a 40-year period, and cancer risks to student receptors were evaluated based on peak annual emissions evaluated over a 6 year period.

Maximum annual emission rates during project construction and operation were conservatively used to determine chronic non-cancer effects, given that the chronic exposure period for non-cancer effects is assumed to be approximately 12% of a 70-year lifetime, or 8 or more years (OEHHA, 2002). Maximum 1-hour emission rates were used to determine the acute hazard index because the acute exposure period is 1 hour for most TACs.

The extended period of analysis (up to 70 years for cancer risk) required predictions of the future operational characteristics of the proposed emission sources. Two of the more important factors that would affect future emissions from Project sources and that were integrated into the analysis are:

- Reductions in emissions due to (a) the incidental phase-in of cleaner vehicles or equipment due to normal fleet turnover; (b) the future phase-in of cleaner fuels as required by existing regulations or agreements; and (c) the future phase-in of cleaner engines as required by existing regulations or agreements
- Increased vehicle and equipment activity levels due to anticipated increases in container throughput.

Based on the future trends in these factors, this HRA developed annualized 70-year TAC emission rates for each emission source category by using the methods described in Sections 2.3, 2.4, and 2.5. The approaches for estimating maximum annual and 1-hour emissions are described in Sections 2.6 and 2.7, respectively.

The year-by-year particulate matter (PM) and volatile organic compound (VOC) emission calculations by source are attached to this Appendix.

2.3 Emission Factor Trends

The following methods were used in this HRA to develop the 70-year trends in annual emission factors for unmitigated emissions.

1. **Trucks**. Due to the promulgation of future USEPA and CARB emission standards, the San Pedro Bay Ports Clean Truck Program (CTP), coupled with normal truck fleet turnover, unmitigated emission factors for trucks will decrease with time. The emission factors also assume the use of CARB ULSD (maximum 15ppm sulfur) starting September 1, 2006, in accordance with existing California Diesel Fuel Regulations (CARB, 2004b). Composite truck emission factors were developed using the EMFAC2007 emission factor model (CARB, 2006b). Emission factors were calculated for several analysis years between 2005 and 2046. Actual inventory data for on-road trucks that serviced the San Pedro Bay ports container terminals in the year 2005 were used to develop the truck fleet age distribution used in EMFAC2007 for the Baseline analysis (Starcrest, 2007). Inventory projections developed for the San Pedro Bay Port CAAP were used to develop fleet age distributions for future years. This approach accounts for a small percentage of older trucks being retired each year and replaced with newer, cleaner trucks through normal fleet turnover, and the accelerated turnover effects of the Ports' CTP and the CARB drayage truck rule and in-use truck and bus rule. Emission factors for years between the calculated years were estimated by interpolation. Given a lack of information on how emission factors would change beyond the year 2046, emission factors after the year 2046 were held constant at 2046 levels.
2. **Locomotives.** Locomotive future-year emission factors were developed considering the 1998 and 2005 CARB MOUs and the fleet average requirements and forecasting developed as part of the MOU analyses. The 2005 CARB railyard MOU was used to determine 2016 opening year locomotive fleet mixes, which require a Tier 2 linehaul locomotive average standard. Forecasts of the linehaul locomotive fleet mix from the 2005 CARB Railyard MOU were used as a basis for projecting the fleet mix to future years until 2019, after which the projections were matched with those of the USEPA nationwide locomotive emission standard implementation schedule for future years beyond 2019 (USEPA, 1998). In general, locomotive emission factors decline in future years as older locomotives gradually are replaced with newer locomotives meeting the USEPA tiered emission standards. The emission factors also assume the use of ULSD with 15 ppm sulfur, which is nationally required for locomotives by the opening year of the SCIG facility. Emission factors after the year 2046 were held constant at 2046 levels.
3. **Rail Yard and Cargo-Handling Equipment.** Emission factors for rail yard equipment, including the emergency generator and TRU's, and cargo-handling equipment were calculated to year 2046 using methodology from the CARB OFFROAD2007 Emissions Model (CARB, 2007). For cargo-handling equipment, this methodology accounts for the tiered implementation of future engine standards from existing CARB and USEPA rules, coupled with an assumed equipment-fleet turnover rate. To estimate future year emission factors for tenant cargo-handling equipment, the OFFROAD model was run using the actual Baseline equipment population at the existing tenant sites in 2005. With each future analysis year, the equipment population was allowed to age in the OFFROAD model until it would reach its useful lifetime, at which point it would be assumed to be replaced by new equipment meeting current emission standards. The new replacement equipment would then age in a similar manner. As a result, emission factors for cargo-handling equipment tend to gradually increase with time as equipment ages, followed by a sharp reduction in emission factors upon replacement with new equipment. The emission factors also assume the use of CARB ULSD fuel (maximum 15ppm sulfur), for the purposes of the risk assessment), in accordance with California Diesel Fuel

Regulations (CARB, 2004b). Emission factors after the year 2046 were held constant at 2046 levels. The emissions for off-road equipment have been adjusted from the OFFROAD2007 output to account for a 33% reduction attributable to overestimation of load factors, which CARB has indicated to be appropriate (CARB, 2010). For the emergency generator, the generator was assumed to meet EPA Tier 4 emissions levels for all analysis years. The TRU's were modeled using the OFFROAD2007 model and considering the CARB air toxics control measure (ACTM) for TRU's.

4. **Light-Duty Gasoline Vehicles**. Emissions factors for light-duty gasoline vehicles, including light-duty gasoline service trucks operating at the SCIG facility and light-duty gasoline automobiles used for employee commutes at the SCIG facility and tenant facilities were developed using the EMFAC2007 model. Vehicles were assumed to meet the default South Coast Air Basin fleet mixes by vehicle type, and the EMFAC2007 model was used to calculate emission factors for each analysis year, considering normal fleet turnover.
5. **Construction Equipment**. Emissions from diesel-powered construction equipment were calculated using emission factors derived from OFFROAD2007. Using South Coast Air Basin fleet information, the OFFROAD model was run for each of the construction years from 2013 through 2015. Emission factors were calculated based on each type of equipment and horsepower rating of the equipment. The emissions for off-road equipment have been adjusted from the OFFROAD2007 output to account for a 33% reduction attributable to overestimation of load factors, which CARB has indicated to be appropriate (CARB, 2010).

2.4 Activity Level Trends

The second parameter needed to develop source category emission rates is the annual source activity levels expected each year over the 70-year period. Examples of activity levels include the container throughput at the SCIG facility, the subsequent required number of train and truck trips, on-site equipment usage, truck vehicle miles traveled (VMT), and truck travel speeds.

For the Baseline scenario, tenant activity levels in 2005 were held constant over the entire 70-year period.

2.5 70-Year and 40-Year Average Emission Rates

For diesel internal combustion engines (ICEs), which represent the majority of emission sources associated with SCIG, DPM is the only pollutant needed for the cancer risk analysis (which uses 70-year-average emission rates for residential, recreational, and sensitive receptor risks and the 40-year average emission rates for worker risk). The cancer slope factor established by OEHHA for the assessment of DPM cancer risk includes consideration of the individual toxic species that could be adsorbed onto DPM particles.

For all other source types (tire and brake wear and alternative-fueled engines) speciating combustion emissions into individual TAC components was necessary. Speciation profiles based on those developed by the CARB were used in this study (CARB, 2011). Table C3-2-1 presents the speciation profiles that were used to convert total organic gas (TOG) and particulate matter (PM) combustion emissions into individual TAC emissions.

Table C3-2-1. Speciation Profiles for Diesel and Alternative Fuel Combustion Sources.[a]

Pollutant	CAS Number	Weight Percent					
		PM_{10} Profile Diesel No. 425[b]	PM_{10} Profile LNG No. 123[b]	PM_{10} Profile Propane No. 123[b]	TOG Profile Diesel[c,d,e] No. 818	TOG Profile LNG[c,d,f] No. 719	TOG Profile Propane[c,d,f] No. 719
Acetaldehyde	75070	--	--	--	7.35	0.03	0.03
Acetone	67641	--	--	--	7.51	0.0	0.0
Acetylene	74862	--	--	--	4.25	0.32	0.32
Alkene Ketone		--	--	--	1.75	0.0	0.0
Benzaldehyde	100527	--	--	--	0.70	0.0	0.0
Benzene	71432	--	--	--	2.00	0.11	0.11
Bromine	7726956	--	0.05	0.05	--	0.0	0.0
1,3-Butadiene	106990	--	--	--	0.19	--	--
N-Butane	106978	--	--	--	0.10	1.00	1.00
1-Butene	106989	--	--	--	0.67	0.01	0.01
cis-2-Butene	590181	--	--	--	0.094	0.02	0.02
trans-2-Butene	624646	--	--	--	0.20	0.13	0.13
Butyraldehyde	123728	--	--	--	1.87	0.02	0.02
C10 Aromatics		--	--	--	0.079	0.0	0.0
C10 Dialkyl benzenes		--	--	--	--	0.01	0.01
C10 Internal alkenes		--	--	--	--	0.02	0.02
C5 Aldehyde		--	--	--	0.11	--	--
C6 Aldehydes		--	--	--	3.80	--	--
C9 Aromatics		--	--	--	0.50	0.01	0.01
C9 Internal alkenes		--	--	--	--	0.04	0.04
Calcium	7440702	--	0.55	0.55	--	--	--
Carbon Elemental	7440440	--	20.0	20.0	--	--	--
Chlorine	7782505	--	7.0	7.0	--	--	--
Chromium	7440473	--	0.05	0.05	--	--	--
Cobalt	7440484	--	0.05	0.05	--	--	--
Copper	7440508	--	0.05	0.05	--	--	--
Cyclohexane	110827	--	--	--	0.026	0.01	0.01
Cyclohexanone	108941	--	--	--	0.11	--	--
Cyclopentane	287923	--	--	--	0.012	0.02	0.02
N-Decane	124185	--	--	--	0.53	0.01	0.01
1,2-Diethylbenzene (Ortho)	135013	--	--	--	0.086	--	--
2,3-Dimethyl-1-butene	563780	--	--	--	0.028	--	--
3,3-Dimethyl-1-butene	558372	--	--	--	2.82	--	--
2,2-Dimethylbutane	75832	--	--	--	0.061	0.01	0.01
2,3-Dimethylhexane	584941	--	--	--	0.011	--	--
2,4-Dimethylhexane	589435	--	--	--	0.036	--	--
2,3-Dimethylpentane	565593	--	--	--	0.073	--	--
2,4-Dimethylpentane	108087	--	--	--	0.019	0.01	0.01
DPM	9901	100.00	--	--	--	--	--
Ethane	74840	--	--	--	0.57	13.99	13.99
Ethanol	64175	--	--	--	0.009	--	--
Ethylbenzene	100414	--	--	--	0.31	0.01	0.01
Ethylene	74851	--	--	--	14.38	0.63	0.63
Ethylhexane		--	--	--	0.061	--	--
Formaldehyde	50000	--	--	--	14.71	0.81	0.81
N-Heptane	142825	--	--	--	0.068	0.02	0.02
1-Heptene	592767	--	--	--	--	0.01	0.01
N-Hexane	110543	--	--	--	0.16	0.02	0.02
Hexavalent chromium[g]	18540299	--	0.0025	0.0025	--	--	--
Indan	496117	--	--	--	0.19	--	--
Iron	7439896	--	0.05	0.05	--	--	--
Isobutane	75285	--	--	--	1.22	0.43	0.43

Pollutant	CAS Number	Weight Percent					
		PM_{10} Profile Diesel No. 425[b]	PM_{10} Profile LNG No. 123[b]	PM_{10} Profile Propane No. 123[b]	TOG Profile Diesel[c,d,e] No. 818	TOG Profile LNG[c,d,f] No. 719	TOG Profile Propane[c,d,f] No. 719
Isobutylene	115117	--	--	--	0.92	0.02	0.02
Isomers Of Butene		--	--	--	--	0.26	0.26
Isomers Of Butylbenzene		--	--	--	0.13	--	--
Isomers Of Decane		--	--	--	--	0.02	0.02
Isomers Of Diethylbenzene		--	--	--	0.14	--	--
Isomers Of Heptane		--	--	--	--	0.04	0.04
Isomers Of Hexane		--	--	--	--	0.02	0.02
Isomers Of Nonane		--	--	--	--	0.01	0.01
Isomers Of Octane		--	--	--	--	0.02	0.02
Isomers Of Pentane		--	--	--	--	0.13	0.13
Isomers Of Xylene	1330207	--	--	--	--	0.02	0.02
Isopentane	78784	--	--	--	0.60	--	--
Isopropylbenzene (Cumene)	98828	--	--	--	0.015	--	--
Manganese	7439965	--	0.05	0.05	--	--	--
Methane	74828	--	--	--	4.08	76.64	76.64
Methyl Alcohol	67561	--	--	--	0.030	--	--
Methyl Ethyl Ketone (MEK) (2-Butanone)	78933	--	--	--	1.48	--	--
Methyl N-Butyl Ketone	591786	--	--	--	0.90	--	--
2-Methyl-1-Pentene	763291	--	--	--	--	0.02	0.02
2-Methyl-2-Butene	513359	--	--	--	--	0.01	0.01
1-Methyl-2-Ethylbenzene	611143	--	--	--	0.14	0.01	0.01
1-Methyl-3-Ethylbenzene	620144	--	--	--	0.25	0.01	0.01
Methylcyclohexane	108872	--	--	--	0.068	0.02	0.02
Methylcyclopentane	96377	--	--	--	0.15	0.04	0.04
2-Methylheptane	592278	--	--	--	0.057	--	--
3-Methylheptane	589811	--	--	--	--	0.02	0.02
2-Methylhexane	591764	--	--	--	0.12	--	--
3-Methylhexane	589344	--	--	--	0.35	0.01	0.01
2-Methylpentane	107835	--	--	--	0.39	--	--
3-Methylpentane	96140	--	--	--	0.12	0.02	0.02
(1-Methylpropyl)Benzene	135988	--	--	--	0.051	--	--
(2-Methylpropyl)Benzene	538932	--	--	--	0.13	--	--
B-Methylstyrene	637503	--	--	--	0.047	0.0	0.0
Naphthalene	91203	--	--	--	0.085	--	--
Nickel	7440020	--	0.05	0.05	--	--	--
Nitrates	14797558	--	0.55	0.55	--	--	--
N-Nonane	111842	--	--	--	0.23	0.01	0.01
1-Nonene	124118	--	--	--	--	0.01	0.01
N-Octane	111659	--	--	--	0.14	0.02	0.02
1-Octene	111660	--	--	--	--	0.01	0.01
Other		--	25.95	25.95	--	--	--
N-Pentane	109660	--	--	--	0.18	0.13	0.13
1-Pentene	109671	--	--	--	0.32	0.01	0.01
Cis-2-Pentene	627203	--	--	--	0.030	--	--
Trans-2-Pentene	646048	--	--	--	0.040	0.01	0.01
Potassium	7440097	--	0.55	0.55	--	--	--
1,2-Propadiene	463490	--	--	--	0.47	--	--

Pollutant	CAS Number	Weight Percent					
		PM_{10} Profile Diesel No. 425[b]	PM_{10} Profile LNG No. 123[b]	PM_{10} Profile Propane No. 123[b]	TOG Profile Diesel[c,d,e] No. 818	TOG Profile LNG[c,d,f] No. 719	TOG Profile Propane[c,d,f] No. 719
Propane	74986	--	--	--	0.19	2.91	2.91
Propionaldehyde	123386	--	--	--	0.97	--	--
N-Propylbenzene	103651	--	--	--	0.12	--	--
Propylene	115071	--	--	--	2.60	1.69	1.69
Styrene	100425	--	--	--	0.058	--	--
Sulfates	9960	--	45.0	45.0	--	--	--
T-Butylbenzene	98066	--	--	--	0.006	--	--
Toluene	108883	--	--	--	1.47	0.04	0.04
1,2,3-Trimethylbenzene	526738	--	--	--	0.12	0.01	0.01
1,2,4-Trimethylbenzene	95636	--	--	--	0.53	0.01	0.01
1,3,5-Trimethylbenzene	108678	--	--	--	0.19	0.02	0.02
2,2,4-Trimethylpentane	540841	--	--	--	0.30	--	--
2,3,4-Trimethylpentane	565753	--	--	--	0.015	--	--
N-Undecane	1120214	--	--	--	0.26	--	--
Unidentified		--	--	--	13.86	--	--
M-Xylene	108383	--	--	--	0.61	0.01	0.01
O-Xylene	95476	--	--	--	0.34	0.01	0.01
P-Xylene	106423	--	--	--	0.10	--	--
Zinc	7440666	--	0.05	0.05	--	--	--
Applicable Emissions Sources:		Locomotives, switchers, cargo handling equipment, emergency generator, trucks – diesel fuel.	Locomotives switchers, cargo handling equipment, emergency generator, trucks – diesel fuel.	Locomotives, switchers, cargo handling equipment, emergency generator, trucks – diesel fuel.	Cargo handling equipment and hostlers	Cargo handling equipment and hostlers	Cargo handling equipment and hostlers

Notes:

a Other speciation profiles used in the HRA but not shown in this table are PM10 Profile No. 472 (Truck Tire Wear) and PM10 Profile No. 473 (Truck Brake Wear).

b CARB 2008

c CARB 2011

d TOG – total organic gas.

e For Profile No. 818, TOG is 87.85 percent VOC.

f For Profile No. 719, TOG is 9.14 percent VOC.

g Hexavalent chromium is assumed to be 5 percent of total chromium, in accordance with the CARB AB2588 Technical Support Document (1989), page 57.

Sources:

California Environmental Protection Agency Air Resources Board (CARB). 2008. Speciation Profiles Used in ARB Modeling.

California Environmental Protection Agency Air Resources Board (CARB). 2011. Speciation Profiles Used in ARB Modeling.

For each emission source category, PM and TOG emissions were calculated for specific analysis years (2005 for Baseline, 2013-2015 for construction, and 2016, 2023, 2035, and 2046 for each Project alternative) by multiplying the source activity level by the emission factors for that particular year. The resulting annual emission rates for each pollutant were then averaged to produce the 70-year average PM and TOG emission rates, to be used for the residential, recreational, and sensitive receptor risk calculations, and the 40-year average PM and TOG emission rates, to be used for the worker receptor risk calculations. Maximum annual emissions, described in Section 2.6 below, were used for the student risk calculations. For the 70- and 40-year average emissions, it was assumed that emissions change linearly between analysis years and remain at the 2046 emission rate until the end of the period, where the 70-year period runs from 2013 through 2082 and the 40-year period runs from 2013 through 2052. The only exception is that the Baseline 70-year average emission rate and 40-year average emission rate are simply the 2005 emission rate. Tables C3-2-2 through C3-2-7 present the 70-year average, 40-year average, maximum annual, and maximum hourly TAC emission rates used in this HRA for the Baseline, Unmitigated Proposed Project, Mitigated Proposed Project, No Project Alternative, Unmitigated Reduced Project Alternative, and Mitigated Reduced Project Alternative, respectively.

For the information-only floating Baseline cancer risk analysis, the 70-year averaging period is 2005-2074. The 70-year average TAC emission rates for the floating Baseline cancer risk analysis are presented in Table C3-2-8.

Table C3-2-2. Toxic Air Contaminant Emissions by Source - CEQA Baseline.

Emission Source[a]	70-Year-Average Emissions (lb/yr)[b,c]	40-Year-Average Emissions (lb/yr)[b,c]	Maximum Annual Emissions (lb/yr)[c,d]	Maximum 1-Hour Emissions (lb/hr)[e]	
	DPM	DPM	DPM	Acetaldehyde	Formaldehyde
Hobart Trucks	3.6E+04	3.6E+04	3.6E+04	5.8E-01	1.2E+00
Tenant CHE	7.2E+03	7.2E+03	7.2E+03	2.1E-01	4.4E-01
Tenant Offsite Gasoline Vehicles	0.0E+00	0.0E+00	0.0E+00	2.6E-03	1.5E-02
Tenant Offsite Trucks	1.2E+04	1.2E+04	1.2E+04	5.3E-01	1.1E+00
Tenant Onsite Gasoline Vehicles	0.0E+00	0.0E+00	0.0E+00	1.9E-04	1.1E-03
Tenant Onsite Locomotives	1.4E+01	1.4E+01	1.4E+01	1.5E-03	3.0E-03
Tenant Onsite Trucks	4.4E+03	4.4E+03	4.4E+03	4.9E-01	9.9E-01
Total - All Sources	**6.0E+04**	**6.0E+04**	**6.0E+04**	**1.8E+00**	**3.7E+00**

Notes:

a This HRA evaluated emissions of all toxic air contaminants (TACs) listed in Table C3-5-1. However, for brevity, only those TACs contributing at least 2 percent to the estimated health endpoint results are presented in this table.

b Seventy-year-average emissions were used to determine individual residential, recreational, and sensitive receptor lifetime cancer risk. Forty-year-average emissions were used to determine individual worker lifetime cancer risk.

c Maximum annual emissions were used to determine noncancer chronic hazard indexes and were used to determine individual student lifetime cancer risk, as a conservative estimate of 6-year-average emissions.

d For 70-year average, 40-year average, and maximum annual emissions, only DPM emissions were modeled in the HRA for diesel equipment.

e Maximum 1-hour emissions were used to determine noncancer acute hazard indices.

Table C3-2-3. Toxic Air Contaminant Emissions by Source - Unmitigated Proposed Project.

Emission Sourcea	70-Year-Average Emissions (lb/yr)b,c	40-Year-Average Emissions (lb/yr)b,c	Maximum Annual Emissions (lb/yr)c,d			Maximum 1-Hour Emissions (lb/hr)e	
	DPM	DPM	Chlorine	DPM	Formaldehyde	Acetaldehyde	Formaldehyde
Emergency Generator	7.8E+00	7.6E+00	0.0E+00	8.2E+00	8.3E+00	2.1E-02	4.2E-02
Hostler	0.0E+00	0.0E+00	1.6E+01	0.0E+00	5.1E+02	2.4E-03	6.6E-02
Onsite Refueling Trucks	9.2E-02	9.9E-02	1.7E-04	1.5E-01	6.4E-01	3.7E-05	7.5E-05
SCIG CHE/TRU	5.1E+00	4.8E+00	0.0E+00	7.6E+00	6.8E+00	6.6E-03	1.3E-02
SCIG Construction	1.7E+02	3.0E+02	1.9E-02	7.7E+03	4.4E+03	1.2E+00	2.4E+00
SCIG Offsite Gasoline Vehicles	0.0E+00	0.0E+00	3.9E+00	0.0E+00	1.4E+00	2.9E-05	1.6E-04
SCIG Offsite Locomotives	3.6E+02	4.8E+02	0.0E+00	2.5E+03	1.0E+03	6.0E-02	1.2E-01
SCIG Offsite Trucks	2.0E+03	2.0E+03	7.1E+00	2.3E+03	1.5E+03	9.7E-02	1.9E-01
SCIG Onsite Gasoline Vehicles	0.0E+00	0.0E+00	4.4E-01	0.0E+00	2.1E+00	4.3E-05	2.4E-04
SCIG Onsite Locomotives	2.1E+02	2.7E+02	0.0E+00	6.3E+02	1.8E+02	4.1E-02	8.2E-02
SCIG Onsite Trucks	9.2E+02	8.8E+02	2.7E+00	9.9E+02	2.3E+03	1.5E-01	3.0E-01
Tenant CHE	2.6E+02	3.8E+02	0.0E+00	1.3E+03	7.3E+02	9.6E-02	1.9E-01
Tenant Construction	6.5E+00	1.1E+01	0.0E+00	4.6E+02	2.2E+02	5.8E-02	1.2E-01
Tenant Offsite Gasoline Vehicles	0.0E+00	0.0E+00	9.7E+00	0.0E+00	8.2E+00	4.2E-04	2.4E-03
Tenant Offsite Trucks	5.0E+02	5.2E+02	4.6E+00	2.2E+03	1.6E+03	2.4E-01	4.9E-01
Tenant Onsite Gasoline Vehicles	0.0E+00	0.0E+00	2.8E-01	0.0E+00	2.3E-01	1.3E-05	7.6E-05
Tenant Onsite Locomotives	1.9E+00	1.8E+00	0.0E+00	1.9E+00	1.1E+00	1.4E-04	2.9E-04
Tenant Onsite Trucks	6.6E+01	7.9E+01	4.7E-02	1.2E+02	5.6E+02	8.9E-02	1.8E-01
Three Rivers Underpass	1.3E+01	1.3E+01	1.8E-02	1.9E+01	6.7E+01	9.4E-03	1.9E-02
Total - All Sources	**4.6E+03**	**4.9E+03**	**4.5E+01**	**1.8E+04**	**1.3E+04**	**2.1E+00**	**4.2E+00**

Notes:

a This HRA evaluated emissions of all toxic air contaminants (TACs) listed in Table C3-5-1. However, for brevity, only those TACs contributing at least 2 percent to the estimated health endpoint results are presented in this table.

b Seventy-year-average emissions were used to determine individual residential, recreational, and sensitive receptor lifetime cancer risk. Forty-year-average emissions were used to determine individual worker lifetime cancer risk.

c Maximum annual emissions were used to determine noncancer chronic hazard indexes and were used to determine individual student lifetime cancer risk, as a conservative estimate of 6-year-average emissions.

d For maximum annual emissions, only nondiesel ICE emissions (i.e., alternative fueled engines, tire wear, and brake wear) are shown for chlorine and formaldehyde. Diesel ICE emissions are modeled only with DPM emissions. For 70-year average and 40-year average emissions, only DPM emissions were modeled in the HRA.

e Maximum 1-hour emissions were used to determine noncancer acute hazard indices.

Table C3-2-4. Toxic Air Contaminant Emissions by Source - Mitigated Proposed Project.

Emission Source[a]	70-Year-Average Emissions (lb/yr)[b,c]	40-Year-Average Emissions (lb/yr)[b,c]	Maximum Annual Emissions (lb/yr)[c,d]			Maximum 1-Hour Emissions (lb/hr)[e]	
	DPM	DPM	Chlorine	DPM	Formaldehyde	Acetaldehyde	Formaldehyde
Emergency Generator	7.8E+00	7.6E+00	0.0E+00	8.2E+00	8.3E+00	2.1E-02	4.2E-02
Hostler	0.0E+00	0.0E+00	1.6E+01	0.0E+00	5.1E+02	2.4E-03	6.6E-02
Onsite Refueling Trucks	9.2E-02	9.9E-02	1.7E-04	1.5E-01	6.4E-01	3.7E-05	7.5E-05
SCIG CHE/TRU	5.1E+00	4.8E+00	0.0E+00	7.6E+00	6.8E+00	6.6E-03	1.3E-02
SCIG Construction	5.7E+01	1.0E+02	1.9E-02	2.5E+03	3.2E+03	9.4E-01	1.9E+00
SCIG Offsite Gasoline Vehicles	0.0E+00	0.0E+00	3.9E+00	0.0E+00	1.4E+00	2.9E-05	1.6E-04
SCIG Offsite Locomotives	3.6E+02	4.8E+02	0.0E+00	2.5E+03	1.0E+03	6.0E-02	1.2E-01
SCIG Offsite Trucks	2.0E+03	2.0E+03	7.1E+00	2.3E+03	1.5E+03	9.7E-02	1.9E-01
SCIG Onsite Gasoline Vehicles	0.0E+00	0.0E+00	4.4E-01	0.0E+00	2.1E+00	4.3E-05	2.4E-04
SCIG Onsite Locomotives	2.1E+02	2.7E+02	0.0E+00	6.3E+02	1.8E+02	4.1E-02	8.2E-02
SCIG Onsite Trucks	9.2E+02	8.8E+02	2.7E+00	9.9E+02	2.3E+03	1.5E-01	3.0E-01
Tenant CHE	2.6E+02	3.8E+02	0.0E+00	1.3E+03	7.3E+02	9.6E-02	1.9E-01
Tenant Construction	4.7E+00	8.1E+00	0.0E+00	3.3E+02	1.8E+02	4.3E-02	8.6E-02
Tenant Offsite Gasoline Vehicles	0.0E+00	0.0E+00	9.7E+00	0.0E+00	8.2E+00	4.2E-04	2.4E-03
Tenant Offsite Trucks	5.0E+02	5.2E+02	4.6E+00	2.2E+03	1.6E+03	2.4E-01	4.9E-01
Tenant Onsite Gasoline Vehicles	0.0E+00	0.0E+00	2.8E-01	0.0E+00	2.3E-01	1.3E-05	7.6E-05
Tenant Onsite Locomotives	1.9E+00	1.8E+00	0.0E+00	1.9E+00	1.1E+00	1.4E-04	2.9E-04
Tenant Onsite Trucks	6.6E+01	7.9E+01	4.7E-02	1.2E+02	5.6E+02	8.9E-02	1.8E-01
Three Rivers Underpass	1.3E+01	1.3E+01	1.8E-02	1.9E+01	6.7E+01	9.4E-03	1.9E-02
Total - All Sources	**4.4E+03**	**4.7E+03**	**4.5E+01**	**1.3E+04**	**1.2E+04**	**1.8E+00**	**3.7E+00**

Notes:

a This HRA evaluated emissions of all toxic air contaminants (TACs) listed in Table C3-5-1. However, for brevity, only those TACs contributing at least 2 percent to the estimated health endpoint results are presented in this table.

b Seventy-year-average emissions were used to determine individual residential, recreational, and sensitive receptor lifetime cancer risk. Forty-year-average emissions were used to determine individual worker lifetime cancer risk.

c Maximum annual emissions were used to determine noncancer chronic hazard indexes and were used to determine individual student lifetime cancer risk, as a conservative estimate of 6-year-average emissions.

d For maximum annual emissions, only nondiesel ICE emissions (i.e., alternative fueled engines, tire wear, and brake wear) are shown for chlorine and formaldehyde. Diesel ICE emissions are modeled only with DPM emissions. For 70-year average and 40-year average emissions, only DPM emissions were modeled in the HRA.

e Maximum 1-hour emissions were used to determine noncancer acute hazard indices.

Table C3-2-5. Toxic Air Contaminant Emissions by Source - No Project Alternative.

Emission Sourcea	70-Year-Average Emissions (lb/yr)b,c	40-Year-Average Emissions (lb/yr)b,c	Maximum Annual Emissions (lb/yr)c,d				Maximum 1-Hour Emissions (lb/hr)e	
	DPM	DPM	Chlorine	DPM	Manganese	Nickel	Acetaldehyde	Formaldehyde
Hobart Trucks	8.5E+03	8.2E+03	2.6E+01	9.2E+03	4.1E+00	1.6E+00	3.0E-01	6.1E-01
Tenant CHE	4.6E+02	6.5E+02	7.7E+01	2.1E+03	5.5E-01	5.5E-01	6.9E-02	1.7E-01
Tenant Offsite Gasoline Vehicles	0.0E+00	0.0E+00	1.5E+01	0.0E+00	5.6E-01	2.8E-01	8.7E-04	4.9E-03
Tenant Offsite Trucks	1.4E+03	1.4E+03	5.2E+00	2.3E+03	8.0E-01	3.2E-01	2.7E-01	5.5E-01
Tenant Onsite Gasoline Vehicles	0.0E+00	0.0E+00	3.9E+00	0.0E+00	3.5E-02	3.0E-02	6.5E-05	3.7E-04
Tenant Onsite Locomotives	1.5E+01	1.5E+01	0.0E+00	1.5E+01	0.0E+00	0.0E+00	1.7E-03	3.3E-03
Tenant Onsite Trucks	1.7E+02	1.9E+02	3.7E-01	6.5E+02	5.7E-02	2.3E-02	2.4E-01	4.8E-01
Total - All Sources	**1.1E+04**	**1.0E+04**	**1.3E+02**	**1.4E+04**	**6.1E+00**	**2.8E+00**	**8.9E-01**	**1.8E+00**

Notes:

a This HRA evaluated emissions of all toxic air contaminants (TACs) listed in Table C3-5-1. However, for brevity, only those TACs contributing at least 2 percent to the estimated health endpoint results are presented in this table.

b Seventy-year-average emissions were used to determine individual residential, recreational, and sensitive receptor lifetime cancer risk. Forty-year-average emissions were used to determine individual worker lifetime cancer risk.

c Maximum annual emissions were used to determine noncancer chronic hazard indexes and were used to determine individual student lifetime cancer risk, as a conservative estimate of 6-year-average emissions.

d For maximum annual emissions, only nondiesel ICE emissions (i.e., alternative fueled engines, tire wear, and brake wear) are shown for chlorine, manganese, and nickel. Diesel ICE emissions are modeled only with DPM emissions. For 70-year average and 40-year average emissions, only DPM emissions were modeled in the HRA.

e Maximum 1-hour emissions were used to determine noncancer acute hazard indices.

Table C3-2-6. Toxic Air Contaminant Emissions by Source - Unmitigated Reduced Project Alternative.

Emission Source[a]	70-Year-Average Emissions (lb/yr)[b,c]	40-Year-Average Emissions (lb/yr)[b,c]	Maximum Annual Emissions (lb/yr)[c,d]			Maximum 1-Hour Emissions (lb/hr)[e]	
	DPM	DPM	Chlorine	DPM	Formaldehyde	Acetaldehyde	Formaldehyde
Emergency Generator	7.8E+00	7.6E+00	0.0E+00	8.2E+00	8.3E+00	2.1E-02	4.2E-02
Hostler	0.0E+00	0.0E+00	1.1E+01	0.0E+00	3.6E+02	1.7E-03	4.6E-02
Onsite Refueling Trucks	7.1E-02	7.8E-02	1.7E-04	1.5E-01	6.4E-01	3.7E-05	7.5E-05
SCIG CHE/TRU	5.1E+00	4.8E+00	0.0E+00	7.6E+00	6.8E+00	6.6E-03	1.3E-02
SCIG Construction	1.7E+02	3.0E+02	1.9E-02	7.7E+03	4.4E+03	1.2E+00	2.4E+00
SCIG Offsite Gasoline Vehicles	0.0E+00	0.0E+00	3.8E+00	0.0E+00	1.1E+00	2.3E-05	1.3E-04
SCIG Offsite Locomotives	2.9E+02	3.9E+02	0.0E+00	1.9E+03	7.8E+02	4.5E-02	9.1E-02
SCIG Offsite Trucks	1.8E+03	2.0E+03	1.2E+01	4.2E+03	2.5E+03	1.6E-01	3.3E-01
SCIG Onsite Gasoline Vehicles	0.0E+00	0.0E+00	2.1E-01	0.0E+00	2.1E+00	4.2E-05	2.4E-04
SCIG Onsite Locomotives	1.7E+02	2.2E+02	0.0E+00	6.3E+02	1.8E+02	3.1E-02	6.2E-02
SCIG Onsite Trucks	6.2E+02	6.1E+02	2.7E+00	9.9E+02	2.3E+03	1.5E-01	3.0E-01
Tenant CHE	2.6E+02	3.8E+02	0.0E+00	1.3E+03	7.3E+02	9.6E-02	1.9E-01
Tenant Construction	6.5E+00	1.1E+01	0.0E+00	4.6E+02	2.2E+02	5.8E-02	1.2E-01
Tenant Offsite Gasoline Vehicles	0.0E+00	0.0E+00	9.7E+00	0.0E+00	8.2E+00	4.2E-04	2.4E-03
Tenant Offsite Trucks	5.0E+02	5.2E+02	4.6E+00	2.2E+03	1.6E+03	2.4E-01	4.9E-01
Tenant Onsite Gasoline Vehicles	0.0E+00	0.0E+00	2.8E-01	0.0E+00	2.3E-01	1.3E-05	7.6E-05
Tenant Onsite Locomotives	1.9E+00	1.8E+00	0.0E+00	1.9E+00	1.1E+00	1.4E-04	2.9E-04
Tenant Onsite Trucks	6.6E+01	7.9E+01	4.7E-02	1.2E+02	5.6E+02	8.9E-02	1.8E-01
Three Rivers Underpass	1.3E+01	1.3E+01	1.8E-02	1.9E+01	6.7E+01	9.4E-03	1.9E-02
Total - All Sources	**3.9E+03**	**4.6E+03**	**4.5E+01**	**1.9E+04**	**1.4E+04**	**2.1E+00**	**4.3E+00**

Notes:

a This HRA evaluated emissions of all toxic air contaminants (TACs) listed in Table C3-5-1. However, for brevity, only those TACs contributing at least 2 percent to the estimated health endpoint results are presented in this table.

b Seventy-year-average emissions were used to determine individual residential, recreational, and sensitive receptor lifetime cancer risk. Forty-year-average emissions were used to determine individual worker lifetime cancer risk.

c Maximum annual emissions were used to determine noncancer chronic hazard indexes and were used to determine individual student lifetime cancer risk, as a conservative estimate of 6-year-average emissions.

d For maximum annual emissions, only nondiesel ICE emissions (i.e., alternative fueled engines, tire wear, and brake wear) are shown for chlorine and formaldehyde. Diesel ICE emissions are modeled only with DPM emissions. For 70-year average and 40-year average emissions, only DPM emissions were modeled in the HRA.

e Maximum 1-hour emissions were used to determine noncancer acute hazard indices.

Table C3-2-7. Toxic Air Contaminant Emissions by Source - Mitigated Reduced Project Alternative.

Emission Source[a]	70-Year-Average Emissions (lb/yr)[b,c]	40-Year-Average Emissions (lb/yr)[b,c]	Maximum Annual Emissions (lb/yr)[c,d]			Maximum 1-Hour Emissions (lb/hr)[e]	
	DPM	DPM	Chlorine	DPM	Formaldehyde	Acetaldehyde	Formaldehyde
Emergency Generator	7.8E+00	7.6E+00	0.0E+00	8.2E+00	8.3E+00	2.1E-02	4.2E-02
Hostler	0.0E+00	0.0E+00	1.1E+01	0.0E+00	3.6E+02	1.7E-03	4.6E-02
Onsite Refueling Trucks	7.1E-02	7.8E-02	1.7E-04	1.5E-01	6.4E-01	3.7E-05	7.5E-05
SCIG CHE/TRU	5.1E+00	4.8E+00	0.0E+00	7.6E+00	6.8E+00	6.6E-03	1.3E-02
SCIG Construction	5.7E+01	1.0E+02	1.9E-02	2.5E+03	3.2E+03	9.4E-01	1.9E+00
SCIG Offsite Gasoline Vehicles	0.0E+00	0.0E+00	3.8E+00	0.0E+00	1.1E+00	2.3E-05	1.3E-04
SCIG Offsite Locomotives	2.9E+02	3.9E+02	0.0E+00	1.9E+03	7.8E+02	4.5E-02	9.1E-02
SCIG Offsite Trucks	1.8E+03	2.0E+03	1.2E+01	4.2E+03	2.5E+03	1.6E-01	3.3E-01
SCIG Onsite Gasoline Vehicles	0.0E+00	0.0E+00	2.1E-01	0.0E+00	2.1E+00	4.2E-05	2.4E-04
SCIG Onsite Locomotives	1.7E+02	2.2E+02	0.0E+00	6.3E+02	1.8E+02	3.1E-02	6.2E-02
SCIG Onsite Trucks	6.2E+02	6.1E+02	2.7E+00	9.9E+02	2.3E+03	1.5E-01	3.0E-01
Tenant CHE	2.6E+02	3.8E+02	0.0E+00	1.3E+03	7.3E+02	9.6E-02	1.9E-01
Tenant Construction	4.7E+00	8.1E+00	0.0E+00	3.3E+02	1.8E+02	4.3E-02	8.6E-02
Tenant Offsite Gasoline Vehicles	0.0E+00	0.0E+00	9.7E+00	0.0E+00	8.2E+00	4.2E-04	2.4E-03
Tenant Offsite Trucks	5.0E+02	5.2E+02	4.6E+00	2.2E+03	1.6E+03	2.4E-01	4.9E-01
Tenant Onsite Gasoline Vehicles	0.0E+00	0.0E+00	2.8E-01	0.0E+00	2.3E-01	1.3E-05	7.6E-05
Tenant Onsite Locomotives	1.9E+00	1.8E+00	0.0E+00	1.9E+00	1.1E+00	1.4E-04	2.9E-04
Tenant Onsite Trucks	6.6E+01	7.9E+01	4.7E-02	1.2E+02	5.6E+02	8.9E-02	1.8E-01
Three Rivers Underpass	1.3E+01	1.3E+01	1.8E-02	1.9E+01	6.7E+01	9.4E-03	1.9E-02
Total - All Sources	**3.8E+03**	**4.4E+03**	**4.5E+01**	**1.4E+04**	**1.3E+04**	**1.8E+00**	**3.7E+00**

Notes:

a This HRA evaluated emissions of all toxic air contaminants (TACs) listed in Table C3-5-1. However, for brevity, only those TACs contributing at least 2 percent to the estimated health endpoint results are presented in this table.

b Seventy-year-average emissions were used to determine individual residential, recreational, and sensitive receptor lifetime cancer risk. Forty-year-average emissions were used to determine individual worker lifetime cancer risk.

c Maximum annual emissions were used to determine noncancer chronic hazard indexes and were used to determine individual student lifetime cancer risk, as a conservative estimate of 6-year-average emissions.

d For maximum annual emissions, only nondiesel ICE emissions (i.e., alternative fueled engines, tire wear, and brake wear) are shown for chlorine and formaldehyde. Diesel ICE emissions are modeled only with DPM emissions. For 70-year average and 40-year average emissions, only DPM emissions were modeled in the HRA.

e Maximum 1-hour emissions were used to determine noncancer acute hazard indices.

Table C3-2-8. Toxic Air Contaminant Emissions by Source - Floating Baseline.

Emission Source[a]	70-Year-Average Emissions (lb/yr)[b,c]
	DPM
SCIG Offsite Trucks	6.7E+03
Tenant Offsite Trucks	2.0E+03
Tenant CHE	9.6E+02
Tenant Onsite Trucks	4.5E+02
Tenant Onsite Locomotives	1.4E+01
Tenant Offsite Gasoline Vehicles	0.0E+00
Tenant Onsite Gasoline Vehicles	0.0E+00
Total - All Sources	**1.0E+04**

Notes:
a This cancer risk analysis evaluated emissions of all toxic air contaminants (TACs) listed in Table C3-5-1. However, for brevity, only those TACs contributing at least 2 percent to the estimated health endpoint results are presented in this table.
b Seventy-year-average emissions were used to determine individual residential lifetime cancer risk.
c For 70-year average emissions, only DPM emissions were modeled in the cancer risk analysis for diesel equipment.

2.6 Maximum Year Emission Rates

Similar to the cancer risk analysis, the chronic hazard index developed to assess non-cancer health effects from diesel ICEs requires only DPM emissions data. Analogous to the DPM unit risk factor, the reference exposure level (REL) established by OEHHA for the assessment of DPM for chronic non-cancer effects includes consideration of the individual toxic species that may be adsorbed onto the DPM particles.

For all other source types (tire and brake wear and alternative-fueled engines), it was necessary to speciate combustion emissions into individual TAC components using the TOG and PM speciation profiles shown in Table C3-2-1.

For the Project alternatives, maximum year emissions were selected from the Project construction years (2013-2015) and analysis years (2016, 2023, 2035, and 2046). To ensure the capture of maximum impacts, the highest annual emissions from each type of source were conservatively modeled together in the HRA, even if the emissions would occur in different analysis years for different source groupings. For Baseline conditions, 2005 emissions were used in the HRA.

Tables C3-2-2 through C3-2-7 present the maximum annual TAC emission rates used in this HRA for the Baseline, Unmitigated Proposed Project, Mitigated Proposed Project, No Project Alternative, Unmitigated Reduced Project Alternative, and Mitigated Reduced Project Alternative, respectively.

2.7 Maximum 1-Hour Emission Rates

For the acute hazard index analysis, which is based on maximum 1-hour emission rates, speciating combustion emissions into individual TAC components was necessary for all source types including diesel ICE because OEHHA has not developed an acute REL for

DPM. Therefore, combustion emissions were speciated into individual TAC components using the TOG and PM speciation profiles shown in Table C3-2-1.

For the Project alternatives, maximum 1-hour emissions were calculated assuming theoretical worst-case hourly activity levels for each source category from the construction years (2013-2015) and analysis years (2016, 2023, 2035, and 2046). To ensure that the health effect calculations included an assessment of maximum impacts, the highest 1-hour emissions from each type of source were conservatively modeled together in the HRA, even if the emissions would occur in different analysis years for different source groupings. Baseline emissions represent activity levels for 2005.

For SCIG facility equipment, maximum 1-hour emissions for TRUs and the on-site emergency generator assumed activity for the entire 1-hour duration. For other on-site equipment, maximum 1-hour emissions were derived from the average daily emissions. For SCIG yard hostlers and gasoline vehicles (service trucks), these were assumed to operate for the entire 1-hour duration. Maximum 1-hour emissions for SCIG locomotives were derived from the detailed locomotive movement emissions, which track every step in the entry, breakdown, build and departure of trains. The movements were analyzed to determine the series of movements representing the maximum 1-hour emissions from all movements.

For SCIG trucks maximum 1-hour emissions were derived from the peak daily emissions. The derivation of peak daily emissions for trucks and terminal equipment is discussed in Section 3.2 of the EIR under Impact AQ-3. Peak daily emissions were estimated using a peaking factor representative of port-wide activities in the Port's 2004 Baseline transportation study.

For construction equipment, maximum 1-hour emissions were estimated by first calculating daily emissions from individual construction elements (for example, PCH grade separation, site construction, lead and storage track construction). Maximum daily emissions then were determined by summing emissions from overlapping construction activities as indicated in the proposed construction schedule (Table 2-2) of the EIR. Maximum 1-hour emissions were derived from the peak daily emissions assuming uniform distribution of emissions over a 10-hour workday.

For relocated tenant activities, maximum 1-hour emissions from on-site cargo-handling equipment assumed that the equipment were operational for the entire 1-hour duration. For relocated tenant trucks, maximum 1-hour emissions were derived from the peak daily emissions which are discussed in Section 3.2 of the EIR under Impact AQ-3. Peak daily emissions were derived using the Port peaking factor described above.

Tables C3-2-2 through C3-2-7 present the maximum 1-hour speciated emissions by source for the CEQA Baseline, Unmitigated Proposed Project, Mitigated Proposed Project, No Project Alternative, Unmitigated Reduced Project Alternative, and Mitigated Reduced Project Alternative, respectively.

3.0 Receptor Locations Used in the HRA

This HRA analyzes the health effects associated with TAC emissions from Project-related sources at a variety of locations (receptors) throughout the project area, including at the locations of potential exposure of residents, offsite workers, recreational users, students, and sensitive members of the public. The analysis utilized a fine grid of 8,603 receptor points spaced every 50 meters (m) apart over the area that extended 250 m outward from the facility boundaries of the Project, relocated tenants, and ICTF. This

fine grid also covered the 250 m buffer around highway I-710 between West Ocean Blvd and CA-91. A medium grid of 691 receptor points spaced every 500 m apart extended roughly 4 kilometers (km) to the east and west, 1 km to the north, and 5.5 km to the south of the fine grid. A coarse grid of 366 receptor points spaced every 1,000 m apart extended up to approximately 16 km from the medium grid. In addition, 49 discrete receptors were placed at sensitive receptor locations of special concern, such as schools, day care centers, convalescent homes, and hospitals within a 5-km radius of the railyard.

Figure C3.3-1 presents the coarse, medium, and fine receptor grids used in the AERMOD modeling analysis discussed in Section 4.0. Figure C3.3-2 shows the locations of the sensitive receptors included in the modeling analysis.

AERMAP, version 09040, was used to calculate source elevations, receptor elevations and the controlling hill height for each receptor.

Maximally exposed individual (MEI) locations were selected from the modeled receptor grids for five different receptor types: residential, occupational, sensitive, student, and recreational. The selection methodology for the MEI locations was:

- The residential MEI was selected from all receptors in residential or residentially-zoned areas.
- The occupational MEI was selected from all receptors outside Port of Los Angeles property (e.g., outside the Project and relocated tenant locations).
- The sensitive MEI was selected from all identified schools, day care centers, convalescent homes, and hospitals in the surrounding area.
- The student MEI was selected from all identified schools in the surrounding area.
- The recreational MEI was selected from all receptors not over water and outside Port of Los Angeles property but including receptors located within the Wilmington and San Pedro Waterfront recreational areas.

4.0 Dispersion Model Selection and Inputs

The air dispersion modeling for the HRA was performed using the USEPA AERMOD dispersion model, version 09292, based on the *Guideline on Air Quality Models* (40 CFR, Part 51, Appendix W; November 9, 2005). The AERMOD model is a steady-state, multiple-source, Gaussian dispersion model designed for use with emission sources situated in terrain where ground elevations can exceed the stack heights of the emission sources. The AERMOD model requires hourly meteorological data consisting of wind vector, wind speed, temperature, stability class, and mixing height. The AERMOD model allows input of multiple sources and source groupings, eliminating the need for multiple model runs. The selection of the AERMOD model is well suited based on (1) the general acceptance by the modeling community and regulatory agencies of its ability to provide reasonable results for large industrial complexes with multiple emission sources, (2) a consideration of the availability of annual sets of hourly meteorological data for use by AERMOD, and (3) the ability of the model to handle the various physical characteristics of project emission sources, including, “point,” “area,” and “volume” source types. AERMOD is a USEPA-approved dispersion model; the SCAQMD approves of its use for mobile source analyses, and CARB’s *Health Risk Assessment Guidance for Rail Yard and Intermodal Facilities* (CARB, 2006a) recommends its use.

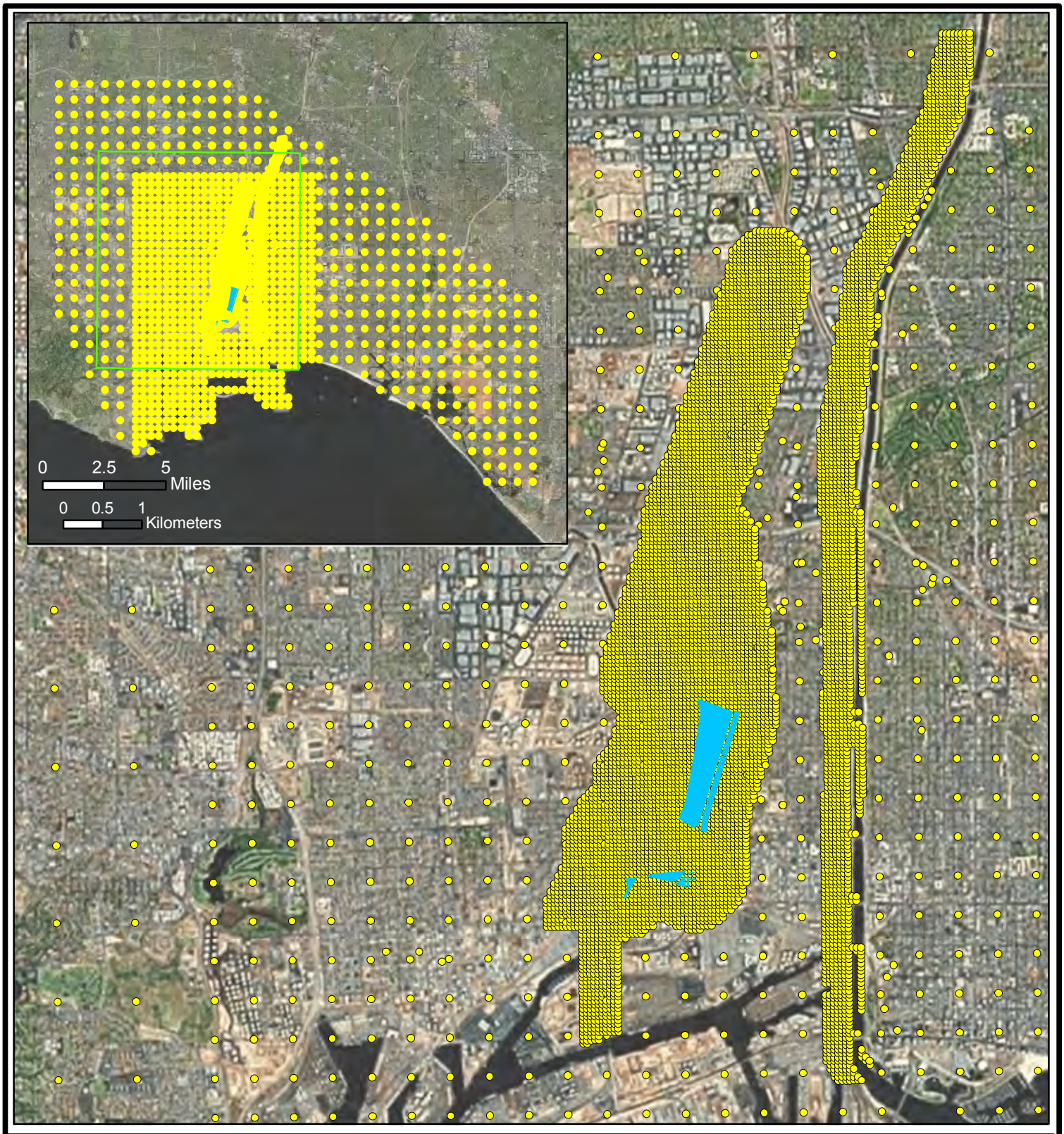
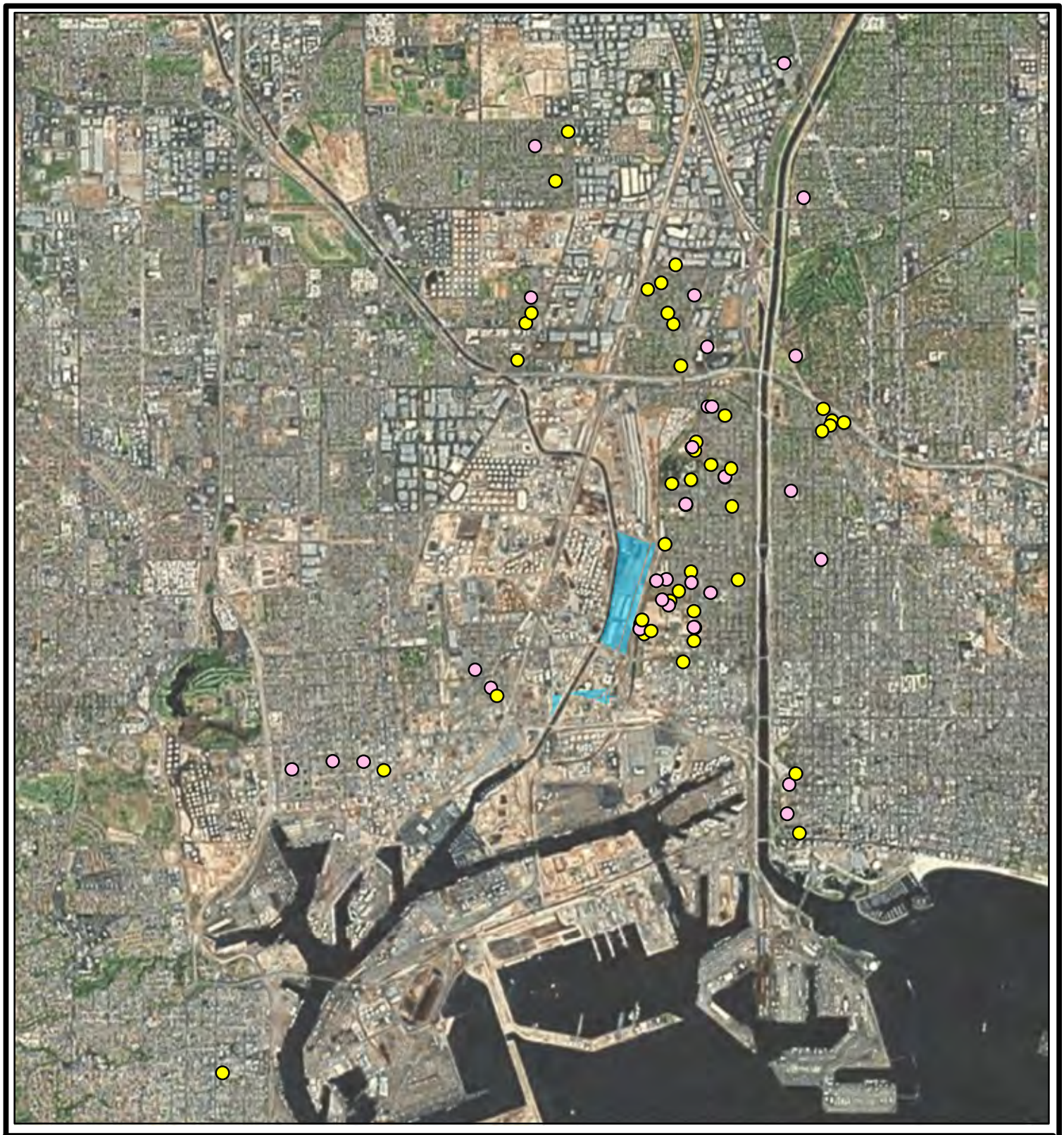


Figure C3.3-1
Coarse, Medium, and Fine Receptor Grids

DRAFT

ENVIRON



Legend

- Non-School Sensitive Receptor
- School Sensitive Receptor
- Site

N

0 0.5 1 Miles

0 0.5 1 Kilometers

Figure C3.3-2
Sensitive Receptor Locations

DRAFT

ENVIRON

4.1 Emission Source Representation

The AERMOD modeling analysis evaluated Project-related construction and operational emission sources, including construction equipment, rail yard equipment, locomotives, and on-road vehicles. The HRA simulated the Project-related emission sources, taking into consideration physical characteristics and operational locations of the sources. Emissions from the movement of locomotives on rail lines, and vehicles on roadways are line-source emissions that were simulated and modeled as a series of separated volume sources. Mobile source operations confined within specific geographic locations, such as the construction equipment, were modeled as a collection of volume sources covering the area. Volume source emissions were simulated by AERMOD as being released and mixed vertically and horizontally within a volume of air prior to being dispersed downwind. The onsite cargo handling equipment emissions were modeled as area sources covering specific geographic locations. Finally, stationary emissions from the emergency generator and rail idling were modeled as point (stack) sources with upward plume velocity and buoyancy.

The operational characteristics of each source type in terms of area of operation and vertical stack height or source height determined the release parameters of each volume or point source. A total of six types of emission sources were simulated in AERMOD. The specific methodology for defining the sources is discussed below.

1. **Construction trucks and equipment.** The areas of SCIG and relocated tenant construction were overlaid with square boxes of various sizes to achieve complete coverage of the surface areas where the construction equipment and truck sources operate. Each of the boxes represents the base of a volume source. The emissions were assumed to be spread uniformly over the entire area represented by the volume sources. Emissions, therefore, were assigned to each volume source in proportion to the base area of the source divided by the total area of all sources. Emissions from construction trucks and equipment were assigned a release height of 15 feet, which is the approximate average height of the exhaust port plus a nominal amount of plume rise.
2. **Cargo handling equipment.** The SCIG rail yard and tenant footprints were covered with polygon area sources to achieve complete coverage of the surface areas where the cargo handling equipment sources operate. The emissions were assumed to be spread uniformly over each area source. Emissions from cargo handling equipment were assigned a release height of 15 feet, which is the approximate average height of the exhaust port plus a nominal amount of plume rise.
3. **Roadways and railways.** Truck and gasoline vehicle movements on roadways and train movements on rail lines were modeled as a series of separated volume sources, as recommended for the simulation of line sources in the AERMOD User's Guide (USEPA, 2004). Roadways were divided into links that have uniform average speeds and widths. Average roadway speeds by roadway link were directly output from the traffic modeling described in Section 3.10. The rail line was assumed to have a width of 9.05 meters where there is only a single track and the combined track width plus 3.05 meters where there are multiple tracks, with uniform emissions per mile of off-site locomotive travel over the entire segment from the SCIG rail yard to I-405. Therefore, the source characteristics for each volume source along a given link are identical except for the centerpoint locations. Total link emissions were divided equally among the number of sources in a given link. Truck idling at the gate was modeled using discrete volume sources.

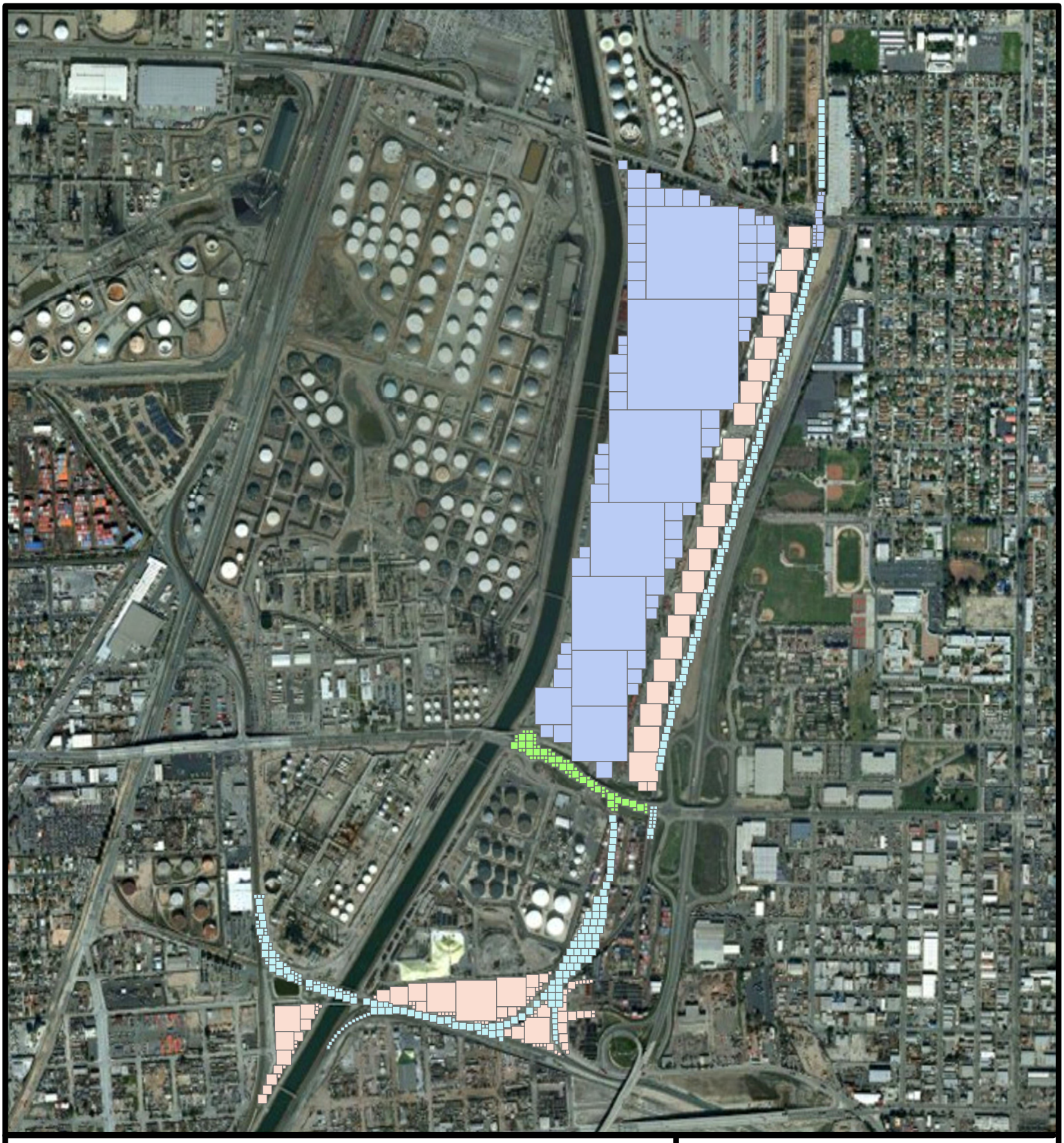
Emissions from trucks were assigned a release height of 15 feet, which is the approximate average height of the exhaust port plus a nominal amount of plume rise, and emissions from gasoline vehicles were assigned a release height of 2 feet. The width of the volume sources for roadways was set equal to the width of the roadway.

Based on the methodology in the Roseville Rail Yard Study, the volume source heights for locomotives in transit were set to between 16 – 20 feet for daytime conditions and 28 – 177 feet for nighttime conditions (CARB, 2004c). Following the same methodology, the volume source height for switcher locomotives was 36 feet for daytime conditions and 51 feet for nighttime conditions. The width of the volume sources for rail lines was set equal to the number of tracks times 3.05 meters per track, except if the rail line had only a single track, in which an additional 3 m was added on each side.

4. **Emergency Generator.** SCIG's emergency generator was modeled as a single point source, with a release height of 3.7 feet, an exit velocity of 10,755 feet per minute, an exit temperature of 879 degrees Fahrenheit, and a stack diameter of 23 feet, based on the Generac Model SD 600 specifications.

The HRA positioned the emission sources by using the Universal Transverse Mercator (UTM) coordinate system (NAD-83) referenced to topographic data obtained from the United States Geological Survey (USGS).

Table C3-4-1 lists the source release parameters used in the AERMOD model. Figures C3.4-1, C3.4-2, and C3.4-3 show the sizes and locations of the emission sources over a base map of the Project vicinity during construction, onsite operation, and offsite operation.

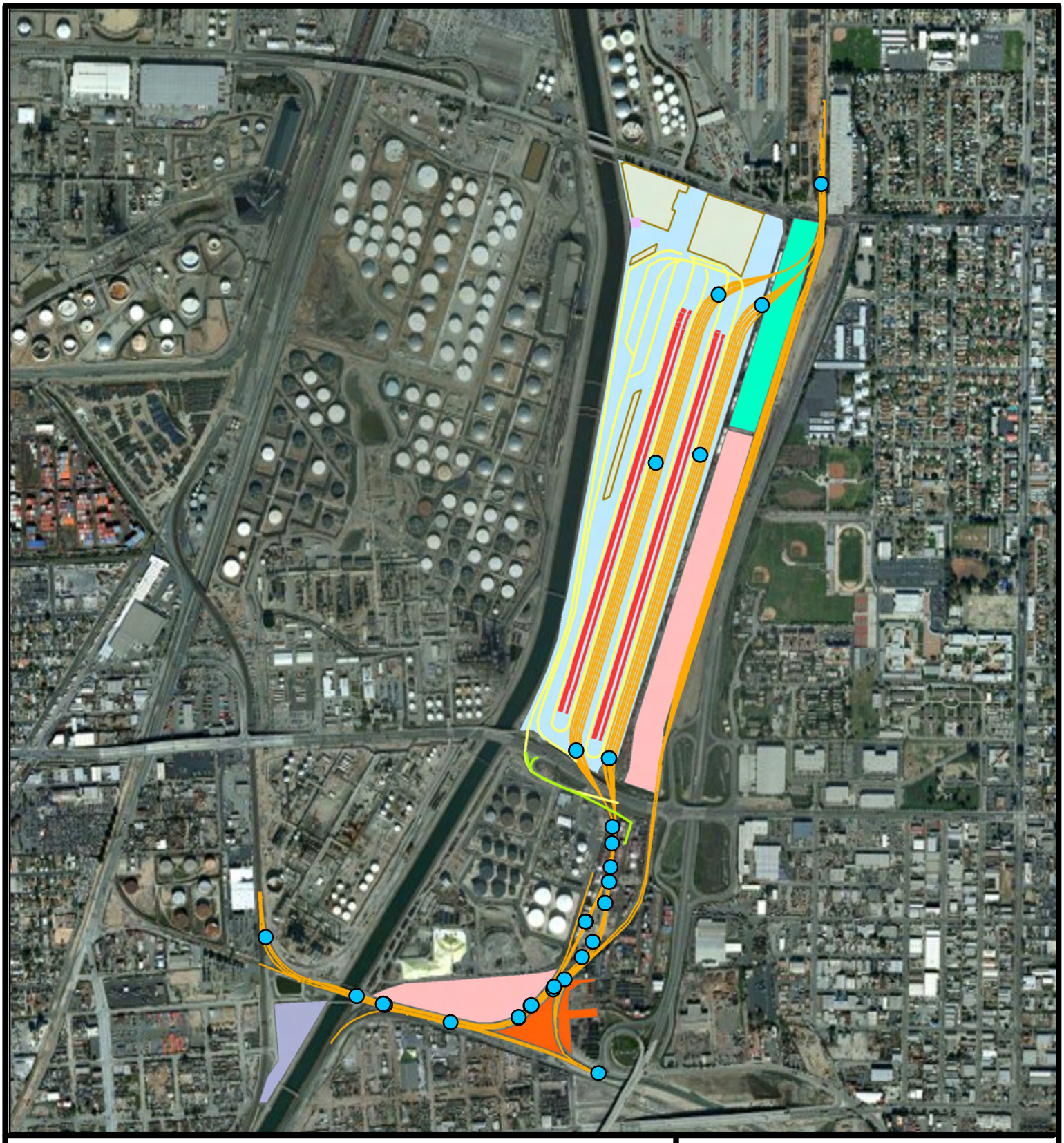


Notes
1. Area sources are modeled for fugitive dust emissions during construction
3. Volume sources are modeled for off-road equipment exhaust emissions during construction

Figure C3.4-1
Source Representation in AERMOD
Construction Sources

DRAFT

ENVIRON

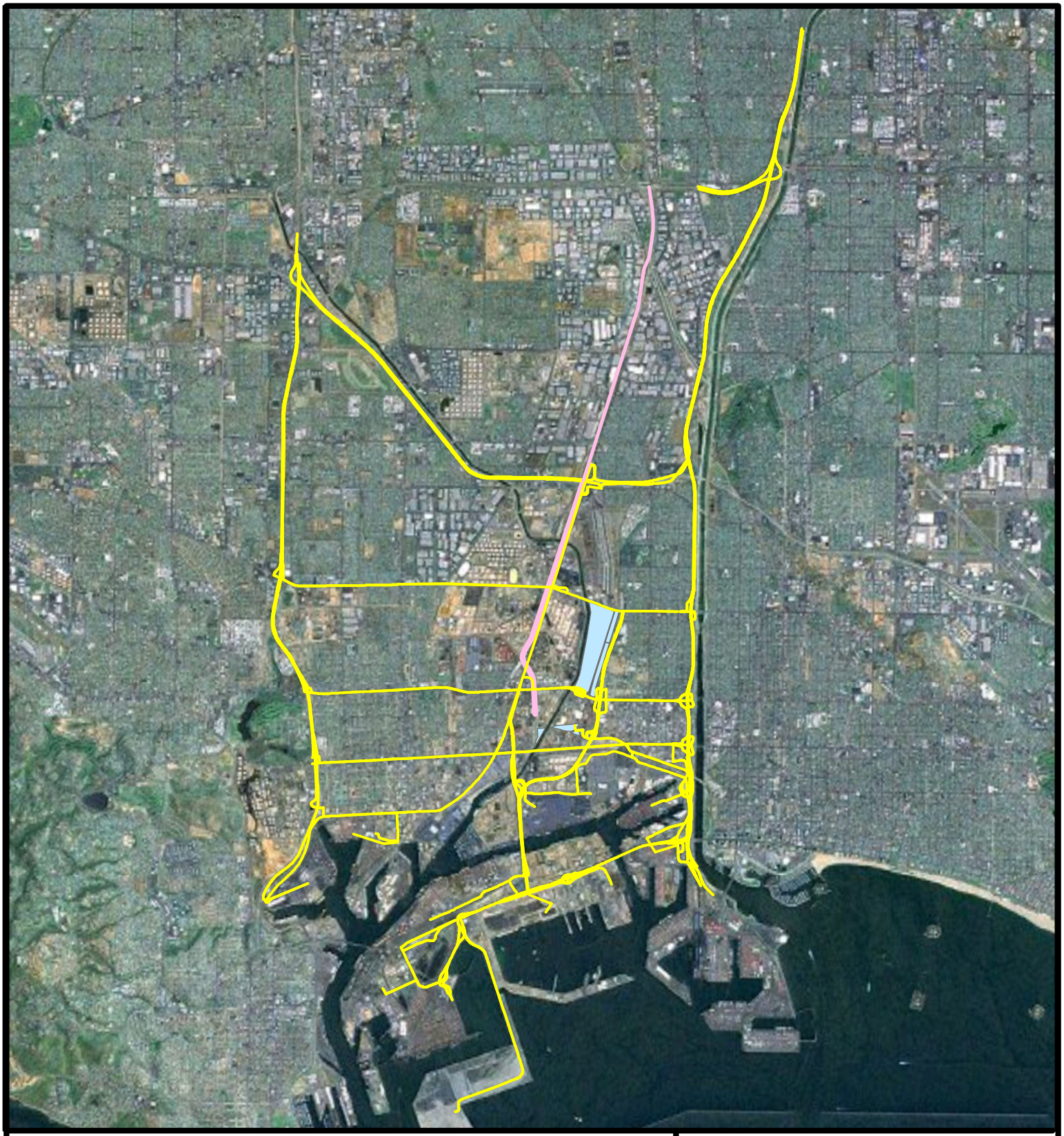


Notes
1. Point sources are modeled for emergency generator and locomotive idling emissions.
2. Area sources are modeled for cargo handling equipment and gasoline vehicle emissions.
3. Volume sources are modeled for locomotive movement and truck emissions.

Figure C3.4-2
Source Representation in AERMOD
Onsite Operational Sources

DRAFT

ENVIRON



Legend

- Project Site
- Alameda Corridor
- Offsite Truck

N

0 0.5 1 Miles

0 0.5 1 Kilometers

Notes
1. Volume sources are modeled for SCIG locomotive emissions on Alameda Corridor and offsite truck traffic between SCIG project site and Port terminals.

Figure C3.4-3
Source Representation in AERMOD
Offsite Operational Sources

DRAFT

ENVIRON

Table C3-4-1. AERMOD Source Release Parameters for the HRA.

Source Type	Source Description	AERMOD Source Type	Release Height (feet)	Source Width (m)	Line Source Spacing (m)	Exit Velocity (fpm)	Exit Temp. (°F)	Stack Diam. (feet)
SCIG and Relocated Tenants Construction	Construction Equipment and Trucks	Volume	15 [a]	Various [b]	—	—	—	—
Cargo Handling Equipment	Wheel Change Out Machines	Area	15[a]	—	—	—	—	—
	Yard Hostler	Area	15[a]	—	—	—	—	—
Locomotives	Line Haul Movement	Volume	Various[c]	Various[e]	50	—	—	—
	Line Haul Idling	Point	15	—	—	684[f]	209[f]	2[f]
	Switcher Movement	Volume	Various[d]	Various[e]	50	—	—	—
	Switcher Idling	Point	15	—	—	3,062[f]	191[f]	0.9[f]
Trucks	Trucks driving between terminals and SCIG or relocated tenants	Volume	15[a]	Various[g]	—	—	—	—
Gasoline Vehicles	Service Truck and Employee Vehicle	Volume	2[h]	Various[g]	50	—	—	—
Emergency Generator	Generac, Model SD600	Point	3.7[i]	—	—	10775[i]	879[i]	0.23[i]

Notes:

a Consistent with the past POLA EIRs.

b It was assumed that construction activities can occur anywhere onsite. Various size of volume sources were used to cover the SCIG and relocated tenant construction area.

c The volume source height for Line Haul locomotives ranges from 16 - 280 feet for daytime and 28 – 177 feet for nighttime conditions, respectively. These heights were derived based on the methodology in the Roseville Railyard Study (CARB, 2004c).

d The volume source height for switcher locomotives was 36 feet for daytime and 51 feet for nighttime conditions, respectively. These heights were derived based on the methodology in the Roseville Railyard Study (CARB, 2004c).

e The width of locomotive volume sources depends on the width of the proposed track lines.

f Source parameters provided by Southwest Research Institute, Steve Fritz, Personal Communication, November 2006.

g The width of trucks and gasoline vehicles depends on the width of the traveled roadways.

h Release height based on CARB Risk Reduction Plan (CARB, 2000) and recommendations from ARB staff.

i Stack Parameters based on a 600 kW generator consistent with parameters used under MOU.

Abbreviations:

fpm feet per minute
m meter
°F degrees Fahrenheit

4.2 Meteorological Data

The dominant terrain features/water bodies that may influence wind patterns in this part of the Los Angeles Basin include the Pacific Ocean to the west, the hills of the Palos Verdes Peninsula to the west/southwest and the San Pedro Bay and shipping channels to the south of the study area. Although the area in the immediate vicinity of the Ports of Los Angeles (POLA or the Port) and Long Beach (POLB) is generally flat, these terrain features/water bodies may result in significant variations in wind patterns over relatively short distances (POLA/POLB, 2010).

POLA and POLB currently are operating monitoring programs that include the collection of meteorological data from several locations within port boundaries (Port, 2004). The data sets contain 8,760 hourly observations of wind speed, wind direction, temperature, atmospheric stability, and mixing height recorded at each of the monitoring stations in the

network. The meteorological data stations to the west of the Palos Verdes Hills and within approximately 5 kilometers of the San Pedro Bay generally exhibit predominant winds from the northwest and from the south or southeast. The consistency of the predominant winds among these stations indicates that the Palo Verdes Hills are channeling the winds from the northwest and that the San Pedro Bay and shipping channels influence the winds from the south and southeast (POLA/POLB, 2010).

Because all of the Long Beach area stations indicate the same general wind patterns (i.e., predominant winds from the northwest and south/southeast), and due to data quality issues identified for most other stations in this area, the Saints Peter and Paul Elementary School (SPPS) meteorological station in Wilmington, about 2.5 miles southwest of the project site, and the Terminal Island Treatment Plant (TITP) meteorological station, about 4 miles southwest of the project site, were selected as representative meteorological stations for the on-Port emissions and out-of-Port truck emissions on major freeways and locomotive emissions on the Alameda Corridor in the northern part of Long Beach. The Berth 47 (B47) station is located at the southern tip of the Port of Los Angeles, where the winds appear to be heavily influenced by the San Pedro Bay and predominant winds are from the southwest. The B47 station is characterized by higher wind speeds and less variation in wind direction than patterns further inland (POLA/POLB, 2010).

To account for the unique wind patterns in the project area, the modeling domain for this analysis was split into inner, middle and outer harbor regions. The inner harbor zone is north of the East Basin Channel, Cerritos Channel, and Vincent Thomas Bridge, and bounded by Interstate 110 on west, Interstate 710 on the east, and an approximate east-west line created by Interstate 405 and 223rd Street in the northern part of Long Beach on the north. The middle harbor zone is the majority of Terminal Island and San Pedro. The outer harbor zone is the terminals on the southern end of Terminal Island and inside breakwater. Emission sources located in the inner harbor region, which includes construction sources and most operational sources, were modeled with the SPPS meteorological data. Emission sources located in the middle harbor region, which includes truck traffic between the project site and the terminals, were modeled with the TITP meteorological data. Emission sources located in the outer harbor region, which includes truck traffic near the breakwater, were not included based on the results of a sensitivity analysis that showed that sources in the outer harbor region contributed less than 0.6% of the risk from DPM at the MEIR. As a result, the B47 meteorological data was not used in the analysis. The modeling results were then summed at each common receptor point.

The meteorological data were processed using the USEPA's approved AERMET (version 06341) meteorological data preprocessor for the AERMOD dispersion model. AERMET uses three steps to preprocess and combine the surface and upper-air soundings to output the data in a format which is compatible with the AERMOD model. The first step extracts the data and performs a brief quality assurance check of the data. The second step merges the meteorological data sets. The third step outputs the data in AERMOD-compatible format while also incorporating surface characteristics surrounding the collection or application site.

The output from the AERMET model consists of two separate files: the surface conditions file and a vertical profile dataset. AERMOD utilizes these two files in the dispersion modeling algorithm to predict pollutant concentrations resulting from a source's emissions.

4.3 Model Options

Technical options selected for the AERMOD model used regulatory defaults. Use of these options follows the USEPA modeling guidance (40 CFR, Appendix W; November 2005).

The following temporal distribution of emissions was modeled for peak 1-hour, peak 8-hour, peak 24-hour, and annual average concentrations:

Construction (SCIG)	100% of emissions 8am – 6pm
Offsite Trucks and Gasoline Vehicles (SCIG), Locomotives (SCIG), Cargo Handling Equipment (SCIG), Emergency Generator (SCIG), Onsite Gasoline Vehicles (SCIG)	Uniform distribution of emissions 24 hr/day
Offsite Gasoline Vehicles (Tenants), Offsite Trucks (California Cartage and Fastlane)	100% of emissions 6am – 6pm
Offsite Trucks (All Tenants Other Than California Cartage and Fastlane)	100% of emissions 8am – 4pm
Construction (Tenants)	100% of emissions 9am – 5pm
Onsite Sources (Tenants)	Variable by Tenant Operation Schedule

These emission distributions are based on the Baseline and Proposed Project operation schedules of SCIG and the affected tenants.

5.0 Calculation of Health Risks

An HRA spanning years 2013-2082 was conducted pursuant to a project-specific Protocol developed by the Port of Los Angeles and reviewed by SCAQMD (POLA, 2008). The period 2013-2082 is the 70-year exposure period with the greatest combined DPM emissions from the proposed Project construction and operation. Seventy-year average TAC concentrations were used to estimate cancer risk to residential receptors, sensitive receptors, and recreational receptor populations (see following). In addition, the HRA evaluated the cancer risk from project emissions to workers based on average emissions calculated over a 40-year period (years 2013 to 2052) and evaluated the cancer risk to students based on peak annual emissions for an exposure duration of 6 years. The HRA was performed in a manner consistent with methodologies specified in:

- *Air Toxics Hot Spots Program Risk Assessment Guidelines (OEHHA, 2003)*
- *Health Risk Assessment Guidance for Analyzing Cancer Risks from Mobile Source Diesel Idling Emissions for CEQA Air Quality Analysis (South Coast Air Quality Management District [SCAQMD], 2003),*
- *Air Resources Board Recommended Interim Risk Management Policy for Inhalation-Based Residential Cancer Risk (Air Resources Board [ARB], 2003)*
- *Supplemental Guidelines for Preparing Risk Assessments for the Air Toxics "Hot Spots" Information and Assessment Act (AB2588) (SCAQMD, 2005),*
- *Health Risk Assessment Protocol for Port of Los Angeles Terminal Improvement Projects (Los Angeles Harbor Department [Port of Los Angeles],* 2005).

In addition to cancer risk and non-cancer hazard, the HRA considered cancer burden, which is the estimated number of cancer cases for a population exposed over a 70-year period to project emissions (OEHHA, 2003; SCAQMD, 2011). Because the proposed

Project would generate DPM during construction and operation, the HRA also discusses and evaluates the effects of PM on mortality and morbidity. .

Chronic and acute non-cancer effects were evaluated by calculating a hazard index (HI). The chronic non-cancer HI is a ratio of the maximum annual average concentration of a TAC to a chronic reference exposure level (REL). Similarly, an acute non-cancer HI is the ratio of the maximum hourly concentration of a TAC to an acute REL.

5.1 Toxicity Factors

The inhalation unit risk factor is the upper-bound excess lifetime cancer risk estimated to result from continuous exposure to a TAC at a concentration of 1 $\mu g/m^3$ in air (US Environmental Protection Agency [USEPA], 2011). The inhalation unit risk factor is used to calculate a potential inhalation cancer risk using risk algorithms defined in OEHHA (2003).

The likelihood for non-cancer effects was evaluated by developing HIs, which, as noted above, represent the ratio of the modeled concentration of each TAC to the REL. RELs are developed by OEHHA (2008) and each is an estimate of the continuous inhalation exposure concentration to which the human population (including sensitive subgroups) may be exposed without appreciable risk of experiencing adverse non-cancer effects. A chronic non-cancer HI below 1.0, or an acute HI below 1.0 indicates that adverse non-cancer health effects from long-term or short-term exposure, respectively, are not expected.

Table C3-5-1 presents the cancer, chronic non-cancer, and acute non-cancer toxicity factors used to assess health risks in this study. As noted in the TAC Emission Calculation Approach (section 2.2), the TACs listed in this table were identified from the speciation of all non-DPM sources (e.g., tire and brake wear and alternate-fueled engines), as well as the speciation of DPM for the assessment of acute health effects.

Table C3-5-1. Toxicity Factors Used in the HRA.

Pollutant	CAS Number	Inhalation Cancer Potency Factor (mg/kg-d)-1 a	Chronic Inhalation REL (µg/m3) b	Target Organ for Chronic Exposure	Acute Inhalation REL (µg/m3) b,c	Target Organ for Acute Exposure
Acetaldehyde	75070	0.01	140	I	470	D,I
Acrolein (2-propenal)	107028	--	0.35	I	2.5	D,I
Ammonia	7664417	--	200	I	3200	D,I
Arsenic	7440382	12	0.015	B,C,G,I,J	0.2	B,C,G
Benzene[c]	71432	0.1	60	C,E,G	1300	C,H
1,3-butadiene	106990	0.6	20	H	--	--
Cadmium	7440439	15	0.02	I,M	--	--
Chlorine	7782505	--	0.2	I	210	D,I
Copper	7440508	--	--	--	100	I
DPM[d]	9901	1.1	5	I	--	--
Ethylbenzene	100414	0.0087	2000	A,L,M	--	--
Formaldehyde	50000	0.021	9	I	55	D,I
Hexavalent chromium	18540299	510	0.2	I	--	--
Isomers of xylene	1330207	--	700	D,G,I	22000	D,G,I
Lead	7439921	0.042	--	--	--	--
Manganese	7439965	--	0.09	G	--	--
Mercury	7439976	--	0.03	G	0.6	G

Pollutant	CAS Number	Inhalation Cancer Potency Factor (mg/kg-d)-1 a	Chronic Inhalation REL (µg/m3) b	Target Organ for Chronic Exposure	Acute Inhalation REL (µg/m3) b,c	Target Organ for Acute Exposure
Methyl alcohol	67561	--	4000	C	28000	G
Methyl ethyl ketone (mek) (2-butanone)	78933	--	--	--	13000	D,I
M-xylene	108383	--	700	D,G,I	22000	D,G,I
Naphthalene	91203	0.12	9	I	--	--
N-hexane	110543	--	7000	G	--	--
Nickel	7440020	0.91	0.05	E,I	6	F,I
O-xylene	95476	--	700	D,G,I	22000	D,G,I
Propylene	115071	--	3000	I	--	--
P-xylene	106423	--	700	D,G,I	22000	D,G,I
Selenium	7782492	--	20	A,B,G	--	--
Styrene	100425	--	900	G	21000	C,D,H,I
Sulfates	9960	--	--	--	120	I
Toluene	108883	--	300	C,G,I	37000	G,I
Vanadium (fume or dust)	7440622	--	--	--	30	D, I

Notes:
a CARB 2011
b OEHHA 2008
c The acute exposure period is 1 hour for all compounds except benzene (6 hours).
d For diesel ICEs and diesel trucks, only DPM emissions were evaluated for cancer risk and chronic hazard indices, because DPM is a surrogate for the combined health effects associated with exposure to diesel exhaust emissions. For all other emission sources (external combustion boilers, alternative fuel engines, tire and brake wear), emissions of the 30 other toxic air contaminants were evaluated for cancer risk and chronic hazard indices. For the acute hazard indices, DPM was not evaluated; rather, emissions of the 30 other toxic air contaminants were evaluated for all emission sources (including diesel ICEs).

Key to noncancer acute and chronic exposure target organs:

A. Alimentary Tract	H. Reproductive System
B. Cardiovascular System	I. Respiratory System
C. Developmental System	J. Skin
D. Eye	K. Bone
E. Hematologic System	L. Endocrine System
F. Immune System	M. Kidney
G. Nervous System	Source: OEHHA 2008

References:

California Environmental Protection Agency Air Resources Board (CARB). 2011. California Consolidated Table of OEHHA/ARB Approved Risk Assessment Health Values. February 14, 2011.
California Environmental Protection Agency Office of Environmental Health Hazard Assessment (OEHHA). 2008. All Chronic Reference Exposure Levels Adopted by OEHHA. Last updated on December 18.

5.2 Health Effects of Particulate Matter

Particulate matter small enough to be inhaled and retained by the lungs is a public health concern. These respirable particles (particulate matter less than about 10 micrometers in diameter [PM_{10}] and particulate matter less than 2.5 micrometers in diameter [$PM_{2.5}$]) can accumulate in the respiratory system or penetrate into the vascular system, causing or aggravating diseases such as asthma, bronchitis, lung disease, and cardiovascular disease. Children, the elderly, and the ill are believed to be especially vulnerable to adverse health effects of PM_{10} and $PM_{2.5}$.

PM in ambient air is a complex mixture that varies in size and chemical composition, as well as varying spatially and temporally. PM is generated from a number of sources such

as the combustion of petroleum-based fuels, forest fires, and re-suspension of soil. At the present time, the PM released from combustion of diesel fuel, diesel exhaust particulate matter (DPM), can't be reliably distinguished from other sources of PM. The CARB and OEHHA consider DPM and PM to have equivalent toxicity.

Numerous studies have been published over the past 15 years that have established a strong correlation between the inhalation of ambient PM and an increased incidence of premature mortality from heart and/or lung diseases (Pope et al., 1995, 2002; 2004; Jerrett et al. 2005; Krewski et al., 2001; Gauderman et al., 2007). Asthma onset and the exacerbation of existing disease have also been linked to PM exposure (Pandaya et al., 2002; Jerrett et al., 2008; Clark et al., 2010). Studies such as these provide the basis for PM air quality standards promulgated by SCAQMD, CARB, EPA, and the World Health Organization.

5.2.1 Quantifying Mortality and Morbidity

The Port has previously included analyses of PM-related mortality in the TraPac, China Shipping, and San Pedro Waterfront EIRs. The latter two documents utilized a methodology published by CARB (2006c), which was primarily developed for large geographic areas such as air basins or the entire state. In CARB (2008), the agency noted that the methods for applying calculations of mortality to a project-level scale were not fully developed, and that such applications should include explicit statements regarding the uncertainties and limitations. Notwithstanding these uncertainties, the Port has received requests from individuals, environmental groups, the SCAQMD, OEHHA, and the CARB to include separate quantitative assessments of project-related PM-attributable mortality as well as morbidity in their CEQA analyses. In response to these requests POLA developed a methodology to calculate mortality and morbidity from project emissions. A complete description of the methodology, including supporting equations and references, is available in POLA (2011).

In brief, the Port has committed to quantifying mortality and morbidity from PM exposure if dispersion modeling of ambient air quality concentrations for operation of the project (project minus Baseline) results in the identification of a significant impact for 24-hour concentrations of $PM_{2.5}$ (Impact AQ-4 in POLA CEQA documents).

No CEQA significance thresholds have been identified for premature mortality or morbidity by any state or local regulatory agency. With the exception of the three previous POLA EIRs, there is no precedent for calculating premature mortality for project-level effects under CEQA, and no precedent for completing project-attributable morbidity calculations from PM under CEQA. As specified in POLA (2011), POLA has determined that mortality and morbidity will be calculated when the incremental operational emissions would result in off-site 24-hour $PM_{2.5}$ concentrations that exceed the SCAQMD significance criterion of 2.5 $\mu g/m^3$. The geographic area of analysis for the mortality and morbidity calculations is all census blocks partially or fully within the 2.5 $\mu g/m^3$ $PM_{2.5}$ peak daily concentration isopleths for the project minus CEQA Baseline. This approach is consistent with the significant impact threshold identified by the SCAQMD for $PM_{2.5}$. Project-specific estimates of the exposed population will be developed based on the residential population within these census blocks.

Mortality will be calculated using the relative risk factor of a 10% increase in premature deaths per year (mortality rate) per 10 $\mu g/m^3$ increase in $PM_{2.5}$ concentration (CARB, 2008). Morbidity calculations will follow the general methodology and available concentration-response data described by CARB (2002, 2006c) and provided in POLA

(2011). Morbidity endpoints that are calculated on an annual basis will be based on project-specific incremental annual $PM_{2.5}$ concentrations (e.g., project minus Baseline). Morbidity endpoints that require estimates of daily impacts will be based on daily average $PM_{2.5}$ concentrations.

The specific health effect endpoints that will be evaluated include:

- Hospital admissions for chronic obstructive pulmonary disease
- Hospital admissions for pneumonia
- Hospital admissions for cardiovascular disease
- Acute bronchitis
- Hospital admissions for asthma
- Emergency Room visits for asthma
- Asthma attacks
- Lower respiratory symptoms
- Work loss days
- Minor restricted activity days

To address mortality and morbidity over the multiple years of a project's lease, the annual incidence for each endpoint will be summed to provide an estimate of the aggregate effects attributable to a project's incremental PM emissions.

5.3 Cancer Burden

The Office of Environmental Health Hazard Assessment defines cancer burden as "an estimate of the number of cancer cases expected from a 70□year exposure …" to current estimated emissions (OEHHA, 2003). Whereas cancer risk represents the probability of an individual to develop cancer, cancer burden multiplies the cancer risk by the exposed population to estimate the number of individuals that would be expected to contract cancer. The exposed population is defined as the number of persons within a facility's zone of impact, which is typically the area within the facility's one in a million cancer risk isopleths. Consistent with this definition, cancer burden will be calculated only if a project alternative is associated with cancer risks of one in a million or above.

5.4 Exposure Scenarios for Individual Lifetime Cancer Risk

For the cancer risk evaluation, the frequency and duration of exposure to TACs are assumed to be directly proportional to the risk. Therefore, this HRA used specific exposure assumptions for each receptor type, as described below.

1. **Residential and Sensitive Receptors.** Cancer risks for residential and sensitive receptors were estimated using the breathing rates described in the *CARB Recommended Interim Risk Management Policy for Inhalation-Based Residential Cancer Risk (October 2003)* (CARB, 2004a). The HRA determined residential and sensitive receptor cancer risks by using a breathing rate of 302 liters per kilogram day (corresponding to an 80th percentile value) and an exposure duration of 24 hours per day, 350 days per year over 70 years. For supplemental information, residential

cancer risks also were calculated using a 65th percentile ("average") breathing rate of 271 L/kg-day and a 95th percentile ("high-end") breathing rate of 393 L/kg-day.

2. **Occupational impacts**. Workers generally do not spend as much time within the region of a project as do residents. The SCAQMD, therefore, allows an exposure adjustment for workers (SCAQMD, 2005a). Lifetime occupational exposure is based on a worker presence of 8 hours per day, 245 days per year for 40 years (as recommended by OEHHA [2003]). The breathing rate for workers is equal to 447 L/kg-day, which equates to 149 L/kg-day over an 8-hour workday (OEHHA, 2003).
3. **Student impacts**. The policy of the SCAQMD is to evaluate student cancer risk based upon a full 70 years of exposure. However, students actually spend a far more limited portion of their lives at a given school than 70 years. Accordingly, student exposures were calculated based on a student presence of 6 hours per day, 180 days per year for 6 years. The breathing rate of children is equal to 581 L/kg-day (OEHHA, 2003).
4. **Recreational user impacts**. Exposures for recreational users were estimated based on an exposure frequency of 2 hours per day, 350 days per year, and an exposure duration of 70 years. The breathing rate of a person engaged in recreational activities is assumed to be a "heavy-activity" rate equal to 1,097 L/kg-day, which was obtained from the USEPA *Exposure Factors Handbook* (USEPA, 1997).

Table C3-5-2 summarizes the primary exposure assumptions used to calculate individual lifetime cancer risk by receptor type.

Table C3-5-2. Exposure Assumptions for Individual Lifetime Cancer Risk.

Receptor Type	Exposure Frequency		Exposure Duration	Breathing Rate
	Hours/Day	Days/Year	(Years)	(L/kg-day)
Residential	24	350	70	302
Occupational	8	245	40	447
Sensitive	24	350	70	302
Student	6	180	6	581
Recreational	2	350	70	1,097

Notes:

aThe residential breathing rate of 302 L/kg BW-day represents the 80th percentile breathing rate. For informational purposes, residential cancer risks were also calculated for a 95th percentile ("high end") breathing rate of 393 L/kg BW-day (OEHHA, 2003).

bThe occupational exposure frequency of 245 days/year represents 5 days/week, 49 weeks/year. The occupational breathing rate of 447 L/kg BW-day equates to 149 L/kg BW-day over an 8-hour work day (OEHHA, 2003).

cThe student breathing rate of 581 L/kg BW-day represents the high end child breathing rate (OEHHA, 2003).

dThe recreational breathing rate of 1,097 L/kg BW-day represents a "heavy activity" breathing rate, which is derived from a breathing rate of 3.2 m3/hr (and assuming a 70-kg adult) as reported in the USEPA Exposure Factors Handbook (USEPA, 1997). This recreational breathing rate is conservative because it assumes that an individual could sustain the maximum hourly breathing rate for 2 consecutive hours.

6.0 Significance Criteria for Project Health Risks

The Port has adopted the significance threshold of 10 in a million as being an acceptable level of risk for receptors. Based on this threshold, a project would produce less than significant cancer risk impacts if the maximum incremental cancer risk due to the project is less than 10 chances in 1 million (10×10^{-6}).

The Port has also adopted the recently-established air quality significance threshold for cancer burden of > 0.5 excess cancer cases in areas with project-attributable cancer risk above one in a million (1×10^{-6}) (SCAQMD, 2011).

For chronic and acute non-cancer exposures, maximum predicted annual and 1-hour TAC concentrations are compared with the RELs developed by OEHHA to yield hazard indices. Hazard indexes above 1.0 represent the potential for an unacceptable health effects, and represent CEQA significance criteria for non-cancer effects.

For the determination of significance from a CEQA standpoint, this HRA determined the incremental increase in health effects values due to the proposed Project by estimating the net change in impacts between each proposed Project and Baseline conditions. These incremental health effects values were compared to the significance thresholds described above.

7.0 Predicted Health Impacts

7.1 Unmitigated Proposed Project Health Impacts

Table C3-7-1 presents a summary of the maximum health impacts that would occur for each receptor type with construction and operation of the Unmitigated Proposed Project. The table also shows the maximum health impacts from the Baseline, as well as the CEQA increment (Unmitigated Proposed Project minus Baseline). Because the results in Table C3-7-1 represent the maximum impacts predicted for each receptor type, the impacts at all other receptors would be less than these values.

The data in Table C3-7-1 show that the CEQA cancer risk increment at the location of the Unmitigated Proposed Project MEI is predicated to be -160 in a million (-160×10^{-6}), at a residential receptor. This risk value, as well as the risk value at all residential receptors, is below the significance threshold of 10 in a million. The CEQA increments would be below the CEQA significance threshold at all receptors, including occupational, sensitive, student, and recreational.

Table C3-7-1. Maximum Health Impacts Associated with the Unmitigated Proposed Project.

Health Impact	Receptor Type	Maximum Predicted Impact			Significance Threshold
		Proposed Project	CEQA Baseline	CEQA Increment	
Cancer Risk	Residential	48×10^{-6} (48 in a million)	568×10^{-6} (568 in a million)	-160×10^{-6} (-160 in a million)	10×10^{-6} (10 in a million)
	Occupational	41×10^{-6} (41 in a million)	215×10^{-6} (215 in a million)	-114×10^{-6} (-114 in a million)	
	Sensitive	41×10^{-6} (41 in a million)	220×10^{-6} (220 in a million)	-179×10^{-6} (-179 in a million)	
	Student	2.7×10^{-6} (2.7 in a million)	4.7×10^{-6} (4.7 in a million)	-2×10^{-6} (-2 in a million)	
	Recreational	62×10^{-6} (62 in a million)	329×10^{-6} (329 in a million)	-175×10^{-6} (-175 in a million)	
Chronic Hazard Index	Residential	0.09	0.36	-0.05	1.0
	Occupational	0.47	0.69	0.11	
	Sensitive	0.11	0.16	-0.06	

Health Impact	Receptor Type	Maximum Predicted Impact			Significance Threshold
		Proposed Project	CEQA Baseline	CEQA Increment	
	Student	0.11	0.16	-0.06	
	Recreational	0.47	0.69	0.11	
Acute Hazard Index	Residential	0.19	0.29	0.01	
	Occupational	0.65	0.79	013	
	Sensitive	0.21	0.27	-0.062	1.0
	Student	0.21	0.27	-0.062	
	Recreational	0.65	0.79	0.13	

a Exceedances of the significance thresholds are in bold. The significance thresholds apply to the CEQA increments only.
b The maximum increments might not occur at the same receptor locations as the maximum impacts. This means that the increments cannot necessarily be determined by subtracting the CEQA Baseline impact from the project impact. Rather, the subtraction must be done at each receptor, for all modeled receptors, and the maximum result selected.
c The CEQA Increment represents Project minus CEQA Baseline.
d When the maximum increment for a receptor type is negative, the maximum increment displayed is the increment at the maximum project receptor location.
e Data represent the receptor locations with the maximum impacts or increments. The impacts or increments at all other modeled receptors would be less than these values for each receptor type.
f The cancer risk values reported in this table for the residential receptor are based on the 80th percentile breathing rate. The risks associated with the 65th percentile (average) breathing rate will be less than these values. The risks associated with the 95th percentile (high end) breathing rate are 63 x 10-6 for the Project impact, 740 x 10-6 for the CEQA Baseline impact, and -208 x 10-6 for the CEQA increment.

The maximum chronic hazard index increments are predicted to be less than the CEQA significance of 1.0 at all receptors.

The maximum acute hazard index increments are predicted to be less than the CEQA significance threshold of 1.0 at each receptor type.

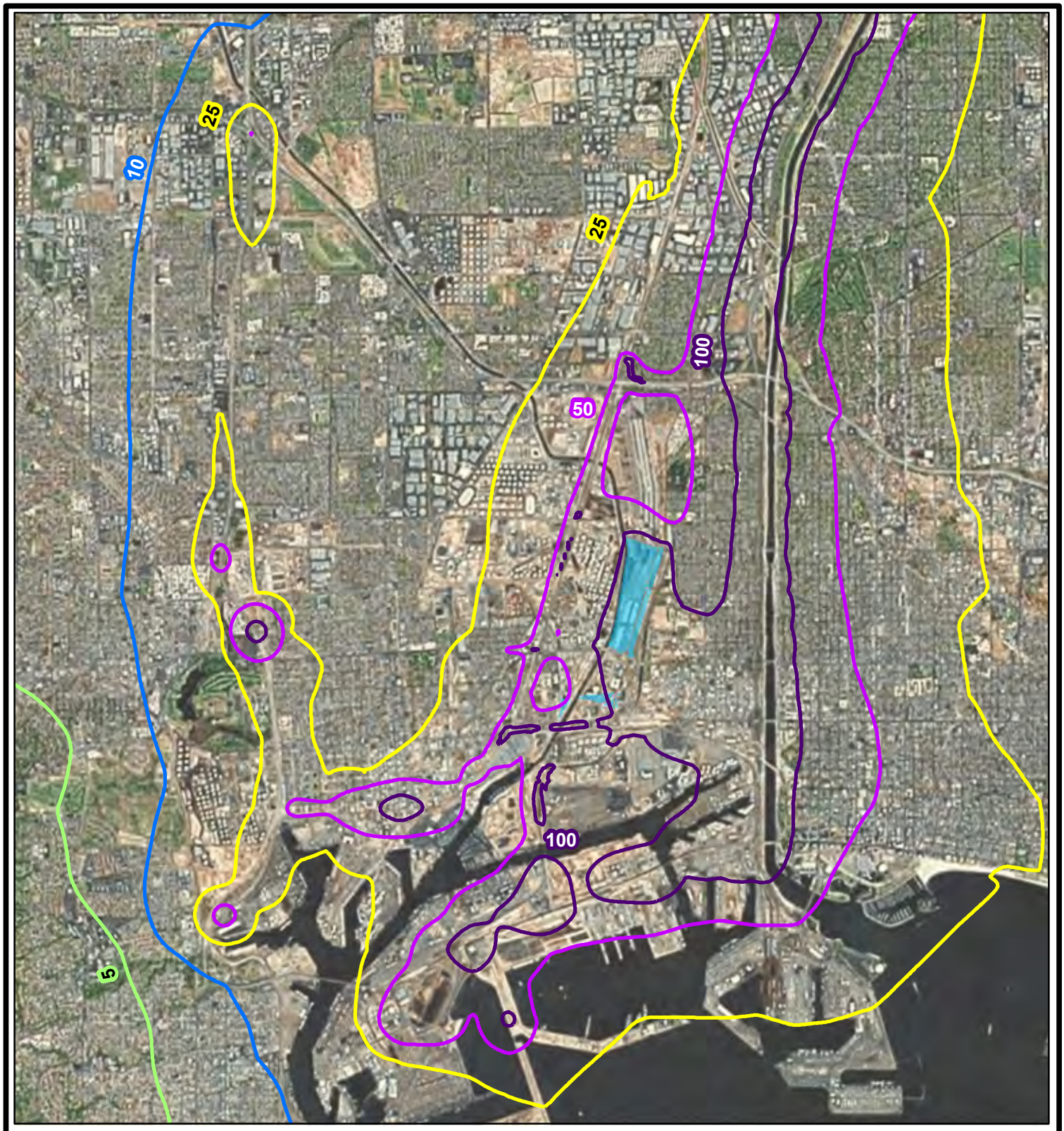
To illustrate the geographical extent of health risk impacts associated with the proposed Project, a series of health risk isopleths (contours) has been prepared. The isopleths show individual lifetime cancer risks overlaid on a map of the surrounding community, assuming residential exposure conditions (24 hours per day, 350 days per year, for 70 years) and an 80th percentile breathing rate. Figure C3.7-1 shows the Baseline residential individual lifetime cancer risk (per million).

Figures C3.7-2, C3.7-3, and C3.7-4 show the maximum receptor locations for the Baseline for cancer risk, chronic HI, and acute HI, respectively. The residential, occupational, and recreational MEIs are not necessarily located directly on existing homes, workplaces, or recreational facilities; rather, they are located in areas that contain these land use types.

Figures C3.7-5 and C3.7-6 show the residential cancer risk isopleths associated with the Unmitigated Proposed Project and Unmitigated Proposed Project minus Baseline, respectively.

Figures C3.7-7, C3.7-8, and C3.7-9 show the maximally exposed receptor locations for the Unmitigated Proposed Project for cancer risk, chronic HI, and acute HI, respectively. The residential, occupational, and recreational MEIs are not necessarily located directly on existing homes, workplaces, or recreational facilities; rather, they are located in areas that contain these land use types.

Table C3-7-2 presents the contributions from each emission source to the maximum health effects values for the Unmitigated Proposed Project. At the maximum residential receptor, the greatest contributor to the cancer risk is SCIG offsite and onsite trucks. The



Legend

- 5
- 10
- 25
- 50
- 100
- Site

N

0 0.5 1 Miles

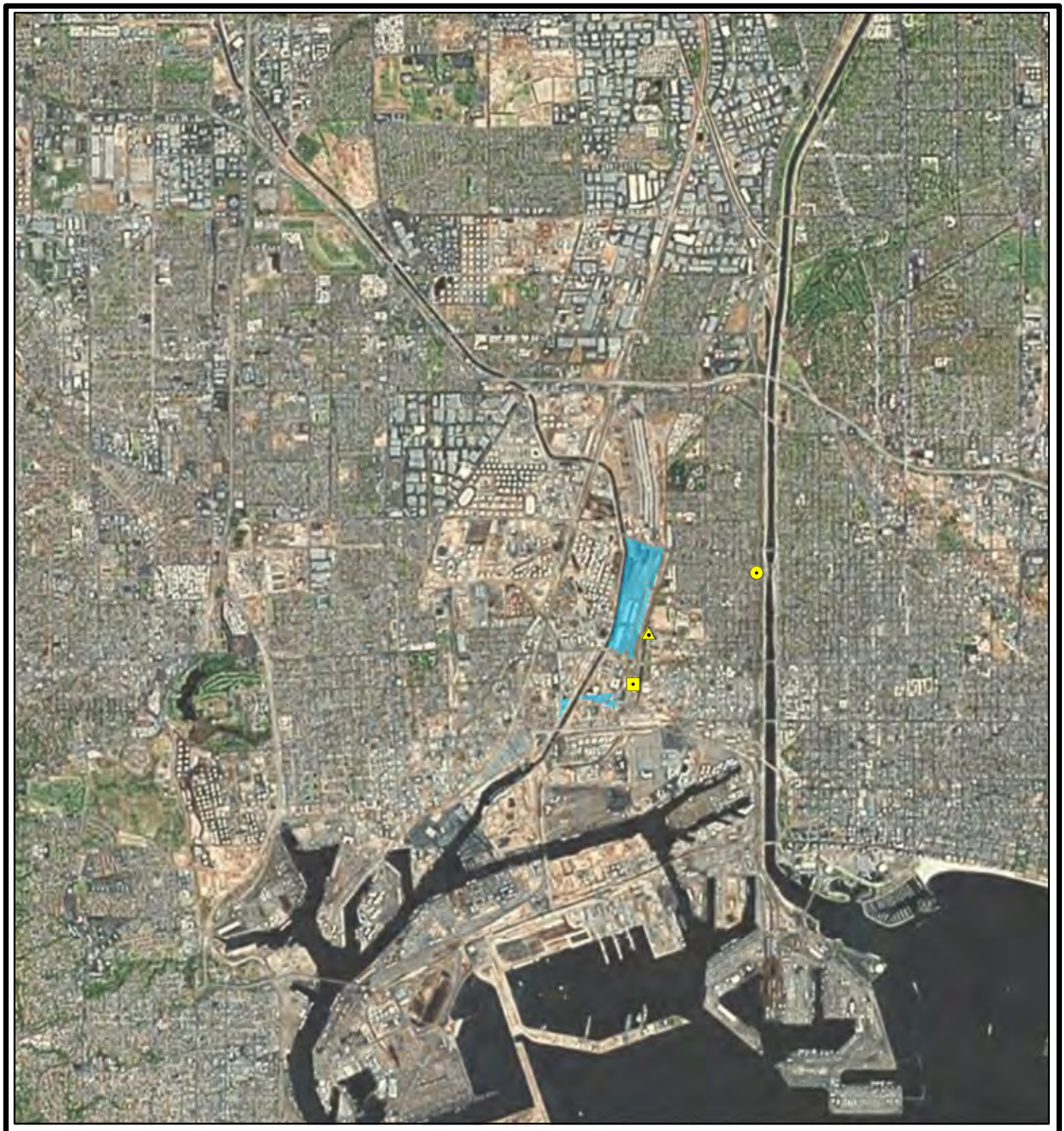
0 0.5 1 Kilometers

Figure C3.7-1
Baseline

Residential Individual Lifetime Cancer Risk (per Million)

DRAFT

ENVIRON



Legend

- Residential MEI - CEQA Baseline
- Occupational and Recreational MEI - CEQA Baseline
- Sensitive and Student MEI - CEQA Baseline
- Site

N

0 0.5 1 Miles

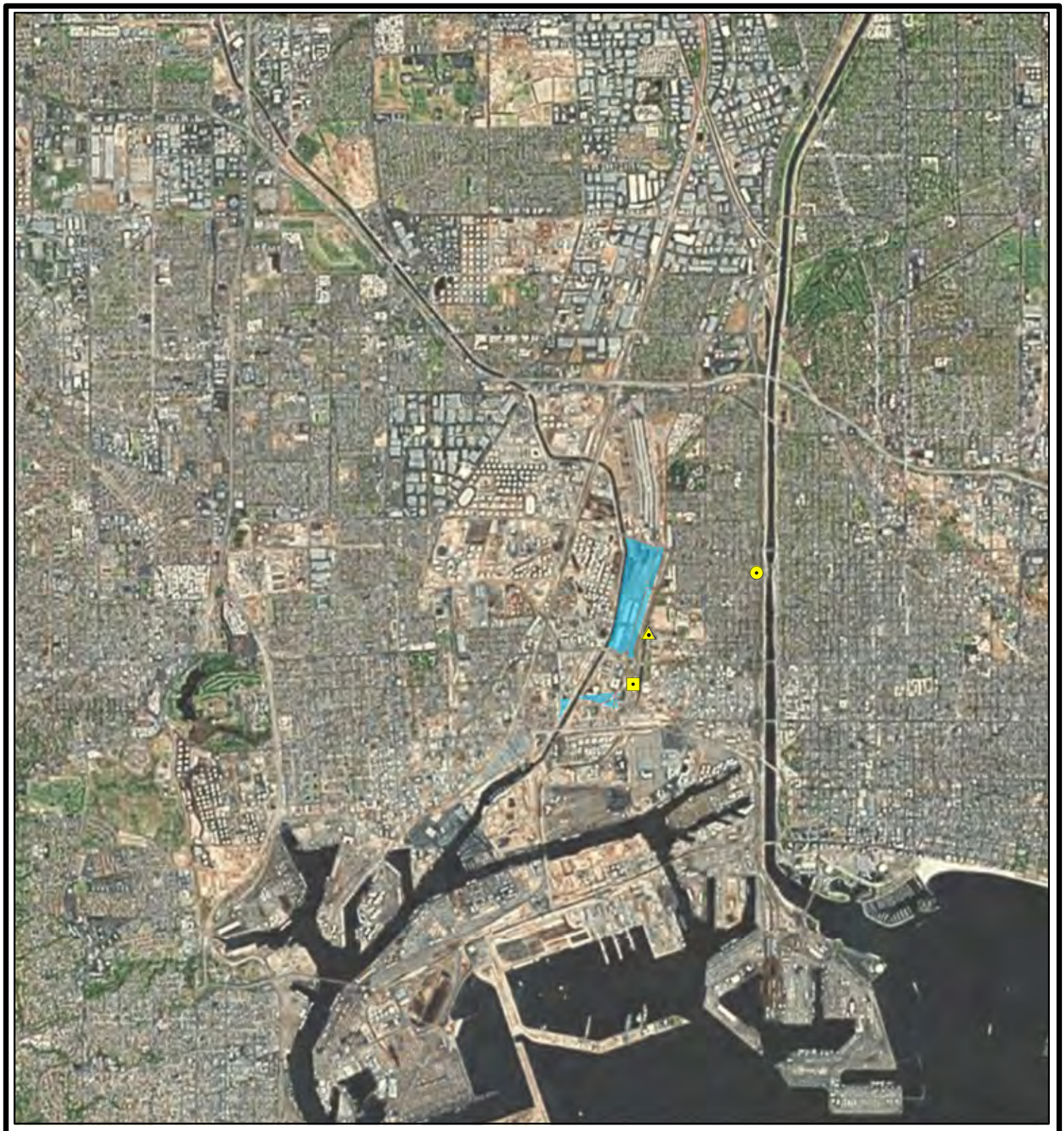
0 0.5 1 Kilometers

Figure C3.7-2
Baseline

Maximum Exposed Individual for Cancer Risk

DRAFT

ENVIRON



Legend

- Residential MEI - CEQA Baseline
- Occupational and Recreational MEI - CEQA Baseline
- Sensitive and Student MEI - CEQA Baseline
- Site

N

0 0.5 1 Miles

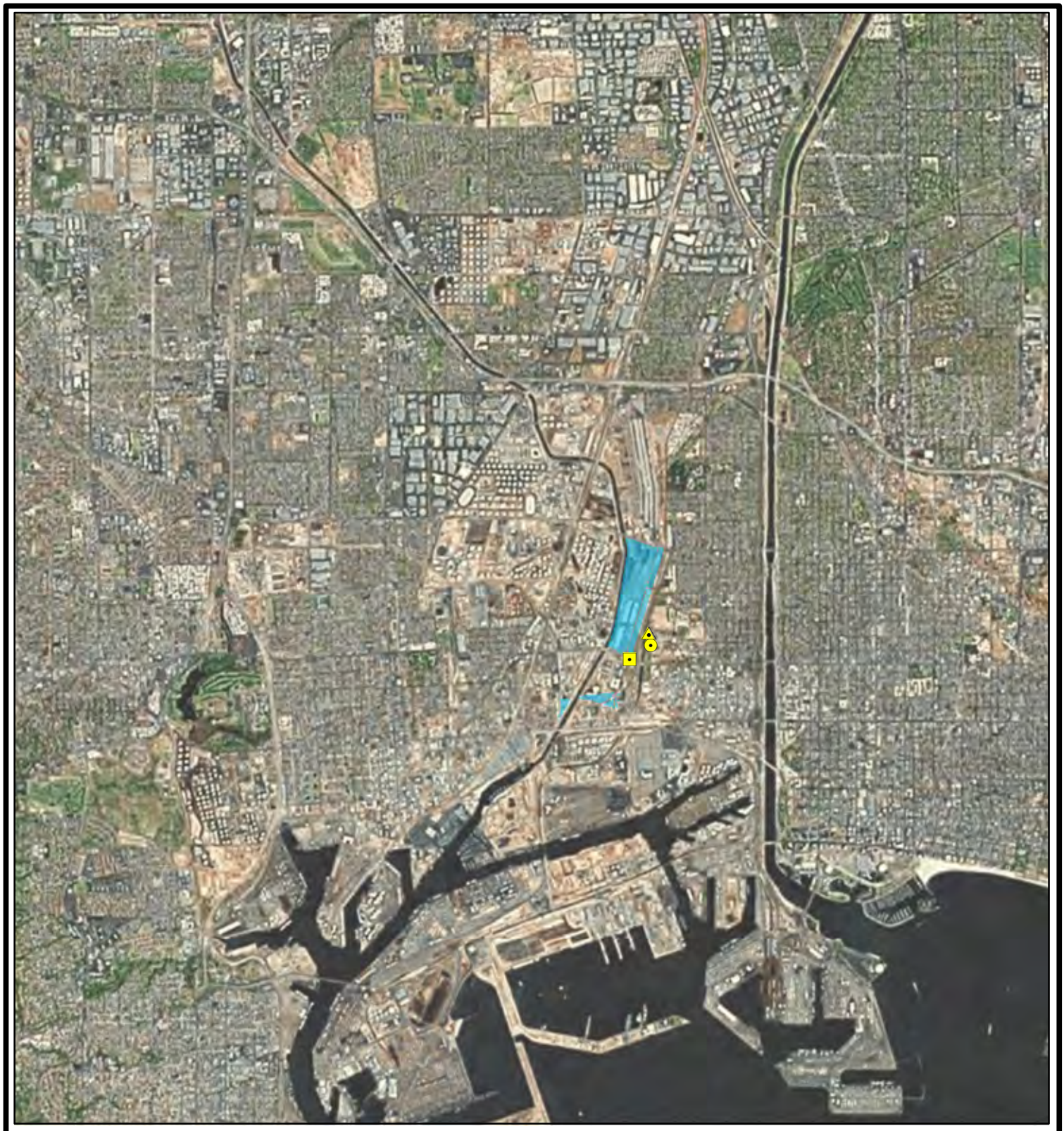
0 0.5 1 Kilometers

Figure C3.7-3
Baseline

Maximum Exposed Individual for Chronic HI

DRAFT

ENVIRON



Legend

- Residential MEI - CEQA Baseline
- Occupational and Recreational MEI - CEQA Baseline
- Sensitive and Student MEI - CEQA Baseline
- Site

N

0 0.5 1 Miles

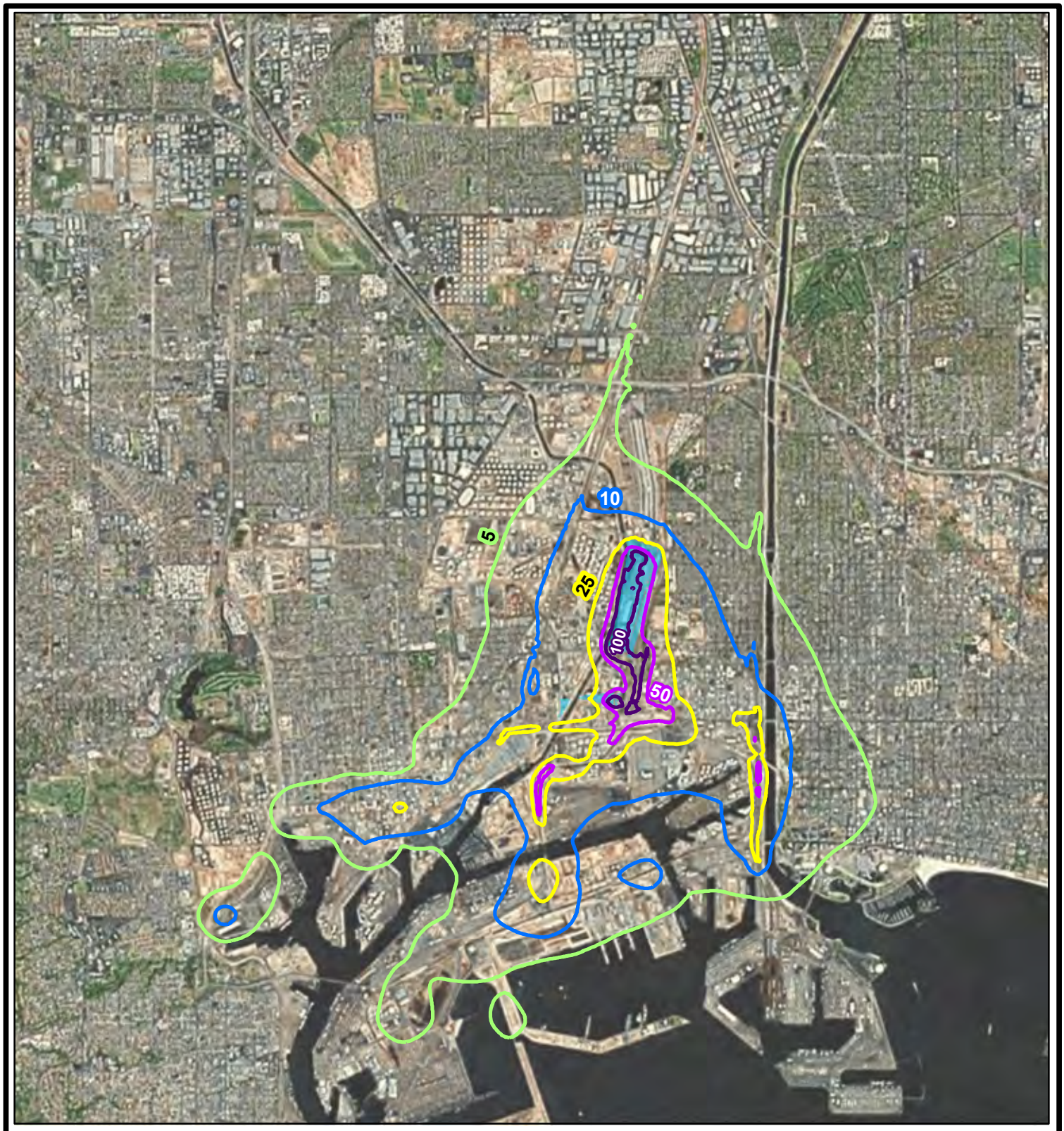
0 0.5 1 Kilometers

Figure C3.7-4
Baseline

Maximum Exposed Individual for Acute HI

DRAFT

ENVIRON



Legend

- 5
- 10
- 25
- 50
- 100
- Site

N

0 0.5 1 Miles

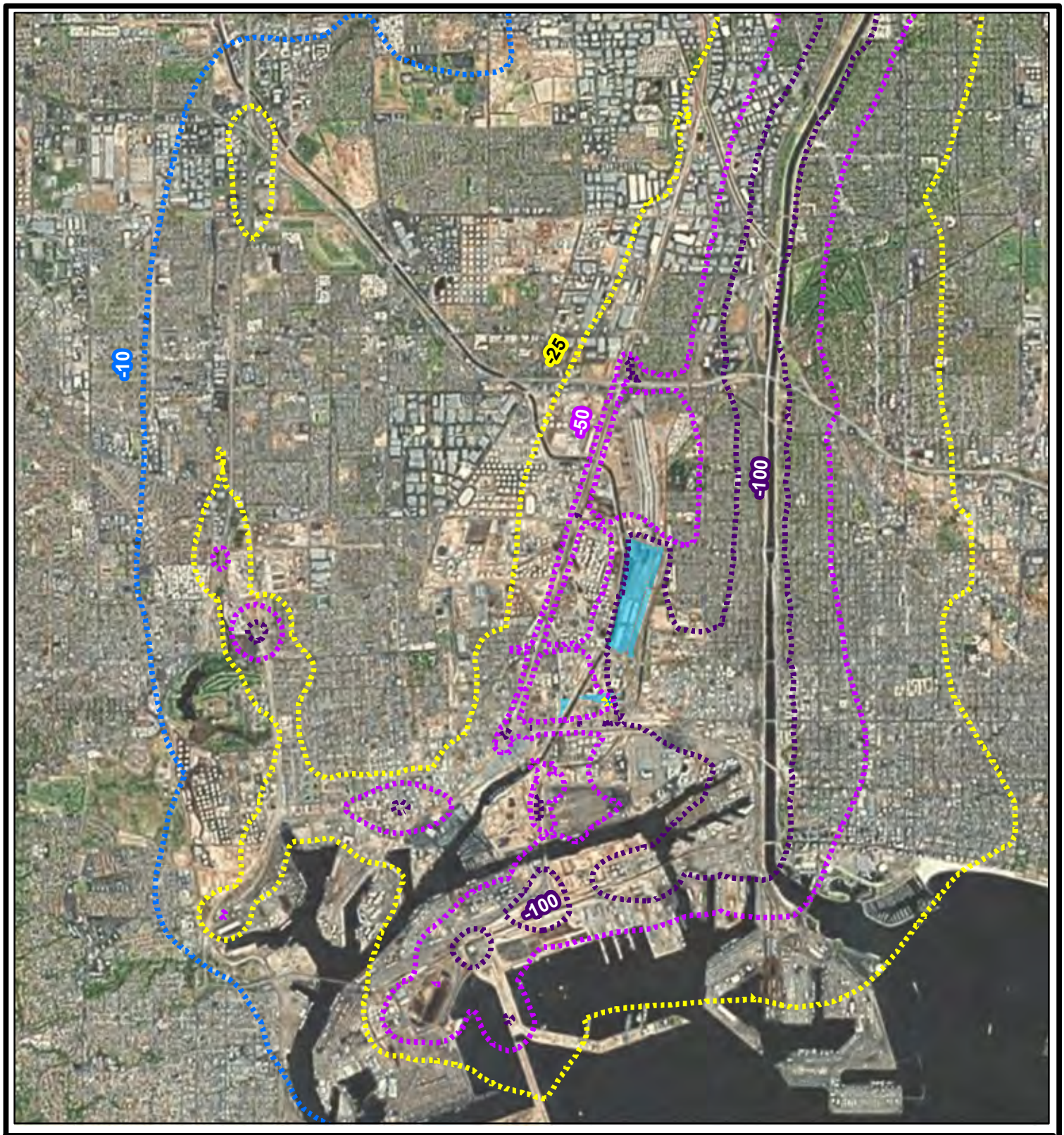
0 0.5 1 Kilometers

Figure C3.7-5
Unmitigated Proposed Project

Residential Individual Lifetime Cancer Risk (per Million)

DRAFT

ENVIRON



Legend

- -10
- -25
- -50
- -100
- Site

N

0 0.5 1 Miles

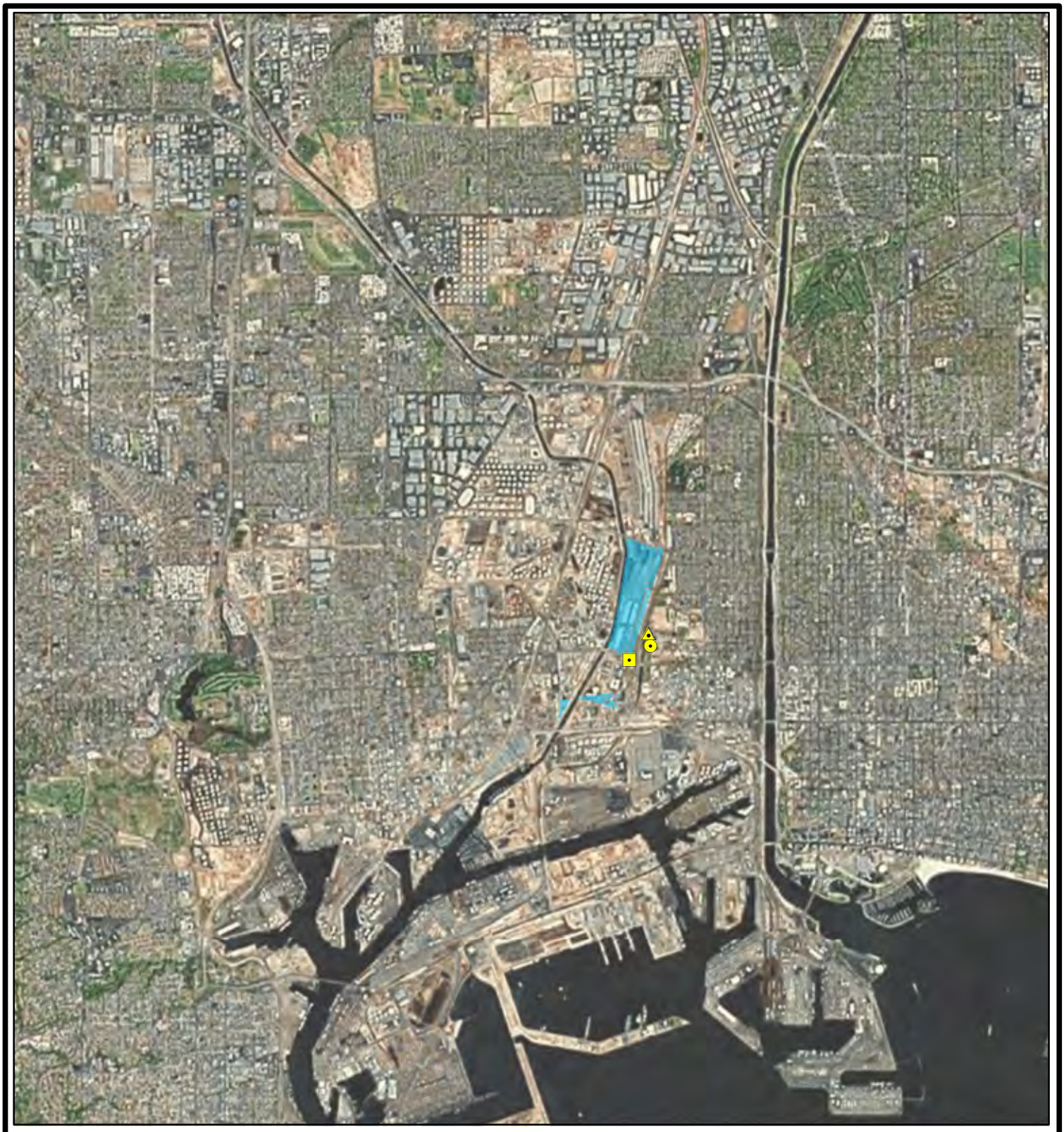
0 0.5 1 Kilometers

Figure C3.7-6
Unmitigated Proposed Project minus Baseline

Residential Individual Lifetime Cancer Risk (per Million)

DRAFT

ENVIRON



Legend

- Residential MEI - Proposed Project[1]
- Occupational and Recreational MEI - Proposed Project[2]
- Sensitive and Student MEI - Proposed Project[3]
- Site

N

0 0.5 1 Miles

0 0.5 1 Kilometers

Notes

1. Also location of the Residential CEQA increment value in Table E3-7-1.
2. Also location of the Occupational and Recreational CEQA increment values in Table E3-7-1.
3. Also location of the Sensitive and Student CEQA increment values in Table E3-7-1.

Figure C3.7-7
Unmitigated Proposed Project

Maximum Exposed Individual for Cancer Risk

DRAFT

ENVIRON

Legend

- Residential MEI - Proposed Project[1]
- Occupational and Recreational MEI - Proposed Project
- Sensitive and Student MEI - Proposed Project[2]
- Occupational and Recreational MEI - CEQA Increment
- Site

N

0 0.5 1 Miles

0 0.5 1 Kilometers

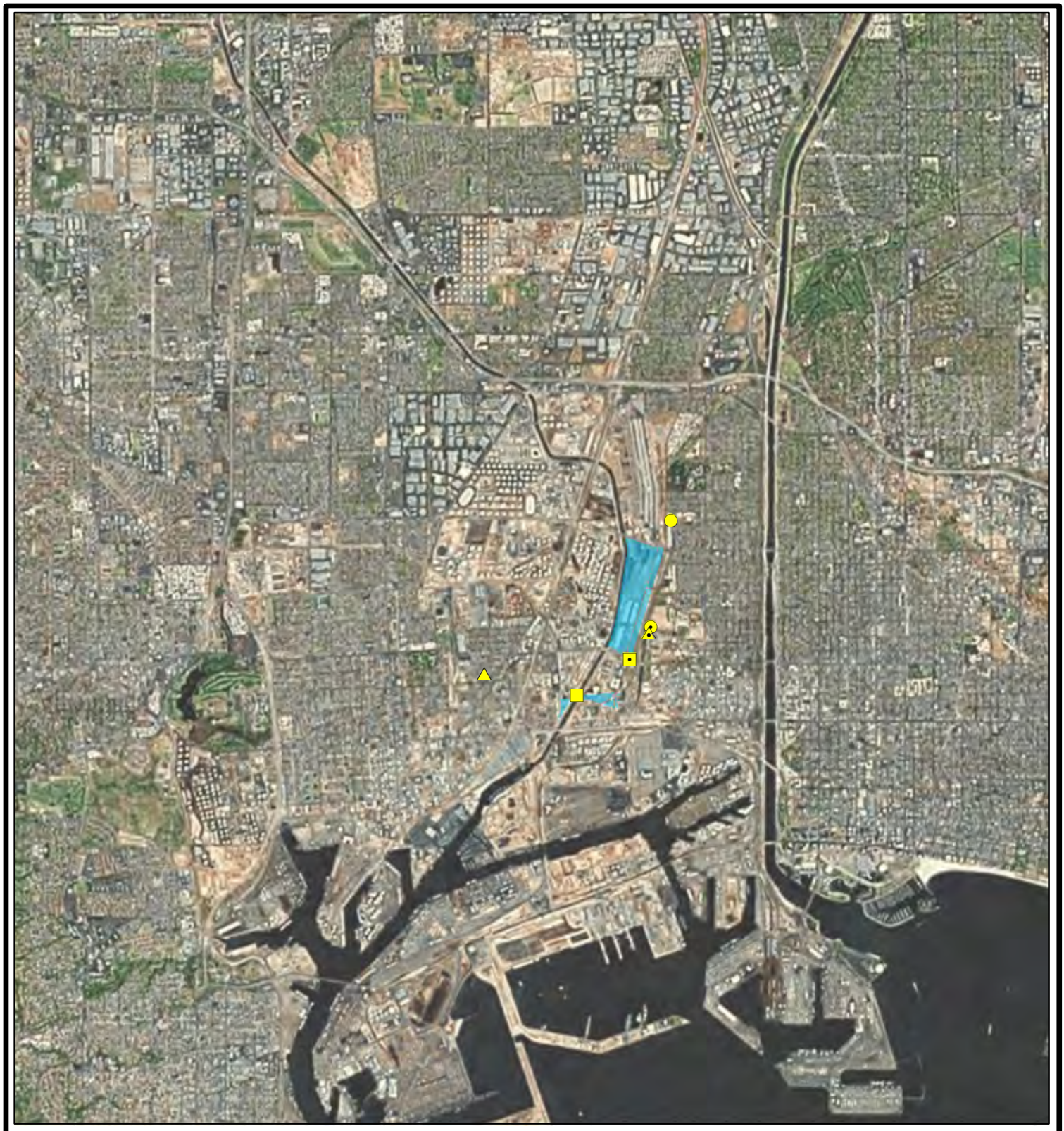
Notes
1. Also location of the Residential CEQA increment value in Table E3-7-1.
2. Also location of the Sensitive and Student CEQA increment values in Table E3-7-1.

Figure C3.7-8
Unmitigated Proposed Project

Maximum Exposed Individual for Chronic HI

DRAFT

ENVIRON



Legend

- Residential MEI - Proposed Project
- Occupational and Recreational MEI - Proposed Project
- Sensitive and Student MEI - Proposed Project
- Residential MEI - CEQA Increment
- Occupational and Recreational MEI - CEQA Increment
- Sensitive and Student MEI - CEQA Increment
- Site

N

0 0.5 1 Miles

0 0.5 1 Kilometers

Figure C3.7-9
Unmitigated Proposed Project

Maximum Exposed Individual for Acute HI

DRAFT

ENVIRON

proximity of the receptor to the on- and off-ramps of Highway 1 (the Pacific Coast Highway) is the dominant contributor to these health risk values. By contrast, the greatest contributor to the chronic hazard index at the maximum residential receptor is a combination of emissions from SCIG construction, Hostler emissions, and SCIG onsite trucks. The greatest contributor to the acute hazard index at the maximum residential receptor is SCIG construction emissions. Locomotives contribute between approximately 1-3% of each health effect endpoint at the maximum residential receptor.

Table C3-7-2. Source Contributions at the Residential and Occupational MEIs for the Unmitigated Proposed Project.

Emission Source	Maximum Residential Receptor			Maximum Occupational Receptor		
	Cancer Risk	Chronic Hazard Index	Acute Hazard Index	Cancer Risk	Chronic Hazard Index	Acute Hazard Index
SCIG Offsite Trucks	53.8%	7.5%	1.4%	72.2%	4.4%	1.6%
SCIG Onsite Trucks	35.5%	17.2%	7.0%	15.6%	1.6%	2.5%
SCIG Onsite Locomotives	1.7%	1.8%	2.6%	1.0%	7.0%	4.1%
SCIG Construction	1.7%	35.0%	55.8%	7.6%	81.4%	46.7%
Tenant CHE	1.7%	5.5%	7.3%	1.1%	0.5%	13.2%
Tenant Offsite Trucks	1.6%	2.6%	4.7%	1.2%	0.9%	4.0%
Hostler	1.5%	24.2%	6.8%	0.2%	0.3%	2.3%
SCIG Offsite Locomotives	1.0%	2.5%	0.7%	0.4%	2.2%	0.7%
Tenant Onsite Trucks	0.7%	0.5%	5.4%	0.3%	<0.1%	17.8%
SCIG CHE/TRU	0.3%	0.2%	1.3%	<0.1%	<0.1%	0.4%
Tenant Construction	<0.1%	2.4%	1.3%	<0.1%	1.3%	6.1%
Emergency Generator	<0.1%	<0.1%	4.3%	<0.1%	<0.1%	0.4%
Three Rivers Underpass	<0.1%	<0.1%	1.1%	<0.1%	<0.1%	0.1%
SCIG Onsite Gasoline Vehicles	<0.1%	0.1%	<0.1%	<0.1%	<0.1%	<0.1%
Tenant Onsite Locomotives	<0.1%	<0.1%	<0.1%	<0.1%	<0.1%	<0.1%
Onsite Refueling Trucks	<0.1%	<0.1%	<0.1%	0.2%	<0.1%	<0.1%
SCIG Offsite Gasoline Vehicles	<0.1%	0.3%	<0.1%	<0.1%	0.1%	<0.1%
Tenant Offsite Gasoline Vehicles	<0.1%	0.2%	0.1%	<0.1%	0.1%	<0.1%
Tenant Onsite Gasoline Vehicles	<0.1%	<0.1%	<0.1%	<0.1%	<0.1%	<0.1%

At the maximum occupational receptor, the greatest contributors to the cancer risk are SCIG offsite and onsite trucks. The greatest contributor to the chronic hazard index is SCIG construction. The greatest contributors to the acute hazard index are SCIG construction, relocated tenant onsite trucks, and relocated tenant cargo handling equipment (CHE) emissions. SCIG onsite and offsite locomotives contribute less than

1.5% of cancer risk while SCIG onsite locomotives contribute 7% to the chronic hazard index and over 4% to the acute hazard index at the maximum occupational receptor

Table C3-7-3 presents the contributions from each TAC to the maximum health effects values for the Unmitigated Proposed Project. Because DPM is a surrogate for all diesel ICE emissions for cancer risk calculations, DPM is the maximum contributor (over 97 percent) to these health risk values at the residential and occupational receptor. DPM contributes nearly 99 percent of the chronic hazard index at the occupational receptor while DPM and chlorine together contribute nearly 90 percent of the chronic hazard index at the residential receptor. The table shows that the greatest acute hazard index contributor is formaldehyde at both the maximum residential and occupational receptors.

Because the calculation of cancer burden is only meaningful where there is an increased (positive) risk of cancer from project emissions, no calculations of cancer burden were completed for the Unmitigated Proposed Project given that all cancer risk increments for the maximally-exposed residential receptor were negative

Table C3-7-3. TAC Contributions at the Residential and Occupational MEIs for the Unmitigated Proposed Project.

Pollutant	Maximum Residential Receptor			Maximum Occupational Receptor		
	Cancer Risk	Chronic Hazard Index[a]	Acute Hazard Index[a]	Cancer Risk	Chronic Hazard Index[a]	Acute Hazard Index[a]
DPM	97.6%	72.4%	0.0%	99.0%	98.7%	0.0%
Hexavalent Chromium	1.0%	<0.1%	0.0%	0.8%	<0.1%	0.0%
Formaldehyde	0.8%	9.6%	93.4%	0.1%	0.1%	93.3%
Benzene	0.5%	0.2%	0.5%	<0.1%	<0.1%	0.5%
Nickel	<0.1%	0.8%	<0.1%	<0.1%	0.1%	<0.1%
1,3-Butadiene	<0.1%	<0.1%	0.0%	<0.1%	<0.1%	0.0%
Acetaldehyde	<0.1%	<0.1%	5.1%	<0.1%	<0.1%	5.3%
Ethylbenzene	<0.1%	<0.1%	0.0%	<0.1%	<0.1%	0.0%
Arsenic	<0.1%	<0.1%	0.2%	<0.1%	<0.1%	0.2%
Naphthalene	<0.1%	<0.1%	0.0%	<0.1%	<0.1%	0.0%
Lead	<0.1%	0.0%	0.0%	<0.1%	0.0%	0.0%
Cadmium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Chlorine	0.0%	15.9%	<0.1%	0.0%	0.9%	<0.1%
Manganese	0.0%	0.8%	0.0%	0.0%	0.2%	0.0%
Acrolein (2-Propenal)	0.0%	<0.1%	0.1%	0.0%	<0.1%	<0.1%
Propylene	0.0%	<0.1%	0.0%	0.0%	<0.1%	0.0%
Toluene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
Isomers Of Xylene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
M-Xylene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
O-Xylene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
N-Hexane	0.0%	<0.1%	0.0%	0.0%	<0.1%	0.0%
Ammonia	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
Styrene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
Methyl Alcohol	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
Mercury	0.0%	0.0%	0.3%	0.0%	0.0%	0.4%
Methyl Ethyl Ketone (MEK) (2-Butanone)	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
Copper	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%

Pollutant	Maximum Residential Receptor			Maximum Occupational Receptor		
	Cancer Risk	Chronic Hazard Index[a]	Acute Hazard Index[a]	Cancer Risk	Chronic Hazard Index[a]	Acute Hazard Index[a]
Vanadium (Fume Or Dust)	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
Vanadium	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
P-Xylene	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
Antimony	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Bromine	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Calcium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Carbon Elemental	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Carbon Organic	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Carbonate Ion	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Chromium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Cobalt	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Iron	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Nitrates	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Other	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Phosphorous	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Potassium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Selenium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Sulfates	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Unidentified	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Unknown Pm	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Zinc	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%

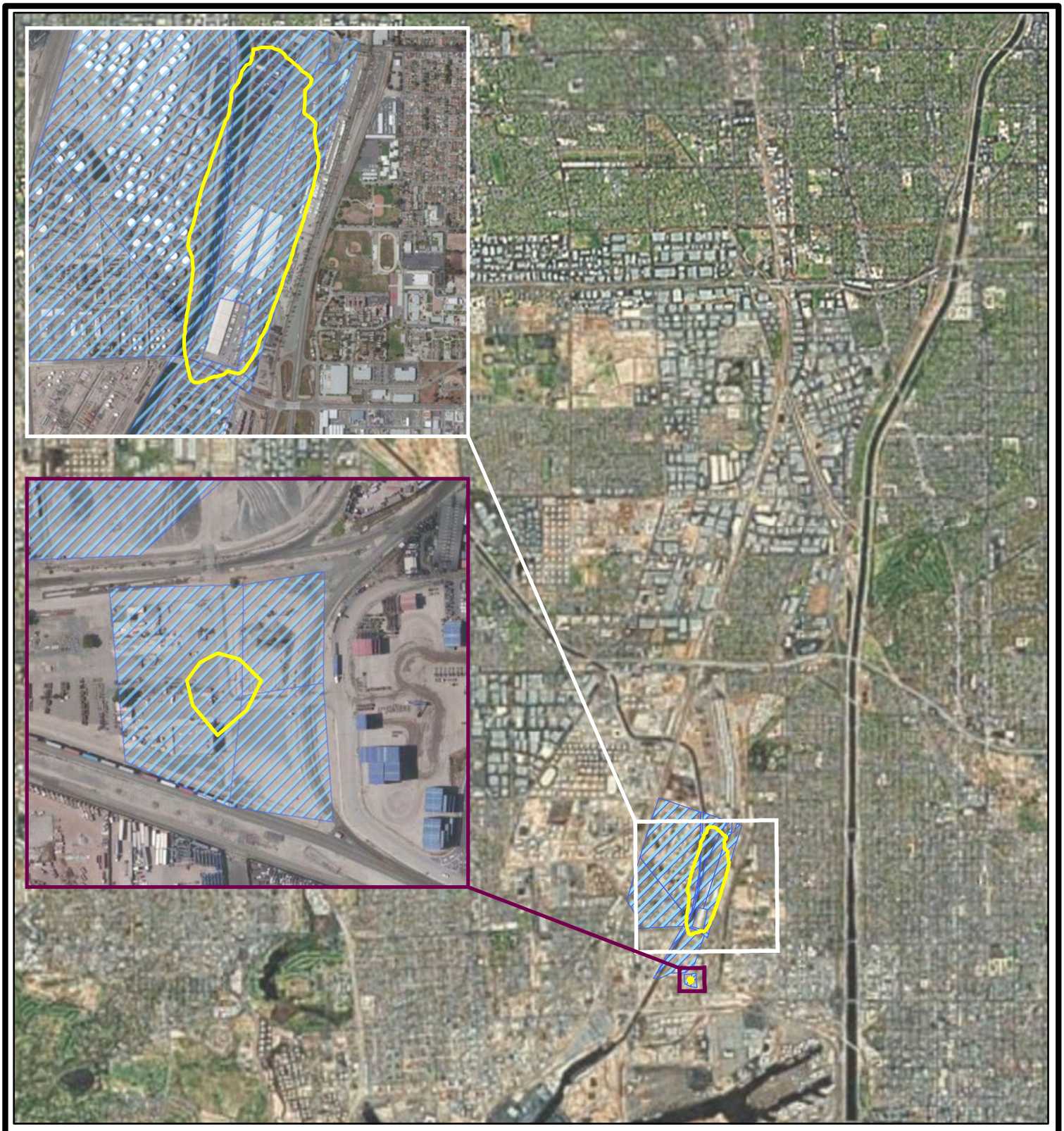
a The chemical contributions for the chronic and acute hazard indices include all chemicals regardless of the target organs they affect. As a result, the contributions may add to greater than 100 percent because not all chemicals affect the same target organ.

b For diesel internal combustion engines, only DPM emissions were evaluated for cancer risk and chronic hazard indices, because DPM is a surrogate for the combined health effects associated with exposure to diesel exhaust emissions. For all other emissions (alternative fuel engines, tire and brake wear), emissions of the 47 other toxic air contaminants were evaluated for cancer and chronic hazard indices. For the acute hazard indices, DPM was not evaluated; rather, emissions of the 47 other toxic air contaminants were evaluated for all emission sources (including diesel ICEs).

7.1.1 $PM_{2.5}$ Effects

As described in Chapter 3-2 (Impact AQ-4), the results of ambient air dispersion modeling indicated that operation of the Unmitigated Proposed Project (project minus Baseline) would result in off-site 24-hour $PM_{2.5}$ concentrations that exceed the SCAQMD significance threshold of 2.5 $\mu g/m^3$ Because of this exceedance, incremental $PM_{2.5}$ concentrations from the project's operations meet the Port's criteria for calculating mortality and morbidity attributable to PM (POLA, 2011), and are discussed here as further elaboration of a $PM_{2.5}$ significance finding identified in Chapter 3-2. This discussion does not identify a new impact, but provides additional information on the potential impact of $PM_{2.5}$ levels identified in AQ-4.

In accordance with the Port's methodology, census blocks lying partially or completely within the project increment 24-h $PM_{2.5}$ $\mu g/m^3$ concentration isopleth were identified (see Figure C3.7-27). All census blocks were found to be located in industrialized areas, and aerial images did not show any residential structures. On the ground observations established that these census blocks are used solely for industrial purposes i.e., that there is no residential use. Because no residential populations inhabit the impacted census



Legend

- 24-Hour $PM_{2.5}$ Concentration ≥ 2.5 µg/m^3
- Census Block

N

0 0.5 1 Miles

0 0.5 1 Kilometers

Figure C3.7-27
Unmitigated Proposed Project minus Baseline

Mortality and Morbidity Applicability

DRAFT

ENVIRON

blocks, the project increment is not expected to have an impact on PM-attributable morbidity or mortality. No calculations of mortality and morbidity were completed.

7.2 Mitigated Proposed Project Health Impacts

This HRA evaluated the effect on health risks resulting from the implementation of the air quality mitigation measures identified in Section 3.2 of the EIR. A summary of the mitigation measure quantified in this HRA for project construction is as follows:

MM AQ-1: The Mitigated Proposed Project assumes that the Port guidelines for reducing emissions from construction equipment operating at the Port are followed; it is otherwise equivalent to the Unmitigated Proposed Project.

Table C3-7-4 presents a summary of the maximum health impacts that would occur for each receptor type with construction and operation of the Mitigated Proposed Project. The table also shows the maximum health impacts from the Baseline, as well as the CEQA increment (Mitigated Proposed Project minus Baseline). Because the results in Table C3-7-4 represent the maximum impacts predicted for each receptor type, the impacts at all other receptors would be less than these values.

The mitigation measure would reduce Project maximum cancer risks by about 1 to 35 percent, depending on the receptor location. Chronic hazard indexes would be reduced by about 20 to 36 percent. Acute hazard indices would be reduced by about 9 to 13 percent.

Table C3-7-4. Maximum Health Impacts Associated with the Mitigated Proposed Project.

Health Impact	Receptor Type	Maximum Predicted Impact			Significance Threshold
		Mitigated Project	CEQA Baseline	CEQA Increment	
Cancer Risk	Residential	48 x 10^{-6} (48 in a million)	568 x 10^{-6} (568 in a million)	-161 x 10^{-6} (-161 in a million)	10 x 10^{-6} (10 in a million)
	Occupational	39 x 10^{-6} (39 in a million)	215 x 10^{-6} (215 in a million)	-116 x 10-6 (-116 in a million)	
	Sensitive	40 x 10^{-6} (40 in a million)	220 x 10^{-6} (220 in a million)	-180 x 10^{-6} (-180 in a million)	
	Student	1.7 x 10^{-6} (1.7 in a million)	4.7 x 10^{-6} (4.7 in a million)	-2.9 x 10^{-6} (-2.9 in a million)	
	Recreational	60 x 10^{-6} (60 in a million)	329 x 10^{-6} (329 in a million)	-177 x 10^{-6} (-177 in a million)	
Chronic Hazard Index	Residential	0.07	0.36	-0.07	1.0
	Occupational	0.30	0.69	0.03	
	Sensitive	0.08	0.16	-0.08	
	Student	0.08	0.16	-0.08	
	Recreational	0.30	0.69	0.03	
Acute Hazard Index	Residential	0.17	0.29	-0.071	1.0
	Occupational	0.60	0.79	0.09	
	Sensitive	0.18	0.27	-0.09	
	Student	0.18	0.27	-0.09	
	Recreational	0.60	0.79	0.09	

[a] Exceedances of the significance thresholds are in bold. The significance thresholds apply to the CEQA increments only.

b The maximum increments might not occur at the same receptor locations as the maximum impacts. This means that the increments cannot necessarily be determined by subtracting the CEQA Baseline impact from the project impact. Rather, the subtraction must be done at each receptor, for all modeled receptors, and the maximum result selected.

c The CEQA Increment represents Project minus CEQA Baseline.

d When the maximum increment for a receptor type is negative, the maximum increment displayed is the increment at the maximum project receptor location.

e Data represent the receptor locations with the maximum impacts or increments. The impacts or increments at all other modeled receptors would be less than these values for each receptor type.

f The cancer risk values reported in this table for the residential receptor are based on the 80th percentile breathing rate. The risks associated with the 65th percentile (average) breathing rate will be less than these values. The risks associated with the 95th percentile (high end) breathing rate are 62 x 10-6 for the Project impact, 740 x 10-6 for the CEQA Baseline impact, and -209 x 10-6 for the CEQA increment.

g The Mitigated Proposed Project assumes that the Port guidelines for reducing emissions from construction equipment operating at the Port are followed; it is otherwise equivalent to the Unmitigated Proposed Project.

The data in Table C3-7-4 show that the CEQA cancer risk increment at the location of the Mitigated Proposed Project MEI is predicated to be -161 in a million (-161 x 10^{-6}), at a residential receptor. This risk value, as well as the risk value at all residential receptors, is below the significance threshold of 10 in a million. The CEQA risk increments would be below the CEQA significance threshold at all receptors, including occupational, sensitive, student, and recreational.

The maximum chronic hazard index increments are predicted to be less than the CEQA significance threshold of 1.0 at all receptors.

The maximum acute hazard index increments are predicted to be less than the CEQA significance threshold of 1.0 at each receptor type.

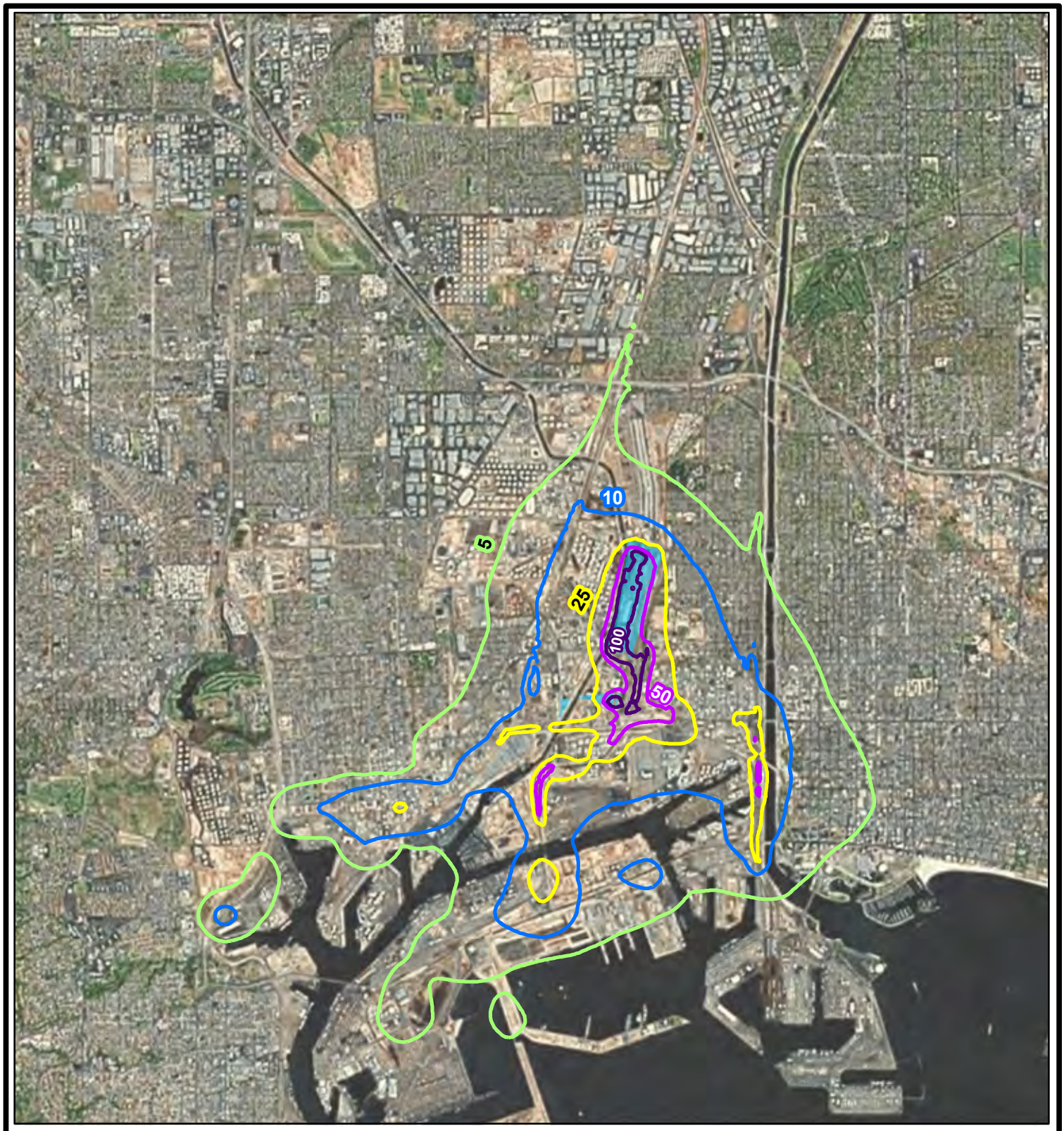
Figures C3.7-10 and C3.7-11 show the cancer risk isopleths associated with the Mitigated Proposed Project and Mitigated Proposed Project minus Baseline residential individual lifetime cancer risk (per million), respectively.

Figures C3.7-12, C3.7-13, and C3.7-14 show the maximum receptor locations for the Mitigated Proposed Project for cancer risk, chronic HI, and acute HI, respectively. It should be noted that the residential, occupational, and recreational MEIs are not necessarily located directly on existing homes, workplaces, or recreational facilities; rather, they are located in areas that contain these land use types.

Table C3-7-5 presents the contributions from each emission source to the maximum health effects impacts for the Mitigated Proposed Project. At the maximum residential receptor, the greatest contributors to cancer risk are SCIG offsite and onsite trucks. The greatest contributors to the chronic hazard index are SCIG offsite trucks, Hostler emissions, SCIG onsite trucks, and SCIG construction. . The greatest contributor to the acute hazard index is SCIG construction. Locomotives contribute between 3-5% to each health effect endpoint at the maximum residential receptor.

Table C3-7-5. Source Contributions at the Residential and Occupational MEIs for the Mitigated Proposed Project.

Emission Source	Maximum Residential Receptor			Maximum Occupational Receptor		
	Cancer Risk	Chronic Hazard Index	Acute Hazard Index	Cancer Risk	Chronic Hazard Index	Acute Hazard Index
SCIG Offsite Trucks	54.5%	26.7%	1.6%	76.0%	17.6%	2.0%
SCIG Onsite Trucks	35.9%	17.2%	8.0%	16.4%	2.3%	3.0%
SCIG Onsite Locomotives	1.8%	1.9%	2.9%	1.1%	2.9%	4.8%
Tenant CHE	1.7%	5.3%	8.5%	1.2%	57.8%	15.7%
Tenant Offsite Trucks	1.6%	6.5%	5.4%	1.3%	3.3%	4.7%
Hostler	1.5%	24.9%	7.9%	0.2%	0.9%	2.7%
SCIG Offsite Locomotives	1.1%	3.1%	0.8%	0.4%	1.9%	0.9%
Tenant Onsite Trucks	0.7%	0.4%	6.3%	0.3%	6.5%	21.2%
SCIG Construction	0.6%	10.4%	49.2%	2.7%	4.1%	40.0%
SCIG CHE/TRU	0.3%	0.2%	1.5%	<0.1%	<0.1%	0.4%
Tenant Construction	<0.1%	2.3%	1.3%	<0.1%	1.7%	3.8%
Emergency Generator	<0.1%	<0.1%	5.0%	<0.1%	<0.1%	0.5%
Three Rivers Underpass	<0.1%	<0.1%	1.2%	<0.1%	<0.1%	0.1%
SCIG Onsite Gasoline Vehicles	<0.1%	0.1%	<0.1%	<0.1%	<0.1%	<0.1%
Tenant Onsite Locomotives	<0.1%	<0.1%	<0.1%	<0.1%	<0.1%	<0.1%
Onsite Refueling Trucks	<0.1%	<0.1%	<0.1%	0.2%	<0.1%	<0.1%
SCIG Offsite Gasoline Vehicles	<0.1%	0.6%	<0.1%	<0.1%	0.1%	<0.1%
Tenant Offsite Gasoline Vehicles	<0.1%	0.4%	0.2%	<0.1%	0.3%	<0.1%
Tenant Onsite Gasoline Vehicles	<0.1%	<0.1%	<0.1%	<0.1%	0.4%	<0.1%



Legend

- 5
- 10
- 25
- 50
- 100
- Site

N

0 0.5 1 Miles

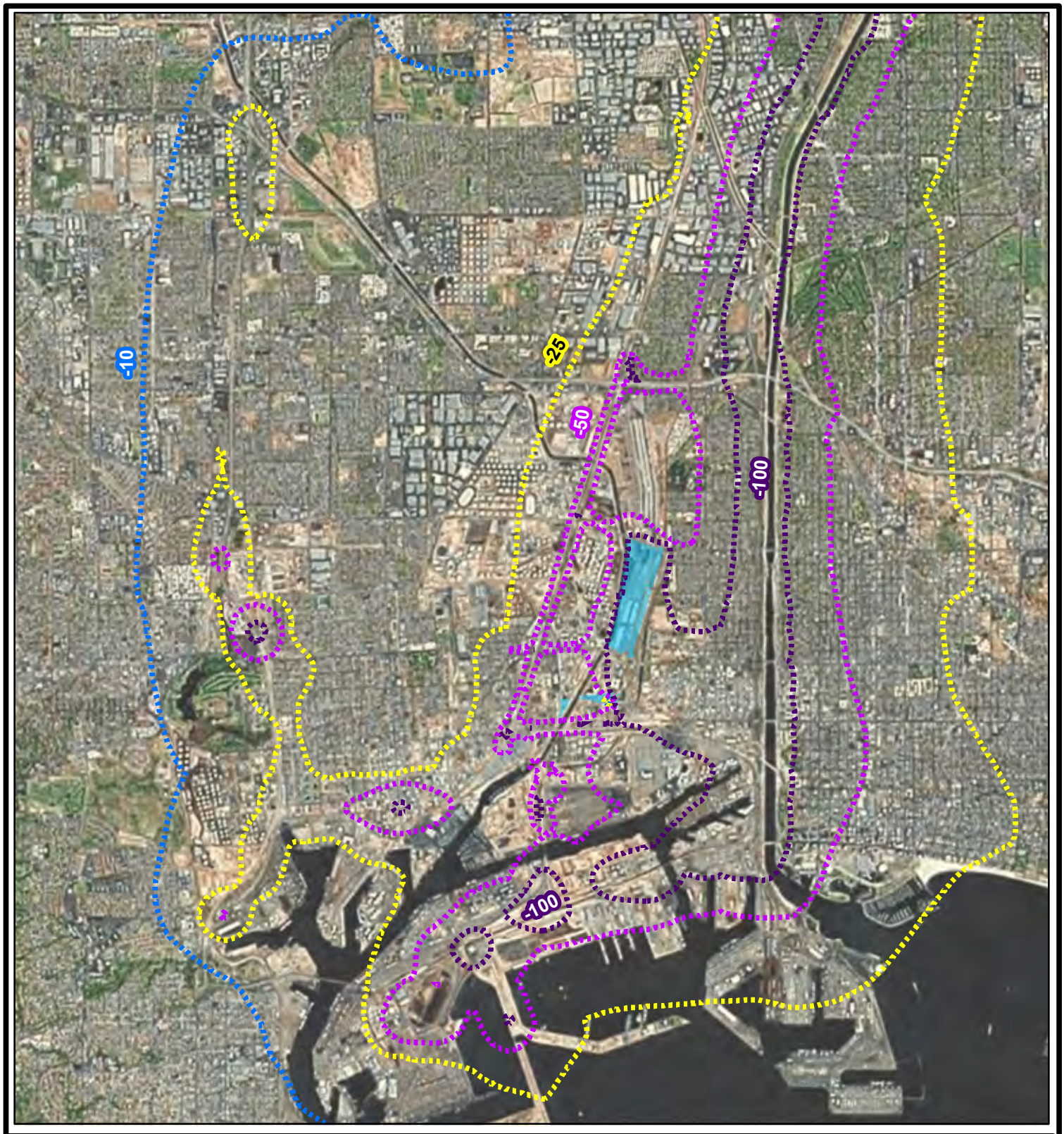
0 0.5 1 Kilometers

Figure C3.7-10
Mitigated Proposed Project

Residential Individual Lifetime Cancer Risk (per Million)

DRAFT

ENVIRON



Legend

- -10
- -25
- -50
- -100
- Site

N

0 0.5 1 Miles

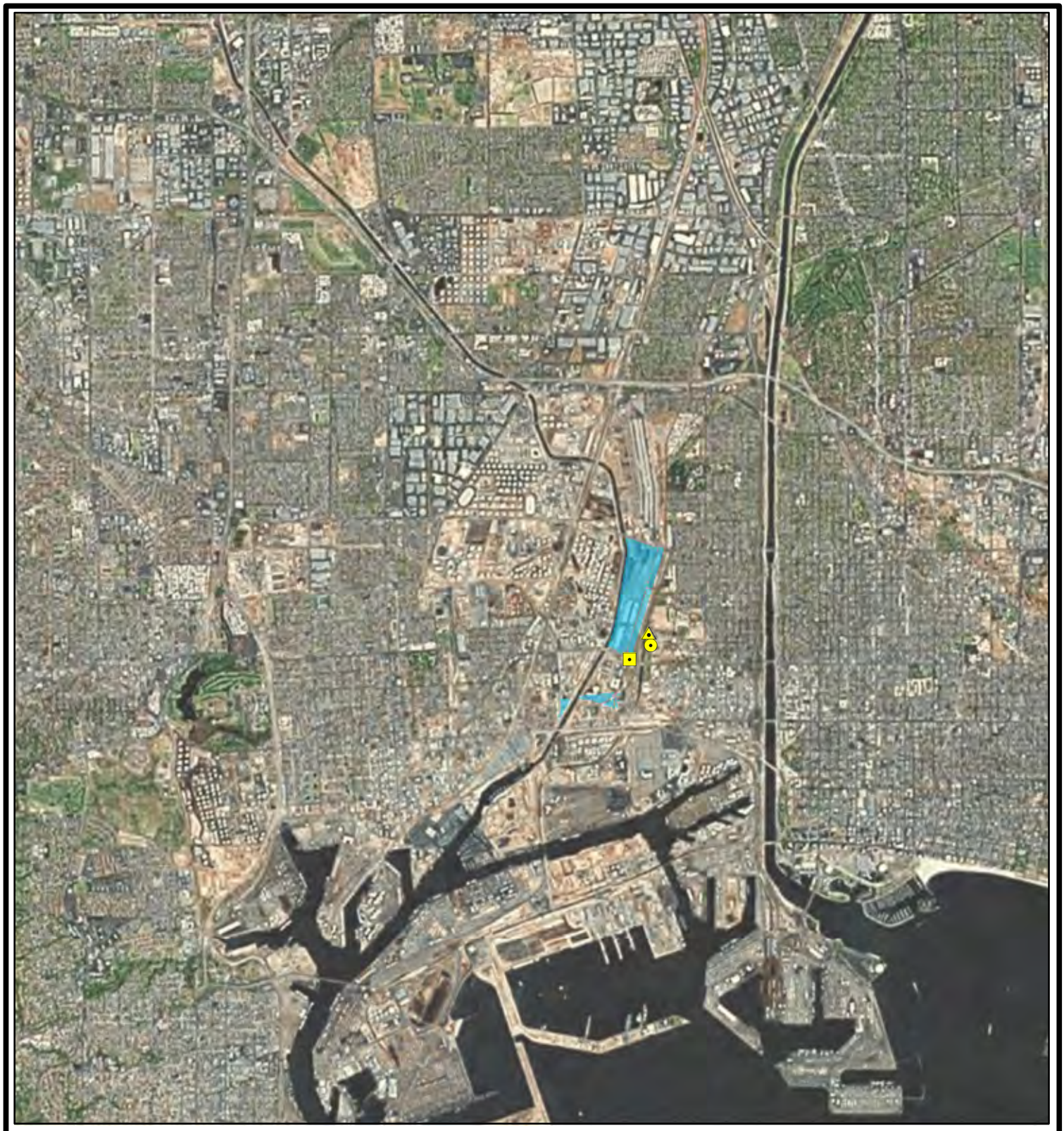
0 0.5 1 Kilometers

Figure C3.7-11
Mitigated Proposed Project minus Baseline

Residential Individual Lifetime Cancer Risk (per Million)

DRAFT

ENVIRON



Legend

- Residential MEI - Mitigated Project[1]
- Occupational and Recreational MEI - Mitigated Project[2]
- Sensitive and Student MEI - Mitigated Project[3]
- Site

N

0 0.5 1 Miles

0 0.5 1 Kilometers

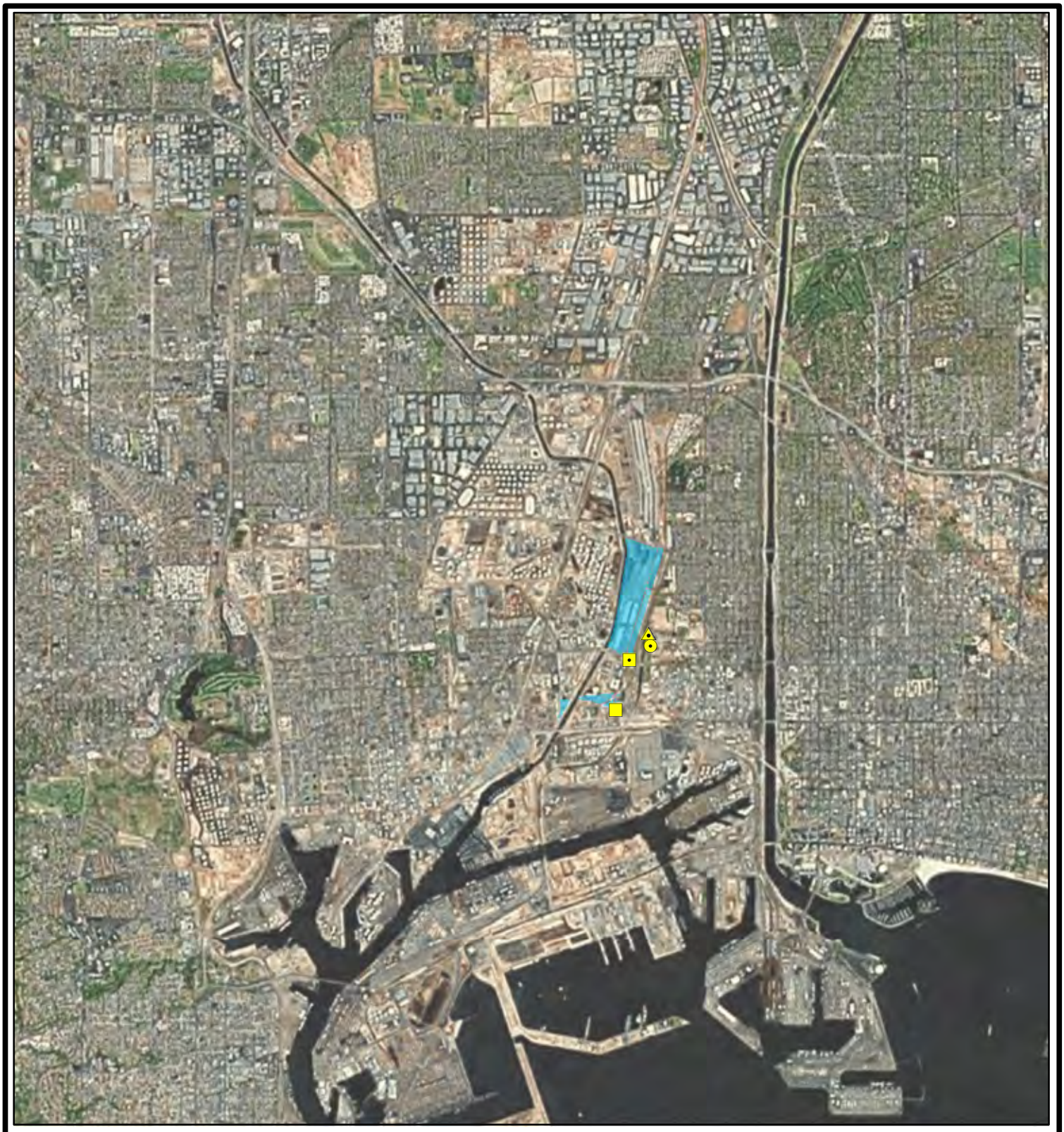
Notes
1. Also location of the Residential CEQA increment value in Table E3-7-4.
2. Also location of the Occupational and Recreational CEQA increment values in Table E3-7-4.
3. Also location of the Sensitive and Student CEQA increment values in Table E3-7-4.

Figure C3.7-12
Mitigated Proposed Project

Maximum Exposed Individual for Cancer Risk

DRAFT

ENVIRON



Legend

- Residential MEI - Mitigated Project[1]
- Occupational and Recreational MEI - Mitigated Project
- Sensitive and Student MEI - Mitigated Project[2]
- Occupational and Recreational MEI - CEQA Increment
- Site

N

0 0.5 1 Miles

0 0.5 1 Kilometers

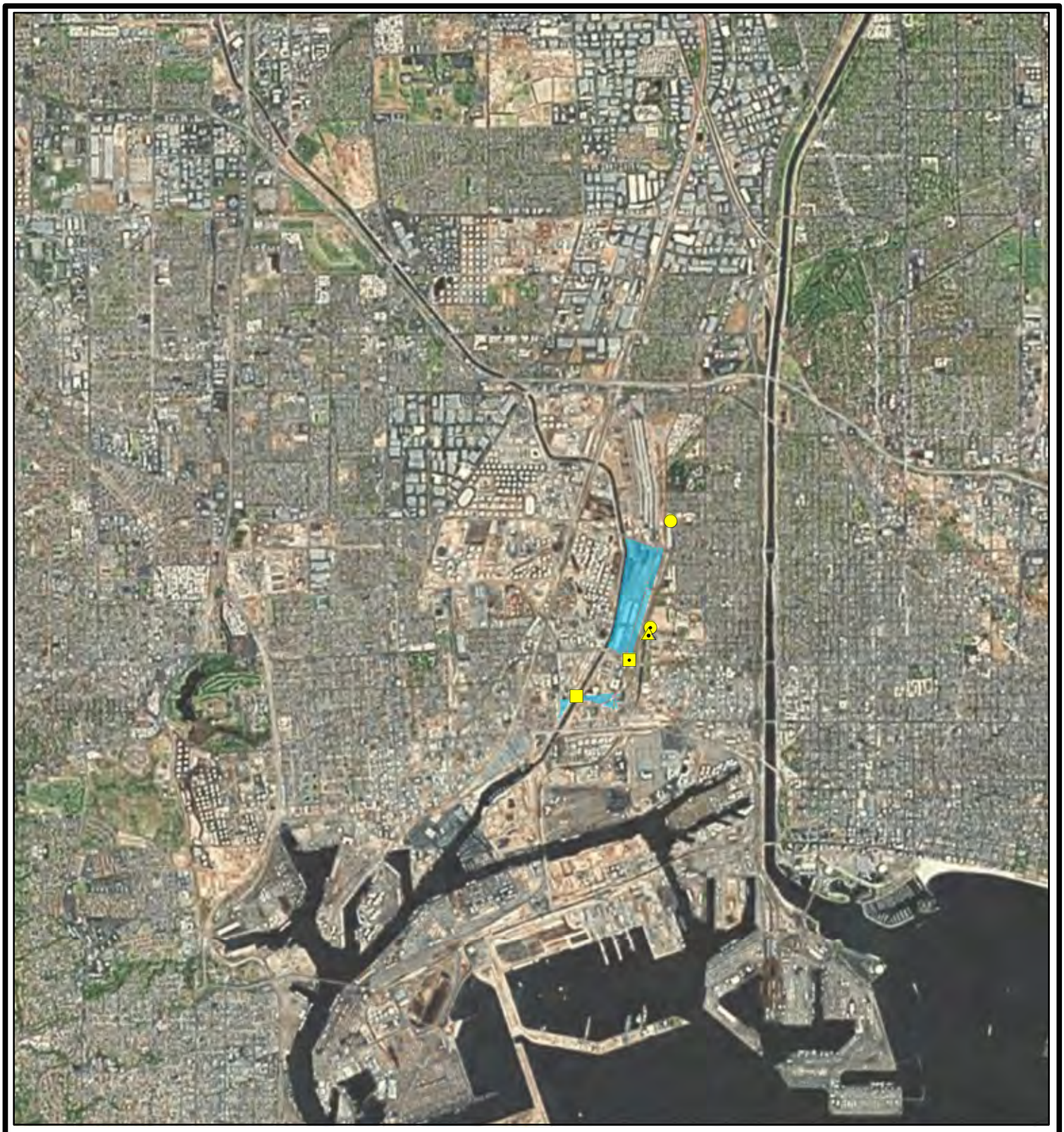
Notes
1. Also location of the Residential CEQA increment value in Table E3-7-4.
2. Also location of the Sensitive and Student CEQA increment values in Table E3-7-4.

Figure C3.7-13
Mitigated Proposed Project

Maximum Exposed Individual for Chronic HI

DRAFT

ENVIRON



Legend

- Residential MEI - Mitigated Project
- Occupational and Recreational MEI - Mitigated Project
- Sensitive and Student MEI - Mitigated Project[1]
- Residential MEI - CEQA Increment
- Occupational and Recreational MEI - CEQA Increment
- Site

N

0 0.5 1 Miles

0 0.5 1 Kilometers

Notes
1. Also location of the Sensitive and Student CEQA increment values in Table E3-7-4.

Figure C3.7-14
Mitigated Proposed Project

Maximum Exposed Individual for Acute HI

DRAFT

ENVIRON

At the maximum occupational receptor, the greatest contributors to cancer risk are SCIG onsite and offsite trucks, as the receptor is located near the Highway 1 off-ramp and the entrance to the SCIG site. The greatest contributors to the chronic hazard index are relocated tenant CHE emissions and SCIG offsite trucks. The greatest contributors to the acute hazard index are SCIG construction, relocated tenant onsite trucks, and relocated tenant CHE emissions. Locomotives contribute between 1.5% and approximately 6% by health effect at the maximum occupational receptor.

Table C3-7-6 presents the contributions from each TAC to the maximum health effects values for the Mitigated Proposed Project. Despite the use of alternative fuels in trucks, DPM remains the primary contributor to cancer risk (greater than 97 percent). The greatest chronic hazard index contributors are DPM at the maximum occupational receptor DPM and chlorine at the maximum residential receptor. The greatest acute hazard index contributor is formaldehyde.

Table C3-7-6. TAC Contributions at the Residential and Occupational MEIs for the Mitigated Proposed Project.

Pollutant	Maximum Residential Receptor			Maximum Occupational Receptor		
	Cancer Risk	Chronic Hazard Index[a]	Acute Hazard Index[a]	Cancer Risk	Chronic Hazard Index[a]	Acute Hazard Index[a]
DPM	97.6%	68.6%	0.0%	98.9%	95.7%	0.0%
Hexavalent Chromium	1.0%	<0.1%	0.0%	0.9%	<0.1%	0.0%
Formaldehyde	0.8%	9.9%	93.6%	0.1%	0.4%	93.5%
Benzene	0.5%	0.2%	0.5%	<0.1%	<0.1%	0.5%
Nickel	<0.1%	1.3%	<0.1%	<0.1%	0.4%	<0.1%
1,3-Butadiene	<0.1%	<0.1%	0.0%	<0.1%	<0.1%	0.0%
Acetaldehyde	<0.1%	<0.1%	5.1%	<0.1%	<0.1%	5.3%
Ethylbenzene	<0.1%	<0.1%	0.0%	<0.1%	<0.1%	0.0%
Arsenic	<0.1%	<0.1%	0.1%	<0.1%	<0.1%	0.1%
Naphthalene	<0.1%	<0.1%	0.0%	<0.1%	<0.1%	0.0%
Lead	<0.1%	0.0%	0.0%	<0.1%	0.0%	0.0%
Cadmium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Chlorine	0.0%	18.3%	0.1%	0.0%	2.8%	<0.1%
Manganese	0.0%	1.4%	0.0%	0.0%	0.6%	0.0%
Acrolein (2-Propenal)	0.0%	<0.1%	0.2%	0.0%	<0.1%	<0.1%
Propylene	0.0%	<0.1%	0.0%	0.0%	<0.1%	0.0%
Toluene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
Isomers Of Xylene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
M-Xylene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
O-Xylene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
N-Hexane	0.0%	<0.1%	0.0%	0.0%	<0.1%	0.0%
Ammonia	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
Styrene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
Methyl Alcohol	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
Mercury	0.0%	0.0%	0.2%	0.0%	0.0%	0.3%
Methyl Ethyl Ketone (MEK) (2-Butanone)	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
Copper	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
Vanadium (Fume Or	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%

Pollutant	Maximum Residential Receptor			Maximum Occupational Receptor		
	Cancer Risk	Chronic Hazard Index[a]	Acute Hazard Index[a]	Cancer Risk	Chronic Hazard Index[a]	Acute Hazard Index[a]
Dust)						
Vanadium	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
P-Xylene	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
Antimony	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Bromine	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Calcium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Carbon Elemental	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Carbon Organic	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Carbonate Ion	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Chromium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Cobalt	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Iron	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Nitrates	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Other	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Phosphorous	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Potassium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Selenium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Sulfates	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Unidentified	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Unknown Pm	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Zinc	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%

a The chemical contributions for the chronic and acute hazard indices include all chemicals regardless of the target organs they affect. As a result, the contributions may add to greater than 100 percent because not all chemicals affect the same target organ.

b For diesel internal combustion engines, only DPM emissions were evaluated for cancer risk and chronic hazard indices, because DPM is a surrogate for the combined health effects associated with exposure to diesel exhaust emissions. For all other emissions (alternative fuel engines, tire and brake wear), emissions of the 47 other toxic air contaminants were evaluated for cancer and chronic hazard indices. For the acute hazard indices, DPM was not evaluated; rather, emissions of the 47 other toxic air contaminants were evaluated for all emission sources (including diesel ICEs).

No calculations of cancer burden were completed for the Mitigated Proposed Project as cancer risks for this alternative were negative.

7.2.1 $PM_{2.5}$ Effects

While the Mitigated Proposed Project will reduce $PM_{2.5}$ concentrations relative to the Unmitigated Proposed Project, 24-hour $PM_{2.5}$ emissions for the Mitigated Proposed Project increment (mitigated project minus Baseline) would still exceed the SCAQMD threshold of 2.5 $\mu g/m^3$. Because of this exceedance, incremental operational $PM_{2.5}$ concentrations meet the Port's criteria for calculating mortality and morbidity attributable to PM.

The area impacted by PM emissions from the Mitigated Proposed Project increment (shown in Figure C2.5-15 of Appendix C2) is similar to that of the Unmitigated Proposed Project increment, although the impacted area is smaller in geographic extent (consistent with the reduced emissions). As discussed with respect to the Unmitigated Proposed Project, no residential populations inhabit the census blocks that are within the zone of

$PM_{2.5}$ exceedance so that the Mitigated Proposed Project increment is not expected to have an impact on PM-attributable morbidity or mortality. As a result, no calculations of morbidity and/or mortality were completed.

7.3 No Project Alternative Health Impacts

The No Project Alternative assumes that the Proposed Project is not built. It accounts for tenant growth and trucks associated with the project going to the BNSF Hobart yard instead (Hobart Trucks).

Table C3-7-7 presents a summary of the maximum health impacts that would occur for each receptor type under the No Project Alternative. The table also shows the maximum health impacts from the Baseline, as well as the CEQA increment (No Project minus Baseline). Because the results in Table C3-7-7 represent the maximum impacts predicted for each receptor type, the impacts at all other receptors would be less than these values.

Table C3-7-7. Maximum Health Impacts Associated with the No Project Alternative.

Health Impact	Receptor Type	Maximum Predicted Impact			Significance Threshold
		No Project	CEQA Baseline	CEQA Increment	
Cancer Risk	Residential	128 x 10^{-6} (128 in a million)	568 x 10^{-6} (568 in a million)	-440 x 10^{-6} (-440 in a million)	10 x 10^{-6} (10 in a million)
	Occupational	37 x 10^{-6} (37 in a million)	215 x 10^{-6} (215 in a million)	-97 x 10^{-6} (-97 in a million)	
	Sensitive	32 x 10^{-6} (32 in a million)	220 x 10^{-6} (220 in a million)	-116 x 10^{-6} (-116 in a million)	
	Student	2.3 x 10^{-6} (2.3 in a million)	4.7 x 10^{-6} (4.7 in a million)	-2.4 x 10^{-6} (-2.4 in a million)	
	Recreational	59 x 10^{-6} (59 in a million)	329 x 10^{-6} (329 in a million)	-146 x 10^{-6} (-146 in a million)	
Chronic Hazard Index	Residential	0.11	0.36	-0.23	1.0
	Occupational	0.18	0.69	-0.51	
	Sensitive	0.03	0.16	-0.10	
	Student	0.03	0.16	-0.13	
	Recreational	0.18	0.69	-0.51	
Acute Hazard Index	Residential	0.12	0.29	-0.17	1.0
	Occupational	0.31	0.79	-0.48	
	Sensitive	0.11	0.27	-0.16	
	Student	0.10	0.27	-0.17	
	Recreational	0.31	0.79	-0.48	

a Exceedances of the significance thresholds are in bold. The significance thresholds apply to the CEQA increments only.

b The maximum increments might not occur at the same receptor locations as the maximum impacts. This means that the increments cannot necessarily be determined by subtracting the CEQA Baseline impact from the project impact. Rather, the subtraction must be done at each receptor, for all modeled receptors, and the maximum result selected.

c The CEQA Increment represents Project minus CEQA Baseline.

d When the maximum increment for a receptor type is negative, the maximum increment displayed is the increment at the maximum project receptor location.

e Data represent the receptor locations with the maximum impacts or increments. The impacts or increments at all other modeled receptors would be less than these values for each receptor type.

f The No Project Alternative assumes that the Proposed Project is not built. It accounts for tenant growth.

The data in Table C3-7-7 show that the CEQA cancer risk increment at the location of the No Project Alternative MEI is predicted to be -440 in a million (-440 x 10^{-6}), at a residential receptor. This risk value, as well as the risk value at all residential receptors, is below the significance threshold of 10 in a million. The CEQA increments would be below the CEQA significance threshold at all receptors, including occupational, sensitive, student, and recreational.

The maximum chronic hazard index increments are predicted to be less than the CEQA significance of 1.0 at all receptors.

The maximum acute hazard index increments are predicted to be less than the CEQA significance threshold of 1.0 at each receptor type.

Figures C3.7-15 and C3.7-16 show the cancer risk isopleths associated with the No Project Alternative and No Project minus Baseline residential individual lifetime cancer risk (per million), respectively.

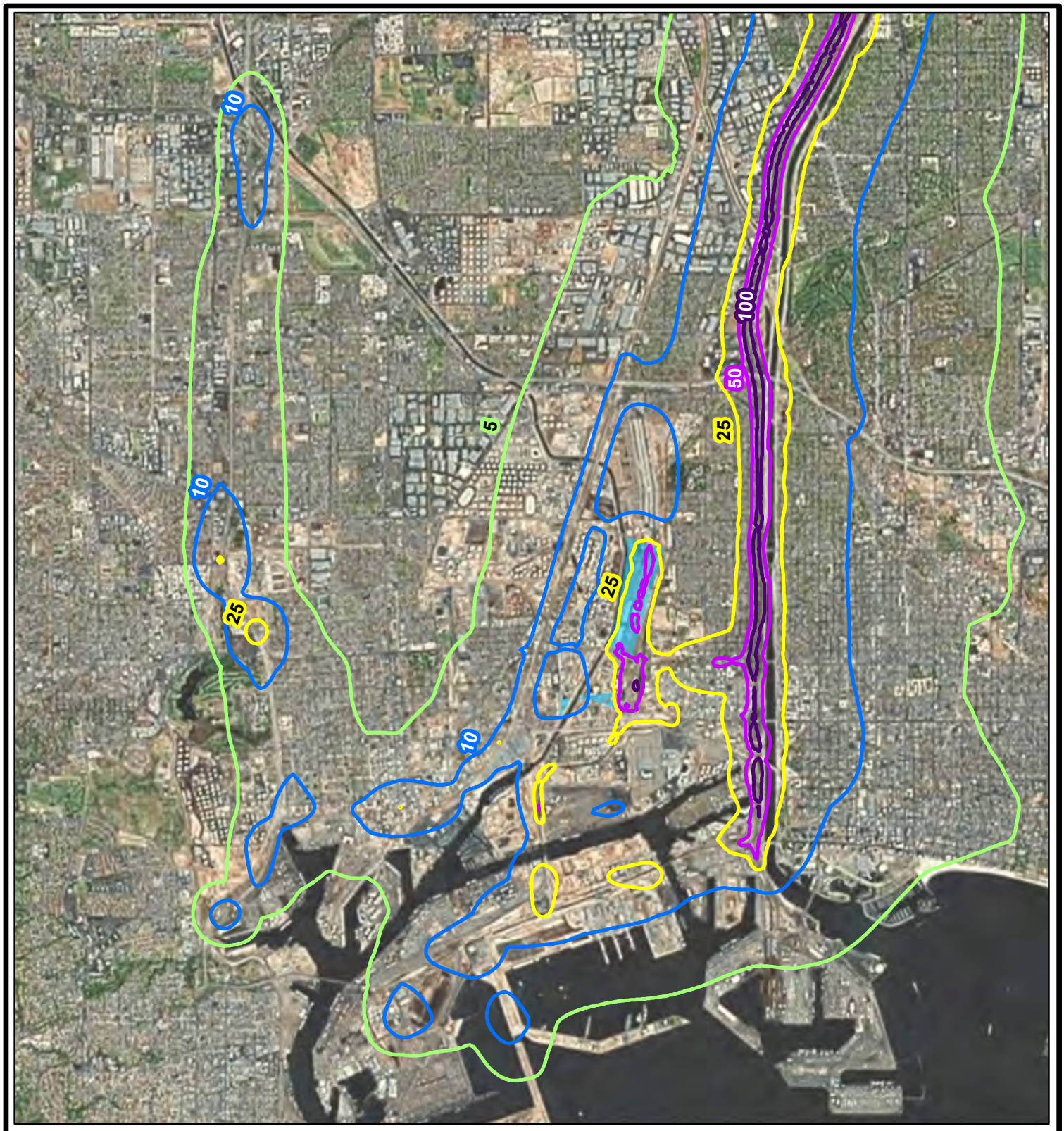
Figures C3.7-17, C3.7-18, and C3.7-19 show the maximum receptor locations for the No Project Alternative for cancer risk, chronic HI, and acute HI, respectively. It should be noted that the residential, occupational, and recreational MEIs are not necessarily located directly on existing homes, workplaces, or recreational facilities; rather, they are located in areas that contain these land use types.

Table C3-7-8 presents the contributions from each emission source to the maximum health effects impacts for the No Project Alternative. At the maximum residential receptor, the greatest contributors to cancer risk are Hobart Trucks. The greatest contributors to the chronic hazard index are Hobart Trucks, as well as tenant offsite gasoline vehicles and tenant offsite trucks. The greatest contributors to the acute hazard index are tenant onsite trucks and tenant CHE emissions, as well as tenant offsite trucks. Tenant onsite locomotives contribute 2% to the acute hazard index and less than 0.5% to the cancer risk and chronic hazard index at the maximum residential receptor.

Table C3-7-8. Source Contributions at the Residential and Occupational MEIs for the No Project Alternative.

Emission Source	Maximum Residential Receptor			Maximum Occupational Receptor		
	Cancer Risk	Chronic Hazard Index	Acute Hazard Index	Cancer Risk	Chronic Hazard Index	Acute Hazard Index
Hobart Trucks	94.6%	74.7%	6.5%	99.9%	7.8%	3.0%
Tenant Offsite Trucks	5.0%	11.6%	15.9%	<0.1%	5.5%	20.9%
Tenant Onsite Locomotives	0.2%	<0.1%	2.0%	<0.1%	<0.1%	1.2%
Tenant CHE	0.1%	0.2%	20.6%	<0.1%	74.3%	20.2%
Tenant Onsite Trucks	<0.1%	<0.1%	54.3%	<0.1%	11.8%	54.4%
Tenant Offsite Gasoline Vehicles	<0.1%	13.4%	0.4%	<0.1%	0.1%	0.3%
Tenant Onsite Gasoline Vehicles	<0.1%	<0.1%	0.2%	<0.1%	0.4%	0.1%

At the maximum occupational receptor, the greatest contributor to cancer risk is Hobart Trucks, as the receptor is located along Interstate 710. The greatest contributor to the chronic hazard index is tenant CHE emissions as well as tenant onsite trucks. The greatest contributors to the acute hazard index are tenant onsite and offsite trucks and tenant CHE emissions. Tenant locomotives contribute approximately 1% to the acute hazard index and less than 0.1% to the cancer risk and chronic hazard index at the maximum occupational receptor.



Legend

- 5
- 10
- 25
- 50
- 100
- Site

N

0 0.5 1 Miles

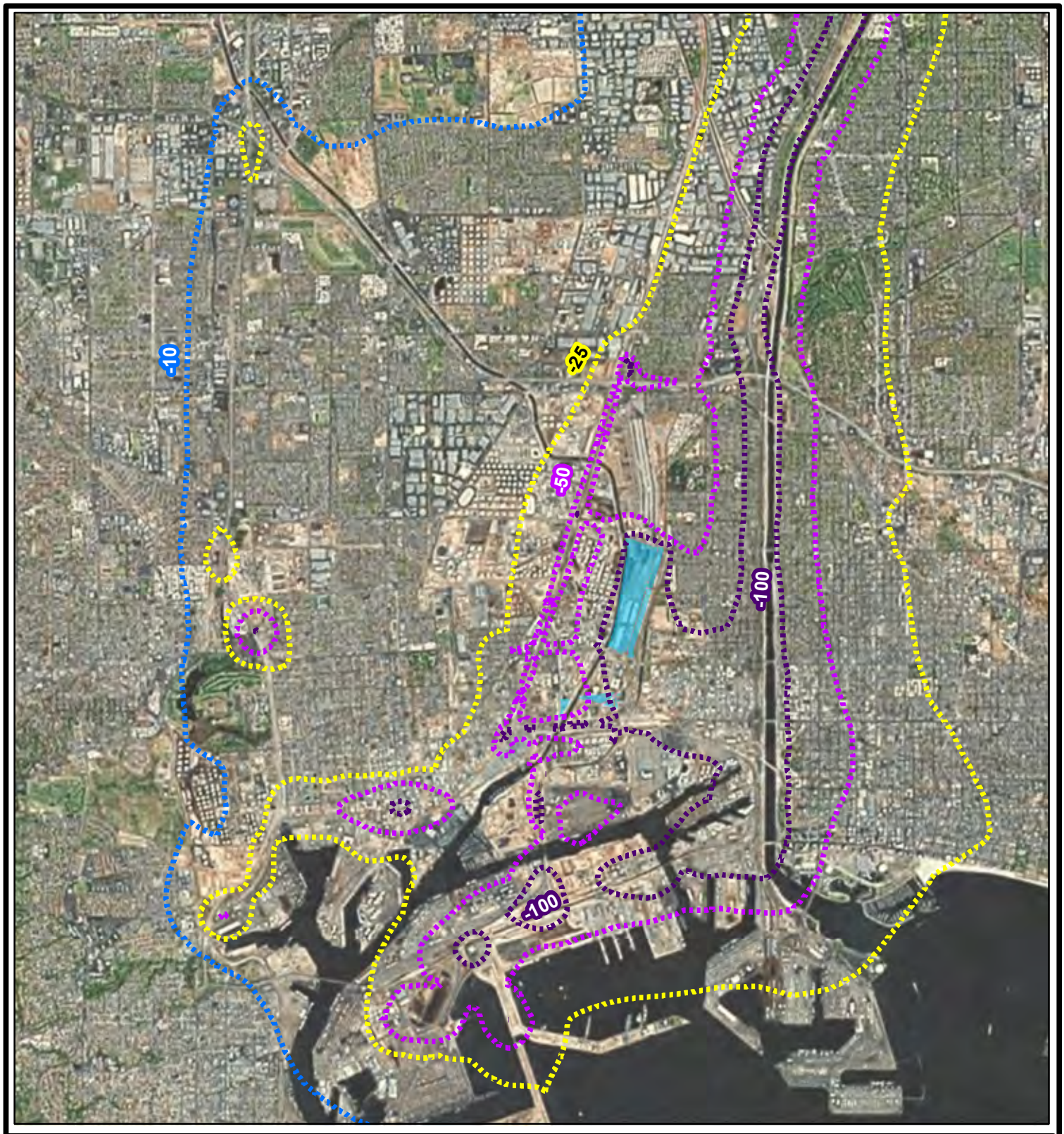
0 0.5 1 Kilometers

Figure C3.7-15
No Project Alternative

Residential Individual Lifetime Cancer Risk (per Million)

DRAFT

ENVIRON



Legend

- -10
- -25
- -50
- -100
- Site

N

0 0.5 1 Miles

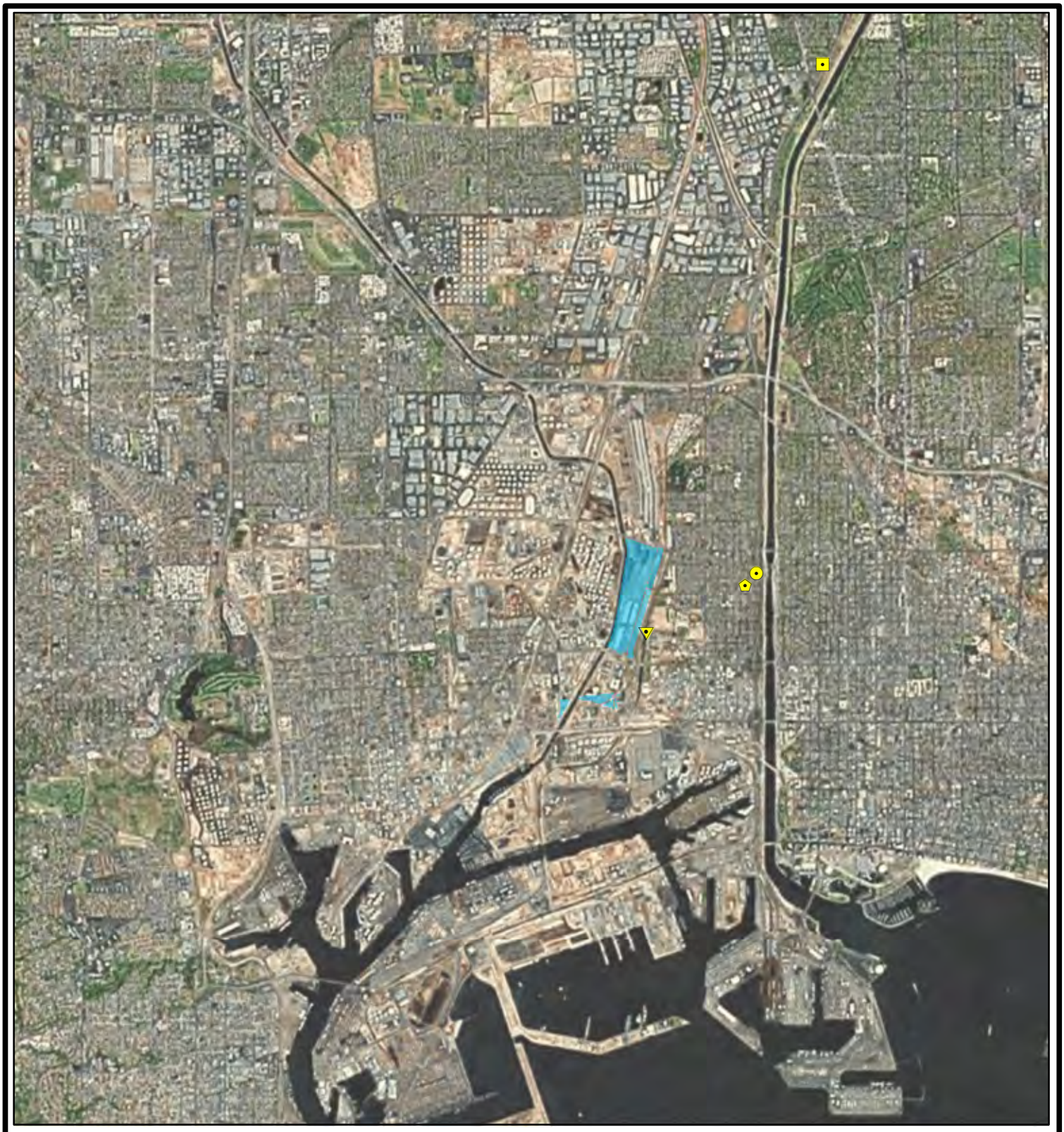
0 0.5 1 Kilometers

Figure C3.7-16
No Project Alternative minus Baseline

Residential Individual Lifetime Cancer Risk (per Million)

DRAFT

ENVIRON



Legend

- Residential MEI - No Project[1]
- Occupational and Recreational MEI - No Project[2]
- Sensitive MEI - No Project[3]
- Student MEI - No Project[4]
- Site

N

0 0.5 1 Miles

0 0.5 1 Kilometers

Notes

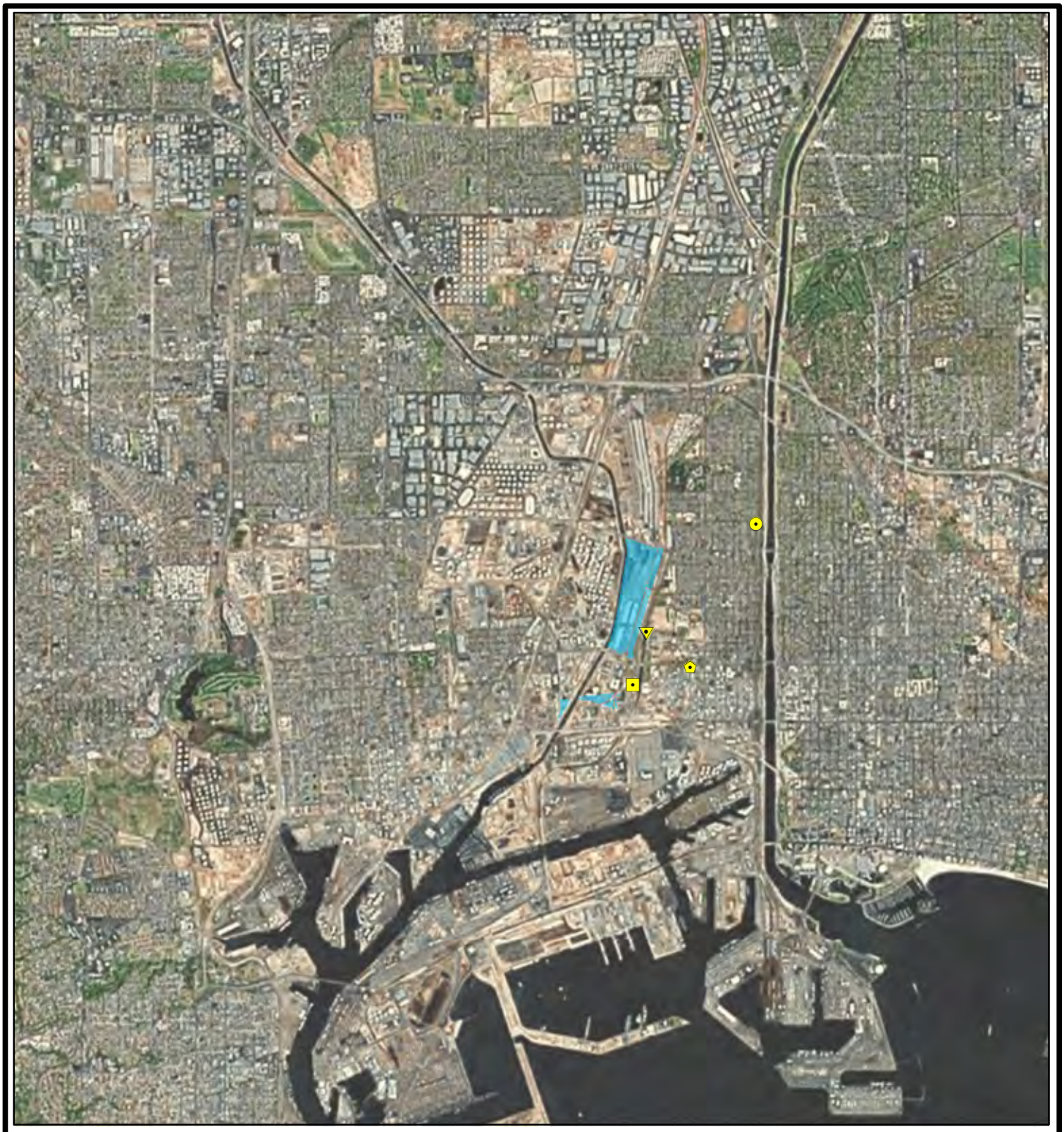
1. Also location of the Residential CEQA increment value in Table E3-7-16.
2. Also location of the Occupational and Recreational CEQA increment values in Table E3-7-16.
3. Also location of the Sensitive CEQA increment value in Table E3-7-16.
4. Also location of the Student CEQA increment value in Table E3-7-16.

Figure C3.7-17
No Project Alternative

Maximum Exposed Individual for Cancer Risk

DRAFT

ENVIRON



Legend

- Residential MEI - No Project[1]
- Occupational and Recreational MEI - No Project[2]
- Sensitive MEI - No Project[3]
- Student MEI - No Project[4]
- Site

N

0 0.5 1 Miles

0 0.5 1 Kilometers

Notes

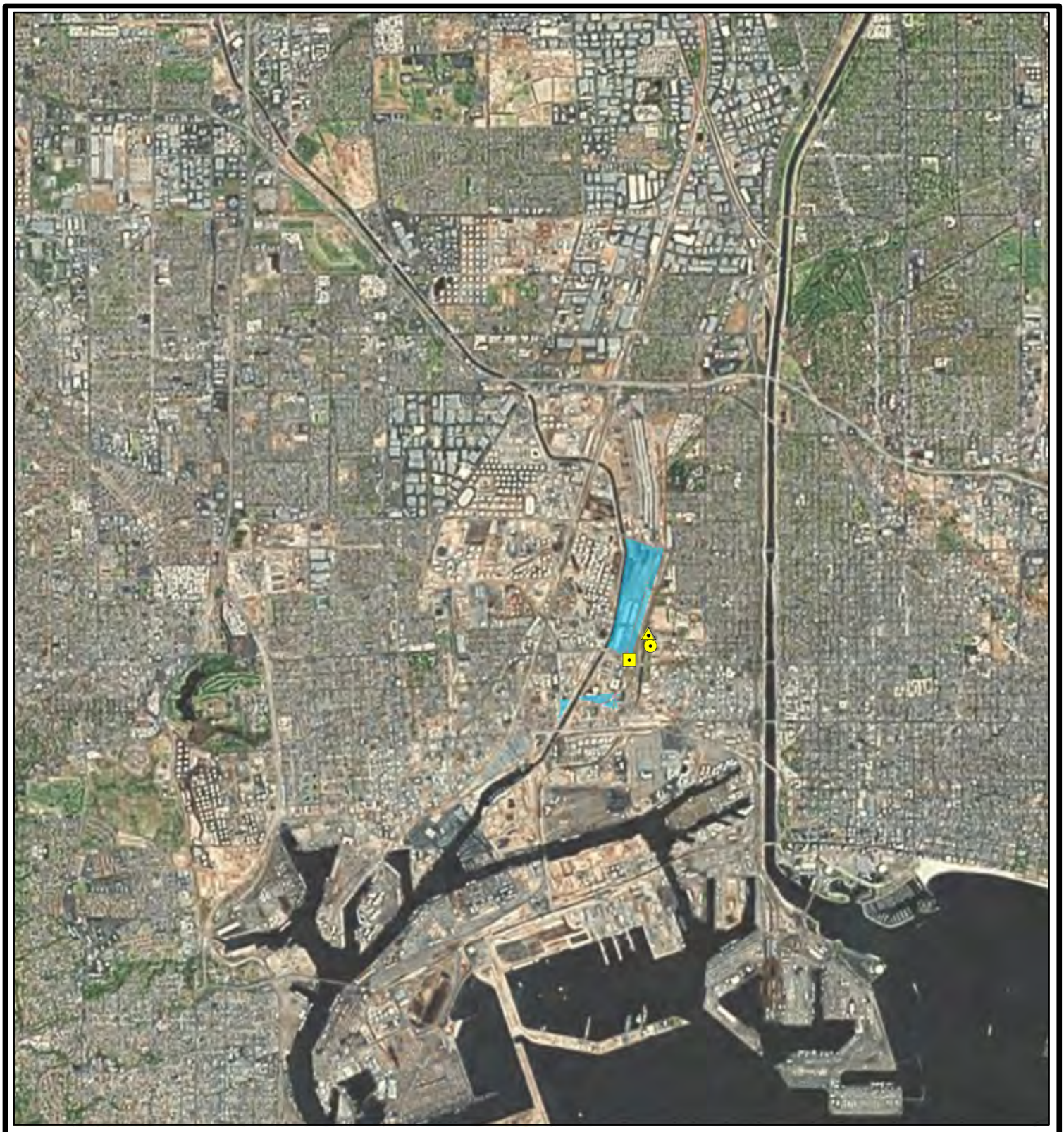
1. Also location of the Residential CEQA increment value in Table E3-7-16.
2. Also location of the Occupational and Recreational CEQA increment values in Table E3-7-16.
3. Also location of the Sensitive CEQA increment value in Table E3-7-16.
4. Also location of the Student CEQA increment value in Table E3-7-16.

Figure C3.7-18
No Project Alternative

Maximum Exposed Individual for Chronic HI

DRAFT

ENVIRON



Legend

- Residential MEI - No Project[1]
- Occupational and Recreational MEI - No Project[2]
- Sensitive and Student MEI - No Project[3]
- Site

N

0 0.5 1 Miles

0 0.5 1 Kilometers

Notes
1. Also location of the Residential CEQA increment value in Table E3-7-16.
2. Also location of the Occupational and Recreational CEQA increment values in Table E3-7-16.
3. Also location of the Sensitive and Student CEQA increment values in Table E3-7-16.

Figure C3.7-19
No Project Alternative

Maximum Exposed Individual for Acute HI

DRAFT

ENVIRON

Table C3-7-9 presents the contributions from each TAC to the maximum health effects values for the No Project Alternative. DPM remains the primary contributor to cancer risk (greater than 99 percent) at both the maximum residential and occupational receptors. The greatest chronic hazard index contributors at the maximum residential receptor are DPM and chlorine, while DPM contributes almost 100 percent of the chronic hazard index at the maximum occupational receptor. The greatest acute hazard index contributor is formaldehyde.

Table C3-7-9. TAC Contributions at the Residential and Occupational MEIs for the No Project Alternative.

Pollutant	Maximum Residential Receptor			Maximum Occupational Receptor		
	Cancer Risk	Chronic Hazard Index[a]	Acute Hazard Index[a]	Cancer Risk	Chronic Hazard Index[a]	Acute Hazard Index[a]
DPM	99.2%	77.6%	0.0%	99.3%	100.0%	0.0%
Hexavalent Chromium	0.8%	<0.1%	0.0%	0.7%	<0.1%	0.0%
Nickel	<0.1%	2.1%	-0.1%	<0.1%	0.2%	-0.1%
Arsenic	<0.1%	<0.1%	0.3%	<0.1%	<0.1%	0.3%
1,3-Butadiene	<0.1%	<0.1%	0.0%	<0.1%	<0.1%	0.0%
Benzene	<0.1%	<0.1%	0.5%	<0.1%	<0.1%	0.5%
Formaldehyde	<0.1%	0.2%	93.5%	<0.1%	<0.1%	93.5%
Lead	<0.1%	0.0%	0.0%	<0.1%	0.0%	0.0%
Ethylbenzene	<0.1%	<0.1%	0.0%	<0.1%	<0.1%	0.0%
Naphthalene	<0.1%	<0.1%	0.0%	<0.1%	<0.1%	0.0%
Acetaldehyde	<0.1%	<0.1%	5.3%	<0.1%	<0.1%	5.3%
Cadmium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Chlorine	0.0%	16.7%	-0.6%	0.0%	-0.6%	-0.5%
Manganese	0.0%	2.7%	0.0%	0.0%	0.3%	0.0%
Acrolein (2-Propenal)	0.0%	0.5%	0.3%	0.0%	<0.1%	0.2%
Toluene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
M-Xylene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
O-Xylene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
Propylene	0.0%	<0.1%	0.0%	0.0%	<0.1%	0.0%
N-Hexane	0.0%	<0.1%	0.0%	0.0%	<0.1%	0.0%
Styrene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
Ammonia	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
Methyl Alcohol	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
Isomers Of Xylene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
Mercury	0.0%	0.0%	0.6%	0.0%	0.0%	0.5%
Copper	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
Methyl Ethyl Ketone (MEK) (2-Butanone)	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
Vanadium	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
Vanadium (Fume Or Dust)	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
P-Xylene	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
Antimony	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Bromine	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Calcium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Carbon Elemental	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Carbon Organic	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Carbonate Ion	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Chromium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Cobalt	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%

Pollutant	Maximum Residential Receptor			Maximum Occupational Receptor		
	Cancer Risk	Chronic Hazard Index[a]	Acute Hazard Index[a]	Cancer Risk	Chronic Hazard Index[a]	Acute Hazard Index[a]
Iron	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Nitrates	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Other	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Phosphorous	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Potassium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Selenium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Sulfates	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Unidentified	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Unknown Pm	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Zinc	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%

a The chemical contributions for the chronic and acute hazard indices include all chemicals regardless of the target organs they affect. As a result, the contributions may add to greater than 100 percent because not all chemicals affect the same target organ.

b For diesel internal combustion engines, only DPM emissions were evaluated for cancer risk and chronic hazard indices, because DPM is a surrogate for the combined health effects associated with exposure to diesel exhaust emissions. For all other emissions (alternative fuel engines, tire and brake wear), emissions of the 47 other toxic air contaminants were evaluated for cancer and chronic hazard indices. For the acute hazard indices, DPM was not evaluated; rather, emissions of the 47 other toxic air contaminants were evaluated for all emission sources (including diesel ICEs).

As discussed above, the cancer risks for the No Project Alternative are negative and thus, no calculations of cancer burden were completed for this Alternative.

7.3.1 $PM_{2.5}$ Effects

The results of ambient air dispersion modeling indicated that operation of the No Project (project minus Baseline) would not result in off-site 24-hour $PM_{2.5}$ concentrations that exceed the SCAQMD significance threshold of 2.5 $\mu g/m^3$. As a result, incremental operational $PM_{2.5}$ concentrations for the No Project do not meet the Port's criteria for calculating mortality and morbidity attributable to PM (POLA, 2011), and no calculations of mortality or morbidity were made.

7.4 Unmitigated Proposed Project minus Floating Baseline Health Impacts

In addition to the CEQA increment based on a static Baseline, an increment was evaluated based on a floating Baseline. Floating Baseline emissions spanning years 2005 to 2074 were estimated by fixing activity levels at the time the NOP was released and allowing for future changes in emission factors for equipment, vehicles, ships, locomotives, and other emissions sources. In this analysis, emissions factors were reduced as regulations were applied requiring lower-emission technology and fleet turnover which introduced new model years of equipment with lower emissions in the future years. A floating Baseline established in this manner results in Baseline emissions that are much lower than the static Baseline used under the evaluation of the CEQA increment.

Given that this evaluation is for informational purposes only and will not be used for the evaluation of significance under CEQA, the health impact analysis for the floating

Baseline increment focused exclusively on the calculation of cancer risk to residential receptors. These calculations were performed following the same approach and using the same exposure assumptions for a residential receptor as described in the evaluation of the Unmitigated Proposed Project. However, the emissions used to model TAC concentrations were based on floating Baseline emissions as distinct from CEQA Baseline emissions. Therefore, the maximum Unmitigated Proposed Project impact remains at 48 in a million (48×10^{-6}) for a residential receptor as seen in Table C3-7-10. The maximum floating Baseline cancer risk is predicted to be 114 in a million (114×10^{-6}) which is approximately 80 percent lower than the CEQA static Baseline. The maximum increment of Unmitigated Proposed Project minus floating Baseline impact is predicted to be 17 in a million (17×10^{-6}), as compared to the CEQA increment associated with the Unmitigated Proposed Project minus the static Baseline of -160 in a million (-160×10^{-6}). The location of maximum risk increment is at the same location as that of the maximum Unmitigated Proposed Project impact.

Table C3-7-10. Maximum Health Impacts Associated with the Unmitigated Proposed Project and the Floating Baseline.

Health Impact	Receptor Type	Maximum Predicted Impact		
		Proposed Project	CEQA Baseline	CEQA Increment
Cancer Risk	Residential	48×10^{-6} (48 in a million)	114×10^{-6} (114 in a million)	17×10^{-6} (17 in a million)

a The maximum increments might not occur at the same receptor locations as the maximum impacts. This means that the increments cannot necessarily be determined by subtracting the CEQA Baseline impact from the project impact. Rather, the subtraction must be done at each receptor, for all modeled receptors, and the maximum result selected.
b The CEQA Increment represents Project minus CEQA floating Baseline.
c When the maximum increment for a receptor type is negative, the maximum increment displayed is the increment at the maximum project receptor location.
d Data represent the receptor locations with the maximum impacts or increments. The impacts or increments at all other modeled receptors would be less than these values for each receptor type.

Figure C3.7-20 shows the Floating Baseline residential individual lifetime cancer risk (per million).

Figure C3.7-21 shows the cancer risk isopleth associated with the Unmitigated Proposed Project minus Floating Baseline residential individual lifetime cancer risk (per million), respectively.

7.5 Unmitigated Reduced Project Alternative Health Impacts

The Unmitigated Reduced Project Alternative is based on a reduced throughput assumption.

Table C3-7-11 presents a summary of the maximum health impacts that would occur for each receptor type with construction and operation of the Unmitigated Reduced Project Alternative. The table also shows the maximum health impacts from the Baseline, as well as the CEQA increment (Unmitigated Reduced Project minus Baseline). Because the results in Table C3-7-11 represent the maximum impacts predicted for each receptor type, the impacts at all other receptors would be less than these values.

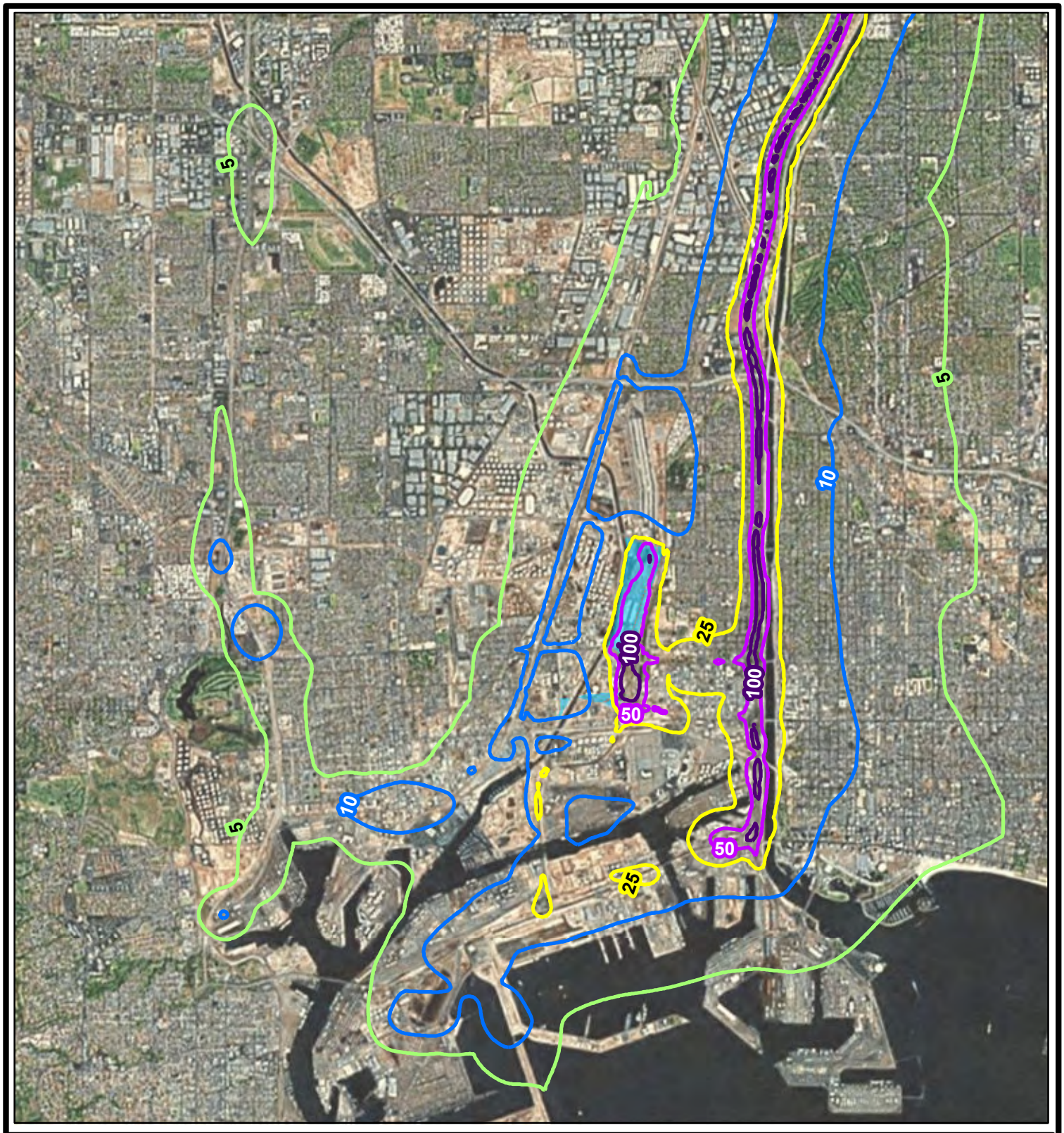
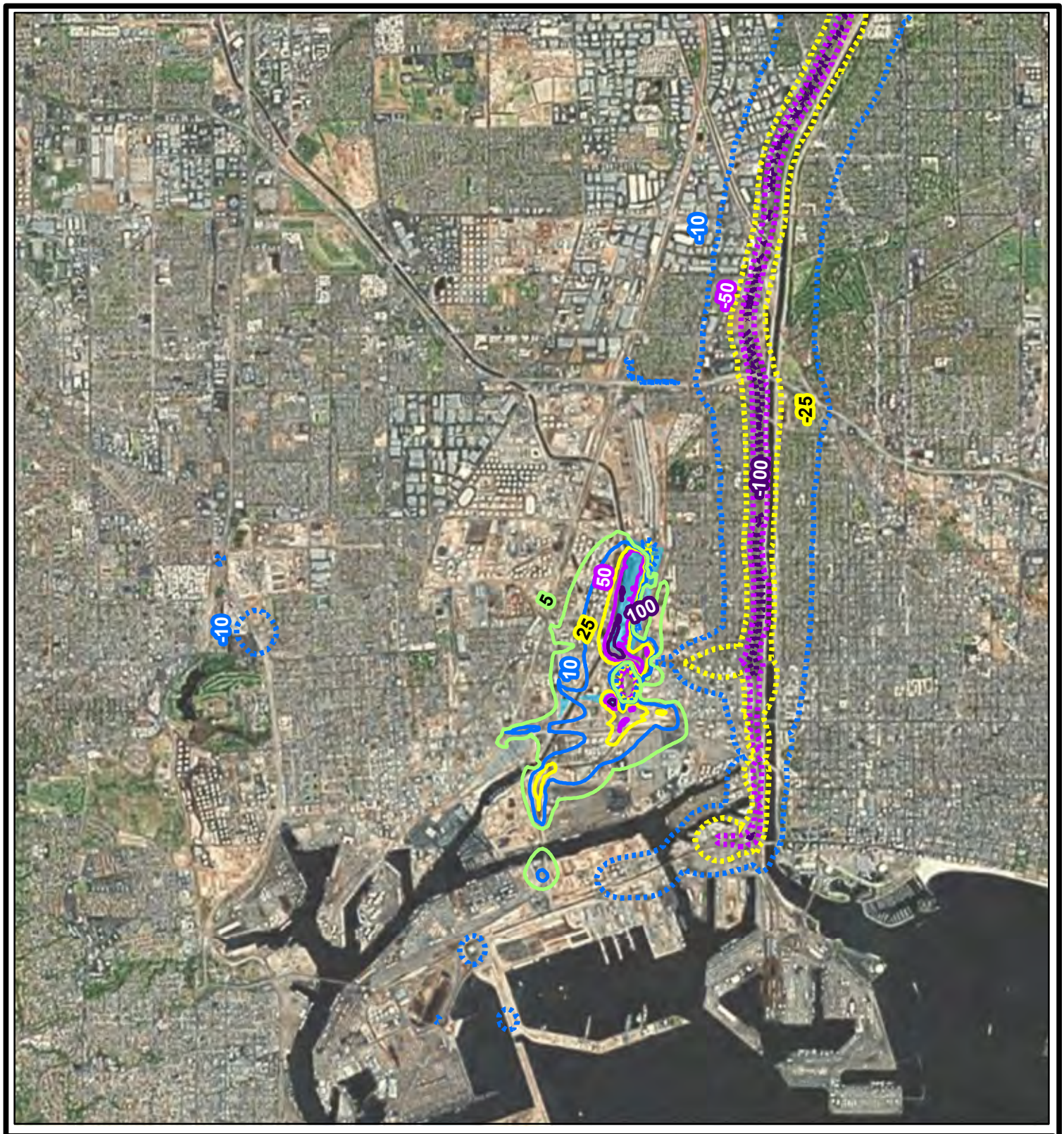


Figure C3.7-20
Floating Baseline

Residential Individual Lifetime Cancer Risk (per Million)

DRAFT

ENVIRON



Legend

- 100
- 50
- 25
- 10
- 5
- -10
- -25
- -50
- -100
- Site

N

0 0.5 1 Miles

0 0.5 1 Kilometers

Figure C3.7-21
Unmitigated Proposed Project minus Floating Baseline

Residential Individual Lifetime Cancer Risk (per Million)

DRAFT

ENVIRON

Table C3-7-11. Maximum Health Impacts Associated with the Unmitigated Reduced Project Alternative.

Health Impact	Receptor Type	Maximum Predicted Impact			Significance Threshold
		Reduced Project	CEQA Baseline	CEQA Increment	
Cancer Risk	Residential	35×10^{-6} (35 in a million)	568×10^{-6} (568 in a million)	-174×10^{-6} (-174 in a million)	10×10^{-6} (10 in a million)
	Occupational	29×10^{-6} (29 in a million)	215×10^{-6} (215 in a million)	-125×10^{-6} (-125 in a million)	
	Sensitive	30×10^{-6} (30 in a million)	220×10^{-6} (220 in a million)	-190×10^{-6} (-190 in a million)	
	Student	2.6×10^{-6} (2.6 in a million)	4.7×10^{-6} (4.7 in a million)	-2.1×10^{-6} (-2.1 in a million)	
	Recreational	43×10^{-6} (43 in a million)	329×10^{-6} (329 in a million)	-194×10^{-6} (-194 in a million)	
Chronic Hazard Index	Residential	0.09	0.36	-0.06	1.0
	Occupational	0.44	0.69	0.10	
	Sensitive	0.10	0.16	-0.07	
	Student	0.10	0.16	-0.07	
	Recreational	0.44	0.69	0.10	
Acute Hazard Index	Residential	0.19	0.29	0.01	1.0
	Occupational	0.65	0.79	0.13	
	Sensitive	0.21	0.27	0.000	
	Student	0.21	0.27	-0.065	
	Recreational	0.65	0.79	0.13	

a Exceedances of the significance thresholds are in bold. The significance thresholds apply to the CEQA increments only.

b The maximum increments might not occur at the same receptor locations as the maximum impacts. This means that the increments cannot necessarily be determined by subtracting the CEQA Baseline impact from the project impact. Rather, the subtraction must be done at each receptor, for all modeled receptors, and the maximum result selected.

c The CEQA Increment represents Project minus CEQA Baseline.

d When the maximum increment for a receptor type is negative, the maximum increment displayed is the increment at the maximum project receptor location.

e Data represent the receptor locations with the maximum impacts or increments. The impacts or increments at all other modeled receptors would be less than these values for each receptor type.

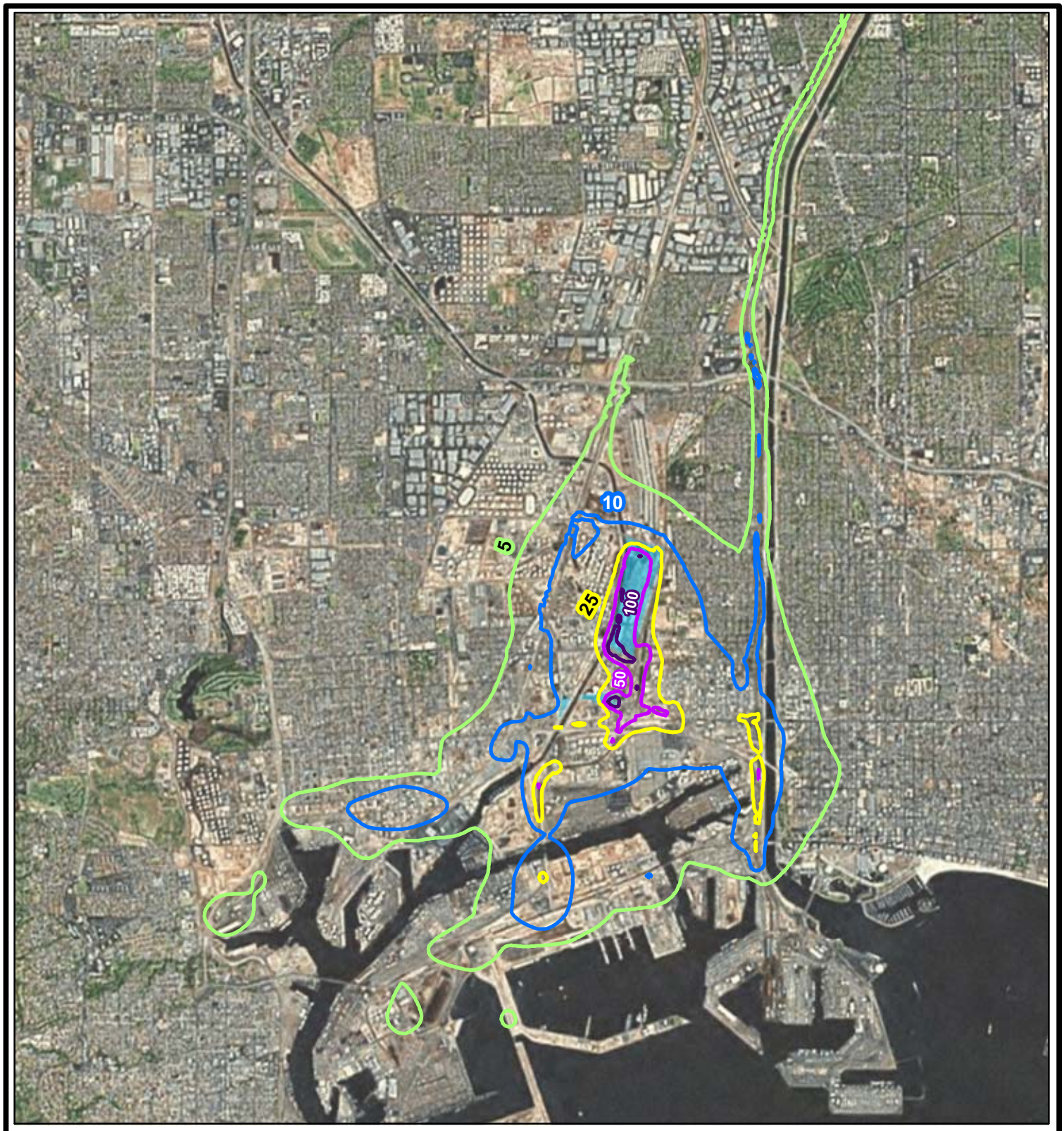
f The Unmitigated Reduced Project scenario is based on a reduced throughput assumption.

The data in Table C3-7-11 show that the CEQA cancer risk increment at the location of the Unmitigated Reduced Project Alternative MEI is predicated to be -174 in a million (-174×10^{-6}), at a residential receptor. This risk value, as well as the risk value at all residential receptors, is below the significance threshold of 10 in a million. The CEQA increments would be below the CEQA significance threshold at all receptors, including occupational, sensitive, student, and recreational.

The maximum chronic hazard index increments are predicted to be less than the CEQA significance of 1.0 at all receptors. The maximum acute hazard index increments are predicted to be less than the CEQA significance threshold of 1.0 at each receptor type.

Figures C3.7-22 and C3.7-23 show the cancer risk isopleths associated with the Unmitigated Reduced Project Alternative and Unmitigated Reduced Project minus Baseline residential individual lifetime cancer risk (per million), respectively.

Figures C3.7-24, C3.7-25, and C3.7-26 show the maximum receptor locations for the Unmitigated Reduced Project Alternative for cancer risk, chronic HI, and acute HI, respectively. It should be noted that the residential, occupational, and recreational MEIs



Legend

- 5
- 10
- 25
- 50
- 100
- Site

N

0 0.5 1 Miles

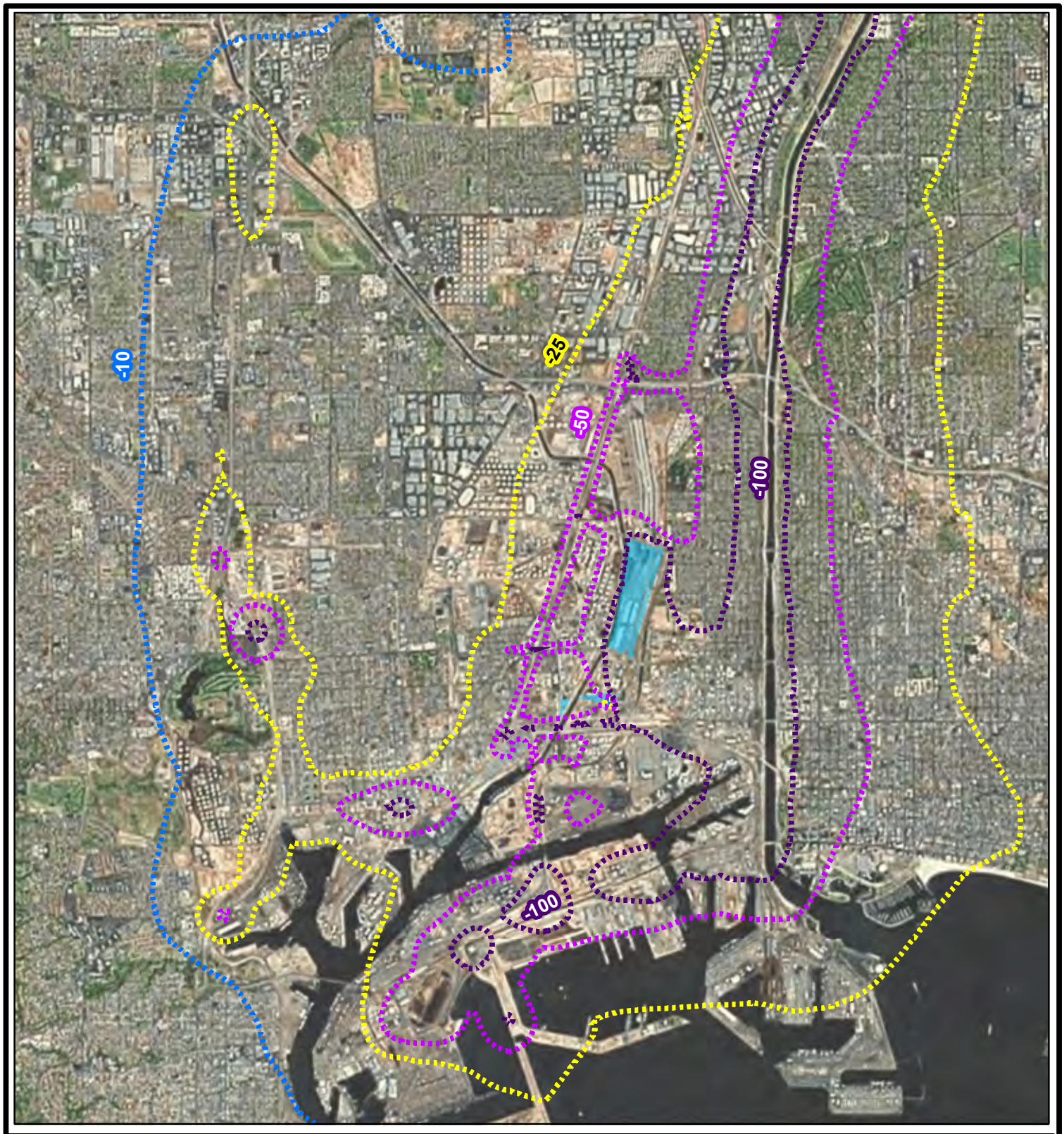
0 0.5 1 Kilometers

Figure C3.7-22
Unmitigated Reduced Project Alternative

Residential Individual Lifetime Cancer Risk (per Million)

DRAFT

ENVIRON



Legend

- -10
- -25
- -50
- -100
- Site

N

0 0.5 1 Miles

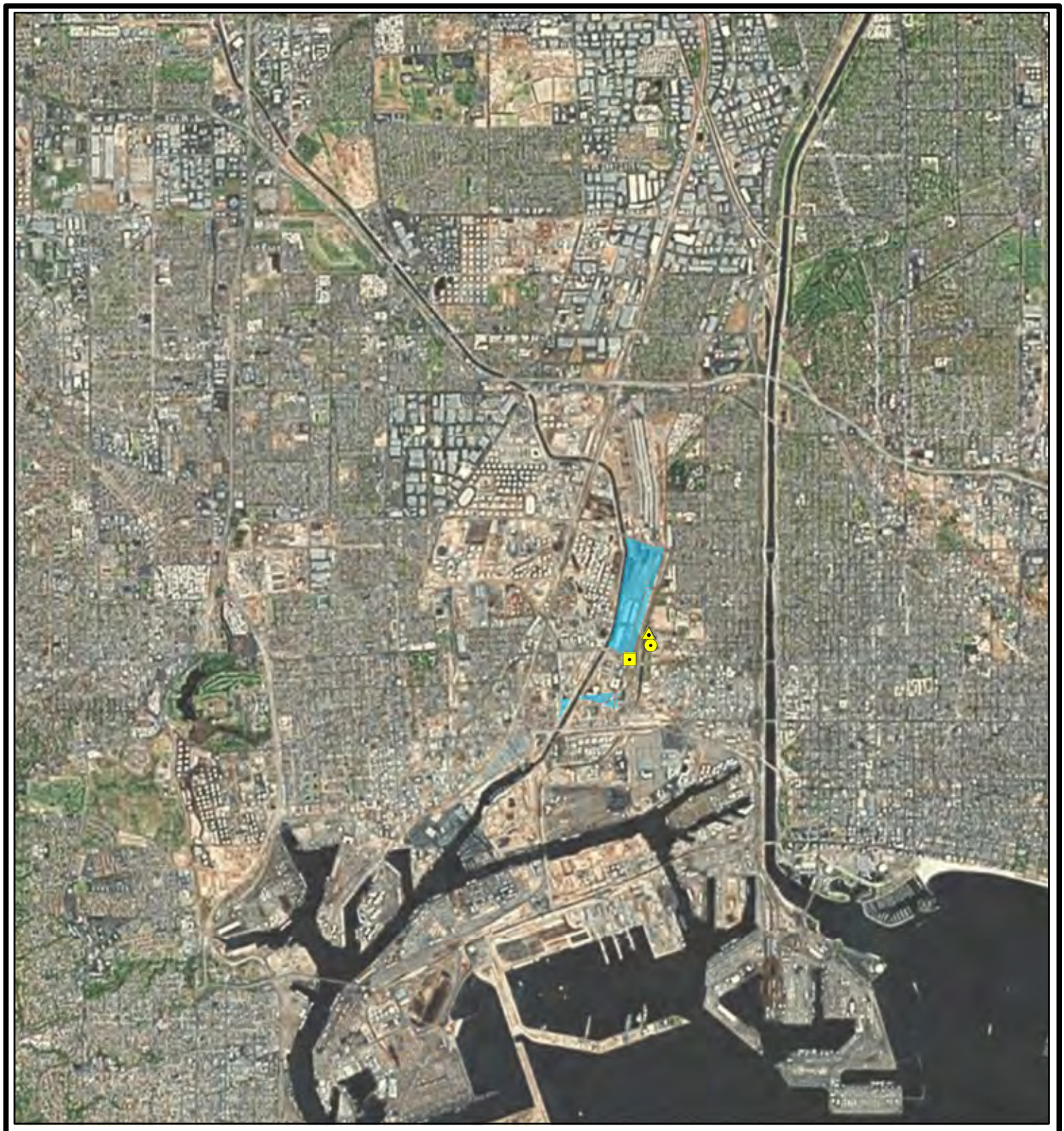
0 0.5 1 Kilometers

Figure C3.7-23
Unmitigated Reduced Project Alternative minus Baseline

Residential Individual Lifetime Cancer Risk (per Million)

DRAFT

ENVIRON



Legend

- Residential MEI - Reduced Project[1]
- Occupational and Recreational MEI - Reduced Project[2]
- Sensitive and Student MEI - Reduced Project[3]
- Site

N

0 0.5 1 Miles

0 0.5 1 Kilometers

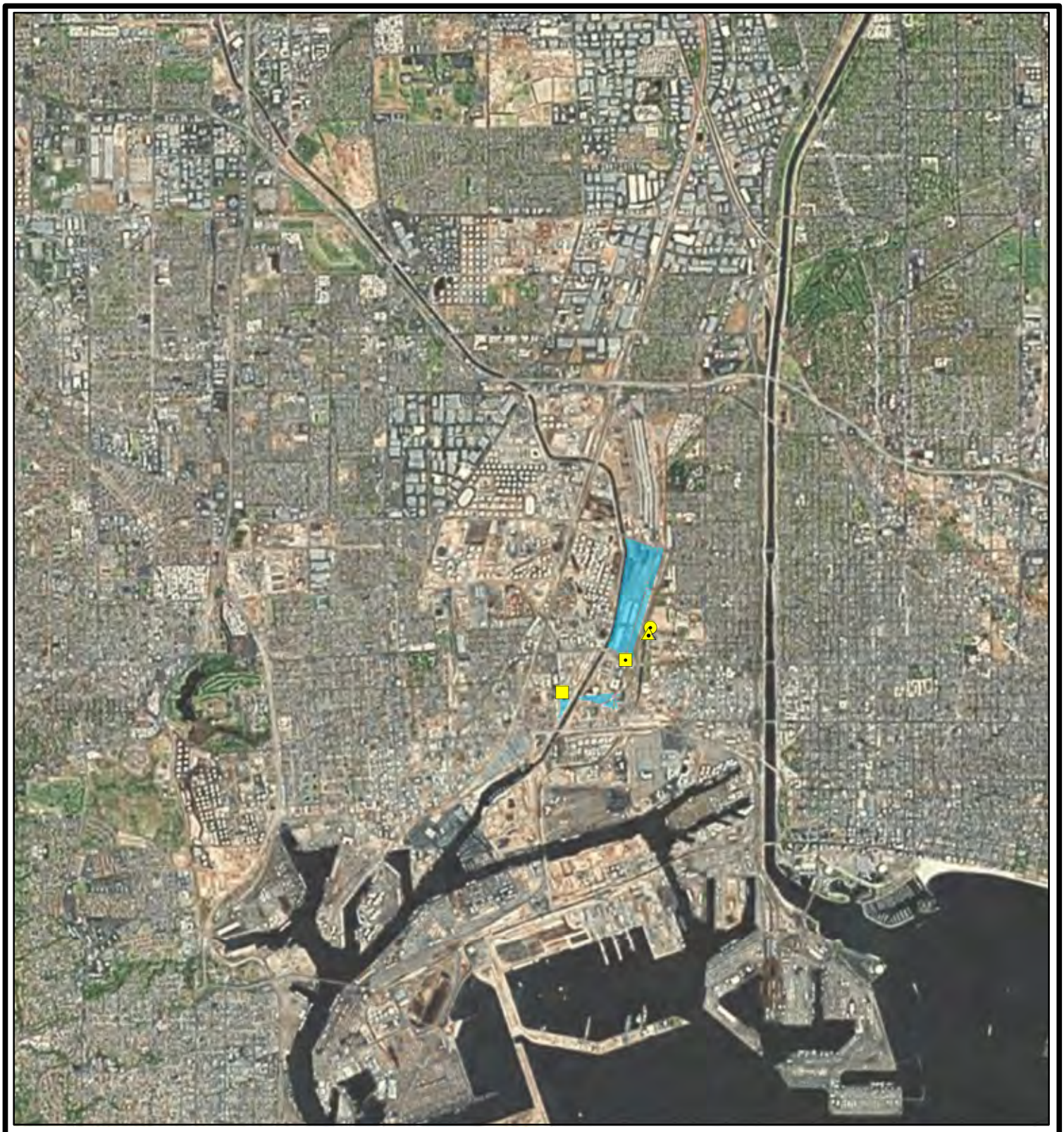
Notes
1. Also location of the Residential CEQA increment value in Table E3-7-10.
2. Also location of the Occupational and Recreational CEQA increment values in Table E3-7-10.
3. Also location of the Sensitive and Student CEQA increment values in Table E3-7-10.

Figure C3.7-24
Unmitigated Reduced Project Alternative

Maximum Exposed Individual for Cancer Risk

DRAFT

ENVIRON



Legend

- Residential MEI - Reduced Project[1]
- Occupational and Recreational MEI - Reduced Project
- Sensitive and Student MEI - Reduced Project[2]
- Occupational and Recreational MEI - CEQA Increment
- Site

N

0 0.5 1 Miles

0 0.5 1 Kilometers

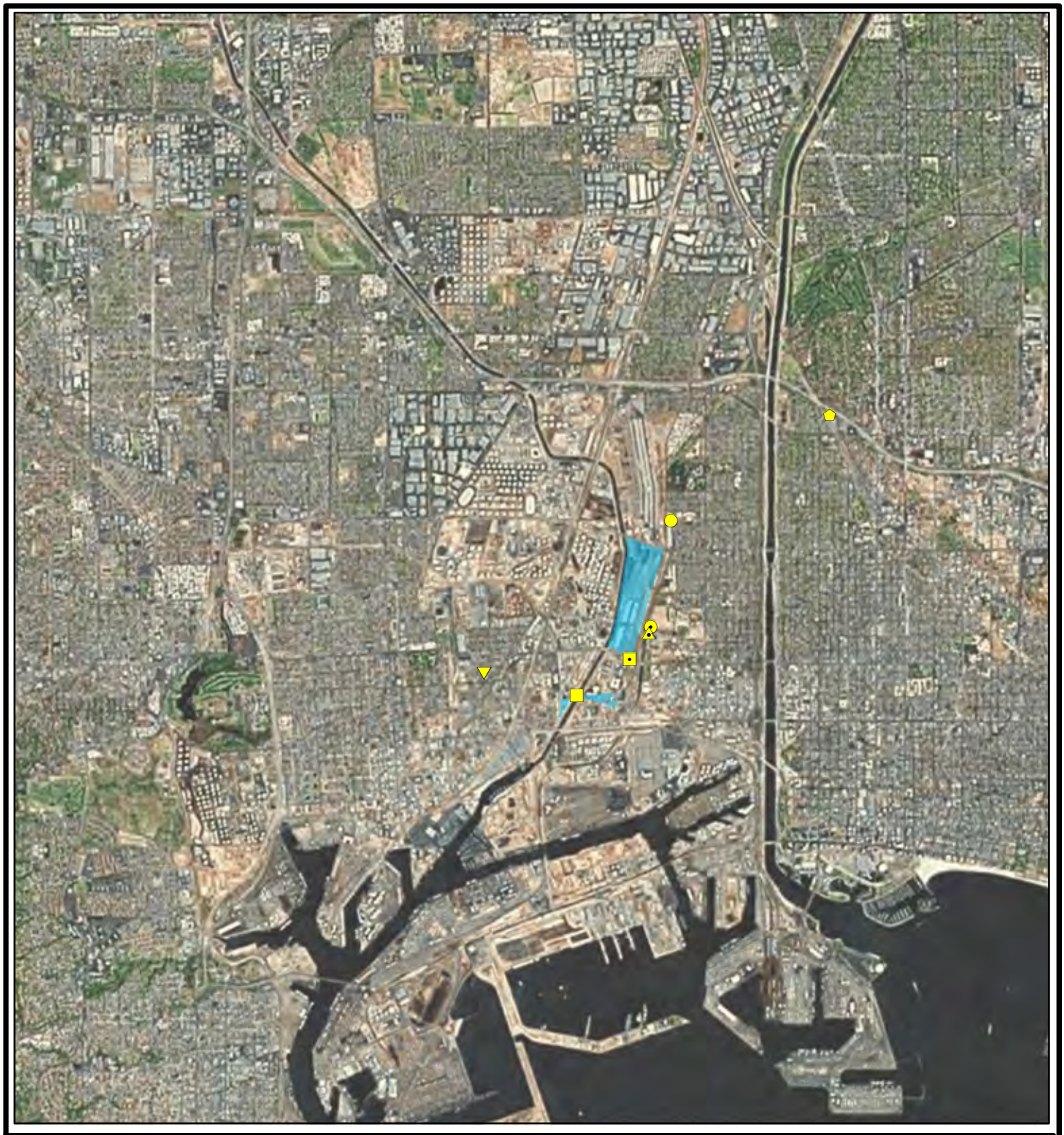
Notes
1. Also location of the Residential CEQA increment value in Table E3-7-10.
2. Also location of the Sensitive and Student CEQA increment values in Table E3-7-10.

Figure C3.7-25
Unmitigated Reduced Project Alternative

Maximum Exposed Individual for Chronic HI

DRAFT

ENVIRON



Legend

- Residential MEI - Reduced Project
- Occupational and Recreational MEI - Reduced Project
- Sensitive and Student MEI - Reduced Project
- Residential MEI - CEQA Increment
- Occupational and Recreational MEI - CEQA Increment
- Sensitive MEI - CEQA Increment
- Student MEI - CEQA Increment
- Site

N

0 0.5 1 Miles

0 0.5 1 Kilometers

Figure C3.7-26
Unmitigated Reduced Project Alternative

Maximum Exposed Individual for Acute HI

DRAFT

ENVIRON

are not necessarily located directly on existing homes, workplaces, or recreational facilities; rather, they are located in areas that contain these land use types.

Table C3-7-12 presents the contributions from each emission source to the maximum health effects impacts for the Unmitigated Reduced Project Alternative. At the maximum residential receptor, the greatest contributors to cancer risk are SCIG offsite and onsite trucks. The greatest contributors to the chronic hazard index are SCIG construction and onsite trucks and Hostler emissions. The greatest contributor to the acute hazard index is SCIG construction. Locomotives contribute between approximately 3-4% by health effect at the maximum residential receptor.

Table C3-7-12. Source Contributions at the Residential and Occupational MEIs for the Unmitigated Reduced Project Alternative.

Emission Source	Maximum Residential Receptor			Maximum Occupational Receptor		
	Cancer Risk	Chronic Hazard Index	Acute Hazard Index	Cancer Risk	Chronic Hazard Index	Acute Hazard Index
SCIG Offsite Trucks	52.7%	7.7%	2.7%	68.9%	4.1%	2.1%
SCIG Onsite Trucks	33.7%	18.8%	7.1%	14.7%	1.6%	2.6%
SCIG Construction	2.4%	38.1%	56.7%	10.5%	82.2%	47.4%
Tenant CHE	2.4%	6.0%	7.4%	1.5%	0.5%	13.4%
Tenant Offsite Trucks	2.2%	2.8%	4.8%	1.7%	0.9%	4.0%
SCIG Onsite Locomotives	2.1%	1.9%	2.0%	1.2%	7.1%	3.1%
Hostler	1.5%	18.5%	4.9%	0.2%	0.2%	1.6%
SCIG Offsite Locomotives	1.2%	2.1%	0.6%	0.4%	1.6%	0.5%
Tenant Onsite Trucks	1.0%	0.5%	5.5%	0.4%	<0.1%	18.0%
SCIG CHE/TRU	0.4%	0.2%	1.3%	<0.1%	<0.1%	0.4%
Tenant Construction	0.1%	2.7%	1.4%	<0.1%	1.3%	6.2%
Emergency Generator	<0.1%	<0.1%	4.4%	<0.1%	<0.1%	0.4%
Three Rivers Underpass	<0.1%	<0.1%	1.1%	<0.1%	<0.1%	0.1%
SCIG Onsite Gasoline Vehicles	<0.1%	0.1%	<0.1%	<0.1%	<0.1%	<0.1%
Tenant Onsite Locomotives	<0.1%	<0.1%	<0.1%	<0.1%	<0.1%	<0.1%
Onsite Refueling Trucks	<0.1%	<0.1%	<0.1%	0.2%	<0.1%	<0.1%
SCIG Offsite Gasoline Vehicles	<0.1%	0.3%	<0.1%	<0.1%	0.1%	<0.1%
Tenant Offsite Gasoline Vehicles	<0.1%	0.2%	0.1%	<0.1%	0.1%	<0.1%
Tenant Onsite Gasoline Vehicles	<0.1%	<0.1%	<0.1%	<0.1%	<0.1%	<0.1%

At the maximum occupational receptor, the greatest contributors to cancer risk are SCIG offsite and onsite trucks and SCIG construction, as the receptor is located near the Highway 1 off-ramp and the entrance to the SCIG site. The greatest contributor to the chronic hazard index is SCIG construction. The greatest contributors to the acute hazard index are SCIG construction, relocated tenant onsite trucks and relocated tenant CHE emissions. Locomotives contribute approximately 2% to cancer risk, 9% to the chronic hazard index, and 4% to the acute hazard index at the maximum occupational receptor.

Table C3-7-13 presents the contributions from each TAC to the maximum health effects values for the Unmitigated Reduced Project Alternative. DPM remains the primary contributor to cancer risk at both the maximum residential and occupational receptor (greater than 97 percent). At the residential receptor, the greatest chronic hazard index contributors are DPM and chlorine while at the occupational receptor DPM drives over 98% of the chronic hazard index. The greatest acute hazard index contributor is formaldehyde.

Estimated cancer risks for the Unmitigated Reduced Project Alternative are negative and thus, no calculations of cancer burden were completed for this Alternative.

Table C3-7-13. TAC Contributions at the Residential and Occupational MEIs for the Unmitigated Reduced Project Alternative.

Pollutant	Maximum Residential Receptor			Maximum Occupational Receptor		
	Cancer Risk	Chronic Hazard Index[a]	Acute Hazard Index[a]	Cancer Risk	Chronic Hazard Index[a]	Acute Hazard Index[a]
DPM	97.6%	77.9%	0.0%	99.0%	98.8%	0.0%
Hexavalent Chromium	1.0%	<0.1%	0.0%	0.8%	<0.1%	0.0%
Formaldehyde	0.8%	7.3%	93.3%	0.1%	<0.1%	93.3%
Benzene	0.5%	0.2%	0.5%	<0.1%	<0.1%	0.5%
Nickel	<0.1%	0.8%	<0.1%	<0.1%	0.1%	<0.1%
1,3-Butadiene	<0.1%	<0.1%	0.0%	<0.1%	<0.1%	0.0%
Acetaldehyde	<0.1%	<0.1%	5.2%	<0.1%	<0.1%	5.4%
Ethylbenzene	<0.1%	<0.1%	0.0%	<0.1%	<0.1%	0.0%
Arsenic	<0.1%	<0.1%	0.2%	<0.1%	<0.1%	0.2%
Naphthalene	<0.1%	<0.1%	0.0%	<0.1%	<0.1%	0.0%
Lead	<0.1%	0.0%	0.0%	<0.1%	0.0%	0.0%
Cadmium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Chlorine	0.0%	12.8%	<0.1%	0.0%	0.8%	<0.1%
Manganese	0.0%	0.8%	0.0%	0.0%	0.2%	0.0%
Acrolein (2-Propenal)	0.0%	<0.1%	0.2%	0.0%	<0.1%	<0.1%
Propylene	0.0%	<0.1%	0.0%	0.0%	<0.1%	0.0%
Toluene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
Isomers Of Xylene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
M-Xylene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
O-Xylene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
N-Hexane	0.0%	<0.1%	0.0%	0.0%	<0.1%	0.0%
Ammonia	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
Styrene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
Methyl Alcohol	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
Mercury	0.0%	0.0%	0.4%	0.0%	0.0%	0.4%
Methyl Ethyl Ketone (MEK) (2-Butanone)	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
Copper	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
Vanadium (Fume Or Dust)	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
Vanadium	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
P-Xylene	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
Antimony	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Bromine	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Calcium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Carbon Elemental	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Carbon Organic	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Carbonate Ion	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Chromium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Cobalt	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Iron	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Nitrates	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Other	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Phosphorous	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Potassium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Selenium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%

Pollutant	Maximum Residential Receptor			Maximum Occupational Receptor		
	Cancer Risk	Chronic Hazard Index[a]	Acute Hazard Index[a]	Cancer Risk	Chronic Hazard Index[a]	Acute Hazard Index[a]
Sulfates	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Unidentified	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Unknown Pm	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Zinc	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%

a The chemical contributions for the chronic and acute hazard indices include all chemicals regardless of the target organs they affect. As a result, the contributions may add to greater than 100 percent because not all chemicals affect the same target organ.

b For diesel internal combustion engines, only DPM emissions were evaluated for cancer risk and chronic hazard indices, because DPM is a surrogate for the combined health effects associated with exposure to diesel exhaust emissions. For all other emissions (alternative fuel engines, tire and brake wear), emissions of the 47 other toxic air contaminants were evaluated for cancer and chronic hazard indices. For the acute hazard indices, DPM was not evaluated; rather, emissions of the 47 other toxic air contaminants were evaluated for all emission sources (including diesel ICEs).

7.5.1 $PM_{2.5}$ Effects

The Unmitigated Reduced Project Alternative will reduce $PM_{2.5}$ concentrations relative to the Unmitigated Proposed Project, but is still predicted to yield incremental operational 24-hour $PM_{2.5}$ emissions that will exceed the SCAQMD 24-hour $PM_{2.5}$ threshold of 2.5 $\mu g/m^3$. Because of this exceedance, incremental operational $PM_{2.5}$ concentrations meet the Port's criteria for calculating mortality and morbidity attributable to PM.

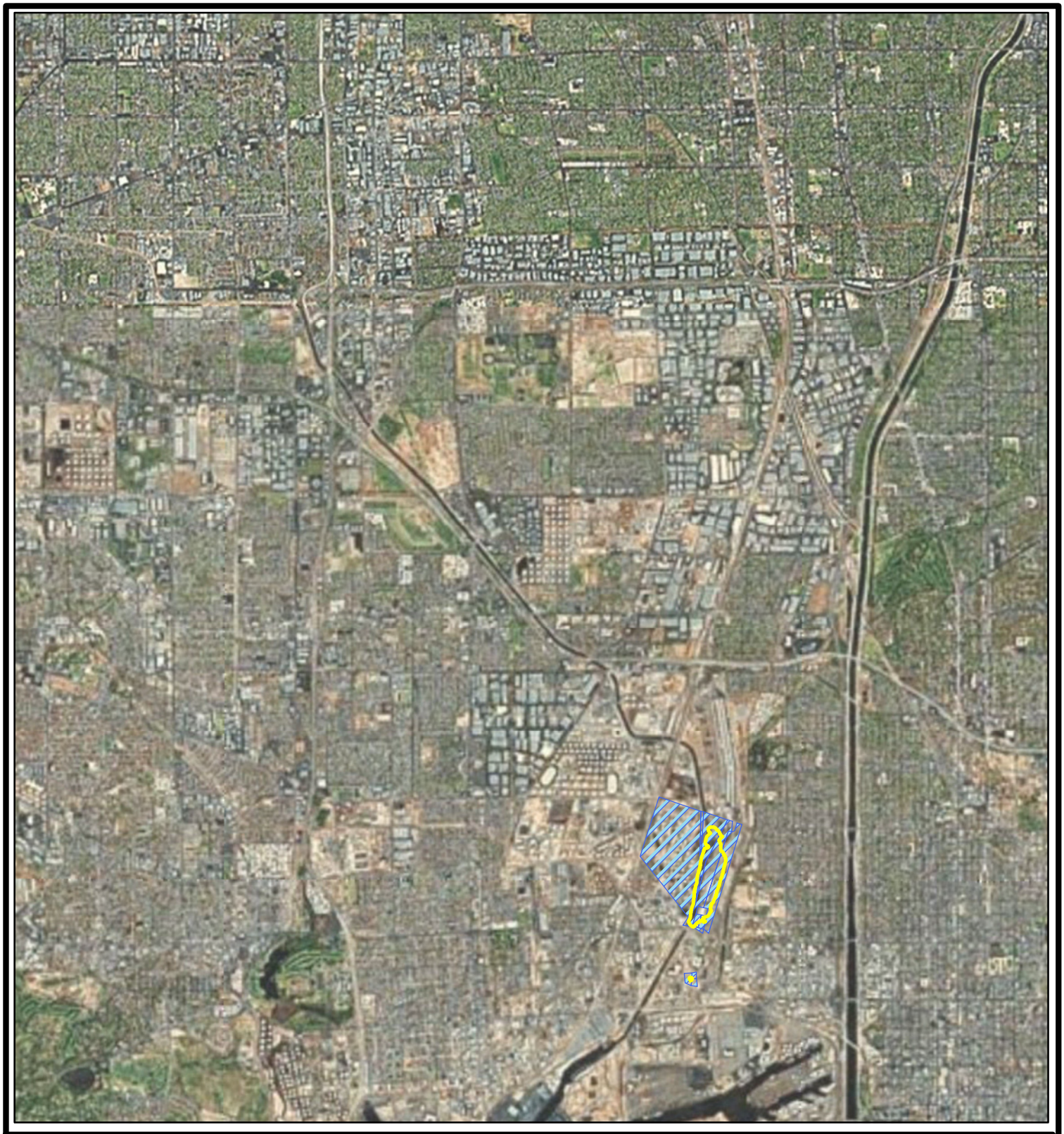
The area impacted by PM emissions from the Unmitigated Reduced Project Alternative is defined as those census blocks lying partially or completely within the project increment peak 24-h $PM_{2.5}$ $\mu g/m^3$ concentration isopleth (shown in Figure C3.7-28).

This area is similar to but smaller in geographic extent than the Unmitigated Proposed Project Area increment. As discussed with respect to the Unmitigated Proposed Project and other project Alternatives, no residential populations inhabit the census blocks of interest, and the Unmitigated Reduced Project Alternative is not expected to have an impact on PM-attributable morbidity or mortality. No calculations of mortality and morbidity were completed.

7.6 Mitigated Reduced Project Alternative Health Impacts

The Mitigated Reduced Project Alternative assumes that the Port guidelines for reducing emissions from construction equipment operating at the Port are followed; it is otherwise equivalent to the Unmitigated Reduced Project Alternative.

Table C3-7-14 presents a summary of the maximum health impacts that would occur for each receptor type with construction and operation of the Mitigated Reduced Project Alternative. The table also shows the maximum health impacts from the Baseline, as well as the CEQA increment (Mitigated Reduced Project minus Baseline). Because the results in Table C3-7-14 represent the maximum impacts predicted for each receptor type, the impacts at all other receptors would be less than these values.



Legend

- 24-Hour $PM_{2.5}$ Concentration ≥ 2.5 µg/m^3
- Census Block

N

0 0.5 1 Miles

0 0.5 1 Kilometers

Figure C3.7-28
Unmitigated Reduced Project Alternative minus Baseline

Mortality and Morbidity Applicability

DRAFT

ENVIRON

Table C3-7-14. Maximum Health Impacts Associated with the Mitigated Reduced Project Alternative.

Health Impact	Receptor Type	Maximum Predicted Impact			Significance Threshold
		Mitigated Reduced Project	CEQA Baseline	CEQA Increment	
Cancer Risk	Residential	34×10^{-6} (34 in a million)	568×10^{-6} (568 in a million)	-174×10^{-6} (-174 in a million)	10×10^{-6} (10 in a million)
	Occupational	27×10^{-6} (27 in a million)	215×10^{-6} (215 in a million)	-127×10^{-6} (-127 in a million)	
	Sensitive	29×10^{-6} (29 in a million)	220×10^{-6} (220 in a million)	-191×10^{-6} (-191 in a million)	
	Student	1.7×10^{-6} (1.7 in a million)	4.7×10^{-6} (4.7 in a million)	-3×10^{-6} (-3 in a million)	
	Recreational	42×10^{-6} (42 in a million)	329×10^{-6} (329 in a million)	-196×10^{-6} (-196 in a million)	
Chronic Hazard Index	Residential	0.06	0.36	-0.08	1.0
	Occupational	0.26	0.69	0.02	
	Sensitive	0.07	0.16	-0.09	
	Student	0.07	0.16	-0.09	
	Recreational	0.26	0.69	0.02	
Acute Hazard Index	Residential	0.16	0.29	-0.073	1.0
	Occupational	0.59	0.79	0.08	
	Sensitive	0.18	0.27	-0.09	
	Student	0.18	0.27	-0.09	
	Recreational	0.59	0.79	0.08	

a Exceedances of the significance thresholds are in bold. The significance thresholds apply to the CEQA increments only.

b The maximum increments might not occur at the same receptor locations as the maximum impacts. This means that the increments cannot necessarily be determined by subtracting the CEQA Baseline impact from the project impact. Rather, the subtraction must be done at each receptor, for all modeled receptors, and the maximum result selected.

c The CEQA Increment represents Project minus CEQA Baseline.

d When the maximum increment for a receptor type is negative, the maximum increment displayed is the increment at the maximum project receptor location.

e Data represent the receptor locations with the maximum impacts or increments. The impacts or increments at all other modeled receptors would be less than these values for each receptor type.

f The Mitigated Reduced Project Alternative assumes that the Port guidelines for reducing emissions from construction equipment operating at the Port are followed; it is otherwise equivalent to the Unmitigated Reduced Project Alternative.

The data in Table C3-7-14 show that the CEQA cancer risk increment at the location of the Mitigated Reduced Project Alternative MEI is predicated to be -174 in a million (-174×10^{-6}), at a residential receptor. This risk value, as well as the risk value at all residential receptors, is below the significance threshold of 10 in a million. The CEQA increments would be below the CEQA significance threshold at all receptors, including occupational, sensitive, student, and recreational.

The maximum chronic hazard index increments are predicted to be less than the CEQA significance of 1.0 at all receptors.

The maximum acute hazard index increments are predicted to be less than the CEQA significance threshold of 1.0 at each receptor type.

Table C3-7-15 presents the contributions from each emission source to the maximum health effects impacts for the Mitigated Reduced Project Alternative. At the maximum

residential receptor, the greatest contributors to cancer risk are SCIG offsite and onsite trucks. The greatest contributors to the chronic hazard index are SCIG offsite and onsite trucks, Hostler emissions, and SCIG construction. The greatest contributor to the acute hazard index is SCIG construction. Locomotives contribute between 3-5% by health effect at the maximum residential receptor.

Table C3-7-15. Source Contributions at the Residential and Occupational MEIs for the Mitigated Reduced Project Alternative.

Emission Source	Maximum Residential Receptor			Maximum Occupational Receptor		
	Cancer Risk	Chronic Hazard Index	Acute Hazard Index	Cancer Risk	Chronic Hazard Index	Acute Hazard Index
SCIG Offsite Trucks	53.6%	24.5%	3.1%	74.0%	10.5%	2.5%
SCIG Onsite Trucks	34.2%	19.9%	8.2%	15.8%	4.1%	3.1%
Tenant CHE	2.4%	6.1%	8.6%	1.6%	1.2%	16.0%
Tenant Offsite Trucks	2.2%	7.5%	5.6%	1.8%	2.3%	4.8%
SCIG Onsite Locomotives	2.1%	2.2%	2.3%	1.3%	18.0%	3.7%
Hostler	1.5%	20.2%	5.6%	0.2%	0.6%	1.9%
SCIG Offsite Locomotives	1.2%	2.7%	0.6%	0.5%	4.1%	0.7%
Tenant Onsite Trucks	1.0%	0.5%	6.4%	0.4%	0.1%	21.5%
SCIG Construction	0.8%	12.0%	50.2%	3.8%	56.8%	40.7%
SCIG CHE/TRU	0.4%	0.2%	1.5%	<0.1%	<0.1%	0.4%
Tenant Construction	0.1%	2.7%	1.3%	<0.1%	1.4%	3.8%
Emergency Generator	<0.1%	<0.1%	5.1%	<0.1%	<0.1%	0.5%
Three Rivers Underpass	<0.1%	<0.1%	1.2%	<0.1%	<0.1%	0.1%
SCIG Onsite Gasoline Vehicles	<0.1%	0.1%	<0.1%	<0.1%	<0.1%	<0.1%
Tenant Onsite Locomotives	<0.1%	<0.1%	<0.1%	<0.1%	<0.1%	<0.1%
Onsite Refueling Trucks	<0.1%	<0.1%	<0.1%	0.2%	<0.1%	<0.1%
SCIG Offsite Gasoline Vehicles	<0.1%	0.6%	<0.1%	<0.1%	0.4%	<0.1%
Tenant Offsite Gasoline Vehicles	<0.1%	0.4%	0.2%	<0.1%	0.3%	<0.1%
Tenant Onsite Gasoline Vehicles	<0.1%	<0.1%	<0.1%	<0.1%	<0.1%	<0.1%

At the maximum occupational receptor, the greatest contributors to cancer risk are SCIG offsite and onsite trucks. The greatest contributors to the chronic hazard index are SCIG construction, SCIG onsite locomotives, and SCIG offsite trucks. The greatest contributors to the acute hazard index are SCIG construction, relocated tenant onsite trucks, and relocated tenant CHE emissions. SCIG onsite and offsite locomotives contribute 18% and 4%, respectively, to the chronic hazard index at the maximum occupational receptor; locomotives also contribute approximately 2% to cancer risk and 4.5% to acute hazard index.

Table C3-7-16 presents the contributions from each TAC to the maximum health effects values for the Mitigated Reduced Project Alternative. DPM remains the primary contributor to cancer risk at both the maximum residential and occupational receptor (greater than 97 percent). The greatest chronic hazard index contributors are DPM and chlorine at the maximum residential receptor, while DPM alone contributes approximately 97% of the chronic hazard index at the maximum occupational receptor. The greatest acute hazard index contributor is formaldehyde at both the maximum residential and occupational receptors.

Estimated cancer risks for the Mitigated Reduced Project Alternative are negative. As a result, no calculations of cancer burden were completed for this Alternative.

Table C3-7-16. TAC Contributions at the Residential and Occupational MEIs for the Mitigated Reduced Project Alternative.

Pollutant	Maximum Residential Receptor			Maximum Occupational Receptor		
	Cancer Risk	Chronic Hazard Index[a]	Acute Hazard Index[a]	Cancer Risk	Chronic Hazard Index[a]	Acute Hazard Index[a]
DPM	97.5%	73.1%	0.0%	98.9%	96.9%	0.0%
Hexavalent Chromium	1.1%	<0.1%	0.0%	0.8%	<0.1%	0.0%
Formaldehyde	0.8%	8.0%	93.5%	0.1%	0.3%	93.4%
Benzene	0.5%	0.2%	0.5%	<0.1%	<0.1%	0.5%
Nickel	<0.1%	1.2%	<0.1%	<0.1%	0.3%	<0.1%
1,3-Butadiene	<0.1%	<0.1%	0.0%	<0.1%	<0.1%	0.0%
Acetaldehyde	<0.1%	<0.1%	5.2%	<0.1%	<0.1%	5.4%
Ethylbenzene	<0.1%	<0.1%	0.0%	<0.1%	<0.1%	0.0%
Arsenic	<0.1%	<0.1%	0.1%	<0.1%	<0.1%	0.1%
Naphthalene	<0.1%	<0.1%	0.0%	<0.1%	<0.1%	0.0%
Lead	<0.1%	0.0%	0.0%	<0.1%	0.0%	0.0%
Cadmium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Chlorine	0.0%	15.8%	<0.1%	0.0%	2.0%	<0.1%
Manganese	0.0%	1.4%	0.0%	0.0%	0.4%	0.0%
Acrolein (2-Propenal)	0.0%	<0.1%	0.2%	0.0%	<0.1%	<0.1%
Propylene	0.0%	<0.1%	0.0%	0.0%	<0.1%	0.0%
Toluene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
Isomers Of Xylene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
M-Xylene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
O-Xylene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
N-Hexane	0.0%	<0.1%	0.0%	0.0%	<0.1%	0.0%
Ammonia	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
Styrene	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
Methyl Alcohol	0.0%	<0.1%	<0.1%	0.0%	<0.1%	<0.1%
Mercury	0.0%	0.0%	0.2%	0.0%	0.0%	0.3%
Methyl Ethyl Ketone (MEK) (2-Butanone)	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
Copper	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
Vanadium	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
Vanadium (Fume Or Dust)	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
P-Xylene	0.0%	0.0%	<0.1%	0.0%	0.0%	<0.1%
Antimony	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Bromine	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Calcium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Carbon Elemental	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Carbon Organic	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Carbonate Ion	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Chromium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Cobalt	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Iron	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Nitrates	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Other	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Phosphorous	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Potassium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Selenium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Sulfates	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Unidentified	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Unknown Pm	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%

Pollutant	Maximum Residential Receptor			Maximum Occupational Receptor		
	Cancer Risk	Chronic Hazard Index[a]	Acute Hazard Index[a]	Cancer Risk	Chronic Hazard Index[a]	Acute Hazard Index[a]
Zinc	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%

a The chemical contributions for the chronic and acute hazard indices include all chemicals regardless of the target organs they affect. As a result, the contributions may add to greater than 100 percent because not all chemicals affect the same target organ.

b For diesel internal combustion engines, only DPM emissions were evaluated for cancer risk and chronic hazard indices, because DPM is a surrogate for the combined health effects associated with exposure to diesel exhaust emissions. For all other emissions (alternative fuel engines, tire and brake wear), emissions of the 47 other toxic air contaminants were evaluated for cancer and chronic hazard indices. For the acute hazard indices, DPM was not evaluated; rather, emissions of the 47 other toxic air contaminants were evaluated for all emission sources (including diesel ICEs).

7.6.1 $PM_{2.5}$ Effects

The Mitigated Reduced Project Alternative will reduce $PM_{2.5}$ concentrations relative to the Unmitigated Reduced Project Alternative, but will still result in incremental (project minus Baseline) 24-hour $PM_{2.5}$ emissions predicted to exceed the SCAQMD 24-hour $PM_{2.5}$ threshold of 2.5 $\mu g/m^3$. Accordingly, operational $PM_{2.5}$ concentrations for the Mitigated Reduced Project Alternative increment meet the Port's criteria for calculating mortality and morbidity attributable to PM (POLA, 2011).

The area impacted by PM emissions from the Mitigated Reduced Project Alternative increment (Figure C2.5-31 from Appendix C2) is similar to that of the Unmitigated Reduced Project Alternative increment, although the impacted area is smaller in geographic extent (consistent with the reduced emissions). Census blocks lying partially or completely within the project increment peak 24-h $PM_{2.5}$ $\mu g/m^3$ concentration isopleths represent the area identified for analysis of PM-attributable mortality and morbidity. Consistent with the information provided in the discussions of the Unmitigated Proposed Project as well as the Reduced Project Alternatives, no residential populations inhabit the impacted census blocks. Consequently, the Mitigated Reduced Project Alternative is not expected to have an impact on PM-attributable morbidity or mortality, and no calculations of mortality and morbidity were completed.

8.0 Risk Uncertainty

Health risk assessments such as the one presented in this Appendix are not intended to provide estimates of the absolute health risk or expected incidence of disease in a population, but instead, are conducted to allow comparisons of the potential health impacts of different alternatives. Consistent with agency guidelines and standard approaches to regulatory risk assessment, this risk assessment used health-protective (conservative) assumptions selected by regulatory agencies to "err on the side of health protection in order to avoid underestimation of risk to the public" (OEHHA, 2003). As an example of the conservative assumptions used in this risk assessment, residential receptors are considered to be exposed to TACs while individuals are present at the same outdoor location for 365 days per year for 70 years, breathing continuously at a rate that is at the 80th percentile of breathing rates for the population.

OEHHA has provided a discussion of risk uncertainty, which is reiterated here (OEHHA, 2003).

There is a great deal of uncertainty associated with the process of risk assessment. The uncertainty arises from lack of data in many areas necessitating the use of assumptions. The assumptions used in these guidelines are designed to err on the side of health protection in order to avoid underestimation of risk to the public. Sources of uncertainty, which may either overestimate or underestimate risk, include: 1) extrapolation of toxicity data in animals to humans, 2) uncertainty in the estimation of emissions, 3) uncertainty in the air dispersion models, and 4) uncertainty in the exposure estimates. Uncertainty may be defined as what is not known and may be reduced with further scientific studies. In addition to uncertainty, there is a natural range or variability in the human population in such properties as height, weight, and susceptibility to chemical toxicants. Scientific studies with representative individuals and large enough sample size can characterize this variability.

Interactive effects of exposure to more than one carcinogen or toxicant are also not necessarily quantified in the HRA. Cancer risks from all emitted carcinogens are typically added, and hazard quotients for substances impacting the same target organ system are added to determine the hazard index (HI). Many examples of additivity and synergism (interactive effects greater than additive) are known. For substances that act synergistically, the HRA could underestimate the risks. Some substances may have antagonistic effects (lessen the toxic effects produced by another substance). For substances that act antagonistically, the HRA could overestimate the risks.

Other sources of uncertainty, which may underestimate or overestimate risk, can be found in exposure estimates where little or no data are available (e.g., soil half-life and dermal penetration of some substances from a soil matrix).

The differences among species and within human populations usually cannot be easily quantified and incorporated into risk assessments. Factors including metabolism, target site sensitivity, diet, immunological responses, and genetics may influence the response to toxicants. The human population is much more diverse both genetically and culturally (e.g., lifestyle, diet) than inbred experimental animals. The intraspecies variability among humans is expected to be much greater than in laboratory animals. Adjustment for tumors at multiple sites induced by some carcinogens could result in a higher potency. Other uncertainties arise 1) in the assumptions underlying the dose-response model used, and 2) in extrapolating from large experimental doses, where, for example, other toxic effects may compromise the assessment of carcinogenic potential, to usually much smaller environmental doses. Also, only single tumor sites induced by a substance are usually considered. When epidemiological data are used to generate a carcinogenic potency, less uncertainty is involved in the extrapolation from workplace exposures to environmental exposures. However, children, a subpopulation whose hematological, nervous, endocrine, and immune systems, for example, are still developing and who may be more sensitive to the effects of carcinogens on their developing systems, are not included in the worker population and risk estimates based on

occupational epidemiological data are more uncertain for children than adults. Finally, the quantification of each uncertainty applied in the estimate of cancer potency is itself uncertain.

Thus, risk estimates generated by an HRA should not be interpreted as the expected rates of disease in the exposed population but rather as estimates of potential risk, based on current knowledge and a number of assumptions. Additionally, the uncertainty factors integrated within the estimates of non-cancer RELs are meant to err on the side of public health protection in order to avoid underestimation of risk. Risk assessment is best used as a ruler to compare one source with another and to prioritize concerns. Consistent approaches to risk assessment are necessary to fulfill this function.

9.0 References

California Air Resources Board (CARB). 2011. "PM and Organic Gas Speciation Profiles." Available online at: http://www.arb.ca.gov/ei/speciate/dnldopt.htm. March 9.

_____2010. *Workshops on Information Regarding the Off-Road, Truck and Bus and Drayage Truck Regulations*. September 3.

_____2008. Methodology for Estimating Premature Deaths Associated with Long-term Exposure to Fine Airborne Particulate Matter in California. October 24, 2008. California Air Resources Board. California Environmental Protection Agency.

_____2007. *User's Guide for OFFROAD2007.* November.

_____2006a. *ARB Health Risk Assessment Guidance for Rail Yard and Intermodal Facilities.* September.

_____2006b. EMFAC2007 version 2.30. *Calculating emission inventories for vehicles in California.* User's Guide. November.

_____2006c. *Emission Reduction Plan for Ports and International Goods Movement*. January 30. Website: www.arb.ca.gov/planning/gmerp/gmerp.htm.

_____2004a. *Recommended Interim Risk Management Policy*. Web site: http://www.arb.ca.gov/toxics/harp/rmpolicyfaq.htm.

_____2004b. *The California Diesel Fuel Regulations*. Title 13, California Code of Regulations, Sections 2281-2285; Title 17, California Code of Regulations, Section 93114. August 14.

_____2004c. *Roseville Rail Yard Study.* Stationary Source Division. October 14.

_____2003. Recommended Interim Risk Management Policy for Inhalation-Based Residential Cancer Risk. October 9.

_____2000a. *Risk Reduction Plan to Reduce Particular Matter Emissions from Diesel-Fueled Engines and Vehicles.* Stationary Source Division and Mobile Source Control Division. October. Web site: http://www.arb.ca.gov/diesel/documents/rrpfinal.pdf

_____2002b. Staff Report: Public Hearing to Consider Amendments to the Ambient Air Quality Standards for Particulate Matter and Sulfates. Prepared by CARB and OEHHA. May 3.Clark, N. A., P. A. Demers, C. J. Karr, M. Koehoorn, C. Lencar, L. Tamburic, and M. Brauer. 2010. "Effect of Early Life Exposure to Air Pollution on Development of Childhood Asthma." Environmental Health Perspectives. Vol. 118, No. 2. pp. 284-290.

Gauderman, W. J., H. Vora, R. McConnell, K. Berhane, F. Gilliland, D. Thomas, F. Lurmann, E. Avol, N. Kunzli, M. Jerrett, and J. Peters. 2007. Effect of exposure to traffic on lung development from 10 to18 years of age: a cohort study. Lancet. 369. pp. 571-577.

Jerrett, M., R.T. Burnett, R. Ma, C.A. Pope III, D. Krewski, K.B. Newbold, G. Thurston, Y. Shi, N. Finkelstein, E.E. Calle, and M.J. Thun. 2005. Spatial analysis of air pollution and mortality in Los Angeles. Epidemiology 16 (6):727-36.

Jerrett, M., K. Shankardass, K. Berhane, W. J. Gauderman, N. Künzli, E. Avol, F. Gilliland, F. Lurmann, J. N. Molitor, J. T. Molitor, D. C. Thomas, J. Peters, and R. McConnell. 2008. Traffic-Related Air Pollution and Asthma Onset in Children: A Prospective Cohort Study with Individual Exposure Measurement. Environmental Health Perspectives. Vol. 116, No. 10. pp. 1433-1438.

Krewski, D., R. Burnett, M.S. Goldberg, K. Koover, J. Siemiatycki, M. Jerrett, et al. 2001. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Research Report of the Health Effects Institute.

Office of Environmental Health Hazard Assessment (OEHHA). 2008. All OEHHA Acute, 8-hour and Chronic Reference Exposure Levels (chRELs) as on December 18, 2008. http://oehha.ca.gov/air/allrels.html

_____2003. Air Toxics Hot Spots Program Risk Assessment Guidelines. *The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments.* August.

_____2002. Air Toxics Hot Spots ProgramRisk Assessment Guidelines Part III. Technical Support Document for the Determination of Non-cancer Chronic Reference Exposure Levels. Air Toxicology and Epidemiology Section Office of Environmental Health Hazard Assessment,California Environmental Protection Agency. February 2000.

Pandya, R. J., G. Solomon, A. Kinner, and J. R. Balmes. 2002. Diesel Exhaust and Asthma: Hypotheses and Molecular Mechanisms of Action. Environmental Health Perspectives. Vol. 110, Supplement 1. pp. 104-112.

Pope, C.A., R. T. Burnett, G. D. Thurston, M. J. Thun, E. E. Calle, D. Krewski, and J. J. Godleski. 2004. Cardiovascular Mortality and Long-Term Exposure to Particulate Air Pollution: Epidemiological Evidence of General Pathophysiological Pathways of Disease. Circulation. 109: 71-77.

Pope, C.A., R.T. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito, and G. Thurston. 2002. Lung Cancer, Cardiopulmonary Mortality, and Long-Term Exposure to Fine Particulate Air Pollution. JAMA. 287: 1123-1141.

Pope, C.A., M.J. Thun, M.M. Namboodiri, D.W. Dockery, J.S. Evans, F.E. Speizer, and C.W. Health. 1995. Particulate Air Pollution as a Predictor of Mortality in a Prospective Study of U.S. Adults. American Journal of Respiratory and Critical Care Medicine. 151: 669-674.

Port of Los Angeles (POLA), 2011. *Methodology for Addressing Mortality and Morbidity in Port of Los Angeles CEQA Documents*. July 22, 2011.

Port of Los Angeles and Port of Long Beach (POLA/POLB). 2006. *Final San Pedro Bay Ports Clean Air Action Plan. http://www.portoflosangeles.org/CAAP/CAAP_Tech_Report_Final.pdf*

_____2010. *Final 2010 San Pedro Bay Ports Clean Air Action Plan Update*. Attachment I to Appendix B - Sphere of Influence Bay-Wide Sphere of Influence Analysis for Surface Meteorological Stations Near the Ports. Web site: http://www.cleanairactionplan.org/civica/filebank/blobdload.asp?BlobID=2439.

_____Port of Los Angeles (POLA). 2011. Methodology for Addressing Mortality and Morbidity in Port of Los Angeles CEQA Documents, Port of Los Angeles, 2011. August.

_____2008. Preliminary Draft Protocol for Air Emission Modeling and Human Health Risk Assessment for the Southern California International Gateway. Prepared by ENVIRON International Corporation, Novato, CA and Emeryville, CA. January 2008

_____2005. *Health Risk Assessment Protocol for Port of Los Angeles Terminal Improvement Projects.* June 27.

_____2004. *Final Air Quality Monitoring Work Plan for the Port of Los Angeles*.

South Coast Air Quality Management District(SCAQMD). 2011. SCAQMD Air Quality Significance Thresholds. http://www.aqmd.gov/ceqa/handbook/signthres.pdf

_____2005. Supplemental Guidelines for Preparing Risk Assessments for the Air Toxics "Hot Spots" Information and Assessment Act (AB2588). July.

_____2003. Health Risk Assessment Guidance for Analyzing Cancer Risks from Mobile Source Diesel Idling Emissions for CEQA Air Quality Analysis. August 2003.

_____2002. *Health Risk Assessment Guidance for Analyzing Cancer Risks from Mobile Source Diesel Emissions.*

Starcrest, 2007 Port of Los Angeles Inventory of Air Emissions 2005, prepared by Starcrest Consulting Group LLC for the Port of Los Angeles. September.U.S. Environmental Protection Agency (USEPA). IRIS Glossary http://www.epa.gov/iris/help_gloss.htm#s Updated May 2 2011; accessed June 3 2011.

_____2004. *User's Guide for the AMS/EPA Regulatory Model – AERMOD*. Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina. EPA-454/B-03/001.

_____1997. *Exposure Factors Handbook*. August.

_____1998. Locomotive Emission Standards. Regulatory Support Document. Office of Mobile Sources. April.