7 RISK CHARACTERIZATION

The risk characterization combines the information presented in the exposure assessment with the information presented in the toxicity assessment to describe the types and magnitudes of potential carcinogenic risk and noncarcinogenic hazard due to exposure to PCAPP and EDS emissions. The magnitude and types of risks depend on the nature, duration, and frequency of exposure to chemicals and the characteristics of the exposed human receptors. The risk characterization for this MPHRA was performed in accordance with USEPA risk assessment guidelines (USEPA, 1989, 1994a, 1998, and 2005a). The risk estimates developed for this MPHRA represent conservative estimates of the incremental or additional risk potentially associated with exposure to environmental media impacted by PCAPP and EDS emissions.

Quantitative estimates of carcinogenic risks and noncarcinogenic hazards have been calculated for direct inhalation exposures and indirect exposures to PCAPP and EDS emissions. Appendix D, Sections D.10 and D.11, discuss the procedures used to quantify the carcinogenic risks and noncarcinogenic hazards, respectively. As detailed below, quantified total carcinogenic risk was compared to the CDPHE acceptable level of 1 case in a million (i.e., 1/1,000,000, 1.00 E-06, or 1×10^{-6}), and total noncarcinogenic hazard was compared to the CDPHE acceptable hazard index (HI) of 0.25 (i.e., total cumulative dose is less than 25 percent of the RfD or RfC, depending on the exposure route). This section presents the results of both the carcinogenic risk and the noncarcinogenic hazard assessments. In addition, an acute hazard analysis was performed; the results of that assessment are also presented.

7.1 CHARACTERIZATION OF CARCINOGENIC HEALTH EFFECTS

For carcinogens, risks were estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to potential carcinogens released from PCAPP and EDS operations (i.e., excess individual lifetime cancer risk). The IURs and CSFs presented in Section 6 were used to make these estimates. For direct (inhalation) exposures, the IUR converts calculated long-term average exposure concentrations to the incremental risk of an individual developing cancer. Similarly, for indirect exposures, the CSF converts estimated daily intakes averaged over a lifetime of exposure to incremental risk of an individual developing cancer. (Section 5 previously presented information on calculating exposures.) A critical assumption of this estimating procedure is that the dose-response relationship is a linear relationship in the low-dose portion of the dose-response curve. In other words, even the smallest dose of a carcinogen presents some risk of inducing cancer in the exposed human receptor. Under this assumption, the IUR and CSF are a constant, and risk is directly related to ambient air concentration or intake.

Current CDPHE CHWRs (6 CCR 1007-3 264.342 (a)(1)(i)) indicate that a site presents acceptable risk if the calculated cumulative cancer risk to a potentially exposed human receptor is less than 1×10^{-6} (i.e., the risk is less than 1 in a million).

7.1.1 Early-Life Exposures to Mutagenic Carcinogens

As introduced in Section 6.1, USEPA published guidance entitled Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens (USEPA, 2005c). The Supplemental Guidance recommends the use of ADAFs for carcinogens acting through a mutagenic mode of action based on analysis of available data. In preparing this guidance, USEPA listed 67 chemicals that had been reviewed by various researchers and found to have carcinogenic effects from prenatal and postnatal exposure in animals. Vinyl chloride is the only COPC included in this MPHRA that is on this list of 67 mutagens. The Toxicological Review of

Vinyl Chloride in Support of Summary Information on the Integrated Risk Information System (IRIS) (USEPA, 2000a) presents procedures for calculating risk from vinyl chloride for early-life exposures. The calculation procedure involves summing the standard cancer risk (pro-rated over an averaging time of 78 years) and the early-life portion of the cancer risk (which is not pro-rated). For a lifetime (78 year) exposure, this calculation procedure effectively doubles the cancer risk due to exposure to vinyl chloride. This early-life adjustment is necessary due to the mutagenic mode of action for vinyl chloride as a carcinogen. Of the COPCs evaluated in this MPHRA, this procedure only applies to vinyl chloride and only applies to the calculation of carcinogenic effects.

In addition to the early-life exposure supplemental guidance cited above, IRIS identifies two additional COPCs, methylene chloride and trichloroethene, that USEPA has concluded are carcinogenic by a mutagenic mode of action and result in increased early-life susceptibility. Unlike vinyl chloride, however, data are insufficient to develop separate risk estimates for childhood exposure; therefore, USEPA recommends the use of ADAFs to estimate risk from early-life exposures. The following default ADAFs were combined with age-specific exposure estimates when assessing cancer risks:

- 10-fold adjustment for exposures during the first 2 years of life (ADAF = 10);
- 3-fold adjustment for exposures from ages 2 to <16 years of age (ADAF = 3); and
- no adjustment for exposures after turning 16 years of age (ADAF = 1).

Appendix D, Section D.10 presents the methodology used to characterize carcinogenic effects, including the incorporation of early-life considerations.

7.1.2 Carcinogenic Effects from Direct Exposures

Cancer risk from direct exposure to (inhalation of) chemical emissions in air was estimated when an IUR was available for the COPC using the procedures presented in Appendix D Section D.10.2. The procedure for calculating the carcinogenic effects from direct exposures involves multiplying the calculated ambient air concentration to which one is exposed by the IUR for that COPC.

7.1.3 Carcinogenic Effects from Indirect Exposures

Cancer risk from indirect exposures (all pathways except inhalation) to COPCs was estimated following the procedures outlined in Appendix D Section D.10.3. Risk from indirect exposures was calculated by multiplying the indirect COPC intake by the oral CSF for that COPC.

7.1.4 Cumulative Carcinogenic Risk Characterization

Total cancer risk from direct exposures was calculated as the sum of the direct exposure cancer risks for each individual COPC. Similarly, total cancer risk from indirect exposures was calculated as the sum of the indirect exposure cancer risks for each individual COPC. Total direct and indirect cancer risks were then summed to determine the overall cancer risk.

Table 7-1 presents the total excess lifetime cancer risk from both direct and indirect exposures and for each individual infant, child, and adult exposure scenario as well as the composite lifetime exposure scenarios. The lifetime exposure scenarios, described in detail in Section 4.2, represent composite exposures based on various combinations of infant, child and adult exposures. Table 7-1 also presents the cancer risk for the adult worker receptor. As presented in this table, the subsistence fisher lifetime (I) exposure scenario results in the highest lifetime risk of all scenarios (0.265 in a million) and is 3.8 times lower than the CDPHE acceptable risk level of 1 in a million. The pathway results presented in Table 7-1 show that, for this receptor, the fish consumption pathway accounts for 65 percent of the total lifetime risk, followed by the breast milk consumption pathway (20 percent) and the inhalation pathway (2 percent). While breast milk consumption is the second largest exposure pathway, it is driven by the nursing mother's exposure from the fish consumption pathway. A total of 76 percent of the risk from the breast milk consumption pathway is contributed by the mother's consumption of fish.

The individual infant, child, and adult subsistence fisher receptors each contribute a portion of the total risk to the cumulative lifetime subsistence fisher risk; therefore, the individual risks for these receptors will always be less than the composite lifetime risk. The highest total cancer risk for each of the individual receptor is identified below, along with the three highest contributing pathways to the total cancer risk:

- Infant subsistence fisher risk is 0.159 in a million: 58% due to ingestion of fish, 35% due to ingestion of breast milk, and 6% due to inhalation.
- Child subsistence fisher risk is 0.177 in a million: 80% due to ingestion of fish, 10% due to inhalation, and 6% due to ingestion of surface water.
- Adult subsistence fisher risk is 0.0601 in a million: 77% due to ingestion of fish, 19% due to inhalation, and 2% due to dermal contact with surface water.

As indicated above, the highest individual receptor contributing to the composite lifetime (I) subsistence fisher risk is the child followed by the infant, and then the adult, and fish consumption is the most significant pathway for each of these individual receptors. The infant and lifetime (I) risk results presented in Table 7-1 are conservative because the infant is assumed to begin consuming solid food (including fish) immediately upon birth. In addition, as noted above, fish consumption also greatly impacts the contribution from breast milk consumption on the risk to both the infant and lifetime (I) subsistence fisher

Appendix G presents detailed tables of risk for each receptor and the risk presented by each individual COPC.

Table 7-2 presents the 10 COPCs contributing the largest lifetime cancer risk to the subsistence fisher lifetime (I) receptor. This table demonstrates that four COPCs produce 99 percent of the cancer risk. These four COPCs include vinyl chloride, cadmium, 1,2-dichloroethane, and HD. This is important when evaluating the risk drivers and the significance of uncertainty, because changes related to uncertainty parameters for these four COPCs will have the highest impact on uncertainty. Conversely, the impact of uncertainty parameters associated with other COPCs will be insignificant. For example, if the high-end uncertainty factors resulted in a 100 times increase in the cancer risk for ethylbenzene (the COPC contributing the 7th highest cancer risk), the total cancer risk would only increase by 3% to 0.274 in a million.

Table 7-1. Total Carcinogenic Risks for Each Human Receptor by Pathway

Receptor	Pathway									
	Inhalation	Dermal Contact with Surface Water	Dermal Contact with Soil	Incidental Ingestion of Soil	Incidental Ingestion of Surface Water	Ingestion of Produce	Ingestion of Terrestrial Animal Products	Ingestion of Fish	Ingestion of Breast Milk	All Pathways[a]
RESIDENT										
Infant resident	9.01E-09	n/a[b]	2.16E-14	8.24E-14	n/a	3.18E-09	2.13E-12	3.66E-08	2.99E-08	0.0787E-06
Child resident	1.83E-08	3.69E-09	2.06E-14	7.36E-14	1.01E-08	3.95E-09	6.41E-12	5.62E-08	n/a	0.0922E-06
Adult resident	1.16E-08	9.78E-10	1.71E-15	4.29E-15	6.02E-10	9.15E-10	2.38E-12	1.84E-08	n/a	0.0325E-06
Resident lifetime (I)[c]	1.83E-08	3.91E-09	2.80E-14	1.00E-13	9.03E-09	5.18E-09	8.85E-12	6.94E-08	2.99E-08	0.136E-06
Resident lifetime (II)[d]	1.83E-08	4.51E-09	2.20E-14	7.71E-14	1.06E-08	4.71E-09	8.40E-12	7.15E-08	n/a	0.110E-06
SUBSISTENCE FISHER										
Infant subsistence fisher	9.01E-09	n/a	2.16E-14	8.24E-14	n/a	3.18E-09	2.13E-12	9.15E-08	5.52E-08	0.159E-06
Child subsistence fisher	1.83E-08	3.69E-09	2.06E-14	7.36E-14	1.01E-08	3.95E-09	6.41E-12	1.40E-07	n/a	0.177E-06
Adult subsistence fisher	1.16E-08	9.78E-10	1.71E-15	4.29E-15	6.01E-10	9.15E-10	2.38E-12	4.60E-08	n/a	0.0601E-06
Subsistence fisher lifetime (I)[c]	1.83E-08	3.91E-09	2.80E-14	1.00E-13	9.03E-09	5.18E-09	8.85E-12	1.74E-07	5.52E-08	0.265E-06
Subsistence fisher lifetime (II)[d]	1.83E-08	4.51E-09	2.20E-14	7.71E-14	1.06E-08	4.71E-09	8.40E-12	1.79E-07	n/a	0.217E-06
SUBSISTENCE FARMER										
Infant subsistence farmer	9.01E-09	n/a	2.16E-14	8.24E-14	n/a	7.94E-09	5.32E-12	3.66E-08	6.26E-08	0.116E-06
Child subsistence farmer	1.83E-08	3.69E-09	2.06E-14	7.36E-14	1.01E-08	9.87E-09	1.60E-11	5.62E-08	n/a	0.0982E-06
Adult subsistence farmer	1.16E-08	1.30E-09	1.71E-15	4.29E-15	8.02E-10	2.35E-09	7.24E-12	2.45E-08	n/a	0.0406E-06
Subsistence farmer lifetime (I)[e]	1.83E-08	4.24E-09	2.81E-14	1.00E-13	9.24E-09	1.31E-08	2.35E-11	7.55E-08	6.26E-08	0.183E-06
Subsistence farmer lifetime (II)[f]	1.83E-08	4.84E-09	2.21E-14	7.73E-14	1.08E-08	1.19E-08	2.24E-11	7.77E-08	n/a	0.124E-06
WORKER										
Adult worker	2.76E-09	n/a	2.02E-15	3.06E-15	n/a	n/a	n/a	n/a	n/a	0.0028E-06

a. CDPHE acceptable risk = 1.00E-06.
b. n/a = Pathway not applicable to receptor.
c. Lifetime (I) exposure consists of: 1 year of inhalation exposure as an infant and 4 years of inhalation exposure as a child (5 years of total inhalation exposure); 1 year of indirect exposure as an infant, 5 years of indirect exposure as a child, and 24 years of indirect exposure as an adult (30 years of total indirect exposure).
d. Lifetime (II) exposure consists of: 5 years of inhalation exposure as a child (5 years of total inhalation exposure); 5 years of indirect exposure as a child and 25 years of indirect exposure as an adult (30 years of total indirect exposure).
e. Lifetime (I) exposure consists of: 1 year of inhalation exposure as an infant and 4 years of inhalation exposure as a child (5 years of total inhalation exposure); 1 year of indirect exposure as an infant, 5 years of indirect exposure as a child, and 34 years of indirect exposure as an adult (40 years of total indirect exposure).
f. Lifetime (II) exposure consists of: 5 years of inhalation exposure as a child (5 years of total inhalation exposure); 5 years of indirect exposure as a child and 35 years of indirect exposure as an adult (40 years of total indirect exposure).

Table 7-2. Top COPCs Contributing to Carcinogenic Risk[a]

COPC	Inhalation	All Indirect Pathways	Total for All Pathways	Percent of Total Risk
vinyl chloride	7.09E-09	1.62E-07	1.69E-07	63.6%
cadmium	1.58E-13	5.23E-08	5.23E-08	19.7%
1,2-dichloroethane	9.47E-09	1.83E-08	2.78E-08	10.5%
HD	1.54E-09	1.26E-08	1.41E-08	5.32%
1,1,2,2-tetrachloroethane	1.80E-10	1.35E-09	1.52E-09	0.58%
trichloroethene	2.85E-11	4.57E-10	4.86E-10	0.18%
ethylbenzene	1.46E-12	8.41E-11	8.55E-11	0.03%
tetrachloroethene	8.09E-13	7.92E-11	8.00E-11	0.03%
lead	8.08E-15	5.30E-11	5.30E-11	0.02%
arsenic	7.55E-13	2.61E-11	2.69E-11	0.01%
all other COPCs combined	6.93E-12	2.82E-11	3.51E-11	0.01%
Total Pathway Risk	0.018E-06	0.247E-06	0.265E-06	100%

a. Data presented are for the subsistence fisher lifetime (I) scenario which represents the highest carcinogenic risk of all exposure scenarios evaluated.

7.2 CHARACTERIZATION OF NONCARCINOGENIC HEALTH EFFECTS

For COPCs with noncarcinogenic effects, the potential for noncarcinogenic toxicity to occur in an individual was evaluated by comparing the estimated exposure level over a specified time period (e.g., lifetime) with the appropriate noncancer toxicity value (i.e., RfD or RfC). The noncarcinogenic hazard quotient (HQ) assumes that there is a level of exposure (e.g., RfD) below which it is unlikely for even sensitive subpopulations to experience appreciable adverse health effects.

7.2.1 Noncarcinogenic Effects from Direct Exposures

The HQ for direct inhalation exposures to COPCs in the air that have noncancer health effects data were estimated using the procedure outlined in Appendix D Section D.11.1. The HQ for direct exposures was calculated by dividing the direct inhalation intake concentration of a COPC by the RfC for that COPC.

7.2.2 Noncarcinogenic Effects from Indirect Exposures

The HQ for indirect exposures to COPCs that have noncancer health effects was calculated using a procedure similar to that used to calculate the direct exposure HQ. The calculation procedure is described in Appendix D Section D.11.2. The averaging time for noncarcinogenic health effects is equal to the exposure duration (as opposed to the 78-year averaging time used in the calculation of carcinogenic health effects).

7.2.3 Cumulative Noncarcinogenic Hazard Characterization

HQs represent the noncarcinogenic hazard associated with a single COPC. Inhalation (direct) HQs for each COPC were summed to calculate the inhalation HI. Indirect HQs for each COPC were summed to calculate the indirect HI. The HI represents total noncarcinogenic hazard from all COPCs to which a human receptor may be exposed by a particular exposure pathway. The total HI was then calculated by summing the individual pathway HIs for a given human receptor. Appendix D Section D.11.3 presents detailed calculations for determining individual pathway HIs and the total HI.

Different chemicals affect specific target organs (liver, kidney, etc.). It is possible (and acceptable according to USEPA risk assessment guidance [USEPA, 1989]) to calculate total HIs for individual target organs. However, for this MPHRA, the simplified approach of summing all HIs, regardless of target organ, was taken (i.e., comparing multiple target organ-specific HIs to the CDPHE acceptable HI, as opposed to a single cumulative HI regardless of target organ).

Table 7-3 presents HIs for direct and indirect exposures and the total HI for each infant, child, adult, and worker exposure scenario as well as the composite lifetime exposure scenarios described in Section 4.2. The subsistence farmer scenario produces the highest total HI for all combined pathways presented in Table 7-3. The infant subsistence farmer has the highest HI of 0.0306, which is 8.2 times lower than the CDPHE acceptable level of 0.25. The adult worker exposure scenario produced the lowest total HI (0.0162) of any receptor. The highest total HI for each individual receptor and the highest lifetime average HI are identified below, along with the three highest contributing pathways:

- Infant subsistence farmer HI is 0.0306: 59% due to ingestion of produce, 22% due to inhalation, and 17% due to ingestion of breast milk.
- Child subsistence farmer HI is 0.0232: 68% due to ingestion of produce, 29% due to inhalation, and 3% due to ingestion of fish.
- Adult subsistence farmer HI is 0.0145: 51% due to ingestion of produce, 47% due to inhalation, and 2% due to ingestion of fish.
- Subsistence farmer lifetime (I) HI is 0.0160: 54% due to ingestion of produce, 43% due to inhalation, and 2% due to ingestion of fish.

As noted in Section 7.1.4, the infant results are conservative because the infant is assumed to begin consuming solid food (including produce) immediately upon birth. Therefore, the infant is impacted by contaminated foodstuff due to its own consumption as well as contamination included in breast milk.

The composite lifetime exposure scenario HI is calculated as the exposure duration-weighted average HI over different stages of life (i.e., infant, child, adult). As a result, the composite lifetime exposure scenario HI will always fall within the range of HIs calculated for individual receptors. This result is illustrated in the list above, wherein the subsistence farmer lifetime (I) HI is lower than both the infant and child subsistence farmer HIs, but more than the adult subsistence farmer receptor HI. The resulting average HI is most heavily weighted by the 34-year adult indirect exposure duration versus the 1-year infant and 5-year child indirect exposure durations.

Appendix H presents detailed tables of HI for each receptor/pathway combination and HI by each individual COPC.

Table 7-4 presents the 10 COPCs contributing the largest lifetime noncarcinogenic hazard to the infant subsistence farmer receptor. This table demonstrates that eight COPCs produce 98 percent of the HI. These eight COPCs include 1,4-dithiane, thiodiglycol, chlorine, 1,2-dichloroethane, ammonia, cadmium, HD, and hydrogen chloride. This is important when evaluating the significance of uncertainty, because changes related to uncertainty parameters for these COPCs will be those resulting in the highest impact on uncertainty. Conversely, the impact of uncertainty parameters associated with other COPCs will be insignificant.

Table 7-3. Total Noncarcinogenic HI for Each Human Receptor by Pathway

Receptor	Pathway									
	Inhalation	Dermal Contact with Surface Water	Dermal Contact with Soil	Incidental Ingestion of Soil	Incidental Ingestion of Surface Water	Ingestion of Produce	Ingestion of Terrestrial Animal Products	Ingestion of Fish	Ingestion of Breast Milk	All Pathways[a]
RESIDENT										
Infant resident	6.82E-03	n/a[b]	9.28E-08	3.58E-07	n/a	7.27E-03	8.08E-06	4.17E-04	4.17E-03	0.0187
Child resident	6.82E-03	1.16E-05	4.77E-08	1.72E-07	4.05E-05	6.30E-03	1.12E-05	5.85E-04	n/a	0.0138
Adult resident	6.82E-03	6.39E-06	6.88E-09	1.74E-08	4.86E-06	2.93E-03	2.83E-06	2.97E-04	n/a	0.0101
Resident lifetime (I)[c]	6.82E-**03**	7.04E-**06**	1.65E-**08**	5.46E-**08**	1.06E-05	3.63E-**03**	4.40E-**06**	3.49E-**04**	1.39E-**04**	0.0110
Resident lifetime (II)[d]	6.82E-03	7.26E-**06**	1.37E-08	4.32E-08	1.08E-05	3.49E-03	4.23E-06	3.45E-04	n/a	0.0107
SUBSISTENCE FISHER										
Infant subsistence fisher	6.82E-03	n/a	9.28E-08	3.58E-07	n/a	7.27E-03	8.08E-06	1.04E-03	5.32E-03	0.0205
Child subsistence fisher	6.82E-03	1.16E-05	4.77E-08	1.72E-07	4.05E-05	6.30E-03	1.12E-05	1.46E-03	n/a	0.0146
Adult subsistence fisher	6.82E-03	6.39E-06	6.88E-09	1.74E-08	4.86E-06	2.93E-03	2.83E-06	7.43E-04	n/a	0.0105
Subsistence fisher lifetime (I)[c]	6.82E-03	7.04E-06	1.65E-08	5.46E-08	1.06E-05	3.63E-03	4.40E-06	8.73E-04	1.77E-04	0.0115
Subsistence fisher lifetime (II)[d]	6.82E-03	7.26E-06	1.37E-08	4.32E-08	1.08E-05	3.49E-03	4.23E-06	8.63E-04	n/a	0.0112
SUBSISTENCE FARMER										
Infant subsistence farmer	6.82E-03	n/a	9.28E-08	3.58E-07	n/a	1.82E-02	2.02E-05	4.17E-04	5.15E-03	0.0306
Child subsistence farmer	6.82E-03	1.16E-05	4.77E-08	1.72E-07	4.05E-05	1.57E-02	2.81E-05	5.85E-04	n/a	0.0232
Adult subsistence farmer	6.82E-03	6.39E-06	6.88E-09	1.74E-08	4.86E-06	7.32E-03	7.08E-06	2.97E-04	n/a	0.0145
Subsistence farmer lifetime (I)[e]	6.82E-03	6.88E-06	1.41E-08	4.53E-08	9.20E-06	8.64E-03	1.00E-05	3.36E-04	1.29E-04	0.0160
Subsistence farmer lifetime (II)[f]	6.82E-03	7.04E-06	1.20E-08	3.68E-08	9.32E-06	8.37E-03	9.70E-06	3.33E-04	n/a	0.0156
WORKER										
Adult worker	1.62E-03	n/a	8.13E-09	1.25E-08	n/a	n/a	n/a	n/a	n/a	0.00162

a. CDPHE acceptable HI = 0.25.
b. n/a = Pathway not applicable to receptor.
c. Lifetime (I) exposure consists of: 1 year of inhalation exposure as an infant and 4 years of inhalation exposure as a child (5 years of total inhalation exposure); 1 year of indirect exposure as an infant, 5 years of indirect exposure as a child, and 24 years of indirect exposure as an adult (30 years of total indirect exposure).
d. Lifetime (II) exposure consists of: 5 years of inhalation exposure as a child (5 years of total inhalation exposure); 5 years of indirect exposure as a child and 25 years of indirect exposure as an adult (30 years of total indirect exposure).
e. Lifetime (I) exposure consists of: 1 year of inhalation exposure as an infant and 4 years of inhalation exposure as a child (5 years of total inhalation exposure); 1 year of indirect exposure as an infant, 5 years of indirect exposure as a child, and 34 years of indirect exposure as an adult (40 years of total indirect exposure).
f. Lifetime (II) exposure consists of: 5 years of inhalation exposure as a child (5 years of total inhalation exposure); 5 years of indirect exposure as a child and 35 years of indirect exposure as an adult (40 years of total indirect exposure).

Table 7-4. Top COPCs Contributing to Total Noncarcinogenic Hazard[a]

COPC	Inhalation	All Indirect Pathways	Total for All Pathways	Percent of Total Hazard
1,4-dithiane	0.0001	0.0098	0.0098	32.2%
thiodiglycol	0.0000	0.0059	0.0059	19.1%
chlorine	0.0052	0.0000	0.0053	17.2%
1,2-dichloroethane	0.0008	0.0035	0.0043	14.0%
ammonia	0.0002	0.0019	0.0022	7.08%
cadmium	0.0000001	0.0012	0.0012	4.06%
HD	0.0003	0.0004	0.0007	2.18%
hydrogen chloride	0.00004	0.0005	0.0005	1.67%
copper	0.000000	0.0003	0.0003	0.99%
trichloroethene	0.00001	0.0002	0.0002	0.55%
all other COPCs combined	0.0001	0.0002	0.0003	0.98%
Total Pathway Hazard	0.0068	0.0238	0.0306	100%

a. Data presented are for the infant subsistence farmer scenario which represents the highest noncarcinogenic hazard of all exposure scenarios evaluated.

7.3 CHARACTERIZATION OF ACUTE HEALTH EFFECTS

Potential acute hazards associated with short-term (1-hour) emission release events were assessed for each COPC that has both a quantified short-term emission rate and an available AIEC value. Maximum acute hazards all occurred at on-site receptors.

The acute HQ (HQ_A) represents the hazard associated with short-term (1-hour) direct exposure to each COPC in air (maximum 1-hour average air concentration) during a short-term emission release event. Like the HQ for direct exposures from chronic (i.e., long-term) exposures, the calculation of HQ_A was based on the air concentration of each COPC to which a human receptor may be exposed. HQ_As for each COPC were summed to calculate the overall acute HI (HI_A). The detailed calculation procedure for determining the HQ_As for each COPC and the total HI_A is presented in Appendix D. Appendix I presents detailed tables of acute HI by each individual COPC for each receptor.

Table 7-5 and Table 7-6 present the total HI_A for the on-site worker and off-site resident receptors, respectively, during short-term emission release events from the PCAPP and EDS operations. The 10 COPCs contributing the largest of the total HI_A also are provided in these tables. The total HI_A of 0.126 for the on-site worker is 7.9 times less than the CDPHE acceptable acute HI of 1.0, and the HI_A of 0.0112 for the off-site resident is 89 times less than the CDPHE acceptable acute HI.

Table 7-5. Acute Hazards for the On-site Worker Pathway

COPC	AIEC (mg/m^3)	Acute Air Concentration ($\mu g/m^3$)	Acute HQ (dimensionless)
arsenic	0.0002	0.0152	0.0762
HD	0.00075	0.0130	0.0173
ammonia	3.2	55.0	0.0172
hydrogen cyanide	0.34	1.37	0.0040
chlorine	0.21	0.743	0.0035
copper	0.1	0.193	0.0019
2-chloroethoxyethane	0.75	1.20	0.0016
1,4-oxathiane	10	8.60	0.0009
acrolein	0.0025	0.00193	0.0008
monoethanolamine	15	11.3	0.0008
		All Other COPCs Combined	0.0020
		Total HI_A[a]	**0.126**

a. CDPHE acceptable total HI = 1.0.

Table 7-6. Acute Hazards for the Off-site Resident Pathway

COPC	AIEC (mg/m^3)	Acute Air Concentration ($\mu g/m^3$)	Acute HQ (dimensionless)
ammonia	3.2	11.7	0.0037
arsenic	0.0002	0.000720	0.0036
HD	0.00075	0.00131	0.0017
chlorine	0.21	0.121	0.0006
2-chloroethoxyethane	0.75	0.433	0.0006
1,4-oxathiane	10	3.012	0.0003
hydrogen cyanide	0.34	0.0648	0.0002
acrolein	0.0025	0.000316	0.0001
copper	0.1	0.00912	0.0001
2-chlorobutane	0.47	0.0399	0.0001
		All Other COPCs Combined	0.0003
		Total HI_A[a]	**0.0112**

a. CDPHE acceptable total HI = 1.0.

7.4 COMPARISON OF LEAD IMPACTS AND RESULTS TO SCREENING LEVELS

USEPA has no consensus RfD or CSF for inorganic lead and considers lead to be a special case because of the difficulty in identifying the classic "threshold" needed to develop an RfD. The USEPA has issued several directives and guidance documents that provide recommended lead soil screening levels. In addition, CDPHE has adopted a lead regulatory level in air. Therefore, in addition to the quantitative lead results presented earlier in this Section, potential exposures to lead also were evaluated qualitatively by comparing calculated lead concentrations in soil and air with levels in those media that are associated with no adverse effects. The USEPA Implementation Guidance recommends comparing lead concentrations in soil to the “health-based” level of 100 mg/kg. (USEPA, 1994b) As of May 2015, this lead soil screening level is reportedly under evaluation, but no revision has yet been proposed. Any future update adopted will be incorporated into future versions of the MPHRA, if necessary. The

USEPA Office of Solid Waste has also released a detailed directive on risk assessment and cleanup of residential soil lead. The directive recommends that soil lead levels less than 400 mg/kg are generally safe for residential use (USEPA, 1994c). The lower soil lead screening level of 100 mg/kg was used for the current MPHRA. The concentration of lead in air was compared with the CDPHE regulatory level of 0.09 µg Pb/m^3, as provided in the Code of Colorado Regulations (6 CCR 1007-3, Section 264.348, Table IV). Table 7-7 presents the results of this qualitative lead evaluation for PCAPP and EDS emissions. These results indicate that the health-based and regulator levels for lead are not exceeded, and, therefore, no additional assessment of lead is warranted.

Table 7-7. Summary of Lead Screening Level Results

Lead Soil Concentration			Lead Air Concentration		
MPHRA Result[a] (mg Pb/kg)	Health-based Screening Level[b] (mg Pb/kg)	Comparison to Screening Level	MPHRA Result[c] (µg Pb/m^3)	Colorado Regulatory Reference Air Concentration[d] (µg Pb/m^3)	Comparison to Reference Air Concentration
2.42E-10	100	413 billion times lower	1.12E-08	0.09	8 million times lower

a. Highest annual soil concentration at the RME location.
b. Health-based screening soil concentration (USEPA, 1994b)
c. Air concentration at the RME location based on AERMOD modeling.
d. Source: 6 CCR 1007-3, Section 264.348, Table IV.