Ecology Review Draft
Tier 2 Health Impact Assessment
Engine Test Laboratory III
PACCAR Technical Center
Skagit County, Washington

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Prepared for

PACCAR Inc.
12479 Farm to Market Road
Mount Vernon, Washington 98273

LANDAU ASSOCIATES
130 2nd Avenue South
Edmonds, WA 98020
(425) 778-0907

TABLE OF CONTENTS

FIGURES

TABLES

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LIST OF ABBREVIATIONS AND ACRONYMS

$\mu g/m^3$	Microgram per Cubic Meter
µm	Micrometer
AERMOD	American Meteorological Society/EPA Regulatory Model
ASIL	Acceptable Source Impact Level
BACT	Best Available Control Technology
DEEP	Diesel Engine Exhaust Particulate
Ecology	Washington State Department of Ecology
EPA	U.S. Environmental Protection Agency
HI	Hazard Index
HIA	Health Impact Assessment
HQ	Hazard Quotient
m	Meter
MIBR	Maximally Impacted Boundary Receptor
MICR	Maximally Impacted Commercial Receptor
MIRR	Maximally Impacted Residential Receptor
NAAQS	National Ambient Air Quality Standards
NOC	Notice of Construction
NO_x	Nitrogen Oxides
OEHHA	California Office of Environmental Health Hazard Assessment
$PM_{2.5}$	Particulate Matter Less Than or Equal to 2.5 Microns in Size
PRIME	Plume Rise Model Enhancements
RBC	Risk-Based Concentration
REL	Reference Exposure Level
RfC	Reference Concentration
SO_2	Sulfur Dioxide
SQER	Small-Quantity Emission Rate
TAP	Toxic Air Pollutant
tBACT	Toxics Best Available Control Technology
TPY	Tons per Year
TWA	Time-Weighted Average
URF	Unit Risk Factor
WAC	Washington Administrative Code

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1.0 EXECUTIVE SUMMARY

1.1 PROPOSED PROJECT

PACCAR Inc. (PACCAR) has proposed the development of a third engine test laboratory (ETL III) at the PACCAR Technical Center (PTC) in Skagit County, Washington. The proposed development will include construction of an approximately 19,000-square-foot building and installation of stationary emission units that include three engine test cells, one power train test cell, three heating and process hydronic boilers, and one cooling tower. The engines to be tested in the test cells are certified for compliance with U.S. Environmental Protection Agency and European Union emission standards for on-highway heavy-duty diesel engines. To meet those emission standards, these engines require the use of in-cylinder combustion controls or exhaust after-treatment control technologies including selective catalytic reduction, particulate filters, and oxidation catalysts; emissions controls are used to achieve and demonstrate compliance with the specific engine programs being tested.

PACCAR evaluated air quality impacts associated with the proposed ETL III project in a Notice of Construction (NOC) air permit application submitted to the Northwest Clean Air Agency (NWCAA) (Landau Associates 2013). As documented in the NOC permit application, potential emissions of diesel engine exhaust particulate (DEEP) from the four test cells within ETL III may cause ambient air impacts that exceed the Washington State Acceptable Source Impact Level (ASIL). Based on that modeled exceedance, PACCAR is required to submit a second-tier petition per Chapter 173-460 of the Washington Administrative Code (WAC).

A cumulative approach to permitting the test cells is warranted at the PTC because the proposed test cells are within close proximity to the existing test cells and other regional background sources of DEEP. As part of the cumulative approach, this second-tier health impact assessment (HIA) considers the cumulative impacts of DEEP from existing permitted and proposed test cells and regional background concentrations of DEEP.

1.2 HEALTH IMPACTS EVALUATION

The HIA demonstrates that the ambient cancer risks and non-cancer risks caused by emissions of DEEP from the proposed project are less than Ecology's approval limits. The four proposed test cells could cause an increased cancer risk of up to 5.1 in one million (5.1×10^{-6}) at the maximally impacted residence, specifically at the closest existing house to the southwest of the PTC on property zoned Rural Reserve (Skagit County website 2013). Because the increase in cancer risk attributable to the proposed project alone is less than the maximum risk allowed by a second-tier review, which is 10 in one million, the project is approvable under WAC 173-460-090.

Based on the cumulative maximum DEEP concentration at a residential location near the PTC, the estimated maximum potential cumulative cancer risk posed by DEEP emitted from the proposed project and local and regional background sources within the area is approximately 46 in one million (46 $\times 10^{-6}$) at an existing house to the southwest of the PTC on property zoned Rural Reserve.

1.3 CONCLUSIONS

Project-related health risks are less than the limits permissible under WAC 173-460-090. Therefore, the project is approvable under WAC 173-460-090.

2.0 PACCAR TECHNICAL CENTER EXPANSION PROJECT

2.1 DESCRIPTION OF PROPOSED DEVELOPMENT OF ENGINE TEST LABORATORY III

PACCAR operates an engine testing facility at the PACCAR Technical Center (PTC) in Skagit County, Washington (Figure 2-1). PACCAR is proposing the development of a third engine test laboratory (ETL III) at the PTC that would include the construction of an approximately 19,000-square-foot building and installation of stationary emission units that include three engine test cells, one power train test cell, three heating and process hydronic boilers, and one cooling tower. The three boilers are exempt from New Source Review; therefore, no further discussion of these emission units is provided. The four proposed test cells would be used to test diesel-fueled internal combustion engines that have a power rating of up to 620 brake horsepower (BHP). The PTC site layout and the proposed location of the ETL III building are presented in Figure 2-2. The four proposed test cells will be located within the ETL III building. Each test cell will have its own 55-foot-tall vertical exhaust stack, with a diameter of 10 inches. Figure 2-3 presents the location of the proposed ETL III test cell exhaust stacks.

Figure 2-2 also shows the locations of the existing engine test labs (ETL I and II). ETL I and II are each comprised of four indoor test cells. The four ETL I test cells are used to test diesel-fueled internal combustion engines that have a power rating of up to 600 BHP. The four ETL II test cells are used to test diesel-fueled internal combustion engines that have a power rating of up to 805 BHP. The location of the existing ETL I and II test cell exhaust stacks are presented on Figure 2-3.

Engine types, engine loads, and testing schedules will vary within each test cell. For example, endurance testing may be conducted on one engine for multiple months, whereas short-term testing on engines can take only minutes or days.

2.2 FORECAST EMISSION RATES

Air pollutant emission rates were calculated for the sources identified in Section 2.1 in accordance with Northwest Clean Air Agency requirements and WAC 173-460-050. Emission rates were quantified for criteria pollutants and TAPs. Detailed emission calculation spreadsheets are provided in the *Notice of Construction Application Supporting Documentation* report (Landau Associates 2013).

The emission estimates presented in this permit application have been calculated to conservatively overestimate emissions by assuming that the four proposed ETL III test cells will be used to test engines with a 620 BHP power rating (highest anticipated power rating) at 100 percent load capacity for 24 hours per day, 365 days per year.

Emissions estimates for the existing ETL I and II test cells were also calculated for use in modeling the cumulative air quality impacts (i.e., project plus background emissions). ETL I and II emissions estimates were conservatively calculated to assume that the test cells would be used to test engines with a maximum permitted potential BHP power rating (i.e., 600 BHP for ETL I and 805 BHP for ETL II) at 100 percent of the rated engine capacity for the maximum permitted number of hours per year (i.e., 6,570 hours per year).

The basis for determining appropriate DEEP emission factors and calculating emission rates is provided in the *Notice of Construction Application Supporting Documentation* report (Landau Associates 2013). The maximum potential DEEP emission rate for ETL III is 0.34 tons per year. Table 2-1 summarizes the calculated emission rates for ETL III.

The maximum annual emission rates presented in Table 2-1 were used in the NOC permit application to model compliance with the annual-average NAAQS, annual-average ASILs, and to evaluate non-cancer health risks.

2.3 LAND USE AND ZONING

Land uses in the vicinity of the PTC are presented in Figure 2-4. Topography in the vicinity of the PTC is relatively flat with elevations ranging between approximately 5 and 150 ft above sea level. The zoning designation for the project site is Bayview Ridge Heavy Industrial (BR-HI). The surrounding land use is rural including undeveloped land, aviation, low density residential, agricultural, and industrial uses.

Detailed zoning information for the area surrounding the PTC is presented on Figure 2-5 (Skagit County website 2013). From a health impacts standpoint, an existing single-family residence is located to the southwest of the PTC on land zoned Rural Reserve (RRv) is of primary interest (see Figure 2-4). Zoning and land use developments for properties surrounding the PTC are presented in Table 2-2.

2.4 SENSITIVE RECEPTORS

The following sensitive receptors are near the PTC:

- The nearest school is Bay View Elementary School, approximately 1.2 miles northeast of the PTC.
- The nearest daycare or pre-school is ABC Kidcare, approximately 2.8 miles southeast of the PTC.
- The nearest church is Avon United Methodist Church, approximately 2.7 miles southeast of the PTC.
- The nearest medical facility is Thyroid Treatment Center, approximately 3 miles southeast of the PTC.

- The nearest convalescent home is Where the Heart Is, approximately 4.2 miles east of the PTC.

3.0 PERMITTING REQUIREMENTS FOR NEW SOURCES OF TOXIC AIR POLLUTANTS

3.1 OVERVIEW OF THE REGULATORY PROCESS

The requirements for performing a toxics screening are established in Chapter 173-460 WAC. This rule requires a review of any non-*de minimis*[1] increase in TAP emissions for all new or modified stationary sources in Washington State. Sources subject to review under this rule must apply best available control technology (BACT) for toxics (tBACT) to control emissions of all TAPs subject to review.

There are three levels of review when processing an NOC application for a new or modified emissions unit emitting TAPs in excess of the *de minimis* levels: 1) first tier (toxic screening); 2) second tier (health impacts assessment); and 3) third tier (risk management decision).

All projects with emissions exceeding the *de minimis* levels are required to undergo a toxics screening (first-tier review) as required by WAC 173-460-080. The objective of the toxics screening is to establish the systematic control of new sources emitting TAPs in order to prevent air pollution, reduce emissions to the extent reasonably possible, and maintain such levels of air quality to protect human health and safety. If modeled emissions exceed the trigger levels called ASILs, a second-tier review is required.

As part of a second-tier petition, described in WAC 173-460-090, the applicant submits a site-specific HIA. The objective of an HIA is to quantify the increase in lifetime cancer risk for persons exposed to the increased concentration of any carcinogen, and to quantify the increased health hazard from any non-carcinogen that would result from the proposed project. Once quantified, the cancer risk is compared to the maximum risk allowed by a second-tier review, which is 10 in 1 million, and the concentration of any non-carcinogen that would result from the proposed project is compared to its effect threshold concentration.

In evaluating a second-tier petition, background concentrations of the applicable pollutants must be considered. If the emissions of a TAP result in an increased cancer risk of greater than 10 in 1 million (equivalent to 1 in 100,000), then an applicant may request that Ecology conduct a third-tier review. For non-carcinogens, a similar path exists, but there is no specified numerical criterion to indicate when a third-tier review is triggered.

If an applicant is unable to demonstrate compliance with the second-tier conditions, then approval can be requested under a third-tier review. A third-tier review (which is not required for the development

[1] If the estimated increase of emissions of a TAP or TAPs from a new or modified project is below the *de minimis* emissions threshold(s) found in WAC 173-460-150, the project is exempt from review under Chapter 173-460 WAC.

of ETL III at the PTC) is a risk management decision in which Ecology makes a decision that the risk of the project is acceptable based on a 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.

3.2 BACT AND TBACT FOR ETL III TEST CELLS

NWCAA is responsible for establishing BACT and tBACT for controlling criteria pollutants and TAPs emitted from the proposed test cells. Engine testing protocols used at the PTC are intended to evaluate engine performance under a wide range of operational scenarios. In some cases, emission control technologies would interfere with the objectives of specific tests that are integral to the process of verifying that the engines meet rigorous performance standards, including emission limits. After-treatment systems and/or in-cylinder combustion control technologies are used to achieve and demonstrate compliance with the specific engine programs being tested. Not every control technology integral to the PACCAR after-treatment system will be used for every test, but every test will be run with after-treatment and/or in-cylinder combustion control technologies required to adhere to the emission limits established for the specific engine being tested.

The proposed BACT and tBACT determinations are summarized in Tables 3-1 and 3-2, respectively.

3.2.1 BACT FOR NITROGEN OXIDES

More than 95 percent of the tests conducted within the proposed test cells will adhere to the EPA 2010 or EuroVI emission standards for heavy-duty highway engines; compliance with these standards involves the use of selective catalytic reduction (SCR) to control emissions of nitrogen oxides (NO_x). Thus, the after-treatment system for most tests cells will include the use of SCR as an emission control technology. Other tests to demonstrate compliance with less stringent standards may involve operation without SCR controls, but will still adhere to the overall emissions described in Section 2.2. The post-combustion SCR control unit integrated into after-treatment systems used at the PTC should be considered BACT for NO_x in most tests conducted at the PTC. For those tests in which SCR is not appropriate, in-cylinder combustion modifications will be used as BACT for controlling NO_x emissions.

3.2.2 BACT FOR CARBON MONOXIDE

More than 95 percent of the tests conducted at the PTC adhere to the EuroVI emission standards for heavy-duty highway engines; compliance with these standards involves the use of diesel oxidation catalysts (DOCs) to control carbon monoxide (CO) emissions. Thus, the after-treatment system for most tests cells will include the use of DOCs as an emission control technology. Other tests to demonstrate compliance with less stringent standards may involve operation without DOCs, but will still adhere to the overall emissions described in Section 2.2. The post-combustion DOCs integrated into after-treatment systems used at the PTC should be considered BACT for CO in most tests conducted at the PTC. For those tests in which DOCs are not appropriate, combustion modifications will be used as BACT for controlling CO emissions.

3.2.3 BACT for Particulate Matter

More than 95 percent of the tests conducted at the PTC adhere to the EPA 2010 and EuroVI emission standards for heavy-duty highway engines; compliance with these standards involves the use of diesel particulate filters (DPFs) to control PM emissions. Thus, the after-treatment system for most tests cells will include the use of DPFs as an emission control technology. Other tests to demonstrate compliance with less stringent standards may involve operation without DPFs, but will still adhere to the overall emissions described in Section 2.2. The post-combustion DPFs integrated into after-treatment systems used at the PTC should be considered BACT for PM in most tests conducted at the PTC. DPFs will be used unless a specific test calls for engine operation without the use of a DPF.

3.2.4 BACT for Sulfur Oxides and Volatile Organic Compounds

Control technologies for internal combustion engines (ICEs) are typically geared toward reductions in NO_x and CO emissions. Emissions of sulfur oxides (SO_x) and VOCs will be controlled by using ultra low sulfur diesel (ULSD) fuel (with no more than 0.0015 weight percent sulfur), except when its use is precluded by the requirements of specific tests, and using appropriate combustion practices. These methods should be considered as BACT for SO_x and VOCs.

3.2.5 BACT for Toxic Air Pollutants (TBACT)

TAPs emitted by diesel engines include DEEP, nitrogen dioxide (NO_2), sulfur dioxide (SO_2), CO, benzene, toluene, xylenes (m-, p-, and o-xylene), propylene, formaldehyde, acetaldehyde, acrolein, naphthalene, and polycyclic aromatic hydrocarbons (PAHs) that include benz(a)anthracene, chrysene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, indeno(1,2,3-cd)pyrene, and dibenz(a,h)anthracene. The proposed tBACT for benzene, toluene, xylenes, propylene, formaldehyde, acetaldehyde, acrolein, naphthalene, and all PAHs includes using appropriate combustion practices. The tBACT analysis for CO, DEEP, NO_2, and SO_2 are accounted for in the BACT analysis for CO (as a criteria pollutant), PM, NO_x and SO_x, respectively.

3.3 FIRST-TIER TOXICS SCREENING REVIEW FOR ETL III TEST CELLS

The first-tier TAP assessment compares the forecast emission rates to the Small-Quantity Emission Rates (SQERs) and compares the maximum ambient impacts at any sensitive receptor to the ASILs.

Table 3-3 shows the calculated emission rates for each TAP emitted from the ETL III test cells, and compares the emission rates to the SQERs. The SQERs are emission thresholds, below which NWCAA does not require an air quality impact assessment for the listed TAP. Table 3-3 lists the "SQER Ratio" of the emission rate for the ETL III test cells compared to the SQER. The maximum emission rates for DEEP, benzene, 1,3-butadiene, and naphthalene exceed their respective SQERs, so an ambient impact assessment based on atmospheric dispersion modeling was required for those pollutants.

Ecology requires facilities to conduct a first-tier screening analysis for each TAP whose emission exceeds its SQER by modeling the 1^{st}-highest 1-hour, 1^{st}-highest 24-hour, or annual impacts (based on the averaging period listed for each TAP in WAC 173-460-150) at or beyond the project boundary, then comparing the modeled values to the ASILs (WAC 173-460-080). For this analysis, annual average impacts were modeled based on a worst-case operational scenario of 24 hours per day for 365 days per year for 5 years, with the American Meteorological Society/EPA Regulatory Model (AERMOD).

Table 3-4 presents the first-tier ambient concentration screening analysis for each TAP whose emission rate exceeds its SQER. Details on the methodologies for the modeling are provided in the *Notice of Construction Application Supporting Documentation* report (Landau Associates 2013). All of the modeled maximum impacts occur at the unoccupied facility boundary. The maximum annual-average DEEP impact at the unoccupied facility boundary far exceeds its ASIL, while the impacts for all TAPs other than DEEP are less than their respective ASILs. Therefore, DEEP is the only TAP triggering a requirement for a second-tier HIA.

3.4 SECOND-TIER REVIEW PROCESSING REQUIREMENTS

In order for Ecology to review the second-tier petition, each of the following regulatory requirements under Chapter 173-460-090 must be satisfied:

(a) The permitting authority has determined that other conditions for processing the NOC Order of Approval have been met, and has issued a preliminary approval order.

(b) Emission controls contained in the preliminary NOC approval order represent at least tBACT.

(c) The applicant has developed an HIA protocol that has been approved by Ecology.

(d) The ambient impact of the emissions increase of each TAP that exceeds ASILs has been quantified using refined air dispersion modeling techniques as approved in the HIA protocol.

(e) The second-tier review petition contains an HIA conducted in accordance with the approved HIA protocol.

Ecology provided comments to Landau Associates' HIA protocol [item (c) above]. Ecology's comments were addressed as part of this HIA.

3.5 SECOND-TIER REVIEW APPROVAL CRITERIA

As specified in WAC 173-460-090(7), Ecology may recommend approval of a project that is likely to cause an exceedance of ASILs for one or more TAPs only if:

- Ecology determines that the emission controls for the new and modified emission units represent tBACT
- The applicant demonstrates that the increase in emissions of TAPs is not likely to result in an increased cancer risk of more than 1 in 100,000
- Ecology determines that the non-cancer hazard is acceptable.

The remainder of this document discusses the HIA conducted by Landau Associates.

4.0 HEALTH IMPACT ASSESSMENT

This HIA was conducted according to the requirements of WAC 173-460-090 and guidance provided by Ecology. The HIA addresses the public health risk associated with exposure to DEEP from PACCAR's proposed test cells and existing sources of DEEP in the vicinity. While the HIA is not a complete risk assessment, it generally follows the four steps of the standard health risk assessment approach proposed by the National Academy of Sciences (NAS 1983, 1994). These four steps are: 1) hazard identification; 2) exposure assessment; 3) dose-response assessment; and 4) risk characterization. As described later in this document, the HIA did not consider exposure pathways other than inhalation.

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?).

Although the second-tier HIA is triggered solely by potential ambient air impacts of DEEP, the toxicity of other TAPs with emission rates exceeding the SQERs was also reviewed to consider whether additive toxicological effects should be considered in the HIA.

4.1.1 OVERVIEW OF DEEP TOXICITY

Diesel engines emit very small fine [<2.5 micrometers (μm)] and ultrafine (<0.1 μm) particles. These particles can easily enter deep into the lungs when inhaled. Mounting evidence indicates that inhaling fine particles can cause numerous adverse health effects.

Studies of humans and animals specifically exposed to DEEP show that diesel particles can cause both acute and chronic health effects including cancer. Ecology has summarized these health effects in a document titled *Concerns about Adverse Health Effects of Diesel Engine Emissions* (Ecology 2008).

The following health effects have been associated with exposure to diesel particles:

- Inflammation and irritation of the respiratory tract

- Eye, nose, and throat irritation along with coughing, labored breathing, chest tightness, and wheezing
- Decreased lung function
- Worsening of allergic reactions to inhaled allergens
- Asthma attacks and worsening of asthma symptoms
- Heart attack and stroke in people with existing heart disease
- Lung cancer and other forms of cancer
- Increased likelihood of respiratory infections
- Male infertility
- Birth defects
- Impaired lung growth in children.

It is important to note that the estimated levels of DEEP emissions from the PTC that will potentially impact people will be much lower than levels associated with many of the health effects listed above. For the purpose of determining whether PACCAR's project-related and cumulative DEEP impacts are acceptable, non-cancer hazards and cancer risks are quantified and presented in the remaining sections of this document.

4.1.2 Overview of Naphthalene Toxicity

Naphthalene is a natural constituent of coal tar. It is a white crystalline power and it is present in gasoline and diesel fuels. Naphthalene is released into the atmosphere as a component of the exhaust from the use of diesel engines. Naphthalene is classified as a possible human carcinogen by the EPA. Studies of human occupational exposure and animal exposure have shown that naphthalene can cause acute and chronic health effects. The EPA has summarized these health effects on its Technology Transfer Network Air Toxics website (EPA 2000; DOHHS 2005).

It is important to note that the estimated levels of naphthalene emissions from the ETL III test cells are below the levels that are associated with the health effects listed above. The EPA identifies the critical effect of naphthalene inhalation exposure as "nasal effects" including hyperplasia of the respiratory epithelium in the nose and metaplasia of the olfactory epithelium. Because critically affected organs/tissue resulting from exposure to naphthalene are different from those critically affected organs/tissue resulting from exposure to DEEP, the non-cancer risks from naphthalene would not be additive to those resulting from exposure to DEEP. However, risks associated with exposure to naphthalene ambient air impacts were conservatively evaluated as additive in this HIA.

4.1.3 OVERVIEW OF BENZENE TOXICITY

Benzene is an organic chemical compound and a component of diesel fuel. Benzene is released into the atmosphere as a component of the exhaust from the use of diesel engines. Benzene is classified as a carcinogen. Toxic effects of exposure are primarily on the hematopoietic system. The affected tissue/organ associated with benzene exposure is bone marrow. The EPA's Integrated Risk Information System (IRIS) database lists the critical effect of chronic inhalation exposure of benzene as decreased lymphocyte count (EPA 2003). Chronic benzene exposure can depress the immune system and lead to leukemia in humans.

Benzene does not present any cross-media impact potential as there are no deposition impacts to soil or water from the vapor phase emissions. Because critically affected organs/tissue resulting from exposure to benzene are different from those critically affected organs/tissue resulting from exposure to DEEP, the non-cancer risks from benzene would not be additive to those resulting from exposure to DEEP. However, risks associated with exposure to benzene ambient air impacts were conservatively evaluated as additive in this HIA.

4.1.4 OVERVIEW OF 1,3-BUTADIENE TOXICITY

1,3-Butadiene is a gas at room temperature and is a component of diesel fuel. 1,3-Butadiene is released into the atmosphere as a component of the exhaust from the use of diesel engines. 1,3-Butadiene is classified as a carcinogen. Toxic effects of exposure are primarily on the reproductive and lymphohematopoietic systems (EPA 2002a). The affected tissue/organ associated with 1,3-butadiene exposure is lymphatic tissue. The EPA's IRIS database lists the critical effect of chronic inhalation exposure of 1,3-butadiene as ovarian atrophy and the dominant lethal effects in humans would likely be manifested as infertility or spontaneous abortions.

1,3-Butadiene does not present any cross-media impact potential as there are no deposition impacts to soil or water from the vapor phase emissions. Because critically affected organs/tissue resulting from exposure to 1,3-butadiene are different from those critically affected organs/tissue resulting from exposure to DEEP, the non-cancer risks from 1,3-butadiene would not be additive to those resulting from exposure to DEEP. However, risks associated with exposure to 1,3-butadiene ambient air impacts were conservatively evaluated as additive in this HIA.

4.2 EXPOSURE ASSESSMENT

An exposure assessment involves estimating the extent that the public is exposed to a chemical substance emitted from a facility. This includes:

- Identifying routes of exposure

- Estimating long- and/or short-term offsite pollutant concentrations
- Identifying exposed receptors.
- Estimating the duration and frequency of receptors' exposure.

4.2.1 Identifying Routes of Potential Exposure

Humans can be exposed to chemicals in the environment through inhalation, ingestion, or dermal contact. The primary route of exposure to most air pollutants is inhalation; however, some air pollutants may also be absorbed through ingestion or dermal contact. Ecology uses guidance provided in California's *Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments* (CalEPA 2003) to determine which routes and pathways of exposure to assess for chemicals emitted from a facility. Chemicals for which Ecology assesses multiple routes and pathways of exposure are presented in Table 4-1.

DEEP consists of ultra-fine particles (approximately 0.1 to 1 micron in size) that behave like a gas and do not settle out of the downwind plume by gravity. DEEP particles will eventually be removed from the atmosphere and be deposited onto the ground surface by either molecular diffusion or by being incorporated into rain droplets, but that deposition process is slow and will likely occur many miles downwind of the PACCAR facility. At those far downwind distances, the resulting DEEP concentrations in the surface soil will likely be indistinguishable from regional background values.

It is possible that very low levels of polycyclic aromatic hydrocarbons (PAHs) and the few other persistent chemicals in DEEP will build up in food crops, soil, and drinking water sources downwind of the PTC. However, given the very low levels of PAHs and other multi-exposure route-type TAPs that will be emitted from ETL III, quantifying exposures via pathways other than inhalation is very unlikely to yield significant concerns. Further, inhalation is the only route of exposure to DEEP that has received sufficient scientific study to be useful in human health risk assessment. Therefore, in the case of PTC test cell emissions, only inhalation exposure to DEEP is evaluated.

4.2.2 Estimating Pollutant Concentrations

DEEP emissions may be carried by the wind and may impact people living and working in the immediate area. The level of these pollutants in offsite air depends in part on how much is emitted, wind direction, and other weather-related variables at the time the pollutants are emitted. To estimate where pollutants will disperse after they are emitted from the PTC, Landau Associates conducted air dispersion modeling, which incorporates emissions, meteorological, geographical, and terrain information to estimate pollutant concentrations downwind from a source.

Each of the proposed ETL III test cells (proposed) and ETL I and II test cells (existing local background) were modeled as individual discharge points. Landau Associates used the following model inputs to estimate ambient impacts:

- AERMOD with Plume Rise Model Enhancements (PRIME) algorithm for building downwash (Version 12345).
- Five years of sequential hourly meteorological data from Skagit Regional Airport (2007 to 2011).
- Twice-daily upper air data from Quillayute, WA (2007 to 2011) to define mixing heights.
- Skagit County area shuttle radar topography mission files (which describe local topography and terrain).
- Skagit County area digital land classification files (which describe surface characteristics).
- Each test cell's emissions were modeled with stack heights of 52 feet (ETL I), 56 feet (ETL II), and 55 feet (ETL III) above ground level and stack inside diameters of 31 inches (ETL I), 16 inches (ETL II), and 10 inches (ETL III). ETL-specific exhaust gas temperatures and velocities were used.
- The building dimensions for the multiple buildings at the PTC were included to account for building downwash.
- The receptor grid for the AERMOD modeling domain at or beyond the facility boundary was established using a variable Cartesian grid:
  - 12.5-meter (m) spacing from emission source to 350 m
  - 25-m spacing from 350 m to 450 m
  - 50-m spacing from 450 m to 950 m
  - 100 m spacing from 950 m to 2,100 m
  - 300 m spacing from 2,100 m to 4,500 m
  - 600 m spacing beyond 4,500 m.

4.2.3 IDENTIFYING POTENTIALLY EXPOSED RECEPTORS

There are several different land use types within the general vicinity of the PTC. Locations where people could be exposed to project-related emissions are identified on Figure 2-4. Most importantly, two single-family residences are on RRv-zoned land within close proximity to the PTC. Additionally, there are two business receptors (a tank farm and an airport building) located on Aviation Related (AVR)-zoned land and one business receptor (agricultural building) located on RRv-zoned land within close proximity to the PTC. The residential and business receptors are potentially at risk for exposure to project-related emissions. Typically, Ecology considers exposures occurring at maximally exposed boundary, residential, and business/commercial areas to capture worst-case exposure scenarios.

4.2.3.1 Receptors Maximally Exposed to DEEP

Maximally exposed receptors of different use types and the direction and distance of those receptors from the PTC are presented on Table 4-2. These receptors represent the locations of various land uses that are most impacted by DEEP emissions from the facility. This table also shows the estimated 70-year average exposure concentration at each maximally exposed receptor for emissions from the proposed ETL III test cells.

Figure 4-1 shows a color-coded map of estimated 70-year annual average DEEP concentrations attributable solely to DEEP emissions from the proposed ETL III test cells. Figure 4-1 presents the ambient impacts of PACCAR's project and each of the maximally exposed receptors representing different land uses. The concentrations at the Maximally Impacted Boundary Receptor (MIBR), Maximally Impacted Residential Receptor (MIRR), and Maximally Impacted Commercial Receptor (MICR) are highlighted. The modeling indicates that ETL III emissions impact multiple existing residences to the southwest and northwest at a level exceeding the ASIL. The blue contour line [0.00333 micrograms per cubic meter ($\mu g/m^3$)] represents the ASIL. Receptors at all locations outside the blue contour are forecast to be exposed to concentrations less than the ASIL.

Figure 4-2 shows a contour map of the 70-year annual average DEEP concentrations attributable to the combined test cells at the PTC, including the existing eight and the four proposed test cells.

4.2.4 EXPOSURE FREQUENCY AND DURATION

The likelihood that someone is exposed to DEEP from test cells at the PTC depends on local wind patterns, the frequency of engine testing, and how much time people spend in the immediate area. As discussed previously, the air dispersion model uses emissions and meteorological information (and other assumptions) to determine ambient DEEP concentrations in the vicinity of the PTC.

This analysis considers the land use surrounding the PTC to estimate the amount of time a given receptor could be exposed. For example, people are more likely to be exposed frequently and for a longer duration if the source impacts residential locations because people spend much of their time at home. People working in offices or on commercial properties in the area are likely exposed to test cell-related emissions only during the hours that they spend working near the facility.

This analysis uses simplified assumptions about receptors' exposure frequency and duration and assumes that people located at residential receptors are potentially continuously exposed, meaning they never leave their property. These behaviors are not typical; however, these assumptions are intended to avoid underestimating exposure so that public health protection is ensured. Workplace and other non-residential exposures are also considered, but adjustments are often made because the amount of time that people spend at these locations is more predictable than time spent at their homes. These adjustments are presented in Section 4.4.2 when quantifying cancer risk from intermittent exposure to DEEP.

4.2.5 Background Exposure to Pollutants of Concern

WAC 173-460-090 states, "Background concentrations of TAPs will be considered as part of a second tier review." The word "background" is often used to describe exposures to chemicals that come from existing sources, or sources other than those being assessed.

To estimate DEEP background concentrations, ambient impacts from existing test cells at the PTC were modeled using the methodology described in Section 4.2.2. Regional DEEP background concentrations were estimated based on regional data from the EPA's National Air Toxics Assessment (NATA) database (EPA 2011).

4.2.6 Cumulative Exposure to DEEP

Table 4-3 shows the calculated cumulative DEEP concentrations near the PTC based on allowable emissions from proposed ETL III, local background emissions (from ETL I and II), and regional background sources.

The maximum 70-year cumulative concentration at a residence near the PTC is estimated at 0.153 $\mu g/m^3$ (approximately 46 times greater than the DEEP ASIL). This is modeled to occur at R-1, which is southwest of the PTC. It is important to note that the estimated ambient levels of DEEP are based on allowable (permitted) emissions instead of actual emissions. Actual emissions are likely to be much lower than what the facilities are permitted for, but worst-case emissions were used to avoid underestimating cumulative DEEP exposure concentrations.

4.3 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.

4.3.1 Dose-Response Assessment for DEEP

The EPA and California Office of Environmental Health Hazard Assessment (OEHHA) developed toxicological values for DEEP evaluated in this project (EPA 2002b; EPA website 2013; CalEPA 1998). These toxicological values are derived from studies of animals that were exposed to a known amount (concentration) of DEEP, or from epidemiological studies of exposed humans, and are intended to represent a level at or below which adverse non-cancer health effects are not expected, and a metric by which to quantify increased risk from exposure to a carcinogen. Table 4-4 shows DEEP non-cancer and cancer toxicity values.

The EPA's reference concentration (RfC) and OEHHA's reference exposure level (REL) for diesel engine exhaust (measured as DEEP) was derived from dose-response data on inflammation and changes in the lung from rat inhalation studies. Each agency established a level of 5 $\mu g/m^3$ as the concentration of DEEP in air at which long-term exposure is not expected to cause adverse non-cancer health effects.

NAAQS and other regulatory toxicological values for short- and intermediate-term exposure to particulate matter have been promulgated, but values specifically for DEEP exposure at these intervals do not currently exist.

OEHHA derived a unit risk factor (URF) for estimating cancer risk from exposure to DEEP. The URF is based on a meta-analysis of several epidemiological studies of humans occupationally exposed to DEEP. URFs are expressed as the upper-bound probability of developing cancer, assuming continuous lifetime exposure to a substance at a concentration of 1 $\mu g/m^3$, and are expressed in units of inverse concentration [i.e., $(\mu g/m^3)^{-1}$]. OEHHA's URF for DEEP is 0.0003 $(\mu g/m^3)^{-1}$ meaning that a lifetime of exposure to 1 $\mu g/m^3$ of DEEP results in an increased individual cancer risk of 0.03 percent or a population cancer risk of 300 excess cancer cases per million people exposed.

4.4 RISK CHARACTERIZATION

Risk characterization involves the integration of data analyses from each step of the HIA to determine the likelihood that the human population in question will experience any of the various health effects associated with a chemical under its known or anticipated conditions of exposure.

4.4.1 EVALUATING NON-CANCER HAZARDS

In order to evaluate the potential for non-cancer adverse health effects that may result from exposure to air pollutants, exposure concentrations at each receptor location are compared to relevant non-cancer toxicological values (i.e., RfC, REL). If a concentration exceeds the RfC or REL, this indicates only the potential for adverse health effects. The magnitude of this potential can be inferred from the degree to which this value is exceeded. This comparison is known as a hazard quotient (HQ) and is given by the equation below:

$$HQ = \frac{\text{Concentration of pollutant in air } (\mu g/m^3)}{\text{RfC or REL}}$$

An HQ of 1 or less indicates that the exposure to a substance is not likely to result in adverse non-cancer health effects. As the HQ increases above 1, the probability of adverse human health effects increases by an undefined amount. However, it should be noted that an HQ above 1 is not necessarily indicative of health impacts due to the application of uncertainty factors in deriving toxicological reference values (e.g., RfC and REL).

4.4.1.1 Hazard Quotient – DEEP

The chronic HQ for DEEP exposure is calculated using the following equation:

$$\text{Chronic HQ} = \frac{\text{Annual average DEEP concentration } (\mu g/m^3)}{5\ \mu g/m^3}$$

HQs were calculated for the maximally exposed residential and workplace receptors. Because chronic toxicity values (RfCs and RELs) are based on a continuous exposure, an adjustment is sometimes necessary or appropriate to account for people working at business/commercial properties who are exposed for only 8 hours per day, 5 days per week. While EPA risk assessment guidance recommends adjusting to account for periodic instead of continuous exposure, OEHHA does not employ this practice. For the purpose of this evaluation, an RfC or REL of 5 $\mu g/m^3$ was used as the chronic risk-based concentration for all scenarios where receptors could be exposed frequently (e.g., residences, work places, or schools).

Table 4-5 shows chronic HQs at the maximally exposed receptors near the PTC attributable to DEEP exposure from all sources. HQs are several-fold lower than 1.0 for all receptors' cumulative exposure to DEEP. This indicates that adverse non-cancer effects are not likely to result from chronic exposure to DEEP emitted from the PTC.

4.4.1.2 Combined Hazard Quotient for All Pollutants Whose Emission Rates Exceed SQER

Four TAPs (i.e., DEEP, naphthalene, benzene, and 1,3-butadiene) emitted by the PTC have emission rates exceeding their respective SQERs and, therefore, have the potential to cause ambient concentrations high enough to cause acute or chronic non-cancer inhalation health risks. The receptor locations of concern are the maximally impacted boundary receptor (MIBR), the maximally impacted commercial receptors (MICR), and the property line of the adjacent residential location or maximally impacted residential receptor (MIRR). Tables 4-6 through 4-8 show modeled concentrations, risk-based concentrations (RBCs), and HQs for each receptor point. All modeled concentrations and RBCs are in $\mu g/m^3$. The chronic hazard index (HI) for each location is the sum of annual time-weighted average (TWA) HQs for DEEP, naphthalene, benzene, and 1,3-butadiene. The acute HQ for each location is the 1-hour TWA HQ for benzene (the only pollutant with an emission rate above the SQER with an acute RBC).

Table 4-6 shows the impacts at the MIBR for DEEP, naphthalene, benzene, and 1,3-butadiene. The acute HQ of approximately 0.0002 and the chronic HI of 0.01 are much lower than 1.0. This indicates that the MIBR is not likely to experience either acute or chronic non-cancer adverse health effects attributable to emissions from the PTC.

Table 4-7 shows the HIs at the Tank Farm, which is the MICR. The acute HQ of approximately 0.00008 and the chronic HI of 0.002 are both lower than 1.0. This indicates that the MICR is not likely to experience either acute or chronic non-cancer adverse health effects attributable to emissions from the PTC.

Table 4-8 shows the HIs at the maximally-impact residential receptor (MIRR). The acute HQ of approximately 0.0001 and the chronic HI of 0.004 are both lower than 1.0. This indicates that the MIRR is not likely to experience either acute or chronic non-cancer adverse health effects attributable to emissions from the PTC.

The information in Table 4-6 through 4-8 suggests that both chronic and acute health effects are unlikely to occur even under worst-case conditions at the maximally impacted locations. At times when unfavorable air dispersion conditions occur coincident with a maximum operating scenario, the combined HQs (i.e., the hazard index) from DEEP, naphthalene, benzene, and 1,3-butadiene are modeled to be less than 1. If the HI is less than 1, then the risk is generally considered acceptable.

4.4.2 QUANTIFYING AN INDIVIDUAL'S INCREASED CANCER RISK

4.4.2.1 Cancer Risk from Exposure to DEEP

Cancer risk is estimated by determining the concentration of DEEP at each receptor point and multiplying it by its respective URF. Because URFs are based on a continuous exposure over a 70-year lifetime, exposure duration and exposure frequency are important considerations.

The formula used to determine cancer risk is as follows:

$$\text{Risk} = \frac{C_{Air} \times URF \times EF1 \times EF2 \times ED}{AT}$$

The exposure frequencies for each receptor type are shown below, based on Ecology's judgment from review of published risk evaluation guidelines.

EXPOSURE FREQUENCIES FOR EACH RECEPTOR TYPE

Parameter	Description	Value Based on Receptor Type					Units
		Residential	Worker	School-Staff	School-Student	Boundary	
CAir	Concentration in air at the receptor	See Table 4-3					$\mu g/m^3$
URF	Unit Risk Factor	0.0003					$(\mu g/m^3)^{-1}$
EF1	Exposure Frequency	365	250	200	180	250	Days/Year
EF2	Exposure Frequency	24	8	8	8	2	Hours/Day
ED	Exposure Duration	70	40	40	7 (Elem) 4 (HS & College)	30	Years
AT	Averaging Time	613,200					Hours

Based on the factors listed above, Table 4-9 shows the resulting Unit Risk Factor for each exposure scenario.

Current regulatory practice assumes that a very small dose of a carcinogen will give a very small cancer risk. Cancer risk estimates are, therefore, not yes or 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 this approach 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. Guidelines on cancer risk from the EPA reflect the potential that thresholds for some carcinogenesis exist. However, the EPA still assumes no threshold unless sufficient data indicate otherwise.

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 1×10^{-6} means that if 1 million people are exposed to a carcinogen, one excess cancer might occur, or a person's chance of getting cancer in their lifetime increases by 1 in 1 million or 0.0001 percent. 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 upper-bound theoretical estimates. In other words, each is the estimate of the plausible upper limit, or highest likely true value of the quantity of risk.

Table 4-10 shows ranges of estimated worst-case residential, business, and fence line receptor increased cancer risks attributable to DEEP exposure near the PTC. Cancer risks attributable to the proposed project are less than 1 in 100,000 (1×10^{-5}). The highest risk occurs at the residential home to the southwest of the PTC (5.1×10^{-6}). Under Chapter 173-460 WAC, Ecology may recommend approval of a project if the applicant demonstrates that the increase in emissions of TAPs is not likely to result in an increased cancer risk of more than 1 in 100,000 (1×10^{-5}).

As part of the second-tier risk evaluation, Ecology also considers the cumulative impacts of DEEP emissions in the project vicinity. Note that Chapter 173-460 WAC does not currently contain a numerical limit on allowable cumulative cancer risks.

The results, as shown in Table 4-10, indicate that the cumulative cancer risk for the maximally impacted current residential receptor near the PTC is approximately 46 in 1 million. This risk occurs at an existing residence to the southwest of the facility. The maximum cumulative cancer risk at an existing commercial business near the PTC is approximately 5.5 in 1 million. This risk occurs at a tank farm located to the southeast of the facility.

4.4.2.2 Cancer Risk from Exposure to All Potential Carcinogens

Based on the estimated emissions of all potentially carcinogenic compounds from the proposed project alone, the emission rates for all of the carcinogenic constituents are less than Ecology's SQERs except for DEEP, naphthalene, benzene, and 1,3-butadiene. The SQERs are Ecology's screening threshold emission rates below which the WAC 173-460 regulation indicates there is negligible potential for ambient air quality impacts. The maximum permitted emission rates for most toxic pollutants emitted from the proposed test cells are less than their respective SQERs. Regardless of the SQER comparison, the emission rate for every carcinogenic constituent was considered in the cumulative cancer analysis, which is shown in Table 4-11.

As indicated in Table 4-11, the cancer risk associated with DEEP alone at MIRR (R-1, the SW home) is 5.1 per million. The other recognized carcinogenic compounds contribute negligibly to the overall cancer risk (i.e., 0.1 per million). The combined cancer risk caused by all constituents is 5.2 per million.

5.0 UNCERTAINTY CHARACTERIZATION

Many factors of the HIA are prone to uncertainty. Uncertainty relates to the lack of exact knowledge regarding many of the assumptions used to estimate the human health impacts of DEEP emissions from ETL III test cells and “background” sources of DEEP. The assumptions used in the face of uncertainty may tend to overestimate or underestimate the health risks estimated in the HIA.

5.1 EMISSION FACTOR AND EXPOSURE UNCERTAINTY

One of the major uncertainties is the emission factors for TAPs emitted by diesel engines. The forecast emission rates for particulate matter used for this analysis were based on the upper range of PACCAR’s estimates for the specific diesel engines to be used. For this analysis, it was conservatively assumed that all of the particulate matter emitted from diesel generators is DEEP, with the highest level of cancer potency. The emission rates for the other TAPs were based on published emission factor data from the EPA, which are believed to be conservatively high because they were developed based on historical testing of older-technology engines.

It is difficult to characterize the amount of time that people can be exposed to DEEP emissions from the proposed ETL III test cells. For simplicity, this analysis assumed that a residential receptor is at one location for 24 hours per day, 365 days per year for 70 years. These assumptions tend to overestimate exposure.

The duration and frequency of engine testing within the test cells is also uncertain. For this permit application PACCAR conservatively estimated that it will use the test cells 24 hours per day and 365 days per year (i.e., 8,760 hours per year) for 70 years. In reality, actual operation of the ETL III test cells is likely to be no more than 3,500 hours per year. It is expected that estimates of cancer risks will be significantly overestimated by assuming test cells will operate annually at the maximum permitted level for 70 consecutive years.

5.2 AIR DISPERSION MODELING UNCERTAINTY

The transport of pollutants through the air is a complex process. Regulatory air dispersion models have been developed to estimate the transport and dispersion of pollutants as they travel through the air. The models are frequently updated as techniques that are more accurate become known, but are developed to avoid underestimating the modeled impacts. Even if all of the numerous input parameters to an air dispersion model are known, random effects found in the real atmosphere will introduce uncertainty. Typical of the class of modern steady-state Gaussian dispersion models, the AERMOD model used for the PTC analysis will likely slightly overestimate the short-term (24-hour average) impacts and somewhat underestimate the annual pollutant concentrations. The expected magnitude of the uncertainty is probably similar to the emissions uncertainty and much lower than the toxicity uncertainty.

5.3 TOXICITY UNCERTAINTY

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. To account for uncertainty when developing toxicity values (e.g., RfCs), the EPA and other agencies apply "uncertainty" factors to doses or concentrations that were observed to cause adverse non-cancer effects in animals or humans. The EPA applies these uncertainty factors so that they derive a toxicity value that is considered protective of humans including susceptible populations. In the case of the EPA's DEEP RfC, EPA acknowledges (EPA 2002b):

"… the actual spectrum of the population that may have a greater susceptibility to diesel exhaust (DE) is unknown and cannot be better characterized until more information is available regarding the adverse effects of diesel particulate matter (DPM) in humans."

Quantifying DEEP cancer risk is also uncertain. Although the EPA classifies DEEP as probably carcinogenic to humans, it has not established a URF for quantifying cancer risk. In its health assessment document, the EPA determined that "human exposure-response data are too uncertain to derive a confident quantitative estimate of cancer unit risk based on existing studies." However, the EPA suggested that a URF based on existing DEEP toxicity studies would range from 1×10^{-5} to 1×10^{-3} per $\mu g/m^3$. OEHHA's DEEP URF (3×10^{-4} per $\mu g/m^3$) falls within this range. Regarding the range of URFs, EPA states in its health assessment document for diesel exhaust (EPA 2002b):

"Lower risks are possible and one cannot rule out zero risk. The risks could be zero because (a) some individuals within the population may have a high tolerance to exposure from [diesel exhaust] and therefore not be susceptible to the cancer risk from environmental exposure, and (b) although evidence of this has not been seen, there could be a threshold of exposure below which there is no cancer risk."

Other sources of uncertainty cited in the EPA's health assessment document for diesel exhaust are:

- Lack of knowledge about the underlying mechanisms of DEEP toxicity.
- The question of whether historical toxicity studies of DEEP based on older engines is relevant to current diesel engines. It is likely that the mixture of pollutants emitted by new-technology diesel engines (such as those proposed by PACCAR) is different from older technology engines.

Table 5-1 presents a summary of how the uncertainty affects the quantitative estimate of risks or hazards.

6.0 OTHER CONSIDERATIONS

6.1 SHORT-TERM EXPOSURE TO DEEP AND $PM_{2.5}$

As discussed previously, exposure to DEEP can cause both acute and chronic health effects. However, as discussed in Section 4.3.1, reference toxicological values specifically for DEEP exposure at short-term or intermediate intervals (e.g., 24-hour values) do not currently exist. Therefore, short-term risks from DEEP exposure are not quantified in this assessment. Regardless, not quantifying short-term health risks in this document does not imply that they have not been considered. Instead, it is assumed that compliance with the 24-hour NAAQS for particulate matter with an aerodynamic diameter less than or equal to 2.5 microns ($PM_{2.5}$) is an indicator of acceptable short-term health effects from DEEP exposure. In our analysis, we assumed all DEEP emissions to be $PM_{2.5}$. The *Notice of Construction Application Supporting Documentation* report (Landau Associates 2013) concludes that emissions from the proposed project are not expected to cause or contribute to an exceedance of any NAAQS.

6.2 SHORT-TERM EXPOSURE TO OTHER TOXIC AIR POLLUTANTS

The impacts of short-term emission rates of other TAPs from the existing unmodified test cells at the PTC have not been evaluated in detail in this document because only DEEP emissions from the project exceeded the ASIL. Because emissions of other TAPs from the project were below the ASIL, no further review was required for those pollutants. Emissions below the ASIL suggest that increased health risks from these project-related pollutants are acceptable.

7.0 DISCUSSION OF ACCEPTABILITY OF RISK WITH REGARD TO SECOND-TIER REVIEW GUIDELINES

7.1 PROJECT-ONLY CANCER RISKS ARE LOWER THAN 10-PER-MILLION

As noted above, the modeled worst-case TAP concentrations at the facility boundary caused solely by emissions from the proposed test cells at the PTC are less than the ASIL values established by Ecology for all pollutants, with the exception of DEEP. The worst-case emission rates are less than the SQERs for most pollutants, with the exception of DEEP, naphthalene, benzene, and 1,3-butadiene. The long-term uncontrolled cancer risks at the nearby homes and businesses range from 0.22 to 5.1 per million for DEEP and are much lower for the other TAPs considered in this analysis. The overall cancer risk at any of the maximally exposed home and business receptors, caused solely by ETL III emissions, is estimated to be less than the 10-per-million threshold that has been established by Ecology under its second-tier review criteria.

7.2 CUMULATIVE CANCER RISK

The total cumulative DEEP cancer risks for the maximally exposed home and business receptors are as follows:

PTC ETL III-only cancer risk (R-1 SW home):	5.1 per million
Local background DEEP cancer risk:	3.6 per million
Regional background DEEP cancer risk:	37 per million
Cumulative DEEP cancer risk:	46 per million
PTC ETL III -only cancer risk (C-1 tank farm):	0.38 per million
Local background DEEP cancer risk:	0.45 per million
Regional background DEEP cancer risk:	4.7 per million
Cumulative DEEP cancer risk:	5.5 per million

7.3 NON-CANCER RISK HAZARD QUOTIENT <1.0

As described previously, the maximum HQ related to ETL III-only annual-average DEEP impacts at any maximum impacted receptor is 0.009. The maximum HQ for cumulative impacts caused by emissions of DEEP, naphthalene, benzene, and 1,3-butadiene is only 0.01. This confirms that emissions from PACCAR's proposed test cells are unlikely to cause non-cancer impacts.

8.0 USE OF THIS REPORT

This Second-Tier Risk Analysis Technical Support Document has been prepared for the exclusive use of PACCAR, Inc. and applicable regulatory agencies for specific application to the PACCAR Technical Center project in Skagit County, Washington. No other party is entitled to rely on the information, conclusions, and recommendations included in this document without the express written consent of Landau Associates. Further, the reuse of information, conclusions, and recommendations provided herein for extensions of the project or for any other project, without review and authorization by Landau Associates, shall be at the user's sole risk. Landau Associates warrants that within the limitations of scope, schedule, and budget, our services have been provided in a manner consistent with that level of care and skill ordinarily exercised by members of the profession currently practicing in the same locality under similar conditions as this project. We make no other warranty, either express or implied.

This document has been prepared under the supervision and direction of the following key staff.

LANDAU ASSOCIATES, INC.

Mark Brunner
Project Planner

Charles P. Halbert, P.E.
Principal

MWB/CPH/ccy

9.0 REFERENCES

CalEPA. 2009. *Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures*. Available at http://www.oehha.ca.gov/air/hot_spots/tsd052909.html. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. May.

CalEPA. 2011. *Air Toxics Hot Spots Risk Assessment Guidelines Part II: Technical Support Document for Cancer Potency Factors, Appendix A*. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. Available at http://oehha.ca.gov/air/hot_spots/tsd052909.html.

CalEPA. 2013. *Chronic Toxicity Summary, Naphthalene*. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. Available at http://oehha.ca.gov/air/chronic_rels/pdf/91203.pdf. Accessed May 3.

CalEPA. 2003. *The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments*. Available at http://oehha.ca.gov/air/hot_spots/HRAguidefinal.html. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. August.

CalEPA. 1998. *Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant*. Available at http://www.arb.ca.gov/toxics/dieseltac/staffrpt.pdf. Air Resources Board and Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. June.

DOHHS. 2005. *Toxicological Profile for Naphthalene, 1-Methylnaphthalene, and 2-Methylnaphthalene*. Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services. August. Available at http://www.atsdr.cdc.gov/toxprofiles/tp67.pdf.

Ecology. 2008. *White Paper: Concerns About Adverse Health Effects of Diesel Engine Emissions*. Publication No. 08-02-032. Available at https://fortress.wa.gov/ecy/publications/publications/0802032.pdf. Air Quality Program, Washington State Department of Ecology. December 3.

EPA. 1998. *Integrated Risk Information System: Naphthalene*. U.S. Environmental Protection Agency. September 17. Available at http://www.epa.gov/iris/subst/0436.htm.

EPA. 2000. *Naphthalene Hazard Summary*. U.S. Environmental Protection Agency. January. Available at http://www.epa.gov/ttnatw01/hlthef/naphthal.html.

EPA. 2002a. *Integrated Risk Information System: 1,3-Butadiene (CASRN 106-99-0)*. U.S. Environmental Protection Agency. Available at http://www.epa.gov/iris/subst/0139.htm. November 5.

EPA. 2011. *2005 Tract-Level Modeled Ambient Concentrations, Exposures, and Risks*. U.S. Environmental Protection Agency. Available at http://www.epa.gov/ttn/atw/nata2005/tables.html.

EPA. 2002b. *Health Assessment Document for Diesel Engine Exhaust*. EPA/600/8-90/057F. Available at http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=29060. National Center for Environmental Assessment, Office of Research and Development, U.S. Environmental Protection Agency. May.

EPA. 2003. *Integrated Risk Information System: Benzene (CASRN 71-43-2)*. U.S. Environmental Protection Agency. Available at http://www.epa.gov/iris/subst/0276.htm. April 17.

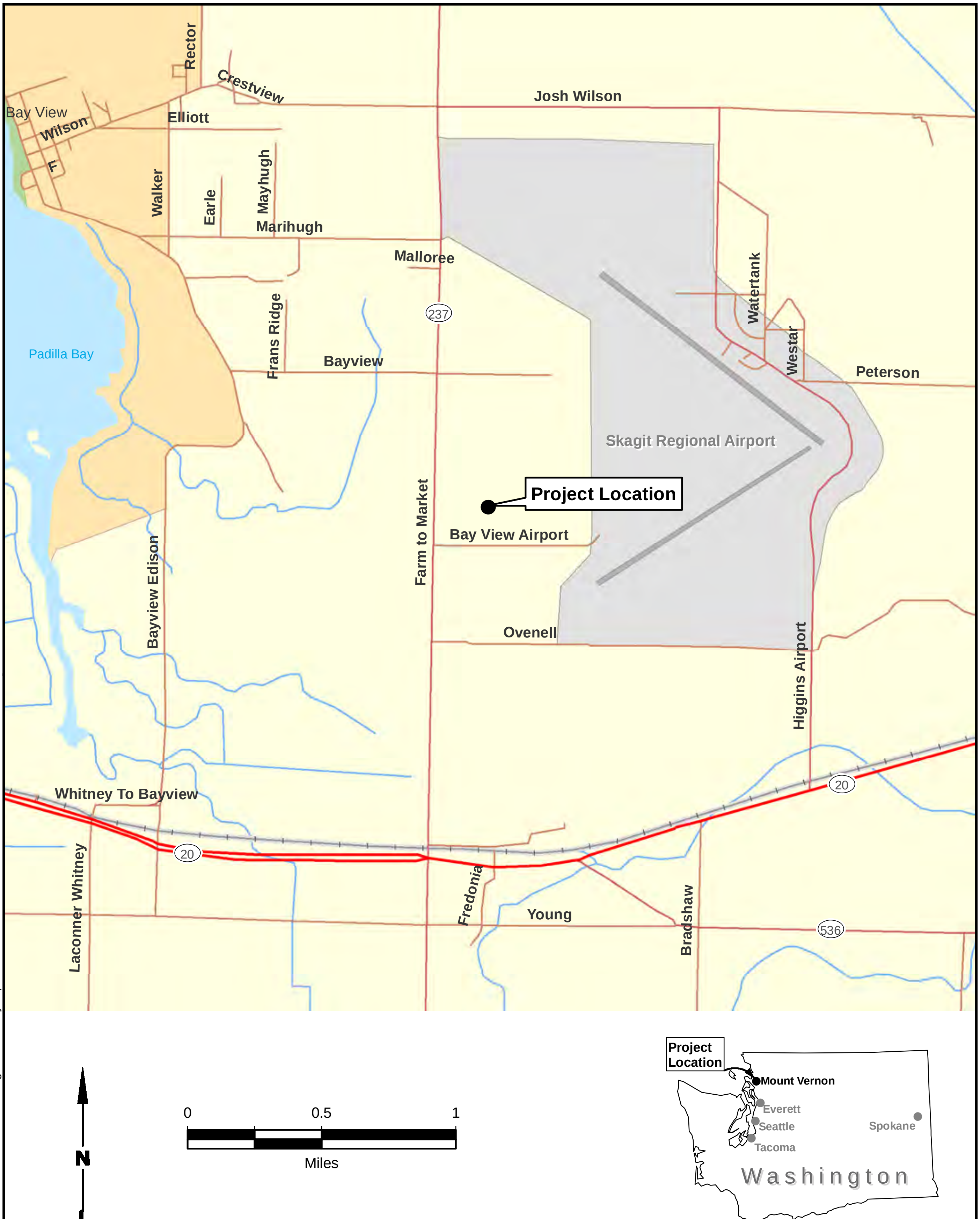
EPA website. 2013. *Integrated Risk Information System, Diesel Engine Exhaust Quickview (CASRN NA)*. http://cfpub.epa.gov/ncea/iris/index.cfm?fuseaction=iris.showQuickView&substance_ nmbr=0642. Accessed May 1.

Landau Associates. 2013. *Notice of Construction Application Supporting Documentation, Engine Test Laboratory III, PACCAR Technical Center, Skagit County, Washington*. Prepared for PACCAR, Inc. May 3.

NAS. 1994. *Science and Judgment in Risk Assessment*. National Research Council, National Academy of Sciences. National Academy Press. Washington, D.C.

NAS. 1983. *Risk Assessment in the Federal Government: Managing the Process*. Available at http://www.nap.edu/catalog.php?record_id=366. National Research Council, National Academy of Sciences. National Academy Press. Washington, D.C.

Skagit County website. 2013. *Skagit County iMAP*. http://www.skagitcounty.net/GIS/Applications/iMap/asp/iMap.asp. Accessed May 1.



Data Source: ESRI 2008

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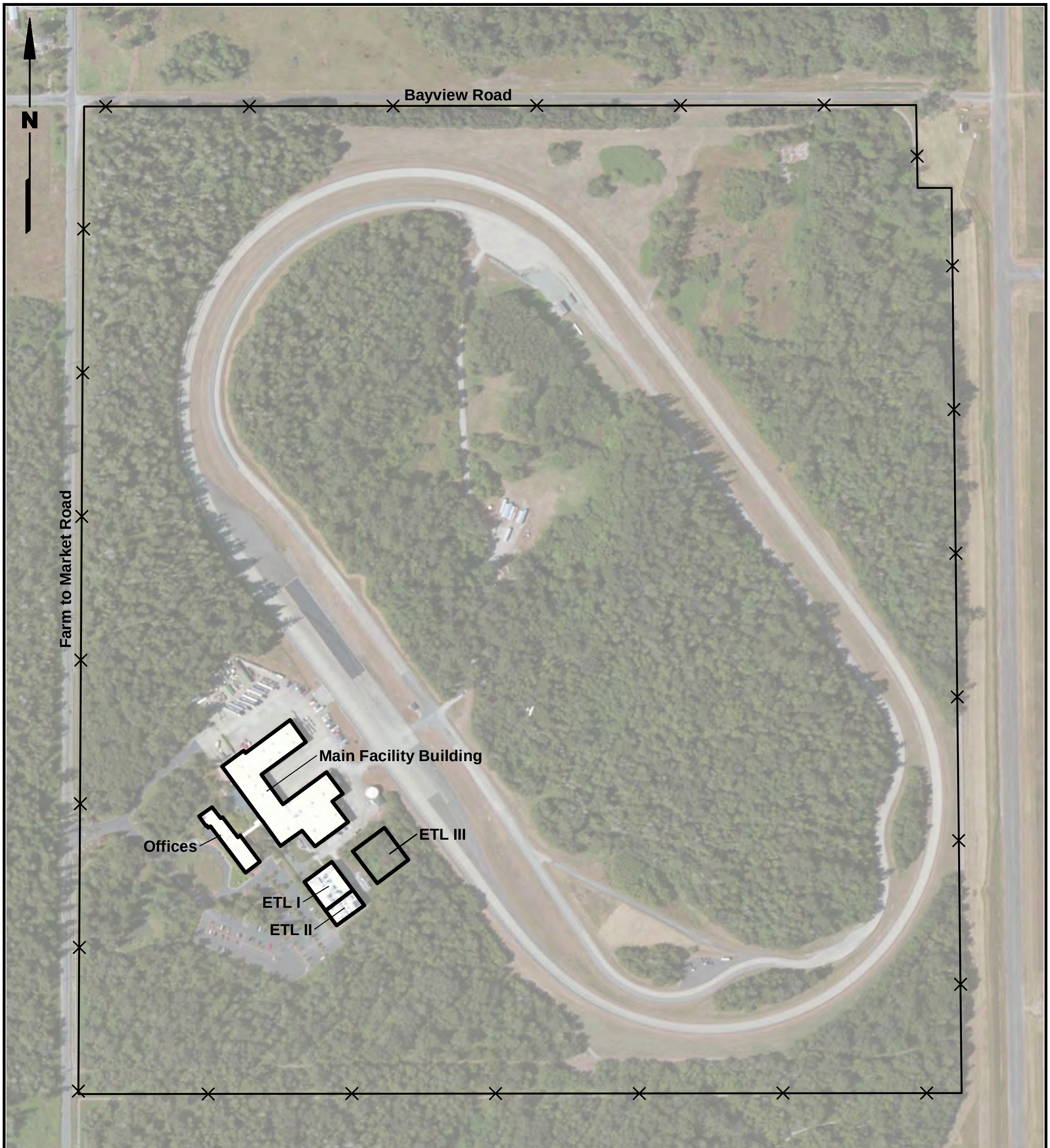
PACCAR Technical Center Skagit County, Washington	**Vicinity Map**	Figure 2-1



Legend

Building Outlines

Fence Line

Note

1. Black and white reproduction of this color original may reduce its effectiveness and lead to incorrect interpretation.

0 500 1,000

Scale in Feet

Data Source: ESRI World Imagery

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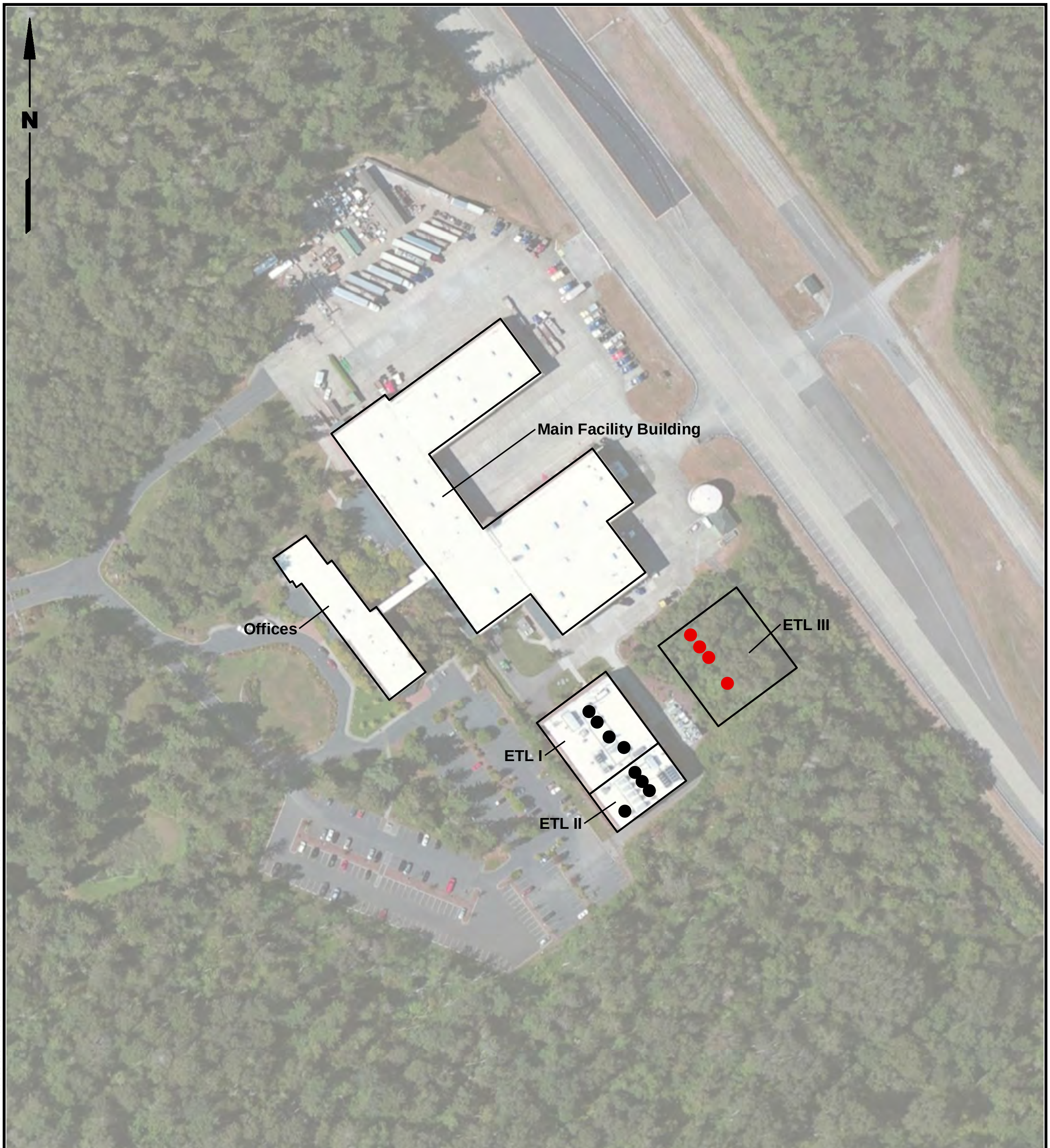
PACCAR Technical Center Skagit County, Washington	**Site Plan**	Figure 2-2



Legend

- ● Existing Test Cell Exhaust Stack
- ● New Test Cell Exhaust Stack
- ▭ Building Outlines

Note
1. Black and white reproduction of this color original may reduce its effectiveness and lead to incorrect interpretation.

0 200 400

Scale in Feet

Data Source: ESRI World Imagery

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PACCAR Technical Center Skagit County, Washington	**Exhaust Stack Locations**	Figure 2-3

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Legend

- Facility Boundary
- Land Use

Note

1. Black and white reproduction of this color original may reduce its effectiveness and lead to incorrect interpretation.

Data Source: ESRI World Imagery.

0 2,000 4,000

Scale in Feet

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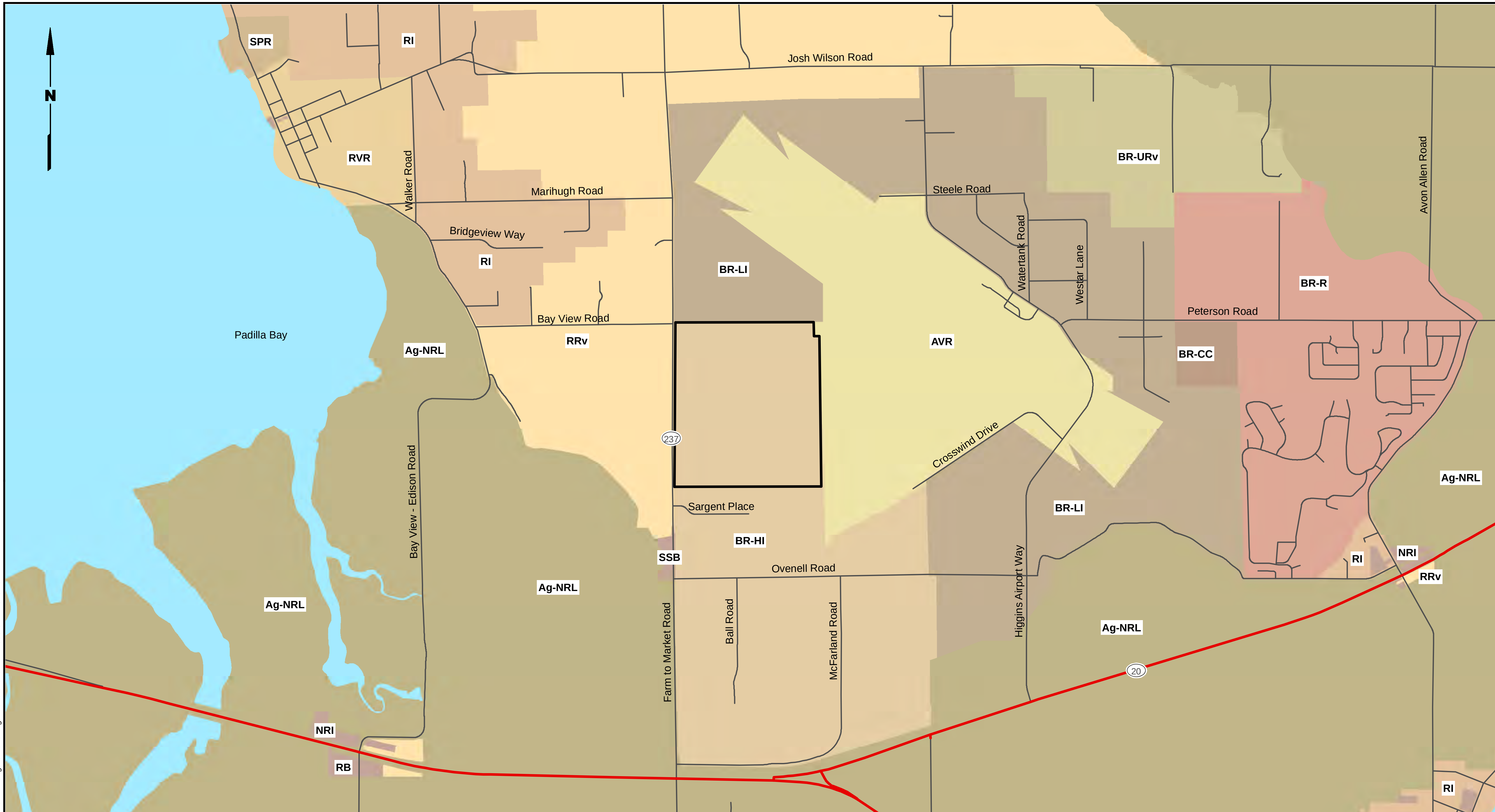
Technical Center
Skagit County, Washington

Land Use in the Vicinity of the PTC

Figure
2-4



Legend

PACCAR Facility Boundary	BR-HI - Bayview Ridge Heavy Industrial
Water	BR-LI - Bayview Ridge Light Industrial
AVR - Aviation Related	BR-R - Bayview Ridge Residential
Ag - NRL Agricultural	BR-URv - Bayview Ridge Urban Reserve
BR-CC - Bayview Ridge Community Center	NRI,RB,RMI,SSB - Commercial/Industrial
OSRSI - Public Open Space Areas of Regional/State Importance	RI - Rural Intermediate
RRv - Rural Reserve	RVR - Rural Village Residential

0 2,000 4,000

Scale in Feet

Note

1. Black and white reproduction of this color original may reduce its effectiveness and lead to incorrect interpretation.

Data Sources: Skagit County GIS; ESRI World Imagery.

Y:\Projects\136500\1010\013\Tier2HIA\Figure2-5Zoning.mxd 6/6/2013 NAD 1983 UTM Zone 10N

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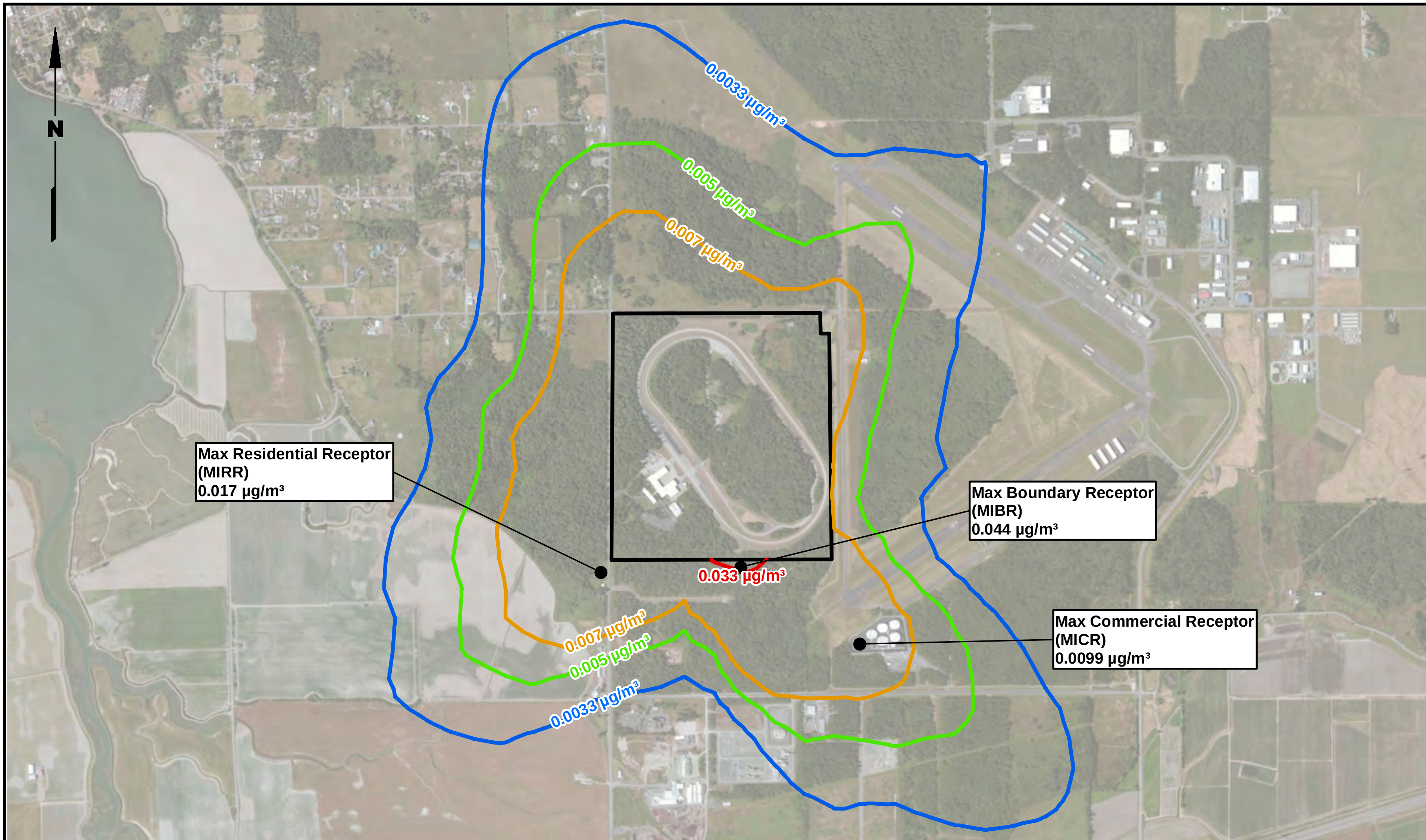
PACCAR Technical Center Skagit County, Washington	**Detailed Zoning Map for the Vicinity of the PTC**	Figure 2-5

Y:\Projects\1365001\010\013\Tier2HIA\Figure4-1DEEPNewTestCells.mxd 6/11/2013 NAD 1983 UTM Zone 10N



Legend

DEEP Concentrations

0.00333 µg/m³

0.005 µg/m³

0.007 µg/m³

0.033 µg/m³

PACCAR Facility Boundary

0 2,000 4,000

Scale in Feet

Data Source: ESRI World Imagery.

Note

1. Black and white reproduction of this color original may reduce its effectiveness and lead to incorrect interpretation.

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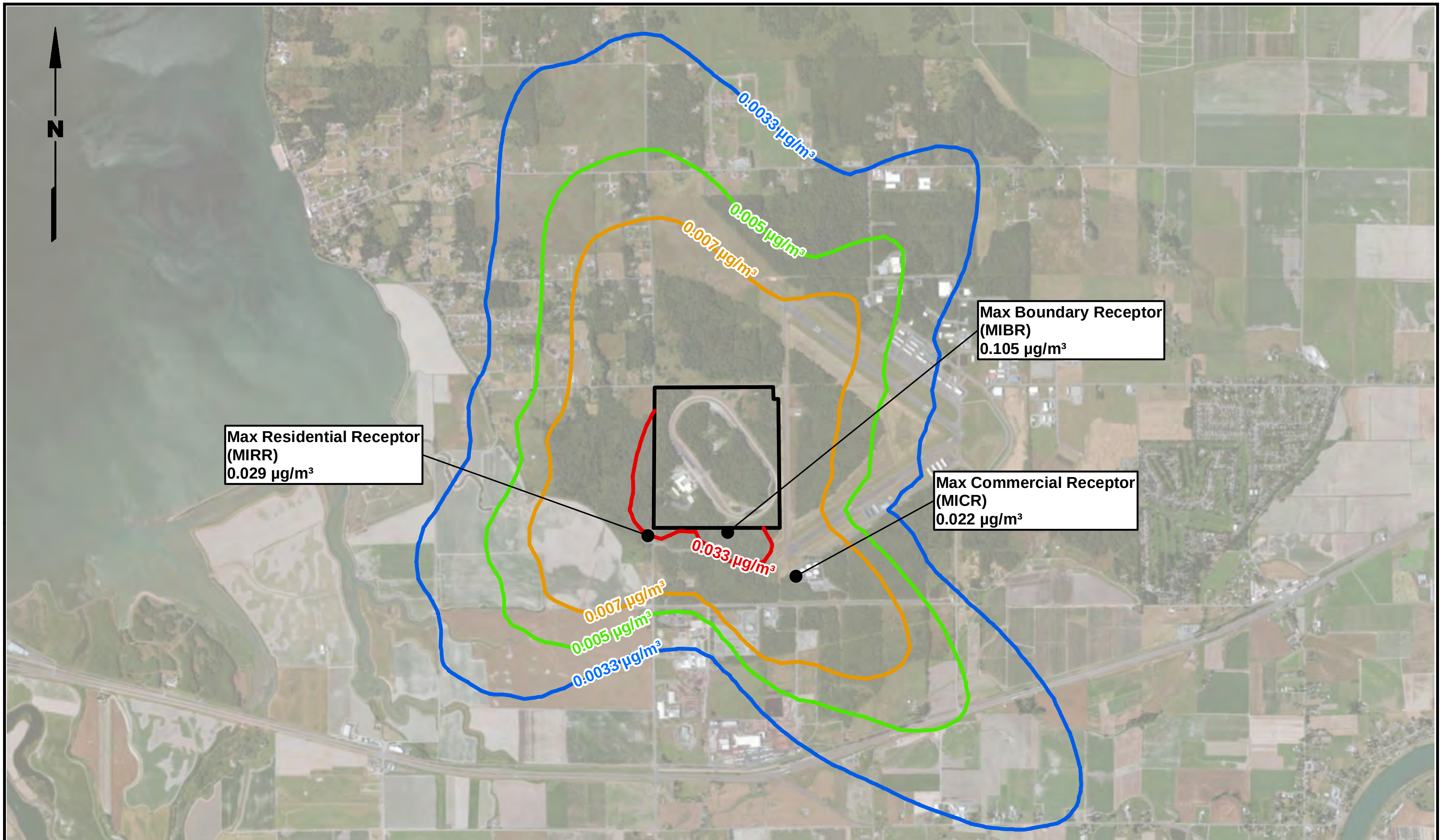
PACCAR
Technical Center
Skagit County, Washington

70-Year Average DEEP Concentrations Caused by Emissions from Four New Test Cells

Figure 4-1

Y:\Projects\1365001\010\013\Tier2HIA\Figure4-2DEEPExistingTestCells.mxd 6/11/2013 NAD 1983 UTM Zone 10N



Legend

DEEP Concentrations

- 0.00333 µg/m³
- 0.005 µg/m³
- 0.007 µg/m³
- 0.033 µg/m³

PACCAR Facility Boundary

Note

1. Black and white reproduction of this color original may reduce its effectiveness and lead to incorrect interpretation.

Data Source: ESRI World Imagery.

0 3,500 7,000

Scale in Feet

LANDAU ASSOCIATES

PACCAR
Technical Center
Skagit County, Washington

70-Year Average DEEP Concentrations Caused by Emissions from Existing and New Test Cells

Figure 4-2

TABLE 2-1
EMISSION RATES FOR FOUR NEW TEST CELLS
PACCAR TECHNICAL CENTER
SKAGIT COUNTY, WASHINGTON

Pollutants	CAS No.	Hourly Emissions (per cell) lb/hr	Hourly Emissions (total) lb/hr	Total Annual Emissions PTE tons/year
NO_x	--	0.64	3	11
SO_2	7446-09-05	7.5E-05	3.01E-04	1.32E-03
CO	630-08-0	1.5	6.1	27
TSP/ PM_{10}/ $PM_{2.5}$/ DEEP (a)	--	0.019	0.1	0.3
HC/VOCs	--	0.2	0.9	3.9
NO_2	10102-44-0	0.1	0.3	1.1
Benzene	71-43-2	2.4E-03	9.8E-03	4.3E-02
Toluene	108-88-3	8.9E-04	3.5E-03	1.6E-02
Xylenes	(b)	6.1E-04	2.4E-03	1.1E-02
Propylene	115-07-1	8.8E-03	3.5E-02	1.5E-01
Formaldehyde	50-00-0	2.5E-04	1.0E-03	4.4E-03
Acetaldehyde	75-07-0	8.0E-05	3.2E-04	1.4E-03
Acrolein	107-02-8	2.5E-05	9.9E-05	4.4E-04
Naphthalene	91-20-3	4.1E-04	1.6E-03	7.2E-03
1,3-Butadiene	106-99-0	1.2E-04	4.9E-04	2.2E-03
Benz(a)anthracene	56-55-3	2.0E-06	7.8E-06	3.4E-05
Chrysene	218-01-9	4.8E-06	1.9E-05	8.5E-05
Benzo(b)fluoranthene	205-99-2	3.5E-06	1.4E-05	6.1E-05
Benzo(k)fluoranthene	207-08-9	6.9E-07	2.8E-06	1.2E-05
Benzo(a)pyrene	50-32-8	8.1E-07	3.2E-06	1.4E-05
Indeno(1,2,3-cd)pyrene	193-39-5	1.3E-06	5.2E-06	2.3E-05
Dibenz(a,h)anthracene	53-70-3	1.1E-06	4.4E-06	1.9E-05

(a) For the purposes of this evaluation, the PM_{10}, $PM_{2.5}$, and DEEP emission factors were conservatively assumed to be equal to the emission factor for total suspended particulates (TSP).

(b) Xylenes is comprised of m-xylene (CAS No. 108-38-3), o-xylene (CAS No. 95-47-6), and p-xylene (106-42-3).

"—" No CAS identification number is available for pollutant.

TABLE 2-2
GENERAL LAND USE ZONES NEAR THE SITE
PACCAR TECHNICAL CENTER
SKAGIT COUNTY, WASHINGTON

Direction From PTC	Zoning (from Skagit County Zoning map)	Notable Development
North	Bayview Ridge Light Industrial; Rural Reserve; and Aviation Related	Receptor R-2, Single-family House
East	Aviation Related	Receptor C-2, Airport Building
West	Rural Reserve; Agricultural	Vacant, Undeveloped Land
South	Bayview Ridge Heavy Industrial; Agricultural; Residential; Aviation Related	Receptor R-1 (Single-family House); C-1 (Tank Farm), and C-3 (Agricultural Building)

TABLE 3-1
SUMMARY OF BACT DETERMINATION
PACCAR TECHNICAL CENTER
SKAGIT COUNTY, WASHINGTON

Pollutant(s)	BACT Determination
Particulate matter (PM)	Use of good combustion practices In-cylinder combustion controls (as testing allows) Diesel particulate filters (as testing allows)
Nitrogen oxides (NO_x)	Use of good combustion practices In-cylinder combustion controls (as testing allows) Selective catalytic reduction (as testing allows)
Carbon monoxide (CO)	Use of good combustion practices In-cylinder combustion controls (as testing allows) Diesel oxidation catalysts (as testing allows)
Sulfur dioxide (SO_2) and volatile organic compounds (VOCs)	Use of good combustion practices Use of ultra-low sulfur diesel fuel containing no more than 15 parts per million by weight of sulfur, except when precluded from use by the requirements of specific tests

TABLE 3-2
SUMMARY OF TBACT DETERMINATION FOR AIR TOXICS
PACCAR TECHNICAL CENTER
SKAGIT COUNTY, WASHINGTON

Toxic Air Pollutant(s)	tBACT Determination
DEEP	Compliance with the PM BACT requirement
CO	Compliance with the CO BACT requirement
Benzene, toluene, xylenes, propylene, formaldehyde, acetaldehyde, acrolein, naphthalene, and total PAHs	Compliance with the VOC BACT requirement
Nitrogen dioxide	Compliance with the NO_x BACT requirement
Sulfur dioxide	Compliance with the SO_2 BACT requirement

TABLE 3-3
SMALL-QUANTITY EMISSION RATES COMPARISON FOR TOXIC AIR POLLUTANTS
PACCAR TECHNICAL CENTER
SKAGIT COUNTY, WASHINGTON

Pollutant	SQER	Units	ETL III Test Cell Emissions	SQER Ratio
DEEP	0.64	lbs/yr	680	1,063
CO	50.2	lbs/1-hour	6.1	0.12
SO_2	1.45	lbs/1-hour	3.0E-04	2.1E-04
Primary NO_2	1.03	lbs/1-hour	0.26	0.25
Benzene	6.62	lbs/yr	86	13
Toluene	657	lbs/24-hr day	0.085	1.3E-04
Xylenes	58	lbs/24-hr day	0.058	1.0E-03
Propylene	394	lbs/24-hr day	0.85	0.0022
1,3-Butadiene	1.13	lbs/yr	4.3	3.8
Formaldehyde	32	lbs/yr	8.7	0.27
Acetaldehyde	71	lbs/yr	2.8	0.039
Acrolein	0.0079	lbs/24-hr day	0.0024	0.30
Naphthalene	5.64	lbs/yr	14	2.5
Benzo(a)pyrene	0.17	lbs/yr	0.028	0.16
Benzo(a)anthracene	1.74	lbs/yr	0.069	0.040
Chrysene	17.4	lbs/yr	0.17	0.0098
Benzo(b)fluoranthene	1.74	lbs/yr	0.12	0.069
Benzo(k)fluoranthene	1.74	lbs/yr	0.024	0.014
Dibenz(a,h)anthracene	0.16	lbs/yr	0.038	0.24
Indeno(1,2,3-cd)pyrene	1.74	lbs/yr	0.046	0.026

Note: Highlighted cells indicate SQER ratios exceeding 1.0.

TABLE 3-4
FIRST-TIER AMBIENT IMPACT ASSESSMENT FOR TOXIC AIR POLLUTANTS
PACCAR TECHNICAL CENTER
SKAGIT COUNTY, WASHINGTON

Toxic Air Pollutant	ASIL (μg/m^3)	Averaging Period	Highest Ambient Concentration (μg/m^3)
DEEP	0.00333	Annual average	0.04439
Benzene	0.0345	Annual average	0.0056
1,3-Butadiene	0.00588	Annual average	0.00028
Naphthalene	0.0294	Annual average	0.00095

TABLE 4-1
CALIFORNIA'S AIR TOXICS HOTSPOTS RISK ASSESSMENT GUIDANCE ON SPECIFIC PATHWAYS TO BE ANALYZED FOR EACH MULTI-PATHWAY SUBSTANCE SIL COMPLIANCE AT FACILITY BOUNDARY
PACCAR TECHNICAL CENTER
SKAGIT COUNTY, WASHINGTON

	Ingestion Pathway									
Substance	**Soil**	**Dermal**	**Meat, Milk & Egg**	**Fish**	**Exposed Veg.**	**Leafy Veg.**	**Protected Veg.**	**Root Veg.**	**Water**	**Breast Milk**
4,4'-Methylene dianiline	X	X		X	X	X	X	X	X	
Creosotes	X	X	X	X	X	X			X	
Diethylhexylphthalate	X	X		X	X	X	X	X	X	
Hexachlorocyclohexanes	X	X		X	X	X			X	
PAHs	X	X	X	X	X	X			X	
PCBs	X	X	X	X	X	X	X	X	X	X
Cadmium & compounds	X	X	X	X	X	X	X	X	X	
Chromium VI & compounds	X	X	X	X	X	X	X	X	X	
Inorganic arsenic & compounds	X	X	X	X	X	X	X	X	X	
Beryllium & compounds	X	X	X	X	X	X	X	X	X	
Lead & compounds	X	X	X	X	X	X	X	X	X	
Mercury & compounds	X	X		X	X	X	X	X	X	
Nickel	X	X	X		X	X	X	X	X	
Fluorides (including hydrogen fluoride)	To be determined									
Dioxins & furans	X	X	X	X	X	X	X		X	X

Veg. = Vegetable

TABLE 4-2
MAXIMALLY EXPOSED RECEPTORS
70-YEAR AVERAGE DEEP, ATTRIBUTABLE TO PROPOSED TEST CELLS
PACCAR TECHNICAL CENTER
SKAGIT COUNTY, WASHINGTON

Receptor Type	Direction From Nearest Project-Specific DEEP Emission Source	Estimated Distance From Nearest Project-Specific DEEP Emission Source		Estimated ETL III-Only Increase in 70-Year Average DEEP Concentration at Receptor Location (μg/m^3)
		Feet	Meters	
Point of Maximum Offsite Impact (a)	South (undeveloped, forested land)	1,056	322	0.044
Maximum Impacted Residence (yard near residential building, R-1)	Southwest	1,588	484	0.017
Maximum Offsite Impacted Business (tank farm, C-1)	Southeast	3,087	941	0.0099

(a) South fence line, approximately 413 meters west of the eastern property line.

TABLE 4-3
MAXIMALLY EXPOSED RECEPTORS
70-YEAR AVERAGE CUMULATIVE ANNUAL DEEP
PACCAR TECHNICAL CENTER
SKAGIT COUNTY, WASHINGTON

Attributable To	70-Year Average Annual DEEP Concentration (μg/m^3) at Various Receptor Locations – PTC Receptors					
	Fence Line Receptor (MIBR)	R-1 SW House (MIRR)	R-2 NW House	C-1 Tank Farm (MICR)	C-2 Airport Building	C-3 Agricultural Buildings
Proposed Four ETL III Test Cells	0.044	0.017	0.011	0.0099	0.0082	0.0058
Existing Eight ETL I and II Test Cells	0.062	0.012	0.012	0.0119	0.0078	0.0041
NATA Regional Background	0.124	0.124	0.124	0.124	0.124	0.124
Cumulative (Post-project)	0.230	0.153	0.147	0.146	0.140	0.134

TABLE 4-4
TOXICITY VALUES USED TO ASSESS AND QUANTIFY NON-CANCER HAZARD AND CANCER RISK
PACCAR TECHNICAL CENTER
SKAGIT COUNTY, WASHINGTON

Pollutant	Agency	Non-Cancer	Cancer
DEEP	U.S. Environmental Protection Agency	RfC = 5 $\mu g/m^3$	NA (a)
	California EPA–Office of Environmental Health Hazard Assessment	Chronic REL = 5 $\mu g/m^3$	URF = 0.0003 per $\mu g/m^3$

(a) The EPA considers DEEP and naphthalene to be probable human carcinogens, but has not established a cancer slope factor or unit risk factor.

TABLE 4-5
DEEP CHRONIC NON-CANCER HAZARD QUOTIENTS FOR RESIDENTIAL AND OCCUPATIONAL SCENARIOS
PACCAR TECHNICAL CENTER
SKAGIT COUNTY, WASHINGTON

	DEEP Chronic Hazard Quotient at Various Receptor Locations					
Attributable To:	**Fence Line Receptor (MIBR)**	**R-1 SW House (MIRR)**	**R-2 NW House**	**C-1 Tank Farm (MICR)**	**C-2 Airport Building**	**C-3 Agricultural Buildings**
Proposed Four ETL III Test Cells	0.0088	0.0034	0.0022	0.0020	0.0016	0.0012
Existing Eight ETL I and II Test Cells	0.012	0.0024	0.0024	0.0024	0.0016	0.00082
NATA Regional Background	0.025	0.025	0.025	0.025	0.025	0.025
Cumulative (Post-project)	0.046	0.031	0.029	0.029	0.028	0.027

TABLE 4-6
NON-CANCER HAZARDS OF ETL III TEST CELL EMISSIONS AT THE MAXIMALLY EXPOSED LOCATION AT OR BEYOND THE FACILITY BOUNDARY (MAXIMALLY IMPACTED BOUNDARY RECEPTOR) PACCAR TECHNICAL CENTER SKAGIT COUNTY, WASHINGTON

DEEP			
DEEP Concentration ($\mu g/m^3$)		0.044 (Max annual TWA)	
RBC ($\mu g/m^3$)		RfC = 5	REL = 5
HQ		0.0088	0.0088
Naphthalene			
Naphthalene Concentration ($\mu g/m^3$)		0.00096 (Max annual TWA)	
RBC ($\mu g/m^3$)		RfC = 3	REL = 9
HQ		0.00032	0.00011
Benzene			
Benzene Concentration ($\mu g/m^3$)	0.25 (Max 1-hour TWA)	0.0056 (Max annual TWA)	
RBC ($\mu g/m^3$)	REL = 1,300	RfC = 30	REL = 60
HQ	0.00019	0.00019	0.000093
1,3-Butadiene			
1,3-Butadiene Concentration ($\mu g/m^3$)		0.00028 (Max annual TWA)	
RBC ($\mu g/m^3$)		RfC = 2	REL = 20
HQ		0.00014	0.000014
Combined Pollutants			
Combined Pollutant Hazard Index	Max 1-hr Acute Hazard	Max Chronic Hazard	
	0.00019	0.0095	

TABLE 4-7
NON-CANCER HAZARDS OF ETL III TEST CELL EMISSIONS AT THE TANK FARM (MAXIMALLY IMPACTED COMMERCIAL RECEPTOR)
PACCAR TECHNICAL CENTER
SKAGIT COUNTY, WASHINGTON

DEEP			
DEEP Concentration (µg/m^3)		0.0099 (Max annual TWA)	
RBC (µg/m^3)		RfC = 5	REL = 5
HQ		0.0020	0.0020
Naphthalene			
Naphthalene Concentration (µg/m^3)		0.00021 (Max annual TWA)	
RBC (µg/m^3)		RfC = 3	REL = 9
HQ		0.000070	0.000023
Benzene			
Benzene Concentration (µg/m^3)	0.097 (Max 1-hour TWA)	0.0013 (Max annual TWA)	
RBC (µg/m^3)	REL = 1,300	RfC = 30	REL = 60
HQ	0.000075	0.000043	0.000022
1,3-Butadiene			
1,3-Butadiene Concentration (µg/m^3)		0.000060 (Max annual TWA)	
RBC (µg/m^3)		RfC = 2	REL = 20
HQ		0.000030	0.0000030
Combined Pollutants			
Combined Pollutant Hazard Index	Max 1-hr Acute Hazard	Max Chronic Hazard	
	0.000075	0.0021	

TABLE 4-8
NON-CANCER HAZARDS OF ETL III TEST CELL EMISSIONS AT THE MAXIMALLY IMPACTED RESIDENTIAL RECEPTOR
PACCAR TECHNICAL CENTER
SKAGIT COUNTY, WASHINGTON

DEEP			
DEEP Concentration (µg/m^3)		0.017 (Max annual TWA)	
RBC (µg/m^3)		RfC = 5	REL = 5
HQ		0.0034	0.0034
Naphthalene			
Naphthalene Concentration (µg/m^3)		0.00038 (Max annual TWA)	
RBC (µg/m^3)		RfC = 3	REL = 9
HQ		0.00013	0.000042
Benzene			
Benzene Concentration (µg/m^3)	0.17 (Max 1-hour TWA)	0.0022 (Max annual TWA)	
RBC (µg/m^3)	REL = 1,300	RfC = 30	REL = 60
HQ	0.00013	0.000073	0.000037
1,3-Butadiene			
1,3-Butadiene Concentration (µg/m^3)		0.00011 (Max annual TWA)	
RBC (µg/m^3)		RfC = 2	REL = 20
HQ		0.000055	0.0000055
Combined Pollutants			
Combined Pollutant Hazard Index	Max 1-hr Acute Hazard	Max Chronic Hazard	
	0.00013	0.0037	

TABLE 4-9
EXPOSURE ASSUMPTIONS AND UNIT RISK FACTORS FOR DIESEL ENGINE EXHAUST PARTICULATE RISK ASSESSMENT
PACCAR TECHNICAL CENTER
SKAGIT COUNTY, WASHINGTON

Receptor Type	Annual Exposure	Exposure Duration	Diesel Particulate Matter Cancer Unit Risk Factor (risk per million, per annual µg/m^3 DEEP)
Unoccupied Land	2 hours/day 250 days/year	30 years	7.3-per-million cancer risk per µg/m^3 DEEP
Residences	24 hours/day 365 days/year	70 years	300-per-million cancer risk per µg/m^3 DEEP
Schools (College Students)	36 hours/week 40 weeks/year	4 years	2.8-per million risk per µg/m^3 DEEP
Schools (High School Students)	36 hours/week 40 weeks/year	4 years	2.8-per-million risk per µg/m^3 DEEP
Schools (Elementary School Students)	36 hours/week 40 weeks/year	7 years	4.9-per-million risk per µg/m^3 DEEP
Schools (All Teachers)	40 hours/week 40 weeks/year	40 years	31-per-million risk per µg/m^3 DEEP
Churches	2 hours/week 52 weeks/year	40 years	2-per-million risk per µg/m^3 DEEP
Business	8 hours/day 250 days/year	40 years	38-per-million risk per µg/m^3 DEEP

TABLE 4-10
ESTIMATED CUMULATIVE INCREASED CANCER RISK
PACCAR TECHNICAL CENTER
SKAGIT COUNTY, WASHINGTON

	70-Year Average Risk Per Million From DEEP Exposure at Various Receptor Locations					
Attributable To:	**Fence Line Receptor (MIBR)**	**R-1 SW House (MIRR)**	**R-2 NW House**	**C-1 Tank Farm (MICR)**	**C-2 Airport Building**	**C-3 Agricultural Buildings**
Proposed Four ETL III Test Cells	0.32	5.1	3.3	0.38	0.31	0.22
Existing Eight ETL I and II Test Cells	0.45	3.6	3.6	0.45	0.30	0.16
NATA Regional Background	0.91	37	37	4.7	4.7	4.7
Cumulative (Post-project)	1.7	46	44	5.5	5.3	5.1

TABLE 4-11
CANCER RISK CAUSED BY ALL EMITTED CARCINOGENS
PACCAR TECHNICAL CENTER
SKAGIT COUNTY, WASHINGTON

Carcinogen	70-Year Average Emission Rate (Tons per Year)	ASIL (μg/m^3)	Cancer Risk at Key Receptors (per Million)		
			MIRR	MIBR	MICR
DEEP	0.339	0.00333	5.1	0.32	0.38
Naphthalene	7.19E-03	2.94E-02	1.2E-02	7.7E-04	9.1E-04
Benzene	4.29E-02	3.50E-02	6.1E-02	3.9E-03	4.6E-03
1,3-Butadiene	2.16E-03	5.88E-03	1.8E-02	1.2E-03	1.4E-03
Formaldehyde	4.36E-03	1.67E-01	1.3E-03	8.2E-05	9.7E-05
Acetaldehyde	1.39E-03	3.70E-01	1.9E-04	1.2E-05	1.4E-05
Benzo(a)pyrene	1.42E-05	9.09E-04	7.8E-04	4.9E-05	5.8E-05
Benzo(a)anthracene	3.44E-05	9.09E-03	1.9E-04	1.2E-05	1.4E-05
Chrysene	8.46E-05	9.09E-02	4.7E-05	2.9E-06	3.5E-06
Benzo(b)fluoranthene	6.14E-05	9.09E-03	3.4E-04	2.1E-05	2.5E-05
Benzo(k)fluoranthene	1.21E-05	9.09E-03	6.6E-05	4.2E-06	4.9E-06
Dibenz(a,h)anthracene	1.91E-05	9.09E-04	1.1E-03	6.6E-05	7.9E-05
Ideno(1,2,3-cd)pyrene	2.29E-05	9.09E-03	1.3E-04	7.9E-06	9.4E-06
Total Risk Per Million	--	--	5.2	0.33	0.39

TABLE 5-1
QUALITATIVE SUMMARY OF THE EFFECTS OF UNCERTAINTY ON QUANTITATIVE ESTIMATES OF RISKS OR HAZARDS
PACCAR TECHNICAL CENTER
SKAGIT COUNTY, WASHINGTON

Source of Uncertainty	How Does it Affect Estimated Risk From This Project?
Exposure assumptions	Likely overestimate of exposure
Emissions estimates	Possible overestimate of emissions concentrations
AERMOD air modeling methods	Possible underestimate of average long-term ambient concentrations and overestimate of short-term ambient concentration
Toxicity of DEEP at low concentrations	Possible overestimate of cancer risk, possible underestimate of non-cancer hazard for sensitive individuals