# DRAFT RECOMMENDED PROCEDURES FOR CONDUCTING TOXIC AIR CONTAMINANT HEALTH RISK ASSESSMENTS

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

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# Table of Contents

1. INTRODUCTION ..... 1
**1.1 Purpose and Organization ..... 1**
**1.2 Process Overview ..... 1**
2. RISK SCREENING AND ASSESSMENT ..... 3
**2.1 Risk Assessment Concepts ..... 3**
**2.2 Risk-Based Concentrations ..... 4**
2.2.1 Exposure Frequency and Duration Considerations ..... 5
2.2.2 Multipathway Considerations ..... 5
2.2.3 Early-Life Exposure ..... 5
**2.3 Risk Assessment Elements ..... 6**
2.3.1 Conceptual Site Model ..... 6
**2.4 Exposure Assessment and Air Dispersion Modeling ..... 7**
2.4.1 Air Dispersion Modeling ..... 7
2.4.2 Use of Air Monitoring Data in Risk Assessments ..... 9
**2.5 Toxicity Assessment ..... 9**
2.5.1 Assessment of Noncancer Health Effects ..... 10
2.5.2 Assessment of Cancer Effects ..... 10
2.5.3 Assessment of Chemical Mixtures ..... 10
2.5.4 Screening Terminology ..... 11
**2.6 Uncertainty Evaluation ..... 11**
3. LEVEL 1 AND 2 SCREENING ..... 12
**3.1 Level 1 Screening Approach ..... 12**
**3.2 Level 2 Screening Approach ..... 14**
4. LEVEL 3 RISK ASSESSMENT AND LEVEL 4 COMPREHENSIVE RISK ASSESSMENT ..... 15
**4.1 Level 3 Risk Assessment ..... 15**
**4.2 Level 4 Comprehensive Risk Assessment ..... 16**
4.2.1 Exposure Assumption Modifications ..... 16
4.2.2 Relative Bioavailability ..... 16
4.2.3 Multipathway Analysis ..... 17
4.2.4 Permit Denial and Immediate Curtailment Levels ..... 17
4.2.5 Risk Assessment Updates ..... 17
5. REFERENCES ..... 19
APPENDIX A ..... 27

**Authoritative Sources of Toxicity Reference Values** ..... **27**
APPENDIX B ..... 29
**Development of Adjustment Factors and Calculation of Risk-Based Concentrations** ..... **29**
APPENDIX C ..... 36
**Derivation of Early-Life Adjustment Factors** ..... **36**
APPENDIX D ..... 43
**Use of the Toxic Equivalency Factor Methodology for Dioxins and Furans, PCBs, and PAHs** ..... **43**
APPENDIX E ..... 50
**Compilation of Target Organs for Toxic Air Contaminants** ..... **50**
APPENDIX F ..... 51
**Procedures for Updating Risk Assessments** ..... **51**

TABLES (from OAR 340-245)

Table 1 – Risk Action Levels (OAR 340-245-8010)
Table 2 – Toxic Air Contaminant Reporting List (OAR 340-245-8020)
Table 3 – Toxicity Reference Values (OAR 340-245-8030)
Table 4 – Risk-Based Concentrations (OAR 340-245-8040)
Table 5 – Level 1 Risk Assessment Tool, Dispersion Factors (OAR 340-245-8050)

# 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 |
| ESRE | Emission Scaled Risk Estimate |
| EQC | Environmental Quality Commission |
| HI | Hazard Index |
| HQ | Hazard Quotient |
| IRIS | EPA's Integrated Risk Information System |
| IUR | Inhalation Unit Risk |
| 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 toxic air contaminant screening and 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 toxic air contaminant 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 will 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 or a Level 4 comprehensive health risk assessment, you should prepare a risk assessment work plan and an air dispersion Modeling Protocol, and meet with DEQ staff to discuss your plans for conducting the 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 340-245-8050 Table 5 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 340-245-8040 Table 4 for residential and non-residential exposure locations, and acute exposure locations. Finally, compare the summed excess cancer risks and hazard indices with the 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, and other facilities, 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 locations, and acute exposure locations.

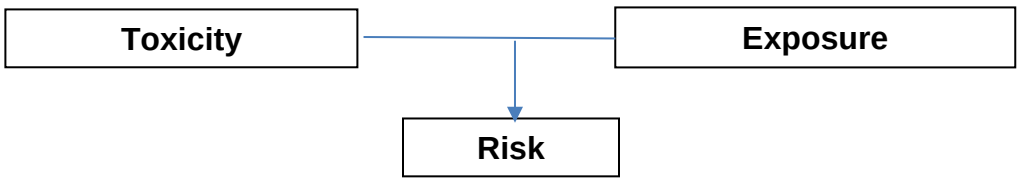
**Level 3 – Simple Risk Assessment**. At this level, you can use detailed site-specific information (stack heights, building heights, topography, distances to various exposure locations) and perform complex modeling using EPA's AERMOD model to calculate air concentrations for comparison with RBCs.

**Level 4 – Comprehensive Risk Assessment**. The most comprehensive risk assessment option 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.

# 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 toxic air contaminants in OAR 340-245-8040 Table 4, and ultimately reduce risk. 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 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 (µg/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).

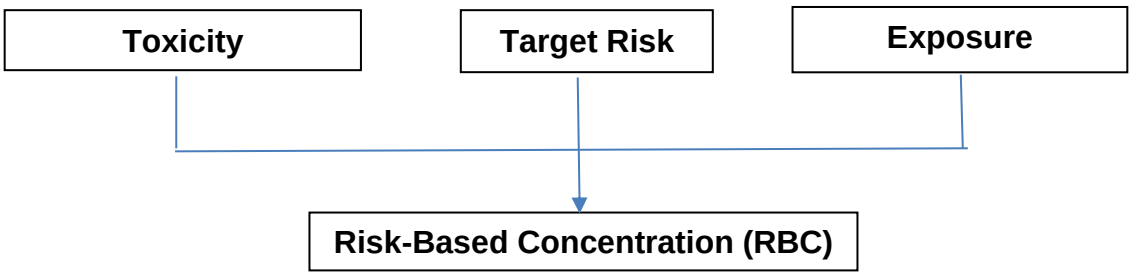
For cancer effects, the assumption is that there is no threshold for adverse effects. Although the risk at a very low concentration of a carcinogen may be very low, we assume it is not zero. Because of this assumption, the toxicity of carcinogens is given not as a threshold concentration, but instead as a probability of getting cancer if exposed continuously to a concentration of 1 microgram per cubic meter, or µg/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 toxic air contaminants from a single facility to be far less than one million, so the calculated excess cancers in the exposed population (called a cancer burden) as a result of emissions from a facility is expected to be much less than 1. To be clear, 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 in the development of RBCs, 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 Table B-1 in Appendix B. Other adjustments to TRVs are discussed in Section 2.2.

DEQ developed RBCs for each toxic air contaminant 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 Table B-1. Appendix B shows how the adjustment factors were used to develop the RBCs shown in OAR 340-245-8040 Table 4. Adjustment factors apply only to chronic exposure. None of the adjustment factors are appropriate or necessary for acute RBCs because of the short period of exposure being considered.

### 2.2.1 Exposure Frequency and Duration Considerations

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

### 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 adjust the cancer TRV. Currently, the chemicals of primary interest for consideration of early-life exposure are listed in Table B-1 with early-life adjustment factors (ELAFs). Appendix C shows the derivation of 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 toxic air contaminant 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. You should consider reasonably likely exposure scenarios based on location. 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. If you know that people do not actually live in the area, you can provide documentation to DEQ that it is not appropriate to evaluate residential use in the area. If DEQ concludes that the documentation is adequate to rebut the presumption of residential use, you can adjust your exposure assessment accordingly. However, you must annually show that the excluded zoned areas continue to not be used in the manner allowed by the land use zoning.

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.
- Nonresident adult exposure, which includes adults in office buildings, commercial buildings, 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 facilities, 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 toxic air contaminants 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 estimate 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, which should not be confused with the term “receptor” commonly used to identify humans in a risk assessment. 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 340-245-8050 Table 5 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 2017), 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. The monitoring protocol should include data quality objectives, and describe how exposure point concentrations will be used to evaluate risk in a revised risk assessment.

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 toxic air contaminants a facility emits or may emit, and to estimate the relationship between the amount of exposure of a toxic air contaminant 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 toxic air contaminants. 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 µg/m$^3$ or mg/m$^3$. The federal Agency for Toxic Substances and Disease Registry calls these concentrations Minimal Risk Levels (MRLs), and California's Office of Environmental Health Hazard Assessment calls them Risk Exposure Levels (RELs). For the purpose of this document, we will use 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. DEQ intends to prepare a table of applicable organ systems for toxic air contaminants and include this as Appendix E.

### 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 when exposed continuously to a concentration of 1 µg/m$^3$. This value is called the inhalation unit risk (IUR) value, in units of risk per microgram per cubic meter (µg/m$^3$)$^{-1}$. For ease of use in assessing risk in the CAO program, IURs were converted to TRVs using a target excess cancer risk level of one in one million.

### 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. Details about how to address chemicals classes are presented in Appendix D.

**<u>Toxicity of CDDs/CDFs and Dioxin-like PCBs</u>**
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 Appendix D. 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 toxic air contaminants is part of the overall risk evaluation approach. Toxic air contaminants 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, toxic air contaminants 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, toxic air contaminants 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 for 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 results of the risk assessment.

# 3. LEVEL 1 AND 2 SCREENING

To conduct screening, you need to calculate concentrations of toxic air contaminants at the exposure locations 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 toxic air contaminant concentrations.

## 3.1 Level 1 Screening Approach

The Level 1 approach allows you to use toxic air contaminant 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, and other facilities, 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 340-245-8050 Tables 5A (annual exposure) and 5B (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. In addition, Level 2 screening may be necessary if the source is in complex terrain, where the elevation of receptors, within a distance equivalent to ten stack heights, is higher than the stack height of the source.

The use of OAR 340-245-8050 Table 5 requires only the stack height and distance to the nearest exposure area. You will likely have four types of exposure areas to evaluate (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 toxic air contaminant emissions at the capacity to emit; 2) if you want to demonstrate that risk at your pre-existing Potential to Emit (PTE) level 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 toxic air contaminant emissions using the requested limit, which may be based on actual emissions if you can continue to comply at that level. You may assess risk both at the actual emission rate and another rate such as PTE. This may assist with communicating risk to the public by showing typical as well as maximum emission rates. 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 units of micrograms per cubic meter, $\mu g/m^3$.

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

To generate the dispersion factors provided in OAR 340-245-8050 Table 5, DEQ first developed a series of reasonable maximum assumptions associated with stack height, such as stack diameter 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 $\mu g/m^3$ per lbs/year for chronic exposure, or $\mu g/m^3$ per lbs/day for acute exposure. The results are shown in OAR 340-245-8050 Tables 5A (annual exposure) and 5B (24-hour exposure).

Next, divide the calculated air concentration by the respective RBC for that toxic from OAR 340-245-0840 Table 4. 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 toxic air contaminants for each of 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.

The Level 1 screening process 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, but it does indicate a need for further evaluation. 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 toxic air contaminant concentrations in $\mu g/m^3$ from all of the individual stacks to estimate an aggregate concentration that is then compared to the RBC for that toxic air contaminant; or 2) group the stacks and their emissions into a single stack, and use the information in OAR 340-245-8050 Table 5 to determine a dispersion factor to apply to the grouped emissions in order to estimate an air concentration. DEQ can assist with information about methods to group stacks.

For a stack height between values shown in OAR 340-245-8050 Table 5, 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. For stack heights greater than 50 meters, use the appropriate dispersion factor for 50 meters. For exposure locations greater than 1,000 meters from your facility, use the appropriate dispersion factor at 1,000 meters.

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 $\mu g/m^3$ / lbs/yr) and daily dispersion factor (8.3 $\mu g/m^3$ / lbs/day) for a stack height of 5 meters and an exposure location distance of 50 meters.

To update an existing risk assessment with new information, see Appendix F for options ranging from simple calculations to a fully revised risk assessment.

# 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 toxic air contaminant 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 toxic air contaminant emissions using the requested limit, which may be based on actual emissions if you can continue to comply at that level. You may assess risk both at the actual emission rate and another rate such as PTE. This may assist with communicating risk to the public by showing typical as well as maximum emission rates.

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 most 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 then 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 340-245-8040 Table 4. To complete the screening step, add the results for all the applicable toxic air contaminants for each of the different types of RBCs (chronic cancer, chronic noncancer, and acute noncancer). 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, but it would indicate a need for further evaluation. 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 toxic air contaminant concentrations.

To update an existing risk assessment with new information, see Appendix F for options ranging from simple calculations to a fully revised risk assessment.

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

The screening steps of Levels 1 and 2 include 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 will be required. DEQ will require you to prepare a risk assessment work plan and modeling protocol for Level 3 and 4 evaluations.

## 4.1 Level 3 Risk 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 toxic air contaminant 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 toxic air contaminant 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 risk assessment 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 toxic air contaminant emissions from the source, including residents, nonresident adults, and nonresident children and other sensitive populations;
- An exposure assessment that models or measures toxic air contaminant concentrations at locations of existing and reasonably likely future human populations that may be exposed to toxic air contaminant emissions from the source. Modifications to default exposure assumptions may be proposed, including but not limited to exposure times, frequencies, and durations, relative bioavailability of chemicals, and multipathway considerations for persistent, bioaccumulative and toxic chemicals;
- A toxicity assessment evaluating the carcinogenicity, noncarcinogenic chronic effects, and noncarcinogenic acute effects of toxic air contaminants 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, and additional multipathway considerations not addressed by the default adjustment factors. 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 by a person, 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 work plan for approval prior to conducting the evaluation.

The particular form of a chemical that exists 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 toxic air contaminant emissions, California's OEHHA's Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments (2015).

### 4.2.4 Permit Denial and Immediate Curtailment Levels

DEQ will not approve a permit for a source if we determine that:

- Emissions from a proposed new source would result in risk at any exposure location that will exceed any Permit Denial Level in OAR 340-245-8010 Table 1; or
- Emissions from an existing source would result in risk at any exposure location that will exceed any Immediate Curtailment Level in OAR 340-245-8010 Table 1

If your risk assessment shows that calculated risks are above Immediate Curtailment Levels, you should immediately plan on addressing the risk.

### 4.2.5 Risk Assessment Updates

To update an existing risk assessment with new information, see Appendix F for options ranging from simple calculations to a fully revised risk assessment.

**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.

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. Finally, DEQ considered risk benchmarks set in Oregon statute following passage of Senate Bill 1541 in the 2018 Oregon Legislature.

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

**Adjusted Hazard Index RAL**

DEQ is allowed to adjust a RAL for noncancer risk to a hazard index value other than 5. To be eligible for this adjustment, a chemical must be expected to have developmental human health effects associated with pre- or post-natal exposure, or other severe human health effects. The adjusted RAL may be no less than a hazard index of 3. To establish an adjusted RAL under rule, the EQC must first establish and consider the recommendations of an advisory committee.

The statutory requirement limiting a noncancer RAL to no less than 3 expires on January 1, 2029, after which the EQC may establish a new noncancer RAL.

# 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 2017. **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 2018. **Regional Screening Levels**. U.S. Environmental Protection Agency, May 20187. 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 340-245-8050 Table 5. 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 340-245-8040 Table 4, 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 340-245-8050 Table 5 | |
|---|---|---|---|
| **Emission Unit 1** | **(meters)** | **Dispersion Factor** | |
| Stack height = | 10 | | |
| Distance to residential = | 100 | 0.00075 | µg/m$^3$ per lb/yr |
| Distance to nonresident child = | 200 | 0.00033 | |
| Distance to nonresident worker = | 200 | 0.00033 | |
| Distance to acute = | 85 | 2.7 | µg/m$^3$ per lb/day |
| | | (interpolated between 2.6 and 2.8) | |
| **Emission Unit 2** | **(meters)** | **Dispersion Factor** | |
| Stack height = | 20 | | |
| Distance to residential = | 150 | 0.00017 | µg/m$^3$ per lb/yr |
| Distance to nonresident child = | 250 | 0.00010 | |
| Distance to nonresident worker = | 250 | 0.00010 | |
| Distance to acute = | 135 | 0.635 | µg/m$^3$ per lb/day |
| | | (interpolated between 0.62 and 0.65) | |

**Example 1-2 –Level 1 Calculation of Air Concentrations**

| Toxic Air Contaminant | 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** | | | | | | |
| Cadmium | 140 | 0.38 | 0.11 | 0.046 | 0.046 | 1.0 |
| Manganese | 70 | 0.25 | 0.053 | 0.023 | 0.023 | 0.68 |
| Nickel (insoluble) | 220 | 0.60 | 0.17 | 0.073 | 0.073 | 1.6 |
| | **Dispersion Factor:** | | **0.00075** (µg/m3 per lb/yr) | **0.00033** (µg/m3 per lb/yr) | **0.00033** (µg/m3 per lb/yr) | **2.7** (µg/m3 per lb/day) |
| **Emission Unit 2** | | | | | | |
| Acetaldehyde | 100,000 | 300 | 17 | 10 | 10 | 191 |
| Acetone | 80,000 | 250 | 14 | 8 | 8 | 159 |
| Acrolein | 10,000 | 50 | 2 | 1 | 1 | 32 |
| | **Dispersion Factor:** | | **0.00017** (µg/m3 per lb/yr) | **0.00010** (µg/m3 per lb/yr) | **0.00010** (µg/m3 per lb/yr) | **0.635** (µg/m3 per lb/day) |

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

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

| | Residential Exposure | | | | | Non-Resident Child Exposure | | | | | Non-Resident Worker Exposure | | | | | Acute Exposure | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Toxic Air Contaminant** | **Annual Avg 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** | | | | | | | | | | | | | | | | | | |
| Cadmium | 0.11 | 0.00056 | 188 | 0.0050 | 21 | 0.046 | 0.014 | 3.3 | 0.037 | 1.3 | 0.046 | 0.0067 | 6.9 | 0.037 | 1.2 | 1.0 | 0.030 | 35 |
| Manganese | 0.053 | | | 0.090 | 0.58 | 0.023 | | | 0.40 | 0.058 | 0.023 | | | 0.40 | 0.06 | 0.68 | 0.30 | 2.3 |
| Nickel (insoluble) | 0.17 | 0.0038 | 43 | 0.014 | 12 | 0.073 | 0.10 | 0.73 | 0.062 | 1.2 | 0.073 | 0.046 | 1.6 | 0.062 | 1.2 | 1.6 | 0.20 | 8.1 |
| **Total Unit 1** | | | **231** | | **33** | | | **4.0** | | **2.5** | | | **8.5** | | **2.5** | | | **45** |
| **Emission Unit 2** | | | | | | | | | | | | | | | | | | |
| Acetaldehyde | 17 | 0.45 | 38 | 140 | 0.12 | 10 | 12 | 0.83 | 620 | 0.016 | 10 | 5.5 | 1.8 | 620 | 0.016 | 191 | 470 | 0.41 |
| Acetone | 14 | | | 31,000 | 0.00044 | 8 | | | 140,000 | 0.000057 | 8 | | | 140,000 | 0.000057 | 159 | 62,000 | 0.003 |
| Acrolein | 2 | | | 0.35 | 4.9 | 1 | | | 1.5 | 0.67 | 1 | | | 1.5 | 0.67 | 32 | 6.9 | 4.6 |
| **Total Unit 2** | | | **38** | | **5.0** | | | **0.83** | | **0.68** | | | **1.8** | | **0.68** | | | **5** |
| **Total Source (Unit 1 and Unit 2)** | | | **269** | | **38** | | | **4.9** | | **3.2** | | | **10** | | **3.2** | | | **50** |
| *Existing Facility Risk Action Level* | | | *50* | | *5* | | | *50* | | *5* | | | *50* | | *5* | | | *5* |

**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 calculated using dispersion factors from OAR 340-245-8050 Table 5.

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

| | Residential Exposure | | | | | Non-Resident Child Exposure | | | | | Non-Resident Worker Exposure | | | | | Acute Exposure | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Toxic Air Contaminant** | **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** | | | | | | | | | | | | | | | | | | |
| Cadmium | 0.032 | 0.00056 | 56 | 0.0050 | 6.3 | 0.014 | 0.014 | 0.99 | 0.037 | 0.37 | 0.014 | 0.0067 | 2.1 | 0.037 | 0.37 | 0.21 | 0.030 | 6.9 |
| Manganese | 0.016 | | | 0.090 | 0.18 | 0.0069 | | | 0.40 | 0.017 | 0.0069 | | | 0.40 | 0.017 | 0.14 | 0.30 | 0.45 |
| Nickel (insoluble) | 0.050 | 0.0038 | 13 | 0.014 | 3.5 | 0.022 | 0.10 | 0.22 | 0.062 | 0.35 | 0.022 | 0.046 | 0.47 | 0.062 | 0.35 | 0.33 | 0.20 | 1.6 |
| **Total Unit 1** | | | **69** | | **10** | | | **1.2** | | **0.74** | | | **2.5** | | **0.74** | | | **9.0** |
| **Emission Unit 2** | | | | | | | | | | | | | | | | | | |
| Acetaldehyde | 5 | 0.45 | 11 | 140 | 0.036 | 3.0 | 12 | 0.25 | 620 | 0.0048 | 3.0 | 5.5 | 0.55 | 620 | 0.0048 | 38 | 470 | 0.081 |
| Acetone | 4 | | | 31,000 | 0.00013 | 2.4 | | | 140,000 | 0.000017 | 2.4 | | | 140,000 | 0.000017 | 32 | 62,000 | 0.00051 |
| Acrolein | 1 | | | 0.35 | 1.5 | 0.30 | | | 1.5 | 0.20 | 0.30 | | | 1.5 | 0.20 | 6.4 | 6.9 | 0.92 |
| **Total Unit 2** | | | **11** | | **1.5** | | | **0.25** | | **0.20** | | | **0.55** | | **0.20** | | | **1.0** |
| **Total Source (Unit 1 and Unit 2)** | | | **81** | | **12** | | | **1.5** | | **0.95** | | | **3.1** | | **0.95** | | | **10** |
| *Existing Facility Risk Action Level* | | | *50* | | *5* | | | *50* | | *5* | | | *50* | | *5* | | | *5* |

**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 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Toxic Air Contaminant** | **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** | | | | | | | | | | | | | | | | | | |
| Cadmium | 0.0019 | 0.00056 | 3.4 | 0.010 | 0.38 | 0.00083 | 0.014 | 0.059 | 0.037 | 0.023 | 0.00083 | 0.0067 | 0.12 | 0.037 | 0.023 | 0.021 | 0.030 | 0.69 |
| Manganese | 0.0009 | | | 0.090 | 0.011 | 0.00042 | | | 0.40 | 0.0010 | 0.00042 | | | 0.40 | 0.0010 | 0.014 | 0.30 | 0.045 |
| Nickel (insoluble) | 0.0030 | 0.0038 | 0.78 | 0.014 | 0.21 | 0.0013 | 0.10 | 0.013 | 0.062 | 0.021 | 0.0013 | 0.046 | 0.028 | 0.062 | 0.021 | 0.033 | 0.20 | 0.16 |
| **Total Unit 1** | | | **4.2** | | **0.60** | | | **0.072** | | **0.045** | | | **0.15** | | **0.045** | | | **0.90** |
| **Emission Unit 2** | | | | | | | | | | | | | | | | | | |
| Acetaldehyde | 0.31 | 0.45 | 0.68 | 140 | 0.0022 | 0.18 | 12 | 0.015 | 620 | 0.00029 | 0.18 | 5.5 | 0.033 | 620 | 0.00029 | 3.8 | 470 | 0.0081 |
| Acetone | 0.24 | | | 31,000 | 0.0000079 | 0.14 | | | 140,000 | 0.0000010 | 0.14 | | | 140,000 | 0.0000010 | 3.2 | 62,000 | 0.000051 |
| Acrolein | 0.031 | | | 0.35 | 0.087 | 0.018 | | | 1.5 | 0.012 | 0.018 | | | 1.5 | 0.012 | 0.6 | 6.9 | 0.092 |
| **Total Unit 2** | | | **0.68** | | **0.09** | | | **0.015** | | **0.012** | | | **0.033** | | **0.012** | | | **0.10** |
| **Total Source (Unit 1 and Unit 2)** | | | **4.8** | | **0.69** | | | **0.087** | | **0.057** | | | **0.19** | | **0.057** | | | **1.0** |
| *Existing Facility Risk Action Level* | | | *50* | | *5* | | | *50* | | *5* | | | *50* | | *5* | | | *5* |

**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 | 269 | 38 | 5 | 3 | 10 | 3 | 50 |
| Level 2 | 81 | 12 | 2 | 1 | 3 | 1 | 10 |
| Level 3 | 5 | 0.7 | 0.09 | 0.06 | 0.2 | 0.06 | 1 |
| | | | | | | | |
| *Risk Action Level[b]* | *50* | *5* | *50* | *5* | *50* | *5* | *5* |
| | | | | | | | |

Note:

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

# APPENDIX A

## Authoritative Sources of Toxicity Reference Values

### A.1 Chronic Values

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

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

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

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

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

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

Acute TRVs are only for non-carcinogenic effects. If no short-term toxicity values were available from the above authoritative 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, ATSDR's intermediate MRL for vinylidene chloride is 79 µg/m$^3$. The chronic noncancer value from IRIS is 200 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 vinylidene chloride.

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

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

Where:

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

# APPENDIX 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 340-245-8040 Table 4. The development of each adjustment factor is discussed below. None of the adjustment factors are appropriate or necessary for acute RBCs because of the short period of exposure being considered.

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 represents someone who is present 8 hr/day, 250 days/yr (5 days/week for 50 weeks):

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

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

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

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

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

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

#### 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-Hodgkin's 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 Table B-1 to the TRVs in OAR 340-245-8030 Table 3 using the following equations to calculate residential risk-based concentrations (RBCs) in OAR 340-245-8040 Table 4.

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

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

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

If multipathway or early-life considerations are not relevant for a 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.

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

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

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

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

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

If multipathway or early-life considerations are not relevant for a 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 = Acute risk-based concentration (µg/m$^3$)

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

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

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

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

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

**Notes**:

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

b <u>Additional adjustment factors:</u>
childNRAFnc = Non-residential adjustment factor, noncancer, child = 4.4.
workerNRAFnc = Non-residential adjustment factor, noncancer, worker = 4.4.
Chronic RBCs are based on continual exposure to residents for 70 years. The adjustment for non-resident exposure is:
(24 hours/day / 8 hours/day) x (365 days/year / 250 days/year) = 4.4
childNRAFc = Non-residential adjustment factor, child, cancer = 26
For carcinogenic effects to children, the non-residential exposure duration assumption is 12 years (infant through elementary school), resulting in a childNRAFc value of:
(70 years / 12 years) x (365 days/year / 250 days/year) x (24 hours/day / 8 hours/day) = 26
workerNRAFc = Non-residential adjustment factor, adult worker, cancer = 12
The adjustment for non-resident worker exposure working for 25 years is:
(70 years / 25 years) x (365 days/year / 250 days/year) x (24 hours/day / 8 hours/day) = 12

c MPAF = multipathway adjustment factor. Sources of multipathway adjustment factors:
South Coast Air Quality Management District, Permit Application Package "M", March 2016, Table 8-1.
South Coast Air Quality Management District, Facility Prioritization Procedures for AB 2588

Program, Nov. 2016, Table 3.
Chemicals for which there are MPAFs are considered persistent, bioaccumulative and toxic substances.

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

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

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

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

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

i Multipathway adjustment factors are for PCDDs.

# APPENDIX 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-Hodgkin's 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 (250 days/year) and exposure time (8 hours/day), are already accounted for in the non-residential adjustment factor. The nonresidential ELAF is the ratio of early-life exposure to general exposure for the same duration.

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

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

The default ELAF values are applied to the list of chemicals with early-life adjustments in Table B-1, 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-Hodgkin's lymphoma. EPA determined that TCE was carcinogenic by a mutagenic mode of action for kidney cancer (renal cell carcinoma). Accordingly, age-dependent adjustment factors should be used to evaluate early-life exposure to TCE for this endpoint, presumed to be initiated by a mutagenic mode of action. However, EPA did not determine that there is a mutagenic mode of action for the other two

cancer endpoints. The precise method for calculating RBCs for TCE is to use slope factors and IURs for each cancer endpoint, determine an RBC for kidney cancer using ADAFs, determine RBCs for liver cancer and non-Hodgkin's lymphoma without assuming early-life exposure, and combine the individual endpoint RBCs to get a comprehensive RBC using the following equation:

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

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

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

**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 $4.4 \times 10^{-6}$ risk per $\mu g/m^3$ for adult exposure, and $8.8 \times 10^{-6}$ risk per $\mu g/m^3$ for adult/child exposure. The Air Toxics Science Advisory Committee chose to use the adult/child IUR in developing an ambient benchmark concentration for vinyl chloride. Because 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 0.114 µg/m$^3$ by 2 to get a non-early-life TRV of 0.228 µg/m$^3$.

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

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

$$= \quad 0.228\ \mu g/m^3$$

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

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

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

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

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

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

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

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

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

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

**Table C-1**
**Toxic Air Contaminant 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, 2018.
b) Although not explicitly included in EPA's list, EPA states that carcinogenic PAHs with a relative potency factor relating the toxicity to the slope factor for benzo[a]pyrene should also be evaluated for early-life exposure.
c) Of the three cancer endpoints considered in the development of the inhalation unit risk (IUR) factor for TCE (kidney cancer, liver cancer, and non-Hodgkin's lymphoma), EPA determined that TCE was carcinogenic by a mutagenic mode of action for kidney cancer (renal cell carcinoma), but not the other endpoints. Age-dependent adjustment factors should be used to evaluate early-life exposure to TCE for kidney cancer, but not the other endpoints.
d) EPA has a specific method for evaluating early-life exposure to vinyl chloride, as presented in EPA's Integrated Risk Information System (www.epa.gov/iris).

**Table 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]<br>nonesidential[d] | 2<br>2 | 4<br>4 | 10<br>6 | 54<br>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 ($mg/cm^2 \cdot event$) | 0.2 | 0.2 | 0.07 | 0.07 |
| SA ($cm^2$)[b] | 2,690 | 2,690 | 6,032 | 6,032 |
| IRA ($m^3/d$)[b] | 10 | 10 | 20 | 20 |

**Notes:**

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

# Appendix D

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

### D.1 Introduction

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

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

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

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

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

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

The Risk Based Concentrations for total dioxins and furans, treated as 2,3,7,8-TCDD TEQ, are $3 \times 10^{-8}$ $\mu g/m^3$ for carcinogenic effects, and $4 \times 10^{-5}$ $\mu g/m^3$ for non-carcinogenic effects. These protective concentrations were obtained using the 1996 OEHHA Inhalation Risk Unit value of 38 per $\mu g/m^3$ for 2,3,7,8-TCDD, and the OEHHA RfC of $4 \times 10^{-5}$ $\mu g/m^3$, respectively.

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

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

### D.3 Polychlorinated Biphenyls (PCBs)

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

#### D.3.1 Total PCBs

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

#### D.3.2 Dioxin-Like PCB Congeners

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

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

Just as with the evaluation of dioxins and furans, the Risk Based Concentrations for the sum of the 12 dioxin-like PCBs, treated as 2,3,7,8-TCDD TEQ, are $3 \times 10^{-8}$ $\mu g/m^3$ for carcinogenic effects, and $4 \times 10^{-5}$ $\mu g/m^3$ for non-carcinogenic effects. These protective concentrations were obtained using the 1996 OEHHA Inhalation Risk Unit value of 38 per $\mu g/m^3$ for 2,3,7,8-TCDD, and the OEHHA RfC of $4 \times 10^{-5}$ $\mu g/m^3$, respectively.

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

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

## D.4 Polycyclic Aromatic Hydrocarbons

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

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

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

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

## D.5 References

Ecology (Washington Department of Ecology). 2007. Evaluating the toxicity and assessing the carcinogenic risk of environmental mixtures using toxic equivalency factors.

Minnesota Department of Health (MDOH). 2014. Guidance for evaluating the cancer potency of polycyclic aromatic hydrocarbon (PAH) mixtures in environmental samples. February 8.

U.S. EPA (Environmental Protection Agency). 2017. Toxicological review of benzo(a)pyrene. January. Posted on Integrated Risk Information System website https://cfpub.epa.gov/ncea/iris2/chemicalLanding.cfm?substance_nmbr=136.

U.S. EPA. . 2010a. Recommended toxicity equivalence factors (TEFs) for human health risk assessments of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin and dioxin-like compounds. EPA/100/R 10/005. December.

U.S. EPA. 2010b. Development of a relative potency factor (RPF) approach for polycyclic aromatic hydrocarbon (PAH) mixtures. EPA/615/R-08/012A. February.

U.S. EPA. 2009. Technical Assistance Document for the National Air Toxics Trends Stations Program, Revision 2. Prepared for U.S. EPA by Eastern Research Group, Inc., Morrisville, NC. April 1.

U.S. EPA. 2000. Supplementary guidance for conducting health risk assessment of chemical mixtures. EPA/630/R-00/002. August.

van den Berg, M; Birnbaum, L; Bosveld, AT; et al. 1998. Toxic equivalency factors (TEFs) for PCBs, PCDDs, PCDFs for humans and wildlife. Environ Health Perspect 106(12): 775-792.

van den Berg, M; Birnbaum, LS; Denison, M: et al. 2006. The 2005 World Health Organization re-evaluation of human and mammalian toxic equivalency factors for dioxins and dioxin-like compounds. Toxicol Sci 93(2): 223-241.

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

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

Note

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

**Table D-2: Dioxin-Like PCB Congeners and Related TEFs**

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

Note

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

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

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

Notes:

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

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

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

[d] Values were obtained from an External Review Draft version of EPA's 2010 *Development of a relative potency factor (RPF) approach for polycyclic aromatic hydrocarbon (PAH) mixtures*. Although this document is not supposed to be cited or quoted, the Air Toxics Science Advisory Committee considers this information to be the best and most current science available on this topic. A portion of the TEFs represent the average range of Potency Equivalency Factors provided in this document.

NA – not listed in either EPA 2010b nor by MnDOH, but is a NATTS-requested PAH.

# APPENDIX E

## Compilation of Target Organs for Toxic Air Contaminants

DEQ intends to create a table summarizing target organs for the list of toxic air contaminants in OAR 340-245-8040 Table 4.

# APPENDIX F

## Procedures for Updating Risk Assessments

If you have a new or modified toxic emission unit and are required to update your risk assessment, you have options ranging from simple scaling of emission rates to simple modeling to re-doing the risk assessment.

### F.1 Simple Scaling of Risk Estimates

If the only changes to your facility are to emission rates for an existing source, and not to any physical characteristics such as stack height, exit velocity, for other factors that will affect air dispersion modeling calculations, then you can conduct a simple scaling of emissions and risk to complete an updated risk assessment. An example of this type of change would be using different chemicals in a production process that otherwise remains unchanged. To conduct this evaluation, estimate the mass emission rate of all chemicals (many of which may not change), and use the following formula to calculate an emission scaled risk estimate (ESRE) for each revised emission unit:

$$ESRE = \frac{\sum \frac{ER_{i,revised}}{RBC_i}}{\sum \frac{ER_{i,existing}}{RBC_i}} Risk_{existing}$$

Where:
ESRE = Emission scaled risk estimate
$ER_{i,revised}$ = Emission rate for chemical i for revised emission unit
$ER_{i,existing}$ = Emission rate for chemical i for existing emission unit
$RBC_i$ = Risk-based concentration for chemical i
$Risk_{existing}$ = Total excess cancer risk or hazard index from risk assessment for emission unit

Calculate ESRE separately for excess cancer risk, chronic noncancer hazard index, and acute noncancer hazard index.

### F.2 Independent Modeling of New Emission Source

If your facility is adding a new source of emissions and not modifying existing emissions, the risk from the new source can be evaluated independently using any risk assessment level (1 to 4), and the results added to the existing risk estimates. The calculated new potential risks should be presented separately for cancer, chronic noncancer, and acute noncancer risks. This approach should be relatively easy to implement, but it may overestimate risk if the results of a lower level modeling calculation are added to the existing calculated risk assuming that maximum concentrations occur at the same location.

### F.3 Revised Comprehensive Modeling and Risk Assessment

Under some circumstances where source emissions are changing, it may be most appropriate to redo the air dispersion modeling and risk assessment. This will likely be the case if there are changes to physical characteristics such as stack height, exit velocity, for other factors that will affect air dispersion modeling calculations. In this case, the best approach would be to conduct comprehensive new modeling to replace the air concentrations used in the risk assessment.