Revised Second-Tier Health Impact Assessment Report H5 Data Center Campus Expansion Quincy, Washington

July 15, 2021

Prepared for

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Revised Second-Tier Health Impact Assessment Report H5 Data Center Campus Expansion Quincy, Washington

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 Date:
 July 15, 2021

 Project No.:
 1904001.010

 File path:
 P:\1904\001\R\HIA Report\Updated Runtime 04-21-2021

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LIST OF ABBREVIATIONS AND ACRONYMS

μg/m ³	microgram per cubic meter
μm	micrometer
AEGL 1	Acute Exposure Guideline Level 1
AERMOD	American Meteorological Society/EPA Regulatory Model
ASIL	acceptable source impact level
BACT	best available control technology
cDPF	catalyzed diesel particulate filter
со	carbon monoxide
DEEP	diesel engine exhaust particulate matter
Ecology	Washington State Department of Ecology
ЕРА	US Environmental Protection Agency
Facility	
g/kWm-hr	grams per mechanical kilowatt-hour
Н5	H5 Data Centers
н	hazard index
HIA	health impact assessment
HQ	hazard quotient
LAI	Landau Associates, Inc.
m	meter
MIBR	maximally impacted boundary receptor
MICR	maximally impacted commercial receptor
MIIR	maximally impacted institutional receptor
MIRR	maximally impacted residential receptor
MTU	MTU Onsite Energy
MWe	megawatts electrical
NAAQS	National Ambient Air Quality Standards
NO	nitric oxide
NO ₂	nitrogen dioxide
NOC	Notice of Construction
NO _x	nitrogen oxides
OEHHACal	ifornia Office of Environmental Health Hazard Assessment
РАН	polycyclic aromatic hydrocarbon
PM	particulate matter
PM _{2.5} particulate matter with a	in aerodynamic diameter less than or equal to 2.5 microns
ppm	parts per million
PUD	public utility district

LIST OF ABBREVIATIONS AND ACRONYMS (Continued)

REL	reference exposure level
RfC	reference concentration
SCR	selective catalytic reduction
SQER	small-quantity emission rate
ТАР	toxic air pollutant
tBACT	best available control technology for toxic air pollutants
URF	unit risk factor
VOC	volatile organic compound
WAC	Washington Administrative Code

1.0 EXECUTIVE SUMMARY

1.1 Project Description

H5 Data Centers (H5) is proposing to install additional diesel-fired emergency generator sets, or emergency generators, at the H5 Data Center (Facility) in Quincy, Washington. The H5 Data Center is located at 1711 M Street NE in Quincy, Washington.

Under air quality regulations promulgated by the Washington State Department of Ecology (Ecology), Landau Associates, Inc. (LAI), on behalf of H5, has submitted a Notice of Construction (NOC) application for installation and operation of 12 new emergency generators. The NOC application and supporting documentation were submitted to the Ecology Eastern Regional Office on April 13, 2021 (LAI 2021).

As documented in the NOC application, potential emissions of diesel engine exhaust particulate matter (DEEP) and nitrogen dioxide (NO₂) from the 12 emergency diesel engine generators may cause ambient air impacts that exceed the Washington State acceptable source impact levels (ASILs). Pursuant to Chapter 173-460 (updated December 30, 2019) of the Washington Administrative Code (WAC), this Health Impact Assessment (HIA) evaluates adverse health effects as a result of the H5 Data Center emergency generator expansion.

1.2 Health Impacts Evaluation

This HIA follows the human health risk assessment approach proposed by the National Academy of Sciences (NRC 1983, 1994) and the requirements of WAC 173-460-090.

The ambient cancer risks caused by emissions of DEEP are less than Ecology's approval limits. Under worst-case exposure assumptions involving residents standing outside a home, for 70 continuous years, DEEP from the emergency generators could cause an increased cancer risk of up to 9.6 in 1 million (9.6 x 10^{-6}) at the maximally impacted residence (MIRR, R-4). Because the increase in cancer risk attributable to the project alone would be less than the maximum risk allowed by a second-tier review, which is 10 in 1 million (10×10^{-6}), the project is approvable under WAC 173-460-090. NO₂ is not classified as a carcinogen; therefore, there is no cancer toxicity value associated with NO₂.

Based on the cumulative maximum DEEP concentration at the maximally impacted residential receptor (MIRR, R-4) location near the Facility, the estimated maximum potential cumulative cancer risk posed by DEEP emitted from the proposed project and background sources within the area would be approximately 44 in 1 million (44×10^{-6}) at the MIRR location.

The non-cancer risk assessment concluded that all receptors exposed to ambient DEEP concentrations would encounter acceptable levels of non-cancer risk as quantified by hazard quotients (HQs) less than 1. Potential NO₂ concentrations (project related + background) correspond to HQs of more than 1 at the MICR location (HQ of 2.1). However, based on the very good electrical grid reliability in Grant

County, the recurrence interval for human exposure to cumulative NO₂ concentrations (project + background) above the acute reference exposure level (REL) ranges between 9 and 10 years at the receptor locations maximally impacted by the project. Additionally, because maximum modeled project-related NO₂ concentrations are below the level at which nearly all individuals could be exposed for up to 1 hour without experiencing more than mild, transient adverse health effects, it is anticipated that no significant adverse health impacts would occur as a result of NO₂ emissions from diesel generators.

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 **PROJECT DESCRIPTION**

2.1 Existing Facility Description

The H5 Data Center, located at 1711 M Street NE in Quincy, Washington (Figure 2-1), has one existing building and six existing emergency generators. The six existing emergency generators are MTU Onsite Energy (MTU) 2.25-megawatt electrical (MWe) diesel-fired emergency generator sets, powered by an MTU Model 16V4000DS2250 engine. These emergency generators provide emergency backup power to additional server equipment at the H5 Data Center. These existing emergency generators were previously permitted by Ecology under Approval Order No. 18AQ-E044.

2.2 **Proposed Expansion Description**

H5 is proposing to install 12 additional emergency generators at the H5 Data Center, increasing the total number of emergency generators to 18. These additional generators will provide emergency backup power to additional server equipment to be located in the existing H5 Data Center. The 12 proposed generators will be powered by either MTU Model 16V4000G84 engines or Kohler model KD 2250 engines. A site map for the proposed development is provided on Figure 2-2.

The equipment to be evaluated in this HIA consists of the following:

- Twelve (12) Tier-2 certified diesel-fired emergency generator sets. The 2.25-MWe electrical generators will have a combined capacity of 27 MWe.
- Eight (8) evaporative cooling towers.

2.3 Facility Emissions

Air pollutant emission rates from the proposed 12 emergency generators were calculated for criteria pollutants and toxic air pollutants (TAPs) in accordance with WAC 173-460-050. This HIA was conducted using the emission estimates and generator runtime assumptions that are described in the NOC application and supporting documentation (LAI 2021).

2.4 Land Use and Zoning

For exposure assessment purposes, land-use and zoning designations are used to classify air modeling receptors. Receptors in residentially-zoned areas are classified as residential receptors. Receptors in areas zoned as commercial, industrial, or agricultural, are classified as commercial receptors. Receptors that surround a sensitive population institution are classified as institutional receptors or sensitive receptors. The topography south and east of the Facility is relatively flat with elevations of approximately 1,230 feet above sea level. North and west of the Facility is an elevated ridge with a maximum elevation of approximately 2,697 feet above sea level.

• **Zoning** in the vicinity of the Facility is used to predict the expected activity conducted by persons at a location and, therefore, the frequency and duration of exposure at a particular location. Zoning codes have been simplified into four groups: residential, commercial,

industrial, and agricultural. A compilation of categorized zoning designations is shown on Figure 2-3. In both the figure, and throughout this HIA, the more conservative zoning designation for a mixed-use parcel is considered. For instance, in a parcel designated as residential and commercial mixed use, this HIA will consider the parcel to be residentially zoned.

• Land use is used to geographically identify sensitive receptors and unoccupied land and to reclassify receptors if the current land use does not match the zoning. Parcels of land with schools or hospitals were considered representative of sensitive populations. Receptors over bodies of water were classified as "unoccupied." In some cases, a parcel of land containing dwellings may be zoned as agricultural or commercial, but a dwelling unit is on the land. Areal maps were compared to zoning maps to reclassify receptors as the most conservative receptor type. In the instance where a location's zoning is more conservative than the land use (i.e., the parcel is zoned as residential but is used for agriculture), the more conservative zoning type is retained.

Land-use and zoning information were acquired from Grant County (Grant County 2018) and the City of Quincy (accessed March 10, 2021). The area immediately surrounding the Facility to the east, west, and south is designated as industrial/commercial and the area immediately surrounding the Facility to the north is designated as agricultural. To the north the zoning is typically agricultural and to the west the land use and zoning is predominantly residential. The city of Quincy comprises the residential areas west of the Facility. See Table 2-1 for a summary of land uses in the vicinity of the Facility.

2.5 Sensitive Receptor Locations

The following sensitive receptor locations are in the vicinity of the Facility:

- Quincy Innovation Academy, located at 404 1st Avenue SW, Quincy, Washington (approximately 2.0 miles southwest of the Facility).
- Quincy Junior High/Middle School, located at 16 Sixth Avenue SE, Quincy, Washington (approximately 1.4 miles southwest of the Facility).
- Monument Elementary School, located at 1400 13th Avenue SW, Quincy, Washington (approximately 3.1 miles southwest of the Facility).
- Ancient Lakes Elementary School, located at 417 C Street SE, Quincy, Washington (approximately 1.6 miles southwest of the Facility).
- Mountain View Elementary School, located at 199 D Street NW, Quincy, Washington (approximately 1.8 miles southwest of the Facility).
- Quincy High School, located at 403 Jackrabbit Street NE, Quincy, Washington (approximately 1.4 miles southwest of the Facility).
- Quincy High School (new), located north of Rd 11 NW and east of Rd Q NW, Quincy, Washington (approximately 1.3 miles west of the Facility).
- Pioneer Elementary School, located at 224 J Street SE, Quincy, Washington (approximately 2.0 miles southwest of the Facility).

• Quincy Valley Medical Center, located at 908 10th Avenue SW, Quincy, Washington (approximately 2.7 miles southwest of the Facility).

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3.0 OVERVIEW OF THE REGULATORY PROCESS

Pursuant to WAC 173-460-080, any source emitting TAPs in excess of *de minimis* levels is subject to tiered review: 1) first-tier (toxics screening); 2) second-tier (HIA); and 3) third-tier (risk management decision).

A first-tier review, or toxics screening analysis, is to establish the systematic control of new sources emitting TAPs to prevent air pollution, reduce emissions to the extent reasonably possible, and maintain such levels of air quality to protect human health and safety. A first-tier review includes an emissions assessment, comparing emission rates to small quantity emission rates (SQERs) and analysis of best available control technology (BACT) for toxics (tBACT). If modeled project impacts exceed acceptable source impact levels (ASILs), a second-tier review, consisting of a site-specific HIA is required.

A second-tier review, or HIA, is intended 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 operations of the facility. 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 project operations is compared to its effective threshold concentration. 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.

In evaluating a second-tier petition, background concentrations of the applicable TAPs must be considered. Ecology sets no numerical limit on cumulative impacts from a facility, local background, and regional background levels.

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4.0 **FIRST-TIER REVIEW OVERVIEW**

First-tier review supporting information is provided in the NOC application and supporting documents. This section provides a brief overview and conclusions of the first-tier review. For a detailed description of the methods used to calculate project emission rates and to conduct BACT and tBACT analyses, see the NOC Supporting Information Report (LAI 2021).

4.1 Best Available Control Technology

H5 conducted BACT and tBACT analyses as presented in the NOC Supporting Information Report for presentation to Ecology, the agency ultimately responsible for determining BACT and tBACT for the Facility. For a detailed description of the methods used to conduct the BACT and tBACT analyses, see the NOC Supporting Information Report (LAI 2021).

The BACT/tBACT analyses concluded that all of the add-on control technology options (the selective catalytic reduction [SCR]/catalyzed diesel particulate filter [cDPF] Tier 4 Integrated Control Package, urea-SCR, active and passive cDPF, and diesel oxidation catalyst-alone) are technically feasible, but each of them failed the BACT cost-effectiveness evaluation. Therefore, the emission controls inherent to US Environmental Protection Agency (EPA) Tier 2-certified diesel engines should be required as BACT. The proposed BACT for particulate matter (PM), nitrogen oxides (NO_x), carbon monoxide (CO), and volatile organic compounds (VOCs) is based on compliance with the EPA's Tier 2 emission standards for non-road diesel engines: 0.20 grams per mechanical kilowatt-hour (g/kWm-hr) for PM, 3.5 g/kWm-hr for CO, and 6.4 g/kWm-hr for combined NO_x plus VOCs. The proposed BACT and tBACT determinations are summarized in Tables 4-1 and 4-2, respectively.

Additional restrictions proposed in the NOC application include:

- Limits on the total number of hours that the emergency diesel engines operate
- Use of ultra-low sulfur diesel fuel (15 parts per million [ppm] sulfur content)
- Compliance with the operation and maintenance restrictions of 40 Code of Federal Regulations Part 60, Subpart IIII.

4.2 Emissions Assessment

Identified pollutants with the potential for emissions from emergency generators are listed in Table 4-3. The table also shows calculated emission rates for each identified TAP and compares them to *de minimis* and SQER thresholds. As indicated in the table below, pollutants with emission rates below *de minimis* require no further analysis; pollutants with emission rates above *de minimis*, but below the SQER are reported; and pollutants with emission rates above the SQER are required to be modeled to assess ambient air impacts.

Pollutants Below <i>de minimis</i>	Pollutants Below the SQER, but Above <i>de minimis</i>	Pollutants Above the SQER
Sulfur dioxide (SO ₂)	Formaldehyde	NO ₂
Toluene	Dibenz(a,h)anthracene	DEEP
Acetaldehyde	Acrolein	со
Benz(a)anthracene	1,3-Butadiene	
Chrysene	Benzene	
Benzo(b)fluoranthene	Naphthalene	
Benzo(k)fluoranthene		
Indeno(1,2,3-cd)pyrene		
Benzo(a)pyrene		
Propylene		
Xylenes		

4.3 Air Dispersion Modeling

Ecology requires facilities to conduct a first-tier screening analysis for each TAP with an emission rate that exceeds its SQER by modeling the 1st-highest 1-hour, 1st-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 or where public receptors could be exposed, then comparing the modeled values to the ASILs (WAC 173-460-080).

Table 4-4 presents the first-tier ambient air concentration screening analysis for each TAP with an emission rate that exceeds its SQER and compares modeled ambient air concentrations to ASILs. A description of the methodology used for modeling the TAPs is provided in the NOC Supporting Information Report (LAI 2021). Pollutants with concentrations below the ASIL require no further analysis and pollutants with concentrations above the ASIL require second-tier review.

Modeled concentrations for CO are below their respective ASILs. The maximum annual average DEEP impact from the project at an offsite receptor location exceeds its ASIL. Additionally, the maximum 1-hour average NO₂ impact from the project at the maximally impacted receptor location exceeds its ASIL. Therefore, DEEP and NO₂ are the only TAPs that trigger a requirement for a second-tier HIA, as indicated in the table below.

Pollutants with Modeled Concentrations Below the ASIL	Pollutants with Modeled Concentrations Above the ASIL
СО	DEEP
	NO ₂

5.0 SECOND-TIER REVIEW OVERVIEW

5.1 **Processing Requirements**

In order for Ecology to review the second-tier petition, each of the following regulatory requirements under WAC 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 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 its ASIL 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 indicated approval of H5's HIA protocol (item [c] above) (Palcisko 2020).

5.2 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 LAI.

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6.0 HEALTH IMPACT ASSESSMENT

This HIA was conducted in accordance with the requirements of WAC 173-460-090 and guidance provided by Ecology. This HIA addresses the public health risk associated with exposure to DEEP and NO₂ from the proposed emergency diesel engine generators and existing sources of DEEP and NO₂ in the vicinity of the project. 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 (NRC 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, this HIA did not consider exposure pathways other than inhalation.

6.1 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 the 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., whether chemicals found to be carcinogenic or teratogenic in experimental animals also would likely be so in adequately exposed humans).

Although the second-tier HIA is triggered solely by potential ambient air impacts of DEEP and NO₂, 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.

6.1.1 Overview of DEEP Toxicity

Diesel engines emit very small, fine (smaller than 2.5 micrometers $[\mu m]$) and ultrafine (smaller than 0.1 μ m) 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 health effects listed below have been associated with exposure to very high concentrations of diesel particles, primarily in industrial workplace settings (e.g., underground mines that use diesel equipment) with concentrations much higher than the ambient levels that will be caused by the project:

- 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 proposed project 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 the Facility'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.

6.1.2 Overview of NO₂ Toxicity

NO₂ is a red-brown gas that is present in diesel exhaust. It forms when nitrogen, present in diesel fuel and a major component of air, combines with oxygen to produce oxides of nitrogen (NO_x). NO₂ and other oxides of nitrogen are of concern for ambient air quality because they are part of a complex chain of reactions responsible for the formation of ground-level ozone. Additionally, exposure to NO₂ can cause both long-term (chronic) and short-term (acute) health effects. Long-term exposure to NO₂ can lead to chronic respiratory illness such as bronchitis and increase the frequency of respiratory illness due to respiratory infections.

Short-term exposure to extremely high concentrations (> 180,000 micrograms per cubic meter $[\mu g/m^3]$) of NO₂ may result in serious effects including death (NAC AEGL Committee 2008). Moderate levels (~30,000 $\mu g/m^3$) may severely irritate the eyes, nose, throat, and respiratory tract, and cause shortness of breath and extreme discomfort. Lower-level NO₂ exposure (< 1,000 $\mu g/m^3$), such as that experienced near major roadways, or perhaps downwind from stationary sources of NO₂, may cause sporadic increased bronchial reactivity in some asthmatics, decreased lung function in patients with chronic obstructive pulmonary disease, and increased risk of respiratory infections, especially in young children (CalEPA 2008). The EPA's Acute Exposure Guideline Level 1 (AEGL 1) for NO₂ is 0.5 ppm (940 $\mu g/m^3$). The AEGL 1 is defined as the level at which notable discomfort, irritation, or certain asymptomatic non-sensory effects may occur, but the effects are not disabling and are transient and

reversible upon cessation of exposure. For this project, the maximum short-term ambient NO_2 concentration has been estimated to be 919 μ g/m³ (1-hour average).

Power outage emissions present the greatest potential for producing high enough short-term concentrations of NO₂ to be of concern for susceptible individuals, such as people with asthma.

6.1.3 **Overview of Toxicity for Other Toxic Air Pollutants**

The TAP with an emission rate exceeding the SQER is carbon monoxide. The REL for CO considers toxic effects for the cardiovascular system (CalEPA 2016; accessed February 19, 2021), not the respiratory system; however, the ambient air impacts associated with CO emissions have been included in the project-specific hazard index (HI) calculated in this HIA to conservatively overestimate health risks.

6.2 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 receptor locations
- Estimating the duration and frequency of receptors' exposure.

6.2.1 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 2015) 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 provided in Table 6-1.

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 can be slowly 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 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 Facility. However, given the very low levels of PAHs and other multi-exposure route-type TAPs

that will be emitted from the proposed project, 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.

NO₂ is formed by nitrogen and oxygen combining at high temperatures during the combustion process. Though both nitric oxide (NO) and NO₂ are produced during the combustion process, NO is oxidized quickly in ambient air, by oxygen, ozone, and VOCs, to form NO₂. NO₂ is then broken down through reactions with sunlight and other substances in the atmosphere (ATSDR 2002).

In both outdoor and indoor conditions, NO₂ exists in gaseous form; therefore, inhalation is the primary route of exposure. High concentrations of NO₂ can cause eye irritation; however, such high concentrations are associated with industrial settings, not ambient air (Jarvis et al. 2010).

In the case of project emissions, only inhalation exposure to DEEP and NO₂ is evaluated.

6.2.2 Estimating DEEP and NO₂ Concentrations

To estimate where pollutants will disperse after they are emitted from the project generators, LAI conducted air dispersion modeling, which incorporates emissions, meteorological, geographical, and terrain information to estimate pollutant concentrations downwind from a source.

DEEP and NO₂ ambient air impacts from the proposed project were modeled using the following air dispersion model inputs:

- The EPA's plume rise model enhancement algorithm for building downwash.
- Five years of sequential hourly meteorological data from Grant County International Airport at Moses Lake (2012 to 2016).
- Twice-daily upper air data from Spokane, Washington (2012 to 2016) to define mixing heights.
- Digital topographical data for the analysis region were obtained from the Web GIS website (www.webgis.com) and processed for use in AERMOD.¹
- The emissions for each proposed diesel engine were modeled with stack heights of 43 feet above grade.
- The dimensions of the building at the Facility were included to account for building downwash.
- Each of the proposed Facility emergency generators was modeled as an individual discharge point.
- NO₂ modeling was conducted using the Plume Volume Molar Ratio Method, with EPA approval.

¹ American Meteorological Society/EPA Regulatory Model

- The receptor grid for the AERMOD modeling domain at or beyond the Facility boundary was established using a tiered Cartesian grid:
 - 12.5-meter (m) spacing from the property boundary to 150 m from the nearest emission source
 - 25-m spacing from 150 m to 400 m
 - 50-m spacing from 400 m to 900 m
 - 100-m spacing from 900 m to 2,000 m
 - 300-m spacing between 2,000 m and 4,500 m
 - 600-m spacing beyond 4,500 m (to 10,000 m maximum extent).

6.2.3 Background Sources of Pollutants of Concern

WAC 173-460-090 states, "Background concentrations of TAPs will be considered as part of a secondtier review." The word "background" is used to describe exposures to chemicals that come from existing sources other than those being assessed. Both regional (county-scale) and local (city-scale) background concentrations were incorporated into the modeling results.

Following guidance from Ecology, the Quincy, Washington online Storymap was used to determine background concentrations of DEEP and NO₂ at each receptor location (Palcisko 2020).

6.2.4 Exposure Frequency and Duration

The likelihood that someone would be exposed to DEEP and NO₂ from the Facility depends on local wind patterns, the frequency of engine testing and power outages, and how much time people spend in the immediate area. As discussed previously, the air dispersion model uses emission and meteorological information (and other assumptions) to determine ambient DEEP and NO₂ concentrations in the vicinity of the Facility.

This analysis considers the land use surrounding the proposed project site 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 at industrial or commercial properties in the area are likely to be exposed to project-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 at residential receptor locations 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. Assumptions for estimated exposure duration for each receptor type are detailed in Table 6-2.

6.2.5 Identifying Potentially Exposed Receptor Locations

Residential, commercial, and institutional receptors are assessed for exposure to Facility emissions. Typically, Ecology considers exposures at maximally exposed residential, commercial, and institutional receptor locations to capture worst-case exposure scenarios. These receptors are collectively called the key risk receptors. The location of each receptor may be different for different modeled pollutants. Individual names for key risk receptors are as follows:

- Maximally Impacted Residential Receptor (MIRR) Location: This is the location with the highest modeled DEEP or NO₂ impact from the Facility with a zoning code designation or land-use observation (through aerial imagery) of residential.
- Maximally Impacted Commercial Receptor (MICR) Location: This is the location with the highest modeled DEEP or NO₂ impact from the Facility with a zoning code designation of commercial, industrial, or agricultural. Aerial imagery was reviewed to identify and reclassify residences that are located within this zoning designation.
- Maximally Impacted Institutional Receptor (MIIR) Location: This is the sensitive receptor location (typically a school) with the highest modeled DEEP or NO₂ impact from the Facility. Zoning designations do not affect a receptor's status as a sensitive receptor.
- Maximally Impacted Boundary Receptor (MIBR) Location: This is the location with the highest modeled DEEP or NO₂ impact from the Facility, regardless of zoning code or land-use designation. The MIBR may coincide with another key risk receptor.

6.2.5.1 Receptors Maximally Exposed to DEEP

Maximally exposed receptor locations of different use types, the direction and distance of those receptor locations from the Facility, and the predicted project-related DEEP impacts at those receptor locations are summarized in Table 6-3.

Figure 6-1 shows a color-coded map of estimated annual-average DEEP concentrations attributable solely to DEEP emissions from the project. The figure also depicts project-related impacts at each of the maximally exposed receptor locations, the MIBR/MICR (C-1), the MIRR (R-4), and the MIIR (I-2). Impacts at the nearest hospital (I-8) are also shown on the figure. The blue contour line $(0.0033 \ \mu g/m^3)$ represents the ASIL. Receptors at all locations outside the blue contour line are forecast to be exposed to concentrations less than the ASIL.

6.2.5.2 Receptors Maximally Exposed to NO₂

Maximally exposed receptor locations of different use types, the direction and distance of those receptors from the Facility, and the predicted project-related NO₂ impacts at those receptor locations are summarized in Table 6-4.

Figure 6-2 shows a color-coded map of estimated 1-hour average NO₂ concentrations attributable solely to emissions from the project, including project-related impacts at each of the maximally exposed receptor locations representing different land uses. The concentrations at the MIBR/MICR

(C-2), the MIRR (R-2), the MIIR (I-4), the nearest hospital (I-8), and the location with maximum ASIL exceedance counts (C-2) are shown on the figures. The blue contour line (470 μ g/m³) represents the ASIL. Receptors at all locations outside the blue contour line are forecast to be exposed to concentrations less than the ASIL.

6.2.6 Short-Term Exposure to DEEP

As discussed previously, exposure to DEEP can cause both acute and chronic adverse health effects. However, reference toxicological values 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 National Ambient Air Quality Standards (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. The NOC Supporting Information Report (LAI 2021) concludes that emissions from the proposed project are not expected to cause or contribute to an exceedance of any NAAQS.

6.2.7 Cumulative Exposure to DEEP

Cumulative exposures to DEEP were modeled with two annual averaging period models. The first model, entitled DPM_ANN, looks at the theoretical maximum year for DEEP emissions. In addition to regular maintenance and power outage operating scenarios for all 12 generators, this model represents single-event operating scenarios, such as engine commissioning, that are not expected to occur more than once in the engine's lifetime. Results from this model are used when comparing exposure to any relevant threshold level. The second model, crDPM_ANN, is used to estimate cancer risk as a result of the project and averages single-event operating scenarios over 70 years. Results from this model are used only in cancer risk calculations.

Both cancer and theoretical maximum year (non-cancer) DEEP impacts were evaluated at key risk receptor locations. Key risk receptor locations are consistent between the two models, but present different concentrations based on the model. Table 6-3 summarizes Facility-related impacts for the two model results at the key risk receptor locations. Figure 6-3 presents cumulative theoretical maximum year DEEP contours within the modeling domain.

Cumulative impacts, which account for local and regional background concentrations, were also assessed for DEEP at key risk receptor locations. The cumulative impact for the theoretical maximum year at each key risk receptor location is as follows:

- MIBR/MICR = 0.49 μg/m³
- MIRR = 0.17 μg/m³
- MIIR = 0.16 μg/m³

• Hospital (I-7) = 0.15 μg/m³.

The cumulative DEEP impact for cancer risk calculations at each key risk receptor location is as follows:

- MIBR/MICR = $0.31 \,\mu\text{g/m}^3$
- MIRR = 0.15 μg/m³
- MIIR = 0.16 μg/m³
- Hospital (I-7) = 0.15 μg/m³.

Cumulative impacts include both local and regional background concentrations for DEEP. The theoretical maximum year cumulative concentration at the MIRR is approximately 45 times greater than the DEEP ASIL. This is modeled to occur approximately 0.25 miles southeast of the nearest emergency generator at the Facility. 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 lower than permitted emissions, but worst-case emissions were used to avoid underestimating cumulative DEEP exposure concentrations.

6.2.8 Cumulative Exposure to NO₂

Project-only NO₂ impacts are presented for the key risk receptor locations in Table 6-4 and on Figure 6-2. Some project-only NO₂ impacts were above the ASIL. However, conditions in which the ASIL is continuously exceeded are not probable since this occurs only during a power outage. The frequency with which these impacts could occur is discussed further in Section 6.4.

Cumulative exposures to NO₂ were modeled with a 1-hour averaging period for a power outage scenario in which all the Facility emergency generators are running concurrently for the full hour. The model used for this assessment is named NO2_1HR_ASIL. Cumulative 1-hour NO₂ impacts at key risk receptor locations were also assessed. Figure 6-4 shows the calculated cumulative NO₂ concentrations near the Facility based on allowable emissions from the proposed project. Cumulative impacts for 1-hour NO₂ at key risk receptor locations are as follows:

- MIBR/MICR = 981 μg/m³
- MIRR = 230 μg/m³
- MIIR = 174 μg/m³
- Hospital = $145 \,\mu g/m^3$.

Cumulative NO₂ impacts were below the ASIL at the MIRR, MIIR, and hospital. Cumulative impacts were above the ASIL at the MIBR/MICR. However, conditions in which the ASIL is continuously exceeded at these locations are not probable since this occurs only during a power outage. The frequency with which these conditions could occur is discussed further in Section 6.4.1.2.

6.2.9 Long-Term Exposure to NO₂

As discussed previously, exposure to NO₂ can cause both acute and chronic adverse health effects. However, reference toxicological values for NO₂ exposure at long-term intervals do not currently exist. Therefore, long-term risks from NO₂ exposure are not quantified in this assessment. Regardless, not quantifying long-term health risks in this document does not imply that they have not been considered. Instead, it is assumed that compliance with the annual NAAQS standard for NO₂ is an indicator of acceptable long-term health effects from NO₂ exposure. The NOC Supporting Information Report (LAI 2021) concludes that emissions from the proposed project are not expected to cause or contribute to an exceedance of any NAAQS.

6.3 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 an adverse health outcome. The process often involves establishing a toxicity value or criterion to use in assessing potential health risk. Table 6-5 shows risk factors used to calculate lifetime cancer risk, as well as non-cancer and cancer toxicity values for all pollutants with maximum emissions exceeding their respective SQERs.

6.3.1 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 (CalEPA 1998; EPA; accessed March 10, 2021; 2002). 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 non-cancer health effects are not expected, and a metric by which to quantify increased risk from exposure to emissions.

The EPA's reference concentration (RfC) and OEHHA's REL for diesel engine exhaust (measured as DEEP) was derived from dose-response data on inflammation and changes in the lungs from rat inhalation studies. Each agency established a level of 5 μ g/m³ as the concentration of DEEP in air at which long-term exposure is not expected to cause non-cancer health effects.

NAAQS and other regulatory toxicological values for short- and intermediate-term exposure to PM 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 μ g/m³, and are expressed in units of inverse concentration (i.e., [μ g/m³]⁻¹). OEHHA's URF for DEEP is 0.0003 (μ g/m³)⁻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 risk of 300 excess cancer cases per million people exposed.

6.3.2 Dose Response Assessment for NO₂

OEHHA developed an acute REL for NO₂ based on inhalation studies of asthmatics exposed to NO₂. These studies found that some asthmatics exposed to about 0.25 ppm (i.e., 470 μ g/m³) experienced increased airway reactivity following inhalation exposure to NO₂ (CalEPA 1998). Not all asthmatic subjects experienced an effect.

The acute REL derived for NO₂ does not contain any uncertainty factor adjustment and, therefore, does not provide any additional buffer between the derived value and the exposure concentration at which effects have been observed in sensitive populations. This implies that exposure to NO₂ at levels equivalent to the acute REL (which is also the same as Ecology's ASIL) could result in increased airway reactivity in a subset of asthmatics. People without asthma or other respiratory disease are not likely to experience effects at NO₂ levels at or below the REL.

6.4 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.

6.4.1 Evaluating Non-Cancer Hazards

The non-cancer health impacts were evaluated based on the conservatively high 1-hour and annualaverage emission rates. In order to evaluate the potential for non-cancer health effects that may result from exposure to TAPs, exposure concentrations at each receptor location were compared to relevant non-cancer toxicological values (i.e., RfC, REL). Table 6-5 lists the non-cancer toxicological values that were used for this assessment. If a concentration exceeds the RfC, minimal risk level, or REL, this indicates only the potential for 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:

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

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

6.4.1.1 Hazard Quotient - DEEP

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

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

HQs were calculated for the maximally exposed residential, workplace, and sensitive receptors. Because chronic toxicity values (RfCs and RELs) are based on a continuous exposure, an adjustment is sometimes necessary or appropriate to account for shorter receptor exposure periods (i.e., 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 µg/m³ 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 6-6 shows chronic HQs at the maximally exposed receptor locations near the project site attributable to DEEP exposure from the Facility and all background sources. HQs are significantly lower than 1 for all receptors' cumulative exposure to DEEP. This indicates that non-cancer health effects are unlikely to result from chronic exposure to DEEP in the vicinity of the Facility.

6.4.1.2 Hazard Quotient - NO₂

To evaluate possible non-cancer effects from exposure to NO_2 , modeled concentrations at receptor locations were compared to their respective non-cancer toxicological values. In this case, maximum-modeled 1-hour NO_2 concentrations were compared to the acute REL (470 µg/m³). The acute HQ for NO_2 exposure was calculated using the following equation:

Acute HQ = $\frac{maximum \ 1 \ hr \ NO_2 \ concentration}{470 \ \mu g/m^3}$

Table 6-7 shows acute HQs at the maximally exposed receptor locations near the project site attributable to NO₂ exposure from the project and all background sources. HQs exceed 1 at the MIBR/MICR.

Given that the acute REL for NO₂ does not provide any additional buffer between the derived value and the exposure concentration at which effects have been observed in sensitive populations, someone with asthma or other respiratory illness present at these locations when both meteorological conditions and engine use during a power outage occurred could experience increased airway reactivity and respiratory symptoms. However, the extremity of exposure symptoms associated with NO₂ exposure at levels contributed by the proposed project are not considered significant.

6.4.1.3 Discussion of Acute Hazard Quotients Greater Than 1

NO₂ HQs may exceed 1 at certain times when unfavorable air dispersion conditions coincide with electrical grid transmission failure. If the HQ is less than 1, then the risk is generally considered acceptable. The more the HQ increases above 1, the more likely it is that adverse health effects will occur by some undefined amount (due in part to how the risk-based concentration is derived).

OEHHA developed an acute REL for NO₂ based on inhalation studies of people with asthma. These studies found that some subjects exposed to about 0.25 ppm (470 μ g/m³) experienced increased airway reactivity following exposure (CalEPA 2008). Not all subjects experienced apparent effects. Like NO₂, DEEP may interact with airways in the respiratory tract. Simultaneous exposure to NO₂ and DEEP components of diesel engine exhaust probably results in a higher risk of adverse respiratory effects than exposure to the NO₂ component alone.

6.4.1.4 Combined Hazard Quotient for All Pollutants with Emission Rates that Exceed the SQERs

The chronic non-cancer health impacts were evaluated based on conservatively high emission rates. Three TAPs (CO, DEEP, and NO₂) to be emitted by the Facility have emission rates that exceed their respective SQERs and, therefore, are subject to further evaluation. The receptor locations of concern are the MIBR/MICR and MIRR.

Since DEEP is the only TAP with emissions above the SQER that can potentially result in chronic non-cancer health impacts, it is not necessary to calculate a hazard index (HI).

The acute combined HI for each location is the sum of the 1-hour time-weighted average HQs for NO_2 and CO. Table 6-8 shows the acute combined HI including and not including NO_2 .

The information in Table 6-8 indicates that an acute health effect from CO is unlikely to occur even under worst-case conditions at maximally impacted receptor locations. When NO_2 is included in the acute combined HI, the HIs at the MIBR/MICR exceed 1. Section 6.4.1.2 discusses the probability of worst-case scenario exceedances.

6.4.1.5 Probability Analysis of NO₂ ASIL Exceedances

LAI analyzed the frequency (number of hours) that meteorological conditions could result in a NO₂ concentration greater than 470 μ g/m³ across the modeling domain. Table 6-9 displays these results graphically by showing the exceedance interval, or number of years between each theoretical occurrence of project-related NO₂ concentrations exceeding 470 μ g/m³, based on Ecology's requirement to use a power outage duration for Grant County Public Utility District (PUD) customers of 8 hours (Palcisko 2020).

LAI conducted an analysis of the duration of each event exceeding 470 μ g/m³ at the MIBR/MICR (C-2), and the time intervals between those exceedance events. The results were as follows:

•	Number of AERMOD modeled hours:	43,800
•	Number of hours in 5 years exceeding 470 μ g/m ³ :	292

This statistical analysis confirms that ASIL exceedances would occur infrequently, even if the generators are assumed to operate continuously for 5 years. The MIBR/MICR is located along the west side of the H5 Data Center fence line, at a location that is unlikely to be occupied other than by passers-by.

To calculate the frequency of occurrence, LAI used the following steps for each maximally impacted receptor location:

- Calculate the hourly probability of occurrence of "poor dispersion conditions" defined as the fraction of hours in the 5-year modeling period when AERMOD predicts a 1-hour NO₂ concentration exceeding the threshold, assuming a power outage occurs continuously during the 5-year period.
- Calculate the hourly probability of occurrence of a power outage based on 8 hours of outage per year, a conservative overestimate calculated based on historical PUD data.
- Calculate the joint probability of those two independent events happening simultaneously and convert the joint probability to an annual recurrence interval.

The results of these calculations are shown in Table 6-9.

Table 6-9 summarizes the probability that the modeled values exceed the selected thresholds for the assumption of 8 hours/year of power outage. The table presents the number of hours that the threshold is exceeded during the 5-year period, the average number of hours per year that the threshold is exceeded, the probability that a power outage will occur for any given hour, the probability of exceeding the threshold during a power outage for any given hour (phr), the overall probability that the threshold will be exceeded in a given year (p1yr), and the estimated recurrence interval. Overall annual probability, p, is calculated as: p = 1 - (1 - phr)n, where n is the total number of hours (e.g., 8,760 hours in 1 year). The annual recurrence interval is the inverse of the overall annual probability, and represents the average number of years between exceedances.

As shown in the table, when taking into account historical Grant County PUD electrical grid reliability, the recurrence interval of cumulative NO₂ impacts above the ASIL at the MIBR/MICR was calculated as 10 years.

The MIBR/MICR is located west along the H5 Data Center fence line at a location that is unlikely to be occupied other than by passers-by. NO₂ concentrations resulting from Facility emissions at the MIRR and MIIR are expected to never exceed the ASIL. This evaluation demonstrates that the probability of a receptor location being exposed to NO₂ concentrations above the acute REL is very low.

6.4.1.6 Probability Analysis of NO₂ AEGL Exceedances

Project-related NO₂ concentrations are not expected to exceed the AEGL 1 of 940 μ g/m³ at any maximally exposed receptor locations.

6.4.2 Quantifying an Individual's Increased Cancer Risk

6.4.2.1 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 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:

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

The exposure frequencies for each receptor type are shown below and provided in Table 6-2, based on Ecology's judgment from review of published risk evaluation guidelines.

		Value Based on Receptor Type						
Parameter	Description	Residential	Worker	School- Staff	School- Student	Hospital	Boundary	Units
C _{Air}	Concentration in air at the receptor location		See Table 6-3				µg/m³	
URF	Unit Risk Factor		0.0003				(µg/m³)-1	
EF1	Exposure Frequency	365	250	200	180	365	250	Days/Year
EF2	Exposure Frequency	24	8	8	8	24	2	Hours/Day
ED	Exposure Duration	70	40	40	7 (Elem) 4 (HS & College)	1	30	Years
AT	Averaging Time	613,200				Hours		

DEEP Exposure Frequencies for Each Receptor Type

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 6-10 shows the estimated cancer risks associated with predicted project-related DEEP concentrations and the URFs. The location with the greatest increased cancer risk associated with project-related DEEP concentrations is the MIRR location. The calculated lifetime cancer risk resulting from Facility emissions is 9.6 per million at the MIRR. This is less than 10 per million, which is the recommended permissible limit for second-tier review under Chapter 176-460 WAC.

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

Also shown in Table 6-10 are the cumulative cancer risks for each maximally impacted receptor location. This accounts for currently permitted DEEP emissions from neighboring sources. The maximum cumulative (project-related and background emissions) cancer risk impact at the MIRR location is estimated to be 44 per million. Cancer risk at the MIBR/MICR is estimated to be 12 per million.

6.4.2.2 Cancer Risk from Exposure to All Pollutants

An evaluation was completed to estimate the increased cancer risk from exposure to all potentially carcinogenic compounds from the proposed project alone. The emission rate for every carcinogenic constituent was considered in this evaluation, which is shown in Table 6-11. As indicated in the table, the cancer risk associated with DEEP alone at the MIRR location is 9.6×10^{-6} . The other recognized carcinogenic compounds contribute negligibly to the overall cancer risk (i.e., 2.0×10^{-8}). The combined cancer risk caused by all constituents is 9.6 per million or 9.6×10^{-6} .

6.4.2.3 Cancer Risk from Exposure to NO₂

Cancer health risk was not evaluated for NO₂ because NO₂ is not considered carcinogenic by the US Department of Health and Human Services, the International Agency for Research on Cancer, or the EPA (ATSDR 2011; EPA; accessed March 10, 2021).
6.5 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 and NO₂ emissions from the proposed project and "background" sources of DEEP and NO₂. The assumptions used in the face of uncertainty may tend to overestimate or underestimate the health risks described in the HIA.

6.5.1 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 PM used for this analysis were based on the upper range of vendor estimates for engines meeting Tier 2 emission criteria. The forecast emission rates for NO₂ were based on the conservatively high assumption that NO₂ makes up 10 percent of the emitted NO_x. 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 will be exposed to DEEP and NO₂ emissions from the proposed Facility. 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 power outages is also uncertain. For this permit application, H5 conservatively estimated that it would use the generators during emergency outages for no more than 8 hours per year. Grant County PUD reports an Average Service Availability Index (or percent of time that a customer has power provided during the year) of more than 99.98 percent each year (2007 to 2019) and a Customer Average Interruption Duration Index (or average duration of power interruption per customer) of 167.5 minutes (2.8 hours) over the same period (Palcisko 2020). While this high level of historical reliability provides some assurance that electrical service is relatively stable, H5 cannot predict future outages with any degree of certainty. H5 proposes a limit of 38 hours average per generator per year for all Facility emergency generator operations (including maintenance and testing), and estimates that this limit should be sufficient to meet its emergency demands. It is expected that calculations of cancer risk will be significantly overestimated by assuming the generators will operate annually at the maximum permitted level for 70 consecutive years.

6.5.2 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, and 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 project 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.

6.5.3 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 non-cancer effects in animals or humans. The EPA applies these uncertainty factors so that it derives a toxicity value that is considered protective of humans including susceptible populations.

6.5.3.1 DEEP Toxicity Uncertainty

In the case of the DEEP RfC, the EPA acknowledges (EPA 2002):

... 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" (EPA 2002). However, the EPA suggested that a URF based on existing DEEP toxicity studies would range from 1×10^{-5} to 1×10^{-3} per µg/m³. OEHHA's DEEP URF (3×10^{-4} per µg/m³) falls within this range. Regarding the range of URFs, the EPA states in its health assessment document for diesel exhaust (EPA 2002):

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.

6.5.3.2 NO₂ Toxicity Uncertainty

Similar to DEEP, uncertainty exists surrounding NO₂ toxicity. In a 2009 review of more than 50 experimental studies regarding human exposure to NO₂, Hesterberg et al. (2009) found that "the reporting of statistically significant changes in lung function and bronchial sensitivity did not show a consistent trend with increasing NO₂ concentrations." Hesterberg et al. (2009) also reported:

The NO₂ epidemiology remains inconsistent and uncertain due to the potential for exposure misclassification, residual confounding, and co-pollutant effects, whereas animal toxicology findings using high levels of NO₂ exposure require extrapolation to humans exposed at low ambient NO₂ levels.

In OEHHA's Acute Toxicity Summary, describing the factors contributing to its determination of an acute REL for NO₂, OEHHA reported uncertainty in NO₂ effects on pulmonary function due to the lack of accidental human exposure data available. High uncertainty factors were used when extrapolating animal test results to humans due to interspecies differences. "Species-specific susceptibility comparisons of experimental animals suggest that humans are less sensitive to the toxic effects of NO₂ than smaller experimental animal species." OEHHA found that exposure levels that resulted in compromised lung function in experimental animal species failed to produce even symptoms of mild irritation in humans with asthma (CalEPA 1999).

It is likely that the mixture of pollutants emitted by new-technology diesel engines (such as those proposed for this project) is different from older-technology engines. Table 7-1 presents a summary of how the uncertainty affects the quantitative estimate of risks or hazards.

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 Facility are less than the ASIL values established by Ecology for all pollutants, with the exception of DEEP and NO₂. The worst-case emission rates are less than the SQERs for most pollutants, with the exception of DEEP, CO, and NO₂. The long-term uncontrolled cancer risks at the nearby residences and businesses range from 7.0 to 9.6 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 residential, business, and sensitive receptor locations, caused solely by emissions from the proposed project, 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 average cumulative DEEP cancer risks for the maximally exposed home, business, and sensitive receptors are as follows:

Facility-only cancer risk (MIBR/MICR):	7.3 per million
Background DEEP cancer risk:	4.4 per million
Cumulative DEEP cancer risk:	12 per million
Facility-only cancer risk (MIRR):	9.6 per million
Background DEED concernicky	24 por million
Background DEEP cancer risk:	34 per million

7.3 Non-Cancer Risk Hazard Quotients

The maximum HQ related to project-only annual-average DEEP at any maximally impacted receptor location is 0.074.

The maximum HQ related to project-only 1-hour average NO₂ at any maximally impacted receptor location is 2.0. The project-only maximum acute HI for impacts caused by emissions of NO₂ and CO is 2.2. As described above, 1-hour NO₂ acute REL exceedances—that would result in an HQ or HI greater than 1—could theoretically occur; however, it would require two infrequent, independent events occurring simultaneously: a full power outage and winds blowing directly toward the receptor location with exceptionally poor atmospheric dispersion. An evaluation of the recurrence interval of HQs greater than 1 concluded that the estimated minimum recurrence interval is 9 years considering the historical power grid reliability in Grant County.

This evaluation demonstrates that the probability that this project could cause non-cancer health impacts is very low. Additionally, the extremity of potential toxicity outcomes associated with NO₂

exposure at levels evaluated for this project are not considered significant (e.g., mild, transient adverse health effects).

8.0 **REFERENCES**

- ATSDR. 2002. Nitrogen Oxides (Nitric Oxide, Nitrogen Dioxide, etc.). Division of Toxicology and Environmental Medicine, Agency for Toxic Substances and Disease Registry, US Department of Health and Human Services. April. http://www.atsdr.cdc.gov/toxfaqs/tfacts175.pdf.
- ATSDR. 2011. "Toxic Substances Portal: Nitrogen Oxides." Agency for Toxic Substances and Disease Registry, US Department of Health and Human Services. March 3. http://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=69.
- CalEPA. 1998. Staff Report: Initial Statement of Reasons for Rulemaking: Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant. Air Resources Board and Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. June. https://www.arb.ca.gov/toxics/dieseltac/staffrpt.pdf.
- CalEPA. 1999. Acute Toxicity Summary: Nitrogen Dioxide. In: Appendix C of The Determination of Acute Reference Exposure Levels in Airborne Toxicants. Air Toxicology and Epidemiology Section, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. March.
- CalEPA. 2008. Appendix D.2: Acute RELs and Toxicity Summaries Using the Previous Version of the Hot Spots Risk Assessment Guidelines. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. December 19. http://oehha.ca.gov/media/downloads/crnr/appendixd2final.pdf.
- CalEPA. 2015. Air Toxics Hot Spots Program Risk Assessment Guidelines: Guidance Manual for Preparation of Health Risk Assessments. Air, Community, and Environmental Research Branch, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. February. http://oehha.ca.gov/media/downloads/crnr/2015guidancemanual.pdf.
- CalEPA. 2016. "OEHHA Acute, 8-Hour and Chronic Reference Exposure Level (REL) Summary." Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. June 28. http://oehha.ca.gov/air/general-info/oehha-acute-8-hour-and-chronic-reference-exposure-levelrel-summary.
- City of Quincy. City of Quincy Zoning and Urban Growth Area Map. City of Quincy, Washington. https://www.quincywashington.us/266/Zoning-Map.
- Ecology. 2008. White Paper: Concerns about Adverse Health Effects of Diesel Engine Emissions. Publication No. 08-02-032. Air Quality Program, Washington State Department of Ecology. December 3. https://fortress.wa.gov/ecy/publications/documents/0802032.pdf.
- EPA. "Integrated Risk Information System." US Environmental Protection Agency. https://www.epa.gov/iris.
- EPA. 2002. Health Assessment Document for Diesel Engine Exhaust. Publication No. EPA/600/8-90/057F. Office of Research and Development National Center for Environmental Assessment, US Environmental Protection Agency. May. https://nepis.epa.gov/Exe/ZyPDF.cgi/300055PV.PDF?Dockey=300055PV.PDF.
- Grant County. 2018. Official Comprehensive Land Use Plan, Grant County, Washington. August 6. http://www.grantcountywa.gov/GIS/MISC/PDF/Maps_11x17/LandUsePlanning/GrantCo_Compre hensivePlan_36x48.pdf.

- Hesterberg, Thomas W., William B. Bunn, Roger O. McClellan, Ali K. Hamade, Christopher M. Long, and Peter A. Valberg. 2009. "Critical Review of the Human Data on Short-Term Nitrogen Dioxide (NO2) Exposures: Evidence for NO2 No-Effect Levels." Critical Reviews in Toxicology. 39 (9):743-781. doi: 10.3109/10408440903294945. http://dx.doi.org/10.3109/10408440903294945. 2009/10/01.
- Jarvis, D.J., Adamkiewicz G., Heroux M.E., R. Rapp, and F.J. Kelley. 2010. Nitrogen Dioxide. In: WHO Guidelines for Indoor Air Quality: Selected Pollutants. World Health Organization. http://www.ncbi.nlm.nih.gov/books/NBK138707/.
- LAI. 2021. Notice of Construction Application Supporting Information Report, H5 Data Center Campus Expansion, Quincy, Washington. Landau Associates, Inc. April 13.
- NAC AEGL Committee. 2008. Interim/Proposed: Acute Exposure Guidelines Levels for Nitrogen Dioxide, Nitrogen Tetroxide. The National Advisory Committee for the Development of Acute Exposure Guideline Levels for Hazardous Substances. December. https://nepis.epa.gov/Exe/ZyPDF.cgi/P1006QYS.PDF?Dockey=P1006QYS.PDF.
- NRC. 1983. Risk Assessment in the Federal Government: Managing the Process. Washington, DC: National Research Council, National Academy of Sciences.
- NRC. 1994. Science and Judgment in Risk Assessment. Washington, DC: National Research Council Committee on Risk Assessment of Hazardous Air Pollutants.
- Palcisko, G. 2020. "Re: H5 Data Centers Air Modeling & HIA Protocols." From Gary Palcisko, Air Quality Program, Washington State Department of Ecology, to Mark Brunner, Landau Associates, Inc.; Ranil Dhammapala, Washington State Department of Ecology; Jenny Filipy, Washington State Department of Ecology. December 4.









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	Notable Development	Direction from Project Site	City / County Zoning	HIA Zoning ID
с	Industrial Zone	North	City Industrial	C-1
o m	Agricultural Zone	North	County Agricultural	C-1
m	Industrial Zone	West	City Industrial	C-2
r c	Industrial Zone	South	City Industrial	C-3
i a	Agricultural Zone	East	County Agricultural	C-4
I	Industrial Zone	East	City Industrial	C-4
e	Residential Zone	North	County Residential	R-1
s i d	Residential Zone	West	City Residential	R-2
e n t	Residential Zone	South	County Residential	R-3
i a	Residential Zone	East	County Residential	R-4
	Quincy Innovation Academy	Southwest	City Residential	I-1
n	Quincy Jr High/Middle School	Southwest	City Residential	I-2
s t	Monument Elementary	Southwest	City Residential	I-3
i t	Ancient Lakes Elementary	Southwest	City Residential	I-4
u t	Mountain View Elementary	Southwest	City Residential	I-5
i o	Quincy High School	Southwest	City Residential	I-6
n	Pioneer Elementary	Southwest	City Residential	I-7
a I	Quincy Valley Medical Center	Southwest	City Residential	I-8
	Quincy High School (new)	West	City Residential	I-9

Table 4-1 Summary of BACT Determination for Diesel Engine Generators H5 Data Center Quincy, Washington

Pollutant(s)	BACT Determination
Particulate matter (PM), carbon monoxide	a. Use of EPA Tier 2-certified engines when installed and operated as
(CO), volatile organic compounds (VOCs), and nitrogen oxides (NO_x)	emergency engines, as defined by 40 CFR 60.4219.
	b. Compliance with the operation and maintenance restrictions of 40 CFR Part 60, Subpart IIII.

Abbreviations and Acronyms:

BACT = Best available control technology

CFR = Code of Federal Regulations

CO = Carbon monoxide

EPA = US Environmental Protection Agency

NO_x = Nitrogen oxides

PM = Particulate matter

VOCs = Volatile organic compounds

Table 4-2 Summary of tBACT Determination for Diesel Engine Generators H5 Data Center Quincy, Washington

Toxic Air Pollutant(s)	tBACT Determination
Particulate matter (PM), carbon monoxide (CO), volatile organic compounds (VOCs), and nitrogen oxides (NO _x)	Use of EPA Tier 2-certified engines when installed and operated as emergency engines, as defined by 40 CFR 60.4219. Compliance with the operation and maintenance restrictions of 40 CFR Part 60, Subpart IIII.
Toxic air pollutants, including primary nitrogen dioxide (NO ₂), diesel engine exhaust particulate matter (DEEP),	Compliance with the proposed BACT requirements for PM, CO, VOCs, NO_x , and SO_2 .
CO, 1,3-butadiene, acrolein, benzene, dibenz(a,h)anthracene, formaldehyde, naphthalene, arsenic, and vanadium	Compliance with the proposed BACT for the cooling towers is the use of high-efficiency drift eliminators that reduce the drift droplet rate to at most 0.0005 percent of the recirculation water flow rate.

Abbreviations and Acronyms:

BACT = best available control technology

CFR = Code of Federal Regulations

CO = carbon monoxide

DEEP = diesel engine exhaust particulate matter

EPA = US Environmental Protection Agency

NO₂ = nitrogen dioxide

NO_x = nitrogen oxides

PM = particulate matter

tBACT = best available control technology for toxic air pollutants

VOC = volatile organic compound

Table 4-3

Project Emissions Compared to Small-Quantity Emission Rates

H5 Data Center

Quincy, Washington

			Project			
		Averaging	Emissions	De Minimis	SQER	Required
Pollutant	CAS No.	Period		(Ibs/averaging perio	od)	Action
Primary NO ₂	10102-44-0	1-hr	57	0.46	0.87	Model
DEEP	DPM	year	1,049	0.027	0.54	Model
со	630-08-0	1-hr	152	1.1	43	Model
SO ₂	7446-09-5	1-hr	0.43	0.46	1.2	
1,3-Butadiene	106-99-0	year	0.73	0.27	5.4	Report
Acetaldehyde	75-07-0	year	0.47	3.0	60	-
Acrolein	107-02-8	24-hr	6.7E-03	1.3E-03	2.6E-02	Report
Benzene	71-43-2	year	15	1.0	21	Report
Benz(a)anthracene	56-55-3	year	0.012	0.045	0.89	-
Benzo(a)pyrene	50-32-8	year	4.8E-03	8.2E-03	0.16	
Benzo(b)fluoranthene	205-99-2	year	0.021	0.045	0.89	
Benzo(k)fluoranthene	207-08-9	year	4.1E-03	0.045	0.89	
Chrysene	218-01-9	year	0.029	0.45	8.9	
Dibenz(a,h)anthracene	53-70-3	year	6.5E-03	4.1E-03	0.082	Report
Formaldehyde	50-00-0	year	1.5	1.4	27	Report
Indeno(1,2,3-cd)pyrene	193-39-5	year	7.8E-03	0.045	0.89	
Naphthalene	91-20-3	year	2.4	0.24	4.8	Report
Propylene	115-07-1	24-hr	2.4	11	220	
Toluene	108-88-3	24-hr	0.24	19	370	
Xylenes	1330-20-7	24-hr	0.16	0.82	16	
Arsenic	_	year	0.011	2.5E-03	0.049	Report
Beryllium	_	year	3.9E-04	3.4E-03	0.068	
Cadmium	—	year	3.9E-04	1.9E-03	0.039	
Chromium ^a	_	24-hr	1.8E-06	3.7E-04	7.4E-03	
Cobalt	7440-48-4	24-hr	3.2E-05	3.7E-04	7.4E-03	
Copper	_	1-hr	1.4E-04	9.3E-03	0.19	
Lead	_	year	0.046	10	14	
Manganese	_	24-hr	1.8E-04	1.1E-03	0.022	
Mercury	7439-97-6	24-hr	2.1E-06	1.1E-04	2.2E-03	
Selenium	—	24-hr	1.8E-05	0.074	1.5	
Vanadium	7440-62-2	24-hr	6.5E-04	3.7E-04	7.4E-03	Report
Total Cyanide	74-90-8	24-hr	1.1E-04	3.0E-03	0.059	
Ammonia	7664-41-7	24-hr	7.4E-04	1.90	37	
Total Phosphorus	7723-14-0	24-hr	7.4E-04	0.074	1.5	

Notes:

Highlighted cells indicate pollutants that require ambient air dispersion modeling analysis

Updated to WAC 460 12/30/2019

^a All chromium was assumed to be Chromium (III), soluble particulates.

Abbreviations and Acronyms:

 $\label{eq:CAS} \begin{array}{l} \mathsf{CAS} = \mathsf{Chemical Abstract Service} \\ \mathsf{CO} = \mathsf{carbon monoxide} \\ \mathsf{DEEP} = \mathsf{diesel engine exhaust particulate matter} \\ \mathsf{hr} = \mathsf{hour} \\ \mathsf{lbs} = \mathsf{pounds} \\ \mathsf{NO}_2 = \mathsf{nitrogen dioxide} \\ \mathsf{SO}_2 = \mathsf{sulfur dioxide} \\ \mathsf{SQER} = \mathsf{small-quantity emission rate} \end{array}$

Table 4-4Estimated Project Impacts Compared to Acceptable Source Impact LevelsH5 Data Center

Quincy, Washington

Pollutant	CAS No.	Averaging Period	AERMOD Filename	ASIL (μg/m³)	Modeled Maximum Facility Impact (μg/m ³)
Nitrogen dioxide (NO ₂)	10102-44-0	1-hr	NO2_1HR_ASIL.ADI	470	919
DEEP ^a	DPM	year	DPM_ANN.ADI	0.0033	0.37
со	630-08-0	1-hr	CO.ADI	23,000	4,945

Notes:

^a Predicted maximum impacts are based on emissions for the theoretical maximum year.

Highlighted cells indicate pollutants that require a human health impact assessment

Abbreviations and Acronyms:

μg/m³ = micrograms per cubic meter AERMOD = American Meteorological Society (AMS)/US Environmental Protection Agency (EPA) Regulatory Model ASIL = acceptable source impact level CAS = Chemical Abstract Service CO = carbon monoxide DEEP = diesel engine exhaust particulate matter hr = hour lbs/avg. period = pounds per averaging period NO₂ = nitrogen dioxide Page 1 of 1

Table 6-1Chemicals Assessed for Multiple Exposure Pathways

H5 Data Center

Quincy, Washington

	Breast		Exposed		Leafy	Meat, Milk	Protected	Root		
Chemical	Milk	Dermal	Vegetable	Fish	Vegetable	& Eggs	Vegetable	Vegetable	Soil	Water
4,4'-Methylene dianiline		Х	Х	Х	Х		Х	Х	Х	Х
Beryllium & compounds		Х	Х	Х	Х	х	Х	Х	Х	Х
Cadmium & compounds		Х	Х	Х	Х	Х	Х	Х	Х	Х
Chromium VI & compounds		Х	Х	Х	Х	х	Х	Х	Х	Х
Creosotes		Х	Х	Х	Х	х			Х	Х
Diethylhexylphthalate		Х	Х	Х	Х		Х	Х	Х	Х
Dioxins & furans	Х	Х	Х	Х	Х	х	Х	Х	Х	Х
Fluorides (including hydrogen fluoride)						To be de	termined			
Hexachlorocyclohexanes		Х	Х	Х	Х				Х	Х
Inorganic arsenic & compounds		Х	Х	Х	Х	х	Х	Х	Х	Х
Lead & compounds		Х	Х	Х	Х	х	Х	х	х	Х
Mercury & compounds		Х	Х	Х	Х		х	х	х	х
Nickel		Х	Х		Х	Х	Х	Х	Х	Х
Polycyclic aromatic hydrocarbons (PAHs)		Х	Х	Х	Х	Х			Х	Х
Polychlorinated biphenyls (PCBs)	Х	Х	Х	Х	Х	Х	Х	Х	Х	Х

Source: CalEPA 2015.

Exposure Assumptions and Unit Risk Factors Used for Lifetime Cancer Risk Assessment H5 Data Center Quincy, Washington

		Exposure	
Receptor Type	Annual Exposure	Duration	Unit Risk Factor
Unoccupied Land	2 hours/day	30 years	7.3 -per-million cancer risk per $\mu g/m^3$ DEEP
	250 days/year	So years	γ.5 -per-minion cancer risk per μg/m DEEP
Residences	24 hours/day	70 years	300 -per-million cancer risk per $\mu g/m^3$ DEEP
Residences	365 days/year	70 years	500 -per-minion cancer risk per µg/m DEEP
Schools	36 hours/week	4 years	2.8 -per-million cancer risk per $\mu g/m^3$ DEEP
(High School Students)	40 week/year	4 years	2.0 -per-minion cancer risk per µg/m DEEP
Schools	36 hours/week	7 years	4.9 -per-million cancer risk per $\mu g/m^3$ DEEP
(Elementary School Students)	40 week/year	7 years	4.9 -per-minion cancer risk per µg/m DEEP
Schools	40 hours/week	40 years	31 -per-million cancer risk per $\mu g/m^3$ DEEP
(All Teachers)	40 week/year	40 years	31 -per-minion cancer risk per μg/m DEEP
Commercial, Industrial, or	8 hours/day	40 years	38 -per-million cancer risk per $\mu g/m^3$ DEEP
Agricultural	250 days/year	40 years	³⁸ -per-million cancer risk per μg/m DEEP
Hospital	24 hours/week	1 year	4.3 -per-million cancer risk per $\mu g/m^3$ DEEP
	365 week/year	туса	4.3 -per-minion cancer risk per µg/m DEEP

Abbreviations and Acronyms:

μg/m³ = micrograms per cubic meter DEEP = diesel engine exhaust particulate matter

Table 6-3 Summary of Project Impacts from Emissions of DEEP H5 Data Center Quincy, Washington

	UTM Zone			Direction From	Approximate Distance From Nearest Project- Generator		for Cancer Risk ^b	Facility DEEP Maximum Year Impact
Key Risk Receptor ^a	Easting (m)	Northing (m)	ID	Facility	Feet	Meters	(μg/m³)	(μg/m³)
MIBR/MICR	286,663	5,237,242	C-1	North	352	107	0.19	0.37
MIRR	286,900	5,236,733	R-4	Southeast	1,296	395	0.032	0.058
MIIR (School)	285,200	5,235,370	I-2	Southwest	7,665	2,336	0.0023	0.0043
Hospital	282,900	5,234,870	I-8	Southwest	14,605	4,451	0.00088	0.0016

Notes:

^a Key Risk Receptors are at the same location between DEEP Annual Impact for Cancer Risk and DEEP Maximum Year Impact.

^b Facility DEEP Annual Impact for Cancer Risk is modeled in crDPM_ANN.

^c Facility DEEP Maximum Year Impact is modeled in DPM_ANN.

Abbreviations and Acronyms:

 $\mu g/m^3$ = micrograms per cubic meter

DEEP = diesel engine exhaust particulate matter

m = meter

MIBR = maximally impacted boundary receptor location

MICR = maximally impacted commercial receptor location

MIIR = maximally impacted institutional receptor location

MIRR = maximally impacted residential receptor location

UTM = Universal Transverse Mercator

Table 6-4Summary of Project Impacts from Emissions of Nitrogen DioxideH5 Data Center

Quincy, Washington

	UTM Zone 11 North			Direction From	Approximate Distance From Nearest Project Generator		Facility NO ₂ 1-hr Impacts	Project Exceedance Counts (5 years)		Project + Background Exceedance Counts (5 years)	
Key Risk Receptor	Easting (m)	Northing (m)	ID	Facility	Feet	Meters	(µg/m³)	ASIL	AEGL	ASIL	AEGL
MIBR/MICR	286,524	5,237,112	C-2	West	589	180	919	292	0	561	10
MIRR	286,150	5,237,370	R-2	Northwest	1,965	599	203	0	0	0	0
MIIR	284,900	5,237,170	I-9	West	5,873	1,790	150	0	0	0	0
Hospital	282,900	5,234,870	I-8	Southwest	14,605	4,451	101	0	0	0	0
Receptor with Maximum ASIL Exceedance Counts	286,522	5,237,050	C-2	West	598	182	779	515	0	620	0

Abbreviations and Acronyms:

 $\mu g/m^3$ = micrograms per cubic meter

AEGL = acute exposure guideline

ASIL = acceptable source impact level

m = meter

MIBR = maximally impacted boundary receptor location

MICR = maximally impacted commercial receptor location

MIIR = maximally impacted institutional receptor location

MIRR = maximally impacted residential receptor location

 NO_2 = nitrogen dioxide

UTM = Universal Transverse Mercator

Toxicity Values Used to Assess and Quantify Non-Cancer Hazard and Cancer Risk H5 Data Center Quincy, Washington

Pollutant	Averaging Time Period	Non-Cancer REL (µg/m³)	Carcinogenic URF (μg/m ³) ⁻¹
DEEP	Acute (1-hr average)	N/A	3.0x10 ⁻⁴
DELF	Chronic (12-month average)	5	5.0X10
NO ₂	Acute (1-hr average)	470	N/A
	Chronic (12-month average)	N/A	N/A
со	Acute (1-hr average)	23,000	N/A
	Chronic (12-month average)	N/A	N/A

Source: California Office of Environmental Health Hazard Assessment (OEHHA)

Abbreviations and Acronyms:

 $\mu g/m^3$ = micrograms per cubic meter

CO = carbon monoxide

DEEP = diesel engine exhaust particulate matter

hr = hour

N/A = not applicable to this toxic air pollutant

NO₂ = nitrogen dioxide

REL = reference exposure level

URF = unit risk factor

Table 6-6 Predicted DEEP Impacts and Chronic Hazard Quotients Summary H5 Data Center Quincy, Washington

	Maximum Year DEEP Impact (µg/m ³)			
	MIBR/MICR	MIRR		
H5 Data Center	0.37	0.058		
Local Background ^a	0.11	0.10		
Cumulative Impact	0.49	0.16		

5 = DEEP REL (μg/m ³)	DEEP - Chronic Hazard Quotient	
	MIBR/MICR	MIRR
H5 Data Center	0.074	0.0115
Local Background	0.57	0.50
Cumulative (post-project) HQ	0.10	0.032

Notes:

^a Local background values determined from Ecology's Quincy Storymap of DEEP concentrations.

Abbreviations and Acronyms:

 $\mu g/m^3$ = micrograms per cubic meter

DEEP = diesel engine exhaust particulate matter

HQ = hazard quotient

MIBR = maximally impacted boundary receptor location

MICR = maximally impacted commercial receptor location

MIRR = maximally impacted residential receptor location

REL = reference exposure level

Predicted Nitrogen Dioxide Impacts and Acute Hazard Quotients Summary H5 Data Center Quincy, Washington

	1-hour NO ₂ Impact (μg/m ³)	
	MIBR/MICR	
H5 Data Center	919	
Local Background ^a	62	
Cumulative Impacts	981	

470 = $NO_2 REL^b (\mu g/m^3)$	Acute (1-hour) NO ₂ Hazard Quotient MIBR/MICR	
H5 Data Center	2.0	
Local Background	0.13	
Cumulative HQ	2.1	

Notes:

 $^{\rm a}$ Local background values determined from Ecology's Quincy Storymap of $\rm NO_2$ concentrations.

^b The NO₂ REL is from the California Office of Environmental Health Hazard Assessment: Acute, 8-hour and Chronic Reference Exposure Level Summary (CalEPA 2016).

Abbreviations and Acronyms:

 $\mu g/m^3$ = micrograms per cubic meter

HQ = hazard quotient

MIBR = maximally impacted boundary receptor location

MICR = maximally impacted commercial receptor location

NO₂ = nitrogen dioxide

REL = reference exposure level

Table 6-8 Acute (1-Hour) Combined Hazard Index for Toxic Air Pollutants

H5 Data Center

Quincy, Washington

1-hour Acute Hazard Index ^{a, b}		MIBR/MICR ^c	MIRR
Nitrogen dioxide (NO ₂)	Ambient Impact (μg/m ³)	919	203
	Risk-Based Toxic Threshold Value (µg/m ³)	470	
Hazard Quotient		2.0	0.43
	Ambient Impact (μg/m³)	4,945	1,305
со	Risk-Based Toxic Threshold Value (µg/m ³)	23,000	
Hazard Quotient		0.22	0.057

Combined Hazard Index (HI)	2.2	0.49
Combined HI (not including NO ₂)	0.22	0.057

Notes:

^a The hazard quotient for DEEP is not applicable to this exposure scenario.

^b The MIBR, MICR, and MIRR are the maximally impacted receptors for NO₂.

^c The NO₂-impacted MIBR and MICR were at the same receptor location.

Abbreviations and Acronyms:

 $\mu g/m^3$ = micrograms per cubic meter

CO = carbon monoxide

DEEP = diesel engine exhaust particulate matter

HI = hazard index

MIBR = maximally impacted boundary receptor location

MICR = maximally impacted commercial receptor location

MIIR = maximally impacted institutional receptor location

MIRR = maximally impacted residential receptor location

NO₂ = nitrogen dioxide

Joint Probability of NO₂ Acceptable Source Impact Level Exceedances

H5 Data Center

Quincy, Washington

Risk Receptor Location: MIBR/MICR

	Historical Occurrence:	
Evaluation Detail	Grant County PUD ^a	
	Project Only	Project + Background ^b
Hours of Power Outage per Year	8	
Total No. of Hrs > Threshold (in 5 Yrs)	292	561
Average No. of Hrs > Threshold Per Year	58	112
Hourly Probability of Poor Wind Dispersion	6.7E-03	1.3E-02
Hourly Probability of a Power Outage	9.1E-04	9.1E-04
Joint Probablility (per Hr) of	6.1E-06	1.2E-05
Exceeding the Threshold During a Power Outage		
Overall Probability in 1 Year	5.2E-02	9.7E-02
Recurrence Interval (yrs)	19	10

Risk Receptor Location: Maximum project-only ASIL Exceedance Counts

	Historical Occurrence:	
	Grant County PUD ^a	
Evaluation Detail	Project Only	Project + Background [∞]
Hours of Power Outage per Year	8	
Total No. of Hrs > Threshold (in 5 Yrs)	515	620
Average No. of Hrs > Threshold Per Year	103	124
Hourly Probability of Poor Wind Dispersion	1.2E-02	1.4E-02
Hourly Probability of a Power Outage	9.1E-04	9.1E-04
Joint Probablility (per Hr) of	1.1E-05	1.3E-05
Exceeding the Threshold During a Power Outage		
Overall Probability in 1 Year	9.0E-02	1.1E-01
Recurrence Interval (yrs)	11	9

Note:

- ^a The average power outage duration for Grant County PUD customers was 167.5 minutes and occurred an average of 2 times per year (Palcisko 2020).
- ^b Background values determined from Ecology's Quincy Storymap of DEEP concentrations.

Abbreviations and Acronyms:

μg/m ³ = micrograms per cubic meter	MIIR - maximally impacted institutiona
ASIL = acceptable source impact level	MIRR = maximally impacted residential
Hr = hour	NO ₂ = nitrogen dioxide
MIBR = maximally impacted boundary receptor location	PUD = Public Utility District
MICR = maximally impacted commercial receptor location	Yr = year

	Units = µg/m ³		
Source	MIBR/MICR	MIRR	
H5 Data Center	0.19	0.032	
Local Background ^a	0.11	0.11	
Cumulative Impact	0.31	0.15	

	MIBR/MICR	MIRR	
DEEP Cancer Risk Unit Risk Factor (µg/m ³) ⁻¹	38	300	
	Units = per million		
H5 Data Center	7.3	9.6	
Local Background	4.4	34	
Cumulative Risk	12	44	

Notes:

^a Local background values determined from Ecology's Quincy Storymap of DEEP concentrations.

Abbreviations and Acronyms:

 $\mu g/m^3$ = micrograms per cubic meter

DEEP = diesel engine exhaust particulate matter

MIBR = maximally impacted boundary receptor location

MICR = maximally impacted commercial receptor location

MIIR = maximally impacted institutional receptor location

MIRR = maximally impacted residential receptor location

NATA = National Air Toxics Assessment Database

Lifetime Cancer Risk Associated with Project-Related Emissions of Carcinogenic Compounds

H5 Data Center

Quincy, Washington

			Estimated Increased Cancer Risk			
	Annual Emissions		at Key Risk Receptor Locations (per Million			
Carcinogen	(TPY)	ASIL (µg/m³)	MIBR/MICR	MIRR		
Diesel Engine Emissions	Diesel Engine Emissions					
DEEP	0.68	0.0033	7.3	9.6		
1,3-Butadiene	1.2E-03	0.033	1.3E-03	0.0017		
Acetaldehyde	7.8E-04	0.37	7.5E-05	9.8E-05		
Benz(a)anthracene	1.9E-05	0.0055	1.2E-04	1.6E-04		
Benzene	0.024	0.13	6.5E-03	8.6E-03		
Benzo(a)pyrene	7.9E-06	0.0010	2.8E-04	3.7E-04		
Benzo(b)fluoranthene	3.4E-05	0.0055	2.2E-04	2.9E-04		
Benzo(k)fluoranthene	6.7E-06	0.0055	4.3E-05	5.7E-05		
Chrysene	4.7E-05	0.055	3.0E-05	4.0E-05		
Dibenz(a,h)anthracene	1.1E-05	0.0005	7.6E-04	9.9E-04		
Formaldehyde	2.4E-03	0.17	5.1E-04	6.7E-04		
Indeno(1,2,3-cd)pyrene	1.3E-05	0.0055	8.2E-05	1.1E-04		
Naphthalene	4.0E-03	0.029	4.9E-03	6.44E-03		
Toluene	0.0086	5,000	6.1E-08	8.1E-08		
Xylenes	0.0059	220	9.6E-07	1.3E-06		
Combined Increased Cano	Combined Increased Cancer Risk 7.3 9.6					

Notes:

^a Estimated cancer risk was calculated based on modeled DEEP cancer risk.

Abbreviations and Acronyms:

 $\mu g/m^3$ = micrograms per cubic meter

ASIL = acceptable source impact level

DEEP = diesel engine exhaust particulate matter

MIBR = maximally impacted boundary receptor location

MICR = maximally impacted commercial receptor location

MIRR = maximally impacted residential receptor location

TPY = tons per year

Table 7-1

Qualitative Summary of the Effects of Uncertainty on Quantitative Estimates of Health Risk H5 Data Center Quincy, 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
AERMOD air modeling methods	Possible underestimate of average long-term ambient air concentrations and overestimate of short-term ambient air concentrations
Toxicity of DEEP at low concentrations	Possible overestimate of cancer risk, possible underestimate of non- cancer hazard for sensitive individuals
Toxicity of NO_2 at low concentrations	Possible overestimate of non-cancer hazard for sensitive individuals

Abbreviations and Acronyms:

AERMOD = American Meteorological Society (AMS)/US Environmental Protection Agency (EPA)

regulatory model

DEEP = diesel engine exhaust particulate matter

NO₂ = nitrogen dioxide

APPENDIX A

Electronic Files (on DVD)