

**April 2016 Second-Tier Health Impact
Assessment for Diesel Engine Exhaust
Particulate Matter and Nitrogen Dioxide
(Response to Incompleteness)
Microsoft Oxford Data Center
Quincy, Washington**

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Prepared for

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LIST OF ABBREVIATIONS AND ACRONYMS

$\mu\text{g}/\text{m}^3$	microgram per cubic meter
$\mu\text{g}/\text{m}^3$	microgram per cubic meter
μm	micrometer
AERMOD	American Meteorological Society/EPA Regulatory Model
ASIL	acceptable source impact level
BACT	best available control technology
CO	carbon monoxide
DEEP	diesel engine exhaust particulate matter
DPF	diesel particulate filter
Ecology	Washington State Department of Ecology
EPA	US Environmental Protection Agency
ft.	feet
g/kWm-hr	grams per mechanical kilowatt-hour
g/m^3	grams per cubic meter
HI	hazard index
HIA	health impacts assessment
HQ	hazard quotient
m	meter
MIBR	maximally impacted boundary receptor
MICR	maximally impacted commercial receptor
MIRR	maximally impacted residential receptor
MW	megawatt
NAAQS	National Ambient Air Quality Standards
NO	nitric oxide
NO ₂	nitrogen dioxide
NOC	Notice of Construction
NO _x	nitrogen oxides
OEHHA	California Office of Environmental Health Hazard Assessment
PAH	polycyclic aromatic hydrocarbon
PM	particulate matter
PM _{2.5}	particulate matter with an aerodynamic diameter less than or equal to 2.5 microns
PUD	Grant County Public Utility District
REL	reference exposure level
RfC	reference concentration
SCR	selective catalytic reduction
SQER	small-quantity emission rate
SR	State Route
TAP	toxic air pollutant
tBACT	best available control technology for toxics
URF	unit risk factor
VOC	volatile organic compound
WAC	Washington Administrative Code

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1.0 EXECUTIVE SUMMARY

1.1 Summary of Changes from Previous January 2016 Report

This revised report updates the previous version submitted in January 2016, to respond to Washington State Department of Ecology (Ecology) comments. The only numerical changes in this document are in Tables 2-1, 4-2, 4-6, and 4-9. The following items have been revised but, as described in this report, the revisions did not affect the conclusions regarding health risks:

- The theoretical maximum annual emission rate and ambient concentration estimates for diesel engine exhaust particulate matter (DEEP) were increased by 8 percent.
- The theoretical maximum annual emission rate and ambient concentration estimates for gaseous pollutants including ammonia were increased by 18 percent.

The following items have not been changed for this revised analysis:

- The estimates for 70-year average emissions, 70-year average ambient concentrations, and cancer risks for DEEP have not been revised.
- The estimates for maximum 1-hour emissions and ambient concentrations of nitrogen oxides (NO_x) and nitrogen dioxide (NO₂) have not been revised.
- The maximum daily emissions and ambient concentrations of gaseous pollutants including ammonia have not been revised.

1.2 Proposed Project

Microsoft Inc. (Microsoft) is currently permitted to construct and operate the Oxford Data Center (Oxford) in Quincy, Washington. Ecology issued Approval Order No. 14AQ-E537 in August 2014 to authorize the installation of 32 2.5-megawatt (MW) emergency diesel engine generators, four 2.0-MW generators, one 0.75-MW emergency diesel engine generator, and 32 cooling towers. All of the engines must be US Environmental Protection Agency (EPA) Tier 2 engines equipped with engine exhaust add-on air pollution control devices designed to achieve the stringent emission standards set by the EPA for Tier 4 (Final) certification. The add-on air pollution controls for each engine will include catalyzed diesel particulate filters (DPFs) and selective catalytic reduction (SCR) for the removal of NO_x. To support issuance of the Approval Order, the modeled ambient concentrations of DEEP exceeded the acceptable source impact level (ASIL), but the modeled ambient concentrations for all other toxic air pollutants (TAPs) were less than the ASILs. Therefore, Ecology prepared a Second-Tier health impact review for DEEP in May 2015 (Ecology 2015).

Microsoft submitted an application to Ecology in January 2016 to revise the current Approval Order (Landau Associates 2016). Microsoft proposes the following general changes:

- Install shorter stacks for the 2.5-MW generators to address back pressure issues for the generators
- Install eight new 2.5-MW reserve emergency generators

- Expand the allowable range of allowable generator loads, allowing each generator to operate at any load between idle (zero electrical load) to 100 percent
- Set a new facility-wide 1-hour emission limit for NO_x of 575 pounds per hour, applicable during the first hour of a facility-wide power outage under cold-start conditions.

Details on the emission rates associated with these changes are presented in Microsoft's Approval Order Revision Letter (Landau Associates 2016). These proposed changes would increase the emission rates for some pollutants, and would cause the modeled ambient concentrations of two TAPs (DEEP and NO₂) to exceed their ASILs. Therefore, Microsoft has prepared this new second-tier HIA report for both DEEP and NO₂.

1.3 Health Impacts Evaluation

This HIA evaluates the "zero baseline" cumulative risks caused by the aggregate emissions from the entire Oxford Data Center after implementation of Microsoft's proposed changes, and not just the impacts caused by incremental emission increases above the levels permitted by the current August 2014 Approval Order. This HIA demonstrates that the ambient cancer risks and non-cancer risks caused by emissions of DEEP and NO₂ from the proposed project meet Ecology's approval criteria.

Ecology has implemented a community-wide approach to evaluating health impacts from Quincy data centers because the engines are within close proximity to other background sources of DEEP. As part of the community-wide approach, this second-tier HIA considers the cumulative impacts from the proposed project along with existing nearby permitted sources and other local background sources, including State Route (SR) 28, 281, and the adjacent railroad line.

Under worst-case exposure assumptions involving residents standing outside their homes for 70 continuous years, DEEP emissions from the proposed project have the potential to increase cancer risk up to 5.9 in 1 million (5.9×10^{-6}) at the maximally impacted residential receptor (MIRR) location. The proposed project is approvable under Washington Administrative Code (WAC) 173-460-090 because the project-related increase in cancer risk is expected to be less than the maximum risk allowed by a second-tier review, which is 10 in 1 million (10×10^{-6}).

Based on the maximum cumulative DEEP concentration estimated at the MIRR in the modeling domain (a house 1.5 miles southeast of the project site, adjacent to SR 281), the maximum cumulative cancer risk (from local DEEP emissions including background sources within the vicinity) is 68 in 1 million (68×10^{-6}). Of this, only a small fraction (only 0.77 percent) of the ambient DEEP impact is attributable to Oxford Data Center emissions and most of the DEEP at this location is from traffic emissions from SR 28 and 281.

This HIA also evaluated the potential for chronic non-cancer health risks from local exposure to project-related emissions as quantified by the magnitude of the pollutant-specific hazard quotient (HQ). The acceptability for annual-average chronic health risks is identified by an annual-average HQ

less than 1. This HIA demonstrates that all receptors chronically exposed to cumulative ambient concentrations of DEEP and all other chronic TAPs are predicted to encounter cumulative chronic HQs much lower than 1, even under the unlikely but theoretically-possible worst-case scenario where the aggregated emissions allowed by the 3-year rolling average permit limit are released entirely within a single year. Therefore, this HIA concludes that the emissions of DEEP and all other chronic air pollutants will not cause unacceptable chronic health risks.

For NO₂ and other pollutants subject to acute (1-hour) toxicity thresholds, acceptability is defined either as a cumulative HQ less than 1, or low frequency of expected occurrence for the acute HQ to exceed 1, by cumulative impacts. For the project-only emissions, the modeled cumulative acute HQ for impacts of NO₂ and other TAPs is 1.4 at an area of unoccupied industrial land adjacent to the Microsoft facility boundary; this indicates the potential for acute health impacts inside that small, unoccupied area. However, the zones with modeled acute HQs exceeding 1 do not extend to any offsite homes or commercial buildings. Furthermore, the predicted frequency of occurrence for an acute HQ exceeding 1 at the unoccupied property line is very low (a recurrence interval of 1,922 years). Therefore, this HIA concludes that the project-only emissions of NO₂ (and combined project emissions of TAPs that may have acute health impacts) are not expected to cause unacceptable acute health risks.

The Oxford Data Center is within 1 mile of existing background NO_x emission sources including two existing data centers, state highways, and a rail line. Ambient dispersion modeling of the existing background sources show elevated background NO₂ concentrations that exceed the acute reference exposure level (REL) for NO₂ at Mountain View School and residential properties east of the existing data centers. The proposed project emissions will have a small impact at those locations. The project-only NO₂ impacts at the maximum cumulatively impacted receptor is 207 µg/m³, which is well below the ASIL for NO₂ (470 µg/m³) and only 18 percent of the total cumulative impact at that location.

1.4 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.

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2.0 MICROSOFT OXFORD DATA CENTER PROJECT

2.1 Description of Proposed Development

Microsoft is currently permitted by Approval Order No. 14AQ-E537 to develop the Oxford Data Center in Quincy, Washington (Figure 2-1). The Oxford Data Center site layout, the locations of the currently-permitted 37 primary emergency generators and cooling towers, and the proposed locations of the 8 new reserve emergency generators are shown on Figure 2-2.

During a power outage, the currently-permitted 2.5-MW and 2.0-MW primary emergency generators will be used to provide electricity to the servers and the 0.75-MW backup diesel engine generator will be used for building safety (i.e., lights and appliances) at the administration building. The eight new 2.5-MW reserve generators will be “backups to the backups,” and will provide electricity to the servers only if one of the primary generators malfunctions. All of the generators will require runtime for scheduled testing and maintenance.

The ambient air impacts associated with installation of the 32 cooling towers are discussed in the original Notice of Construction (NOC) air permit application and Supporting Information Report (Landau Associates 2014). The requested revisions do not increase the currently permitted limits related to the cooling towers and Microsoft will continue to meet those limits. Therefore, no further discussion of the cooling towers is necessary.

2.2 Forecast Emission Rates

Air pollutant emission rates were calculated, in accordance with WAC 173-460-050, for the emission sources described in Section 2.1. Emission rates were quantified for criteria pollutants and TAPs. For a detailed description of the methods used to calculate project emission rates, refer to Microsoft’s April 2016 application package (Landau Associates 2016). Microsoft’s proposed permit modifications will cause the emission rates for DEEP and NO₂ to increase to the following levels:

- DEEP (70-year average for cancer risk calculations): 0.814 tons/year
- DEEP (theoretical maximum annual for chronic non-cancer risk calculations): 2.6 tons/year
- NO₂ (initial cold-start during facility-wide power outage): 57.5 lbs/hour.

The emission estimates presented in this report have been calculated for generators that meet EPA Tier 4 emission limits. Load-specific emission rates were developed from generator manufacturer estimates of “Not to Exceed” and “Potential Site Variation” emissions data for nitrogen oxides (NO_x), particulate matter (PM), carbon monoxide (CO), and total volatile organic compounds (VOCs). An estimate of the “back-half” condensable fraction of the emitted PM was included for calculating DEEP emission rates and DEEP cancer risks. For the TAPs other than DEEP, emission factors from the EPA’s Compilation of Air Pollutant Emission Factors (AP-42), Sections 3.3 and 3.4 were used (EPA 1995). Table 2-1 summarizes the calculated facility-wide emission rates for the proposed project. The

following TAPs exhibit emission rates exceeding the SQERs: DEEP, NO₂, CO, ammonia, benzene, and acrolein.

These estimated emission rates are for cold-start operating conditions, which assumes that a brief “black puff” occurs during each cold start, using the same methodology that was used for previous data center permit applications. The “black puff” factor is based on measurements taken by the California Energy Commission as described in its 2005 document, Air Quality Implications of Backup Emergency Generators in California (Lents et al. 2005). Cold-start emission rates also account for the delay period before the emission control catalysts reach their activation temperatures.

2.3 Land Use and Zoning

The topography in the vicinity of the Oxford site is relatively flat with elevations ranging between 1,300 and 1,400 feet (ft) above sea level. The zoning designation for the site is City of Quincy City Industrial. Zoning designations on adjacent lands include Grant County agricultural to the north, northwest, and far south. City of Quincy City residential/business and Grant County commercial/industrial and residential zones are to the east and southeast of the project site (Grant County website 2015; City of Quincy 2011).

2.4 Sensitive Receptors

A land use zoning map for the project vicinity is provided on Figure 2-3, where locations of commercial, residential, and institutional receptors of interest are also indicated. These receptors of interest are identified and summarized in Table 2-2. From a health impact standpoint, six institutional facilities (schools/medical facilities) are within the vicinity where project emissions may impact the property. These facilities have the potential for ambient exposure to persons with weak or compromised immune systems, the elderly, children, or pregnant women.

The following sensitive receptor locations are within vicinity of proposed project and may be impacted by project-related emissions. Among these, the maximally impacted sensitive receptor was evaluated for potential carcinogenic and non-cancer health impacts:

- The nearest school is Monument Elementary School (I-1), approximately 1 mile southeast of the Oxford Data Center.
- The nearest daycare or preschool is a private home-based facility, approximately 0.6 miles southeast of the Oxford Data Center.
- The nearest church is located approximately 0.7 miles southeast of the Oxford Data Center.
- The nearest medical facility is Quincy Valley Medical Center (I-2), approximately 0.7 miles southeast of the Oxford Data Center.
- The nearest convalescent home is Cambridge, approximately 1 mile southeast of the Oxford Data Center.

3.0 PERMITTING REQUIREMENTS FOR NEW SOURCES OF TOXIC AIR POLLUTANTS

3.1 Overview of the Regulatory Process

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

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

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

As part of a second-tier petition, described in WAC 173-460-090, the applicant submits a site-specific HIA. The objective of an HIA is to quantify the increase in lifetime cancer risk for persons exposed to the project emissions of any carcinogen, and to identify the potential for adverse health hazards from exposures to any non-carcinogen that would result from the emissions of the proposed project. The potential for an adverse health hazard is identified based on the assumption of an effective threshold concentration. The estimated cancer risk is compared to the maximum allowable risk, under a second-tier review, which is an increase of 10 in 1 million (equivalent to the risk of 1 cancer case in 100,000 population). In evaluating a second-tier petition, background concentrations of the applicable TAPs must be considered. Ecology has not set any numerical limit on cumulative impacts (project + background).

For non-carcinogens such as NO₂, a similar path exists for second-tier risk review, but there is no specified numerical criterion. Instead, Ecology's regulations state, "Ecology may recommend approval of a project that is likely to cause an exceedance of acceptable source impact levels for one or more TAPs only if it determines ... that the non-cancer hazard is found to be acceptable." For previous HIAs for NO₂, Ecology has determined the non-cancer hazard to be acceptable if the non-cancer HQ at frequently-populated areas is less than 1, or if the frequency of occurrence for an HQ exceeding 1 is small.

If the emissions of any TAP are expected to result in an unacceptable increased cancer or non-cancer risk, then the applicant may request that Ecology conduct a third-tier review. However, as described

in this report the estimated cancer and non-cancer risks associated with the project emissions have been determined to be acceptable, so the project does not require a third-tier review.

3.2 BACT and tBACT for Oxford Data Center

Ecology is responsible for determining BACT and tBACT for controlling criteria pollutants and TAPs emitted from the proposed project. Microsoft has committed to equipping the proposed diesel engine generators with an integrated add-on control package designed to achieve the stringent emission standards set by the EPA for Tier 4 (Final) certification. The add-on controls for each diesel engine will include a catalyzed DPF for removal of particulate matter with an aerodynamic diameter less than or equal to 2.5 microns (PM_{2.5}), DEEP, and VOCs, and SCR for NO_x removal.

Microsoft conducted an updated BACT and tBACT analysis as presented in the April-2016 NOC Supporting Information Report (Landau Associates 2016). Based on the original 2014 BACT analysis, Ecology found in the 2014 Approval Order that BACT for criteria and toxic air pollutants is application of EPA Tier 2 controls. Approval Order No. 14AQ-E537, Determination No. 2. The more stringent Tier 4 controls required by the Approval Order will achieve a level of control significantly better than BACT.

3.3 First-Tier Toxics Screening Review for Oxford Data Center

The first-tier TAP assessment compares the project forecast emission rates to the SQERs and compares the maximum project ambient air impacts to the ASILs. For this analysis, Microsoft conducted a “zero baseline” first-tier risk review based on the aggregate emissions including the currently permitted emissions plus the emission increases caused by Microsoft’s proposed permit revisions.

Table 2-1 shows calculated emission rates for each TAP emitted from the proposed project, and compares the emission rates to the SQERs. The SQERs are emission thresholds, below which Ecology does not require an air quality impact assessment for the listed TAP. The maximum emission rates for DEEP, CO, ammonia, NO₂, benzene, and acrolein exceed their respective SQERs, so an ambient air impact assessment based on atmospheric dispersion modeling was required for those TAPs.

Ecology requires facilities to conduct a first-tier screening analysis for each TAP whose emissions exceed 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, then compare the modeled values to the ASILs (WAC 173-460-080). For this analysis, the theoretical maximum annual-average impacts were modeled based on a worst-case operational scenario where all of the permitted emissions during a 3-year rolling period occur in a single year. The maximum short-term impacts were modeled based on the worst-case assumption that the short-term emissions occur for 24 hours per day for 365 days per year for 5 years, with the American Meteorological Society/EPA Regulatory Model (AERMOD) set to automatically choose the 1st-highest event.

Table 2-1 presents the first-tier ambient air concentration screening analysis for each TAP whose emission rate exceeds its SQER. Details on the methodologies for the modeling are provided in the permit revision application (Landau Associates 2016). All of the modeled maximum impacts occur at the unoccupied facility boundary (i.e., locations where there are no current buildings). The modeled maximum annual-average DEEP impact from Oxford at the unoccupied facility boundary exceeds the ASIL and the modeled maximum 1-hour average NO₂ impact from Oxford exceeds the ASIL, while the impacts for all TAPs other than DEEP and NO₂ are less than their respective ASILs. Therefore, DEEP and NO₂ are the only TAPs that triggered the requirement for a second-tier HIA.

3.4 Second-Tier Review Processing Requirements

In order for Ecology to review the second-tier petition, each of the following regulatory requirements under 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 described in the preliminary NOC approval order represent at least tBACT.
- (c) The applicant has developed an HIA protocol that has been approved by Ecology.
- (d) The ambient impact of the emissions increase of each TAP that exceeds ASILs has been quantified using refined air dispersion modeling techniques as approved in the HIA protocol.
- (e) The second-tier review petition includes an HIA conducted in accordance with the approved HIA protocol.

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

3.5 Second-Tier Review Approval Criteria

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

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

The remainder of this document discusses the combined (DEEP and NO₂) HIA conducted by Landau Associates.

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4.0 HEALTH IMPACT ASSESSMENT

This HIA was conducted according to the requirements of WAC 173-460-090. The HIA addresses the public health risk associated with exposure to DEEP and NO₂ from Microsoft's proposed project and local background sources within the project vicinity. While this HIA is not a complete risk assessment, it generally follows the four steps of the standard health risk assessment approach proposed by the National Academy of Sciences (NAS 1983, 1994). These four steps are: 1) hazard identification; 2) exposure assessment; 3) dose-response assessment; and 4) risk characterization. As described later in this document, the HIA did not consider exposure pathways other than inhalation.

4.1 Hazard Identification

Hazard identification involves gathering and evaluating toxicity data on the types of health injury or disease that may be produced by a chemical, and on the conditions of exposure under which injury or disease is produced. It may also involve characterization of the behavior of a chemical within the body and the interactions it undergoes with organs, cells, or even parts of cells. This information may be of value in determining whether the forms of toxicity known to be produced by a chemical agent in one population group or in experimental settings are also likely to be produced in human population groups of interest. Note that risk is not assessed at this stage. Hazard identification is conducted to determine whether and to what degree it is scientifically correct to infer that toxic effects observed in one setting will occur in other settings (e.g., 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 project emissions of DEEP and NO₂, the toxicity of other TAPs with emission rates exceeding the SQERs was also reviewed in this HIA, to consider whether additive toxicological effects should be considered in the HIA.

4.1.1 Overview of DEEP Toxicity

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

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

The following health effects have been associated with exposure to 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 project emissions:

- 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 ambient concentrations of DEEP that may potentially impact humans within the project vicinity will be much lower than levels associated with many of the above-described health effects. For the purpose of determining whether project-related ambient impacts are acceptable, both the cancer risk and non-cancer health hazard will be quantified from DEEP impacts and will be evaluated in the remaining sections of this document.

4.1.2 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. 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) adverse 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 [$> 180,000$ grams per cubic meter (g/m³)] of NO₂ may result in serious effects including death (NAC AEGC Committee 2008). Moderate levels ($\sim 30,000$ g/m³) may severely irritate the eyes, nose, throat, and respiratory tract, and cause shortness of breath and extreme discomfort. Lower-level NO₂ exposure ($< 1,000$ g/m³), such as that experienced

near major roadways, or perhaps downwind from stationary sources of NO₂, may cause 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 website). For this project, the maximum short-term ambient NO₂ concentration has been estimated to be 859 µg/m³ (1-hour average).

Project and background emissions during an emergency power outage present the greatest potential for producing high enough acute concentrations of NO₂ to be of concern for susceptible individuals, such as people with asthma.

4.1.3 Overview of Toxicity for TAPs that exceed the SQER

Other TAPs (with emission rates exceeding the SQERs) that may contribute to a combined health hazard as a TAP, which (like DEEP and NO₂) impact the respiratory system, are: CO, ammonia, benzene, and acrolein. The applicability of these TAPs are summarized below.

- Carbon monoxide: The reference exposure level (REL) for CO considers toxic effects for the cardiovascular system (OEHHA website 2007), not the respiratory system; however, the ambient air impacts associated with CO emissions have been conservatively included in the project-specific hazard index calculated in this HIA.
- Ammonia: The reference concentration for ammonia considers toxic effects for the respiratory system (OEHHA website 2007); therefore, the ambient air impacts associated with ammonia emissions are included in the project-specific hazard index calculated in this HIA.
- Benzene: The REL for benzene considers toxic effects for reproductive development, the immune system, and the hematologic system (OEHHA website 2007), not the respiratory system; however, the ambient air impacts associated with benzene emissions have been conservatively included in the project-specific hazard index calculated in this HIA.
- Acrolein: The reference concentration for acrolein considers toxic effects for the eyes at 1-hour acute exposures. Toxic effects to the respiratory system are not anticipated for less than 8-hour acute exposures (OEHHA website 2007). This HIA examines 1-hour acute and annual chronic inhalation exposure scenarios; however, the ambient air impacts associated with acrolein emissions have been conservatively included in the project-specific hazard index (using the 1-hour acute REL) calculated in this HIA.

4.2 Exposure Assessment

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

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

4.2.1 Identifying Routes of Potential Exposure

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

DEEP consists of ultra-fine particles (approximately 0.1 to 1 micron in size) that behave like a gas and do not settle out of the downwind plume by gravity. DEEP particles will eventually be removed from the atmosphere and 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 project site. 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 project site. 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 major 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 Oxford emissions, only inhalation exposure to DEEP and NO₂ is evaluated.

4.2.2 Estimating Particulate Concentrations

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

Each of the proposed Oxford emergency generators was modeled as an individual discharge point. Additionally, local background DEEP and NO₂ contributions were modeled, including existing generators at the existing Oxford buildings, the existing Dell Data Center, the existing Columbia Data Center, diesel truck exhaust from SR 28 and 281, and locomotive emissions from the BNSF railroad line. Emission rates for the existing data centers were calculated based on the permitted maximum emission rates provided in the Ecology approval orders for those facilities. NO₂ and DEEP emission rates for SR 28, SR 281, and the railroad line were provided by Ecology (Dhammapala 2015a, b). Ecology developed highway emissions data using the EPA model MOVES, which incorporates Grant County-wide on-road diesel emissions exhaust data and highway-specific vehicle miles traveled. DEEP and NO₂ ambient air impacts from the proposed project and local background sources 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 (2001 to 2005).
- Twice-daily upper air data from Spokane, Washington (2001 to 2005) 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.
- The emissions for each proposed diesel engine were modeled with a 40-ft-tall vertical exhaust stack (except for the 35.5-ft-tall stack for the 0.75-MW engine). Existing engines at neighboring data centers were modeled at their permitted height of 58 ft above grade for the Dell Data Center and 21 to 38 ft (depending on the engine) for the Columbia Data Center.
- The dimensions of the existing and proposed buildings at the project site were included to account for building downwash.
- The receptor grid for the AERMOD modeling domain at or beyond the facility boundary was established using a variable Cartesian grid:
 - 12.5-meter (m) spacing from 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 6,000-m maximum extent).

4.2.3 Identifying Potentially Exposed Receptors

Typically, Ecology evaluates potential exposures at maximally impacted boundary, residential, and business/commercial areas to capture worst-case exposure scenarios. This evaluation additionally considered potential exposures at sensitive receptor locations, such as schools and medical centers.

Several different land use types are within the general vicinity of the project site, residential, commercial, and institutional locations, where people may be exposed to project-related emissions. These are shown on Figure 2-3, and Table 2-2 characterizes the potential risk receptors (of different use types), the direction, and approximate distances of each receptor location from the nearest project emission source. The project-related impacts at those receptor locations have been estimated and the maximally impacted residential, commercial, and institutional receptor locations have been identified (based on ambient dispersion modeling results). These, along with the maximum project-related impacted receptor locations, are listed in Table 4-1.

4.2.3.1 Receptors Maximally Exposed to DEEP from Project Emissions

Table 4-1 details the maximally impacted risk receptor locations for project-related DEEP impacts. Figure 4-1 is an AERMOD isopleth, which shows a color-coded concentration contour map of estimated maximum annual DEEP concentrations attributable solely to project emissions and identifies the estimated ambient air impacts of DEEP at each of the maximally exposed receptor locations. The concentrations at the maximally impacted boundary receptor (MIBR), maximally impacted residential receptor (MIRR), and maximally impacted commercial receptor (MICR) are shown on Figure 4-1. The modeling indicates that emissions from the proposed project can be expected to extend to multiple existing residences and that ambient air impacts are anticipated at a level that exceeds the ASIL ($0.00333 \mu\text{g}/\text{m}^3$ of DEEP). On Figure 4-1, the purple contour represents this concentration boundary. All receptors located beyond the purple contour are forecast to have concentration impacts less than the ASIL.

4.2.3.2 Receptors Maximally Exposed to NO₂ from Project Emissions

Table 4-1 also details the maximally impacted receptor locations for project-related NO₂ impacts and Figure 4-2 is the AERMOD isopleth, showing the maximum 1-hour NO₂ concentration contours from Project project-only emissions. This figure shows the magnitude of the NO₂ ambient impacts during the first hour of cold-start NO_x emissions during a facility-wide power outage. Note that the characteristic MIBR, MIRR, and MICR (based on NO₂ impacts) differ from those from DEEP emissions. The modeling indicates that project-only NO₂ impacts, which exceed the ASIL would be limited to small, unpopulated areas adjacent to the industrially zoned facility boundary. The blue contour on Figure 4-2 represents the NO₂ ASIL ($470 \mu\text{g}/\text{m}^3$) and receptors located beyond this contour are forecast to have ambient impacts at concentrations less than the ASIL. The blue contours do not extend to any offsite buildings.

4.2.4 Exposure Frequency and Duration

The likelihood that someone would be exposed to project emissions of DEEP and NO₂ depends on local wind dispersion patterns, the frequency of engine operation, and how much time people spend in the immediate vicinity. As discussed previously, the air dispersion model (AERMOD) uses emission rate estimates and historical meteorological data (along with other simplifying assumptions) to predict ambient DEEP and NO₂ concentrations within the project vicinity. However, people are more

likely to be exposed frequently and for a longer duration at residential receptor locations because they spend much more time at home than at commercial/business or institutional receptor locations.

Therefore, this analysis uses simplified assumptions about receptors' exposure frequency and duration and considers the land use surrounding the proposed project to estimate the amount of time persons at that receptor location may be exposed. This evaluation assumes that people at residential receptor locations may potentially be continuously exposed, as if they never leave their place of residence. These behaviors are not typical; however, the assumptions are intended to avoid underestimating exposure so that public health protection is ensured. Workplace and other non-residential receptor locations were also adjusted for potential exposure durations based on the amount of time people can be expected to stay at the receptor location. These exposure durations are more predictable for commercial/business zones than time spent at their homes and are further described in Section 4.4.2, where cancer risk is quantified for intermittent exposures to DEEP.

4.2.5 Background Exposure to Pollutants of Concern

WAC 173-460-090 states, "Background concentrations of TAPs will be considered as part of a second-tier review." The word "background" is often used to describe exposures to chemicals that come from existing sources, or sources other than those being assessed. Regional background DEEP and NO₂ concentrations from the EPA's National Air Toxics Assessment database were not used in this evaluation because Ecology has concluded that site-specific evaluation of local highways and railroad lines provides a more realistic spatial determination of regional background concentrations.

To estimate DEEP and NO₂ background concentrations, ambient air impacts from SR 28 and 281, the railroad line, and the neighboring Columbia and Dell data centers were evaluated as "cumulative impacts" using the methodology described in Section 4.2.2.

4.2.6 Cumulative Exposure to DEEP in Quincy

Figure 4-3 shows the cumulative concentration contours of DEEP within the project vicinity. The cancer risk for project-only emissions is based on the 70-year average DEEP emissions including the condensable front-half. This figure includes the project emissions and local background sources of DEEP, including nearby highways and the railroad line. The maximum cumulative impact (based on the annual average), at any residence within the modeling domain is predicted to be 0.23 µg/m³ (approximately 70 times greater than the DEEP ASIL). This is estimated to occur approximately 1.5 miles southeast of the proposed project (at receptor location R-2, which is adjacent to SR 281).

Table 4-2 shows the estimated cumulative DEEP impact in the project vicinity. The non-cancer HQ for project-only emissions is based on the theoretical maximum emission rate (the permitted emissions during a 3-year rolling period occur in a single year), while the cancer risk for the project emissions is based on the 70-year average DEEP emissions including the condensable front-half. Table 4-2 shows that at the maximum cumulatively impacted residence within the modeling domain, 86 percent of the DEEP exposure is due to traffic emissions from nearby SR 281, and only 2 percent of the DEEP

exposure would be attributable to the proposed project. It is also important to note that the predicted ambient concentrations of DEEP are based on maximum allowable (permitted) emissions instead of actual emissions from nearby facilities. Actual emissions are likely to be lower than what the facilities are permitted for, but worst-case emissions are evaluated here in order to avoid underestimating potential cumulative DEEP exposure.

4.2.7 Cumulative Exposure to NO₂ in Quincy

A similar methodology, as described in Section 4.2.6, was used to estimate the cumulative short-term (1-hour) NO₂ impacts based on a city-wide power outage scenario. The purpose of this effort was to identify worst-case acute exposure scenarios in the event of a system-wide power outage where all neighboring emergency diesel generators would be active. Table 4-3 shows the estimated cumulative NO₂ concentrations at maximally impacted receptor locations, based on allowable emissions from the proposed project and other background sources. This model:

- Assumed that the emissions from the Oxford Data Center will occur during the first hour of a facility-wide power outage, when the generators operate under cold-start conditions
- Assumed simultaneous outage emissions from the Columbia and Dell data center generators for the hour of maximum impact
- Assumed engine operation at loads specified in permits for emergency power outages
- Included local emissions from nearby SR 28, SR 281, and the railroad line.

Figure 4-4 shows the magnitude and frequency of occurrence of city-wide cumulative NO₂ impacts. Modeling results indicate that the worst-case scenarios may result in concentrations greater than the acute NO₂ REL at the Mountain View School, existing homes, and vacant lands (residentially zoned) east of the existing Columbia Data Center and Dell Data Center. Most of the modeled cumulative NO₂ impacts at those eastern receptor locations are attributed to emissions from the Columbia and Dell data centers. The maximum cumulatively impacted residential receptor (adjacent east to the Columbia Data Center) is predicted to reach an ambient concentration of 1,129 µg/m³, which exceeds the NO₂ ASIL (470 µg/m³). However, the Oxford-only ambient impact at that location is only 207 µg/m³ and a small fraction (18 percent) of the total NO₂ impact. The frequency with which these impacts may occur is discussed further in Section 4.4.1.4.

4.3 Dose-Response Assessment

Dose-response assessment describes the quantitative relationship between the amounts of exposure to a substance (the ambient concentrations as “the dose”) and the incidence or occurrence of injury (adverse health impacts as “the response”). The assessment necessitates establishing a toxicity value or criterion to use in evaluating potential health risk. Exposure assumptions and risk factors used to calculate lifetime cancer risk are summarized in Table 4-4, and Table 4-5 shows non-cancer and carcinogenic unit risk toxicity values for those pollutants expected to be emitted from the project at rates that exceed their respective SQERs.

4.3.1 Dose-Response Assessment for DEEP

The EPA and California Office of Environmental Health Hazard Assessment (OEHHA) have developed toxicological values for DEEP (EPA 2002; EPA website 2015; CalEPA 1998). These toxicological values are derived from studies on animals that were exposed to a known amount (ambient concentrations) of DEEP, or from epidemiological studies of incidental human exposure. These values are used in this evaluation 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 inhalation reference concentration (RfC) and the OEHHA's REL for diesel engine exhaust (measured as DEEP) was derived from dose-response data on inflammation and changes in the lung from rat inhalation studies. Each agency established the same level of $5 \mu\text{g}/\text{m}^3$ as the maximum long-term inhalation exposure of DEEP at which adverse (non-cancer) health effects are not expected to occur.

National Ambient Air Quality Standards (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.

The 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 that substance at a concentration of $1 \mu\text{g}/\text{m}^3$, and are expressed in units of inverse concentration [i.e., $(\mu\text{g}/\text{m}^3)^{-1}$]. The OEHHA's URF for DEEP is $0.0003 (\mu\text{g}/\text{m}^3)^{-1}$, meaning that a lifetime of exposure to $1 \mu\text{g}/\text{m}^3$ of DEEP could result in an increased individual cancer risk of 0.03 percent or a population risk of 300 excess cancer cases per million population exposure.

4.3.2 Dose-Response Assessment for NO₂

The OEHHA has developed an acute (1-hour) REL for NO₂ based on inhalation studies of asthmatics exposed to NO₂. These studies found that some asthmatics exposed to approximately 0.25 parts per million (i.e., $470 \mu\text{g}/\text{m}^3$) experienced increased airway reactivity following inhalation of NO₂ (CalEPA 1998). Not all asthmatic subjects experienced an effect.

No uncertainty factor adjustment was used in the development of OEHHA's acute REL for NO₂, and therefore the REL does not provide any additional buffer between the derived value and the exposure concentration at which effects were observed in sensitive populations. This implies that exposure to NO₂ at levels equivalent to the acute REL (which is equivalent to Ecology's NO₂ ASIL) could result in increased airway reactivity in a subset of asthmatics and sensitive subgroups. People without asthma or other respiratory disease are not expected to experience adverse health effects from NO₂ exposure at or below the REL.

4.4 Risk Characterization

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

4.4.1 Evaluating Non-Cancer Hazards

This assessment evaluates adverse health effects from DEEP and NO₂ exposure. Because the pollutants of interest present exposure responses based on annual and 1-hour exposure scenarios, the potential non-cancer health effects were evaluated based on the following:

- The theoretical maximum 12-month emission rate, which assumes the worst-case scenario that the 3-year rolling average permit limit is released entirely within a single year, was used in evaluating chronic (non-cancer) health hazards from exposure to DEEP and other TAPs expected to be emitted at rate that exceed their SQER. These theoretical maximum year impacts were revised for this April-2016 report.
- The acute (non-cancer, 1-hour average) health assessment was based on the peak hourly emission impacts of NO₂ and other TAPs expected to be emitted at rates that exceed their SQERs.

Table 4-5 lists the non-cancer toxicological values that were used for this assessment.

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). If a concentration exceeds the RfC, minimal risk level, or REL, this indicates only the potential for adverse health effects. The magnitude of this potential can be inferred from the degree to which this value is exceeded. This comparison is known as a hazard quotient (HQ) and is given by the equation below:

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

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

4.4.1.1 Hazard Quotient – DEEP

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

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

HQs were calculated for the maximally exposed residential, workplace, and sensitive receptor locations. 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, the OEHHA does not employ this practice. For the purpose of this evaluation, an RfC or REL of $5 \mu\text{g}/\text{m}^3$ was used as the chronic risk-based concentration for all scenarios where receptors could be exposed frequently (e.g., residences, work places, or schools).

Tables 4-2 and 4-6 show chronic HQs attributable to project DEEP emission at the maximally exposed receptor locations. Table 4-2 quantifies the cumulative hazard index, based on combined emissions from local background DEEP sources and the theoretical maximum emission impacts from the proposed project. Table 4-6 quantifies the combined hazard index of all TAPs expected to be emitted at rates that exceed the SQER (theoretical maximum emission rate). As shown, all hazard quotients and hazard indices calculated from chronic exposure of the theoretical maximum emission impacts are well below the threshold of 1 at each risk receptor location. This concludes that non-cancer health effects are not likely to result from chronic exposure to project-related emissions of DEEP.

4.4.1.2 Hazard Quotient – NO₂

To evaluate possible non-cancer health effects from exposure to NO₂, modeled concentrations at receptor locations were compared to their respective non-cancer toxicological values. In this case, maximum-modeled 1-hour NO₂ concentrations were compared to the acute REL ($470 \mu\text{g}/\text{m}^3$). The acute HQ for NO₂ exposure was calculated using the following equation:

$$\text{Acute HQ} = \frac{\text{peak 1 – hour NO}_2 \text{ emission impact}}{470 \mu\text{g}/\text{m}^3}$$

Tables 4-3 and 4-7 show acute HQs attributable to project NO₂ emission at the maximally exposed receptor locations. Table 4-2 quantifies the cumulative hazard index, based on combined emissions from local background NO₂ sources. Table 4-6 quantifies the combined hazard index of all TAPs expected to be emitted at rates that exceed the SQER.

Given that the acute REL for NO₂ does not provide any additional buffer between the derived value and the exposure concentration at which adverse health effects have been observed in sensitive populations, emissions exposure to individuals at these locations who have asthma or other respiratory illness are likely to experience increased airway reactivity and respiratory symptoms when the following two independent, intermittent events occur, when an emergency power outage occurs to activate all of the diesel generators, and when exceptionally poor meteorological conditions cause

characteristic dispersion patterns. However, that combination of two worst-case conditions is modeled to occur very infrequently, as described in Section 4.4.1.4.

4.4.1.3 Combined Hazard Quotient for All TAPs Whose Emission Rates Exceed Their SQERs

The non-cancer health impacts were evaluated based on the conservatively high emission rates. Based on these emission rate estimates, six TAPs (DEEP, CO, ammonia, NO₂, benzene, and acrolein) are expected to be emitted by the proposed project at levels that exceed their respective SQERs and, therefore, are included in this evaluation. Tables 4-6 and 4-7 show the modeled ambient concentrations, risk-based concentrations, and corresponding HQs for each risk receptor location.

The chronic combined hazard index (HI) is the sum of the HQs calculated for DEEP, ammonia, benzene, and acrolein because these pollutants each present a chronic health hazard. The acute HI was calculated for each risk receptor location and accounts for those TAPs that present an acute (1-hour) health hazard (CO, ammonia, NO₂, benzene, and acrolein). As shown in Table 4-6, the combined HI calculated for chronic exposure was modeled to be less than 1 for each risk receptor location. If the chronic HI is less than 1, then the chronic non-cancer risk is considered acceptable.

Table 4-7 shows the acute combined HI at each risk receptor and indicates an exceedance of 1 at both the MIBR and MICR. Note that the acute HQ of NO₂ expected at the unoccupied land of the MIBR, is greater than 1, and the MICR is very near to the threshold (HQ_{MICR} = 0.97) and, if one considers the combined HI for all pollutants except for NO₂ (as shown in Table 4-8), the HIs are well below 1. This shows that the combined emissions of CO, ammonia, benzene, and acrolein from the proposed project are not significant and that acute adverse health effects from emissions of these TAPs are unlikely to occur even under worst-case conditions at maximally impacted receptor locations. Section 4.4.1.4 discusses the probability of NO₂ ambient impacts exceeding the REL (indicated by an HQ greater than or equal to 1).

4.4.1.4 Probability Analysis of NO₂ REL Exceedances

This section demonstrates that the worst-case NO₂ impact is expected to exceed the REL very infrequently at all modeling receptor locations, including the unpopulated vacant land adjacent to the Oxford Data Center, and at the school, homes, and residentially zoned vacant land east of the Dell and Columbia data centers.

Methodology to Calculate Recurrence Interval of an NO₂ REL Exceedance

At any receptor location, an adverse acute NO₂ risk is defined as an ambient NO₂ concentration exceeding 470 µg/m³. Since the regional background NO₂ concentration is 16 µg/m³, an acute NO₂ risk would occur if the cumulative NO₂ concentration caused by the data centers exceeds 454 µg/m³ (470 – 16 = 454). At any given receptor location, this could occur only if three worst-case events occur simultaneously:

- The wind blows in a direction from the data center generators to the receptor location.
- Atmospheric dispersion conditions are exceptionally poor.
- A city-wide power outage occurs, activating all emergency generators at the local data centers.

The frequency of occurrence of the first two conditions listed above (wind direction directly toward the receptor location, during exceptionally poor dispersion conditions) was calculated from AERMOD modeling data. Landau Associates used AERMOD to estimate the frequency (number of hours) that exceptionally poor meteorological conditions would cause characteristic dispersion patterns to result in an NO₂ concentration greater than 454 µg/m³¹ at the modeling receptor location. This “count” of hours of exceedances of 454 µg/m³ (if emergency power outage operations were to occur all day and every day year-round for 5 straight years) is listed on Figures 4-2 and Figure 4-4 for both project-only and cumulative impacts, respectively.

The annual frequency of power outages was calculated from historical records of power outages in Quincy. In reality, according to the Grant County Public Utility District (PUD), the actual average total outage time per year, from 2008 to 2014, for customers who experienced an outage throughout PUD’s service area was only about 152 minutes per year or 2.5 hours per year (Grant County PUD 2015). However, to provide an alternate worst-case analysis an upper-bound frequency of power outages was also set to an assumed value of 24 hours per year. That upper-bound power outage value was originally used by Microsoft in the 2014 air quality application to estimate worst-case annual DEEP emissions.

Landau Associates conducted an analysis of the duration of each event exceeding 454 µg/m³ at the MIBR, and the time intervals between those exceedance events. The results were as follows:

- | | |
|---|--------|
| • Number of AERMOD modeled hours: | 43,825 |
| • Number of hours in 5 years exceeding 454 µg/m ³ : | 14 |
| • Number of events with 2 or more sequential hours of NO ₂ > 454 µg/m ³ : | 0 |

This ambient air impact simulation demonstrates that a REL exceedance at the MIBR is not expected to last for more than 1 hour, even if the generators were to operate continuously for 5 years.

Similarly at the MICR, the duration of each event exceeding 454 µg/m³ and the time intervals between those exceedance events were:

- | | |
|---|---|
| • Number of hours in 5 years exceeding 454 µg/m ³ : | 1 |
| • Number of events with 2 or more sequential hours of NO ₂ > 454 µg/m ³ : | 0 |

¹ Although the NO₂ level of interest is 470 µg/m³, concentrations that exceed 454 µg/m³ are noteworthy because Ecology estimates that a prevailing NO₂ concentration of 16 µg/m³ could exist in Quincy at any given time (WSU website 2015).

To calculate the frequency of occurrence of an NO₂ impact exceeding 454 µg/m³, Landau Associates used the following steps for each maximally impacted receptor location:

- Calculated 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 454 µg/m³, assuming the power outage occurs continuously during the 5-year period. This frequency was calculated by the 5-year “count” (AERMOD modeled hours of exceedance in 5 years) divided by 43,800 (modeling hours in 5 years).
- Calculated the hourly probability of occurrence of a power outage based on an “average case” of 152 minutes of outage per year based on PUD data from 2008 to 2014, and an upper-bound case of 24 hours of outage every year based on Microsoft’s previous upper-bound estimate in the 2014 application.
- Calculated the joint probability of those two independent events happening simultaneously and converted the joint probability to an annual recurrence interval.

Table 4-8 summarizes the probability that the modeled values exceed 454 µg/m³ for the worst-case assumption of 24 hours/year of power outage and the average-case assumption of 152 minutes/year of power outage. Table 4-8 presents the modeled 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 fraction of total hours 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 (p_{hr}), the overall probability that the threshold will be exceeded in a given year (p_{1yr}), and the estimated recurrence interval. Overall annual probability, p, was calculated as:

$$p = 1 - (1 - p_{hr})^n$$

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 predicts the average number of years between a REL exceedance. These calculations are shown in Table 4-8.

Project-only Frequency of NO₂ Impacts that exceed the REL

As shown on Figure 4-2 and Table 4-8, when taking into account historical Grant County PUD electrical grid reliability, the recurrence interval for ambient NO₂ concentrations to exceed 454 µg/m³ at the MIBR is once every 1,235 years. Likewise, the recurrence interval for ambient NO₂ concentrations to exceed 454 µg/m³ at the MICR is once every 17,290 years.

Cumulative Frequency of NO₂ Impacts that exceed the REL

As shown on Figure 4-4, cumulative NO₂ impacts exceeding the 454 µg/m³ threshold were modeled to occur at homes, at the Mountain View School, and at residentially zoned vacant lands east of the existing Dell and Columbia data centers. Those receptor locations are adjacent to the uncontrolled generators at the Dell and Columbia data centers, approximately 1.5 miles from the Oxford Data

Center. Therefore, most of the modeled NO₂ impacts at those eastern receptor locations are caused by emissions from the Dell and Columbia data centers.

The maximum cumulatively impacted residential receptor (adjacent east to the Columbia Data Center) is predicted to reach an ambient concentration of 1,129 µg/m³. Only 18 percent (207 µg/m³) of that NO₂ impact is attributed to project-related emissions. Although, the project-related emission impacts at these eastern locations are all below the NO₂ ASIL (470 µg/m³), the cumulative ambient concentration is expected to exceed the REL. Therefore, the estimated frequency of occurrence for ambient concentrations to exceed 470 µg/m³ was evaluated (in the same manner as shown in Table 4-). As shown on Figure 4-4, the predicted frequency of occurrence for such an exceedance would be once every 206 years at the Mountain View School, and no more than once every 30 years at the maximum cumulatively impacted residential receptor.

This evaluation demonstrates that the average frequency of occurrence of a risk receptor to be exposed to NO₂ concentrations greater than the acute REL is very low. Therefore, it is concluded that the Oxford Data Center satisfies Ecology's criteria for air quality permitting related to acute non-cancer health impacts.

4.4.2 Quantifying Increased Cancer Risk

4.4.2.1 Cancer Risk from Exposure to DEEP

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

The formula used to determine cancer risk is as follows:

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

The exposure frequencies for each receptor type are shown below and provided in Table 4-5, based on Ecology's judgment from review of published risk evaluation guidelines.

EXPOSURE FREQUENCIES FOR EACH RECEPTOR TYPE

Parameter	Description	Value Based on Receptor Type						Units
		Residential	Worker	School-Staff	School-Student	Hospital	Boundary	
C _{Air}	Concentration in air at the receptor location	See Table 4-2						µg/m ³
URF	Unit Risk Factor	0.0003						(µg/m ³) ⁻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

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 do exist. However, the EPA 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 specified carcinogen concentration, 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.

The permitted annual-average emission rate for the Oxford Data Center (0.81 tons per year including condensable back-half) was used to evaluate the 70-year lifetime cancer risk from exposure to DEEP. Table 4-2 shows the estimated cancer risks associated with predicted project-only DEEP concentrations and the URFs (from Table 4-4). Although the highest annual-average DEEP concentration was predicted to occur at the MIBR, the greatest cancer risk estimate is at the MIRR. This is due to considerations of duration and frequency of potential exposure incorporated in the

corrected URFs. The calculated project-only lifetime cancer risk at the MIRR is 5.9 per million (5.9×10^{-6}). This is less than 10 per million, which is the 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. However, Ecology has indicated that new sources of DEEP may not be approved to locate in Quincy if the resulting cumulative cancer risk is above 100 per million (100×10^{-6}). Table 4-2 lists the cumulative cancer risks for each maximally impacted receptor location. This accounts for currently permitted DEEP emissions from neighboring data centers, railroad and roadway diesel traffic emissions, and proposed project emissions. The maximum cumulative cancer risk for persons exposed to ambient air at the MIRR is estimated to be 13 per million. The maximum cumulative cancer risk at the school (I-1) is estimated to be 13 per million (13×10^{-6}). The maximum cumulative impacted house in the modeling domain is 68 per million (68×10^{-6}); however, the contribution to the ambient DEEP at this location that is attributable to project-only emissions is only 0.77 percent of the total ambient impact. Most of the cancer risk at this receptor location is from traffic on SR 28 and SR 281, as shown in Table 4-2.

4.4.2.2 Cancer Risk from Exposure to All Carcinogenic Emissions

The cancer risk evaluation was completed by estimating the increased cancer risk from exposure to all potentially carcinogenic compounds to be emitted from the proposed project. The permitted annual-average emission rate was used to evaluate the (70-year) lifetime cancer risk from exposure to all carcinogens. Table 4-9 lists the individual pollutant and combined pollutant cancer risks at the MIRR and shows a total combined risk of 5.9 per million (5.9×10^{-6}). As shown, all recognized carcinogenic compounds other than DEEP contribute negligibly to the overall cancer risk.

4.4.2.3 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 (ASTDR website 2014; EPA website 2015).

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5.0 UNCERTAINTY CHARACTERIZATION

Many factors of the HIA are prone to uncertainty. Uncertainty relates to the lack of exact knowledge regarding many of the assumptions used to estimate the human health impacts of DEEP 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 evaluated in this HIA.

5.1 Emission Factor and Exposure Uncertainty

One of the major uncertainties is the emission factors for TAPs emitted by diesel engines. The forecast emission rates for 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 project. 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, Microsoft conservatively estimated that it would use the generators during emergency outages for no more than 84 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 over 99.99 percent each year (2008 to 2014) and a Customer Average Interruption Duration Index (or average duration of power interruption per customer) of 76 to 300 minutes (1.3 to 5 hours) over the same period (Grant County PUD 2015). While this high level of historical reliability provides some assurance that electrical service is relatively stable, Microsoft cannot predict future outages with any degree of certainty. Microsoft accepted a limit of 84 hours per year for emergency operations, and estimated that this limit should be more than sufficient to meet its emergency demands. It is expected that estimates of cancer risk will be significantly overestimated by assuming the generators will operate annually at the maximum permitted level for 70 consecutive years.

5.2 Air Dispersion Modeling Uncertainty

The transport of pollutants through the air is a complex process. Regulatory air dispersion models have been developed to estimate the transport and dispersion of pollutants as they travel through the air. The models are frequently updated as techniques that are more accurate become known, but are developed to avoid underestimating the modeled impacts. Even if all of the numerous input

parameters to an air dispersion model are known, random effects found in the real atmosphere will introduce uncertainty. Typical of the class of modern steady-state Gaussian dispersion models, the AERMOD model used for the 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.

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.

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 $\mu\text{g}/\text{m}^3$. The OEHHA's DEEP URF (3×10^{-4} per $\mu\text{g}/\text{m}^3$) 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 (EPA 2002) 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.

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, the 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.” The 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 (OEHHA 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 5-1 presents a summary of how the uncertainty affects the quantitative estimate of risks or hazards.

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6.0 SHORT-TERM EXPOSURE TO DEEP AND PM_{2.5}

As discussed previously, exposure to DEEP can cause both acute and chronic adverse health effects. However, as discussed in Section 4.3.1, reference toxicological values specifically for DEEP exposure at short-term or intermediate intervals (e.g., 24-hour values) do not currently exist. Therefore, health risks from acute DEEP exposure are not quantified in this assessment. Regardless, not quantifying acute health risks in this document does not imply that they have not been considered. Instead, it is assumed that compliance with the 24-hour NAAQS for particulate matter with an aerodynamic diameter less than or equal to 2.5 microns (PM_{2.5}) is an indicator of acceptable short-term emission impacts and that no adverse health effects may be anticipated from acute DEEP exposure. Microsoft's Approval Order Revision Letter (Landau Associates 2016) concludes that emissions from the proposed project are not expected to cause or contribute to an exceedance of any NAAQS.

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7.0 DISCUSSION OF ACCEPTABILITY OF RISK

7.1 Project-Only Cancer Risks Are Lower Than 10-Per-Million

The estimated cancer risks (due to project-only DEEP emissions) at the nearby residences, businesses, and sensitive receptor locations range between 0.02 per million (at the MIBR) to 5.9 per million (at the MIRR). The combined pollutant cancer risk at the MIRR (from all carcinogenic emissions from the project) is also 5.9 per million, indicating that DEEP is the only significant carcinogen.

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 Risks Are Lower Than 100-Per-Million

The residence that will be exposed to the highest cumulative cancer risk is located approximately 1.5 miles southeast of the project site, near SR 281, at a location where most of the cancer risk is attributable to traffic and unrelated to project emissions. The total average cumulative DEEP cancer risks for the maximally exposed residences based on maximum project impacts and maximum cumulative impacts are as follows:

Project-only cancer risk (R-2 maximally impacted residence)	0.5 per million
Background DEEP cancer risk	67.5 per million
Cumulative DEEP cancer risk	68 per million
Project-only cancer risk (R-1 northward house)	5.9 per million
Background DEEP cancer risk	7.1 per million
Cumulative DEEP cancer risk	13 per million

The increased cancer risk associated with DEEP emissions from the proposed project is approximately 0.77 percent of the total cumulative DEEP cancer risk at receptor location R-2 and 44 percent at R-1. The total average cumulative DEEP cancer risks for the maximally exposed home, business, and sensitive receptor locations are each less than the residential risks listed above and less than 100 per million.

7.3 Non-Cancer Risk Hazard Quotients

7.3.1 Project-only Impacts

The chronic HQ for DEEP at the MIBR is only 0.077. The chronic HI for all combined non-cancer TAPs at the MIBR is only 0.09.

The acute HQ for NO₂ at the unpopulated MIBR is 1.3, but the modeled frequency of occurrence for an HQ exceeding 1 is 1,922 years. The acute HI for all combined non-cancer TAPs at the MIBR is 1.34.

7.3.2 Cumulative Impacts Including Other Quincy Data Centers

The highest cumulative chronic HQ for DEEP at the Oxford MIBR is only 0.09 ($0.13 \mu\text{g}/\text{m}^3 \div 5 \mu\text{g}/\text{m}^3$).

The highest cumulative acute HQ (indicating an ambient impact greater than the REL = $470 \mu\text{g}/\text{m}^3$) for NO₂ occurs at residential property at the eastern boundary of the Columbia Data Center, and is caused primarily by emissions from the uncontrolled generators at the Columbia Data Center. At that location, the modeled impact attributable to Oxford emissions is $207 \mu\text{g}/\text{m}^3$, which is only 18 percent of the cumulative impact. Although the maximum cumulative acute HQ for NO₂ at that location is 2.4 ($1,129 \mu\text{g}/\text{m}^3 \div 470 \mu\text{g}/\text{m}^3$), the estimated frequency of occurrence for the ambient concentration to exceed the REL at this location is only once every 30 years.

7.3.3 Conclusion for Non-Cancer Risks

This evaluation demonstrates that the probability that this project could cause non-cancer adverse health impacts is very low. The spatial zones where the project-only impacts exceed the REL (indicated by an HQ that exceeds 1) are very small and limited to adjacent vacant land near the property boundary. Furthermore, the calculated frequency of occurrence for the ambient impacts to exceed the REL is very low (recurrence interval of 1,922 years at the MIBR). Therefore, it is concluded that the non-cancer risks caused by the Oxford Data Center satisfy Ecology's permitting criteria.

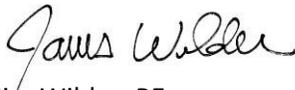
8.0 SIGNATURES

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

LANDAU ASSOCIATES, INC.



Christel Olsen, EIT
Senior Staff Engineer-In-Training



Jim Wilder, PE
Senior Associate Engineer

CO/JMW/ccy

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9.0 REFERENCES

- ATSDR. 2002. Nitrogen Oxides (Nitric Oxide, Nitrogen Dioxide, etc.). Division of Toxicology, Agency for Toxic Substances and Disease Registry. Available at <http://www.atsdr.cdc.gov/toxfaqs/tfacts175.pdf>. April.
- ATSDR website. 2014. Toxic Substances Portal – Nitrogen Oxides.. Updated October 21. Agency for Toxic Substances and Disease Registry. <http://www.atsdr.cdc.gov/mmg/mmg.asp?id=394&tid=69>. Accessed October 1, 2015.
- CalEPA. 2003. The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments. Available at http://oehha.ca.gov/air/hot_spots/HRAguidefinal.html. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. August.
- CalEPA. 1998. Initial Statement of Reasons for Rulemaking, Staff Report: Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant. Available at <http://www.arb.ca.gov/toxics/dieseltac/staffrpt.pdf>. Air Resources Board and Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. June.
- CalEPA website. 2008. Appendix D.2: Acute RELs and Toxicity Summaries using the Previous Version of the Hot Spots Risk Assessment Guidelines. http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD2_final.pdf. California Environmental Protection Agency. Accessed October 15, 2015.
- Dhammapala, R. 2015a. Email message from Ranil Dhammapala, Washington State Department of Ecology, to Christel Olsen, Landau Associates. Re: AERMOD Input File for SR28. August 13.
- Dhammapala, R. 2015b. Email message from Ranil Dhammapala, Washington State Department of Ecology, to Mark Brunner, Landau Associates. Re: Project Genesis NOC Application. November 9.
- Ecology. 2015. Revised Health Impact Assessment Review Document for Microsoft Oxford Data Center, Quincy, Washington. Air Quality Program, Washington State Department of Ecology. Available at <http://www.ecy.wa.gov/programs/air/quincydatacenter/docs/HIAreccs.pdf>. May 13.
- 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. Available at <https://fortress.wa.gov/ecy/publications/publications/0802032.pdf>. December 3.
- EPA. 2002. Health Assessment Document for Diesel Engine Exhaust. EPA/600/8-90/057F. National Center for Environmental Assessment, Office of Research and Development, U.S. Environmental Protection Agency. Available at <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=29060>. May.
- EPA. 1995. Compilation of Air Pollutant Emission Factors, Volume I: Stationary Point and Area Sources. Fifth Edition. AP-42. Office of Air Quality Planning and Standards, Office of Air and Radiation, U.S. Environmental Protection Agency. January.

-
- EPA website. 2015. Integrated Risk Information System. <http://cfpub.epa.gov/ncea/iris/search/index.cfm>. Accessed October 18.
- Grant County PUD. 2015. Grant County PUD System Reliability Indices Numbers. Grant County Public Utility District.
- Grant County website. 2015. Grant County GIS. <http://www.grantcountywa.gov/GIS/>. Accessed September 18.
- Hesterberg T.W., W.B. Bunn, R.O. McClellan, A.K. Hamade, C.M. Long, and P.A. Valberg PA. 2009. "Critical Review of the Human Data on Short-Term Nitrogen Dioxide (NO₂) Exposures: Evidence for NO₂ No-Effect Levels." *Crit Rev. Toxicol.* 39(9):743-81. DOI: 10.3109/10408440903294945.
- Jarvis D.J., G. Adamkiewicz, M.E. Heroux, R. Rapp, and F.J. Kelly. 2010. "Nitrogen Dioxide." In: WHO Guidelines for Indoor Air Quality: Selected Pollutants. World Health Organization. Available at <http://www.ncbi.nlm.nih.gov/books/NBK138707/>.
- Landau Associates. 2016. Letter: April 2016 Request for Revisions to Approval Order No. 14AQ-E537, Microsoft Oxford Data Center, Quincy, Washington. From Jim Wilder, PE, to Gary Huitsing, Washington State Department of Ecology. April 8.
- Landau Associates. 2014. Final: Notice of Construction Supporting Information Report, Proposed Microsoft Project Oxford Data Center, Quincy, Washington. Prepared for The Microsoft Corporation. June 11.
- Lents, J.M., L. Arth, M. Boretz, M. Chitjian, K. Cocker, N. Davis, K. Johnson, Y. Long, J.W. Miller, U. Mondragon, R.M. Nikkila, M. Omary, D. Pacocha, Y. Qin, S. Shah, G. Tonnesen, Z.S. Wang, M. Wehrey, and X. Zhu. 2005. Air Quality Implications of Backup Generators in California – Volume One: Generation Scenarios, Emissions and Atmospheric Modeling, and Health Risk Analysis. California Energy Commission, PIER Energy-Related Environmental Research. CEC-500-2005-048. March.
- NAC AEGL Committee. 2008. Interim Acute Exposure Guideline Levels for Nitrogen Dioxide. The National Advisory Committee for the Development of Acute Exposure Guideline Levels for Hazardous Substances (AEGL Committee). Available at http://www.epa.gov/oppt/aegl/pubs/nitrogen_dioxide_interim_nitrogen_tetroxide_proposed_dc_2008.v1.pdf. December.
- NAS. 1994. Science and Judgment in Risk Assessment. National Research Council, National Academy of Sciences. National Academy Press. Washington, DC.
- NAS. 1983. Risk Assessment in the Federal Government: Managing the Process. National Research Council, National Academy of Sciences. Available at http://www.nap.edu/catalog.php?record_id=366. The National Academy Press. Washington, DC.

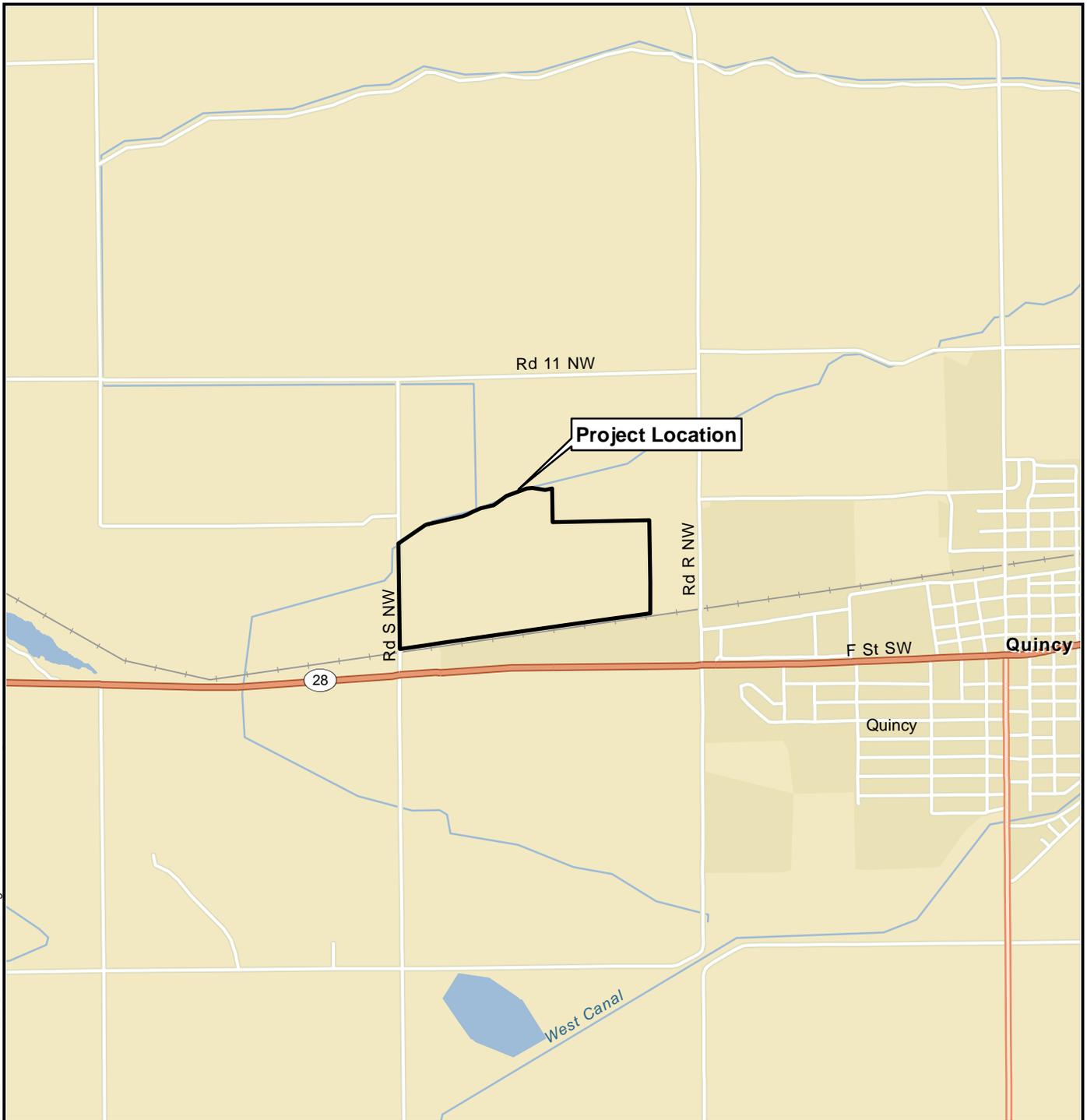
OEHHA. 1999. Acute Toxicity Summary, Nitrogen Dioxide. Determination of Acute Reference Exposure Levels for Airborne Toxicants. Available at http://oehha.ca.gov/air/acute_rels/pdf/10102440A.pdf.

OEHHA website. 2007. Air Toxicology and Epidemiology. <http://oehha.ca.gov/air/allrels.html>. California Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. Accessed October 12, 2015.

Quincy, City of. 2011. Quincy, Washington Comprehensive Plan and Zoning. City of Quincy, Washington. Effective Date: February 8.

WSU website. 2015. Lookup 2009-2011 Design Values of Criteria Pollutants. <http://lar.wsu.edu/nw-airquest/lookup.html>. Northwest International Air Quality Environmental Science and Technology Consortium, Washington State University. Accessed October 12.

G:\Projects\1409\001\010\011\Figure2-1VicMap.mxd 1/16/2014 NAD 1983 StatePlane Washington South FIPS 4602 Feet



Data Source: Esri 2012.



Project Oxford Data Center
Quincy, Washington

Vicinity Map

Figure
2-1



Data Source: Microsoft



Project Oxford Data Center
Quincy, Washington

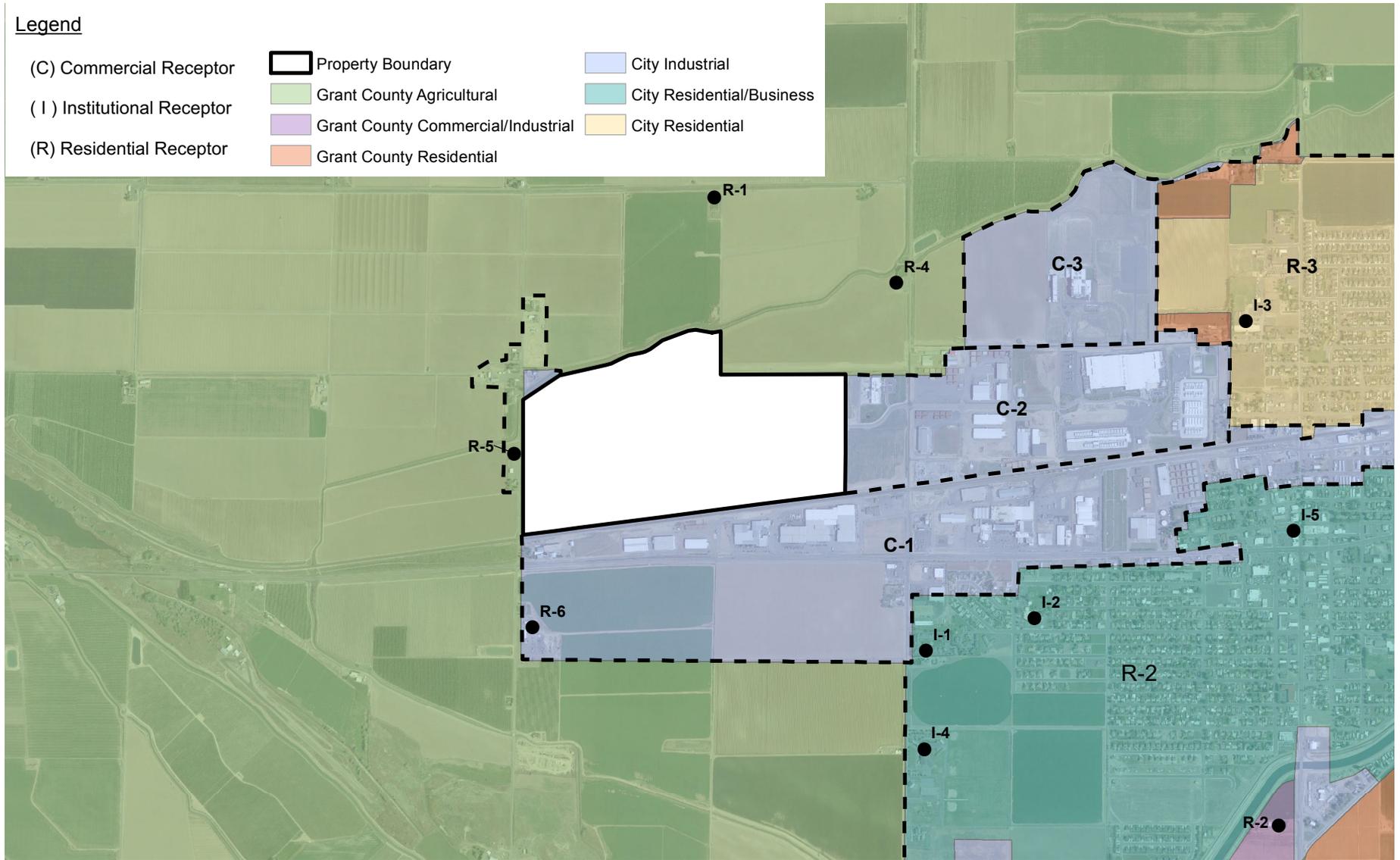
Site Plan

Figure
2-2

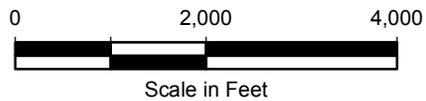


Legend

- | | | |
|----------------------------|------------------------------------|---------------------------|
| (C) Commercial Receptor | Property Boundary | City Industrial |
| (I) Institutional Receptor | Grant County Agricultural | City Residential/Business |
| (R) Residential Receptor | Grant County Commercial/Industrial | City Residential |
| | Grant County Residential | |



Data Source: © Google Earth Pro 2015; Grant County GIS;



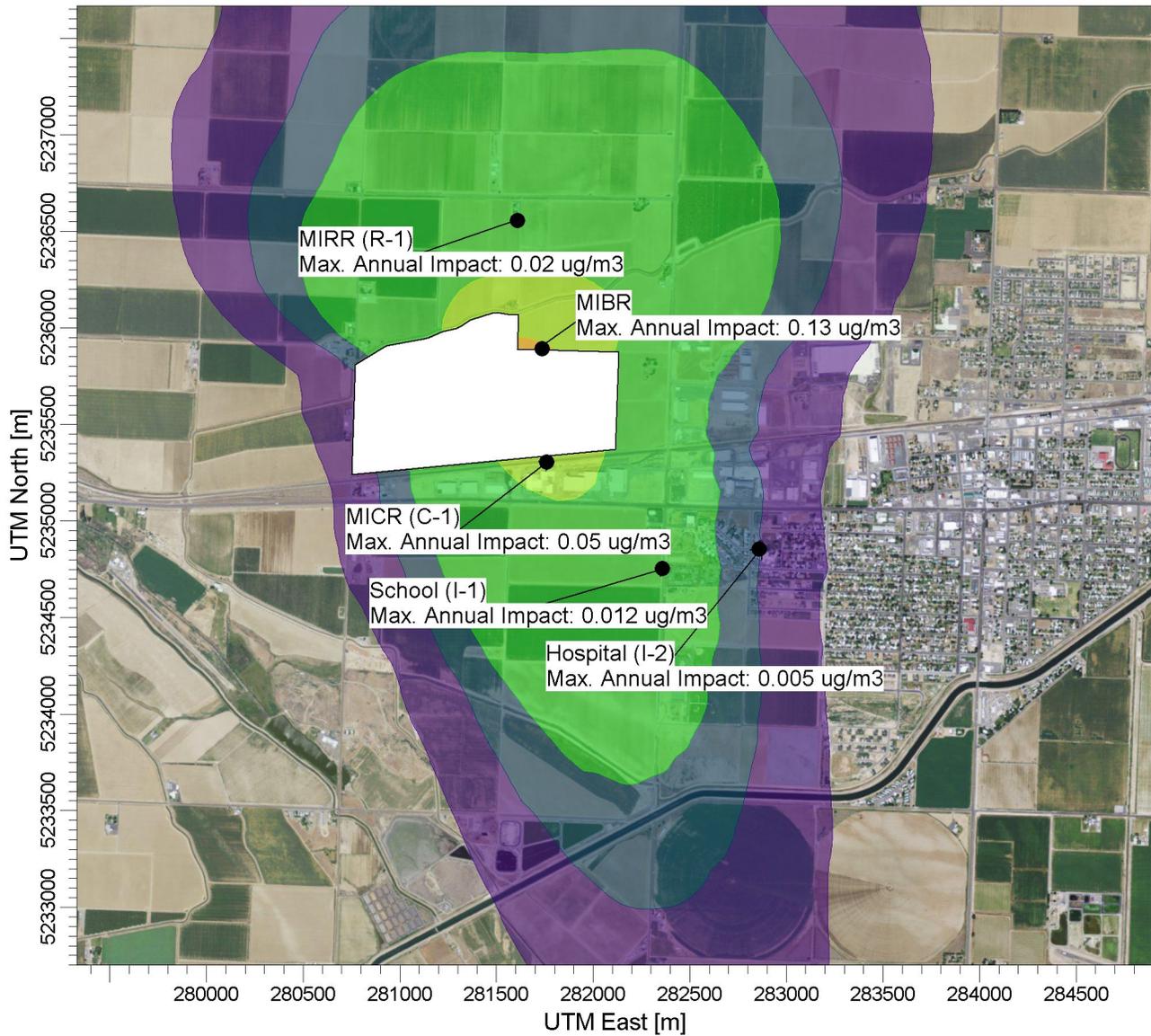
Project Oxford Data Center
Quincy, Washington

Land Use Zoning Map

Figure
2-3

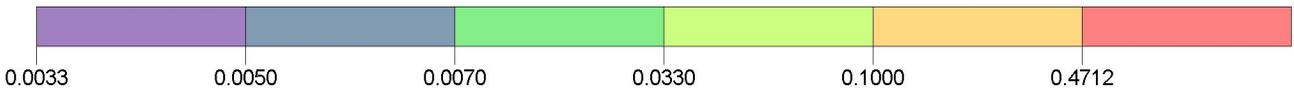
PROJECT TITLE:

**FIGURE 4-1: AMBIENT IMPACTS FROM PROJECT ONLY DEEP EMISSIONS
PROJECT OXFORD DATA CENTER OF QUINCY, WASHINGTON**



PLOT FILE OF ANNUAL VALUES AVERAGED ACROSS 5 YEARS FOR SOURCE GROUP: MSOXFORD

ug/m³



COMMENTS:

Modeled max. ambient impacts from project related emissions, only.

SOURCES:

290

RECEPTORS:

3430

OUTPUT TYPE:

Concentration

MAX:

0.1288 ug/m³

COMPANY NAME:

Landau Associates, Inc.

MODELER:

Christel Olsen

SCALE:

1:35,000

0

1 km

DATE:

12/28/2015

PROJECT NO.:

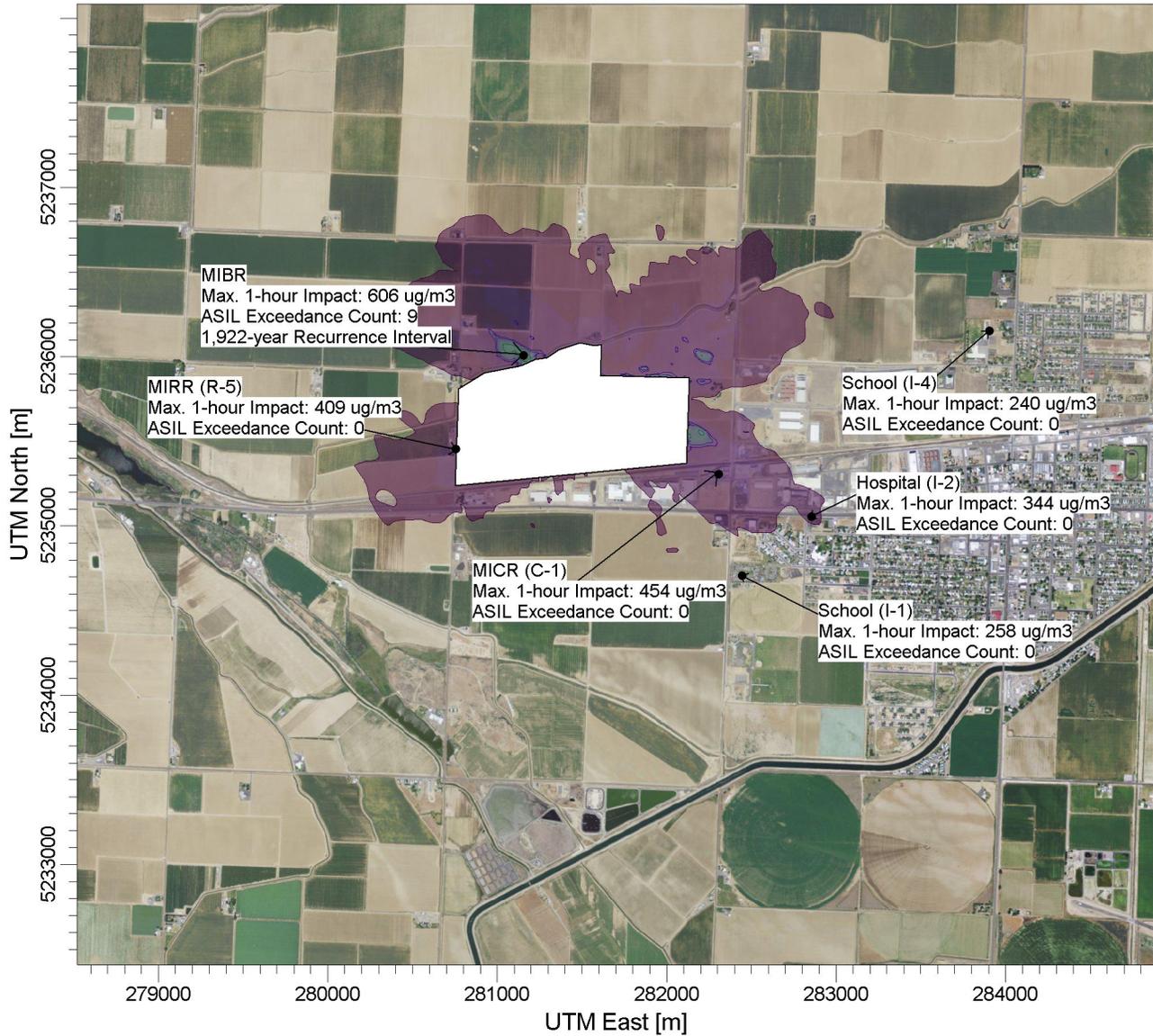
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LANDAU ASSOCIATES

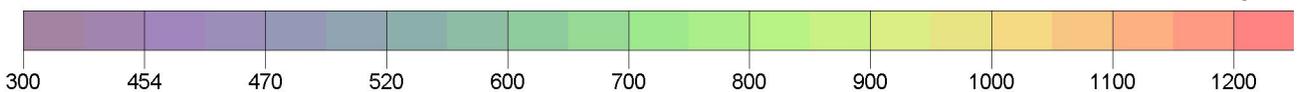
PROJECT TITLE:

**FIGURE 4-2: AMBIENT IMPACTS FROM PROJECT ONLY NO2 EMISSIONS
PROJECT OXFORD DATA CENTER OF QUINCY, WASHINGTON**



PLOT FILE OF HIGH 1ST HIGH 1-HR VALUES FOR SOURCE GROUP: ALL

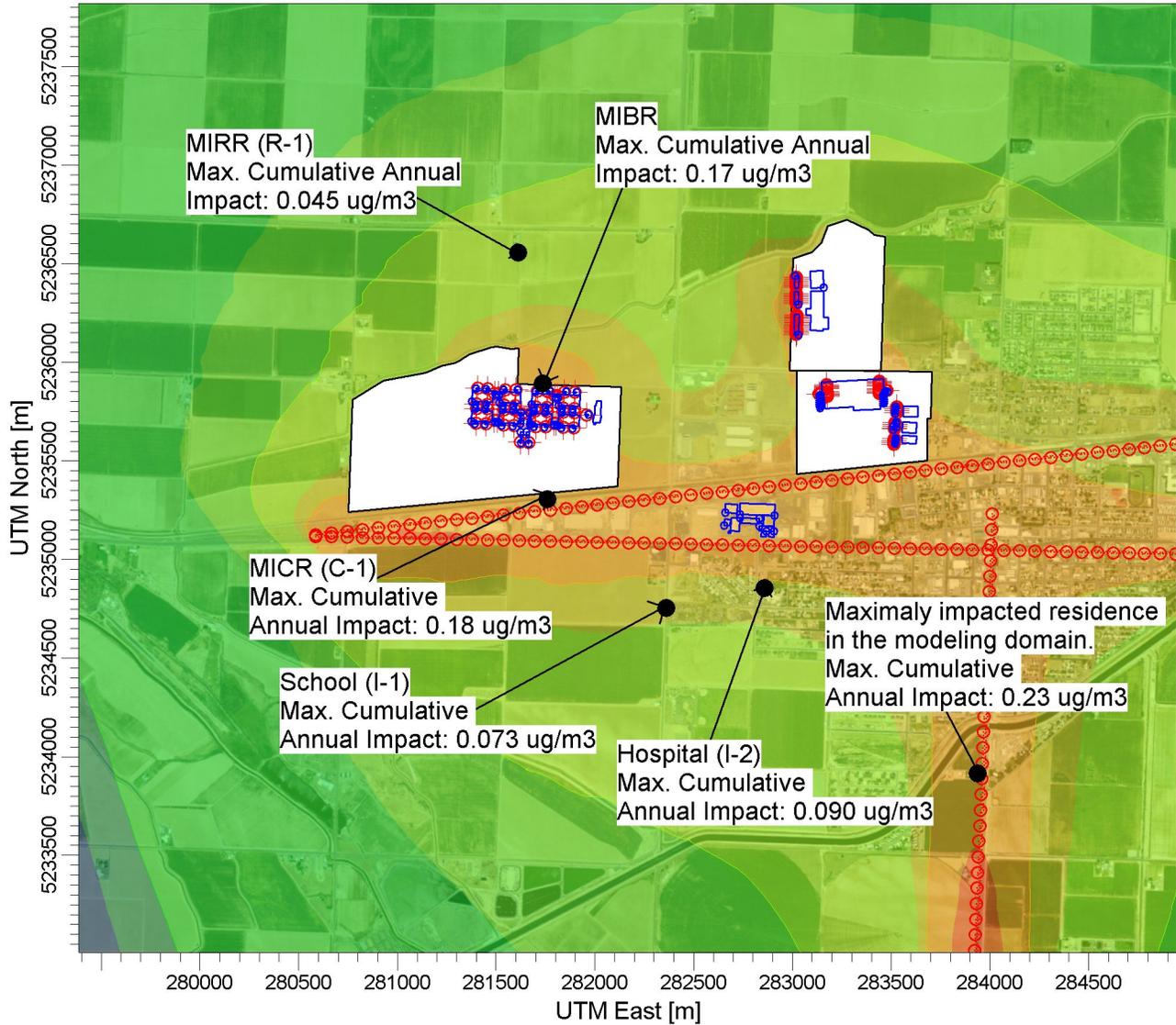
ug/m³



<p>COMMENTS:</p> <p>Recurrence Interval (years) is based on 152 minutes of power outage per year, according to Grant County PUD.</p>	<p>SOURCES:</p> <p>45</p>	<p>COMPANY NAME:</p> <p>Landau Associates, Inc.</p>	
	<p>RECEPTORS:</p> <p>3872</p>	<p>MODELER:</p> <p>Christel Olsen</p>	<p>LANDAU ASSOCIATES</p>
	<p>OUTPUT TYPE:</p> <p>Concentration</p>	<p>SCALE:</p> <p>1:40,000</p> <p>0 1 km</p>	
	<p>MAX:</p> <p>606 ug/m³</p>	<p>DATE:</p> <p>12/28/2015</p>	<p>PROJECT NO.:</p> <p>1409001.010</p>

PROJECT TITLE:

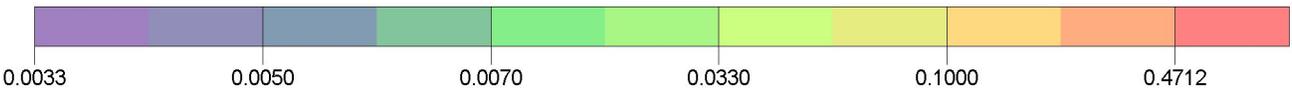
**FIGURE 4-3: CUMULATIVE AMBIENT IMPACTS FROM LOCAL DEEP EMISSIONS
PROJECT OXFORD DATA CENTER OF QUINCY, WASHINGTON**



PLOT FILE OF ANNUAL VALUES AVERAGED ACROSS 5 YEARS FOR SOURCE GROUP: ALL

ug/m³

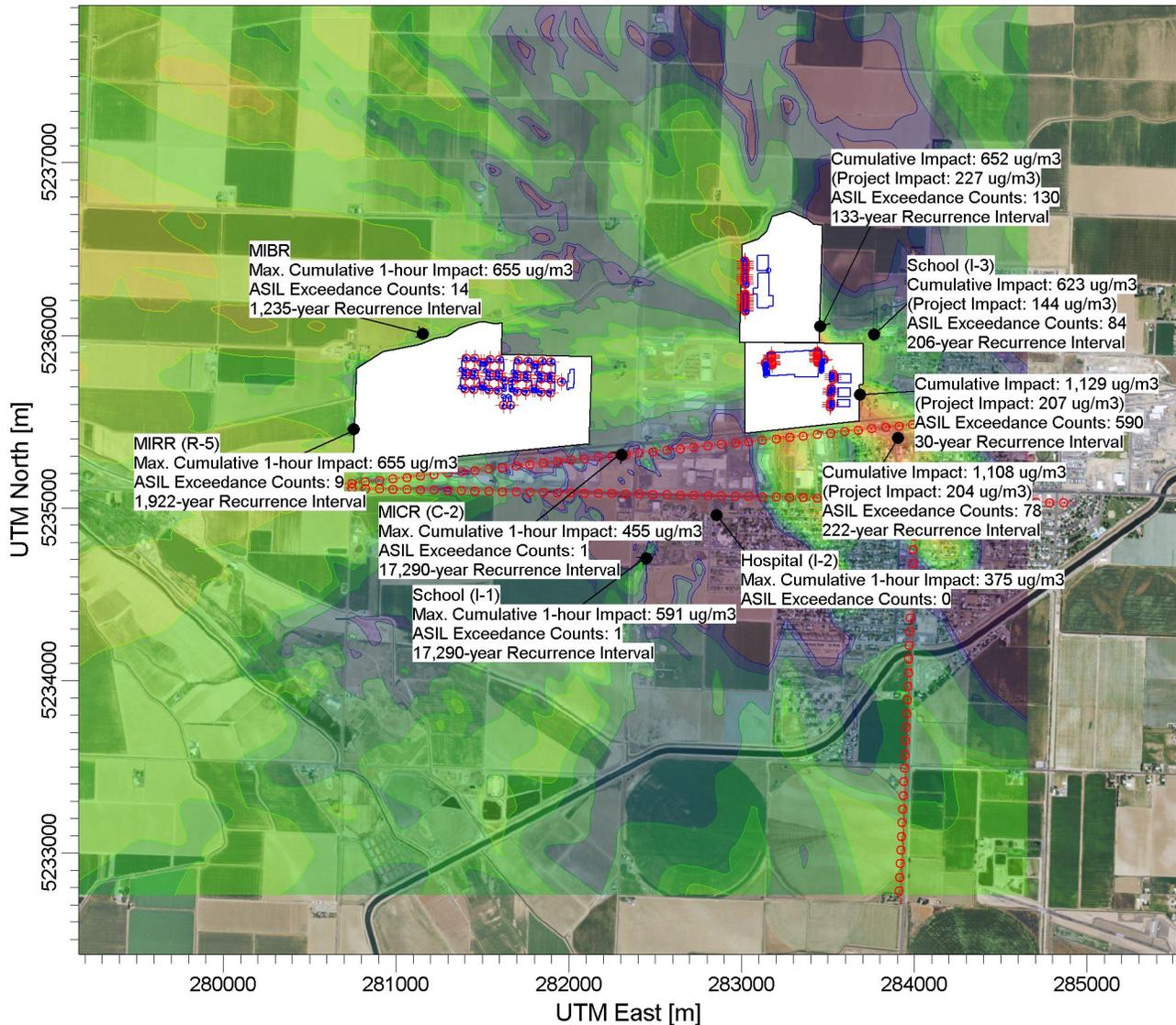
Max: 0.4713 [ug/m³] at (283937.63, 5233028.00)



<p>COMMENTS:</p> <p>Modeled max. cumulative ambient impacts include emissions from the proposed project, DELL data center, Columbia data center, SR 28, SR 281, and BNSF railroad.</p>	<p>SOURCES:</p> <p>291</p>	<p>COMPANY NAME:</p> <p>Landau Associates, Inc.</p>		
	<p>RECEPTORS:</p> <p>3810</p>	<p>MODELER:</p> <p>Christel Olsen</p>		
	<p>OUTPUT TYPE:</p> <p>Concentration</p>	<p>SCALE:</p> <p>1:35,000</p> <p>0  1 km</p>	 <p>LANDAU ASSOCIATES</p>	
	<p>MAX:</p> <p>0.4713 ug/m³</p>	<p>DATE:</p> <p>12/28/2015</p>		

PROJECT TITLE:

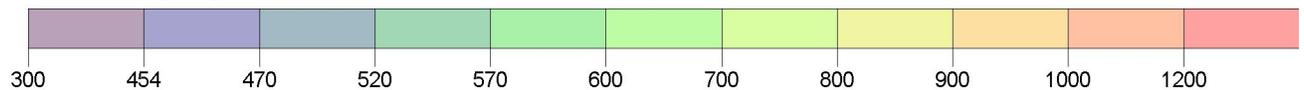
**FIGURE 4-4: CUMULATIVE AMBIENT IMPACTS FROM LOCAL NO2 EMISSIONS
PROJECT OXFORD DATA CENTER OF QUINCY, WASHINGTON**



PLOT FILE OF HIGH 1ST HIGH 1-HR VALUES FOR SOURCE GROUP: ALL

ug/m³

Max: 1233 [ug/m³] at (283155.28, 5235757.00)



COMMENTS:

Recurrence Interval (years) is based on 152 minutes of power outage per year, according to Grant County PUD.

SOURCES:

252

COMPANY NAME:

Landau Associates, Inc.

RECEPTORS:

3907

MODELER:

Christel Olsen

OUTPUT TYPE:

Concentration

SCALE:

1:40,000

0 1 km

MAX:

1233 ug/m³

DATE:

1/6/2016

PROJECT NO.:

1409001.010



**TABLE 2-1
APRIL 2016 EMISSION RATES FOR TOXIC AIR POLLUTANTS
OXFORD DATA CENTER
QUINCY, WASHINGTON**

Pollutant	CAS Number	SQER	Facility Emissions	SQER Exceeded?	Ambient Concentration (µg/m ³)	ASIL (µg/m ³)	ASIL Exceeded?
Diesel Generator TAPs							
70-year Average DEEP	None	0.639 lbs/yr	1,628 lbs/yr	Yes	0.139	0.0033	Yes
Theo. Max Year DEEP (a)	None	0.639 lbs/yr	5,285 lbs/yr	Yes	0.417	0.0033	Yes
CO	630-08-0	50.2 lbs/hour	126 lbs/hour	Yes	421	23,000	No
Ammonia	7664-41-7	9.3 lbs/day	518 lbs/day	Yes	25.9	70.8	No
SO ₂		1.45 lbs/hour	1.3 lbs/hour	No	-	-	-
NO ₂	10102-44-0	1.03 lbs/hour	57.5 lbs/hour	Yes	606	470	Yes
Benzene (a)	71-43-2	6.62 lbs/yr	20.8 lbs/yr	Yes	9.70E-04	0.034	No
Toluene	108-88-3	657 lbs/day	0.592 lbs/day	No	-	-	-
Xylenes	95-47-6	58 lbs/day	0.407 lbs/day	No	-	-	-
1,3-Butadiene (a)	106-99-0	1.13 lbs/yr	1.05 lbs/yr	No	-	-	-
Formaldehyde (a)	50-00-0	32 lbs/yr	1.79 lbs/yr	No	-	-	-
Acetaldehyde (a)	75-07-0	71 lbs/yr	0.68 lbs/yr	No	-	-	-
Acrolein	107-02-8	0.00789 lbs/day	0.016 lbs/day	Yes	7.90E-04	0.06	No
Benzo(a)pyrene (a)	50-32-8	0.174 lbs/yr	6.9E-03 lbs/yr	No	-	-	-
Benzo(a)anthracene (a)	56-55-3	1.74 lbs/yr	1.7E-02 lbs/yr	No	-	-	-
Chrysene (a)	218-01-9	17.4 lbs/yr	4.1E-02 lbs/yr	No	-	-	-
Benzo(b)fluoranthene (a)	205-99-2	1.74 lbs/yr	3.0E-02 lbs/yr	No	-	-	-
Benzo(k)fluoranthene (a)	207-08-9	1.74 lbs/yr	5.9E-03 lbs/yr	No	-	-	-
Dibenz(a,h)anthracene (a)	53-70-3	0.16 lbs/yr	9.3E-03 lbs/yr	No	-	-	-
Ideno(1,2,3-cd)pyrene (a)	193-39-5	1.74 lbs/yr	1.1E-02 lbs/yr	No	-	-	-
Naphthalene (a)	91-20-3	5.64 lbs/yr	3.5E+00 lbs/yr	No	-	-	-
Propylene	115-07-1	394 lbs/yr	6.9E+00 lbs/yr	No	-	-	-
Cooling Tower TAPs							
Fluoride	---	1.71 lbs/day	0.0260 lbs/day	No	-	-	-
Manganese	---	0.0053 lbs/day	0.00252 lbs/day	No	-	-	-
Copper	---	0.219 lbs/hour	3.5E-05 lbs/hour	No	-	-	-
Chloroform	67-66-3	8.35 lbs/year	0.526 lbs/year	No	-	-	-
Bromodichloromethane	75-27-4	5.18 lbs/year	0.526 lbs/year	No	-	-	-
Bromoform	75-25-2	174 lbs/year	13.8 lbs/year	No	-	-	-

Notes:

- DEEP = Diesel engine exhaust particulate matter.
- CO = Carbon monoxide.
- SO₂ = Sulfur dioxide.
- NO₂ = Nitrogen dioxide.
- SQER = Small-quantity emission rate.
- ASIL = Acceptable source impact level.
- TAP = Toxic air pollutant.

Shaded rows indicate the emission rate exceeds the SQER.
(a) Based on theoretical maximum year emissions.

TABLE 2-2
SUMMARY OF LAND USES NEAR OXFORD DATA CENTER
OXFORD DATA CENTER
QUINCY, WASHINGTON

Receptor Type	ID	Direction From Project Site	Approximate Distance From Nearest Project-Emission Source	
			Feet	Meters
Residence	R-1	north	2,418	737
Residencial Zone	R-2	southeast	3,051	930
Residencial Zone	R-3	east	5,184	1,580
Residence	R-4	northeast	1,919	585
Residences	R-5	adjacent north & northwest	2,986	910
Residence	R-6	southwest	3,166	965
Commercial Zone	C-1	southeast & adjacent south	984	300
Commercial Zone (including Columbia Data Center)	C-2	adjacent east	558	170
Commercial Zone (including Dell Data Center)	C-3	east	3,609	1,100
Quincy Valley School	I-1	southeast	3,379	1,030
Quincy Valley Hospital	I-2	southeast	3,904	1,190
Mountain View Elementary School	I-3	northeast	5,955	1,815
Monument Elementary School	I-4	southeast	4,577	1,395
Quincy High Tech High School	I-5	east	6,841	2,085

TABLE 3-1
SUMMARY OF BACT DETERMINATION FOR DIESEL ENGINE GENERATORS
OXFORD DATA CENTER
QUINCY, WASHINGTON

Pollutant(s)	BACT Determination
Particulate matter (PM)	Use of good combustion practices; Use of EPA Tier 2-certified engines; and Compliance with the operation and maintenance restrictions of 40 CFR Part 60, Subpart IIII
Nitrogen oxides (NO _x)	Use of good combustion practices; Use of EPA Tier 2-certified engines; and Compliance with the operation and maintenance restrictions of 40 CFR Part 60, Subpart IIII
Carbon monoxide (CO) and volatile organic compounds (VOCs)	Use of good combustion practices; Use of EPA Tier 2-certified engines; and Compliance with the operation and maintenance restrictions of 40 CFR Part 60, Subpart IIII
Sulfur dioxide (SO ₂)	Use of ultra-low sulfur diesel fuel containing no more than 15 parts per million by weight of sulfur

Notes:

BACT = Best available control technology.

PM = Particulate matter.

EPA = US Environmental Protection Agency.

CFR = Code of Federal Regulations.

NO_x = Nitrogen oxides.

CO = Carbon monoxide.

VOCs = Volatile organic compounds.

SO₂ = Sulfur dioxide

TABLE 3-2
SUMMARY OF TBACT DETERMINATION FOR DIESEL ENGINE GENERATORS
OXFORD DATA CENTER
QUINCY, WASHINGTON

Toxic Air Pollutant(s)	tBACT Determination
DEEP	Compliance with the PM BACT requirement
Acetaldehyde, carbon monoxide, acrolein, benzene, benzo(a)pyrene, 1,3-butadiene, formaldehyde, propylene, toluene, total PAHs, xylenes	Compliance with the VOC BACT requirement
Nitrogen dioxide	Compliance with the NO _x BACT requirement
Sulfur dioxide	Compliance with the SO ₂ BACT requirement

Notes:

tBACT = Best available control technology for toxics.

DEEP = Diesel engine exhaust particulate matter.

PM = Particulate matter.

BACT = Best available control technology.

PAHs = Polycyclic aromatic hydrocarbons.

VOC = Volatile organic compound.

NO_x = Nitrogen oxides.

SO₂ = Sulfur dioxide.

TABLE 4-1
SUMMARY OF IDENTIFIED RISK RECEPTORS
OXFORD DATA CENTER
QUINCY, WASHINGTON

Receptor Type	ID	UTM Zone 11		Project-only DEEP Annual Impact	Project-only NO ₂ 1-hour Impact
		E (m)	N (m)	(µg/m ³)	(µg/m ³)
NO ₂ -MIBR	--	281,155	5,236,007	--	606
NO ₂ -MICR	C-1	282,305	5,235,307	--	454
NO ₂ -MIRR	R-5	280,755	5,235,457	--	409
Maximally Impacted School Yard	I-1	282,447	5,234,708	0.012 (a)	258
Maximally Impacted Medical Center	I-2	282,855	5,234,957	0.005 (b)	300
DEEP-MIBR	--	281,735	5,235,893	0.13	--
DEEP-MICR	C-1	281,760	5,235,306	0.048	--
DEEP-MIRR	R-1	281,610	5,236,556	0.0197	--
DEEP-maximum cumulatively impacted house in the modeling domain (c)	R-2	283,938	5,233,915	0.0017	--

Notes:

UTM = Universal Transverse Mercator coordinate system.

DEEP = Diesel engine exhaust particulate matter.

NO₂ = Nitrogen dioxide.

MIBR = Maximally impacted boundary receptor.

MICR = Maximally impacted commercial receptor.

MIRR = Maximally impacted residential receptor.

µg/m³ = Micrograms per cubic meter.

(a) The maximum DEEP impact at the I-1 receptor was found at 282,359.8 m (E), 5,234,755.5 m (N).

(b) The maximum DEEP impact at the I-2 receptor was found at 282,859.8 m (E), 5,234,855.5m (N).

(c) The maximum cumulative DEEP impact at the maximum cumulatively impacted house was 0.23 µg/m³.

TABLE 4-2
APRIL 2016 MODELED IMPACTS AT THE MAXIMALLY IMPACTED DEEP RISK RECEPTORS
OXFORD DATA CENTER
QUINCY, WASHINGTON

Source	70-Year Average Annual DEEP Impact ($\mu\text{g}/\text{m}^3$)					
	MIBR	MIRR (R-1)	MICR (C-1)	School (I-1)	Hospital (I-2)	R-2 (a)
Project Oxford-Only (70-yr average)	0.129	0.020	0.048	0.012	5.0E-03	1.7E-03
Dell Data Center	1.2E-03	2.0E-03	9.0E-04	1.4E-03	2.7E-03	2.3E-03
Columbia Data Center	1.7E-03	2.3E-03	1.3E-03	2.2E-03	5.1E-03	5.3E-03
State Route 28	0.021	2.8E-03	0.059	0.034	0.049	0.013
State Route 281	0.004	0.010	0.004	0.007	0.010	0.196
BNSF Railroad Line	0.016	7.6E-03	0.067	0.016	0.018	7.4E-03
Cumulative (including local background) Impacts	0.172	0.045	0.182	0.073	0.090	0.226

5 = DEEP REL ($\mu\text{g}/\text{m}^3$)	DEEP - Chronic Hazard Quotient (Theoretical Max Year)					
	MIBR	MIRR (R-1)	MICR (C-1)	School (I-1)	Hospital (I-2)	R-2 (a)
Project Oxford-Only (b)	0.083	0.013	0.031	0.007	0.003	0.001
Dell Data Center	2.4E-04	4.0E-04	1.8E-04	2.8E-04	5.4E-04	4.6E-04
Columbia Data Center	3.4E-04	4.5E-04	2.5E-04	4.3E-04	1.0E-03	1.1E-03
State Route 28	0.004	5.5E-04	0.012	6.9E-03	0.010	0.003
State Route 281	7.5E-04	2.1E-03	8.9E-04	1.5E-03	2.1E-03	0.039
BNSF Railroad Line	3.2E-03	1.5E-03	0.013	0.003	0.004	0.001
Cumulative (including local background) HQ	0.091	0.018	0.058	0.020	0.020	0.046

DEEP Cancer Risk Unit Risk Factor ($\mu\text{g}/\text{m}^3$) ⁻¹	MIBR	MIRR (R-1)	MICR (C-1)	School (I-1)	Hospital (I-2)	R-2 (a)
	Lifetime Cancer Risk per Million Population					
Project Oxford-Only	0.02	5.9	2	0.36	0.02	0.52
Cumulative (including local background) Impacts	1.3	13	6.9	2.3	0.4	68

Notes:

DEEP = Diesel engine exhaust particulate matter.

HQ = Hazard quotient.

MIBR = Maximally impacted boundary receptor.

MICR = Maximally impacted commercial receptor.

MIRR = Maximally impacted residential receptor.

R-2 = Maximum cumulatively impacted house in the modeling domain.

I-1 = Quincy Valley School.

I-2 = Quincy Valley Medical Center.

 $\mu\text{g}/\text{m}^3$ = Micrograms per cubic meter.

(a) The maximum cumulatively impacted residence is located at R-2 (283937.6 m E 5233914.5 m N).

(b) Based on theoretical maximum year emissions.

TABLE 4-3
MODELED IMPACTS AT THE MAXIMALLY IMPACTED NO₂ RISK RECEPTORS
OXFORD DATA CENTER
QUINCY, WASHINGTON

	1-hour NO ₂ Impact (µg/m ³)				
	MIBR	MIRR (R-5)	MICR (C-1)	School (I-1)	Hospital (I-2)
Project Oxford-Only	606	409	454	258	300
Dell Data Center	316	360	363	237	178
Columbia Data Center	476	292	445	407	345
State Route 28	13	47	58	35	87
State Route 281	2.4	4.1	6.7	7.1	8.8
BNSF Railroad Line	7.3	26.1	114	13	16
Cumulative (including local background) Impacts	655	655	455	527	375

470 = NO ₂ REL (µg/m ³)	Acute (1-hour) NO ₂ Hazard Quotient				
	MIBR	MIRR (R-5)	MICR (C-1)	School (I-1)	Hospital (I-2)
Project Oxford-Only	1.3	0.87	0.97	0.55	0.64
Dell Data Center	0.67	0.77	0.77	0.50	0.38
Columbia Data Center	1.0	0.62	0.95	0.87	0.73
State Route 28	2.81E-02	0.10	0.12	7.44E-02	0.18
State Route 281	5.00E-03	8.79E-03	1.42E-02	1.51E-02	1.88E-02
BNSF Railroad Line	1.54E-02	5.55E-02	0.24	2.73E-02	3.42E-02
Cumulative (including local background) Impacts	1.4	1.4	0.97	1.1	0.80

Notes:

NO₂ = Nitrogen dioxide.

HQ = Hazard quotient.

MIBR = Maximally impacted boundary receptor.

MICR = Maximally impacted commercial receptor.

MIRR = Maximally impacted residential receptor.

I-1 = Quincy Valley School.

I-2 = Quincy Valley Medical Center.

µg/m³ = Micrograms per cubic meter.

Highlighted cells indicate an HQ or cumulative HI greater than 1.

TABLE 4-4
EXPOSURE ASSUMPTIONS AND UNIT RISK FACTORS USED FOR LIFETIME CANCER RISK ASSESSMENT
OXFORD DATA CENTER
QUINCY, WASHINGTON

Receptor Type	Annual Exposure	Exposure Duration	Unit Risk Factor
Unoccupied Land	2 hours/day 250 days/year	30 years	7.3-per-million cancer risk per $\mu\text{g}/\text{m}^3$ DEEP
Residences	24 hours/day 365 days/year	70 years	300-per-million cancer risk per $\mu\text{g}/\text{m}^3$ DEEP
Schools (College Students)	36 hours/week 40 weeks/year	4 years	2.8-per million risk per $\mu\text{g}/\text{m}^3$ DEEP
Schools (High School Students)	36 hours/week 40 weeks/year	4 years	2.8-per-million risk per $\mu\text{g}/\text{m}^3$ DEEP
Schools (Elementary School Students)	36 hours/week 40 weeks/year	7 years	4.9-per-million risk per $\mu\text{g}/\text{m}^3$ DEEP
Schools (All Teachers)	40 hours/week 40 weeks/year	40 years	31-per-million risk per $\mu\text{g}/\text{m}^3$ DEEP
Churches	2 hours/week 52 weeks/year	40 years	2-per-million risk per $\mu\text{g}/\text{m}^3$ DEEP
Business	8 hours/day 250 days/year	40 years	38-per-million risk per $\mu\text{g}/\text{m}^3$ DEEP
Hospital	24 hours/day 365 days/year	1 year	4.3-per-million risk per $\mu\text{g}/\text{m}^3$ DEEP

Notes:

DEEP = Diesel engine exhaust particulate matter.

$\mu\text{g}/\text{m}^3$ = Micrograms per cubic meter.

TABLE 4-5
EXPOSURE ASSUMPTIONS AND UNIT RISK FACTORS USED FOR HEALTH IMPACT ASSESSMENT
OXFORD DATA CENTER
QUINCY, WASHINGTON

Pollutant	Agency	Non-Cancer REL ($\mu\text{g}/\text{m}^3$)	Carcinogenic URF ($\mu\text{g}/\text{m}^3$) ⁻¹
DEEP	Acute (1-hr average)	N/A	3.0×10^{-4}
	Chronic (12-month average)	5	
CO	Acute (1-hr average)	23,000	N/A
	Chronic (12-month average)	N/A	
Ammonia	Acute (1-hr average)	3,200	N/A
	Chronic (12-month average)	200	
NO ₂	Acute (1-hr average)	470	N/A
	Chronic (12-month average)	N/A	
Benzene	Acute (1-hr average)	27	2.9×10^{-5}
	Chronic (12-month average)	3	
Acrolein	Acute (1-hr average)	2.5	N/A
	Chronic (12-month average)	0.35	

Notes:

DEEP = Diesel engine exhaust particulate matter.

CO = Carbon monoxide.

NO₂ = Nitrogen dioxide.

REL = Reference exposure level.

URF = Unit risk factor.

 $\mu\text{g}/\text{m}^3$ = Micrograms per cubic meter.

N/A = Not applicable to this toxic air pollutant.

Source: California Office of Environmental Health Hazard Assessment.

TABLE 4-6
APRIL 2016 CHRONIC (NON-CANCER) HEALTH IMPACT ASSESSMENT FOR TOXIC AIR POLLUTANTS
OXFORD DATA CENTER
QUINCY, WASHINGTON

Annual Average Hazard Index (a)		MIBR (b)	MIRR (R-1)	MICR (C-1)	School (I-1)	Hospital (I-2)
DEEP (c)	Max 3-Year Annual Ambient Impact ($\mu\text{g}/\text{m}^3$)	4.2E-01	6.4E-02	1.6E-01	3.8E-02	1.6E-02
	Risk-Based Toxic Threshold Value ($\mu\text{g}/\text{m}^3$)	5				
	Hazard Quotient	8.4E-02	1.3E-02	3.1E-02	7.6E-03	3.3E-03
Ammonia (c,d)	Max 3-Year Annual Ambient Impact ($\mu\text{g}/\text{m}^3$)	2.5E+00	3.9E-01	9.5E-01	2.3E-01	9.9E-02
	Risk-Based Toxic Threshold Value ($\mu\text{g}/\text{m}^3$)	200				
	Hazard Quotient	1.3E-02	1.9E-03	4.7E-03	1.1E-03	4.9E-04
Benzene (c,d)	Max 3-Year Annual Ambient Impact ($\mu\text{g}/\text{m}^3$)	7.2E-03	1.1E-03	2.7E-03	6.5E-04	2.8E-04
	Risk-Based Toxic Threshold Value ($\mu\text{g}/\text{m}^3$)	3				
	Hazard Quotient	2.4E-03	3.7E-04	9.0E-04	2.2E-04	9.4E-05
Acrolein (c,d)	Max 3-Year Annual Ambient Impact ($\mu\text{g}/\text{m}^3$)	7.3E-05	1.1E-05	2.7E-05	6.6E-06	2.9E-06
	Risk-Based Toxic Threshold Value ($\mu\text{g}/\text{m}^3$)	0.35				
	Hazard Quotient	2.1E-04	3.2E-05	7.8E-05	1.9E-05	8.2E-06
Combined Hazard Index (HI)		0.10	1.5E-02	0.04	8.9E-03	3.9E-03

Notes:

MIBR = Maximally impacted boundary receptor.

MICR = Maximally impacted commercial receptor.

MIRR = Maximally impacted residential receptor.

I-1 = Quincy Valley School.

I-2 = Quincy Valley Medical Center.

$\mu\text{g}/\text{m}^3$ = micrograms per cubic meter.

(a) The hazard quotients for NO_2 , SO_2 , and CO are not applicable to this exposure scenario.

(b) The MIBR, MICR, and MIRR are the maximally impacted risk receptors for DEEP.

(c) Evaluated ambient impacts are the theoretical worst-case maximum impacts to account for the hypothetical situation if all 3-years of permitted operating hours were to be run within a single year.

(d) Predicted impacts based on dispersion factors and annual potential-to-emit.

TABLE 4-7
ACUTE (NON-CANCER) HEALTH IMPACT ASSESSMENT FOR TOXIC AIR POLLUTANTS
OXFORD DATA CENTER
QUINCY, WASHINGTON

Annual Average Hazard Index (a)		MIBR (b)	MIRR (R-5)	MICR (C-1)	School (I-1)	Hospital (I-2)
CO	Ambient Impact ($\mu\text{g}/\text{m}^3$)	3.8E+02	2.4E+02	2.7E+02	1.3E+02	1.6E+02
	Risk-Based Toxic Threshold Value ($\mu\text{g}/\text{m}^3$)	23,000				
	Hazard Quotient	1.7E-02	1.1E-02	1.2E-02	5.8E-03	7.1E-03
Ammonia	Ambient Impact ($\mu\text{g}/\text{m}^3$)	7.7E+01	4.9E+01	5.5E+01	2.7E+01	3.2E+01
	Risk-Based Toxic Threshold Value ($\mu\text{g}/\text{m}^3$)	3,200				
	Hazard Quotient	2.4E-02	1.5E-02	1.7E-02	8.4E-03	1.0E-02
NO ₂	Ambient Impact ($\mu\text{g}/\text{m}^3$)	6.1E+02	4.1E+02	4.5E+02	2.6E+02	3.0E+02
	Risk-Based Toxic Threshold Value ($\mu\text{g}/\text{m}^3$)	470				
	Hazard Quotient	1.3	0.87	0.97	0.55	0.64
Benzene (c)	Ambient Impact ($\mu\text{g}/\text{m}^3$)	2.3E-01	1.5E-01	1.7E-01	8.2E-02	9.9E-02
	Risk-Based Toxic Threshold Value ($\mu\text{g}/\text{m}^3$)	27				
	Hazard Quotient	8.6E-03	5.5E-03	6.2E-03	3.0E-03	3.7E-03
Acrolein (c)	Ambient Impact ($\mu\text{g}/\text{m}^3$)	2.4E-03	1.5E-03	1.7E-03	8.3E-04	1.0E-03
	Risk-Based Toxic Threshold Value ($\mu\text{g}/\text{m}^3$)	2.50				
	Hazard Quotient	9.4E-04	6.0E-04	6.7E-04	3.3E-04	4.0E-04
Combined Hazard Index (HI)		1.3	0.90	1.0	0.57	0.66
Combined HI (not including NO ₂)		5.0E-02	3.2E-02	3.6E-02	1.8E-02	2.1E-02

Notes:

DEEP = Diesel engine exhaust particulate matter.

CO = Carbon monoxide.

NO₂ = Nitrogen dioxide.

MIBR = Maximally impacted boundary receptor.

MICR = Maximally impacted commercial receptor.

MIRR = Maximally impacted residential receptor.

I-1 = Quincy Valley School.

I-2 = Quincy Valley Medical Center.

 $\mu\text{g}/\text{m}^3$ = Micrograms per cubic meter.

Highlighted cells indicate an HQ or HI greater than or equal to 1.

(a) The hazard quotients for DEEP and naphthalene are not applicable to this exposure scenario.

(b) The MIBR, MICR, and MIRR are the maximally impacted risk receptors for NO₂.

(c) Predicted impacts based on dispersion factors and annual potential-to-emit.

TABLE 4-8
JOINT PROBABILITY OF NO₂ ASIL EXCEEDANCES
OXFORD DATA CENTER
QUINCY, WASHINGTON

Exceedance Threshold Value ($\mu\text{g}/\text{m}^3$)	454			
Max. Project Impact	606			
Project Oxford --> MIBR	Assumed Power Outage Occurrence		Historical Occurrence: Grant County PUD (a)	
Hours of Power Outage per Year	24		2.5	
Contributing Source	Project Oxford	ALL	Project Oxford	ALL
Total #No. of Hrs > Threshold (in 5 years)	9	14	9	14
Average No. of Hrs > Threshold Per Year	2	3	2	3
Hourly Probability of Poor Wind Dispersion	2.05E-04	3.20E-04	2.05E-04	3.20E-04
Hourly Probability of a Power Outage	2.74E-03	2.74E-03	2.89E-04	2.89E-04
Joint Probability (per Hr) of Exceeding the Threshold During a Power Outage	5.63E-07	8.76E-07	5.94E-08	9.24E-08
Overall Probability in 1 Yr	4.92E-03	7.64E-03	5.20E-04	8.09E-04
Recurrence Interval (years)	203	131	1,922	1,235

Exceedance Threshold Value ($\mu\text{g}/\text{m}^3$)	454			
Max. Project Impact	453.8			
Project Oxford --> MICR (C-1)	Assumed Power Outage Occurrence		Historical Occurrence: Grant County PUD (a)	
Hours of Power Outage per Year	24		2.5	
Contributing Source	Project Oxford	ALL	Project Oxford	ALL
Total #No. of Hrs > Threshold (in 5 years)	0	1	0	1
Average No. of Hrs > Threshold Per Year	0	0	0	0
Hourly Probability of Poor Wind Dispersion	--	2.28E-05	--	2.28E-05
Hourly Probability of a Power Outage	2.74E-03	2.74E-03	2.89E-04	2.89E-04
Joint Probability (per Hr) of Exceeding the Threshold During a Power Outage	--	6.26E-08	--	6.60E-09
Overall Probability in 1 Yr	--	5.48E-04	--	5.78E-05
Recurrence Interval (years)	--	1,825	--	17,290

Notes:

NO₂ = Nitrogen dioxide.

ASIL = Acceptable source impact level.

MIBR = Maximally impacted boundary receptor.

MICR = Maximally impacted commercial receptor.

 $\mu\text{g}/\text{m}^3$ = Micrograms per cubic meter.

(a) The average power outage duration for Grant County PUD customers, between 2008 and 2014 was 152 minutes per year (Grant County PUD 2015).

TABLE 4-9
APRIL 2016 LIFETIME (70-YEAR AVERAGE) CANCER RISK FROM TOXIC AIR POLLUTANTS
OXFORD DATA CENTER
QUINCY, WASHINGTON

Carcinogen	Annual Emissions (Tons per Year)	ASIL ($\mu\text{g}/\text{m}^3$)	Combined Cancer Risk per Million Population
			MIRR
DEEP	0.814	0.0033	5.9
Benzene	1.61E-04	0.0345	1.1E-04
Toluene	1.04E-04	5,000	5.0E-10
Xylenes	3.21E-03	221	3.5E-07
1,3-Butadiene	2.57E-06	0.0059	1.1E-05
Formaldehyde	1.06E-06	1.7E-01	1.5E-07
Acetaldehyde	4.59E-06	3.7E-01	3.0E-07
Benzo(a)pyrene	9.01E-07	9.1E-04	2.4E-05
Benzo(a)anthracene	6.32E-06	9.1E-03	1.7E-05
Chrysene	1.43E-06	9.1E-02	3.8E-07
Benzo(b)fluoranthene	3.26E-04	9.1E-03	8.7E-04
Benzo(k)fluoranthene	1.71E-06	9.1E-03	4.6E-06
Dibenz(a,h)anthracene	5.37E-04	9.1E-04	1.4E-02
Ideno(1,2,3-cd)pyrene	1.16E-03	9.1E-03	3.1E-03
Naphthalene	7.97E-04	0.0294	6.6E-04
Combined Lifetime Cancer Risk (per Million Population)			5.94

Notes:

DEEP = Diesel engine exhaust particulate matter.

MIRR = Maximally impacted residential receptor.

ASIL = Acceptable source impact level.

TABLE 5-1
QUALITATIVE SUMMARY OF THE EFFECTS OF UNCERTAINTY
OXFORD 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 concentration
Toxicity of DEEP at low concentrations	Possible overestimate of cancer risk, possible underestimate of non-cancer hazard for sensitive individuals

Notes:

AERMOD = American Meteorological Society/US Environmental Protection Agency regulatory model.

DEEP = Diesel engine exhaust particulate matter.