DRAFT RECOMMENDED PROCEDURES FOR TOXIC AIR CONTAMINANT HEALTH RISK ASSESSMENTS

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DEQ is a leader in restoring, maintaining and enhancing the quality of Oregon's air, land and water.

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LIST OF ABBREVIATIONS

ABC	DEQ Ambient Benchmark Concentration
ADAF	Age Dependent Adjustment Factor
AERMOD	American Meteorological Society/EPA preferred air dispersion modeling program
AERSCREEN	Program to run AERMOD in screening mode
ATSAC	DEQ's Air Toxics Science Advisory Committee
ATSDR	Agency for Toxic Substances and Disease Registry
CAO	Cleaner Air Oregon
CDDs/CDFs	Chlorinated Dibenzo-*p*-dioxins and Chlorinated Dibenzofurans
CSM	Conceptual Site Model
DEQ	Oregon Department of Environmental Quality
DF	Dispersion Factor
ED	Exposure Duration
ELAF	Early-Life Adjustment Factor
EPA	U.S. Environmental Protection Agency
ER	Emission Rate
ESRE	Emission Scaled Risk Estimate
EQC	Environmental Quality Commission
HI	Hazard Index
HQ	Hazard Quotient
IRIS	EPA's Integrated Risk Information System
IUR	Inhalation Unit Risk
LRAPA	Lane Regional Air Protection Agency
MAKEMET	Program that generates a site-specific matrix of meteorological conditions for input to AERMOD or AERSCREEN
MPAF	Multipathway Adjustment Factor
MRL	ATSDR's Minimal Risk Level
NRAF	Nonresident Adjustment Factor
NSR	New Source Review
OAR	Oregon Administrative Rules
OEHHA	California's Office of Environmental Health Hazard Assessment
OHA	Oregon Health Authority
OSHA	Occupational Safety and Health Administration
PAH	Polycyclic Aromatic Hydrocarbon
PBT	Persistent, Bioaccumulative, and Toxic air contaminant
PCB	Polychlorinated Biphenyl
PPRTV	EPA's Provisional Peer-Reviewed Toxicity Value
RAL	Risk Action Level
RBC	Risk-Based Concentration
RBDM	Risk-Based Decision Making
REER	Risk Equivalent Emission Rate
RfC	Reference Concentration
SCAQMD	California's South Coast Air Quality Management District
TBACT	Toxics Best Available Control Technology
TAC	Toxic Air Contaminant
TEF	Toxic Equivalency Factor
TEU	Toxics Emission Unit
TRV	Toxicity Reference Value

1. INTRODUCTION

1.1 Purpose and Organization

This document provides DEQ's recommended procedures for conducting toxic air contaminant (TAC) risk assessments in compliance with OAR chapter 340, division 245. A risk assessment can range from a simple risk assessment using screening-type modeling (Levels 1 and 2), to a risk assessment requiring approved air dispersion models (Level 3), or finally a complex risk assessment (Level 4) that may include site-specific adjustments to exposure assumptions.

The methods to perform human health risk assessments at sources that emit toxic air contaminants in Oregon are based primarily on U.S. Environmental Protection Agency guidance (EPA 1989), and are consistent with and make reference to human health risk assessment guidance under DEQ's Cleanup Program (DEQ 2010). In general, the exposure factors and equations described in this document are sufficient for calculating exposure and risk from existing, modified, reconstructed, and new facilities.

Section 2 outlines general risk assessment concepts, including the development of the Risk Based Concentrations (RBCs) DEQ will use to assess risk from a facility. This section also provides an overview of the risk assessment process, including development of a conceptual site model, and a brief discussion on air dispersion modeling. Section 3 presents detailed discussion on conducting a risk assessment, and provides calculation methodologies and suggested guidance for each level of risk assessment. The appendices contain walk-through examples of risk assessment calculations at different levels and using different approaches, as well as a discussion of the development of Multipathway Adjustment Factors and RBCs. Also contained in the appendicies are tables of target organs for use in noncancer risk evaluations.

DEQ intends this document to serve as a guide for facilities preparing risk assessments for the Cleaner Air Oregon program by outlining approvable, recommended procedures. Additionally, DEQ strongly encourages facilities to establish and maintain an open and collaborative communication with DEQ during this process to ensure an efficient and successful risk assessment.

1.2 Process Overview

The overall recommended human health risk assessment process involves the general steps discussed below. Information on existing site conditions and the nature of properties potentially impacted by facility emissions are key prerequisites for screening steps and risk assessments.

All levels of risk assessment involve calculating three separate risk numbers: for cancer, for chronic noncancer, and for acute noncancer. The calculations use the Risk-Based Concentrations (RBCs) listed in OAR 340-245-8040 Table 4. Risks associated with individual toxic air contaminants are summed and then compared with the Risk Action Levels (RALs) in OAR 340-245-8010 Table 1 to determine what action is needed. Different RALs apply to new/reconstructed and existing facilities.

The owner or operator of the source can choose to start with any level of risk assessment and is not required to do all four levels. While each of the four levels is considered a risk assessment, Levels 1 and 2 function more as risk screening assessments that can be further refined as desired by a Level 3

or 4 risk assessment. More information about these risk assessment levels is included in Section 3. The elements of the different levels of evaluation are the following:

Level 1. This risk assessment level involves choosing dispersion factors from OAR 340-245-8050 Table 5 based on site-specific information. This includes stack height and distances to various exposure locations for stack emissions, and building height, dimensions, and distances to exposure locations for fugitive emissions. In the absence of site-specific information, you can use a default dispersion factor. The default dispersion factor is located in the upper left hand corner of each Table 5 A, B, C, and D, and uses the most conservative assumptions based on the lowest emission release height and the closest exposure location. To screen your emissions, multiply each toxic air contaminant emission rate by the dispersion factor, and compare the resulting calculated ambient air concentration with the appropriate RBC in OAR 340-245-8040 Table 4 for residential and non-residential exposure locations and acute exposure locations. Finally, compare the summed excess cancer risks and hazard indices with the RALs in OAR 340-245-8010 Table 1.

Level 2. At this level, you can use site-specific information (such as stack height, other stack parameters, and distances to various exposure locations) and perform simple modeling using EPA's AERSCREEN model or AERMOD model in screening mode to calculate ambient air concentrations for comparison with RBCs.

Level 3. At this level, you use detailed site-specific information (such as stack heights, building heights, topography, and distances to various exposure locations) and site-specific meteorological data to perform complex modeling using EPA's AERMOD model to calculate ambient air concentrations for comparison with RBCs.

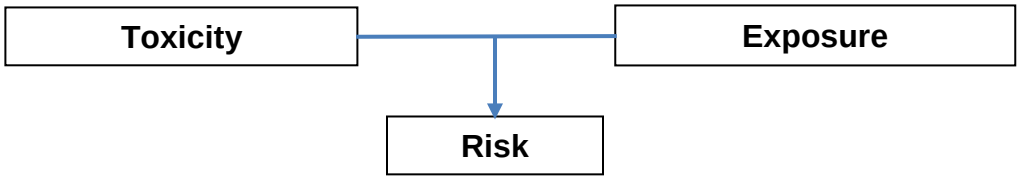
Level 4. The most comprehensive risk assessment option uses the same air dispersion modeling conducted in Level 3, with detailed site-specific information. In addition, you can consider factors to refine the exposure assessment. These factors can include modified exposure assumptions, relative bioavailability of toxic air contaminants, or multipathway considerations not covered by the values used to develop RBCs.

2. RISK ASSESSMENT OVERVIEW

2.1 Risk Assessment Concepts

The goal of the Cleaner Air Oregon program is to evaluate risk to people near facilities that emit regulated toxic air contaminants in OAR 340-245-8040 Table 4, and manage risk consistent with established Risk Action Levels and related regulatory requirements

Risk considers both exposure and toxicity:



Exposure is how much contact someone has with a toxic air contaminant. This mainly includes the concentration of the toxic air contaminant in air, typically expressed as micrograms of toxic air contaminant per cubic meter of air ($\mu g/m^3$). The greater the concentration of a toxic air contaminant in air, the greater the risk. Other considerations for exposure include how long the exposure occurs, which for chronic exposure includes both exposure frequency, such as 8 hours per day for workers, and exposure duration, such as 25 years. Acute effects are evaluated using 24-hour averages for exposure to a toxic air contaminant at locations where people may spend several hours of one day (OAR 340-245-0020(3) and (4)).

Toxicity is a measure of how harmful a toxic air contaminant is if someone is exposed to it. The two general types of toxic effects, noncancer and cancer, are evaluated separately. For noncancer effects, we assume there is a threshold below which toxic effects are unlikely to occur. This level is called a reference concentration (RfC).

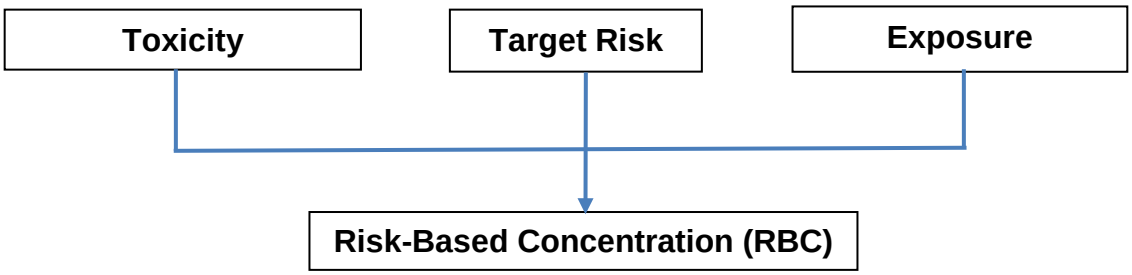
For cancer effects, the assumption is that there is no threshold for adverse effects. Although the risk at a very low concentration of a carcinogen may be very low, we assume it is not zero. Because of this assumption, the toxicity of carcinogens is given not as a threshold concentration, but instead as a probability of getting cancer as a result of being exposed continuously to a concentration of 1 $\mu g/m^3$. This value is called the Inhalation Unit Risk (IUR) value. For ease of use in assessing risk in the Cleaner Air Oregon program, DEQ converted IURs to concentrations using a target excess lifetime cancer risk level of one in one million.

Roughly speaking, if one million people are exposed to an excess cancer risk of one in one million, we would expect about one additional cancer in the population, compared with the already-existing nationwide background level of approximately 400,000 cancers per million people. We expect the number of people exposed to toxic air contaminants from a single facility to be far less than one million, so the calculated excess cancers in the exposed population (called a cancer burden) as a result of emissions from a facility is expected to be much less than 1. To be clear, in the Cleaner Air Oregon program DEQ looks at individual probabilities resulting from exposure to toxic air contaminants at specific exposure locations, and not a total population cancer burden.

Toxic air contaminants may have both noncancer and cancer effects. As a general term, we use Toxicity Reference Value (TRV) to mean either the noncancer reference concentration, or a

concentration based on the cancer inhalation unit risk value. Toxicity reference values only consider risks from direct inhalation of toxic air contaminants in air. They do not consider risks from cross-media exposure, such as eating vegetables grown in soil where toxic air contaminants settled out of air into the soil and were taken up into the vegetables. To address cross-media risk in the development of RBCs, an adjustment is made to certain toxicity reference values as described in Section 2.2.2, and presented in Table C-1 in Appendix C. Other adjustments to TRVs are discussed in Section 2.2.

DEQ developed RBCs for each toxic air contaminant using standard exposure and toxicity assumptions, generally from EPA, and selected risk levels discussed below. DEQ developed RBCs using the same toxicity and exposure information used in a risk assessment, along with target risk levels. There are separate RBCs for cancer risk, chronic noncancer risk, and acute noncancer risk.



For establishing RBCs for noncarcinogens, target risk is set at a hazard quotient, or HQ, of 1. A hazard quotient is the ratio of the concentration of toxic air contaminant in air to the RBC. An HQ below 1 means there is little likelihood that even sensitive people will experience serious adverse health effects. To establish RBCs for carcinogens, target risk is set at an excess cancer risk of one in one million.

The choices of a HQ of one and an excess lifetime cancer risk level of one in one million are for convenience in establishing RBCs. These levels are different from acceptable risk levels, or RALs. OAR 340-245-8010 Table 1 shows RALs, which are the levels at which facilities must take action to address risk. DEQ developed separate RALs for new/reconstructed and existing facilities.

2.2 Risk-Based Concentrations

TRVs serve as the basis for RBCs. To establish TRVs for each toxic air contaminant, DEQ relied on the scientific conclusions of agencies like the U.S. Environmental Protection Agency and the Agency for Toxic Substances and Disease Registry. DEQ used the authoritative sources of chronic and acute TRVs identified in Appendix B. Section 2.5 provides additional information on toxicity.

Three adjustments of TRVs were made, if appropriate, to calculate RBCs. The first addresses scenario-specific consideration of exposure frequency and duration that are appropriate for chronic exposure scenarios. Another adjustment considers deposition and bioaccumulation of toxic air contaminants, which involves exposure other than by inhalation alone. This is a multipathway adjustment. The third adjustment is for early-life exposure to toxic air contaminants that exhibit greater toxicity to infants and children. These three types of adjustments are described in more detail in the sections below.

Adjustment factors for Risk-Based Concentrations are provided in Table C-1. Appendix C shows how DEQ used the adjustment factors to develop the RBCs shown in OAR 340-245-8040 Table 4. Adjustment factors apply only to chronic exposure. None of the adjustment factors is appropriate or necessary for acute RBCs because of the short period of exposure being considered.

2.2.1 Exposure Frequency and Duration Considerations

Residential exposure assumes continual, long-term exposure. Because continual, long-term exposure is the basis of most chronic toxicity values, chronic TRVs are most directly applicable for residential exposure. For other types of exposure, including shorter term, nonresidential child exposure such as at schools, and worker exposure at commercial or industrial facilities, adjustments to TRVs are needed to take into consideration differences in exposure frequency and duration. Adjustment factors for chronic exposure are discussed in Appendix C.

2.2.2 Multipathway Adjustment Factors

If your facility emits persistent, bioaccumulating, and toxic (PBT) air contaminants, it is important to consider exposure through pathways other than air. For PBT toxic air contaminants, DEQ considered multipathway effects on residents in developing RBCs, which are used in all levels of risk assessment. DEQ developed multipathway adjustment factors (MPAFs) for residential exposure scenarios that consider:

- Inhalation of toxic air contaminants in air
- Deposition of airborne toxic air contaminants to backyard soil
- Contact with soil by incidental ingestion and dermal exposure
- Uptake into garden vegetables, and ingestion of vegetables, and
- Bioaccumulation into women, and infant ingestion of breastmilk

For nonresidential exposure, different MPAFs are used because some considerations, such as uptake into garden vegetables, are not appropriate. MPAFs do not include exposure scenarios that incorporate airborne deposition of toxic air contaminants to:

- Agricultural land
- Livestock grazing areas
- Drinking water reservoirs
- Bodies of water used for fishing

If PBT toxic air contaminant emissions from your facility could impact the above areas, DEQ may require a more complex evaluation of risk considering multipathway exposure even if emissions screen out at the Level 1, 2, or 3 risk assessments using default MPAFs [OAR 340-245-0050(12)]. DEQ will consider toxic air contaminant deposition rates, and may consider other factors such as size of impacted area and degree of use. DEQ recommends that the process for calculating risk from PBT toxic air contaminants start with development of a conceptual site model more extensive than the default assumptions. This additional multipathway risk evaluation may sufficiently address DEQ's concerns without requiring the need for a Level 4 risk assessment.

2.2.3 Early-Life Exposure Adjustment Factors

Carcinogens that act by a mutagenic mode of action can have greater toxicity during early-life stages (EPA 2005a). In these cases, we need to adjust the cancer TRV. Currently, the toxic air contaminants of primary interest for consideration of early-life exposure are listed in Table C-1 with early-life adjustment factors (ELAFs). Appendix D shows the derivation of ELAFs. As more information becomes available, EPA may determine that additional carcinogens act by a mutagenic mode of action. If this happens, DEQ may undertake rulemaking to expand the list of toxic air contaminants for which ELAF values are needed, and revise RBCs accordingly.

2.3 Risk Assessment Process

A number of elements are important to conducting a risk assessment. The first risk assessment elements are important even at a simple evaluation level. Some of the later elements are important only in a Level 4 risk assessment. The first element is the establishment of a conceptual site model to understand the various toxic air contaminant emission sources and types of exposure areas near your site. To evaluate the types of exposure areas, DEQ recommends that you identify the land use near your facility. If significant airborne deposition to water is expected, you should also identify uses of water. A conceptual site model will establish exposure locations, which are necessary for developing a modeling protocol (see Section 2.4.1). Therefore, the modeling protocol should include the conceptual site model. The modeling protocol (for all levels) and risk assessment workplan (for Levels 3 and 4) need to be approved by DEQ prior to preparing the risk assessment.

The conceptual site model should include a list of chemicals being emitted from the facility. This list is derived from the emission inventory, which is the first submittal required under the CAO program. DEQ provides assistance on how an emission inventory should be prepared and presented.

Once you establish a conceptual site model, you can proceed with the main exposure and toxicity assessments, and risk characterizations. Finally, you should include an uncertainty section to document qualitative or quantitative evaluations of variability and uncertainty. In particular, Level 3 and Level 4 risk assessments should include a discussion of the uncertainty associated with emitted toxic air contaminants or other chemicals for which there are no RBCs.

DEQ may approve a risk assessment that follows the approach using RBCs presented here, or one that follows the traditional method where risk is calculated without benefit of RBCs (EPA 1989). DEQ recommends using the RBC approach, given the relative simplicity of the method and DEQ's confidence in the appropriateness of the default values and equations.

A risk assessment includes:

- A problem formulation step to determine a conceptual site model (CSM) describing toxic air contaminant releases and relevant exposure scenarios based on current exposure populations;
- An exposure analysis, which includes quantifying exposure concentrations based on the CSM, selecting exposure model equations, and selecting exposure factor values;
- A toxicity analysis evaluating the inherent toxicity of toxic air contaminants;
- A risk characterization combining the results of the exposure and toxicity analyses to evaluate risk; and
- A quantitative or qualitative uncertainty analysis covering all aspects of the risk assessment.

If you document the risk assessment results in a clear and consistent manner, it will be easier for DEQ staff to review it quickly. To further expedite review of the risk assessment, we recommend that you provide DEQ with electronic copies of spreadsheets of data and calculations with functioning (unlocked) formulas as part of the documentation.

2.3.1 Conceptual Site Model

Before you proceed to the risk assessment, it is important to have a detailed understanding of the locations and configurations of all toxic air contaminant Toxics Emissions Units (TEUs), exposure pathways, routes of exposure, and types of exposure locations near your facility. A good way of presenting a conceptual site model (CSM) is in a chart, although for most air emission evaluations a brief narrative should be sufficient. If your facility emits PBT toxic air contaminants, and therefore may be required to conduct a multipathway risk assessment, we recommend you describe the site with a

more extensive CSM. Figure 2 in DEQ's Risk Assessment Guidance for the Cleanup Program (DEQ 2010) provides an example of a multipathway conceptual site model.

A high-quality CSM should combine information on toxic air contaminants, exposure locations, and exposure pathways to summarize relevant site information for use in the risk assessment. If land is zoned for uses allowing residents, include residential exposure, except as described below and in the CAO rules (OAR chapter 340, division 245). You should consider reasonably likely exposure scenarios based on location. For example, in farmland where a residence is allowed, include exposure to any current houses. However, it is not necessary to consider an unlikely future addition of a house in an agricultural field.

For areas zoned only for residential use, evaluate residential exposure to the entire area. If you know that people do not actually live in the area, you can provide documentation to DEQ that there is no current residential use in the area. If DEQ concludes that the documentation is adequate to rebut the presumption of residential use, you can adjust your exposure assessment accordingly. However, you must annually demonstrate that the excluded zoned areas continue to not be used in the manner allowed by the land use zoning.

Knowing how nearby land and water are being used is an important starting point for identifying potentially exposed populations for a risk assessment. For complex facilities, especially those emitting PBT toxic air contaminants, it may be useful to follow DEQ's guidance on land and water use determinations (DEQ 1998a, DEQ 1998b). A specific combination of exposure locations, exposure routes, and land and water uses can be described as an exposure scenario. Once you have determined potential risks for the set of land and water use designations appropriate to the facility, any changes to designations means that risks should be re-evaluated in some manner. The key point is that if land and water uses change without a reassessment of risk appropriate for that site, the risk assessment may no longer be accurate.

2.4 Exposure Assessment and Air Dispersion Modeling

Estimation of exposure involves the identification of exposure pathways, scenarios, and routes. The initial identification of these elements is in the conceptual site model, which should be included in the modeling protocol. An exposure pathway is the course a toxic air contaminant takes from a source to an exposed organism (EPA 1989). Exposure scenarios (designated "residential", "industrial", etc.) are comprised of one or more exposure routes appropriate to the potentially exposed population. An exposure route is the way a toxic air contaminant comes in contact with a person. Inhalation is the primary exposure route for air emissions, although other routes (ingestion, dermal contact) may be important for PBT toxic air contaminants.

DEQ developed RBCs for the following exposure scenarios:

- Residential exposure, which includes long-term exposure to children and adults.
- Nonresident adult exposure, which includes workers in office buildings, commercial buildings, or industrial facilities.
- Nonresident child exposure, which includes schools and daycare facilities.
- Acute exposure, which includes areas where people may spend all or a portion of a day, such as parks, sports facilities, or agricultural fields.

Level 1, 2, 3 and 4 risk assessments include evaluating potential exposures for all relevant exposure scenarios through some form of air dispersion modeling, from lookup tables, simple models, or complex models.

When completing a Level 4 risk assessment for a source that emits PBT toxic air contaminants, additional scenarios such as agricultural or recreational use may be relevant. Details about how to evaluate these exposure scenarios are not provided in this document, and we recommend discussing them with DEQ prior to submitting a modeling protocol and a risk assessment work plan.

2.4.1 Air Dispersion Modeling

This section provides a brief overview of air dispersion modeling and its relationship to risk assessments. More complete recommended protocols for modeling are found in DEQ's *Recommended Procedures for Air Dispersion Modeling* (DEQ 2019).

A primary element of an exposure assessment for toxic air contaminants is air quality dispersion modeling. As it provides exposure concentrations, dispersion modeling underlies the analyses at all levels of the risk assessment, from Level 1 through Level 4. These exposure concentrations, and their comparison to the RBCs, are the foundation of the risk assessment. Essentially, a dispersion model is a mathematical approximation of the physical, and sometimes chemical, processes in the atmosphere that disperse emissions, and calculates air concentrations of toxic air contaminants. Dispersion modeling estimates these concentrations at specific geographic points called modeling receptors. These modeling receptors can be considered virtual monitors, and the dispersion model calculates concentrations at these locations to simulate measurements as if made by real monitors. Modeling receptors can be positioned to coincide with exposure locations, for example residential or worker areas. Modeling receptors are typically arrayed in a grid, although they can also be positioned at specific locations, such as houses or schools. Modeling receptors are virtual points in space and should not be confused with the term "receptor" commonly used to identify humans in a risk assessment..

There are four risk assessment levels. The role of dispersion modeling for each level is briefly described below.

Level 1
For Level 1, DEQ developed lookup tables of dispersion factors (OAR 340-245-8050 Table 5) that are based on pre-run modeling results, so it is not necessary for facilities to conduct modeling. Facilities can calculate concentrations for comparison to the RBCs to assess risk using emissions and dispersion factors obtained from the tables.

Level 2
Risk assessments at Level 2 require the direct use of the dispersion model AERMOD, or other approved model. AERMOD is an EPA approved dispersion model for regulatory modeling, and is the primary refined dispersion model for the Cleaner Air Oregon program. AERMOD-MAKEMET and AERSCREEN are screening versions of AERMOD using worst case screening meteorology. There are distinct advantages to using AERMOD-MAKEMET, including the ability to model multiple emission points in in a single model run. AERSCREEN requires separate runs for each emission unit, and may be more conservative since the individual maximum concentrations from single runs must be added to get a total concentration. In addition, if after the Level 2 analysis a Level 3 analysis is preferred, the AERMOD-MAKEMET model input file can be re-used for a full AERMOD analysis with the replacement of MAKEMET data with actual meteorology. DEQ recommends AERMOD-MAKEMET for the Level 2 analysis.

Levels 3 and 4
For risk assessment Levels 3 and 4, the full AERMOD model should be used with actual representative terrain information and a gridded array or field of modeling receptors where concentrations will be evaluated.

Modeling Protocol and Modeling Report

Prior to conducting air dispersion modeling, the owner or operator must prepare and get DEQ approval of a modeling protocol. Detailed information on developing a modeling protocol and for using AERMOD-MAKEMET, AERSCREEN, and their pre and post-processors, is in DEQ's *Recommended Procedures for Air Dispersion Modeling* (DEQ 2019). After the modeling work plan is approved and implemented, a report must be prepared providing the results of the modeling. Because air dispersion modeling is the exposure assessment element of the risk assessment, the modeling report can be an attachment to the risk assessment report. The level of detail required in the modeling report varies by the risk assessment level selected.

2.4.2 Use of Air Monitoring Data in Risk Assessments

You may request to conduct ambient air monitoring to supplement air modeling after completion of an initial Level 3 or Level 4 risk assessment. However, there are a number of complexities to using air monitoring data in a risk assessment. The presence of multiple sources of toxic air contaminants near the facility can complicate ambient monitoring results. This requires simultaneous monitoring upwind and downwind of a facility. This is further complicated by varying wind directions over the year. A year of monitoring results may reasonably provide an annual average concentration at the monitoring station, suitable for comparison with chronic RBCs; however, it is far more difficult to determine the highest daily concentration that could occur at a monitoring location. This uncertainty could underestimate acute risks.

Ambient monitoring would likely take a minimum of 1.5 years to: 1) develop an adequate monitoring protocol, 2) receive DEQ approval for monitoring, and 3) obtain and deploy sampling equipment. Another six months may be required to analyze the data, develop conclusions, and obtain DEQ approval of the conclusions in a final, revised risk assessment based on the monitoring data. The monitoring protocol should include data quality objectives, and describe how exposure concentrations will be used to evaluate risk in a revised risk assessment.

In consideration of the above complexities, if DEQ approves monitoring results, DEQ may allow the results to be used as part of comparing facility risk with Risk Action Levels. Sometimes source testing and ambient air monitoring will result in non-detect values for toxic air contaminants. Non-detect values can be handled according to the approach presented in Appendix G.

2.5 Toxicity Assessment

The purpose of the toxicity assessment is to compile toxicity data for the toxic air contaminants a facility emits, and to estimate the relationship between the amount of exposure to a toxic air contaminant and the likelihood of adverse effects. You should evaluate the potential cumulative cancer risks and noncancer risks from all toxic air contaminants your facility emits. In most cases, facilities performing a toxic air contaminant risk assessment can use the RBCs listed in OAR 340-245-8040 Table 4 to assess the toxicity of toxic air contaminants they emit, and will not need the additional information in this section. However, the following information may be useful to facilities performing a Level 4 risk assessment or wanting to evaluate noncancer effects by target organ or organ system in a lower level risk assessment.

2.5.1 Assessment of Noncancer Health Effects

The potential for noncancer health effects, such as organ damage, immunological effects, birth defects, or skin irritation, is assessed by comparison with what EPA calls a Reference Concentration (RfC) in

units of µg/m^3 or mg/m^3. The federal Agency for Toxic Substances and Disease Registry calls these concentrations Minimal Risk Levels (MRLs), and California's Office of Environmental Health Hazard Assessment calls them Risk Exposure Levels (RELs). For the purpose of this document, we will use the term noncancer toxicity reference value (TRV) for the selected RfC, MRL, or REL (see Appendix B). Cleaner Air Oregon rules require that risk assessments performed for the program use the TRVs provided in OAR 340-245-8030 Table 3.

A noncancer TRV is considered a threshold below which adverse effects are not likely even in sensitive groups. Often, TRVs are based on data from test animals. Because the goal of human health risk assessments is to protect humans, including sensitive humans, toxicologists use uncertainty factors to develop reference concentrations to ensure that the levels are protective of sensitive people.

For simplicity, noncancer effects are evaluated by summing hazard quotients, as discussed in Section 3. The sum of hazard quotients for multiple toxic air contaminants is known as a hazard index. In some cases, this will be an overestimate of risk if the toxic air contaminants act on different organ systems such that the effects are not additive. If your noncancer hazard index is greater than 1, you can refine the evaluation by summing ratios of toxic air contaminants with effects on the same organ system. Note that many toxic air contaminants cause effects on more than one organ. DEQ and OHA determined that the health effect, or health effects, used to derive the TRV should be considered when determining whether to include a toxic air contaminant in an organ-specific hazard index evaluation. Chemicals may cause effects on other organs at concentrations higher than the TRV, but there is greater uncertainty associated with quantifying these effects, and it is not necessary to include them in this analysis. Some TRVs are set based on impacts to more than one target organ. In these cases, assess risk to all target organs that are the basis for the TRV. Appendix F contains suggested tables of applicable organ systems for toxic air contaminants that will be acceptable to DEQ. Table F-1 is for chronic effects, and Table F-2 is for acute effects.

2.5.2 Assessment of Cancer Effects

For cancer effects, the assumption is that there is no threshold for adverse effects. That is, we assume that exposure to even very small concentrations of the toxic air contaminant could contribute a small amount towards cancer risk. Because of this assumption, the toxicity of carcinogens is given not as a threshold concentration, but instead as a probability of getting cancer when exposed continuously to a concentration of 1 µg/m^3. This value is called the inhalation unit risk (IUR) value, in units of risk per microgram per cubic meter (µg/m^3)$^{-1}$. For ease of use in assessing risk in the CAO program, IURs were converted to TRVs using a target excess cancer risk level of one in one million. Therefore, each TRV for cancer risk represents a one-in-one-million excess cancer risk.

2.5.3 Assessment of Toxic Air Contaminant Mixtures

For some chemical classes, it is preferable to evaluate risk as a single value for the entire class because the class exhibits toxicity by the same mechanism. DEQ's recommendations on how to conduct evaluations for two important chemical classes are provided below. Appendix E presents details about how to address chemical classes.

Toxicity of CDDs/CDFs and Dioxin-like PCBs

Consistent with EPA, DEQ recommends use of toxicity equivalency factors (TEFs) to evaluate toxic effects of chlorinated dibenzo-*p*-dioxins (CDDs), polychlorinated dibenzofurans (CDFs), and co-planar (dioxin-like) chlorinated biphenyl (PCB) congeners relative to the toxicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD). Concentrations of congeners are multiplied by their TEFs to estimate the toxicity of these congeners relative to 2,3,7,8-TCDD; the resulting concentrations may be summed into a total 2,3,7,8-TCDD toxic equivalent (TEQ) concentration. EPA and DEQ use World Health Organization TEFs for humans from Van den Berg 2006.

Toxicity of Polycyclic Aromatic Hydrocarbons

DEQ recommends use of TEFs to evaluate cancer risk of polycyclic aromatic hydrocarbons (PAHs) relative to the toxicity of benzo[a]pyrene. Concentrations of other PAHs are multiplied by their TEFs to estimate their toxicity relative to benzo[a]pyrene; the resulting concentrations may be summed into a total benzo[a]pyrene toxic equivalent concentration. TEFs for humans are provided in Appendix E. Typically, however, IURs and slope factors based on the TEFs are available from EPA for the individual PAHs, so an evaluation of total carcinogenic PAHs using TEFs is not necessarily required.

2.6 Uncertainty Evaluation

CAO rules require that a quantitative or qualitative uncertainty evaluation be included in a Level 3 and Level 4 risk assessment. In this section, uncertainty in the exposure analysis (including the conceptual site model and air dispersion modeling), toxicity analysis, and risk characterization results should be evaluated qualitatively or quantitatively (if possible). For example, the residential assumption of continual 24 hours/day exposure may overestimate risk at some residences. If there are no RBCs for some of the emitted chemicals at a facility, this will result in an underestimation of risk. This evaluation will allow DEQ to consider the uncertainty associated with the results of the risk assessment. In some cases it may be helpful to collect additional data to reduce uncertainty related to emissions and associated risk.

3. CONDUCTING LEVEL 1 THROUGH LEVEL 4 RISK ASSESSMENTS

3.1 Introduction

To conduct a risk assessment, you need to calculate concentrations of toxic air contaminants at the exposure locations identified in the conceptual site model. You may assess risk under the CAO rules without conducting dispersion modeling for emissions from your facility by using the Level 1 Risk Assessment Tool of dispersion factors (Section 3.2). If you are unable to use a Level 1 approach, or want to have a more refined assessment, then you may complete an assessment using the Level 2 simple model to calculate toxic air contaminant concentrations (Section 3.3).

Level 1 and Level 2 risk assessments include conservative assumptions that are very likely to overestimate risk. If you want to further refine your risk assessment and eliminate these conservative assumptions, you may conduct more detailed site-specific air dispersion modeling (Levels 3 and 4). This will allow you to more accurately quantify the actual risks posed by toxic air contaminants at your facility in order to create a more site-specific evaluation of potential exposure.

The key element of a Level 3 or Level 4 risk assessment is the use of more sophisticated air dispersion modeling. This is discussed in Section 3.4. If you decide to undertake a more detailed evaluation beyond air dispersion modeling, or if DEQ requires consideration of non-inhalation pathways that are important and not covered by the assumptions incorporated into RBCs, a Level 4 risk assessment will be required (Section 3.5). You are required to prepare a risk assessment work plan and modeling protocol for either a Level 3 or 4 risk assessment, and you must receive DEQ approval before performing the risk assessment. The results of Level 3 and Level 4 risk assessments are estimates of risk, not measurements of actual risk.

Figure 1 provides an overview of the exposure modeling and risk assessment process.

3.1.1 Emission Rate Determination

A common element in all levels of risk assessment is the estimation of emission rates for toxic air contaminants identified on the emission inventory. DEQ has a form available for completing the emission inventory, with instructions to assist facilities. For the emission rates at any level of risk assessment, select from among the following:

- All sources must evaluate risk based on actual emissions. If you want to be permitted at a requested Potential to Emit (PTE) that is different from your actual emissions, you must assess risk at the requested level of emissions.
- Requested PTE activity levels must be included, along with your actual activity levels, in your submitted emission inventory as noted in OAR 340-245-0040(3)(a)(B) and (3)(b)(B), and must be approved by DEQ using criteria outlined in OAR 340-245-0110(2)(b).

- In order to evaluate if you are a *de minimis* source, you must assess toxic air contaminant emissions at the capacity to emit, as defined in OAR 340-200-0020(19).

Note that DEQ will evaluate and approve each facility's PTE when reviewing emission inventories. A source has the choice to have DEQ set a Source Risk Limit based on the source's actual emissions, existing PTE used to calculate Plant Site Emission Limits, or a requested PTE (as noted above) for toxic air contaminants.

In some cases, it will be important to clearly identify the form of the chemical being emitted, because there can be large differences in the toxicity of different forms of chemicals. This is also discussed in Section 4.2.2.

3.1.2 Comparison of Air Concentrations with RBCs

Another common element in all levels of risk assessment is the comparison of modeled or measured air concentrations with RBCs. How air concentrations are estimated varies by the level of risk assessment, as discussed below for Levels 1 through 4 in Sections 3.2 to 3.5.

The evaluation should consider total air emissions from all non-exempt toxic emission units (TEUs) at the facility. In Level 1 (using the lookup table) and Level 2 (using simple modeling), air concentrations will be calculated for each TEU. You need to sum these concentrations to get the total risk from the facility. In Levels 3 and 4, the more complex modeling evaluates emissions from all TEUs at the same time, without the need for later summation of calculated concentrations. Air concentrations need to be estimated for all applicable exposure locations (residential, commercial/industrial, school/daycare, and acute).

Once an air concentration for each chemical is estimated at all relevant exposure locations, divide the maximum calculated air concentration by the respective RBC for that toxic air contaminant from OAR 340-245-8040 Table 4. This will determine the potential risk for that single toxic air contaminant emitted from a single TEU. Sum the results for all the relevant toxic air contaminants, and then sum the results for all the different TEUs to get a facility risk. You should calculate a facility risk for each of the different types of RBCs (chronic cancer, chronic noncancer, and acute noncancer).

In summary, for each exposure location, calculate the following:

$$Excess\ Cancer\ Risk = \sum_{j=1}^{TEU\ m} \sum_{i=1}^{chemical\ n} \frac{ltConc_{ij}}{caRBC_i}$$

$$Chronic\ Hazard\ Index = \sum_{j=1}^{TEU\ m} \sum_{i=1}^{chemical\ n} \frac{ltConc_{ij}}{ncRBC_i}$$

$$Acute\ Hazard\ Index = \sum_{j=1}^{TEU\ m} \sum_{i=1}^{chemical\ n} \frac{stConc_{ij}}{acuteRBC_i}$$

Where:
Excess cancer risk = probability of developing cancer (above background rate), expressed as a per million rate
Chronic hazard index = sum of hazard quotients used to evaluate chronic noncancer health risk (can be calculated separately by target organ)
Acute hazard index = sum of hazard quotients used to evaluate acute noncancer health risk (can be calculated separately by target organ)
ltConcij = Calculated long-term (annual) concentration of toxic air contaminant i from emission source j
stConcij = Calculated short-term (daily) concentration of toxic air contaminant i from emission source j
caRBCi = Cancer Risk-Based Concentration for toxic air contaminant i
ncRBCi = Noncancer Risk-Based Concentration for toxic air contaminant i
acuteRBCi = Acute noncancer Risk-Based Concentration for toxic air contaminant i

Finally, compare these calculated total risk values with the RALs in OAR 340-245-8010 Table 1.

Risk Action Levels

RALs are levels of risk that prescribe the actions a regulated facility are required by DEQ to undertake under Cleaner Air Oregon rules. As risks from the source increase, so do the more corrective actions the facility is required to take, as specified by the applicable RAL.

The RALs reflect the challenges existing facilities could face in retrofitting existing equipment to meet new lower emissions requirements under the Cleaner Air Oregon program. Cleaner Air Oregon rules include higher RALs for existing facilities than for new or reconstructed facilities, which are easier to design with a target emission rate in mind.

In recommending RALs to the Environmental Quality Commission (EQC), DEQ considered risk levels used in decision making by federal agencies like the EPA and the Agency for Toxic Substances and Disease Registry. DEQ also considered risk action levels used by other states that already have health risk-based industrial air toxics programs. In addition, DEQ considered the overall non-industrial level of risk from air toxics in Oregon. Finally, DEQ considered risk benchmarks set in Oregon statute following passage of Senate Bill 1541 in the 2018 Oregon Legislature.

Cleaner Air Oregon considers risk for three categories: chronic cancer, chronic noncancer, and acute noncancer. Risk from toxic air contaminants that contribute to each of those categories of risk are calculated and compared against the appropriate RAL separately. Cancer and noncancer risks are calculated and expressed differently. The RALs for Cleaner Air Oregon reflect those differences in that there are separate sets of RALs for cancer and noncancer risks. RALs are shown in OAR 340-245-8010 Table 1.

Senate Bill 1541 included a January 1, 2029 sunset provision for its requirements that set minimum levels for the existing source TBACT (toxics best available control technology) level and Risk Reduction Level. After that date, the Environmental Quality Commission may establish new risk levels for those RALs through rulemaking. The TBACT RALs could be set at values not lower than 25 in one million excess cancer risk and a hazard index value of 1.

3.1.3 Alternative Modeling Approach for Risk Calculations

Standard Approach

The general approach for calculating risk is to:

- Develop a toxic air contaminant emission rate
- Apply a dispersion factor from a lookup table, or use a computer model to calculate an air concentration, and
- Divide the concentration by an RBC to calculate risk.

This is shown by the following sets of equations:

Level 1 Lookup Table

$$ER\ (lb/yr) \times DF\ (\mu g/m^3\ per\ lb/yr) = C_{air}\ (\mu g/m^3)$$

$$C_{air}\ (\mu g/m^3) / RBC\ (\mu g/m^3) = Risk\ (per\ million\ cancer\ risk\ or\ hazard\ quotient)$$

Level 2, 3, or 4 Modeling

$$ER\ (g/s) \rightarrow AERMOD \rightarrow C_{air}\ (\mu g/m^3)$$

$$C_{air}\ (\mu g/m^3) / RBC\ (\mu g/m^3) = Risk\ (per\ million\ cancer\ risk\ or\ hazard\ quotient)$$

Where:
ER = emission rate (lb/yr, lb/day, or g/s)
DF = dispersion factor ($\mu g/m^3$ per lb/yr, or $\mu g/m^3$ per lb/day)
C_{air} = air concentration ($\mu g/m^3$)
RBC = risk-based concentration ($\mu g/m^3$)

If actual emission rates are used in AERMOD or another air dispersion model, the resulting concentration output in $\mu g/m^3$ is available for each toxic air contaminant. For convenience in modeling, typically a unit emission rate of 1 gram/second (g/s) is used. The resulting air concentration must be converted for each toxic air contaminant for a given emission unit. These calculations need to be performed in a spreadsheet or database after modeling. Using actual emission rates, or a unit emission rate with post-processing, will provide calculated air concentrations, not risk. To calculate risk, an additional calculation of dividing the concentration by the appropriate RBC is needed for each toxic air contaminant at each appropriate exposure location (residential, non-residential child, non-residential adult, and acute).

Alternative Approach

There is an alternative method that will require additional model runs, but will greatly reduce the post-modeling calculation effort. The above equations for risk can be equivalently expressed as:

Level 1 Lookup Table

$$[ER\ (g/s) / RBC\ (\mu g/m^3)] \times DF\ (\mu g/m^3\ per\ g/s) = Risk\ (per\ million\ cancer\ risk\ or\ hazard\ quotient)$$

Level 2, 3, or 4 Modeling

$$\frac{\text{ER (g/s)} \rightarrow \text{AERMOD} \rightarrow C_{air}\ (\mu g/m^3)}{\text{RBC } (\mu g/m^3)} = \text{Risk}$$

ER (g/s) / RBC ($\mu g/m^3$) → AERMOD → Risk (per million cancer risk or hazard quotient)

For a Level 1 evaluation, the risk calculations are generally simple, and there is no need to use another approach. To implement this alternative approach with AERMOD dispersion modeling in Levels 2, 3, and 4, emission rates for each toxic air contaminant at each emission unit are normalized to risk by dividing by the appropriate RBC. This calculation needs to be performed for each toxic air contaminant, at each emission unit, for all relevant exposure locations (RBCs). The result is a Risk Equivalent Emission Rate (REER) in units of g/s per $\mu g/m^3$.

ER (g/s) / RBC ($\mu g/m^3$) = REER (g/s per $\mu g/m^3$)

REER (g/s per $\mu g/m^3$) → AERMOD → Risk (per million cancer risk or hazard quotient)

The calculation of a REER normalizes the emission rate to risk, either an excess cancer risk of one in one million for carcinogens, or a hazard quotient of 1 for noncarcinogens. Because REER is directly proportional to risk, REERs for the various toxic air contaminants can be added together at each exposure unit, unlike regular emission rates. After running a model such as AERMOD, the concentration results reported as $\mu g/m^3$ are now equivalent to units of risk (risk per million for carcinogens, and hazard index for noncarcinogens). This approach greatly simplifies risk calculations. DEQ considers this method mathematically equivalent to modeling concentrations and then performing separate risk calculations. Appendix A provides example calculations for both approaches. The approaches are also presented in DEQ's *Draft Recommended Procedures for Air Dispersion Modeling* (DEQ 2019).

In summary, there are two approaches to modeling risk. In the standard approach, a unit emission rate is modeled using multiple source groups, and the risk calculation is then calculated in spreadsheets or databases. In the alternative approach, a risk-equivalent emission rate is used to model risk directly. In this case, the risk calculation is pre-processed prior to air dispersion modeling by normalizing the emission rate to risk. There are distinct advantages to modeling using the alternative approach: 1) it is computationally more efficient to pre-process risk calculations for relatively few emission units and toxic air contaminants than to post-process the risk calculations at hundreds of modeling receptors identified as exposure locations, and 2) areas of highest risk for each exposure location are identified immediately in the model output, and subsequent analysis can focus on locations and exposure locations of most concern. For these reasons, DEQ recommends using the alternative approach to directly model risk.

3.2 Modeling Protocol and Risk Assessment Work Plan

As discussed in Section 2.4.1, prior to conducting air dispersion modeling, the owner or operator must prepare a modeling protocol. This applies to modeling at all levels of risk assessment. In addition, you are required to prepare a risk assessment work plan for Level 3 and Level 4 risk assessments. Because the modeling protocol covers the exposure assessment portion of the risk assessment, the risk assessment work plan can include the modeling protocol to avoid duplication of efforts. Details on the modeling protocol and report are included in DEQ's modeling procedures document (DEQ 2019).

The modeling protocol should include a map with topographic features, a facility plot plan with site features identified, and maps showing exposure locations and modeling receptors. Modeling receptor locations for CAO should extend from no less than 2 km and up to 10 km from the facility, but must include all areas where modeled risk is at or above 0.5 in 1 million excess cancer risk, or at a hazard index of 0.5 for chronic and acute noncancer risk. For point, area, and volume sources, provide details relevant to modeling emissions, such as location and dimensions.

An important factor in modeling and risk assessment is the decision of when to link the modeling function, which predicts level and location of impacts, and the assignment of risk to these modeled impacts. As discussed in Section 3.1.3, the standard approach is to use unit emission rates (1 g/s) in the model. The resulting modeled concentrations are then post-processed using the actual emission rates for each toxic air contaminant (TAC) by exposure scenario. When modeling across many receptors, this can be extremely time and resource intensive and may prove computationally challenging.

Another approach is to normalize TAC emissions to risk by dividing by their RBCs in $\mu g/m^3$. This approach is computationally simpler than the standard unit emission rate, and it provides an efficient method to identify the highest risk areas, by exposure scenario, as well as the TACs driving risk. These REER modeled emissions can be used at all risk assessment levels, including the Level 1 lookup tables. Appendix A provides a description of the unit emission rate and REER emissions approaches.

The risk assessment level selected will affect the information submitted in the modeling protocol, as shown in Figure 1. Submit the following information in the modeling protocol (and risk assessment work plan, if relevant):

- Identify all Toxic Air Contaminants (TACs), with and without RBCs
  - The total annual and acute emissions from the facility of each TAC.
  - The RBC for each TAC, from OAR 340-245-8040 Table 4.

- Identify Toxic Emission Units (TEU) and their respective TACs – Level 1
  - Location of each TEU in a figure.
  - Emission type (point or fugitive) for each TEU.
  - Stack height for point sources.
  - Building dimensions for fugitive sources.
  - Annual and acute emission rates, in lbs/yr or lbs/day, respectively, of each TAC by TEU.

- Identify Toxic Emission Units (TEU) and their respective TACs – Levels 2-4
  - Location of each TEU.
  - Emission type (point, area, volume, etc.) for each TEU.
  - The model-ready stack parameters for each TEU.
  - Annual and acute emission rates, in g/s, of each TAC by TEU

- Identify Exposure Locations
  - Levels 1, and 2 (AERSCREEN), distances to all exposure locations should be identified
  - Levels 2 (AERMOD-MAKEMET), 3, and 4, define a receptor grid and identify exposure locations

- Identify Meteorological Dataset
  - Level 1, no need to develop meteorology
  - Level 2, worst case meteorological dataset (MAKEMET) is required
  - Levels 3-4, meteorological data for input to AERMOD should be representative of the facility location

- Calculate Exposure Concentrations
  - Level 1, use lookup table OAR 340-245-8050 Table 5.
  - Level 2, convert 1-hour concentrations to annual average and maximum 24-hour concentrations.
  - Levels 3 and 4, for each exposure location, use maximum concentrations (annual average and maximum 24-hour concentration).

3.3 Level 1 Risk Assessment

The Level 1 approach allows you to use toxic air contaminant emission rates from your facility to calculate air concentrations. If you use a Level 1 risk assessment, a simple modeling protocol, including methods to combine stacks and basis for distance to exposure locations, must be approved by DEQ before the Level 1 risk assessment is performed.

To perform a Level 1 risk assessment, use the dispersion factors listed in OAR 340-245-8050 Table 5. If your emissions come from a stack, use Table 5A to estimate chronic risk and Table 5B to estimate acute risk. If you have fugitive emissions that do not come from a stack, use Table 5C to estimate chronic risk and Table 5D to estimate acute risk. Because DEQ has done the dispersion modeling to develop these dispersion factors, it is not necessary for you to run an air dispersion model. The table is designed for both point source emissions from discrete stacks that have quantifiable dimensions, and fugitive emissions that do not have a discrete emission point, including emissions from building doors and windows, or from areas where paint, solvent, or other emissions are generated. These factors are shown in OAR 340-245-8050 Tables 5A (annual exposure) and 5B (24-hour exposure) for stack emissions, and Tables 5C (annual exposure) and 5D (24-hour exposure) for fugitive emissions.

OAR 340-245-8050 Table 5 may not be used if there is elevated terrain higher than the stack height within a distance of 1.5 kilometers from the source. In this case, the assumptions used to develop the dispersion factors in Table 5 are not valid for the source, and Level 2 or Level 3 modeling will be necessary.

For stack emissions, the use of OAR 340-245-8050 Tables 5(A,B) requires the stack height and distance from the stack to the nearest exposure locations. For fugitive emissions, Tables 5(C,D) require the building area and height, and distances from the building to the nearest exposure locations. For sources that do not readily fit the scenarios presented in Table 5, refer to the *Draft Recommended Procedures for Air Quality Modeling* (DEQ 2019). For example, a wastewater treatment plant will have ponds that emit toxic air contaminants that would be evaluated as area sources using Level 2 or Level 3 air dispersion modeling.

You will likely have four types of exposure locations to evaluate (residential, commercial/industrial, school/daycare, and acute), as discussed in Section 2.4. Determine the closest distance to all the relevant exposure locations. Find the dispersion factor in the table for the exposure location distance and a given stack height for stacks, or building height and dimensions for fugitive emissions. For each exposure location, multiply the emission rate from a facility stack or fugitive emitting process by the dispersion factor.

Stack and fugitive emission rates must be in the same units as the table, such as pounds/day for toxic air contaminants with acute effects, and pounds/year for those with chronic effects. The result of the calculation will be air concentrations in units of micrograms per cubic meter, $\mu g/m^3$.

Next, for each exposure location, divide the calculated air concentration by the respective RBC for that toxic air contaminant from OAR 340-245-8040 Table 4, and sum the results as discussed in Section 3.1.2. Compare these calculated total risk values with the RALs in OAR 340-245-8010 Table 1.

DEQ's Development of Dispersion Factors in OAR 340-245-8050 Table 5

To generate the dispersion factors provided in OAR 340-245-8050 Table 5, DEQ first developed a series of reasonable maximum assumptions associated with stack height, such as stack diameter, stack flow rate, and building dimensions. We used meteorology data from six airport sites representing different regions of the state (Portland, Salem, Eugene, Medford, Redmond, and Hermiston). We then used AERMOD to estimate air concentrations at distances from 50 meters to 1,000 meters from the stack. We averaged the results for each of the exposure location distances from the six meteorological sites to develop dispersion factors in units of $\mu g/m^3$ per pounds/year for chronic exposure. For acute exposure, we used the maximum result at each exposure location distance to develop dispersion factors in units of $\mu g/m^3$ per pounds/day. The results are shown in OAR 340-245-8050 Tables 5A (annual exposure) and 5B (24-hour exposure).

DEQ developed fugitive emission dispersion factors in a similar fashion using a series of reasonable maximum assumptions associated with building area and height, and a single set of meteorology data that was a conservative representation of areas in the state. As with stack emissions, we used AERMOD to estimate air concentrations at distances from 50 meters to 1,000 meters from the building. The results were used to develop tables of dispersion factors in $\mu g/m^3$ per pounds/year for chronic exposures (OAR 340-245-8050 Table 5C), and $\mu g/m^3$ per pounds/day for acute exposures (OAR 340-245-8050 Table 5D)

Example 1 in Appendix A shows a simple Level 1 risk assessment.

The Level 1 risk assessment process is meant to be conservatively protective, and was designed primarily to assist smaller facilities in their risk assessments. It is likely that for larger, more complex facilities, a Level 1 analysis may overestimate risk. DEQ does not expect any health concerns at facilities that use a Level 1 analysis and calculate values below Source Permit Level RALs. Because risk estimated using a Level 1 risk assessment is generally much higher than actual risk, higher results obtained using this method may indicate a need for further evaluation. Completing a Level 2 or 3 riskassessment will likely show lower, more accurate estimates of risk.

For a Level 1 assessment, you can treat multiple stacks at your facility in one of two ways:

- Add the toxic air contaminant concentrations in $\mu g/m^3$ from all of the individual stacks to estimate an aggregate concentration obtained using the procedures above and then compare the concentration to the RBC for that toxic air contaminant; or
- Group the stacks and their emissions into a single stack, and use the information in OAR 340-245-8050 Table 5 to determine a dispersion factor to apply to the grouped emissions in order to estimate an air concentration for comparison to the RBC for that toxic air contaminant.

Similarly, you can address fugitive emissions from multiple buildings by either adding toxic air contaminant air concentrations to estimate an aggregate concentration, or grouping emissions into a single building. DEQ can assist with information about methods to group stacks and buildings.

For a stack height between the values shown in OAR 340-245-8050 Tables 5A and 5B, you may either use the next lowest stack height, or interpolate the dispersion factor. Similarly, for an exposure location distance between values shown in the table, you may either use the next lower distance, or interpolate the dispersion factor. For stack heights greater than 50 meters, use the appropriate dispersion factor for 50 meters. For exposure locations greater than 1,000 meters from your facility, use the appropriate dispersion factor at 1,000 meters.

Obtain stack heights and distances to exposure locations for your facility. However, in the absence of a known stack height and exposure location distance, you may use the annual dispersion factor (0.0033 $\mu g/m^3$ / pounds/year) and daily dispersion factor (8.3 $\mu g/m^3$ / pounds/day) for a stack height of 5 meters and an exposure location distance of 50 meters.

In evaluating fugitive emissions, for an exposure location distance between the values shown in the OAR 240-245-8050 Tables 5C and 5D, you may either use the next lowest distance, or interpolate the dispersion factor. For exposure locations greater than 1,000 meters from the building, you may use the appropriate dispersion factor at 1,000 meters. In the absence of known building dimensions and exposure location distance, you may use as a default, the annual dispersion factor (0.0045 $\mu g/m^3$ / pounds/year) and daily dispersion factor (4.8 $\mu g/m^3$ / pounds/day) for a building area of ≤3,000 ft^2, height of ≤20 feet, and exposure location distance of 50 meters.

3.4 Level 2 Risk Assessment

A Level 2 risk assessment is similar to a Level 1 risk assessment, except that it is less conservative and therefore more accurate. Level 2 assessments are based on air dispersion modeling using AERSCREEN or AERMOD-MAKEMET. AERSCREEN is the easier model to use, and is more appropriate for relatively basic sources with one or a few stacks in flat terrain. AERMOD-MAKEMET can consider the effects of elevated terrain and multiple stacks. Both use conservative screening meteorology. Assuming flat terrain and a single stack, both models will provide the same results. If you plan to conduct modeling, you must develop an air dispersion modeling protocol, and obtain DEQ approval before completing any modeling. DEQ can assist in preparing this simple protocol. A Level 2 risk assessment submitted without an approved modeling protocol may not be accepted.

The model results from AERSCREEN are estimated at the nearest distance from the stack or building to each exposure location, such as a residence. The results from AERMOD-MAKEMET are estimated at specific exposure locations. AERMOD-MAKEMET will provide concentrations at receptors located on a grid, and receptors at specific locations, such as schools or daycare centers, which are important to evaluate in the risk assessment.

Because of the nature of the conservative screening meteorology, Level 2 assessment models only estimate 1-hr concentrations, which must then be converted to daily (24-hour) and annual concentrations. EPA conversion factors should be used to convert the modeled 1-hr concentrations to annual and 24-hour concentrations. This calculation is done automatically for AERSCREEN, but must be done by the user for AERMOD-MAKEMET. These factors to convert 1-hr to 24-hr and annual average concentrations are 0.6 and 0.1, respectively (EPA 2016).

Once you have calculated air concentrations at the various exposure locations, divide the values by the respective RBCs for the toxic air contaminants from OAR 340-245-8040 Table 4, and sum the results as discussed in Section 3.1.2. Compare these calculated total risk values with the RALs in OAR 340-245-8010 Table 1. Example 2 in Appendix A shows an example Level 2 risk assessment.

As with Level 1, calculated risks at Level 2 remain conservative. If further refinement is desired, you can use a Level 3 risk assessment with a more complex model and actual meteorology, which will more accurately characterize toxic air contaminant concentrations.

3.5 Level 3 Risk Assessment

The key feature of a Level 3 risk assessment is site-specific air dispersion modeling conducted using a

program such as EPA's AERMOD. Because it is important to agree on receptor grids, appropriate meteorological data, and other elements necessary for effectively running a sophisticated model, DEQ requires that you first submit an air dispersion modeling protocol and risk assessment work plan for DEQ approval prior to conducting the modeling and risk assessment. We recommend that you have at least one meeting with DEQ to agree on scope, and make sure there are common understandings regarding the modeling and risk assessment. DEQ will provide approval of the modeling protocol and work plan in writing. Section 2.4.1 and DEQ's *Draft Recommended Procedures for Air Dispersion Modeling* (DEQ 2019) provide information that will be helpful in preparing the modeling protocol.

Once you have modeled ambient air concentrations at the various exposure locations, you can proceed with the calculations as discussed above in Section 3.1.2. Divide the calculated air concentration by the respective RBC in OAR 340-245-8040 Table 4. Add the results for all the applicable toxic air contaminants for each of the different types of RBCs (chronic cancer, chronic noncancer, and acute noncancer). By using an air dispersion model such as AERMOD, there is no need to conduct a summation of emissions over TEUs because the aggregate effect of multiple TEUs is already considered. Finally, compare calculated total risk values with the RALs in OAR 340-245-8010 Table 1. Example 3 in Appendix A shows an example Level 3 risk assessment.

3.6 Level 4 Risk Assessment

There are two general reasons for conducing Level 4 risk assessments: 1) if you consider it important to incorporate site-specific considerations to more accurately represent risk that may be over-estimated by default assumptions used to develop RBCs, and 2) if DEQ determines that airborne deposition of Persistant, Bioaccumulative, or Toxic (PBT) toxic air contaminants could be important for scenarios not included in the default multipathway adjustment factor assumptions used to develop RBCs. One of DEQ's goals in selecting reasonably protective assumptions for developing RBCs was to minimize the need for Level 4 risk assessments.

A Level 4 risk assessment should have the same elements as Level 3, with some additional considerations. A Level 4 risk assessment should include the following:

- A problem formulation step ending with a conceptual site model identifying TEUs and populations that may be exposed to toxic air contaminant emissions from the source, including residents, nonresident adults, and nonresident children and other sensitive populations;
- An exposure assessment that models or measures toxic air contaminant concentrations at locations of populations that may be exposed to toxic air contaminant emissions from the source. Modifications to default exposure assumptions may be proposed, including but not limited to exposure times, frequencies, and durations, relative bioavailability of toxic air contaminants, and multipathway considerations for persistent, bioaccumulative and toxic air contaminants;
- A toxicity assessment evaluating the carcinogenicity, noncarcinogenic chronic effects, and noncarcinogenic acute effects of toxic air contaminants to which populations will be exposed, including quantifying noncarcinogenic effects separately for different organ systems, and determining persistence and bioaccumulation potential. Facilities may not consider TRVs other than those listed in OAR 340-245-8030 Table 3 [OAR 340-245-0210(2)(e)].
- A risk characterization presenting a quantitative evaluation of potential cumulative health risks associated with exposure to all emissions from the source; and
- A quantitative or qualitative uncertainty evaluation of appropriate elements of the risk assessment.

Elements specific to a Level 4 evaluation include modifications to default exposure assumptions, relative bioavailability of toxic air contaminants, and additional multipathway considerations not

addressed by the default adjustment factors. These elements are discussed below.

3.6.1 Exposure Assumption Modifications

The default exposure assumptions for exposure times, frequencies, and durations used in the development of RBCs for residents and workers are typical of those used in risk assessments. However, there may be special circumstances where it is appropriate to modify these assumptions. An example could be a nearby facility that is known to contain workers for only a fraction of the default assumption for exposure time. In this case, you should document the circumstances, and propose modified exposure parameter values for use in the risk assessment.

3.6.2 Relative Bioavailability

The toxicity of a toxic air contaminant can depend on how much of the chemical is actually absorbed by a person, not just on the measured concentration in air (or soil or water). If the form of a toxic air contaminant is less bioavailable to a human than it was in the animal test used as the basis for its TRV, this can be taken into account. For example, under some circumstances, you may want to propose a relative bioavailability test to quantify these differences. There are few standard laboratory tests, and animal tests can be time-consuming and expensive, so relative bioavailability tests are not commonly performed. If you decide to pursue testing, DEQ will request a detailed work plan for approval prior to conducting the evaluation.

Consideration of the particular form of a toxic air contaminant is the main reason for differences in bioavailability and is usually considered when the TRV is established. For example, DEQ has an RBC for chromium based on the toxicity of hexavalent chromium. The hexavalent form of chromium is substantially more toxic than the other forms, such as trivalent chromium (for which no RBC is available). If you can characterize the specific chemical form of your emissions, you can use the appropriate RBC for that chemical form of the toxic air contaminant at any risk evaluation level. This may make it unnecessary to proceed to a Level 4 evaluation.

3.6.3 Multipathway Analysis

If your source emits PBT toxic air contaminants, it may be important to evaluate air deposition and additional exposure scenarios not included in the development of RBCs that could include contact with soil and water (see Section 2.2.2). Contact DEQ to discuss how to proceed. Available protocols include EPA's Risk Assessment Guidance for Superfund (1989), Guidelines for Exposure Assessment (1992), and more specifically for toxic air contaminant emissions, California's OEHHA's Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments (2015).

3.7 Risk Assessment Report

The risk assessment report should include the information provided in the modeling protocol and risk assessment work plan (if required), as well as information to further support the risk calculations. The level of detail required in the modeling results varies by the risk assessment level selected. You should provide sufficient information to allow DEQ to duplicate the results of the modeling and risk assessment during DEQ's review process. The following sections outline the recommendations by risk assessment level. Figure 1 includes a summary of this information.

3.7.1. Level 1 and Level 2 AERSCREEN

Report the following information:

- Provide a map depicting the source location and all relevant exposure locations

- For each TAC, provide the RBCs from OAR 340-245-8040 Table 4, dispersion factors from OAR 340-245-8050 Table 5, maximum exposure concentrations, and total excess cancer risk and hazard quotients across all exposure scenarios reported by individual TEU and for the facility as a whole.
- Demonstrate how the total risk across the entire facility was calculated and compared to the risk action levels.

3.7.2. Level 2 AERMOD-MAKEMET, Level 3, and Level 4

Report the following information:

- For each TAC, provide the RBCs from OAR 340-245-8040 Table 4, location of maximum exposure concentration, maximum exposure concentration, total excess cancer risk and hazard quotients across all exposure scenarios, reported for both TEUs and for the facility as a whole.
- Demonstrate how the total risk across the entire facility was calculated and compared to the risk action levels.
- Provide figures showing the concentration/risk plots and gradients around the facility for each exposure scenario.
- For modeling risk using the REER approach, present results in units of risk. Isopleths should represent total risk for each exposure scenario.
- Provide all modeling input and output files to DEQ. Specifically, DEQ requests the following files:
    - AERMOD input file
    - AERMOD source and receptor files (SOU and ROU)
    - Terrain data files
    - BPIP files
    - Met data (sfc and pfl files)
    - Submit other modeling files needed for running input file
    - Table listing any referenced receptor IDs, geocoordinates (UTM, lat/long), and assigned exposure location.

DEQ's air dispersion modeling procedures document (DEQ 2019) provides examples of requested report figures. DEQ prefers that contour plots show total facility risk for each exposure scenario. Figure 2 shows an example of a figure showing residential excess cancer risk for a facility.

3.7.3. Risk Assessment Results

Appendix A provides example tables for presenting the results of Level 1, 2, and 3 risk assessments. The summary tables in the risk assessment report should identify locations of maximum risk, and show a direct comparison of calculated risk values with appropriate risk action levels.

By providing all the supporting information identified above, DEQ will be able to review a facility's submitted risk assessment more efficiently. In addition, the documentation will provide a transparent way to show how a facility is meeting the requirements of the Cleaner Air Oregon program.

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Figure 1. Exposure Modeling and Risk Assessment Overview

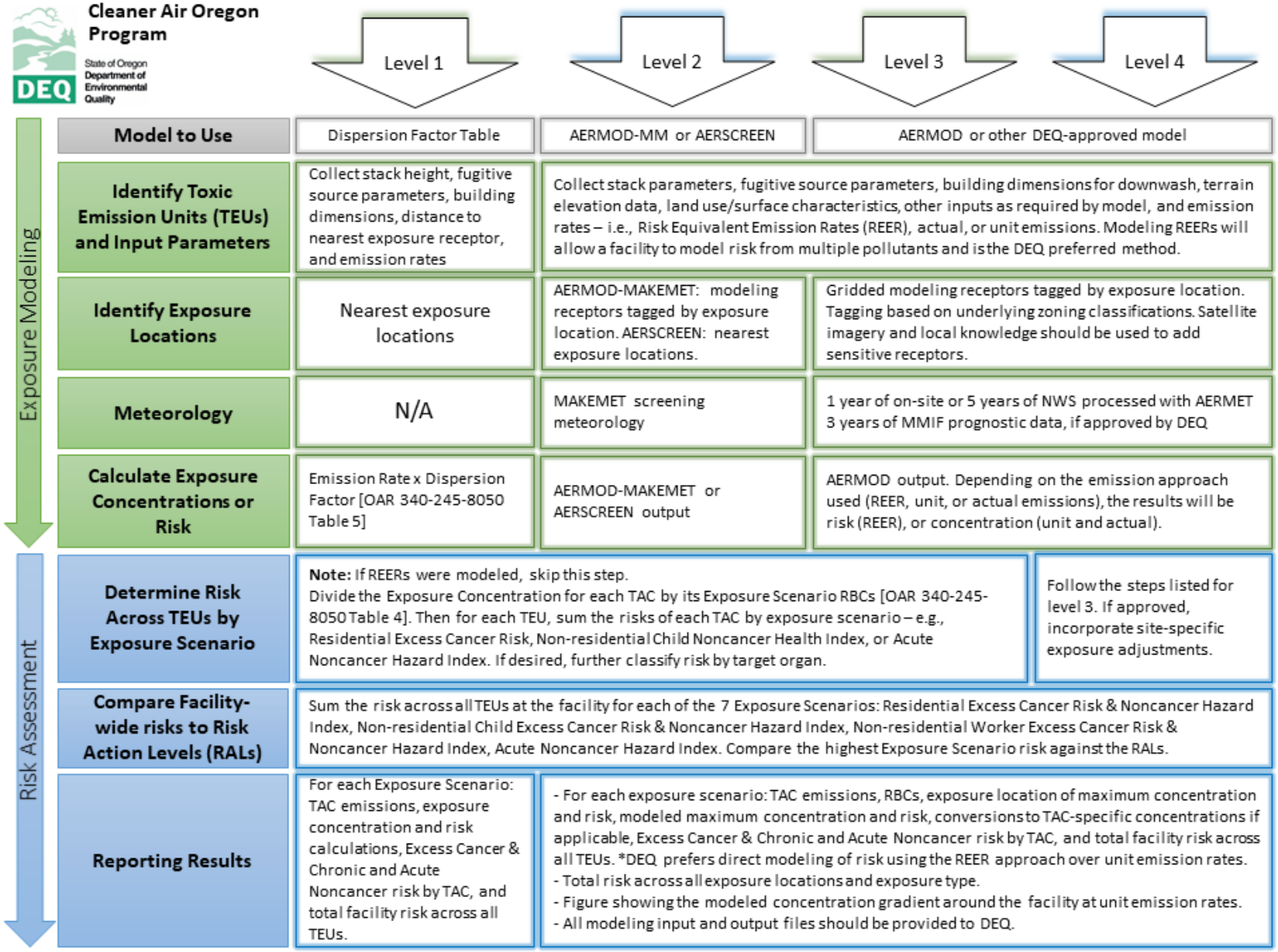

Figure 2. Example Figure Showing Excess Cancer Risk in Proxyland, Oregon

APPENDIX A

Risk Assessment Examples

A.1 Introduction

The examples in this appendix show how to perform Level 1, Level 2, and Level 3 risk assessments. The first tables show how to conduct the risk assessments according to the explicit procedures presented in rule. Later tables show a more efficient approach that DEQ considers equivalent to the approach presented in rule. See DEQ's air dispersion modeling procedures document (DEQ 2019) for example figures and more information about how to document the air dispersion modeling.

A.2 Standard Approach

Example 1 illustrates the different steps in performing a Level 1 risk assessment. Table A-1 shows the stack heights of two emission units at an existing facility, the nearest distances to various exposure locations, and the corresponding dispersion factors obtained from OAR 340-245-8050 Table 5. Table A-2 shows the calculation of air concentrations using the dispersion factors and site-specific emission rates for each chemical. Table A-3 shows the comparison of calculated air concentrations with Risk-Based Concentrations from OAR 340-245-8040 Table 4, and the resulting risk calculations. Because the risks exceed Risk Action Levels for an existing facility, additional evaluations were performed using Level 2 and Level 3 risk assessment procedures.

Table A-4 shows how a Level 2 risk assessment would be done using air concentrations obtained from AERSCREEN air dispersion modeling, the comparison with RBCs, and the resulting risk calculations. Using more site-specific modeling resulted in more accurate, lower air concentrations, but the risks are still above RALs. In practice, the air dispersion model is generally run using a unit emission rate of 1 g/s for each emission unit. Air concentrations are later calculated in a spreadsheet or database by multiplying the model output (as $\mu g/m^3$ per g/s) by the actual emission rate (g/s).

Table A-5 shows how a Level 3 risk assessment would be done using air concentrations obtained from more sophisticated AERMOD air dispersion modeling, the comparison with RBCs, and the resulting risk calculations. In a Level 3 risk assessment there is no need to sum concentrations resulting from two or more emission sources because the air dispersion model calculates concentrations resulting from all emission sources at the same time. Using the more realistic modeling resulted in more accurate, lower air concentrations such that risks are not above RALs.

Table A-6 is a summary of calculated risk in the examples at each risk assessment level.

An owner or operator of a source can start at any level of risk assessment, even Level 4. If the owner or operator starts at Level 1 and finds that risk is greater than Risk Action Levels, they can skip directly to a Level 4 risk assessment if they choose to do so.

A.2.1 Target Organs

Noncancer hazards can be evaluated by target organs at any level of risk assessment. In the standard approach, this would involve using the calculated air concentrations to calculate a hazard index for each relevant organ. Table A-7 shows an example residential chronic noncancer risk calculation using the information for a Level 2 evaluation in Example 2. Nickel's chronic TRV applies to two target organ

systems, immune and respiratory. A hazard quotient is calculated for each organ system, but the hazard quotients are not additive. Therefore, the sum of the hazard quotients for individual organ systems does not equal the initial hazard index of 12 for residential exposure calculated in Table A-4.

Table A-8 shows an example residential exposure hazard index evaluation for target organs based on acute effects.

A.3 Alternative Approach – Risk Equivalent Emission Rate

The example calculations for the standard approach using two emission units and six chemicals may not be extensive depending on the number of receptors on the modeling grid. DEQ recognizes that results of AERMOD modeling using a unit emission rate of 1 g/s from many emission units for many chemicals will require considerable post-modeling processing to determine the maximum annual average risk and maximum daily risk at each exposure location. With a large number of receptors, the post-modeling risk assessment effort may be onerous. As an alternative, DEQ will accept a mathematically equivalent approach that uses AERMOD to model risk instead of chemical concentrations. This approach will require more model runs, but no post-model calculations are required, which will result in considerable savings in time and effort for the risk assessment.

The key to the alternative approach is to model modified emission rates that incorporate risk, by dividing the emission rate for each toxic air contaminant by the RBC appropriate to an exposure location, creating a Risk Equivalent Emission Rate (REER). Because REERs are directly proportional to risk, REERs from different toxic air contaminants can be summed to calculate a total REER for each toxics emission unit. With this approach, the values AERMOD calculates at each modeling receptor is a unitless value corresponding to either cancer risk per million for carcinogens, or hazard index for noncarcinogens. This is because using REER inputs, AERMOD calculations will convert a toxic air contaminant's g/s emission rate to $\mu g/m^3$ at each receptor. Each exposure scenario and toxic endpoint combination (residential cancer, residential chronic noncancer, nonresidential child cancer, nonresidential child chronic noncancer, worker cancer, worker chronic noncancer, and acute noncancer) will need to be run separately. Noncancer risk evaluation can be further resolved by separate target organ system. If the number of target organs is large, and some of the organ systems do not substantially contribute to risk, the low-risk target organs can be conservatively evaluated in a combined category of "other target organs".

Table A-9 uses the same information in Example 2, and shows how REERs are calculated using RBCs. REERs are calculated separately for each relevant exposure unit. After modeling, the result is in risk units, which makes preparing summary Table A-10 a simple task. For chronic RBCs, the model is run as if annual concentrations are being calculated. For acute RBCs, the model is run as if daily concentrations are being calculated. The maximum values for each exposure location are presented for comparison with RALs.

As with the standard approach, noncancer hazards can be evaluated by target organs at any level of risk assessment. In the alternative approach, this would first involve calculating REERs for each target organ. Table A-11 shows example REER calculations for residential chronic noncancer risk, and Table A-12 shows the calculations for acute risk. Separate REERs would need to be calculated for the other exposure areas (non-residential child and non-residential worker). Running AERMOD using the REERs for each target organ will create an output that corresponds to hazard index. This will make summarizing risk a simple task of taking the highest hazard index for each exposure location. Table A-13 shows an example summary for residential exposure. Separate tables would be needed for non-residential child and non-residential worker exposure locations.

Table A-1. Example 1 – Emission Unit Information and Dispersion Factors

	Value	OAR 340-245-8050 Table 5	
Emission Unit 1	**(meters)**	**Dispersion Factor**	
Stack height =	10		
Distance to residential =	100	0.00075	µg/m^3 per lb/yr
Distance to nonresident child =	200	0.00033	
Distance to nonresident worker =	200	0.00033	
Distance to acute =	85	2.7	µg/m^3 per lb/day (interpolated between 2.6 and 2.8)
Emission Unit 2	**(meters)**	**Dispersion Factor**	
Stack height =	20		
Distance to residential =	150	0.00017	µg/m^3 per lb/yr
Distance to nonresident child =	250	0.00010	
Distance to nonresident worker =	250	0.00010	
Distance to acute =	135	0.635	µg/m^3 per lb/day (interpolated between 0.62 and 0.65)

Table A-2. Example 1 – Level 1 Calculation of Air Concentrations

Toxic Air Contaminant	Annual Emission Rate (lb/yr)	24-Hour Emission Rate (lb/day)	Calc. Annual Residential Concentration ($\mu g/m^3$)	Calc. Annual Nonresidential Child Conc. ($\mu g/m^3$)	Calc. Annual Nonresidential Worker Conc. ($\mu g/m^3$)	Calculated 24-Hour Concentration ($\mu g/m^3$)
Emission Unit 1						
Cadmium	140	0.38	0.11	0.046	0.046	1.0
Manganese	70	0.25	0.053	0.023	0.023	0.68
Nickel (insoluble)	220	0.60	0.17	0.073	0.073	1.6
	Dispersion Factor:		**0.00075** (µg/m3 per lb/yr)	**0.00033** (µg/m3 per lb/yr)	**0.00033** (µg/m3 per lb/yr)	**2.7** (µg/m3 per lb/day)
Emission Unit 2						
Acetaldehyde	100,000	300	17	10	10	191
Acetone	80,000	250	14	8	8	159
Acrolein	10,000	50	2	1	1	32
	Dispersion Factor:		**0.00017** (µg/m3 per lb/yr)	**0.00010** (µg/m3 per lb/yr)	**0.00010** (µg/m3 per lb/yr)	**0.635** (µg/m3 per lb/day)

Notes:
Dispersion factors from OAR 340-245-8050 Table 5. See Table A-1.
Concentration = Emission Rate * Dispersion Factor

Table A-3. Example 1 –Summary Risk Table for Level 1 Risk Assessment

	Residential Exposure					Non-Resident Child Exposure					Non-Resident Worker Exposure					Acute Exposure		
Toxic Air Contaminant	Annual Avg Conc. ($\mu g/m^3$)	RBC Cancer ($\mu g/m^3$)	Excess Cancer Risk	RBC Noncancer ($\mu g/m^3$)	Hazard Quotient	Annual Average Conc. ($\mu g/m^3$)	RBC Cancer ($\mu g/m^3$)	Excess Cancer Risk	RBC Noncancer ($\mu g/m^3$)	Hazard Quotient	Annual Average Conc. ($\mu g/m^3$)	RBC Cancer ($\mu g/m^3$)	Excess Cancer Risk	RBC Noncancer ($\mu g/m^3$)	Hazard Quotient	24-Hour Average Conc. ($\mu g/m^3$)	Acute RBC ($\mu g/m^3$)	Hazard Quotient
Emission Unit 1																		
Cadmium	0.11	0.00056	188	0.0050	21	0.046	0.014	3.3	0.037	1.3	0.046	0.0067	6.9	0.037	1.2	1.0	0.030	35
Manganese	0.053	NA	--	0.090	0.58	0.023	NA	--	0.40	0.058	0.023	NA	--	0.40	0.06	0.68	0.30	2.3
Nickel (insoluble)	0.17	0.0038	43	0.014	12	0.073	0.10	0.73	0.062	1.2	0.073	0.046	1.6	0.062	1.2	1.6	0.20	8.1
Total Unit 1			**231**		**33**			**4.0**		**2.5**			**8.5**		**2.5**			**45**
Emission Unit 2																		
Acetaldehyde	17	0.45	38	140	0.12	10	12	0.83	620	0.016	10	5.5	1.8	620	0.016	191	470	0.41
Acetone	14	NA	--	31,000	0.00044	8	NA	--	140,000	0.000057	8	NA	--	140,000	0.000057	159	62,000	0.003
Acrolein	2	NA	--	0.35	4.9	1	NA	--	1.5	0.67	1	NA	-	1.5	0.67	32	6.9	4.6
Total Unit 2			**38**		**5.0**			**0.83**		**0.68**			**1.8**		**0.68**			**5**
Total Source (Unit 1 and Unit 2)			**269**		**38**			**5**		**3**			**10**		**3**			**50**
Existing Facility Risk Action Level			*50*		*5*			*50*		*5*			*50*		*5*			*5*

Notes:
Excess Cancer Risk = Annual conc. ($\mu g/m^3$) / Cancer RBC ($\mu g/m^3$) expressed as risk per million
Chronic Hazard Quotient = Annual conc. ($\mu g/m^3$) / Noncancer RBC ($\mu g/m^3$) x 1
Acute Hazard Quotient = 24-hr conc. ($\mu g/m^3$) / Acute RBC ($\mu g/m^3$) x 1

Table A-4. Example 2 – Summary Risk Table for Level 2 Risk Assessment

	Residential Exposure					Non-Resident Child Exposure					Non-Resident Worker Exposure					Acute Exposure		
Toxic Air Contaminant	**Annual Average Conc. ($\mu g/m^3$)**	**RBC Cancer ($\mu g/m^3$)**	**Excess Cancer Risk**	**RBC Noncancer ($\mu g/m^3$)**	**Hazard Quotient**	**Annual Average Conc. ($\mu g/m^3$)**	**RBC Cancer ($\mu g/m^3$)**	**Excess Cancer Risk**	**RBC Noncancer ($\mu g/m^3$)**	**Hazard Quotient**	**Annual Average Conc. ($\mu g/m^3$)**	**RBC Cancer ($\mu g/m^3$)**	**Excess Cancer Risk**	**RBC Noncancer ($\mu g/m^3$)**	**Hazard Quotient**	**24-Hour Average Conc. ($\mu g/m^3$)**	**Acute RBC ($\mu g/m^3$)**	**Hazard Quotient**
Emission Unit 1																		
Cadmium	0.032	0.00056	56	0.0050	6.3	0.014	0.014	0.99	0.037	0.37	0.014	0.0067	2.1	0.037	0.37	0.21	0.030	6.9
Manganese	0.016			0.090	0.18	0.0069	NA	--	0.40	0.017	0.0069	NA	--	0.40	0.017	0.14	0.30	0.45
Nickel (insoluble)	0.050	0.0038	13	0.014	3.5	0.022	0.10	0.22	0.062	0.35	0.022	0.046	0.47	0.062	0.35	0.33	0.20	1.6
Total Unit 1			**69**		**10**			**1.2**		**0.74**			**2.5**		**0.74**			**9.0**
Emission Unit 2																		
Acetaldehyde	5	0.45	11	140	0.036	3.0	12	0.25	620	0.0048	3.0	5.5	0.55	620	0.0048	38	470	0.081
Acetone	4	NA	--	31,000	0.00013	2.4	NA	--	140,000	0.000017	2.4	NA	--	140,000	0.000017	32	62,000	0.00051
Acrolein	0.5	NA	--	0.35	1.5	0.30	NA	--	1.5	0.20	0.30	NA	--	1.5	0.20	6.4	6.9	0.92
Total Unit 2			**11**		**1.5**			**0.25**		**0.20**			**0.55**		**0.20**			**1.0**
Total Source (Unit 1 and Unit 2)			**81**		**12**			**2**		**1**			**3**		**1**			**10**
Existing Facility Risk Action Level			*50*		*5*			*50*		*5*			*50*		*5*			*5*

Notes:
Air concentrations modeled using AERSCREEN.
Excess Cancer Risk = Annual conc. ($\mu g/m^3$) / Cancer RBC ($\mu g/m^3$) expressed as risk per million
Chronic Hazard Quotient = Annual conc. ($\mu g/m^3$) / Noncancer RBC ($\mu g/m^3$) x 1 (HQ)
Acute Hazard Quotient = 24-hr conc. ($\mu g/m^3$) / Acute RBC ($\mu g/m^3$) x 1 (HQ)

Table A-5. Example 3 – Summary Risk Table for Level 3 Risk Assessment

	Residential Exposure					Non-Resident Child Exposure					Non-Resident Worker Exposure					Acute Exposure		
Toxic Air Contaminant	**Annual Average Conc. ($\mu g/m^3$)**	**RBC Cancer ($\mu g/m^3$)**	**Excess Cancer Risk**	**RBC Noncancer ($\mu g/m^3$)**	**Hazard Quotient**	**Annual Average Conc. ($\mu g/m^3$)**	**RBC Cancer ($\mu g/m^3$)**	**Excess Cancer Risk**	**RBC Noncancer ($\mu g/m^3$)**	**Hazard Quotient**	**Annual Average Conc. ($\mu g/m^3$)**	**RBC Cancer ($\mu g/m^3$)**	**Excess Cancer Risk**	**RBC Noncancer ($\mu g/m^3$)**	**Hazard Quotient**	**24-Hour Average Conc. ($\mu g/m^3$)**	**Acute RBC ($\mu g/m^3$)**	**Hazard Quotient**
Emission Units 1 and 2																		
Location of Maximum Risk			**R2105**		**R2041**			**C3535**		**C3501**			**W1121**		**W1007**			**A1090**
Cadmium	0.0019	0.00056	3.4	0.010	0.38	0.00083	0.014	0.059	0.037	0.023	0.00083	0.0067	0.12	0.037	0.023	0.021	0.030	0.69
Manganese	0.0009	NA	--	0.090	0.011	0.00042	NA	--	0.40	0.0010	0.00042	NA	--	0.40	0.0010	0.014	0.30	0.045
Nickel (insoluble)	0.0030	0.0038	0.78	0.014	0.21	0.0013	0.10	0.013	0.062	0.021	0.0013	0.046	0.028	0.062	0.021	0.033	0.20	0.16
Acetaldehyde	0.31	0.45	0.68	140	0.0022	0.18	12	0.015	620	0.00029	0.18	5.5	0.033	620	0.00029	3.8	470	0.0081
Acetone	0.24	NA	--	31,000	0.0000079	0.14	NA	--	140,000	0.0000010	0.14	NA	--	140,000	0.0000010	3.2	62,000	0.000051
Acrolein	0.031	NA	--	0.35	0.087	0.018	NA	--	1.5	0.012	0.018	NA	--	1.5	0.012	0.6	6.9	0.092
Total Source (Unit 1 and Unit 2)			**5**		**0.69**			**0.087**		**0.057**			**0.19**		**0.057**			**1**
Existing Facility Risk Action Level			*50*		*5*			*50*		*5*			*50*		*5*			*5*

Notes:
Air concentrations modeled using AERMOD.
Excess Cancer Risk = Annual conc. ($\mu g/m^3$) / Cancer RBC ($\mu g/m^3$) expressed as risk per million
Chronic Hazard Quotient = Annual conc. ($\mu g/m^3$) / Noncancer RBC ($\mu g/m^3$) x 1
Acute Hazard Quotient = 24-hr conc. ($\mu g/m^3$) / Acute RBC ($\mu g/m^3$) x 1
UTM locations of maximum risk:
R2105 = 10T 421046 5021013
R2041 = 10T 421040 5021010
C3535 = 10T 421046 5020913
C3501 = 10T 421048 5020920
W1121 = 10T 421040 5020902
W1007 = 10T 421038 5020907
A1090 = 10T 421046 5021028

Table A-6. Summary of Calculated Risk[a] at Different Risk Assessment Levels

	Residential Exposure		Non-Resident Child Exposure		Non-Resident Worker Exposure		Acute Exposure
Risk Assessment Level	**Excess Cancer Risk**	**Hazard Index**	**Excess Cancer Risk**	**Hazard Index**	**Excess Cancer Risk**	**Hazard Index**	**Hazard Index**
Level 1	269	38	5	3	10	3	50
Level 2	81	12	2	1	3	1	10
Level 3	5	0.7	0.09	0.06	0.2	0.06	1
Risk Action Level[b]	*50*	*5*	*50*	*5*	*50*	*5*	*5*

Note:

a) Calculated risks are total calculated risks for the example facility, taken from Examples 1, 2, and 3.
b) RALs for an existing facility, taken from OAR 340-245-8010 Table 1.

Table A-7. Example Level 2 Target Organ Chronic Noncancer Risk Assessment for Residential Exposure

Chronic Noncancer Residential Exposure								
Target Organ:		**Kidney**		**Nervous System**		**Immune System**		**Respiratory System**	
Chemical	**Annual Average Concentration (µg/m³)**	**RBC (µg/m³)**	**Hazard Quotient**	**RBC (µg/m³)**	**Hazard Quotient**	**RBC (µg/m³)**	**Hazard Quotient**	**RBC (µg/m³)**	**Hazard Quotient**
Emission Unit 1									
Cadmium	0.032	0.005	6.4						
Manganese	0.016			0.09	0.18				
Nickel (insoluble)	0.05					0.014	3.6	0.014	3.6
Total Unit 1			**6.4**		**0.18**		**3.6**		**3.6**
Emission Unit 2									
Acetaldehyde	5							140	0.036
Acetone	4			31,000	0.00013				
Acrolein	0.5							0.35	1.4
Total Unit 2					**0.00013**				**1.5**
Total Source (Unit 1 and Unit 2)			**6.4**		**0.18**		**3.6**		**5.0**
Existing Facility Risk Action Level			*5*		*5*		*5*		*5*

Notes:
RBC = risk-based concentration
Chronic Hazard Quotient = Annual conc. (µg/m³) / Noncancer Chronic RBC (µg/m³) x 1
Hazard indices for specific target organs will not necessarily sum to the total hazard index for all organs.

Table A-8. Example Level 2 Target Organ Acute Noncancer Risk Assessment for Residential Exposure

Acute Noncancer Residential Exposure									
Target Organ:		**Eyes**		**Nervous System**		**Immune System**		**Respiratory System**	
Chemical	**Daily Average Concentration (µg/m³)**	**RBC (µg/m³)**	**Hazard Quotient**	**RBC (µg/m³)**	**Hazard Quotient**	**RBC (µg/m³)**	**Hazard Quotient**	**RBC (µg/m³)**	**Hazard Quotient**
Emission Unit 1									
Cadmium	0.21							0.030	7.0
Manganese	0.14			0.30	0.47				
Nickel (insoluble)	0.33					0.20	1.7		
Total Unit 1					**0.47**		**1.7**		**7.0**
Emission Unit 2									
Acetaldehyde	38	470	0.081					470	0.082
Acetone	32			62,000	0.00053				
Acrolein	6.4							6.9	0.93
Total Unit 2			**0.081**		**0.00053**				**1.0**
Total Source (Unit 1 and Unit 2)			**0.08**		**0.47**		**1.7**		**8.0**
Existing Facility Risk Action Level			*5*		*5*		*5*		*5*

Notes:
RBC = risk-based concentration
Acute Hazard Quotient = Daily conc. (µg/m³) / Noncancer Acute RBC (µg/m³) x 1
Hazard quotients for specific target organs will not necessarily sum to the total HI for all organs.

Table A-9. Example of Level 3 Emissions Calculation for Risk Modeling Approach

					Residential				Non-Residential Child				Non-Residential Worker				Acute	
					Cancer		Noncancer		Cancer		Noncancer		Cancer		Noncancer			
	Annual Emission Rate		Daily Emission Rate		RBC Cancer	Annual REER	RBC Noncancer	Annual REER	RBC Cancer	Annual REER	RBC Noncancer	Annual REER	RBC Cancer	Annual REER	RBC Noncancer	Annual REER	Acute RBC	Daily REER
Toxic Air Contaminant	**(lb/yr)**	**(g/s)**	**(lb/yr)**	**(g/s)**	**(µg/m³)**	**(g/s per µg/m³)**	**(µg/m³)**	**(g/s per µg/m³)**	**(µg/m³)**	**(g/s per µg/m³)**	**(µg/m³)**	**(g/s per µg/m³)**	**(µg/m³)**	**(g/s per µg/m³)**	**(µg/m³)**	**(g/s per µg/m³)**	**(µg/m³)**	**(g/s per µg/m³)**
Emission Units 1 and 2																		
Cadmium	140	0.00201	0.38	0.00201	0.00056	3.6	0.0050	0.403	0.014	0.144	0.037	0.054	0.0067	0.301	0.037	0.054	0.030	0.067
Manganese	70	0.00101	0.25	0.00131			0.090	0.0112			0.40	0.0025			0.40	0.0025	0.30	0.0044
Nickel (insoluble)	220	0.00316	0.60	0.00316	0.0038	0.833	0.014	0.226	0.10	0.032	0.062	0.051	0.046	0.069	0.062	0.051	0.20	0.016
Acetaldehyde	100,000	1.44	300	1.58	0.45	3.196	140	0.0103	12	0.1199	620	0.0023	5.5	0.2615	620	0.0023	470	0.0034
Acetone	80,000	1.15	250	1.31			31,000	0.0000371			140,000	0.0000082			140,000	0.0000082	62,000	0.000021
Acrolein	10,000	0.144	50	0.263			0.35	0.411			1.5	0.096			1.5	0.096	6.9	0.038
Total Source (Units 1 and 2)						**7.6**		**1.06**		**0.295**		**0.206**		**0.631**		**0.206**		**0.129**

Notes:
RBC = Risk Based Concentration
REER = Risk Equivalent Emission Rate
Annual Emission Rate (g/s) = Annual Emission Rate (lb/yr) x 453.6 g/lb / (60 sec/min x 60 min/hr x 24 hr/day x 365 day/yr)
Daily Emission Rate (g/s) = Daily Emission Rate (lb/yr) x 453.6 g/lb / (60 sec/min x 60 min/hr x 24 hr/day)

Table A-10. Example Summary Risk Table for Level 3 Risk Assessment using Modeled Risk

	Residential		Non-Residential Child		Non-Residential Worker		Acute
	Excess Cancer Risk	**Chronic Hazard Quotient**	**Excess Cancer Risk**	**Chronic Hazard Quotient**	**Excess Cancer Risk**	**Chronic Hazard Quotient**	**Acute Hazard Quotient**
Location of Maximum Risk	**R2105**	**R2041**	**C3535**	**C3501**	**W1121**	**W1007**	**A1090**
Total Source (Units 1 and 2)	**5**	**0.7**	**0.09**	**0.06**	**0.2**	**0.06**	**1**
Existing Facility Risk Action Level	*50*	*5*	*50*	*5*	*50*	*5*	*5*

Note:
Values shown are the maximum outputs from AERMOD for each exposure location using modeled risk equivalent emission rates (Table A-9).
UTM locations of maximum risk:
R2105 = 10T 421046 5021013
R2041 = 10T 421040 5021010
C3535 = 10T 421046 5020913
C3501 = 10T 421048 5020920
W1121 = 10T 421040 5020902
W1007 = 10T 421038 5020907
A1090 = 10T 421046 5021028

Table A-11. Example of Level 3 Calculation of Emission Rates for Risk Modeling of Target Organ Chronic Noncancer Risk for Residential Exposure

			Cancer		Kidney		Nervous System		Immune System		Respiratory System	
	Annual Emission Rate		RBC	Annual REER	RBC	Annual REER	RBC	Annual REER	RBC	Annual REER	RBC	Annual REER
Toxic Air Contaminant	**(lb/yr)**	**(g/s)**	**(µg/m^3)**	**(g/s per µg/m^3)**	**(µg/m^3)**	**(g/s per µg/m^3)**	**(µg/m^3)**	**(g/s per µg/m^3)**	**(µg/m^3)**	**(g/s per µg/m^3)**	**(µg/m^3)**	**(g/s per µg/m^3)**
Emissions Unit 1 and 2												
Cadmium	140	0.00201	0.00056	3.6	0.0050	0.40						
Manganese	70	0.00101					0.09	0.011				
Nickel (insoluble)	220	0.00316	0.0038	0.83					0.014	0.23	0.014	0.23
Acetaldehyde	100,000	1.44	0.45	3.2							140	0.010
Acetone	80,000	1.15					31,000	0.000037				
Acrolein	10,000	0.144									0.35	0.41
Total Source (Units 1 and 2)				**7.6**		**0.40**		**0.011**		**0.23**		**0.65**

Notes:
RBC = Risk Based Concentration
REER = Risk Equivalent Emission Rate
Annual Emission Rate (g/s) = Annual Emission Rate (lb/yr) x 453.6 g/lb / (60 sec/min x 60 min/hr x 24 hr/day x 365 day/yr)
REER (g/s per µg/m^3) = Annual Emission Rate (g/s) / Chronic RBC (µg/m^3)

Table A-12. Example of Level 3 Calculation of Emission Rates for Risk Modeling of Target Organ Acute Noncancer Risk for Residential Exposure

			Eyes		Nervous System		Immune System		Respiratory System	
	Daily Emission Rate		RBC	Annual REER	RBC	Annual REER	RBC	Annual REER	RBC	Annual REER
Toxic Air Contaminant	**(lb/day)**	**(g/s)**	**(µg/m^3)**	**(g/s per µg/m^3)**	**(µg/m^3)**	**(g/s per µg/m^3)**	**(µg/m^3)**	**(g/s per µg/m^3)**	**(µg/m^3)**	**(g/s per µg/m^3)**
Emission Units 1 and 2										
Cadmium	0.38	0.0020							0.03	0.11
Manganese	0.25	0.0013			0.3	0.0044				
Nickel (insoluble)	0.60	0.0032					0.2	0.016		
Acetaldehyde	300	1.58	470	0.0034					470	0.0034
Acetone	250	1.313			62,000	0.000021				
Acrolein	50	0.263							6.9	0.038
Total Source (Units 1 and 2)				**0.0034**		**0.0044**		**0.016**		**0.15**

Notes:
RBC = Risk Based Concentration
REER = Risk Equivalent Emission Rate
Daily Emission Rate (g/s) = Daily Emission Rate (lb/yr) x 453.6 g/lb / (60 sec/min x 60 min/hr x 24 hr/day
REER (g/s per µg/m^3) = Daily Rate (g/s) / Acute RBC (µg/m^3)

Table A-13. Example Summary Residential Risk Table for Level 3 Risk Assessment using Modeled Risk

	Chronic Residential Exposure					Acute Residential Exposure			
	Excess Cancer Risk	**Kidney Hazard Index**	**Nervous System Hazard Index**	**Immune System Hazard Index**	**Respiratory System Hazard Index**	**Eyes Hazard Index**	**Nervous System Hazard Index**	**Immune System Hazard Index**	**Respiratory System Hazard Index**
Location of Maximum Risk	**R2105**	**R2020**	**R2062**	**R2016**	**R2112**	**A1001**	**A1092**	**A1044**	**A998**
Total Source (Units 1 and 2)	**5**	**0.4**	**0.01**	**0.2**	**0.3**	**0.008**	**0.05**	**0.2**	**0.8**
Existing Facility Risk Action Level	*50*	*5*	*5*	*5*	*5*	*5*	*5*	*5*	*5*

Note:
Values shown are the maximum outputs from AERMOD for the residential exposure location using modeled risk equivalent emission rates (Tables A-11 and A-12).
UTM locations of maximum risk:
R2105 = 10T 421046 5021013
R2020 = 10T 421035 5021002
R2062 = 10T 421051 5021018
R2016 = 10T 421053 5021007
R2112 = 10T 421044 5021011
A1001 = 10T 421044 5021033
A1092 = 10T 421050 5021035
A1044 = 10T 421052 5021040
A998 = 10T 421054 5021046

APPENDIX B

Authoritative Sources of Toxicity Reference Values

B.1 Chronic Values

DEQ used the following authoritative sources of chronic toxicity reference values (TRVs) to make its recommendations to the EQC, which the EQC adopted:

- DEQ Ambient Benchmark Concentrations (ABCs) adopted by the Environmental Quality Commission (EQC)
- EPA Integrated Risk Information System (IRIS) database (www.epa.gov/iris)
- EPA Provisional Peer-Reviewed Toxicity Value (PPRTV) database (www.hhpprtv.ornl.gov)
- Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profiles (www.atsdr.cdc.gov)
- California's Office of Environmental Health Hazard Assessment (OEHHA)(www.oehha.ca.gov)

DEQ and OHA selected the most recently published TRV from among the authoritative sources for each toxic air contaminant. This ensures that chronic TRVs are based on the most recent review of scientific studies. Chronic TRVs were developed separately for noncarcinogenic and carcinogenic effects. For cases where DEQ's ABCs were the most recent values, and DEQ's Air Toxics Science Advisory Committee (ATSAC) decided it was inappropriate to develop an ABC based on carcinogenic effects, we did not obtain a cancer TRV from the other authoritative sources. Similarly, if the ATSAC decided it was inappropriate to develop an ABC based on noncarcinogenic effects, DEQ did not obtain a TRV from the other authoritative sources if the ABC was the most recent value.

Inhalation toxicity information for noncancer effects are typically provided as threshold values, and are given different names by different authoritative bodies. For example, EPA calls them Reference Concentrations (RfCs), the federal Agency for Toxic Substances and Disease Registry calls them Minimal Risk Levels (MRLs), and California's Office of Environmental Health Hazard Assessment calls them Risk Exposure Levels (RELs). For the purposes of this document, all of these will be given the general name "reference concentrations" (RfCs). For noncancer, Toxicity Reference Values are equal to the Reference Concentrations.

$$TRV_{noncancer,\ chronic}\ (\mu g/m^3) = RfC_{\ chronic}\ (\mu g/m^3)$$

Where:

$TRV_{noncancer,\ chronic}$ = toxicity reference value for chronic exposures leading to noncancer health effects
RfC = reference concentration for chronic exposures leading to noncancer health effects

Inhalation toxicity values for carcinogens are typically provided as inhalation unit risk (IUR) values. For ease of use in developing RBCs, IURs were converted to TRV concentrations using a consistent target excess cancer risk level of one in one million.

$$TRV_{cancer}\ (\mu g/m^3) = \text{Target Risk } (1 \times 10^{-6}) \,/\, IUR\ (\mu g/m^3)^{-1}$$

Where:

TRV_{cancer} = Toxicity Reference Value for cancer

IUR = Inhalation Unit Risk

B.2 Acute Values

The approach used to develop acute TRVs is different from the approach for chronic TRVs because fewer authoritative sources create them, and because the authoritative sources make different assumptions about how long people are exposed. Health risks from inhaling toxic air contaminants are the result not only of how concentrated the contaminants are in the air, but also the amount of time people spend breathing them. In CAO risk assessments, DEQ and OHA assume 24 hours of exposure for acute TRVs. CAO is not intended to be a mechanism to address emergencies where exposures of an hour or less could affect health. There are other mechanisms to address emergencies caused by very high accidental releases. Therefore, when making its recommendations to the EQC, DEQ and OHA selected acute TRVs from among authoritative sources by preference for the authoritative source that used assumptions about exposure times that best matched DEQ and OHA's assumed exposure time of 24 hours. The following authoritative sources are listed in order of preference based on how well their TRVs match DEQ and OHA's assumed 24-hours of exposure.

1. DEQ alone or in consultation with DEQ's ATSAC and/or OHA
2. Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profiles, Acute Minimal Risk Levels (MRLs)
3. California's Office of Environmental Health Hazard Assessment (OEHHA) Acute Reference Level (REL)
4. Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profiles, Intermediate Minimal Risk Levels (MRLs)
5. If no short-term Reference Concentration was available from sources listed here, no short-term Toxicity Reference Value was recommended or proposed

Acute TRVs are only for non-carcinogenic effects. If no short-term toxicity values were available from the above authoritative sources, no short-term TRVs were established. If the short-term TRV was lower than the chronic TRV, the chronic TRV was used for the short-term TRV, because there is generally more confidence in chronic toxicity values. For example, ATSDR's intermediate MRL for vinylidene chloride is 79 $\mu g/m^3$. The chronic noncancer value from IRIS is 200 $\mu g/m^3$. Given the greater confidence in the chronic value, and because it would be inconsistent to have an acute TRV less than a chronic TRV, the chronic noncancer value was used as the acute TRV for vinylidene chloride.

As with chronic noncancer effects, TRVs for acute effects are equal to the RfCs.

$$TRV_{noncancer,\ acute}\ (\mu g/m^3) = RfC_{acute}\ (\mu g/m^3)$$

Where:

$TRV_{noncancer,\ acute}$ = toxicity reference value for acute exposures leading to noncancer health effects
RfC_{acute} = reference concentration for acute exposures leading to noncancer health effects

APPENDIX C

Development of Adjustment Factors and Calculation of Risk-Based Concentrations

C.1 Introduction

When making its recommendations to the EQC, DEQ calculated risk-based concentrations (RBCs) for the following receptors for chronic exposure:

- Residential, including single family homes, apartments, and condominiums
- Non-residential children, including schools and daycare facilities
- Non-residential adults, including commercial and industrial facilities

DEQ also considered short-term acute exposure.

DEQ made three adjustments of Toxicity Reference Values, if appropriate, to calculate RBCs. The first adjustment is for a scenario-specific consideration of exposure frequency and duration. Another adjustment is for deposition and bioaccumulation of toxic air contaminants, which involve exposure routes other than inhalation alone; this is a multipathway adjustment. The third adjustment considers early-life exposure to toxic air contaminants that exhibit greater toxicity to infants and children. These adjustments are reflected in the chronic RBCs listed in OAR 340-245-8040 Table 4. The development of each adjustment factor is discussed below. None of the adjustment factors is appropriate or necessary for acute RBCs because of the short period of exposure being considered.

DEQ may recommend that the EQC update the RBC tables in this protocol periodically as toxicity values are revised by the authoritative sources. Revised RBCs will be adopted by rule. In addition, DEQ may also recommend that exposure factors may be revised as new information becomes available.

C.2 Development of Adjustment Factors

C.2.1 Scenario-Specific Exposure Frequency and Duration Adjustments

Residential exposure assumes continual, long-term exposure. Because continual, long-term exposure is the basis of most chronic toxicity values, TRVs are most directly appropriate for residential exposure. In this case, no exposure modifications of TRVs are necessary for calculating RBCs. For other exposure, including shorter term, nonresidential child exposure such as at schools, and worker exposure at commercial or industrial facilities, modifications to TRVs are needed to take into consideration the differences in exposure frequency and duration.

For non-residential exposure, DEQ used factors for more limited exposure to calculate RBCs, as follows. For noncarcinogenic effects for either workers or children in schools or daycare, the value of the adjustment factor for childNRAFnc and workerNRAFnc represents someone who is present 8 hrs/day and 250 days/yr (5 days/week for 50 weeks):

childNRAFnc = workerNRAFnc = (24 hrs/day / 8 hrs/day) x (365 days/yr / 250 days/yr) = 4.4

Where:
childNRAFnc = Nonresident adjustment factor, child noncancer (unitless)
workerNRAFnc = Nonresident adjustment factor, worker noncancer (unitless)

These factors apply to chronic RBCs for noncarcinogenic effects because we assume effects may occur after a year of exposure. For carcinogens, we also include factors for exposure duration because we assume nonresidents are not present at one location for an entire lifetime of 70 years. We assume that non-resident children may be exposed from infancy through elementary school, for a total of 12 years. The standard assumption for worker exposure duration is 25 years. The exposure frequency assumption is 250 days/yr (5 days/week for 50 weeks). The NRAF values for cancer effects are:

childNRAFc = (24 hrs/day / 8 hrs/day) x (365 days/yr / 250 days/yr) x (70 yrs / 12 yrs) = 26

workerNRAFc = (24 hrs/day / 8 hrs/day) x (365 days/yr / 250 days/yr) x (70 yrs / 25 yrs) = 12

Where:
childNRAFc = Nonresident adjustment factor, child cancer (unitless)
workerNRAFc = Nonresident adjustment factor, worker cancer (unitless)

C2.1.1. Life Expectancy

When making its recommendation to the EQC, DEQ and OHA decided that the value of 70 years used above is an appropriate estimate of lifetime despite EPA's determination in the 2011 Exposure Factors Handbook that average life expectancy is now 78 years (EPA 2011). A change in lifetime only matters for evaluating carcinogenic effects for less than lifetime exposure (such as workers) because residential exposure is evaluated for a lifetime regardless of duration, and noncancer effects are evaluated in a manner that does not incorporate life expectancy. Considering a change in life expectancy involves deciding between two inconsistencies. A decision to stay with a 70-year life expectancy used in risk assessments since the 1980s is inconsistent with current knowledge. A decision to change to a 78-year life expectancy would make current risk assessments for workers inconsistent with prior risk assessments, even though actual risks have not changed. EPA recommends continued use of a 70-year lifetime for Superfund risk assessments; DEQ's Cleanup Program follows this recommendation. DEQ determined that it is appropriate for Cleaner Air Oregon risk assessments to use a 70-year lifetime, and the EQC adopted that approach. This decision is slightly more protective than assuming a 78-year lifetime.

C.2.2 Multipathway Adjustment Factors

DEQ considered developing Multipathway Adjustment Factors (MPAFs) specific to Oregon, but determined that the agency had neither the time nor resources to undertake this effort. After evaluating Multipathway Adjustment Factors from other agencies, including Minnesota (MPCA 2016) and California's South Coast Air Quality Management District (SCAQMD 2016b), DEQ decided to use the Multipathway Adjustment Factors from SCAQMD because of the extensive modeling performed for the development of the Multipathway Adjustment Factors, and the large list of toxic air contaminants evaluated. DEQ acknowledges that exposure conditions may not be the same in Oregon, but considers the MPAFs appropriately protective.

DEQ only applied MPAFs in development of chronic RBCs, and not in development of acute RBCs. Acute RBCs are equal to acute TRVs in OAR 340-245-8030 Table 3. Acute RBCs only consider risks posed by direct inhalation. Assessment of acute risk need not include multipathway analysis.

C.2.3 Early-Life Adjustment Factors

Carcinogens that harm a cell's genetic material can have greater toxicity during early-life stages such as infancy and early childhood than in adulthood (EPA 2005a). In these cases, we cannot use the cancer Toxicity Reference Value without modification. For most carcinogenic toxic air contaminants acting by a mutagenic mode of action, we use EPA's general approach to account for early-life exposure using age-dependent adjustment factors, ADAFs. The approach is different for two toxic air contaminants. For trichloroethene (TCE), EPA considers early-life appropriate for liver cancer only, and not kidney cancer or non-Hodgkin's Lymphoma. This makes the development of an early-life adjustment factor for TCE more complicated. For vinyl chloride, EPA determined that it should continue to be evaluated using a specific procedure for evaluating early-life exposure. Because of the many details necessary in evaluating early-life exposure, we provide the development of early-life adjustment factors (ELAFs) separately, in Appendix D.

C.3 Calculation of RBCs

C.3.1 Residential RBCs

DEQ applied the multipathway adjustment factor (MPAF) and early-life adjustment factor (ELAF) values shown in Table C-1 to the TRVs in OAR 340-245-8030 Table 3 using the following equations to calculate residential risk-based concentrations (RBCs) in OAR 340-245-8040 Table 4.

$$residRBCc = \frac{TRVc}{ELAFr \cdot MPAFrc}$$

$$residRBCnc = \frac{TRVnc}{MPAFrnc}$$

Where:
residRBCc = Residential risk-based concentration for cancer effects (μg/m^3)
residRBCnc = Residential risk-based concentration for noncancer effects (μg/m^3)
TRVc = Toxicity reference value for cancer effects (μg/m^3)
TRVnc = Toxicity reference value for noncancer effects (μg/m^3)
ELAFr = Early-life adjustment factor, resident (unitless)
MPAFrc = multipathway adjustment factor, resident cancer (unitless)
MPAFrnc = multipathway adjustment factor, resident noncancer (unitless)

If multipathway or early-life considerations are not relevant for a toxic air contaminant, these adjustments are omitted. For most toxic air contaminants, this is the case, and the residential RBC is equal to the TRV.

C.3.2 Non-Residential RBCs

In addition to considerations of MPAF and ELAF for chronic exposure, exposure frequency and exposure duration are also included for non-residential scenarios where exposure will be less than continual exposure for a lifetime. DEQ used the following equations to calculate non-residential RBCs.

$$nrchildRBCc = \frac{TRVc \cdot childNRAFc}{ELAFnr \cdot MPAFnrc}$$

$$nrchildRBCnc = \frac{TRVnc \cdot childNRAFnc}{MPAFnrnc}$$

$$workerRBCc = \frac{TRVc \cdot workerNRAFc}{MPAFnrc}$$

$$workerRBCnc = \frac{TRVnc \cdot workerNRAFnc}{MPAFnrnc}$$

Where:
nrchildRBCc = Nonresidential child risk-based concentration for cancer effects (µg/m^3)
nrchildRBCnc = Nonresidential child risk-based concentration for noncancer effects (µg/m^3)
workerRBCc = Nonresidential worker risk-based concentration for cancer effects (µg/m^3)
workerRBCnc = Nonresidential worker risk-based concentration for noncancer effects (µg/m^3)
TRVc = Toxicity reference value for cancer effects (µg/m^3)
TRVnc = Toxicity reference value for noncancer effects (µg/m^3)
ELAFnr = Early-life adjustment factor, non-resident (unitless)
MPAFnrc = Multipathway adjustment factor, nonresident cancer (unitless)
MPAFnrnc = Multipathway adjustment factor, nonresident noncancer (unitless)
childNRAFc = Nonresident adjustment factor, child cancer (26) (unitless)
childNRAFnc = Nonresident adjustment factor, child noncancer (4.4) (unitless)
workerNRAFc = Nonresident adjustment factor, worker cancer (12) (unitless)
workerNRAFnc = Nonresident adjustment factor, worker noncancer (4.4) (unitless)

If multipathway or early-life considerations are not relevant for a toxic air contaminant, these adjustments are omitted.

C.3.3 Acute RBCs

The acute Toxicity Reference Value is used directly as the acute Risk-Based Concentration.

$$acuteRBC = TRVa$$

Where:

acuteRBC = Acute risk-based concentration (µg/m^3)

TRVa = Toxicity reference value for acute effects (µg/m^3)

Table C-1
Adjustment Factors for Risk-Based Concentrations[a,b]
Multipathway, Early-Life, and Non-Resident Adjustment Factors

	Chronic Cancer				Chronic Noncancer	
	Early-Life[d]		Multipathway[c]		Multipathway[c]	
Toxic Air Contaminant	**Resident ELAFr**	**Non-Resident ELAFnr**	**Resident MPAFrc**	**Non-Resident MPAFnrc**	**Resident MPAFrnc**	**Non-Resident MPAFnrnc**
Acrylamide	1.7	4.2				
Arsenic			9.7	4.5	88	28
Benzidine (and its salts)	1.7	4.2				
Benzo[a]pyrene	1.7	4.2	23	6.6		
Bis-(2-ethylhexyl)phthalate (DEHP)			5.2	1		
Cadmium			1	1	2.0	1.2
Chromium VI[g]	1.7	4.2	1.6	1	2.4	1
Coke Oven Emissions	1.7	4.2				
1,2-Dibromo-3-chloropropane (DBCP)	1.7	4.2				
Dichloromethane (methylene chloride)	1.7	4.2				
Ethylene oxide	1.7	4.2				
Fluorides					5.7	2.9
Hexachlorocyclohexanes (mixture)			5.4	1.3	1	1
alpha-Hexachlorocyclohexane			5.4	1.3	1	1
beta-Hexachlorocyclohexane			5.4	1.3	1	1
gamma-Hexachlorocyclohexane			5.4	1.3	1	1
Hydrogen fluoride					6.1	3.0
Lead			11	5.8		
Mercury					3.9	2.1
4,4'-Methylene dianiline (and its dichloride)			7.2	2.5	1	1
Naphthalene			1	1	1	1

Table C-1
Adjustment Factors for Risk-Based Concentrations[a,b]
Multipathway, Early-Life, and Non-Resident Adjustment Factors

	Chronic Cancer				Chronic Noncancer	
	Early-Life[d]		Multipathway[c]		Multipathway[c]	
Toxic Air Contaminant	**Resident ELAFr**	**Non-Resident ELAFnr**	**Resident MPAFrc**	**Non-Resident MPAFnrc**	**Resident MPAFrnc**	**Non-Resident MPAFnrnc**
N-Nitrosodiethylamine	1.7	4.2				
N-Nitrosodimethylamine	1.7	4.2				
Polychlorinated biphenyls (PCBs)			19	13	240	11
Polychlorinated biphenyls (PCBs) TEQ[h]			26[i]	7.6[i]	310[i]	6.7[i]
Polychlorinated dibenzo-*p*-dioxins (PCDDs) & Polychlorinated dibenzofurans (PCDFs) TEQ[h]			26[i]	7.6[i]	310[i]	6.7[i]
Polycyclic aromatic hydrocarbons (PAHs)	1.7	4.2	23	6.6		
Trichloroethene (TCE)	1.2[e]	1.8[e]				
Urethane (ethyl carbamate)	1.7	4.2				
Vinyl chloride	2[f]	27[f]				

Notes:

a Application of adjustments factors in calculating RBCs:
Resident RBC cancer = TRVc / ELAFr / MPAFrc
Resident RBC noncancer = TRVnc / MPAFrnc
Non-resident RBC child cancer = TRVc x childNRAFc / ELAFnr / MPAFnrc
Non-resident RBC child noncancer = TRVnc x childNRAFnc / MPAFnrnc
Worker RBC cancer = TRVc x workerNRAFc / MPAFnrc
Worker RBC noncancer = TRVnc x workerNRAFnc / MPAFnrnc
TRVc = Toxicity reference value, cancer
TRVnc = Toxicity reference value, noncancer

b <u>Additional adjustment factors:</u>
childNRAFnc = Non-residential adjustment factor, noncancer, child = 4.4
workerNRAFnc = Non-residential adjustment factor, noncancer, worker = 4.4
Chronic RBCs are based on continual exposure to residents for 70 years. The adjustment for non-resident exposure is:
(24 hours/day / 8 hours/day) x (365 days/year / 250 days/year) = 4.4
childNRAFc = Non-residential adjustment factor, child, cancer = 26
For carcinogenic effects to children, the non-residential exposure duration assumption is 12 years (infant through elementary school), resulting in a childNRAFc value of:

(70 years / 12 years) x (365 days/year / 250 days/year) x (24 hours/day / 8 hours/day) = 26
workerNRAFc = Non-residential adjustment factor, adult worker, cancer = 12
The adjustment for non-resident worker exposure working for 25 years is:
(70 years / 25 years) x (365 days/year / 250 days/year) x (24 hours/day / 8 hours/day) = 12

c MPAF = multipathway adjustment factor. Sources of multipathway adjustment factors:
South Coast Air Quality Management District, Permit Application Package "M", March 2016, Table 8-1.
South Coast Air Quality Management District, Facility Prioritization Procedures for AB 2588 Program, Nov. 2016, Table 3.
Toxic air contaminants for which there are MPAFs are considered persistent, bioaccumulative and toxic substances.

d ELAF = early-life adjustment factor. ELAFs apply to toxic air contaminants determined by EPA to be carcinogens acting by a mutagenic mode of action. The standard ELAF approach is to use age-dependent adjustment factors (ADAFs) of 10 for infants up to 2 years old, and 3 for children aged 2 to 16, unless EPA determines that a chemical-specific approach is appropriate. For applicable toxic air contaminants, ELAFs are incorporated in the derivation of residential and nonresident child RBCs.

e Early-life adjustment factor for TCE developed by applying ADAFs to one of three toxic endpoints for TCE.

f Early-life adjustment factor for vinyl chloride developed by assuming exposure during early-life doubles the lifetime cancer risk without early-life exposure. These ELAF values apply to the IUR of 4.4×10^{-6} $(\mu g/m^3)^{-1}$ [TRV = 0.22 $\mu g/m^3$], not the adult/child IUR of 8.8×10^{-6} $(\mu g/m^3)^{-1}$ used to calculate the TRV of 0.11 $\mu g/m^3$.

g Adjustment factors for chromium VI apply to both chromate and dichromate particulates, and chromic acid aerosol mist.

h TEQ = toxic equivalency (relative to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin)

i Multipathway adjustment factors are for PCDDs.

APPENDIX D

Derivation of Early-Life Adjustment Factors

D.1 Introduction

This appendix covers the development of early-life adjustment factors (ELAFs) and the evaluation of early-life exposure for certain compounds. For toxic air contaminants that are carcinogens having a mutagenic mode of action, risk may not be fully assessed without incorporation of early-life exposure. Such toxic air contaminants are shown in Table D-1. We provide a general discussion below, with specific evaluations of TCE and vinyl chloride. In the future, as more information becomes available, early-life exposure may need to be considered for other toxic air contaminants. Early-life exposure is included in the derivation of RBCs for residential and non-residential child exposure scenarios.

D.2 Background

In March 2005, EPA issued new *Guidelines for Carcinogenic Risk Assessment* (EPA 2005a), updating the 1986 guidelines and 1999 interim final guidelines. Also included was *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens* (EPA 2005b). In the *Supplemental Guidance,* EPA concluded that some toxic air contaminants, specifically carcinogens acting by a mutagenic mode of action, have a greater cancer impact if exposure occurs during childhood. DEQ included early-life exposure in the derivation of RBCs for the relevant toxic air contaminants. The general approach is to evaluate cancer risk using different adjusted potency factors for three life stages (0 – 2 years, 2 – 16 years, and adult).

EPA created workgroups to provide additional information on how to implement the *Supplemental Guidance*, and provide consistency. One outcome of the workgroups is an EPA memorandum clarifying which toxic air contaminants should be evaluated for early-life exposure (EPA 2006). The list of mutagenic toxic air contaminants, with updates, is provided in Table D-1. Most toxic air contaminants with early-life exposure considerations are evaluated using the default approach. For TCE, EPA considers early-life appropriate for liver cancer only, and not kidney cancer or non-Hodgkin's Lymphoma. Because of this complication, the approach for TCE is discussed separately. In addition, EPA determined that vinyl chloride should continue to be evaluated using a specific procedure for evaluating early-life exposure, so vinyl chloride is also discussed separately.

D.3 Default Early-Life Adjustment Factors

Risk assessments for carcinogens acting by a mutagenic mode of action (excluding vinyl chloride discussed below) include a term called an age dependent adjustment factor (ADAF) to account for increased carcinogenic potency during early life stages. For ages up to 2 years, the ADAF is 10, indicating a ten-fold increase in carcinogenic potency during this period. For ages from 2 years to 16 years, the ADAF is 3. For ages 16 years and older, the ADAF is 1. Using ADAFs, the differences in potency are incorporated by a factor separate from the inhalation unit risk factor, so only one cancer IUR is needed. In the CAO program, DEQ uses the corresponding TRV rather than the IUR. Risk assessments for carcinogens that do not act by a

mutagenic mode of action should be conducted using the TRV without adjustments for age.

In developing exposure parameters for children, EPA decided that it would be more accurate to divide the 2- to 16-year-old stage into two stages (2 to 6 years, and 6 to 16 years). Both stages have the same ADAF value. For inhalation exposure, it is not necessary to separate these age groups, so they are combined in the equation below.

The incorporation of ADAFs is best included in the calculation of age-adjusted exposure duration for inhalation exposure. These factors are used both in forward risk assessments and calculations of RBCs. Equations for age-adjusted intake factors are presented in DEQ's RBDM guidance (DEQ 2003). For carcinogens acting by a mutagenic mode of action, these equations should be modified as follows:

$$ED_{adj} = ED_2\ ADAF_2 + ED_{16}\ ADAF_{16} + ED_{adult}\ ADAF_{adult}$$

Where:

$ADAF_2$ = Age-dependent Adjustment Factor, child 0 to <2 years old (unitless)
$ADAF_{16}$ = Age-dependent Adjustment Factor, child 2 to <16 years old (unitless)
$ADAF_{adult}$ = Age-dependent Adjustment Factor, adult (unitless)
ED_2 = Exposure duration, child 0 to <2 years old (yr)
ED_{16} = Exposure duration, child 2 to <16 years old (yr)
ED_{adult} = Exposure duration, adult (yr)

This approach is discussed in DEQ's risk assessment guidance for the Cleanup Program (DEQ 2010). Default parameter values are shown in Table D-2.

The early-life adjustment factor for residential exposure is the ratio of early-life exposure duration to general exposure.

ELAFr = (EDadj-r / EDr = [(2 yr x 10) + (14 yr x 3) + (54 yr x 1)] / (70 yr) = 116 yr / 70 yr = 1.66

Where:
ELAFr = Early-life adjustment factor for residential exposure
EDadj-r = Exposure duration, adjusted for early-life, residential
EDr = Exposure duration for residential

For nonresidential child exposure, we assume exposure from infancy through elementary school, for a total exposure duration of 12 years. Other factors, such as exposure frequency (250 days/year) and exposure time (8 hours/day), are already accounted for in the non-residential adjustment factor. The nonresidential ELAF is the ratio of early-life exposure to general exposure for the same duration.

ELAFnr = (EDadj x EFnr) / ED = [(2 yr x 10) + (10 yr x 3)] / 12 yr = 50 yr / 12 yr = 4.2

Where:
ELAFnr = Early-life adjustment factor for nonresidential exposure
EDadj-nr = Exposure duration, adjusted for early-life, nonresidential
EDnr = Exposure duration for nonresidential

The default ELAF values are applied to the list of toxic air contaminants with early-life

adjustments in Table D-1, with the exception of TCE and vinyl chloride, which are addressed using the approaches described below.

D.4 Calculation of ELAFs for TCE

One issue that complicates the derivation of RBCs for TCE concerns the incorporation of early-life exposure. There are three cancer endpoints considered in the development of the carcinogenic slope factor and inhalation unit risk (IUR) factor for TCE: kidney cancer, liver cancer, and non-Hodgkin's lymphoma. EPA determined that TCE was carcinogenic by a mutagenic mode of action for kidney cancer (renal cell carcinoma). Accordingly, age-dependent adjustment factors should be used to evaluate early-life exposure to TCE for this endpoint, presumed to be initiated by a mutagenic mode of action. However, EPA did not determine that there is a mutagenic mode of action for the other two cancer endpoints. The precise method for calculating RBCs for TCE is to use slope factors and IURs for each cancer endpoint, determine an RBC for kidney cancer using ADAFs, determine RBCs for liver cancer and non-Hodgkin's lymphoma without assuming early-life exposure, and combine the individual endpoint RBCs to get a comprehensive RBC using the following equation:

$$RBC_{TCE\text{-}total} = \frac{1}{(1/RBC_{TCE\text{-}kidney}) + (1/RBC_{TCE\text{-}liver}) + (1/RBC_{TCE\text{-}lymphoma})}$$

DEQ used this approach to develop the current RBCs for TCE using default exposure assumptions. To develop site-specific RBCs for TCE, we determined RBCs separately for each toxic endpoint, and then combined the RBCs to derive a total RBC as shown above. The toxicity values for each endpoint are the following:

Toxic Endpoint	TCE Inhalation Unit Risk IUR $(\mu g/m^3)^{-1}$	TCE Toxicity Reference Value TRV $(\mu g/m^3)$
Kidney cancer	1.0×10^{-6}	1
Liver cancer	1.0×10^{-6}	1
Non-Hodgkin's lymphoma	2.1×10^{-6}	0.476
Total	4.1×10^{-6}	0.244

Note:
TRV based on one-in-one-million excess cancer risk.

D.5 Calculation of ELAFs for Vinyl Chloride

EPA's Integrated Risk Information System (IRIS) report for vinyl chloride includes two derivations of IUR factors, one based on the linearized multistage (LMS) procedure, and one based on the LED_{10} approach (EPA 2000). The LED_{10} is the lower 95% limit on a dose that is estimated to cause a 10% response. The results are similar, but the LMS approach is used here because that is what is currently used by the EPA regions. For vinyl chloride, LMS values are slightly less conservative than IUR factors based on the LED_{10} approach.

EPA provided IUR factors separately for lifetime exposure as an adult, and lifetime exposure beginning from birth. The values differ by a factor of 2. The unit risk factors provided in IRIS for inhalation exposure are 4.4×10^{-6} risk per $\mu g/m^3$ for adult exposure, and 8.8×10^{-6} risk per $\mu g/m^3$ for adult/child exposure. The Air Toxics Science Advisory Committee chose to use the

adult/child IUR in developing an ambient benchmark concentration for vinyl chloride. Because the ATSAC decision on the ABC was selected as the basis for the vinyl chloride TRV, we used the adult/child IUR for developing RBCs. This simplifies the development of an RBC for residential exposure, but complicates a non-residential child RBC.

An example is presented below for the calculation of the inhalation RBC for vinyl chloride. You can use similar concepts in a forward risk assessment.

For vinyl chloride, EPA concludes that because the effects of early-life exposure are qualitatively and quantitatively different from those of later exposures, it is not appropriate to prorate early-life exposures as if they were received at a proportionately lesser rate over a full lifetime. This feature of vinyl chloride toxicity must be considered in the derivation of RBCs for nonresidential exposure. It is already covered in the derivation of the residential RBC.

Following EPA's example, early-life exposure is estimated assuming a lifetime of exposure using the lower (adult) slope factor. For an exposure scenario involving both early-life and additional exposure, the early-life exposure (which is a single value and is not pro-rated for reduced exposure time) is added to a child's nonresidential exposure (which can be pro-rated).

To show explicitly how early-life and adult exposure are incorporated, the following is the general RBC equation:

$$RBC_{air} = \frac{AT_c \cdot 365\,\text{days/yr} \cdot TRV}{\text{ED} \cdot \text{EF}_\text{r}}$$

Where:
RBC_{air} = Risk based concentration for inhalation of air (µg/m^3)
ATc = Averaging time, carcinogens (70 years)
ED = Exposure duration (yr)
EF_r = Exposure frequency, residential (365 days/year)
TRV = Toxicity reference value (µg/m^3)

Because DEQ followed the ATSAC recommendation to develop a vinyl chloride TRV that includes early-life exposure, for this more detailed calculation we multiplied the early-life TRV of 0.114 µg/m^3 by 2 to get a non-early-life TRV of 0.228 µg/m^3.

The RBC equation was applied separately for early-life exposure, and exposure other than early-life. Early-life exposure is assumed equivalent to a lifetime of adult exposure (70 years).

$$RBC_{early-life} = \frac{70\ yr\ \cdot 365\frac{days}{yr} \cdot 0.228\ \mu g/m^3}{70\ yr\ \cdot 365\frac{days}{yr}}$$

$$= \quad 0.228\ \mu\text{g/m}^3$$

For the other exposure to a nonresidential child, the RBC is:

$$RBC_{child} = \frac{70\ yr\ \cdot 365\frac{days}{yr}\cdot 24\frac{hrs}{day}\cdot\ 0.228\ \mu g/m^3}{12\ yr\ \cdot 250\frac{days}{yr}\cdot 8\frac{hrs}{day}}$$

$$= \quad 5.83\ \mu g/m^3$$

Because the definition of "early-life" is not clearly defined for vinyl chloride, including the full non-residential child exposure duration assumption of 12 years for this calculation may slightly overestimate risk.

The RBC for combined exposure as a child and adult is calculated using the following relationship:

$$\frac{1}{RBC_{early-life/child}} = \frac{1}{RBC_{early-life}} + \frac{1}{RBC_{child}}$$

$$RBC_{early-life/child} = \frac{1}{\frac{1}{RBC_{early-life}} + \frac{1}{RBC_{child}}}$$

$$RBC_{early-life/child} = \frac{1}{\frac{1}{0.228} + \frac{1}{5.83}}$$

$$= \ 0.22\ \mu g/m^3$$

This same approach can be used for other scenarios in performing a risk assessment for vinyl chloride.

Table D-1
Toxic Air Contaminant Determined by EPA to be Carcinogens Having a Mutagenic Mode of Action

Toxic air contaminant[a]	Chemical Abstract Service Registration Number
Acrylamide	79-06-1
Benzidine	92-87-5
Coke Oven Emissions	
1,2-Dibromo-3-chloropropane (DBCP)	96-12-8
Ethylene oxide	75-21-8
N-Nitrosodiethylamine	55-18-5
N-Nitrosodimethylamine	62-75-9
Polycyclic aromatic hydrocarbons	
Benz[a]anthracene[b]	56-55-3
Benzo[b]fluoranthene[b]	205-99-2
Benzo[k]fluoranthene[b]	207-08-9
Benzo[a]pyrene	50-32-8
Chrysene[b]	218-01-9
Dibenz[a,h]anthracene	53-70-3
Indeno[1,2,3-cd]pyrene[b]	193-39-5
Trichloroethene[c] (TCE)	79-01-6
1,2,3-Trichloropropane	96-18-4
Urethane (ethyl carbamate)	51-79-6
Vinyl chloride[d]	75-01-4

Notes:

a) Source: EPA 2006, and EPA Regional Screening Level table, 2018.
b) Although not explicitly included in EPA's list, EPA states that carcinogenic PAHs with a relative potency factor relating the toxicity to the slope factor for benzo[a]pyrene should also be evaluated for early-life exposure.
c) Of the three cancer endpoints considered in the development of the inhalation unit risk (IUR) factor for TCE (kidney cancer, liver cancer, and non-Hodgkin's lymphoma), EPA determined that TCE was carcinogenic by a mutagenic mode of action for kidney cancer (renal cell carcinoma), but not the other endpoints. Age-dependent adjustment factors should be used to evaluate early-life exposure to TCE for kidney cancer, but not the other endpoints.
d) EPA has a specific method for evaluating early-life exposure to vinyl chloride, as presented in EPA's Integrated Risk Information System (www.epa.gov/iris).

Table D-2
Default Parameter Values for Early-Life Exposure

Parameter	<2 Years Old	2 to <6 Years Old	6 to <16 Years Old	Adult
ADAF (unitless)[a]	10	3	3	1
ED (yr)[b] residential[c]	2	4	10	54
nonesidential[d]	2	4	6	0
BW (kg)[b]	15	15	80	80
IRS (mg/d)[b]	200	200	100	100
IRW (L/d)[b]	0.78	0.78	2.5	2.5
AF (mg/cm^2·event)	0.2	0.2	0.07	0.07
SA (cm^2)[b]	2,690	2,690	6,032	6,032
IRA (m^3/d)[b]	10	10	20	20

Notes:

a) Age-dependent adjustment factor (ADAF) values taken from EPA 2005b.
b) Exposure values taken from *Exposure Factors Handbook* (EPA 2011), *Risk Assessment Guidance for Superfund* (EPA 1989), and EPA Regional Screening Levels (EPA 2018).
ED = exposure duration BW = body weight
IRS = ingestion rate, soil IRW = ingestion rate, water
AF = adherence factor SA = skin surface area
IRA = inhalation rate, air
c) The standard residential default exposure duration is 70 years.
d) The nonresidential default exposure duration is 12 years, infancy through elementary school.

Appendix E

Use of the Toxic Equivalency Factor Methodology for Dioxins and Furans, PCBs, and PAHs

E.1 Introduction

The toxicity equivalency factor (TEF) methodology was developed by the U.S. Environmental Protection Agency (EPA) to evaluate the toxicity and assess the risks of a mixture of structurally-related chemicals with a common mechanism of action. Both EPA and the World Health Organization (WHO) use TEFs to evaluate mixtures of polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzo-*p*-furans (PCDDs and PCDFs) and mixtures of dioxin-like polychlorinated biphenyls (PCBs). TEF methodology specific to mixtures of polycyclic aromatic hydrocarbons (PAHs) are used by the California EPA, the Washington Department of Ecology, and DEQ's Cleanup Program. Further details for each of these three types of mixtures are presented below.

E.2 Polychlorinated Dibenzo-*p*-dioxins (PCDDs) and Dibenzofurans (PCDFs)

There are 7 distinct PCDD compounds and 10 distinct PCDF compounds, all of which are referred to as congeners. All 17 dioxin/furan congeners are structurally similar and have the same mechanism of toxicity. Because of their similarities, the combined toxicity of these 17 compounds can be estimated using the sum of their doses, which are scaled for potency relative to one component of the mixture for which adequate dose-response toxicity information is available (EPA 2000); this compound is referred to as an "index" chemical. Of these 17 congeners, the toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (commonly referred to as 2,3,7,8-TCDD) has been the most extensively studied, and is used as the index chemical.

Each of the congeners is assigned a TEF which represents the relative potency, or toxicity, of each congener to 2,3,7,8-TCDD. Thus, for 2,3,7,8-TCDD, the TEF is 1.0. The TEFs assigned to each dioxin/furan congener are presented in Table E-1.

To evaluate cumulative risk, the concentrations of each of the 17 congeners is multiplied by its specific TEF. Then those 17 adjusted concentrations are summed to produce a Toxic Equivalency, or TEQ, concentration. The TEQ concentration is then compared to the toxicity value for 2,3,7,8-TCDD to determine whether dioxins and furans are present at levels that will cause unacceptable impacts to human health.

The TEF normalization process described above is based on the use of oral toxicity factors. EPA states that TEFs may be applied to other exposure routes, including inhalation, as an interim estimate (*Recommended Toxicity Equivalence Factors (TEFs) for Human Health Risk Assessments of 2,3,7,8-Tetrachlorodibenzo-p-dixoin and Dioxin-Like Compounds*, EPA 2010a).

The Risk Based Concentrations for total dioxins and furans, treated as 2,3,7,8-TCDD TEQ, are 3×10^{-8} $\mu g/m^3$ for carcinogenic effects, and 4×10^{-5} $\mu g/m^3$ for non-carcinogenic effects. These

protective concentrations were obtained using the 1996 OEHHA Inhalation Risk Unit value of 38 per $\mu g/m^3$ for 2,3,7,8-TCDD, and the OEHHA RfC of 4×10^{-5} $\mu g/m^3$, respectively.

In formula form, the TEQ for PCDDs and PCDFs is calculated as:

$$TEQ_{PCDDs,PCDFs} = \sum_{i=1}^{7}\left(PCDD_i \cdot TEF_i\right) + \sum_{i=1}^{10}\left(PDCF_i \cdot TEF_i\right)$$

E.3 Polychlorinated Biphenyls (PCBs)

Polychlorinated biphenyls are comprised of a group of 209 congeners, 12 of which are considered dioxin-like in terms of their structural similarity and mechanism of toxicity. Most people are more familiar with the term “PCB Aroclors”. Aroclors are specific mixtures of portions of the 209 PCB congeners (for example, Aroclor 1260, Aroclor 1254), and were created by Monsanto and used commercially to insulate and cool electrical equipment from the 1930s up to 1977, when Monsanto ceased production. EPA banned the use of Aroclors in 1979. However, because PCBs are extremely persistent and bioaccumulate through the food chain, the residual PCBs related to past Aroclor use still exist today, and have spread globally. As Aroclor mixtures deteriorate, their original mixture of PCB congeners changes over time. Therefore, there is a need to be able to evaluate mixtures of PCB congeners rather than Aroclors, although Aroclors are still evaluated in special cases. The term “Total PCBs” is used in different ways, depending on the situation: 1) for a sum of all 209 PCB congeners; 2) for a sum of Aroclors. Additionally and separately, a sum of normalized concentrations of the 12 dioxin-like PCB congeners can be evaluated using a Toxic Equivalency Factor (TEF) methodology.

E.3.1 Total PCBs

The Risk Based Concentration for total polychlorinated biphenyls is 0.01 $\mu g/m^3$, and should be compared to a straight summed concentration of all 209 PCB congeners in a mixture.

E.3.2 Dioxin-Like PCB Congeners

The 12 dioxin-like PCB congeners are evaluated by applying a TEF methodology, using the dioxin 2,3,7,8-TCDD as the “index” chemical to which the TEQ for the 12 dioxin-like PCB congeners are compared. The 12 dioxin-like congeners are known to be carcinogenic, and typically are assumed to be of more concern than the remaining 197 PCB congeners. Each of the 12 dioxin-like PCB congeners has an assigned TEF (World Health Organization 2005, EPA 2010a); please refer to Table E-2.

To evaluate the concentration of a PCB mixture which contains the dioxin-like congeners, each dioxin-like PCB congener is multiplied by its assigned TEF, and then the results for all 12 are summed to produce a Toxic Equivalency (TEQ) concentration, which is then compared to the toxicity value for 2,3,7,8-TCDD.

Just as with the evaluation of dioxins and furans, the Risk Based Concentrations for the sum of the 12 dioxin-like PCBs, treated as 2,3,7,8-TCDD TEQ, are 3×10^{-8} $\mu g/m^3$ for carcinogenic effects, and 4×10^{-5} $\mu g/m^3$ for non-carcinogenic effects. These protective concentrations were

obtained using the 1996 OEHHA Inhalation Risk Unit value of 38 per μg/m^3 for 2,3,7,8-TCDD, and the OEHHA RfC of 4×10^{-5} μg/m^3, respectively.

In formula form, the TEQ for dioxin-like PCB congeners is calculated as:

$$TEQ_{pcb} = \sum_{i=1}^{12} \left(PCB_i \cdot TEF_i \right)$$

E.4 Polycyclic Aromatic Hydrocarbons

Polycyclic aromatic hydrocarbons (PAHs) are produced whenever fossil fuels or organic matter is combusted. PAHs can also exist as contaminants in uncombusted petroleum products. Several PAHs can increase the risk of developing cancers, and one PAH (benzo[a]pyrene) can also impair normal fetal development. Concentrations of individual PAHs should be normalized to a concentration of the PAH benzo[a]pyrene using TEFs. Once this normalization is completed and the TEF results summed, the resulting TEQ concentration can be compared to the toxicity value for benzo[a]pyrene.

The list of 26 PAHs shown in Table E-3 should be used to generate a concentration for total PAHs. Please note that current laboratory analytical methods are available for only a subset of the PAHs in Table E-3.

Because benzo[a]pyrene has both cancer and non-cancer effects, the concentration of benzo[a]pyrene as an individual PAH should also be compared separately against the non-cancer RBC for benzo[a]pyrene, which is an RfC value of 0.002 μg/m^3 (EPA 2017). Because the RfC is based on developmental effects, this value for benzo[a]pyrene should be compared to 24-hour-based concentrations as well as annual averaged concentrations of this PAH.

Naphthalene is both a representative volatile PAH and was the single most emitted PAH in Oregon circa 2005. Thus, at that time, naphthalene was evaluated separately from the other PAHs. Naphthalene is still evaluated separately, and is not included in the summed total carcinogenic PAHs.

E.5 References

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Table E-1: Toxicity Equivalency Factors for Dioxin/Furan Congeners

Congener	Toxicity Equivalency Factor[1]
PCDDs	
2,3,7,8-TetraCDD	1
1,2,3,7,8-PentaCDD	1
1,2,3,4,7,8-HexaCDD	0.1
1,2,3,6,7,8-HexaCDD	0.1
1,2,3,7,8,9-HexaCDD	0.1
1,2,3,4,6,7,8-HeptaCDD	0.01
1,2,3,4,6,7,8,9-OctaCDD	0.0003
PCDFs	
2,3,7,8-TetraCDF	0.1
1,2,3,7,8-PentaCDF	0.03
2,3,4,7,8-PentaCDF	0.3
1,2,3,4,7,8-HexaCDF	0.1
1,2,3,6,7,8-HexaCDF	0.1
2,3,4,6,7,8-HexaCDF	0.1
1,2,3,7,8,9-HexaCDF	0.1
1,2,3,4,6,7,8-HeptaCDF	0.01
1,2,3,4,7,8,9-HeptaCDF	0.01
1,2,3,4,6,7,8,9-OctaCDF	0.0003

Note

1) From Van den Berg et al. (2006); adopted for use by the World Health Organization and by USEPA (2010).

Table E-2: Dioxin-Like PCB Congeners and Related TEFs

Congener	TEF[1]
3,3',4,4'-Tetrachlorinated biphenyl (PCB 77)	0.0001
3,4,4',5-TetraCB (PCB 81)	0.0003
3,3',4,4',5-PentaCB (PCB 126)	0.1
3,3',4,4',5,5'-HexaCB (PCB 169)	0.03
2,3,3',4,4'-PentaCB (PCB 105)	0.00003
2,3,4,4',5-PentaCB (PCB 114)	0.00003
2,3',4,4',5-PentaCB (PCB 118)	0.00003
2',3,4,4',5-PentaCB (PCB 123)	0.00003
2,3,3',4,4',5-HexaCB (PCB 156)	0.00003
2,3,3',4,4',5'-HexaCB (PCB 157)	0.00003
2,3',4,4',5,5'-HexaCB (PCB 167)	0.00003
2,3,3',4,4',5,5'-HeptaCB (PCB 189)	0.00003

Note

1) From Van den Berg et al. (2006); adopted for use by the World Health Organization and by USEPA (2010).

Table E-3. Recommended Revised List of PAHs and Related TEFs (Reduction from 2005 list of 32 PAHs to proposed 26)

#	PAH	EPA Required (1) [a]	EPA Requested (14) [b]	From MN list (11) [c]	TEF [c,d]
1	5-Methylchrysene			⊙	1 [d]
2	6-Nitrochrysene			⊙	10 [d]
3	Acenaphthene		⊙		NA
4	Acenaphthylene		⊙		NA
5	Anthanthrene			⊙	0.4
6	Anthracene		⊙		0
7	Benz(a)anthracene		⊙		0.2
8	Benzo(a)pyrene	⊙			1
9	Benzo(b)fluoranthene		⊙		0.8
10	Benzo(c)fluorene			⊙	20
11	Benzo(e)pyrene		⊙		NA
12	Benzo(g,h,i)perylene			⊙	0.009
13	Benzo(j)fluoranthene			⊙	0.3
14	Benzo(k)fluoranthene		⊙		0.03
15	Chrysene		⊙		0.1
16	Cyclopenta[c,d]pyrene			⊙	0.4
17	Dibenz(a,h)anthracene		⊙		10
18	Dibenzo(a,e)pyrene			⊙	0.4
19	Dibenzo(a,h)pyrene			⊙	0.9
20	Dibenzo(a,i)pyrene			⊙	0.6
21	Dibenzo(a,l)pyrene			⊙	30
22	Fluoranthene		⊙		0.08
23	Fluorene		⊙		NA
24	Indeno(1,2,3-c,d)pyrene		⊙		0.07
25	Phenanthrene		⊙		0
26	Pyrene		⊙		0

Notes:

[a] Naphthalene is also required, but already has its own risk-based concentration.

[b] Per EPA National Air Toxics Trend Sites (NATTS) Technical Assistance Document (TAD) 2009, Revision 2, Table 1.1-1. Note that the most-current version of NATTS, published in 2016, requests the same list of PAHs as those presented in the 2009 NATTs.

[c] PAHs on Minnesota Department of Health 2014 list of 19 priority cPAHs that are not already required or requested by EPA.

[d] Values were obtained from an External Review Draft version of EPA's 2010 *Development of a relative potency factor (RPF) approach for polycyclic aromatic hydrocarbon (PAH) mixtures*. Although this document is not supposed to be cited or quoted, the Air Toxics Science Advisory Committee considers this information to be the best and most current science available on this topic. A portion of the TEFs represent the average range of Potency Equivalency Factors provided in this document.
NA – not listed in either EPA 2010b nor by MnDOH, but is a NATTS-requested PAH.

APPENDIX F

Compilation of Target Organs for Toxic Air Contaminants

DEQ and OHA determined that the health effect, or health effects, used to derive the TRV should be considered when determining whether to include a toxic air contaminant in an organ-specific hazard index evaluation. Chemicals may cause effects on other organs at concentrations higher than the TRV, but there is greater uncertainty associated with quantifying these effects, and it is not necessary to include them in the analysis. Table F-1 (chronic effects) and Table F-2 (acute effects) show applicable organ systems for toxic air contaminants that will be acceptable to DEQ for organ-specific evaluations.

Table F-1
Chronic Target Organ Systems

CASRN	Chemical	Kidney	Liver	Blood	Endo	Musc	Eyes	Skin	Nerv	Cardio	Immune	Resp	Gastro	Develop
75-07-0	Acetaldehyde											X		
67-64-1	Acetone								X					
75-05-8	Acetonitrile								X			X		
107-02-8	Acrolein											X		
79-06-1	Acrylamide								X					
79-10-7	Acrylic acid											X		
107-13-1	Acrylonitrile											X		
107-05-1	Allyl chloride								X					
7429-90-5	Aluminum and compounds								X					
7664-41-7	Ammonia											X		
62-53-3	Aniline										X			
7440-36-0	Antimony and compounds											X		
7440-38-2	Arsenic and compounds								X					
7784-42-1	Arsine								X					
71-43-2	Benzene			X										
100-44-7	Benzyl chloride											X		
7440-41-7	Beryllium and compounds										X	X		
111-44-4	Bis(2-chloroethyl) ether (BCEE)													
542-88-1	Bis(chloromethyl) ether													
74-83-9	Bromomethane (Methyl bromide)											X		
106-94-5	1-Bromopropane (n-propyl bromide)								X					
106-99-0	1,3-Butadiene													X
78-93-3	2-Butanone (Methyl ethyl ketone)													X
78-92-2	sec-Butyl alcohol													X
7440-43-9	Cadmium and compounds	X												
105-60-2	Caprolactam											X		
75-15-0	Carbon disulfide								X					
56-23-5	Carbon tetrachloride		X											
463-58-1	Carbonyl sulfide								X					
57-74-9	Chlordane		X											
7782-50-5	Chlorine											X		
10049-04-4	Chlorine dioxide											X		
532-27-4	2-Chloroacetophenone											X		
108-90-7	Chlorobenzene	X												

Table F-1
Chronic Target Organ Systems

CASRN	Chemical	Kidney	Liver	Blood	Endo	Musc	Eyes	Skin	Nerv	Cardio	Immune	Resp	Gastro	Develop
75-68-3	1-Chloro-1,1-difluoroethane								X					
75-45-6	Chlorodifluoromethane (Freon 22)	X			X									
75-00-3	Chloroethane (Ethyl chloride)					X								X
67-66-3	Chloroform	X	X											
74-87-3	Chloromethane (Methyl chloride)								X					
76-06-2	Chloropicrin											X		
126-99-8	Chloroprene										X	X		
18540-29-9	Chromium VI, chromate and dichromate particulate											X		
18540-29-9	Chromium VI, chromic acid aerosol mist											X		
7440-48-4	Cobalt and compounds											X		
7440-50-8	Copper and compounds													
1319-77-3	Cresols (mixture), including m-cresol, o-cresol, p-cresol								X					
74-90-8	Cyanide, Hydrogen				X									
110-82-7	Cyclohexane													X
333-41-5	Diazinon													
96-12-8	1,2-Dibromo-3-chloropropane (DBCP)													X
106-46-7	p-Dichlorobenzene (1,4-Dichlorobenzene)											X		
156-60-5	trans-1,2-dichloroethene													
75-09-2	Dichloromethane (Methylene chloride)		X											
78-87-5	1,2-Dichloropropane (Propylene dichloride)											X		
542-75-6	1,3-Dichloropropene											X		
62-73-7	Dichlorovos (DDVP)								X					
	Diesel Particulate Matter											X		
111-42-2	Diethanolamine											X		
112-34-5	Diethylene glycol monobutyl ether		X											
111-90-0	Diethylene glycol monoethyl ether											X		
75-37-6	1,1-Difluoroethane								X					
68-12-2	Dimethyl formamide		X											

Table F-1
Chronic Target Organ Systems

CASRN	Chemical	Kidney	Liver	Blood	Endo	Musc	Eyes	Skin	Nerv	Cardio	Immune	Resp	Gastro	Develop
57-14-7	1,1-Dimethylhydrazine													
123-91-1	1,4-Dioxane											X		
298-04-4	Disulfoton													
106-89-8	Epichlorohydrin											X		
106-88-7	1,2-Epoxybutane											X		
140-88-5	Ethyl acrylate											X		
100-41-4	Ethyl benzene	X												
106-93-4	Ethylene dibromide (EDB, 1,2-Dibromoethane)											X		
107-06-2	Ethylene dichloride (EDC, 1,2-Dichloroethane)								X					
107-21-1	Ethylene glycol											X		
111-76-2	Ethylene glycol monobutyl ether											X		
110-80-5	Ethylene glycol monoethyl ether			X										X
111-15-9	Ethylene glycol monoethyl ether acetate													X
109-86-4	Ethylene glycol monomethyl ether													X
110-49-6	Ethylene glycol monomethyl ether acetate													X
75-21-8	Ethylene oxide								X					
	Fluorides					X								
7782-41-4	Fluorine gas													
50-00-0	Formaldehyde											X		
111-30-8	Glutaraldehyde											X		
77-47-4	Hexachlorocyclopentadiene											X		
67-72-1	Hexachloroethane								X					
822-06-0	Hexamethylene-1,6-diisocyanate											X		
110-54-3	Hexane								X					
302-01-2	Hydrazine		X											
7647-01-0	Hydrochloric acid											X		
7664-39-3	Hydrogen fluoride													
7783-06-4	Hydrogen sulfide											X		
78-59-1	Isophorone													X
67-63-0	Isopropyl alcohol													X
98-82-8	Isopropylbenzene (Cumene)	X			X									

Table F-1
Chronic Target Organ Systems

CASRN	Chemical	Kidney	Liver	Blood	Endo	Musc	Eyes	Skin	Nerv	Cardio	Immune	Resp	Gastro	Develop
7439-92-1	Lead and compounds													X
108-31-6	Maleic anhydride											X		
7439-96-5	Manganese and compounds								X					
7439-97-6	Mercury and compounds								X					
67-56-1	Methanol													X
101-77-9	4,4'-Methylenedianiline (and its dichloride)						X							
108-10-1	Methyl isobutyl ketone (MIBK, Hexone)													X
624-83-9	Methyl isocyanate											X		
80-62-6	Methyl methacrylate											X		
1634-04-4	Methyl tert-butyl ether	X					X							
101-68-8	Methylene diphenyl diisocyanate (MDI)											X		
91-20-3	Naphthalene								X			X		
	Nickel compounds, insoluble										X	X		
	Nickel compounds, soluble										X	X		
7697-37-2	Nitric acid													
98-95-3	Nitrobenzene											X		
79-46-9	2-Nitropropane		X											
8014-95-7	Oleum (fuming sulfuric acid)													
56-38-2	Parathion													
108-95-2	Phenol		X						X					
75-44-5	Phosgene											X		
7803-51-2	Phosphine	X		X					X			X	X	
7664-38-2	Phosphoric acid											X		
12185-10-3	Phosphorus, white											X		
85-44-9	Phthalic anhydride						X					X		
	Polybrominated diphenyl ethers (PBDEs)													
	Polychlorinated biphenyls (PCBs) TEQ		X	X							X	X		
32598-13-3	*PCB 77 [3,3',4,4'-tetrachlorobiphenyl]*		X	X							X	X		
70362-50-4	*PCB 81 [3,4,4',5-tetrachlorobiphenyl]*		X	X							X	X		
32598-14-4	*PCB 105 [2,3,3',4,4'-pentachlorobiphenyl]*		X	X							X	X		

Table F-1
Chronic Target Organ Systems

CASRN	Chemical	Kidney	Liver	Blood	Endo	Musc	Eyes	Skin	Nerv	Cardio	Immune	Resp	Gastro	Develop
74472-37-0	*PCB 114 [2,3,4,4',5-pentachlorobiphenyl]*		X	X							X	X		
31508-00-6	*PCB 118 [2,3',4,4',5-pentachlorobiphenyl]*		X	X							X	X		
65510-44-3	*PCB 123 [2,3',4,4',5'-pentachlorobiphenyl]*		X	X							X	X		
57465-28-8	*PCB 126 [3,3',4,4',5-pentachlorobiphenyl]*		X	X							X	X		
38380-08-4	*PCB 156 [2,3,3',4,4',5-hexachlorobiphenyl]*		X	X							X	X		
69782-90-7	*PCB 157 [2,3,3',4,4',5'-hexachlorobiphenyl]*		X	X							X	X		
52663-72-6	*PCB 167 [2,3',4,4',5,5'-hexachlorobiphenyl]*		X	X							X	X		
32774-16-6	*PCB 169 [3,3',4,4',5,5'-hexachlorobiphenyl]*		X	X							X	X		
39635-31-9	*PCB 189 [2,3,3',4,4',5,5'-heptachlorobiphenyl]*		X	X							X	X		
	Polychlorinated dibenzo-p-dioxins (PCDDs) & dibenzofurans (PCDFs) TEQ		X	X							X	X		
1746-01-6	*2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD)*		X	X							X	X		
40321-76-4	*1,2,3,7,8-Pentachlorodibenzo-p-dioxin (PeCDD)*		X	X							X	X		
39227-28-6	*1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin (HxCDD)*		X	X							X	X		
57653-85-7	*1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin (HxCDD)*		X	X							X	X		
19408-74-3	*1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin (HxCDD)*		X	X							X	X		
35822-46-9	*1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin (HpCDD)*		X	X							X	X		
3268-87-9	*Octachlorodibenzo-p-dioxin (OCDD)*		X	X							X	X		

Table F-1
Chronic Target Organ Systems

CASRN	Chemical	Kidney	Liver	Blood	Endo	Musc	Eyes	Skin	Nerv	Cardio	Immune	Resp	Gastro	Develop
51207-31-9	*2,3,7,8-Tetrachlorodibenzofuran (TcDF)*		X	X							X	X		
57117-41-6	*1,2,3,7,8-Pentachlorodibenzofuran (PeCDF)*		X	X							X	X		
57117-31-4	*2,3,4,7,8-Pentachlorodibenzofuran (PeCDF)*		X	X							X	X		
70648-26-9	*1,2,3,4,7,8-Hexachlorodibenzofuran (HxCDF)*		X	X							X	X		
57117-44-9	*1,2,3,6,7,8-Hexachlorodibenzofuran (HxCDF)*		X	X							X	X		
72918-21-9	*1,2,3,7,8,9-Hexachlorodibenzofuran (HxCDF)*		X	X							X	X		
60851-34-5	*2,3,4,6,7,8-Hexachlorodibenzofuran (HxCDF)*		X	X							X	X		
67562-39-4	*1,2,3,4,6,7,8-Heptachlorodibenzofuran (HpCDF)*		X	X							X	X		
55673-89-7	*1,2,3,4,7,8,9-Heptachlorodibenzofuran (HpCDF)*		X	X							X	X		
39001-02-0	*Octachlorodibenzofuran (OCDF)*		X	X							X	X		
50-32-8	Benzo[a]pyrene													X
123-38-6	Propionaldehyde											X		
115-07-1	Propylene											X		
6423-43-4	Propylene glycol dinitrate			X										
107-98-2	Propylene glycol monomethyl ether		X											
75-56-9	Propylene oxide											X		
	Refractory Ceramic Fibers											X		
7783-07-5	Selenide, hydrogen													
7782-49-2	Selenium and compounds													

Table F-1
Chronic Target Organ Systems

CASRN	Chemical	Kidney	Liver	Blood	Endo	Musc	Eyes	Skin	Nerv	Cardio	Immune	Resp	Gastro	Develop
7631-86-9	Silica, crystalline (respirable)											X		
1310-73-2	Sodium hydroxide													
100-42-5	Styrene								X					
505-60-2	Sulfur Mustard													
7446-71-9	Sulfur trioxide											X		
7664-93-9	Sulfuric acid											X		
127-18-4	Tetrachloroethene (Perchloroethylene)								X					
811-97-2	1,1,1,2-Tetrafluoroethane													X
7550-45-0	Titanium tetrachloride											X		
108-88-3	Toluene								X					
26471-62-5	Toluene diisocyanates (2,4- and 2,6-)											X		
71-55-6	1,1,1-Trichloroethane (Methyl chloroform)		X											
79-01-6	Trichloroethene (TCE, Trichloroethylene)										X			X
96-18-4	1,2,3-Trichloropropane											X		
121-44-8	Triethylamine		X				X					X		
526-73-8	1,2,3-Trimethylbenzene								X					
95-63-6	1,2,4-Trimethylbenzene								X					
108-67-8	1,3,5-Trimethylbenzene								X					
7440-62-2	Vanadium (fume or dust)											X		
1314-62-1	Vanadium pentoxide											X		
108-05-4	Vinyl acetate											X		
593-60-2	Vinyl bromide		X											
75-01-4	Vinyl chloride		X											
75-35-4	Vinylidene chloride		X											
1330-20-7	Xylene (mixture), including m-xylene, o-xylene, p-xylene						X		X			X		

Notes for Table F-1:
CASRN = Chemical Abstracts Service Registry Number
Endo = Endocrine system
Musc = Musculo-skeletal system
Nerv = Nervous system
Cardio = Cardiovascular system
Immune = Immune system
Resp = Respiratory system
Gastro = Gastrointestinal system
Develop = Developmental or reproductive effects
Bold text = Category name
Italic text = Chemical within a categoryResp = Respiratory system

Table F-2
Acute Target Organ Systems

CASRN	Chemical	Kidney	Liver	Blood	Endo	Musc	Eyes	Skin	Nerv	Cardio	Immune	Resp	Gastro	Develop
75-07-0	Acetaldehyde						X					X		
67-64-1	Acetone								X					
75-05-8	Acetonitrile													
107-02-8	Acrolein											X		
79-06-1	Acrylamide													
79-10-7	Acrylic acid											X		
107-13-1	Acrylonitrile								X					
107-05-1	Allyl chloride													
7429-90-5	Aluminum and compounds													
7664-41-7	Ammonia						X					X		
62-53-3	Aniline													
7440-36-0	Antimony and compounds											X		
7440-38-2	Arsenic and compounds													X
7784-42-1	Arsine													X
71-43-2	Benzene										X			
100-44-7	Benzyl chloride						X					X		
7440-41-7	Beryllium and compounds										X	X		
111-44-4	Bis(2-chloroethyl) ether (BCEE)								X			X		
542-88-1	Bis(chloromethyl) ether											X		
74-83-9	Bromomethane (Methyl bromide)								X					
106-94-5	1-Bromopropane (n-propyl bromide)								X					
106-99-0	1,3-Butadiene													X
78-93-3	2-Butanone (Methyl ethyl ketone)													X
78-92-2	sec-Butyl alcohol													
7440-43-9	Cadmium and compounds											X		
105-60-2	Caprolactam						X							
75-15-0	Carbon disulfide								X					X
56-23-5	Carbon tetrachloride													X
463-58-1	Carbonyl sulfide								X					
57-74-9	Chlordane		X											
7782-50-5	Chlorine											X		
10049-04-4	Chlorine dioxide											X		
532-27-4	2-Chloroacetophenone													

Table F-2
Acute Target Organ Systems

CASRN	Chemical	Kidney	Liver	Blood	Endo	Musc	Eyes	Skin	Nerv	Cardio	Immune	Resp	Gastro	Develop
108-90-7	Chlorobenzene													
75-68-3	1-Chloro-1,1-difluoroethane													
75-45-6	Chlorodifluoromethane (Freon 22)													
75-00-3	Chloroethane (Ethyl chloride)													X
67-66-3	Chloroform		X											
74-87-3	Chloromethane (Methyl chloride)								X					
76-06-2	Chloropicrin											X		
126-99-8	Chloroprene													
18540-29-9	Chromium VI, chromate and dichromate particulate											X		
18540-29-9	Chromium VI, chromic acid aerosol mist											X		
7440-48-4	Cobalt and compounds													
7440-50-8	Copper and compounds											X		
1319-77-3	Cresols (mixture), including m-cresol, o-cresol, p-cresol													
74-90-8	Cyanide, Hydrogen								X					
110-82-7	Cyclohexane													
333-41-5	Diazinon								X					
96-12-8	1,2-Dibromo-3-chloropropane (DBCP)													X
106-46-7	p-Dichlorobenzene (1,4-Dichlorobenzene)						X					X		
156-60-5	trans-1,2-dichloroethene		X											
75-09-2	Dichloromethane (Methylene chloride)								X					
78-87-5	1,2-Dichloropropane (Propylene dichloride)											X		
542-75-6	1,3-Dichloropropene											X		
62-73-7	Dichlorovos (DDVP)								X					
	Diesel Particulate Matter													
111-42-2	Diethanolamine													
112-34-5	Diethylene glycol monobutyl ether													
111-90-0	Diethylene glycol monoethyl ether													
75-37-6	1,1-Difluoroethane													

Table F-2
Acute Target Organ Systems

CASRN	Chemical	Kidney	Liver	Blood	Endo	Musc	Eyes	Skin	Nerv	Cardio	Immune	Resp	Gastro	Develop
68-12-2	Dimethyl formamide													
57-14-7	1,1-Dimethylhydrazine		X											
123-91-1	1,4-Dioxane						X					X		
298-04-4	Disulfoton								X					
106-89-8	Epichlorohydrin						X					X		
106-88-7	1,2-Epoxybutane													
140-88-5	Ethyl acrylate													
100-41-4	Ethyl benzene								X					
106-93-4	Ethylene dibromide (EDB, 1,2-Dibromoethane)													
107-06-2	Ethylene dichloride (EDC, 1,2-Dichloroethane)													
107-21-1	Ethylene glycol											X		
111-76-2	Ethylene glycol monobutyl ether			X										
110-80-5	Ethylene glycol monoethyl ether													X
111-15-9	Ethylene glycol monoethyl ether acetate													X
109-86-4	Ethylene glycol monomethyl ether													X
110-49-6	Ethylene glycol monomethyl ether acetate													
75-21-8	Ethylene oxide	X												
	Fluorides											X		
7782-41-4	Fluorine gas						X					X		
50-00-0	Formaldehyde											X		
111-30-8	Glutaraldehyde											X		
77-47-4	Hexachlorocyclopentadiene											X		
67-72-1	Hexachloroethane								X					
822-06-0	Hexamethylene-1,6-diisocyanate											X		
110-54-3	Hexane													
302-01-2	Hydrazine		X											
7647-01-0	Hydrochloric acid											X		
7664-39-3	Hydrogen fluoride											X		
7783-06-4	Hydrogen sulfide											X		
78-59-1	Isophorone													
67-63-0	Isopropyl alcohol						X					X		

Table F-2
Acute Target Organ Systems

CASRN	Chemical	Kidney	Liver	Blood	Endo	Musc	Eyes	Skin	Nerv	Cardio	Immune	Resp	Gastro	Develop
98-82-8	Isopropylbenzene (Cumene)													
7439-92-1	Lead and compounds													X
108-31-6	Maleic anhydride													
7439-96-5	Manganese and compounds								X					
7439-97-6	Mercury and compounds								X					X
67-56-1	Methanol								X					
101-77-9	4,4'-Methylenedianiline (and its dichloride)													
108-10-1	Methyl isobutyl ketone (MIBK, Hexone)													
624-83-9	Methyl isocyanate													
80-62-6	Methyl methacrylate													
1634-04-4	Methyl tert-butyl ether								X					
101-68-8	Methylene diphenyl diisocyanate (MDI)											X		
91-20-3	Naphthalene											X		
	Nickel compounds, insoluble										X			
	Nickel compounds, soluble										X			
7697-37-2	Nitric acid											X		
98-95-3	Nitrobenzene													
79-46-9	2-Nitropropane													
8014-95-7	Oleum (fuming sulfuric acid)											X		
56-38-2	Parathion								X					
108-95-2	Phenol						X					X		
75-44-5	Phosgene											X		
7803-51-2	Phosphine													
7664-38-2	Phosphoric acid													
12185-10-3	Phosphorus, white											X		
85-44-9	Phthalic anhydride													
	Polybrominated diphenyl ethers (PBDEs)				X									
	Polychlorinated biphenyls (PCBs) TEQ													
32598-13-3	*PCB 77 [3,3',4,4'-tetrachlorobiphenyl]*													
70362-50-4	*PCB 81 [3,4,4',5-tetrachlorobiphenyl]*													
32598-14-4	*PCB 105 [2,3,3',4,4'-pentachlorobiphenyl]*													

Table F-2
Acute Target Organ Systems

CASRN	Chemical	Kidney	Liver	Blood	Endo	Musc	Eyes	Skin	Nerv	Cardio	Immune	Resp	Gastro	Develop
74472-37-0	*PCB 114 [2,3,4,4',5-pentachlorobiphenyl]*													
31508-00-6	*PCB 118 [2,3',4,4',5-pentachlorobiphenyl]*													
65510-44-3	*PCB 123 [2,3',4,4',5'-pentachlorobiphenyl]*													
57465-28-8	*PCB 126 [3,3',4,4',5-pentachlorobiphenyl]*													
38380-08-4	*PCB 156 [2,3,3',4,4',5-hexachlorobiphenyl]*													
69782-90-7	*PCB 157 [2,3,3',4,4',5'-hexachlorobiphenyl]*													
52663-72-6	*PCB 167 [2,3',4,4',5,5'-hexachlorobiphenyl]*													
32774-16-6	*PCB 169 [3,3',4,4',5,5'-hexachlorobiphenyl]*													
39635-31-9	*PCB 189 [2,3,3',4,4',5,5'-heptachlorobiphenyl]*													
	Polychlorinated dibenzo-p-dioxins (PCDDs) & dibenzofurans (PCDFs) TEQ													
1746-01-6	*2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD)*													
40321-76-4	*1,2,3,7,8-Pentachlorodibenzo-p-dioxin (PeCDD)*													
39227-28-6	*1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin (HxCDD)*													
57653-85-7	*1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin (HxCDD)*													
19408-74-3	*1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin (HxCDD)*													
35822-46-9	*1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin (HpCDD)*													
3268-87-9	*Octachlorodibenzo-p-dioxin (OCDD)*													

Table F-2
Acute Target Organ Systems

CASRN	Chemical	Kidney	Liver	Blood	Endo	Musc	Eyes	Skin	Nerv	Cardio	Immune	Resp	Gastro	Develop
51207-31-9	*2,3,7,8-Tetrachlorodibenzofuran (TcDF)*													
57117-41-6	*1,2,3,7,8-Pentachlorodibenzofuran (PeCDF)*													
57117-31-4	*2,3,4,7,8-Pentachlorodibenzofuran (PeCDF)*													
70648-26-9	*1,2,3,4,7,8-Hexachlorodibenzofuran (HxCDF)*													
57117-44-9	*1,2,3,6,7,8-Hexachlorodibenzofuran (HxCDF)*													
72918-21-9	*1,2,3,7,8,9-Hexachlorodibenzofuran (HxCDF)*													
60851-34-5	*2,3,4,6,7,8-Hexachlorodibenzofuran (HxCDF)*													
67562-39-4	*1,2,3,4,6,7,8-Heptachlorodibenzofuran (HpCDF)*													
55673-89-7	*1,2,3,4,7,8,9-Heptachlorodibenzofuran (HpCDF)*													
39001-02-0	*Octachlorodibenzofuran (OCDF)*													
50-32-8	Benzo[a]pyrene													
123-38-6	Propionaldehyde													
115-07-1	Propylene													
6423-43-4	Propylene glycol dinitrate								X					
107-98-2	Propylene glycol monomethyl ether													
75-56-9	Propylene oxide											X		
	Refractory Ceramic Fibers													
7783-07-5	Selenide, hydrogen						X					X		
7782-49-2	Selenium and compounds						X	X	X			X		

Table F-2
Acute Target Organ Systems

CASRN	Chemical	Kidney	Liver	Blood	Endo	Musc	Eyes	Skin	Nerv	Cardio	Immune	Resp	Gastro	Develop
7631-86-9	Silica, crystalline (respirable)													
1310-73-2	Sodium hydroxide						X	X				X		
100-42-5	Styrene								X					
505-60-2	Sulfur Mustard						X							
7446-71-9	Sulfur trioxide											X		
7664-93-9	Sulfuric acid											X		
127-18-4	Tetrachloroethene (Perchloroethylene)								X					
811-97-2	1,1,1,2-Tetrafluoroethane													
7550-45-0	Titanium tetrachloride											X		
108-88-3	Toluene								X					
26471-62-5	Toluene diisocyanates (2,4- and 2,6-)											X		
71-55-6	1,1,1-Trichloroethane (Methyl chloroform)								X					
79-01-6	Trichloroethene (TCE, Trichloroethylene)										X			X
96-18-4	1,2,3-Trichloropropane											X		
121-44-8	Triethylamine						X							
526-73-8	1,2,3-Trimethylbenzene													
95-63-6	1,2,4-Trimethylbenzene													
108-67-8	1,3,5-Trimethylbenzene													
7440-62-2	Vanadium (fume or dust)											X		
1314-62-1	Vanadium pentoxide											X		
108-05-4	Vinyl acetate											X		
593-60-2	Vinyl bromide													
75-01-4	Vinyl chloride													X
75-35-4	Vinylidene chloride		X											
1330-20-7	Xylene (mixture), including m-xylene, o-xylene, p-xylene								X			X		

Notes for Table F-2:
CASRN = Chemical Abstracts Service Registry Number
Endo = Endocrine system
Musc = Musculo-skeletal system
Nerv = Nervous system
Cardio = Cardiovascular system
Immune = Immune system
Resp = Respiratory system
Gastro = Gastrointestinal system
Develop = Developmental or reproductive effects
Bold text = Category name
Italic text = Chemical within a category

APPENDIX G

Handling of Non-Detect Values in Risk Assessment

For source tests and ambient monitoring, it is possible that some of the toxic air contaminants will not be detected above the method detection limit. The following is DEQ's preferred approach for handling non-detect values in risk assessments. For calculations involving toxicity equivalency factors, follow DEQ's Source Sampling Manual (Vol. 1, January, 1976, revised November, 2018, https://www.oregon.gov/deq/FilterDocs/SSMI.pdf).

G.1 Source Testing

When considering whether source test data provided by a facility should be accepted for use in a Cleaner Air Oregon risk assessment, DEQ's expectations are the following:

1) Source testing will comply with DEQ's Source Sampling Manual; and
2) The toxic air contaminants to be tested for, test methods, test conditions, and detection limits will be approved by a DEQ source test coordinator prior to conducting the test; and
3) Non-detects will show the actual analytical limit of detection for each source test run; and
4) The source test was performed less than two years prior to submittal of the CAO emissions inventory, unless the facility demonstrates that earlier test data remain valid; and
5) For cyclic operations or variations in feedstock, tests are representative of variations in loads, feed rates, and seasons, if applicable. An adequate number of tests must be conducted for all cyclic or seasonal operations.

DEQ's Source Sampling Manual requires that the detection limit be used for non-detect values in averaging data, except that substitutions at less than the detection limit may be used in CAO risk assessments if approved by DEQ. Provided the above conditions are met, DEQ will accept the following:

If a toxic air contaminant is not detected in any source test runs or samples, you can consider the toxic air contaminant not present, and treat its concentration as zero in that portion of the risk assessment.

If a toxic air contaminant is detected in less than 10% of the test runs or samples, assign a concentration of **zero** to those test runs or samples that were non-detect. Average the non-detect values (zeros) with the detected values, and report the final average value for use in the risk assessment.

If a toxic air contaminant is detected in 10% or more of the test runs or samples, assign a concentration of **one-half the detection limit** to those test runs or samples that were non-detect. Average the detected values with ½ detection limit values for the non-detect samples, and report the final average value for use in the risk assessment.

G.2 Ambient Monitoring

Non-detects in all test samples
If all samples consistently show levels below detection limits, consider the toxic air contaminant not present, and treat its concentration as **zero** in that portion of the risk assessment .

Non-detects in some test samples
If toxic air contaminants are detected in some samples but not others, and there are sufficient samples, use an appropriate statistical method such as Kaplan Meier to calculate an exposure concentration. If there are insufficient samples for a meaningful statistical evaluation, handle the non-detect samples consistent with the procedures for source tests listed above.

For both source testing and ambient monitoring, DEQ may make other site-specific decisions regarding non-detect values if, for example, detection limits are found to be unreasonably high after DEQ approved a monitoring plan with reasonable detection limits.