9 SUMMARY AND CONCLUSION

This screening-level MPHRA evaluated the estimated emissions of COPCs from the operation of PCAPP and EDS based on the latest design information, data acquired from sampling during bench-scale and pilot-scale process testing, and existing data from other similar facilities to calculate direct and indirect exposures for various pathways by which human receptors would be potentially exposed. The estimated cancer risk and chronic noncancer hazards resulting from exposure to the estimated long-term emissions of COPCs were compared with the CDPHE acceptable levels of 1 in a million cancer risk and 0.25 noncancer hazard level. In addition, the acute hazard estimate resulting from exposure to estimated maximum short-term (1-hour) COPC emissions were compared to the CDPHE acceptable level of 1.0.

The highest estimated cancer risks from all exposure scenarios evaluated was 0.265 in a million, which was attributed to the subsistence fisher lifetime (I) receptor. This maximum cancer risk is 3.8 times lower than, or approximately 27 percent of, the CDPHE acceptable level. The results for the maximum cancer risk demonstrate that the predominant exposure pathways are ingestion of fish (65%), ingestion of breast milk (20%), and inhalation (7%). The following four (4) COPCs collectively contribute 99 percent of the total risk:

1. vinyl chloride (64 percent of total risk)
2. cadmium (20 percent of total risk)
3. 1,2-dichloroethane (11 percent of total risk)
4. HD (5 percent of total risk)

While 25 percent of the risk is attributed to the breast milk pathway, 76 percent of the breast milk pathway risk is attributed to the nursing mother's ingestion of fish. Therefore, ingestion of fish actually contributes more than 83 percent of the total risk for the subsistence fisher lifetime (I).

The highest calculated noncancer HI from all exposure scenarios evaluated was 0.0306 for the infant subsistence farmer. This maximum noncancer HI is 8.2 times lower than, or 12 percent of, the CDPHE acceptable level. The results for the maximum noncancer HI demonstrate that the predominant exposure pathways are ingestion of produce (59%), inhalation (22%), and ingestion of breast milk (17%). The COPCs that drive the noncancer HI include the following eight (8) COPCs, which collectively contribute 98 percent of the total noncancer HI:

1. 1,4-dithiane (32 percent of total HI)
2. thiodiglycol (19 percent of total HI)
3. chlorine (17 percent of total HI)
4. 1,2-dichloroethane (14 percent of total HI)
5. ammonia (7 percent of total HI)
6. cadmium (4 percent of total HI)
7. HD (2 percent of total HI)
8. hydrogen chloride (2 percent of total HI)

The acute HI for the on-site worker resulting from the maximum estimated short-term (1-hour) PCAPP and EDS emissions was 0.126, which is 7.9 times lower than, or approximately 13 percent of, the CDPHE acceptable level. Seven (7) COPCs each contribute more than 1 percent of the total acute HI on an individual basis and collectively contribute more than 96 percent of the total acute HI.

The seven COPCs that drive the acute HI for the on-site worker receptor include:

1. arsenic (60 percent of total acute HI)
2. HD (14 percent of total acute HI)
3. ammonia (14 percent of total acute HI)
4. hydrogen cyanide (3 percent of total acute HI)
5. chlorine (3 percent of total acute HI)
6. copper (2 percent of total acute HI)
7. 2-chloroethoxyethane (1 percent of total acute HI)

The acute HI for the off-site resident resulting from the maximum estimated short-term (1-hour) PCAPP and EDS emissions was 0.0112, which is 89 times lower than, or approximately 1 percent of, the CDPHE acceptable level. Eight (8) COPCs each contribute more than 1 percent of the total acute HI on an individual basis and collectively contribute more than 95 percent of the total acute HI.

The eight COPCs that drive the acute HI for the off-site resident receptor include:

1. ammonia (33 percent of total acute HI)
2. arsenic (32 percent of total acute HI)
3. HD (16 percent of total acute HI)
4. chlorine (5 percent of total acute HI)
5. 2-chloroethoxyethane (5 percent of total acute HI)
6. 1,4-oxathiane (3 percent of total acute HI)
7. hydrogen cyanide (2 percent of total acute HI)
8. acrolein (1 percent of total acute HI)

In addition to the results summarized above for the baseline analysis, results were also obtained for a subset of identified sources of uncertainty that presented quantifiable ranges (i.e., baseline and maximum) for the risk and hazard. Although some uncertainty exists that cannot be quantified in this MPHRA, the estimated risks and hazards are all well below the CDPHE acceptable levels after taking into account major sources of underestimations that can be quantified.

Based on these results, it can be concluded that this MPHRA has demonstrated that the chronic and acute health effects resulting from PCAPP and EDS emissions are below all CDPHE specified cancer risk and noncancer hazard levels. Therefore, no further refinement of the conservative assumptions employed in this screening-level MPHRA is deemed necessary at this time. The MPHRA will be updated and rerun using the test results obtained during pilot-scale operations.