Oregon Department of Environmental Quality
July 14, 2017

Notice of Proposed Rulemaking

Air Toxics Benchmarks Review

Table of Contents

Introduction 2
Overview 5
Statement of need 9
Rules affected, authorities, supporting documents 10
Fee Analysis 12
Statement of fiscal and economic impact 13
Federal relationship 16
Land use 17
Stakeholder and public involvement 18
Public notice and hearings 21
Draft Rules - With Edits Highlighted 24
Draft Rules – With Edits Incorporated 43
Supporting documents 61

Introduction

DEQ invites public input on proposed permanent rule amendments to chapter 340 of the Oregon Administrative Rules.

Background

When the Oregon State Air Toxics Program was adopted by the EQC in 2003, DEQ was required to form, with the agreement of the EQC, an Air Toxics Science Advisory Committee. The purpose of the ATSAC is to provide DEQ, and in Lane County the Lane Regional Air Protection Agency, with advice on the state air toxics program that is scientifically sound, independent, balanced, useful, and timely. By rule, the Air Toxics Science Advisory Committee is convened every five years to review any new toxicity information available for the 52 chemicals assigned Ambient Benchmark Concentrations. The latest iteration of the ATSAC performed benchmark reviews from December 2014 through June 2017.

By rule, the Air Toxics Science Advisory Committee is convened every five years to review any new toxicity information available for the 52 chemicals assigned Ambient Benchmark Concentrations and to review toxicity information for any new chemicals requested by DEQ. Most recently, the ATSAC was reconvened in December 2014 and met periodically through March 2017 to review the Ambient Benchmark Concentrations for which new toxicity information had become available since approximately 2006. The ATSAC reviewed new toxicity information that was available for 32 of the Ambient Benchmark Concentration and then recommended revisions to 23 benchmarks and retention of 9 benchmarks.

DEQ proposal

DEQ proposes changes to OAR 340, division number 246 that will make revisions to 23 standing Ambient Benchmark Concentrations, and add new benchmarks for phosgene, n-propyl bromide, and styrene. DEQ is also proposing some minor plain language edits and to add which statutes the rules are implementing. DEQ recommends that the EQC adopt the 23 revised and three new air toxics benchmark concentrations along with the plain language edits, as presented in the draft rules, into the current administrative rules. These rules are being proposed for adoption as required under Division 246 of the Oregon Air Toxics Program.

More information

Information about this rulemaking is on this rulemaking's web page: Air Toxics Benchmarks Review 2017

Public Hearings

DEQ will hold a public hearing on this rulemaking at the time and location below.

Anyone can attend the public hearing, either in person or through a webinar or teleconference. The details are listed below

9:30 a.m., August 17, 2017
700 NE Multnomah St., Suite 600
Third Floor, Conference Room A
Portland, OR 97232

Teleconference/Webinar Information
Call-in number: 888-278-0296
Participant ID code: 8040259

How to join webinar or teleconference: Webinar instructions

What will happen next?
DEQ will consider all comments received and include a written response to comments in a staff report DEQ will submit to the Environmental Quality Commission. DEQ may modify the rule proposal based on the comments.

Present proposal to the EQC
Proposed rules only become effective if the Environmental Quality Commission adopts them. DEQ plans to present the proposed rules to the commission for a decision at its meeting on Nov. 1-2, 2017.

How to comment on this rulemaking proposal

DEQ is asking for public comment on the proposed rules. Anyone can submit comments and questions about this rulemaking. A person can submit comments through an online web page, by regular mail or at the public hearing.

Comment deadline
DEQ will only consider comments on the proposed rules that DEQ receives by 4 p.m., on October 2, 2017.

Submit comment online
Air Toxics Benchmarks Review 2017 Comment Page

Note for public university students:
ORS 192.501(29) allows Oregon public university and OHSU students to protect their university email addresses from disclosure under Oregon's public records law. If you are an Oregon public university or OHSU student you may omit your email address when you complete the online form to submit a comment.

By mail
Oregon DEQ
Attn: Sue MacMillan
700 NE Multnomah Street, Suite 600
Portland, OR 97232

At hearing
August 17, 2017 (Thursday), 9:30 a.m., Conference Room A, Third Floor, 700 N.E. Multnomah Street, Portland, Oregon 97232.

Sign up for rulemaking notices
Get email updates about future DEQ rulemaking by signing up through GovDelivery or on the rulemaking web site.

Accessibility information
You may review copies of all documents referenced in this announcement at:

Oregon Department of Environmental Quality
700 NE Multnomah Street, Suite 600
Portland, OR, 97232

To schedule a review of all websites and documents referenced in this announcement, call Sue MacMillan, Portland, at 503-229-6458 (800-452-4011, ext. 5622 toll-free in Oregon).

Please notify DEQ of any special physical or language accommodations or if you need information in large print, Braille or another format. To make these arrangements, contact DEQ, Portland, at 503-229-5696 or call toll-free in Oregon at 1-800-452-4011, ext. 5696; fax to 503-229-6762; or email to deqinfo@deq.state.or.us. Hearing impaired persons may call 711.

Overview

Short summary

DEQ proposes the Oregon Environmental Quality Commission adopt the proposed rules that contain revisions to 23 standing Ambient Benchmark Concentrations, and new benchmarks for phosgene, n-propyl bromide, and styrene. DEQ is also proposing some minor plain language edits and to add what statutes are being implemented by the rules.

Brief history

In October 2003, the EQC adopted the framework for Oregon's Air Toxics Program (OAR 340-246-0010 to -0230). In September 2004, DEQ first convened the ATSAC to assist in determining ambient benchmark concentrations for a list of air toxics. At that time, the ATSAC began looking at a list of 262 air toxics obtained from Oregon's 1999 emissions inventory, which became available in 2003. Based on certain criteria, including whether a compound had been emitted at one pound per year or more and whether toxicity information was available for the compound, the ATSAC identified 164 air toxics for prioritization. As stated in rule (OAR 340-246-0090), prioritization includes the relative toxicity or potency of a pollutant; the degree of exposure and number of people at risk; the impact to sensitive human populations; the number and degree of predicted ambient benchmark exceedances; and the potential to cause harm through pollutant persistence and bioaccumulation. Through the prioritization process, the ATSAC identified 52 air toxics for which ambient benchmark concentrations needed to be developed.

By rule, the Air Toxics Science Advisory Committee is convened every five years to review any new toxicity information available for the 52 chemicals assigned Ambient Benchmark Concentrations and to review toxicity information for any new chemicals requested by DEQ. Most recently, the ATSAC was reconvened in December 2014 and met periodically through March 2017 to review the Ambient Benchmark Concentrations for which new toxicity information had become available since approximately 2006. The ATSAC reviewed new toxicity information that was available for 32 of the Ambient Benchmark Concentration and then recommended revisions to 23 benchmarks and retention of 9 benchmarks.

Of the 23 revisions to benchmarks and recommendations for three new benchmarks being proposed, only four garnered substantial attention during the ATSAC meetings, either in terms of time spent on review of toxicity information by the ATSAC and/or concerns expressed by the audience. These include diesel particulate matter, lead, polycyclic aromatic hydrocarbons, and trichloroethylene. Details for each are provided below.

Diesel Particulate Matter

The benchmark for this air toxic has received high interest since 2006 as well as during this current iteration of the ATSAC. The bulk of six ATSAC meetings were devoted to this topic, and a comprehensive review of recent scientific literature was conducted. The ATSAC explored whether any new research or analysis would suggest changing DEQ's existing

ambient benchmark value for diesel particulate. The ATSAC ultimately concluded that there is no new decisive information that would warrant revising the current diesel benchmark. The ATSAC recommended that DEQ retain its current ambient benchmark concentration for Diesel Particulate Matter. A concise description of this work is provided for Diesel Particulate Matter in Attachment B, and comprehensive documentation of the ATSAC's work on Diesel Particulate Matter can be provided upon request.

Lead

The ATSAC spent considerable time exploring new toxicity information for lead. There is significant scientific information indicating that there is no safe concentration of lead to which people can be exposed without harm, particularly in regard to diminished cognitive abilities in children. Exposure to lead *in utero* and during the early years of life causes impairment of neural development and decreased mental functional capacity. In later years, associations with impaired academic performance and Attention Deficit Hyperactive Disorder have been reported, and these effects persist into adulthood. Impaired neurodevelopment and functioning is the most sensitive endpoint, and these effects have been demonstrated in multiple studies, so there is a high confidence in a causal relationship.

However, there is also significant uncertainty in the health science, making it difficult to establish a definitive protective threshold for lead as an air toxic. The EPA has chosen to continue to use its National Ambient Air Quality Standard of 0.15 microgram per cubic meter as its threshold for protecting public health from lead exposure. The ATSAC recommended that DEQ use that same value as the benchmark for lead as an air toxic based on the rationale presented below.

The Clean Air Act directs that NAAQS be set at a level with an adequate margin of safety to protect the most sensitive groups in the population. In the case of lead, the relevant sensitive population group is children under five years of age, including fetuses. The ATSAC recognizes that the current federal lead health standard does provide a comprehensive level of public health protection.

The ATSAC discussed that identifying a lead threshold other than zero will leave a portion of the population to face some elevated health risk (i.e., as related to a potential decrease of one to three IQ points). The committee also pointed out that lead levels monitored in air in Oregon are much lower than the benchmark for lead. In addition, some lead is produced naturally, so the concentration of lead in air will never be zero. Using a benchmark of 0.15 ug/m^3 would thus help the DEQ to identify trouble spots of highest concern.

The ATSAC felt that the NAAQS value of 0.15 ug/m^3 should be retained as the benchmark for lead, as this level represents the best available scientific and technical evidence. The committee acknowledged that their recommendation of a benchmark for lead is based on the current state of the available science, and that lead should be evaluated again as the available health science advances.

Polycyclic aromatic hydrocarbons

The benchmark for Polycyclic Aromatic Hydrocarbons is based on the summation of toxicity-adjusted concentrations of 32 individual PAHs. However, the ATSAC has recommended changing the underlying list of individual PAHs to include new PAHs that are more directly related to air exposure, and to remove some of the PAHs from the original list, resulting in a proposed list of 26 individual PAHs.
The toxicity adjustment mentioned earlier includes the application of specific Toxicity Equivalency Factors which are specific to each individual PAH, and which adjust to align with the toxicity of one of the most-toxic and best-researched PAH, benzo(a)pyrene. In addition to the recommended change to the benchmark for total PAHs and to the underlying list of individual PAHs, new Toxicity Equivalency Factors were proposed. The proposed revised list of individual PAHs and their respective Toxicity Equivalency Factors are presented in Table B-1 of Attachment B.

Trichloroethylene

The benchmark for this air toxic was discussed at length by the ATSAC. Originally, the previous ATSAC chose a cancer-based Unit Risk Estimate value of 2×10^{-6} per ug/m^3, which resulted in a benchmark of 0.5 ug/m^3 for Trichloroethylene. The Unit Risk Estimate value was published in 1990 by the California Office of Environmental Health Hazard Assessment. However, in 2011, new toxicity information became available from EPA indicating that a cancer-based Unit Risk Estimate value of 5×10^{-6} per ug/m^3 was preferable, resulting in a proposed revised benchmark for TCE of 0.2 ug/m^3. In addition, the new toxicity information indicated that the non-cancer effects of TCE were of great concern, due to a few studies that indicated that pregnant mothers exposed during a 21-day period of their gestation were likely to produce fetuses or infants with fetal heart malformation. However, because the proposed benchmark based on TCE cancer effects is set at a lower (more stringent) concentration than would have been required for non-cancer effects, the proposed benchmark is considered protective of both cancer and non-cancer chronic effects of TCE.

In addition, ambient benchmark concentrations were recommended by the ATSAC for three new chemicals: phosgene, n-propyl bromide, and styrene. Toxicity information for chronic exposure to selenium, although discussed by the ATSAC, was inadequate, and the ATSAC declined to make a recommendation for this chemical.

This proposal is limited in scope to adopting revised and new ambient benchmark concentrations as administrative rules. The ambient benchmarks proposed in this rulemaking will function within Oregon's existing air toxics program as goal reference values. Three separate actions could be triggered under the Toxics Program if monitoring data shows ambient air toxics concentration to be above a benchmark. These include:

a) The development of emission reduction strategies for specific emission source categories (like diesel engines or woodstoves),
b) Evaluation of a major industrial facility under the "Safety-Net" program, or
c) Community planning work in select geographic areas.

Currently, DEQ and the Oregon Health Authority are developing a risk-based air toxics permitting program called Cleaner Air Oregon. Under the proposed framework, Ambient

Benchmark Concentrations could be used as a first-tier preference in a longer list of enforceable risk-based concentrations.

Regulated parties

Because the Ambient Benchmarks Concentrations are used as goals by the DEQ to prioritize resources based on air toxics exceedances, no parties are directly regulated by the proposed rule changes.

The proposed amendment of Oregon Administrative Rule 340-246-0090 to incorporate revised and new ambient benchmarks into rule does not change the regulated parties.

Request for other options

During the public comment period, DEQ requests public comment on whether to consider other options for achieving the rules' substantive goals while reducing any identified negative economic impact on business.

Statement of need

What need would the proposed rule address?

Since 2006, ambient benchmark concentrations have been used by DEQ to evaluate the degree of human health risks associated with emissions of 52 chemicals. DEQ uses these benchmarks to assess the levels of air toxics in Oregon, and to prioritize which problems to address first. Although only used as goals, these benchmarks are utilized by emissions sources and the public to better understand what kinds of human health risks are potentially associated with monitored or modeled emissions of air toxics. In addition, there is a regulatory requirement for the Ambient Benchmark Concentrations to be reviewed and updated as necessary every five years; this proposed rule will serve to meet this requirement.

How would the proposed rule address the need?

Because toxicity information for chemicals is constantly changing due to new study results becoming available, the ATSAC's review of the toxicity information behind the current ambient benchmark concentrations insures that the most up-to-date and scientifically defensible toxicity information is used to generate or revise the benchmarks. Making sure that these benchmarks reflect the current, best science allows DEQ and other entities to utilize the benchmarks with confidence in making technical and policy decisions around levels of toxics in air.

How will DEQ know the rule addressed the need?

Updating the ambient benchmark concentrations provides DEQ and external users of the benchmarks confidence that the benchmarks reflect the best, most-current science, as recommended by the ATSAC. The proposed rules provide updated values for existing benchmarks and new benchmark values for n-propyl bromide, phosgene, and styrene.

Rules affected, authorities, supporting documents

Lead division
Solutions

Program or activity
Air Toxics

Chapter 340 action

Amend - OAR

340-246-0090	340-246-0010	340-246-0030	340-246-0050	340-246-0070
340-246-0110	340-246-0130	340-246-0150	340-246-0170	340-246-0190
340-246-0210				

Statutory authority - ORS
468.020, 468.065, 468.035, 468A.010(1), 468A.015

Statute implemented - ORS
468A.015 468A.025

Legislation

Not applicable

Documents relied on for rulemaking

DEQ relied on ATSAC's consensus recommendations for updates to the Ambient Benchmark Concentrations. The ATSAC relied upon credible information from a variety of peer-reviewed and technical documents, the most important being those from the:

Document title	Document location
USEPA Integrated Risk Information System (IRIS) cancer and non-cancer toxicity values	https://www.epa.gov/iris

California Office of Environmental Health Hazard Assessment (OEHHA) cancer and non-cancer toxicity values	https://www.arb.ca.gov/toxics/healthval/contable.pdf
Agency for Toxic Substances and Disease Registry cancer and non-cancer toxicity values	https://www.atsdr.cdc.gov/mrls/mrllist.asp
ATSAC meeting minutes	http://www.oregon.gov/deq/aq/air-toxics/Pages/ATSAC-Meetings.aspx

Fee Analysis

This rulemaking does not involve fees.

Statement of fiscal and economic impact

Fiscal and Economic Impact

Adoption of the proposed benchmarks will not, in and of itself, have a direct fiscal or economic impact. But, because their adoption will move the air toxics program itself forward, their adoption is expected to eventually cause some indirect impacts. However, there could be indirect impacts dependent upon future decisions that are unquantifiable at the time of benchmark adoption.

The proposed rules are limited to adopting ambient benchmarks as administrative rules. The ambient benchmarks proposed in this rulemaking will function within Oregon's existing air toxics program as triggers for, and clean air goals within, other facets (Geographic, Local Air Toxics Emissions Reduction Planning, Source Category Strategy, Safety Net) of the program. DEQ requests public comment on whether other options should be considered for achieving the rule's substantive goals while reducing negative economic impact of the rule on business. The substantive goal of this rulemaking is to establish ambient reference values (Air Benchmark Concentration) for the purposes of identifying, evaluating, and addressing air toxics problems. The benchmarks are only a single component of the overall air toxics program. Any specific implementation, compliance, enforcement, financial, land use, or resource issues are expected to be associated with the existing overall program and subsequent community emission reduction planning, and not with adoption of these ambient benchmarks.

Statement of Cost of Compliance

DEQ is unable to quantify the impact at this time because we do not have available data to make this estimate. Please refer to the Fiscal and Economic Impact section above for further details.

State agencies

Adopting ambient benchmarks as administrative rules will have no impact on FTE's, revenues, or expenses.

Local governments

No direct or indirect fiscal or economic impacts.

Public

No direct or indirect fiscal or economic impacts.

Large businesses - businesses with more than 50 employees

No direct or indirect fiscal or economic impacts.

Small businesses – businesses with 50 or fewer employees

No direct or indirect fiscal or economic impacts.

a. Estimated number of small businesses and types of businesses and industries with small businesses subject to proposed rule.

Not applicable.

b. Projected reporting, recordkeeping and other administrative activities, including costs of professional services, required for small businesses to comply with the proposed rule.

Not applicable.

c. Projected equipment, supplies, labor and increased administration required for small businesses to comply with the proposed rule.

Not applicable.

d. Describe how DEQ involved small businesses in developing this proposed rule.

ATSAC meetings were open to the public, including representatives of small businesses. Any comments made during the audience participation period were recorded and considered.

Advisory committee

DEQ used the Air Toxics Science Advisory Committee to establish the ambient benchmark concentrations to be adopted as administrative rules. DEQ did not appoint an advisory committee to ascertain fiscal and economic impacts from this rule, because there are not expected to be any.

The primary assumption is that any fiscal and economic impacts will result from the operation of Oregon's air toxics program, which relies on the benchmarks, and not from simply adopting ambient benchmarks as administrative rules.

Housing cost

As ORS 183.534 requires, DEQ evaluated whether the proposed rules would have an effect on the development cost of a 6,000-square-foot parcel and construction of a 1,200-square-foot detached, single-family dwelling on that parcel. DEQ determined that this proposed rulemaking will have no effect on the cost of development of a 6,000 square foot parcel and the construction of a 1,200 square foot detached single family dwelling on that parcel.

Federal relationship

Relationship to federal requirements

The proposed rules are not different from or in addition to federal requirements. The EPA does not currently have uniform ambient benchmark concentrations for use as reference and planning values. The proposed rule changes will allow DEQ to address threats to public health from toxic air pollutants that remain after the technology-based strategies of the federal air toxics program. Although not a requirement, these changes are consistent with implementing the Federal Integrated Urban Air Toxics Strategy. They are not expected to affect existing federal standards for evaluating criteria pollutants.

Land use

Land-use considerations

In adopting new or amended rules, ORS 197.180 and OAR 340-018-0070 require DEQ to determine whether the proposed rules significantly affect land use. If so, DEQ must explain how the proposed rules comply with state wide land-use planning goals and local acknowledged comprehensive plans.

Under OAR 660-030-0005 and OAR 340 Division 18, DEQ considers that rules affect land use if:

- The statewide land use planning goals specifically refer to the rule or program, or
- The rule or program is reasonably expected to have significant effects on:
    - Resources, objectives or areas identified in the statewide planning goals, or
    - Present or future land uses identified in acknowledged comprehensive plans

To determine whether the proposed rules involve programs or actions that affect land use, DEQ reviewed its Statewide Agency Coordination plan, which describes the DEQ programs that have been determined to significantly affect land use. DEQ considers that its programs specifically relate to the following statewide goals:

Goal	Title
5	Open Spaces, Scenic and Historic Areas, and Natural Resources
6	Air, Water and Land Resources Quality
9	Ocean Resources
11	Public Facilities and Services
16	Estuarial Resources

Statewide goals also specifically reference the following DEQ programs:

- Nonpoint source discharge water quality program – Goal 16
- Water quality and sewage disposal systems – Goal 16
- Water quality permits and oil spill regulations – Goal 19

Determination

DEQ determined that these proposed rules do not affect land use under OAR 340-018-0030 or DEQ's State Agency Coordination Program

Stakeholder and public involvement

Advisory committee

DEQ used the Air Toxics Science Advisory Committee to establish the ambient benchmarks to be adopted as administrative rules.

Background

When the Oregon State Air Toxics Program was adopted by the EQC in 2003, DEQ was required to form, with the agreement of the EQC, an Air Toxics Science Advisory Committee. The purpose of the ATSAC is to provide DEQ, and in Lane County the Lane Regional Air Protection Agency, with advice on the state air toxics program that is scientifically sound, independent, balanced, useful, and timely. A seven-member ATSAC was formed in September 2004. Members were selected for their relevant air toxics experience, as required by rule, in: toxicology; environmental science or engineering; risk assessment, epidemiology and biostatistics, public health medicine; and air pollution modeling, monitoring, meteorology, or engineering.

This same set of requirements was used to select the member of the 2014-2017 ATSAC. The present iteration of the ATSAC included three members from academia, two members from the consulting sector, and two members from state government, including a staff person from the Oregon Health Authority. Please refer to Attachment C for details on each member's experience. The committee's web page is located at: http://www.oregon.gov/deq/aq/air-toxics/Pages/ATSAC.aspx

The committee members were:

Air Toxics Science Advisory Committee	
Name	**Representing**
Dr. Bill Lambert	Oregon Health Sciences University, public health medicine, toxicology
Dr. Dean Atkinson	Portland State University, air pollution monitoring, modeling, meteorology, engineering
Dr. Kent Norville	Air Sciences Inc., air pollution monitoring, modeling, meteorology, engineering

Dr. Dave Farrer	Oregon Health Authority,, toxicology, environmental science
Dr. Bruce Hope	Former toxicologist for DEQ and for CH2MHill, toxicology, environmental science
Dr. David Stone	Oregon State University, environmental science, toxicology, air pollution monitoring and modeling
Mr. Max Hueftle	Lane Regional Air Protection Agency, environmental science, air pollution monitoring, modeling, meteorology, and engineering

Meeting notifications

To notify people about the advisory committee's activities, DEQ:

- Sent GovDelivery bulletins, a free e-mail subscription service, to the following lists:
    - Air Toxics State-wide
- Added advisory committee announcements to DEQ's calendar of public meetings at DEQ Calendar.

Committee discussions

The ATSAC was convened specifically to perform comprehensive review of relevant information from recognized authoritative bodies and the scientific literature in order to recommend to DEQ ambient benchmark concentrations protective of human health for a large list of air toxics. The current iteration of the ATSAC spent 12 three-hour meetings discussing toxicity information for 32 of the 52 standing benchmarks, and identifying benchmarks for three new chemicals. In addition, individual ATSAC members conducted their own individual reviews of assigned materials and prepared summaries to the present to the committee during meeting times. Minutes for all ATSAC meetings can be accessed at http://www.oregon.gov/deq/aq/air-toxics/Pages/ATSAC-Meetings.aspx .

EQC prior involvement

DEQ shares general rulemaking information with EQC through the monthly Director's Report.

DEQ shared information about this rulemaking with the EQC through the Director's Report as an informational item on the November 7, 2014 EQC agenda. At this time, Director Pederson informed the EQC of the seven appointees to the ATSAC, with two recommended alternates.

During the April 15, 2015 EQC meeting, Director Pederson informed the EQC through a Director's Report presented as an informational item that a new committee member, Dr. David Stone, would replace a departing committee member, Dr. Kim Anderson.

Public notice and hearing

Public notice

DEQ provided notice of the proposed rulemaking and rulemaking hearing on July 14, 2017 by:

- On July 14, 2017 Filing notice with the Oregon Secretary of State for publication in the Aug. 1, 2017 Oregon Bulletin;
- Notifying the EPA by mail;
  Posting the Notice, Invitation to Comment and Draft Rules on the web page for this rulemaking, located at: Air Toxics Benchmarks Review 2017;
- Emailing approximately 9,976 interested parties on the following DEQ lists through GovDelivery:
  - Rulemaking
  - Air Toxics State-wide
  - DEQ Public Notices
  - Portland Air Toxics Solutions
  - Cleaner Air Oregon Regulatory Overhaul
  - Dry Cleaner Program Advisory Committee Updates
- Emailing the following key legislators required under ORS 183.335:
  - Senator Michael Dembrow, Chair, Senate Environment and Natural Resources Committee
  - Representative Ken Helm, Chair, House Energy and Environment Committee
- Emailing advisory committee members,
- Postings on Twitter and Facebook
- Posting on the DEQ event calendar: DEQ Calendar

Public hearings

DEQ plans to hold one public hearing. The details are listed below. Anyone can attend the hearing in person, or by webinar or teleconference.

DEQ will consider all written and oral comments received at the hearings listed below before completing the draft rules. DEQ will summarize all comments and respond to comments in the Environmental Quality Commission staff report.

Hearing 1	
Date	Aug. 17, 2017
Time	9:30 a.m.
Street Address	Conference Room A, Third Floor, 700 NE Multnomah St., Suite 600
City	Portland, OR 97232

Presiding Officer	DEQ Staff
Staff Presenter	Sue MacMillan
Call-in Phone Number	888-278-0296
Participant ID	8040259
Instructions on how to access webinar and teleconference	Webinar instructions

How to comment on the proposed rules:

Submit comment online

Air Toxics Benchmarks Review 2017 Comment Page

Note for public university students:

ORS 192.501(29) allows Oregon public university and OHSU students to protect their university email addresses from disclosure under Oregon's public records law. If you are an Oregon public university or OHSU student you may omit your email address when you complete the online form to submit a comment.

By mail

Oregon DEQ
Attn: Sue MacMillan
700 NE Multnomah St., Suite 600
Portland, OR 97232

At the hearing

Close of public comment period

The comment period will close 4 p.m. on October 2, 2017.

Accessibility Information

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Oregon Department of Environmental Quality
700 NE Multnomah Street
Portland, OR, 97204

To schedule a review of all websites and documents referenced in this announcement, call Sue MacMillan, Portland, 503-229-6458 (800-452-4100 toll-free in Oregon).

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Key to Identifying Changed Text:
~~Deleted Text~~
New/inserted text
~~Text deleted from one location~~ - and moved to another location

DEPARTMENT OF ENVIRONMENTAL QUALITY

DIVISION 246

Oregon State Air Toxics Program

340-246-0010

Policy and Purpose

The purpose of Oregon's state air toxics program is to address threats to public health and the environment from toxic air pollutants that remain after implementing the state delegated technology-based strategies of the federal air toxics program. Oregon's program meets the goals of the federal Urban Air Toxics Strategy by using a community-based effort that focuses on geographic areas of concern. It also addresses cases of elevated health risks from unregulated air toxics emissions at stationary sources and source categories of air toxics emissions.

Stat. Auth.: ORS 468.035, 468A.010(1), 468A.015
Stats. Implemented: ORS 468A.015, 468A.025
Hist.: DEQ 15-2003, f. & cert. ef. 11-3-03

340-246-0030

Definitions

The definitions in OAR 340-200-0020, 340-218-0030, 340-244-0030 and this rule apply to this division. If the same term is defined in this division and elsewhere, the definition in this division applies.

(1) "Air toxics" means those pollutants known or suspected to cause cancer or other serious health effects, including but not limited to "hazardous air pollutants" or "HAPs" listed by the EPA ~~pursuant to~~under section 112(b) of the Federal Clean Air Act.

(2) "Ambient benchmark" means the concentration of an air toxic in outdoor air that would result in an excess lifetime cancer risk level of one in a million (1 x 10-6) or a non-cancer hazard quotient of one.

(3) "Bio-accumulation" means the net accumulation of a substance by an organism as a result of uptake from all routes of exposure (e.g., ingestion of food, intake of drinking water, direct contact, or inhalation).

(4) "Geographic area" means an area identified by ~~the Department~~DEQ where air toxics concentrations are estimated or measured at levels that exceed ambient benchmark concentrations.

(5) "Hazard quotient" means the ratio of the potential exposure to a single air toxic to the reference concentration for that pollutant. If the hazard quotient is calculated to be less than or equal to 1, then no adverse health effects are expected as a result of exposure. If the hazard quotient is greater than 1, then adverse health effects are possible.

(6) "High priority geographic area" means an area identified by ~~the Department~~DEQ where air toxics concentrations are estimated or measured at levels that exceed ambient benchmark concentrations and pose excess cancer risk above ten in a million, or non-cancer risk above a hazard quotient of one with the potential for serious adverse health effects.

(7) "Public receptor" means any outdoor area where members of the public have unrestricted access, including but not limited to residences, institutions (e.g. schools, hospitals), industrial, commercial, or office buildings, parks, recreational areas, public lands, streets or sidewalks.

(8) "Reference concentration" means an estimate of a continuous exposure or a daily exposure to the human population (including sensitive populations) that is likely to be without an appreciable risk of adverse non-cancer effects during a lifetime. The reference concentration can be derived from various types of human or animal data, with uncertainty factors generally applied to reflect limitations of the data used.

(9) "Sensitive human populations" means humans with increased susceptibility to the adverse effects of air toxics, including humans in prenatal or postnatal periods of development.

(10) "Source" means:

(a) An activity conducted by a person at a point, area, on-road mobile, or off-road mobile operation that emits air toxics; or

(b) Any building, structure, facility, installation or combination thereof that emits or is capable of emitting air contaminants to the atmosphere, is located on one or more contiguous or adjacent properties and is owned or operated by the same person or by persons under common control. The term includes all pollutant emitting activities that belong to a single major industrial group (i.e., that have the same two-digit code) as described in the **Standard Industrial Classification Manual**, (U.S. Office of Management and Budget, 1987) or that support the major industrial group.

(11) "Source Category" means:

(a) A source or group of sources that emit air toxics due to the use of the same or similar processes, including commercial, residential, public or private processes, which as a group can reduce air toxics emissions by employing similar control or prevention strategies or;

(b) All the pollutant emitting activities that belong to the same industrial grouping (i.e., that have the same two-digit code) as described in the **Standard Industrial Classification Manual**, (U.S. Office of Management and Budget, 1987).

(12) "Toxics Best Available Retrofit Technology", or "TBART" means an air toxics emissions limitation based on the maximum degree of reduction of air toxics, determined on a case-by-case basis, that is feasible taking into consideration:

(a) What has been achieved in practice for that source category, or for similar processes or emissions;

(b) Energy and non-air quality health or environmental impacts; and

(c) Economic impacts, including the costs of changing existing processes or equipment or adding equipment or controls to existing processes and equipment. Such limitation may be based on a design, equipment, work practice or other operational standard, or combination thereof.

[Publications: Publications referenced are available from the agency.]

Stat. Auth.: ORS 468.035, 468A.010(1), 468A.015
Stats. Implemented: ORS 468A.015, 468A.025
Hist.: DEQ 15-2003, f. & cert. ef. 11-3-03

340-246-0050

Pollution Prevention

The Environmental Quality Commission encourages the use of pollution prevention for all sources of air toxics statewide. The Commission encourages use of the following hierarchy to reduce air toxics:

(1) Modify the process, raw materials, or product to reduce the quantity and toxicity of air contaminants generated;

(2) Capture and reuse air contaminants;

(3) Treat to reduce the quantity and toxicity of air contaminants released; or

(4) Otherwise control air toxics emissions.

Stat. Auth.: ORS 468.035, 468A.010(1), 468A.015
Stats. Implemented: ORS 468A.015, 468A.025
Hist.: DEQ 15-2003, f. & cert. ef. 11-3-03

340-246-0070,

Air Toxics Science Advisory Committee

(1) Purpose. The Commission recognizes the many scientific uncertainties associated with the effects of air toxics, and the continuing development of new information in this field. An Air Toxics Science Advisory Committee (ATSAC), will advise ~~the Department~~DEQ, and in its jurisdiction, the Lane Regional Air Pollution Authority, on technical issues and evaluation of the state air toxics program. The ATSAC will provide advice on the technical aspects of risk assessment. It will not provide risk management or policy recommendations. The ATSAC will perform the following functions:

(a) Review ambient benchmarks for the state air toxics program;

(b) Advise ~~the Department~~DEQ on developing a risk assessment methodology to be used in the Safety Net Program in OAR 340-246-0190 (5) and (6);

(c) Advise ~~the Department~~DEQ on selecting sources for the Safety Net program. The ATSAC will evaluate potential Safety Net sources identified by ~~the Department~~DEQ to determine whether they qualify for the Safety Net Program, as specified in OAR 340-246-0190 through 0230;

(d) Evaluate overall progress in reducing emissions of and exposure to air toxics by considering trends in emissions and ambient concentrations of air toxics. The ATSAC will periodically advise ~~the Department~~DEQ on air toxics program effectiveness and make technical recommendations for program development concerning the possible adverse environmental effects of air toxics and risk from exposure to multiple air toxics; and

(e) Provide advisory opinions on questions requiring scientific expertise, as requested by ~~the Department~~DEQ.

(2) Membership. The ATSAC will be composed of highly qualified members with experience relevant to air toxics. There will be at least five but no more than seven members. The following disciplines will be represented on the ATSAC:

(a) Toxicology;

(b) Environmental Science or Environmental Engineering;

(c) Risk Assessment;

(d) Epidemiology/Biostatistics;

(e) Medicine (Physician) with training or experience in Public Health; and

(f) Air Pollution Modeling, Monitoring, Meteorology or Engineering.

(3) Appointment. ~~The Department~~DEQ's Air Quality Division Administrator will nominate potential members to the Director. Before making these nominations, the Administrator will develop a list of candidates by consulting with government, public, and private organizations involved in work relevant to air toxics. The Director will appoint ATSAC members with concurrence by the Commission.

(4) Term. Air Toxics Science Advisory Committee members will serve a three-year term. Initial terms will be staggered for continuity and transfer of work so that members of the first ATSAC may serve more or less than three years.

(5) Operation.

(a) No member may have an actual or potential conflict of interest, as those terms are defined by ORS 244.020.

(b) The ATSAC will meet as necessary.

(6) Procedures, Bylaws, and Decision-making Process. At a minimum, the ATSAC will observe the procedures specified below. The ATSAC will develop other necessary procedures and bylaws in consultation with ~~the Department~~DEQ.

(a) Final decisions must be made by a quorum of members, based on consensus when possible. If consensus is not possible, decisions will be made by majority vote with a quorum present.

(b) If necessary, ~~the Department~~DEQ may obtain a facilitator to assist the ATSAC.

(c) The bylaws will include provisions for removing a member for cause, with concurrence by the Commission.

Stat. Auth.: ORS 468.035, 468A.010(1), 468A.015
Stats. Implemented:
Hist.: DEQ 15-2003, f. & cert. ef. 11-3-03

340-246-0090,

Ambient Benchmarks for Air Toxics

(1) Purpose. Ambient benchmarks are concentrations of air toxics that serve as goals in the Oregon Air Toxics Program. They are based on human health risk and hazard levels considering sensitive populations. Ambient benchmarks are not regulatory standards, but reference values by which air toxics problems can be identified, addressed and evaluated. ~~The Department~~DEQDEQ will use ambient benchmarks as indicated in these rules, to implement the Geographic, Source

Category, and Safety Net Programs. Ambient benchmarks set by the procedures described in this rule apply throughout Oregon, including that area within the jurisdiction of the Lane Regional Air Protection Agency. Ambient benchmarks are subject to public notice and comment before adoption by the Environmental Quality Commission as administrative rules.

(2) Establishing Ambient Benchmarks

(a) ~~The Department~~DEQDEQ will consult with the ATSAC to prioritize air toxics for ambient benchmark development. Highest priority air toxics are those that pose the greatest risk to public health.

(b) To prioritize air toxics, ~~the Department~~DEQDEQ will apply the criteria described in OAR 340-246-0090(2)(c) to modeling, monitoring, and emissions inventory data.

(c) Ambient benchmark prioritization criteria will include at least the following:

(A) Toxicity or potency of a pollutant;

(B) Exposure and number of people at risk;

(C) Impact on sensitive human populations;

(D) The number and degree of predicted ambient benchmark exceedances; and

(E) Potential to cause harm through persistence and bio-accumulation.

(d) ~~The Department~~DEQDEQ will develop ambient benchmarks for proposal to the ATSAC based upon a protocol that uses reasonable estimates of plausible upper-bound exposures that neither grossly underestimate nor grossly overestimate risks.

(e) Within three months of the first meeting of the ATSAC, ~~the Department~~DEQDEQ will propose ambient benchmark concentrations for the highest priority air toxics for review by the ATSAC. ~~The Department~~DEQDEQ will propose additional and revised air toxics ambient benchmarks for review by the ATSAC based on the prioritization criteria in OAR 340-246-0090(2)(c). Once the ATSAC has completed review of each set of proposed ambient benchmarks, ~~the Department~~DEQDEQ will, within 60 days, begin the process to propose ambient benchmarks as administrative rules for adoption by the Environmental Quality Commission.

(f) If ~~the Department~~DEQDEQ is unable to propose ambient benchmarks to the ATSAC by the deadlines specified in OAR 340-246-0090(2)(e), the ATSAC will review the most current EPA ambient benchmarks. If EPA ambient benchmarks are not available, the ATSAC will review the best available information from other states and local air authorities.

(g) The ATSAC will consider proposed ambient benchmarks and evaluate their adequacy for meeting risk and hazard levels, considering human health, including sensitive human

populations, scientific uncertainties, persistence, bio-accumulation, and, to the extent possible, multiple exposure pathways. The ATSAC will conduct this review consistent with the criteria in OAR 340-246-0090(2)(c) and (d). The ATSAC will report these findings to ~~the Department~~DEQDEQ. If the ATSAC unanimously disagrees with ~~the Department~~DEQ~~'s~~ DEQ's recommendation, ~~the Department~~DEQDEQ will re-consider and re-submit its recommendation at a later date.

(h) The ATSAC will complete review of and report findings on each set of ambient benchmarks as ~~expeditiously~~ quickly as possible, but no later than 12 months after ~~the Department~~DEQDEQ has proposed them. If the ATSAC is unable to complete review of ambient benchmarks within 12 months after ~~the Department~~DEQDEQ's proposal, ~~the Department~~DEQDEQ will initiate rulemaking to propose ambient benchmarks.

(i) ~~The Department~~DEQDEQ will review all ambient benchmarks at least every five years and, if necessary, propose revised or additional ambient benchmarks to the ATSAC. At its discretion, ~~the Department~~DEQDEQ may review and propose a benchmark for review by the ATSAC at any time when new information is available.

(3) Ambient Benchmarks. Benchmark concentrations are in units of micrograms of air toxic per cubic meter of ambient air, on an average annual basis. The Chemical Abstract Service Registry Number (CASRN) is shown in parentheses.

(a) The ambient benchmark for acetaldehyde (75-07-0) is 0.45 micrograms per cubic meter.

(b) The ambient benchmark for acrolein (107-02-8) is 0.35~~02~~ micrograms per cubic meter.

(c) The ambient benchmark for acrylonitrile (107-13-1) is 0.01 micrograms per cubic meter.

(d) The ambient benchmark for ammonia (7664-41-7) is ~~200~~500 micrograms per cubic meter.

(e) The ambient benchmark for arsenic (7440-38-2) is 0.0002 micrograms per cubic meter.

(f) The ambient benchmark for benzene (71-43-2) is 0.13 micrograms per cubic meter.

(g) The ambient benchmark for beryllium (7440-41-7) is 0.0004 micrograms per cubic meter.

(h) The ambient benchmark for 1,3-butadiene (106-99-0) is 0.03 micrograms per cubic meter.

(i) The ambient benchmark for cadmium and cadmium compounds (7440-43-9) is 0.0006 micrograms per cubic meter.

(j) The ambient benchmark for carbon disulfide (75-15-0) is 800 micrograms per cubic meter.

(k) The ambient benchmark for carbon tetrachloride (56-23-5) is 0.2~~07~~ micrograms per cubic meter.

(l) The ambient benchmark for chlorine (7782-50-5) is 0.1~~2~~ micrograms per cubic meter.

(m) The ambient benchmark for chloroform (67-66-3) is 300~~98~~ micrograms per cubic meter.

(n) The ambient benchmark for chromium, hexavalent (18540-29-9) is 0.00008 micrograms per cubic meter.

(o) The ambient benchmark for cobalt and cobalt compounds (7440-48-4) is 0.1 micrograms per cubic meter.

(p) The ambient benchmark for 1,4-dichlorobenzene (106-46-7) is 0.09 micrograms per cubic meter.

(q) The ambient benchmark for 1,3-dichloropropene (542-75-6) is 0.25 micrograms per cubic meter.

(r) The ambient benchmark for diesel particulate matter (none) is 0.1 micrograms per cubic meter. The benchmark for diesel particulate matter applies only to such material from diesel-fueled internal combustion sources.

(s) The ambient benchmark for dioxins and furans (1746-01-6) is 0.00000003 micrograms per cubic meter. The benchmark for dioxin is for total chlorinated dioxins and furans expressed as 2,3,7,8-TCDD toxicity equivalents.

(t) The ambient benchmark for ethyl benzene (100-41-4) is 0.4 micrograms per cubic meter.

(u) The ambient benchmark for ethylene dibromide (106-93-4) is 0.002 micrograms per cubic meter.

(v) The ambient benchmark for ethylene dichloride (107-06-2) is 0.04 micrograms per cubic meter.

(w) The ambient benchmark for ethylene oxide (75-21-8) is 0.0003 ~~0.01~~ micrograms per cubic meter.

(x) The ambient benchmark for formaldehyde (50-00-0) is ~~3~~ 0.2 micrograms per cubic meter.

(y) The ambient benchmark for n-hexane (110-54-3) is 700~~0~~ micrograms per cubic meter.

(z) The ambient benchmark for hydrogen chloride (7647-01-0) is 20 micrograms per cubic meter.

(aa) The ambient benchmark for hydrogen cyanide (74-90-8) is 0.8~~9~~ micrograms per cubic meter.

(bb) The ambient benchmark for ~~hydrogen~~ fluoride anion (7664-39-3) is 13~~4~~ micrograms per cubic meter.

(cc) The ambient benchmark for lead and lead compounds (7439-92-1) is 0.15 micrograms per cubic meter.

(dd) The ambient benchmark for manganese and manganese compounds (7439-96-5) is 0.09 micrograms per cubic meter.

(ee) The ambient benchmark for elemental mercury (7439-97-6) is 0.3 micrograms per cubic meter.

(ff) The ambient benchmark for methyl bromide (74-83-9) is 5 micrograms per cubic meter.

(gg) The ambient benchmark for methyl chloride (74-87-3) is 90 micrograms per cubic meter.

(hh) The ambient benchmark for methyl chloroform (71-55-6) is ~~1000~~ 5,000 micrograms per cubic meter.

(ii) The ambient benchmark for methylene chloride (75-09-2) is ~~2.1~~ 100 micrograms per cubic meter.

(jj) The ambient benchmark for naphthalene (91-20-3) is 0.03 micrograms per cubic meter.

~~(kk) The ambient benchmark for nickel refinery dust (7440-02-0) is 0.004 micrograms per cubic meter.~~

~~(ll) The ambient benchmark for nickel subsulfide (12035-72-2) is 0.002 micrograms per cubic meter.~~

~~(mm) The ambient benchmark for soluble nickel compounds (various) is 0.05 micrograms per cubic meter, where soluble nickel compounds may include any or all of the following: nickel acetate (373-02-4), nickel chloride (7718-54-9), nickel carbonate (3333-39-3), nickel carbonyl (13463-39-3), nickel hydroxide (12054-48-7), nickelocene (1271-28-9), and nickel sulfate (7786-81-4).~~

(kk) The benchmark for soluble nickel compounds (various) is 0.01 micrograms per cubic meter, where soluble nickel compounds include nickel acetate (373-20-4), nickel chloride (7718-54-9), nickel carbonate (3333-39-3), nickel carbonyl (13463-39-3), nickel hydroxide (12054-48-7), nickelocene 1271-28-9), nickel sulfate 7786-81-4), nickel sulfate hexahydrate 10101-97-0), nickel nitrate hexahydrate (13478-00-7), and nickel carbonate hydroxide (12607-70-4).

(ll) The ambient benchmark for insoluble nickel compounds (various) is 0.004 micrograms per cubic meter, where insoluble nickel compounds include nickel subsulfide (12035-72-2), nickel oxide (1313-99-1), nickel sulfide (11113-75-0), and nickel metal (7440-02-0).

(~~nn~~mm) The ambient benchmark for phosphine (7803-51-2) is ~~0.3~~ 0.8 micrograms per cubic meter.

(~~oo~~nn) The ambient benchmark for phosphoric acid (7664-38-2) is 10 micrograms per cubic meter.

(~~pp~~oo) The ambient benchmark for total (as the sum of congeners) polychlorinated biphenyls (1336-36-3) is 0.01 micrograms per cubic meter.

(~~qq~~pp) The ambient benchmark for total polycyclic aromatic hydrocarbons (none) is ~~0.0009~~ 0.002 micrograms per cubic meter, where total polycyclic aromatic hydrocarbons are the sum of the toxicity equivalency factor (with respect to benzo(a)pyrene (50-32-8)) adjusted concentrations for all of the following individual 26 polycyclic aromatic hydrocarbons:~~-~~ 5-methylchrysene (3697-24-3); 6-nitrochrysene (7496-02-8); acenaphthene (83-32-9); acenaphthylene (208-96-8); anthanthrene (191-26-4); anthracene (120-12-7); benz(a)anthracene (56-55-3); benzo(a)pyrene (50-32-8); benzo(b)fluoranthene (205-99-6); benzo(c)fluoranthene (243-17-4); benzo(e)pyrene (192-97-2); benzo(g,h,i)perylene (191-24-2); benzo(j)fluoranthene (205-82-3); benzo(k)fluoranthene (207-08-9); chrysene (218-01-9); cyclopenta(c,d)pyrene (27208-37-3); dibenz(a,h)anthracene (226-36-8); dibenzo(a,e)pyrene (192-65-4); dibenzo(a,h)pyrene (189-64-0); dibenzo(a,i)pyrene (189-55-9); dibenzo(a,l)pyrene (191-30-0); fluoranthene (206-44-0); fluorene (86-73-7); indeno(1,2,3-c,d)pyrene (193-39-5); phenanthrene (85-01-8); and pyrene (129-00-0).

~~benzo(a)anthracene (56-55-3), benzo(a)pyrene (50-32-8), benzo(b)fluoranthene (205-99-2), benzo(k)fluoranthene (207-08-9), carbazole (86-74-8), chrysene (218-01-9), dibenz(a,h)acridine (226-36-8), dibenz(a,h)anthracene (226-36-8), dibenz(a,j)acridine (224-42-0), 7H-dibenzo(c,g)carbazole (194-59-2), dibenzo(a,e)pyrene (192-65-4), dibenzo(a,i)pyrene (189-55-9), dibenzo(a,l)pyrene (191-30-0), 7,12-dimethylbenz(a)anthracene (57-97-6), 1,6-dinitropyrene (42397-64-8), 1,8-dinitropyrene (42397-65-9), indeno(1,2,3-c,d)pyrene (193-39-5), 3-methylcholanthrene (56-49-5), 5-methylchrysene (3697-24-3), 1-nitropyrene (5522-43-0), 2-nitrofluorene (607-57-8), 4-nitropyrene (59865-13-3), 5-nitroacenaphthene (607-87-9) 6-nitrochrysene (7496-02-8), acenaphthene (83-32-9), acenaphthylene (208-96-8), anthracene (120-12-7), benzo(g,h,i)perylene (191-24-2), fluoranthene (206-44-0), fluorene (86-73-7), phenanthrene (85-01-8), and pyrene (129-00-0)~~.

(~~rr~~qq) The ambient benchmark for tetrachloroethylene (127-18-4) is ~~35~~ 4 micrograms per cubic meter.

(~~ss~~rr) The ambient benchmark for toluene (108-88-3) is ~~400~~ 5,000 micrograms per cubic meter.

(ss~~tt~~) The ambient benchmark for 2,4- & 2,6 toluene diisocyanate, mixture (26471-62-5) is ~~0.07~~ 0.02 micrograms per cubic meter.

(tt~~uu~~) The ambient benchmark for trichloroethylene (79-01-6) is ~~0.5~~ 0.2 micrograms per cubic meter.

(uu~~vv~~) The ambient benchmark for vinyl chloride (75-01-4) is 0.1 micrograms per cubic meter.

(vv~~ww~~) The ambient benchmark for white phosphorus (7723-14-0) is ~~0.07~~ 9 micrograms per cubic meter.

(ww~~xx~~) The ambient benchmark for xylenes, mixed (1330-20-7) is ~~700~~ 200 micrograms per cubic meter.

(xx~~yy~~) The ambient benchmark for hydrogen sulfide (7783-06-4) is 2.0 micrograms per cubic meter.

(yy~~zz~~) T~~-~~he ambient benchmark for methanol (67-56-1) is 4,000 micrograms per cubic meter.

(zz) The ambient benchmark for phosgene (75-44-5) is 0.3 micrograms per cubic meter.

(aaa) The ambient benchmark for n-propyl bromide (106-94-5) is 0.5 micrograms per cubic meter.

(bbb) The ambient benchmark concentration for styrene (100-42-5) is 1,000 micrograms per cubic meter.

Stat. Auth.: ORS 468.035, 468A.010(1) & 468A.015
Stats. Implemented: ORS 468A.015, 468A.025
Hist.: DEQ 15-2003, f. & cert. ef. 11-3-03; DEQ 12-2006, f. & cert. ef. 8-15-06

340-246-0110

Source Category Rules and Strategies

(1) ~~The Department~~DEQ may identify the need for source category rules and strategies through the following methods:

(a) The emissions inventory, modeling or monitoring, shows air toxics emissions from point, area, or mobile sources associated with public health risk at public receptors;

(b) Development of a local air toxics reduction plan provides source category controls that could be effectively applied to sources existing in other parts of the state; or

(c) When implementing the Safety Net Program, ~~the Department~~DEQ establishes air toxics emissions reductions for a source and determines that there are other similar sources in the state to which the reductions ~~should~~ must apply.

(2) Subject to the requirements in this rule, the Lane Regional Air Pollution Authority is designated by the Commission as the agency responsible for implementing Source Category Rules and Strategies within its area of jurisdiction. The requirements and procedures contained in this rule must be used by the Regional Authority to implement Source Category Rules and Strategies unless the Regional Authority adopts superseding rules that are at least as restrictive as the rules adopted by the Commission.

(3) ~~The Department~~DEQ will consider the following criteria in determining whether to propose source category strategies under this division:

(a) Whether air toxics emissions from the source category are not, or will not, be addressed by other regulations or strategies, including emissions reduction requirements under the Geographic Program (OAR 340-246-0130 through 340-246-0170), or the Safety Net Program (OAR 340-246-0190 through 340-246-0230);

(b) Whether air toxic emissions from the source category can be effectively reduced through regulations or voluntary strategies; and

(c) Whether the source category contributes to ambient benchmark exceedances at public receptors statewide, in multiple geographic areas, or in multiple counties

Stat. Auth.: ORS 468.035, 468A.010(1), 468A.015
Stats. Implemented: ORS 468A.015, 468A.025
Hist.: DEQ 15-2003, f. & cert. ef. 11-3-03

340-246-0130

Geographic Program (0130 through 0170)

(1) Purpose. The Geographic Program addresses emissions from multiple sources of air toxics. It requires prioritizing and selecting geographic areas of concern, forming a local advisory committee, developing a specific local plan to control air toxics, a public participation and comment process, EQC adoption or approval, implementing reduction strategies, and periodically evaluating the effectiveness by ~~the Department~~DEQ.

(2) Subject to the requirements in OAR 340-246-0130 through 0170, the Lane Regional Air Pollution Authority is designated by the Commission as the agency to implement the Geographic Program within its area of jurisdiction. The requirements and procedures contained in this rule ~~shall~~ must be used by the Regional Authority to implement the Geographic Program unless the Regional Authority adopts superseding rules which are at least as restrictive as state rules. The Regional Authority will address geographic areas as resources allow, considering the prioritization criteria in 340-246-0150.

Stat. Auth.: ORS 468.035, 468A.010(1), 468A.015
Stats. Implemented: ORS 468A.015, 468A.025
Hist.: DEQ 15-2003, f. & cert. ef. 11-3-03

340-246-0150

Prioritizing and Selecting Geographic Areas

(1) ~~The Department~~DEQ will prioritize geographic areas by considering the total cancer and non-cancer risk from air toxics to the population in the area, as indicated by:

(a) The number and degree of ambient benchmark exceedances;

(b) The toxicity or potency of air toxics exceeding ambient benchmarks;

(c) The level of exposure and number of people at risk in areas of concern;

(d) The presence of sensitive populations;

(e) The effectiveness of local control strategies; and

(f) To the extent known, the risk posed by multiple pollutants and pollutant mixtures.

(2) Not later than 18 months after the first set of benchmarks is adopted, ~~the Department~~DEQ will select the first geographic area for air toxics reduction planning. ~~The Department~~DEQ will base selection on representative monitoring compared to the ambient benchmark concentrations at public receptors. To the extent possible, geographic areas will be identified using monitoring data generated following EPA monitoring guidelines. Subsequent geographic areas will be selected after completion of monitoring. A geographic area is formally selected upon publication of a notice in the Oregon Secretary of State's Bulletin. Once an area is selected for air toxics reduction planning, it will retain the status of a selected geographic area until ~~the Department~~DEQ determines through an evaluation of data that a reduction plan is no longer necessary for the area to meet all air toxics ambient benchmarks.

(3) ~~The Department~~DEQ will first select for emissions reduction planning the high priority geographic areas, where concentrations of air toxics are more than ten times above the ambient benchmarks or above a hazard quotient of one with the potential for serious adverse health effects. ~~The Department~~DEQ will select all other geographic areas, where air toxics concentrations are above benchmarks, after air toxics emissions reduction plans have been approved for the high priority geographic areas.

(4) Geographic Area Boundaries. ~~The Department~~DEQ will establish general geographic area boundaries on a neighborhood or urban area scale. ~~The Department~~DEQ will consider feasibility of administration when setting the boundaries of a geographic area. In setting geographic area boundaries, ~~the Department~~DEQ will consider criteria including but not limited to the following:

(a) Areas of impact (where people are exposed);

(b) Population density;

(c) Areas of influence (where sources are located);

(d) Meteorology;

(e) Geography and topography;

(f) Including all air toxics exceeding ambient benchmarks; and

(g) Coordination with criteria pollutant boundaries for attainment of the National Ambient Air Quality Standards (NAAQS).

Stat. Auth.: ORS 468.035, 468A.010(1), 468A.015
Stats. Implemented: ORS 468A.015, 468A.025
Hist.: DEQ 15-2003, f. & cert. ef. 11-3-03

340-246-0170

Local Air Toxics Emissions Reduction Planning

(1) ~~The Department~~DEQ will develop air toxics reduction plans for selected geographic areas with the advice of local advisory committees. The main role of a local advisory committee is to consider air toxics reduction options and to recommend a specific air toxics reduction plan for their geographic area. The Director will appoint a local air toxics advisory committee.

(a) Local advisory committees will generally be composed of a balanced representation of members from affected local government, local health departments, the public, small businesses (50 or fewer employees), larger businesses (if present in the area), and interest groups represented in the area.

(2) Local Advisory Committee Tasks.

(a) Within 18 months of their first meeting, the committee will evaluate options for reducing emissions of air toxics that exceed ambient benchmarks, and recommend a local air toxics reduction plan to ~~the Department~~DEQ.

(b) ~~The Department~~DEQ may grant an extension of time to the local committee if requested by the committee, if ~~the Department~~DEQ believes the extension is technically justified and the committee is making reasonable progress in developing a local air toxics reduction plan.

(c) If the committee is unable to recommend a local air toxics reduction plan to ~~the Department~~DEQ within 18 months, or the date of an extension, ~~the Department~~DEQ will formulate a plan for the area within six months.

(d) ~~The Department~~DEQ and the local advisory committee will seek local government support for the proposed local air toxics emissions reduction plan.

(e) The local advisory committee will evaluate the plan's effectiveness as it is implemented and recommend changes to ~~the Department~~DEQ.

(f) At ~~the Department~~DEQ's request, the local advisory committee will reconvene to implement contingency planning and recommend contingency measures as specified by OAR 340-246-0170(4)(l).

(g) If the committee is unable to recommend contingency measures within 18 months, ~~the Department~~DEQ will formulate contingency measures for the area within 6 months.

(3) Public Notice, Comment, Approval and Adoption by the Environmental Quality Commission. ~~The Department~~DEQ will provide an opportunity for public notice and comment on proposed local emissions reduction plans. After the public notice and comment process is complete, ~~the Department~~DEQ will present local air toxics reduction plans to the Commission for approval, including adoption of appropriate administrative rules. The Environmental Quality Commission may delegate the approval of plans that do not contain administrative rules to the Director of ~~the Department~~DEQ.

(4) Elements of an Air Toxics Reduction Plan:

(a) Local air toxics reduction plans must focus on the air toxic or air toxics measured or modeled above the ambient benchmarks.

(b) Local air toxics reduction plans must be based on sound data analysis. This includes developing enhanced emissions inventory information for the local area using source-specific information to the extent possible. This may also include enhanced modeling and monitoring to better characterize ambient concentrations. Plans also must rely on sound analysis of the effectiveness and cost of air toxics emissions reduction options. Where needed to fill specific information gaps, ~~the Department~~DEQ may require air toxics emissions reporting for specific sources or source categories within the geographic area on a case-by-case basis.

(c) The emissions reduction goals for individual air toxics are ambient benchmarks in local air toxics reduction plans.

(d) Local air toxics reduction plans must be designed to reduce air toxics emissions in a timely manner.

(A) When feasible, local air toxics reduction plans will be designed to reach levels that are equal to or below ambient benchmark concentrations. Plans will be designed to achieve emissions reductions within ten years, beginning at the date the Commission approves the plan. Local plans must provide for the timeliest reductions possible for each air toxic exceeding ambient benchmarks.

(B) Local air toxics reduction plans must include specific three-year milestones that ~~the Department~~DEQ and the local advisory committee will evaluate every three years, in coordination with ~~the Department~~DEQ's air toxics emissions inventory update.

(e) Every three years, ~~the Department~~DEQ will assess the effectiveness of local plans and make recommendations for plan revision based on progress meeting milestones or new information. If ~~the Department~~DEQ finds lack of progress at year three, it will work with the local advisory committee to provide corrective measures. If ~~the Department~~DEQ finds lack of progress at year six and projects that ten-year goals in OAR 340-246-0170(4)(d)(A) will not be met, it will implement the contingency plan in 340-246-0170(4)(l). If at year nine ~~the Department~~DEQ projects that ten year goals in 340-246-0170(4)(d)(A) will not be met, it will work with the local advisory committee to propose and seek adoption of measures necessary to reach these goals.

(f) Local air toxics reduction plans must evaluate air toxics emissions from all types of sources, including point, area, and mobile sources. Plans must require emissions reductions from the most significant sources of air toxics. Mandatory emissions reduction strategies will be commensurate with source contributions, considering relative emissions, toxicity, technical feasibility, cost-effectiveness and equity.

(g) Local air toxics reduction plans must include strategies to reduce high concentrations of air toxics that are limited to smaller portions of a geographic area as well as pollutants causing public health risk throughout the area.

(h) Local air toxics reduction plans may include a variety of mandatory and voluntary approaches to reducing emissions of air toxics. Depending on the type of source, local air toxics

reduction plans may include public education, pollution prevention alternatives, economic incentives and disincentives, technical assistance and regulatory requirements.

(i) ~~The Department~~DEQ will ensure the opportunity for public involvement during the plan development process. This includes involving those affected by the air toxics emissions and those affected by the proposals to reduce air toxics emissions. Proposed local air toxics reduction plans must be available for public hearing and comment.

(j) Local air toxics reduction plans must be coordinated with other local, state, and federal requirements to the extent possible. This includes considerations of any ozone or particulate control requirements for the area, any federal standard applicable to sources in the area, any strategies that are federally pre-empted, and any impacts on water or land, such as water pollution or hazardous waste.

(k) Local air toxics reduction plans will include specific recommendations for developing ongoing emissions inventory or ambient air monitoring to track local trends in air toxics.

(l) Local air toxics reduction plans must include a contingency plan that will be implemented if evaluation at year six shows that an area is not meeting milestones and will not achieve the ten year goals established under OAR 340-246-0170(4)(d)(A). The contingency plan, like the original plan, must require emissions reductions from the most significant sources of air toxics. Mandatory emissions reduction strategies will be commensurate with source contributions, considering relative emissions, toxicity, technical feasibility cost-effectiveness and equity. Contingency plans must include but are not limited to:

(i) Re-evaluation of planning assumptions, such as emissions factors, motor vehicle data and background pollutants;

(ii) Evaluation of existing conditions and effectiveness of emissions reduction strategies, including reasons for success or failure; and

(iii) New or progressively more mandatory strategies that will be considered.

Stat. Auth.: ORS 468.035, 468A.010(1), 468A.015
Stats. Implemented: ORS 468A.015, 468A.025
Hist.: DEQ 15-2003, f. & cert. ef. 11-3-03

340-246-0190

Air Toxics Safety Net Program (0190 through 0230)

(1) The purpose of the Air Toxics Safety Net Program is to address human exposures at public receptors to air toxics emissions from stationary sources that are not addressed by other regulatory programs or the Geographic Program. It is the Commission's expectation that the Safety Net Program in OAR 340-246-0190 through 340-246-0230 will apply only rarely.

(2) Subject to the requirements contained in OAR 340-246-0190 through 340-246-0230, the Lane Regional Air Pollution Authority is designated by the Commission as the agency responsible for implementing the Air Toxics Safety Net Program within its area of jurisdiction. The requirements and procedures contained in this rule must be used by the Regional Authority

to implement the Air Toxics Safety Net Program unless the Regional Authority adopts superseding rules, which are at least as restrictive as the rules adopted by the Commission.

(3) Selection of Sources. ~~The Department~~DEQ will select a source for the Air Toxics Safety Net Program if all of the following criteria are met:

(a) ~~The Department~~DEQ has ambient monitoring information, gathered using appropriate EPA or other published international, national, or state standard methods that concentrations of air toxics have caused an exceedance of at least one ambient benchmark at a site representing expected human exposure to air toxics from the source at a public receptor in a location outside of the source's ownership or control.

(b) ~~The Department~~DEQ has information that the source's air toxics emissions alone have caused an exceedance of at least one ambient benchmark at a site representing expected human exposure to air toxics from the source at a public receptor, in a location outside of the source's ownership or control. This could be based on emissions inventory, modeling or other information.

(c) The source is not subject to or scheduled for a federal residual risk assessment under the federal Clean Air Act section 112(f)(2) through (6).

(d) The source is not subject to an emissions limit or control requirement imposed as the result of modeling or a risk assessment performed or required by ~~the Department~~DEQ prior to November 1, 2003 for the air toxics that exceed the ambient benchmarks.

(e) The source is located outside of a selected geographic area, as designated in OAR 340-246-0130 through 0170.

(4) Air Toxics Science Advisory Committee Review. Before requiring a source to conduct a source-specific risk assessment, ~~the Department~~DEQ will present its analysis to the ATSAC. Within 120 days, the ATSAC will review the analysis and make a finding. If the ATSAC concurs with ~~the Department~~DEQ or takes no action, ~~the Department~~DEQ may proceed ~~pursuant to~~under this rule. If the ATSAC objects, ~~the Department~~DEQ will not proceed until it receives concurrence from the Commission.

(5) Source-Specific Exposure Modeling and Risk Assessment. Upon written notification by ~~the Department~~DEQ, a source must conduct a risk assessment including exposure modeling for the air toxics measured at levels above ambient benchmarks. The source must use a risk assessment methodology provided by ~~the Department~~DEQ. This risk assessment will provide the basis for establishing air toxics emissions reductions or demonstrating that at public receptors in areas outside of a source's ownership or control, people are not being exposed to air toxics at levels that exceed the ambient benchmarks.

(6) Risk Assessment Methodology ~~The Department~~DEQ will provide guidance on the methods to be used. The risk assessment methodology will be developed in consultation with the ATSAC and will result in a protocol that:

(a) Uses reasonable estimates of plausible upper-bound exposures that neither grossly underestimate nor grossly overestimate risks;

(b) Considers the range of probabilities of risks actually occurring, the range of size of the populations likely to be exposed to the risk, and current and reasonably likely future land uses;

(c) Defines the use of high-end and central-tendency exposure cases and assumptions;

(d) Develops values associated with chronic exposure for carcinogens; and

(e) Addresses both carcinogenic and non-carcinogenic air toxics and allows for detailed exposure assessments to the extent possible.

(7) Review and Acceptance by ~~the Department~~DEQ ~~The Department~~DEQ will evaluate the risk assessment for adequacy and completeness before accepting the results. If the results demonstrate that the source is not causing human exposures to air toxics at levels that exceed the ambient benchmarks at public receptors, in areas outside the source's ownership or control, and ~~the Department~~DEQ has received concurrence from the ATSAC, ~~the Department~~DEQ will notify the source that air toxics emissions reductions will not be required ~~pursuant to~~under this rule.

Stat. Auth.: ORS 468.035, 468A.010(1), 468A.015
Stats. Implemented: ORS 468A.015, 468A.025
Hist.: DEQ 15-2003, f. & cert. ef. 11-3-03

340-246-0210

Safety Net Source Air Toxics Emissions Reductions

(1) Air Toxics Emissions Reduction Analysis:

(a) If source-specific exposure modeling and risk assessment show that the source is causing exceedances of ambient benchmarks at public receptors in areas outside the source's ownership or control, the source must perform an analysis showing how air toxics could be reduced to meet ambient benchmarks. ~~The Department~~DEQ and the safety net source will develop proposed air toxics emissions reduction measures based on modeling and, when available, monitoring information.

(b) As part of the air toxics emissions reduction analysis, the source will analyze pollution prevention options, and is encouraged to use the hierarchy stated in OAR 340-246-0050.

(2) Air Toxics Emissions Reduction Requirements:

(a) A safety net source emitting air toxics causing exposure resulting in excess lifetime cancer risk greater than one in a million (1x10-6) or a hazard quotient of one for non-carcinogens must, as soon as practicable but no later than three years after the effective date of the permit imposing such conditions, meet toxics best available retrofit technology (TBART) for each air toxic that exceeds an ambient benchmark.

(b) A safety net source may use a means of air toxics reduction, other than TBART, if it can demonstrate to ~~the Department~~DEQ that it will achieve a risk level at or below one in a million, or a hazard quotient at or below one, within three years of using the other means of air toxics emissions reductions.

(c) A safety net source emitting a carcinogenic air toxic causing excess lifetime cancer risk at or above one hundred in a million (1x10-4) must reduce its air toxic emissions to achieve a risk level below one hundred in a million as soon practicable but no later than one year after the effective date of the permit imposing such conditions.

(d) A safety net source emitting a non-carcinogenic air toxic at a level above a hazard quotient of one that ~~the Department~~DEQ finds to have a potential for causing very serious or irreversible adverse health effects must reduce its air toxic emissions below this level as soon practicable, but no later than one year after the effective date of the permit imposing such conditions.

(3) If a safety net source cannot reach a risk level at or below excess lifetime cancer risk of one in a million, or a hazard quotient at or below one in three years, even though it meets TBART, the TBART determination for the source will be subject to periodic review under this section until the source achieves a risk level at or below one in a million or a hazard quotient at or below one. Upon each renewal of the source's permit, TBART for the source must be reviewed, taking into consideration retrofit costs and the remaining useful life of controls installed or other measures taken to meet a prior TBART determination. Upon renewal of the source's permit, ~~the Department~~DEQ must include conditions requiring the source to meet TBART as determined for that permit renewal.

Stat. Auth.: ORS 468.035, 468A.010(1), 468A.015
Stats. Implemented: ORS 468A.015, 468A.025
Hist.: DEQ 15-2003, f. & cert. ef. 11-3-03

DEPARTMENT OF ENVIRONMENTAL QUALITY

DIVISION 246

Oregon State Air Toxics Program

340-246-0010, Policy and Purpose

The purpose of Oregon's state air toxics program is to address threats to public health and the environment from toxic air pollutants that remain after implementing the state delegated technology-based strategies of the federal air toxics program. Oregon's program meets the goals of the federal Urban Air Toxics Strategy by using a community-based effort that focuses on geographic areas of concern. It also addresses cases of elevated health risks from unregulated air toxics emissions at stationary sources and source categories of air toxics emissions.

Stat. Auth.: ORS 468.035, 468A.010(1), 468A.015
Stats. Implemented: ORS 468A.015, 468A.025
Hist.: DEQ 15-2003, f. & cert. ef. 11-3-03

340-246-0030, Definitions

The definitions in OAR 340-200-0020, 340-218-0030, 340-244-0030 and this rule apply to this division. If the same term is defined in this division and elsewhere, the definition in this division applies.

(1) "Air toxics" means those pollutants known or suspected to cause cancer or other serious health effects, including but not limited to "hazardous air pollutants" or "HAPs" listed by the EPA under section 112(b) of the Federal Clean Air Act.

(2) "Ambient benchmark" means the concentration of an air toxic in outdoor air that would result in an excess lifetime cancer risk level of one in a million (1 x 10-6) or a non-cancer hazard quotient of one.

(3) "Bio-accumulation" means the net accumulation of a substance by an organism as a result of uptake from all routes of exposure (e.g., ingestion of food, intake of drinking water, direct contact, or inhalation).

(4) "Geographic area" means an area identified by DEQ where air toxics concentrations are estimated or measured at levels that exceed ambient benchmark concentrations.

(5) "Hazard quotient" means the ratio of the potential exposure to a single air toxic to the reference concentration for that pollutant. If the hazard quotient is calculated to be less than or equal to 1, then no adverse health effects are expected as a result of exposure. If the hazard quotient is greater than 1, then adverse health effects are possible.

(6) "High priority geographic area" means an area identified by DEQ where air toxics concentrations are estimated or measured at levels that exceed ambient benchmark concentrations and pose excess cancer risk above ten in a million, or non-cancer risk above a hazard quotient of one with the potential for serious adverse health effects.

(7) "Public receptor" means any outdoor area where members of the public have unrestricted access, including but not limited to residences, institutions (e.g. schools, hospitals), industrial, commercial, or office buildings, parks, recreational areas, public lands, streets or sidewalks.

(8) "Reference concentration" means an estimate of a continuous exposure or a daily exposure to the human population (including sensitive populations) that is likely to be without an appreciable risk of adverse non-cancer effects during a lifetime. The reference concentration can be derived from various types of human or animal data, with uncertainty factors generally applied to reflect limitations of the data used.

(9) "Sensitive human populations" means humans with increased susceptibility to the adverse effects of air toxics, including humans in prenatal or postnatal periods of development.

(10) "Source" means:

(a) An activity conducted by a person at a point, area, on-road mobile, or off-road mobile operation that emits air toxics; or

(b) Any building, structure, facility, installation or combination thereof that emits or is capable of emitting air contaminants to the atmosphere, is located on one or more contiguous or adjacent properties and is owned or operated by the same person or by persons under common control. The term includes all pollutant emitting activities that belong to a single major industrial group (i.e., that have the same two-digit code) as described in the **Standard Industrial Classification Manual**, (U.S. Office of Management and Budget, 1987) or that support the major industrial group.

(11) "Source Category" means:

(a) A source or group of sources that emit air toxics due to the use of the same or similar processes, including commercial, residential, public or private processes, which as a group can reduce air toxics emissions by employing similar control or prevention strategies or;

(b) All the pollutant emitting activities that belong to the same industrial grouping (i.e., that have the same two-digit code) as described in the **Standard Industrial Classification Manual**, (U.S. Office of Management and Budget, 1987).

(12) "Toxics Best Available Retrofit Technology", or "TBART" means an air toxics emissions limitation based on the maximum degree of reduction of air toxics, determined on a case-by-case basis, that is feasible taking into consideration:

(a) What has been achieved in practice for that source category, or for similar processes or emissions;

(b) Energy and non-air quality health or environmental impacts; and

(c) Economic impacts, including the costs of changing existing processes or equipment or adding equipment or controls to existing processes and equipment. Such limitation may be based on a design, equipment, work practice or other operational standard, or combination thereof.

[Publications: Publications referenced are available from the agency.]

Stat. Auth.: ORS 468.035, 468A.010(1), 468A.015
Stats. Implemented: ORS 468A.015, 468A.025
Hist.: DEQ 15-2003, f. & cert. ef. 11-3-03

340-246-0050, Pollution Prevention

The Environmental Quality Commission encourages the use of pollution prevention for all sources of air toxics statewide. The Commission encourages use of the following hierarchy to reduce air toxics:

(1) Modify the process, raw materials, or product to reduce the quantity and toxicity of air contaminants generated;

(2) Capture and reuse air contaminants;

(3) Treat to reduce the quantity and toxicity of air contaminants released; or

(4) Otherwise control air toxics emissions.

Stat. Auth.: ORS 468.035, 468A.010(1), 468A.015
Stats. Implemented: ORS 468A.015, 468A.025
Hist.: DEQ 15-2003, f. & cert. ef. 11-3-03

340-246-0070, Air Toxics Science Advisory Committee

(1) Purpose. The Commission recognizes the many scientific uncertainties associated with the effects of air toxics, and the continuing development of new information in this field. An Air Toxics Science Advisory Committee (ATSAC), will advise DEQ, and in its jurisdiction, the Lane Regional Air Pollution Authority, on technical issues and evaluation of the state air toxics program. The ATSAC will provide advice on the technical aspects of risk assessment. It will not

provide risk management or policy recommendations. The ATSAC will perform the following functions:

(a) Review ambient benchmarks for the state air toxics program;

(b) Advise DEQ on developing a risk assessment methodology to be used in the Safety Net Program in OAR 340-246-0190 (5) and (6);

(c) Advise DEQ on selecting sources for the Safety Net program. The ATSAC will evaluate potential Safety Net sources identified by DEQ to determine whether they qualify for the Safety Net Program, as specified in OAR 340-246-0190 through 0230;

(d) Evaluate overall progress in reducing emissions of and exposure to air toxics by considering trends in emissions and ambient concentrations of air toxics. The ATSAC will periodically advise DEQ on air toxics program effectiveness and make technical recommendations for program development concerning the possible adverse environmental effects of air toxics and risk from exposure to multiple air toxics; and

(e) Provide advisory opinions on questions requiring scientific expertise, as requested by DEQ.

(2) Membership. The ATSAC will be composed of highly qualified members with experience relevant to air toxics. There will be at least five but no more than seven members. The following disciplines will be represented on the ATSAC:

(a) Toxicology;

(b) Environmental Science or Environmental Engineering;

(c) Risk Assessment;

(d) Epidemiology/Biostatistics;

(e) Medicine (Physician) with training or experience in Public Health; and

(f) Air Pollution Modeling, Monitoring, Meteorology or Engineering.

(3) Appointment. DEQ's Air Quality Division Administrator will nominate potential members to the Director. Before making these nominations, the Administrator will develop a list of candidates by consulting with government, public, and private organizations involved in work relevant to air toxics. The Director will appoint ATSAC members with concurrence by the Commission.

(4) Term. Air Toxics Science Advisory Committee members will serve a three-year term. Initial terms will be staggered for continuity and transfer of work so that members of the first ATSAC may serve more or less than three years.

(5) Operation.

(a) No member may have an actual or potential conflict of interest, as those terms are defined by ORS 244.020.

(b) The ATSAC will meet as necessary.

(6) Procedures, Bylaws, and Decision-making Process. At a minimum, the ATSAC will observe the procedures specified below. The ATSAC will develop other necessary procedures and bylaws in consultation with DEQ.

(a) Final decisions must be made by a quorum of members, based on consensus when possible. If consensus is not possible, decisions will be made by majority vote with a quorum present.

(b) If necessary, DEQ may obtain a facilitator to assist the ATSAC.

(c) The bylaws will include provisions for removing a member for cause, with concurrence by the Commission.

Stat. Auth.: ORS 468.035, 468A.010(1), 468A.015
Stats. Implemented:
Hist.: DEQ 15-2003, f. & cert. ef. 11-3-03

340-246-0090, Ambient Benchmarks for Air Toxics

(1) Purpose. Ambient benchmarks are concentrations of air toxics that serve as goals in the Oregon Air Toxics Program. They are based on human health risk and hazard levels considering sensitive populations. Ambient benchmarks are not regulatory standards, but reference values by which air toxics problems can be identified, addressed and evaluated. DEQ will use ambient benchmarks as indicated in these rules, to implement the Geographic, Source Category, and Safety Net Programs. Ambient benchmarks set by the procedures described in this rule apply throughout Oregon, including that area within the jurisdiction of the Lane Regional Air Protection Agency. Ambient benchmarks are subject to public notice and comment before adoption by the Environmental Quality Commission as administrative rules.

(2) Establishing Ambient Benchmarks

(a) DEQ will consult with the ATSAC to prioritize air toxics for ambient benchmark development. Highest priority air toxics are those that pose the greatest risk to public health.

(b) To prioritize air toxics, DEQ will apply the criteria described in OAR 340-246-0090(2)(c) to modeling, monitoring, and emissions inventory data.

(c) Ambient benchmark prioritization criteria will include at least the following:

(A) Toxicity or potency of a pollutant;

(B) Exposure and number of people at risk;

(C) Impact on sensitive human populations;

(D) The number and degree of predicted ambient benchmark exceedances; and

(E) Potential to cause harm through persistence and bio-accumulation.

(d) DEQ will develop ambient benchmarks for proposal to the ATSAC based upon a protocol that uses reasonable estimates of plausible upper-bound exposures that neither grossly underestimate nor grossly overestimate risks.

(e) Within three months of the first meeting of the ATSAC, DEQ will propose ambient benchmark concentrations for the highest priority air toxics for review by the ATSAC. DEQ will propose additional and revised air toxics ambient benchmarks for review by the ATSAC based on the prioritization criteria in OAR 340-246-0090(2)(c). Once the ATSAC has completed review of each set of proposed ambient benchmarks, DEQ will, within 60 days, begin the process to propose ambient benchmarks as administrative rules for adoption by the Environmental Quality Commission.

(f) If DEQ is unable to propose ambient benchmarks to the ATSAC by the deadlines specified in OAR 340-246-0090(2)(e), the ATSAC will review the most current EPA ambient benchmarks. If EPA ambient benchmarks are not available, the ATSAC will review the best available information from other states and local air authorities.

(g) The ATSAC will consider proposed ambient benchmarks and evaluate their adequacy for meeting risk and hazard levels, considering human health, including sensitive human populations, scientific uncertainties, persistence, bio-accumulation, and, to the extent possible, multiple exposure pathways. The ATSAC will conduct this review consistent with the criteria in OAR 340-246-0090(2)(c) and (d). The ATSAC will report these findings to DEQ. If the ATSAC unanimously disagrees with DEQ's recommendation, DEQ will re-consider and re-submit its recommendation at a later date.

(h) The ATSAC will complete review of and report findings on each set of ambient benchmarks as quickly as possible, but no later than 12 months after DEQ has proposed them. If the ATSAC is unable to complete review of ambient benchmarks within 12 months after DEQ's proposal, DEQ will initiate rulemaking to propose ambient benchmarks.

(i) DEQ will review all ambient benchmarks at least every five years and, if necessary, propose revised or additional ambient benchmarks to the ATSAC. At its discretion, DEQ may review and propose a benchmark for review by the ATSAC at any time when new information is available.

(3) Ambient Benchmarks. Benchmark concentrations are in units of micrograms of air toxic per cubic meter of ambient air, on an average annual basis. The Chemical Abstract Service Registry Number (CASRN) is shown in parentheses.

(a) The ambient benchmark for acetaldehyde (75-07-0) is 0.45 micrograms per cubic meter.

(b) The ambient benchmark for acrolein (107-02-8) is 0.35 micrograms per cubic meter.

(c) The ambient benchmark for acrylonitrile (107-13-1) is 0.01 micrograms per cubic meter.

(d) The ambient benchmark for ammonia (7664-41-7) is 500 micrograms per cubic meter.

(e) The ambient benchmark for arsenic (7440-38-2) is 0.0002 micrograms per cubic meter.

(f) The ambient benchmark for benzene (71-43-2) is 0.13 micrograms per cubic meter.

(g) The ambient benchmark for beryllium (7440-41-7) is 0.0004 micrograms per cubic meter.

(h) The ambient benchmark for 1,3-butadiene (106-99-0) is 0.03 micrograms per cubic meter.

(i) The ambient benchmark for cadmium and cadmium compounds (7440-43-9) is 0.0006 micrograms per cubic meter.

(j) The ambient benchmark for carbon disulfide (75-15-0) is 800 micrograms per cubic meter.

(k) The ambient benchmark for carbon tetrachloride (56-23-5) is 0.2 micrograms per cubic meter.

(l) The ambient benchmark for chlorine (7782-50-5) is 0.1 micrograms per cubic meter.

(m) The ambient benchmark for chloroform (67-66-3) is 300 micrograms per cubic meter.

(n) The ambient benchmark for chromium, hexavalent (18540-29-9) is 0.00008 micrograms per cubic meter.

(o) The ambient benchmark for cobalt and cobalt compounds (7440-48-4) is 0.1 micrograms per cubic meter.

(p) The ambient benchmark for 1,4-dichlorobenzene (106-46-7) is 0.09 micrograms per cubic meter.

(q) The ambient benchmark for 1,3-dichloropropene (542-75-6) is 0.25 micrograms per cubic meter.

(r) The ambient benchmark for diesel particulate matter (none) is 0.1 micrograms per cubic meter. The benchmark for diesel particulate matter applies only to such material from diesel-fueled internal combustion sources.

(s) The ambient benchmark for dioxins and furans (1746-01-6) is 0.00000003 micrograms per cubic meter. The benchmark for dioxin is for total chlorinated dioxins and furans expressed as 2,3,7,8-TCDD toxicity equivalents.

(t) The ambient benchmark for ethyl benzene (100-41-4) is 0.4 micrograms per cubic meter.

(u) The ambient benchmark for ethylene dibromide (106-93-4) is 0.002 micrograms per cubic meter.

(v) The ambient benchmark for ethylene dichloride (107-06-2) is 0.04 micrograms per cubic meter.

(w) The ambient benchmark for ethylene oxide (75-21-8) is 0.0003 micrograms per cubic meter.

(x) The ambient benchmark for formaldehyde (50-00-0) is 0.2 micrograms per cubic meter.

(y) The ambient benchmark for n-hexane (110-54-3) is 700 micrograms per cubic meter.

(z) The ambient benchmark for hydrogen chloride (7647-01-0) is 20 micrograms per cubic meter.

(aa) The ambient benchmark for hydrogen cyanide (74-90-8) is 0.8 micrograms per cubic meter.

(bb) The ambient benchmark for fluoride anion (7664-39-3) is 13micrograms per cubic meter.

(cc) The ambient benchmark for lead and lead compounds (7439-92-1) is 0.15 micrograms per cubic meter.

(dd) The ambient benchmark for manganese and manganese compounds (7439-96-5) is 0.09 micrograms per cubic meter.

(ee) The ambient benchmark for elemental mercury (7439-97-6) is 0.3 micrograms per cubic meter.

(ff) The ambient benchmark for methyl bromide (74-83-9) is 5 micrograms per cubic meter.

(gg) The ambient benchmark for methyl chloride (74-87-3) is 90 micrograms per cubic meter.

(hh) The ambient benchmark for methyl chloroform (71-55-6) is 5,000 micrograms per cubic meter.

(ii) The ambient benchmark for methylene chloride (75-09-2) is 100 micrograms per cubic meter.

(jj) The ambient benchmark for naphthalene (91-20-3) is 0.03 micrograms per cubic meter.

(kk) The benchmark for soluble nickel compounds (various) is 0.01 micrograms per cubic meter, where soluble nickel compounds include nickel acetate (373-20-4), nickel chloride (7718-54-9), nickel carbonate (3333-39-3), nickel carbonyl (13463-39-3), nickel hydroxide (12054-48-7), nickelocene 1271-28-9), nickel sulfate 7786-81-4), nickel sulfate hexahydrate 10101-97-0), nickel nitrate hexahydrate (13478-00-7), and nickel carbonate hydroxide (12607-70-4).

(ll) The ambient benchmark for insoluble nickel compounds (various) is 0.004 micrograms per cubic meter, where insoluble nickel compounds include nickel subsulfide (12035-72-2), nickel oxide (1313-99-1), nickel sulfide (11113-75-0), and nickel metal (7440-02-0).

(mm) The ambient benchmark for phosphine (7803-51-2) is 0.8 micrograms per cubic meter.

(nn) The ambient benchmark for phosphoric acid (7664-38-2) is 10 micrograms per cubic meter.

(oo) The ambient benchmark for total (as the sum of congeners) polychlorinated biphenyls (1336-36-3) is 0.01 micrograms per cubic meter.

(pp) The ambient benchmark for total polycyclic aromatic hydrocarbons (none) is 0.002 micrograms per cubic meter, where total polycyclic aromatic hydrocarbons are the sum of the toxicity equivalency factor (with respect to benzo(a)pyrene (50-32-8)) adjusted concentrations for all of the following individual 26 polycyclic aromatic hydrocarbons: 5-methylchrysene (3697-24-3); 6-nitrochrysene (7496-02-8); acenaphthene (83-32-9); acenaphthylene (208-96-8); anthanthrene (191-26-4); anthracene (120-12-7); benz(a)anthracene (56-55-3); benzo(a)pyrene (50-32-8); benzo(b)fluoranthene (205-99-6); benzo(c)fluoranthene (243-17-4); benzo(e)pyrene (192-97-2); benzo(g,h,i)perylene (191-24-2); benzo(j)fluoranthene (205-82-3); benzo(k)fluoranthene (207-08-9); chrysene (218-01-9); cyclopenta(c,d)pyrene (27208-37-3); dibenz(a,h)anthracene (226-36-8); dibenzo(a,e)pyrene (192-65-4); dibenzo(a,h)pyrene (189-64-0); dibenzo(a,i)pyrene (189-55-9); dibenzo(a,l)pyrene (191-30-0); fluoranthene (206-44-0); fluorene (86-73-7); indeno(1,2,3-c,d)pyrene (193-39-5); phenanthrene (85-01-8); and pyrene (129-00-0).

(qq) The ambient benchmark for tetrachloroethylene (127-18-4) is 4 micrograms per cubic meter.

(rr) The ambient benchmark for toluene (108-88-3) is 5,000 micrograms per cubic meter.

(ss) The ambient benchmark for 2,4- & 2,6 toluene diisocyanate, mixture (26471-62-5) is 0.02 micrograms per cubic meter.

(tt) The ambient benchmark for trichloroethylene (79-01-6) is 0.2 micrograms per cubic meter.

(uu) The ambient benchmark for vinyl chloride (75-01-4) is 0.1 micrograms per cubic meter.

(vv) The ambient benchmark for white phosphorus (7723-14-0) is 9 micrograms per cubic meter.

(ww) The ambient benchmark for xylenes, mixed (1330-20-7) is 200 micrograms per cubic meter.

(xx) The ambient benchmark for hydrogen sulfide (7783-06-4) is 2.0 micrograms per cubic meter.

(yy) The ambient benchmark for methanol (67-56-1) is 4,000 micrograms per cubic meter.

(zz) The ambient benchmark for phosgene (75-44-5) is 0.3 micrograms per cubic meter.

(aaa) The ambient benchmark for n-propyl bromide (106-94-5) is 0.5 micrograms per cubic meter.

(bbb) The ambient benchmark concentration for styrene (100-42-5) is 1,000 micrograms per cubic meter.

Stat. Auth.: ORS 468.035, 468A.010(1) & 468A.015
Stats. Implemented: ORS 468A.015, 468A.025
Hist.: DEQ 15-2003, f. & cert. ef. 11-3-03; DEQ 12-2006, f. & cert. ef. 8-15-06

340-246-0110, Source Category Rules and Strategies

(1) DEQ may identify the need for source category rules and strategies through the following methods:

(a) The emissions inventory, modeling or monitoring, shows air toxics emissions from point, area, or mobile sources associated with public health risk at public receptors;

(b) Development of a local air toxics reduction plan provides source category controls that could be effectively applied to sources existing in other parts of the state; or

(c) When implementing the Safety Net Program, DEQ establishes air toxics emissions reductions for a source and determines that there are other similar sources in the state to which the reductions must apply.

(2) Subject to the requirements in this rule, the Lane Regional Air Pollution Authority is designated by the Commission as the agency responsible for implementing Source Category Rules and Strategies within its area of jurisdiction. The requirements and procedures contained in this rule must be used by the Regional Authority to implement Source Category Rules and Strategies unless the Regional Authority adopts superseding rules that are at least as restrictive as the rules adopted by the Commission.

(3) DEQ will consider the following criteria in determining whether to propose source category strategies under this division:

(a) Whether air toxics emissions from the source category are not, or will not, be addressed by other regulations or strategies, including emissions reduction requirements under the Geographic

Program (OAR 340-246-0130 through 340-246-0170), or the Safety Net Program (OAR 340-246-0190 through 340-246-0230);

(b) Whether air toxic emissions from the source category can be effectively reduced through regulations or voluntary strategies; and

(c) Whether the source category contributes to ambient benchmark exceedances at public receptors statewide, in multiple geographic areas, or in multiple counties

Stat. Auth.: ORS 468.035, 468A.010(1), 468A.015
Stats. Implemented: ORS 468A.015, 468A.025
Hist.: DEQ 15-2003, f. & cert. ef. 11-3-03

340-246-0130, Geographic Program (0130 through 0170)

(1) Purpose. The Geographic Program addresses emissions from multiple sources of air toxics. It requires prioritizing and selecting geographic areas of concern, forming a local advisory committee, developing a specific local plan to control air toxics, a public participation and comment process, EQC adoption or approval, implementing reduction strategies, and periodically evaluating the effectiveness by DEQ.

(2) Subject to the requirements in OAR 340-246-0130 through 0170, the Lane Regional Air Pollution Authority is designated by the Commission as the agency to implement the Geographic Program within its area of jurisdiction. The requirements and procedures contained in this rule must be used by the Regional Authority to implement the Geographic Program unless the Regional Authority adopts superseding rules which are at least as restrictive as state rules. The Regional Authority will address geographic areas as resources allow, considering the prioritization criteria in 340-246-0150.

Stat. Auth.: ORS 468.035, 468A.010(1), 468A.015
Stats. Implemented: ORS 468A.015, 468A.025
Hist.: DEQ 15-2003, f. & cert. ef. 11-3-03

340-246-0150, Prioritizing and Selecting Geographic Areas

(1) DEQ will prioritize geographic areas by considering the total cancer and non-cancer risk from air toxics to the population in the area, as indicated by:

(a) The number and degree of ambient benchmark exceedances;

(b) The toxicity or potency of air toxics exceeding ambient benchmarks;

(c) The level of exposure and number of people at risk in areas of concern;

(d) The presence of sensitive populations;

(e) The effectiveness of local control strategies; and

(f) To the extent known, the risk posed by multiple pollutants and pollutant mixtures.

(2) Not later than 18 months after the first set of benchmarks is adopted, DEQ will select the first geographic area for air toxics reduction planning. DEQ will base selection on representative monitoring compared to the ambient benchmark concentrations at public receptors. To the extent possible, geographic areas will be identified using monitoring data generated following EPA monitoring guidelines. Subsequent geographic areas will be selected after completion of monitoring. A geographic area is formally selected upon publication of a notice in the Oregon Secretary of State's Bulletin. Once an area is selected for air toxics reduction planning, it will retain the status of a selected geographic area until DEQ determines through an evaluation of data that a reduction plan is no longer necessary for the area to meet all air toxics ambient benchmarks.

(3) DEQ will first select for emissions reduction planning the high priority geographic areas, where concentrations of air toxics are more than ten times above the ambient benchmarks or above a hazard quotient of one with the potential for serious adverse health effects. DEQ will select all other geographic areas, where air toxics concentrations are above benchmarks, after air toxics emissions reduction plans have been approved for the high priority geographic areas.

(4) Geographic Area Boundaries. DEQ will establish general geographic area boundaries on a neighborhood or urban area scale. DEQ will consider feasibility of administration when setting the boundaries of a geographic area. In setting geographic area boundaries, DEQ will consider criteria including but not limited to the following:

(a) Areas of impact (where people are exposed);

(b) Population density;

(c) Areas of influence (where sources are located);

(d) Meteorology;

(e) Geography and topography;

(f) Including all air toxics exceeding ambient benchmarks; and

(g) Coordination with criteria pollutant boundaries for attainment of the National Ambient Air Quality Standards (NAAQS).

Stat. Auth.: ORS 468.035, 468A.010(1), 468A.015
Stats. Implemented: ORS 468A.015, 468A.025
Hist.: DEQ 15-2003, f. & cert. ef. 11-3-03

340-246-0170, Local Air Toxics Emissions Reduction Planning

(1) DEQ will develop air toxics reduction plans for selected geographic areas with the advice of local advisory committees. The main role of a local advisory committee is to consider air toxics reduction options and to recommend a specific air toxics reduction plan for their geographic area. The Director will appoint a local air toxics advisory committee.

(a) Local advisory committees will generally be composed of a balanced representation of members from affected local government, local health departments, the public, small businesses

(50 or fewer employees), larger businesses (if present in the area), and interest groups represented in the area.

(2) Local Advisory Committee Tasks.

(a) Within 18 months of their first meeting, the committee will evaluate options for reducing emissions of air toxics that exceed ambient benchmarks, and recommend a local air toxics reduction plan to DEQ.

(b) DEQ may grant an extension of time to the local committee if requested by the committee, if DEQ believes the extension is technically justified and the committee is making reasonable progress in developing a local air toxics reduction plan.

(c) If the committee is unable to recommend a local air toxics reduction plan to DEQ within 18 months, or the date of an extension, DEQ will formulate a plan for the area within six months.

(d) DEQ and the local advisory committee will seek local government support for the proposed local air toxics emissions reduction plan.

(e) The local advisory committee will evaluate the plan's effectiveness as it is implemented and recommend changes to DEQ.

(f) At DEQ's request, the local advisory committee will reconvene to implement contingency planning and recommend contingency measures as specified by OAR 340-246-0170(4)(l).

(g) If the committee is unable to recommend contingency measures within 18 months, DEQ will formulate contingency measures for the area within 6 months.

(3) Public Notice, Comment, Approval and Adoption by the Environmental Quality Commission. DEQ will provide an opportunity for public notice and comment on proposed local emissions reduction plans. After the public notice and comment process is complete, DEQ will present local air toxics reduction plans to the Commission for approval, including adoption of appropriate administrative rules. The Environmental Quality Commission may delegate the approval of plans that do not contain administrative rules to the Director of DEQ.

(4) Elements of an Air Toxics Reduction Plan:

(a) Local air toxics reduction plans must focus on the air toxic or air toxics measured or modeled above the ambient benchmarks.

(b) Local air toxics reduction plans must be based on sound data analysis. This includes developing enhanced emissions inventory information for the local area using source-specific information to the extent possible. This may also include enhanced modeling and monitoring to better characterize ambient concentrations. Plans also must rely on sound analysis of the effectiveness and cost of air toxics emissions reduction options. Where needed to fill specific information gaps, DEQ may require air toxics emissions reporting for specific sources or source categories within the geographic area on a case-by-case basis.

(c) The emissions reduction goals for individual air toxics are ambient benchmarks in local air toxics reduction plans.

(d) Local air toxics reduction plans must be designed to reduce air toxics emissions in a timely manner.

(A) When feasible, local air toxics reduction plans will be designed to reach levels that are equal to or below ambient benchmark concentrations. Plans will be designed to achieve emissions reductions within ten years, beginning at the date the Commission approves the plan. Local plans must provide for the timeliest reductions possible for each air toxic exceeding ambient benchmarks.

(B) Local air toxics reduction plans must include specific three-year milestones that DEQ and the local advisory committee will evaluate every three years, in coordination with DEQ's air toxics emissions inventory update.

(e) Every three years, DEQ will assess the effectiveness of local plans and make recommendations for plan revision based on progress meeting milestones or new information. If DEQ finds lack of progress at year three, it will work with the local advisory committee to provide corrective measures. If DEQ finds lack of progress at year six and projects that ten-year goals in OAR 340-246-0170(4)(d)(A) will not be met, it will implement the contingency plan in 340-246-0170(4)(l). If at year nine DEQ projects that ten year goals in 340-246-0170(4)(d)(A) will not be met, it will work with the local advisory committee to propose and seek adoption of measures necessary to reach these goals.

(f) Local air toxics reduction plans must evaluate air toxics emissions from all types of sources, including point, area, and mobile sources. Plans must require emissions reductions from the most significant sources of air toxics. Mandatory emissions reduction strategies will be commensurate with source contributions, considering relative emissions, toxicity, technical feasibility, cost-effectiveness and equity.

(g) Local air toxics reduction plans must include strategies to reduce high concentrations of air toxics that are limited to smaller portions of a geographic area as well as pollutants causing public health risk throughout the area.

(h) Local air toxics reduction plans may include a variety of mandatory and voluntary approaches to reducing emissions of air toxics. Depending on the type of source, local air toxics reduction plans may include public education, pollution prevention alternatives, economic incentives and disincentives, technical assistance and regulatory requirements.

(i) DEQ will ensure the opportunity for public involvement during the plan development process. This includes involving those affected by the air toxics emissions and those affected by the proposals to reduce air toxics emissions. Proposed local air toxics reduction plans must be available for public hearing and comment.

(j) Local air toxics reduction plans must be coordinated with other local, state, and federal requirements to the extent possible. This includes considerations of any ozone or particulate control requirements for the area, any federal standard applicable to sources in the area, any strategies that are federally pre-empted, and any impacts on water or land, such as water pollution or hazardous waste.

(k) Local air toxics reduction plans will include specific recommendations for developing ongoing emissions inventory or ambient air monitoring to track local trends in air toxics.

(l) Local air toxics reduction plans must include a contingency plan that will be implemented if evaluation at year six shows that an area is not meeting milestones and will not achieve the ten year goals established under OAR 340-246-0170(4)(d)(A). The contingency plan, like the original plan, must require emissions reductions from the most significant sources of air toxics. Mandatory emissions reduction strategies will be commensurate with source contributions, considering relative emissions, toxicity, technical feasibility cost-effectiveness and equity. Contingency plans must include but are not limited to:

(i) Re-evaluation of planning assumptions, such as emissions factors, motor vehicle data and background pollutants;

(ii) Evaluation of existing conditions and effectiveness of emissions reduction strategies, including reasons for success or failure; and

(iii) New or progressively more mandatory strategies that will be considered.

Stat. Auth.: ORS 468.035, 468A.010(1), 468A.015
Stats. Implemented: ORS 468A.015, 468A.025
Hist.: DEQ 15-2003, f. & cert. ef. 11-3-03

340-246-0190, Air Toxics Safety Net Program (0190 through 0230)

(1) The purpose of the Air Toxics Safety Net Program is to address human exposures at public receptors to air toxics emissions from stationary sources that are not addressed by other regulatory programs or the Geographic Program. It is the Commission's expectation that the Safety Net Program in OAR 340-246-0190 through 340-246-0230 will apply only rarely.

(2) Subject to the requirements contained in OAR 340-246-0190 through 340-246-0230, the Lane Regional Air Pollution Authority is designated by the Commission as the agency responsible for implementing the Air Toxics Safety Net Program within its area of jurisdiction. The requirements and procedures contained in this rule must be used by the Regional Authority to implement the Air Toxics Safety Net Program unless the Regional Authority adopts superseding rules, which are at least as restrictive as the rules adopted by the Commission.

(3) Selection of Sources. DEQ will select a source for the Air Toxics Safety Net Program if all of the following criteria are met:

(a) DEQ has ambient monitoring information, gathered using appropriate EPA or other published international, national, or state standard methods that concentrations of air toxics have caused an exceedance of at least one ambient benchmark at a site representing expected human exposure to air toxics from the source at a public receptor in a location outside of the source's ownership or control.

(b) DEQ has information that the source's air toxics emissions alone have caused an exceedance of at least one ambient benchmark at a site representing expected human exposure to air toxics

from the source at a public receptor, in a location outside of the source's ownership or control. This could be based on emissions inventory, modeling or other information.

(c) The source is not subject to or scheduled for a federal residual risk assessment under the federal Clean Air Act section 112(f)(2) through (6).

(d) The source is not subject to an emissions limit or control requirement imposed as the result of modeling or a risk assessment performed or required by DEQ prior to November 1, 2003 for the air toxics that exceed the ambient benchmarks.

(e) The source is located outside of a selected geographic area, as designated in OAR 340-246-0130 through 0170.

(4) Air Toxics Science Advisory Committee Review. Before requiring a source to conduct a source-specific risk assessment, DEQ will present its analysis to the ATSAC. Within 120 days, the ATSAC will review the analysis and make a finding. If the ATSAC concurs with DEQ or takes no action, DEQ may proceed under this rule. If the ATSAC objects, DEQ will not proceed until it receives concurrence from the Commission.

(5) Source-Specific Exposure Modeling and Risk Assessment. Upon written notification by DEQ, a source must conduct a risk assessment including exposure modeling for the air toxics measured at levels above ambient benchmarks. The source must use a risk assessment methodology provided by DEQ. This risk assessment will provide the basis for establishing air toxics emissions reductions or demonstrating that at public receptors in areas outside of a source's ownership or control, people are not being exposed to air toxics at levels that exceed the ambient benchmarks.

(6) Risk Assessment Methodology. DEQ will provide guidance on the methods to be used. The risk assessment methodology will be developed in consultation with the ATSAC and will result in a protocol that:

(a) Uses reasonable estimates of plausible upper-bound exposures that neither grossly underestimate nor grossly overestimate risks;

(b) Considers the range of probabilities of risks actually occurring, the range of size of the populations likely to be exposed to the risk, and current and reasonably likely future land uses;

(c) Defines the use of high-end and central-tendency exposure cases and assumptions;

(d) Develops values associated with chronic exposure for carcinogens; and

(e) Addresses both carcinogenic and non-carcinogenic air toxics and allows for detailed exposure assessments to the extent possible.

(7) Review and Acceptance. DEQ will evaluate the risk assessment for adequacy and completeness before accepting the results. If the results demonstrate that the source is not causing human exposures to air toxics at levels that exceed the ambient benchmarks at public receptors, in areas outside the source's ownership or control, and DEQ has received concurrence from the ATSAC, DEQ will notify the source that air toxics emissions reductions will not be required under this rule.

Stat. Auth.: ORS 468.035, 468A.010(1), 468A.015
Stats. Implemented: ORS 468A.015, 468A.025
Hist.: DEQ 15-2003, f. & cert. ef. 11-3-03

340-246-0210, Safety Net Source Air Toxics Emissions Reductions

(1) Air Toxics Emissions Reduction Analysis:

(a) If source-specific exposure modeling and risk assessment show that the source is causing exceedances of ambient benchmarks at public receptors in areas outside the source's ownership or control, the source must perform an analysis showing how air toxics could be reduced to meet ambient benchmarks. DEQ and the safety net source will develop proposed air toxics emissions reduction measures based on modeling and, when available, monitoring information.

(b) As part of the air toxics emissions reduction analysis, the source will analyze pollution prevention options, and is encouraged to use the hierarchy stated in OAR 340-246-0050.

(2) Air Toxics Emissions Reduction Requirements:

(a) A safety net source emitting air toxics causing exposure resulting in excess lifetime cancer risk greater than one in a million (1x10-6) or a hazard quotient of one for non-carcinogens must, as soon as practicable but no later than three years after the effective date of the permit imposing such conditions, meet toxics best available retrofit technology (TBART) for each air toxic that exceeds an ambient benchmark.

(b) A safety net source may use a means of air toxics reduction, other than TBART, if it can demonstrate to DEQ that it will achieve a risk level at or below one in a million, or a hazard quotient at or below one, within three years of using the other means of air toxics emissions reductions.

(c) A safety net source emitting a carcinogenic air toxic causing excess lifetime cancer risk at or above one hundred in a million (1x10-4) must reduce its air toxic emissions to achieve a risk level below one hundred in a million as soon practicable but no later than one year after the effective date of the permit imposing such conditions.

(d) A safety net source emitting a non-carcinogenic air toxic at a level above a hazard quotient of one that DEQ finds to have a potential for causing very serious or irreversible adverse health effects must reduce its air toxic emissions below this level as soon practicable, but no later than one year after the effective date of the permit imposing such conditions.

(3) If a safety net source cannot reach a risk level at or below excess lifetime cancer risk of one in a million, or a hazard quotient at or below one in three years, even though it meets TBART, the TBART determination for the source will be subject to periodic review under this section until the source achieves a risk level at or below one in a million or a hazard quotient at or below one. Upon each renewal of the source's permit, TBART for the source must be reviewed, taking into consideration retrofit costs and the remaining useful life of controls installed or other measures taken to meet a prior TBART determination. Upon renewal of the source's permit, DEQ must include conditions requiring the source to meet TBART as determined for that permit renewal.

Stat. Auth.: ORS 468.035, 468A.010(1), 468A.015
Stats. Implemented: ORS 468A.015, 468A.025
Hist.: DEQ 15-2003, f. & cert. ef. 11-3-03

Attachment B – Summary of ATSAC Consensus Recommendations on Benchmark Revisions

Supporting documents

Summary of ATSAC Consensus Recommendations on Benchmark Revisions

1 Acrolein

Acrolein is known to have non-carcinogenic effects. The current Ambient Benchmark Concentration, or ABC, for acrolein is 0.02 micrograms per cubic meter, or µg/m^3. It is based on a 2002 EPA IRIS Reference Concentration of 0.02 µg/m^3.

Currently, OEHHA, recommends a Reference Exposure Level, sometimes referred to as a Reference Concentration, value of 0.35 µg/m^3 for acrolein; this value incorporates an uncertainty factor related to potential exposure of asthmatic children. Due to acrolein's high volatility, applying a generous protective factor for asthmatic children is appropriate. The OEHHA value of 0.35 µg/m3 is based on new information since the value of 0.02 µg/m^3 was identified as the ABC for acrolein.

The new study upon which OEHHA bases its new Reference Exposure Level is 30 years newer than the study that OEHHA previously depended on. The fact that a credible no-observed-adverse-effect level, or NOAEL, is available that does not require extrapolation from a lowest-observed-adverse-effect level, or LOAEL, is important, and the Dorman et al. 2008 study from which the NOAEL was drawn appears to be a robust and well-done study; this makes the related OEHHA Reference Exposure Level very credible.

The Air Toxics Science Advisory Committee, or ATSAC, unanimously recommended 0.35 µg/m^3 as the new ABC for acrolein.

2 Ammonia

Ammonia is known to have non-cancer effects. A new Reference Concentration of 500 µg/m^3 for ammonia became available from EPA's IRIS in September 2016, and was discussed by the ATSAC in March 2017. Although the new number is draft, committee members agreed it might be prudent to recommend its use, rather than waiting until the next ABC cycle begins in 2020; it is also unlikely that the draft number will change. Previously in January 2015, the committee set the ABC for ammonia at 200 µg/m^3, based on the OEHHA Reference Effect Level from 2000. Both values used the same occupational worker study from Holness et al. as their basis. The new IRIS value represents a new look at this information 16 years later, and thus is a much more recent number. The same base study was used to obtain two differing values based on the application of different uncertainty factors, as well as different toxicological points of departure, and the new IRIS value is within an order of magnitude of the OEHHA value that was used for the ABC for ammonia. The committee decided to use the September 2016 number available from IRIS, and noted that the committee has always prioritized IRIS over OEHHA. The committee unanimously voted to recommend that the ABC for ammonia be set at 500 µg/m^3.

3 Cadmium

Cadmium is a heavy metal that has both cancer and non-cancer effects, but the ABC is based on the more-stringent protective value for cancer effects. Cancers of the respiratory tract drive

cadmium cancer risk; for the non-cancer effects of cadmium, the kidney is the target organ, and impacts to the kidney can result in brittle bone syndrome. The standing cancer-based ABC for cadmium of 0.0006 µg/m^3 is based on EPA's IRIS URE of 1.8 x 10^{-3} per µg/m^3, related to lung cancer. California's OEHHA has a different cancer value for cadmium, but that value hasn't changed since the previous iteration of the ATSAC reviewed toxicity information for cadmium. The OEHHA value is based on an upper-bound value, while the IRIS value is based on the use of a point-estimate approach (Thun et al. 1985). The Agency for Toxic Substances and Disease Registry, or ATSDR, and EPA are identical in their recommendations.

The consensus of the committee was to retain the current ABC of 0.0006 µg/m^3 for cadmium.

4 Carbon Tetrachloride

Carbon tetrachloride has both cancer and non-cancer effects, but the standing ABC of 0.07 µg/m^3 is based on cancer effects; specifically, on a 1991 EPA IRIS URE value of 1.5 x 10^{-5} per µg/m^3. Carbon tetrachloride is a Class B2 carcinogen, which means it is probably carcinogenic to humans, but has little or no available supporting data. In 2010, EPA re-evaluated carbon tetrachloride using a 104-week-long inhalation study that used both rats and mice; the resulting URE value is 6 x 10^{-6} per µg/m^3, which converts to a protective value of 0.017 per µg/m^3.

The ATSAC unanimously recommended a revised ABC of 0.17 µg/m^3. The committee will round this value up to 0.2 µg/m^3, to be consistent with previous ATSAC decisions along these lines.

5 Chlorine

Chlorine is known to have non-cancer effects. The OEHHA 2000 Reference Exposure Level, which was obtained from a 1995 rat study, was used as the basis of the existing ABC for chlorine of 0.2 µg/ m^3. A protective level of 0.1 µg/m^3 is recommended by the Agency for Toxic Substances and Disease Registry (ATSDR), and is based on a 1987 study that used monkeys as test subjects. The way that monkeys uptake chlorine through inhalation is more similar to the way human uptake occurs, and therefore the study that used monkeys is preferred by the ATSAC over the study that used rats. Uncertainty factors for both the 1987 and 1995 studies resulted in a total uncertainty factor of 30, so both studies have similar levels of uncertainty. However, the monkeys were tested with lower doses than those used in the rat study, and nasal lesions still occurred; this makes the monkey study more credible to the ATSAC.

Five of the six ATSAC members voted to revise the ABC for chlorine to reflect the ATSDR value of 0.1 µg/m^3, while one member, Kent Norville, abstained because he felt that OEHHA had used good science to choose their value based on the rat study, and that the ATSAC had inconsistently applied their decision protocol in regard to choosing one study over another.

6 Chloroform

Chloroform is known to have non-cancer effects. The current ABC for chloroform is 98 µg/m^3, which was based on the 1998 Minimal Risk Level from ATSDR. A committee member pointed out that notes made during the prior ATSAC in 2006 stated that the committee didn't use the OEHHA value of 300 µg/m^3 available at that time because it was thought to be based on an oral study; but this is not correct. The OEHHA study was actually an inhalation study that assessed intermittent exposure; the committee member thought this study was very credible. The OEHHA study was not used by the ATSAC earlier because committee policy does not allow extrapolation

of inhalation toxicity values from oral studies, and the ATSAC mistakenly thought that the OEHHA value was based on an oral study. No IRIS values are currently available for chloroform.

The committee unanimously recommended that the ABC for chloroform be revised to 300 $\mu g/m^3$, based on the OEHHA 2000 chronic inhalation reference concentration.

7 Chromium, hexavalent

The current benchmark value for hexavalent chromium is 0.00008 $\mu g/m^3$, which is based on a 1998 IRIS URE value of 1.2×10^{-2} per $\mu g/m^3$. The committee discussed whether an additional benchmark value for trivalent chromium should be identified. The hexavalent chromium studies upon which the standing ABC is based were conducted within an experimental atmosphere containing primarily hexavalent chromium; however this point is not explicitly reported. Thus, the committee is of the opinion that hexavalent chromium is the species of relevance to adverse health effects. Therefore it was decided to retain the ABC for hexavalent chromium of 0.00008 $\mu g/m^3$. This benchmark concentration should not be applied to total chromium data. Because no new carcinogenic potency information is available for hexavalent chromium, the 1998 IRIS toxicity information is considered representative of the best-available science. The committee voted unanimously to retain the ABC of 0.00008 $\mu g/m^3$ for hexavalent chromium.

8 Cobalt

Cobalt is known to have non-cancer effects. The standing ABC for cobalt of 0.1 $\mu g/m^3$ is based on an ATSDR 2001 non-cancer Minimal Risk Level value; not much other toxicity information for cobalt was available during the first ATSAC review of cobalt information in 2005. Formerly, the protective level for cobalt exposure was based on particulate exposure, and related cobalt toxicity studies were based on exposure of occupational workers to cobalt. In more-recent studies, animals were exposed over a lifetime to atomized sprays of soluble cobalt sulfate or cobalt chloride. These studies demonstrated the carcinogenic properties of cobalt. However, under non-controlled conditions, such as those that would occur with releases of cobalt to the atmosphere, soluble cobalt sulfate undergoes rapid environmental breakdown. The ATSAC decided that the environmentally relevant form of cobalt is particulate cobalt, in terms of potential human exposure.

EPA has not weighed in on cobalt toxicity in IRIS, but does provide cobalt toxicity information in their Provisional Peer-Reviewed Toxicity Values, or PPRTVs. The alternative Reference Concentration of 0.006 $\mu g/m^3$ available as a PPRTV is the most-current EPA value available.

Both the 2001 ATSDR value of 0.1 $\mu g/m^3$ and the currently available PPRTV value of 0.006 $\mu g/m^3$ are based on the same study (Nemery et al., 1992), but EPA applied additional uncertainty factors to obtain their PPRTV. Per ATSAC policy, the higher of two values based on the same study is chosen, all other things being equal. The committee voted unanimously to recommend retaining the current value of 0.1 $\mu g/m^3$ as the ABC for cobalt.

9 Diesel particulate matter

Diesel particulate matter, or DPM, has both carcinogenic and non-carcinogenic effects. This compound was discussed in great detail by the ATSAC over the course of six committee meetings which took place in May, June, July, September, and October of 2015, and March of 2017.

The single published 1998 URE value currently available is from OEHHA. The OEHHA URE value is 0.0003 per $\mu g/m^3$; when converted to a concentration that is protective at a one-in-one-million risk, the value 0.003 $\mu g/m^3$ is obtained. The more-stringent OEHHA value was available back in 2005, and was originally published in 1998, but there was controversy surrounding the technical credibility of this number. The OEHHA 1998 URE value has been questioned for years by a number of technical agencies, including the International Agency for Research on Cancer, and by some of the researchers whose data was used by OEHHA to calculate the value. EPA and the World Health Organization both tried to calculate a URE value, but gave up because they considered the available data to be inadequate. No other agency or researcher has been able to replicate the California value, which further detracts from its credibility. Although the California value has been adopted and used by Washington state and a metropolitan agency in Vancouver, B.C., it seems that the California value was accepted because there was no other estimate available for diesel emissions. Also, the California value is based on total particulate mass, which is most likely not the component in diesel exhaust that can cause cancer. The study behind the California value does not include an exposure response, which further detracts from the credibility of the California value. Because the OEHHA value is older (nearly 20 years old at this point) and because the ATSAC has not been able to determine exactly how OEHHA calculated the value, the committee continues, as it did circa 2006, to refuse consideration of the OEHHA URE value as a basis for a new ABC for DPM.

A number of draft Geometric Mean values, which represented a number of potentially credible DPM values from the scientific and regulatory literature, were calculated and discussed by the ATSAC. Among many different results obtained through different groupings of the available study results, the calculated Geometric Mean values fell within a range of 0.001 $\mu g/m^3$ to 0.003 $\mu g/m^3$. However, more discussion by the committee cast doubt on which studies should be included, and the ATSAC dropped the idea of a Geometric Mean approach as a way to identify an ABC for DPM.

In 2005, the ATSAC chose an ABC for DPM of 0.1 $\mu g/m^3$ for DPM and made a statement that, at the time, the committee's pick was a reasonable choice, particularly because it recognized at that time that DPM was a carcinogen. The committee set the standard near the World Health Organization values that were available at that time. Other, newer information on DPM has become available since then, but so far none of the new information allows the current committee to quantitatively identify a new URE for DPM. Thus, the committee voted unanimously in March 2017 to retain the current ABC of 0.1 $\mu g/m^3$ for DPM.

10 Ethylene oxide

Ethylene oxide is known to have both cancer and non-cancer effects. The current ABC of 0.01 $\mu g/m^3$ for ethylene oxide is based on the URE for ethylene chloride that was available in 1987 from OEHHA. This was the only toxicity factor available for ethylene oxide in 2005. The 1987 URE value is 8.8×10^{-5} per $\mu g/m^3$, which converts to an ABC of 0.01 $\mu g/m^3$. OEHHA had applied a multi-stage model to a rodent study in order to extrapolate a URE value relevant to the protection of human receptors.

Two different UREs were published by the EPA in the IRIS database in December 2016 in a document entitled *Evaluation of the Inhalation Toxicity of Ethylene Oxide*, EPA 635/R-16/350Fc. One URE of 3×10^{-3} per $\mu g/m^3$ was based on adult exposure with the endpoints of lymphoid cancer and breast cancer in females. If an ABC were calculated using this URE value,

the ABC would be 0.0003 µg/m^3. EPA calculated this adult-based URE by first subtracting 16 years from the lifetime assumption of 70 years to make sure only the adult exposure period was being considered; then the results were averaged over a 70-year period of time.

The second EPA IRIS URE value considered by the ATSAC was 5 x 10^{-3} per µg/m^3, which would result in an ABC of 0.0002 µg/m^3. This URE value was calculated using the same data and most of the same assumptions as the first URE value, but age-dependent adjustment factors known as ADAFs were applied to account for the greater vulnerability of children to ethylene oxide. Ethylene oxide has mutagenic effects, which means that early-life DNA mutations caused by exposure to this chemical at the more-vulnerable child life stages increases the lifetime potency of this carcinogen. However, per previous ATSAC policy, ABCs will not be adjusted using ADAFs. The ATSAC continues to recommend instead that ADAFs be used within the context of any human health risk assessment that is conducted.

The December 2016 URE values are considered by the ATSAC to be based on robust and well-conducted epidemiological studies (Steenland 2003; Steenland 2004; Stayman 1993, among others) using a very large National Institute for Occupational Safety and Health, or NIOSH, cohort of workers exposed to ethylene oxide during use of the compound for sterilization purposes. These values are nearly 30 years newer than the 1987 OEHHA value used to identify the current ABC for ethylene oxide, and are based on direct epidemiological (human) exposure data, rather than the rodent data which served as the basis of the 1987 OEHHA value. Because human data rather than animal data was used to generate the 2016 URE values, there is significantly less uncertainty associated with the 2016 URE values. In addition, the ATSAC tends to choose an IRIS value over those from other authoritative bodies, all other things being equal.

The difference between the two ABC values related to the two 2016 values is very small, 0.0003 µg/m^3 versus 0.0002 µg/m^3. Although epidemiological data are considered more credible than animal data, the results of the animal studies which have been conducted for ethylene oxide provide strong support of the fact that ethylene oxide is indeed carcinogenic, and also a mutagen.

The ATSAC voted unanimously to recommend the adoption of the value of 0.0003 µg/m^3 as the new ABC for ethylene oxide. This value will be based on the EPA IRIS December 2016 URE value of 3 x 10^{-3} µg/m^3.

11 Formaldehyde

Formaldehyde is known to have both cancer and non-cancer effects. The current ABC for formaldehyde is 3 µg/m^3, which was based on the non-cancer 2000 OEHHA Reference Exposure Level value; in approximately 2005, the ATSAC decided not to use cancer-based toxicity values as potential toxicity values for formaldehyde, because the committee felt that the issue of its cancer potency had not been resolved.. EPA IRIS (typically referred to as IRIS) currently lists a cancer-based inhalation unit risk factor, aka URE, of 1.3 x 10^{-5} per µg/m^3, which, when converted to a concentration that is protective of human health at a risk target of 1 x 10^{-6}, is 0.08 µg/m^3.

For non-cancer effects, OEHHA revised its Reference Exposure Level in 2008 to 9 µg/m^3, based on additional studies and evaluation of the data. Formaldehyde previously had been estimated to have a high risk of cancer, based on information from EPA's National Air Toxics Assessment program; and based on the newer pre-public review of the 2011 data, formaldehyde will still

have one of the highest cancer risks estimated. The related, modeled value from EPA is 17.8 in a million excess incidents of cancer risk over a lifetime for Oregon populations statewide. This is an estimate similar to what's been calculated for formaldehyde in Lane County, Oregon. Thus it is important to determine protective levels for formaldehyde in air.

The OHHEA-based cancer value of 0.2 per $\mu g/m^3$ was discussed by the committee as a potential ABC for formaldehyde; this value was based on the OEHHA URE of 6×10^{-6} per $\mu g/m^3$. By default, because the cancer-based value is more stringent than the non-cancer-based values for formaldehyde, the OEHHA-based cancer value of 0.2 $\mu g/m^3$ would also be protective of non-cancer effects. Note that the current IRIS URE (which converts to a protective concentration of 0.08 $\mu g/m^3$) and OEHHA unit risk estimate (which converts to a protective concentration of 0.17 $\mu g/m^3$, which in turns rounds up to 0.2 $\mu g/m^3$) are both based on a rat inhalation study conducted by Kerns, et al. back in 1983.This was a rat inhalation study that IRIS felt provided reliable information, and later on California OEHHA also utilized it. It should be noted that the EPA classified formaldehyde as a Class B1 (probable human) carcinogen, while the International Agency for Research on Cancer classifies formaldehyde as a 2A (probable human) carcinogen.

In 2014, in the National Toxicology Program's *13th Report on Carcinogens (RoC),* formaldehyde was classified as a known human carcinogen. Cancer is induced at levels much lower than levels at the level at which non-cancer effects occur, such as irritation of the mucus membranes and inflammation of the respiratory track. In May 2014, EPA held a formaldehyde workshop, which was convened in order to consider other health outcomes from formaldehyde, as well as evidence supporting concern about the induction of leukemia and lymphomas due to exposure to formaldehyde. No consensus publication resulted from that workshop. There is some discussion of leukemia and lymphoma in the National Toxicology Program's RoC, but IRIS and OHHEA benchmarks for formaldehyde are based on the endpoint effects of nasal cancers.

The endogenous (inside the body) production of formaldehyde was considered, because although formaldehyde is a naturally-occurring substance outside the body, it is also produced inside the body, which means it's always present and people are always being exposed to it at some level. This type of mechanism infers that a certain threshold concentration for adverse health effects probably exists. The other concern discussed at the meeting was formaldehyde exposure causing leukemia, lymphomas, and hepatotoxicity (liver toxicity). However, there appear to be some animal studies related to this question, but at this point in time, there is not much hard evidence, although studies are in progress. Interest and funding for formaldehyde toxicity research was spurred by the unfortunate situation that occurred with the FEMA trailers deployed after Katrina to New Orleans. After the trailers were no longer needed in New Orleans, they were not scrapped, but instead were resold. These trailers unfortunately continue to be used for housing around the United States, particularly in the Midwest. The formaldehyde released to air from these trailers is still occurring at levels that cause adverse health effects, although over time the concentration of formaldehyde in air in these trailers has decreased to about 15 parts per million. Because of all this, new formaldehyde research is in the process of being published, but it's not available right now.

A committee member presented some information from the National Toxicology Program's Report on Carcinogens. The Report on Carcinogens is a science-based, public health document prepared by the National Toxicology Program that identifies substances in our environment considered to be cancer hazards. The Report on Carcinogens considered human, animal, and

mechanistic studies published through November 8, 2013 that focused on the potential for formaldehyde exposure to cause nasopharyngeal cancer. Based on these findings, the National Toxicology Program committee concluded that formaldehyde is *known to be a human carcinogen* based on sufficient evidence of carcinogenicity from studies in humans and supporting data on the mechanisms of carcinogenicity. The committee member explained that, in worker studies, we see an outcome of nasopharyngeal cancers squamous cell, cancers, and other dysplasias related to formaldehyde exposure. We see intermediate endpoints too, of dysplasias in the epithelium of the nasoturbinate and pharyngeal passages. The unit risk estimate being considered for use as an ABC by the committee is based on the study by Kerns et al. in 1983, which was conducted with 120 rats exposed for a six-month period, six hours a day, at environmental levels of exposure, rather than occupational worker exposure levels.

In summary, a committee member recommended that the OEHHA-based cancer value of 0.2 $\mu g/m^3$ be used as the ABC for formaldehyde. The IRIS URE results, when converted, in a lower value of 0.08 $\mu g/m^3$, but is based on the same study that OEHHA reviewed. It is committee policy to choose the higher (less stringent) protective value when two different value choices are based on the same study. The OEHHA concentration of 0.2 $\mu g/m^3$ is about an order of magnitude more stringent than the current ABC of 3 $\mu g/m^3$.

The committee voted unanimously to recommend an ABC of 0.2 $\mu g/m^3$ for formaldehyde.

12 n-Hexane

The compound n-hexane is known to have non-cancer effects. The current ABC for n-hexane is 7,000 $\mu g/m^3$, which is based on the 2000 OEHHA Reference Exposure Level. N-hexane is a simple mid-length carbon chain used to extract vegetable oils from seeds, such as sunflower seeds or safflower seeds. It's also used as an industrial solvent, and is a common component of gasoline. When gasoline is spilled, the n-hexane in it will volatilize to air. This chemical also has neurological effects, with the critical endpoint in most studies being peripheral neuropathy.

The IRIS Reference Concentration of 700 $\mu g/m^3$ for n-hexane is the most recent toxicity value available, and is based a study done by Huang et al. 1989, using four dose groups of eight mice each. The mice were dosed for 12 hours a day, seven days a week, for 16 weeks. The study measured biochemical endpoints, including changes the in nervous-system-specific proteins neuron-specific enolase and beta S-100. The researchers weren't sure what to do with this information, so the toxicological point of departure they chose to use was based on the velocity of motor neuron conductance. All three studies' toxicological points of departure were similar, at concentrations of 204, 205, and 215 milligrams per cubic meter. But due to differing application of uncertainty factors, the three studies identified different toxicity values. There was general agreement among the committee members that the IRIS number was the most credible toxicity value of the three.

Researchers who were very active in toxicology labs back in the1960s and 1970s, and who used n-hexane before its toxicological properties were fully understood, worked with n-hexane without using gloves. Many of them developed very painful peripheral neuropathy, and attribute it dermal absorption of n-hexane during laboratory work.

The committee voted unanimously to recommend 700 $\mu g/m^3$ as the ABC for n-hexane, which is an order of magnitude more stringent than the current ABC for n-hexane of 7,000 $\mu g/m^3$.

Attachment B – Summary of ATSAC Consensus Recommendations on Benchmark Revisions

13 Hydrogen cyanide

Hydrogen cyanide is known to have non-cancer effects, and the current ABC for this compound is 9 µg/m^3. It is a colorless or pale blue liquid or gas above 78 degrees Fahrenheit, and is known to have a bitter almond-like odor. Hydrogen cyanide is used in gold and silver mining, and electroplating of those metals. The key epidemiology studies of n-hexane came from evaluation of electroplate workers in Egypt.

Hydrogen cyanide is also present in vehicle exhaust, tobacco smoke, and wood smoke and in fact appears in the Oregon Emission Inventory at fairly high mass amounts. In terms of mass, hydrogen cyanide is the 20th highest mass amount emitted, attributable to structural fires and to open burning, including residential open burning. The pits found in some fruits, such as apricots and chokecherries, contain hydrogen cyanide, and if eaten, can cause death in humans. Burning cigarettes and the resulting smoke also contain hydrogen cyanide. Fire fighters face being poisoned, depending on the type of fire they are fighting.

In 2005 the EPA IRIS Reference Concentration of 3 µg/m^3 and the OEHHA Reference Concentration of 9 µg/m^3 were available; the ATSAC chose the OEHHA number of 9 µg/m^3 as the ABC for hydrogen cyanide. The committee chose the OEHHA Reference Concentration because it was the more recent of the two available Reference Concentrations in 2005.

Since then, EPA has reviewed their 1993 Reference Concentration of 3 µg/m^3. Currently, both the IRIS and OEHHA are basing their Reference Concentrations on the same study, which was a study that looked at thyroid effects and iodine uptake from 36 male workers at three Egyptian electroplating facilities. The worker exposure durations in this case ranged from three to five years, which is more of a sub-chronic timeframe, although one person in the study was exposed for up to 15 years. The resulting number from this study identified a NOAEL of 7.1 milligrams per cubic meter, which became the point of departure for both the IRIS and OEHHA studies, but the two agencies applied different total uncertainty factors of 3,000 and 300, respectively. So the question is: Does EPA's application of an additional uncertainty factor warrant a reconsideration of the EPA benchmark as a potential ABC for hydrogen cyanide?

A lengthy and complex discussion by the committee of the reasons for each uncertainty factor ensued, including discussion of hydrogen cyanide effects on pregnant women; evidence that subclinical hyper-thyroid rates cause deficits in offspring; the fact that cyanide causes a deficit in thyroid function, and a deficit in thyroid function can lead to health outcomes, *ipso facto* cyanide becomes the link to adverse health outcomes; the fact that the toxicological point of departure is based on impacts to healthy male workers rather than pregnant women; that the ATSAC committee is charged with protection of vulnerable sub-groups, which would include women and fetuses; the possibility that the hypothyroidism induced by hydrogen cyanide exposure may be a different "flavor" than the hypothyroidism that the other studies are based upon, which may be related to endocrine disruption.

The two choices that the committee considered included either retaining the current ABC for hydrogen cyanide or accepting EPA's value, which is based on the use of an additional uncertainty factor of 10. Use of the additional uncertainty factor of 10 would account for the uncertainty around known effects to hypothyroidism outcomes and the potential that higher exposure could contribute to those. A suggestion was made to recommend 0.8 µg/m3 as the ABC for hydrogen cyanide, and the committee felt that the suggestion was supportable because

it is likely that a larger database on the effects of hypothyroidism and intergenerational effects due to hydrogen cyanide exists than is represented by the three studies discussed. The rapidly growing endocrine disruption literature would be expected to contribute additional studies to back up the committee's confidence in this mechanism occurring during pregnancy and *in utero*.

The ATSAC unanimously recommended to revise the current ABC of 9 $\mu g/m^3$ to 0.8 $\mu g/m^3$, which would make the ABC consistent with the value from EPA IRIS.

14 Hydrogen fluoride and fluorides

Hydrogen fluoride is known to have non-cancer effects. The current ABC for hydrogen fluoride is 14 $\mu g/m^3$, which is based on a 2003 OEHHA Reference Exposure Level. Fluoride is referred to in different ways by different sources. *Fluorine* is a gas that is exceptionally toxic and reactive. A person would encounter fluorine gas only in an industrial situation, or perhaps through an accident of some kind. Exposure to this gas would almost certainly result in death.

Fluorine, although highly toxic, doesn't exist in the gaseous form for very long. The gas form rapidly combines with water, hydrogen, or methane in the atmosphere to produce *hydrogen fluoride*. It is important to keep track of what type of fluoride is being discussed by using the Chemical Abstracts Service (CAS) numbers, which are specific to each type of chemical. The current ABC is specific to hydrogen fluoride. Hydrogen fluoride can exist as a gas or as *hydrofluoric acid*; in fact, they have the same CAS numbers. There are other types of fluoride compounds, such as fluoride-chlorine compounds. Sodium fluorides are relatively water-soluble, and is released from aluminum filters. Fluorine's toxicity comes from the fact that fluorine displaces calcium in the blood, and calcium is necessary for proper heart action. Thus, the presence of fluorine in the human body causes heart failure and death. However, the compound sulfur hexafluoride is completely inert and does not cause damage, and in fact is used in cataract surgery to seal wounds.

Fluoride exists as an anion, and combines with other ions to form stable compounds, and is present in toothpaste and drinking water; it has its own CAS number. Fluorine, on the other hand, is an element, and as mentioned previously, is highly reactive and toxic. Fluorine is the entity in fluoride compounds that causes toxic responses. As stated earlier, fluorine displaces calcium in the blood, leading to heart problems. It also displaces calcium during bone formation, which leads to one clearly recognized health effect from fluoride, which is skeletal fluorosis, indicating damage to joints and bones. The measurement of fluoride in air is restricted to the technical ability to measure the fluoride anion. Among all the different fluoride compounds, the only measurable type is the fluoride anion.

One state program exists which has separate benchmarks for many different fluoride compounds, based on a compound equivalency approach. This is done simply by evaluating the molecular weight ratio of the fluorine (the toxic component) in each compound versus the molecular weight of everything else in the compound. However, this method does not address the fact that many of the compounds for which benchmarks were generated are not soluble, and solubility facilitates the toxic effects. Nor does this method address the fact that in some cases, the reactive gases don't last long enough in air to be a health problem. Thus, it is important to regulate the fluoride anion, because its availability in air is what drives toxic responses.

Hydrogen fluoride makes up about 35% of the fluoride present in the atmosphere on a global basis. In the presence of water, hydrochloric acid tends to form aqueous hydro fluoride, which

exists as an aerosol. It can be "scrubbed" out of the atmosphere by wet deposition --for example, rainfall -- or through dry deposition. The aerosol form lasts about 12 to 14 hours after it's been emitted; so it's not a long-lasting compound, and thus not of much concern.

Twenty-four percent of the fluoride in the atmosphere is actually in the form of fluoride particles. If a person were standing next to an operating primary smelter, the smelter would be emitting particles of sodium fluoride. These large particles can condense and dry-deposit out of air onto other media, or can be "scrubbed out" of air if moisture is present. So under dry conditions, these particles last quite a while. But under wet conditions they last only about 50 hours. Because they don't have a long residence time in the atmosphere, they cannot travel very far.

Volcanoes and certain soils are natural sources of hydrogen fluoride. Fluorides in general are naturally- occurring compounds. Coal fire and electrical utilities are anthropogenic sources of hydrogen fluoride. One of these types of sources is still present in Oregon but will close soon. Another source of hydrogen fluoride is as emissions from primary aluminum smelters; but Oregon no longer has any of these facilities in residence.

Because fluorides are naturally occurring, there are ambient levels of fluorides present even in isolated areas at a concentration of about 0.05 $\mu g/m^3$. Within most urban areas, the concentration is between 0.01 or 0.005 $\mu g/m^3$ and 1 or 2 $\mu g/m^3$. In heavily industrialized areas, the concentration can be present at 2 or 3 $\mu g/m^3$, and in some cases at 7 to 10 $\mu g/m^3$. However, a person gets fluoride in their system primarily from food consumption. The tea plant is one of the few plants that can accumulate fluoride; therefore ingestion of large amounts of tea includes ingestion of fluorides.

There is not any clear epidemiological evidence that fluoride causes cancer. EPA hasn't evaluated fluoride, and the International Agency for Research on Cancer has not classified fluoride as a carcinogen. Although people have asserted that fluoride causes a number of other diseases, there is no clear scientific evidence of that. Thus, the strongest evidence available for health effects relies on studies regarding skeletal fluorosis.

There are multiple protective values available for fluoride. All the different sources coalesce on the same value of 13 or 14 $\mu g/m^3$. In three cases, a value of 14 $\mu g/m^3$ is applied to hydrogen fluoride, with 13 $\mu g/m^3$ of this being attributed to fluorine. The State of Texas recently reassessed the Derryberry et al. (1963) results using a benchmark dose model, and established a chronic effects screening level of 27 $\mu g/m^3$, which is higher (less stringent) than the information from the other agencies discussed above. The ATSAC discussed recommending 13 $\mu g/m^3$ as the ABC for the fluoride anion, with the related critical effect being skeletal fluorosis. Related respiratory and pulmonary issues were also noted.

Washington State has adopted 14 $\mu g/m^3$ as its value for fluoride. Most regulatory agencies recognize a value around 13 $\mu g/m^3$ for the protection of human health. Many reviewers other than the members of the ATSAC have looked at this issue and settled near the same number. Also, the committee is considering choosing to set a benchmark for total fluoride anion in the air, regardless of its source, which allow use of a protective value for nearly any fluoride compound being emitted.

The fluoride anion is an inorganic form of fluoride, but that there are quite a few organic fluoride compounds which are not covered by the ABC currently being considered by the committee for

the fluoride anion. The parent compounds are not really what the committee is interested in, but rather the fluoride anion. Setting an ABC for the fluoride anion will be protective of any other fluoride compound where fluorine contributes a significant amount of toxicity.

With the more complicated fluoride organic compounds, inhalation problems are rare unless a person is exposed to a fire; rather, human exposure to organic fluoride compounds tends to occur through exposure to ground water or soil contamination. In regard to impacts to human health through the inhalation pathway, these chemicals don't appear to be a concern, based on the current state of the science. The Emissions Inventory report lists fluoride-related compounds at about 9 tons per year (as compared to 30 tons of hydrogen fluoride), which are emitted from residential wood stove combustion, and predominantly from non-EPA-certified wood stoves.

The committee voted unanimously to discard the current ABC for hydrogen fluoride (which is 14 $\mu g/m^3$) and set a new ABC based on the fluoride anion of 13 $\mu g/m^3$.

15 Hydrogen sulfide

Hydrogen sulfide is known to have non-cancer effects, and the current ABC for hydrogen sulfide is 2 $\mu g/m^3$, which is based on 2003 IRIS Reference Concentration. There's been no change in available toxicity information for hydrogen sulfide since the standing ABC was chosen circa 2006. ATSDR did put out a revised value, but it applies only to intermediate, rather than chronic, exposure. Thus, the existing ABC is based on chronic exposure to hydrogen sulfide, and is also much lower (more stringent) than the ATSDR value. Because there is no new, applicable toxicity information for hydrogen sulfide, the committee recommended that the current ABC of 2 $\mu g/m^3$ for hydrogen sulfide be retained.

16 Lead

Lead is known to have non-carcinogenic effects, and there is some evidence that it may also have cancer effects. The standing ABC for lead is 0.15 $\mu g/m^3$, which is consistent with the National Ambient Air Quality Standard for lead, defined as a Criteria Pollutant under NAAQS. The Clean Air Act directive is to protect public health with an adequate margin of safety that is protective of the most sensitive population groups. In the case of lead, the relevant sensitive population group is children under 5 years of age, including fetuses.

Exposure to lead *in utero* and during the early years of life causes impairment of neural development and decreased mental functional capacity. In later years, associations with impaired academic performance and ADHD have been reported, and these effects persist into adulthood. Impaired neurodevelopment and functioning is the most sensitive endpoint for exposure to lead, and these effects have been demonstrated in multiple studies, so there is a high confidence in a causal relationship.

Children's hand-to-mouth behavior significantly increases their exposure to lead; this route of exposure is not as large in older children and teenagers, so children who are about 5 years old and younger are of the most concern in regard to ingestion of lead. The deposition of inhaled lead particles in expected to be observed in the upper and mid-respiratory tract; from there, mucocilia remove lead from the respiratory tract and into the pharynx, where the particles trapped in mucous are swallowed. Therefore, the primary route of lead entry into a child's body is through the gastrointestinal tract.

Attachment B – Summary of ATSAC Consensus Recommendations on Benchmark Revisions

The Centers for Disease Control's lead *reference level* (new term in past three years that replaces "action level") is a blood lead level of 5 micrograms of lead per deciliter of blood, or µg/dL. The current reference blood lead level of 5 µg/dL replaces the former action level of 10 µg/dL. Recent epidemiological studies show detrimental effects below 5 µg/dL of lead, at 3 µg/dL.

Evidence for carcinogenicity of lead is limited, although it has been recognized by EPA as a B2 carcinogen (probable human carcinogen). Even so, the available cancer risk-based level for chronic exposure to lead is higher (less stringent) than the toxicity-based level of lead associated with adverse neurological outcomes in children. Therefore, basing the ABC for lead on non-cancer effects, rather than cancer effects, will protect against both types of effects

Malnutrition can be related to lead exposure, because the valence of lead is +2, which is the same valence state as calcium. Therefore, certain metabolic pathways, such as the active transport mechanisms in the epithelium of the small intestine, selectively absorb lead as well as calcium; thus, the body uptakes lead under these conditions, which further adversely impacts health. A similar physiologic state exists in pregnant women, who more actively absorb calcium and therefore, also absorb lead from their gastrointestinal tracts. Furthermore, the metabolic characteristics associated with pregnancy mobilize calcium from maternal bone stores, which also releases previously sequestered lead, which can then cross the placenta and impact the fetus.

The EPA focused their lead modeling effort on the air concentration (µg/m^3)-to-blood (µg/dL) ratio in developing its analysis for rulemaking. Ideally, EPA would set air-related lead exposure concentrations that would allow no loss of IQ in the population. However, neurodevelopmental impacts to children are predicted at all levels of population exposure; therefore the air concentration would have to be zero to prevent any loss of IQ points in the population. At 0.15 µg/m^3, which is the level of the current NAAQS and the ABC, EPA models estimate an average loss of 1 to 2 IQ points in the subset of children in the upper tail of the distribution exposed to a lead concentration of 0.15 µg/m^3. In rulemaking, the EPA Administrator accepted this level of estimated harm.

In its modeling, the EPA relied upon the critical epidemiologic studies by Bellinger, Canfield, Lanpher, and Tellez-Rojo, who demonstrated quantifiable lead effects below the blood level of 5 µg/dL. These studies show losses of 1.5 to 2.5 IQ points per µg/dL blood lead for children with blood lead levels in the range of 0.5 to 9 µg/dL. As a point of comparison, current NHANES data indicate the 5 µg/dL blood lead concentration represents the 97.5th percentile for US children ages 1-5 years old. California's *LeadSpread* model considered particulate phase-to-blood level-to-IQ points and estimated an IQ decrease of 1 to 3 points at the level of the federal NAAQS; similarly, a loss of 1 to 3 IQ points is associated with the most-exposed populations.

The air concentration of 0.15 µg/m^3 was established in 2008 as the NAAQS for lead. The ATSAC followed suit in 2008 by establishing an ABC of 0.15 µg/m^3, which was more stringent than the previous ABC for lead of 0.5 µg/m^3.

In remote areas, an air lead concentration of 1 nanogram per cubic meter ng/m^3 is present, identified specifically in an Arctic (pristine) remote-region study; 1 nanogram per cubic meter is equivalent to 0.001 µg/m^3. Therefore, the ABC for lead of 0.15 µg/m^3 is 150 times the remote-region background concentration of lead in air. Oregon air data indicates that lead levels in air statewide in Oregon, as estimated, are well below the ABC of 0.15 µg/m^3.

Attachment B – Summary of ATSAC Consensus Recommendations on Benchmark Revisions

The ATSAC doesn't have the resources to carefully evaluate other studies in the scientific literature that might support a more-stringent standard for lead. Identifying any concentration threshold other than zero (0) will always leave a portion of the population unprotected (i.e., in terms of a possible loss of 1 to 3 IQ points). Note that lead levels monitored in air are much lower than the benchmark for lead. Nature itself produces some lead, so it is infeasible to decrease lead levels to zero, outside of any industrial emissions. The reality is, it would cost a lot of money to get the concentration of lead in air to zero, and it simply might not be possible technically.

One ATSAC member felt that the committee should retain 0.15 $\mu g/m^3$ as the ABC for lead, as it represents the best available scientific and technical evidence, although there is a trade-off in terms of lowered IQ points for people who are exposed to this concentration. This is an acknowledged compromise position. Alignment with the federal NAAQS for lead makes sense, based on the ATSAC's limited resources and subsequent inability to conduct independent research analysis and modeling.

If the ATSAC was to arbitrarily divide the ABC for lead by 2 or 5 or some other arbitrary safety factor in order to make the ABC lower (more stringent), it would be impossible to provide a legally defensible reason why the ATSAC chose to do this, and in fact the time it would take to go through that justification process would take funds away from regulatory efforts that are actually protecting people.

The ATSAC was unable to locate more-recent high quality epidemiologic studies that would change the interpretation of the adequacy of the EPA analysis and rulemaking. However, if any new peer-reviewed information becomes available, then the ATSAC could use it to re-evaluate the ABC for lead at that time. It was the consensus of the committee to recommend retaining the standing ABC for lead of 0.15 $\mu g/m^3$, in alignment with the federal NAAQS.

17 Manganese

Manganese is known to have non-cancer effects. The original ABC for manganese of 0.2 $\mu g/m^3$ was set in 2006, and revised to 0.09 $\mu g/m^3$ in 2009 to be consistent with the 2008 OEHHA Reference Exposure Level for non-cancer effects. The ATSAC at that time was focusing on sensitive subgroups, specifically children.

The central nervous system and the lungs are affected by exposure to manganese. The central nervous system is the primary target, and exposure to manganese is related to the development of Parkinson's disease, as well as a variety of psychiatric and motor disturbances, collectively termed manganism.

LOAELs for manganese were obtained from a published study of workers exposed to high levels. Manganese is regarded as a toxic compound, but it is also an essential nutrient and trace element, so some level of manganese uptake is critical for good health. Therefore, the main concern with potential health risks should be related to chronic over-exposure to manganese.

EPA critical studies for manganese include two studies and an additional analysis performed by Roels et al. in the mid-1980s and early 1990s. Since then, no new studies based on exposure of humans to manganese have become available.

There are two ways to estimate toxicity effects from manganese: 1) Look at the occupational epidemiology results first, then down to extrapolate population exposure; or 2) use

physiologically-based pharmacokinetic models to estimate the dose received at the target site of action in the brain, understand what the dose is at the target site, and then relate this dose to observed adverse effect levels of exposure. Either way, significant assumptions are necessary to include in the calculations, which injects a large amount of uncertainty regarding decisions made based on either of these two protocols.

In the ATSAC chair ' s opinion, the ATSAC should continue to rely on epidemiology studies, including the critical epidemiology studies conducted by Roels, which looked at exposure to respirable dust in the form of manganese dioxide. A report on manganese toxicity submitted by an outside party to the ATSAC was based on the use of a physiologically-based pharmokinetic model, which the chair considered too is questionable to consider.

In 1992, the related lowest-observed-adverse-effect level for manganese was estimated as 0.05 $\mu g/m^3$. In 1997, the same authors came up with a different estimate of 0.34 $\mu g/m^3$. Since then the median concentration of the two studies, 0.15 $\mu g/m^3$, is the value that has been relied upon to represent a LOAEL related to manganese exposure effects of short-term memory, hand tremor, and reaction time. In 2006, set the ATSAC ABC for manganese at 0.2 $\mu g/m^3$; then ATSAC revised it in 2009 to 0.09 $\mu g/m^3$ in order to be consistent with the OHHEA Reference Exposure Level, which specifically applied an uncertainty factor to protect children.

The consensus of the committee was to retain the standing ABC of 0.09 $\mu g/m^3$ for manganese.

18 Mercury (elemental)

Elemental mercury is known to have non-cancer effects. The current ABC for elemental mercury is 0.3 $\mu g/m^3$, which is based on the 1995 EPA IRIS Reference Concentration. Currently, there are two Reference Concentrations for elemental mercury available to the committee for consideration. However, both values have the same target health endpoints, are based on the same critical study, and on the same toxicological point of departure; but then OEHHA added another uncertainty factor of 10 to the mix.

In the case of mercury, the route of exposure is important, because mercury exists in many different forms, and each form has a critical route of exposure. In regard to inhalation of elemental mercury, the most toxic component is inhalation of the vapor that comes from elemental mercury. Mercury is neurotoxic, and can also damage kidneys and the immune system. Organic forms of mercury, methylmercury in particular, are created when inorganic mercury enters an aquatic environment, where microbes can convert inorganic mercury into organic methylmercury through methylation. Once mercury is methylated, it is able to biomagnify up the food chain. Many of the fish advisories in Oregon are based on methylmercury concentrations that accumulate in the top tier of predators in the aquatic environment, such as large game fish. Methylmercury is also readily absorbed into nervous system tissue and the brain. The critical endpoint is neurodevelopmental damage to developing fetuses, which can occur when pregnant mothers eat fish tissue containing methylmercury, or are otherwise exposed to this compound. The inorganic salt forms of mercury affect the kidneys and the immune system, rather than the brain.

The form of mercury in air to which most people are likely to be exposed is elemental mercury vapor. In 2010, the ATSAC reviewed the same values that are currently available for mercury. An EPA IRIS Reference Concentration of 0.3 $\mu g/m^3$was used as the basis of the current ABC for elemental mercury. The ATSDR has a chronic Minimal Risk Level of 0.2 $\mu g/m^3$ for elemental

mercury. The Minimal Risk Level is based on one of the same studies in adult human workers as was utilized by EPA in IRIS, circa 1999. OEHHA has a chronic Reference Exposure Level of 0.03 $\mu g/m^3$ and this value is based on exactly the same set of studies. In the studies, occupational workers exposed to mercury were evaluated, and neurological toxicity endpoints such as hand tremor and memory disturbance were assessed. Hand tremor and memory disturbance are classical signs of mercury vapor neurotoxicity in an occupational setting.

Thus, all three agencies used the same studies and the same point of departure to come up with their respective but differing protective values. However, OEHHA applied an additional uncertainty factor to account for the potential for young children to have increased vulnerability to mercury exposure as their brains go through development; this mechanism was assumed to happen in the same way that it would occur if children were exposed to methylmercury. In 2010, the ATSAC had all the same information in front of them, but decided to not adopt the OEHHA value, because the committee felt that the additional uncertainty factor of 10 was excessive.

Individual variability is usually broken down into four toxicokinetic differences between people and three toxicodynamic differences between people. So the term toxicokinetic refers to how and where the compound moves in the body: how the chemical is taken up, how it's metabolized, where it moves to; and how it's excreted. Toxicodynamics refers to the damage that's caused while the chemical is at the site of action in the body. Therefore, based on the toxicological information used by OEHHA. OEHHA is saying that a standard uncertainty factor of 10 is insufficient to cover the potential toxicodynamic risk related to exposure of a pregnant adult worker the related effects to her developing fetus.

A detailed discussion followed about how each uncertainty factor was applied by EPA and by OEHHA to come up with their separate values for mercury. Fairly sophisticated modeling conducted by DEQ lead in 2010 regarding the potential multi-pathway effects of mercury resulted in the ATSAC choosing the 1995 EPA IRIS Reference Concentration of 0.3 $\mu g/m^3$ as the ABC for elemental mercury.

The 2010 modeling included estimates of overall mercury intake from air at 20% to 40% contribution, with 80% of the air contribution being due to global transport. The 2010 decision on the ABC for mercury assumed that other exposure pathways, such as fish consumption after methymercury biomagnifies in fish tissue, are not as significant as was assumed then. Elemental mercury itself first has to undergo methylation to become methylmercury, and then that methylmercury has to be taken up in the food chain before it impacts humans. Another committee member said that this committee previously discussed the fact that there is a relationship between the amount of elemental mercury in air and what shows up in fish tissue consumed by humans.

The assumption with methylmercury is that it has a toxicodynamic difference between how it affects adults versus how it affects the developing fetus; and elemental mercury vapor exposure can be considered to have a similar mechanism because it also affects the brain. Researchers have assumed that inorganic elemental mercury travels to all the same areas in the body that organic methylmercury does. One committee member countered by saying that elemental (inorganic) mercury vapor does not cross the blood brain barrier, and is excreted unchanged, for the most part, from the body.

The ATSAC unanimously decided that because the scientific literature on mercury effects is voluminous and technically comprehensive, the current ABC of 0.3 µg/m^3 for elemental mercury should be retained.

19 Methanol

Methanol has non-cancer, developmental effects. The existing ABC of 4,000 µg/m^3 for methanol is based on an OEHHA 2000 Reference Exposure Level. Draft guidance published by EPA in 2010 suggests that IRIS will soon revise its methanol Reference Concentration to 2,000 µg/m^3, which is two times more stringent than the standing ABC of 4,000 µg/m^3. A better protocol using the Benchmark Dose Model was used to obtain the new draft EPA value. But the new number is still provisional, and so far the ATSAC has opted not to recommend provisional values for chemicals, because the guiding principle is to work with protective levels already established by recognized scientific/regulatory bodies. Also, the 2014 Emissions Inventory listed an emission rate state-wide for methanol of 8,000 tons/year, so the existing ABC of 4,000 µg/m^3 is still protective.

The ATSAC voted unanimously to retain the ABC for methanol of 4,000 µg/m^3.

20 Methyl chloroform / 1,1,1-trichloroethane

Methyl chloroform, also known as 1,1,1-trichloroethane, is known to have non-cancer effects. Methyl chloroform is used as a solvent, and is a colorless liquid with a fairly high vapor pressure. The current ABC for methyl chloroform is 1,000 µg/m^3, which is the OEHHA 2000 Reference Exposure Concentration. Back in 2006, when the committee chose the current ABC based on the OEHHA value, there was no IRIS Reference Concentration available, so the committee chose the 2000 OEHHA Reference Concentration as the ABC for methyl chloroform. In 2007, IRIS published a Reference Concentration for methyl chloroform of 5,000 µg/m^3, based on histopathologic changes in the liver in rats exposed over a two-period, which is consistent with results from human epidemiological studies. All epidemiological studies identified effect endpoints that are associated with decreases in cognitive response.

In addition, the experimental literature suggests that chronic exposure to methyl chloroform induces hepatocellular hypertrophy at concentrations of 1,370 to 1,460 milligrams per cubic meter, and that these effects do not appear to progress in severity or incidence with exposure duration and are considered a physiological rather than adverse response. Methyl chloroform is not a particularly toxic compound.

It is unlikely that monitored concentrations of 1,1,1-trichloroethane in Oregon statewide will exceed either of the Reference Concentrations available for this chemical. There is only a difference of a factor of five between the two values, which is less than a difference of one order of magnitude.

One committee member mentioned that some of the technical information available for this chemical indicates that the chronic studies point to a protective target value that is even higher than the 5,000 µg/m^3 value from IRIS. But at the time these studies were performed, a value protective of acute exposures was found to be lower (more stringent) than the protective chronic level; so the researchers basically lowered the chronic level down to match the acute level, because it seemed counterintuitive that chronic exposure would cause lesser effects than acute

exposure. Acute exposure levels are nearly always less stringent than chronic exposure levels, due to the shorter exposure duration that occurs with acute exposure.

Another committee member said that two studies were conducted for 1,1,1-trichloroethane to look for cancer outcomes. But the rats used in the studies didn't survive long enough to determine whether or not cancer would occur, so 1,1,1-trichloroethane was deemed a Class D carcinogen because the data was considered inadequate.

The committee voted unanimously to recommend a new ABC of 5,000 $\mu g/m^3$ for methyl chloroform/1,1,1-trichloroethane.

21 Methylene Chloride

Methylene chloride, also known as dichloromethane, has both cancer and non-cancer effects, but cancer is the most sensitive endpoint for methylene chloride. The standing ABC for methylene chloride is 2.1 $\mu g/m^3$, which is based on the 1997 EPA IRIS URE value of 4.7×10^{-7} per $\mu g/m^3$.

This chemical was recently determined to be a mutagenic carcinogen. The best toxicity data for methylene chloride comes from the 2011 IRIS reassessment, which utilized pharmacokinetic modeling of methylene chloride toxicity from animals to humans, at the tissue sites of action, which is considered by the ATSAC to be very credible information. The resulting URE for methylene chloride was 1×10^{-8} per $\mu g/m^3$, which converts to a protective value of 100 $\mu g/m^3$. The conversion is performed by dividing the acceptable risk limit of 1×10^{-6}, which is 0.000001 in decimal form, by the URE value.

The committee discussed the fact that methylene chloride is a recognized mutagen, meaning that early-life exposure results in a higher rate of cancer in children exposed from zero to two years and from three to 16 years of age, than for adults. However, incorporating this consideration into a recommendation for an ABC requires more than just adjusting a toxicity-based value, which the ATSAC has chosen not to do for mutagenic chemicals. Thus, the ATSAC will continue to use toxicity factors based on adult exposure in their review of appropriate toxicity information for ABCs.

The ATSAC unanimously recommended that the IRIS-based value of 100 $\mu g/m^3$ be used as the new ABC for methylene chloride, which is also consistent with the Agency for Toxic Substances and Disease Registry Minimal Risk Level and with the OEHHA Reference Exposure Level.

22, 23, 24Nickel and nickel compounds

Nickel and nickel compounds cause both cancer and non-cancer effects, depending on the specific compound. In 2005, the ATSAC adopted a single ABC of 0.004 $\mu g/m^3$ for nickel; comments in 2006 facilitated the adoption of ABCs for three nickel compounds instead: nickel refinery dust (ABC of 0.004 $\mu g/m^3$, based on cancer effects); nickel subsulfide (ABC of 0.002 $\mu g/m^3$, based on cancer effects); and "nickel compounds, soluble" (ABC of 0.05 $\mu g/m^3$, based on non-cancer effects of nickel acetate, nickel chloride, nickel carbonate, nickel carbonyl, nickel hydroxide, nickelocene, and nickel sulfate).

Nickel compounds can be separated into two general categories: (1) those that are insoluble in water are thought to be carcinogenic, and are generally emitted as particulates primarily from smelting, refining, and metals processing operations, such as nickel refinery dust, nickel subsulfide, nickel sulfide, and nickel oxide; and (2) those nickel compounds that are soluble in

water and which are thought to be non-carcinogenic or somewhat carcinogenic are generally emitted as aerosols primarily from nickel plating operations, such as nickel sulfate, nickel chloride, and several other nickel compounds.

In 2015, the ATSAC proposed that nickel be separated into insoluble and soluble compounds, with a separate ABC for each type, with specific recommendations including:

(1) Nickel refinery dust – drop this compound from the ABC list because it lacks a CASRN number and is an uncharacterized mixture of various nickel compounds, although nickel subsulfide is the primary nickel compound in nickel refinery dust.

(2) "Insoluble" nickel compounds, most likely emitted as particles and more carcinogenic than the "soluble" nickel compounds: Set the ABC for insoluble nickel compounds at 0.004 $\mu g/m^3$, based on the newer OEHHA 2011 value for nickel subsulfide. This ABC would encompass the following Ni compounds:

(a) Nickel subsulfide (CAS 12035-72-2)

(b) Nickel oxide (1313-99-1) - during the meeting, the committee discussed assigning this compound the same ABC assigned to soluble nickel compound, but a recent paper suggests that nickel oxide should remain in the category of insoluble nickel compounds. It's also more likely to be emitted during refinery/metal working processes.

(c) Nickel sulfide (11113-75-0) – this compound is listed by OEHHA list of Ni compounds, and placing this compound under the insoluble nickel category will provide Oregon with a better coverage of nickel compounds in general.

(d) Nickel metal (7440-02-0) – added because this nickel compound is also on the OEHHA list.

(3) "Soluble" nickel compounds, most likely emitted as aerosols (through operations such as nickel plating) and less carcinogenic than the "insoluble" nickel compounds. Proposal was made to set the ABC for soluble nickel compounds at 0.01 $\mu g/m^3$, based on the OEHHA 2011 Reference Exposure Level, and as supported by similar values established in other jurisdictions.

(a) Nickel acetate (373-20-4)

(b) Nickel chloride (7718-54-9)

(c) Nickel carbonate (3333-39-3)

(d) Nickel carbonyl (13463-39-3)

(e) Nickel hydroxide (12054-48-7)

(f) Nickelocene (1271-28-9) - an organometallic nickel compound.

(g) Nickel sulfate (7786-81-4)

(h) Nickel sulfate hexahydrate (10101-97-0) - added from the OEHHA list.

(i) Nickel nitrate hexahydrate (13478-00-7) - added from the OEHHA list.

(j) Nickel carbonate hydroxide (12607-70-4) - added from the OEHHA list.

The consensus of the committee was to recommend two new ABCs related to nickel: 0.004 $\mu g/m^3$ for insoluble nickel compounds, and 0.01 $\mu g/m^3$ for soluble nickel compounds**.** The committee also recommended that the ABC for nickel refinery dust be dismissed.

25 Perchloroethylene /Tetrachloroethylene

Perchloroethylene, also referred to as PCE or its chemical synonym tetrachloroethylene, has both cancer and non-cancer effects. The standing ABC for PCE is 35 $\mu g/m^3$, which is based on a 1991 OEHHA Reference Exposure Level for non-cancer effects. Back in 2005, the ATSAC acknowledged the carcinogenicity of PCE but also acknowledged that there was no clear evidence of carcinogenicity in humans at that time. Therefore, the ATSAC chose to assign an ABC to PCE based on its non-cancer effects at that time, pending better cancer potency information becoming available.

The EPA IRIS database values for PCE, updated in 2012, include both cancer and non-cancer toxicity values. When the IRIS cancer-based URE value of 2.6×10^{-7} per $\mu g/m^3$ is converted to a concentration which is protective to an excess lifetime cancer risk of 1×10^{-6}, the resulting value is 4 $\mu g/m^3$.

The IRIS URE cancer value is based on a weight-of-evidence approach using epidemiological and rat studies with a value derived from the $BMCL_{10}$, by dividing the risk [as a fraction] by the $BMCL_{10}$. A $BMCL_{10}$ is the lower bound on the 95 percent confidence limit of the10 percent response limit related to a benchmark concentration. To obtain a cancer slope factor, the extrapolation method used was a multistage model with linear extrapolation from the point of departure (i.e., $BMCL_{10}$), followed by extrapolation to humans using the physiologically-based pharmacokinetic (PBPK) model of Chiu and Ginsberg (2011).

The ATSAC made an initial recommendation to use 4 $\mu g/m^3$ as the new ABC for PCE, based on the IRIS URE value. However, one committee member was concerned because even though human epidemiological data is available for this chemical, which is typically considered the most credible form of toxicity data, a total uncertainty factor of 1,000 had nonetheless been assigned to the IRIS URE value. The committee then discussed exactly why each of the three uncertainty factors of 10 (which, when multiplied together, result in a total uncertainty factor of 1,000) had been applied in the study, but also decided that the ATSAC cannot arbitrarily remove uncertainty factors from the calculation of a value conducted by another agency. If the ATSAC chose to do that in this case, an undesirable precedent would be set for the toxicity information available for other chemicals the ATSAC is reviewing.

The ATSAC then unanimously agreed to recommend revising the ABC for PCE to 4 $\mu g/m^3$, based on the EPA IRIS URE value.

26 Phosgene

Phosgene is known to have non-cancer effects. DEQ asked the ATSAC to review the toxicity information for phosgene and identify an ABC, if appropriate. Phosgene is an infamous chemical used during World War I in gas attacks, and is an acutely toxic material. Phosgene is also an important precursor chemical for many compounds, particularly isocyanides. It is also used as a basis material for many other compounds, as well. There were only two available toxicology values for the committee to consider for phosgene: 1) an EPA IRIS Reference Concentration that was developed in 2008, and 2) a California EPA OEHHA Reference Concentration that was

derived a couple of years later. The EPA used a newer study conducted in 1997 as the basis of their number, while the OEHHA number is based on a 1985 study. There is a 10-fold difference between the EPA and OEHHA values, with the EPA value being the lower (more stringent) of the two.

There were no chronic studies identified for phosgene, so the committee has to rely on multiple sub-chronic studies that all seem to support each other's results. The critical study upon which the EPA IRIS number is based is from a sub-chronic in inhalation study done by Kodavanti, et al., in 1997, where rats were used as experimental subjects. No mortality was reported, but sub-chronic effects to the lung were documented. An increase in collagen staining in the lungs was observed, which is a good indicator for the presence of fibrosis.

The data was fit to a multi-stage model to obtain a BMDL (an acronym for a Benchmark Dose Level) of about 0.007 parts per million; then they applied a total uncertainty factor of 100, as did OEHHA. The total uncertainty factor of 100 is based on an uncertainty factor of 10 to account for human variation; an uncertainty factor of 3 to account of animal-to-human uncertainty; and an uncertainty factor of 3 to account for the uncertainty related to extrapolating a chronic value from a sub-chronic value. Using toxicology protocols, a total uncertainty factor of 100 is obtained, which results in a value of 0.3 $\mu g/m^3$ as the Reference Concentration. Two ATSAC members felt that this EPA number is more supportable than the number available from OHHEA. Although there is nothing fundamentally wrong with the OEHHA value, one of its limitations is that it was based on an acute duration exposure, whereas the EPA IRIS value is based on sub-chronic exposure. The study defined the chronic exposure data using rats and looking at an outcome of early scarring in the lungs, and this critical endpoint represents damage from chronic inflammation from this irritant. Phosgene is well-known to be a powerful inflammatory agent that affects mucous membranes and the respiratory tract, so the fibrosis-related scarring in rodents is a reasonable endpoint to use for phosgene.

The committee unanimously recommended that the ABC for phosgene be set at 0.3 $\mu g/m^3$, which is based on the IRIS Reference Concentration.

27 Phosphine

Phosphine causes non-cancer effects. It is a respiratory irritant and is used as a fumigant for crops and tobacco, meth labs, and semi-conductor manufacturing. The standing ABC for phosphine is 0.3 $\mu g/m^3$, which is based on the 1995 IRIS Reference Concentration related to a mouse study conducted by Barbosa, et al. The OEHHA Reference Exposure Level of 0.8 $\mu g/m^3$ is less stringent, and newer (2002), and is also related to the Barbosa et al. study, but uses additional uncertainty factors. Originally, the ATSAC rejected the OEHHA value because they did not accept the uncertainty factors applied by OEHHA.

As of January 2015, Reference Concentrations from IRIS and OEHHA had not changed. However, because the two values represent two different interpretations of the same study, it is ATSAC policy to choose the higher (less stringent) of the two values.

The ATSAC voted unanimously to recommend 0.8 $\mu g/m^3$ as the ABC for phosphine.

28 Polycyclic Aromatic Hydrocarbons (PAHs)

PAHs were discussed at the ATSAC meetings in June, July, and September of 2015, as was new toxicity information for the PAH benzo(a)pyrene in March 2017. PAHs are a group of chemicals

containing both carcinogenic and non-carcinogenic compounds. The standing ABC for total PAHs is 0.0009 $\mu g/m^3$, which is a cancer-based value obtained through conversion of the OEHHA 1999 URE value for benzo(a)pyrene of 1.1×10^{-3} per $\mu g/m^3$. In order to utilize this ABC in the evaluation of air sample results, the ABC was compared to the toxic-equivalency-factor-weighted sum of concentrations for 32 PAHs.

Five main concerns related to the toxicity of PAHs were emphasized during committee discussion at the June 2015 ATSAC meeting: 1) the fact that an index PAH exists, which is benzo(a)pyrene, and this PAH is used as the basis of toxicity equivalency factor calculations for total PAHs; 2) the cancer-based URE value used to identify an ABC for total PAHs is related to the index PAH, which is benzo(a)pyrene; 3) identification of which PAHs should be monitored and measured; 4) whether relative potency factors exist for all relevant individual PAHs, and are used to relate other PAHs to the index PAH; and 5) consideration of how source attribution might be assessed based on the composition and type of PAHs present in air samples.

EPA's IRIS recently looked at some new toxicity information, and then proposed a new URE value of 6×10^{-4} per $\mu g/m^3$ for benzo(a)pyrene in their 2014 External Review Draft document, a value which converts to a protective concentration of 0.002 $\mu g/m^3$. This conversion is calculated by dividing the acceptable carcinogenic risk, 10^{-6}, or 0.000001, by the URE value. This newly-published URE appears to indicate that the committee could now recommend the use of the related higher concentration as the ABC for total PAHs. However, in the intervening years, a subset of the PAHs have been recognized as having mutagenic properties, including the index PAH benzo(a)pyrene, which is the PAH by which other PAHs are judged in terms of relative toxicity. In these cases, EPA recommends that the UREs be age-adjusted based on mutagenic effects, which are theorized to impact children more drastically than adults. As a policy decision, the committee decided not to apply age-dependent adjustment factors (ADAFs) to the ABC for benzo(a)pyrene. As discussed earlier, the ABC for benzo(a)pyrene is used as the ABC for total PAHs. However, the committee recommended that ADAFs be used to assess toxicity of PAHs within the context of a human health risk assessment.

The current ABC value for total PAHs is based on the assumption that the mixture of total PAHs (to which the ABC applies) contains 32 PAHs. A committee member proposed that a revised ABC should be based on only 25 PAHs. Later on in the review process, corrections were made to this list that included removing the PAH perylene; adding the PAH pyrene, and adding the PAH anthracene which had been inadvertently left off the list, to bring the list to 26. The proposed list of 26 PAHs would include benzo(a)pyrene, which is one of two PAHs that are EPA-required for DEQ to monitor, along with naphthalene. However, naphthalene has its own ABC and so is not included in the list of total PAHs; 14 PAHs requested for monitoring by EPA, as was discussed by Anthony Barnack of DEQ at the May ATSAC meeting; and 10 PAHs that are specifically relevant to air which were obtained from the list provided by the Minnesota Department of Health. Please refer to Table B-1 for the proposed list of 26 PAHs and their related potency equivalency factors.

In 2014, the Minnesota Department of Health published a revised guidance document suggesting that the measurement of a list of 19 PAHs would be relevant for identifying PAHs emitted from air sources. Some of the PAHs included in the Minnesota Department of Health list are 30 to 50 times more potent (in terms of carcinogenicity) than benzo(a)pyrene. Minnesota goes beyond the

typical EPA list of 16 PAHs, which has served well for soil and sediment studies historically, but the 16 PAHs are not all relevant to PAHs in air.

The proposed list of 26 PAHs attempts to capture PAHs that are from different sources, whether they are internal combustion engines, wood stoves, ether oil boilers, or others. Thus, although the number of PAHs in the proposed list have decreased from 32 to 26, the 26 proposed PAHS are specifically relevant to air. In order to be able to relate the toxicity of each of the proposed 26 PAHs to the index PAH benzo(a)pyrene, published toxic equivalency factors (typically referred to as TEFs) for each of the 26 PAHS need to be identified. In the particular case of PAHs, TEFs are based on cancer potency, and are referred to as potency equivalency factors, or PEFs.

The 2010 EPA draft document and the Minnesota Department of Health provide PEFs for most of the PAHs in the proposed list. The concentration of each of the 26 PAHs is adjusted based on each compound's PEF, and then the adjusted concentrations are summed. That summed concentration of adjusted concentrations for 26 PAHs is then compared to the ABC for total PAHs.

For each PAH, a range of PEF values are available in Table 1, page xi of EPA's February 2010 draft guidance, which in the case of carcinogenic PAHs are also referred to as relative potency factors. A committee member proposed that the upper-bound PEF value from each range be used as the PEF for each PAH in the proposed list of 26. Use of the upper-bound PEF values would allow the use of values that estimate the maximum toxicity of the group of 26 PAHs, relative to benzo(a)pyrene. In some cases, as with the PAHs 5-methylchrysene and 6-nitrochrysene, the relative potency factor provided by Minnesota would be used, as it is the only available relative potency factor available. The Minnesota PEFs are each based on a single-point value, rather than on a range of values.

Previously, 15 PAHs were considered in the report published by the Portland Air Toxics Solutions (PATS) committee; naphthalene was considered separately from the other 15 PAHs. The PATS report indicated that wood smoke was the biggest contributor to PAHs in the Portland Air Toxics Solution work.

The committee has not yet considered nitro-PAHs and oxygenated PAHs, and it would be good to have an extra protective factor for these types of PAHs at some point. The committee may want to augment the proposed list of PAHs list with nitro-PAHs and oxygenated PAH in the future, if more information on their toxicities becomes available in the future.

The committee reviewed the history behind the original list of 32 PAHs, and chose to recommend that the list be revised to reflect 26 PAHs that are most relevant to air. All 26 PAHs other than benzo(a)pyrene will be adjusted using PEFs prior to being compared to the ABC for total PAHs. The committee considered application of ADAFs to PAHs and decided that this approach was not appropriate. In addition, the committee reached a consensus to retain the current ambient benchmark concentration for total PAHs of 0.0009 $\mu g/m^3$.

Attachment B – Summary of ATSAC Consensus Recommendations on Benchmark Revisions

At the September 2015 ATSAC meeting, Sue MacMillan, DEQ lead for the ATSAC, discussed the fact that a decision to use the upper-bound value of a range of PEF values had been approved by the ATSAC at the previous meeting. She requested that the average value of each of the PEF ranges be used rather than the upper-bound value, and explained that using the average value of each PEF range will make the ATSAC toxic equivalency factor protocol consistent with how EPA and other state agencies use this information, including DEQ's Cleanup Program. The result of using an average rather than the upper-bound TEF value for each PAH is a slightly lower summed concentration once all the PAHs are adjusted, which in turn means that the total PAH concentration is slightly less likely to exceed the ABC. But this is the approach most other agencies use. The committee chair asked whether this change would only alter the way in which PAH sample results would be calculated, but not alter the ABC itself. She responded that the ABC that the ATSAC voted unanimously to accept at the July 2015 ATSAC meeting would not change.

The committee unanimously voted to accept the proposed change of choosing PEFs based on the averages of ranges, rather than the upper-bound values.

The draft cancer-based URE value of 0.0006 per $\mu g/m^3$ for benzo(a)pyrene proposed in 2010 EPA guidance became final in IRIS in January 2017, and so was discussed by the ATSAC at their March 2017 meeting. Use of this URE value would result in an ABC of 0.002 $\mu g/m^3$, as discussed earlier. Because the benzo(a)pyrene toxicity value serves as the basis for calculating the carcinogenicity of total PAHs using a PEF approach, the toxicological information for benzo(a)pyrene is critically important. Benzo(a)pyrene has not been assigned its own ABC; rather, benzo(a)pyrene is used as the basis of the ABC for total PAHs, as discussed above. In response to a committee member's question about the non-carcinogenic effects of benzo(a)pyrene, it was explained that if the more-stringent toxicity value that is based on cancer effects is used, then that same value would automatically be protective of non-cancer effects as well.

David Farrer, a committee member from the Oregon Health Authority, said that he had looked at the January 2017 benzo(a)pyrene information especially carefully, and found the new number to be a much newer number, and one that uses a much more modern method, that is, a benchmark dose, a benchmark concentration. He favors the new number.

The standing ABC for total PAHs is 0.0009 $\mu g/m^3$, which rounds up to the current ABC of 0.001 $\mu g/m^3$, The new ABC value of 0.002 $\mu g/m^3$ would not be much different, and both values are based on the same study by Thyssen et al. (1981). The committee discussed at some length whether to retain the current ABC for total PAHs and wait until the next assemblage of the ATSAC in 2020 to make a decision to use the new EPA IRIS number, especially since the two numbers don't differ by very much. Instead, the committee decided to use the new IRIS number as the ABC for total PAHs, because IRIS had utilized all the all available data from the Thyssen study in its calculation of a URE value, while the 1999 OEHHA value was based on only a portion of the study data.

The committee voted unanimously to use the new IRIS-based protective concentration of 0.002 $\mu g/m^3$ for benzo(a)pyrene as the new ABC for total PAHs.

29 n- Propyl Bromide

Attachment B – Summary of ATSAC Consensus Recommendations on Benchmark Revisions

DEQ requested that the ATSAC review toxicological information for n-propyl bromide and identify an ABC, if appropriate. This chemical has recently been considered by the dry cleaning industry as a possible less-toxic replacement for their use of perchloroethylene (also called tetrachloroethylene). N-propyl bromide has been a relatively obscure industrial solvent used for degreasing and cleaning. There is no extensive toxicological evaluation required for the use of commercial chemicals, prior to their release into the marketplace.

N-Propyl bromide was put into commercial use as an industrial degreasing agent 25 years ago. There was a minimal amount of toxicology evaluation done prior to this. Later, when perchloroethylene and Stoddard Solvent became challenged as safe dry cleaning fluids, the industry began considering the use of alternative chemicals.

The dry-cleaning industry's requirements for the type of dry cleaning fluid to use are specific to cleaning clothes, such as identifying a chemical that specifically cleans clothes well without destroying them or leaving residue behind. Thus, they identified n-propyl bromide as a potentially good dry-cleaning fluid. N-propyl bromide was then put into commercial use under a couple of different trade names, for example "DryFall" and "Fabersall".

Then the National Toxicology Program evaluated n-propyl bromide and discovered that it had non-cancer chronic effects, primarily damaging the central nervous system. There was also some evidence that n-propyl bromide might be a carcinogen. Nonetheless, the producers of the n-propyl-bromide-based chemicals continued to claim that the chemicals weren't toxic.

The National Toxicology Program again evaluated n-propyl bromide, which, according to their studies, turned out to be a really powerful reproductive toxicant. Also, over the past couple of years, n-propyl bromide has been identified as "reasonably anticipated to be a carcinogen", and is regulated by the European Union through the Registration, Evaluation, Authorisation and Restriction of *Chemicals* program. New York State has banned the use of n-propyl bromide.

The committee tried to find toxicological information for n-propyl bromide, but discovered that there wasn't much available. Initially, the committee found a chronic health-based protective value of 20 $\mu g/m^3$ from the Minnesota Department of Health, related to non-cancer lesions in the respiratory system from exposures, using rats and mice. But the following year, Exponent (a well-regarded consulting firm that works only for commercial private clients) published a report on n-propyl bromide. The report stated that n-propyl bromide was a carcinogen, and a related URE value of 2.1×10^{-6} per $\mu g/m^3$ was calculated by Exponent. When the URE is converted to a concentration usable as an ABC, it results in a value of 0.5 $\mu g/m^3$. A committee member suggested that the cancer-based URE calculated by Exponent be used to calculate an ABC of 0.5 $\mu g/m^3$ for n-propyl bromide.

Exponent primarily used studies conducted in 2011 by the National Toxicology Program to identify their URE value for n-propyl bromide. The National Toxicology Program is a premier program, recognized as producing highly credible study results. Thus, the basis of the Exponent work is expected to be credible.

The committee discussed the potential for a conflict of interest in regard to Exponent estimating the URE for a paying client. The committee chair had dealt with Exponent epidemiologists directly on other issues, and found them to be very good, highly qualified scientists, but stated that they do represent their clients' interests vigorously. Many of the epidemiologists and toxicologists at Exponent have published in the peer-reviewed scientific literature, which lends

credibility to their work. Also, with Exponent being a client advocate, they would be expected to come up with a URE that resulted in a much higher (less stringent) value than the 20 $\mu g/m^3$ listed by the Minnesota Department of Health – but they didn't, Instead, they came up with a much lower (more stringent) number, and this fact makes the Exponent value a little more trustworthy.

No estimates of emissions for n-propyl bromide in Oregon were give in the recently-published data from the 2011 National Air Toxics Assessment inventory, nor was there any relevant information for n-propyl bromide available in the Oregon Emissions Inventory. DEQ explained that the reason they had requested that the ATSAC review n-propyl bromide was because it appeared that dry cleaning facilities might be deciding to use this chemical in their operations, and a protective number would be needed.

Sarah Armitage, senior DEQ air quality planner, stated that based on coordination with the state dry cleaner program, there is at least one dry cleaner in Oregon using n-propyl bromide in their operations, and there may be others. The dry-cleaning industry is now aware that n-propyl bromide is not the perfect replacement chemical they hoped for, and so seem to be moving on and considering other chemicals.

The ATSAC voted unanimously to recommend an ABC of 0.5 $\mu g/m^3$ for n-propyl bromide.

30 Selenium

At the request of DEQ, the ATSAC agreed that selenium should be reviewed to determine if an ABC could be identified for this element. There appears to be little new information available for chronic toxicity values for selenium, based on the information available from other agencies as of January 2016.

The only toxicology number available for selenium in regard to inhalation exposure is a number from OEHHA. However, OEHHA derived its inhalation reference concentration from an ingestion study. Although OEHHA chose to extrapolate an inhalation value from that oral ingestion study, it is ATSAC's policy not to extrapolate an inhalation-based toxicity value from an oral ingestion study. OEHHA itself mentioned in its assessment that the inhalation effects from selenium exposure seemed to be different than the effects caused by ingestion exposure to selenium. The irritation of upper airways and related pulmonary symptoms are not accounted for when only oral exposure to selenium is considered.

The ATSAC's recommendation is to not develop an ABC for selenium at this time, because the only toxicity value available is one that was extrapolated from an oral ingestion study, rather than an inhalation study.

31 Styrene

DEQ requested that the ATSAC review toxicological information for styrene and identify an ABC, if appropriate. Styrene is known to cause non-cancer effects, and is a colorless oily liquid that readily evaporates. It has a sweet smell initially but can be rather unpleasant at higher concentrations. Manufactured homes often smell of styrene. Some manufacturers of marine bearings in the Eugene, Oregon area also use styrene in their operations. It is also used in the manufacturing of tile and faux marble. Styrene appears in the emissions inventory primarily from fiberglass manufacturing, but it is also a product of incomplete combustion. Styrene occurs in cigarette smoke, and from some naturally-occurring sources in small amounts.

Attachment B – Summary of ATSAC Consensus Recommendations on Benchmark Revisions

Chronic exposure to styrene affects the central nervous system, and the peripheral nervous system in particular. There are basically two values available for styrene: an EPA IRIS Reference Concentration and an OEHHA Reference Concentration. Both values are based on the same study by Moody, which is a study that evaluated exposure of non-exposed occupational workers to 50 styrene-exposed occupational workers. Moody et al. matched the exposed and unexposed workers groups in regard to age, sex, educational level, and excluded/limited the workers who used alcohol or cigarettes to help narrow down some of the uncertainties involved. The worker exposure to styrene wasn't directly measured, but rather was determined through the monitoring of metabolites in urine. A number of studies have shown that there is good correlation between styrene exposure through inhalation and the resulting styrene metabolites found in urine, so this protocol is considered credible. Using this protocol, they identified a NOAEL of 34 milligrams per cubic meter. EPA IRIS used this number for their calculations, after applying an uncertainty factor of 30, to obtain a Reference Concentration of 1,000 $\mu g/m^3$. An uncertainty factor of 3 was applied due to database inadequacy; another UF of 3 was applied due to intraspecies variability; and a third uncertainty factor of 3 was applied due to lack of information on chronic studies. Multiplying these together creates a total uncertainty factor of 27, which is rounded up to 30 in keeping with toxicology protocols.

OEHHA used the same study, but instead of using the NOAEL as a starting point, instead chose to use a benchmark concentration which approximated the dose response using a model-fitting process, and then applied an uncertainty factor of 9 to obtain a Reference Concentration of 900 $\mu g/m^3$. Thus, the EPA IRIS value and the OEHHA value are very similar. Based on ATSAC policy, which is to use the higher of two numbers if they are based on the same study, the committee considered using the value of 1,000 $\mu g/m^3$ as the ABC for styrene. The National Toxicology Program has identified styrene as a likely carcinogen, but there are no numeric values available that quantify the carcinogenic effects of styrene, so no ABC can be calculated for the carcinogenic effects of styrene using the National Toxicity Program information. The committee can consider re-evaluating the ABC for styrene if quantified information on the carcinogenicity of styrene becomes available in the future.

The odor from styrene is unpleasant. When a chemical smells bad, it is easy to assume it must be toxic, but this is only true in some cases. Although styrene isn't highly toxic, it does have a strong odor, and perception of the odor alone can trigger health effects like headache and nausea. These are hard-wired, involuntary neurological reactions/reflexes to odors that are real, and not psychosomatic. Thus, exposure to the smell of styrene can create a lot of stress and real discomfort, independent of classical toxicological mechanisms. This kind of chronic stress has its own set of negative health effects, so odors are a real concern from a public health perspective. However, there are not any evidence-based methods that provide a numeric quantification of these impacts that would allow the incorporation of odor-induced symptoms and health effects into health benchmarks. Therefore, whatever value the committee chooses as an ABC will have to be based on the direct toxicological effects of styrene. One DEQ staff member commented that it is always helpful for DEQ to have a risk-based number, especially for a high-odor chemical like styrene. In the past, having a risk-based number available for a high-odor compound seemed to have lowered people's stress levels when smelling the odor, once they are shown monitoring results in comparison with that protective level.

Attachment B – Summary of ATSAC Consensus Recommendations on Benchmark Revisions

The ATSAC unanimously recommended that the IRIS-based value of 1,000 µg/m^3 be used as the ABC for styrene.

32 Toluene

Toluene has non-cancer effects, and a current ABC of 400 µg/m3, which was based on a 1995 IRIS Reference Concentration. The committee discussed the various studies and toxicity values currently available from multiple agencies for toluene. On this basis, it was unanimously recommended by the committee that the current ABC of 400 µg/m^3 be revised to match the new 2005 EPA IRIS Inhalation Reference Concentration value of 5,000 µg/m^3.

33 2,4-/2,6-toluene diisocyanate (mixture)

The current ambient benchmark for a 2,4-/2,6-toluene diisocyanate mixture is 0.07 µg/m^3, and this value is based on the 1995 IRIS review. In September 2015, ATSDR updates became available, including a Minimum Risk Level of 0.02 µg/m^3 for 2,4/2,6-toluene diisocynate mixtures. The 1995 IRIS value relied upon the Diem et al. study as its basis; ATSDR, on the other hand, considered the Diem study but then chose instead to use the Clark et al. study from 1998 as the basis of their Minimal Risk Level. The Clark study wasn't available to IRIS originally. The 2015 ATSDR Minimal Risk Level represents essentially a 20-year update, using a more-recent study. A committee member said that ATSDR had focused on a more-sensitive endpoint with a lower LOAEL; he thought this was very important to note.

The lung function decline parameter used in the Clark study indicates a reduction in "FEV 1", in these workers. "FEV 1" is the forced expiatory volume that occurs in one second, which is a highly sensitive measure of pulmonary function. It is arguably the best indicator of sensitivity of the lung to an irritant that with chronic exposure would lead to scarring and stiffening of the lung. So, loss of FEV 1 is directly related to restriction of activities of daily living and function. The committee agreed that the Clark study was exceptionally well done.

The ATSAC recommended unanimously to revise the current ABC for 2,4/2,6-toluene diisocyanate to 0.02 µg/m^3.

34 Trichloroethylene

TCE is a chlorinated solvent that has both cancer and non-cancer effects, but the benchmark is based on cancer because it is more stringent than the related protective value for non-cancer effects. TCE can cause kidney cancer, liver cancer, and non-Hodgkins Lymphoma. The standing ABC for TCE is 0.5 µg/m^3, based on the 1990 URE value from OEHHA of 2.0 x10^{-6} per µg/m^3. Many more epidemiological studies have become available since then, including a study of occupational workers in France (Charbotel, 2006) developing renal cell cancer after exposure to TCE; this study is the principal study used by IRIS.

TCE also causes similar cancers in animal studies. There are also non-cancer endpoints for TCE, so an inhalation Reference Concentration for non-cancer effects was developed, based on consideration of a 21-day window for fetal cardiac malformation in rats. Protection against fetal cardiac malformation is of course critical; however, use of the more-stringent Inhalation Unit Risk for cancer effects, rather than the less-stringent non-cancer-base Reference Concentration, will also be protective of non-cancer risks.

An ABC of 0.24 µg/m^3 can be generated from the EPA IRIS URE of 4.1 x 10^{-6} per µg/m^3. Rounding the ABC value per ATSAC policy would result in a value of 0.2 µg/m^3. The ATSAC unanimously recommended 0.2 µg/m^3 as the new ABC for TCE**,** based largely on new epidemiology studies of highly exposed workers, and new molecular biology methods which have shown causal relationship with cancer as an outcome of exposure to TCE.

35 White phosphorus

White phosphorus is known to have non-cancer effects, and has a standing ABC of 0.07 µg/m^3, which is value based on the 1991 OEHHA Reference Exposure Concentration. California has since withdrawn that value. EPA does not currently provide an inhalation value for white phosphorus. ATSDR identified a value based on respiratory irritation occurring to adults over a period of five minutes. The National Research Council looked at a study based on longer-term exposures in rats.

White phosphorus is not used much anymore. Initially it was used to make matches, and the military tried to use it to generate smoke, but white phosphorus turned out to a bad option for generating smoke. So the military switched to using red phosphorus instead. The only current use of white phosphorus is as a weapon. White phosphorus looks like a block of wax, and if it's left out in the air it will volatilize slowly, creating an odor similar to garlic. People exposed to these vapors in industrial settings back at the turn of the last century led to the development of a disease called phossy jaw, where the jaw rotted away due to exposure to white phosphorus. But this situation can only occur in industrial settings, because if a block of wax-like white phosphorus sits there a little longer, it ignites and generates a cloud of smoke made up primarily of phosphorus pentoxide. Phosphorus pentoxide reacts with water in the air to form phosphoric acid. So the committee's focus should be on protecting people from inhalation of white phosphorus smoke and subsequent irritation of the upper respiratory tract.

Based on the rat study evaluated by the National Research Council mentioned above, the Council came up with a value they called their repeated public exposure guidance level. This level was based on the fact, again, that white phosphorus is rarely used outside of an industrial setting. The military is the only organization that ever looked closely at using white phosphorus.

The number being considered as an ABC for white phosphorus is 9 µg/m^3, which is based on the rat study with a series of uncertainty factors added to protect people from irritation from inhalation of the smoke. Members of the general public are highly unlikely to be exposed to white phosphorus smoke, but if they were, this protective value would prevent upper respiratory irritation.

The committee unanimously recommended that 9 µg/m^3 be used as the ABC for white phosphorus.

36 Xylenes (mixed)

Xylenes are known to have non-cancer effects, and has a standing ABC of 700 µg/m^3 based on a 2000 OEHHA Reference Exposure Concentration. A new Minimal Risk Level for xylenes became available in 2007 from ATSDR of 200 µg/m^3. Both values are based on a Uchida et al. 1993 study.

A committee member said that the Uchida et al. study looks like it's the best study available. It's an occupational study where the subjects were exposed to some other chemicals, but xylene was

the predominant one that they were exposed to. The critical endpoints, which included eye irritation, sore throat, a floating sensation, and a poor appetite, were based on the subjective self-reports of the workers for respiratory and neurologic effects.

Respiratory effects include respiratory irritation, mucus membrane irritation, and headache. ATSDR applied uncertainty factors of 10 for use of a LOAEL, 10 for human variability, and 3 for lack of supporting studies evaluating the neurotoxicity of xylene. It appears that ATSDR used the LOAEL value of 14.2, and then divided it by the total uncertainty factor of 300 to get 0.05. A committee member agreed and added that OEHHA had applied an uncertainty factor to adjust the LOAEL value of 14.2 down to 5.1, based on the presumption of continuous exposure. No adjustment was made by ATSDR for continuous exposure.

If ATSDR had chosen to use an uncertainty factor of 3 to account for the use of a LOAEL, rather than the uncertainty factor of 10 that they did use, then the ATSDR value would then be about 600 $\mu g/m^3$, rather than 200 $\mu g/m^3$. OEHHA applied an uncertainty factor of 10 to their use of a LOAEL and got a value of 700 $\mu g/m^3$. When ATSDR choose an uncertainty factor of 3 rather than 10, it is likely because they considered the severity of the effect to be less, and also they used the unadjusted LOAEL as a point of departure. But identifying something as a severe effect versus identifying it as not severe is somewhat subjective. David Farrer of the Oregon Health Authority is inclined to use the ATSDR value because it is newer and more protective.

The difference of 3.5-fold between 200 $\mu g/m^3$ and 700 $\mu g/m^3$ is not large. Not only was the same study used to obtain both values, but even the actual departure point used to calculate both values was the same. ATSDR chose to add an uncertainty factor of 3 because of the lack of supporting studies to show that xylene can cause neurotoxic effects. Although the respiratory and neurologic effects in the Uchida study are subjective endpoints that were identified by the study participants, there are animal models and considerable other toxicological evidence to support the idea that exposure to xylene results in impaired neurologic function, even in the animal studies.

The 2010 ATSAC deliberations on an ABC for xylene resulted in choosing the OEHHA number of 700 $\mu g/m^3$, rather than the IRIS number of 100 $\mu g/m^3$ available at the time. However, the IRIS number was based on an animal study rather than a human study. So if the committee chooses to use the ATSDR value of 200 $\mu g/m^3$, which is at least partly based on the assumption of neurologic effects in animals, it would be closer to the IRIS value of 100 $\mu g/m^3$, which the committee rejected in 2010.

Kent Norville is concerned with the committee leaning toward choosing the new 200 $\mu g/m^3$ value as the ABC for xylenes, because it does not appear that the ATSAC is following its own policy in this case. Other committee members were comfortable with the ATSDR 2007 value of 200 $\mu g/m^3$. If the three different values for xylene available from IRIS, ATSDR and OEHHA are compared, the ATSDR value is more of a mid-range value on the more health-protective side

The committee voted unanimously to use the ATSDR value of 200 $\mu g/m^3$ as the new ABC for xylene. It was noted that Kent Norville still had some reservations about how and why the ATSDR value was chosen, rather than simply retaining the existing ABC for xylene of 700 $\mu g/m^3$. His particular concern is related to the uncertainty and assumptions about the chronic neurotoxicity of xylene.

Attachment B – Summary of ATSAC Consensus Recommendations on Benchmark Revisions

LIST OF ACRONYMS

ABC ambient benchmark concentration

ADAF age-dependent adjustment factor

ATSAC Air Toxics Science Advisory Committee

ATSDR Agency for Toxic Substances and Disease Registry

BMDL benchmark dose level

DEQ Oregon Department of Environmental Quality

DPM diesel particulate matter

EPA U.S. Environmental Protection Agency

IRIS EPA's Integrated Risk Information System

LOAEL lowest-observed-adverse-effect level

NOAEL no-observed-adverse-effect level

OEHHA California's Office of Environmental Health Hazard Assessment

PAHs polycyclic aromatic hydrocarbons

PATS Portland Air Toxics Solution

PEF potency equivalency factor (related to carcinogenic potency)

PPRTV EPA's Provisional Peer-Reviewed Toxicity Values

TCE trichloroethylene

TEF toxic equivalency factor

$\mu g/m^3$ micrograms of chemical per cubic meter of air

URE unit risk estimate (related to carcinogenic chemicals)

TABLE B-1: Recommended List of 26 PAHs and Related Potency Equivalency Factors

#	PAH	CASRN	EPA Required (1)*	EPA Requested (14)**	From MN list (11) §	PEF ǂ
1	5-Methylchrysene	3697-24-3			⊙	1 ♯
2	6-Nitrochrysene	7496-02-8			⊙	10 ♯
3	Acenaphthene	83-32-9		⊙		--
4	Acenaphthylene	208-96-8		⊙		--
5	Anthanthrene	191-26-4			⊙	0.4
6	Anthracene	120-12-7		⊙		0
7	Benz(a)anthracene	56-55-3		⊙		0.2
8	Benzo(a)pyrene	50-32-8	⊙			1
9	Benzo(b)fluoranthene	205-99-2		⊙		0.8
10	Benzo(c)fluorene	243-17-4			⊙	20
11	Benzo(e)pyrene	192-97-2		⊙		--
12	Benzo(g,h,i)perylene	191-24-2			⊙	0.009
13	Benzo(j)fluoranthene	205-82-3			⊙	0.3
14	Benzo(k)fluoranthene	207-08-9		⊙		0.03
15	Chrysene	218-01-9		⊙		0.1
16	Cyclopenta[c,d]pyrene	27208-37-3			⊙	0.4
17	Dibenz(a,h)anthracene	226-36-8		⊙		10
18	Dibenzo(a,e)pyrene	192-65-4			⊙	0.4
19	Dibenzo(a,h)pyrene	189-64-0			⊙	0.9
20	Dibenzo(a,i)pyrene	189-55-9			⊙	0.6
21	Dibenzo(a,l)pyrene	191-30-0			⊙	30
22	Fluoranthene	206- 44-0		⊙		0.08
23	Fluorene	86-73-7		⊙		--
24	Indeno(1,2,3-c,d)pyrene	193-39-5		⊙		0.07
25	Phenanthrene	85-01-8		⊙		0
26	Pyrene	129-00-0		⊙		0

List recommended by the ATSAC in 2015.

* Naphthalene is also required but already has its own ABC.
** Per NATTS TAD 2009, Revision 2, Table 1.1-1.
§ PAHs on MN MDH 2014 list of 19 priority cPAHs that are not already required or requested by EPA.
ǂ Unless footnoted otherwise, these PEF values are the average from the ranges given in EPA/635/R-08/012A (2010), External Review Draft.
♯ Relative to PEF for benzo(a)pyrene as presented in Minnesota Department of Health *Guidance for Evaluating the Cancer Potency of Polycyclic Aromatic Hydrocarbons (PAH) Mixtures in Environmental Samples* (2016).