Appendix B3

Health Risk Assessment

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1.0 Introduction

This appendix describes the methods and results of a health risk assessment (HRA) that evaluates potential public health effects from toxic air contaminant (TAC) emissions that would be generated during the construction and operation of the proposed Project and alternatives for the Shell MOTEMS project at Berths 167-169. 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 risks associated with the following scenarios:

- Proposed Project construction and operation;
- Reduced Project Alternative (Alternative 2) construction and operation; and
- No Project Alternative (Alternative 1) operation.

The HRA was prepared as a Tier 1 risk assessment in accordance with OEHHA's *Guidance Manual for Preparation of Health Risk Assessments* (OEHHA, 2015) and the SCAQMD's *Supplemental Guidelines for Preparing Risk Assessments for the Air Toxics "Hot Spots" Information and Assessment Act* (SCAQMD, 2015a). The HRA includes an evaluation of four different types of health effects: individual cancer risk, population cancer burden, chronic noncancer hazard index, and acute noncancer hazard index.

- Individual cancer risk is the additional chance for a person to contract cancer after long-term exposure to project emissions (for example, 30 years for a resident and 25 years for a worker).

- Population cancer burden is the expected number of additional cancer cases in the population exposed to an individual cancer risk of 1 in a million or greater from the project, based on 70 years of exposure.

- The chronic hazard index is a ratio of annual average concentrations of TACs in the air to established reference exposure levels. A chronic hazard index below 1.0 indicates that adverse noncancer health effects from long-term exposure are not expected.

- The acute hazard index is a ratio of maximum 1-hour average concentrations of TACs in the air to established reference exposure levels. An acute hazard index below 1.0 indicates that adverse noncancer health effects from infrequent short-term exposure are not expected.

The OEHHA HRA guidelines also provide a methodology for determining an 8-hour chronic hazard index, which evaluates repeated 8-hour exposures over a significant fraction of a lifetime (OEHHA, 2015). This health value is applicable primarily to off-site workers with work schedules that align with the emitting facility's operational schedule. Because the Shell terminal operates continuously, the average 8-hour concentrations to which off-site workers would be exposed would not be substantially different than the annual concentrations used to calculate the chronic hazard index. Moreover, the toxicity factors for the 8-hour chronic hazard index are generally less stringent and apply to fewer TACs than the toxicity factors for the chronic hazard index. As a result, the 8-hour chronic hazard indices associated with the proposed Project and alternatives would be less than the chronic

hazard indices. Therefore, this HRA does not quantify 8-hour chronic hazard indices, and instead uses chronic hazard indices as a conservative health value for off-site workers.

The USEPA dispersion model AERMOD (USEPA, 2015; 2017a) was used to predict maximum ambient pollutant concentrations outside the Project site. The Hotspots Analysis and Reporting Program (HARP2) (CARB, 2016a; 2017a) was used to perform the health risk calculations based on output from the AERMOD dispersion model.

The HRA was developed using a five-step process to estimate incremental health impact results: (1) quantify proposed Project, alternative, and baseline emissions; (2) identify ground-level receptor locations that may be affected by emissions, including a regular receptor grid as well as specific sensitive receptor locations nearby such as grade schools, hospitals, elder care facilities, or child care facilities; (3) perform dispersion modeling analyses to estimate ambient TAC concentrations at each receptor location; (4) characterize the potential health risks at each receptor location; and (5) evaluate incremental health risk values by comparing potential health risks posed by the proposed Project and alternatives relative to CEQA baseline. The following sections provide additional details on the methods used to complete the HRA.

2.0 Emission Estimation Approach

The following construction emission sources were included in the HRA:

- Off-road equipment: land-based and dredging equipment;
- On-road trucks: driving and idling on-site;
- Harborcraft: tugboats used to position dredging barges and scows while adjacent to the terminal;
- Source control program: the refurbishment of existing storage tanks. Modeled sources include off-road construction equipment, storage tank degassing, and a thermal oxidizer for vapor treatment during degassing.

In accordance with SCAQMD guidance (SCAQMD, 2005), construction emission sources were modeled with their on-site emissions only, which include emissions within the terminal and adjacent waters.

The following operational emission sources were included in the HRA:

- Tanker ships transiting between the SCAQMD overwater boundary and the terminal (about 40 nautical miles), anchoring while waiting for an available berth, and hoteling while at berth. Tanker emission sources include propulsion engines, auxiliary engines, and boilers.
- Integrated tug-barges (ITBs)/articulated tug-barges (ATBs) transiting between the SCAQMD overwater boundary and the terminal (about 40 nautical miles), anchoring while waiting for an available berth, and hoteling while at berth. ITB/ATB emission sources include propulsion and auxiliary engines.

- Tugboats used to assist tankers and ITBs/ATBs while arriving and departing the Port, between the berths and breakwater. Tugboat emission sources include propulsion and auxiliary engines.

- Fugitive VOC emissions from on-site storage tanks and associated piping.

- Fugitive VOC and vapor destruction unit (VDU) combustion emissions from future vessel loading activities. These sources do not pertain to the baseline scenario because product was not loaded onto vessels in the baseline condition.

2.1 Emissions Used for Cancer Risk

To estimate cancer risk impacts for the proposed Project and alternatives, annual VOC and PM_{10} emissions from construction and operation were estimated for each year of several long-term exposure periods. The HRA assumed exposure periods of 30 years for residential and sensitive receptors; 25 years for occupational receptors; and 70 years for population cancer burden. For the proposed Project and alternatives, the first year of each exposure period was assumed to be 2019, the anticipated first complete year of proposed Project construction and overlapping operation. For example, the 30-year residential exposure period was assumed to occur during the years 2019-2048.

Annual emissions were estimated using the methodology and assumptions described in Section 3.1.4.1 of the EIR and Sections 1.2 and 1.4 of Appendix B2. The emissions account for the future growth in product throughput and associated vessel calls, and the future reduction in emission factors for construction equipment and tugboats as required by existing regulations and assumptions regarding equipment fleet turnover. Annual construction emissions were calculated for each year of construction (2018-2023). For the proposed Project and Reduced Project Alternative (Alternative 2), all construction emissions were conservatively assumed to occur within the first 5 years of the cancer risk exposure period, even those construction emissions that may begin prior to 2019. Annual operational emissions were calculated directly for years 2019, 2023 (Alternative 1 - No Project Alternative only), 2031, and 2048. Emissions for intermediate years were interpolated. Operational emissions beyond 2048 were assumed to remain constant at 2048 levels.

To better apprise the public and decision makers of the cancer risk impacts under CEQA, the predicted cancer risks for the proposed Project and alternatives were compared to both a CEQA baseline and a future CEQA baseline. For both baselines, the first year of every exposure period was assumed to be 2015. The CEQA baseline cancer risk was evaluated using average 2011 – 2015 activity levels and 2015 emission factors for each year of every exposure period. In other words, the CEQA baseline was evaluated with constant emissions during every exposure period.

The future CEQA baseline cancer risk also uses average 2011-2015 activity levels for each year of every exposure period, but the emission factors vary year-by-year, starting with 2015, to account for the future beneficial effects of existing air quality regulations. For example, the 30-year residential exposure period for the future CEQA baseline uses average 2011-2015 activity levels applied to year-by-year emission factors that vary by year from 2015-2044. In this study, the future CEQA baseline cancer risk is slightly lower than the CEQA baseline cancer risk, resulting in a higher project increment, because the

tugboat emission factors decline starting in 2023 in response to existing air quality regulations and assumptions regarding equipment fleet turnover.

The use of both the CEQA baseline and future CEQA baseline helps to resolve the complexity of evaluating a fixed point in time (the baseline condition) over decades-long exposure periods. This issue does not exist for the chronic and acute hazard indices because they are based on modeled TAC concentrations of one year and one hour, respectively, which fit entirely within the baseline period. Therefore, the future CEQA baseline is not necessary for the evaluation of chronic and acute hazard indices; the CEQA baseline by itself is adequate.

2.2 Emissions Used for Non-Cancer Hazard Indices

To estimate chronic and acute noncancer hazard indices for the proposed Project and alternatives, annual and peak hour construction emissions of VOC and PM_{10} were calculated for each year of construction (2018-2023). Annual and peak hour operational emissions were calculated directly for years 2019, 2023 (Alternative 1 - No Project Alternative only), 2031, and 2048. The emissions were estimated using the methodology and assumptions described in Section 3.1.4.1 of the EIR and Sections 1.2 and 1.4 of Appendix B2. Because prior Port projects have shown that the chronic and acute hazard indexes are unlikely to exceed the significance thresholds, a conservative screening approach was used where each AERMOD source was modeled with its maximum construction and operational emissions from these analysis years even if the emissions would not occur at the same time.

To estimate chronic and acute noncancer hazard indices for the CEQA baseline, annual and peak hour emissions of VOC and PM_{10} were calculated using 2011 – 2015 activity levels and 2015 emission factors.

Appendix B1 of this EIR documents the overall emission calculation methodology and assumptions for the proposed Project, project alternatives, and CEQA baseline.

2.3 TAC Speciation

Diesel internal combustion (IC) engines represent the biggest source of TAC emissions associated with the proposed Project and alternatives. Diesel IC engines include off-road construction equipment, most on-road trucks, tanker and ITB/ATB propulsion and auxiliary engines, and harborcraft. For the determination of cancer risk and chronic hazard indices, OEHHA and CARB use diesel particulate matter (DPM) from IC engines as a surrogate for total diesel exhaust (CARB, 2017b). The toxicity factors for DPM that were established by OEHHA and CARB account for the individual toxic species contained in total diesel IC engine exhaust. Therefore, diesel IC engine exhaust was not speciated into its chemical components for the determination of cancer risk and chronic noncancer hazard indices.

Sources other than diesel IC engines include tanker boilers, vehicle tire and brake wear, the VDU and thermal oxidizer, storage tank and piping vapors, and vessel loading fugitives. For these sources, VOC and PM_{10} emissions were speciated into their individual TAC components for the determination of cancer risk and chronic hazard indices. The speciation profiles used in the HRA were developed by CARB (2016b). Table B3-1

presents the speciation profiles that were used to convert PM_{10} emissions into individual TACs. Table B3-2 presents the speciation profiles that were used to convert VOC emissions into individual TACs.

OEHHA and CARB have not established acute toxicity factors for DPM. Therefore, peak hour VOC and PM_{10} emissions from all sources, including diesel IC engines, were speciated into their individual TAC components for the determination of acute hazard indices.

Table B3-1. Speciation Profiles for PM_{10}

Toxic Air Contaminant [c]	HARP2 TAC ID	Speciation Profile ID and TAC Weight Fraction [d]							
		Profile 42514: Diesel Vehicles [a]	Profile 119: Marine Vessels Liquid Fuel [a]	Profile 4251: Marine Vessels MGO [a]	Profile 116: Diesel IC Engine [a]	Profile 473: Brake Wear	Profile 112: Fuel Combustion Distillate	Profile 162: Incineration Gaseous Fuel	Profile 472: Tire Wear
Arsenic	7440382	0	0	0	0	0.00001	0.005418	0	0
Cadmium	7440439	0	0	0	0	0	0.0005	0	0
Chlorine	7782505	0	0	0	0	0.0015	0	0	0.0078
Copper	7440508	0.000356	0	0	0	0.011485	0	0	0.00049
Hexavalent Chromium [b]	18540299	0.00003035	0	0	0	0.00006	0.0002709	0	0.0000015
Lead	7439921	0	0	0	0	0.00005	0.0055	0	0.00016
Manganese	7439965	0	0	0	0	0.0017	0	0	0.0001
Nickel	7440020	0	0	0	0	0.00066	0.0005	0	0.00005
Selenium	7782492	0	0	0	0	0.00002	0.0005	0	0.00002
Sulfates	9960	0.285538	0.15	0.08	0.15	0.0334	0.25	0.2	0.0025
Vanadium	7440622	0	0.0055	0	0.0055	0.00066	0	0	0
Applicable sources:		On-road trucks, off-road equipment	Harborcraft, ITB/ATB main engines	Tanker main and auxiliary engines	ITB/ATB auxiliary engines	Brake Wear	Tanker boilers	Thermal oxidizer, VDU	Tire wear

Notes:
a. Profiles No. 42514, 119, 4251, and 116 are associated with diesel IC engines and therefore were only used for the determination of the acute hazard index. For the determination of cancer risk and the chronic hazard index, DPM emissions were used without speciation because CARB provides toxicity factors for DPM as a whole (CARB, 2017b).
b. Hexavalent chromium is assumed to be 5 percent of total chromium, according to CARB's AB2588 Technical Support Document (CARB, 1989), page 57.
c. Only TACs that have OEHHA/CARB toxicity factors are shown in the table.
d. Source for speciation profiles: CARB, 2016b.

Table B3-2. Speciation Profiles for VOC

Toxic Air Contaminant [c]	HARP2 TAC ID	Speciation Profile and Weight Fraction [b,d]			
		Profile 818: Diesel IC Engines [a]	Profile 316: Petroleum Vapors	Profile 504: Boilers	Profile 79: Flares
Acetaldehyde	75070	0.083665	0	0	0.007
Acrylonitrile	107131	0	0	0	0.03
Benzene	71432	0.022766	0.001524	0.022833	0.1
1,3-Butadiene	106990	0.002163	0	0	0
Chlorobenzene	108907	0	0	0.000529	0
Ethyl benzene	100414	0.003529	0	0.00074	0
Ethyl chloride	75003	0	0	0	0.072
Ethylene oxide	75218	0	0	0	0.046
Formaldehyde	50000	0.167445	0	0.001057	0.017
Hexane	110543	0.001821	0.051829	0.016808	0
Isopropyl alcohol	67630	0	0	0	0.025
Methanol	67561	0.000341	0	0	0.054
Methyl ethyl ketone	78933	0.016847	0	0	0
Naphthalene	91203	0.001024	0	0.00074	0
Phenol	108952	0	0	0	0.02
Propylene	115071	0.029596	0.001524	0.048203	0.09
Propylene oxide	75569	0	0	0	0.014
Styrene	100425	0.000683	0	0	0.034
Toluene	108883	0.016733	0.007622	0.022727	0.041
Vinyl chloride	75014	0	0	0	0.006
Xylenes	1330207	0.011952	0.003049	0.011628	0.013
Applicable sources:		On-road trucks, off-road equipment, harborcraft, ITB/ATB main and auxiliary engines, tanker main and auxiliary engines	Storage tanks and piping vapors, vessel loading vapors	Tanker boilers	Thermal oxidizer, VDU

Notes:
a. Profile No. 818 is associated with diesel IC engines and therefore was only used for the determination of the acute hazard index. For the determination of cancer risk and the chronic hazard index, DPM emissions were used without speciation because CARB provides toxicity factors for DPM as a whole (CARB, 2017b).
b. The speciation profiles were originally expressed by CARB as total organic gas (TOG) profiles; they were converted to VOC profiles by dividing by the fraction VOC provided with each profile.
c. Only TACs that have OEHHA/CARB toxicity factors are shown in the table.
d. Source for speciation profiles: CARB, 2016b.

3.0 Receptors

The HRA modeled TAC concentrations and health effects at 2,307 locations (receptors) throughout the project area, including the locations of potential exposure for residents, off-site workers, and sensitive members of the public. The analysis used an inner coarse grid, with receptors positioned every 250 meters and covering an area of 5.5 km x 7.5 km, surrounded by an outer grid, with receptors positioned every 500 meters and covering an area of 16.5 km x 12.5 km. Receptor points were also placed along the Shell terminal boundary at 50 meter intervals. Multiple fine grids, with receptors positioned every 50 meters, were placed over the maximum coarse grid receptors to obtain HRA results to the nearest 50 meters. In addition, receptor points were positioned directly over specific sensitive receptor locations, including grade schools, child care centers, elder care facilities, hospitals, and recreational uses in the vicinity of the terminal.

Figures B3-1 and B3-2 show all receptor points modeled in the HRA. Figure B3-3 shows the modeled sensitive receptors within 2 miles of the Project site, classified by receptor type (schools, child care, elder care). There were no hospitals identified within 2 miles of the Project site. For visual clarity, modeled recreational receptors are not shown in Figure B3-3. Table B3-3 describes the sensitive receptors shown in Figure B3-3.

Table B3-3. Sensitive Receptors within 2 Miles of the Project Site

ID No.	UTM X (m)	UTM Y (m)	Receptor Description	Street Address	City, State, Zip	Category
1	380863	3732991	15th Street Elementary School	1527 Mesa St	San Pedro, CA 90731	Grade School
2	379660	3734797	Bandini Street Elementary School	425 N. Bandini St	San Pedro, CA 90731	Grade School
3	380681	3734795	Barton Hill Elementary School	423 N. Pacific Ave	San Pedro, CA 90731	Grade School
4	380154	3733793	Cabrillo Avenue Elementary School	732 S. Cabrillo Ave	San Pedro, CA 90731	Grade School
5	382757	3737606	Gang Alternative Program	231 Island Ave	Wilmington, CA 90744	Grade School
6	382820	3738093	George de la Torre Jr. Elementary School	500 Island Ave	Wilmington, CA 90744	Grade School
7	382180	3739100	Gulf Avenue Elementary School	828 W. L St	Wilmington, CA 90744	Grade School
8	380824	3735170	Harbor Occupational Center	740 N. Pacific Ave.	San Pedro, CA 90731	Grade School
9	381835	3737984	Hawaiian Avenue Elementary School	540 Hawaiian Ave	Wilmington, CA 90744	Grade School
10	379760	3736916	J F Cooper High School	2210 N. Taper Ave	San Pedro, CA 90731	Grade School
11	380014	3733758	Mary Star of the Sea Elementary School	717 S. Cabrillo Ave	San Pedro, CA 90731	Grade School
12	379495	3737075	Mary Star of the Sea High School	810 W. 8th St	San Pedro, CA 90731	Grade School
13	379279	3735517	Park Western Place Elementary School	1214 Park Western Place	San Pedro, CA 90732	Grade School
14	381187	3734118	Port of Los Angeles High School	250 W 5th St	San Pedro, CA 90731	Grade School
15	382425	3738317	Saints Peter & Paul School	706 Bay View Ave	Wilmington, CA 90744	Grade School
16	379935	3736833	San Pedro MST Center	2201 Barrywood Ave	San Pedro, CA 90731	Grade School
17	379821	3736524	Taper Avenue Elementary School	1824 N. Taper Ave	San Pedro, CA 90731	Grade School
18	379694	3736911	William J. Johnston Community Day School	2210 N Taper Ave	San Pedro, CA 90731	Grade School
19	380186	3733726	Cabrillo Early Education Center	741 W. 8th St	San Pedro, CA 90731	Child Care
20	381264	3732980	Carmen's Cry Baby Care	1509 S. Palos Verdes St	San Pedro, CA 90731	Child Care

ID No.	UTM X (m)	UTM Y (m)	Receptor Description	Street Address	City, State, Zip	Category
21	380158	3734275	Comprehensive Child Development	2565 Pacific Ave.	Long Beach, CA 90806	Child Care
22	380440	3733911	Day Star Early Learning Center	631 W. 6th St	San Pedro, CA 90731	Child Care
23	380825	3733609	Harbor Area YWCA	437 W 9th St	San Pedro, CA 90731	Child Care
24	380519	3733982	Harbor Day Preschool	580 W 6th St	San Pedro, CA 90731	Child Care
25	381827	3738004	Hawaiian Avenue Children's Center	909 W. D St	Wilmington, CA 90744	Child Care
26	380203	3736551	Kidazzle Preschool	1921 N Gaffey St	San Pedro, CA 90731	Child Care
27	383107	3737969	Lil Cowpoke Preschool	445 N Avalon Blvd	Wilmington, CA 90744	Child Care
28	380807	3733760	Merry Go Round Nursery School	446 W 8th St	San Pedro, CA 90731	Child Care
29	382152	3737824	New Harbor Vista Child Development Center	909 W D St	Wilmington, CA 90744	Child Care
30	381871	3738620	Reece Family Day Care	911 King Ave	Wilmington, CA 90744	Child Care
31	380385	3733112	Robin's Nest Day Care	645 W. 14th St	San Pedro, CA 90731	Child Care
32	379822	3735045	San Pedro Child Care	926 W Elberon Ave	San Pedro, CA 90731	Child Care
33	380096	3734974	Smith Family Daycare	787 W Elberon Ave	San Pedro, CA 90731	Child Care
34	380473	3734491	Toberman Child Care Center	131 N. Grand Ave	San Pedro, CA 90731	Child Care
35	381437	3734112	World Tots LA Day Care Center	100 W. 5th St	San Pedro, CA 90731	Child Care
36	382217	3738795	Yvette's Daycare	815 W. Opp St	Wilmington, CA 90744	Child Care
37	380194	3736308	YWCA Venture Park Pre-School	1921 N. Gaffey St	San Pedro, CA 90731	Child Care
38	383100	3738224	American AAA Health Care Center	629 N Avalon Blvd	Wilmington, CA 90744	Elder Care
39	380445	3733657	Crow Flora Boarding & Care Homes	624 W. 9th St	San Pedro, CA 90731	Elder Care
40	381762	3737740	Grandma's House	1218 W D St	Wilmington, CA 90744	Elder Care

ID No.	UTM X (m)	UTM Y (m)	Receptor Description	Street Address	City, State, Zip	Category
41	381348	3733563	Harbor View House	921 S. Beacon St	San Pedro, CA 90731	Elder Care
42	381635	3738433	Wilmington Gardens	1311 W Anaheim St	Wilmington, CA 90744	Elder Care

Sources: California Department of Social Services licensed care search form (www.cdss.ca.gov/), California Department of Education California School Directory (www.cde.ca.gov/re/sd/), California Office of Statewide Health Planning and Development Healthcare Information Division Facility Listings (www.oshpd.ca.gov/HID/Facility-Listing.html), Los Angeles Times California Schools Guide (schools.latimes.com), Yellow Pages (www.yellowpages.com), Los Angeles County Department of Public Social Services (www.ladpss.org/dpss/childcare/search.cfm), Cribsters (www.cribsters.com), and Google Maps (www.google.com/maps).
Note: The receptors listed in this table correspond to Figure B3-3.

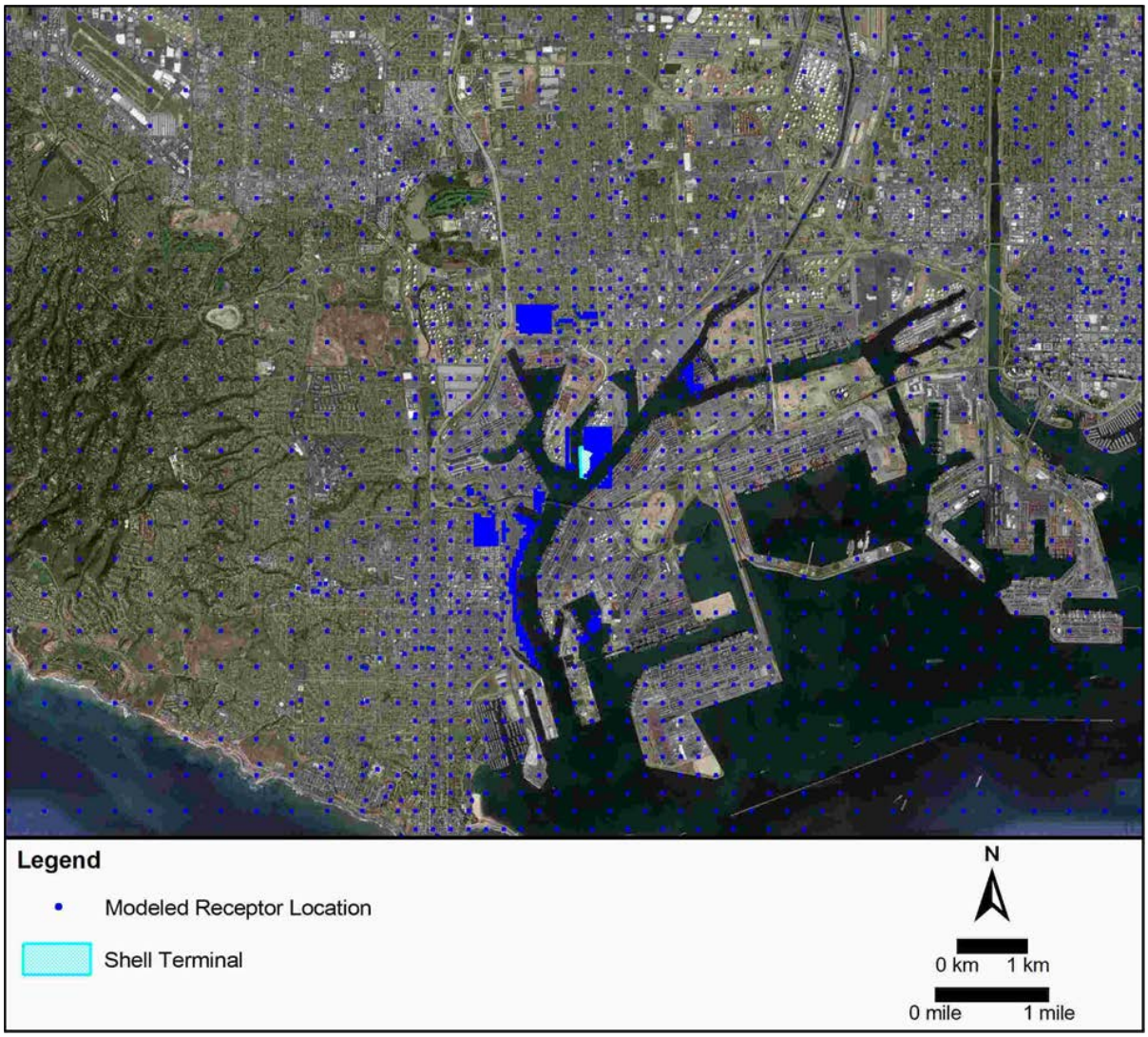

Figure B3-1. HRA Receptor Locations (Far Field)

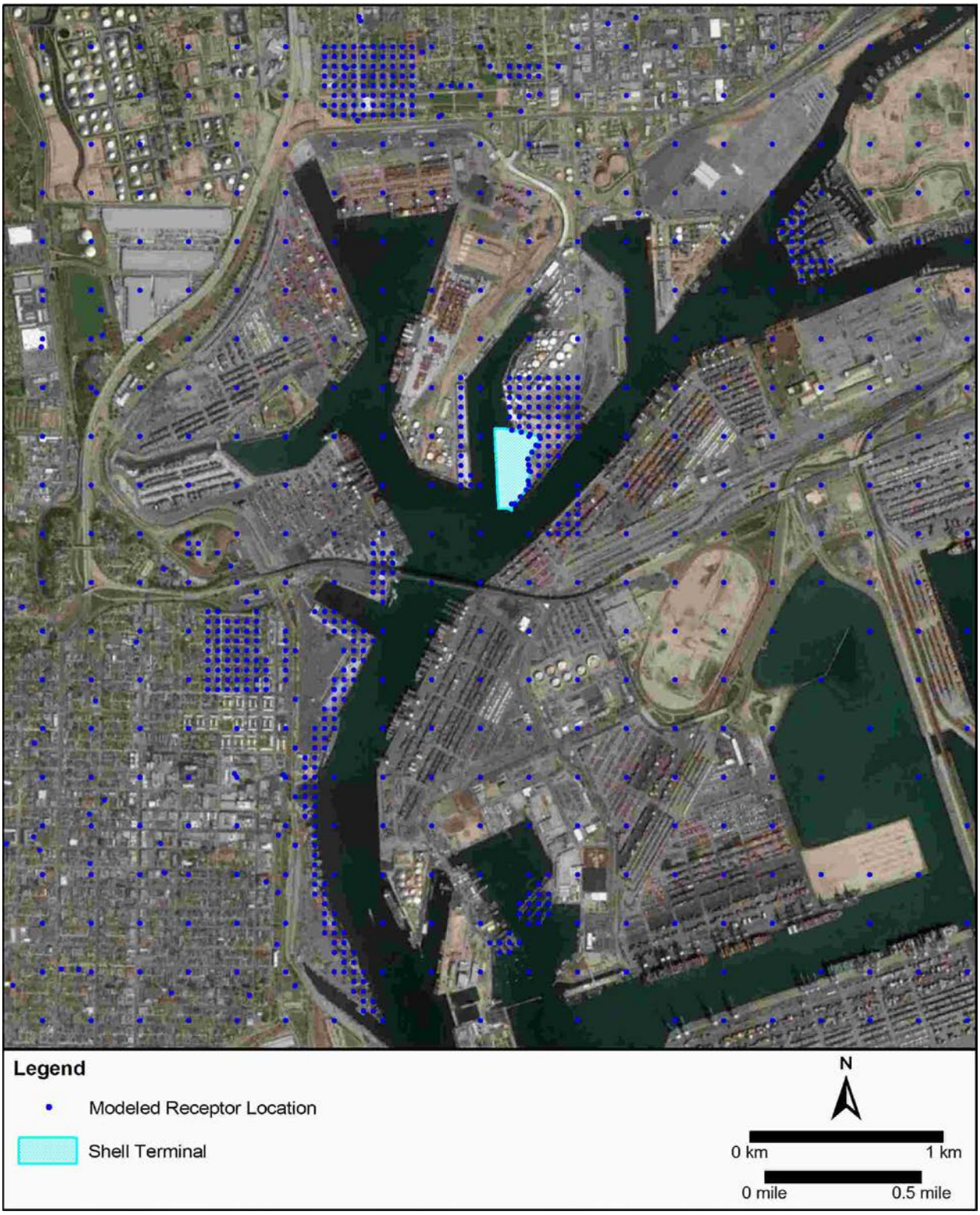

Figure B3-2. HRA Receptor Locations (Near Field)

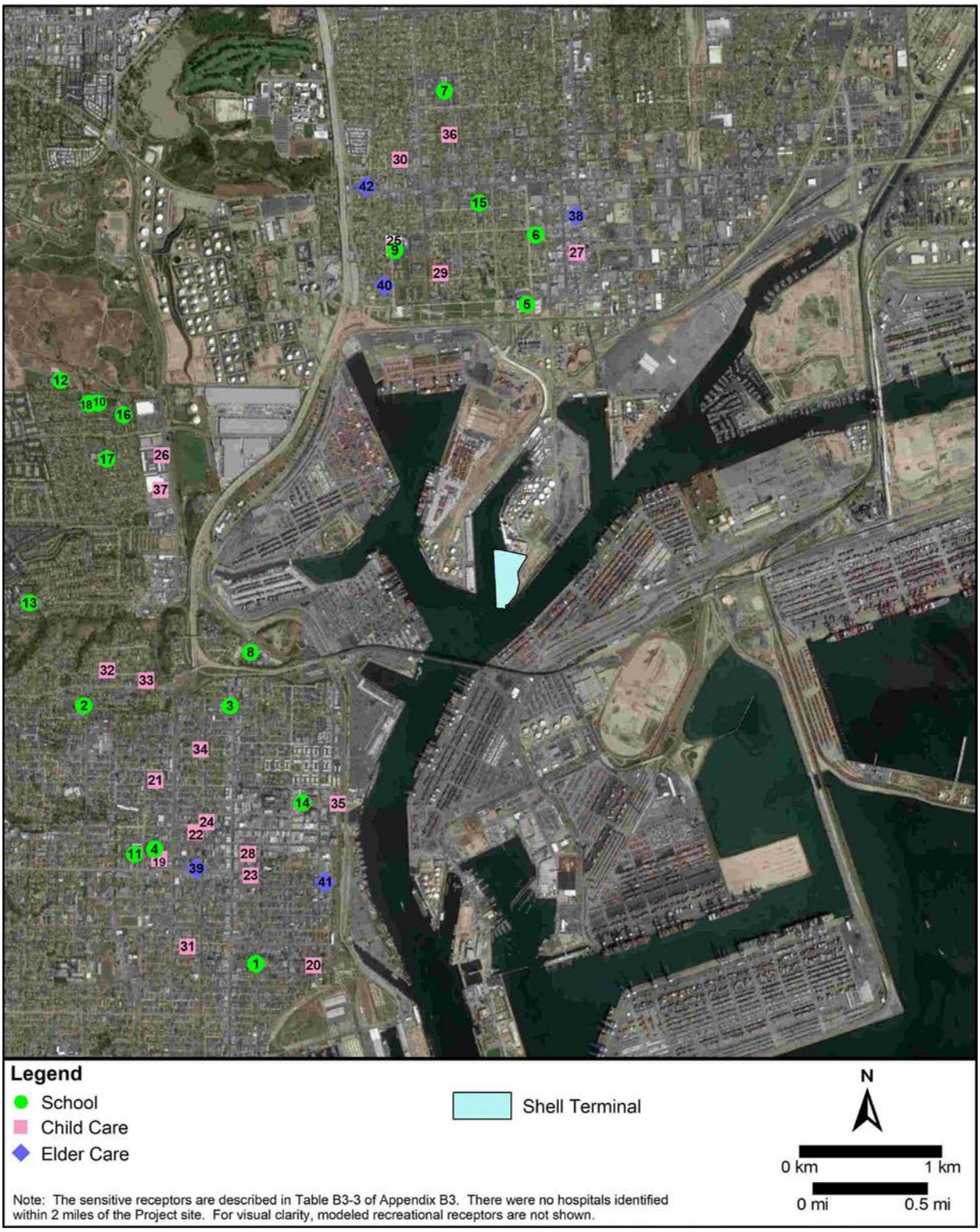

Figure B3-3. Sensitive Receptor Locations within 2 Miles of the Project Site

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

- The residential MEI was selected from all receptors in residential or residentially-zoned areas. Marinas where live-aboards may be present were treated as valid residential receptors.
- The occupational MEI was selected from all receptors on or outside the proposed Project boundary.
- The sensitive MEI was selected from all modeled grade schools, child care centers, elder care facilities, hospitals, and dedicated recreational areas such as parks, marinas, and public waterfront areas.

Receptor points located on water (except for marinas) or within modeled roadways were excluded from MEI selection (SCAQMD, 2008).

4.0 Health Risk Calculation Approach

4.1 Model Selection

The air dispersion modeling was performed using the USEPA AERMOD dispersion model (USEPA, 2015; 2017a), based on the *Guideline on Air Quality Models* (USEPA, 2017b). The emission source parameters, meteorological data, model options, and temporal distribution assumptions used in the HRA are the same as described in Appendix B2. For compatibility with HARP2, each source group in AERMOD was modeled with a 1 gram per second "unit" emission rate. The actual TAC emission rates for each source group were modeled in HARP2.

The health risk calculations were performed using HARP2 (CARB, 2016a; 2017a), based on the TAC concentrations predicted by AERMOD. HARP2 calculated values for individual cancer risk, chronic hazard index, and acute hazard index at each modeled receptor for the CEQA baseline, future CEQA baseline, proposed Project, and project alternatives. For each health value calculated by HARP2, the HRA determined a CEQA increment and future CEQA increment by subtracting the CEQA baseline and future CEQA baseline health value, respectively, from the project health value at each modeled receptor. For each receptor type (residential, occupational, and sensitive), the modeled receptor with the highest increment was selected for reporting and comparison to the appropriate significance threshold.

The most current available versions of AERMOD and HARP2 were used at the time the analyses were performed. AERMOD version 16216r and HARP2 version 17320 were used to calculate population cancer burden and chronic and acute hazard indices. Because individual cancer risk was evaluated at an earlier time, previous versions of AERMOD (version 15181) and HARP2 (version 16088) were used to calculate individual cancer risk. After the modeling of individual cancer risk was completed, some of the construction and operational emissions were updated as follows:

- The construction emissions were revised to reflect a change in the assumed construction activities.
- The assumed vessel hoteling times during product loading were increased;
- The proposed Project horizon year was changed from 2047 to 2048, resulting in an additional 2 percent assumed increase in annual terminal throughput and associated emissions;
- The VDU emissions were updated based on the SCAQMD permit conditions and increased fuel consumption; and
- The start year for the cancer risk exposure periods for the proposed Project and alternatives was updated from 2017 to 2019.

An analysis of these emission changes resulted in a conservative scaling factor adjustment to the original cancer risk results without the need to re-model. The scaling factors applied to the original cancer risk results were 1.28 for the proposed Project, 1.29 for the Reduced Project Alternative (Alternative 2), and 1.16 for the No Project Alternative (Alternative 1). These scaling factors conservatively reflect the highest increase in emissions during any portion of the cancer risk exposure period. The CEQA baseline modeling results were not affected by these emission revisions; therefore, no scaling was applied to baseline. These emission revisions were included in the calculation of population cancer burden and chronic and acute hazard indices, so no scaling was necessary for those health values.

To test the similarity of AERMOD versions 15181 and 16216r, baseline emissions were modeled with both versions of AERMOD, and the resulting concentrations differed by 0.0 to 0.8 percent depending on the pollutant and averaging time. Therefore, the use of either AERMOD version would produce essentially the same predicted concentrations. Furthermore, a review of the version history of HARP2 (CARB, 2017b) indicates that there would be no difference in the calculated risks between the two HARP2 versions as applied to this project.

4.2 Toxicity Factors

An inhalation cancer potency factor represents the probability that a person will contract cancer from the continuous inhalation of one milligram (mg) of a chemical per kilogram (kg) of body weight per day over a period of 70 years. Inhalation potency factors were used by HARP2 to calculate individual cancer risk using the risk assessment algorithms defined in OEHHA (2015).

To assess the potential for non-cancer health effects resulting from chronic and acute inhalation exposure, OEHHA has established Reference Exposure Levels (REL) (CARB, 2017b). An REL 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. The chronic hazard index is the sum of the chemical-specific chronic hazard quotients affecting a particular target organ. The acute hazard index is the sum of the chemical-specific acute hazard quotients affecting a particular target organ. A hazard quotient is a chemical's predicted concentration divided by its REL. A separate hazard index is calculated for each target

organ affected by the TACs because not all TACs affect the same target organ. A hazard index below 1.0 for all affected target organs indicates that adverse non-cancer health effects are not expected.

In addition to the inhalation exposure pathway, several noninhalation exposure pathways were also incorporated in the HRA, including dermal adsorption, soil ingestion, home-grown produce ingestion (residential and sensitive receptors only), and mother's milk ingestion (residential and sensitive receptors only). The TACs evaluated for noninhalation pathways include arsenic, cadmium, hexavalent chromium, lead, nickel, and selenium from all sources except diesel IC engines. For diesel IC engines, the inhalation toxicity factors for DPM already include the effects from exposure to whole diesel exhaust, so a separate evaluation of noninhalation pathways is not required. The various exposure parameters and settings used in HARP2 for the noninhalation exposure pathways are consistent with OEHHA default recommendations (OEHHA, 2015). The results of this analysis show that the contributions of the noninhalation exposure pathways to the HRA results are small compared to the inhalation pathway.

Table B3-4 presents the toxicity factors used to assess health risks in this study.

Table B3-4. Toxicity Factors Used in the HRA

Toxic Air Contaminant	HARP2 TAC ID	Inhalation Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Chronic Inhalation REL ($\mu g/m^3$)	Target Organ for Chronic Exposure[b]	Acute Inhalation REL ($\mu g/m^3$)	Target Organ for Acute Exposure[b]
Acetaldehyde	75070	0.01	140	I	470	D,I
Acrylonitrile	107131	1	5	I	—	—
Arsenic [a]	7440382	12	0.015	B,C,G,I,J	0.2	B,C,G
Benzene	71432	0.1	3	E	27	C,E,F
1,3-Butadiene	106990	0.6	2	C	660	C
Cadmium [a]	7440439	15	0.02	I,M	—	—
Chlorine	7782505	—	0.2	I	210	D,I
Chlorobenzene	108907	—	1,000	A,C,M	—	—
Copper	7440508	—	—	—	100	I
Diesel PM (DPM)	9901	1.1	5	I	—	—
Ethyl benzene	100414	0.0087	2,000	A,C,L,M	—	—
Ethyl chloride	75003	—	30,000	A,C	—	—
Ethylene oxide	75218	0.31	30	G	—	—

Toxic Air Contaminant	HARP2 TAC ID	Inhalation Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Chronic Inhalation REL ($\mu g/m^3$)	Target Organ for Chronic Exposure[b]	Acute Inhalation REL ($\mu g/m^3$)	Target Organ for Acute Exposure[b]
Formaldehyde	50000	0.021	9	I	55	D
Hexane	110543	—	7,000	G	—	—
Hexavalent chromium [a]	18540299	510	0.2	E,I	—	—
Isopropyl alcohol	67630	—	7,000	C,M	3,200	D,I
Lead [a]	7439921	0.042	—	—	—	—
Manganese	7439965	—	0.09	G	—	—
Methanol	67561	—	4,000	C	28,000	G
Methyl ethyl ketone	78933	—	—	—	13,000	D,I
Naphthalene	91203	0.12	9	I	—	—
Nickel [a]	7440020	0.91	0.014	C,E,I	0.2	F
Phenol	108952	—	200	A,B,G,M	5,800	D,I
Propylene	115071	—	3,000	I	—	—
Propylene oxide	75569	0.013	30	I	3,100	C,D,I
Selenium [a]	7782492	—	20	A,B,G	—	—
Styrene	100425	—	900	G	21,000	C,D,I
Sulfates	9960	—	—	—	120	I
Toluene	108883	—	300	C,G,I	37,000	C,D,G,I
Vanadium	7440622	—	—	—	30	D,I
Vinyl chloride	75014	0.27	—	—	180,000	D,G,I
Xylenes	1330207	—	700	D,G,I	22,000	D,G,I

Notes:

[a] Arsenic, cadmium, hexavalent chromium, lead, nickel, and selenium were also evaluated for noninhalation exposure pathways. For arsenic, the cancer risk oral slope factor is 1.5 $(mg/kg/day)^{-1}$, and the noncancer chronic oral REL is 0.0000035 mg/kg/day. For cadmium, the noncancer chronic oral REL is 0.0005 mg/kg/day. For hexavalent chromium, the cancer risk oral slope factor is 0.5 $(mg/kg/day)^{-1}$, and the noncancer chronic oral REL is 0.02 mg/kg/day. For lead, the cancer risk oral slope factor is 0.0085 $(mg/kg/day)^{-1}$. For nickel, the noncancer

Toxic Air Contaminant	HARP2 TAC ID	Inhalation Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$	Chronic Inhalation REL ($\mu g/m^3$)	Target Organ for Chronic Exposure[b]	Acute Inhalation REL ($\mu g/m^3$)	Target Organ for Acute Exposure[b]

chronic oral REL is 0.011 mg/kg/day. For selenium, the noncancer chronic oral REL is 0.005 mg/kg/day. The deposition rate was assumed to be the HARP2 default of 0.02 meters per second (controlled sources).

[b] Key to non-cancer acute and chronic exposure target organs:

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

Source: CARB, 2017b.

4.3 Exposure Scenarios for Individual Cancer Risk

According to OEHHA (2015), individual cancer risk is directly proportional to the frequency and duration of exposure to TACs, modified by age sensitivity factors. The age sensitivity factors multiply the risk by 10 for 3rd-trimester fetuses through infants age 1 (labeled by OEHHA as "0 < 2"); by 3 for children from age 2 through 15 ("2 < 16"), and by 1 for persons age 16 and older.

Tables B3-5 summarizes the primary exposure assumptions used in this HRA to calculate individual cancer risks by receptor type. The exposure assumptions for residential and occupational receptors were obtained from OEHHA (2015). The exposure assumptions for sensitive receptors are not explicitly provided by OEHHA (2015); therefore, LAHD conservatively evaluated sensitive receptors with 30-year residential exposure assumptions. LAHD evaluated recreational receptors, which are classified as sensitive receptors in this HRA, with reasonable worst case exposure assumptions of 250 days/year, 2 hr/day, for 30 years.

Because the future CEQA baseline, proposed Project, and project alternatives have emissions that change over time in the HRA, it was necessary to subdivide the exposure durations listed in Table B3-5 into smaller time periods (sub-periods) and run HARP2 separately for each sub-period. These sub-periods correspond to the receptor age groups listed in Table B3-6. Each receptor age group uses a unique set of exposure assumptions and age sensitivity factors, which are presented in Table B3-6.

For each receptor type, the youngest expected age range was modeled in the HRA to produce the most conservative (highest) risk result. For example, the calculation of 30-year residential cancer risk assumes that the exposed person is in the 3rd trimester before birth at the beginning of the 30-year exposure period. This assumption maximizes the use of the childhood age sensitivity factors in the cancer risk calculation. Moreover, the calculated cancer risk is increased even further during childhood years by using higher breathing rates per body weight than adults.

For each receptor age group in Table B3-6, HARP2 modeled the average annual emissions that would occur over that specified period of time. The HARP2 cancer risk results for each age group were then summed to obtain the cancer risk for the entire exposure duration. For example, the 30-year residential cancer risk for the proposed Project was determined by

running HARP2 once for each of three receptor age groups. The first age group is third trimester < 2, assumes an exposure duration of 2 ¼ years, and uses Project emissions averaged over the time period 2019-2020. The second age group is 2 < 16, assumes an exposure duration of 14 years, and uses Project emissions averaged over the time period 2021-2034. The third age group is 16 < 30, assumes an exposure duration of 14 years, and uses Project emissions averaged over the time period 2035-2048. The cancer risks calculated by HARP2 for these three receptor age groups were then summed to obtain the total cancer risks for the entire 30-year exposure duration.

Table B3-5. Cancer Risk Exposure Assumptions by Receptor Type

Receptor Type	Exposure Duration			Breathing Rate Category	Exposed Person's Age Range [5]
	Days per Year	Hours per Day	Years		
Residential - For Individual Cancer Risk [1]	350	24	30	95th percentile residential	3TM[4] < 30
Residential - For Population Cancer Burden [1]	350	24	70	95th percentile residential	3TM < 70
Occupational [1]	250	8	25	95th percentile moderate intensity	≥ 16
Sensitive (except Recreational) [2]	350	24	30	95th percentile residential	3TM < 30
Recreational [6]	250	2	30	95th percentile moderate intensity[3]	0 < 30

Notes:
1. The exposure assumptions for residential and occupational receptors were obtained from OEHHA (2015).
2. Thirty-year residential exposure assumptions were conservatively used for sensitive receptors.
3. The breathing rate for recreational receptors is scaled from the 95th percentile 8-hour moderate intensity breathing rate (OEHHA 2015) by a factor of 2 hr/8 hr.
4. 3TM = third trimester (prior to birth).
5. The exposed person's age ranges were conservatively selected to maximize the cancer risk (i.e., the youngest age range).
6. Recreational receptors are classified as sensitive receptors in this HRA.

Table B3-6. Cancer Risk Exposure Assumptions by Age Group

Exposure Parameter	Receptor Age Group					
	3rd Trimester	0 < 2 Years	2 < 9 Years	2 < 16 Years	16 < 30 Years	16 - 70 Years
ASF: Age Sensitivity Factor	10	10	3	3	1	1
Fraction of Time Spent at Receptor Location (FAH) (unitless): [4]						
Residential [1] and Sensitive (except Recreational) [2]	1	1	n/a [6]	1	0.73	0.73
Occupational [1]	n/a	n/a	n/a	n/a	n/a	1
Recreational [2]	n/a	1	n/a	1	1	n/a
Breathing Rate per Body Weight (BR/BW) (L/kg/day): [5]						
Residential [1] and Sensitive (except Recreational) [2]	361	1,090	n/a	745	335	290
Occupational [1]	n/a	n/a	n/a	n/a	n/a	230
Recreational [2,3]	n/a	300	n/a	130	60	n/a

Notes:
1. The exposure assumptions for residential and occupational receptors were obtained from OEHHA (2015).
2. The exposure assumptions for sensitive receptors are not explicitly provided by OEHHA (2015); LAHD conservatively evaluated sensitive receptors (except recreational) with 30-year residential exposure assumptions. LAHD evaluated recreational receptors, which are also classified as sensitive receptors in this HRA, with reasonable worst case exposure assumptions (see notes 3 and 5).
3. The breathing rate for recreational receptors is scaled from the 95th percentile 8-hour moderate intensity breathing rate (OEHHA 2015) by a factor of 2 hr/8 hr.
4. Per OEHHA (2015), a value of 1 for FAH for ages <16 should be used if a school exists within the 1 per million cancer risk isopleth (OEHHA, 2015).
5. L/kg/day is liters of air per kilogram body weight per day. These daily breathing totals reflect 24 hours of exposure for residential and sensitive receptors (except recreational); 8 hours of exposure for occupational receptors; and 2 hours of exposure for recreational receptors.
6. "n/a" means the value is not used in the cancer risk calculation for that particular receptor type.

Population Cancer Burden Methodology

Population cancer burden is defined by OEHHA as an estimate of the number of cancer cases expected from a 70-year exposure to emissions (OEHHA, 2015). Whereas individual cancer risk represents the probability of a single exposed person to develop cancer, population cancer burden estimates the number of individuals that would be expected to contract cancer by multiplying the cancer risk by the exposed population. The exposed population is defined as the number of persons within a facility's zone of impact, which is defined by the LAHD and SCAQMD as the area within the Project's one in a million incremental cancer risk isopleth. Population cancer burden was calculated using census block population data contained in HARP2, which are based on the 2010 U.S. Census.

5.0 Significance Criteria

The LAHD has adopted a significance threshold of 10 in a million for individual cancer risk (project increment). 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 in 1 million (10×10^{-6}) relative to the CEQA baseline and, for cancer risk, the future CEQA baseline. The LAHD has also adopted the air quality significance threshold for cancer burden of 0.5 excess cancer cases in areas with project-attributable individual cancer risk above one in a million (1×10^{-6}) (SCAQMD, 2015b). In addition, the LAHD has adopted the significance threshold of 1.0 for chronic and acute non-cancer

hazard indices; a project would produce less than significant non-cancer impacts if the chronic and acute hazard indices are less than 1.0 (SCAQMD, 2015b).

6.0 Predicted Incremental Health Impacts

6.1 Proposed Project without Mitigation

Table B3-7 presents the maximum predicted health impacts associated with construction and operation of the proposed Project without mitigation. The table includes estimates of individual cancer risk, chronic noncancer hazard index, and acute noncancer hazard index at the maximally exposed residential, occupational, and sensitive receptors. Results are presented for the terminal with the proposed Project (before subtracting baseline), the two CEQA baselines, the CEQA increment (terminal with proposed Project minus CEQA baseline), and future CEQA increment (terminal with proposed Project minus future CEQA baseline). The table also presents the population cancer burden for the CEQA increment and future CEQA increment. Significance findings are made by comparing the increments to the significance thresholds.

Each health value in the table represents its maximum predicted receptor location. The receptor locations are not necessarily the same from one health value to the next. To illustrate this, the following example shows how the maximum proposed Project CEQA increment for cancer risk at a residential receptor (3.3 in a million), shown in the first row of results in the table, was determined. This result is predicted to occur at modeled Receptor No. 1124, in Wilmington.

Example—Determine Proposed Project CEQA Increment at Receptor No. 1124:

- Terminal with Proposed Project cancer risk, Receptor No. 1124 = 8.0 in a million (shown in the table)
- CEQA baseline cancer risk, Receptor No. 1124 = 4.7 in a million (not shown in the table because Receptor No. 1124 is not the location of the maximum CEQA baseline cancer risk)
- Proposed Project CEQA increment, Receptor No. 1124 = 8.0 - 4.7 = 3.3 in a million (shown in the table)

After performing an increment calculation similar to the above example at every modeled receptor, it was determined that Receptor No. 1124 has the highest proposed Project CEQA increment of any residential receptor. Therefore, its CEQA increment of 3.3 in a million is reported in Table B3-7. However, in this example, Receptor 1124 is *not* the maximum residential receptor for the CEQA baseline by itself (its maximum of 5.3 in a million occurs at Receptor No. 425). The CEQA increment (Project minus baseline) at Receptor No. 425 is 2.3 in a million, which is less than the maximum increment of 3.3 in a million shown in the table.

Although the above example shows the cancer risk increment being calculated at one modeled receptor, the complete determination of the maximum increment involves this same type of calculation at hundreds of modeled receptors. The chronic and acute noncancer hazard index increments are determined in the same way.

6.1.1 Individual Cancer Risk

Table B3-7 shows that the maximum individual cancer risk is predicted to be less than the significance threshold at all receptors for both the proposed Project CEQA increment and proposed Project future CEQA increment.

Table B3-8 shows the emission source contributions to cancer risk from the unmitigated proposed Project at the receptor locations with the highest predicted residential and occupational CEQA increments. The highest source contributor is vessel (i.e., tanker and ITB/ATB) hoteling, which would contribute 77 percent of the cancer risk at the maximum residential receptor, and 72 percent of the cancer risk at the maximum occupational receptor. These contribution percentages are approximations because they were derived from the original modeling results for individual cancer risk, prior to the emission revisions and scaling adjustments described in Section 4.1.

6.1.2 Population Cancer Burden

Table B3-7 shows that the population cancer burden is predicted to be less than the significance threshold for both the proposed Project CEQA increment and proposed Project future CEQA increment.

6.1.3 Chronic and Acute Impacts

Table B3-7 shows that the maximum chronic and acute hazard index CEQA increments associated with the proposed Project are predicted to be less than the significance thresholds at all receptors.

Because all health impacts are predicted to be less than the significance thresholds without mitigation, an evaluation of the proposed Project with the mitigation measures prescribed in Section 3.1 was not necessary.

Table B3-7. Maximum Health Impacts Estimated for Construction and Operation of the Proposed Project without Mitigation

Health Impact [d,e]	Receptor Type	Terminal with Proposed Project	CEQA Baseline	Proposed Project CEQA Increment[b]	Future CEQA Baseline[c]	Proposed Project Future CEQA Increment[b]	Significance Threshold[a]	Threshold Exceeded?
Individual Cancer Risk	Residential	8.0 × 10-6	5.3 × 10-6	3.3 × 10-6	4.8 × 10-6	3.4 × 10-6	10×10^{-6}	No
	Occupational	13.2 × 10-6	8.2 × 10-6	6.8 × 10-6	8.1 × 10-6	6.9 × 10-6		No
	Sensitive[f]	7.3 × 10-6	4.8 × 10-6	3.0 × 10-6	4.3 × 10-6	3.1 × 10-6		No
Chronic Hazard Index	Residential	0.14	0.04	0.10	n/a	n/a	1.0	No
	Occupational	0.87	0.30	0.65	n/a	n/a		No
	Sensitive	0.15	0.04	0.10	n/a	n/a		No
Acute Hazard Index	Residential	0.08	0.02	0.06	n/a	n/a	1.0	No
	Occupational	0.85	0.18	0.77	n/a	n/a		No
	Sensitive	0.11	0.02	0.09	n/a	n/a		No
Population Cancer Burden			**Proposed Project CEQA Increment[b]**		**Proposed Project Future CEQA Increment[b]**		0.5	No
			0.12		0.14			

Notes:
[a]The significance thresholds apply only to the Proposed Project CEQA increment and Proposed Project Future CEQA increment.
[b]The Proposed Project CEQA increment represents the Terminal with Proposed Project minus CEQA Baseline. The Proposed Project Future CEQA increment represents the Terminal with Proposed Project minus Future CEQA Baseline.
[c]The Future CEQA baseline (and, therefore, the Proposed Project Future CEQA increment) is applicable only to cancer risk because cancer risk has a uniquely long exposure period (30 years for residential exposure and 70 years for population cancer burden). By contrast, the baseline chronic and acute hazard indices are derived from annual and peak hour emissions, respectively, and therefore reflect the baseline at the time of the NOP (i.e., CEQA baseline).
[d]Each result shown in the table for cancer risk, chronic hazard index, and acute hazard index represents the modeled receptor location with the maximum impact or increment. The impacts or increments at all other receptors would be less than the values in the table.
[e]Health values include contributions from both construction and operation.
[f]The sensitive receptor category in this table includes grade schools, child care centers, hospitals, elder care facilities, and dedicated recreational areas such as parks, marinas, and public waterfront areas. The maximum health value from all of these receptor types is presented in the table.

Table B3-8. Source Contributions to Cancer Risk at the Maximum Residential and Occupational Increment Receptors – Proposed Project without Mitigation

Source Category	Maximum Residential Receptor		Maximum Occupational Receptor	
	CEQA Increment	Future CEQA Increment	CEQA Increment	Future CEQA Increment
Construction				
Off-road Equipment	2.7%	2.7%	14.1%	14.1%
On-road Trucks	<0.1%	<0.1%	<0.1%	<0.1%
Harborcraft	3.7%	3.7%	9.2%	9.2%
Source Control Program	0.3%	0.3%	0.9%	0.9%
Operation				
Vessel Transit	4.5%	4.5%	0.8%	0.8%
Vessel Anchorage	7.8%	7.8%	0.5%	0.5%
Vessel Hotelling	77.5%	77.5%	72.1%	72.1%
Tugboats	3.3%	3.3%	1.0%	1.0%
Storage Tanks and Piping	0.1%	0.1%	1.1%	1.1%
Vessel Loading Fugitives and VDU	0.1%	0.1%	0.3%	0.3%

Note: Contributions are from proposed Project sources prior to subtracting baseline.

6.2 Reduced Project Alternative without Mitigation

Table B3-9 presents the maximum predicted health impacts associated with construction and operation of the Reduced Project Alternative (also known as Alternative 2) without mitigation.

Individual Cancer Risk

Table B3-9 shows that the maximum individual cancer risk is predicted to be less than the significance threshold at all receptors for both the Reduced Project CEQA increment and Reduced Project future CEQA increment.

Population Cancer Burden

Table B3-9 shows that the population cancer burden is predicted to be less than the significance threshold for both the Reduced Project CEQA increment and Reduced Project future CEQA increment.

Chronic and Acute Impacts

Table B3-9 shows that the maximum chronic and acute hazard index CEQA increments associated with the Reduced Project Alternative are predicted to be less than the significance thresholds at all receptors.

Because all health impacts are predicted to be less than the significance thresholds without mitigation, an evaluation of the Reduced Project Alternative with the mitigation measures prescribed in Section 3.1 was not necessary.

Table B3-9. Maximum Health Impacts Estimated for Construction and Operation of the Reduced Project Alternative without Mitigation

Health Impact [d,e]	Receptor Type	Terminal with Reduced Project	CEQA Baseline	Reduced Project CEQA Increment[b]	Future CEQA Baseline[c]	Reduced Project Future CEQA Increment[b]	Significance Threshold[a]	Threshold Exceeded ?
Individual Cancer Risk	Residential	8.2×10^{-6}	5.3×10^{-6}	3.5×10^{-6}	4.8×10^{-6}	3.6×10^{-6}	10×10^{-6}	No
	Occupational	13.7×10^{-6}	8.2×10^{-6}	5.5×10^{-6}	8.1×10^{-6}	5.6×10^{-6}		No
	Sensitive[f]	7.4×10^{-6}	4.8×10^{-6}	3.2×10^{-6}	4.3×10^{-6}	3.3×10^{-6}		No
Chronic Hazard Index	Residential	0.18	0.04	0.14	n/a	n/a	1.0	No
	Occupational	1.23	0.30	0.93	n/a	n/a		No
	Sensitive	0.19	0.04	0.15	n/a	n/a		No
Acute Hazard Index	Residential	0.07	0.02	0.06	n/a	n/a	1.0	No
	Occupational	0.82	0.18	0.74	n/a	n/a		No
	Sensitive	0.10	0.02	0.08	n/a	n/a		No
Population Cancer Burden			**Reduced Project CEQA Increment[b]**		**Reduced Project Future CEQA Increment[b]**		0.5	No
			0.12		0.14			

Notes:
[a]The significance thresholds apply only to the Reduced Project CEQA increment and Reduced Project Future CEQA increment.
[b]The Reduced Project CEQA increment represents the Terminal with Reduced Project minus CEQA Baseline. The Reduced Project Future CEQA increment represents the Terminal with Reduced Project minus Future CEQA Baseline.
[c]The Future CEQA baseline (and, therefore, the Reduced Project Future CEQA increment) is applicable only to cancer risk because cancer risk has a uniquely long exposure period (30 years for residential exposure and 70 years for population cancer burden). By contrast, the baseline chronic and acute hazard indices are derived from annual and peak hour emissions, respectively, and therefore reflect the baseline at the time of the NOP (i.e., CEQA baseline).
[d]Each result shown in the table for cancer risk, chronic hazard index, and acute hazard index represents the modeled receptor location with the maximum impact or increment. The impacts or increments at all other receptors would be less than the values in the table.
[e]Health values include contributions from both construction and operation.
[f]The sensitive receptor category in this table includes grade schools, child care centers, hospitals, elder care facilities, and dedicated recreational areas such as parks, marinas, and public waterfront areas. The maximum health value from all of these receptor types is presented in the table.

6.3 No Project Alternative

Table B3-10 presents the maximum predicted health impacts associated with operation of the No Project Alternative (also known as Alternative 1).

6.3.1 Individual Cancer Risk

Table B3-10 shows that the maximum individual cancer risk is predicted to be less than the significance threshold at all receptors for both the No-Project CEQA increment and No-Project future CEQA increment. The negative values for the cancer risk increments indicate that the cancer risk associated with the No Project Alternative would be less than the baseline risks. This risk reduction is primarily a result of operational emissions ending after 2023, which more than compensates for the assumed growth in annual product throughput from 2019 to 2023.

6.3.2 Population Cancer Burden

Because the individual cancer risk increments associated with the No Project Alternative would be less than zero in all areas, there would be no zone of impact, and population cancer burden would be zero.

6.3.3 Chronic and Acute Impacts

Table B3-10 shows that the maximum chronic and acute hazard index CEQA increments associated with the No Project Alternative are predicted to be less than the significance thresholds at all receptors.

Table B3-10. Maximum Health Impacts Estimated for Operation of the No Project Alternative

Health Impact[e]	Receptor Type	Terminal with No Project	CEQA Baseline	No Project CEQA Increment[b]	Future CEQA Baseline[c]	No Project Future CEQA Increment[b,d]	Significance Threshold[a]	Threshold Exceeded?
Individual Cancer Risk	Residential	3.8×10^{-6}	5.3×10^{-6}	-1.5×10^{-6}	4.8×10^{-6}	-0.9×10^{-6}	10×10^{-6}	No
	Occupational	2.7×10^{-6}	8.2×10^{-6}	-5.5×10^{-6}	8.1×10^{-6}	-5.4×10^{-6}		No
	Sensitive[f]	3.4×10^{-6}	4.8×10^{-6}	-1.4×10^{-6}	4.3×10^{-6}	-0.9×10^{-6}		No
Chronic Hazard Index	Residential	0.09	0.04	0.05	n/a	n/a	1.0	No
	Occupational	0.61	0.30	0.31	n/a	n/a		No
	Sensitive	0.09	0.04	0.05	n/a	n/a		No
Acute Hazard Index	Residential	0.02	0.02	0.002	n/a	n/a	1.0	No
	Occupational	0.19	0.18	0.03	n/a	n/a		No
	Sensitive	0.02	0.02	0.002	n/a	n/a		No
Population Cancer Burden			**No Project CEQA Increment[b]**		**No Project Future CEQA Increment[b]**		0.5	No
			0.0		0.0			

Notes:
[a]The significance thresholds apply only to the No Project CEQA increment and No Project Future CEQA increment.
[b]The No Project CEQA increment represents the Terminal with No Project minus CEQA Baseline. The No Project Future CEQA increment represents the Terminal with No Project minus Future CEQA Baseline.
[c]The Future CEQA baseline (and, therefore, the No Project Future CEQA increment) is applicable only to cancer risk because cancer risk has a uniquely long exposure period (30 years for residential exposure and 70 years for population cancer burden). By contrast, the baseline chronic and acute hazard indices are derived from annual and peak hour emissions, respectively, and therefore reflect the baseline at the time of the NOP (i.e., CEQA baseline).
[d]Negative values for increments denote health risk reductions relative to baseline.
[e]Each result shown in the table for cancer risk, chronic hazard index, and acute hazard index represents the modeled receptor location with the maximum impact or increment except for the negative cancer risk increments. Because the negative cancer risk increments are predicted to be less than zero at all modeled receptors, they would approach a maximum value of zero infinitely far away from the Project site. To provide more meaningful results, the cancer risk increments shown in the table correspond to the maximum receptor locations for the Terminal with No Project.
[f]The sensitive receptor category in this table includes grade schools, child care centers, hospitals, elder care facilities, and dedicated recreational areas such as parks, marinas, and public waterfront areas. The maximum health value from all of these receptor types is presented in the table.

7.0 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 to each other and to significance criteria. Consistent with agency guidelines and standard approaches to regulatory risk assessment, this risk assessment used health-protective (conservative) assumptions to provide a margin of safety with respect to human health. OEHHA has provided a discussion of risk uncertainty, which is reiterated here (OEHHA 2015):

OEHHA has striven to use the best science available in developing these risk assessment guidelines. However, 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 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. In addition to uncertainty, there is a natural range or variability in measured parameters defining the exposure scenario. Scientific studies with representative sampling and large enough sample sizes can characterize this variability. In the specific context of a Hot Spots risk assessment, the source of variability with the greatest quantitative impact is variation among the human population in such properties as height, weight, food consumption, breathing rates, and susceptibility to chemical toxicants. OEHHA captures at least some of the variability in exposure by developing data driven distributions of intake rates, where feasible, in the TSD for Exposure Assessment (OEHHA, 2012).

Interactive effects of exposure to more than one carcinogen or toxicant are addressed in the risk assessment with default assumptions of additivity. Cancer risks from all carcinogens addressed in the HRA are added. Similarly, non-cancer hazard quotients for substances impacting the same target organ/system are added to determine the hazard index (HI). Although such effects of multiple chemicals are assumed to be additive by default, several examples of 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. In most cases, cancer potency values have been estimated only for the single most affected tumor site. This

represents a source of uncertainty in the cancer risk assessment. Adjustment for tumors at multiple sites induced by some carcinogens may result in a higher potency. Some recent assessments of carcinogens include such adjustments. Other uncertainties arise 1) in the assumptions underlying the dose-response model used, and 2) in extrapolating from large experimental doses, where other toxic effects may compromise the assessment of carcinogenic potential, to usually much smaller environmental doses.

When occupational epidemiological data are used to generate a carcinogenic potency or a health protective level for a non-carcinogen, less uncertainty is involved in the extrapolation from workplace exposures to environmental exposures. When using human data, no interspecies extrapolation is necessary, eliminating a significant source of uncertainty. However, children are a subpopulation whose hematological, nervous, endocrine, and immune systems, for example, are still developing and who may be more sensitive to the effects of toxicants on their developing systems. The worker population and risk estimates based on occupational epidemiological data are more uncertain for children than adults. Current risk assessment guidelines include procedures designed to address the possibly greater sensitivity of infants and children, but there are only a few compounds for which these effects have actually been measured experimentally. In most cases, the adjustment relies on default assumptions which may either underestimate or overestimate the true risks faced by infants and children exposed to toxic substances or carcinogens.

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 for disease, based on current knowledge and a number of assumptions.

In the Hot Spots program, cancer risk is often expressed as the maximum number of new cases of cancer projected to occur in a population of one million people due to exposure to the cancer-causing substance over a 30-year residential period. However, there is uncertainty associated with the cancer risk estimate. An individual's risk of contracting cancer from exposure to facility emissions may be less or more than the risk calculated in the risk assessment. An individual's risk not only depends on the individual's exposure to a specific chemical but also on his or her genetic background, health, diet, lifestyle choices and other environmental and workplace exposures. OEHHA uses health-protective exposure assumptions to avoid underestimating risk. For example, the risk estimate for airborne exposure to chemical emissions uses the health protective assumption that the individual has a high breathing rate and exposure began early in life when cancer risk is highest.

A Reference Exposure Level (REL) is the concentration level at or below which no adverse non-cancer health effects are anticipated for the specified exposure duration. RELs are based on the most sensitive, relevant, adverse health effect reported in the medical and toxicological literature. RELs are designed to protect the most sensitive individuals in the population by the inclusion of factors that account for uncertainties as well as individual differences in human susceptibility to chemical exposures. The factors used in the calculation of RELs are meant to err on the side of public health protection in order to avoid underestimation of non-cancer hazards. Exceeding the REL does not automatically indicate an adverse health impact. However, increasing concentrations above the REL value increases the likelihood that the health effect will occur.

Risk assessments under the Hot Spots program are often used to compare one source with another and to prioritize concerns. Consistent approaches to risk assessment are necessary to fulfill this function.

8.0 References

CARB 1989. *Technical Guidance Document for the Emission Inventory Criteria and Guidelines Regulation for AB 2588*. Technical Support Division. August.

CARB, 2016a. Hotspots Analysis and Reporting Program (HARP2). Version 16088. Air Dispersion Modeling and Risk Tool (ADMRT). March 28.

CARB, 2016b. Speciation Profiles Used in ARB Modeling. The following files were downloaded: orgprofile15apr16.xlsx, fraction15apr16.xlsx, sccassignfrxn2016thru2035vv10001xx20120920.xls, sccassignfrxn1996thru2015vv10001xx20120920.xls, pmchemprofile15apr16.xlsx, and pmsizeprofile15apr16.xlsx. April 15.

CARB, 2017a. Hotspots Analysis and Reporting Program (HARP2). Version 17320. Air Dispersion Modeling and Risk Tool (ADMRT). November 16.

CARB, 2017b. Consolidated Table of OEHHA/ARB Approved Risk Assessment Health Values. February 23.

CARB, 2017c. Hotspots Analysis and Reporting Program (HARP) Software. Air Dispersion Modeling and Risk Tool (ADMRT). Version History. December 6.

OEHHA, 2012. *Air Toxics Hot Spots Program Risk Assessment Guidelines. Technical Support Document for Exposure Assessment and Stochastic Analysis*. August.

OEHHA, 2015. *Air Toxics Hot Spots Program Risk Assessment Guidelines. Guidance Manual for Preparation of Health Risk Assessments.* February.

SCAQMD, 2005. Personal communication with J. Koizumi. September 21.

SCAQMD, 2008. *Localized Significance Threshold Methodology*. Final Revised. July.

SCAQMD, 2015a. *Supplemental Guidelines for Preparing Risk Assessments for the Air Toxics "Hot Spots" Information and Assessment Act.* June 5.

SCAQMD, 2015b. SCAQMD Air Quality Significance Thresholds. March.

USEPA, 2015. AERMOD Modeling System. Version 15181. Technology Transfer Network. Support Center for Regulatory Atmospheric Modeling. Release date: June 30.

USEPA, 2017a. AERMOD Modeling System. Version 16216r. Technology Transfer Network. Support Center for Regulatory Atmospheric Modeling. Release date: January 17.

USEPA, 2017b. *Guideline on Air Quality Models.* 40 CFR Appendix W to Part 51. Federal Register Vol. 82, No. 10. January 17.