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 Berths 226-236 [Everport] Container Terminal Improvements Project and alternatives. 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 construction and operation of the following alternatives:

- Proposed Project
- Alternative 1, No Federal Action Alternative
- Alternative 2, No Project Alternative (there is no construction associated with this alternative)
- Alternative 3, Reduced Project Alternative: Reduced Wharf Improvements
- Alternative 4, Reduced Project Alternative: No Backland Improvements
- Alternative 5, Expanded On-Dock Railyard: Wharf and Backland Improvements with an Expanded TICTF

This HRA evaluated the incremental change in health values associated with the proposed Project and alternatives relative to the CEQA and NEPA baselines. The CEQA baseline represents terminal operation in 2013, as described in Section 3.1.4.2 of the EIS/EIR. The NEPA baseline, for purposes of this Draft EIS/EIR, is the same as the mitigated No Federal Action Alternative (Alternative 1), as described in Section 3.1.4.3 of the EIS/EIR.

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, 2015). 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 an off-site 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.
- 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 Everport terminal operates 24 hours per day, the average 8-hour concentrations to which off-site workers would be exposed would approximate 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 EPA dispersion model AERMOD, version 15181, was used to predict maximum ambient pollutant concentrations outside the Project site. The Hotspots Analysis and Reporting Program (HARP2), version 16088 (CARB, 2016), 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 schools, hospitals, elder care facilities, child care centers, or recreational areas; (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 the CEQA and NEPA baselines. 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:

- Offroad construction equipment: engine exhaust from land-based equipment and marine-based equipment (dredging and pile driving equipment);
- On-road construction vehicles: engine exhaust, tire wear, and brake wear from haul and delivery trucks while traveling and idling onsite;
- Crane delivery ship: auxiliary engine and boiler exhaust while hoteling at berth;
- Harborcraft: engine exhaust from harborcraft used in dredging and pile driving; and
- Asphalt paving: fugitive volatile organic compound (VOC) offgas emissions during the on-site paving process.

In accordance with SCAQMD guidance, the HRA included on-site construction emissions that would occur within the project site or adjacent waters (SCAQMD 2005). Construction emissions would not occur under Alternative 2.

The following operational emission sources were included in the HRA:

- Container ships transiting to and from berth. Ship transit emission sources are comprised of propulsion engine, auxiliary engine, and boiler exhaust. Ship transit in SCAQMD waters consists of transit in the fairway, precautionary zone, and harbor. Ships were modeled as far as the SCAB overwater boundary, approximately 40 nautical miles from berth.
- Container ships hoteling while at berth and at anchorage in the harbor. Ship hoteling emission sources are comprised of ship auxiliary engine exhaust (except when using AMP at berth) and boiler exhaust; propulsion engines would be turned off.
- Tugboats used to assist container ships between the Port breakwater and the berth. Two tugboats were assumed to assist each ship. Tugboat emission sources are comprised of propulsion and auxiliary engines.
- On-road trucks driving on near-Port roads and inside the Everport terminal, and idling on-terminal and at the Everport terminal in- and out-gates. Truck transit emission sources are comprised of exhaust, tire wear, and brake wear. Trucks were modeled as far as approximately 4 miles north of the terminal, a distance established in prior LAHD NEPA/CEQA documents as sufficient to capture maximum concentrations for container terminal projects (LAHD, 2011).
- Locomotives switching and idling at the TICTF on-dock rail yard, and line haul locomotives pulling trains between the TICTF on-dock rail yard and the Alameda Corridor. Locomotives traveling were modeled as far as approximately 4 miles north of the terminal.
- Cargo handling equipment (CHE) operating at the Everport terminal and TICTF, including forklifts, rubber-tired gantry cranes, top handlers, and yard tractors.
- Worker vehicles driving to and from the Everport terminal. Worker vehicle emissions include exhaust, tire wear, and brake wear. Worker vehicles were modeled as far as approximately 4 miles north of the terminal.

2.1 Emissions Used for Cancer Risk

To estimate cancer risk impacts for the proposed Project, project alternatives, and NEPA baseline, annual total organic gas (TOG) and PM10 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 2018, the anticipated first year of proposed Project construction and overlapping operation. For example, the 30-year residential exposure period was assumed to occur during the years 2018-2047.

Annual emissions were estimated using the methodology and assumptions described in Section 3.1.4.1 of the EIR and Sections 2.2 and 2.4 of Appendix B2. The emissions account for the projected future growth in container throughput, and the future reduction in emission factors for most equipment in response to existing regulations.

Annual construction emissions were calculated for each year of construction, 2018 and 2019. The annual emissions reflect the land disposal option for dredged materials because the criteria pollutant dispersion modeling analysis (Appendix B2) found that land disposal yielded higher modeled concentrations than ocean disposal for long-term averages. Annual operational emissions were calculated directly for years 2018, 2019,

2026, 2033, and 2038. Emissions for intermediate years were interpolated. Operational emissions beyond 2038 were assumed to remain constant at 2038 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 2013. The CEQA baseline cancer risk was evaluated using 2013 activity levels and 2013 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 2013 activity levels for each year of every exposure period, but the emission factors vary year-by-year, starting with 2013, 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 2013 activity levels applied to emission factors that vary year-by-year from 2013-2042. This approach causes the future CEQA baseline cancer risk to be lower than the CEQA baseline cancer risk, resulting in a higher project increment, because of the declining trend in future emission factors for most sources.

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, project alternatives, and NEPA baseline, annual and peak hour construction emissions of TOG and PM10 were calculated for each year of construction, 2018 and 2019. The annual emissions reflect the land disposal option for dredged materials, because the criteria pollutant dispersion modeling analysis (Appendix B2) found that land disposal yielded the highest modeled concentrations for long-term averages. Conversely, the peak hour emissions reflect the ocean disposal for dredged materials, because the criteria pollutant dispersion modeling analysis (Appendix B2) found that ocean disposal yielded the highest modeled concentrations for short-term averages. Annual and peak hour operational emissions were calculated directly for years 2018, 2019, 2026, 2033, and 2038. The emissions were estimated using the methodology and assumptions described in Section 3.1.4.1 of the EIR and Sections 2.2 and 2.4 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 emissions from these analysis years even if the emissions would not occur at the same time as other sources.

To estimate chronic and acute noncancer hazard indices for the CEQA baseline, annual and peak hour emissions of TOG and PM10 were calculated using 2013 activity levels and 2013 emission factors.

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

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 offroad construction equipment, onroad construction and drayage trucks, cargo and container ship propulsion and auxiliary engines, harborcraft, locomotives, CHE, and some worker vehicles. 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, 2016c). 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 cargo and container ship boilers, vehicle tire and brake wear, asphalt paving offgas, and most worker vehicles. For these sources, TOG and PM10 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 PM10 emissions into individual TACs for all emission sources. Table B3-2 presents the speciation profiles that were used to convert TOG emissions into individual TACs for all emission sources.

OEHHA and CARB have not established acute toxicity factors for DPM. Therefore, peak hour TOG and PM10 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 PM10

Toxic Air Contaminant	HARP2 TAC ID	Speciation Profile and TAC Weight Fraction						
		Profile 42514: Diesel Vehicles[a]	Profile 119: Marine Vessels Liquid Fuel[a]	Profile 4251: Marine Vessels MGO[a]	Profile 112: Fuel Combustion Distillate	Profile 400: Gasoline Vehicles	Profile 473: Brake Wear	Profile 472: Tire Wear
Arsenic	7440382	0	0	0	0.00542	0	0.00001	0
Cadmium	7440439	0	0	0	0.0005	0	0	0
Chlorine	7782505	0	0	0	0	0.07	0.0015	0.0078
Copper	7440508	0.000356	0	0	0	0.0005	0.0115	0.00049
Hexavalent Chromium [b]	18540299	0.0000304	0	0	0.000271	0.000025	0.00006	0.0000015

Lead	7439921	0	0	0	0.0055	0	0.00005	0.00016
Manganese	7439965	0	0	0	0	0.0005	0.0017	0.0001
Nickel	7440020	0	0	0	0.0005	0.0005	0.00066	0.00005
Selenium	7782492	0	0	0	0.0005	0	0.00002	0.00002
Sulfates	9960	0.286	0.15	0.08	0.25	0.45	0.0334	0.0025
Vanadium	7440622	0	0.0055	0	0	0	0.00066	0
Applicable sources:		Construction equipment, trucks, locomotives, diesel automobiles, CHE	Harbor-craft	Ship main and auxiliary engines	Ship boilers	Gasoline automobiles	Brake wear	Tire wear

Notes:
a. Profiles No. 42514, 119, and 4251 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 2016c).
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 TOG

Toxic Air Contaminant	HARP2 TAC ID	Speciation Profile and TAC Weight Fraction			
		Profile 818: Diesel IC Engines [a]	Profile 504: Boilers	Profile 2113: Automobiles	Profile 715: Asphalt Offgas
Acetaldehyde	75070	0.0735	0	0.0026	0
Acrolein	107028	0	0	0.0012	0
Acrylonitrile	107131	0	0	0	0
Benzene	71432	0.02	0.0216	0.0227	0
1,3-Butadiene	106990	0.0019	0	0.005	0
Chlorobenzene	108907	0	0.0005	0	0
Ethyl benzene	100414	0.0031	0.0007	0.0096	0
Ethyl chloride	75003	0	0	0	0
Ethylene oxide	75218	0	0	0	0
Formaldehyde	50000	0.147	0.001	0.0145	0
Hexane	110543	0.0016	0.0159	0.0147	0
Isopropyl alcohol	67630	0	0	0	0
Methanol	67561	0.0003	0	0.0011	0
Methyl ethyl ketone	78933	0.0148	0	0.0002	0
Naphthalene	91203	0.0009	0.0007	0.0004	0.0653
Phenol	108952	0	0	0	0
Propylene	115071	0.026	0.0456	0.0281	0
Propylene oxide	75569	0	0	0	0
Styrene	100425	0.0006	0	0.0011	0
Toluene	108883	0.0147	0.0215	0.0529	0
Vinyl chloride	75014	0	0	0	0
Xylenes	1330207	0.0105	0.011	0.0441	0
Applicable sources:		Construction equipment, trucks, locomotives, CHE, harbor-craft, ship main and auxiliary engines	Ship boilers	Auto-mobiles	Asphalt offgas

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 2016c).
b. Only TACs that have OEHHA/CARB toxicity factors are shown in the table.
c. Source for speciation profiles: CARB, 2016b.

3.0 Receptors

The HRA modeled TAC concentrations and health effects at 3,564 locations (receptors) throughout the project area, including the locations of potential exposure for residents, offsite 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 14.5 km. Receptor points were also placed along the Everport 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 schools, child care centers, elder care facilities, hospitals, and recreational areas in the vicinity of the terminal. Figures B3-1 and B3-2 show the receptor points modeled in the HRA.

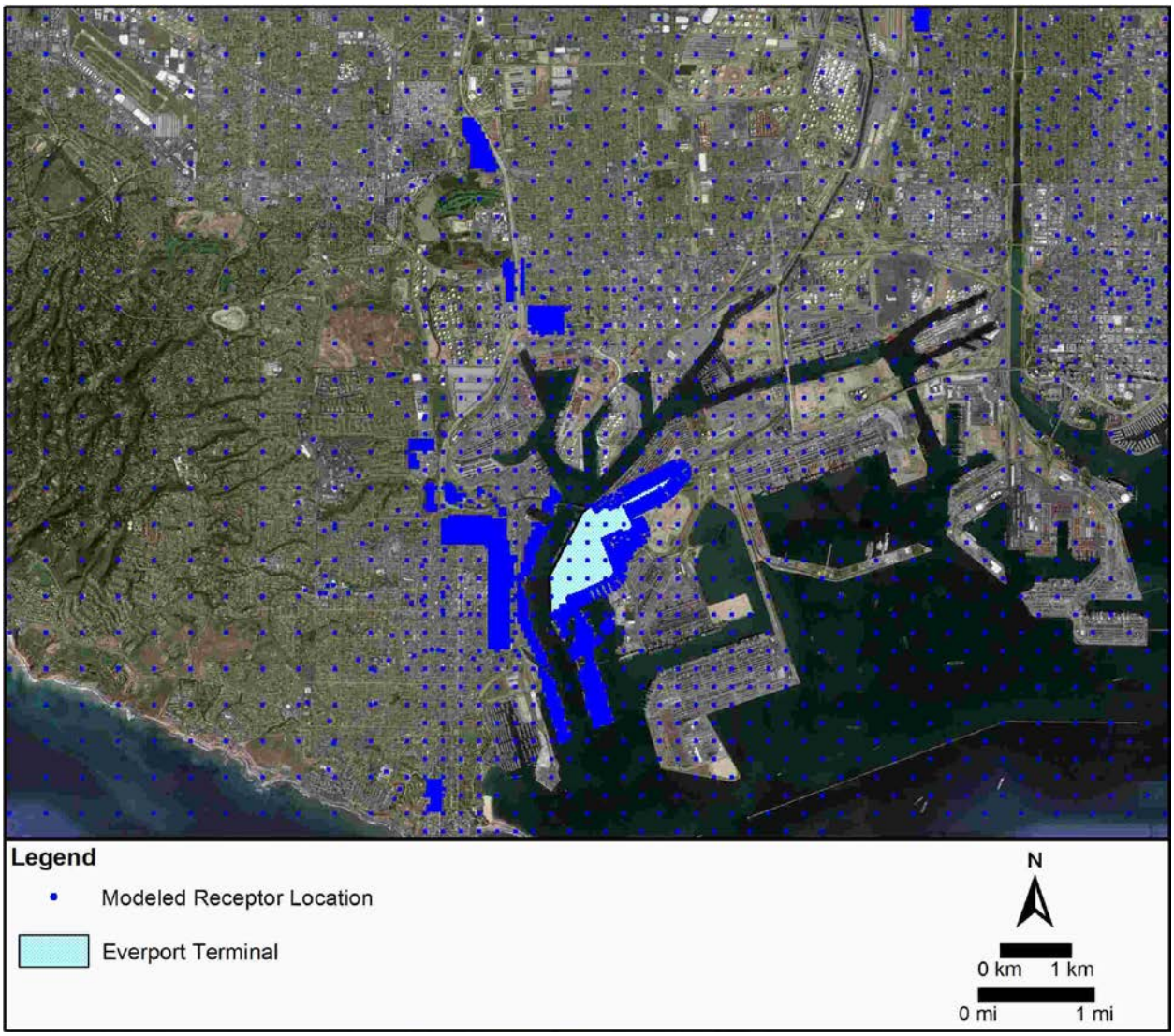

Figure B3-1: HRA Receptor Locations (Far Field)

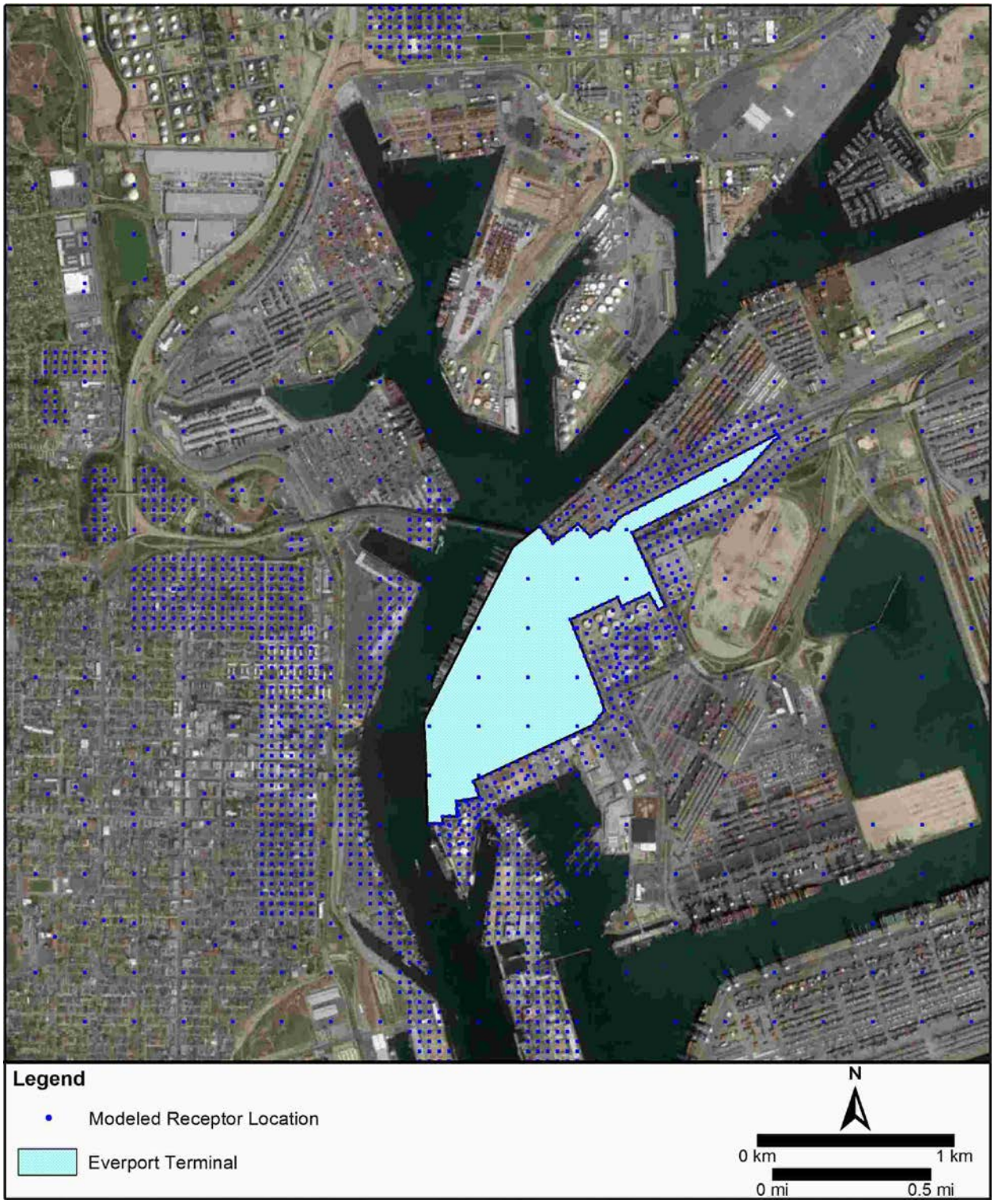

Figure B3-2: HRA Receptor Locations (Near Field)

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 that are not located within modeled roadways or railways. 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 that are not located on water or within modeled roadways or railways.
- The sensitive MEI was selected from all modeled schools, child care centers, elder care facilities, hospitals, and recreational areas such as parks, marinas, and public waterfront areas.

4.0 Health Risk Calculation Approach

4.1 Model Selection

The air dispersion modeling was performed using the USEPA AERMOD dispersion model, version 15181 (USEPA, 2015), based on the Guideline on Air Quality Models (USEPA, 2005). 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, version 16088 (CARB, 2016), 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, NEPA baseline, proposed Project, and project alternatives. For each health value calculated by HARP2, the HRA determined a CEQA increment, future CEQA increment, and NEPA increment by subtracting the CEQA baseline, future CEQA baseline, and NEPA 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.

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,

2016c). 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, and mother's milk ingestion (the latter two pathways were evaluated only for residential and the following sensitive receptors: schools, hospitals, child care, and elder care). 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 B3-3 presents the toxicity factors used to assess health risks in this study.

Table B3-3: 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
Acrolein	107028	—	0.35	I	2.5	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	—	—
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 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, 2016c.

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 to age 2 (labeled by OEHHA as "0 < 2"); by 3 for children from age 2 to 16 ("2 < 16"), and by 1 for persons age 16 and older.

Table B3-4 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) and SCAQMD (2015). The exposure assumptions for sensitive receptors are not explicitly provided by OEHHA (2015) and SCAQMD (2015). Therefore, LAHD conservatively evaluated schools, hospitals, elder care facilities, and child care centers with 30-year residential exposure

assumptions, and recreational receptors with reasonable worst case exposure assumptions of 250 days/year, 2 hr/day, for 30 years.

Because the future CEQA baseline, NEPA 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 B3-4 into smaller time periods (sub-periods) and run HARP2 separately for each sub-period. These sub-periods correspond to the years when the modeled receptor's age falls within the ranges defined by the age sensitivity factors ($0 < 2$, $2 < 16$, and ≥ 16). For residential exposures, the range $0 < 2$ also includes the 3rd trimester before birth.

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 sub-period modeled in HARP2, the average annual project or baseline emissions that would occur during that sub-period were used by HARP2. The HARP2 cancer risk results for each sub-period 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 sub-periods. The first sub-period represents a receptor age of $0 < 2$, assumes an exposure duration of 2 years, and uses Project emissions averaged over the time period 2018-2019. The second sub-period represents a receptor age of $2 < 16$, assumes an exposure duration of 14 years, and uses Project emissions averaged over the time period 2020-2033. The third sub-period represents a receptor age of $16 < 30$, assumes an exposure duration of 14 years, and uses Project emissions averaged over the time period 2034-2047. The cancer risks calculated by HARP2 for these three sub-periods were then summed to obtain the total cancer risks for the entire 30-year exposure duration.

Other HARP2 assumptions for the calculation of cancer risk include: residential and sensitive receptors except recreational were evaluated with inhalation, soil ingestion, dermal contact, mother's milk ingestion, and homegrown garden ingestion pathways. Occupational and recreational receptors were evaluated with inhalation, soil ingestion, and dermal contact pathways. A deposition settling velocity of 0.02 meters per second was assumed in HARP2 for all noninhalation exposure pathways (SCAQMD, 2015).

Table B3-4: Cancer Risk Exposure Assumptions by Receptor Type

Receptor Type	Exposure Duration			Cancer Risk Calculation Approach	Exposed Person's Age Range [6]
	Days per Year	Hours per Day	Years		
Residential					
Individual Cancer Risk	350	24	30	RMP Using the Derived Method [2]	3TM[5] < 30
Population Cancer Burden	350	24	70	RMP Using the Derived Method	3TM < 70
Occupational	250	8	25	OEHHA Derived Method [3]	≥ 16
Sensitive					
Schools, Hospitals, Elder Care, Child Care	350	24	30	RMP Using the Derived Method	3TM < 30
Recreational [4]	250	2	30	OEHHA Derived Method	0 < 30

Notes:

1. The exposure assumptions for residential and occupational receptors were obtained from OEHHA (2015) and SCAQMD (2015). The exposure assumptions for sensitive receptors are not explicitly provided by OEHHA (2015) and SCAQMD (2015). Therefore, LAHD conservatively evaluated schools, hospitals, elder care, and child care with 30-year residential exposure assumptions, and recreational receptors with reasonable worst case exposure assumptions.
2. The "RMP Using the Derived Method" uses CARB's Risk Management Policy (RMP), and is recommended by the SCAQMD (2015) for residential receptors. It uses high end breathing rates (95th percentile) for children from the 3rd trimester through age 2, and 80th percentile breathing rates for all other ages.
3. The "OEHHA Derived Method" is recommended by the SCAQMD (2015) for occupational receptors. For cancer risk, it uses high end (95th percentile) exposure parameters for the top two exposure pathways (one of which is nearly always inhalation), and mean (65th percentile) exposure parameters for the remaining pathways.
4. Recreational receptors were modeled in HARP2 with occupational exposure assumptions, which reflect 8 hours per day of pollutant exposure. Therefore, the HARP2-calculated risk values for recreational receptors were scaled by 2 hr/8 hr to reflect 2 hours per day of pollutant exposure.
5. 3TM = third trimester (prior to birth).
6. The exposed person's age ranges were conservatively selected to maximize the cancer risk (i.e., the youngest expected age range).

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.

4.4 Exposure Scenarios for Non-Cancer Hazard Indices

Chronic hazard indices were calculated in HARP2 using the "OEHHA Derived" method, which evaluates inhalation exposure, the two most dominant noninhalation exposure pathways using high-end (95th percentile) intake rates, and the remaining noninhalation exposure pathways using mean (65th percentile) intake rates (SCAQMD, 2015). All receptors were conservatively evaluated with inhalation, soil ingestion, dermal contact,

mother's milk ingestion, and homegrown garden ingestion pathways. A deposition settling velocity of 0.02 meters per second was assumed in HARP2 for all noninhalation exposure pathways (SCAQMD, 2015).

Acute hazard indices were calculated in HARP2 using the conservative "simple" approach, whereby the highest pollutant concentrations generated by each modeled source group in AERMOD are summed, even if they would not occur at the same time. Although this approach can produce a substantial overstatement of the acute hazard index, it is sufficient to use as a screening approach to demonstrate that the significance threshold would not be exceeded. HARP2 evaluates only the inhalation exposure pathway for the acute hazard index.

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 and NEPA baselines 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

CEQA Impacts without Mitigation

Table B3-5 presents the maximum predicted CEQA health impacts associated with the unmitigated proposed Project. 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 proposed Project (before subtracting baseline), the two CEQA baselines, the CEQA increment (proposed Project minus CEQA baseline), and the future CEQA increment (proposed Project minus future CEQA baseline). The table also presents the CEQA increment and future CEQA increment for the population cancer burden.

Figure B3-3 shows the maximum CEQA increment and future CEQA increment receptor locations for individual cancer risk, chronic hazard index, and acute hazard index for the unmitigated proposed Project.

Table B3-5: Maximum CEQA Health Impacts Estimated for Construction and Operation of the Proposed Project Without Mitigation

Health Impact	Receptor Type	Proposed Project[a]	CEQA Baseline	CEQA Increment[b,d,e]	Future CEQA Baseline	Future CEQA Increment[c,d]	Significance Threshold[f]	Significant?[g]
Individual Cancer Risk	Residential	59.2 × 10-6 59.2 in a million	104.0 × 10-6 104.0 in a million	< 0	64.7 × 10-6 64.7 in a million	1.3 × 10-6 1.3 in a million	10×10^{-6} 10 in a million	No
	Occupational	21.5 × 10-6 21.5 in a million	66.0 × 10-6 66.0 in a million	< 0	22.0 × 10-6 22.0 in a million	5.8 × 10-6 5.8 in a million		No
	Sensitive	45.8 × 10-6 45.8 in a million	94.1 × 10-6 94.1 in a million	< 0	57.6 × 10-6 57.6 in a million	0.8 × 10-6 0.8 in a million		No
Chronic Hazard Index	Residential	0.23	0.16	0.07	n/a[h]	n/a[h]	1.0	No
	Occupational	0.41	0.38	0.16	n/a	n/a		No
	Sensitive	0.40	0.28	0.12	n/a	n/a		No
Acute Hazard Index	Residential	0.11	0.06	0.06	n/a	n/a	1.0	No
	Occupational	0.27	0.16	0.20	n/a	n/a		No
	Sensitive	0.16	0.07	0.10	n/a	n/a		No
Population Cancer Burden			**CEQA Increment**		**Future CEQA Increment**		0.5	No
			0.0		0.1			

Notes:
[a]The Proposed Project column represents the maximum health values prior to subtracting the CEQA baseline or Future CEQA Baseline.
[b]The CEQA Increment column represents the maximum difference of the Proposed Project minus the CEQA baseline.
[c]The Future CEQA Increment column represents the maximum difference of the Proposed Project minus the Future CEQA baseline.
[d]The maximum health values for the Proposed Project, CEQA Baseline, and CEQA Increment may not all occur at the same receptor location. Therefore, the displayed values for the Proposed Project and CEQA Baseline may not necessarily subtract to equal the CEQA Increment. The same is true for the Proposed Project, Future CEQA Baseline, and Future CEQA Increment. The example given in the text illustrates how the increments are calculated.
[e]A CEQA Increment less than zero means that the Proposed Project health values would be less than the CEQA Baseline health values at all modeled receptors.
[f]The significance thresholds apply only to the CEQA increment and Future CEQA increment.
[g]Exceedances of the thresholds are indicated in **bold**. An impact is marked significant if either the CEQA Increment or Future CEQA Increment exceeds the threshold.
[h]The Future CEQA baseline and Future CEQA increment are applicable only to cancer risk because cancer risk has a uniquely long exposure period (30 years for residential and sensitive, and 25 years for occupational).
[i]Each positive 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.

The health values for the proposed Project (before subtracting baseline), CEQA baselines, and CEQA increments in Table B3-5 often occur at different modeled receptor locations from one another. This means that the displayed CEQA increments are not necessarily equal to the displayed proposed Project results minus the displayed CEQA baseline results, although all displayed values are correct. Instead, an increment must be calculated at each of the hundreds of modeled receptors, and the receptor with the highest increment is presented in the table. The following example shows how the maximum future CEQA increment for cancer risk at a residential receptor (1.3 in a million), shown in the first row of results in Table B3-5, was determined. This result is predicted to occur at modeled Receptor No. 3044, in Harbor City, adjacent to the Harbor Freeway.

Example—Determine Future CEQA Increment at Receptor No. 3044:

- Proposed Project cancer risk, Receptor No. 3044 = 14.8 in a million (not shown in the table because this receptor is not the location of the maximum proposed Project cancer risk before subtracting baseline)
- Future CEQA baseline cancer risk, Receptor No. 3044 = 13.5 in a million (not shown in the table because this receptor is not the location of the maximum future CEQA baseline cancer risk)
- Future CEQA increment, Receptor No. 3044 = 14.8 – 13.5 = 1.3 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. 3044 has the highest future CEQA increment of any residential receptor. Therefore, its value of 1.3 in a million is reported in Table B3-5. However, in this example, Receptor 3044 is *not* the maximum residential receptor for either the proposed Project before subtracting baseline (its maximum of 59.2 in a million occurs at Receptor No. 2613) or the future CEQA baseline (its maximum of 64.7 in a million also occurs at Receptor No. 2613). The CEQA increment at Receptor No. 2613 is -5.5 in a million (a risk reduction), less than the maximum increment of 1.3 in a million at Receptor No. 3044.

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 more than 3,500 modeled receptors prior to selection of the maximum receptor. All of the CEQA and NEPA increments for individual cancer risk, chronic hazard index, and acute hazard index are determined in a similar way.

Table B3-5 shows that the unmitigated proposed Project would produce the following health risk impacts under CEQA:

- Individual Cancer Risk

In relation to the CEQA baseline, the maximum incremental cancer risk is predicted to be less than the significance threshold at all receptors. Moreover, the negative values for the CEQA increment indicate that the cancer risk from the proposed Project would be less than the cancer risk from the CEQA baseline at all modeled residential, occupational, and sensitive receptors, due in large part to the beneficial effect of existing air quality rules and regulations on future emissions. In relation to the future CEQA baseline, the

maximum incremental cancer risk is predicted to be less than the significance threshold at all receptors.

Figure B3-4 shows individual cancer risk contours of the future CEQA increment for the unmitigated proposed Project, assuming residential (30-year) exposure parameters. As shown in the figure, the maximum residential receptor for individual cancer risk is located outside the 10 in a million contour line, indicating a less than significant impact.

Residential cancer risk contours of the CEQA increment (as opposed to the future CEQA increment) are not shown because, as stated in the previous paragraph, the CEQA increment is predicted to be less than zero at all modeled residential receptors.

- Population Cancer Burden

In relation to the CEQA baseline, the cancer burden increment would be zero because the individual cancer risk associated with the proposed Project would be less than the CEQA baseline at all modeled receptors. In relation to the Future CEQA baseline, the cancer burden increment is predicted to be less than the significance threshold.

- Chronic and Acute Hazard Indices

The maximum chronic and acute hazard index increments are predicted to be less than the significance threshold for all receptor types.

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

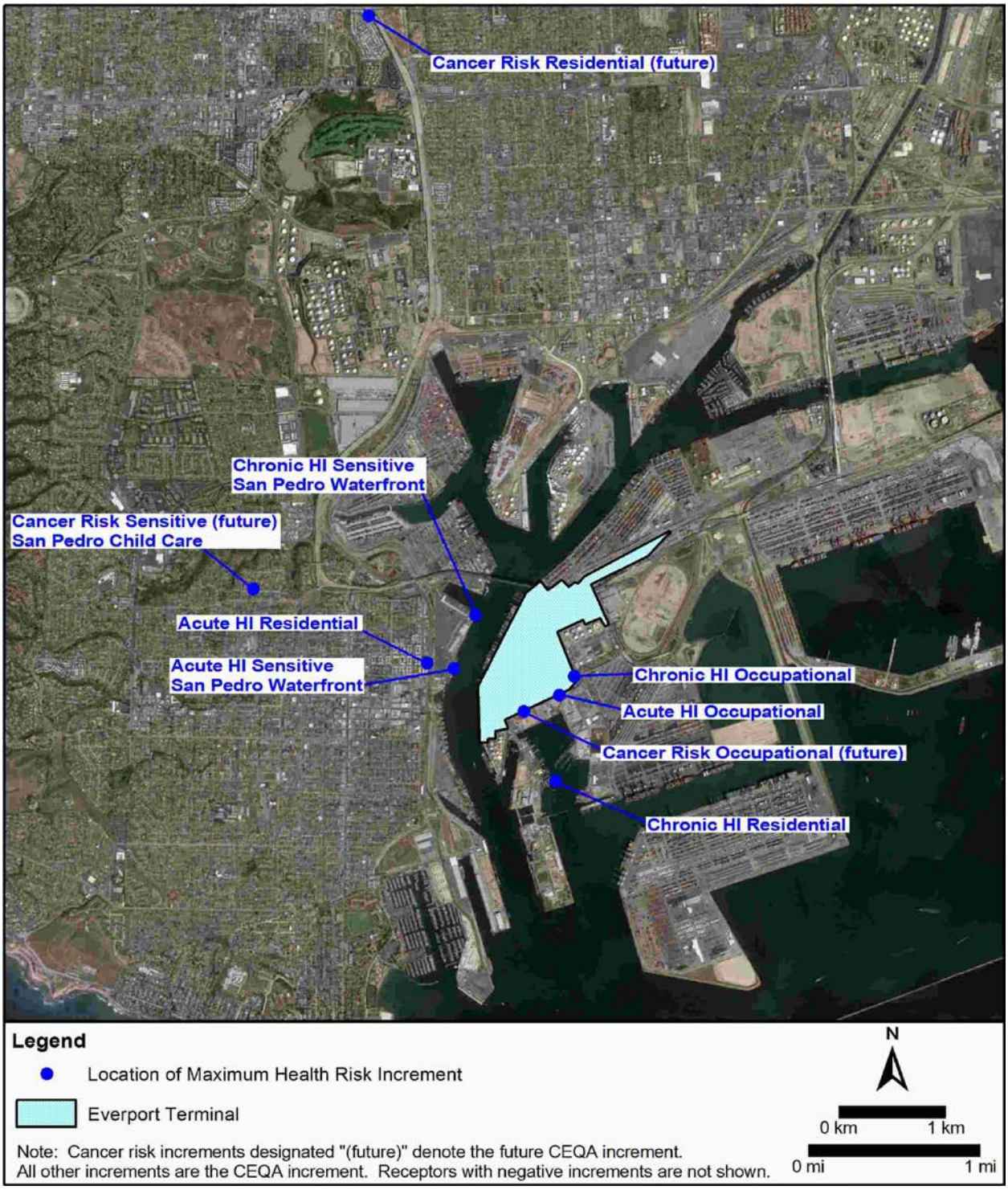

Figure B3-3: Locations of Maximum CEQA Health Impacts Estimated for Construction and Operation of the Proposed Project without Mitigation

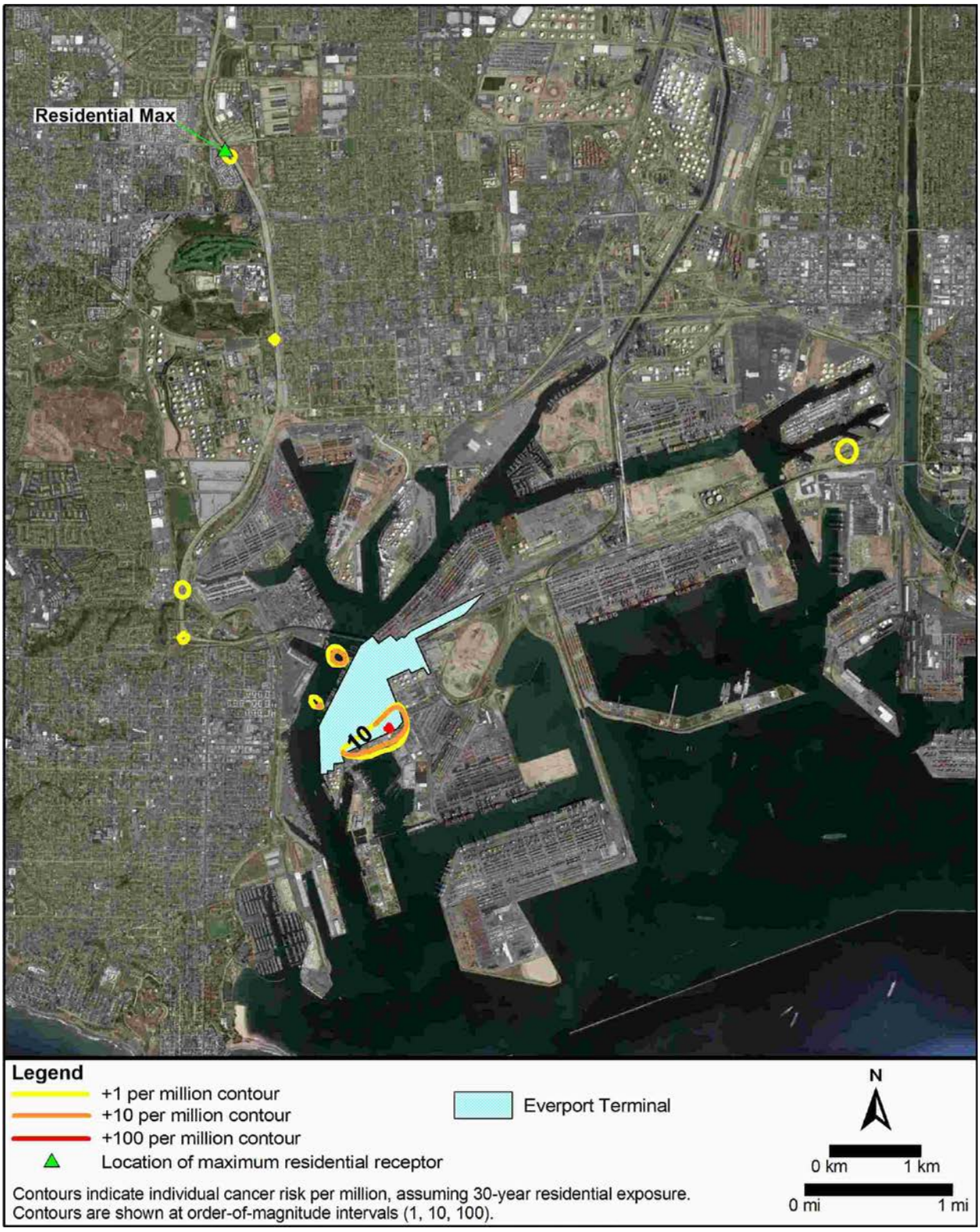

Figure B3-4: Isopleths of 30-Year Residential Cancer Risk – Proposed Project without Mitigation – Future CEQA Increment

NEPA Impacts without Mitigation

Table B3-6 presents the maximum predicted NEPA health impacts associated with the unmitigated proposed Project. 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 proposed Project (before subtracting baseline), the NEPA baseline, and the NEPA increment (proposed Project minus NEPA baseline). The table also presents the NEPA increment for the population cancer burden.

Figure B3-5 shows the maximum NEPA increment receptor locations for individual cancer risk, chronic hazard index, and acute hazard index for the unmitigated proposed Project.

Table B3-6: Maximum NEPA Health Impacts Estimated for Construction and Operation of the Proposed Project Without Mitigation

Health Impact	Receptor Type	Proposed Project[a]	NEPA Baseline	NEPA Increment[b,c]	Significance Threshold[d]	Significant?[e]
Individual Cancer Risk	Residential	59.2 × 10-6 59.2 in a million	43.0 × 10-6 43.0 in a million	**16.1 × 10-6** **16.1 in a million**	10×10^{-6} 10 in a million	**Yes**
	Occupational	21.5 × 10-6 21.5 in a million	17.0 × 10-6 17.0 in a million	4.6 × 10-6 4.6 in a million		No
	Sensitive	45.8 × 10-6 45.8 in a million	34.0 × 10-6 34.0 in a million	**11.7 × 10-6** **11.7 in a million**		**Yes**
Chronic Hazard Index	Residential	0.23	0.18	0.05	1.0	No
	Occupational	0.41	0.31	0.13		No
	Sensitive	0.40	0.30	0.11		No
Acute Hazard Index	Residential	0.11	0.07	0.06	1.0	No
	Occupational	0.27	0.26	0.09		No
	Sensitive	0.16	0.07	0.09		No
Population Cancer Burden				**0.6**	0.5	**Yes**

Notes:
[a]The Proposed Project column represents the maximum health values prior to subtracting the NEPA baseline.
[b]The NEPA Increment column represents the maximum difference of the Proposed Project minus the NEPA baseline.
[c]The maximum health values for the Proposed Project, NEPA Baseline, and NEPA Increment may not all occur at the same receptor location. Therefore, the displayed values for the Proposed Project and NEPA Baseline may not necessarily subtract to equal the NEPA Increment. The example given in the text illustrates how the increments are calculated.
[d]The significance thresholds apply only to the NEPA increment.
[e]Exceedances of the thresholds are indicated in **bold**.
[f]Each positive 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.

Table B3-6 shows that the unmitigated proposed Project would produce the following health risk impacts under NEPA:

- Individual Cancer Risk

In relation to the NEPA baseline, the maximum incremental cancer risk is predicted to be greater than the significance threshold at the maximally impacted residential and sensitive receptors. The cancer risk impact would be less than the threshold at occupational receptors.

Table B3-7 shows the emission source contributions to cancer risk from the unmitigated proposed Project at the residential and sensitive receptor locations with the highest predicted NEPA increments. The highest source contributor is container ships (in transit, at berth, and at anchorage), which would contribute a combined 41 percent of the cancer risk at the maximum residential receptor, and 38 percent of the cancer risk at the maximum sensitive receptor.

Figure B3-6 shows individual cancer risk contours of the NEPA increment for the unmitigated proposed Project, assuming residential (30-year) exposure parameters.

Table B3-7: Source Contributions to Cancer Risk at the Maximum Residential and Sensitive NEPA Increment Receptors – Proposed Project Without Mitigation

Source Category	Maximum Residential Receptor (percent)	Maximum Sensitive Receptor (percent)
Construction		
Offroad Equipment	14.9	13.3
Onroad Vehicles	0.04	0.03
Crane Delivery Ship	0.1	0.1
Harborcraft	2.2	2.7
Asphalt Paving	0.007	0.005
Operation		
Ships in Transit	27.9	27.7
Ships at Berth	12.9	9.8
Ships at Anchorage	0.2	0.2
Tugboats	3.9	5.1
Trucks at Gates and On-Terminal	2.1	1.7
Trucks Driving Off-Terminal	3.4	4.2
Locomotives	4.0	4.8
Cargo Handling Equipment	28.1	30.2
Worker Vehicles	0.1	0.2

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

- Population Cancer Burden

In relation to the NEPA baseline, the cancer burden increment is predicted to be greater than the significance threshold.

- Chronic and Acute Hazard Indices

The maximum chronic and acute hazard index increments are predicted to be less than the significance threshold for all receptor types.

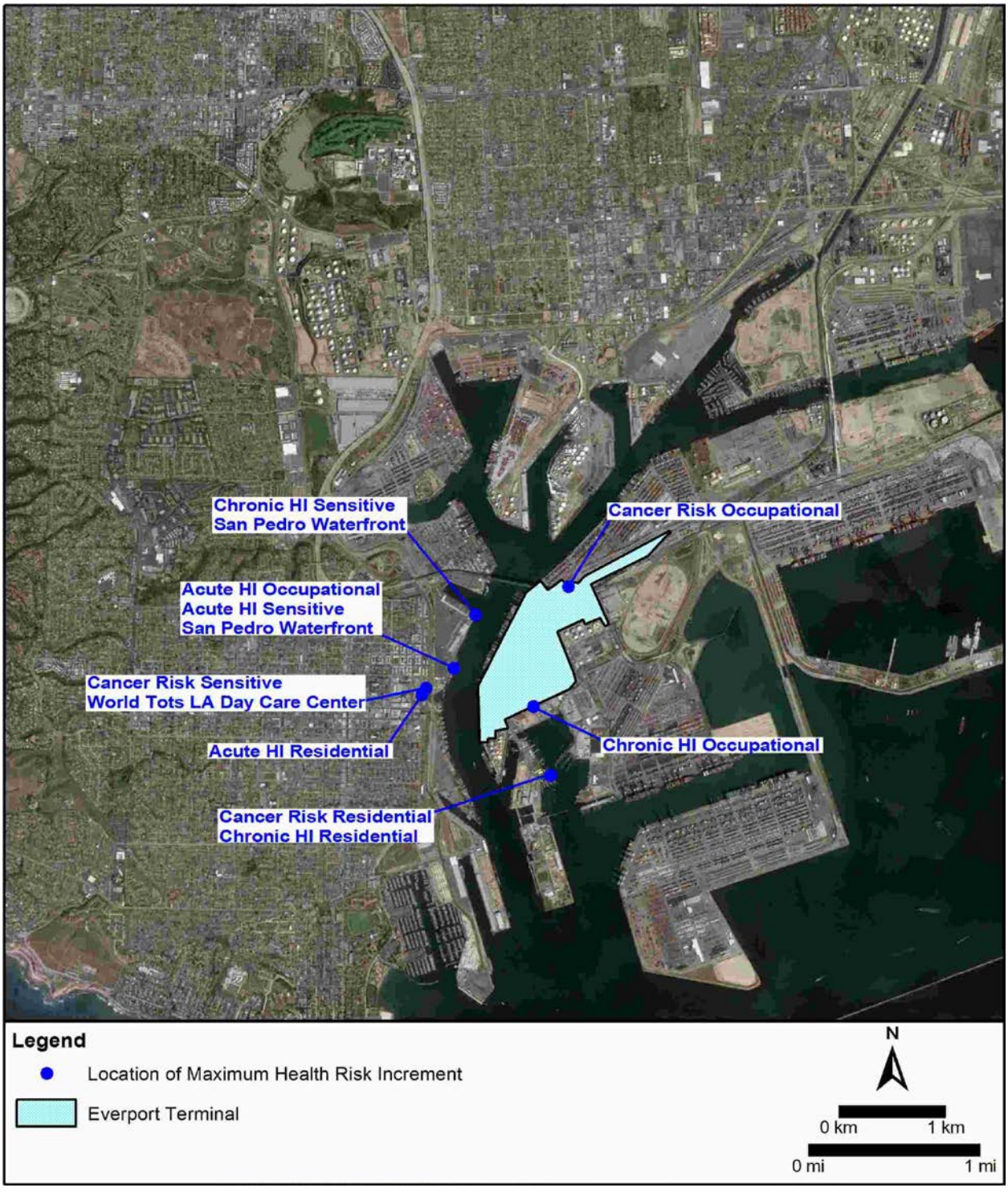

Figure B3-5: Locations of Maximum NEPA Health Impacts Estimated for Construction and Operation of the Proposed Project without Mitigation

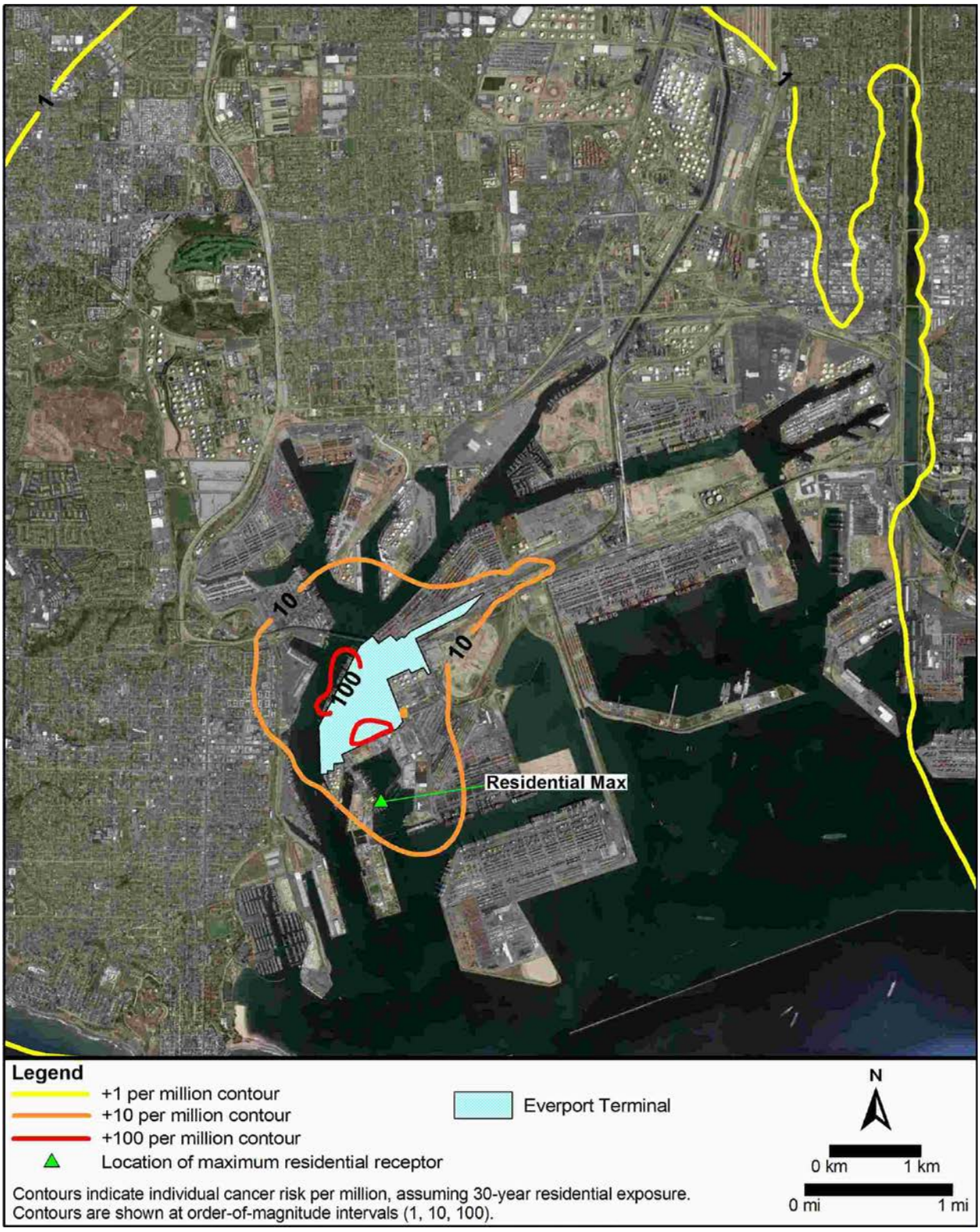

Figure B3-6: Isopleths of 30-Year Residential Cancer Risk – Proposed Project without Mitigation – NEPA Increment

NEPA Impacts with Mitigation

To reduce the NEPA health risk impacts associated with the proposed Project, MM AQ-1 through AQ-5 would be applied during construction, and MM AQ-6 and MM AQ-7 would be applied during operation. The mitigation measures are described in Impacts AQ-1 and AQ-3 of Section 3.1.

Table B3-8 presents the maximum predicted NEPA health impacts associated with the mitigated proposed Project. The table shows that, with mitigation, the maximum incremental cancer risk at residential and sensitive receptors would be reduced to less than the significance thresholds. The population cancer burden would also be reduced to less than the threshold. All other health risk values would remain less than the thresholds.

Figure B3-7 shows the maximum NEPA increment receptor locations for individual cancer risk, chronic hazard index, and acute hazard index for the mitigated proposed Project.

Figure B3-8 shows individual cancer risk contours of the NEPA increment for the mitigated proposed Project, assuming residential (30-year) exposure parameters. As shown in the figure, the maximum residential receptor for individual cancer risk is located outside the 10 in a million contour line, indicating a less than significant impact.

Table B3-8: Maximum NEPA Health Impacts Estimated for Construction and Operation of the Proposed Project With Mitigation

Health Impact	Receptor Type	Proposed Project[a]	NEPA Baseline	NEPA Increment[b,c]	Significance Threshold[d]	Significant?[e]
Individual Cancer Risk	Residential	50.9 × 10-6 50.9 in a million	43.0 × 10-6 43.0 in a million	9.0 × 10-6 9.0 in a million	10×10^{-6} 10 in a million	No
	Occupational	21.3 × 10-6 21.3 in a million	17.0 × 10-6 17.0 in a million	4.3 × 10-6 4.3 in a million		No
	Sensitive	41.0 × 10-6 41.0 in a million	34.0 × 10-6 34.0 in a million	7.0 × 10-6 7.0 in a million		No
Chronic Hazard Index	Residential	0.22	0.18	0.05	1.0	No
	Occupational	0.40	0.31	0.10		No
	Sensitive	0.39	0.30	0.10		No
Acute Hazard Index	Residential	0.11	0.07	0.05	1.0	No
	Occupational	0.26	0.26	0.09		No
	Sensitive	0.15	0.07	0.09		No
Population Cancer Burden				0.3	0.5	No

Notes:
[a]The Proposed Project column represents the maximum health values prior to subtracting the NEPA baseline.
[b]The NEPA Increment column represents the maximum difference of the Proposed Project minus the NEPA baseline.
[c]The maximum health values for the Proposed Project, NEPA Baseline, and NEPA Increment may not all occur at the same receptor location. Therefore, the displayed values for the Proposed Project and NEPA Baseline may not necessarily subtract to equal the NEPA Increment. The example given in the text illustrates how the increments are calculated.
[d]The significance thresholds apply only to the NEPA increment.
[e]Exceedances of the thresholds are indicated in **bold**.
[f]Each positive 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.

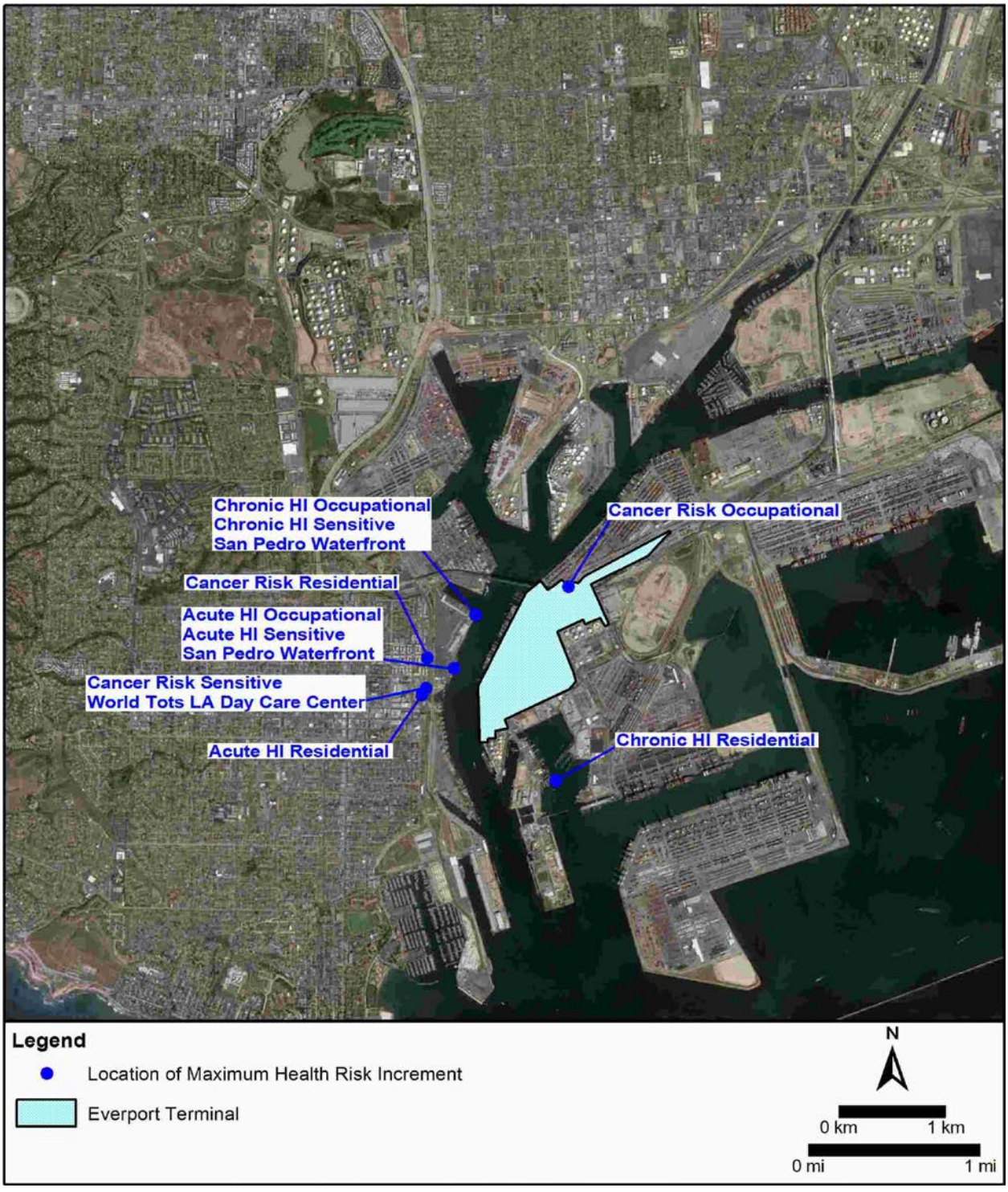

Figure B3-7: Locations of Maximum NEPA Health Impacts Estimated for Construction and Operation of the Proposed Project with Mitigation

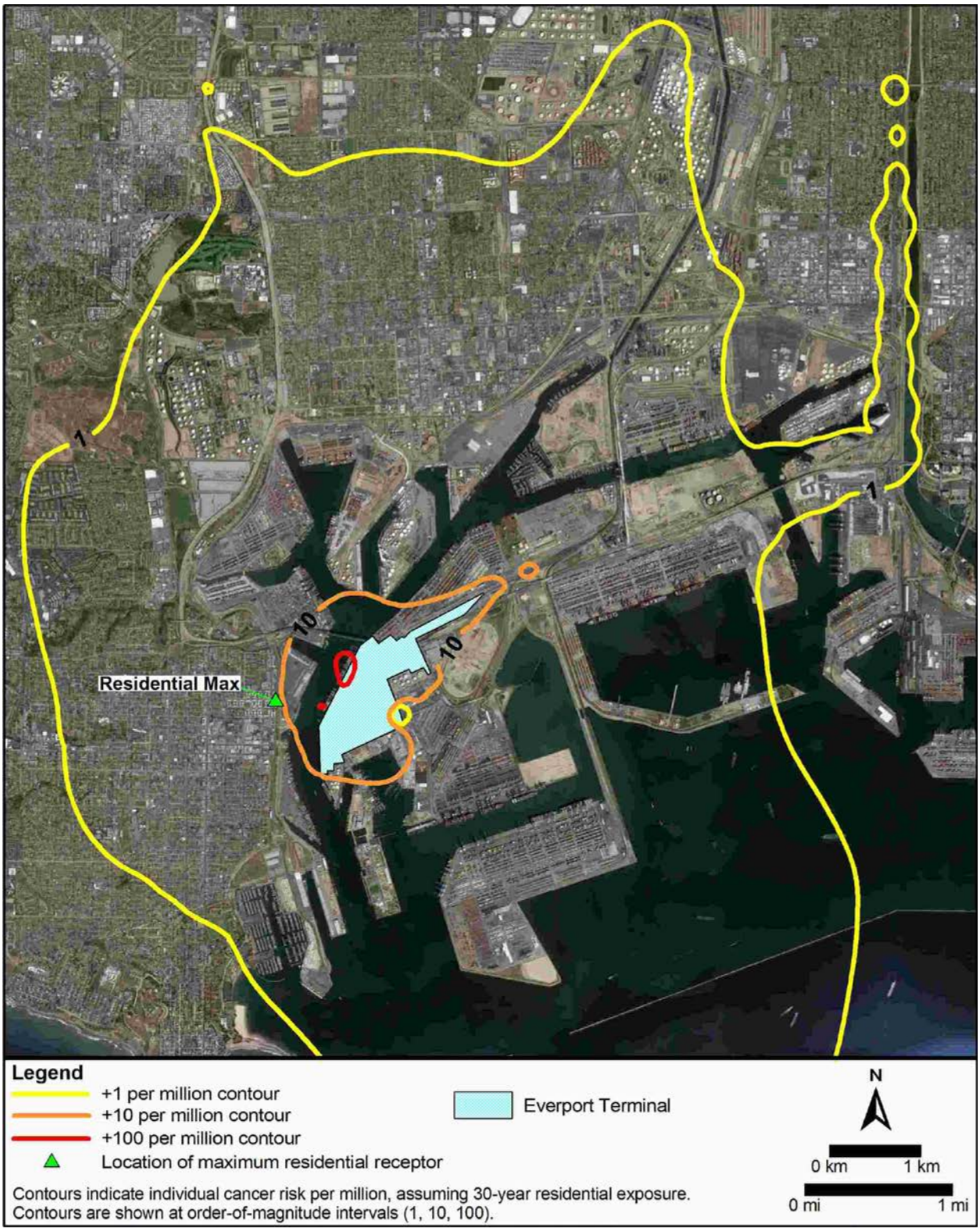

Figure B3-8: Isopleths of 30-Year Residential Cancer Risk – Proposed Project with Mitigation – NEPA Increment

6.2 Alternative 1 (No Federal Action)

CEQA Impacts without Mitigation

This alternative would include development activities in the backlands similar to the proposed Project but no construction in/over/under waters of the United States because under this alternative, no DA permit would be issued. In light of backland development authorized under local and state approvals, impacts under CEQA would occur. Table B3-9 presents the maximum predicted CEQA health impacts associated with unmitigated Alternative 1. 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 Alternative 1 (before subtracting baseline), the two CEQA baselines, the CEQA increment (Alternative 1 minus CEQA baseline), and the future CEQA increment (Alternative 1 minus future CEQA baseline). The table also presents the CEQA increment and future CEQA increment for the population cancer burden.

Figure B3-9 shows the maximum CEQA increment and future CEQA increment receptor locations for individual cancer risk, chronic hazard index, and acute hazard index for unmitigated Alternative 1.

Table B3-9: Maximum CEQA Health Impacts Estimated for Construction and Operation of Alternative 1 Without Mitigation

Health Impact	Receptor Type	Alternative 1[a]	CEQA Baseline	CEQA Increment[b,d,e]	Future CEQA Baseline	Future CEQA Increment[c,d,e]	Significance Threshold[f]	Significant?[g]
Individual Cancer Risk	Residential	49.2 × 10-6 49.2 in a million	104.0 × 10-6 104.0 in a million	< 0	64.7 × 10-6 64.7 in a million	< 0	10×10^{-6} 10 in a million	No
	Occupational	17.2 × 10-6 17.2 in a million	66.0 × 10-6 66.0 in a million	< 0	22.0 × 10-6 22.0 in a million	4.4 × 10-6 4.4 in a million		No
	Sensitive	37.1 × 10-6 37.1 in a million	94.1 × 10-6 94.1 in a million	< 0	57.6 × 10-6 57.6 in a million	< 0		No
Chronic Hazard Index	Residential	0.18	0.16	0.02	n/a[h]	n/a[h]	1.0	No
	Occupational	0.33	0.38	0.13	n/a	n/a		No
	Sensitive	0.30	0.28	0.02	n/a	n/a		No
Acute Hazard Index	Residential	0.07	0.06	0.01	n/a	n/a	1.0	No
	Occupational	0.25	0.16	0.18	n/a	n/a		No
	Sensitive	0.07	0.07	0.02	n/a	n/a		No
Population Cancer Burden			**CEQA Increment**		**Future CEQA Increment**		0.5	No
			0.0		0.0			

Notes:
[a]The Alternative 1 column represents the maximum health values prior to subtracting the CEQA baseline or Future CEQA Baseline.
[b]The CEQA Increment column represents the maximum difference of Alternative 1 minus the CEQA baseline.
[c]The Future CEQA Increment column represents the maximum difference of Alternative 1 minus the Future CEQA baseline.
[d]The maximum health values for Alternative 1, the CEQA Baseline, and the CEQA Increment may not all occur at the same receptor location. Therefore, the displayed values for Alternative 1 and the CEQA Baseline may not necessarily subtract to equal the CEQA Increment. The same is true for Alternative 1, the Future CEQA Baseline, and the Future CEQA Increment. The example given in the text under Impact AQ-7 for the proposed Project illustrates how the increments are calculated.
[e]A CEQA Increment less than zero means that the Alternative 1 health values would be less than the CEQA Baseline health values at all modeled receptors.
[f]The significance thresholds apply only to the CEQA increment and Future CEQA increment.
[g]Exceedances of the thresholds are indicated in **bold**. An impact is marked significant if either the CEQA Increment or Future CEQA Increment exceeds the threshold.
[h]The Future CEQA baseline and Future CEQA increment are applicable only to cancer risk because cancer risk has a uniquely long exposure period (30 years for residential and sensitive, and 25 years for occupational).
[i]Each positive 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.

Table B3-9 shows that unmitigated Alternative 1 would produce the following health risk impacts under CEQA:

- Individual Cancer Risk

In relation to the CEQA baseline, the maximum incremental cancer risk is predicted to be less than the significance threshold at all receptors. Moreover, the negative values for the CEQA increment indicate that the cancer risk from Alternative 1 would be less than the cancer risk from the CEQA baseline at all modeled residential, occupational, and sensitive receptors, due in large part to the beneficial effect of existing air quality rules and regulations on future emissions.

In relation to the future CEQA baseline, the maximum incremental cancer risk is predicted to be less than the significance threshold at all receptors. Moreover, the negative values for the future CEQA increment at residential and sensitive receptors indicate that the cancer risk from Alternative 1 would be less than the cancer risk from the future CEQA baseline at all modeled residential and sensitive receptors, due in large part to the beneficial effect of existing air quality rules and regulations on future emissions.

Residential cancer risk contours of the CEQA increment and future CEQA increment are not shown because, as stated in the previous paragraphs, the increments are predicted to be less than zero at all modeled residential receptors.

- Population Cancer Burden

In relation to the CEQA baseline, the cancer burden increment would be zero because the individual cancer risk associated with Alternative 1 would be less than the CEQA baseline at all modeled receptors. In relation to the Future CEQA baseline, the cancer burden increment is predicted to be less than the significance threshold.

- Chronic and Acute Hazard Indices

The maximum chronic and acute hazard index increments are predicted to be less than the significance threshold for all receptor types.

Because all CEQA health impacts are predicted to be less than the significance thresholds without mitigation, a CEQA evaluation of Alternative 1 with the mitigation measures prescribed in Section 3.1 was not necessary.

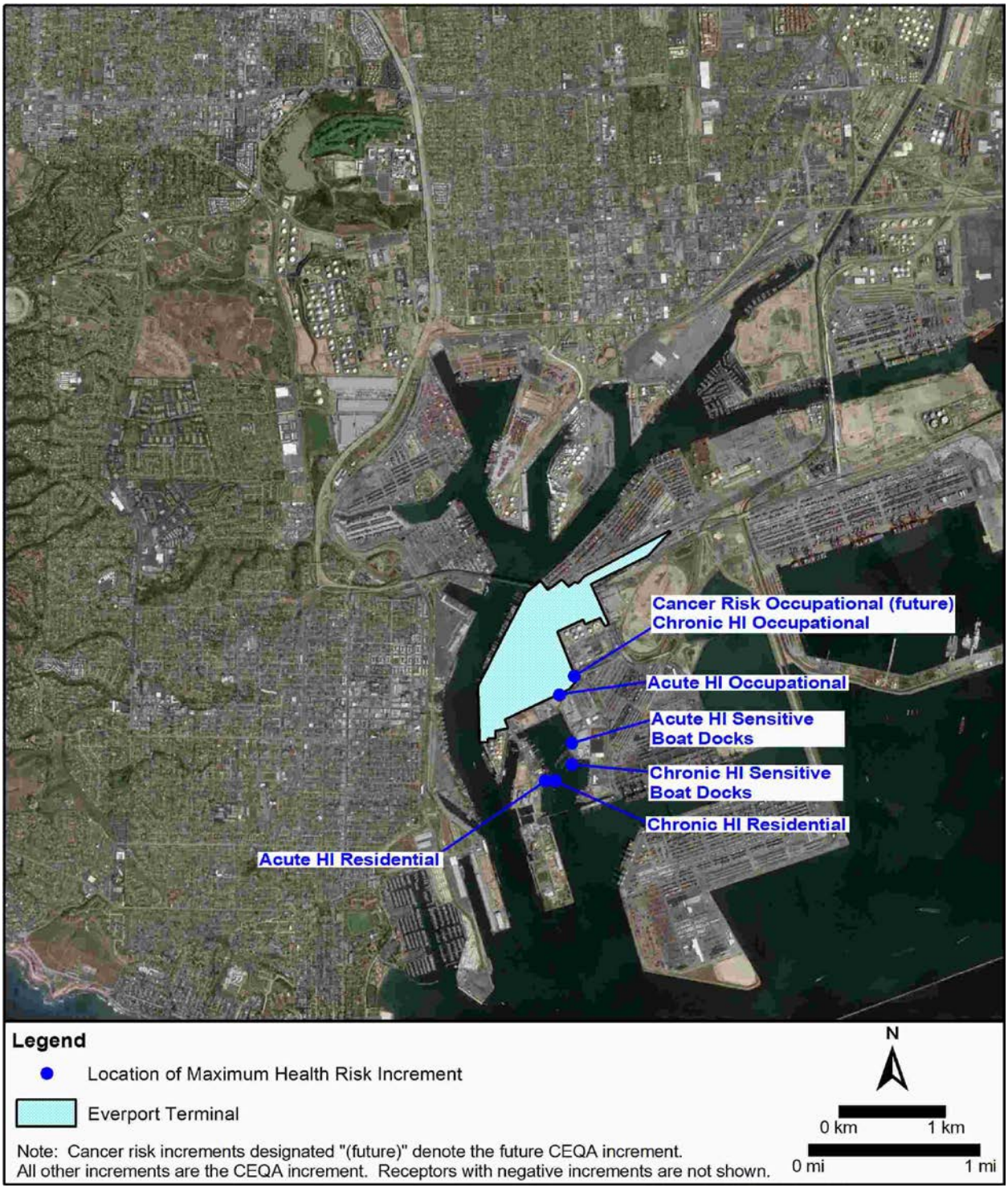

Figure B3-9: Locations of Maximum CEQA Health Impacts Estimated for Construction and Operation of Alternative 1 without Mitigation

NEPA Impacts

Alternative 1 (No Federal Action) would involve the same operational activities, at the same activity levels, as would occur under the NEPA baseline. Therefore, there would be no incremental difference between Alternative 1 and the NEPA baseline.

6.3 Alternative 2 (CEQA No Project)

CEQA Impacts

Table B3-10 presents the maximum predicted CEQA health impacts associated with operation of Alternative 2. 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 Alternative 2 (before subtracting baseline), the two CEQA baselines, the CEQA increment (Alternative 2 minus CEQA baseline), and the future CEQA increment (Alternative 2 minus future CEQA baseline). The table also presents the CEQA increment and future CEQA increment for the population cancer burden.

Figure B3-10 shows the maximum CEQA increment and future CEQA increment receptor locations for individual cancer risk, chronic hazard index, and acute hazard index for Alternative 2.

Table B3-10: Maximum CEQA Health Impacts Estimated for Operation of Alternative 2

Health Impact	Receptor Type	Alternative 2[a]	CEQA Baseline	CEQA Increment[b,d,e]	Future CEQA Baseline	Future CEQA Increment[c,d,e]	Significance Threshold[f]	Significant?[g]
Individual Cancer Risk	Residential	41.3 × 10-6 41.3 in a million	104.0 × 10-6 104.0 in a million	< 0	64.7 × 10-6 64.7 in a million	< 0	10×10^{-6} 10 in a million	No
	Occupational	17.0 × 10-6 17.0 in a million	66.0 × 10-6 66.0 in a million	< 0	22.0 × 10-6 22.0 in a million	0.8 × 10-6 0.8 in a million		No
	Sensitive	33.5 × 10-6 33.5 in a million	94.1 × 10-6 94.1 in a million	< 0	57.6 × 10-6 57.6 in a million	< 0		No
Chronic Hazard Index	Residential	0.18	0.16	0.02	n/a[h]	n/a[h]	1.0	No
	Occupational	0.31	0.38	0.02	n/a	n/a		No
	Sensitive	0.30	0.28	0.02	n/a	n/a		No
Acute Hazard Index	Residential	0.06	0.06	0.006	n/a	n/a	1.0	No
	Occupational	0.14	0.16	0.01	n/a	n/a		No
	Sensitive	0.06	0.07	0.005	n/a	n/a		No
Population Cancer Burden			**CEQA Increment**		**Future CEQA Increment**		0.5	No
			0.0		0.0			

Notes:
[a]The Alternative 2 column represents the maximum health values prior to subtracting the CEQA baseline or Future CEQA Baseline.
[b]The CEQA Increment column represents the maximum difference of Alternative 2 minus the CEQA baseline.
[c]The Future CEQA Increment column represents the maximum difference of Alternative 2 minus the Future CEQA baseline.
[d]The maximum health values for Alternative 2, the CEQA Baseline, and the CEQA Increment may not all occur at the same receptor location. Therefore, the displayed values for Alternative 2 and the CEQA Baseline may not necessarily subtract to equal the CEQA Increment. The same is true for Alternative 2, the Future CEQA Baseline, and the Future CEQA Increment. The example given in the text under Impact AQ-7 for the proposed Project illustrates how the increments are calculated.
[e]A CEQA Increment less than zero means that the Alternative 2 health values would be less than the CEQA Baseline health values at all modeled receptors.
[f]The significance thresholds apply only to the CEQA increment and Future CEQA increment.
[g]Exceedances of the thresholds are indicated in **bold**. An impact is marked significant if either the CEQA Increment or Future CEQA Increment exceeds the threshold.
[h]The Future CEQA baseline and Future CEQA increment are applicable only to cancer risk because cancer risk has a uniquely long exposure period (30 years for residential and sensitive, and 25 years for occupational).
[i]Each positive 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.

Table B3-10 shows that Alternative 2 would produce the following health risk impacts under CEQA:

- Individual Cancer Risk

In relation to the CEQA baseline, the maximum incremental cancer risk is predicted to be less than the significance threshold at all receptors. Moreover, the negative values for the CEQA increment indicate that the cancer risk from Alternative 2 would be less than the cancer risk from the CEQA baseline at all modeled residential, occupational, and sensitive receptors, due in large part to the beneficial effect of existing air quality rules and regulations on future emissions.

In relation to the future CEQA baseline, the maximum incremental cancer risk is predicted to be less than the significance threshold at all receptors. Moreover, the negative values for the future CEQA increment at residential and sensitive receptors indicate that the cancer risk from Alternative 2 would be less than the cancer risk from the future CEQA baseline at all modeled residential and sensitive receptors, due in large part to the beneficial effect of existing air quality rules and regulations on future emissions.

Residential cancer risk contours of the CEQA increment and future CEQA increment are not shown because, as stated in the previous paragraphs, the increments are predicted to be less than zero at all modeled residential receptors.

- Population Cancer Burden

In relation to the CEQA baseline, the cancer burden increment would be zero because the individual cancer risk associated with Alternative 2 would be less than the CEQA baseline at all modeled receptors. In relation to the Future CEQA baseline, the cancer burden increment is predicted to be less than the significance threshold.

- Chronic and Acute Hazard Indices

The maximum chronic and acute hazard index increments are predicted to be less than the significance threshold for all receptor types.

There are no project components or discretionary actions under this alternative; therefore, no mitigation is applicable or required.

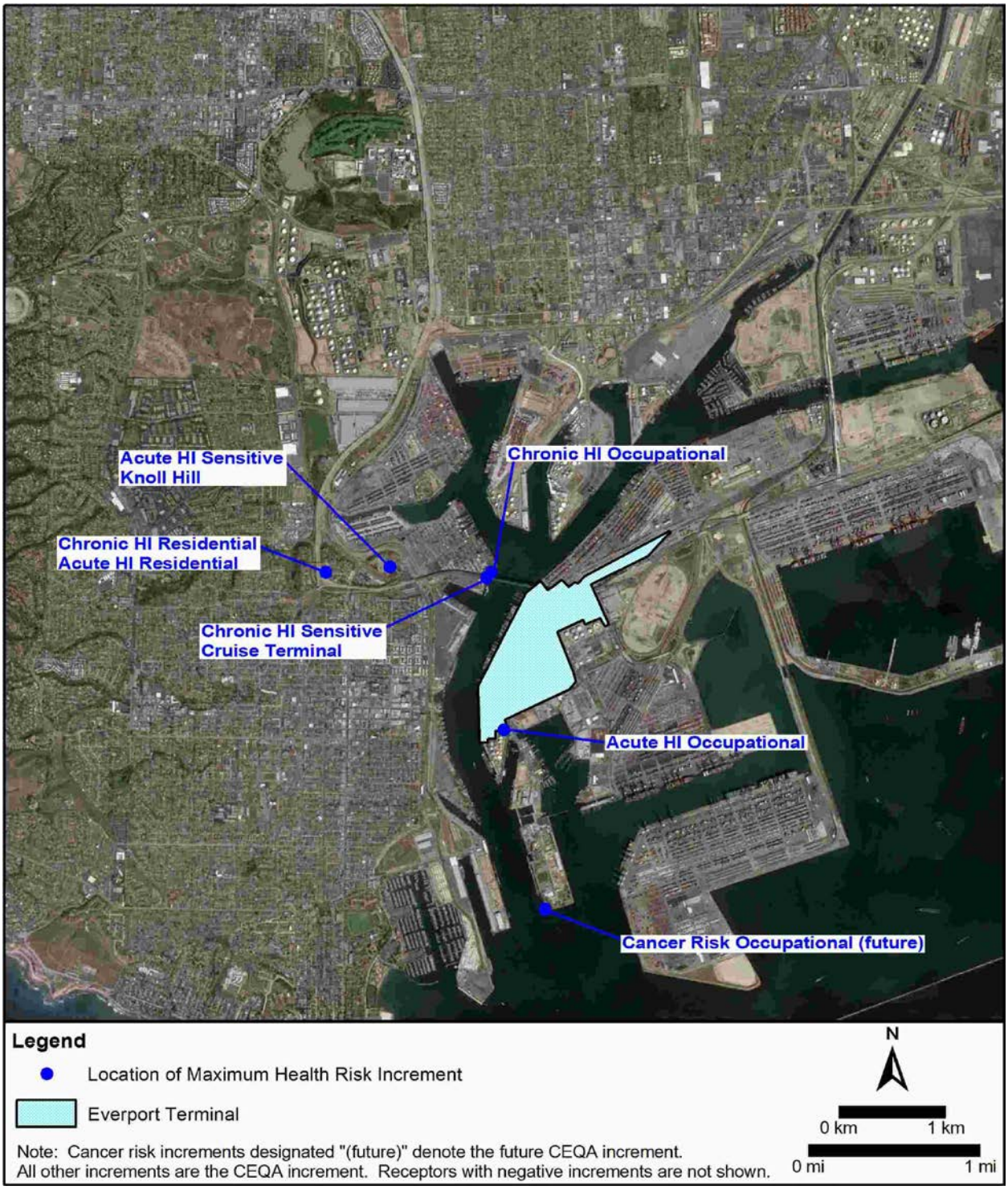

Figure B3-10: Locations of Maximum CEQA Health Impacts Estimated for Operation of Alternative 2

NEPA Impacts

NEPA does not require analysis of the No Project Alternative (Alternative 2).

6.4 Alternative 3 (Reduced Project: Reduced Wharf Improvements)

CEQA Impacts without Mitigation

Table B3-11 presents the maximum predicted CEQA health impacts associated with unmitigated Alternative 3. 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 Alternative 3 (before subtracting baseline), the two CEQA baselines, the CEQA increment (Alternative 3 minus CEQA baseline), and the future CEQA increment (Alternative 3 minus future CEQA baseline). The table also presents the CEQA increment and future CEQA increment for the population cancer burden.

Figure B3-11 shows the maximum CEQA increment and future CEQA increment receptor locations for individual cancer risk, chronic hazard index, and acute hazard index for unmitigated Alternative 3.

Table B3-11: Maximum CEQA Health Impacts Estimated for Construction and Operation of Alternative 3 Without Mitigation

Health Impact	Receptor Type	Alternative 3[a]	CEQA Baseline	CEQA Increment[b,d,e]	Future CEQA Baseline	Future CEQA Increment[c,d]	Significance Threshold[f]	Significant?[g]
Individual Cancer Risk	Residential	55.4 × 10-6 55.4 in a million	104.0 × 10-6 104.0 in a million	< 0	64.7 × 10-6 64.7 in a million	0.8 × 10-6 0.8 in a million	10×10^{-6} 10 in a million	No
	Occupational	20.6 × 10-6 20.6 in a million	66.0 × 10-6 66.0 in a million	< 0	22.0 × 10-6 22.0 in a million	5.3 × 10-6 5.3 in a million		No
	Sensitive	42.0 × 10-6 42.0 in a million	94.1 × 10-6 94.1 in a million	< 0	57.6 × 10-6 57.6 in a million	0.3 × 10-6 0.3 in a million		No
Chronic Hazard Index	Residential	0.21	0.16	0.04	n/a[h]	n/a[h]	1.0	No
	Occupational	0.37	0.38	0.14	n/a	n/a		No
	Sensitive	0.35	0.28	0.07	n/a	n/a		No
Acute Hazard Index	Residential	0.09	0.06	0.05	n/a	n/a	1.0	No
	Occupational	0.25	0.16	0.19	n/a	n/a		No
	Sensitive	0.13	0.07	0.07	n/a	n/a		No
Population Cancer Burden			**CEQA Increment**		**Future CEQA Increment**		0.5	No
			0.0		0.02			

Notes:
[a]The Alternative 3 column represents the maximum health values prior to subtracting the CEQA baseline or Future CEQA Baseline.
[b]The CEQA Increment column represents the maximum difference of Alternative 3 minus the CEQA baseline.
[c]The Future CEQA Increment column represents the maximum difference of Alternative 3 minus the Future CEQA baseline.
[d]The maximum health values for Alternative 3, the CEQA Baseline, and the CEQA Increment may not all occur at the same receptor location. Therefore, the displayed values for Alternative 3 and the CEQA Baseline may not necessarily subtract to equal the CEQA Increment. The same is true for Alternative 3, the Future CEQA Baseline, and the Future CEQA Increment. The example given in the text under Impact AQ-7 for the proposed Project illustrates how the increments are calculated.
[e]A CEQA Increment less than zero means that the Alternative 3 health values would be less than the CEQA Baseline health values at all modeled receptors.
[f]The significance thresholds apply only to the CEQA increment and Future CEQA increment.
[g]Exceedances of the thresholds are indicated in **bold**. An impact is marked significant if either the CEQA Increment or Future CEQA Increment exceeds the threshold.
[h]The Future CEQA baseline and Future CEQA increment are applicable only to cancer risk because cancer risk has a uniquely long exposure period (30 years for residential and sensitive, and 25 years for occupational).
[i]Each positive 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.

Table B3-11 shows that unmitigated Alternative 3 would produce the following health risk impacts under CEQA:

- Individual Cancer Risk

In relation to the CEQA baseline, the maximum incremental cancer risk is predicted to be less than the significance threshold at all receptors. Moreover, the negative values for the CEQA increment indicate that the cancer risk from Alternative 3 would be less than the cancer risk from the CEQA baseline at all modeled residential, occupational, and sensitive receptors, due in large part to the beneficial effect of existing air quality rules and regulations on future emissions. In relation to the future CEQA baseline, the maximum incremental cancer risk is predicted to be less than the significance threshold at all receptors.

Figure B3-12 shows individual cancer risk contours of the future CEQA increment for unmitigated Alternative 3, assuming residential (30-year) exposure parameters. As shown in the figure, the maximum residential receptor for individual cancer risk is located outside the 10 in a million contour line, indicating a less than significant impact.

Cancer risk contours of the CEQA increment (as opposed to the future CEQA increment) are not shown because, as stated in the previous paragraph, the CEQA increment is predicted to be less than zero at all modeled residential receptors.

- Population Cancer Burden

In relation to the CEQA baseline, the cancer burden increment would be zero because the individual cancer risk associated with Alternative 3 would be less than the CEQA baseline at all modeled receptors. In relation to the Future CEQA baseline, the cancer burden increment is predicted to be less than the significance threshold.

- Chronic and Acute Hazard Indices

The maximum chronic and acute hazard index increments are predicted to be less than the significance threshold for all receptor types.

Because all CEQA health impacts are predicted to be less than the significance thresholds without mitigation, a CEQA evaluation of Alternative 3 with the mitigation measures prescribed in Section 3.1 was not necessary.

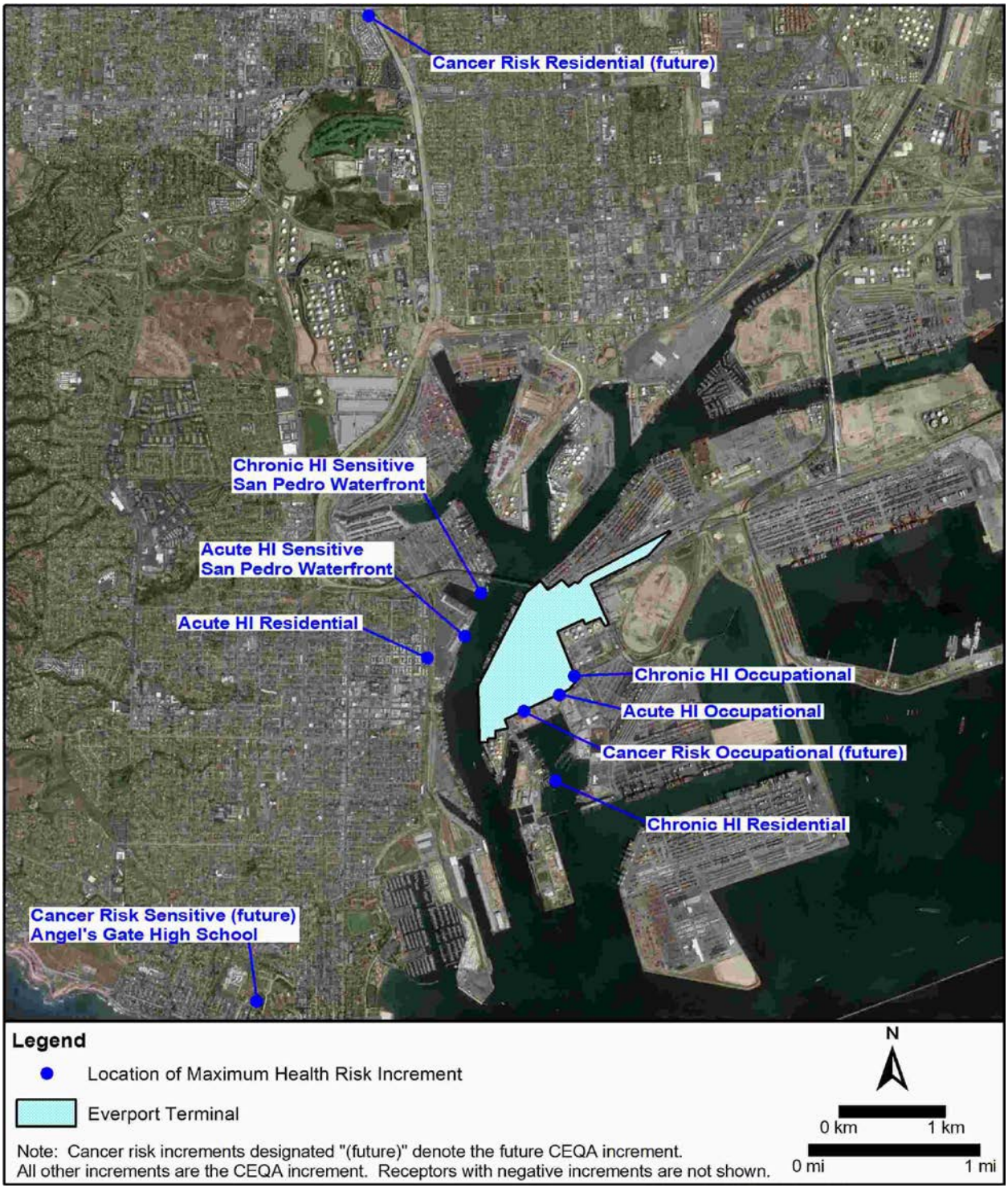

Figure B3-11: Locations of Maximum CEQA Health Impacts Estimated for Construction and Operation of Alternative 3 without Mitigation

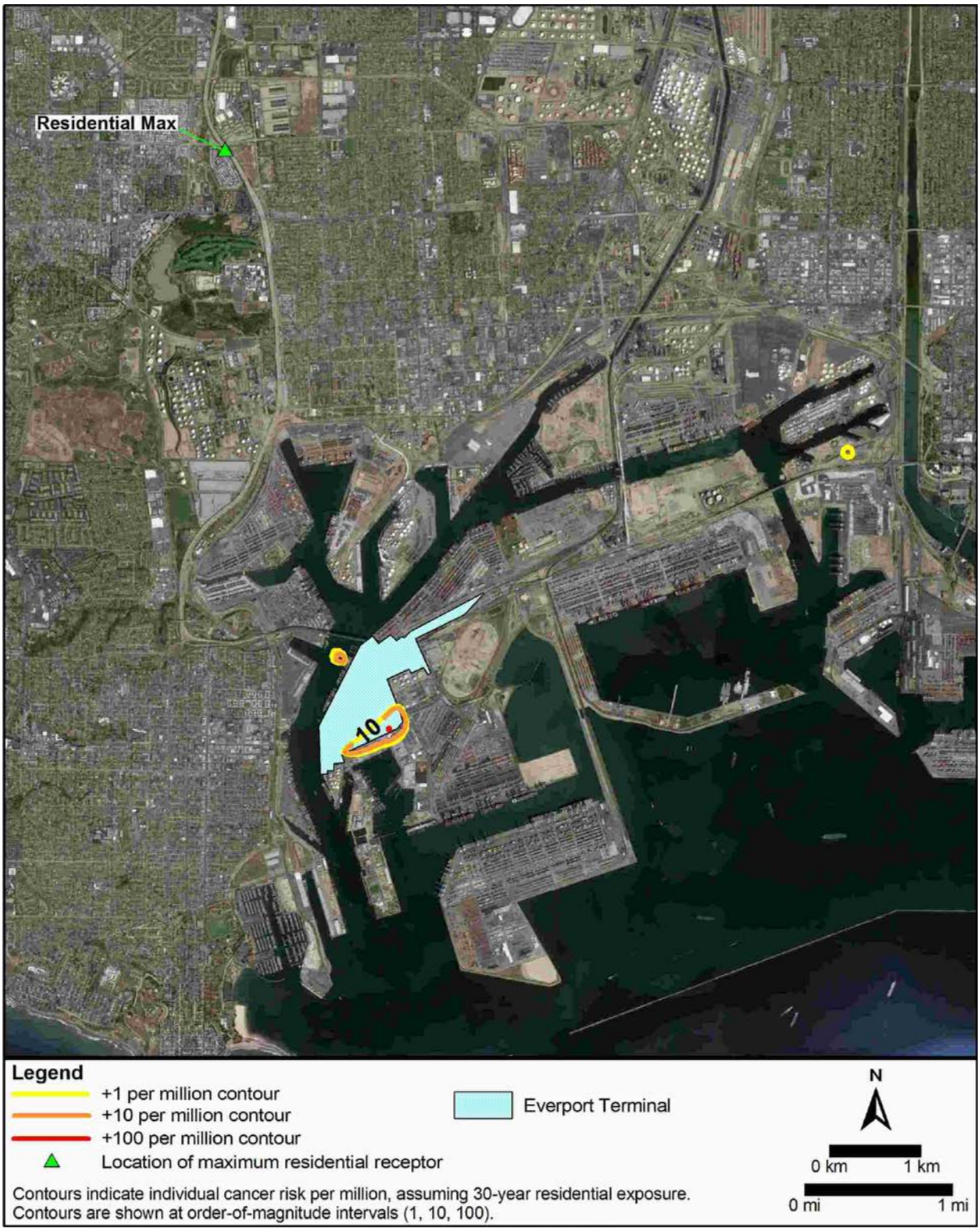

Figure B3-12: Isopleths of 30-Year Residential Cancer Risk – Alternative 3 without Mitigation – Future CEQA Increment

NEPA Impacts without Mitigation

Table B3-12 presents the maximum predicted NEPA health impacts associated with unmitigated Alternative 3. 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 Alternative 3 (before subtracting baseline), the NEPA baseline, and the NEPA increment (Alternative 3 minus NEPA baseline). The table also presents the NEPA increment for the population cancer burden.

Figure B3-13 shows the maximum NEPA increment receptor locations for individual cancer risk, chronic hazard index, and acute hazard index for unmitigated Alternative 3.

Table B3-12: Maximum NEPA Health Impacts Estimated for Construction and Operation of Alternative 3 Without Mitigation

Health Impact	Receptor Type	Alternative 3[a]	NEPA Baseline	NEPA Increment[b,c]	Significance Threshold[d]	Significant?[e]
Individual Cancer Risk	Residential	55.4 × 10-6 55.4 in a million	43.0 × 10-6 43.0 in a million	**12.3 × 10-6** **12.3 in a million**	10×10^{-6} 10 in a million	**Yes**
	Occupational	20.6 × 10-6 20.6 in a million	17.0 × 10-6 17.0 in a million	3.8 × 10-6 3.8 in a million		No
	Sensitive	42.0 × 10-6 42.0 in a million	34.0 × 10-6 34.0 in a million	7.9 × 10-6 7.9 in a million		No
Chronic Hazard Index	Residential	0.21	0.18	0.03	1.0	No
	Occupational	0.37	0.31	0.10		No
	Sensitive	0.35	0.30	0.06		No
Acute Hazard Index	Residential	0.09	0.07	0.04	1.0	No
	Occupational	0.25	0.26	0.10		No
	Sensitive	0.13	0.07	0.07		No
Population Cancer Burden				0.4	0.5	No

Notes:
[a]The Alternative 3 column represents the maximum health values prior to subtracting the NEPA baseline.
[b]The NEPA Increment column represents the maximum difference of Alternative 3 minus the NEPA baseline.
[c]The maximum health values for Alternative 3, the NEPA Baseline, and the NEPA Increment may not all occur at the same receptor location. Therefore, the displayed values for Alternative 3 and the NEPA Baseline may not necessarily subtract to equal the NEPA Increment. The example given in the text under Impact AQ-7 for the proposed Project illustrates how the increments are calculated.
[d]The significance thresholds apply only to the NEPA increment.
[e]Exceedances of the thresholds are indicated in **bold**.
[f]Each positive 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.

Table B3-12 shows that unmitigated Alternative 3 would produce the following health risk impacts under NEPA:

- Individual Cancer Risk

In relation to the NEPA baseline, the maximum incremental cancer risk is predicted to be greater than the significance threshold at the maximally impacted residential receptor. The cancer risk impact would be less than the threshold at occupational and sensitive receptors.

Figure B3-14 shows individual cancer risk contours of the NEPA increment for unmitigated Alternative 3, assuming residential (30-year) exposure parameters.

- Population Cancer Burden

In relation to the NEPA baseline, the cancer burden increment is predicted to be less than the significance threshold.

- Chronic and Acute Hazard Indices

The maximum chronic and acute hazard index increments are predicted to be less than the significance threshold for all receptor types.

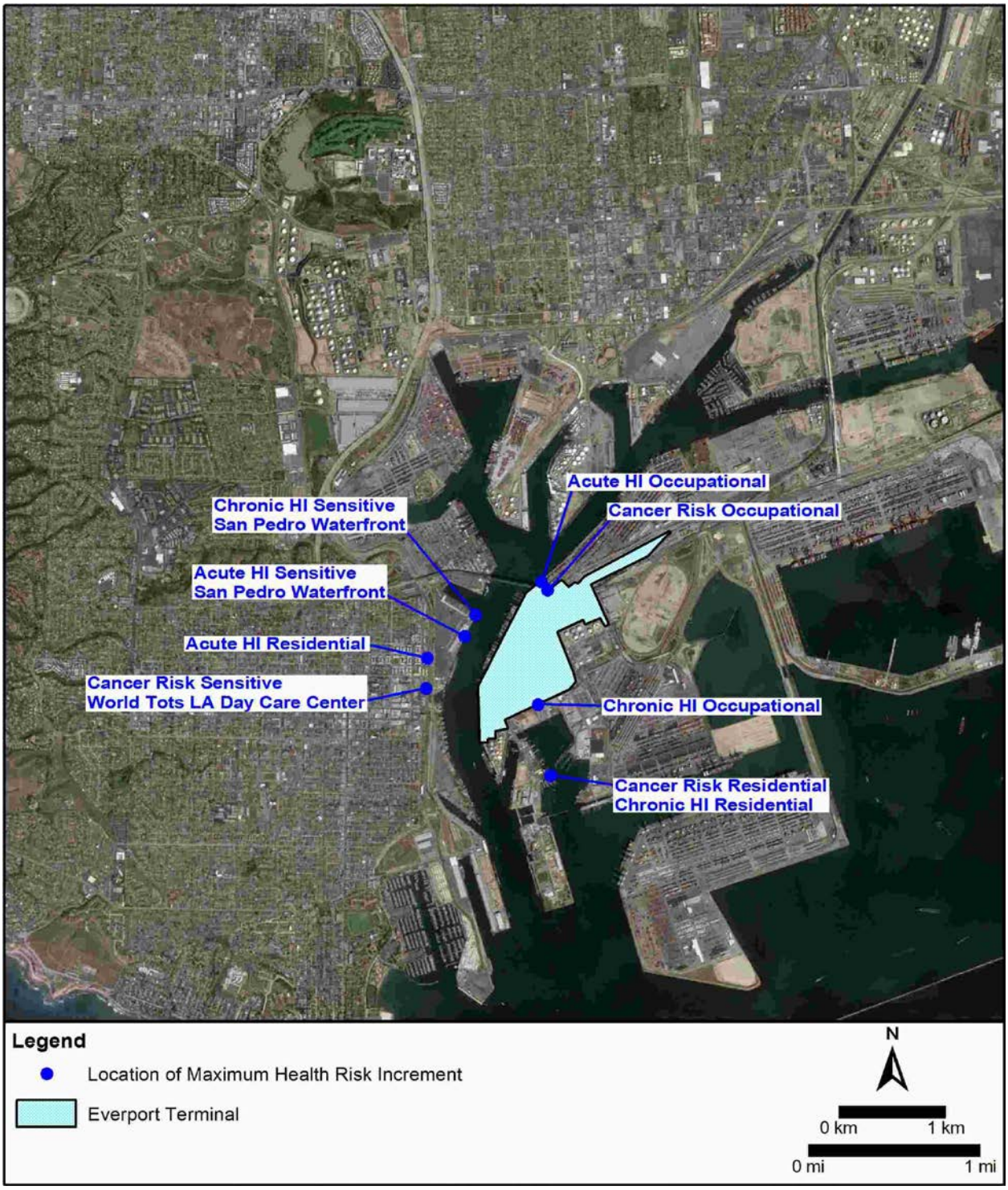

Figure B3-13: Locations of Maximum NEPA Health Impacts Estimated for Construction and Operation of Alternative 3 without Mitigation

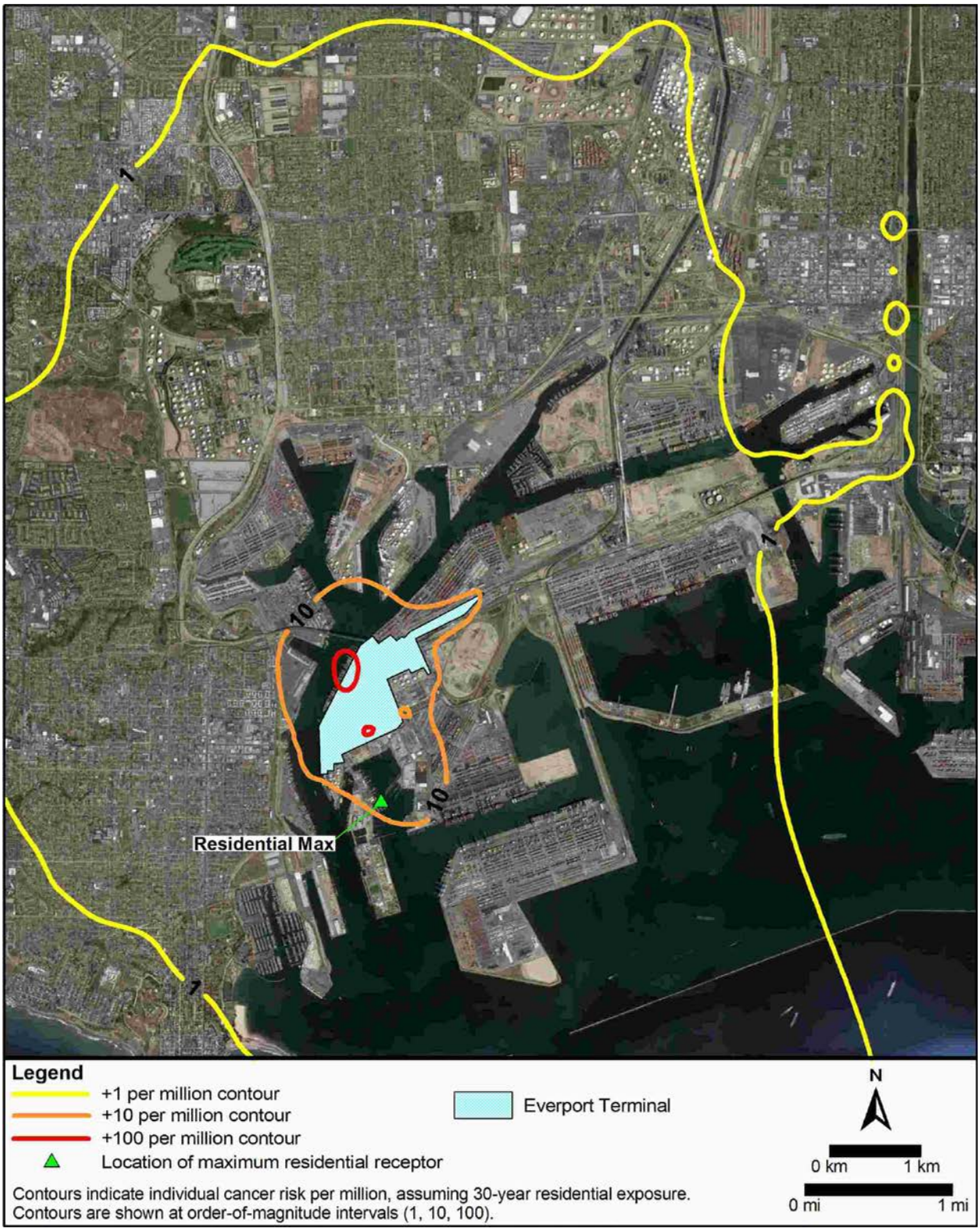

Figure B3-14: Isopleths of 30-Year Residential Cancer Risk – Alternative 3 without Mitigation – NEPA Increment

NEPA Impacts with Mitigation

To reduce the NEPA health risk impacts associated with Alternative 3, MM AQ-1 through AQ-5 would be applied during construction, and MM AQ-6 and MM AQ-7 would be applied during operation. The mitigation measures are described in Impacts AQ-1 and AQ-3 of Section 3.1.

Table B3-13 presents the maximum predicted NEPA health impacts associated with mitigated Alternative 3. The table shows that, with mitigation, the maximum incremental cancer risk at a residential receptor would be reduced to less than the significance threshold. All other health risk values would remain less than the thresholds.

Figure B3-15 shows the maximum NEPA increment receptor locations for individual cancer risk, chronic hazard index, and acute hazard index for mitigated Alternative 3.

Figure B3-16 shows individual cancer risk contours of the NEPA increment for mitigated Alternative 3, assuming residential (30-year) exposure parameters. As shown in the figure, the maximum residential receptor for individual cancer risk is located outside the 10 in a million contour line, indicating a less than significant impact.

Table B3-13: Maximum NEPA Health Impacts Estimated for Construction and Operation of Alternative 3 With Mitigation

Health Impact	Receptor Type	Alternative 3[a]	NEPA Baseline	NEPA Increment[b,c]	Significance Threshold[d]	Significant?[e]
Individual Cancer Risk	Residential	47.6 × 10-6 47.6 in a million	43.0 × 10-6 43.0 in a million	4.6 × 10-6 4.6 in a million	10×10^{-6} 10 in a million	No
	Occupational	20.4 × 10-6 20.4 in a million	17.0 × 10-6 17.0 in a million	3.4 × 10-6 3.4 in a million		No
	Sensitive	37.7 × 10-6 37.7 in a million	34.0 × 10-6 34.0 in a million	3.7 × 10-6 3.7 in a million		No
Chronic Hazard Index	Residential	0.20	0.18	0.02	1.0	No
	Occupational	0.36	0.31	0.05		No
	Sensitive	0.34	0.30	0.05		No
Acute Hazard Index	Residential	0.09	0.07	0.04	1.0	No
	Occupational	0.26	0.26	0.10		No
	Sensitive	0.12	0.07	0.06		No
Population Cancer Burden				0.1	0.5	No

Notes:
[a]The Alternative 3 column represents the maximum health values prior to subtracting the NEPA baseline.
[b]The NEPA Increment column represents the maximum difference of Alternative 3 minus the NEPA baseline.
[c]The maximum health values for Alternative 3, the NEPA Baseline, and the NEPA Increment may not all occur at the same receptor location. Therefore, the displayed values for Alternative 3 and the NEPA Baseline may not necessarily subtract to equal the NEPA Increment. The example given in the text under Impact AQ-7 for the proposed Project illustrates how the increments are calculated.
[d]The significance thresholds apply only to the NEPA increment.
[e]Exceedances of the thresholds are indicated in **bold**.
[f]Each positive 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.

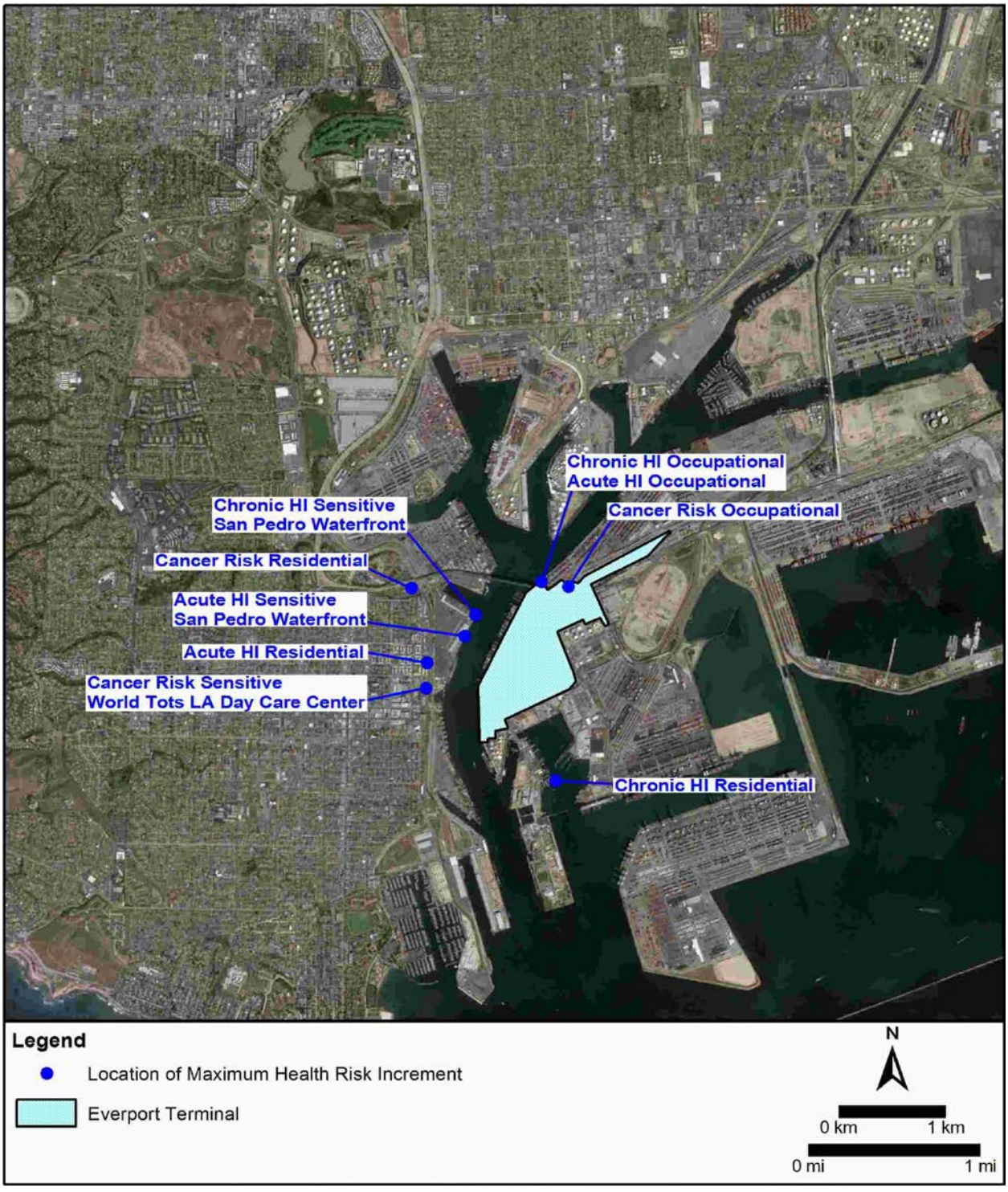

Figure B3-15: Locations of Maximum NEPA Health Impacts Estimated for Construction and Operation of Alternative 3 with Mitigation

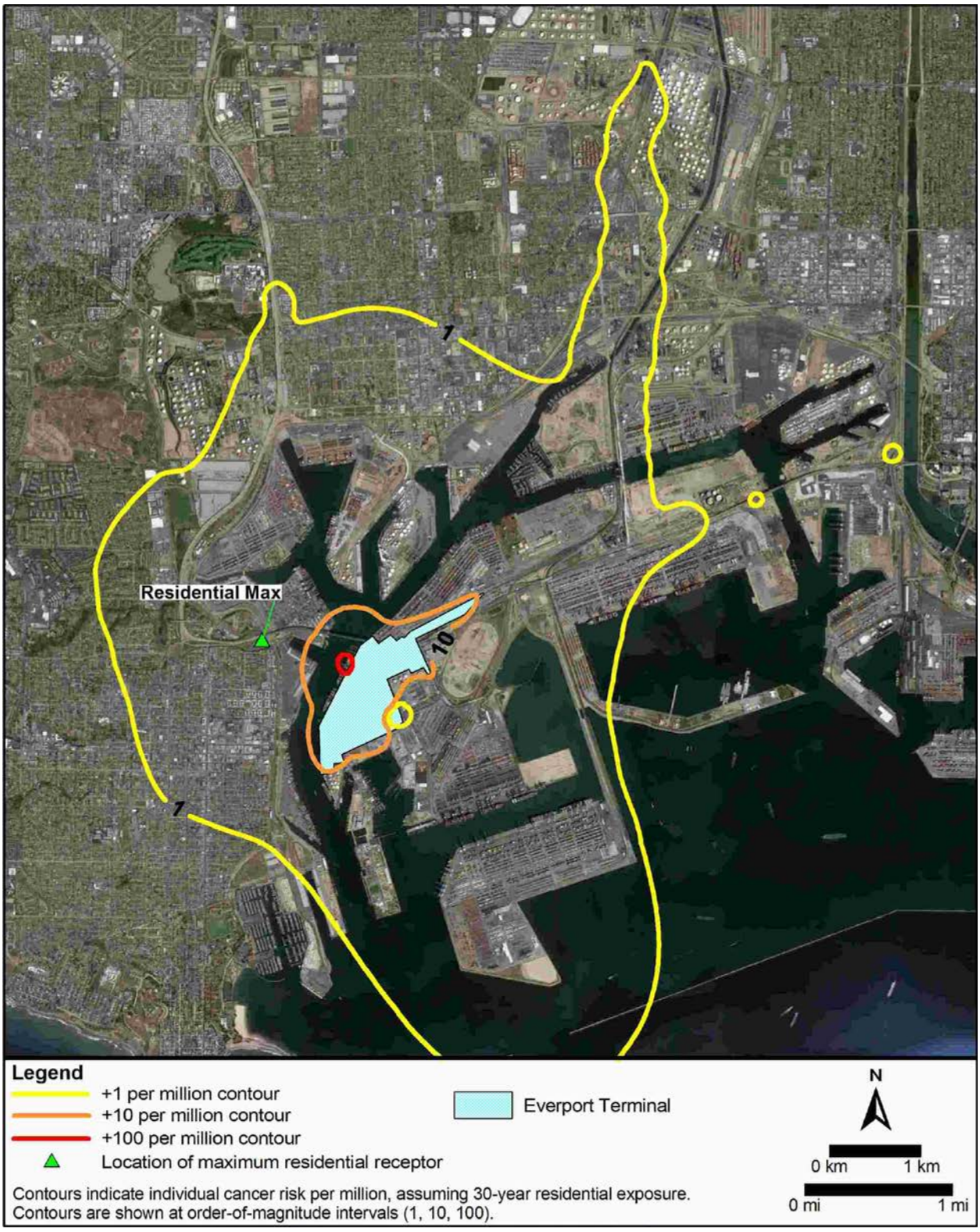

Figure B3-16: Isopleths of 30-Year Residential Cancer Risk – Alternative 3 with Mitigation – NEPA Increment

6.5 Alternative 4 (Reduced Project: No Backland Improvements)

CEQA Impacts without Mitigation

Table B3-14 presents the maximum predicted CEQA health impacts associated with unmitigated Alternative 4. 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 Alternative 4 (before subtracting baseline), the two CEQA baselines, the CEQA increment (Alternative 4 minus CEQA baseline), and the future CEQA increment (Alternative 4 minus future CEQA baseline). The table also presents the CEQA increment and future CEQA increment for the population cancer burden.

Figure B3-17 shows the maximum CEQA increment and future CEQA increment receptor locations for individual cancer risk, chronic hazard index, and acute hazard index for unmitigated Alternative 4.

Table B3-14: Maximum CEQA Health Impacts Estimated for Construction and Operation of Alternative 4 Without Mitigation

Health Impact	Receptor Type	Alternative 4[a]	CEQA Baseline	CEQA Increment[b,d,e]	Future CEQA Baseline	Future CEQA Increment[c,d,e]	Significance Threshold[f]	Significant?[g]
Individual Cancer Risk	Residential	49.0 × 10-6 49.0 in a million	104.0 × 10-6 104.0 in a million	< 0	64.7 × 10-6 64.7 in a million	0.04 × 10-6 0.04 in a million	10×10^{-6} 10 in a million	No
	Occupational	21.0 × 10-6 21.0 in a million	66.0 × 10-6 66.0 in a million	< 0	22.0 × 10-6 22.0 in a million	1.9 × 10-6 1.9 in a million		No
	Sensitive	40.7 × 10-6 40.7 in a million	94.1 × 10-6 94.1 in a million	< 0	57.6 × 10-6 57.6 in a million	0.0007 × 10-6 0.0007 in a million		No
Chronic Hazard Index	Residential	0.21	0.16	0.05	n/a[h]	n/a[h]	1.0	No
	Occupational	0.38	0.38	0.09	n/a	n/a		No
	Sensitive	0.37	0.28	0.09	n/a	n/a		No
Acute Hazard Index	Residential	0.11	0.06	0.06	n/a	n/a	1.0	No
	Occupational	0.19	0.16	0.10	n/a	n/a		No
	Sensitive	0.16	0.07	0.10	n/a	n/a		No
Population Cancer Burden			**CEQA Increment**		**Future CEQA Increment**		0.5	No
			0.0		0.0			

Notes:
[a]The Alternative 4 column represents the maximum health values prior to subtracting the CEQA baseline or Future CEQA Baseline.
[b]The CEQA Increment column represents the maximum difference of Alternative 4 minus the CEQA baseline.
[c]The Future CEQA Increment column represents the maximum difference of Alternative 4 minus the Future CEQA baseline.
[d]The maximum health values for Alternative 4, the CEQA Baseline, and the CEQA Increment may not all occur at the same receptor location. Therefore, the displayed values for Alternative 4 and the CEQA Baseline may not necessarily subtract to equal the CEQA Increment. The same is true for Alternative 4, the Future CEQA Baseline, and the Future CEQA Increment. The example given in the text under Impact AQ-7 for the proposed Project illustrates how the increments are calculated.
[e]A CEQA Increment less than zero means that the Alternative 4 health values would be less than the CEQA Baseline health values at all modeled receptors.
[f]The significance thresholds apply only to the CEQA increment and Future CEQA increment.
[g]Exceedances of the thresholds are indicated in **bold**. An impact is marked significant if either the CEQA Increment or Future CEQA Increment exceeds the threshold.
[h]The Future CEQA baseline and Future CEQA increment are applicable only to cancer risk because cancer risk has a uniquely long exposure period (30 years for residential and sensitive, and 25 years for occupational).
[i]Each positive 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.

Table B3-14 shows that unmitigated Alternative 4 would produce the following health risk impacts under CEQA:

- Individual Cancer Risk

In relation to the CEQA baseline, the maximum incremental cancer risk is predicted to be less than the significance threshold at all receptors. Moreover, the negative values for the CEQA increment indicate that the cancer risk from Alternative 4 would be less than the cancer risk from the CEQA baseline at all modeled residential, occupational, and sensitive receptors, due in large part to the beneficial effect of existing air quality rules and regulations on future emissions. In relation to the future CEQA baseline, the maximum incremental cancer risk is predicted to be less than the significance threshold at all receptors.

Figure B3-18 shows individual cancer risk contours of the future CEQA increment for unmitigated Alternative 4, assuming residential (30-year) exposure parameters. As shown in the figure, the maximum residential receptor for individual cancer risk is located outside the 10 in a million contour line, indicating a less than significant impact.

Residential cancer risk contours of the CEQA increment (as opposed to the future CEQA increment) are not shown because, as stated in the previous paragraph, the CEQA increment is predicted to be less than zero at all modeled residential receptors.

- Population Cancer Burden

In relation to the CEQA baseline, the cancer burden increment would be zero because the individual cancer risk associated with Alternative 4 would be less than the CEQA baseline at all modeled receptors. In relation to the Future CEQA baseline, the cancer burden increment is predicted to be less than the significance threshold.

- Chronic and Acute Hazard Indices

The maximum chronic and acute hazard index increments are predicted to be less than the significance threshold for all receptor types.

Because all CEQA health impacts are predicted to be less than the significance thresholds without mitigation, a CEQA evaluation of Alternative 4 with the mitigation measures prescribed in Section 3.1 was not necessary.

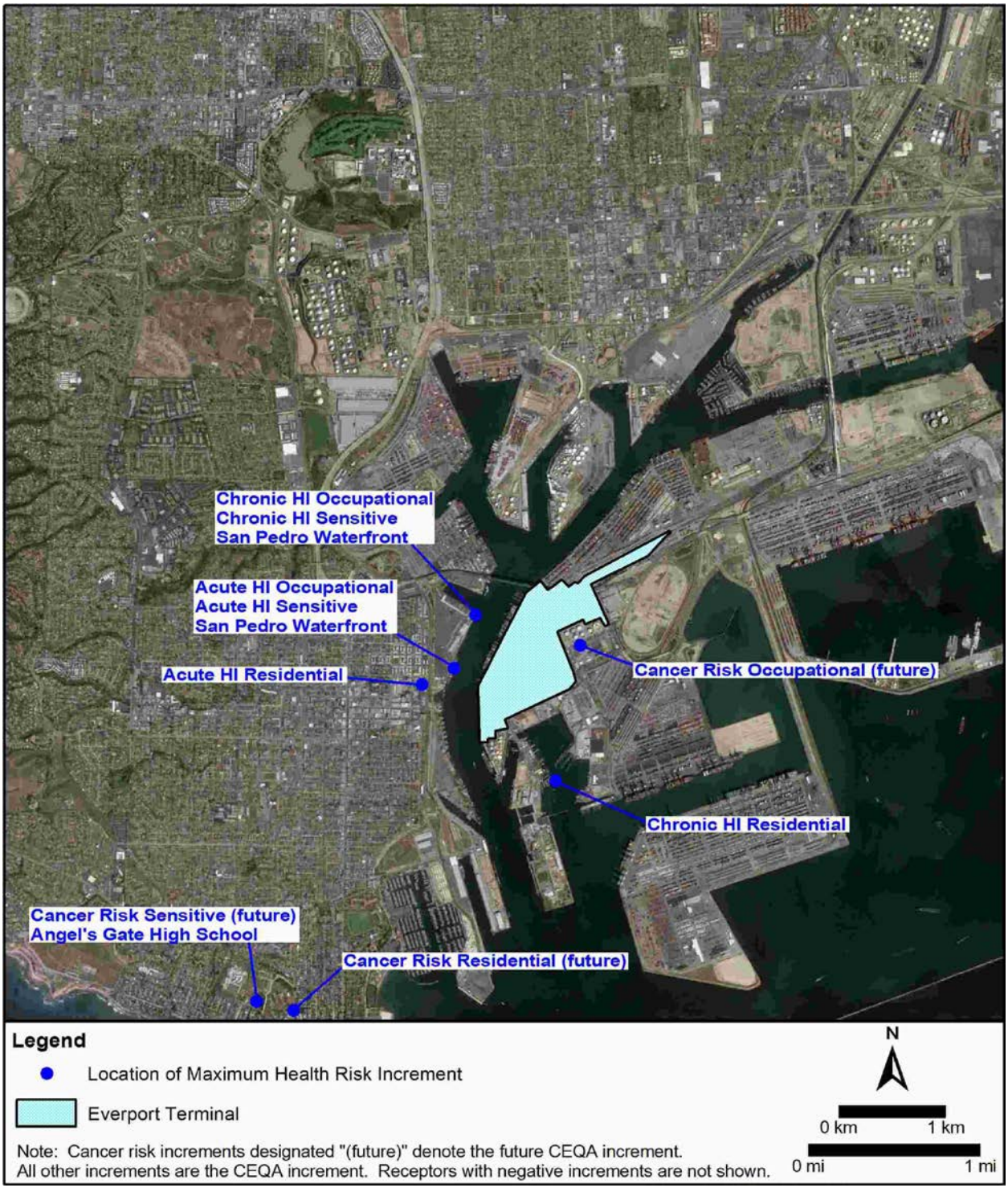

Figure B3-17: Locations of Maximum CEQA Health Impacts Estimated for Construction and Operation of Alternative 4 without Mitigation

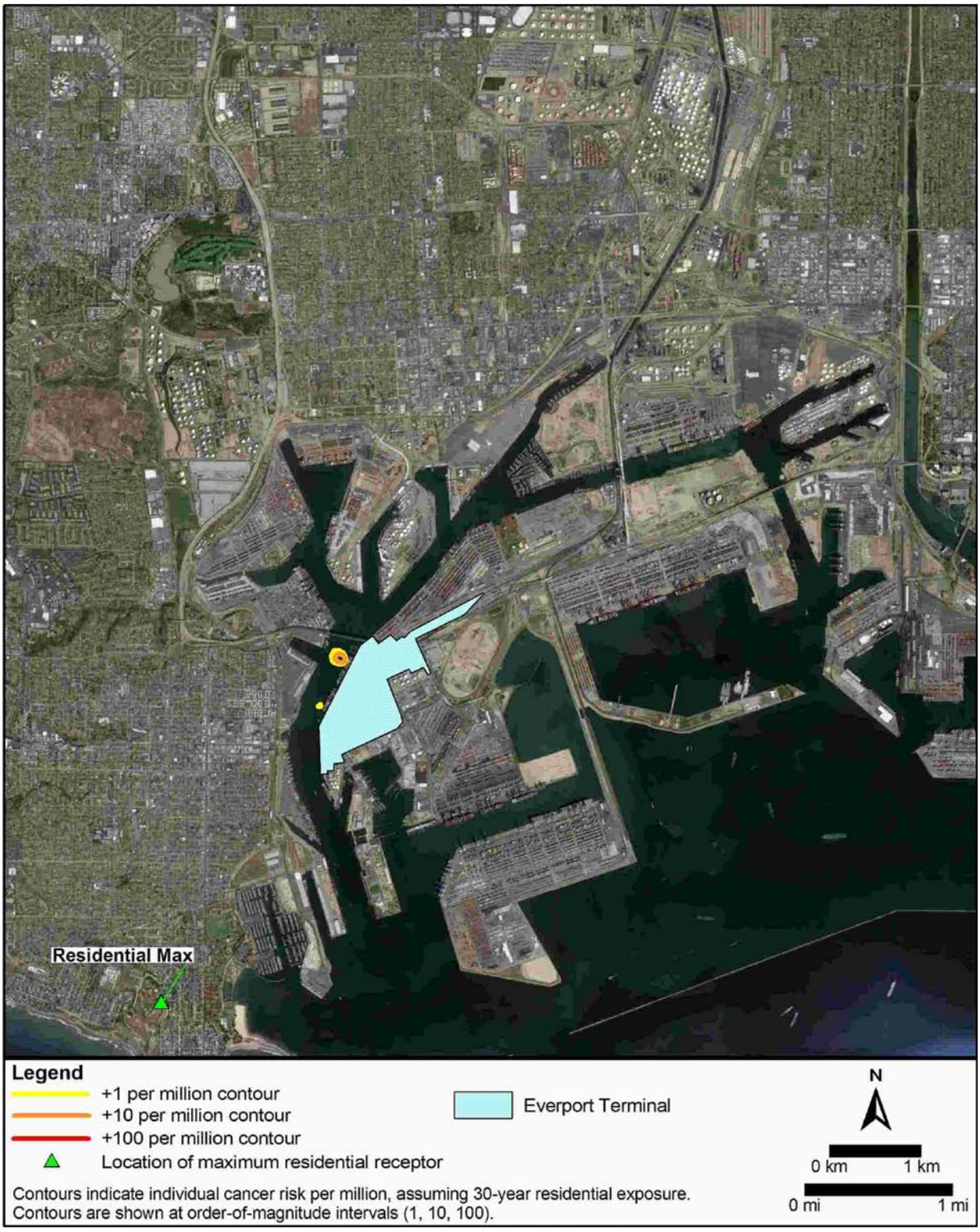

Figure B3-18: Isopleths of 30-Year Residential Cancer Risk – Alternative 4 without Mitigation – Future CEQA Increment

NEPA Impacts without Mitigation

Table B3-15 presents the maximum predicted NEPA health impacts associated with unmitigated Alternative 4. 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 Alternative 4 (before subtracting baseline), the NEPA baseline, and the NEPA increment (Alternative 4 minus NEPA baseline). The table also presents the NEPA increment for the population cancer burden.

Figure B3-19 shows the maximum NEPA increment receptor locations for individual cancer risk, chronic hazard index, and acute hazard index for unmitigated Alternative 4.

Table B3-15: Maximum NEPA Health Impacts Estimated for Construction and Operation of Alternative 4 Without Mitigation

Health Impact	Receptor Type	Alternative 4[a]	NEPA Baseline	NEPA Increment[b,c]	Significance Threshold[d]	Significant?[e]
Individual Cancer Risk	Residential	49.0 × 10-6 49.0 in a million	43.0 × 10-6 43.0 in a million	9.2 × 10-6 9.2 in a million	10×10^{-6} 10 in a million	No
	Occupational	21.0 × 10-6 21.0 in a million	17.0 × 10-6 17.0 in a million	4.8 × 10-6 4.8 in a million		No
	Sensitive	40.7 × 10-6 40.7 in a million	34.0 × 10-6 34.0 in a million	6.6 × 10-6 6.6 in a million		No
Chronic Hazard Index	Residential	0.21	0.18	0.03	1.0	No
	Occupational	0.38	0.31	0.08		No
	Sensitive	0.37	0.30	0.08		No
Acute Hazard Index	Residential	0.11	0.07	0.05	1.0	No
	Occupational	0.19	0.26	0.09		No
	Sensitive	0.16	0.07	0.09		No
Population Cancer Burden				0.2	0.5	No

Notes:
[a]The Alternative 4 column represents the maximum health values prior to subtracting the NEPA baseline.
[b]The NEPA Increment column represents the maximum difference of Alternative 4 minus the NEPA baseline.
[c]The maximum health values for Alternative 4, the NEPA Baseline, and the NEPA Increment may not all occur at the same receptor location. Therefore, the displayed values for Alternative 4 and the NEPA Baseline may not necessarily subtract to equal the NEPA Increment. The example given in the text under Impact AQ-7 for the proposed Project illustrates how the increments are calculated.
[d]The significance thresholds apply only to the NEPA increment.
[e]Exceedances of the thresholds are indicated in **bold**.
[f]Each positive 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.

Table B3-15 shows that unmitigated Alternative 4 would produce the following health risk impacts under NEPA:

- Individual Cancer Risk

In relation to the NEPA baseline, the maximum incremental cancer risk is predicted to be less than the significance threshold at all receptors.

Figure B3-20 shows individual cancer risk contours of the NEPA increment for unmitigated Alternative 4, assuming residential (30-year) exposure parameters. As shown in the figure, the maximum residential receptor for individual cancer risk is located outside the 10 in a million contour line, indicating a less than significant impact.

- Population Cancer Burden

In relation to the NEPA baseline, the cancer burden increment is predicted to be less than the significance threshold.

- Chronic and Acute Hazard Indices

The maximum chronic and acute hazard index increments are predicted to be less than the significance threshold for all receptor types.

Because all NEPA health impacts are predicted to be less than the significance thresholds without mitigation, a NEPA evaluation of Alternative 4 with the mitigation measures prescribed in Section 3.1 was not necessary.

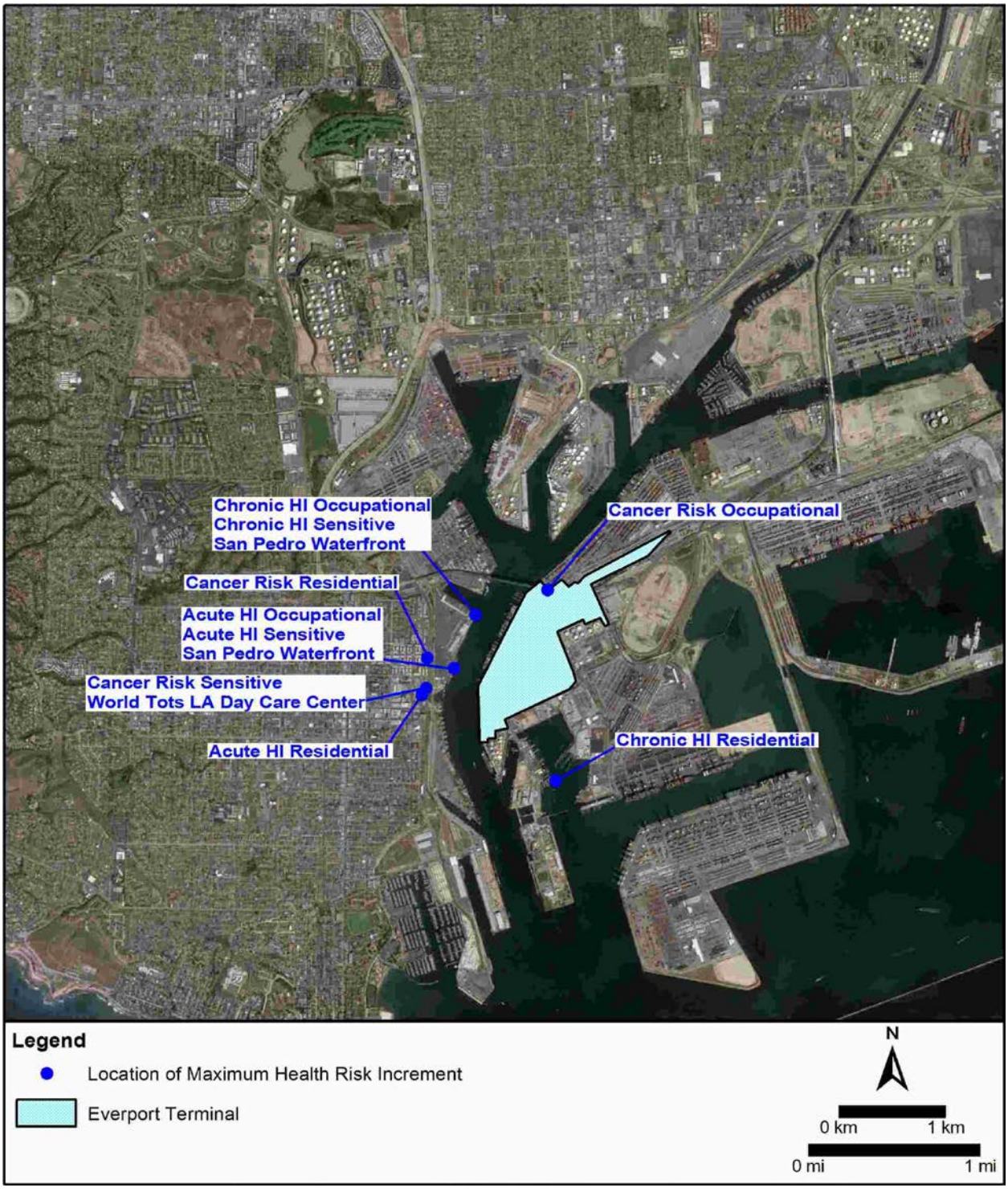

Figure B3-19: Locations of Maximum NEPA Health Impacts Estimated for Construction and Operation of Alternative 4 without Mitigation

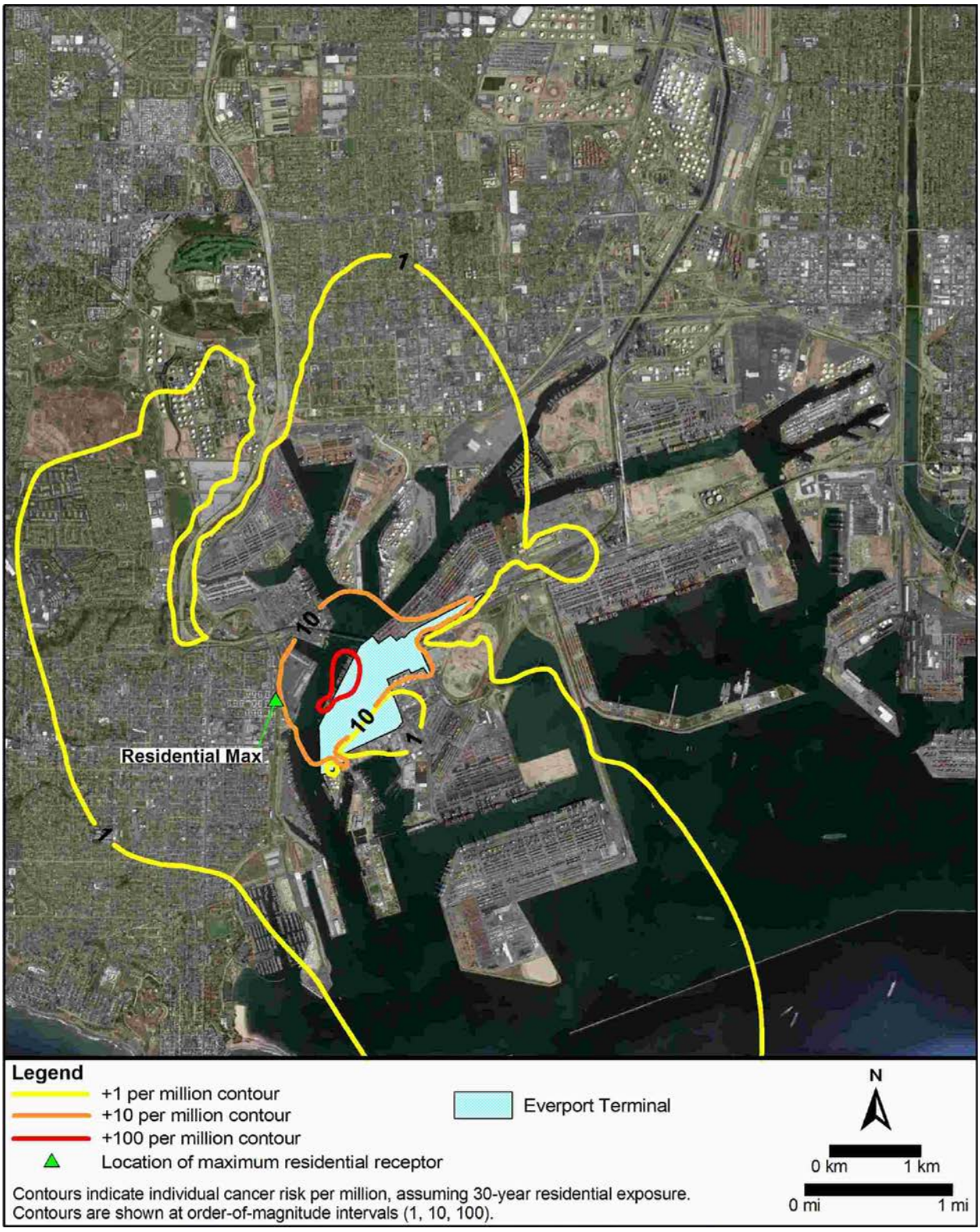

Figure B3-20: Isopleths of 30-Year Residential Cancer Risk – Alternative 4 without Mitigation – NEPA Increment

6.6 Alternative 5 (Expanded On-Dock Railyard: Wharf and Backland Improvements with an Expanded TICTF)

CEQA Impacts without Mitigation

Table B3-16 presents the maximum predicted CEQA health impacts associated with unmitigated Alternative 5. 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 Alternative 5 (before subtracting baseline), the two CEQA baselines, the CEQA increment (Alternative 5 minus CEQA baseline), and the future CEQA increment (Alternative 5 minus future CEQA baseline). The table also presents the CEQA increment and future CEQA increment for the population cancer burden.

Figure B3-21 shows the maximum CEQA increment and future CEQA increment receptor locations for individual cancer risk, chronic hazard index, and acute hazard index for unmitigated Alternative 5.

Table B3-16: Maximum CEQA Health Impacts Estimated for Construction and Operation of Alternative 5 Without Mitigation

Health Impact	Receptor Type	Alternative 5[a]	CEQA Baseline	CEQA Increment[b,d,e]	Future CEQA Baseline	Future CEQA Increment[c,d]	Significance Threshold[f]	Significant?[g]
Individual Cancer Risk	Residential	59.3 × 10-6 59.3 in a million	104.0 × 10-6 104.0 in a million	< 0	64.7 × 10-6 64.7 in a million	1.5 × 10-6 1.5 in a million	10×10^{-6} 10 in a million	No
	Occupational	22.0 × 10-6 22.0 in a million	66.0 × 10-6 66.0 in a million	< 0	22.0 × 10-6 22.0 in a million	5.8 × 10-6 5.8 in a million		No
	Sensitive	46.0 × 10-6 46.0 in a million	94.1 × 10-6 94.1 in a million	< 0	57.6 × 10-6 57.6 in a million	0.9 × 10-6 0.9 in a million		No
Chronic Hazard Index	Residential	0.23	0.16	0.07	n/a[h]	n/a[h]	1.0	No
	Occupational	0.41	0.38	0.16	n/a	n/a		No
	Sensitive	0.40	0.28	0.12	n/a	n/a		No
Acute Hazard Index	Residential	0.11	0.06	0.07	n/a	n/a	1.0	No
	Occupational	0.27	0.16	0.20	n/a	n/a		No
	Sensitive	0.16	0.07	0.10	n/a	n/a		No
Population Cancer Burden			**CEQA Increment**		**Future CEQA Increment**		0.5	No
			0.0		0.1			

Notes:
[a]The Alternative 5 column represents the maximum health values prior to subtracting the CEQA baseline or Future CEQA Baseline.
[b]The CEQA Increment column represents the maximum difference of Alternative 5 minus the CEQA baseline.
[c]The Future CEQA Increment column represents the maximum difference of Alternative 5 minus the Future CEQA baseline.
[d]The maximum health values for Alternative 5, the CEQA Baseline, and the CEQA Increment may not all occur at the same receptor location. Therefore, the displayed values for Alternative 5 and the CEQA Baseline may not necessarily subtract to equal the CEQA Increment. The same is true for Alternative 5, the Future CEQA Baseline, and the Future CEQA Increment. The example given in the text under Impact AQ-7 for the proposed Project illustrates how the increments are calculated.
[e]A CEQA Increment less than zero means that the Alternative 5 health values would be less than the CEQA Baseline health values at all modeled receptors.
[f]The significance thresholds apply only to the CEQA increment and Future CEQA increment.
[g]Exceedances of the thresholds are indicated in **bold**. An impact is marked significant if either the CEQA Increment or Future CEQA Increment exceeds the threshold.
[h]The Future CEQA baseline and Future CEQA increment are applicable only to cancer risk because cancer risk has a uniquely long exposure period (30 years for residential and sensitive, and 25 years for occupational).
[i]Each positive 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.

Table B3-16 shows that unmitigated Alternative 5 would produce the following health risk impacts under CEQA:

- Individual Cancer Risk

In relation to the CEQA baseline, the maximum incremental cancer risk is predicted to be less than the significance threshold at all receptors. Moreover, the negative values for the CEQA increment indicate that the cancer risk from Alternative 5 would be less than the cancer risk from the CEQA baseline at all modeled residential, occupational, and sensitive receptors, due in large part to the beneficial effect of existing air quality rules and regulations on future emissions. In relation to the future CEQA baseline, the maximum incremental cancer risk is predicted to be less than the significance threshold at all receptors.

Figure B3-22 shows individual cancer risk contours of the future CEQA increment for unmitigated Alternative 5, assuming residential (30-year) exposure parameters. As shown in the figure, the maximum residential receptor for individual cancer risk is located outside the 10 in a million contour line, indicating a less than significant impact.

Residential cancer risk contours of the CEQA increment (as opposed to the future CEQA increment) are not shown because, as stated in the previous paragraph, the CEQA increment is predicted to be less than zero at all modeled residential receptors.

- Population Cancer Burden

In relation to the CEQA baseline, the cancer burden increment would be zero because the individual cancer risk associated with Alternative 5 would be less than the CEQA baseline at all modeled receptors. In relation to the Future CEQA baseline, the cancer burden increment is predicted to be less than the significance threshold.

- Chronic and Acute Hazard Indices

The maximum chronic and acute hazard index increments are predicted to be less than the significance threshold for all receptor types.

Because all CEQA health impacts are predicted to be less than the significance thresholds without mitigation, a CEQA evaluation of Alternative 5 with the mitigation measures prescribed in Section 3.1 was not necessary.

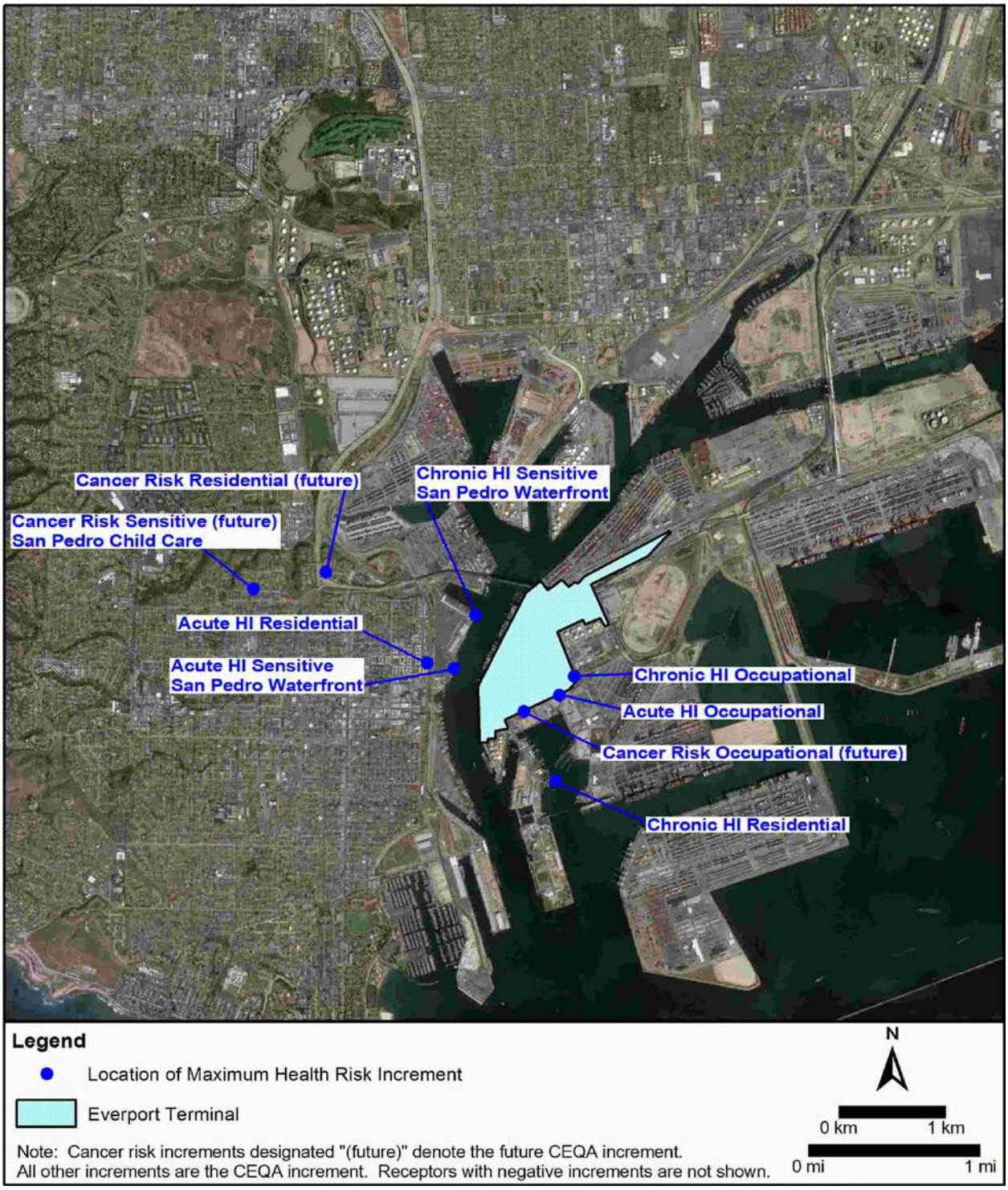

Figure B3-21: Locations of Maximum CEQA Health Impacts Estimated for Construction and Operation of Alternative 5 without Mitigation

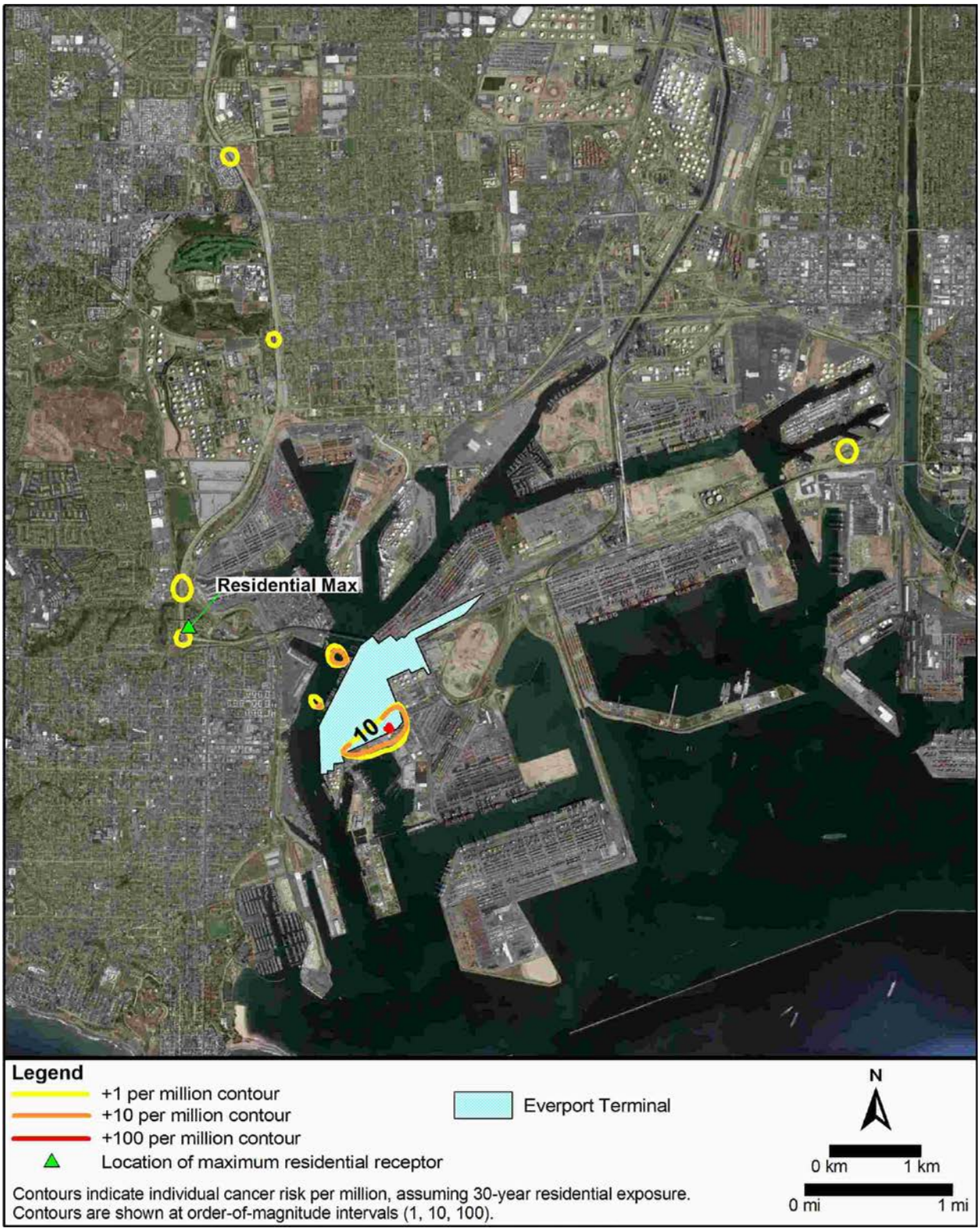

Figure B3-22: Isopleths of 30-Year Residential Cancer Risk – Alternative 5 without Mitigation – Future CEQA Increment

NEPA Impacts without Mitigation

Table B3-17 presents the maximum predicted NEPA health impacts associated with unmitigated Alternative 5. 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 Alternative 5 (before subtracting baseline), the NEPA baseline, and the NEPA increment (Alternative 5 minus NEPA baseline). The table also presents the NEPA increment for the population cancer burden.

Figure B3-23 shows the maximum NEPA increment receptor locations for individual cancer risk, chronic hazard index, and acute hazard index for unmitigated Alternative 5.

Table B3-17: Maximum NEPA Health Impacts Estimated for Construction and Operation of Alternative 5 Without Mitigation

Health Impact	Receptor Type	Alternative 5[a]	NEPA Baseline	NEPA Increment[b,c]	Significance Threshold[d]	Significant?[e]
Individual Cancer Risk	Residential	59.3 × 10-6 59.3 in a million	43.0 × 10-6 43.0 in a million	**16.3 × 10-6** **16.3 in a million**	10×10^{-6} 10 in a million	**Yes**
	Occupational	22.0 × 10-6 22.0 in a million	17.0 × 10-6 17.0 in a million	5.0 × 10-6 5.0 in a million		No
	Sensitive	46.0 × 10-6 46.0 in a million	34.0 × 10-6 34.0 in a million	**12.0 × 10-6** **12.0 in a million**		**Yes**
Chronic Hazard Index	Residential	0.23	0.18	0.05	1.0	No
	Occupational	0.41	0.31	0.13		No
	Sensitive	0.40	0.30	0.11		No
Acute Hazard Index	Residential	0.11	0.07	0.06	1.0	No
	Occupational	0.27	0.26	0.10		No
	Sensitive	0.16	0.07	0.10		No
Population Cancer Burden				**0.7**	0.5	**Yes**

Notes:
[a]The Alternative 5 column represents the maximum health values prior to subtracting the NEPA baseline.
[b]The NEPA Increment column represents the maximum difference of Alternative 5 minus the NEPA baseline.
[c]The maximum health values for Alternative 5, the NEPA Baseline, and the NEPA Increment may not all occur at the same receptor location. Therefore, the displayed values for Alternative 5 and the NEPA Baseline may not necessarily subtract to equal the NEPA Increment. The example given in the text under Impact AQ-7 for the proposed Project illustrates how the increments are calculated.
[d]The significance thresholds apply only to the NEPA increment.
[e]Exceedances of the thresholds are indicated in **bold**.
[f]Each positive 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.

Table B3-17 shows that unmitigated Alternative 5 would produce the following health risk impacts under NEPA:

- Individual Cancer Risk

In relation to the NEPA baseline, the maximum incremental cancer risk is predicted to be greater than the significance threshold at the maximally impacted residential and sensitive receptors. The cancer risk impact would be less than the threshold at occupational receptors.

Figure B3-24 shows individual cancer risk contours of the NEPA increment for unmitigated Alternative 5, assuming residential (30-year) exposure parameters.

- Population Cancer Burden

In relation to the NEPA baseline, the cancer burden increment is predicted to be greater than the significance threshold.

- Chronic and Acute Hazard Indices

The maximum chronic and acute hazard index increments are predicted to be less than the significance threshold for all receptor types.

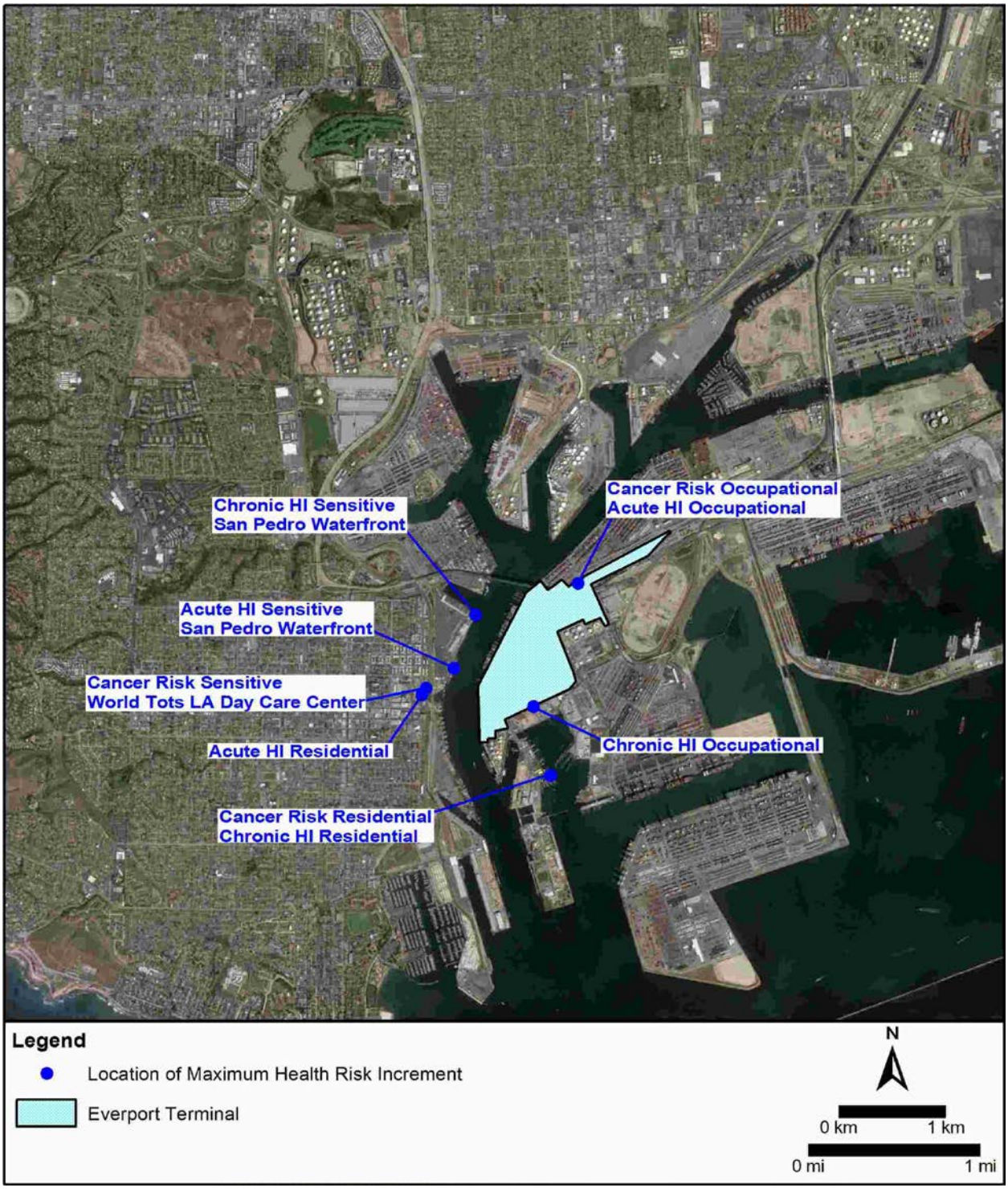

Figure B3-23: Locations of Maximum NEPA Health Impacts Estimated for Construction and Operation of Alternative 5 without Mitigation

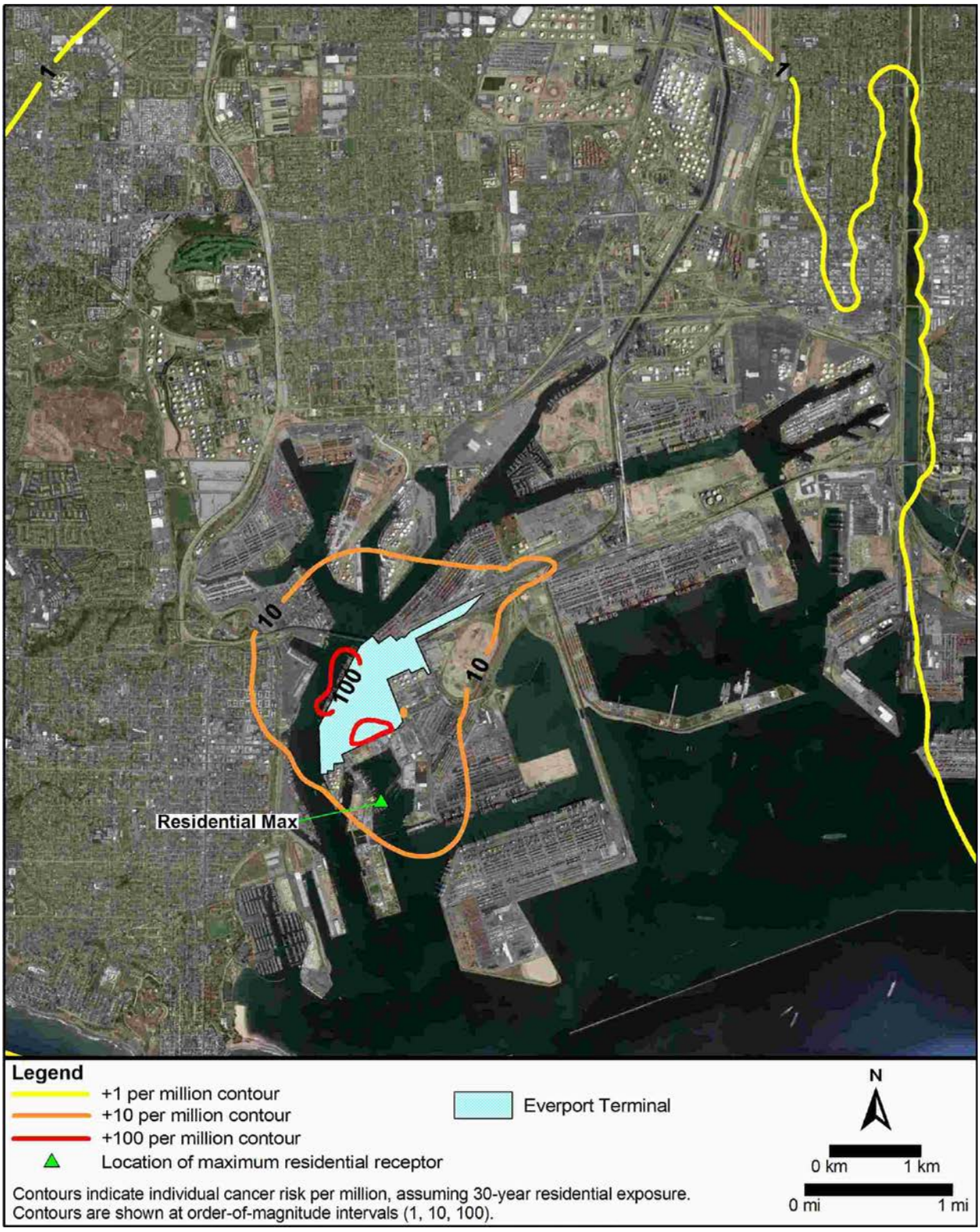

Figure B3-24: Isopleths of 30-Year Residential Cancer Risk – Alternative 5 without Mitigation – NEPA Increment

NEPA Impacts with Mitigation

To reduce the NEPA health risk impacts associated with Alternative 5, MM AQ-1 through AQ-5 would be applied during construction, and MM AQ-6 and MM AQ-7 would be applied during operation. The mitigation measures are described in Impacts AQ-1 and AQ-3 of Section 3.1.

Table B3-18 presents the maximum predicted NEPA health impacts associated with mitigated Alternative 5. The table shows that, with mitigation, the maximum incremental cancer risk at residential and sensitive receptors would be reduced to less than the significance thresholds. The population cancer burden would also be reduced to less than the threshold. All other health risk values would remain less than the thresholds.

Figure B3-25 shows the maximum NEPA increment receptor locations for individual cancer risk, chronic hazard index, and acute hazard index for mitigated Alternative 5.

Figure B3-26 shows individual cancer risk contours of the NEPA increment for mitigated Alternative 5, assuming residential (30-year) exposure parameters. As shown in the figure, the maximum residential receptor for individual cancer risk is located outside the 10 in a million contour line, indicating a less than significant impact.

Table B3-18: Maximum NEPA Health Impacts Estimated for Construction and Operation of Alternative 5 With Mitigation

Health Impact	Receptor Type	Alternative 5[a]	NEPA Baseline	NEPA Increment[b,c]	Significance Threshold[d]	Significant?[e]
Individual Cancer Risk	Residential	50.9 × 10-6 50.9 in a million	43.0 × 10-6 43.0 in a million	9.1 × 10-6 9.1 in a million	10×10^{-6} 10 in a million	No
	Occupational	21.4 × 10-6 21.4 in a million	17.0 × 10-6 17.0 in a million	4.3 × 10-6 4.3 in a million		No
	Sensitive	41.0 × 10-6 41.0 in a million	34.0 × 10-6 34.0 in a million	7.0 × 10-6 7.0 in a million		No
Chronic Hazard Index	Residential	0.22	0.18	0.05	1.0	No
	Occupational	0.40	0.31	0.10		No
	Sensitive	0.39	0.30	0.10		No
Acute Hazard Index	Residential	0.11	0.07	0.06	1.0	No
	Occupational	0.27	0.26	0.14		No
	Sensitive	0.16	0.07	0.09		No
Population Cancer Burden				0.3	0.5	No

Notes:
[a]The Alternative 5 column represents the maximum health values prior to subtracting the NEPA baseline.
[b]The NEPA Increment column represents the maximum difference of Alternative 5 minus the NEPA baseline.
[c]The maximum health values for Alternative 5, the NEPA Baseline, and the NEPA Increment may not all occur at the same receptor location. Therefore, the displayed values for Alternative 5 and the NEPA Baseline may not necessarily subtract to equal the NEPA Increment. The example given in the text under Impact AQ-7 for the proposed Project illustrates how the increments are calculated.
[d]The significance thresholds apply only to the NEPA increment.
[e]Exceedances of the thresholds are indicated in **bold**.

[f]Each positive 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.

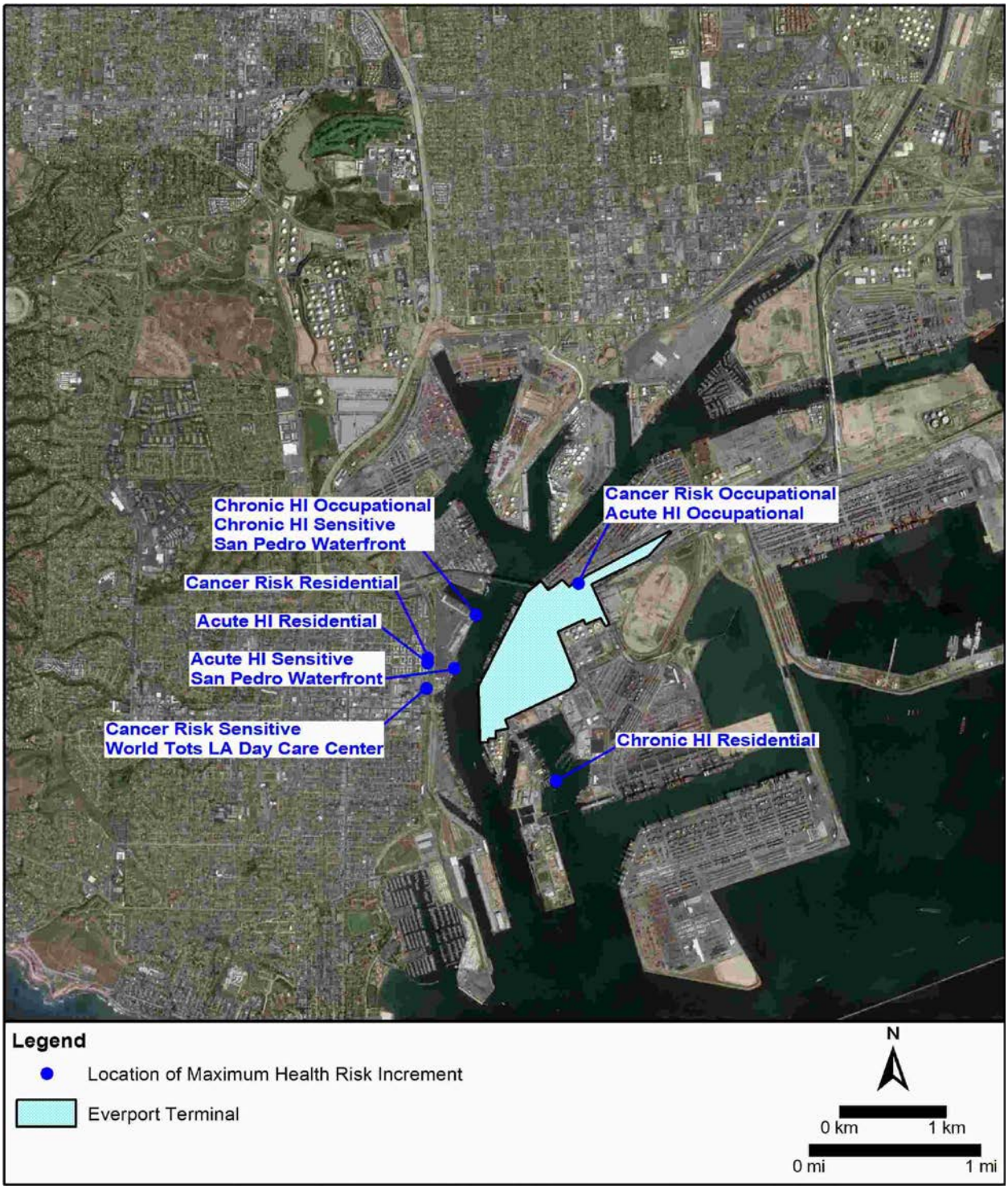

Figure B3-25. Locations of Maximum NEPA Health Impacts Estimated for Construction and Operation of Alternative 5 with Mitigation

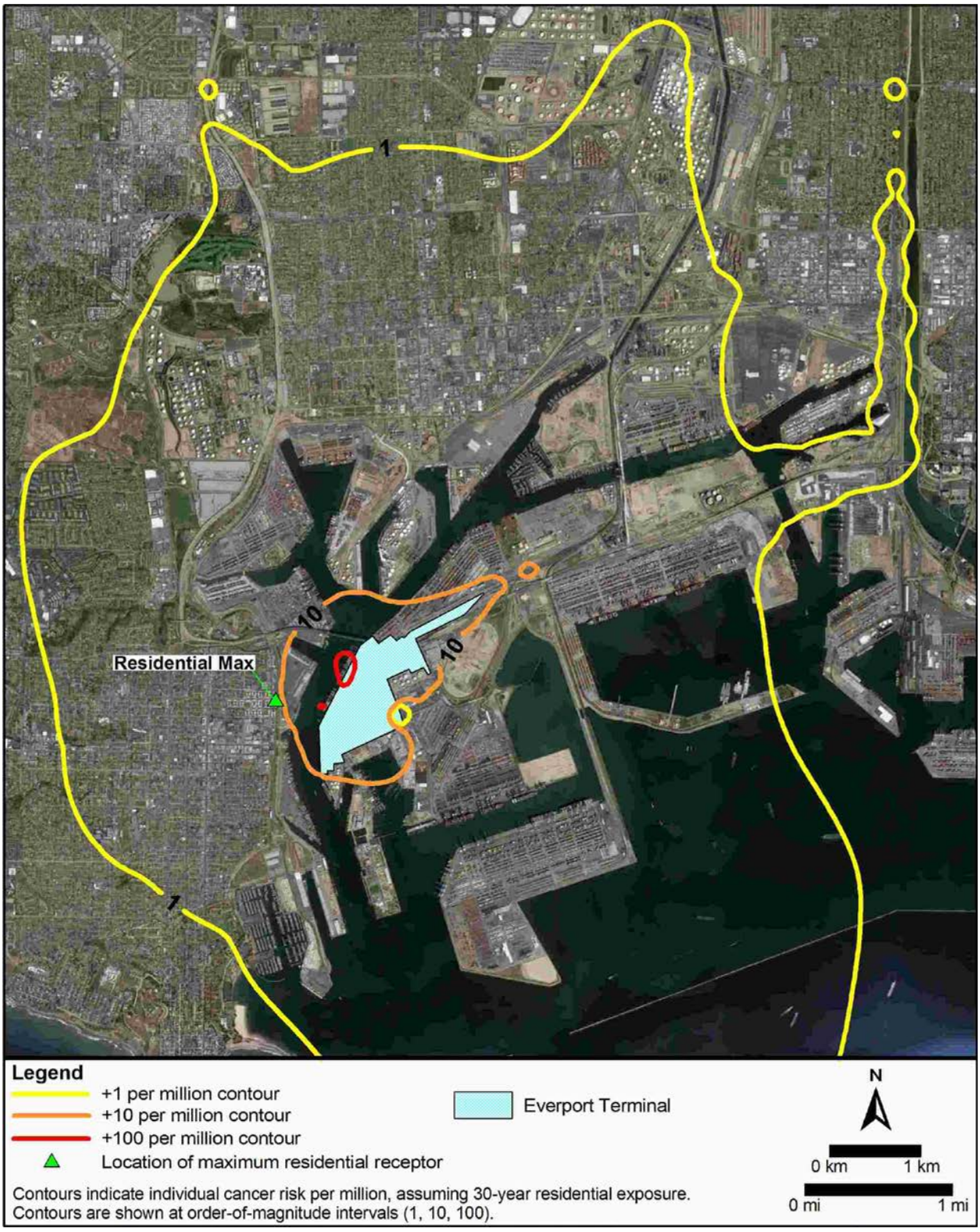

Figure B3-26: Isopleths of 30-Year Residential Cancer Risk – Alternative 5 with Mitigation – NEPA Increment

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

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