

**TECHNICAL SUPPORT DOCUMENT  
FOR THE SECOND TIER ANALYSIS**

**SIERRA PACIFIC INDUSTRIES (BURLINGTON)  
LUMBER PRODUCTION INCREASE  
BURLINGTON, WASHINGTON**

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## **1. EXECUTIVE SUMMARY**

Proposed acetaldehyde, acrolein, and formaldehyde emissions from a project proposed by the Sierra Pacific Industries Burlington (SPI Burlington) exceed a regulatory trigger level called an Acceptable Source Impact Level (ASIL). The project was therefore required to undergo a Second Tier analysis per Chapter 173-460 Washington Administrative Code (WAC).

On the basis of the Second Tier analysis described here and the modeled acetaldehyde, acrolein, and formaldehyde concentrations, the Washington State Department of Ecology (Ecology) has determined the health risks are within the range that Ecology may approve for proposed new sources of Toxic Air Pollutants (TAP) under Chapter 173-460 WAC.

This document describes the technical analysis performed by Ecology.

## **2. THE PROCESS**

### **2.1. The Regulatory Process**

The requirements for performing a toxics screening are established in Chapter 173-460 WAC. These rules require a review of any increase in toxic emissions for all new or modified stationary sources in the state of Washington.

#### **2.1.1. The Three Tiers of Toxic Air Permitting**

The objectives of toxics air permitting are to establish the systematic control of new sources emitting toxic air pollutants in order to prevent air pollution, reduce emissions to the extent reasonably possible, and maintain such levels of air quality as will protect human health and safety.

There are three levels of review when processing a new or modified emissions unit emitting TAPs: (1) First Tier (toxic screening), (2) Second Tier (health impacts assessment), and (3) Third Tier (risk management decision).

All projects are required to undergo a toxic screening (First Tier analysis) as required by WAC 173-460-040. There are two ways to perform a First Tier analysis. If proposed emissions are below the Small Quantity Emission Rate (SQER) tables, no further analysis is required. If emissions are greater than the SQER table or no value exists in the SQER table, those emissions must be modeled and the resultant ambient concentration compared against the appropriate ASIL. If the ambient concentration is below the ASIL, then no further analysis is required.

A Second Tier analysis, promulgated in WAC 173-460-090, is a site-specific health impacts assessment. The objective of a Second Tier analysis is to quantify the increase in lifetime cancer risk for persons exposed to the increased concentration of any Class A TAP and to quantify the increased health hazard from any Class B TAP in ambient air that would result from the

proposed project. Once quantified, the cancer risk is compared to the maximum risk allowed by a Second Tier analysis, which is one in one hundred thousand, and the concentration of any Class B TAP that would result from the proposed project is compared to a Risk Based Concentration (RBC).

If the emissions of a toxic pollutant(s) result in a cancer risk of greater than one in one hundred thousand, then an applicant may request Ecology perform a Third Tier analysis. A Third Tier analysis is basically a risk management decision in which the director of Ecology makes a decision that the risk of the project is acceptable based on determination that emissions will be maximally reduced through available preventive measures, assessment of environmental benefit, disclosure of risk at a public hearing and related factors associated with the facility and the surrounding community.

Since Class B TAPs are not classified as carcinogens, there is no Third Tier analysis performed. All non-cancer risks are evaluated in the Second Tier analysis.

## **2.2. Processing Requirements**

Ecology shall evaluate a source's Second Tier analysis only if:

- The Northwest Clean Air Agency (NWCAA) has advised Ecology that other conditions for processing the Notice of Construction Order of Approval (NOC) have been met,
- Emission controls contained in the conditional NOC represent at least Best Available Control Technology for Toxics (T-BACT), and
- Ambient concentrations exceed acceptable source impact levels after using more refined emission quantification and air dispersion modeling techniques.

NWCAA submitted the three items listed above to Ecology on December 11, 2008.

## **3. THE PROJECT**

### **3.1. Permitting History**

In December 2005, SPI Burlington received approval to construct a lumber manufacturing facility from the NWCAA and Ecology. NWCAA issued Order of Approval to Construct (OAC) #938 and Ecology issued Prevention of Significant Deterioration (PSD) 05-04. In January 2008, NWCAA issued a revised OAC (#938a), which allowed SPI Burlington to produce approximately 300 million board feet of green lumber per year and dry up to 180 million board feet of lumber in six dry kilns with steam from the on-site cogeneration unit.

### **3.2. The Proposed Project**

Today's project does not propose any physical changes to the facility. The five primary emission sources: the dry kilns, the cogeneration unit, the anti-mold spray system, fugitive dust, and the

planer mill dust collection system will have increases in emissions. Those emissions increases will be a result of an increase in both green and dry lumber production. NWCAA has incorporated these changes in OAC #938b. After the modification, the facility will be able to produce and dry up to 400 million board feet of lumber, depending upon the species dried. The property for the project is bordered on the west by the Fredonia Grange and several industrial facilities, to the east and northeast by farm and forest land, on the north by Ovenell Road, a Puget Sound Energy generating station and a metal fabrication company, and on the south by State Road 20 and Burlington Northern Santa Fe railroad tracks. The United States Geographical Survey coordinates are North  $48^{\circ} 26' 56''$ , West  $122^{\circ} 25' 59''$ . The North American Datum of 1927 (NAD 27) coordinates are 5,366,150 meters northing, 541,950 meters easting, Zone 10.



### 3.3. Emissions

SPI Burlington has estimated its emissions from the project and they are compared to the SQER tables below:

Pollutant	Class A or B Pollutant	Total Emissions from Boiler and six Kilns		SQER		Emissions Above SQER? Yes or No
		lb/hr	lb/yr	lb/hr	lb/yr	
Acetaldehyde	A	4.91	42,993	-	50	Yes
Acetone	B	0.09	810	5	43,748	No
Acrolein	B	0.082	719	0.02	175	Yes
Ammonia	B	28.2	247,435	2	17,500	Yes
Antimony	B	0.0098	86.3	0.02	175	No
Arsenic	A	0.00242	21.2	-	-	Yes
Barium	B	0.149	1308	0.02	175	Yes
Benzene	A	0.319	2,796	-	20	Yes
Beryllium	A	0.00067	5.85	-	-	Yes
Bis(2-ethylhexyl)phthalate	A	0.00002	0.175	-	500	No
Methyl bromide	B	0.012	106	0.02	175	No
2-Butanone	B	0.00232	20.3	5	43,748	No
Cadmium	A	0.00125	10.9	-	-	Yes
Carbon tetrachloride	A	0.0195	171	-	20	Yes
Chlorine	B	0.341	2983	0.02	175	Yes
Chlorobenzene	B	0.0143	125	0.02	175	No
Chloroform	A	0.0118	104	-	10	Yes
Chloromethane	B	0.0099	87	5	43,748	No
Chlorophenols	A	0.0000145	0.127	-	50	No
Chromium, hexavalent	A	0.00134	11.8	-	-	Yes
Chromium (III)	B	0.000661	5.79	0.02	175	No
Cobalt	B	0.0000538	0.471	0.02	175	No
Copper	B	0.00320	28.0	0.02	175	No
Crotonaldehyde	B	0.00426	37.3	0.20	1,750	No
1,2-Dichloroethane	A	0.0126	110	-	10	Yes
Dichloromethane	A	0.123	1,082	-	50	Yes
1,2-Dichloropropane	A	0.0143	125	-	-	Yes
Ethyl benzene	B	0.0135	118	5	43,748	No
Formaldehyde	B	0.792	6,938	-	20	Yes
Hydrogen chloride	B	1.51	13,189	0.02	175	Yes
Lead	A	0.0213	186	-	50	Yes
Manganese dust	B	0.0422	370	0.02	175	Yes
Mercury	B	0.000179	1.57	0.02	175	No
Methyl alcohol	B	3.87	33,878	5	43,748	No
Napthalene	B	0.0407	356	2.6	22,750	No
Nickel	A	0.00109	9.52	-	0.5	Yes
Nitric oxide	B	64.5	565,234	2	17,500	Yes
Pentachlorophenol	A	0.00000976	0.0855	-	50	No
Phenol	B	0.00539	47.3	1.2	10,500	No
Phosphorous	B	0.0152	133	0.02	175	No
PAH	A	0.00000776	0.0680	-	-	Yes
Selenium	B	0.000750	6.57	0.02	175	No

Pollutant	Class A or B Pollutant	Total Emissions from Boiler and six Kilns		SQER		Emissions Above SQER? Yes or No
		lb/hr	lb/yr	lb/hr	lb/yr	
Silver	B	0.746	6538	0.02	175	Yes
Styrene	B	0.8	7009	50	43,748	No
Sulfuric acid	B	0.864	7,565	0.02	175	Yes
2,3,7,8-Tetrachlorodibenzo-p-dioxin	A	0.000000088	0.0000771	-	-	Yes
Turpentine	B	14.6	128,000	5	43,748	Yes
Perchloroethylene	A	0.0164	144	-	500	No
Tin	B	0.00285	25.0	0.02	175	No
Toluene	B	0.00914	80.1	5	43,748	No
1,1,2-Trichloroethane	B	0.0132	116	2.6	22,750	No
Trichloroethylene	A	0.0130	114	-	50	Yes
Trichlorofluoromethane	B	0.0174	153	5	43,748	No
2,4,6-Trichlorophenol	A	0.00000488	0.0428	-	50	No
Vanadium	B	0.000585	5.12	0.02	175	No
Vinyl chloride	A	0.00791	69.3	-	10	Yes
Xylenes	B	0.0105	92.3	5	43,748	No
Yttrium	B	0.00013	1.14	0.02	175	No

Emissions of acetaldehyde, acrolein, ammonia, arsenic, barium, benzene, beryllium, cadmium, carbon tetrachloride, chlorine, chloroform, hexavalent chromium, 1,2-dibromoethene, 1,2-dichloroethane, dichloromethane, 1,2-dichloropropane, formaldehyde, hydrogen chloride, lead, manganese dust, nickel, nitric oxide, PAH, silver, sulfuric acid, 2,3,7,8-tetrachlorodibenzo-p-dioxin, turpentine, trichloroethylene, and vinyl chloride exceed the values listed in SQER tables. The applicant then modeled these TAPs and compared them to their respective ASILs as shown in Section 3.5.

### 3.4. Point of Compliance

Assessment of potential health risks from the project were based on the maximum modeled concentration of acetaldehyde, acrolein, ammonia, arsenic, barium, benzene, beryllium, cadmium, carbon tetrachloride, chlorine, chloroform, chromium, hexavalent, 1,2-dibromoethene, 1,2-dichloroethane, dichloromethane, 1,2-dichloropropane, formaldehyde, hydrogen chloride, lead, manganese dust, nickel, nitric oxide, PAH, silver, sulfuric acid, 2,3,7,8-tetrachlorodibenzo-p-dioxin, turpentine, trichloroethylene, and vinyl chloride at an assumed point of public exposure (nearest point of ambient air) at the property fence line and at the maximally-impacted residence.

### 3.5. Emission Concentrations

Below is the modeling results of the pollutants that exceeded the SQERs compared to their respective ASILs.

Pollutant	Class A or B TAP?	Averaging Time	Highest Modeled Concentration ( $\mu\text{g}/\text{m}^3$ )	ASIL ( $\mu\text{g}/\text{m}^3$ )
Acetaldehyde	A	Annual	12.5	0.45
Acrolein	B	24-hr	1.23	0.02
Ammonia	B	24-hr	7.22	100
Arsenic	A	Annual	4.03E-05	0.00023
Barium	B	24-hr	0.0382	1.7
Benzene	A	Annual	0.00533	0.12
Beryllium	A	Annual	1.11E-05	0.00042
Cadmium	A	Annual	2.08E-05	0.00056
Carbon tetrachloride	A	Annual	0.000326	0.067
Chlorine	B	24-hr	0.0870	5.0
Chloroform	A	Annual	0.000198	0.043
Chromium, hexavalent	A	Annual	2.24E-05	0.000083
1,2-Dichloroethane	A	Annual	0.000210	0.038
Dichloromethane	A	Annual	0.00206	0.56
1,2-Dichloropropane	A	24-hr	0.00366	4.0
Formaldehyde	B	Annual	0.167	0.077
Hydrogen chloride	B	Annual	0.385	0.0021
Lead	A	24-hr	0.000355	0.5
Manganese dust	B	24-hr	0.010788	0.4
Nickel	A	Annual	1.81E-05	0.0021
Nitric oxide	B	24-hr	14.3	100
PAH	A	Annual	1.29E-07	0.00048
Silver	B	24-hr	0.191	0.33
Sulfuric acid	B	24-hr	0.221	3.3
2,3,7,8-Tetrachlorodibenzo-p-dioxin	A	Annual	1.47E-09	0.00000003
Turpentine	B	24-hr	337	1900
Trichloroethylene	A	Annual	0.000218	0.59
Vinyl chloride	A	Annual	0.000132	0.012

### 3.6. Pollutants Subject to Second Tier Analysis

Emissions of ammonia, arsenic, barium, benzene, beryllium, cadmium, carbon tetrachloride, chlorine, chloroform, hexavalent chromium, 1,2-dichloroethane, dichloromethane, hydrogen chloride, lead, manganese dust, nickel, nitric oxide, PAH, silver, sulfuric acid, 2,3,7,8-tetrachlorodibenzo-p-dioxin, turpentine, trichloroethylene, and vinyl chloride are below the ASIL after being modeled. Acetaldehyde, acrolein, and formaldehyde are subject to review under this Second Tier analysis.

### 3.7. Background Emissions

Acetaldehyde, acrolein, and formaldehyde are produced during combustion. As a result, these pollutants can be measured in ambient air. Higher levels of these pollutants are found

immediately downwind of combustion sources, especially near heavy traffic in urban atmospheres.

Combustion, including wood combustion in fireplaces and wood stoves, coffee roasting, burning of tobacco, and vehicle exhaust, is the primary source of ambient acetaldehyde.<sup>1</sup> Acetaldehyde is also released from numerous consumer products. As a result, indoor levels of acetaldehyde often exceed outdoor levels due to numerous sources indoors.

Acrolein can be formed from the breakdown of other pollutants found in ambient air. Combustion of fuels represents the major source of emissions of acrolein to the atmosphere.<sup>2</sup> Acrolein may also be released while cooking foods, especially while using cooking oils.

Formaldehyde is released into the atmosphere during combustion. Although formaldehyde is found in ambient air, higher levels of formaldehyde are expected in indoor air, where it is released from building materials and indoor furnishings.<sup>3</sup>

Estimates of average acetaldehyde, acrolein, and formaldehyde levels in the census tract relevant to SPI Burlington's proposed lumber kiln are available from EPA's 1999 National Air Toxics Assessment (NATA). For comparison, estimates from a more urban environment (Seattle) are presented along with monitoring results from 2007. Generally, estimated pollutant levels are two to 10 times lower in the tract associated with the project compared to Beacon Hill. According to NATA, background and on-road sources account for the majority of estimated acetaldehyde and formaldehyde concentrations near Burlington, WA (census tract 53057951900). On-road sources are the main contributors to estimated acrolein concentrations at the same census tract.

Pollutant	NATA 1999		2000-2007 Monitored Average Concentration
	Tract 53057951900 (Near Burlington, WA)	Tract 53033010000 (Beacon Hill – Seattle)	Beacon Hill (Seattle)
Acetaldehyde	0.84	2.9	1.4
Acrolein	0.028	0.21	0.45
Formaldehyde	0.89	3.1	1.6

### 3.8. T-BACT

The NWCAA has selected a kiln maximum drying temperature of 200 degrees Fahrenheit as T-BACT for controlling emissions of acetaldehyde, acrolein, and formaldehyde. As a result, numerical limits are proposed for emissions from the wood-fired boiler, kiln-drying Western hemlock, and kiln-drying Douglas fir. Setting this upper limit on drying temperature will

<sup>1</sup> <http://www.inchem.org/documents/ehc/ehc/ehc167.htm>

<sup>2</sup> <http://www.inchem.org/documents/ehc/ehc/ehc127.htm#PartNumber:3>

<sup>3</sup> <http://www.atsdr.cdc.gov/toxprofiles/tp111-c5.pdf>

minimize the emissions of acetaldehyde, acrolein, and formaldehyde. Ecology concurs with the T-BACT proposed by NWCAA.

### **3.9. Air Dispersion Modeling**

The air quality dispersion model used for this project was EPA's AERMOD model, with EPA's PRIME algorithm for building downwash. Meteorological data from Shell Oil Company's Puget Sound Refinery in Anacortes, WA between January 1995 and December 1999 were combined with National Weather Service's (NWS) upper air data from Quillayute, Washington. These data were supplemented by NWS observations from nearby airports including Whidbey Island Naval Air Station, Arlington Municipal Airport, and Burlington-Skagit Regional Airport. Data included hourly wind speed and wind direction.

## **4. GENERIC HEALTH IMPACTS ASSESSMENT PROCESS**

A health impacts assessment was prepared by the applicant and it was reviewed and approved by Ecology. Ecology has put together a project team consisting of an engineer, a toxicologist, and a modeler.

Below are descriptions of the content of each part of the Health Impacts Assessment.

### **4.1. Hazard Identification**

Hazard identification involves gathering and evaluating toxicity data on the types of health injury or disease that may be produced by a chemical and on the conditions of exposure under which injury or disease is produced. It may also involve characterization of the behavior of a chemical within the body and the interactions it undergoes with organs, cells, or even parts of cells. This information may be of value in determining whether the forms of toxicity known to be produced by a chemical agent in one population group or in experimental settings are also likely to be produced in human population groups of interest. Note that risk is not assessed at this stage; hazard identification is conducted to determine whether and to what degree it is scientifically correct to infer that toxic effects observed in one setting will occur in other settings (e.g., are chemicals found to be carcinogenic or teratogenic in experimental animals also likely to be so in adequately exposed humans?).

### **4.2. Exposure Assessment**

This step involves describing the nature and size of the various populations exposed to a chemical agent in the vicinity of the proposed project. The evaluation could include past exposures, current exposures, or exposures expected in the future.

### **4.3. Dose-Response Assessment**

Dose-response assessment is the process of characterizing the relationship between exposure to a chemical and incidence of an adverse health effect in exposed populations. This step involves the identification of the toxicological profiles of all toxic air pollutants that exceed the ASIL. It includes a discussion of the toxicological effects of hazardous substances, chemicals, and compounds. Each profile includes an examination, summary, and interpretation of available toxicological and epidemiological data evaluations on the hazardous substance.

### **4.4. Risk Characterization**

This step involves the integration of data analyses from each step of the health impact assessment to determine the likelihood that the human population of interest will experience any of the various forms of toxicity associated with a chemical under its known or anticipated conditions of exposure.

### **4.5. Uncertainty Characterization**

In almost all risk assessments undertaken in support of regulatory decisions, especially concerning chronic hazards, risk assessors are required to go beyond available data and make inferences about risks expected for conditions of exposure under which direct evidence of risk cannot now be collected. When scientific uncertainty is encountered in a risk assessment, the integration of any assumptions is required to fill information gaps. The following are examples of components that constitute gaps in the scientific basis for assessing human cancer risk:

- How relevant is the data to humans?
- How relevant to humans are results from animal studies using a different route of exposure?
- How relevant are results from studies using an exposure regimen (in terms of frequency and duration) that differs from the human situation?
- Which species/strains of animals are most appropriate for dose-response assessment in humans?
- How should risk estimates be developed?
- Using most sensitive species/strain/sex.
- Combining incidents of benign and malignant tumors.
- Using pooled tumor incidence (tumor bearing animals).
- Can results of an animal study that does not extend over a lifetime be extrapolated to lifetime?
- How does the dose-response relation relate to the unobservable dose-response relation in the dose region of concern for the human population under study?
- How should low-dose risk be modeled?
- Do agents operate by threshold or non-threshold mechanisms?

## 5. HEALTH IMPACTS ASSESSMENT

### 5.1. Introduction

The Second Tier analysis described below was conducted according to the requirements promulgated in Chapter 173-460 WAC. It addressed the public health risk associated with exposure to acetaldehyde, acrolein, and formaldehyde emissions from increased lumber production at Sierra Pacific's lumber mill in Burlington, WA. The health impacts assessment was prepared by a consultant (Environ International Corporation) for SPI Burlington.

### 5.2. Hazard Identification

#### 5.2.1. Acute and Chronic Effects

The primary acute effects of human exposure to acetaldehyde in air consist of irritation to the eyes, skin, and respiratory tract.<sup>4</sup> Asthmatics exposed to acetaldehyde may experience a decrease in lung function due to bronchoconstriction.

There is little information regarding health outcomes in humans related to long-term exposure to acetaldehyde. In animals, chronic inhalation exposure to acetaldehyde has produced changes in the mucus membranes of the nose and trachea, growth retardation, slight anemia, and increased kidney weight. EPA derived a reference concentration based on the degeneration of a layer of cells lining the tissue responsible for smell in the noses (olfactory epithelium) of rats.<sup>5</sup> There is currently insufficient human data regarding the carcinogenic effects of acetaldehyde. Animal studies involving inhalation of acetaldehyde have shown an increased rate of nasal tumors in rats and laryngeal tumors in hamsters. EPA has classified acetaldehyde as a Group B2, probable human carcinogen.

Low levels of formaldehyde can cause irritation of the eyes, nose, throat, and skin. It is possible that people with asthma exposed to formaldehyde can experience respiratory symptoms such as wheezing, shortness of breath, and reduced pulmonary function consistent with bronchoconstriction.<sup>6</sup> 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, but these workers may have been exposed

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<sup>4</sup> [http://www.arb.ca.gov/toxics/id/summary/acetaldehyde\\_b.pdf](http://www.arb.ca.gov/toxics/id/summary/acetaldehyde_b.pdf)

<sup>5</sup> <http://www.epa.gov/ncea/iris/subst/0290.htm>

<sup>6</sup> [http://www.oehha.ca.gov/air/toxic\\_contaminants/pdf\\_zip/formaldehyde\\_112508.pdf](http://www.oehha.ca.gov/air/toxic_contaminants/pdf_zip/formaldehyde_112508.pdf)

to multiple different 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 a type of epithelial cell in the nose (nasal squamous cell carcinoma). The United States Department of Health and Human Services has determined that formaldehyde may reasonably be anticipated to be a carcinogen.<sup>7</sup> EPA has classified formaldehyde as a Group B1, probable human carcinogen.

Acrolein is a potent irritant to skin and mucous membranes. Effects of acrolein typically occur at the point of exposure (i.e., nasal passages, eyes). Short-term exposure to acrolein can cause eye and nasal irritation at relatively low concentrations (< 1ppm [ $\leq 2.3 \text{ mg/m}^3$ ]) in air.<sup>8</sup> Higher concentrations may also irritate the entire respiratory tract. Accidental exposures to extremely high levels of acrolein result in high fever, dyspnea, coughing, foam expectoration, cyanosis, pulmonary edema, and death.<sup>9</sup> Animals exposed to higher acrolein concentrations showed signs of lesions in the respiratory tract and respiratory distress. These effects became more severe with increasing concentrations. At higher levels, respiratory distress resulted in death.

There are no available studies of humans exposed to acrolein over long periods of time. Longer-term studies in laboratory animals at higher concentrations have demonstrated severe nasal lesions as well as pronounced adverse effects on lung function leading to lethality. Studies indicated that rats were most sensitive species. The potential carcinogenicity of acrolein cannot be determined because the existing data are inadequate for an assessment of human carcinogenic potential for either the oral or the inhalation route of exposure.

### 5.2.2. Reproductive/Developmental Effects

No specific information is available on the reproductive or developmental effects of acetaldehyde in humans; however, acetaldehyde is the primary metabolite of ethanol, and therefore it is not clear if acetaldehyde plays a role in fetal alcohol syndrome. In animals, acetaldehyde has been shown to cross the placenta to the fetus. Developmental effects were noted in studies where animals were injected with acetaldehyde.

In humans, there are few data on the association of teratogenicity or adverse reproductive effects with formaldehyde exposure. Existing data do not suggest that formaldehyde, by inhalation or oral routes, produces significant teratogenic or reproductive effects.<sup>10</sup>

No studies were located regarding developmental effects in humans or animals after inhalation exposure to acrolein. The World Health Organization determined that acrolein is not likely to affect the developing embryo based on animal studies where acrolein injected intravenously had no effect on embryonic development.<sup>11</sup>

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<sup>7</sup> <http://www.atsdr.cdc.gov/tfacts111.html#bookmark06>

<sup>8</sup> [http://www.oehha.ca.gov/air/toxic\\_contaminants/pdf\\_zip/acrolein\\_112508.pdf](http://www.oehha.ca.gov/air/toxic_contaminants/pdf_zip/acrolein_112508.pdf)

<sup>9</sup> <http://www.atsdr.cdc.gov/toxprofiles/tp124.html>

<sup>10</sup> <http://www.atsdr.cdc.gov/toxprofiles/tp111.pdf>

<sup>11</sup> <http://www.inchem.org/documents/ehc/ehc/ehc127.htm#SectionNumber:10.1>

### 5.2.3. Terrestrial Fate

Acetaldehyde will volatilize rapidly in near surface and surface soils.<sup>12</sup> Formaldehyde is also biodegraded in soil in a relatively short time.<sup>13</sup> Acrolein in soil can be mobile, but a large portion is expected to volatilize or be broken down by microorganisms or other reactive processes.<sup>14</sup> Therefore, none of these chemicals as emitted from SPI Burlington is likely to build up in soil.

### 5.2.4. Aquatic Fate

Acetaldehyde mixes with water, but will not reside long in surface water as it either will volatilize or be broken down by microbes. Formaldehyde dissolves easily in water, but it does not reside long in water and is not commonly found in drinking water supplies. Acrolein dissolves readily in water but levels are reduced through volatilization, aerobic biodegradation, and hydration to other compounds that subsequently biodegrade. Half-lives of <1–3 days for small amounts of acrolein in surface water have been observed. None of these chemicals as emitted from SPI Burlington is likely to build up in aquatic environments.

### 5.2.5. Atmospheric Fate

Generally, acetaldehyde, formaldehyde, and acrolein are not persistent in air. They react with other chemicals in air (mainly sunlight-derived radicals). The estimated half-life for the reaction of acetaldehyde with hydroxyl produced by ultra violet light is 6.2 hours.

Most formaldehyde in the air also breaks down during the day. The breakdown products of formaldehyde in air include formic acid and carbon monoxide.

When released into air, acrolein is broken down by chemicals generated in sunlight producing carbon monoxide, formaldehyde, and glycolaldehyde. Acrolein also reacts with nitrogen oxides to form peroxyxynitrate and nitric acid.<sup>15</sup>

## 5.3. Exposure Assessment

In order for pollutants to cause harm, people first must be exposed. To assess exposure, it is important to identify locations where people might be exposed, estimate the concentration of pollutants at places where people might be exposed, and estimate how much time they might be at a location. In the case of SPI Burlington's lumber kiln emissions, inhalation and dermal exposure (eye irritation) are the primary routes of exposure because acetaldehyde, formaldehyde, and acrolein emission from the project are not likely to build up in food, soil, and water.

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<sup>12</sup> <http://www.inchem.org/documents/ehc/ehc/ehc167.htm>

<sup>13</sup> <http://www.atsdr.cdc.gov/toxprofiles/tp111-c5.pdf>

<sup>14</sup> <http://www.atsdr.cdc.gov/toxprofiles/tp124-c6.pdf>

<sup>15</sup> <http://www.inchem.org/documents/ehc/ehc/ehc127.htm#PartNumber:4>

### 5.3.1. Estimating Concentration

Air modeling as described in Section 3.9 was used to estimate maximum 1-hr, 24-hr, and annual average concentrations of acetaldehyde, acrolein, and formaldehyde in air near SPI Burlington. The model uses emissions from the project along with meteorological data to estimate worst-case exposure concentrations outside the facility's property boundary.

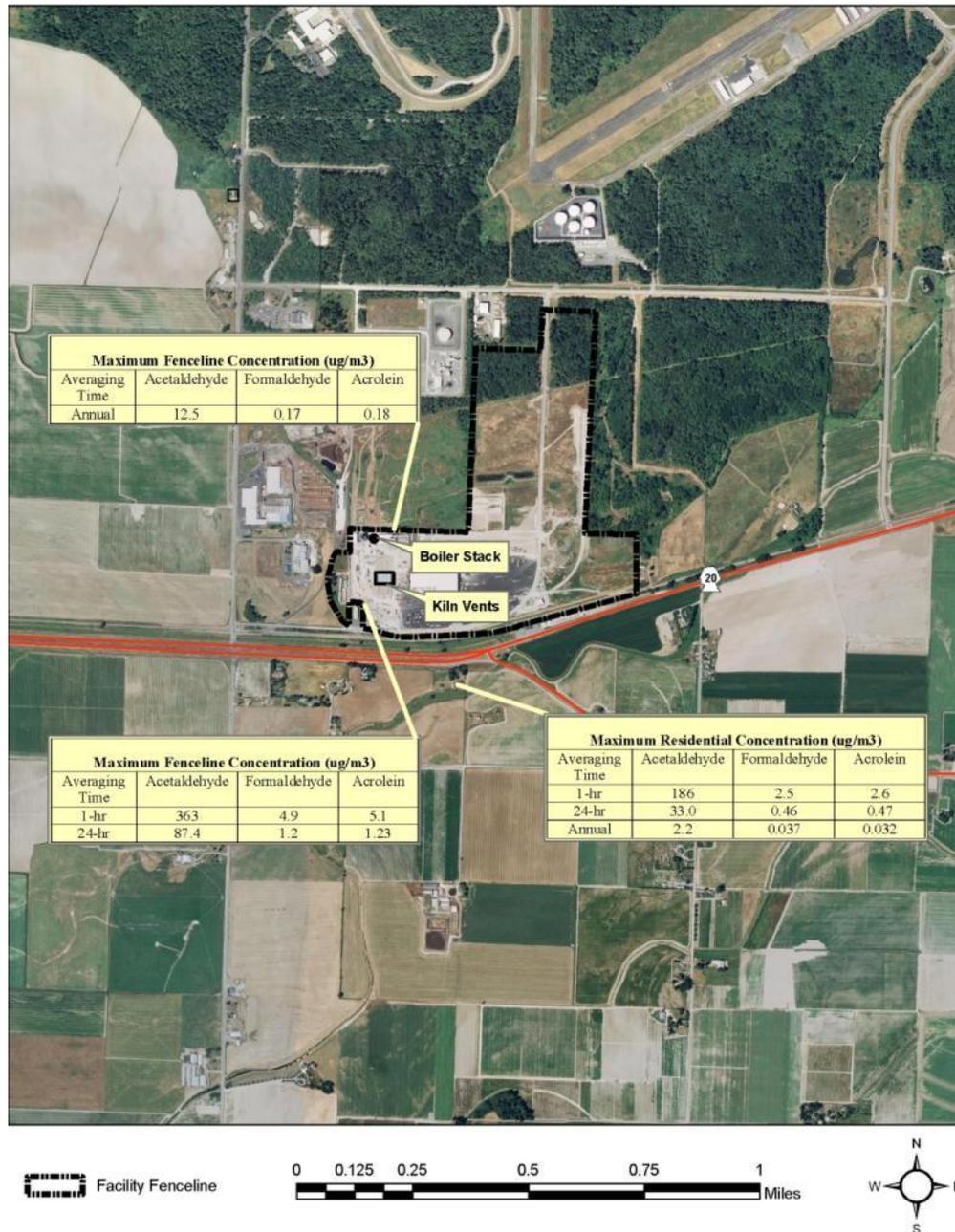
### 5.3.2. Identification of Exposed Populations

Current aerial photographs and land use designations are useful for identifying potentially exposed populations. The table below shows the distances to the exposed fence line and residential receptors.

#	Receptor	Direction from Center of Lumber Kiln	Estimated Distance in Feet from Center of Lumber Kiln	Estimated Distance in Meters from Center of Lumber Kiln
F1	Fence line 1	NNW	570	174
F2	Fence line 2	SSW	371	113
R1	Maximum Residential	SE	1,450	442

### 5.3.3. Discussion of TAP Exposure Concentrations

Air modeling was used to estimate pollutant concentrations at the point of highest concentration (i.e., the fence line) and residences near the facility. Maximum 1-hr, 24-hr, and annual average concentrations at the maximum impacted areas are shown in the figure below.



### 5.3.4. Discussion of Exposure Duration

Exposure duration has implications with regard to health risk that a chemical poses on human health. In most cases, a person continuously exposed to a chemical cannot tolerate as high of concentrations as a person that is exposed for only a short time. Having identified potentially exposed populations, it is also important to consider the amount of time a person might be exposed. People who work at commercial or industrial locations near SPI Burlington are likely only to be exposed for up to the duration of their workday (e.g., eight hours per day). Residents living near SPI Burlington have the potential to be exposed for a longer period (e.g., 24 hours per

day). Residents and occupants of commercial properties both have the opportunity to be exposed for short-term durations (e.g., one hour).

#### 5.4. Dose-Response Assessment

Dose-response assessment describes the quantitative relationship between the amounts of exposure to a substance (the dose) and the incidence or occurrence of injury (the response). The process often involves establishing a toxicity value or criterion to use in assessing potential health risk.

The U.S. Environmental Protection Agency (EPA), California's Office of Environmental Health Hazard Assessment (OEHHA), and the Agency for Toxic Substances and Diseases Registry (ATSDR) have developed toxicological values for the chemicals evaluated in this project. These toxicological values are derived from studies of animals and humans that were exposed to a known amount (concentration) of a chemical and are intended to represent a level at or below which adverse health effects are not expected. Toxicological values derived for cancer and non-cancer effects for the chemicals of concern are shown below. These values in turn are used to quantify hazards and risk associated with exposure.

Chemical	Agency	Type	Value	Animal or Human	Critical Effect	UF	Date
Acetaldehyde	EPA	Chronic RfC	9 µg/m <sup>3</sup>	Rats	Degeneration olfactory epithelium	1000	10/91
		URF	2.2x10 <sup>-6</sup> per µg/m <sup>3</sup>	Rats Hamsters	Nasal, Laryngeal Tumors	NA	10/91
	OEHHA	Chronic REL	9 µg/m <sup>3</sup>	Rats	Degeneration olfactory epithelium	1000	5/93
		URF	2.7x10 <sup>-6</sup> per µg/m <sup>3</sup>	Rats	Nasal tumors	NA	4/99

Chemical	Agency	Type	Value	Animal or Human	Critical Effect	UF	Date
Acrolein	EPA	RfC	0.02 µg/m <sup>3</sup>	Rats	Nasal lesions	1000	6/2003
	OEHHA	Acute REL	0.19 µg/m <sup>3</sup>	Human	Eye irritation	60	4/99
		Chronic REL	0.06 µg/m <sup>3</sup>	Rats	Histological lesions upper airway	300	1/2001
	ATSDR	Acute MRL	6.9 µg/m <sup>3</sup>	Human	Nasal and throat irritation Decreased respiratory rate	100	8/2007
		Intermediate MRL	0.09 µg/m <sup>3</sup>	Rats	Nasal epithelial metaplasia Bronchial inflammation	300	8/2007

Chemical	Agency	Type	Value	Animal or Human	Critical Effect	UF	Date
Formaldehyde	EPA	URF	$1.3 \times 10^{-5}$ per $\mu\text{g}/\text{m}^3$	Rats	Nasal squamous cell carcinomas	NA	5/91
	OEHHA	Acute REL	$94 \mu\text{g}/\text{m}^3$	Human	Eye irritation	10	4/99
		Chronic REL	$3 \mu\text{g}/\text{m}^3$	Human Workers	Nasal and eye irritation, nasal obstruction, and lower airway discomfort; histopathological nasal lesions including rhinitis, squamous metaplasia, and dysplasia	10	2/2000
		URF	$6.6 \times 10^{-6}$ per $\mu\text{g}/\text{m}^3$	Rats	Nasal squamous cell carcinomas	NA	3/92
	ATSDR	Acute MRL	$49 \mu\text{g}/\text{m}^3$	Human	Nasal and eye irritation	9	7/99
		Intermediate MRL	$37 \mu\text{g}/\text{m}^3$	Monkey	Nasopharyngeal irritation (hoarseness and nasal congestion and discharge) and lesions in the nasal epithelium	30	7/99
		Chronic MRL	$9.8 \mu\text{g}/\text{m}^3$	Human Workers	Mild irritation of the eyes and upper respiratory tract and mild damage to the nasal epithelium	30	7/99

#### 5.4.1. Risk Based Concentrations for Exposed Populations

To evaluate possible non-cancer effects from exposure to acetaldehyde, acrolein, and formaldehyde from SPI Burlington's lumber kiln emissions, modeled concentrations were compared to their respective non-cancer comparison value [EPA inhalation reference concentration (RfC), OEHHA reference exposure level (REL) or ATSDR chronic minimal risk level (MRL)]. The RfC, REL, and MRL are concentrations in air below which non-cancer health effects are not expected.

RfCs, RELs, and MRLs are set well below toxic effect levels in order to provide an added measure of safety. The higher the chemical concentration is above the RfC, REL, or MRL, the closer it will be to an actual toxic effect level.

Because chronic RfCs, RELs, and MRLs are based on a continuous exposure, an adjustment was made to account for people working at commercial properties exposed for only eight hours per day, five days per week. This adjustment is shown below:

$$\text{Chronic RBCs} = \frac{\text{AT} \times \text{Chronic RfC, REL, or MRL}}{\text{EF (days per year)} \times \text{EF (hours per 24-hr day)} \times \text{ED}}$$

Scenario	Pollutant	Value	Source	EF (days /yr)	EF (hrs/ 24-hr)	ED (yr)	AT	Chronic Risk Based Concentration
Commercial/ Industrial Worker	Acetaldehyde	9	EPA, OEHHA	250	8 /24	1	365	39.4
	Formaldehyde	3	OEHHA					13.1
	Acrolein	0.02	EPA					0.09
		0.06	OEHHA					0.27
Residential	Acetaldehyde	9	EPA, OEHHA	365	24/24	1	365	9
	Formaldehyde	3	OEHHA					3
	Acrolein	0.02	EPA					0.02
		0.06	OEHHA					0.06

The resulting risk based concentrations for non-cancer health effects are concentrations at or below which health adverse effects are not likely to occur. Risk based concentrations should reflect the exposure characteristics of the various receptors. In this case, the two types of receptors are residential and commercial/industrial workers.

The following table shows the non-cancer risk based concentrations derived for exposure to acetaldehyde, acrolein, and formaldehyde for acute and chronic exposures at residential and commercial settings.

Scenario	Averaging Time	Acute and Chronic Risk Based Concentration ( $\mu\text{g}/\text{m}^3$ )			Source
		Acetaldehyde	Acrolein	Formaldehyde	
Residential	1-hr	NA	6.9	49	ATSDR Acute MRL
	24-hr	NA	6.9	49	ATSDR Acute MRL
	annual	9	0.02 to 0.06	3	EPA RfC and OEHHA chronic REL
Workers at Commercial/ Industrial Properties	1-hr	NA	6.9	49	ATSDR Acute MRL
	annual	39.4	0.09 to 0.27	13.1	EPA RfC and OEHHA chronic REL adjusted for exposure frequency

#### 5.4.2. Estimating Cancer Risk

Some chemicals have the ability to cause cancer. Cancer risk is estimated by determining the concentration of acetaldehyde and formaldehyde at each receptor point and multiplying it by its respective unit risk factor (URF). URFs are expressed as the upper bound probability of developing cancer assuming continuous lifetime exposure to a substance at a concentration of one microgram per cubic meter, and are expressed in units of inverse concentration [i.e.,  $(\mu\text{g}/\text{m}^3)^{-1}$ ].

<sup>15</sup>]. Some URFs are derived from human population data. Others are derived from laboratory animal studies involving doses or concentrations much higher than are encountered in the environment. Use of animal data requires extrapolation of the cancer potency obtained from these high dose studies down to real-world exposures. This process involves much uncertainty.

Because URFs are based on a continuous exposure over a 70 year lifetime, exposure duration and exposure frequency should be considered in occupational or other scenarios.

The formula for determining cancer risk is as follows:

$$\text{Risk} = \frac{C_{\text{Air}} \times \text{URF} \times \text{EF} \times \text{ED}}{\text{AT}}$$

Where:

$C_{\text{Air}}$  = Concentration in air at the receptor ( $\mu\text{g}/\text{m}^3$ )

URF = Unit Risk Factor ( $\mu\text{g}/\text{m}^3$ )<sup>-1</sup>

EF1 = Exposure Frequency (days per year)

EF2 = Exposure Frequency (hours per day)

ED = Exposure Duration (years)

AT = Averaging Time (days)

Current regulatory practice assumes that there is no “safe dose” of a carcinogen and that a very small dose of a carcinogen will give a very small cancer risk. Cancer risk estimates are, therefore, not yes/no answers but measures of chance (probability). Such measures, however uncertain, are useful in determining the magnitude of a cancer threat because any level of a carcinogenic contaminant carries an associated risk. The validity of the “no safe dose” assumption for all cancer-causing chemicals is not clear. Some evidence suggests that certain chemicals considered carcinogenic must exceed a threshold of tolerance before initiating cancer. For such chemicals, risk estimates are not appropriate. Recent guidelines on cancer risk from EPA reflect the potential that thresholds for some carcinogenesis exist. However, EPA still assumes no threshold unless sufficient data indicate otherwise.<sup>16</sup>

## 5.5. Risk Characterization

In this step, non-cancer hazards and cancer risk are quantified to determine if possible health threats exist.

### 5.5.1. Hazard Quotient

Hazard quotients were calculated for different scenarios and averaging periods depending on land use and varying durations of exposure. A hazard quotient (HQ) is the ratio of the potential

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<sup>16</sup> U.S. Environmental Protection Agency. Guidelines for Carcinogen Risk Assessment (Review Draft). NCEA-F-0644 July 1999. Web address available at: <http://www.epa.gov/NCEA/raf/cancer.htm>

exposure to a substance compared to the exposure level that is considered “safe” (e.g., risk based concentration).

$$HQ = \frac{\text{maximum 1-hr, 24-hr, or annual average concentration } (\mu\text{g}/\text{m}^3)}{\text{Corresponding 1-hr, 24-hr, or annual RBC } (\mu\text{g}/\text{m}^3)}$$

A HQ of one or less indicates that adverse health effects are not expected to result from exposure to emissions of that substance. As the HQ increases above one, the probability of human health effects increases by an undefined amount. However, it should be noted that a HQ above one is not necessarily indicative of health impacts due to the application of uncertainty factors in deriving toxicological reference values (e.g., RfC, MRL, and REL).

The following table shows modeled concentrations, RBCs, and HQs at each receptor point. In most cases, HQs are less than one, and therefore of no concern for non-cancer effects. The chronic HQs for acrolein exposure exceed one at residential and fence line receptors.

	Maximum Impacted Residential			Maximum Impacted Point (fence line)		
	Acetaldehyde	Formaldehyde	Acrolein	Acetaldehyde	Formaldehyde	Acrolein
1-hr concentration	186	2.6	2.6	363	4.9	5.1
1-hr RBC	N/A	49	6.9	N/A	49	6.9
1-hr HQ	N/A	0.05	0.38	N/A	0.1	0.74
24-hr concentration	33.0	0.46	0.47	87.4	1.2	1.23
24-hr RBC	N/A	49	6.9	N/A	N/A	N/A
24-hr HQ	N/A	0.01	0.07	N/A	N/A	N/A
Annual concentration	2.2	0.04	0.032	12.5	0.17	0.18
Annual RBC	9	3	0.02	39.4	13.1	0.09
Annual HQ	0.24	0.01	<b>1.5</b>	0.32	0.01	<b>2.0</b>

### 5.5.2. Discussion of Hazard Quotients that Exceed One

Hazard quotients related to chronic exposure to acrolein exceed one (1.5 at residential receptor, and 2.0 at the fence line). As previously noted, a HQ above one is not necessarily indicative of an exposure that will result in health impacts due to the application of uncertainty factors in deriving toxicological reference values (e.g., RfC and REL). The more a HQ exceeds one, the more it becomes a concern for public health. As shown previously in Section 5.4 of this document, acrolein has two available chronic toxicity values (EPA’s RfC of 0.02  $\mu\text{g}/\text{m}^3$  and OEHHA’s REL of 0.06  $\mu\text{g}/\text{m}^3$ ). In this evaluation, use of EPA’s RfC as the toxicological reference value resulted in a HQ slightly greater than one. Contrarily, if OEHHA’s chronic REL were used as the toxicological reference value, the chronic HQs for acrolein would be less than

one. The fact that these two values were based on the same study of rats exposed to acrolein illustrates the difficulty with interpreting and implementing uncertainty. Currently, OEHHA is in the process of updating acrolein's RELs based on studies that are more recent. The proposed chronic REL for acrolein is  $0.35 \mu\text{g}/\text{m}^3$ . Given these considerations along with the fact that highly conservative assumptions were used to estimate receptors' exposures, it is unlikely that chronic exposure to acrolein near the facility will result in adverse non-cancer health effects.

It should be noted that had other toxicological values been used to derive acute RBCs for acrolein, 1-hr HQs would have exceeded one. In addition to ATSDR's acute MRL (the basis for the acute RBC), other public agencies have established toxicological values for acute exposure to acrolein. Minnesota Department of Health established an acute health based value (HBV) for acrolein in air<sup>17</sup> at  $2 \mu\text{g}/\text{m}^3$ . California's OEHHA developed an acute REL of ( $0.19 \mu\text{g}/\text{m}^3$ ) based on mild eye irritation in human volunteers exposed to acrolein, however, this REL is currently undergoing review and may be increased to  $2.5 \mu\text{g}/\text{m}^3$ .<sup>18</sup> Considering the different acute toxicological values derived by each agency, adverse acute health effects are unlikely, but there may be a minor risk of eye irritation from acrolein.

### 5.5.3. Cancer Risk

In this document, cancer risks are reported using scientific notation to quantify the increased cancer risk of an exposed person, or the number of excess cancers that might result in an exposed population. For example, a cancer risk of  $1 \times 10^{-6}$  means that if 1,000,000 people were exposed to a carcinogen, one excess cancer might occur, or a person's chance of getting cancer in their life increases by 0.0001 percent. The reader should note that these estimates are for excess cancers that might result in addition to those normally expected in an unexposed population. Cancer risks quantified in this document are an upper-bound theoretical estimate. Actual risks are likely to be much lower.

The following table shows ranges of estimated worst-case residential and off-site worker cancer risks from exposure to acetaldehyde and formaldehyde near the SPI Burlington facility. The maximum annual pollutant concentrations at the fence line and EPAs and OEHHA's URFs were used to estimate a range of risks to off-site occupational workers (e.g., farmers). In all cases, cancer risks fall below the  $1 \times 10^{-5}$  risk level. This risk level is considered acceptable in chapter 173-460 WAC.

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<sup>17</sup> <http://www.health.state.mn.us/divs/eh/risk/guidance/acroleinmemo.html>

<sup>18</sup> [http://www.oehha.ca.gov/air/toxic\\_contaminants/pdf\\_zip/acrolein\\_112508.pdf](http://www.oehha.ca.gov/air/toxic_contaminants/pdf_zip/acrolein_112508.pdf)

Location	Pollutant	Annual Cair	URF	EF1 (days /yr)	EF2 (hrs/ 24-hr)	ED (yr)	AT (days)	Risk				
Maximum Concentration Fence Line	Acetaldehyde	12.5	2.2x10 <sup>-6</sup>	250	8 /24	25	25550	2.24 x 10 <sup>-6</sup>				
			2.7x10 <sup>-6</sup>					2.75 x 10 <sup>-6</sup>				
	Formaldehyde	0.17	6.6x10 <sup>-6</sup>					9.15 x 10 <sup>-8</sup>				
			1.3x10 <sup>-5</sup>					1.80 x 10 <sup>-7</sup>				
	Total							2.33 x 10 <sup>-6</sup>				
								2.93 x 10 <sup>-6</sup>				
Maximum Impacted Residence	Acetaldehyde	2.2	2.2x10 <sup>-6</sup>	365	24/24	70	25550	4.64 x 10 <sup>-6</sup>				
			2.7x10 <sup>-6</sup>					5.70 x 10 <sup>-6</sup>				
	Formaldehyde	0.037	6.6x10 <sup>-6</sup>					2.34 x 10 <sup>-7</sup>				
			1.3x10 <sup>-5</sup>					4.61 x 10 <sup>-7</sup>				
	Total							4.8810 <sup>-6</sup>				

### 5.6. Uncertainty Characterization

To the extent that an individual will be exposed to emissions of acetaledehyde, acrolein, and formaldehyde from this proposed project, the applicant submitted the following uncertainty analysis:

- TAP emission rates for the proposed project have been estimated using an approach consistent with that used in the NOC permit application submitted to NWCAA for the dried lumber production capacity increase. These emission factors are averages of the results of relatively few tests conducted on laboratory-scale lumber drying equipment, and, in some cases, the range of results being averaged is considerable.
- An air dispersion model was used to predict the off-site concentrations of formaldehyde emission increases expected to result from the proposed project. Site-specific or site-representative inputs were used in the model where appropriate and defaults that are generally conservative were incorporated when such information was not available. The modeling methodology was also consistent with that presented in the NOC permit application submitted to NWCAA.
- The focus of the evaluation was on potential exposure at the maximum fence line location, which is protective of actual receptors located farther from the facility.
- Exposure assumptions for receptors are highly conservative as they assume a person is at one location for 24 hours per day, 365 days per year for 70 years. These assumptions overestimate the actual exposure and risk.

- One of the largest sources of uncertainty in any risk evaluation is associated with the scientific community's limited understanding of the toxicity of most chemicals in humans following exposure to the low concentrations generally encountered in the environment. The majority of available toxicity data are from animal studies, which generate toxicity criteria used to predict what might occur in humans.
- There is much toxicological uncertainty with regard to establishing toxicological reference values. In the case of acrolein, the reference values derived by regulatory and health agencies for both acute and chronic durations varied between public agencies.

## 6. CONCLUSION

Emissions of acetaldehyde and formaldehyde could result in a combined cancer risk of up to  $6 \times 10^{-6}$  (six in one million). This risk falls below Ecology's threshold of maximum acceptable risk (one in one hundred thousand) as defined in chapter 173-460 WAC. Additionally, acute and chronic exposure to acrolein, formaldehyde, and acetaldehyde emissions from the proposed project is not likely to result in adverse non-cancer health effects.

The project will not have a significant adverse impact on air quality. The Washington State Department of Ecology finds that the applicant, Sierra Pacific Industries, has satisfied all requirements for Second Tier analysis.

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## 7. LIST OF ABBREVIATIONS

AERMOD	Air dispersion model
AT	Averaging Time (days)
ATSDR	Agency for Toxic Substance Disease Registry
ASIL	Acceptable Source Impact Level
ATSDR	Agency for Toxic Substances and Diseases Registry
BACT	Best Available Control Technology
C	Celsius
C <sub>Air</sub>	Concentration in air
EPA	United States Environmental Protection Agency
ED	Exposure Duration (years)
EF	Exposure Frequency
EF1	Exposure Frequency (days per year)
EF2	Exposure Frequency (hours per day)
Ecology	Washington State Department of Ecology, Headquarters Office
EPA	United States Environmental Protection Agency
HBV	Health Based Value
HQ	Hazard Quotient
hr	Hour
LOAEL	Lowest Observable Adverse Effect Level
MRL	ATSDR Minimal Risk Level
mg/m <sup>3</sup>	Milligrams per Cubic Meter
NAD27	North American Data of 1927
NATA	National Air Toxics Assessment
NOC	Notice of Construction Order of Approval
NWCAA	Northwest Clean Air Agency
NWS	National Weather Service
OAC	Order of Approval to Construct
OEHHA	California's Office of Environmental Health Hazard
PSD	Prevention of Significant Deterioration
REL	OEHHA Reference Exposure Level
RBC	Risk Based Concentration
RfC	Reference Concentration
SPI Burlington	Sierra Pacific Industries Burlington
SQER	Small Quaintly Emission Rate
TAP	Toxic Air Pollutants
T-BACT	Best Available Control Technology for Toxics
WAC	Washington Administrative Code
UF	Uncertainty Factor
URF	Unit Risk Factor
ug/m <sup>3</sup>	Micrograms per Cubic Meter