Appendix O

Human Health Risk Assessment

CP2 LNG Terminal and Mobile Sources Human Health Risk Assessment (HHRA)

June 30, 2023

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Section 1
Background

Venture Global CP2 LNG, LLC and Venture Global CP Express, LLC (together, the "Applicants") filed a joint application for authorization for new liquefied natural gas facilities in Cameron Parish, Louisiana, and related pipeline facilities in Louisiana and east Texas (collectively, the "Project") with the Federal Energy Regulatory Commission ("FERC"). CP2 LNG proposes to site, construct, and operate natural gas liquefaction, storage, and export facilities on 631.7 acres of the mainland and Monkey Island shoreline east of the Calcasieu Ship Channel.

This report responds to a request from the FERC for a risk assessment of the hazardous air pollutants (HAPs) potentially emitted from the proposed CP2 LNG Terminal and associated marine Mobile Sources (LNG Carriers and Tugboats).

The National Environmental Policy Act (NEPA) requires federal agencies, such as FERC, to assess the environmental effects of proposed actions prior to making decisions. Specifically, federal agencies are required to prepare detailed assessments of the environmental impact of actions with the potential to significantly affect the environment, such as approving permit applications. These statements are commonly referred to as Environmental Impact Statements (EIS).

On January 19, 2023, FERC issued a draft EIS for the Project. This Human Health Risk Assessment (HHRA) was prepared for the final EIS for the proposed CP2 LNG project.

Section 2
Air Modeling Analysis

2.1 HAP Emissions

Facility-wide Maximum Hourly (pounds per hour or lb/hr) and Annual Emission rates (pounds per year or lb/yr) for each HAP, as well as sources of the emissions, were provided in **Attachment 11-1, "**CP2 LNG Terminal and Mobile Sources (LNG Carriers and Tugboats) Hazardous Air Pollutants Emissions Summary" of the Applicants' response to Environmental Information Request No. 11[1].

Facility-wide HAP emissions included:

- CP2 LNG Terminal Stationary Sources; and
- Mobile Sources (LNG Carriers and Tugs).

The CP2 LNG Terminal stationary sources included emissions from combustion turbines, hot oil heaters, thermal oxidizers, flares, equipment leaks, emergency generators, and storage tanks. The mobile sources included emissions from LNG carrier engines, auxiliary boilers, and gas combustion units and tugboat engines. The emission sources and rates for the stationary sources were obtained from the "CP2 LNG Terminal Title V Permit and PSD Permit Application" ("the Permit"). The emission rates for the LNG carriers and tugs were obtained from Appendix 9D of the Applicants' Resource Report 9.[2]

The summary provided in **Attachment 11-1** also identified certain HAPs (i.e., 1,4-dichlorobenzene, ethylbenzene, naphthalene, toluene, and xylenes) that are exempted from ambient air dispersion modeling under the Louisiana Administrative Code (LAC), Title 33, Part III, Chapter 51, Comprehensive Toxic Air Pollutant Emission Control Program (LAC 33:III, Chapter 51) based on comparison of each HAP's annual emission rate against the relevant Minimum Emission Rate listed in the regulation. Although this subset of HAPs qualified for exemption from further review, all HAPs were included in the Applicants' dispersion modeling analysis, as discussed in the next section.

[1] Venture Global CP2 LNG, LLC and CP Express, LLC. Accession No. 20230526-5223, Attachment 11-1. May 26, 2023.
[2] Venture Global CP2 LNG, LLC and CP Express, LLC. Accession No. 20211202-5105. Resource Report 9 – Air and Noise Quality. December 2021.

2.2 Modeling of HAPs

FERC requested that the Applicants provide the maximum modeled 1-hour and annual off-property concentrations of all HAPs emitted from the proposed CP2 LNG Terminal and associated mobile sources identified in the Permit, including those exempted from ambient air dispersion modeling under the Louisiana Air Toxic regulations (Title 33.III, Chapter 51),[3] and the Applicants complied with the request.

The modeled ground-level concentrations (GLCs) of HAPs that serve as the bases of the HHRA were obtained from **Table 3-2** of Venture Global Hazardous Air Pollutants Air Quality Modeling Analysis Report for the CP2 LNG Terminal (the "Venture Global Modeling Report").[4]

The air quality modeling analysis was performed in accordance with the U.S. EPA's (EPA's) Guideline on Air Quality Models[5] and the Louisiana Department of Environmental Quality's (LDEQ's) Air Quality Modeling Procedures.[6] A detailed description of the air dispersion modeling methodology can be found in **Section 2** of the Venture Global Modeling Report.[7] As shown in **Appendix C** of the Venture Global Modeling Report (**Figures C-1 and C-2**), the maximum modeled impacts are not co-located for all HAPs because of the different locations and emissions characteristics (emission rate magnitude, stack height, propensity for aerodynamic plume downwash, etc.) of each source, both stationary and mobile, at the CP2 LNG Terminal.

The Venture Global Modeling Report provided modeled GLCs for total polycyclic aromatic hydrocarbons (PAHs) emitted from the facility rather than speciated PAHs needed for the HHRA. A review of Venture Global's HAPs analysis model output for PAH impacts from CP2 LNG emissions showed that the sources responsible for the maximum 1-hour, 8-hour, and annual GLCs for PAHs are the tugboats (operating within the security zone). The HAP emission rates for the tugboats used in the modeling were taken from Appendix 9D of Venture Global's Resource Report 9.[8] The total PAH emission rates shown in Table 9.D.1.4

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[3] LAC 33:III.5112.

[4] Venture Global CP2 LNG, LLC and CP Express, LLC. Accession No. 20230526-5223, Attachment 11-2 - Hazardous Air Pollutants Air Quality Modeling Analysis Report for the CP2 LNG Terminal. May 26, 2023.

[5] U.S. EPA Revisions to the Guideline on Air Quality Models: Enhancements to the AERMOD Dispersion Modeling System & Incorporation of Approaches to Address Ozone & Fine Particulate Matter, 82 Fed. Reg. 5182 (Jan. 17, 2017) (codified at 40 CFR Part 51, Appendix W).

[6] Air Quality Modeling Procedures, Louisiana Department of Environmental Quality (August 2006). Available at: https://deq.louisiana.gov/assets/docs/Air/ModelingProcedures0806.pdf. Accessed May 2023.

[7] Venture Global CP2 LNG, LLC and CP Express, LLC. Accession No. 20230526-5223, Attachment 11-2 - Hazardous Air Pollutants Air Quality Modeling Analysis Report for the CP2 LNG Terminal. May 26, 2023.

[8] Venture Global CP2 LNG, LLC and CP Express, LLC. Accession No. 20211202-5105. Resource Report 9 – Air and Noise Quality. December 2021.

of Appendix 9D were derived using information provided in Table D.1 (HAP Speciation Profiles for Commercial Marine Engines) of EPA's 2020 Port Emissions Inventory Guidance.[9] Table D.1 provided the fraction and basis (either VOC or $PM_{2.5}$) for marine vessel engine-based HAPs, including individual PAH species. The fractions for the individual PAH species were applied to the appropriate hourly and annual VOC or $PM_{2.5}$ emission rates presented in Table 9.D.1.4 of Appendix 9D. The resulting emission rates for the individual PAH species were divided by the total PAH emission rate, with that fraction then applied to the maximum total PAH impact of interest (e.g., 1-hour GLC) from the Venture Global Modeling Report to calculate the maximum impact for each individual PAH species for each averaging period of interest.

The maximum modeled 1-hour and annual concentrations (in micrograms per cubic meter or $\mu g/m^3$) for each HAP that provide the bases for this HHRA are provided in **Table 1**.

[9] EPA. 2020. Port Emissions Inventory Guidance: Methodologies for Estimating Port-Related and Goods Movement Mobile Source Emissions, Office of Transportation and Air Quality, U.S. Environmental Protection Agency. September 2020. EPA-420-B-20-046.

Table 1
Maximum Modeled Off-Property Concentrations

Pollutant	Annual Concentration ($\mu g/m^3$)	Hourly Concentration ($\mu g/m^3$)
1,3-Butadiene	0.00015	0.0079
1,4-Dichlorobenzene	0.00002	0.00109
Acetaldehyde	0.00194	0.10445
Acrolein	0.00035	0.01672
Benzene	0.00355	4.528
Ethylbenzene	0.00143	0.08968
Formaldehyde	0.00726	0.33295
Hexane	0.03038	10.80628
Acenaphthene	0.0000072	0.0003968
Acenaphthylene	0.0000168	0.0009199
Anthracene	0.0000489	0.0026816
Benz[a]Anthracene	0.0000003	0.0000147
Benzo[a]Pyrene	0.0000001	0.0000070
Benzo[b]Fluoranthene	0.0000003	0.0000139
Benzo[k]Fluoranthene	0.0000001	0.0000070
Benzo[g,h,i]Fluoranthene	0.0000040	0.0002198
Chrysene	0.0000005	0.0000271
Dibenzo[a,h]Anthracene	0.0000003	0.0000144
Fluoranthene	0.0000027	0.0001494
Fluorene	0.0000233	0.0012785
Indeno[1,2,3-cd]Pyrene	0.0000003	0.0000139
Naphthalene	0.0044514	0.2440294
Phenanthrene	0.0001928	0.0105707
Pyrene	0.0000010	0.0000561
Propylene Oxide	0.00122	0.07566
Toluene	0.00566	0.67132
Xylenes	0.00306	0.35867
Cadmium	0.00002	0.001
Chromium	0.00002	0.00127
Nickel	0.00003	0.0019

Section 3
Human Health Risk Assessment

Due to the level of concern regarding potential health effects associated with HAPs emissions from the CP2 LNG Terminal, as well as impacts on environmental justice communities, FERC requested that a HHRA be conducted to evaluate the potential for short- (acute) and long-term (chronic) health effects from inhalation of HAPs potentially emitted from the Project using nationally recognized methods.

3.1 Methodology for Characterizing Human Health Risk

This HHRA was conducted in accordance with methods outlined in EPA's 2005 "Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities" (HHRAP).[10] The HHRAP provides a standardized methodology for conducting combustion risk assessments and was, therefore, chosen as appropriate guidance for this HHRA. Back-up risk calculations are provided in the appendices of this report.

3.1.1 Exposure Assessment

Exposure Setting

CP2 LNG proposes to site, construct, and operate natural gas liquefaction, storage, and export facilities on 631.7 acres of the mainland and Monkey Island shoreline east of the Calcasieu Ship Channel in Cameron Parish, Louisiana.

The LNG Terminal site will be the permanent mainland-based portion of the LNG facility, which will include pretreatment facilities, a liquefaction plant and support facilities, LNG storage tanks, power generation facilities, and ancillary facilities. The Marine Facilities will be on the southwest shoreline of Monkey Island, between the Calcasieu Ship Channel and Calcasieu Pass, and will include the LNG carrier loading docks and an LNG carrier berthing area. LNG transfer lines and utilities will be installed between the Terminal Site and Marine Facilities.

The closest residence is approximately 330 feet northeast of the Terminal Site floodwall. Due to recent hurricanes in the Project area, many residences became uninhabitable and most of the occupied residences near the Terminal Site are approximately 0.8 miles northwest in the Town of Cameron. However, there are several recreational vehicle (RV) camping sites in Cameron and Calcasieu Parishes less

[10] EPA. 2005. U.S. Environmental Protection Agency. Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities. EPA530-R-05-006. https://archive.epa.gov/epawaste/hazard/tsd/td/web/html/risk.html.

than 0.25 miles from the Project. No planned residential developments have been identified within 0.25 mile of the Project. There are several proposed commercial developments within 2 miles of the Terminal Site, including other LNG terminals and natural gas infrastructure.[11]

According to the EIS, construction and operation of the Project would not cross or directly affect any national or state-designated Wild and Scenic Rivers, national or state historic landmarks, national forests, national parks, national recreation trails, Indian lands, land managed under the Wetland Reserve Program or Conservation Reserve Program, state parks, preservation areas, other state-recognized public areas (refuges, wetland conservation areas), private conservation lands or land trusts, or wilderness areas. [12]

Exposure Pathways

An exposure "pathway" is the course a chemical takes from its source to the person potentially exposed and consists of:

1. A source (e.g., combustion turbine, engine, flare, etc.) and mechanism of HAP release (i.e., stack or fugitive emissions);

2. A receiving medium (e.g., air);

3. A point of potential human contact (e.g., property boundary); and

4. An exposure route (e.g., inhalation).

This HHRA estimated chronic (long-term) cancer risk and non-cancer hazard, as well as acute (short-term) hazard via inhalation of compounds potentially emitted from stationary combustion sources, marine mobile sources, and fugitive emissions from CP2 LNG Terminal equipment.

Exposure Scenario and Location

This HHRA evaluated inhalation exposure of hypothetical adult and child Residents for which Reasonable Maximum Exposure (RME) was assumed.

RME means that the hypothetical Resident is conservatively assumed to be exposed 24 hours/day, 350 days/year (two weeks assumed for travel) for 30 years for the adult Resident (represents ~ 95th percentile

[11] Accession No. 20230119-3072, CP2 LNG and CP Express Project Draft Environmental Impact Statement. FERC/DEIS-0328. January 2023. p. 4-156 and 4-159.
[12] Accession No. 20230119-3072, CP2 LNG and CP Express Project Draft Environmental Impact Statement. FERC/DEIS-0328. January 2023. p. 4-158.

residency time for the U.S. population)[13] and six years for the child Resident.[14]In addition, residential inhalation exposures were assumed to occur at the area (i.e., receptor) of greatest contaminant concentration (i.e., maximum modeled 1-hour and annual concentrations), to maximize estimated exposure. This exposure scenario is intended to evaluate potential risk/hazard with a level of protectiveness to address the possibility of exposures not directly evaluated in the HHRA.

Exposure Concentrations

Chronic exposures occur over time. To calculate an average inhalation exposure per unit of time (Exposure Concentration, or EC), the maximum modeled annual GLC or air concentration was multiplied by the Exposure Frequency (EF) and Exposure Duration (ED) and divided by the time over which exposure is averaged, which differs for carcinogens (70 years) and non-carcinogens (30 years). Estimating ECs in air does not involve or require adjustment for differences in respiration rates for adults and children, as those are inherent to inhalation toxicity factors.[15] The equation for calculating chronic ECs is provided below.

$$EC = \frac{CA\ x\ EF\ x\ ED}{AT_c\ or\ AT_{nc}}$$

Where:			**Value**
EC	=	Exposure concentration (µg/m^3)	*Calc*
CA	=	Air concentration (µg/m^3)	*Model*
EF	=	Exposure frequency (days/year)	350
ED_{adult}	=	Adult Exposure duration (years)	30
ED_{child}	=	Child Exposure duration (years)	6
AT_c	=	Carcinogen (70 years x 365 days/year) averaging time (days)	25550
$AT_{nc\ adult}$	=	Non-Cancer (30 years x 365 days/year) averaging time (days)	10950
$AT_{nc\ child}$	=	Non-Cancer (6 years x 365 days/year) averaging time (days)	2190

For acute exposures, the maximum modeled 1-hour concentration is used without any adjustment since acute exposures occur intermittently.

The ECs calculated for use in this HHRA are provided in **Table 2**.

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[13] EPA. 2011. United States Environmental Protection Agency. "Exposure Factors Handbook: 2011 Edition". EPA/600/R-090/052F. Tables 16-108. September.

[14] EPA. 2005. U.S. Environmental Protection Agency. Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities. EPA530-R-05-006. p. 6 – 20.

[15] EPA. 2005. U.S. Environmental Protection Agency. Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities. EPA530-R-05-006. p. 6-2.

Table 2
Exposure Concentrations (ECs)

Pollutant	Exposure Concentrations (µg/m^3)				
	Chronic				Acute
	Carcinogen		Non-Carcinogen		
	Adult	Child	Adult	Child	
1,3-Butadiene	6.16E-05	1.23E-05	1.44E-04	1.44E-04	7.90E-03
1,4-Dichlorobenzene	8.22E-06	1.64E-06	1.92E-05	1.92E-05	1.09E-03
Acetaldehyde	7.97E-04	1.59E-04	1.86E-03	1.86E-03	1.04E-01
Acrolein	1.44E-04	2.88E-05	3.36E-04	3.36E-04	1.67E-02
Benzene	1.46E-03	2.92E-04	3.40E-03	3.40E-03	4.53E+00
Ethylbenzene	5.88E-04	1.18E-04	1.37E-03	1.37E-03	8.97E-02
Formaldehyde	2.98E-03	5.97E-04	6.96E-03	6.96E-03	3.33E-01
Hexane	1.25E-02	2.50E-03	2.91E-02	2.91E-02	1.08E+01
Acenaphthene	2.97E-06	5.95E-07	6.94E-06	6.94E-06	3.97E-04
Acenaphthylene	6.90E-06	1.38E-06	1.61E-05	1.61E-05	9.20E-04
Anthracene	2.01E-05	4.02E-06	4.69E-05	4.69E-05	2.68E-03
Benz[a]Anthracene	1.09E-07	2.19E-08	2.55E-07	2.55E-07	1.47E-05
Benzo[a]Pyrene	5.18E-08	1.04E-08	1.21E-07	1.21E-07	6.96E-06
Benzo[b]Fluoranthene	1.04E-07	2.07E-08	2.42E-07	2.42E-07	1.39E-05
Benzo[k]Fluoranthene	5.18E-08	1.04E-08	1.21E-07	1.21E-07	6.96E-06
Benzo[g,h,i]Fluoranthene	1.64E-06	3.27E-07	3.82E-06	3.82E-06	2.20E-04
Chrysene	2.02E-07	4.04E-08	4.71E-07	4.71E-07	2.71E-05
Dibenzo[a,h]Anthracene	1.07E-07	2.14E-08	2.50E-07	2.50E-07	1.44E-05
Fluoranthene	1.11E-06	2.22E-07	2.59E-06	2.59E-06	1.49E-04
Fluorene	9.58E-06	1.92E-06	2.24E-05	2.24E-05	1.28E-03
Indeno[1,2,3-cd]Pyrene	1.04E-07	2.07E-08	2.42E-07	2.42E-07	1.39E-05
Naphthalene	1.83E-03	3.66E-04	4.27E-03	4.27E-03	2.44E-01
Phenanthrene	7.92E-05	1.58E-05	1.85E-04	1.85E-04	1.06E-02
Pyrene	4.18E-07	8.35E-08	9.75E-07	9.75E-07	5.61E-05
Propylene Oxide	5.01E-04	1.00E-04	1.17E-03	1.17E-03	7.57E-02
Toluene	2.33E-03	4.65E-04	5.43E-03	5.43E-03	6.71E-01
Xylenes	1.26E-03	2.52E-04	2.93E-03	2.93E-03	3.59E-01
Cadmium	8.22E-06	1.64E-06	1.92E-05	1.92E-05	1.00E-03
Chromium	8.22E-06	1.64E-06	1.92E-05	1.92E-05	1.27E-03
Nickel	1.23E-05	2.47E-06	2.88E-05	2.88E-05	1.90E-03

3.1.2 Toxicity Assessment

This HHRA involves multiple HAPs and multiple toxic end points.

Toxicity factors used to estimate chronic (long-term) cancer risk are Inhalation Unit Risk Factors (IURFs) and those used to estimate chronic non-cancer hazards include Reference Concentrations (RfCs) or Minimal Risk Levels (MRLs). Toxicity factors for estimating acute (short-term) inhalation hazards are comprised of California EPA Acute Reference Exposure Levels (Ca RELs) and EPA 1-Hour Acute Exposure Guideline Levels (AEGLs).

Chronic (Long-Term) Toxicity Factors

A hierarchical approach was used to select the appropriate toxicity criteria for use in estimating chronic (long-term) cancer risk and non-cancer hazards. Chronic toxicity criteria for the HAPs were selected from the following sources, in order of preference:

1. Cancer IURFs and non-cancer RfCs from EPA's Integrated Risk Information System (IRIS) at https://www.epa.gov/iris.

2. Cancer IURFs and non-cancer RfCs from EPA's Provisional Peer Reviewed Toxicity Values (PPRTVs) at https://www.epa.gov/pprtv/provisional-peer-reviewed-toxicity-values-pprtvs-assessments.

3. Chronic Minimal Risk Levels (MRLs) located in Toxicological Profiles published by the Agency for Toxic Substances Disease Registry (ATSDR) at https://www.atsdr.cdc.gov/toxprofiledocs/index.html.

Carcinogen IURFs are expressed in terms of risk per concentration for inhalation exposures (i.e., risk per $\mu g/m^3$ or $(\mu g/m^3)^{-1}$). Non-cancer RfCs and MRLs are expressed as air concentrations and have been converted from their original units of mg/m^3 to $\mu g/m^3$ for ease of use with the modeled air concentrations, which are expressed in units of $\mu g/m^3$.

EPA defines the IURF as an upper-bound estimate of the increased cancer risk from inhalation exposure to a concentration of 1 $\mu g/m^3$ for a lifetime. RfCs are defined as an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.[16] The ATSDR defines an MRL as an estimate of the daily human exposure to a hazardous

[16] EPA website. "Basic Information about the Integrated Risk Information System". https://www.epa.gov/iris/basic-information-about-integrated-risk-information-system. Visited on June 6, 2023; "Integrated Risk Information System (IRIS) Glossary". https://sor.epa.gov/sor_internet/registry/termreg/searchandretrieve/glossariesandkeywordlists/search.do?

substance that is likely to be without appreciable risk of adverse non-cancer health effects over a specified duration of exposure.[17]

PAHs (polycyclic aromatic hydrocarbons) are members of the same chemical family and exhibit similar toxicological properties. However, they differ in the degree of toxicity. Relative potency factors (RPFs) have been developed for individual PAH species with carcinogenic properties.[18] These RPFs are based on the carcinogenic potency of each PAH species relative to that of benzo[a]pyrene. In deriving the RPFs, it was assumed that the PAHs have similar dose-response curves, but that it takes a proportionally larger concentration of non-benzo[a]pyrene PAHs to induce an equivalent tumor response. Since they are specific to carcinogenic potency, RPFs are not used to estimate non-cancer toxicity factors. Consistent with the approach used by EPA in developing its regional screening levels,[19] the RPFs have been applied to the IURF for benzo[a]pyrene to calculate IURFs for each carcinogenic PAH, as shown in **Table 3.**

Table 3
Carcinogenic Polycyclic Aromatic Hydrocarbon (PAH) Inhalation Unit Risk Factors (IURFs)

Calculation of IURFs for Carcinogenic PAHs			
PAH	**RPF**	**Benzo[a]Pyrene IURF $(\mu g/m^3)^{-1}$**	**Calculated IURF $(\mu g/m^3)^{-1}$**
Benzo(a)pyrene	1	6.00E-04	6.00E-04
Benz(a)anthracene	0.1		6.00E-05
Benzo(b)fluoranthene	0.1		6.00E-05
Benzo(k)fluoranthene	0.01		6.00E-06
Chrysene	0.001		6.00E-07
Dibenz(a,h)anthracene	1		6.00E-04
Indeno(1,2,3-c,d)pyrene	0.1		6.00E-05

Source: Table 8 of EPA's *Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons*.[20]

details=&glossaryName=IRIS%20Glossary#:~:text=Definition%3A%20The%20probability%20that%20an,1%20%C2%B5g%2Fm%C2%B3%20in%20air. Visited on June 30, 2023

[17] ATSDR website. "Minimal Risk Levels (MRLs) – For Professionals". https://www.atsdr.cdc.gov/mrls/index.html#:~:text=An%20MRL%20is%20an%20estimate,a%20specified%20duration%20of%20exposure. Visited on June 7, 2023.

[18] EPA. 1993. Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons. EPA/600/R-93/089. Table 8, p. 17. https://www.epa.gov/risk/regional-screening-levels-rsls-users-guide#toxicity.

[19] EPA website. Regional Screening Levels (RSLs) - User's Guide. https://www.epa.gov/risk/regional-screening-levels-rsls-users-guide#toxicity. Visited on June 23, 2023.

[20] EPA. 1993. Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons. EPA/600/R-93/089. Table 8, p. 17. https://www.epa.gov/risk/regional-screening-levels-rsls-users-guide#toxicity.

Chronic toxicity factors used in this HHRA and their sources are provided in **Table 4**. Also provided in **Table 4** are the critical effects on which non-cancer toxicity factors are based.

Acute Toxicity Factors (ATFs)

The following sources were searched for 1-hour toxicity criteria, in order of preference.

1. California EPA Acute Reference Exposure Levels (RELs) at https://oehha.ca.gov/air/general-info/oehha-acute-8-hour-and-chronic-reference-exposure-level-rel-summary.
2. EPA 1-hour Acute Exposure Guidelines (AEGL-1) values at https://www.epa.gov/aegl/access-acute-exposure-guideline-levels-aegls-values.

California EPA Acute RELs are defined as the concentration in air at or below which no adverse health effects are anticipated in the general population, including sensitive individuals, for a specified exposure period (i.e., 1-hour) on an intermittent basis.[21] EPA AEGL-1 values are concentrations in air above which the general population, including susceptible individuals, could experience notable discomfort, irritation, or certain asymptomatic non-sensory effects. However, the effects are not disabling and are transient and reversible upon cessation of exposure. AEGL-2 values are the airborne concentration of a substance above which it is anticipated that the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape.[22] An AEGL-2 value was only selected if a MRL or AEGL-1 value was not available for a HAP.

[21] OEHHA. 2015. California Environmental Protection Agency Office of Environmental Health Hazard Assessment. Air Toxics Hot Spots Program. Risk Assessment Guidelines. February 2015. p. 6-3.
[22] EPA website. "About Acute Exposure Guideline Levels (AEGLs)". https://www.epa.gov/aegl/about-acute-exposure-guideline-levels-aegls. Visited on June 6, 2023.

Table 4
Chronic Inhalation Toxicity Factors

Pollutant	Cancer IURF $(\mu g/m^3)^{-1}$	Reference	Non-Cancer RfC $(\mu g/m^3)$	Effect	Reference
1,3-Butadiene	3.00E-05	IRIS	2.00E+00	ovarian atrophy	IRIS
1,4-Dichlorobenzene	NA	NA	8.00E+02	liver/reproductive toxicity	IRIS
Acetaldehyde	2.20E-06	IRIS	9.00E+00	degeneration of olfactory epithelium	IRIS
Acrolein	NA	NA	2.00E-02	nasal lesions	IRIS
Benzene	7.80E-06	IRIS	3.00E+01	decreased lymphocytes	IRIS
Ethylbenzene	NA	NA	1.00E+03	developmental effects	IRIS
Formaldehyde	1.30E-05	IRIS	9.80E+00	histological changes in nasal epithelium	ATSDR
Hexane	NA	NA	7.00E+02	peripheral neuropathy	IRIS
Acenaphthene	NA	NA	NA	NA	NA
Acenaphthylene	NA	NA	NA	NA	NA
Anthracene	NA	NA	NA	NA	NA
Benz[a]Anthracene	6.00E-05	RPF	NA	NA	NA
Benzo[a]Pyrene	6.00E-04	IRIS	2.00E-03	developmental effects	IRIS
Benzo[b]Fluoranthene	6.00E-05	RPF	NA	NA	NA
Benzo[k]Fluoranthene	6.00E-06	RPF	NA	NA	NA
Benzo[g,h,i]Fluoranthene	NA	NA	NA	NA	NA
Chrysene	6.00E-07	RPF	NA	NA	NA
Dibenzo[a,h]Anthracene	6.00E-04	RPF	NA	NA	NA
Fluoranthene	NA	NA	NA	NA	NA
Fluorene	NA	NA	NA	NA	NA
Indeno[1,2,3-cd]Pyrene	6.00E-05	RPF	NA	NA	NA
Naphthalene	NA	NA	3.00E+00	hyperplasia nasal epithelium	IRIS
Phenanthrene	NA	NA	NA	NA	NA
Pyrene	NA	NA	NA	NA	NA
Propylene Oxide	3.70E-06	IRIS	3.00E+01	nasal epithelial infolds	IRIS
Toluene	NA	NA	5.00E+03	CNS	IRIS
Xylenes	NA	NA	1.00E+02	CNS	IRIS
Cadmium	1.80E-03	IRIS	NA	NA	NA
Chromium	1.20E-02	IRIS	8.00E-03	nasal septum atrophy	IRIS
Nickel	4.80E-04	IRIS	9.00E-02	lung inflammation	IRIS

ATSDR – Agency for Toxic Substances and Disease Registry
CNS – Central Nervous System
IRIS – Integrated Risk Information System
IURF – Inhalation Unit Risk Factor
RfC – Reference Concentration
RPF – PAH Relative Potency Factor

Acute toxicity factors used in this HHRA and their sources are provided in **Table 5**.

Table 5
Acute Inhalation Toxicity Factors

Pollutant	Acute Toxicity Factor ($\mu g/m^3$)	Effect	Reference
1,3-Butadiene	6.60E+02	developmental effects	Ca REL
1,4-Dichlorobenzene	NA	NA	NA
Acetaldehyde	4.70E+02	eye/respiratory irritation	Ca REL
Acrolein	2.50E+00	eye/respiratory irritation	Ca REL
Benzene	2.70E+01	developmental, immune, hematological effects	Ca REL
Ethylbenzene	1.43E+05	CNS	AEGL-1 (Interim)
Formaldehyde	5.50E+01	eye irritation	Ca REL
Hexane	1.00E+04	CNS	AEGL-2
Acenaphthene	NA	NA	NA
Acenaphthylene	NA	NA	NA
Anthracene	NA	NA	NA
Benz[a]Anthracene	NA	NA	NA
Benzo[a]Pyrene	NA	NA	NA
Benzo[b]Fluoranthene	NA	NA	NA
Benzo[k]Fluoranthene	NA	NA	NA
Benzo[g,h,i]Fluoranthene	NA	NA	NA
Chrysene	NA	NA	NA
Dibenzo[a,h]Anthracene	NA	NA	NA
Fluoranthene	NA	NA	NA
Fluorene	NA	NA	NA
Indeno[1,2,3-cd]Pyrene	NA	NA	NA
Naphthalene	NA	NA	NA
Phenanthrene	NA	NA	NA
Pyrene	NA	NA	NA
Propylene Oxide	3.10E+03	eye/respiratory irritation, developmental effects	Ca REL
Toluene	5.00E+03	eye/respiratory irritation, CNS	Ca REL
Xylenes	2.20E+04	eye/respiratory irritation, CNS	Ca REL
Cadmium	4.60E+02	respiratory irritation	AEGL-1 (Interim)
Chromium	2.00E-01	respiratory irritation	Ca REL
Nickel	2.00E-01	immune system effects	Ca REL

AEGL – Acute Exposure Guideline Levels
Ca REL – California EPA Acute Reference Exposure Levels
CNS – Central Nervous System effects

3.1.3 Risk Characterization

Chronic cancer risks and non-cancer hazards as well as acute hazards associated with inhalation exposure are estimated using ECs (provided in **Table 2**) with the appropriate inhalation toxicity factors (chronic toxicity factors are provided in **Table 4**, while acute toxicity factors are provided in **Table 5**).

Chronic Cancer Risk

Cancer risk estimates represent the incremental probability that an individual will develop cancer over a lifetime due to exposure to a carcinogenic HAP. HAP-specific cancer risks were estimated by multiplying the chronic carcinogen EC for the HAP (EC_c provided in **Table 2**) by the IURF (provided in **Table 4**) for the HAP, as shown in the equation below.

$$Cancer\ Risk = EC_c\ x\ IURF$$

Where:			**Value**
Cancer Risk =		Probability of developing cancer over a lifetime (unitless)	*Calculated*
EC_C	=	Chronic carcinogen exposure concentration (µg/m^3)	*Table 2*
IURF	=	Inhalation Unit Risk Factor (µg/m^3)$^{-1}$	*Table 4*

Although different carcinogenic PAHs have different potencies, they produce similar tumor responses[23] (i.e., similar cellular origin and mechanism). Therefore, the total cancer risk associated with inhaling all carcinogenic PAHs was estimated as follows.

$$Cancer\ Risk_{TPAH} = \sum_{PAHi}^{n} Cancer\ Risk_{PAHi}$$

Where:			**Value**
Cancer Risk$_{TPAH}$ =		Total PAH cancer risk across all carcinogenic PAHs (unitless)	*Calculated*
Cancer Risk$_{PAHi}$ =		Cancer risk for individual PAH$_i$ (unitless)	*Calculated*

In addition, because it is possible for receptors (i.e., Residents) to be exposed to multiple carcinogenic HAPs via a single exposure pathway (i.e., inhalation), the total cancer risk associated with inhaling all carcinogenic HAPs was estimated as follows.

$$Cancer\ Risk_{THAP} = \sum_{HAPi}^{n} Cancer\ Risk_{HAPi}$$

Where:			**Value**
Cancer Risk$_{THAP}$ =		Total cancer risk across all carcinogenic HAPs (unitless)	*Calculated*
Cancer Risk$_{HAPi}$ =		Cancer risk for individual HAP$_i$ (unitless)	*Calculated*

[23] EPA. 1993. Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons. EPA/600/R-93/089. Table 8, p. 17. https://www.epa.gov/risk/regional-screening-levels-rsls-users-guide#toxicity.

The summing of individual HAP cancer risks is performed to account for the possibility of joint or combined effects from exposure to multiple HAPs and is based on an assumption that doses of different HAPs can be treated as roughly additive with regard to inducing adverse health effects. However, a key problem with the strategy of simply adding risk across HAPs is that in the absence of complete scientific data on certain aspects of exposure and toxicity and in keeping with the EPA's mission of protecting public health, default assumptions are often made in HHRAs and those defaults are generally selected with the goal of ensuring that risk is not underestimated.[24] As a result, many assumptions used in HHRAs are upper-bound estimates, which is a value that is so large that most other values in the dataset are less than the value chosen. For example, this HHRA assumed that an adult Resident lives at the location of maximum impact (i.e., the location where the highest concentration of each HAP was predicted, even though maximum impacts are not co-located for all HAPs) for 30 years. This 30-year residential occupancy approximates the 95th percentile residency time for the U.S. population,[25] which means that 94 percent of the U.S. population resides in a single home for less 30 years. The average residency tenure in the U.S. is substantially less at 11.7 years.[26] IURFs also represent upper-bound estimates (of the potential risk at low doses).[27] In addition to using upper-bound estimates for the residential occupancy time and IURF, as well as assuming that residents live at the maximum impact location for all HAPs (i.e., are maximally exposed to all HAPs simultaneously), it was also assumed that residents are outdoors being exposed to emissions from the CP2 LNG facility 24-hours per day, seven days per week, which is not only unrealistic, it is impossible. Hence, the estimated cancer risk for each individual HAP is a high-end estimate that is almost certainly grossly overstated. With many individual risk estimates, it becomes highly unlikely that they are all at their upper end estimate.[28] Therefore, as the number of carcinogenic HAPs being added increases, the sum of the upper bound risk estimates becomes increasingly improbable as a realistic estimate of overall risk.[29] In conclusion, summing cancer risk across all carcinogenic HAPs is an extremely conservative approach (i.e., health protective) that in all likelihood substantially overestimates risk from a particular source for the following reasons: 1) maximum modeled annual concentrations for all HAPs are not co-

[24] EPA. 2004. United States Environmental Protection Agency. An Examination of EPA Risk Assessment Principles and Practices, EPA/100/B-04/001. p. 11. https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=100045MJ.TXT; EPA . 2014. Framework for Human Health Risk Assessment to Inform Decision Making. EPA/100/001. R-14. p. 44. https://www.epa.gov/sites/default/files/2014-12/documents/hhra-framework-final-2014.pdf.

[25] EPA. 2011. United States Environmental Protection Agency. "Exposure Factors Handbook: 2011 Edition". EPA/600/R-090/052F. Table 16-108. September.

[26] EPA. 2011. United States Environmental Protection Agency. "Exposure Factors Handbook: 2011 Edition". EPA/600/R-090/052F. Table 16-108. September.

[27] EPA. 2005. United States Environmental Protection Agency. Guidelines for Carcinogen Risk Assessment. EPA/630/P-03/001F. p. 3-23.

[28] Cogliano, V. J. (1997). Plausible Upper Bounds: Are Their Sums Plausible? 1. Risk Analysis, 17(1), 77-84.

[29] EPA. 2004. United States Environmental Protection Agency. An Examination of EPA Risk Assessment Principles and Practices, EPA/100/B-04/001. p. 20.

located (i.e., exposure to them does not occur at the same location or simultaneously); 2) estimated cancer risks for individual HAPs are upper-bound estimates and as the number of HAPs increases, it becomes increasingly improbable that their summation represents a valid estimate of overall risk;[30] and 3) cancer is not a single disease, but a variety of neoplastic disorders with different characteristics that occur in different tissues at different life stages;[31] and for this reason, cancers that occur at different sites within the body, or with different cellular origin, likely have independent mechanisms of causation and are, therefore, not necessarily additive.[32]

Chronic Non-Cancer Hazard

Standard risk assessment methodology is to assume that, for most chemicals that cause adverse health effects other than cancer, there is a level of exposure below which no adverse effects will be observed. Therefore, estimating non-cancer hazard typically involves comparing an estimated chronic exposure concentration in air or the EC_{nc} (provided in **Table 2**) to the RfC (provided in **Table 4**), which is an estimate of the continuous inhalation exposure that is likely to be without an appreciable risk of deleterious effects. In some instances, HAP-specific RfCs were not available and a MRL was used instead. The comparisons of inhalation exposure estimates to RfCs (or MRLs) are known as chronic hazard quotients (HQ), which are calculated as follows:

$$HQ_{chronic} = \frac{EC_{nc}}{RfC\ or\ MRL}$$

Where:			**Value**
$HQ_{chronic}$	=	Chronic Hazard Quotient (unitless)	*Calculated*
EC_{nc}	=	Chronic Non-Cancer Exposure Concentration (µg/m^3)	*Table 2*
RfC	=	Reference Concentration (µg/m^3)	*Table 4*
MRL	=	Minimal Risk Level (µg/m^3)	*Table 4*

[30] EPA. 2004. United States Environmental Protection Agency. An Examination of EPA Risk Assessment Principles and Practices, EPA/100/B-04/001. p. 20.

[31] NRC. 1994. National Research Council, Committee on Risk Assessment of Hazardous Air Pollutants, Commission on Life Sciences. United States National Academies of Sciences, Engineering, and Medicine. Science and Judgment in Risk Assessment (1994). p. 230. https://nap.nationalacademies.org/download/2125.

[32] NRC. 1994. National Research Council, Committee on Risk Assessment of Hazardous Air Pollutants, Commission on Life Sciences. United States National Academies of Sciences, Engineering, and Medicine. Science and Judgment in Risk Assessment (1994). p. 230. https://nap.nationalacademies.org/download/2125; Salmon, A. G., & Roth, L. A. 2010. Cancer risk based on an individual tumor type or summing of tumors. Cancer Risk Assessment: Chemical Carcinogenesis, Hazard Evaluation, and Risk Quantification, 716-735.

As with carcinogenic HAPs, a receptor (i.e., a Resident) might be exposed to multiple HAPs associated with non-cancer health effects by the same pathway. Therefore, the total chronic hazard for the exposure pathway (i.e., inhalation) is estimated by summing the individual HAP HQs that have similar effects (e.g., reproductive or developmental effects) or affect the same target organ (e.g., CNS, nasal epithelium) to obtain a total pathway Hazard Index (HI). Summing only the HQs for HAPs that have similar health effects is referred to as segregating the HI.

$$HI_{chronic} = \sum_{i}^{n} HQ_i$$

Where:		**Value**
$HI_{chronic}$ =	Chronic Hazard Index across all HAPs with similar effects (unitless)	*Calculated*
HQ_i =	Hazard Quotients for individual HAP_i (unitless)	*Calculated*

As shown in **Table 4**, chronic health effects that are associated with more than one HAP include: 1) effects on nasal epithelium (acetaldehyde, acrolein, formaldehyde, naphthalene, propylene oxide, and chromium); 2) developmental toxicity (benzo[a]pyrene and ethylbenzene); and 3) CNS effects (toluene and xylenes). Therefore, non-cancer HIs will be estimated for these endpoints by summing individual HAP HQs for these effects. However, it should be noted that summing chronic HQs across HAPs, even those that have similar effects or affect the same target organ, is a conservative (i.e., health protective) approach that likely overestimates non-cancer hazard because: 1) maximum modeled annual concentrations for all HAPs are not co-located (i.e., simultaneous exposure does not necessarily occur); 2) for the same reasons described for cancer risks above, estimated HQs for individual HAPs tend to be high-end estimates and as the number of HAPs increases, it becomes increasingly improbable that their summation represents a valid estimate of the $HI_{chronic}$; and 3) non-cancer effects such as developmental toxicity affect different body parts, have different cellular origins, and likely have independent mechanisms of action, which means that they are not necessarily additive.

Acute Hazard

The potential for adverse health effects from acute inhalation exposure to HAP emissions were estimated by comparing the EC_{acute} (**Table 2**) to the HAP-specific Acute Toxicity Factors (ATFs consisting of Ca RELs and AEGLs) provided in **Table 5**. This comparison is known as the acute hazard quotient (HQ_{acute}) and is calculated as follows.

$$HQ_{acute} = \frac{EC_{acute}}{ATF}$$

Where:		**Value**
HQ_{acute} =	Acute Hazard Quotient (unitless)	*Calculated*
EC_{acute} =	Acute Exposure Concentration (µg/m^3)	*Table 2*
ATF =	Acute Toxicity Factor (µg/m^3)	*Table 5*

Acute HQs (HQ_{acute}) from individual HAPs are summed for HAPs that have similar effects (e.g., eye irritation, CNS effects, etc.) to obtain an acute Hazard Index (HI_{acute}), as shown below.

$$HI_{acute} = \sum_{i}^{n} HQ_i$$

Where:		**Value**
HI_{acute} =	Acute Hazard Index across all HAPs with similar acute effects (unitless)	*Calculated*
HQ_i =	Acute Hazard Quotients for individual HAP_i (unitless)	*Calculated*

As shown in **Table 5**, adverse acute effects that are common across HAPs include: 1) eye irritation (acetaldehyde, acrolein, formaldehyde, propylene oxide, toluene, and xylenes); 2) respiratory irritation (acetaldehyde, acrolein, propylene oxide, toluene, xylenes, cadmium, and chromium); 3) CNS effects (ethylbenzene, hexane, toluene, and xylenes); 4) immune system effects (benzene and nickel); and 5) developmental effects (1,3-butadiene, benzene and propylene oxide). Therefore, acute HIs will be estimated for these endpoints by summing individual HAP HQs based on these effects.

3.1.4 Context for Interpreting Risk Assessment Results

EPA has established a target cancer risk range of 1-in-1 million (1E-06) to 1-in-10 thousand (1E-04) within which it strives to manage long-term risk from environmental exposures.[33] EPA often strives to manage risk from environmental exposure by limiting the cancer risk from individual HAPs from a single source (i.e., via a single exposure pathway from a single facility) to 1-in-1 million (1E-06) and limiting aggregate risk (risk from a single HAP from a single facility summed across multiple exposure pathways) and cumulative risk (i.e., risk summed across multiple HAPs from all possible sources) to 1-in-10 thousand (1E-04). The EPA Region 6 Risk Management Addendum,[34] a companion document to the HHRAP, recommends reducing the upper-bound target risk from 1-in-10 thousand (1E-04) to 1-in-100 thousand

[33] EPA. 1990. U.S. Environmental Protection Agency. National Contingency Plan. Federal Register Volume 55, Number 46. March 8.

[34] EPA. 1998. Region 6 Risk Management Addendum – Draft Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities. EPA-R6-98-002. p. ADD-3. https://archive.epa.gov/region6/6pd/rcra_c/pd-o/web/pdf/r6add.pdf.

(1E-05) to account for exposure to background levels of air contaminants. Therefore, per the EPA Region 6 Risk Management Addendum, the RME risk associated with potential carcinogens released from a single facility should not exceed 1-in-100 thousand (1E-05). This 1-in-100 thousand risk level is ten times more stringent than the highest level that EPA deems acceptable (1E-04) and, therefore, represents a highly conservative risk management objective.

A risk of 1E-06 indicates a 1-in-1 million chance of developing cancer due to lifetime exposure to a particular substance. According to the American Cancer Society, the overall risk of developing cancer over a lifetime in the U.S. is 40.9%,[35] or approximately 1-in-2 chance for men (or 500,000-in-1 million chance), and 30.9%[36] or approximately 1-in-3 (or 333,333-in-1 million) chance for women.[37] Therefore, the range within which EPA manages risks posed by environmental exposures is very small by comparison to a person's typical background risk of developing cancer.

With regard to potential hazards posed by long-term exposure to non-carcinogenic HAPs, a HQ (HQ = EC/RfC or MRL) of less than or equal to 1 is generally considered protective of health.[38] Because they represent exposures that are likely to be without an appreciable risk of deleterious effects during a lifetime, if the EC_{nc} (non-cancer exposure concentration) is less than the RfC or MRL, no adverse health effects are expected. It is important to recognize, however, that an EC_{nc} that exceeds the RfC or MRL does not indicate that adverse health effects will occur, or that they should be expected. This is because RfCs and MRLs do not represent threshold exposures above which illness or disease is expected. They instead represent exposures below which such effects are NOT expected.[39]

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[35] The American Cancer Society lists the risk of developing cancer in men as 40.9% but rounds that up to a 1-in-2 chance of developing cancer, which would technically be a 50% risk.
[36] The American Cancer Society lists the risk of developing cancer in women as 31.9% but rounds that up to a 1-in-3 chance of developing cancer, which would technically be a 33.3% risk.
[37] American Cancer Society website. "Lifetime Risk of Developing or Dying From Cancer". https://www.cancer.org/cancer/risk-prevention/understanding-cancer-risk/lifetime-probability-of-developing-or-dying-from-cancer.html.
[38] EPA. 2005. U.S. Environmental Protection Agency. Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities. EPA530-R-05-006. p. 7-6. https://archive.epa.gov/epawaste/hazard/tsd/td/web/html/risk.html; ATSDR website. Calculating Hazard Quotients and Cancer Risk Estimates. https://www.atsdr.cdc.gov/pha-guidance/conducting_scientific_evaluations/epcs_and_exposure_calculations/hazardquotients_cancerrisk.html#:~:text=HQs%20less%20than%201%20indicate,in%2Ddepth%20toxicological%20effects%20analysis. Visited on June 20, 2023.
[39] EPA website. Basic Information about the Integrated Risk Information System. https://www.epa.gov/iris/basic-information-about-integrated-risk-information-system. Visited on June 20, 2023; EPA. 2005. U.S. Environmental Protection Agency. Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities. EPA530-R-05-006. p. 7-6. https://archive.epa.gov/epawaste/hazard/tsd/td/web/html/risk.html.

In developing toxicity factors for non-carcinogenic effects, the upper bound tolerance range is identified. Because variability exists in the human population, attempts are made to identify a sub-threshold level protective of sensitive individuals in the population. One way in which sub-threshold levels are established is through the application of uncertainty factors to the underlying toxicity data. Therefore, an HQ above one is not necessarily indicative of health impacts because of the application of these uncertainty factors to the underlying data and subsequent ratcheting down of the adverse effect levels in deriving the RfCs.[40] Similar logic applies to short-term exposures. Ca RELs are concentrations in air at or below which no serious adverse health effects are anticipated in the general population from intermittent (i.e., 1-hour) exposures[41] Therefore, if an EC_{acute} exceeds the CA REL, it does not necessarily mean that there is cause for concern. However, AEGL-1 values represent air concentrations at which notable discomfort, irritation, or certain asymptomatic non-sensory effects may occur, although the effects are not disabling and are transient and reversible upon cessation of exposure. Therefore, an EC_{acute} that exceeds an AEGL-1 could cause mild/transient irritation.[42]

Because the agencies tasked with setting these limits (e.g., U.S. and California EPA, ATSDR) are tasked with protecting human health and the environment, these toxicity factors are set at very conservative (highly health protective) levels. Therefore, a risk or hazard estimate that exceeds a target value should trigger more careful consideration of the underlying scientific basis for the calculation. It does not automatically mean that it is not safe or that it presents an unacceptable risk.[43]

3.2 Human Health Risk Assessment Results

Input variables for the risk calculations are provided in Appendix A. Back-up calculations for chronic cancer risks, chronic non-cancer hazards, and acute hazards are provided in Appendices B, C, and D, respectively.

3.2.1 Chronic Cancer Risks

Estimated inhalation cancer risks are provided in **Table 6**. As shown in **Table 6**, no individual HAP has an estimated adult or child Resident inhalation cancer risk above EPA's lower-bound target risk of 1-in-1

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[40] EPA. 1989. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part A). EPA/540/1-89/002. p. 7-6. https://www.epa.gov/risk/risk-assessment-guidance-superfund-rags-part.

[41] OEHHA. 2015. California Environmental Protection Agency Office of Environmental Health Hazard Assessment. Air Toxics Hot Spots Program. Risk Assessment Guidelines. February 2015. p. 6-3.

[42] EPA website. About Acute Exposure Guideline Levels (AEGLs). https://www.epa.gov/aegl/about-acute-exposure-guideline-levels-aegls. Visited on June 20, 2023.

[43] EPA. 2005. U.S. Environmental Protection Agency. Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities. EPA530-R-05-006. p. 7-10. https://archive.epa.gov/epawaste/hazard/tsd/td/web/html/risk.html.

Table 6
Estimated Chronic Inhalation Cancer Risks

Pollutant	Adult Chronic Carcinogen Exposure Concentration ($\mu g/m^3$)	Child Chronic Carcinogen Exposure Concentration ($\mu g/m^3$)	IURF $(\mu g/m^3)^{-1}$	Adult Cancer Risk	Child Cancer Risk
1,3-Butadiene	6.16E-05	1.23E-05	3.00E-05	1.85E-09	3.70E-10
1,4-Dichlorobenzene	8.22E-06	1.64E-06	NA	NA	NA
Acetaldehyde	7.97E-04	1.59E-04	2.20E-06	1.75E-09	3.51E-10
Acrolein	1.44E-04	2.88E-05	NA	NA	NA
Benzene	1.46E-03	2.92E-04	7.80E-06	1.14E-08	2.28E-09
Ethylbenzene	5.88E-04	1.18E-04	NA	NA	NA
Formaldehyde	2.98E-03	5.97E-04	1.30E-05	3.88E-08	7.76E-09
Hexane	1.25E-02	2.50E-03	NA	NA	NA
Acenaphthene	2.97E-06	5.95E-07	NA	NA	NA
Acenaphthylene	6.90E-06	1.38E-06	NA	NA	NA
Anthracene	2.01E-05	4.02E-06	NA	NA	NA
Benz[a]Anthracene	1.09E-07	2.19E-08	6.00E-05	6.56E-12	1.31E-12
Benzo[a]Pyrene	5.18E-08	1.04E-08	6.00E-04	3.11E-11	6.22E-12
Benzo[b]Fluoranthene	1.04E-07	2.07E-08	6.00E-05	6.21E-12	1.24E-12
Benzo[k]Fluoranthene	5.18E-08	1.04E-08	6.00E-06	3.11E-13	6.22E-14
Benzo[g,h,i]Fluoranthene	1.64E-06	3.27E-07	NA	NA	NA
Chrysene	2.02E-07	4.04E-08	6.00E-07	1.21E-13	2.42E-14
Dibenzo[a,h]Anthracene	1.07E-07	2.14E-08	6.00E-04	6.43E-11	1.29E-11
Fluoranthene	1.11E-06	2.22E-07	NA	NA	NA
Fluorene	9.58E-06	1.92E-06	NA	NA	NA
Indeno[1,2,3-cd]Pyrene	1.04E-07	2.07E-08	6.00E-05	6.21E-12	1.24E-12
Naphthalene	1.83E-03	3.66E-04	NA	NA	NA
Phenanthrene	7.92E-05	1.58E-05	NA	NA	NA
Pyrene	4.18E-07	8.35E-08	NA	NA	NA
Propylene Oxide	5.01E-04	1.00E-04	3.70E-06	1.86E-09	3.71E-10
Toluene	2.33E-03	4.65E-04	NA	NA	NA
Xylenes	1.26E-03	2.52E-04	NA	NA	NA
Cadmium	8.22E-06	1.64E-06	1.80E-03	1.48E-08	2.96E-09
Chromium	8.22E-06	1.64E-06	1.20E-02	9.86E-08	1.97E-08
Nickel	1.23E-05	2.47E-06	4.80E-04	5.92E-09	1.18E-09
Total PAH Cancer Risk				**1.15E-10**	**2.30E-11**
Total Cancer Risk				**1.75E-07**	**3.50E-08**

EC_c – Chronic carcinogen exposure concentration
IURF – Inhalation Unit Risk Factor
Total Cancer Risk – Cancer risk obtained by summing inhalation cancer risk for each individual HAP.

million (1E-06). Moreover, the total inhalation cancer risks summed across all HAPs (adult Resident = 1.75E-07, child Resident = 3.5E-08) are well below (by almost 100-fold) the EPA Region 6 Risk Management

Addendum[44] target of 1-in-100 thousand (1E-05) for a single facility.[45] This 1-in-100 thousand individual facility risk management objective is ten times more stringent than the highest cancer risk that EPA deems acceptable (1E-04) to account for potential exposure to background levels of air contaminants (i.e., cumulative risk) and, therefore, represents a highly conservative target risk. Moreover, summing cancer risk (even for a single exposure pathway) across all carcinogenic HAPs is an extremely conservative approach (i.e., health protective) and in all likelihood substantially overestimates total risk from a particular source because: 1) maximum modeled annual concentrations for all HAPs are not co-located (i.e., exposure to them does not occur simultaneously); 2) cancer risks estimated for individual HAPs are upper-bound estimates that when summed, become highly improbable as estimates of overall risk; and 3) cancers that occur at different sites within the body, or with different cellular origin, likely have independent mechanisms of causation and are, therefore, not necessarily additive.[46]

3.2.2 Chronic Non-Cancer Hazards

Estimated HAP-specific chronic inhalation HQ ($HQ_{chronic}$) values and total chronic inhalation HI ($HI_{chronic}$) (summed across HAPs with similar chronic effects) are provided in **Table 7**.

As shown in **Table 7**, no $HQ_{chronic}$ for any HAP is greater than 1. In addition, all segregated $HI_{chronic}$ values (derived by summing $HQ_{chronic}$ values for all HAPs with similar chronic effects) are also well below 1 (by almost 100-fold). It should be noted that summing chronic inhalation HQ values across HAPs, even those that have similar effects or that affect the same target organ, is a conservative (i.e., health protective) approach that likely overestimates non-cancer hazard because: 1) maximum modeled annual concentrations for all HAPs are not co-located (i.e., simultaneous exposure does not necessarily occur); and 2) HQs estimated for individual HAPs are upper-bound estimates that when summed, become highly improbable as estimates of overall $HI_{chronic}$; and 3) non-cancer effects such as developmental toxicity affect different body parts, have different cellular origins, and likely have independent mechanisms of action, which means that they are not necessarily additive.

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[44] EPA. 1998. Region 6 Risk Management Addendum – Draft Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities. EPA-R6-98-002. p. ADD-3. https://archive.epa.gov/region6/6pd/rcra_c/pd-o/web/pdf/r6add.pdf.

[45] EPA. 1998. Region 6 Risk Management Addendum – Draft Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities. EPA-R6-98-002. p. ADD-3. https://archive.epa.gov/region6/6pd/rcra_c/pd-o/web/pdf/r6add.pdf.

[46] Salmon, A. G., & Roth, L. A. 2010. Cancer risk based on an individual tumor type or summing of tumors. Cancer Risk Assessment: Chemical Carcinogenesis, Hazard Evaluation, and Risk Quantification, 716-735.

Table 7
Estimated Chronic Inhalation Non-Cancer Hazards

Pollutant	Adult Chronic Non-Cancer Exposure Concentration ($\mu g/m^3$)	Child Chronic Non-Cancer Exposure Concentration ($\mu g/m^3$)	Non-Cancer RfC/MRL ($\mu g/m^3$)	Adult Non-Cancer $HQ_{chronic}$	Child Non-Cancer $HQ_{chronic}$
1,3-Butadiene	1.44E-04	1.44E-04	2.00E+00	7.19E-05	7.19E-05
1,4-Dichlorobenzene	1.92E-05	1.92E-05	8.00E+02	2.40E-08	2.40E-08
Acetaldehyde	1.86E-03	1.86E-03	9.00E+00	2.07E-04	2.07E-04
Acrolein	3.36E-04	3.36E-04	2.00E-02	1.68E-02	1.68E-02
Benzene	3.40E-03	3.40E-03	3.00E+01	1.13E-04	1.13E-04
Ethylbenzene	1.37E-03	1.37E-03	1.00E+03	1.37E-06	1.37E-06
Formaldehyde	6.96E-03	6.96E-03	9.80E+00	7.10E-04	7.10E-04
Hexane	2.91E-02	2.91E-02	7.00E+02	4.16E-05	4.16E-05
Acenaphthene	6.94E-06	6.94E-06	NA	NA	NA
Acenaphthylene	1.61E-05	1.61E-05	NA	NA	NA
Anthracene	4.69E-05	4.69E-05	NA	NA	NA
Benz[a]Anthracene	2.55E-07	2.55E-07	NA	NA	NA
Benzo[a]Pyrene	1.21E-07	1.21E-07	2.00E-03	6.05E-05	6.05E-05
Benzo[b]Fluoranthene	2.42E-07	2.42E-07	NA	NA	NA
Benzo[k]Fluoranthene	1.21E-07	1.21E-07	NA	NA	NA
Benzo[g,h,i]Fluoranthene	3.82E-06	3.82E-06	NA	NA	NA
Chrysene	4.71E-07	4.71E-07	NA	NA	NA
Dibenzo[a,h]Anthracene	2.50E-07	2.50E-07	NA	NA	NA
Fluoranthene	2.59E-06	2.59E-06	NA	NA	NA
Fluorene	2.24E-05	2.24E-05	NA	NA	NA
Indeno[1,2,3-cd]Pyrene	2.42E-07	2.42E-07	NA	NA	NA
Naphthalene	4.27E-03	4.27E-03	3.00E+00	1.42E-03	1.42E-03
Phenanthrene	1.85E-04	1.85E-04	NA	NA	NA
Pyrene	9.75E-07	9.75E-07	NA	NA	NA
Propylene Oxide	1.17E-03	1.17E-03	3.00E+01	3.90E-05	3.90E-05
Toluene	5.43E-03	5.43E-03	5.00E+03	1.09E-06	1.09E-06
Xylenes	2.93E-03	2.93E-03	1.00E+02	2.93E-05	2.93E-05
Cadmium	1.92E-05	1.92E-05	NA	NA	NA
Chromium	1.92E-05	1.92E-05	8.00E-03	2.40E-03	2.40E-03
Nickel	2.88E-05	2.88E-05	9.00E-02	3.20E-04	3.20E-04
Nasal HI				**2.16E-02**	**2.16E-02**
CNS HI				**3.04E-05**	**3.04E-05**
Developmental HI				**6.18E-05**	**6.18E-05**

CNS HI – –Segregated Hazard Index obtained by summing inhalation HQs for all HAPs that affect the Central Nervous System. HAPS for which HQs were summed include toluene and xylenes.
Developmental HI – – Segregated Hazard Index obtained by summing inhalation HQs for all HAPs that cause developmental toxicity. HAPs for which HQs were summed include ethylbenzene and PAHs.
EC_{nc} – Chronic non-cancer exposure concentration.

MRL – Minimal Risk Level
Nasal HI – Segregated Hazard Index obtained by summing inhalation HQs for all HAPs that affect the nasal epithelium. HAPs for which HQs were summed include acetaldehyde, acrolein, formaldehyde, naphthalene, propylene oxide, and chromium.
RfC – Reference Concentration.

3.2.3 Acute Hazards

Estimated HAP-specific acute inhalation HQ values and total inhalation HI (summed across HAPs with similar effects) are provided in **Table 8**.

As shown in **Table 8**, all individual HAP acute HQ (HQ_{acute}) values and segregated acute HI (HI_{acute}) values, which are derived by summing HQ values for all HAPs with similar acute effects, are well below 1 (by almost 100-fold). Summing acute inhalation HQ values across HAPs, even those that have similar effects, is a conservative (i.e., health protective) approach that likely overestimates acute hazard because while maximum modeled hourly concentrations for some HAPs may occur at the same location, they do not all occur at the same location (i.e., exposure to them does not occur simultaneously).

Table 8
Estimated Acute Inhalation Hazards

Pollutant	Hourly Concentration ($\mu g/m^3$)	Acute Toxicity Factor ($\mu g/m^3$)	Adult/Child HQ_{acute}
1,3-Butadiene	7.90E-03	6.60E+02	1.20E-05
1,4-Dichlorobenzene	1.09E-03	NA	NA
Acetaldehyde	1.04E-01	4.70E+02	2.22E-04
Acrolein	1.67E-02	2.50E+00	6.69E-03
Benzene	4.53E+00	2.70E+01	1.68E-01
Ethylbenzene	8.97E-02	1.43E+05	6.26E-07
Formaldehyde	3.33E-01	5.50E+01	6.05E-03
Hexane	1.08E+01	1.00E+04	1.08E-03
Acenaphthene	3.97E-04	NA	NA
Acenaphthylene	9.20E-04	NA	NA
Anthracene	2.68E-03	NA	NA
Benz[a]Anthracene	1.47E-05	NA	NA
Benzo[a]Pyrene	6.96E-06	NA	NA
Benzo[b]Fluoranthene	1.39E-05	NA	NA
Benzo[k]Fluoranthene	6.96E-06	NA	NA
Benzo[g,h,i]Fluoranthene	2.20E-04	NA	NA
Chrysene	2.71E-05	NA	NA
Dibenzo[a,h]Anthracene	1.44E-05	NA	NA
Fluoranthene	1.49E-04	NA	NA
Fluorene	1.28E-03	NA	NA
Indeno[1,2,3-cd]Pyrene	1.39E-05	NA	NA
Naphthalene	2.44E-01	NA	NA
Phenanthrene	1.06E-02	NA	NA
Pyrene	5.61E-05	NA	NA
Propylene Oxide	7.57E-02	3.10E+03	2.44E-05
Toluene	6.71E-01	5.00E+03	1.34E-04
Xylenes	3.59E-01	2.20E+04	1.63E-05
Cadmium	1.00E-03	4.60E+02	2.17E-06
Chromium	1.27E-03	2.00E-01	6.35E-03
Nickel	1.90E-03	2.00E-01	9.50E-03
Eye Irritation HI			**1.31E-02**
Respiratory Irritation HI			**1.34E-02**
CNS HI			**1.23E-03**
Immune System HI			**1.77E-01**
Developmental HI			**1.68E-01**

CNS HI – – Hazard Index obtained by summing inhalation HQs for all HAPs that affect the Central Nervous System. HAPs for which HQs were summed include ethylbenzene, hexane, toluene, and xylenes.
Developmental HI – – Hazard Index obtained by summing inhalation HQs for all HAPs that cause developmental toxicity. HAPs for which HQs were summed include 1,3-butadiene, benzene, and propylene oxide.
Eye Irritation HI – Hazard Index obtained by summing inhalation HQs for all HAPs that cause eye irritation. HAPs for which HQs were summed include acetaldehyde, acrolein, formaldehyde, propylene oxide, toluene, and xylenes.
EC_{acute} – Acute exposure concentration

Immune system HI – Hazard Index obtained by summing inhalation HQs for all HAPs that affect the immune system. HAPs for which HQs were summed include benzene and nickel.
Respiratory Irritation HI – Hazard Index obtained by summing inhalation HQs for all HAPs that cause respiratory irritation. HAPs for which HQs were summed include acetaldehyde, acrolein, propylene oxide, toluene, xylenes, cadmium, and chromium.

Section 4
Summary and Conclusions

4.1 Summary

Chronic inhalation cancer risks, non-cancer hazards, and acute hazards estimated in this HHRA are summarized in **Table 9**.

As shown in **Table 9,** the estimated adult and child Resident inhalation cancer risk for each HAP is at least an order of magnitude (i.e., 10x) below EPA's risk management objective of 1-in-1 million (1E-06) for individual HAPs. Moreover, the total inhalation cancer risks summed across all HAPs are well below (by almost 100-fold) EPA's target of 1-in-100 thousand (1E-05) for a single facility.[47] As previously discussed, this 1-in-100 thousand individual facility risk management objective is ten times more stringent than the highest cancer risk that EPA deems acceptable to account for potential exposure to background levels of air contaminants. Therefore, this facility risk management objective is intended to address the potential for cumulative risks (i.e., risks associated with multiple pollutants and other sources in the area).

Table 9 also indicates that no inhalation $HQ_{chronic}$ value for any HAP is greater than the non-cancer risk management objective of 1 for individual HAPs. In addition, all segregated inhalation $HI_{chronic}$ values (derived by summing $HQ_{chronic}$ values for all HAPs with similar chronic effects) are well below 1 (by almost 100-fold). Similarly, all inhalation HQ_{acute} and segregated inhalation HI_{acute} values are well below the acute risk management objective of 1 (by almost 100-fold).

It is important to recognize that the inhalation cancer risks, $HQ_{chronic}$ values and HQ_{acute} values for the adult and child Resident in this HHRA were estimated at the maximum impacted off-property location for each HAP, not necessarily at occupied residences. In addition, summing inhalation cancer risk across all carcinogenic HAPs is an extremely conservative approach (i.e., health protective) that is likely to substantially overestimate total cancer risk from a particular source.[48] Likewise, summing inhalation $HQ_{chronic}$ values or HQ_{acute} values across HAPs, even those that have similar effects, is highly conservative and likely overestimates chronic and acute hazard.

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[47] EPA. 1998. Region 6 Risk Management Addendum – Draft Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities. EPA-R6-98-002. p. ADD-3. https://archive.epa.gov/region6/6pd/rcra_c/pd-o/web/pdf/r6add.pdf.

[48] Salmon, A. G., & Roth, L. A. 2010. Cancer risk based on an individual tumor type or summing of tumors. Cancer Risk Assessment: Chemical Carcinogenesis, Hazard Evaluation, and Risk Quantification, 716-735.

Table 9
Inhalation Risk and Hazard Summary

Pollutant	Adult Cancer Risks	Child Cancer Risks	Adult Non-Cancer $HQ_{chronic}$	Child Non-Cancer $HQ_{chronic}$	Adult/Child HQ_{acute}
1,3-Butadiene	1.85E-09	3.70E-10	7.19E-05	7.19E-05	1.20E-05
1,4-Dichlorobenzene	NA	NA	2.40E-08	2.40E-08	NA
Acetaldehyde	1.75E-09	3.51E-10	2.07E-04	2.07E-04	2.22E-04
Acrolein	NA	NA	1.68E-02	1.68E-02	6.69E-03
Benzene	1.14E-08	2.28E-09	1.13E-04	1.13E-04	1.68E-01
Ethylbenzene	NA	NA	1.37E-06	1.37E-06	6.26E-07
Formaldehyde	3.88E-08	7.76E-09	7.10E-04	7.10E-04	6.05E-03
Hexane	NA	NA	4.16E-05	4.16E-05	1.08E-03
Acenaphthene	NA	NA	NA	NA	NA
Acenaphthylene	NA	NA	NA	NA	NA
Anthracene	NA	NA	NA	NA	NA
Benz[a]Anthracene	6.56E-12	1.31E-12	NA	NA	NA
Benzo[a]Pyrene	3.11E-11	6.22E-12	6.05E-05	6.05E-05	NA
Benzo[b]Fluoranthene	6.21E-12	1.24E-12	NA	NA	NA
Benzo[k]Fluoranthene	3.11E-13	6.22E-14	NA	NA	NA
Benzo[g,h,i]Fluoranthene	NA	NA	NA	NA	NA
Chrysene	1.21E-13	2.42E-14	NA	NA	NA
Dibenzo[a,h]Anthracene	6.43E-11	1.29E-11	NA	NA	NA
Fluoranthene	NA	NA	NA	NA	NA
Fluorene	NA	NA	NA	NA	NA
Indeno[1,2,3-cd]Pyrene	6.21E-12	1.24E-12	NA	NA	NA
Naphthalene	NA	NA	1.42E-03	1.42E-03	NA
Phenanthrene	NA	NA	NA	NA	NA
Pyrene	NA	NA	NA	NA	NA
Propylene Oxide	1.86E-09	3.71E-10	3.90E-05	3.90E-05	2.44E-05
Toluene	NA	NA	1.09E-06	1.09E-06	1.34E-04
Xylenes	NA	NA	2.93E-05	2.93E-05	1.63E-05
Cadmium	1.48E-08	2.96E-09	NA	NA	2.17E-06
Chromium	9.86E-08	1.97E-08	2.40E-03	2.40E-03	6.35E-03
Nickel	5.92E-09	1.18E-09	3.20E-04	3.20E-04	9.50E-03
TOTAL CANCER RISK	**1.75E-07**	**3.50E-08**			
Nasal HI			**2.16E-02**	**2.16E-02**	
CNS HI			**3.04E-05**	**3.04E-05**	**1.23E-03**
Developmental HI			**6.18E-05**	**6.18E-05**	**1.68E-01**
Eye Irritation HI					**1.31E-02**
Respiratory Irritation HI					**1.34E-02**
Immune System HI					**1.77E-01**

CNS HI – Hazard Index obtained by summing HQs for all HAPs that affect the Central Nervous System.
Developmental HI – – Hazard Index obtained by summing HQs for all HAPs that cause developmental toxicity.
Eye Irritation HI – Hazard Index obtained by summing HQs for all HAPs that cause eye irritation.
EC_{acute} – Acute exposure concentration
Immune System HI – – Hazard Index obtained by summing HQs for all HAPs that adversely affect the immune system.
Nasal HI – Hazard Index obtained by summing HQs for all HAPs that cause nasal epithelial toxicity.
Respiratory Irritation HI – Hazard Index obtained by summing HQs for all HAPs that cause respiratory irritation.

4.2 Conclusions

This HHRA demonstrates that estimated inhalation cancer risks and non-cancer hazards, as well as short-term acute hazards, potentially associated with long-term (annual) or short-term (hourly) emissions of HAPs from the CP2 LNG Terminal and associated mobile marine sources are well below levels deemed acceptable by EPA. Based on this information, it is concluded that there is no need for concern about health effects potentially associated with exposures to these emissions, even from a cumulative risk perspective, for the following reasons:

- The hypothetical adult and child Resident evaluated in this HHRA were assumed to be continuously exposed to outdoor air (24-hours/day, 7 days/week) for six (child) or 30 (adult) years.
    - These exposure assumptions grossly exaggerate exposure because people:
        - spend 85 to 90% of their time indoors[49] and the modeled concentrations that serve as the basis for this HHRA are in outdoor air (concentrations indoors will be less);
        - do not spend 24 hours/day, 7 days/week at home; and
        - few families live in the same residence for 30 years.

- Cancer risks, $HQ_{chronic}$ and HQ_{acute} values for the adult and child Resident in this HHRA were estimated at the maximum impacted off-property location for each HAP, not necessarily at occupied residences.

- The inhalation cancer risk for each HAP is at least an order of magnitude (i.e., 10x) below EPA's most stringent risk management objective.

- The total inhalation cancer risk (summed across all HAPs) is almost 100-fold lower than the individual facility risk management objective and almost 1,000-fold lower than EPA's upper-bound target cancer risk. Summing cancer risks across individual HAPs overstates risk because:
    - Maximum modeled annual concentrations for all HAPs are not co-located;
    - Estimated cancer risks for individual HAPs are upper-bound estimates and as the number of HAPs increases, it becomes increasingly improbable that their summation represents a valid estimate of overall risk; and

[49] Klepeis, N. E., Nelson, W. C., Ott, W. R., Robinson, J. P., Tsang, A. M., Switzer, P., ... & Engelmann, W. H. (2001). The National Human Activity Pattern Survey (NHAPS): a resource for assessing exposure to environmental pollutants. Journal of Exposure Science & Environmental Epidemiology, 11(3), 231-252.

- Cancers that occur at different sites within the body, or with different cellular origins likely have independent mechanisms of causation and are, therefore, not necessarily additive.

- All inhalation $HQ_{chronic}$ values for individual HAPs are at least two orders of magnitude (i.e., 100x) below EPA's non-cancer risk management objective.

- All segregated inhalation $HI_{chronic}$ values (derived by summing $HQ_{chronic}$ values for all HAPs with similar chronic effects) are also well below EPA's non-cancer risk management objective. Summing $HQ_{chronic}$ values for individual HAPs overestimates chronic hazard because:

    - Maximum modeled annual concentrations for all HAPs are not co-located;

    - Estimated $HQ_{chronic}$ values for individual HAPs are upper-bound estimates and as the number of HAPs increases, it becomes increasingly improbable that their summation represents a valid estimate of $HI_{chronic}$; and

    - Non-cancer effects such as developmental toxicity affect different body parts, have different cellular origins, and likely have independent mechanisms of action, which means that they are not necessarily additive.

- All inhalation HQ_{acute} and segregated inhalation HI_{acute} values are well below EPA's acute risk management objective (by almost 100-fold). Summing HQ_{acute} values for individual HAPs, even for those with similar effects, overestimates acute hazard because maximum modeled hourly concentrations for all HAPs are not co-located (i.e., exposure does not necessarily occur simultaneously).

APPENDICES

Appendix A
Risk Assessment Input Values

Risk Assessment Input Values

Pollutant	Annual Concentration ($\mu g/m^3$)	Hourly Concentration ($\mu g/m^3$)	Cancer IURF $(\mu g/m^3)^{-1}$	Ref.	Non-Cancer RfC ($\mu g/m^3$)	Effect	Ref.
1,3-Butadiene	1.50E-04	7.90E-03	3.00E-05	IRIS	2.00E+00	ovarian atrophy	IRIS
1,4-Dichlorobenzene	2.00E-05	1.09E-03	NA	NA	8.00E+02	liver/repro	IRIS
Acetaldehyde	1.94E-03	1.04E-01	2.20E-06	IRIS	9.00E+00	degen olfactory epithelium	IRIS
Acrolein	3.50E-04	1.67E-02	NA	NA	2.00E-02	nasal lesions	IRIS
Benzene	3.55E-03	4.53E+00	7.80E-06	IRIS	3.00E+01	decreased lymphocyte	IRIS
Ethylbenzene	1.43E-03	8.97E-02	NA	NA	1.00E+03	developmental	IRIS
Formaldehyde	7.26E-03	3.33E-01	1.30E-05	IRIS	9.80E+00	histological changes in nasal epithelium	ATSDR
Hexane	3.04E-02	1.08E+01	NA	NA	7.00E+02	peripheral neuropathy	IRIS
Acenaphthene	7.24E-06	3.97E-04	NA	NA	NA	NA	NA
Acenaphthylene	1.68E-05	9.20E-04	NA	NA	NA	NA	NA
Anthracene	4.89E-05	2.68E-03	NA	NA	NA	NA	NA
Benz[a]Anthracene	2.66E-07	1.47E-05	6.00E-05	RPF	NA	NA	NA
Benzo[a]Pyrene	1.26E-07	6.96E-06	6.00E-04	IRIS	2.00E-03	developmental	IRIS
Benzo[b]Fluoranthene	2.52E-07	1.39E-05	6.00E-05	RPF	NA	NA	NA
Benzo[k]Fluoranthene	1.26E-07	6.96E-06	6.00E-06	RPF	NA	NA	NA
Benzo[g,h,i]Fluoranthene	3.98E-06	2.20E-04	NA	NA	NA	NA	NA
Chrysene	4.92E-07	2.71E-05	6.00E-07	RPF	NA	NA	NA
Dibenzo[a,h]Anthracene	2.61E-07	1.44E-05	6.00E-04	RPF	NA	NA	NA
Fluoranthene	2.71E-06	1.49E-04	NA	NA	NA	NA	NA

Risk Assessment Input Values

Pollutant	Annual Concentration ($\mu g/m^3$)	Hourly Concentration ($\mu g/m^3$)	Cancer IURF $(\mu g/m^3)^{-1}$	Ref.	Non-Cancer RfC ($\mu g/m^3$)	Effect	Ref.
Flourene	2.33E-05	1.28E-03	NA	NA	NA	NA	NA
Indeno[1,2,3-cd]Pyrene	2.52E-07	1.39E-05	6.00E-05	RPF	NA	NA	NA
Naphthalene	4.45E-03	2.44E-01	NA	NA	3.00E+00	hyperplasia resp/nasal epithelium	IRIS
Phenanthrene	1.93E-04	1.06E-02	NA	NA	NA	NA	NA
Pyrene	1.02E-06	5.61E-05	NA	NA	NA	NA	NA
Propylene Oxide	1.22E-03	7.57E-02	3.70E-06	IRIS	3.00E+01	nasal epithelial infolds	IRIS
Toluene	5.66E-03	6.71E-01	NA	NA	5.00E+03	CNS	IRIS
Xylenes	3.06E-03	3.59E-01	NA	NA	1.00E+02	CNS	IRIS
Cadmium	2.00E-05	1.00E-03	1.80E-03	IRIS	NA	NA	NA
Chromium	2.00E-05	1.27E-03	1.20E-02	IRIS	8.00E-03	nasal septum atrophy	IRIS
Nickel	3.00E-05	1.90E-03	4.80E-04	IRIS	9.00E-02	lung inflammation	IRIS

Risk Assessment Input Values

Pollutant	Acute Toxicity Criterion (µg/m^3)	Effect	Ref.	Exceeds Long-Term Toxicity Criterion		Exceeds Acute Toxicity Criterion
				Cancer	Non-Cancer	
1,3-Butadiene	6.60E+02	develop	Ca REL	NO	NO	NO
1,4-Dichlorobenzene	NA	NA	NA	NO	NO	NO
Acetaldehyde	4.70E+02	eye/resp irritation	Ca REL	NO	NO	NO
Acrolein	2.50E+00	eye/resp irritation	Ca REL	NO	NO	NO
Benzene	2.70E+01	developmental, immune system, hematological effects	Ca REL	NO	NO	NO
Ethylbenzene	1.43E+05	CNS	AEGL-1 (Interim)	NO	NO	NO
Formaldehyde	5.50E+01	eye irritation	Ca REL	NO	NO	NO
Hexane	1.00E+04	CNS	AEGL-2	NO	NO	NO
Acenaphthene	NA	NA	NA	NO	NO	NO
Acenaphthylene	NA	NA	NA	NO	NO	NO
Anthracene	NA	NA	NA	NO	NO	NO
Benz[a]Anthracene	NA	NA	NA	NO	NO	NO
Benzo[a]Pyrene	NA	NA	NA	NO	NO	NO
Benzo[b]Fluoranthene	NA	NA	NA	NO	NO	NO
Benzo[k]Fluoranthene	NA	NA	NA	NO	NO	NO
Benzo[g,h,i]Fluoranthene	NA	NA	NA	NO	NO	NO
Chrysene	NA	NA	NA	NO	NO	NO
Dibenzo[a,h]Anthracene	NA	NA	NA	NO	NO	NO
Fluoranthene	NA	NA	NA	NO	NO	NO

Risk Assessment Input Values

Pollutant	Acute Toxicity Criterion (µg/m^3)	Effect	Ref.	Exceeds Long-Term Toxicity Criterion		Exceeds Acute Toxicity Criterion
				Cancer	Non-Cancer	
Flourene	NA	NA	NA	NO	NO	NO
Indeno[1,2,3-cd]Pyrene	NA	NA	NA	NO	NO	NO
Naphthalene	NA	NA	NA	NO	NO	NO
Phenanthrene	NA	NA	NA	NO	NO	NO
Pyrene	NA	NA	NA	NO	NO	NO
Propylene Oxide	3.10E+03	eye/resp irritation, developmental	Ca REL	NO	NO	NO
Toluene	5.00E+03	eye/resp irritation, CNS	Ca REL	NO	NO	NO
Xylenes	2.20E+04	eye/resp irritation, CNS	Ca REL	NO	NO	NO
Cadmium	4.60E+02	resp irritation	AEGL-1 (Interim)	NO	NO	NO
Chromium	2.00E-01	resp irritation	Ca REL	NO	NO	NO
Nickel	2.00E-01	immune	Ca REL	NO	NO	NO

Appendix B
Cancer Risk Backup Calculations

Cancer Risk Estimates

Pollutant	Annual Concentration ($\mu g/m^3$)	Adult Chronic Carcinogen Exposure Concentration ($\mu g/m^3$)	Child Chronic Carcinogen Exposure Concentration ($\mu g/m^3$)	IURF $(\mu g/m^3)^{-1}$	Adult Cancer Risk	Child Cancer Risk
1,3-Butadiene	1.50E-04	6.16E-05	1.23E-05	3.00E-05	1.85E-09	3.70E-10
1,4-Dichlorobenzene	2.00E-05	8.22E-06	1.64E-06	NA	NA	NA
Acetaldehyde	1.94E-03	7.97E-04	1.59E-04	2.20E-06	1.75E-09	3.51E-10
Acrolein	3.50E-04	1.44E-04	2.88E-05	NA	NA	NA
Benzene	3.55E-03	1.46E-03	2.92E-04	7.80E-06	1.14E-08	2.28E-09
Ethylbenzene	1.43E-03	5.88E-04	1.18E-04	NA	NA	NA
Formaldehyde	7.26E-03	2.98E-03	5.97E-04	1.30E-05	3.88E-08	7.76E-09
Hexane	3.04E-02	1.25E-02	2.50E-03	NA	NA	NA
Acenaphthene	7.24E-06	2.97E-06	5.95E-07	NA	NA	NA
Acenaphthylene	1.68E-05	6.90E-06	1.38E-06	NA	NA	NA
Anthracene	4.89E-05	2.01E-05	4.02E-06	NA	NA	NA
Benz[a]Anthracene	2.66E-07	1.09E-07	2.19E-08	6.00E-05	6.56E-12	1.31E-12
Benzo[a]Pyrene	1.26E-07	5.18E-08	1.04E-08	6.00E-04	3.11E-11	6.22E-12
Benzo[b]Fluoranthene	2.52E-07	1.04E-07	2.07E-08	6.00E-05	6.21E-12	1.24E-12
Benzo[k]Fluoranthene	1.26E-07	5.18E-08	1.04E-08	6.00E-06	3.11E-13	6.22E-14
Benzo[g,h,i]Fluoranthene	3.98E-06	1.64E-06	3.27E-07	NA	NA	NA
Chrysene	4.92E-07	2.02E-07	4.04E-08	6.00E-07	1.21E-13	2.42E-14
Dibenzo[a,h]Anthracene	2.61E-07	1.07E-07	2.14E-08	6.00E-04	6.43E-11	1.29E-11
Fluoranthene	2.71E-06	1.11E-06	2.22E-07	NA	NA	NA
Flourene	2.33E-05	9.58E-06	1.92E-06	NA	NA	NA
Indeno[1,2,3-cd]Pyrene	2.52E-07	1.04E-07	2.07E-08	6.00E-05	6.21E-12	1.24E-12
Naphthalene	4.45E-03	1.83E-03	3.66E-04	NA	NA	NA
Phenanthrene	1.93E-04	7.92E-05	1.58E-05	NA	NA	NA
Pyrene	1.02E-06	4.18E-07	8.35E-08	NA	NA	NA
Propylene Oxide	1.22E-03	5.01E-04	1.00E-04	3.70E-06	1.86E-09	3.71E-10
Toluene	5.66E-03	2.33E-03	4.65E-04	NA	NA	NA
Xylenes	3.06E-03	1.26E-03	2.52E-04	NA	NA	NA
Cadmium	2.00E-05	8.22E-06	1.64E-06	1.80E-03	1.48E-08	2.96E-09
Chromium	2.00E-05	8.22E-06	1.64E-06	1.20E-02	9.86E-08	1.97E-08
Nickel	3.00E-05	1.23E-05	2.47E-06	4.80E-04	5.92E-09	1.18E-09
Total Cancer Risk					**1.75E-07**	**3.50E-08**

Cancer Risk Estimates

Equations

$$\frac{\mathbf{EC = CA \times EF \times ED}}{\mathbf{AT}}$$

Risk = EC x IURF

Definitions		**Value**
EC	Exposure concentration (µg/m^3)	*Calculated*
CA	Maximum modeled annual air concentration (µg/m^3)	*Calculated*
EF	Exposure frequency (days/year)	350
ED_{child}	Exposure duration (years)	6
ED	Exposure duration (years)	30
AT	Carcinogen (70 years x 365 days/year) averaging time (days)	25550

Appendix C
Non-Cancer Hazard Backup Calculations

Non-Cancer Hazard Estimates

Pollutant	Annual Concentration (µg/m3)	Adult Chronic Non-Cancer Exposure Concentration (µg/m^3)	Child Chronic Non-Cancer Exposure Concentration (µg/m^3)	RfC or MRL (µg/m^3)	Adult Non-Cancer HQ	Child Non-Cancer HQ	Effect
1,3-Butadiene	0.00015	1.44E-04	1.44E-04	2.00E+00	7.19E-05	7.19E-05	ovarian atrophy
1,4-Dichlorobenzene	0.00002	1.92E-05	1.92E-05	8.00E+02	2.40E-08	2.40E-08	liver/repro
Acetaldehyde	0.00194	1.86E-03	1.86E-03	9.00E+00	2.07E-04	2.07E-04	degen olfactory epithelium
Acrolein	0.00035	3.36E-04	3.36E-04	2.00E-02	1.68E-02	1.68E-02	nasal lesions
Benzene	0.00355	3.40E-03	3.40E-03	3.00E+01	1.13E-04	1.13E-04	decreased lymphocyte
Ethylbenzene	0.00143	1.37E-03	1.37E-03	1.00E+03	1.37E-06	1.37E-06	developmental
Formaldehyde	0.00726	6.96E-03	6.96E-03	9.80E+00	7.10E-04	7.10E-04	histological changes in nasal epithelium
Hexane	0.03038	2.91E-02	2.91E-02	7.00E+02	4.16E-05	4.16E-05	peripheral neuropathy
Acenaphthene	7.238E-06	6.94E-06	6.94E-06	NA	NA	NA	NA
Acenaphthylene	1.67797E-05	1.61E-05	1.61E-05	NA	NA	NA	NA
Anthracene	4.89169E-05	4.69E-05	4.69E-05	NA	NA	NA	NA
Benz[a]Anthracene	2.66044E-07	2.55E-07	2.55E-07	NA	NA	NA	NA
Benzo[a]Pyrene	1.26084E-07	1.21E-07	1.21E-07	2.00E-03	6.05E-05	6.05E-05	developmental
Benzo[b]Fluoranthene	2.51867E-07	2.42E-07	2.42E-07	NA	NA	NA	NA
Benzo[k]Fluoranthene	1.26084E-07	1.21E-07	1.21E-07	NA	NA	NA	NA
Benzo[g,h,i]Fluoranthene	3.98161E-06	3.82E-06	3.82E-06	NA	NA	NA	NA
Chrysene	4.91669E-07	4.71E-07	4.71E-07	NA	NA	NA	NA
Dibenzo[a,h]Anthracene	2.60916E-07	2.50E-07	2.50E-07	NA	NA	NA	NA
Fluoranthene	2.70569E-06	2.59E-06	2.59E-06	NA	NA	NA	NA
Flourene	2.33209E-05	2.24E-05	2.24E-05	NA	NA	NA	NA
Indeno[1,2,3-cd]Pyrene	2.51867E-07	2.42E-07	2.42E-07	NA	NA	NA	NA
Naphthalene	0.004451442	4.27E-03	4.27E-03	3.00E+00	1.42E-03	1.42E-03	hyperplasia resp/nasal epithelium
Phenanthrene	0.000192824	1.85E-04	1.85E-04	NA	NA	NA	NA
Pyrene	1.01652E-06	9.75E-07	9.75E-07	NA	NA	NA	NA
Propylene Oxide	0.00122	1.17E-03	1.17E-03	3.00E+01	3.90E-05	3.90E-05	nasal epithelial infolds
Toluene	0.00566	5.43E-03	5.43E-03	5.00E+03	1.09E-06	1.09E-06	CNS
Xylenes	0.00306	2.93E-03	2.93E-03	1.00E+02	2.93E-05	2.93E-05	CNS
Cadmium	0.00002	1.92E-05	1.92E-05	NA	NA	NA	NA
Chromium	0.00002	1.92E-05	1.92E-05	8.00E-03	2.40E-03	2.40E-03	nasal septum atrophy
Nickel	0.00003	2.88E-05	2.88E-05	9.00E-02	3.20E-04	3.20E-04	lung inflammation
Nasal HI							**2.16E-02**
Developmental HI							**6.18E-05**
CNS HI							**3.04E-05**

Non-Cancer Hazard Estimates

Equations

$$\frac{\text{EC = CA x EF x ED}}{\text{AT}}$$

$$\text{HQ} = \frac{\text{EC}}{\text{RfC}}$$

Definitions		**Value**
EC	Exposure concentration ($\mu g/m^3$)	*Calculated*
CA	Maximum modeled annual air concentration ($\mu g/m^3$)	*Calculated*
EF	Exposure frequency (days/year)	350
ED_{child}	Exposure duration (years)	6
ED	Exposure duration (years)	30
AT_{CHILD}	Residential (6 years x 365 days/year) averaging time (days)	2190
AT	Residential (30 years x 365 days/year) averaging time (days)	10950

Appendix D
Acute Hazard Backup Calculations

Acute Hazard Estimates

Pollutant	Hourly Concentration (µg/m3)	Acute Toxicity Factor (µg/m3)	Acute HQ	Effect
1,3-Butadiene	7.90E-03	6.60E+02	1.20E-05	develop
1,4-Dichlorobenzene	1.09E-03	NA	NA	NA
Acetaldehyde	1.04E-01	4.70E+02	2.22E-04	eye/resp irritation
Acrolein	1.67E-02	2.50E+00	6.69E-03	eye/resp irritation
Benzene	4.53E+00	2.70E+01	1.68E-01	developmental, immune system, hematological effects
Ethylbenzene	8.97E-02	1.43E+05	6.26E-07	CNS
Formaldehyde	3.33E-01	5.50E+01	6.05E-03	eye irritation
Hexane	1.08E+01	1.00E+04	1.08E-03	CNS
Acenaphthene	3.97E-04	NA	NA	NA
Acenaphthylene	9.20E-04	NA	NA	NA
Anthracene	2.68E-03	NA	NA	NA
Benz[a]Anthracene	1.47E-05	NA	NA	NA
Benzo[a]Pyrene	6.96E-06	NA	NA	NA
Benzo[b]Fluoranthene	1.39E-05	NA	NA	NA
Benzo[k]Fluoranthene	6.96E-06	NA	NA	NA
Benzo[g,h,i]Fluoranthene	2.20E-04	NA	NA	NA
Chrysene	2.71E-05	NA	NA	NA
Dibenzo[a,h]Anthracene	1.44E-05	NA	NA	NA
Fluoranthene	1.49E-04	NA	NA	NA
Flourene	1.28E-03	NA	NA	NA
Indeno[1,2,3-cd]Pyrene	1.39E-05	NA	NA	NA
Naphthalene	2.44E-01	NA	NA	NA
Phenanthrene	1.06E-02	NA	NA	NA
Pyrene	5.61E-05	NA	NA	NA
Propylene Oxide	7.57E-02	3.10E+03	2.44E-05	eye/resp irritation, developmental
Toluene	6.71E-01	5.00E+03	1.34E-04	eye/resp irritation, CNS
Xylenes	3.59E-01	2.20E+04	1.63E-05	eye/resp irritation, CNS
Cadmium	1.00E-03	4.60E+02	2.17E-06	resp irritation
Chromium	1.27E-03	2.00E-01	6.35E-03	resp irritation
Nickel	1.90E-03	2.00E-01	9.50E-03	immune
Eye Irritation HI			**1.31E-02**	
Repiratory Irritation HI			**1.34E-02**	
CNS HI			**1.23E-03**	
Developmental HI			**1.68E-01**	
Immune System HI			**1.77E-01**	