Prepared for:
McKinley Paper Company
Port Angeles, Washington

Prepared by:
Ramboll US Corporation
Lynnwood, Washington

Date:
May 2019

Project Number:
1690012265

HEALTH IMPACT ASSESSMENT REPORT

STOCK PREPARATION PROJECT
PORT ANGELES, WASHINGTON

CONTENTS

TABLES

FIGURES

1. INTRODUCTION

McKinley Paper Company (McKinley) owns and operates an integrated pulp and paper mill located at 1902 Marine Drive in Port Angeles, Washington (hereafter, "the Facility"). McKinley plans to upgrade the Facility's existing pulping and stock preparation system to enable the use of alternative recovered fiber sources and the production of more commercially-competitive paper grades (hereafter, "the proposed project").

Because Clallam County is within the jurisdiction of the Olympic Region Clean Air Agency (ORCAA), the Facility must comply with regulations adopted by that agency, as applicable. To accommodate the proposed new equipment and operations, a Notice of Construction (NOC) permit application was submitted to ORCAA on February 8, 2019 and supplemental information was submitted on February 26, 2019 and April 25, 2019 in response to information requests from ORCAA.

Toxic air pollutant (TAP) net emission rate changes attributable to the project calculated using representative emission factors and maximum potential operating schedules were provided in the NOC permit application and supplemental information request response. Of the 12 TAPs considered, the net emission changes of 2 – formaldehyde and methylene chloride – were determined to exceed the Small Quantity Emission Rates (SQERs) provided in Washington Administrative Code (WAC) 173-460-150 for those substances. A dispersion modeling analysis, using the AERMOD modeling system, was employed to predict ambient concentrations attributable to these SQER-exceeding TAP emission changes. The modeling analysis indicated that the net emission change of formaldehyde attributable to the project exceeded the Acceptable Source Impact Level (ASIL) assigned in WAC 173-460-150.

This Health Impact Assessment (HIA) is based on the modeling protocol conditionally approved by Gary Palcisko from the Department of Ecology via email correspondence on April 1, 2019. The remainder of this HIA document provides a description of the project, identification of potentially exposed populations, a discussion of the toxicity of the TAPs of concern, an outline of the air dispersion modeling methodology used to estimate exposure, a description of the calculations used to quantify increased hazards and risk as well as the results of those calculations, and a discussion of uncertainty and conclusions developed as a result of the assessment.

2. PROJECT DESCRIPTION

2.1 Project Location

The Facility is located at 1902 Marine Drive in Port Angeles, Washington. Aerial photos showing the location and layout of the Facility are provided in Figures 2-1 and 2-2.

Service Layer Credits: Source: Esri, DigitalGlobe, GeoEye, Earthstar Geographics, CNES/Airbus DS, USDA, USGS, AeroGRID, IGN, and the GIS User Community

Figure 2-1: Locations of Facility and Modeling Domain

Figure 2-2: Facility Layout

The demographics of Clallam County, as well as the city of Port Angeles, are summarized in Table 2-1. All data were obtained from the U.S. Census Bureau, and represent data from the 2017 census.

Table 2-1: Demographics of Nearby Jurisdictions

Metric	Clallam County[1]	Port Angeles[2]
Population, 2017	75,474	19,872
Percent of persons under 5 years, 2017	3,547	1,331
Percent of persons under 18 years, 2017	13,057	4,292
Percent of persons 65 years and over, 2017	21,737	3,955

Notes:

[1] https://www.census.gov/quickfacts/fact/table/clallamcountywashington

[2] https://www.census.gov/quickfacts/portangelescitywashington

The Facility is located roughly 2 miles northwest of Port Angeles city center. Figure 2-3 shows the 2018 zoning districts surrounding the Facility.[1] In Figure 2-3, the Facility boundaries are outlined in pink. The lagoon within the facility boundary is not readily accessible to the public and was considered on site for this analysis. The area immediately to the east and northeast of the Facility is zoned industrial heavy, and much of the region to the south of the Facility is zoned residential, along with the Strait of Juan de Fuca to the north.

The nearest residential areas lie less than 45 meters (150 feet) from the southwest property boundary of the Facility.

---

[1] https://www.cityofpa.us/Search?searchPhrase=zoning%20map

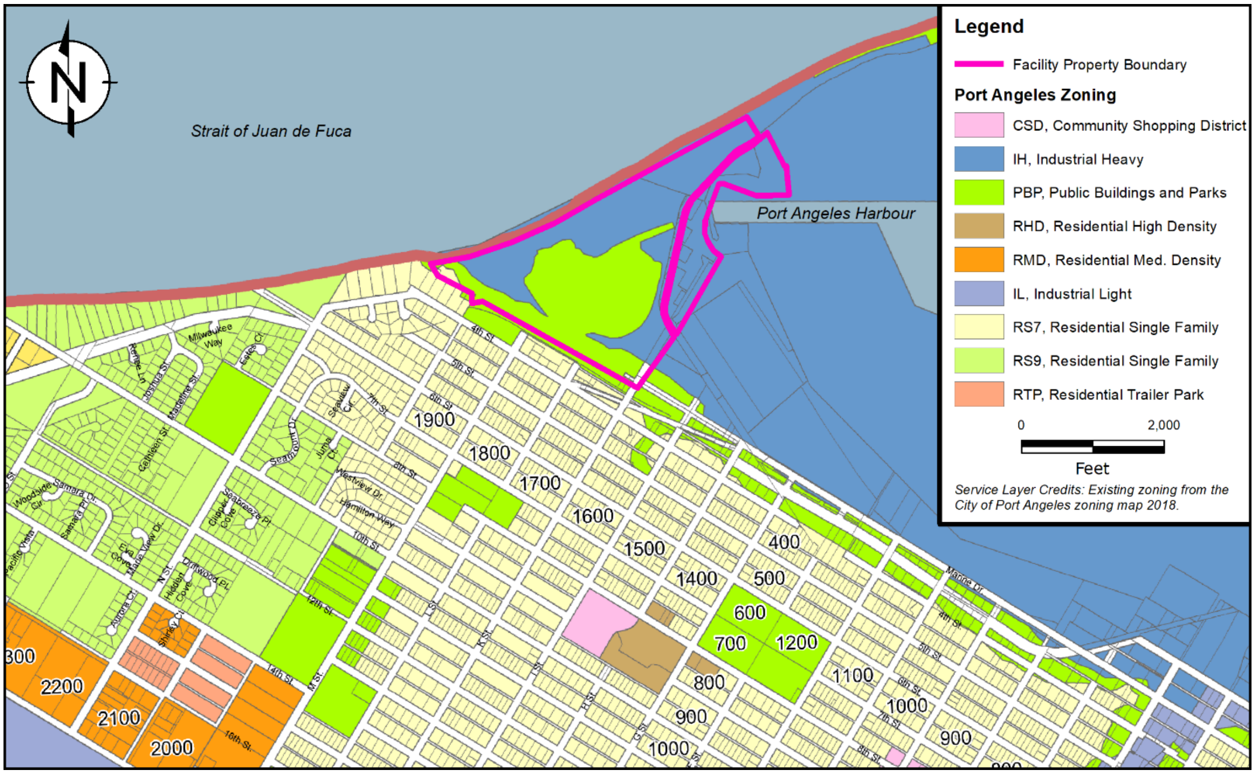


Figure 2-3: Zoning of Area Surrounding Facility

2.2 Emission Units

The existing Facility includes a stock preparation system which is comprised of an old newsprint (ONP) drum pulper, an old corrugated container (OCC) tub pulper, a deinking plant, two mechanical refiner lines, and a purchased kraft re-pulper. The Facility also includes two paper machines and a cogeneration boiler.

Proposed changes to the facility that are expected to affect air pollutant emissions include:

- Replacement of the existing ONP pulper with a new single-line continuous pulper that has a maximum capacity of 900 tons of paper per day (tpd);
- Upgrades to the pulp cleaning, screening, rejects, and dewatering systems;
- Decommissioning the existing OCC tub pulper and refiners; and
- Increased utilization of the existing paper machines.

The proposed project does not directly affect the existing kraft re-pulper, and no increase in steam production by the cogeneration boiler is anticipated, therefore this equipment will not be described further in this analysis.

2.3 Emission Rate Calculations

The proposed continuous pulper associated with the stock preparation system will have a maximum capacity of 900 oven dried tons per day (ODT/day). The existing paper machine will have an increased utilization of up to 840 air dried tons per day (ADT/day). The stock preparation system and paper machine operations will be in continuous, year-round operation.

Total TAP emission rates associated with the proposed modifications are presented in Table 2-2. Maximum potential hourly, daily, or annual emission rates are provided to correspond with the averaging period assigned to each TAP in WAC 173-460-150. Table 2-2 also provides a comparison of the maximum potential emission rate and the SQER for each TAP, and an indication of whether or not the maximum emission rate exceeds the SQER. As shown in Table 2-2, the calculated maximum potential emissions of 2 TAPs exceeded the applicable SQER.

Per the definition of a SQER provided in WAC 173-460-020(7), TAPs with maximum potential emission rates that are less than the applicable SQER are not required to demonstrate compliance with the ambient impact requirement in WAC 173-460-070. However, TAPs with maximum potential emission rates that are equal to or greater than the applicable SQER must assess compliance with the ambient impact requirement using dispersion modeling as indicated in WAC 173-460-080(2)(a). The dispersion modeling is discussed in Section 4.

Table 2-2: Facility-Wide Potential Toxic Air Pollutant Emissions

Pollutant	CAS #	HAP?	Avg. Period[1]	Proposed Emission Rate[2] (lb/averaging period)				Over SQER? (Y/N)
				Stock Prep	Paper Machine	Total	SQER	
Acetaldehyde	75-07-0	Yes	Annual	-4.01E+03	-3.17E+02	-4.33E+03	7.10E+01	N
Carbon Disulfide	75-15-0	Yes	24-hour	1.21E+00	7.55E-01	1.96E+00	1.05E+02	N
Chloroform	67-66-3	Yes	Annual	-1.46E+03	1.06E+03	-4.03E+02	8.35E+00	N
Cumene	98-82-8	Yes	24-hour	-1.66E+00	1.18E+00	-4.86E-01	5.26E+01	N
Formaldehyde	50-00-0	Yes	Annual	-1.05E+03	1.87E+03	8.18E+02	3.20E+01	Y
Methanol	67-56-1	Yes	24-Hour	-6.08E+01	-6.14E+01	-1.22E+02	5.26E+02	N
Methyl Ethyl Ketone	78-93-3	Yes	24-Hour	-6.28E+00	-1.49E+00	-7.77E+00	6.57E+02	N
Methylene Chloride	75-09-2	Yes	Annual	-7.37E+00	7.43E+02	7.36E+02	1.92E+02	Y
Naphthalene	91-20-3	Yes	Annual	-2.35E+03	-3.02E+02	-2.65E+03	5.64E+00	N
Phenol	108-95-2	Yes	24-Hour	-1.66E+01	-2.27E+01	-3.93E+01	2.63E+01	N
Toluene	108-88-3	Yes	24-Hour	7.42E-02	1.32E+01	1.32E+01	6.57E+02	N

Notes:

[1] The averaging period basis for each TAP is assigned in WAC 173-460-150.

[2] The values in the ”SQER” column are the Small Quantity Emission Rates from WAC 173-460-150.

2.4 Control Technology

Per WAC 173-460-060, new or modified sources that increase TAP emission rates must employ Best Available Control Technology for toxics (tBACT). The NOC application submitted to ORCAA included a BACT analysis which addressed tBACT. A summary of the submitted tBACT proposal is provided here.

In the submitted permit application, McKinley indicates that there are no thermal oxidizers, scrubbers, or other add-on control devices demonstrated to be effective at reducing emissions from mechanical pulpers. As a result, both BACT and tBACT for the MP pulper associated with the stock preparation system is proper operation with no added control.

McKinley proposed in the NOC application that, because add-on control technologies are not feasible for paper machines, BACT for VOC and TAP emissions is a work practice standard based on operating in a manner consistent with good air pollution control practices (i.e., minimizing usage rates and the VOC content of paper machine chemical additives, where feasible).

3. HAZARD IDENTIFICATION

The maximum potential emission rates of 2 TAPs, formaldehyde and methylene chloride, exceed the assigned SQER. However, only formaldehyde was predicted by the dispersion modeling to exceed the assigned ASIL. This HIA will assess the cumulative increased cancer risk associated with formaldehyde and methylene chloride emission changes attributable to the project. Non-cancer risks will be evaluated for TAPs that exceed the SQER and have reference concentrations based on the same target organ system as TAPs that exceed the ASIL.

A summary of the potential health effects for each TAP that is expected to exceed the assigned SQER is presented in Table 3-1. The organ or organ system upon which each TAP's toxicity values are based are also provided for each chemical. Both formaldehyde and methylene chloride are considered potentially carcinogenic to humans.

This section presents the potential adverse health effects, physical properties, environmental fate and transport, and general health effects associated with formaldehyde, the only TAP predicted by modeling to exceed the assigned ASIL. Principal sources of information include the U.S Environmental Protection Agency's Integrated Risk Information System (EPA IRIS), Agency for Toxic Substances and Diseases Registry (ATSDR), and California Office of Environmental Health Hazard Assessment (OEHHA) toxic air contaminant databases.

Table 3-1: Potential Effects of Chemicals that Exceed the SQER

Toxic Air Pollutant	CAS #	Critical Effects
Formaldehyde	50-00-0	Irritation of mucous membranes of eyes, nose, and throat, inflammation, epithelial degeneration, respiratory epithelial hypertrophy, and squamous metaplasia, nasal obstruction, pulmonary edema, and dyspnea, allergic sensitization, cough, wheeze, dyspnea, histopathological changes in respiratory epithelium, decrements in lung function, nasopharyngeal and respiratory tract cancer
		Target organ systems: Respiratory system, eyes
Methylene Chloride	75-09-2	Depression of the central nervous system, confusion, incoordination, decreased visual and psychomotor performance, decrease in auditory function, paresthesia, cephalgia, dizziness, nausea, amnesia, narcosis, respiratory depression, cough, breathlessness, chest tightness, dyspnea, hyperactive airways, vomiting, dilation of the stomach, and kidney degeneration
		Target organ systems: Liver, kidney, central nervous system, cardiovascular system

3.1 Potential Effects of Chemicals that Exceed the ASIL

Formaldehyde , which is predicted to exceed the ASIL, can adversely affect the respiratory system and eyes. Studies have found an increased incidence of lung and nasopharyngeal cancer associated with exposure to formaldehyde. The primary acute effects of human exposure to formaldehyde by inhalation consist of irritation to the eyes, nose, and throat and effects on the nasal cavity.[2]

Low levels of formaldehyde can cause irritation of the eyes, nose, throat, and skin. Other noncancer health effects include nausea, headaches, and long-term allergic sensitization. [3] At concentrations that typically occur in ambient air, effects occur in tissues where formaldehyde enters the body (i.e., nose or mouth). At higher levels, coughing, wheezing, bronchitis, nasal obstruction, pulmonary edema, choking, dyspnea, and chest tightness may occur.

People chronically exposed to formaldehyde by inhalation have experienced respiratory symptoms and eye, nose, and throat irritation. Animal studies have reported effects on the nasal respiratory epithelium and lesions in the respiratory system from chronic inhalation exposure to formaldehyde. Some studies of people exposed to formaldehyde in workplace air found more cases of cancer of the nose and throat than expected. However, these workers may have been exposed to a variety of chemicals, so it is not clear if formaldehyde was the chemical that caused this increased rate. In animal studies, rats exposed to high levels of formaldehyde in air developed cancer in nose epithelial cells (nasal squamous cell carcinoma). The ATSDR has determined that formaldehyde may reasonably be anticipated to be a carcinogen. EPA has classified formaldehyde as a Group B1, probable human carcinogen.

The methylene chloride emission increase attributable to the project was estimated exceed the SQER; however, unlike formaldehyde, it was not predicted by the modeling to exceed the ASIL. Routes of exposure include inhalation, ingestion and dermal contact. Exposure to methylene chloride may lead to decreased visual and psychomotor performance, depression of the central nervous system, and a decrease in auditory function.[5,6,8]

When inhaled, low exposure levels of methylene chloride could lead to eye, nose, and throat irritation as well as effects on the nasal cavity. Humans may also experience coughing, wheezing, chest pains, and bronchitis.[4] A human study exposing twelve healthy adults to 195 ppm for four hours resulted in signs of diminished performance on auditory and dual-task functions.[5,6]

When chronically exposed to methylene chloride via inhalation, humans may experience increased blood carboxyhemoglobin and impairment of the respiratory system, as well as eye, nose, and throat irritation. One study documented an adult male had developed gait, dysarthria, and memory loss after

---

[2] https://www.epa.gov/sites/production/files/2016-09/documents/formaldehyde.pdf

[3] https://oehha.ca.gov/media/downloads/air/document/formaldehyde.pdf

[4] U.S. Environmental Protection Agency (USEPA). n.d. Hazard summary – formaldehyde. https://www.epa.gov/sites/production/files/2016-09/documents/formaldehyde.pdf

[5] Putz VR, Johnson BL, Setzer JV. 1976. A comparative study of the effects of carbon monoxide and methylene chloride on human performance. J Environ Path Toxicol;2:97-112, as cited in OEHHA 2008.

[6] Office of Environmental Health Hazard Assessment (OEHHA). 2008. Appendix D.2 Acute RELs and toxicity summaries using the previous version of the Hot Spots Risk Assessment guidelines. https://oehha.ca.gov/media/downloads/crnr/appendixd2final.pdf

having worked with 15-50 liters of methylene chloride every day for three years.[7,8] Animal studies have reported effects on the liver. One study exposing rats to various concentrations of methylene chloride lead to histopathologic lesions in the livers of rats.[8,9] Another study noted elevations in liver triglycerides after exposing mice to 100 ppm of methylene chloride for 2 weeks.[8,10]

3.2 Atmospheric Fate

Generally, formaldehyde, is not persistent in air. Formaldehyde reacts with other chemicals in air (mainly sunlight-derived radicals) and is removed via direct photolysis and oxidation.[11] The breakdown products of formaldehyde include formic acid and carbon monoxide. Methylene chloride readily evaporates and the majority releases into the air.[12] Methylene chloride is broken down by chemicals generated in sunlight

3.3 Terrestrial Fate

Formaldehyde is biodegraded in soil in a relatively short time. Methylene chloride loosely attaches to soil particles but often moves from the soil and into the air.

3.4 Aquatic Fate

Over a few days, formaldehyde will biodegrade to low levels when released to water.[11] Methylene chloride does not easily dissolve in water; in an aqueous setting, the half-life ranges from about 1 to 6 days, with assistance from other chemicals and bacteria.[4]

---

[7] Barrowcliff DF. 1978. Chronic carbon monoxide poisoning caused by methylene chloride painstripper. Med. Sci. Law 18(4):238, as cited in OEHHA 2000.

[8] Office of Environmental Health Hazard Assessment (OEHHA). 2000. Appendix D3. Chronic RELs and toxicity summaries using the previous version of the Hot Spots Risk Assessment guidelines. https://oehha.ca.gov/media/downloads/crnr/appendixd3final.pdf

[9] Nitschke KD, Burek JD, Bell TJ, Kociba RJ, Rampy LW, and McKenna MJ. 1988. Methylene chloride: A 2-year inhalation toxicity and oncogenicity study in rats. Fundam. Appl. Toxicol. 11:48-59, as cited in OEHHA 2000.

[10] Weinstein RS, and Diamond SS. 1972. Hepatotoxicity of dichloromethane (methylene chloride) with continuous inhalation exposure at a low dose level. Proceedings of the 3rd Annual Conference on Environmental Toxicology, 25, 26, and 27 October, 1972. Aerospace Medical Research Laboratory, Aerospace Medical Division, Air Force Systems Command, WrightPatterson Air Force Base, OH, as cited in OEHHA 2000.

[11] Agency for Toxic Substances and Disease Registry.1999. Toxicological profile for formaldehyde. https://www.atsdr.cdc.gov/toxprofiles/tp111.pdf

[12] https://www.atsdr.cdc.gov/toxprofiles/tp14-c1-b.pdf

4. POLLUTANT CONCENTRATION CALCULATIONS

4.1 Modeling Methodology

Air dispersion modeling is frequently used to provide ambient air concentrations for calculating inhalation exposure to airborne toxic compounds. This section provides the methodology used to calculate ambient concentrations and the results of the modeling analysis.

4.1.1 Model Selection

Regulatory modeling techniques were reviewed to select the most appropriate air quality dispersion model to simulate dispersion of air pollutant emissions attributable to the proposed project. AERMOD, the preferred model in the U.S. Environmental Protection Agency's (USEPA's) "Guideline on Air Quality Models" (codified as Appendix W to 40 CFR Part 51, hereafter referred to as the "Guideline"), was selected for the modeling analysis primarily because it is the most up-to-date dispersion model currently available, and is recommended for use in Ecology's air toxics review guidance document.[13]

4.1.2 Modeling Procedures

AERMOD was applied using regulatory defaults and the options and data discussed in this section.

4.1.2.1 Setup and Application

The most up-to-date version of AERMOD (Version 18081) available was applied using the default options for dispersion that depend on local meteorological data, regional upper air data, and the local physical characteristics of land use surrounding the Facility. An archive of modeling files is provided for review.

4.1.2.2 Averaging Periods

The TAPs listed in WAC 173-460-150 have assigned averaging periods (i.e., 1-hour, 24-hour, or annual) which apply to both the SQER and the ASIL for a given TAP. Both TAPs included in this assessment are assigned an annual averaging period, and AERMOD was executed to calculate ambient concentrations on that basis. However, based on the different exposure periods of nearby receptors and the possibility for acute effects, AERMOD was also configured to provide short-term averaging period (i.e., 1-hour, 8-hour, and 24-hour) results for both TAPs.

4.1.3 Terrain Elevation Data and Receptor Network

The 12-km-by-12-km domain used for the modeling simulations is shown in Figure 2-1. Terrain elevations for receptors, as well as the base elevations of onsite structures and emission units, were prepared using available data from the National Elevation Dataset (NED) developed by the United States Geological Survey (USGS); these data have a horizontal spatial resolution of approximately 10 meters (m). The elevation and hill height scale for each receptor were determined using the AERMOD terrain preprocessor, AERMAP (version 18081). All receptor locations are in Universal Transverse Mercator (UTM) coordinates using the spatial reference of NAD 83, Zone 10.

---

[13] Department of Ecology, "Guidance Document: First, Second, and Third Tier Review of Toxic Air Pollution Sources (Chapter 173-460 WAC)." Publication Number 08-02-025, revised August 2015.

Receptors spaced 600 m apart were placed in a grid pattern throughout the 12-km-by-12-km modeling domain. Nested grids of receptors with 12-m, 25-m, 50-m, 100-m, and 300-m spacing were within 300-m, 800-m, 1.8-km, 4-km, and 9-km square areas, respectively, with the Facility at the center of each. Receptors were also located at 10-m intervals along the Facility boundary.

The receptor locations are shown in Figure 4-1. All receptors were located 1.5 m above grade (i.e., “flagpole” receptors) to conservatively estimate inhalation exposure in the human breathing zone.

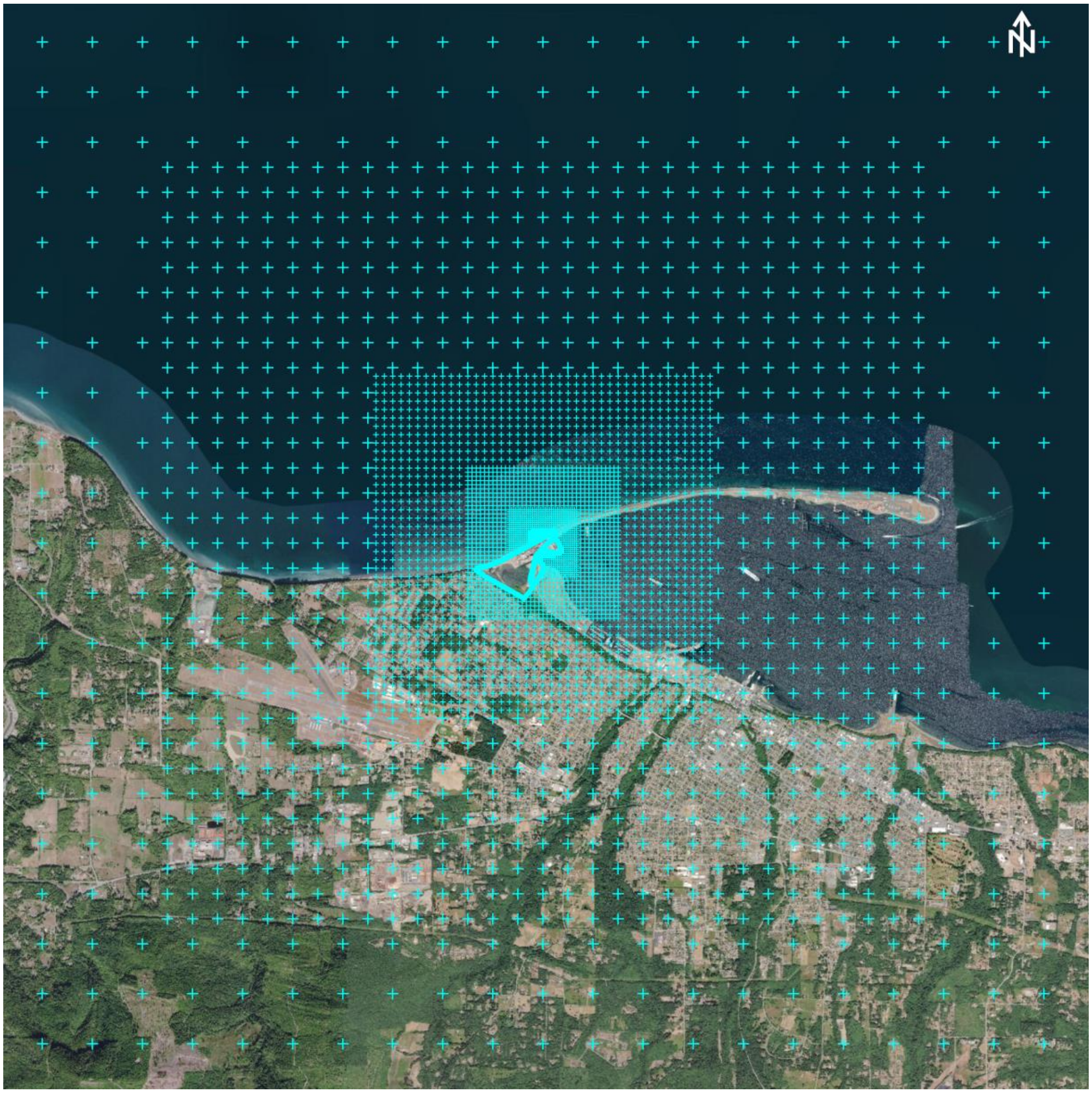


Figure 4-1: Final Receptor Locations

4.1.4 Meteorological Data

Previous dispersion modeling analyses conducted for the Facility used site-specific meteorological data. A description of the on-site meteorological data set (hereafter, “the McKinley dataset”) and how it was processed using the AERMET meteorological program for use with AERMOD was included in the dispersion modeling analysis documentation. This description (hereafter, “the AERMET memo”) is provided as Attachment A to this document. Except as noted in this section, the data and the processing methodology used for this modeling analysis are consistent with those outlined in the AERMET memo and used in modeling analyses in support of permit applications previously submitted to ORCAA by the facility.

The EPA meteorological program AERMET was used to combine surface meteorological observations with twice-daily upper air soundings to calculate the meteorological variables and profiles required by AERMOD. The current version of AERMET (Version (18081) was used for this modeling analysis, and the option to adjust the surface friction velocity (U*) for low-wind or stable conditions was used, without the Bulk Richardson Number option. The site-specific surface data used for the analysis does not include a measurement of the standard deviation of horizontal wind direction (a.k.a., "sigma theta"); based on EPA guidance, it is acceptable to use the adjust U* option when processing the meteorological data as a result of not using sigma theta data, and that option was used in this case.

A representative meteorological data set was prepared using the McKinley dataset, which was collected by ORCAA between 2002-2005 and contemporaneous upper air data from the National Weather Service (NWS) station in Quillayute, Washington. The surface data were collected at a meteorological station located at 1815 Marine Drive, which is adjacent to the northeast side of the Facility.

Regional meteorological data, such as could cover, were obtained from the NWS station at the William R Fairchild International Airport (Fairchild Airport), which is located approximately two miles southwest of the facility. A windrose summarizing the wind speed and wind direction data from the McKinley data set along with wind data statistics is provided in Figure 4-2.

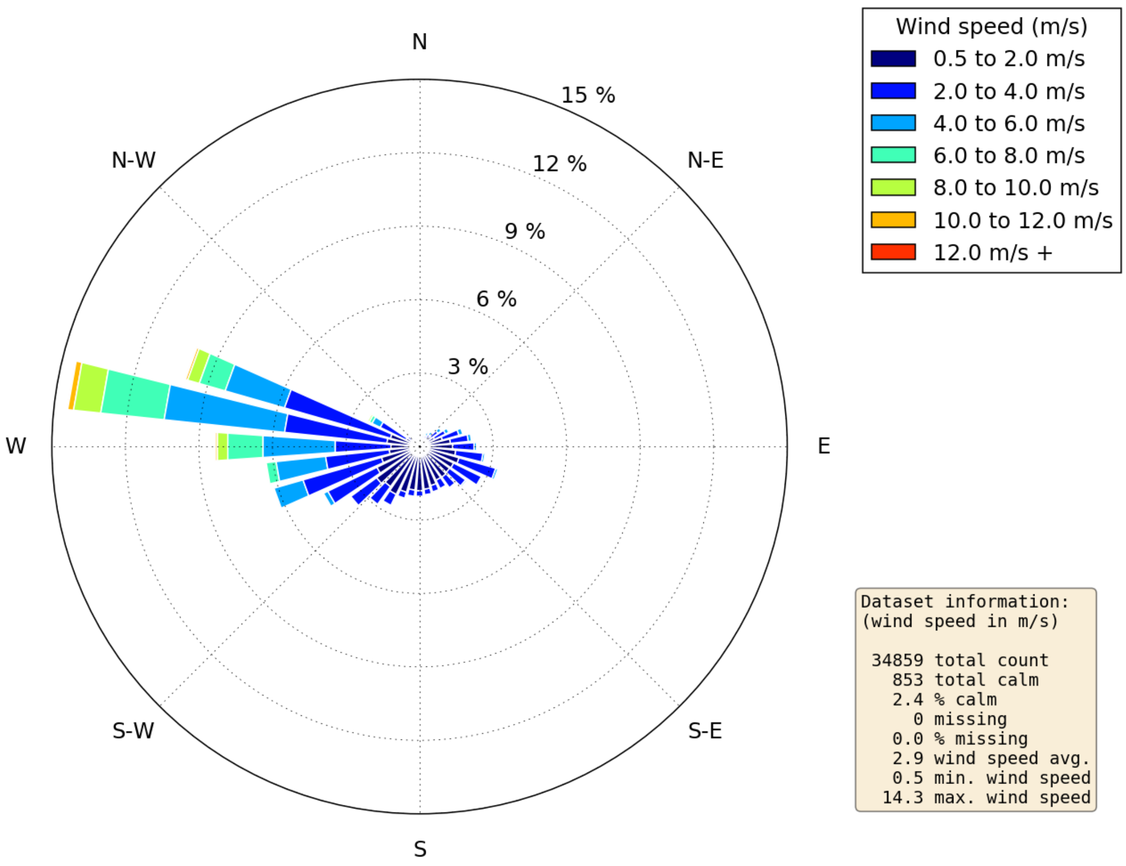


Figure 4-2: McKinley Dataset Windrose

Additional meteorological variables and geophysical parameters are required for the AERMOD dispersion model to estimate the surface energy fluxes and construct boundary layer profiles. Surface characteristics including albedo, Bowen ratio, and surface roughness length were determined for the area surrounding the Facility and the Fairchild Airport meteorological station using the AERMET surface characteristic pre-processor, AERSURFACE (Version 13061), and the USGS National Land Cover Dataset (NLCD) landuse data.

According to the AERMET memo, previous dispersion modeling analyses at the McKinley facility used varying moisture conditions for the four years of data when running AERSURFACE. The AERMET memo specified that the years 2002 and 2003 experienced “average” moisture conditions, whereas the years 2004 and 2005 experienced “dry” conditions. A review of the monthly precipitation throughout these four years indicate that 2004 and 2005 had lower annual averages than other years; however, these annual totals were missing one or more full months of data.[14] Hence, because the annual precipitation data is incomplete, these two years were assigned “average” moisture conditions, instead of “dry” conditions. All other AERSURFACE inputs specified in the AERMET memo were followed for this dispersion modeling analysis.

---

[14] https://wrcc.dri.edu/cgi-bin/cliMAIN.pl?wa6624

4.1.5 Emission Unit Characterization

Emissions of formaldehyde and methylene chloride from stock preparation activities and the paper machines are expected to change as a result of the project. The locations of the vents that exhaust these two emission units to the atmosphere are shown in Figure 4-3. Emissions from the stock preparation in the recycled paper plant will exhaust through six roof vents; each vent was represented in the modeling as a point source. Emissions from the paper machines (Paper Machine 1 [PM1] and Paper Machine 2 [PM2]) will exhaust through seven vents on the roof of the paper machine building. The current PM1 was formerly called Paper Machine 3 (PM3) in previous facility configurations. PM1 is associated with four roof vents, and PM2 is associated with three roof vents; each vent was represented in the modeling as a point source. The parameters used to characterize emissions from these point sources in the modeling are summarized in Table 4-1.

Table 4-1: Emission Release Parameters

Emission Unit	Stack Height (ft / m)	Temperature (°F / K)	Exit Velocity (ft/s / m/s)	Diameter (ft / m)
PM1_3010	58.9 / 18.0	110 / 316	53.05 / 16.17	5 / 1.52
PM1_3020	60.7 / 18.5	110 / 316	65.78 / 20.05	5 / 1.52
PM1_3030	60.7 / 18.5	110 / 316	65.78 / 20.05	5 / 1.52
PM1_3040	59.2 / 18.0	110 / 316	53.05 / 16.17	5 / 1.52
PM2_3010	60.3 / 18.4	110 / 316	53.05 / 16.17	5 / 1.52
PM2_3020	59.9 / 18.3	110 / 316	65.78 / 20.05	5 / 1.52
PM2_3030	59.8 / 18.2	110 / 316	65.78 / 20.05	5 / 1.52
RPP_5005	80.8 / 24.6	ambient	47.75 / 14.55	3.33 / 1.02
RPP_5010	80.6 / 24.6	ambient	47.75 / 14.55	3.33 / 1.02
RPP_5015	80.6 / 24.6	ambient	47.75 / 14.55	3.33 / 1.02
RPP_5020	80.6 / 24.6	ambient	47.75 / 14.55	3.33 / 1.02
RPP_5025	101.5 / 30.9	ambient	47.75 / 14.55	3.33 / 1.02
RPP_5030	80.6 / 24.6	ambient	47.75 / 14.55	3.33 / 1.02

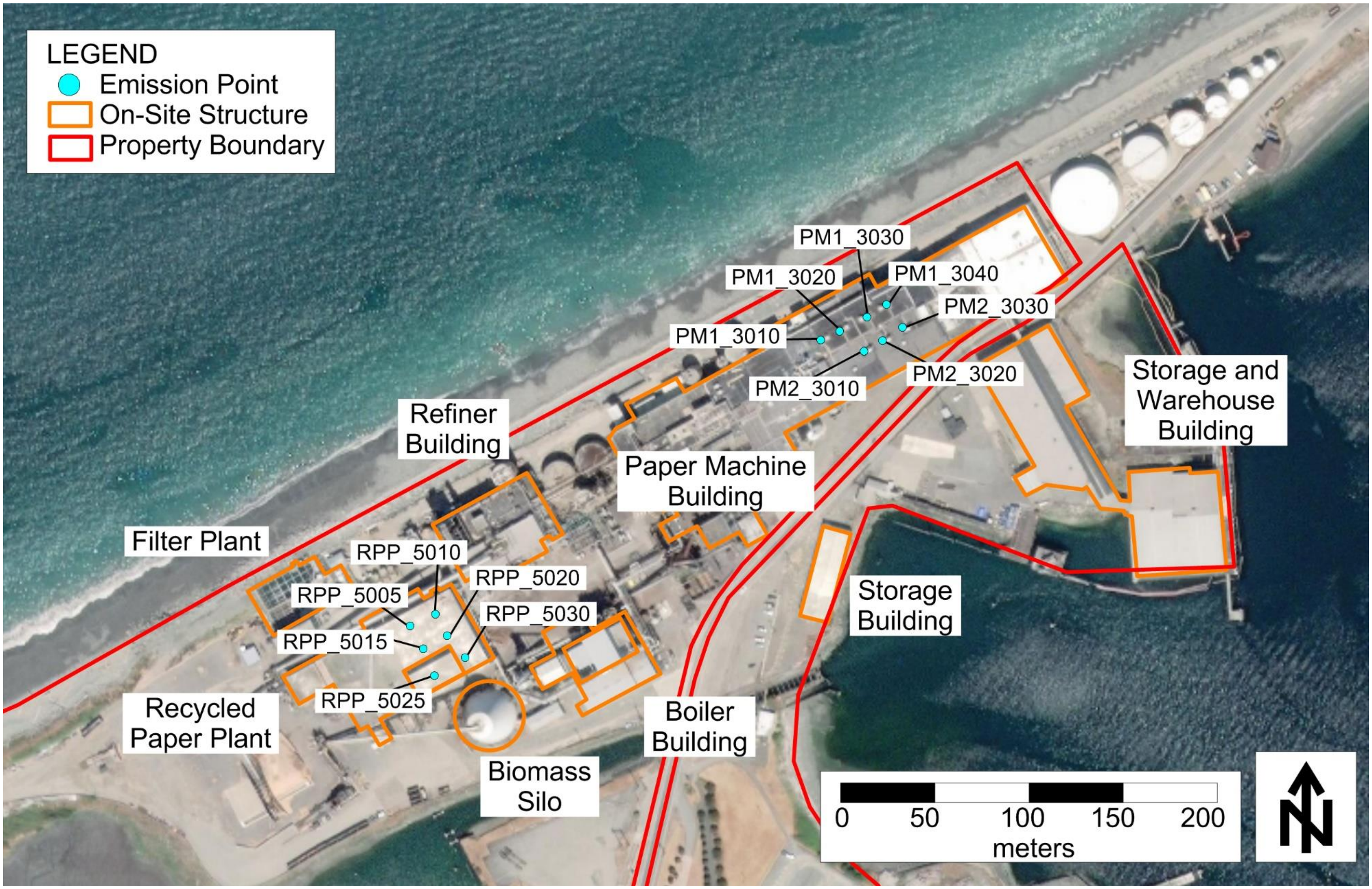


Figure 4-3: Facility Layout for Modeling

Downwash algorithms incorporated into AERMOD account for the plume dispersion effects of the aerodynamic wakes and eddies produced by buildings and structures. In addition to providing a Good Engineering Practice (GEP) evaluation, EPA's Building Profile Input Program for the PRIME algorithm (BPIP-PRIME) was used to determine direction-specific downwash parameters for each point source. Using the output from BPIP-PRIME, AERMOD calculates fields of turbulence intensity, wind speed, and slopes of the mean streamlines as a function of projected structure shape. AERMOD also uses a numerical plume rise model to determine the change in plume centerline location and the rate of plume dispersion with downwind distance. Concentrations are predicted in both the near and far wake regions, with the plume mass captured by the near wake treated separately from the un-captured primary plume, and re-emitted to the far wake as a volume source. The locations and dimensions of each on-site structure shown in Figure 4-3 were provided to BPIP PRIME. The heights for existing structures are presented in Table 4-2.

Table 4-2: Significant Onsite Structure Heights

Structure	Description	Height Above Grade	
		(ft)	(m)
Building 1	Paper Machine Building	50	15.2
Building 2	Filter Plant	27	8.2
Building 3 (2 tiers)	Recycled Paper Plant	75 / 96	22.9 / 29.3
Building 4	Refiner Building	77	23.5
Building 5	Storage and Warehouse	30	9.1
Building 6	Storage	20	6.3
Boiler (2 tiers)	Boiler, Sludge Press, and Steam Turbine Generator	60 / 110	18.3 / 33.5
Biomass	Biomass Silo	121	36.9

4.1.6 Emission Rates

To evaluate the ambient concentration of each TAP emission increase attributable to the proposed project, the emission rates described in Section 2.3 were used. Table 4-3 shows the emission rates for all sources as they were input into AERMOD for all pollutants modeled.

Table 4-3: Modeled Emission Rates

Emission Unit	Formaldehyde (g/s)	Methylene Chloride (g/s)
PM1_3010	3.84E-03	1.53E-03
PM1_3020	3.84E-03	1.53E-03
PM1_3030	3.84E-03	1.53E-03
PM1_3040	3.84E-03	1.53E-03
PM2_3010	3.84E-03	1.53E-03
PM2_3020	3.84E-03	1.53E-03

Emission Unit	Formaldehyde (g/s)	Methylene Chloride (g/s)
PM2_3030	3.84E-03	1.53E-03
RPP_5005	-2.52E-03	-1.77E-05
RPP_5010	-2.52E-03	-1.77E-05
RPP_5015	-2.52E-03	-1.77E-05
RPP_5020	-2.52E-03	-1.77E-05
RPP_5025	-2.52E-03	-1.77E-05
RPP_5030	-2.52E-03	-1.77E-05

4.2 Modeling Results

To evaluate the ambient concentration of each TAP emission increase attributable to the proposed project, the emission rates and source release parameters described in the previous sections were applied using the modeling methodology outlined above. The maximum predicted concentrations for the two TAPs that exceed the assigned SQERs are presented in Table 4-4.

Table 4-4: Maximum Predicted Project TAP Concentrations

Toxic Air Pollutant	CAS #	Averaging Period	Maximum Concentration (μg/m³)	ASIL (μg/m³)	Over ASIL?
Formaldehyde	50-00-0	1-hr	5.59	--	--
		8-hr	2.98	--	--
		24-hr	1.96	--	--
		Annual	0.73	0.167	Yes
Methylene Chloride	75-09-2	1-hr	2.22	--	--
		8-hr	1.24	--	--
		24-hr	0.78	--	--
		Annual	0.31	1.0	No

Figures 4-3 through 4-11 show 1-hr, 8-hr, and annual average isopleths for formaldehyde and methylene chloride.

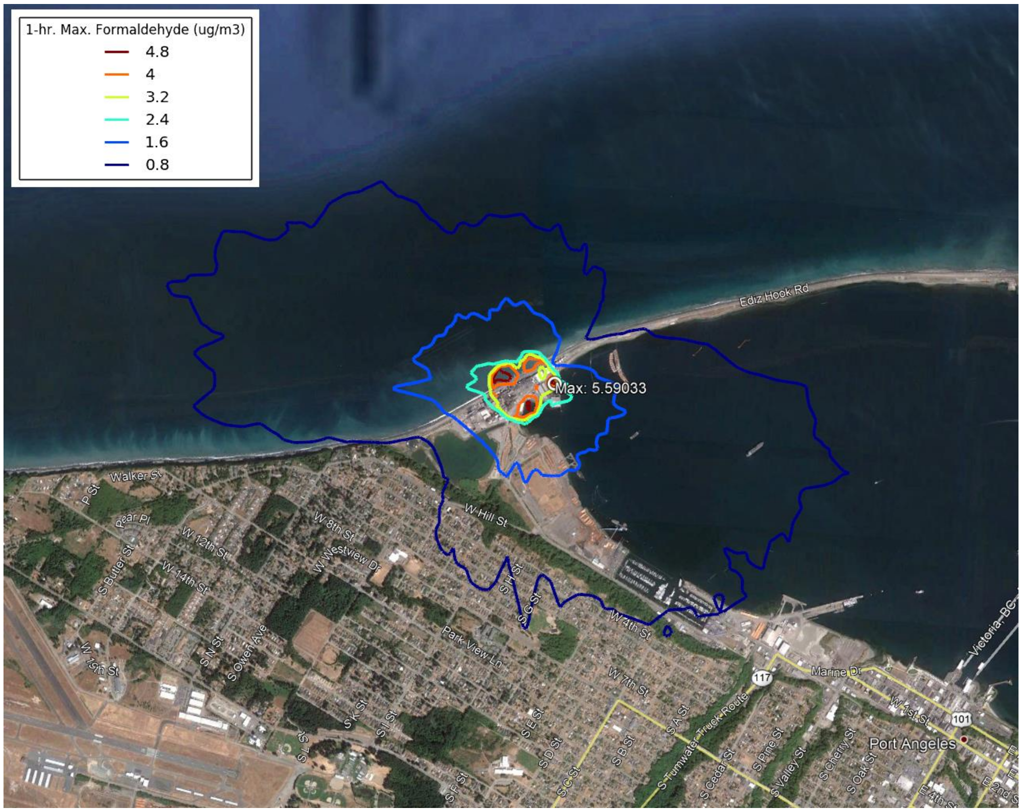


Figure 4-4: Max. Predicted 1-hr Average Formaldehyde Conc. (µg/m³)

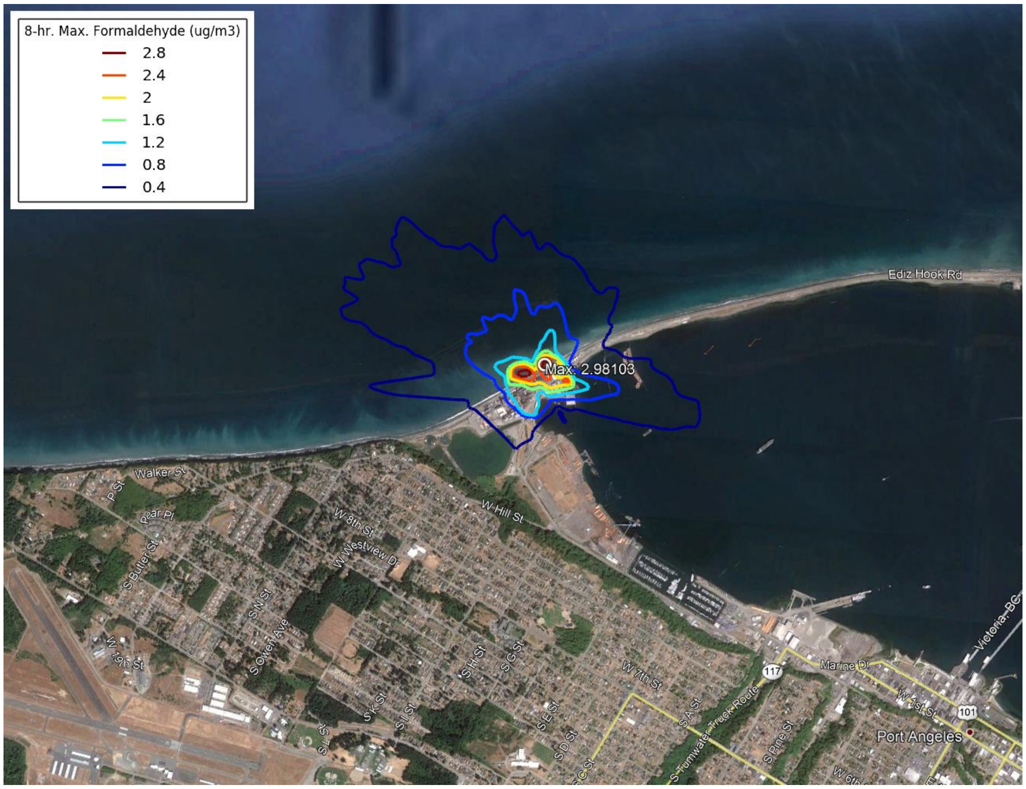


Figure 4-5: Max. Predicted 8-hr Average Formaldehyde Conc. (µg/m³)

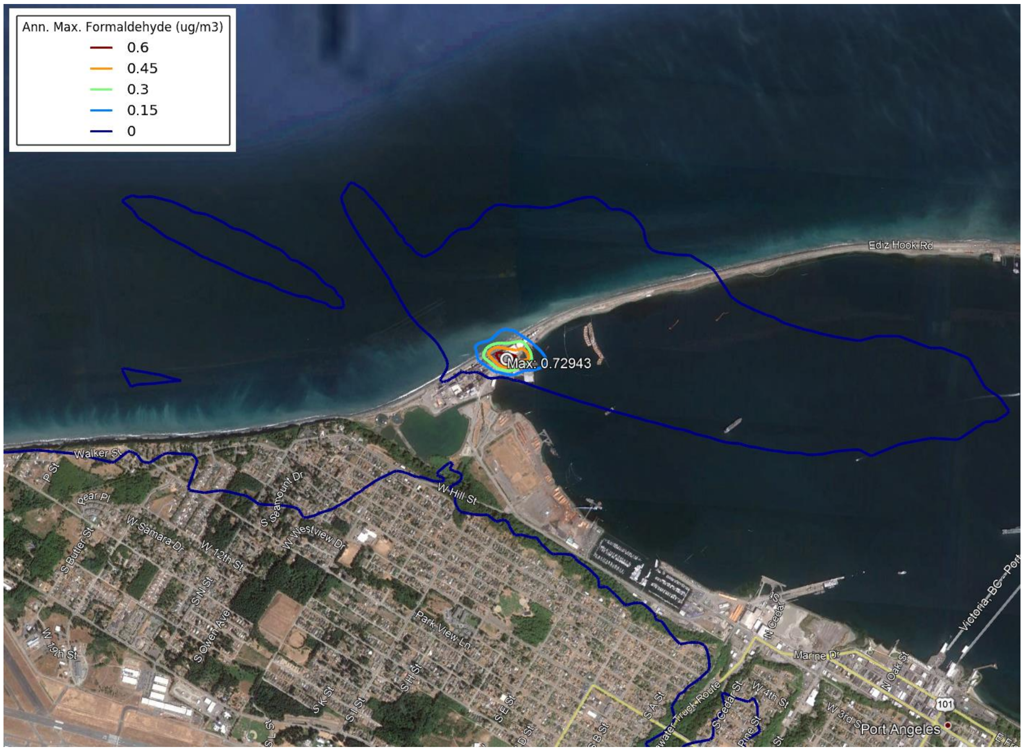


Figure 4-6: Max. Predicted Annual Average Formaldehyde Conc. (µg/m^3)

Figure 4-7: Max. Predicted 1-hr Average Methylene Chloride Conc. (µg/m^3)

Figure 4-8: Max. Predicted 8-hr Average Methylene Chloride Conc. (µg/m^3)

Figure 4-9: Max. Predicted Annual Average Methylene Chloride Conc. (µg/m^3)

4.3 Background Concentration

The EPA has developed, and periodically updates, the National-Scale Air Toxics Assessment (NATA) to identify and prioritize air toxics, sources, and locations of concern. The most recently issued NATA was for 2014, and the TAPs background concentrations within the census tract in which the Facility is located are presented in Table 4-5.

Table 4-5: NATA 2014 Predicted Toxic Air Pollutant Concentrations

Toxic Air Pollutant	CAS #	Annual Average Concentration [1] (μg/m^3)
Formaldehyde	50-00-0	0.973
Methylene Chloride	75-09-2	0.301

Notes:

[1] Background ambient concentration data presented for census tract 53009000700.

5. IDENTIFICATION OF POTENTIALLY EXPOSED POPULATIONS

The HIA evaluates potential airborne exposure to modeled TAP concentration increases attributable to the project. The potentially exposed populations within the simulation domain are identified in this section. These population groups include residents and workers as well as sensitive subpopulations.

5.1 Receptors of Concern

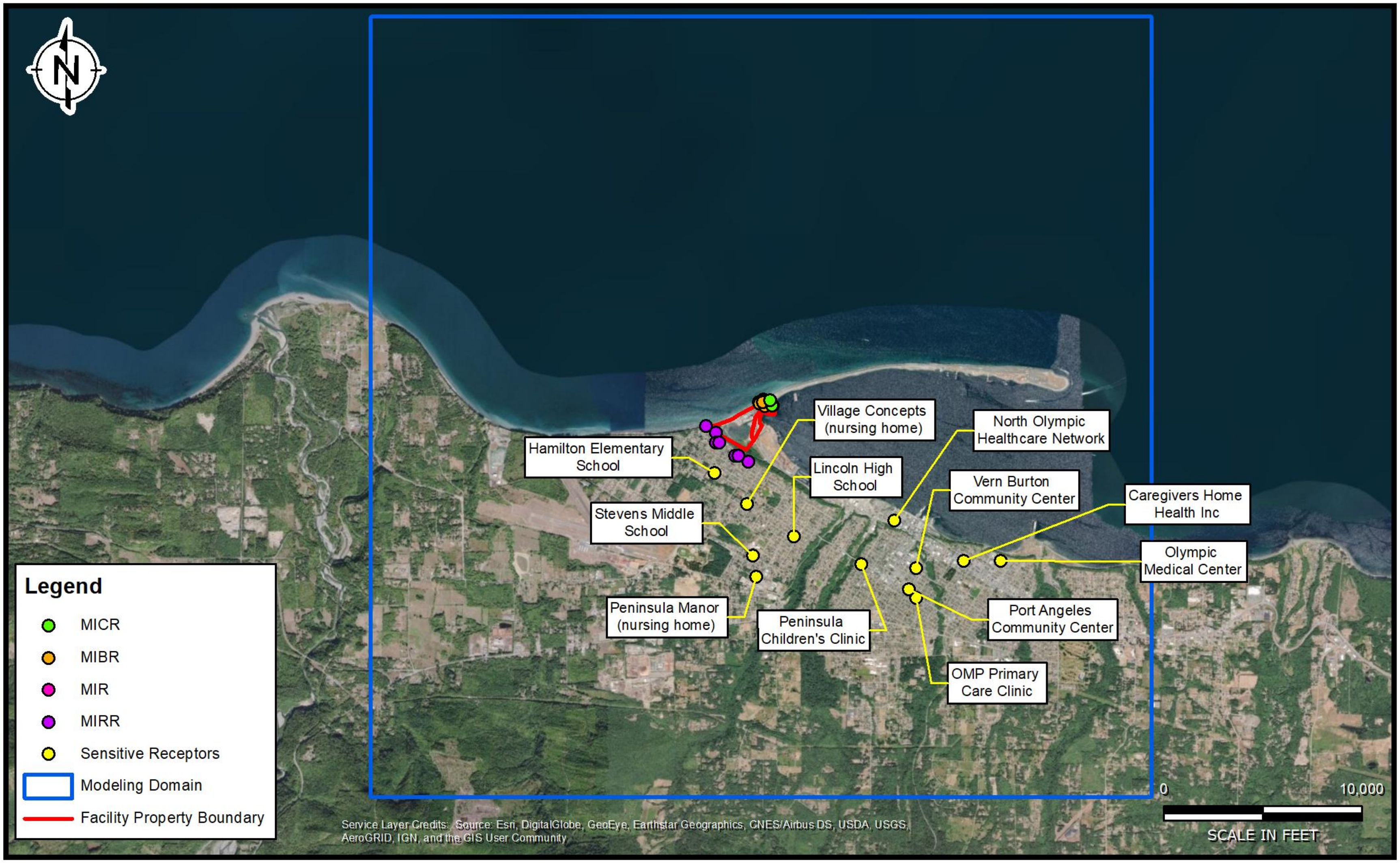
The primary populations that may be exposed to project emissions include residents and workers. The maximally impacted receptor (MIR), residential receptor (MIRR), commercial receptor (MICR), and boundary receptor (MIBR) locations are shown in Figures 5-1 and 5-2.

The MIBR is the location that experiences the highest TAPs concentration along the Facility perimeter, which serves as the boundary for publicly-accessible land. Potential receptors that may be periodically present around the perimeter of the Facility include employees or customers of the Facility or adjacent businesses.

For all TAP concentrations and averaging periods, impacts at the overall maximally impacted receptor (MIR) are quantified to provide an upper-bound estimate of potential exposures within the vicinity of the Facility.

5.2 Sensitive Populations

For the purpose of this HIA, sensitive populations are identified as children, the infirm, and elderly persons. These populations may be more sensitive to the effects of TAPs. The nearest identified sensitive receptors are listed in Table 5-1, and the locations relative to the Facility are presented in Figure 5-1. Because the closest sensitive receptors are more distant from the Facility than the MIR, MIBR, MIRR, and MICR, which are shown in Figure 5-2, risk increases at the sensitive receptors are expected to be less than those identified at the maximally-impacted receptors.



Service Layer Credits: Source: Esri, DigitalGlobe, GeoEye, Earthstar Geographics, CNES/Airbus DS, USDA, USGS, AeroGRID, IGN, and the GIS User Community

Figure 5-1: Locations of Sensitive Receptors Nearest to the Facility

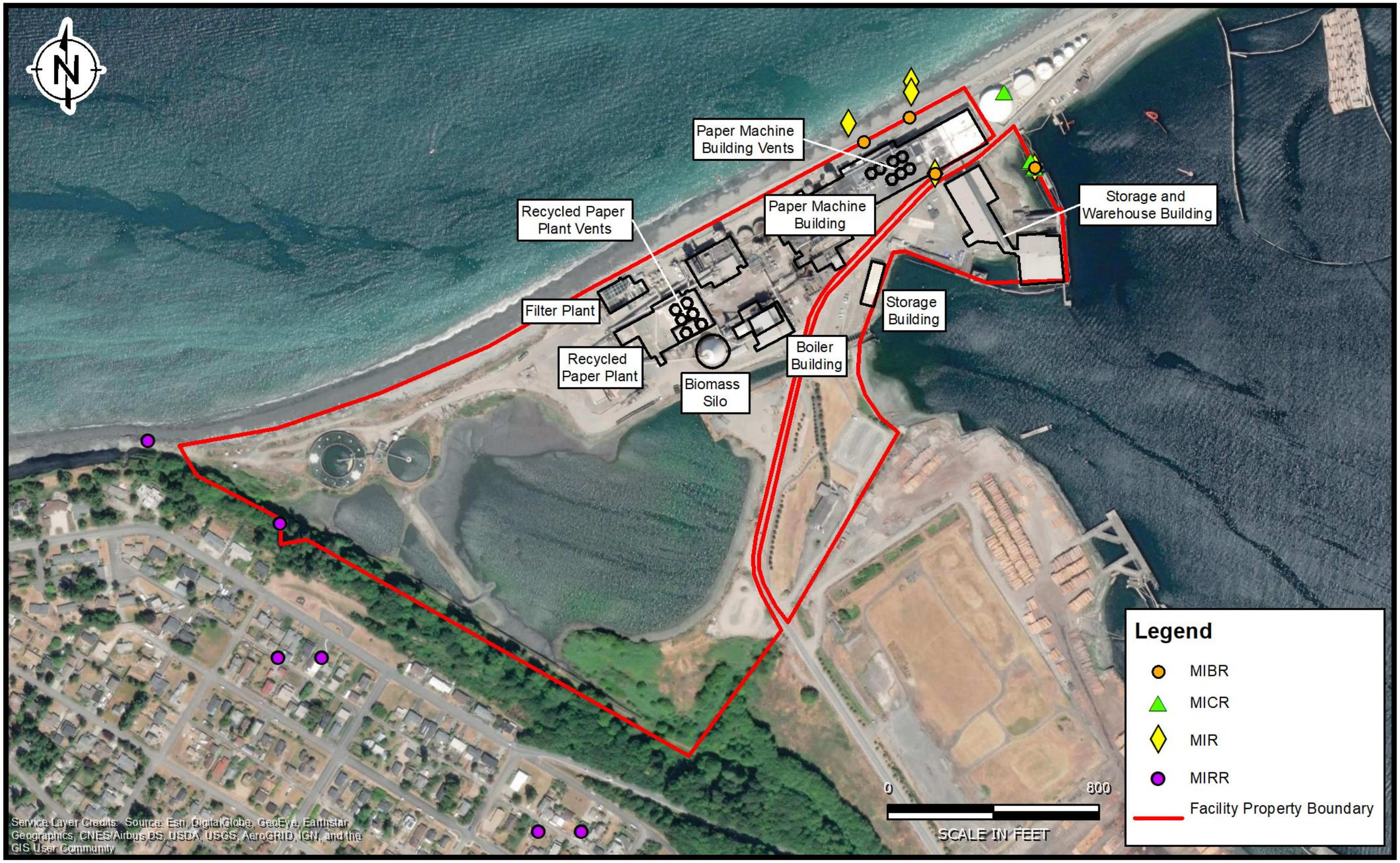


Figure 5-2: Locations of Receptors of Interest

Table 5-1: Nearest Sensitive Receptors

Type of Receptor	Name	Address	Distance (km / mi)
Convalescent Home	Village Concepts of Port Angeles	1430 Park View Ln, Port Angeles, WA 98363	1.3 / 0.8
	Peninsula Manor	1017 W 17th St., Port Angeles, WA 98363	2.4 / 1.5
	Caregivers Home Health Inc.	622 E. Front St., Port Angeles, WA 98363	3.7 / 2.3
Medical Facility	North Olympic Healthcare Network	240 W Front St. A., Port Angeles, WA 98362	2.5 / 1.6
	Peninsula Children's Clinic	303 W 8th St., Port Angeles, WA 98362	2.7 / 1.7
	OMP Primary Care Clinic	433 E 8th St., Port Angeles, WA 98362	3.6 / 2.2
	Olympic Medical Center	939 Caroline St., Port Angeles, WA 98362	4.1 / 2.5
School	Hamilton Elementary School	1822 W. 7th St., Port Angeles, WA 98363	0.9 / 0.6
	Lincoln High School	924 W 9th St., Port Angeles, WA 98363	1.9 / 1.2
	Stevens Middle School	1139 W. 14th St., Port Angeles, WA 98363	4.3 / 2.7
Other	Vern Burton Community Center	308 E. 4th St., Port Angeles, WA 98362	3.2 / 2.0
	Port Angeles Community Center	328 E. 7th St., Port Angeles, WA 98362	3.4 / 2.1

6. EXPOSURE ASSESSMENT

The exposure assessment describes the routes and manner by which receptors identified in the previous section may be exposed to TAPs emitted from the Facility. Concentration increases to which receptor populations may be exposed and key exposure assumptions are also described.

6.1 Identification of Exposure Pathways

Receptors presented in the previous section, residents, workers, and sensitive subpopulations, may be exposed to chemicals in the environment. Specifically, contact with emissions from the Facility may occur primarily through direct inhalation. Contact with emissions attributable to the Facility may also occur indirectly, through incidental ingestion of and skin contact with emissions deposited on area surface soils. However, for the TAPs of interest to this study, indirect exposures through ingestion and skin contact pathways are not considered significant in comparison with the direct inhalation pathway.

Ecology's air toxics review guidance document references California Air Toxic Hot Spots Program guidance[15] to assess the need for consideration of other indirect exposure pathways in addition to consideration of inhalation exposure. Formaldehyde and methylene chloride are not chemicals for which the California Air Toxic Hot Spots Program recommends consideration of multiple exposure pathways. Typically, chemicals considered for alternate ingestion pathways (e.g., soil, produce, breast milk, livestock/game, etc.) are those that are persistent and bio-accumulative. Formaldehyde and methylene chloride do not bio-accumulate, and are, therefore, not prioritized for multi-pathway evaluation. Based on Ecology and California Air Toxic Hot Spots Program guidance, inhalation is the only exposure pathway assessed in this HIA.

6.2 Exposure Concentrations

Tables 6-1 and 6-2 present maximum airborne exposure concentrations (ECs) of the one TAP that exceed the ASIL (formaldehyde) along with the one TAP that exceeds the SQER and has potential health impacts (methylene chloride) at the MIBR, MIR, MIRR, and MICR. For non-cancer hazards associated with the two TAPs, acute exposures were estimated using the model-predicted 1-hour and 8-hour average concentrations at the maximally-impacted receptors, and the annual average concentrations at the maximally-impacted receptors were used to estimate chronic exposures.

---

[15] Office of Environmental Health Hazard Assessment (OEHHA), California Environmental Protection Agency, "Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments." February 2015. https://oehha.ca.gov/media/downloads/crnr/2015guidancemanual.pdf

Table 6-1: Exposure Concentrations for the MIBR and MIR

TAP	MIBR			MIR		
	Period	Concentration (µg/m³)	Zone 10 UTM Coordinates (m)	Period	Concentration (µg/m³)	Zone 10 UTM Coordinates (m)
Formaldehyde	1-hr	5.59E+00	465639, 5331555	1-hr	5.59E+00	465639, 5331555
	8-hr	2.73E+00	465639, 5331555	8-hr	2.98E+00	465496, 5331654
	24-hr	1.77E+00	465442, 5331586	24-hr	1.96E+00	465424, 5331606
	Annual	7.29E-01	465524, 5331548	Annual	7.29E-01	465524, 5331548
Methylene Chloride	1-hr	2.22E+00	465639, 5331555	1-hr	2.22E+00	465639, 5331555
	8-hr	1.14E+00	465495, 5331614	8-hr	1.24E+00	465496, 5331642
	24-hr	7.05E-01	465442, 5331586	24-hr	7.81E-01	465424, 5331606
	Annual	3.15E-01	465524, 5331548	Annual	3.15E-01	465524, 5331548

Table 6-2: Exposure Concentrations for the MIRR and MICR

TAP	MIRR			MICR		
	Period[1]	Concentration (µg/m³)	Zone 10 UTM Coordinates (m)	Period	Concentration (µg/m³)	Zone 10 UTM Coordinates (m)
Formaldehyde	1-hr	1.06E+00	465266, 5330690	1-hr	5.59E+00	465639, 5331555
	8-hr	2.79E-01	465066, 5330790	8-hr	2.73E+00	465639, 5331555
	24-hr	1.24E-01	465066, 5330790	24-hr	1.41E+00	465639, 5331555
	Annual	1.63E-03	465116, 5330790	Annual	5.46E-01	465635, 5331564
Methylene Chloride	1-hr	5.30E-01	464769, 5331146	1-hr	2.22E+00	465639, 5331555
	8-hr	1.30E-01	464816, 5330990	8-hr	1.09E+00	465639, 5331555
	24-hr	5.70E-02	464766, 5330990	24-hr	5.71E-01	465604, 5331642
	Annual	2.94E-03	464616, 5331240	Annual	2.38E-01	465635, 5331564

Notes:

[1] This table presents the 4-year maximum 1-hr, 8-hr, 24-hr, and annual-average concentrations. These concentrations are used to calculate the non-cancer and cancer risks which are presented in Section 8.

6.2.1 Calculating ECs

It is important to note that EPA and OEHHA offer slightly different guidance for assessing chronic hazards to offsite workers. EPA recommends adjusting the long-term exposure concentration to account for the fact that workers may not be present in the vicinity of a facility on a continuous basis.[16] In the absence of 8-hour reference exposure levels (RELs), OEHHA recommends using the chronic REL and the annual average air concentration at maximally impacted commercial receptors without adjustments to estimate chronic hazards at nearby workplace. OEHHA guidance also notes that if available, "the 8-hour RELs can be used to evaluate the potential for health impacts (including effects of repeated exposures) in offsite workers, and to children and teachers exposed during school hours."[17] In this analysis, an 8-hour REL is available for formaldehyde from OEHHA and used to estimate the potential for health impacts at the MICR. An 8-hour REL is not available for methylene chloride from OEHHA, and therefore, the chronic REL was used to estimate the potential for health impacts, in accordance with OEHHA guidance.

ECs for increased cancer risk are based on the maximum annual modeled air concentration, modified by a representative exposure time (ET), exposure frequency (EF), exposure duration (ED), and averaging time (AT), as shown in the following equation:

$$EC\left(\frac{\mu g}{m^3}\right) = \frac{modeled\ air\ concentration\ \left(\frac{\mu g}{m^3}\right) \times ET \times EF \times ED}{AT}$$

Exposure parameter values used to calculate the increased cancer risk ECs are presented in Table 6-3. The exposure parameters applied to maximum impact receptors are dependent on the zoning in the region where the receptor resides. The majority of MIRs are located in commercial/industrial zones and intermittent commercial exposure parameters are used to calculate EC values at these receptors.

Table 6-3: Exposure Parameters Used to Calculate ECs for Increased Cancer Risk

Exposure Parameter	Residential	Commercial / Industrial Worker (Continuous)	Commercial / Industrial Worker (Intermittent)
ET (hours per day)	24	8	2
EF (days per year)	365	250	250
ED (years)	70	40	30
AT (hours; 70 years x 365 days/year x 24 hr/day)	613,200	613,200	613,200
Fraction of 70-Year Continuous Exposure	1	0.13	0.024

---

[16] United States Environmental Protection Agency (EPA), "Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part F, Supplemental Guidance for Inhalation Risk Assessment) Final," EPA-540-R-070-002.January 2009. https://www.epa.gov/sites/production/files/2015-09/documents/partf_200901_final.pdf

[17] Office of Environmental Health Hazard Assessment (OEHHA), California Environmental Protection Agency, "Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments." February 2015. https://oehha.ca.gov/media/downloads/crnr/2015guidancemanual.pdf

6.2.2 Background ECs

Ambient background concentrations were estimated using the latest NATA data (see Section 4.3) for the area in which the Facility is located.

6.2.3 Combining Calculated and Background ECs

WAC 173-460-090(5) requires that TAP background concentrations be "considered" as part of an HIA. To that end, calculated ECs based on the modeled concentrations were combined with background air concentrations from the NATA. Although the resulting combined ECs were used to estimate non-cancer hazards and cancer risk for all identified receptor groups, EPA states that NATA assessments should not be used "to pinpoint specific risk values in small areas such as a census tract."[18]

---

[18] https://www.epa.gov/national-air-toxics-assessment/nata-overview#what-nata-is-not

7. TOXICITY ASSESSMENT

This section contains specific information on the toxicity of the TAPs of concern: formaldehyde and methylene chloride. This evaluation includes a description of the toxic effects and the general levels of exposure associated with these effects in order to evaluate the risks. Additionally, a summary of the toxicokinetics of exposure via inhalation is included. Toxicity estimates from EPA (IRIS and the National Ambient Air Quality Standards (NAAQS)), ATSDR, and OEHHA were compiled to quantify estimates of acute and chronic non-cancer hazard and cancer risk. Table 7-1 provides the non-cancer values for the one TAP over the ASIL and the one TAP over the SQER that acts upon the same primary target organ system. Table 7-2 provides the cancer unit risk values for the carcinogenic TAPs considered in this analysis.

Table 7-1: Non-Cancer, Toxicity Values from EPA, OEHHA, and ATSDR

Toxic Air Pollutant (TAP) [1]	**Source** [2]	**Type**	**Inhalation Value ($\mu g/m^3$)**
Formaldehyde [3]	OEHHA	Acute REL	5.50E+01
		8-Hour REL	9.00E+00
		Chronic REL	9.00E+00
	ATSDR	Acute MRL	5.04E+01
		Intermediate MRL	3.78E+01
		Chronic MRL	1.01E+00
Methylene Chloride	EPA	Chronic RfC	6.00E+02
	OEHHA	Acute REL	1.40E+04
		Chronic REL	4.00E+02
	ATSDR	Acute MRL	2.12E+03
		Intermediate MRL	1.06E+03
		Chronic MRL	1.06E+03

Notes:

[1] TAPs included in this table may exceed the SQER and have toxicity values based on the same target organs or target organ system as those that exceed ASIL

[2] EPA IRIS Assessments updated Nov 18, 2011. https://cfpub.epa.gov/ncea/iris2/chemicalLanding.cfm?&substance_nmbr=70. Accessed Feb 25, 2019.

OEHHA Reference Exposure Levels (RELs) updated June 28, 2016. https://oehha.ca.gov/air/general-info/oehha-acute-8-hour-and-chronic-reference-exposure-level-rel-summary

ATSDR Minimal Risk Levels (MRLs) updated August 2018. https://www.atsdr.cdc.gov/mrls/pdfs/atsdr_mrls.pdf

[3] EPA chronic Reference Concentration (RfC) for chronic inhalation exposure for formaldehyde of 9.8 ug/m^3 not listed as assessed under IRIS program, however available through EPA sources (RSL table)

Table 7-2: Cancer Inhalation Unit Risk Values

Toxic Air Pollutant (TAP)[1]	**Inhalation Unit Risk (($\mu g/m^3)^{-1}$)**	
	EPA[2]	**OEHHA**[3]
Formaldehyde	1.30E-05	6.00E-06
Methylene Chloride	1.00E-08	1.00E-06

Notes:

[1] Chemicals included in this table exceed the SQER and have toxicity values based on the same target organs or target organ system as those that may exceed the ASIL

[2] EPA inhalation unit risk levels as of February 2019. https://www.epa.gov/iris

[3] OEHHA inhalation unit risk levels as of February 2019. https://oehha.ca.gov/chemicals

7.1 Formaldehyde

Formaldehyde is a respiratory irritant, as described in Section 3. The EPA does not provide an RfC for formaldehyde exposure by inhalation. However, OEHHA provides an acute, 8-hour, and an annual REL, and ATSDR provides an acute, intermediate, and annual MRL.

The chronic and 8-hour RELs defined by OEHHA are the same, at 9 $\mu g/m^3$, and are based on the same study showing nasal and eye irritation from occupational exposure over an average of 10 years.[19] The Lowest Observed Adverse Effect Level (LOAEL) from the study was identified as 0.26 mg/m^3, and the No Observed Adverse Effect Level (NOAEL) was found to be 0.09 mg/m^3. The NOAEL was used to determine both the chronic and 8-hour RELs, after applying an uncertainty factor of 10 to account for sensitive populations (asthma in children).

The ATSDR chronic MRL is 10 $\mu g/m^3$ (8 ppb). It is based on a study of occupational exposures resulting in histological changes in nasal tissue over an average of 10 years of exposure.[20] This toxicity value is very similar to OEHHA's chronic REL.

An acute REL of 55 $\mu g/m^3$ was derived from a human study of 19 healthy subjects given short-term exposures to formaldehyde with an endpoint of eye irritation.[21] OEHHA chose a NOAEL of 0.5 ppm and a LOAEL of 1 ppm, from which a benchmark concentration of 0.44 ppm was derived. An uncertainty factor of 10 was added to account for asthma exacerbation in children. The ATSDR acute MRL is a similar value, at 50 $\mu g/m^3$ (40 ppb). This value is based on a LOAEL of 400 ppb from a study of human volunteers reporting itching, sneezing, mucosal congestion, and a burning sensation in the

---

[19] Wilhelmsson B, and Holmstrom M. 1992. Possible mechanisms of formaldehyde-induced discomfort in the upper airway. Scand. J. Work. Environ. Health 18(6):403-407.

[20] Holmstrom M, Wilhelmsson B, Hellquist H, et al. 1989. Histological changes in the nasal mucosa in persons occupationally exposed to formaldehyde alone and in combination with wood dust. Acta Otolaryngol (Stockh) 107:120-129.

[21] Kulle TJ, Sauder LR, Hebel JR, Green DJ and Chatham MD. 1987. Formaldehyde dose-response in healthy nonsmokers. Japca 37(8): 919-24.

eyes and nasal passages after a 2-hour exposure.[22] An uncertainty factor of 9 was applied for use of a LOAEL instead of a NOAEL and to account for sensitive populations.

The intermediate MRL was derived by ATSDR based on lesions in the nasal epithelium and other signs of nasopharyngeal irritation in Cynomolgus monkeys exposed to formaldehyde by inhalation for 26 weeks for 7 days/week, 22 hours/day.[23] A LOAEL of 2.95 ppm was identified. ATSDR applied an uncertainty factor of 10 for human variability and 3 for interspecies extrapolation to result in an MRL of 0.03 ppm (37 µg/m^3). This exposure time is not standard for air modeling; this toxicity value was not used quantitatively in the Risk Characterization.

The EPA's cancer weight-of-evidence characterization for formaldehyde states that there is limited human evidence and sufficient animal evidence to classify it as a probable human carcinogen. Limited human studies of carcinogenicity focused on cancers of the lung and nasopharynx from persons exposed occupationally. EPA's inhalation unit risk factor is 1.3E-5 per µg/m^3. The OEHHA inhalation unit risk factor is 6E-6 per µg/m^3, based on nasal squamous carcinoma data in rats and supported by a human occupational exposure study.[24,25]

7.2 Methylene Chloride

The EPA's RfC (600 µg/m^3) is derived from a study cited in the EPA's chemical assessment summary in which 180 rats (90 female, 90 male) were exposed to various levels of dichloromethane for 6 hours/day, 5 days/week for 2 years.[26,27]

The acute and chronic RELs defined by the OEHHA are 14,000 µg/m^3 and 400 µg/m^3, respectively. The acute REL was obtained from a study in which twelve healthy adults were exposed to 195 ppm of methylene chloride for 90 minutes.[28,29] The chronic REL was based on results from a study of workers

---

[22] Pazdrak K, Gorski P, Krakowiak A, et al. 1993. Changes in nasal lavage fluid due to formaldehyde inhalation. Int Arch Occup Environ Health 64:515-519.

[23] Rusch GM, Clary JJ, Rinehart WE, et al. 1983. A 26-week inhalation toxicity study with formaldehyde in the monkey, rat, and hamster. Toxicol Appl Pharmacol 68:329-343.

[24] Kerns WD, Pavkov KL, Donofrio DJ, Gralla EJ and Swenberg JA. 1983. Carcinogenicity of formaldehyde in rats and mice after long-term inhalation exposure. Cancer Res 43:4382-4392.

[25] U.S. Environmental Protection Agency (EPA) 1987. Assessment of Health Risks to Garment Workers and Certain Home Residents from Exposure to Formaldehyde. Office of Pesticide and Toxic Substances.

[26] Nitschke, KD; Burek, JD; Bell, TJ; et al. 1988a. Methylene chloride: a 2-year inhalation toxicity and oncogenicity study in rats. Fundam Appl Toxicol 11:48–59, as cited in USEPA 2011.

[27] U.S. Environmental Protection Agency (USEPA). 2011. Chemical assessment summary – methylene chloride. https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0070_summary.pdf#nameddest=rfc

[28] Putz VR, BL Johnson, and J V Setzer. 1979. A comparative study of the effects of carbon monoxide and methylene chloride on human performance. Journal of environmental pathology and toxicology 2:97–112, as cited in OEHHA 1999a.

[29] Office of Environmental Health Hazard Assessment (OEHHA). 1999a. Acute RELs and toxicity summaries using the previous version of the Hot Spots Risk Assessment guidelines. https://oehha.ca.gov/media/downloads/crnr/appendixd2final.pdf

who were exposed to average measured concentrations of 40 ppm during the workday.[30,31] Three of the participants wore personal monitors which assisted in determining the LOAEL of 33 ppm. Several factors contributed to uncertainty including age, background carboxyhemoglobin status, and activity level.

Acute, intermediate, and chronic MRLs are provided by the ATSDR. The acute MRL is 2,120 µg/m^3 (0.6 ppm). To support these values, ATSDR cited a randomized blind experiment in which volunteers were exposed to either filtered air or concentrations of methylene chloride vapors (300, 500, or 800 ppm) for 3-4 hours over 45-minute intervals.[32,33] A LOAEL of 300 ppm was established after physical effects were observed at 500 ppm and beyond. An uncertainty factor of 10 was assigned for use of a LOAEL and an additional 10 was applied for human variability.

The intermediate and chronic MRLs are the same value 1,060 µg/m^3 (0.3 ppm), for methylene chloride. The intermediate MRL was derived from a study in which rats were exposed to methylene chloride for 14 weeks.[34,35] A LOAEL of 25 ppm was derived from data supporting hepatic effects of fatty infiltration and cytoplasmic vacuolization. An uncertainty factor of 16 was assigned which included 3 for use of a minimal LOAEL, 3 for extrapolation from animals to humans, and 10 for human variability. ATSDR determined the chronic MRL via a bioassay study cited in the toxicology profile for methylene chloride published by the ATSDR.[36,37] Rats were exposed to different concentrations (0,50,200, and 500 ppm) of methylene chloride for 6 hours/day, 5 days/week for 2 years. A NOAEL of 50 ppm was determined. The uncertainty factor of the NOAEL was 13.

The EPA has concluded that methylene chloride is likely to be carcinogenic to humans. Multiple studies exposing mice to methylene chloride and monitoring health outcomes are the basis for this characterization. Several of these studies provide evidence of increases in malignant mammary

---

[30] DiVincenzo GD, and CJ Kaplan. 1981. Uptake, metabolism, and elimination of methylene chloride vapor by humans. Toxicology and applied pharmacology 59:130–140, as cited in OEHHA 1999b.

[31] Office of Environmental Health Hazard Assessment (OEHHA). 1999b. Chronic RELs and toxicity summaries using the previous version of the Hot Spots Risk Assessment guidelines. https://oehha.ca.gov/media/downloads/crnr/appendixd3final.pdf

[32] Winneke G. 1974. Behavioral effects of methylene chloride and carbon monoxide as assessed by sensory and psychomotor performance. In: Behavioral Toxicology. Early Detection of Occupational Hazards, Eds. C Xintaras, B Johnson, I deGroot. U.S. Department of Health, Education, and Welfare, as cited in ATSDR 2000.

[33] Agency for Toxic Substances ad Disease Registry (ATSDR). 2000. Toxicological profile for methylene chloride. https://www.atsdr.cdc.gov/toxprofiles/tp14.pdf

[34] Haun CC, Vernot EH, Darmer KI, et al. 1972. Continuous animal exposure to low levels of dichloromethane. AMRL-TR-72-130, paper no. 12, as cited in ATSDR 2000.

[35] Burek, JD; Nitschke, KD; Bell, TJ; et al. 1984. Methylene chloride: a two-year inhalation toxicity and oncogenicity study in rats and hamsters. Fundam Appl Toxicol 4:30–47, as cited in USEPA 2011.

[36] Nitschke KD, Burek JD, Bell TJ, et al. 1988b. Methylene Chloride: A 2-year inhalation toxicity and oncogenicity study in rats. Fundam Appl Toxicol 11:60-67, as cited in ATSDR 2000.

[37] Agency for Toxic Substances and Disease Registry (ATSDR). 2000. Toxicology profile for methylene chloride. https://www.atsdr.cdc.gov/toxprofiles/tp14.pdf

tumors.[38,39] The EPA's inhalation unit risk factor is 1.00E-08 µg/m^3. Studies in humans provided evidence of carcinogenicity. The OEHHA inhalation unit risk factor is 1.0E-06 µg/m^3. human and animal studies showcasing relationships between exposure to methylene chloride and cancer mortality were referenced by the OEHHA to support this value.[40,41,42,43]

---

[38] NTP (National Toxicology Program). 1986. Toxicology and carcinogenesis studies of dichloromethane (methylene chloride) (CAS No. 75-09-2) in F344/N rats and B6C3F1 mice (inhalation studies). Public Health Service, U.S. Department of Health and Human Services; NTP TR 306. Available from the National Institute of Environmental Health Sciences, Research Triangle Park, NC. Available online at http://ntp.niehs.nih.gov/ntp/htdocs/LTrpts/tr306.pdf (210 pp, 9M), as cited in USEPA 2011.

[39] Maltoni, C; Cotti, G; Perino, G. 1988. Long-term carcinogenicity bioassays on methylene chloride administered by ingestion to Sprague-Dawley rats and Swiss mice and by inhalation to Sprague-Dawley rats. Ann NY Acad Sci 534:352–366, as cited in USEPA 2011.

[40] Hearne FT, Pifer JW, Friedlander BR and Raleigh RL. 1987. Methylene chloride mortality study: dose response to characterization and animal model comparison. J Occup Med 29:217- 228, as cited in OEHHA 2011.

[41] Friedlander BR, Pifer JW and Hearne FT. 1985. 1964 Methylene Chloride Cohort Mortality Study: Update Through 1984. Eastman Kodak Company, Rochester, NY, as cited in OEHHA 2011.

[42] Dow Chemical Company. 1980. Methylene Chloride: A Two-Year Inhalation Toxicity and Oncogenicity Study in Rats and Hamsters, Follow-Up Response A. FYI-OTS-0281-0097. U.S. Environmental Protection Agency, Office of Toxic Substances, Washington, DC, as cited in OEHHA 2011.

[43] Office of Environmental Health Hazard Assessment (OEHHA). 2011. Appendix B: Chemical Specific Summaries of the Information Used to Drive Unit Risk and Cancer Potency Values. https://oehha.ca.gov/media/downloads/crnr/appendixb.pdf

8. RISK CHARACTERIZATION

For the risk characterization, the results of the exposure and toxicity assessments described in Sections 6 and 7 were integrated into quantitative or qualitative estimates of potential health hazards. Cancer risk and non-cancer hazard estimates attributable to TAP emission increases attributable to the project were quantified at the MIBR, MIR, MIRR, and MICR. Where available, background concentrations described in Section 4.3 were combined with the estimated concentration increase attributable to the project, and combined non-cancer hazard quotients and cancer risks were calculated.

8.1 Calculation of Non-Cancer Hazards

The potential for non-cancer adverse health effects from exposure to formaldehyde and methylene chloride emission increases attributable to the project was investigated by comparing exposure concentration increases at the identified receptor locations to relevant non-cancer toxicity values presented in Table 7-1. A concentration increase that exceeds the relevant value indicates the potential for an adverse health effect. The magnitude of the potential for an adverse health effect is quantified by the hazard quotient (HQ), which is calculated by dividing the exposure concentration by the relevant toxicity value. An HQ of one (1) or less indicates that the predicted exposure is unlikely to result in adverse non-cancerous health effects. Values greater than one do not necessarily indicate that health effects are certain, but do warrant a need for further evaluation.

The non-cancer hazards associated with the exposure concentrations from formaldehyde and methylene chloride compounds were calculated and summed. The summed hazard quotients for each averaging period and receptor result in a hazard index (HI) that accounts for overall hazards for all non-cancer health effects of each TAP combined. Non-cancer hazards are presented with ranges where the toxicity values from two major agencies (OEHHA, and ATSDR) are carried through the analysis, as described in Section 7.

Maximally Impacted Receptor (MIR)

Risk characterization at the maximally impacted receptor (MIR) is located along the facility boundary. At the MIR, a person was assumed to be intermittently exposed, 2 hours per day, 250 days per year, to project emission increases for 30 years. All identified MIRs were located in a commercial/industrial zone.

Table 8-1 presents the hazard quotients and hazard indices for the 1-hr and 8-hr averaging period. The hazard indices for 1-hour and 8-hour exposures are generally consistent and are all less than one.

Due to the variability in toxicity values provided by multiple agencies, the highest and lowest annual non-cancer hazards for the MIR vary, as shown in Table 8-2. The annual hazard index is less than one (no adverse health effects would be expected to occur). The combined non-cancer hazard quotients are slightly higher compared to facility-only hazard quotients but also less than one.

Table 8-1: Non-Cancer Hazard Quotients for MIR, 1-hour and 8-hour

TAP	1-hour		8-hour	
	OEHHA	**ATSDR**	**OEHHA**	**ATSDR**
Formaldehyde	1.02E-01	1.11E-01	3.31E-01	7.89E-02
Methylene Chloride	1.59E-04	1.05E-03	3.09E-03	1.17E-03
Hazard Index	**0.10-0.11**		**0.08-0.33**	

Notes:
- The 8-hour OEHHA methylene chloride hazard index is based on the chronic REL and maximum 8-hour modeled concentration, in accordance with OEHHA gudiance.
- EPA IRIS does not report 1-hour or 8-hour toxicity values, thus hazard indices were not included in Table 8-1.

Table 8-2: Non-Cancer Hazard Quotients for MIR, Annual

TAP	Project Only			Combined (Project + Background)		
	EPA	**OEHHA**	**ATSDR**	**EPA**	**OEHHA**	**ATSDR**
Formaldehyde		8.10E-02	7.24E-02		1.89E-01	1.69E-01
Methylene Chloride		7.87E-04	2.97E-04		1.54E-03	5.81E-04
Hazard Index	**0.07-0.08**			**0.17-0.19**		

Notes:
- Grayed cells indicate no toxicity value is available from respective agencies, and thus hazard indices are not calculated.

Maximally Impacted Boundary Receptor (MIBR)

Hazard quotients for the MIBR are presented in Tables 8-3 and 8-4 and are similar to HQs calculated for the MIR. Similar to results at the MIR, HI values for formaldehyde and methylene chloride are less than 1. At the MIBR, it is assumed that a human is located at the MIBR for 2 hours a day, 250 days per year, for 30 years.

Table 8-3: Non-Cancer Hazard Quotients for MIBR, 1-hour and 8-hour

TAP	1-hour		8-hour	
	OEHHA	**ATSDR**	**OEHHA**	**ATSDR**
Formaldehyde	1.02E-01	1.11E-01	3.03E-01	7.22E-02
Methylene Chloride	1.59E-04	1.05E-03	2.84E-03	1.07E-03
Hazard Index	**0.10-0.11**		**0.07-0.31**	

Notes:
- The 8-hour OEHHA methylene chloride hazard index is based on the chronic REL and maximum 8-hour modeled concentration, in accordance with OEHHA gudiance.
- EPA IRIS does not report 1-hour or 8-hour toxicity values, thus hazard indices were not included in Table 8-3.

Table 8-4: Non-Cancer Hazard Quotients for MIBR, Annual

TAP	Project Only			Combined (Project + Background)		
	EPA	OEHHA	ATSDR	EPA	OEHHA	ATSDR
Formaldehyde		8.10E-02	7.24E-02		1.89E-01	1.69E-01
Methylene Chloride		7.87E-04	2.97E-04		1.54E-03	5.81E-04
Hazard Index	**0.07-0.08**			**0.17-0.19**		

Notes:
- Grayed cells indicate no toxicity value is available from respective agency, and thus hazard index is not calculated.

Maximally Impacted Residential Receptor (MIRR)

The MIRR represents a worst-case scenario for a residential receptor. The non-cancer hazards at the MIRR are lower than non-cancer hazards at the MIR and MIBR. As shown in Table 8-5, the acute 1-hour hazard index is less than 1 (range 0.019 to 0.021), as is the 8-hour hazard index (range 0.01 to 0.03). The annual facility-only scenario hazard index shown in Table 8-6 is less than 1 (0.0002) and the combined hazard index ranges from 0.01 to 0.03. As with the MIR and MIBR, no adverse health effects would be expected to occur at the MIRR.

Table 8-5: Non-Cancer Hazard Quotients for MIRR, 1-hour and 8-hour

TAP	1-hour		8-hour	
	OEHHA	ATSDR	OEHHA	ATSDR
Formaldehyde	1.93E-02	2.11E-02	3.10E-02	7.39E-03
Methylene Chloride	3.78E-05	2.50E-04	3.24E-04	1.23E-04
Hazard Index	**0.019-0.021**		**0.01-0.03**	

Notes:
- The 8-hour OEHHA methylene chloride hazard index is based on the chronic REL and maximum 8-hour modeled concentration, in accordance with OEHHA gudiance.
- EPA IRIS does not report 1-hour or 8-hour toxicity values, thus hazard indices were not included in Table 8-5.

Table 8-6: Non-Cancer Hazard Quotients for MIRR, Annual

TAP	Project Only			Combined (Project + Background)		
	EPA	OEHHA	ATSDR	EPA	OEHHA	ATSDR
Formaldehyde		1.81E-04	1.62E-04		1.08E-01	9.67E-02
Methylene Chloride		7.35E-06	2.78E-06		7.60E-04	2.87E-04
Hazard Index	**0.0002**			**0.10-0.11**		

Notes:
- Grayed cells indicate no toxicity value is available from respective agency, and thus hazard index is not calculated.

Maximally Impacted Commercial Receptor (MICR)

The maximally impacted commercial receptor (MICR) is located where a commercial facility currently operates. The MICR hazard indices are similar compared to the hazard indices at the MIR and MIBR. The hazard index shows that for 1-hour and 8-hour exposures, no adverse health effects are expected to occur (Table 8-7). The annual non-cancer hazard indices at the MICR range from 0.05 to 0.06 for the facility-only scenario and 0.15 to 0.17 for the combined scenario. Again, the hazard indices are less than one and no adverse health effect are expected to occur.

Table 8-7: Non-Cancer Hazard Quotients for MICR, 1-hour and 8-hour

TAP	**1-hour**		**8-hour**	
	OEHHA	**ATSDR**	**OEHHA**	**ATSDR**
Formaldehyde	1.02E-01	1.11E-01	3.03E-01	7.22E-02
Methylene Chloride	1.59E-04	1.05E-03	2.71E-03	1.02E-03
Hazard Index	**0.10-0.11**		**0.07-0.31**	

Notes:
- The 8-hour OEHHA methylene chloride hazard index is based on the chronic REL and maximum 8-hour modeled concentration, in accordance with OEHHA gudiance.
- EPA IRIS does not report 1-hour or 8-hour toxicity values, thus hazard indices were not included in Table 8-7.

Table 8-8: Non-Cancer Hazard Quotients for MICR, Annual

TAP	**Project Only**			**Combined (Project + Background)**		
	EPA	**OEHHA**	**ATSDR**	**EPA**	**OEHHA**	**ATSDR**
Formaldehyde	▨	6.07E-02	5.42E-02	▨	1.69E-01	1.51E-01
Methylene Chloride	▨	5.95E-04	2.25E-04	▨	1.35E-03	5.09E-04
Hazard Index	**0.05-06**			**0.15-0.17**		

Notes:
- Grayed cells indicate no toxicity value is available from respective agency, and thus hazard indices are not calculated.

8.2 Quantifying Increased Cancer Risks

Increased cancer risks represent the hypothetical increase in cancers per number of people exposed. For example, a cancer risk of 1E-06 means that one additional cancer may occur for one million people exposed. According to Ecology, the acceptable increased cancer risk is no more than 10 per million or 1E-05. An increased cancer risk value at a given location is calculated by multiplying the annual air concentration increase predicted for a TAP at that location by the inhalation unit risk factor for that TAP (provided in Table 7-2).

This calculation was performed for formaldehyde and methylene chloride at the MIR, MIBR, MIRR, and MICR. For the TAPs presented in the following tables, EPA and OEHHA provide different inhalation unit risk factors and separate risk calculations were performed using these values. Combined cancer risks were calculated by combining the total cancer risk increase attributable to project emission increases with the risk attributable to NATA background concentrations and summing across all TAPs.

Maximally Impacted Receptor (MIR)

The MIRs for annual-averaged formaldehyde and methylene chloride are located in a commercial/industrial zone and intermittent commercial exposure parameters (Table 6-3) were used to calculate cancer risks associated with these TAPs. The sum of increased cancer risks at the MIR ranges from 0.1 per million to 0.2 per million for the project-only scenario, which is less than the Ecology cancer risk threshold. For the combined scenario, cancer risks range from 0.3 per million to 0.5 per million.

Table 8-9: Cancer Risks for MIR

TAP	Project Only		Combined (Project + Background)	
	EPA	OEHHA	EPA	OEHHA
Formaldehyde	2.32E-07	1.08E-07	5.41E-07	2.52E-07
Methylene Chloride	7.70E-11	7.70E-09	1.51E-10	1.51E-08
Sum of Cancer Risk	**2E-07**	**1E-07**	**5E-07**	**3E-07**

Maximally Impacted Boundary Receptor (MIBR)

Calculated increased cancer risks at the MIBR attributable to project emission increases do not exceed the Ecology cancer risk threshold of 10 per million.

Table 8-10: Cancer Risks for MIBR

TAP	Project Only		Combined (Project + Background)	
	EPA	OEHHA	EPA	OEHHA
Formaldehyde	2.32E-07	1.08E-07	5.41E-07	2.52E-07
Methylene Chloride	7.70E-11	7.70E-09	1.51E-10	1.51E-08
Sum of Cancer Risk	**2E-07**	**1E-07**	**5E-07**	**3E-07**

Maximally Impacted Residential Receptor (MIRR)

At the MIRR, cancer risks range from 0.01 per million to 0.02 per million for the project-only scenario, when considering the EPA and OEHHA unit risk factors. When the background concentrations are combined with those attributable to the project, cancer risks range from 6 to 10 per million. Exposure at residential receptors was conservatively assumed to occur every hour for 70 years, which is conservative, so it is likely that the actual cancer risk increase is less than that presented in Table 8-11.

Table 8-11: Cancer Risks for MIRR

TAP	Project Only		Combined (Project + Background)	
	EPA	OEHHA	EPA	OEHHA
Formaldehyde	2.12E-08	9.88E-09	1.27E-05	5.91E-06
Methlyene Chloride	2.94E-11	2.94E-09	3.04E-09	3.04E-07
Sum of Cancer Risk	**2E-08**	**1E-08**	**1E-05**	**6E-06**

Maximally Impacted Commercial Receptor (MICR)

Increased cancer risks at the MICR are less than the Ecology cancer risk threshold when either EPA and OEHHA toxicity values are used.

Table 8-12: Cancer Risks for MICR

TAP	Project Only		Combined (Project + Background)	
	EPA	OEHHA	EPA	OEHHA
Formaldehyde	9.27E-07	4.32E-07	2.58E-06	1.20E-06
Methylene Chloride	3.10E-10	3.10E-08	7.03E-10	7.03E-08
Sum of Cancer Risk	**9E-07**	**5E-07**	**3E-06**	**1E-06**

9. DISCUSSION AND CONCLUSIONS

9.1 Uncertainty Characterization

The HIA involves several assumptions, each with an associated uncertainty. In particular, there are uncertainties associated with emission rate calculations, air dispersion modeling, background concentrations, and toxicity values.

9.1.1 Emissions Rate Calculations

An emission rate, which is a quantity of pollutant per unit time (e.g., pounds per hour), is calculated from an emission factor, which is a quantity of pollutant per unit of an activity (e.g., pounds per ton of paper produced), and an activity rate, which is a measure of an activity per unit time (e.g., tons of paper produced per day).

For analyses conducted in support of a permitting action, worst-case emission factors and activity rates are employed to ensure that regulatory limits or levels are not exceeded. Regarding activity rates, all equipment were assumed to operate at the maximum capacity in continuous, year-round operation.

As a result of these highly health-protective assumptions, the exposures calculated by the model and the risk characterizations presented in this report are likely to overstate, rather than underestimate, the potential risks to all receptors.

9.1.2 Air Dispersion Modeling

Any attempt to mathematically model a physical process will involve uncertainties. In this case, potential exposures were based on short-term and annual average ambient concentrations calculated using AERMOD, a regulatory model designed and demonstrated to over-predict ambient concentrations. In addition, the concentrations used to calculate exposure are outdoor concentrations, which do not account for effects that tend to diminish concentrations as air migrates indoors (e.g., absorption by building materials, deterioration, chemical reactions, or filtration by ventilation systems). Uncertainty associated with the design of the dispersion model is most likely characterized as the degree to which the predicted concentrations overestimate the actual concentrations.

The meteorological data provided to the model can be a source of uncertainty, related to the quality of the data, and whether the selected data are representative of conditions at the area of interest. In this case, the meteorological data used was collected adjacent to the Facility and is the most representative. Although 1-minute National Weather Service ASOS data is not available to supplement the meteorological dataset, the meteorological dataset passed standard checks for quality control. Based on the quality of the data and the proximity of the source to the location where the data were collected, the meteorological data is not considered a significant source of uncertainty.

While there are uncertainties associated with estimating ambient concentrations using an air dispersion model, we believe that reasonable care has been taken to consistently err on the side of more exposure rather than less.

9.1.3 Background Concentrations

Background concentrations of a compound are typically added to modeled concentration attributable to emissions from a given source to obtain a more realistic estimate of the exposure that a population of interest will experience. Because no monitoring data are available in the vicinity of the proposed facility site, background concentrations for most compounds of interest were estimated using an annual average concentration from the 2014 NATA. The NATA provides only annual average concentrations, so short-term background concentrations were not estimated. Furthermore, the EPA says that the NATA should not be used to pinpoint specific risk values in small areas such as census tracts.

Formaldehyde and methylene chloride degrade in the atmosphere, a fact that was not considered in the model. The degradation half-life of formaldehyde is less than 24 hours[44] whereas methylene chloride degrades within 53 – 127 days;[45] therefore the annual exposure concentrations are overestimated. However, some of the degradation by-products may also have toxicity that can increase risk to the population. Because there are several by-products and environmental and seasonal conditions affect the degradation pathway, it was beyond the scope of this assessment to quantify the risks from these by-products.

9.1.4 Toxicity Values

There is uncertainty associated with development of toxicity values. To derive non-cancer toxicity values, agencies such as the EPA, OEHHA, and ATSDR choose critical studies that show effects from exposure to the chemical of interest. Agencies do not always choose the same studies, which may result in variation between the animal species or chemical formulation tested, the exposure duration, and the exposure concentrations. These differences can result in different LOAEL and NOAEL values. Some studies also may not present a NOAEL if only high concentrations of the chemicals were tested. The database of studies on any given chemical expands over time and new studies may present different NOAEL or LOAEL values. Even if two agencies choose the same critical study, if benchmark dose methodology is used in place of a NOAEL or LOAEL, the resulting toxicity values will differ.

Once a LOAEL/NOAEL or benchmark concentration is chosen, the agency then extrapolates to a value relevant to humans for a particular exposure duration (acute or chronic). This requires the use of uncertainty factors. The magnitude of the uncertainty factors is often based on professional judgment, and may differ between agencies.

9.2 Conclusions

Our conclusions, based on the results from the risk characterization as well as the uncertainties explained above, are presented for the non-cancer hazards and the cancer risks.

9.2.1 Non-Cancer Hazards

Based on the risk characterization, acute (1-hour and 8-hour) health effects are not of concern for the McKinley Paper Company facility. The hazard quotients at the maximum impact receptor is less than

---

[44] https://www.atsdr.cdc.gov/phs/phs.asp?id=218&tid=39

[45] https://www.atsdr.cdc.gov/PHS/PHS.asp?id=232&tid=42

one. Similarly, the annual hazard indices for the facility-only and cumulative scenarios do not exceed one.

9.2.2 Cancer Risks

The sum of the increased cancer risks from TAP emissions attributable to the project does not exceed 10 per million for the MIR, MIRR, MIBR, or MICR, which is the acceptable cancer risk threshold provided by Ecology in WAC 173-460-090(7).

ATTACHMENT A

2011 Modeling AERMET Description

AERMET

The AERMET (Version 06341) pre-processor was used to prepare the meteorological data set. Guidance provided in the most recent *AERMOD Implementation Guide* [Environmental Protection Agency (EPA), March 2009] was used.

AERMET uses three steps to preprocess and combine the surface and upper-air soundings to output the data in a format which is compatible with the AERMOD model. The first step extracts the data and performs a brief quality assurance check of the data. The second step merges the meteorological data sets. The third step outputs the data in the AERMOD compatible format while also incorporating surface characteristics surrounding the data collection or application site.

The output from the AERMET model consists of two separate files: the surface conditions file and a vertical profile dataset. AERMOD utilizes these two files in the dispersion modeling algorithm to predict pollutant concentrations resulting from a source's emissions.

The mid-day albedo, daytime Bowen ratio, and surface roughness length are considered when conducting the third step of AERMET processing. Collectively, these factors are described as surface characteristics. Surface characteristics can vary by season and region (sector) around the data collection site.

The mid-day albedo is the fraction of total incident solar radiation reflected by the surface back to space without absorption. The daytime Bowen ratio is an indicator of surface moisture, which is the ratio of the sensible heat flux to the latent heat flux. The Bowen ratio is used to determine the planetary boundary layer parameters for convective conditions. Surface roughness length is related to the height of obstacles to the wind flow and is the height at which the mean horizontal wind speed is zero. The AERMOD model uses the surface characteristics to define dispersion coefficients in the model.

AERSURFACE

The AERSURFACE program (Version 08009) was used to determine the surface characteristics surrounding the monitoring site. AERSURFACE was developed by the EPA to assist in determining surface characteristics by using U.S. Geological Survey (USGS) land use maps and converting the land use type to values described in the *AERMET User's Guide* (EPA, November 2004, revised December 2006).

AERSURFACE uses a 1-kilometer (km) radius surrounding the data collection site to determine surface roughness values for each sector and a 10x10-km area to determine the mid-day albedo and daytime Bowen ratio.

The surface roughness, mid-day albedo, and Bowen ratio are affected by seasonal variations due to the yearly cycle of trees blooming and shedding leaves. The tree density affects the surface roughness while canopy leaf cover affects the amount of solar radiation reflected or absorbed as well as the amount of retained moisture. AERSURFACE accounts for these variations by assigning different seasons to specific months. The impact of these variations depends on the land use surrounding the data collection site.

Nippon Dataset

To prepare the AERMET meteorological data set, surface observations from Port Angeles, Washington, and twice daily upper-air soundings data from the Quillayute, Washington, upper air station (WBAN # 94240) were used to prepare the AERMET meteorological data set.

The surface data were collected by ORCAA and meet EPA's requirements in its *Meteorological Monitoring Guidance for Regulatory modeling Applications* [EPA, February 2000]. The surface data were collected at 1815 Marine Drive, adjacent to the northeast side of the Nippon property line. This data was obtained from EPA's Air Quality System (AQS) database, accessible via the AQS Data Mart (http://www.epa.gov/ttn/airs/aqsdatamart/access/interface.htm), which is available for public use. Additional cloud cover data was obtained from the William R Fairchild International Airport NWS station, located approximately 2 miles southwest of the project site. The surface data towers are located on the north coast of the Olympic Peninsula, within a mile of the Strait of Juan de Fuca. The terrain is flat in the immediate vicinity of the project site with the foothills of the Olympic Mountains beginning about five miles to the south. Land use surrounding the airport is residential with large forested areas.

The Quillayute upper air station is approximately 50 miles west of the project site. The Olympic Mountains lie between the two locations, but they are both located at lower elevations near the coast. Quillayute upper air station is the nearest upper air sounding station to Port Angeles.

Wind conditions at the surface station are predominantly from the west. Winds conditions are generally consistent throughout the year, with more variability in winds during the winter months (December through February)

When running the AERSURFACE program, the seasonal variations assumed no snow cover in the winter, a transitional spring with partial green coverage, a mid-summer with lush vegetation, and an autumn with un-harvested cropland. The moisture conditions varied according to the year: 2002 and 2003 experienced average conditions, and 2004 and 2005 experienced dry conditions. The following months were assigned to each season:

- Winter: December, January, and February
- Spring: March, April, and May
- Summer: June, July, and August
- Autumn: September, October, and November

Table 1 summarizes the albedo and surface roughness output from the AERSURFACE program and the parameters used in the third step of AERMET processing for the Nippon dataset. Table 2 summarizes the Bowen ratio, which varies by moisture conditions.

Attachment 1 displays the annual wind rose for the Nippon dataset.

TABLE 1

Surface Characteristics for the Nippon Dataset – Albedo and Surface Roughness

	ALBEDO				SURFACE ROUGHNESS			
SECTOR[1]	SPRING	SUMMER	FALL	WINTER	SPRING	SUMMER	FALL	WINTER
1	0.12	0.12	0.12	0.13	0.009	0.009	0.009	0.003
2	0.12	0.12	0.12	0.13	0.003	0.004	0.004	0.002
3	0.12	0.12	0.12	0.13	0.002	0.003	0.003	0.005
4	0.12	0.12	0.12	0.13	0.005	0.009	0.009	0.03
5	0.12	0.12	0.12	0.13	0.03	0.073	0.073	0.298
6	0.12	0.12	0.12	0.13	0.298	0.456	0.456	0.385
7	0.12	0.12	0.12	0.13	0.385	0.547	0.547	0.287
8	0.12	0.12	0.12	0.13	0.287	0.383	0.383	0.065
9	0.12	0.12	0.12	0.13	0.065	0.075	0.075	0.007
10	0.12	0.12	0.12	0.13	0.007	0.008	0.008	0.006
11	0.12	0.12	0.12	0.13	0.006	0.007	0.007	0.005
12	0.12	0.12	0.12	0.13	0.005	0.005	0.005	0.003

Note:

[1] Each sector is a 30 degree segment from true north.

TABLE 2
Bowen Ratio by Moisture Conditions for the Nippon Dataset

	Average Moisture Conditions				Dry Moisture Conditions			
SECTOR[1]	SPRING	SUMMER	FALL	WINTER	SPRING	SUMMER	FALL	WINTER
1	0.27	0.20	0.27	0.27	0.35	0.29	0.38	0.38
2	0.27	0.20	0.27	0.27	0.35	0.29	0.38	0.38
3	0.27	0.20	0.27	0.27	0.35	0.29	0.38	0.38
4	0.27	0.20	0.27	0.27	0.35	0.29	0.38	0.38
5	0.27	0.20	0.27	0.27	0.35	0.29	0.38	0.38
6	0.27	0.20	0.27	0.27	0.35	0.29	0.38	0.38
7	0.27	0.20	0.27	0.27	0.35	0.29	0.38	0.38
8	0.27	0.20	0.27	0.27	0.35	0.29	0.38	0.38
9	0.27	0.20	0.27	0.27	0.35	0.29	0.38	0.38
10	0.27	0.20	0.27	0.27	0.35	0.29	0.38	0.38
11	0.27	0.20	0.27	0.27	0.35	0.29	0.38	0.38
12	0.27	0.20	0.27	0.27	0.35	0.29	0.38	0.38

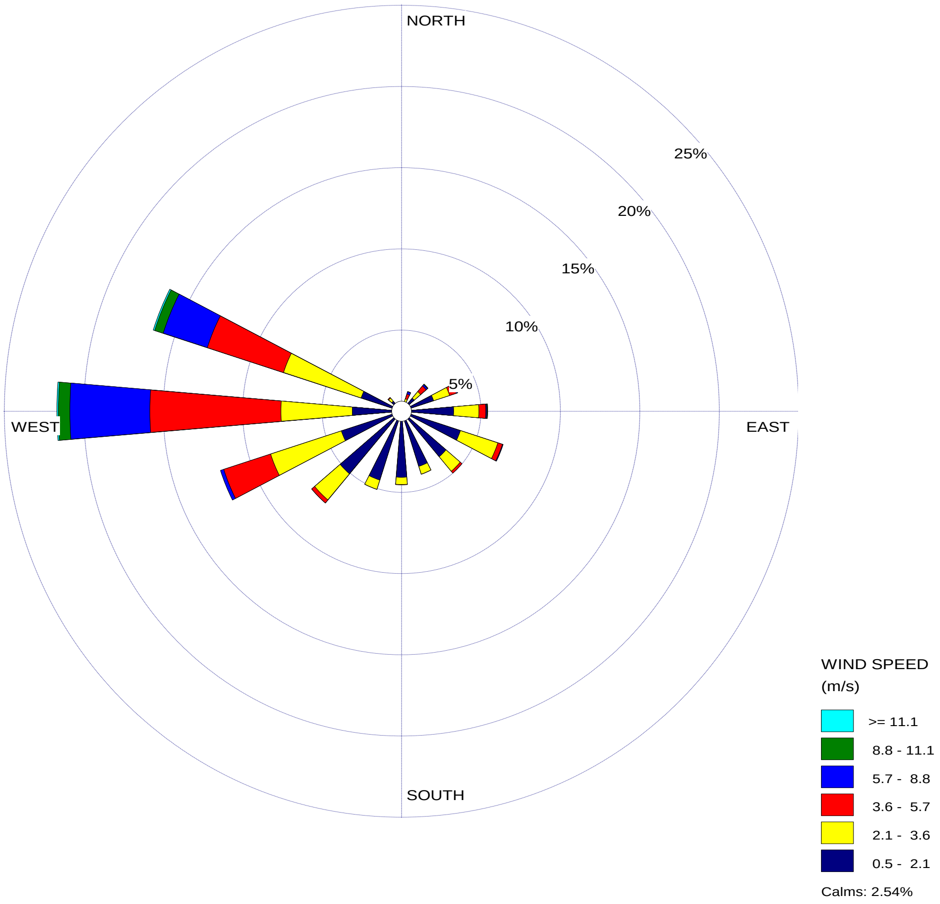
Note:
[1] Each sector is a 30 degree segment from true north.

WIND ROSE PLOT:

Attachment 1
Nippon Paper Wind Rose 2004-2008

DISPLAY:

Wind Speed
Direction (blowing from)



COMMENTS:

CALM WINDS:
2.54%

TOTAL COUNT:
35050 hrs.

AVG. WIND SPEED:
2.88 m/s

PROJECT NO.:

WRPLOT View - Lakes Environmental Software