DRAFT RECOMMENDED PROCEDURES FOR CONDUCTING AIR TOXICS 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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SCREENING EXAMPLES

TABLES (from OAR 340-245)

LIST OF ABBREVIATIONS

ADAF	Age Dependent Adjustment Factor
AERMOD	American Meteorological Society/EPA preferred air dispersion modeling program
AERSCREEN	Program to run AERMOD in screening mode
ANRAL	Alternate Noncancer Risk Action Level
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
COC	Chemical of Concern
COI	Chemical of Interest
COPC	Chemical of Potential Concern
CSM	Conceptual Site Model
DEQ	Oregon Department of Environmental Quality
ED	Exposure Duration
ELAF	Early-Life Adjustment Factor
EPA	U.S. Environmental Protection Agency
EQC	Environmental Quality Commission
HI	Hazard Index
HQ	Hazard Quotient
IRIS	EPA's Integrated Risk Information System
IUR	Inhalation Unit Risk
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 chemical
PCB	Polychlorinated Biphenyl
PPRTV	EPA's Provisional Peer-Reviewed Toxicity Value
RAL	Risk Action Level
RBC	Risk-Based Concentration
RBDM	Risk-Based Decision Making
RfC	Reference Concentration
SCAQMD	California's South Coast Air Quality Management District
TEF	Toxic Equivalency Factor
TRV	Toxicity Reference Value

1. INTRODUCTION

1.1 Purpose and Organization

This document provides DEQ's recommended procedures for conducting air toxics screening and health risk assessments in compliance with OAR chapter 340, division 245. A risk assessment can be a screening risk assessment (Levels 1 and 2), a simple risk assessment (Level 3), or a complex risk assessment (Level 4). Risk screening values are based on both short-term and long-term exposure, and are established in rule.

The methods to perform human health risk assessments at air toxics sites 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, or new facilities.

Screening procedures are presented first. It is possible that your facility may screen out at Level 1 or 2, and you do not need to do more complicated modeling and risk assessment. Section 2 provides an overview of the risk assessment process, including development of a conceptual site model.

Prior to conducting a Level 3 simple risk assessment, you should prepare an air dispersion Modeling Protocol, and meet with DEQ staff to discuss your plans for conducting the assessment. A Level 4 comprehensive health risk assessment will require preparation of a risk assessment workplan and discussions with DEQ before approval is provided to complete the 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 site emissions are key prerequisites for screening steps and risk assessments.

The elements of the different levels of evaluation are the following.

Level 1 – Screening Level Risk Assessment. The first screening step involves choosing dispersion factors from OAR 340-245-8060 Table 6 based on site-specific information (stack height and distances to various receptors). In the absence of site-specific information, you can use a default dispersion factor. To screen your emissions, multiply your chemical-specific emission rates by the dispersion factor, and compare the resulting calculated air concentrations with appropriate Risk-Based Concentrations (RBCs) in OAR 340-245-8050 Table 5. Finally, compare the summed excess cancer risks and hazard indices with the Risk Action Levels (RALs) in OAR 340-245-8010 Table 1. Separate RALs apply to new and existing facilities.

RBCs are developed both for chronic exposure (for cancer and noncancer) and acute exposure. DEQ will conduct a Level 1 evaluation based on emission information provided by your facility during initiation of the program to identify a priority order for DEQ to contact sources that must demonstrate compliance under the rules. After that, facilities contacted by DEQ will conduct their own emission rate screening.

Level 2 – Augmented Screening Level Risk Assessment. If you think it will be helpful, you can use site-specific information (stack height and distances to various exposure locations) and perform simple modeling using EPA's AERSCREEN model to calculate air concentrations for comparison with RBCs. The RBCs are the same as those for Level 1, covering residential and non-residential scenarios, and acute exposure.

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

Level 4 – Comprehensive Health Risk Assessment. The most comprehensive risk assessment uses the same air dispersion modeling conducted in Level 3, with detailed site-specific information and more complex air dispersion modeling. In addition, factors can be considered to refine the exposure assessment. These factors can include exposure assumptions, relative bioavailability of chemicals, or multipathway considerations not covered by the values used to develop RBCs. At this level, you may also propose Alternate Noncancer Risk Assessment Levels, discussed in Section 4.2.4.

2. RISK SCREENING AND ASSESSMENT

2.1 Risk Assessment Concepts

The goal of the Cleaner Air Oregon program is to evaluate potential risks to people near facilities that emit any of the regulated air toxics in OAR 340-245-8050 Table 5, and ultimately reduce unacceptable risks to acceptable levels. Because the concept of risk is fundamental to the program, we begin with a consideration of risk. Basically, risk is a combination of exposure and toxicity:

Exposure • Toxicity → Risk

Exposure is how much contact someone has with a chemical. This mainly includes the concentration of the chemical in air, typically expressed as micrograms of chemical per cubic meter of air ($\mu g/m^3$). The greater the concentration of a chemical in air, the greater the risk. Other considerations for exposure include how long the exposure occurs, which for chronic exposure would include both exposure frequency, such as 8 hours per day for workers, and exposure duration, such as 25 years. Acute effects are evaluated for exposure to a chemical for a day or less.

Toxicity is a measure of how harmful a chemical 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 if exposed to a concentration of 1 microgram per cubic meter, or $\mu g/m^3$. This value is called the Inhalation Unit Risk (IUR) value. For ease of use in assessing risk in the Cleaner Air Oregon program, IURs were converted 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 air toxics 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, DEQ looks at individual probabilities resulting from exposure to air toxics, and not a total population cancer burden.

Chemicals 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 some concentration of a chemical in the air. They do not consider risks from cross-media exposure, such as eating vegetables grown in soil where chemicals settled out of air into the soil and got taken up into the vegetables. To address cross-media risk, an adjustment is made to the toxicity reference value like the ones described in Section 2.2.2 later in this document, and presented in OAR 340-245-8040

Table 4.

DEQ developed RBCs for each air toxic from a selected risk level using standard exposure and toxicity assumptions. Separate RBCs are identified for cancer risk, acute noncancer risk, and chronic noncancer risk. Developing RBCs is essentially the inverse of calculating risk.

For the purpose of 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 chemical in air to the RBC. An HQ below 1 means there is little likelihood that even sensitive people will experience 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 not necessarily intended to be 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 and existing facilities.

2.2 Risk-Based Concentrations

TRVs serve as the basis for RBCs. To establish TRVs for each chemical, 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 preferred sources of chronic and acute TRVs identified in Appendix A. 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 chemicals, which involves exposure other than by inhalation alone. This is a multipathway adjustment. The third adjustment is for early-life exposure to chemicals that exhibit greater toxicity to infants and children.

Adjustment factors are provided in OAR 340-245-8040 Table 4. Appendix B shows how the adjustment factors were used to develop the RBCs shown in OAR 340-245-8050 Table 5.

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 appropriate for residential exposure. 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. Modifying factors for chronic exposure are discussed in Appendix B. There are no modifying factors for acute exposure.

2.2.2 Multipathway Considerations

DEQ recommends use of nearby land use information, along with that on existing site conditions, to help identify potentially exposed human receptors, including any sensitive groups. For example,

pregnant women and infants are sensitive groups for mercury exposure. Information on potentially exposed populations allows for the identification of site-specific exposure scenarios and exposure routes. This is especially important if your facility is emitting persistent, bioaccumulating, and toxic (PBT) chemicals. For bioaccumulating chemicals, DEQ considered multipathway effects on residents in developing RBCs. DEQ developed multipathway adjustment factors (MPAFs) for residential exposure scenarios that consider:

- Inhalation of chemicals in air
- Deposition of airborne chemicals to backyard soil
- Contact with soil by incidental ingestion and dermal exposure
- Uptake into garden vegetables, and ingestion of vegetables
- 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 chemicals to:

- Agricultural land
- Livestock grazing areas
- Drinking water reservoirs
- Lakes or ponds used for fishing

If PBT chemical emissions from your facility could impact the above areas, DEQ may require a more complex Level 4 multipathway risk assessment even if your emissions pass the Level 1 to Level 3 screens using default MPAFs. DEQ recommends that this process start with development of a more extensive conceptual site model.

2.2.3 Early-Life Exposure

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 modify the cancer TRV. Currently, the chemicals of primary interest for consideration of early-life exposure are listed in OAR 340-245-8020 Table 2 with early-life adjustment factors (ELAFs). As more information becomes available, EPA may determine that additional carcinogens act by a mutagenic mode of action. For this reason, DEQ may in the future recommend that the Environmental Quality Commission (EQC) expand the list of chemicals for which ELAF values are needed.

2.3 Risk Assessment Elements

A number of elements are important to conducting a risk assessment. The first risk assessment elements are important even at a simple screening evaluation. 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 chemical sources, exposure pathways, routes of exposure, and types of exposure areas near your site. To evaluate the types of exposure areas, DEQ recommends that you conduct a land use determination, and, if appropriate, a water use determination if significant airborne deposition to water is expected. Once you establish a conceptual site model, you can proceed with the main exposure and toxicity assessments, and risk characterizations. Finally, you may want to include an uncertainty section to document qualitative or quantitative evaluations of variability and uncertainty.

The risk assessment can be conducted with the RBC approach presented here, or follow the traditional method (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 ending with a conceptual site model (CSM) describing air toxic releases and relevant exposure scenarios based on current and reasonably likely future exposure populations;
- an exposure analysis, which includes calculating exposure point concentrations based on the CSM, selecting exposure model equations, and selecting exposure factor values;
- a toxicity analysis evaluating the inherent toxicity of chemicals;
- 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 clear conceptual understanding of the various chemical sources, exposure pathways, routes of exposure, and types of receptors at your site. A good way of presenting a conceptual site model (CSM) is in a chart, although for most air emission evaluations, a brief narrative is sufficient. If you have bioaccumulating chemicals, and therefore may be required to conduct a multipathway risk assessment, we recommend a more extensive CSM to adequately describe the site. An example multipathway conceptual site model is provided as Figure 2 in DEQ's risk assessment guidance for the Cleanup Program (DEQ 2010).

A high-quality CSM should combine information on contaminants, receptors, and exposure pathways to summarize relevant site information and set the stage for the risk assessment. It is important that you consider both current and potential future exposure when identifying exposure routes at your site. Future exposure for residential exposure includes consideration of land which is zoned, or documented as planned to be zoned, for uses allowing residents. Include reasonably likely exposure areas. For example, for farmland where a residence is allowed, include exposure to any current houses. However, it is not necessary to consider an unreasonable future addition of a house in an agricultural field. For areas zoned only for residential use, evaluate residential exposure to the entire area.

Land and water use determinations are important starting points for identifying potentially exposed populations for a risk assessment. For complex facilities, especially those with bioaccumulating chemicals, it may be useful to follow DEQ's guidance on land and water use determinations (DEQ 1998a, DEQ 1998b). A specific combination of receptors, 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 are changed without a reassessment of risk appropriate for that site, the risk assessment may no longer be protective.

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. An exposure pathway is the course a chemical or physical agent 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 chemical or physical agent comes in contact with a person. Inhalation is the primary route of exposure for air emissions, although other routes (ingestion, dermal contact) may be important for bioaccumulating chemicals.

RBCs were developed for the following scenarios:

- Residential exposure, which includes long-term exposure to children and adults, in areas where residential houses already exist, or are zoned, or documented as planned to be zoned, for uses that include residential.
- Nonresident adult exposure, which includes adults in commercial or industrial facilities.
- Nonresident child exposure, which includes schools and daycare facilities
- Acute exposure, which includes areas where someone may spend all or a portion of a day, such as parks, sports fields, or agricultural fields.

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

For bioaccumulating chemicals, additional scenarios such as agricultural or recreational use may be relevant. Details about these exposure scenarios are not provided in this document, and should be discussed with DEQ prior to conducting a risk assessment.

2.4.1 Air Dispersion Modeling

The primary element of an exposure assessment for air toxics is dispersion modeling. Air quality dispersion modeling underlies the analyses in all levels of the risk assessment, from Level 1 through Level 4. A dispersion model is a mathematical way to emulate the physical, and sometimes chemical, processes in the atmosphere that produce an ambient air concentration of a compound based on emissions data. More simply put, a model calculates a concentration of a pollutant as a result of an emission of that pollutant, and physical dispersion in air. Modeled concentrations are estimated at specific locations called modeling receptors. These modeling receptors can be considered virtual monitors, and the dispersion model calculates modeled concentrations at these locations to simulate concentrations as if measured by real monitors at those locations. Modeling receptors can also be positioned where sensitive receptors, such as houses or schools, are located.

OAR 340-245-8060 Table 6 in the Level 1 analysis was developed using dispersion modeling, although running a dispersion model directly is not required for its use. Risk assessments at Level 2 through Level 4 require the direct use of dispersion modeling, ranging from the relatively simple EPA AERSCREEN at Level 2, to the refined analysis at Level 3 or 4 using EPA’s AERMOD.

AERSCREEN is a screening version of AERMOD, and with the exception of actual meteorology and topographic data required for AERMOD, the data necessary to run either model is the same. These include the following parameters:

- Emission rate (may vary by period, such as by month or season)

- Stack Height
- Stack Diameter
- Stack Velocity or Flow Rate
- Stack Temperature
- Building Heights
- Building Horizontal Dimensions
- Land surface characteristics for estimating albedo, Bowen ratio, and surface roughness

AERMOD is an EPA approved dispersion model for regulatory modeling in the New Source Review (NSR) program, and is the primary refined dispersion model for the CAO program. In addition to the parameters shared with AERSCREEN, AERMOD is a refined model that requires actual, representative meteorology, terrain information, and a gridded field of modeling receptors where concentrations will be evaluated. This meteorology can be obtained from the following sources:

- Onsite collection
- Representative National Weather Service (NWS) data
- DEQ weather data
- Weather Research and Forecasting model (WRF) prognostic data produced by the University of Washington

All meteorological data, and options for its use, that may be proposed for running in AERMOD, must be approved by DEQ.

AERMOD can also be run in screening mode using screening meteorology generated by MAKEMET, the meteorological pre-processor in AERSCREEN. DEQ considers the results as equivalent to running AERSCREEN in a Level 2 assessment. As in AERSCREEN, the MAKEMET screening meteorology is a worst-case set of conditions that, together with stack and building data, is designed to produce the highest or most conservative concentrations. The advantages of using MAKEMET in AERMOD are the ability to incorporate terrain information and to model multiple stacks from the facility being studied.

Details for using EPA's AERSCREEN or AERMOD, and their pre and post-processors, can be found in their respective user's manuals (EPA 2016a, EPA 2016b). In addition, dispersion modeling used in the CAO program should follow the EPA's *Guideline on Air Quality Models* (EPA 2017a), commonly called Appendix W. Prior to conducting air dispersion modeling, develop and prepare a Modeling Protocol for DEQ approval. Although the Protocol is site-specific to each source, there are common elements that may include, but are not limited to, the following:

- Annual and short term emission rates, depending on the toxic chemicals emitted by the facility.
- Stack parameters for all operating conditions.
- Building dimensions for evaluating building downwash.
- Dispersion and associated models: including but not limited to AERMOD, AERMET, and their preprocessor programs AERMAP, AERSURFACE, BPIP. You may propose other equivalent models for approval by DEQ.
- Modeling parameters such as terrain, dispersion options, surface characteristics including albedo, Bowen ratio, and surface roughness.
- Extent and refinement of the modeling receptor grid.
- Identification of modeling receptors at locations of exposure populations, such as residential, commercial/industrial, schools/daycare facilities, and sensitive populations.

2.4.2 Use of Air Monitoring Data in Risk Assessments

You may request to conduct ambient air monitoring to supplement air modeling. However, there are a number of complexities to using air monitoring data in a risk assessment. Ambient monitoring results can be complicated by the presence of multiple sources of the air contaminants. This may require the 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 consideration of the above complexities, if DEQ approves monitoring results, and a facility shows that they are in compliance with applicable RALs, then they can request to withdraw the Risk Reduction Plan on the basis that it is not necessary. If the monitoring results show that emissions exceed a RAL, then they would need to follow a compliance schedule based on the Risk Reduction Plan, putting their schedule on the same basis as a facility that did not perform air monitoring.

Cleaner Air Oregon rules allow you to request additional time to complete a Risk Reduction Plan. However, there is also a provision that DEQ may not consider time lost in performing ambient air monitoring as a reason for granting a time extension.

2.5 Toxicity Assessment

The purpose of the toxicity assessment is to compile toxicity data for the air toxics chemicals a facility emits or may emit, and to estimate the relationship between the amount of exposure of an air toxic and the likelihood of adverse effects. You should evaluate the potential cumulative cancer risks and noncancer risks from all chemicals your facility emits. You should also provide qualitative descriptions of the potential toxic properties of the air toxics. These details are most appropriate in a Level 4 risk assessment, unless you want to evaluate noncancer effects by 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 ug/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 Reference Concentration as a general term. CAO rules require that RfCs are developed using the hierarchy in Appendix A, and that risk assessments performed for the program use the RfCs provided in OAR 340-245-8030 Table 3 as TRVs.

An RfC is considered a threshold below which adverse effects are not likely even in sensitive groups. Often, RfCs 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 chemicals is known as a hazard index. In some cases, this will be an overestimate of risk if the chemicals 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 chemicals with effects on the same organ system. Note, however, that many chemicals cause effects on more than one organ. All health effects, regardless of whether the effects were used to derive the RfC, should be considered when determining whether to include a chemical in an organ-specific hazard index evaluation.

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 chemical 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 at 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 (ug/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. For ingestion exposure, EPA provides oral cancer slope factors (SFs), which are expressed as risk per milligram per kilogram body weight per day (mg/kg/day)$^{-1}$.

2.5.3 Assessment of Chemical Mixtures

For some chemical classes, it is preferable to evaluate risk as a single value for the entire class. DEQ's recommendations on how to conduct evaluations for two important chemical classes are provided below.

Toxicity of CDDs/CDFs and Dioxin-like PCBs

Consistent with EPA, DEQ recommends use of toxicity equivalency factors (TEFs) to evaluate toxic effects of polychlorinated dibenzo-*p*-dioxins (CDDs), polychlorinated dibenzofurans (CDFs), and co-planar (dioxin-like) polychlorinated 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 ATSDR 1995. 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.

2.5.4 Screening Terminology

Screening of air toxics is part of the overall risk evaluation approach. Air toxics emitted at the site which

have not been screened should be designated as “Chemicals of Interest” (COIs). If RALs are exceeded at screening Level 1 or 2, air toxics contributing to risk should be designated as “Chemicals of Potential Concern” (COPCs). At the more advanced evaluations, Level 3 or 4, if RALs are exceeded, air toxics contributing to risk should be designated as “Chemicals of Concern” (COCs).

2.6 Uncertainty Evaluation

A section on uncertainty should be included in a risk assessment, especially a Level 4 comprehensive health 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). This evaluation will allow managers to consider the uncertainty associated with the risk assessment.

3. LEVEL 1 AND 2 SCREENING

To conduct screening, you need to calculate concentrations of air toxics at the exposure areas identified in the conceptual site model. You may demonstrate compliance under the CAO rules without conducting dispersion modeling for emissions from your facility by using the Level 1 approach with a lookup table of dispersion factors. If you are unable to demonstrate compliance using a Level 1 approach, then you may attempt to demonstrate compliance using the Level 2 simple model to calculate air toxics concentrations.

3.1 Level 1 Screening Approach

The Level 1 approach allows you to use air toxics emission rates from your facility to calculate air concentrations. Initially, DEQ will conduct a Level 1 evaluation based on emission information provided by your facility during initiation of the program. After that, facilities will conduct their own emission rate screening.

To accomplish Level 1 screening, DEQ developed dispersion factors to do the conversion from chemical emission rates to air concentrations. These factors are shown in OAR 340-245-8060 Tables 6A (annual exposure) and 6B (24-hour exposure). Because DEQ has done these calculations, it is not necessary for you to directly run an air dispersion model. The table is designed for point source emissions only, that is, from discrete stacks that have quantifiable dimensions. If you have fugitive emissions, such as those from building doors and windows, or from areas where paint, solvent, other emissions are generated, you cannot use the table, and must instead, at a minimum, conduct Level 2 modeling using AERSCREEN or a comparable methodology.

The use of OAR 340-245-8060 Table 6 requires only the stack height and distance to the nearest exposure area. You will likely have four types of exposure areas to be evaluated (residential, commercial/industrial, school/daycare, and acute), as discussed in Section 2.4. Determine the closest distance to all the relevant exposure areas. Find the dispersion factor in the table for a given stack height and distance. For each exposure area, multiply the emission rate from a facility stack by the dispersion factor.

DEQ will base Level 1 ranking on reported actual emission rates for facilities. For the emission rates you should use in a Level 1 assessment, select from among the following: 1) if you want to be considered a *de minimis* source, you need to assess air toxics emissions at the capacity to emit; 2) if you want to demonstrate that risk at your pre-existing Potential to Emit (PTE) does not exceed the applicable RALs, evaluate emissions at the pre-existing PTE; or 3) if you want to request a PTE or risk limit, assess your air toxics emissions using the requested limit, which may be based on actual emissions if you can continue to comply at that level. Stack emission rates must be in the same units as the table, such as lbs/day for acute effects toxics, and lbs/year for chronic effects. The result of the calculation will be air concentrations in micrograms per cubic meter, $\mu g/m^3$.

Next, divide the calculated air concentration by the respective RBC for that toxic from OAR 340-245-0830 Table 3. This will determine the potential risk for that single toxic from a single stack. To complete the screening step, add the results for all the applicable air toxic chemicals for the different types of RBCs (chronic cancer, chronic noncancer, and acute noncancer). Finally, compare these calculated total risk values with the RALs in OAR 340-245-8010 Table 1. Example 1 shows a simple Level 1 screening evaluation.

DEQ's Development of Dispersion Factors in OAR 340-245-8060 Table 6

To generate the dispersion factors provided in OAR 340-245-8060 Table 6, DEQ first developed a series of reasonable maximum assumptions associated with stack height, such as stack diameter and building dimensions. We used meteorology data from six cities representing different regions of the state (Portland, Salem, Eugene, Medford, Redmond, and Hermiston). We then used AERMOD to model air concentrations at distances from 50 meters to 1,000 meters from the stack. Hourly concentrations were converted to annual and 24-hour concentrations using standard EPA conversion factors in which calculated 1-hour concentrations were multiplied by 0.1 to estimate an annual concentration, and by 0.6 to estimate a 24-hour concentration (EPA 2016). The results for the six meteorological areas were averaged to develop dispersion factors in units of ug/m^3 per lbs/year for chronic exposure, or ug/m^3 per lbs/day for acute exposure. The results are shown in OAR 340-245-8060 Tables 6A (annual exposure) and 6B (24-hour exposure).

Level 1 is meant to be conservatively protective, such that facilities with values below RALs are not expected to represent a health concern. An exceedance of a RAL does not necessarily constitute a health risk. If you are unable to demonstrate compliance using a Level 1 approach, then you can attempt to demonstrate compliance using the Level 2 simple model to more accurately characterize air concentrations and risk.

You can treat multiple stacks at your facility in one of two ways; 1) add the air toxics concentrations in μg/m^3 from all of the individual stacks to estimate an aggregate concentration that is then compared to the RBC for that toxic; or 2) group the stacks and their emissions into a single stack, and use the information in OAR 340-245-8060 Table 6 to determine a dispersion factor to apply to the grouped emissions in order to estimate an air concentration. DEQ can assist with information about methods to group stacks.

For a stack height between values shown in OAR 340-245-8040 Table 4, either use the next lowest stack height, or interpolate the dispersion factor. Similarly, for an exposure location between values shown in the table, either use the next lower distance, or interpolate the dispersion factor.

Most likely you can readily 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 ug/m^3 / lbs/yr) and daily dispersion factor (8.3 ug/m^3 / lbs/day) for a stack height of 5 meters and an exposure location distance of 50 meters.

3.2 Level 2 Screening Approach

A Level 2 evaluation is similar to a Level 1 evaluation, except that you may add more precision by performing your own 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 allow for elevated terrain and multiple stacks. Both use worst-case meteorology, and provide the same results assuming flat terrain and a single stack. If you plan to conduct modeling, we recommend that you develop an air dispersion Modeling Protocol, and obtain DEQ approval prior to use of the models, as that will increase the likelihood the risk analysis will be approved.

For the emission rates you should use in a Level 2 assessment, select from among the following: 1) if you want to be considered a *de minimis* source, you need to assess air toxics emissions at the capacity to emit; 2) if you want to demonstrate that risk at your pre-existing Potential to Emit (PTE) does not exceed the applicable RALs, evaluate emissions at the pre-existing PTE; or 3) if you want to request a PTE or risk limit, assess your air toxics emissions using the requested limit, which may be based on actual emissions if you can continue to comply at that level.

The model results from AERSCREEN are given by distance from the stack. The results from AERMOD-MAKEMET are estimated at modeling receptors. For AERSCREEN, the concentration at the distance to the nearest sensitive human receptor, such as a residence, is the one of interest. In AERMOD-MAKEMET, you can explicitly include sensitive human receptors as modeling receptors, and the concentration at that modeling receptor, such as a residence, is used in the risk analysis.

Because of the nature of the worst-case screening meteorology, these Level 2 screening models only estimate 1-hr concentrations that must be converted to daily (24-hr) and annual concentrations using EPA conversion factors, in which the 1-hour concentration is multiplied by 0.1 to estimate an annual concentration, and by 0.6 to estimate a 24-hour concentration (EPA 2016).

Once you have calculated air concentrations at the various exposure locations, you can proceed with the screening as discussed above for Level 1. Divide the calculated air concentration by the respective RBC in OAR 340-245-8050 Table 5. To complete the screening step, add the results for all the applicable air toxic chemicals for the different types of RBCs (chronic cancer, chronic noncancer, and acute noncancer). Finally, compare these calculated total risk values with the RALs in OAR 340-245-8010 Table 1. Example 2 shows an example Level 2 screening evaluation.

As with Level 1, exceeding RALs does not necessarily mean that there are adverse health effects due to air emissions from your facility. If you are unable to demonstrate compliance using a Level 2 approach, then you can attempt to demonstrate compliance using the Level 3 approach with a more complex model to more accurately characterize air toxics concentrations.

4. LEVEL 3 ASSESSMENT AND LEVEL 4 COMPREHENSIVE HEALTH RISK ASSESSMENT

The screening steps of Levels 1 and 2 make conservative assumptions that are likely to overestimate risk at many facilities. If results of these screening steps indicate that your emissions are above RALs, you may conduct a more detailed site-specific air dispersion modeling and risk assessment to show compliance with CAO rules. This will allow you to better quantify the potential risks posed by chemicals at your site, and determine if these risks are indeed above RALs after considering a more realistic evaluation of potential exposure.

There are two general approaches for a baseline risk assessment. The traditional approach follows EPA guidance for conducting an exposure assessment, toxicity assessment, and risk characterization (EPA 1989). An alternative to the traditional approach is the Risk-Based Decision Making (RBDM) approach (DEQ 2003). This approach, which involves making a comparison of air concentrations with RBCs, has many benefits in terms of time and effort to prepare a risk assessment. For these reasons, it is the approach used in Level 1 and 2 screening. It is also the approach DEQ recommends for Level 3 and 4. However, you may prefer to conduct some risk assessments, such as those involving pathways not evaluated by air RBCs, using the standard risk assessment approach. Guidance for the standard risk assessment approach is presented in risk assessment guidance for DEQ's Cleanup Program (DEQ 2010).

The key element of a Level 3 or Level 4 evaluation is the use of more sophisticated air dispersion modeling. This is addressed in Section 4.1. If more detailed evaluation is needed beyond air dispersion modeling, a Level 4 risk assessment is required. If you plan to propose Alternate Noncancer Risk Action Levels (ANRALs), you will need a Level 4 evaluation, discussed in Section 4.2. ANRALs are discussed in detail in Section 4.2.4.2. DEQ will require you to prepare protocols for Level 3 and 4 evaluations.

4.1 Level 3 Assessment

The key feature of a Level 3 assessment is extensive 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 you to submit an air dispersion Modeling Protocol prior to conducting the modeling. You should plan on having at least one meeting with DEQ to agree on scope and make sure there are common understandings regarding the modeling. DEQ's approval of the Protocol will make it more likely that the results of the air dispersion modeling will be accepted by DEQ. Section 2.4.1 provides references to EPA modeling guidance that will be helpful in preparing the Modeling Protocol.

For the emission rates you should use in a Level 3 assessment, select from among the following: 1) if you want to be considered a *de minimis* source, you need to assess air toxics emissions at the capacity to emit; 2) if you want to demonstrate that risk at your pre-existing Potential to Emit (PTE) does not exceed the applicable RALs, evaluate emissions at the pre-existing PTE; or 3) if you want to request a

PTE or risk limit, assess your air toxics emissions using the requested limit, which may be based on actual emissions if you can continue to comply at that level.

4.2 Level 4 Comprehensive Risk Assessment

A Level 4 risk assessment should have the same elements as the prior levels, with some additional elements. The required workplan should include the following:

- Identifying information, including the owner or operator of the source, the owner's or operator's mailing address, the source address, the nature of business, name and phone number of the primary contact at the source, permit number, and SIC or NAICS code of the source;
- A problem formulation step ending with a conceptual site model identifying emission sources and existing and reasonably likely future human populations that may be exposed to air toxics emissions from the source, including residents, nonresident adults, and nonresident children and other sensitive populations;
- An exposure assessment that models or measures air toxics concentrations at locations of existing and reasonably likely future human populations that may be exposed to air toxics 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 chemicals, and multipathway considerations for persistent, bioaccumulative and toxic chemicals;
- A toxicity assessment evaluating the carcinogenicity, noncarcinogenic chronic effects, and noncarcinogenic acute effects of air toxics to which human populations will be exposed, including quantifying noncarcinogenic effects separately for different organ systems, and determining persistence and bioaccumulation potential;
- A risk characterization presenting a quantitative evaluation of potential cumulative health risks associated with human 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 chemicals, additional multipathway considerations not addressed by the default adjustment factors, and toxicity considerations relevant to proposing ANRALs. These elements are discussed below.

4.2.1 Exposure Assumption Modifications

The default exposure assumptions for exposure times, frequencies, and durations used in the development of RBCs for residents and workers are standard. 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, document the circumstances, and propose modified exposure parameter values for use in the risk assessment. Note, however, that DEQ will base decisions on reasonably likely future exposure as well as current exposure.

4.2.2 Relative Bioavailability

The toxicity of a chemical can depend on how much of the chemical is actually absorbed, not just on

the measured concentration in air (or soil or water). If the form of a chemical 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. 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 a time-consuming and expensive, so relative bioavailability tests are not commonly performed. If you decide to pursue testing, DEQ will request a detailed workplan for approval prior to conducting the evaluation.

The particular form of a chemical is the main reason for differences in bioavailability. Often this is taken into account in establishing a TRV. For example, DEQ has separate RBCs for total chromium (based on the toxicity of trivalent chromium +3), and hexavalent chromium (+6). The hexavalent form of chromium is substantially more toxic than the other forms. If you can characterize the chemical form of your emissions, you can use the appropriate RBC at any risk evaluation level. This may make it unnecessary to proceed to a Level 4 evaluation.

4.2.3 Multipathway Analysis

If you have bioaccumulating chemicals, it is important to evaluate air deposition and evaluate additional exposure scenarios that could include contact with soil and water. 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 air toxics emissions, California's OEHHA's Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments (2015).

4.2.4 Alternate Noncancer Risk Action Level

In some cases, DEQ will allow Risk Action Levels to be modified for noncarcinogens based on consideration of the severity and permanence of health effects, and uncertainty regarding the toxicity factors. You may propose Alternate Noncancer Risk Action Levels (ANRALs) to DEQ after considering the issues presented in this section.

Some RALs listed in OAR 340-245-8010 Table 1 are presented as a number followed by "or ANRAL up to 10" or "ANRAL+3" or "ANRAL+5." In each case, the number presented serves as the default RAL. DEQ may accept a higher ANRAL up to the number listed in parenthesis on a case-by-case basis for a specific set of air toxics at a specific facility. This approach reflects the fact that the TRVs for different chemicals vary significantly in the severity of health effects they are designed to prevent, and the magnitude of the uncertainty around the value.

You are required to complete a Level 4 risk assessment before you can request an ANRAL. If granted, this ANRAL would be a target organ-specific HI greater than 1 but less than or equal to 10 (depending on criteria listed below). If your facility's risk is higher than the ANRAL, DEQ would consider whether it was also higher than the facility-specific ANRAL+3, which would mean that it was over the RAL for an accelerated schedule as well. If the facility's risk was higher than its granted ANRAL+5 and the facility were requesting to stay at that level of risk with a Conditional Risk Level, then the DEQ Director's approval would be required to grant that conditional risk level. Examples of applying ANRALs are provided in this section.

Risk Action Levels

RALs are levels of risk that inform the actions a regulated facility would be required by DEQ to undertake under Cleaner Air Oregon rules. The higher the exceedance of a RAL at a facility, the more corrective actions the facility would be required to take. Timelines for a facility to come into compliance would also be shorter in these cases.

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

In setting RALs, 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.

Cleaner Air Oregon considers risk in three categories: chronic cancer, chronic noncancer, and acute noncancer. 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.

Examples of Applying Alternate Noncancer Risk Action Levels

ANRAL Example 1:

- Actual chronic non-cancer risk includes a respiratory-specific hazard index of 9 based on Level 4 risk assessment.
- The facility requests and is granted a respiratory effect-specific ANRAL of 3.
- Because the facility's actual risk is less than 10, the facility could apply for a Conditional Risk Level to continue at that level of risk without DEQ Director approval.
- If the facility submits a risk reduction plan to get down to or below their ANRAL of 3, then no Conditional Risk Level would be needed.
- However, the risk reduction plan would have to get them to 6 or less on an accelerated schedule because their risk (9) is greater than the accelerated schedule RAL of ANRAL+3 (which in this case is 3+3 = 6).

ANRAL Example 2:

- Actual chronic non-cancer risk includes a respiratory-specific hazard index of 16 based on Level 4 risk assessment.
- The facility requests and is granted a respiratory effect-specific ANRAL of 10.
- Because the facility's actual risk is greater than the ANRAL+5 (10+5 = 15), the facility could only apply for a Conditional Risk Level to continue at that level of risk with DEQ Director approval.
- If the facility submits a risk reduction plan to get down to or below their ANRAL of 10, then no Conditional Risk Level or director consultation would be needed, but the risk reduction plan would have to get them to 13 or less on an accelerated schedule because their risk (16) is greater than the accelerated schedule RAL of ANRAL+3 (10+3 = 13).

OAR 340-245-0100 outlines the set of criteria DEQ may consider in determining whether or not an ANRAL above the default value may be acceptable. ANRALs will not be considered for chemicals that alter development or cause irreversible health effects. Four criteria define hard limits on the extent to which DEQ has discretion to accept a higher RAL in a specific case:

- **Limits to agency discretion.** There are upper limits on the HIs DEQ can accept as ANRALs for existing sources. This is intended to put a reasonable upper bound around the extent to which DEQ can exercise discretion.
- **Uncertainty around the level of exposure that will cause harm**. To account for uncertainty in the health effects data, TRVs incorporate uncertainty factors that provide a buffer between concentrations where health effects are expected to occur and the level identified as health protective. They are applied to account for uncertainty around differences between individuals or, for values derived from animal studies, differences between species. The magnitude of these uncertainty factors can vary substantially from chemical to chemical. In general, the smaller the uncertainty factor is, the closer the toxicity reference value is to the actual concentration that caused a measurable effect in a study. To prevent a scenario where any community is exposed to concentrations that have been demonstrated to cause health effects, the rule does not allow DEQ to accept an ANRAL that would erode more than 10% of this uncertainty.
- **Severity of health effects.** The severity and permanence of the health effect underlying the toxicity reference value are also taken into account. Some TRVs are based on relatively minor reversible effects, while others are based on severe, irreversible effects. Developmentally toxic chemicals in particular deserve special consideration due to the potential for lasting impacts from short-term exposures during critical windows of development. In recognition of the potential severity of this type of health endpoint, DEQ does not have authority to consider allowing an ANRAL for air toxics with toxicity reference values based on developmental toxicity or otherwise irreversible health outcomes.
- **Use of toxicity reference values.** The TRVs used in Cleaner Air Oregon were developed by authoritative bodies, such as EPA and ATSDR, through an extensive peer review processes. DEQ does not have the resources to perform this level of analysis for each of the chemicals covered by the program and must rely on the values already developed by other authoritative bodies. DEQ, in consultation with OHA, will evaluate whether an ANRAL may be appropriate on a case-by-case basis. The agencies will not consider TRVs other than those presented in OAR 340-245-8030 Table 3.

When the definitive requirements of all four of these criteria are met, DEQ may consider allowing an ANRAL for a specific facility. DEQ identified additional qualitative criteria to inform case-by-case decisions:

- **Whether toxicity reference values are being applied in a manner consistent with the way they were designed.** TRVs were derived from several different authoritative sources that generate values based on different assumptions about routes of exposure and duration of exposure. Some of these assumptions are better suited to the purposes of the CAO program than others. For example, in some cases, toxicity values based on intermediate exposure durations (occurring over periods of weeks or months) were used as TRVs for acute exposure (over 24 hours) because no acute values were available.
- **Potential for exposure through other pathways.** In some cases, emissions occur through water as well as through air. If a community may be exposed to the chemicals impacting the same health endpoints through water, soil, or food, greater caution is warranted.
- **Sensitivity of potentially exposed populations.** Some groups are more susceptible to the effects of air toxics due to biological and social factors. Vulnerable populations may include low income communities, communities of color, pregnant women and developing fetuses, children, the elderly, and people with pre-existing health conditions. Greater caution is warranted when sensitive populations may be exposed.

- **Whether potential health effects are associated with acute or chronic exposure.** Noncancer HIs are calculated for both acute endpoints occurring following short-term exposure over the course of a day, and chronic health endpoints occurring after months or years of continuous exposure. DEQ may be particularly cautious when calculated concentrations would exceed RALs for acute health effects.

4.2.4.1 Setting Limits on DEQ Director Discretion Risk Action Levels for Existing Facilities

Director Consultation Risk Action Levels are intended to provide an opportunity for existing facilities with particularly high risk to get on a path towards compliance when there is support from the community. DEQ rules define an upper limit for cancer and noncancer risks above which the Director does not have discretion to allow a permit. These upper limits were designed to balance the need for some discretion in response to community-specific needs, while still ensuring that health risks are minimized.

The permit denial level for excess cancer risk for existing facilities is 500 in 1 million. Based on conversations with regulators in other states with risk-based industrial air programs, DEQ anticipates that most, if not all facilities in Oregon will be able to reduce risk below this level.

The permit denial level for noncancer risk from existing facilities is a target organ-specific HI of 30. It is difficult to define a single Hazard Index that is unacceptable for all chemicals due to the substantial variation in the severity of health effects and uncertainty contained in toxicity reference values for each chemical. The hard cap for noncancer risk is designed to allow DEQ's Director discretion to reach an HI as high as 30 in cases where it may be appropriate, but there are some chemicals for which reaching this level would fail to protect public health. For some chemicals with severe, acute or irreversible effects, it may be inappropriate to reach this permit denial level for noncancer health endpoints. DEQ's Director must consider chemical-specific concerns with exceeding noncancer RALs, and consult with DEQ and OHA toxicologists, prior to allowing any permits above default RALs.

5. REFERENCES

ATSDR 1995. **Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHs) (Update)**. Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services, Atlanta, Georgia, August 1995.

CalEPA 2015. **Air Toxics Hot Spots Program, Risk Assessment Guidelines, Guidance Manual for Preparation of Health Risk Assessments**. Air, Community, and Environmental Research Branch, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, February 2015.

DEQ 1998a. **Guidance for Consideration of Land Use**. Oregon Department of Environmental Quality, July 1, 1998.

DEQ 1998b. **Guidance for Conducting Beneficial Water Use Determinations at Environmental Cleanup Sites**. Oregon Department of Environmental Quality, July 1, 1998.

DEQ 2003. **Risk-Based Decision Making for the Remediation of Contaminated Sites**. Oregon Department of Environmental Quality, September 22, 2003, updated title October 2, 2017.

DEQ 2010. **Human Health Risk Assessment Guidance**. Oregon Department of Environmental Quality, October 2010.

DEQ/OHA 2017. **Proposed Short-Term Guideline Concentrations for Air Toxics**. Oregon Department of Environmental Quality and Oregon Health Authority. Submitted for public comment on February 28, 2017.

EPA 1989. **Risk Assessment Guidance for Superfund: Volume 1 – Human Health Evaluation Manual (Part A)**. U.S. Environmental Protection Agency, EPA/540/1-89/002, October 1989, Interim Final.

EPA 1992. **Guidelines for Exposure Assessment**. U.S. Environmental Protection Agency, EPA/600/Z-92/001, May 29, 1992. http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=15263.

EPA 2000. **Chemical Assessment Summary for Vinyl Chloride**. U.S. Environmental Protection Agency, Integrated Risk Information System (IRIS). August 7, 2000.

EPA 2005a. **Guidelines for Carcinogenic Risk Assessment**. U.S. Environmental Protection Agency, EPA/630/P-03/001B, March 2005.

EPA 2005b. **Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens**. U.S. Environmental Protection Agency. EPA 630-R-03-003F. March 2005.

EPA 2006. **Implementation of the Cancer Guidelines and Accompanying Supplemental Guidance – Science Policy Council Cancer Guidelines Implementation Workgroup Communication II: Performing Risk Assessments that Include Carcinogens Described in the Supplemental Guidance as Having a Mutagenic Mode of Action**. U.S. Environmental Protection Agency, Memorandum dated June 14, 2006.

EPA 2011. **Exposure Factors Handbook: 2011 Edition**. U. S. Environmental Protection Agency, EPA/600/R-09/052F, September 2011.

EPA 2016a. **AERSCREEN User's Guide.** U.S. Environmental Protection Agency. EPA-454/B-16-004, December 2016.

EPA 2016b. **User's Guide for the AMS/EPA Regulatory Model (AERMOD).** U.S. Environmental Protection Agency. EPA-454-B-16-011, December 2016.

EPA 2017a. **40 CFR Part 51. Revisions to the Guideline on Air Quality Models: Enhancements to the AERMOD Dispersion Modeling System and Incorporation of Approaches to Address Ozone and Fine Particulate Matter.** U. S. Environmental Protection Agency, January 17, 2017.

EPA 2017b. **Regional Screening Levels**. U.S. Environmental Protection Agency, June 2017. https://www.epa.gov/risk/regional-screening-levels-rsls#_ga=1.61411557.813403371.1432240980

MPCA 2016. **Air Emissions Risk Analysis (AERA) Guidance**. Minnesota Pollution Control Agency. Accessed December 2016. https://www.pca.state.mn.us/air/aera-guide

SCAQMD 2016a. **Permit Application Package "M". For use in conjunction with the Risk Assessment Procedures for Rules 1401, 1401.1, and 212, Version 8.0**. South Coast Air Quality Management District. Revised March 2016.

SCAQMD 2016b. **Rule 1402. Control of Toxic Air Contaminants from Existing Sources**. South Coast Air Quality Management District. Adopted April 8, 1994. Last amendment October 7, 2016.

Van den Berg 2006. **The 2005 World Health Organization Re-evaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-like Compounds**. Martin van den Berg, et al. Published by Oxford University Press on behalf of the Society of Toxicology, ToxSci Advance Access, July 7, 2006.

Screening Examples

These examples show how to summarize an air toxics screening evaluation. Example 1-1 shows the characteristics of two emission units at an existing facility, the nearest distances to various exposure locations, and the corresponding dispersion factors obtained from OAR 340-245-8060 Table 6. Example 1-2 shows the calculation of air concentrations using the dispersion factors and site-specific emission rates for each chemical. Example 1-3 shows the comparison of calculated air concentrations with Risk-Based Concentrations from OAR 340-245-8050 Table 5, and the resulting risk calculations. Because the risks exceed Risk Action Levels for an existing facility, an additional evaluation was performed.

Example 2 shows air concentrations obtained from AERSCREEN air dispersion modeling, the comparison with RBCs, and the resulting risk calculations. Using more site-specific modeling resulted in lower air concentrations, but the risks are still above RALs.

Example 3 shows air concentrations obtained from more sophisticated AERMOD air dispersion modeling, the comparison with RBCs, and the resulting risk calculations. Using the more realistic modeling resulted in lower air concentrations such that risks are not above RALs.

Example 4 is a summary of calculated risk in the example at each risk assessment level.

Example 1-1 – Emission Unit Information and Dispersion Factors

	Value	OAR 340-245-8060 Table 6
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)

Example 1-2 –Level 1 Calculation of Air Concentrations

Air Toxic	Annual Emission Rate (lb/yr)	24-Hour Emission Rate (lb/day)	Calc. Annual Residential Concentration (µg/m^3)	Calc. Annual Nonresidential Child Conc. (µg/m^3)	Calc. Annual Nonresidential Worker Conc. (µg/m^3)	Calculated 24-Hour Concentration (µg/m^3)
Emission Unit 1						
Cobalt	40	0.15	0.030	0.013	0.013	0.41
Manganese	70	0.25	0.053	0.023	0.023	0.68
Nickel	80	0.30	0.060	0.026	0.026	0.81
Dispersion Factor:			**0.00075** (ug/m3 per lb/yr)	**0.00033** (ug/m3 per lb/yr)	**0.00033** (ug/m3 per lb/yr)	**2.7** (ug/m3 per lb/day)
Emission Unit 2						
Acetaldehyde	100,000	300	17	10	10	191
Acetone	80,000	250	14	8	8	159
Acrolein	25,000	75	4	3	3	48
Dispersion Factor:			**0.00017** (ug/m3 per lb/yr)	**0.00010** (ug/m3 per lb/yr)	**0.00010** (ug/m3 per lb/yr)	**0.635** (ug/m3 per lb/day)

Notes:
Concentration = Emission Rate * Dispersion Factor
Dispersion factors from OAR 340-245-8060 Table 6.

Example 1-3 –Summary Risk Table for Level 1 Screening Risk Assessment

	Residential Exposure					Non-Resident Child Exposure					Non-Resident Worker Exposure					Acute Exposure		
Air Toxic	Annual Avg Conc. (µg/m^3)	RBC Cancer (µg/m^3)	Excess Cancer Risk	RBC Noncancer (ug/m3)	Hazard Quotient	Annual Average Conc. (µg/m^3)	RBC Cancer (µg/m^3)	Excess Cancer Risk	RBC Noncancer (µg/m^3)	Hazard Quotient	Annual Average Conc. (µg/m^3)	RBC Cancer (µg/m^3)	Excess Cancer Risk	RBC Noncancer (µg/m^3)	Hazard Quotient	24-Hour Average Conc. (µg/m^3)	Acute RBC (µg/m^3)	Hazard Quotient
Emission Unit 1																		
Cobalt	0.030	0.00011	273	0.10	0.30	0.013	0.0047	3	0.42	0.031	0.013	0.0013	10	0.42	0.031	0.41		
Manganese	0.053			0.090	0.58	0.023			0.38	0.061	0.023			0.38	0.06	0.68	0.30	2.3
Nickel	0.060	0.0040	15	0.014	4.3	0.026	0.16	0.17	0.042	0.63	0.026	0.046	0.57	0.042	0.6	0.81	0.20	4.1
Total Unit 1			**288**		**5**			**3**		**0.72**			**11**		**0.7**			**6**
Emission Unit 2																		
Acetaldehyde	17	0.45	38	9	1.9	10	11	0.9	38	0.26	10	5.4	2	38	0.26	191	470	0.4
Acetone	14			31,000	0.00044	8			130,000	0.00006	8			130,000	0.00006	159	62,000	0.003
Acrolein	4			0.35	12	3			1.5	1.7	3			1.5	1.7	48	6.9	6.9
Total Unit 2			**38**		**14**			**0.9**		**1.9**			**2**		**1.9**			**7**
Total Source (Unit 1 and Unit 2)			**326**		**19**			**4**		**3**			**13**		**3**			**14**
Existing Facility Risk Action Level			*25*		*1*			*25*		*1*			*25*		*1*			*1*

Notes:
Excess Cancer Risk = Annual conc. (µg/m^3) / Cancer RBC (µg/m^3)
Expressed as risk per million
Chronic Hazard Quotient = Annual conc. (µg/m^3) / Noncancer RBC (µg/m^3) x 1
Acute Hazard Quotient = 24-hr conc. (µg/m^3) / Acute RBC (µg/m^3) x 1
Air concentrations calculated using dispersion factors from OAR 340-245-8060 Table 6.

Example 2 –Summary Risk Table for Level 2 Screening Risk Assessment

	Residential Exposure					Non-Resident Child Exposure					Non-Resident Worker Exposure					Acute Exposure		
Air Toxic	**Annual Average Conc. (µg/m³)**	**RBC Cancer (µg/m³)**	**Excess Cancer Risk**	**RBC Noncancer (µg/m³)**	**Hazard Quotient**	**Annual Average Conc. (µg/m³)**	**RBC Cancer (µg/m³)**	**Excess Cancer Risk**	**RBC Noncancer (µg/m³)**	**Hazard Quotient**	**Annual Average Conc. (µg/m³)**	**RBC Cancer (µg/m³)**	**Excess Cancer Risk**	**RBC Noncancer (µg/m³)**	**Hazard Quotient**	**24-Hour Average Conc. (µg/m³)**	**Acute RBC (µg/m³)**	**Hazard Quotient**
Emission Unit 1																		
Cobalt	0.015	0.00011	136	0.10	0.15	0.0066	0.0047	1	0.42	0.016	0.0066	0.0013	5.1	0.42	0.016	0.20		
Manganese	0.026			0.09	0.29	0.012			0.38	0.030	0.012			0.38	0.030	0.34	0.30	1.1
Nickel	0.030	0.0040	8	0.014	2.1	0.013	0.16	0.1	0.042	0.31	0.013	0.0460	0.29	0.042	0.31	0.41	0.20	2.0
Total Unit 1			**144**		**2.6**			**1**		**0.36**			**5.4**		**0.36**			**3.2**
Emission Unit 2																		
Acetaldehyde	9	0.45	19	9	0.94	5	11	0.45	38	0.13	5	5.4	0.9	38	0.13	95	470	0.2
Acetone	7			31,000	0.00022	4			130,000	0.000031	4			130,000	0.000031	79	62,000	0.0013
Acrolein	2			0.35	6	1			1.5	0.83	1			1.5	0.83	24	6.9	3.5
Total Unit 2			**19**		**7**			**0.5**		**0.97**			**0.9**		**0.97**			**3.7**
Total Source (Unit 1 and Unit 2)			**163**		**10**			**2**		**1**			**6**		**1**			**7**
Existing Facility Risk Action Level			*25*		*1*			*25*		*1*			*25*		*1*			*1*

Notes:
Excess Cancer Risk = Annual conc. (µg/m³) / Cancer RBC (µg/m³)
Expressed as risk per million
Chronic Hazard Quotient = Annual conc. (µg/m³) / Noncancer RBC (µg/m³) x 1
Acute Hazard Quotient = 24-hr conc. (µg/m³) / Acute RBC (µg/m³) x 1
Air concentrations modeled using AERSCREEN.

Example 3 –Summary Risk Table for Level 3 Risk Assessment

	Residential Exposure					Non-Resident Child Exposure					Non-Resident Worker Exposure					Acute Exposure		
Air Toxic	**Annual Average Conc. (µg/m³)**	**RBC Cancer (µg/m³)**	**Excess Cancer Risk**	**RBC Noncancer (µg/m³)**	**Hazard Quotient**	**Annual Average Conc. (µg/m³)**	**RBC Cancer (µg/m³)**	**Excess Cancer Risk**	**RBC Noncancer (µg/m³)**	**Hazard Quotient**	**Annual Average Conc. (µg/m³)**	**RBC Cancer (µg/m³)**	**Excess Cancer Risk**	**RBC Noncancer (µg/m³)**	**Hazard Quotient**	**24-Hour Average Conc. (µg/m³)**	**Acute RBC (µg/m³)**	**Hazard Quotient**
Emission Unit 1																		
Cobalt	0.0015	0.00011	14	0.10	0.015	0.00066	0.0028	0.24	0.42	0.0016	0.0007	0.0013	0.51	0.42	0.0016	0.020		
Manganese	0.0026			0.090	0.029	0.0012			0.38	0.0030	0.0012			0.38	0.0030	0.034	0.30	0.11
Nickel	0.0030	0.0040	1	0.014	0.21	0.0013	0.10	0.013	0.059	0.022	0.0013	0.048	0.028	0.059	0.022	0.041	0.20	0.20
Total Unit 1			**14**		**0.26**			**0.2**		**0.027**			**0.54**		**0.027**			**0.32**
Emission Unit 2																		
Acetaldehyde	0.9	0.45	2	9	0.094	0.50	11	0.05	38	0.013	0.50	5.4	0.09	38	0.013	10	470	0.02
Acetone	0.7			31,000	0.000022	0.40			130,000	0.0000031	0.40			130,000	0.0000031	8	62,000	0.00013
Acrolein	0.2			0.35	0.61	0.13			1.5	0.083	0.13			1.5	0.083	2	6.9	0.35
Total Unit 2			**2**		**0.7**			**0.05**		**0.097**			**0.09**		**0.097**			**0.37**
Total Source (Unit 1 and Unit 2)			**16**		**1**			**0.3**		**0.1**			**0.6**		**0.1**			**0.7**
Existing Facility Risk Action Level			*25*		*1*			*25*		*1*			*25*		*1*			*1*

Notes:
Excess Cancer Risk = Annual conc. (µg/m³) / Cancer RBC (µg/m³)
Expressed as risk per million
Chronic Hazard Quotient = Annual conc. (µg/m³) / Noncancer RBC (µg/m³) x 1
Acute Hazard Quotient = 24-hr conc. (µg/m³) / Acute RBC (µg/m³) x 1
Air concentrations modeled using AERMOD.

Example 4 – 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	326	19	4	3	13	3	14
Level 2	163	10	2	1	6	1	7
Level 3	16	1	0.3	0.1	0.6	0.1	0.7
Risk Action Level[b]	*25*	*1*	*25*	*1*	*25*	*1*	*1*

Note:

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

APPENDIX A

Hierarchy for Authoritative Sources of Toxicity Reference Values

A.1 Chronic Values

DEQ used the following hierarchy of sources of chronic toxicity reference values (TRVs):

1. DEQ alone or in consultation with DEQ's Air Toxics Science Advisory Committee (ATSAC) and/or Oregon Health Authority (OHA), including Ambient Benchmark Concentrations (ABCs)
2. EPA Integrated Risk Information System (IRIS) database (www.epa.gov/iris)
3. EPA Provisional Peer-Reviewed Toxicity Value (PPRTV) database (www.hhpprtv.ornl.gov)
4. Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profiles (www.atsdr.cdc.gov)
5. California's Office of Environmental Health Hazard Assessment (OEHHA)(www.oehha.ca.gov)

Chronic TRVs were developed separately for noncarcinogenic and carcinogenic effects.

Inhalation toxicity information for noncancer effects are typically provided as threshold values 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 Concentration.

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

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

A.2 Acute Values

DEQ used the following hierarchy of sources of acute toxicity reference values (TRVs):

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 bodies, 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, in DEQ and OHA's draft short-term guideline concentrations, the recommended short-term guideline concentration for selenium of 2 µg/m^3 was developed by dividing the Occupational Safety and Health Administration (OSHA) value of 200 µg/m^3 for long-term daily worker exposure by a factor of 100 (DEQ/OHA 2017). The chronic noncancer value from OEHAA is 20 µg/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 selenium.

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 B

Development of Adjustment Factors and Calculation of Risk-Based Concentrations

B.1 Introduction

Risk-based concentrations (RBCs) were calculated 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

Short-term acute exposure was also considered.

Three adjustments of Toxicity Reference Values were made, 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 chemicals, which involve exposure routes other than inhalation alone. This is a multipathway adjustment. The third adjustment considers early-life exposure to chemicals that exhibit greater toxicity to infants and children. These adjustments are reflected in the chronic RBCs listed in OAR 340-245-8050 Table 5. The development of each adjustment factor is discussed below.

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

B.2 Development of Adjustment Factors

B.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, factors for more limited exposure were used 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 for someone present 8 hr/day, 5 day/week is:

$$\text{childNRAFnc} = \text{workerNRAFnc} = (24 \text{ hrs/day} / 8 \text{ hrs/day}) \times (7 \text{ days/wk} / 5 \text{ days/wk}) = 4.2$$

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 worker exposure duration assumption is 25 years. The NRAF values for cancer effects are:

childNRAFc = (24 hrs/day / 8 hrs/day) x (7 days/wk / 5 days/wk) x (70 yrs / 12 yrs) = 25

workerNRAFc = (24 hrs/day / 8 hrs/day) x (7 days/wk / 5 days/wk) x (70 yrs / 25 yrs) = 12

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

B2.1.1. Life Expectancy

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's recommendation for Superfund risk assessments is to continue using a 70-year lifetime, and this recommendation is used by DEQ's Cleanup Program. DEQ determined that it is appropriate for Cleaner Air Oregon risk assessments to use a 70-year lifetime. This decision is slightly more protective than assuming a 78-year lifetime.

B.2.2 Multipathway Adjustment Factors

DEQ considered developing Multipathway Adjustment Factors (MPAFs) specific to Oregon, but determined that the agency did not have the time or 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 chemicals evaluated. DEQ acknowledges that exposure conditions may not be the same in Oregon, but considers the MPAFs to be appropriately protective.

MPAFs were only applied 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.

B.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 chemicals acting by a mutagenic mode of action, we use EPA's general approach for account for early-life exposure using age-dependent adjustment factors, ADAFs. The approach is different for two chemicals. For

trichloroethene (TCE), EPA considers early-life appropriate for liver cancer only, and not kidney cancer or non-Hodgkins Lymphoma. The 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 C.

B.3 Calculation of RBCs

B.3.1 Residential RBCs

DEQ applied the multipathway adjustment factor (MPAF) and early-life adjustment factor (ELAF) values shown in OAR 340-245-8040 Table 4 to the TRVs in OAR 340-245-8030 Table 3 using the following equations to calculate residential risk-based concentrations (RBCs) in OAR 340-245-8050 Table 5. The acute TRV is used directly as the acute RBC.

$$residRBCc = \frac{TRVc}{ELAF * MPAFrc}$$

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

$$acuteRBC = TRVa$$

Where:
residRBCc = Residential risk-based concentration for cancer effects (μg/m^3)
residRBCnc = Residential risk-based concentration for noncancer effects (μg/m^3)
acuteRBC = Short-term toxicity reference concentration (μg/m^3)
TRVc = Toxicity reference value for cancer effects (μg/m^3)
TRVnc = Toxicity reference value for noncancer effects (μg/m^3)
TRVa = Toxicity reference value for acute effects (μg/m^3)
ELAF = early-life adjustment factor (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 chemical, these adjustments are omitted. For most chemicals, this is the case, and the residential RBC is equal to the TRV.

B.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. The following equations were used to calculate non-residential RBCs.

$$nrchildRBCc = \frac{TRVc * childNRAFc}{ELAF * MPAFnrc}$$

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

$$workerRBCc = \frac{TRVc * worker NRAFc}{MPAFnrc}$$

$$workerRBCnc = \frac{TRVnc * worker NRAFnc}{MPAFnrnc}$$

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)
TRVa = Toxicity reference value for acute effects (µg/m^3)
ELAF = early-life adjustment factor (unitless)
MPAFnrc = multipathway adjustment factor, nonresident cancer (unitless)
MPAFnrnc = multipathway adjustment factor, nonresident noncancer (unitless)
childNRAFc = Nonresident adjustment factor, child cancer (42) (unitless)
childNRAFnc = Nonresident adjustment factor, child noncancer (4.2) (unitless)
workerNRAFc = Nonresident adjustment factor, worker cancer (12) (unitless)
workerNRAFnc = Nonresident adjustment factor, worker noncancer (4.2) (unitless)

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

B.3.3 Acute RBCs

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

$$acuteRBC = TRVa$$

Where:

acuteRBC = Short-term toxicity reference concentration (µg/m^3)

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

APPENDIX C

Derivation of Early-Life Adjustment Factors

C.1 Introduction

This appendix covers the development of early-life adjustment factors (ELAFs) and the evaluation of early-life exposure for certain compounds. The chemicals for which incorporation of early-life exposure will be necessary are shown in Table C-1. A general discussion is provided 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 chemicals. Early-life exposure is included in the derivation of RBCs for residential and non-residential child exposure scenarios.

C.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 chemicals, 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 chemicals. 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 chemicals should be evaluated for early-life exposure (EPA 2006). The list of chemicals, with updates, is provided in Table C-1. Most chemicals are evaluated using the standard approach. For TCE, EPA considers early-life appropriate for liver cancer only, and not kidney cancer or non-Hodgkins Lymphoma. Because of this complication, the approach for TCE is discussed separately. Also, 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.

C.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 C-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 (260 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 chemicals with early-life adjustments in Table 2, with the exception of TCE and vinyl chloride, which are addressed using the approaches described below.

C.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-Hodgkins 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-Hodgkins 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-Hodgkins lymphoma	2.1×10^{-6}	0.48
Total	4.1×10^{-6}	0.24

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

C.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 ug/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 DEQ decisions, including consultation with ATSAC, are the top tier in the TRV selection hierarchy, we use 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 0.1 µg/m^3 by 2 to get a non-early-life TRV of 0.2 µg/m^3.

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

$$RBC_{early-life} = \frac{70\,\text{yr} \cdot 365\,\text{days/yr} \cdot 0.2\,\mu\text{g/m}^3}{70\text{yr} \cdot 365\,\text{days/yr}}$$

$$= \quad 0.20\ \mu\text{g/m}^3$$

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

$$RBC_{child} = \frac{70\,\text{yr} \cdot 365\,\text{days/yr} \cdot 24 hrs/day \cdot 0.2\,\mu\text{g/m}^3}{12\text{yr} \cdot 260\,\text{days/yr} \cdot 8 hrs/day}$$

$$= \qquad 4.9\ \mu\text{g/m}^3$$

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:

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

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

$$RBC_{early-life/child} = \frac{1}{\frac{1}{4.9} + \frac{1}{0.20}}$$

$$= 0.19\ \mu g/m^3$$

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

Table C-1
Air Toxics Chemicals Determined by EPA to be Carcinogens Having a Mutagenic Mode of Action

Chemical[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, 2017.
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-Hodgkins 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 C-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 2017).
   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.