State of Oregon
Department of
Environmental
Quality

Proposed 24-Hour Screening Levels for Oregon Health Authority and Oregon Department of Environmental Quality

For Peer Review

October 3, 2016 (Updated October 12, 2016)

Background

24 hour screening levels are intended to help state agencies determine whether measured ambient concentrations of air toxics pose an immediate health risk to people that requires immediate action. Existing 24 hour screening levels were initially developed very quickly to guide decision making in the immediate aftermath of the detection of high concentrations of heavy metals around glass manufacturing facilities. Subsequent review of these values concluded that some of the screening levels were derived from inappropriate source data or were either overly conservative or not conservative enough. Therefore, OHA and DEQ have undertaken a more deliberate and peer reviewed process to identify science-based screening levels.

The short-term screening levels currently being reviewed are distinct from Oregon's Ambient Benchmark Concentrations (ABCs), which are designed to be protective of potential chronic effects from long-term air toxics exposures. Oregon's ABCs were developed by the Air Toxics Science Advisory Committee (ATSAC). While the ATSAC has previously considered the potential to derive short-term screening levels from ABCs, the committee ultimately concluded that this extrapolation is inappropriate since the relevant health endpoints for acute and chronic exposures are not always the same. Instead, the committee recommended that Oregon adopt short-term screening values from those previously identified by federal agencies and other states.

General Approach to Selection of 24 hour Screening Levels

OHA and DEQ toxicologists collaborated to compile existing federal and state human health benchmarks and the underlying justification behind each of them. Specifically we considered existing benchmarks from ATSDR MRLs, ERA IRIS, CA OEHHA, Minnesota, Michigan, Rhode Island, New York, Texas, New Jersey, and other states when available. We summarized the critical studies used, the health endpoints evaluated, the species tested, the intended averaging time, and the uncertainty factors applied for each benchmark value. We reviewed the available benchmarks and applied the following guiding principles to select the most appropriate value as our proposed 24 hour screening level:

Overall principles used to guide selection of 24 hour screening levels

- ATSDR acute MRLs were the first choice whenever available because of the relevance of the averaging time, route of exposure in critical studies, and population they are designed to protect.
- We adopted chronic values as they were when relevant chronic endpoints have the potential to be impacted over short-term exposures (e.g. sensitive neurodevelopmental developmental processes).
- We only used values derived from non-cancer endpoints.
- We avoided short-term values that were time adjusted from chronic values because the justification behind specific time adjustments is often obscure and the chronic endpoints are not always reflective of potential acute endpoints.
- We avoided values extrapolated from occupational exposure limits, but did use them when no other option was viable (selenium).
- We avoided values derived from route-to-route extrapolation (e.g. Cal OEHHA's chronic REL for selenium).
- In order to make the derivation of 24 hr screening levels fully transparent, we only selected values for which clear documentation is available.
- We rounded final values to avoid implying that screening levels reflect a greater degree of precision than they really do.

In the attached tables, we summarize the available benchmarks considered for each chemical. Some tables contain gaps in details for chronic benchmarks and occupational benchmarks that were deemed less relevant for our short-term screening levels early in the process. Below each table, we provide a rationale for our selected short-term screening level.

Specific Rationale for Proposed Lead and Total Chromium Screening Levels

For two chemicals (lead and total chromium) a slightly modified approach was required.

For lead we simply propose adopting the federal National Ambient Air Quality Standard (NAAQS) as our short-term screening level. As a criteria pollutant, lead has a strong federal standard. It would be inappropriate to consider any short-term screening values for lead that are above this federal standard for chronic exposures. Furthermore, in the case of lead, a chronic exposure value is appropriate for a short-term screening value due to the sensitivity of the developing brain to lead exposure.

For total chromium approach we propose to assume that 100% of the total chromium has the potential to be hexavalent. In any monitoring scenario where total chromium concentrations exceed this threshold, subsequent hexavalent chromium monitoring would be recommended to determine whether the hexavalent chromium screening level was being exceeded. While the total chromium screening level would inform prioritization of further monitoring, any emission control decisions would require direct monitoring of hexavalent chromium.

Peer Review Process

We are now seeking comments from five external reviewers:

Bruce Hope, Ph.D. Toxicologist – Member of Air Toxics Science Advisory Committee (ATSAC) and retired from DEQ and Ch2MHill

Julie Wroble, Toxicologist, EPA Region 10

Michael Stewart, Ph.D. – EPA in RTP, NC

Fredrick Berman, Ph.D., DVM – Toxicologist, Oregon Health Sciences University

William Lambert, Ph.D. Epidemiologist, Oregon Health Sciences University and Member of Air Toxics Science Advisory Committee (ATSAC)

Reviewers will submit their comments within 3 weeks (by October 25). Peer reviewer comments will be addressed, and the revised proposed 24-hour screening levels and rationale. Peer reviewer comments will also go out for public comment as we receive them.

Summary of Proposed 24 hour Screening Concentrations for Air Toxics

COMPOUND	PROPOSED Oregon 24-hr VALUE (ng/m^3)	RATIONALE
Acetone	62,000,000	Based on ATSDR acute MRL and related to neurological effects in human subjects.
Arsenic	200	Based on OEHHA acute REL and related to developmental effects.
Beryllium	20	Based on 24 hour values used in RIDEM and MIDEQ, which are based on an OEL value related to exposure of residential community living next to beryllium ceramics plant; TCEQ short-term health-based Interim AMCV (for air monitoring, not air permitting) used as a 1-hr value, and also based on OEL mentioned above
Cadmium	30	Based on ATSDR acute MRL and related to lung inflammation in rodents
Chromium (hexavalent)	5	Based on ATSDR intermediate MRL and related to respiratory effects observed in an occupational setting.
Chromium (total)	---	No value recommended. Assume 100% of total chromium is hexavalent. When total chromium levels exceed 24 hour screening level for hexavalent chromium, recommend direct hexavalent chromium monitoring to inform appropriate response.
Cobalt	100	Based on ATSDR chronic MRL and related to respiratory effects in people. In the absence of acute toxicity data, we select the chronic MRL as a conservative screening level.
Hydrogen sulfide	98,000	Based on ATSDR acute MRL, which is derived from data on short-term respiratory effects in asthmatic people
Lead	150	This is the federal NAAQS, which is based on extensive EPA analysis. The federal standard for chronic exposure is appropriate for short-term health concerns because the critical endpoint of neurodevelopment may be impacted over relatively short-term exposures.
Manganese	90	CA OEHHA chronic REL (and existing Oregon ABC). The REL is derived from data on human nervous system effects following occupational exposures. Use of the chronic REL is protective of potential neurodevelopmental effects during sensitive windows of development.

Methyl ethyl ketone (MEK)	5,000	Based on EPA RfC; value used as a 24-hr value by both Michigan DEQ and Rhode Island DEM. The chronic non-cancer value is appropriate because the critical endpoint is developmental toxicity.
Naphthalene	200,000	Based on Minnesota acute HBV, reflects respiratory effects in rats and is consistent with odor thresholds related to nausea and vomiting in humans.
Nickel	200	Based on ATSDR intermediate MRL (> 14 days up to 1 year) and Cal OEHHA acute REL value (1 hr exposure duration). RI DEM uses this as a 24-hr Ambient Air Level. These levels are derived from rodent data on respiratory effects and immune response.
Selenium	2,000	Based on TCEQ short-term health-based Interim AMCV (for air monitoring, not air permitting) and MIDEQ ITSLs based on OEL known to be based on non-cancer effects; This is more conservative than the OEHHA chronic MRL and ATSDR chronic MRL which required route-to-route extrapolation from oral human exposures.
Styrene	21,000,000	Consistent with ATSDR acute MRL as well as acute standards adopted by CA, MN, NJ, and TX. Values are derived from human studies on neurotoxic effects

Acetone

Chemical	Value (µg/m³)	Source	Species	Point of Departure	Chemical form	Exposure duration	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
Acetone **CASRN:** 67-64-1	62,000	ATSDR MRLs, 2016 Acute	Humans	LOAEL (237 ppm)	Acetone	4 hrs	1994	LOEAL UF = 3 Human variability UF = 3 Total UFs = 9	Dick et al. 1989	Neurological (increases in response and percent false negatives in auditory discrimination; increased anger, hostility)	Acute (2 weeks)
	31,000	ATSDR MRLs, 2016 Intermediate	Humans	LOAEL (1,250 ppm)	Acetone	1 to 7.5 hrs/day, 2 to 5 days/week for 6 weeks	1994	LOEAL UF = 10 Human variability UF = 10 Total UFs = 100	Stewart et al. 1975	Neurological (increased visual evoked response)	Intermediate (2 weeks up to 1 year)
	31,000	ATSDR MRLs, 2016 Chronic	Humans	LOAEL (1,250 ppm)	Acetone	1 to 7.5 hrs/day, 2 to 5 days/week for 6 weeks	1994	LOEAL UF = 10 Human variability UF = 10 Total UFs = 100	Stewart et al. 1975	Neurological (increased visual evoked response)	Chronic

Chemical	Value (µg/m³)	Source	Species	Point of Departure	Chemical form	Exposure duration	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
	475,000	AEGL-1, Interim, 2005 (10 min, 30 min, 1 hr, 4 hr, 8 hr)	Humans	NOAEL (200 ppm)	Acetone	1 to 7.5 hrs/day, 2 to 5 days/week for 6 weeks	2005	Intraspecies UF = 1 Total UF = 1	Stewart et al. 1975	Subjective symptoms including eye and throat irritation were not reported more often than in control group.	Exposure times which are typically related to acute exposure
	5,900	MIDEQ ITSL (Updated Value, November 2015)	Humans	NIOSH REL of 590 ug/m3. Used to obtain RfC-related ITSL.	Acetone	NA	1985	Using MI rule 232(1)(c), divide NIOSH REL by 100, to get 5,900 ug/m3	NA	NA	8-hr averaging time
	62,000*	NJDEP short-term RFC		ATSDR 2008			2008				24-hr averaging time
	60,000	RIDEM STVs (AALs)		ATSDR						Neurology	1-hr AAL (Acute)
	30,000	RIDEM STVs (AALs)		ATSDR						Neurology	24-hr AAL (Intermediate)

Chemical	Value (µg/m³)	Source	Species	Point of Departure	Chemical form	Exposure duration	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
	30,000 *(18,000 ug/m3 is SGC, but based on ACGIH STEL)*	NYSDEC Annual Guideline Concentration (Se is low-toxicity chemical)		Derived by NYSDEC, probably based on similarity to surrogate compound							Chronic
	26,000	TCEQ AMCV, short-term, health-based, FInal	Humans	LOAEL (free-standing)	Acetone	4 hrs	2013	Intraspecies UF = 10 LOAEL UF = 2 Total UFs = 20	Dick et al., 1989	Primarily neurobehavioral effects, secondarily sensory irritation in human volunteers	1 hr (Acute AMCV)

*No Oregon ABC value available for acetone.

No OHHEA RELs or ERPGs available for acetone. No Minnesota DOH HRVs or HBVs.

*Note from NJDEP: "ATSDR values used as short-term RfCs are "acute" MRLs, based on an exposure duration of 1 to 14 days. Here, they are used with a 24-hr averaging time."

Acetone has a molecular weight of 58.05.

Proposed 24-hour screening level

62,000 µg/m3 based on ATSDR acute MRL.

Rationale

ATSDR values have undergone significant review and applying the 2 week averaging time to a 24-hour monitoring period offers another degree of health protectiveness. ATSDR values are derived with the health of general public in mind as opposed to a derivation from occupational studies, which may not reflect the sensitivities of vulnerable populations. ATSDR acute values are designed for exposure periods lasting "up to 14 days" which can include a single 24-hour period.

Acetone Background Information

Description and uses

Acetone is manufactured and also occurs naturally in the environment. It is used to manufacture plastic, fibers, drugs, and other chemicals, and as a general solvent. Acetone is also naturally emitted by plants, trees, volcanoes, forest fires, and the breakdown of body fat. It is present in vehicle exhaust, tobacco smoke, and landfill sites. Industrial processes contribute more acetone to the environment than do natural processes.

Tons per year emitted to Oregon's air

From regulated sources: not available.

Modeled air data for Acetone

None available.

Health Effects

Causes adverse effects to the hematological system and nervous system. Nervous system effects are primarily a transient central nervous system depression and irritability. At higher concentrations can cause mucous membrane irritation.

Sensitive populations

Based on studies done with mice (Dey & Cedebaum 2007), obese mice were more susceptible to the toxicity of acetone, which is known to cause extensive fatty changes and mild necrosis in the livers of the obese mice vs. control mice. This infers the potential for obese humans to be more susceptible to the toxicity of acetone.

Chemical	Value (ng/m^3)	Source	Species	Point of Departure	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time	Chemical form used in critical tox study	Exposure duration
Arsenic **CASRN:** 7440-38-2	200	OEHHA acute REL (March 2016)	Mice (pregnant) exposed to aerosol of arsenic trioxide	LOAEL	2008	UF for LOAEL (no NOAEL) = 10 Interspecies UFs Toxicokinetic UF = √10 Toxicodynamic UF = √10 Intraspecies UFs Toxicokinetic UF = √10 Toxicodynamic UF = √10 Total UF = 1,000	Nagymajtenyi et al., 1985	Decreased fetal body weight (developmental effects)	Acute REL is related to 1 hour of exposure	As_2O_3(Arsenic trioxide)	4 hrs/day on gestation days 9 through 12
	15	OEHHA REL (March 2016)	Humans	LOAEL(based on 10-year-old children drinking water Oral REL)	2008	UF for LOAAL = 3 Interspecies UFs Toxicokinetic UF = √10 Toxicodynamic UF = √10 Total UF = 30	Wassermen et al, 2004; Tsai et al., 2003	Decreased intellectual function; adverse effects on neurobehavioral development	8 hrs and Chronic [1]	Arsenic	Continuous exposure for 9.5 yrs to 10.5 yrs
	200	RI DEM AAL (from CAL 2008)	Please refer to information for OEHHA acute REL, above.					Effects on reproduction,	Short term, 1 hr	Please refer to information for OEHHA acute REL, above.	

Chemical	Value (ng/m^3)	Source	Species	Point of Departure	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time	Chemical form used in critical tox study	Exposure duration
								developmental			
	200	NJDEP short-term RfC (from CAL 2008)	Please refer to information for OEHHA acute REL, above.						1 hr	Please refer to information for OEHHA acute REL, above.	
	9,900	TCEQ Final AMCV, acute ReV	Rats	NOAEL	2012	3 = interspecies UF 10 = intraspecies UF 1 = LOAEL UF 10 = incomplete database UF Total UF = 300	Holson et al., 1999	Maternal effects: rales and decrease in maternal weight gain.	Used by TCEQ for 24 hrs or less of exposure.	Arsenic trioxide acute ReV of 13 ug/m3 adjusted to Rev of 9.9 ug/m3 for Arsenic	6 hrs/day for multiple days
Arsenic trioxide	3.7 mg/m3 3.0 mg/m3 1.9 mg/m3 1.2 mg/m3	AEGL-2 values (2016); no AEGL-1 values available. Note that these are also *Interim* AEGL-2 values.[2]	Rats	NOEL lethality data related to AEGL-3 values; AEGL-2 values are simply 1/3 of the AEGL-3 values	2009	Safety factor of 10 applied to the rate NOEL concentration of 50 mg/m3 arsenic trioxide results in 6-hr AEGL-3 value is 5 mg/m3.	Holson et al., 1999	For AEGL-3 values: 100% fatality at 100 mg/m3 arsenic trioxide after a single 6-hr exposure; NOEL of 50 mg/m3	10 min and 30 min; 1 hr; 4 hrs; 8 hrs	Arsenic trioxide	Appears to have been multiple days of 6 hr/day exposure to 25 mg/m3 arsenic trioxide

Chemical	Value (ng/m^3)	Source	Species	Point of Departure	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time	Chemical form used in critical tox study	Exposure duration
								arsenic trioxide			

*Oregon ABC for Arsenic is 0. 2 ng/m3, based on cancer effects (chronic exposure).

No ERPGs, NYSDEC SGC, acute or subchronic Minnesota Dept. of Health HRVs, or ATSDR MRLs are available for arsenic short-term non-cancer effects.

1 = Due to the possibility of repeated exposure and the relatively slow clearance of arsenic compounds, the 8-hr REL is taken to be equivalent to the chronic REL (OEHHA, 2008).

2 = No data available for estimation of AEGL-1 levels. No AEGL-2 effects were reported after acute inhalation exposure to arsenic trioxide. As an alternative, AEGL-2 values are based on 1/3 of the AEGL-3 values. Proposed AEGL-2 values are supported by the absence

Proposed 24-hour screening level

200 ng/m3 based on California OEHHA's Acute REL

Rationale

This value is based on an exposure pathway and duration that is very relevant to 24-hour exposures in humans and is based on a developmental endpoint that is protective of sensitive populations.

Arsenic Background Information

Description and uses

Arsenic is an element, found naturally in soils and water world-wide, and in most seafood. It is used/produced in ore refining processes, including the smelting of copper and lead. Pesticides have constituted the largest single use (50%) historically of arsenic compounds, but this practice has diminished significantly. Arsenic is also used to treat and preserve wood products. Majority of atmospheric arsenic is highly respirable inorganic arsenic bound to particulate matter smaller than 2.5 microns. Organic forms of arsenic are considered to be less toxic than inorganic forms.

Tons per year emitted to Oregon's Air

From regulated sources: 0.6

At least in the Portland metro area, emissions of arsenic come from motor vehicle exhaust, oil and natural gas combustion, metal processing, agricultural pesticides, and soil dust (soils in PNW are naturally high in arsenic).

Modeled air data for Arsenic

The Portland Air Toxics Assessment found that most instances of arsenic exceedances of air benchmark concentrations in the Portland metro area were related to on-road mobile non-point sources.

Health Effects

The pulmonary system is the target region for arsenic non-cancer effects. Arsenic is also a potent carcinogen, and known to cause cancer in humans. Chronic low-dose exposure to arsenic can alter genes and proteins associated with oxidative stress and inflammation. Hyperpigmentation and hyperkeratinization have occurred in workers exposed to 0.4 to 1 mg/m3 inorganic arsenic for two or more years. Can cause peripheral vascular disease (narrowing of arteries, followed by skin lesions and gangrene mainly in the lower extremities. In infants and children, exposure to inorganic arsenic can cause skin lesions, adverse effects on IQ, lung disease expressed in later years, and reproductive effects (decreased birth weight, spontaneous abortion, neonatal death).

Sensitive populations

Anyone with pre-existing asthma or other pulmonary complications would be more vulnerable to exposure to arsenic via inhalation (i.e., a sensitive population). Infants and children have also been documented as being disproportionately vulnerable to exposure to arsenic in air. Babies developing in the womb may be another sensitive population based on the critical study used to support the proposed 24-hour screening level

Chemical	Value (ng/m^3)	Source	Species	Point of Departure	Chemical form	Exposure duration	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
Beryllium **CASRN:** 7440-41-7	7	OEHHA REL, 2016	Humans	LOAEL (0.55 ug/m3; median exposure of sensitized workers)	Beryllium emitted from ceramics plant near residential community	5 months up to 10 years	2001	Intraspecies UF = 3 LOAEL UF = 10 Total UFs = 30	Kreiss et al., 1996	Beryllium sensitization ; respiratory and immune system effects	Chronic
	20	RIDEM AAL, 24-hr	Humans	Source: EPA RfC of 2E-2 ug/m3, based on a LOAEL. (Chronic AAL is 0.0004 ug/m3)	Beryllium	Not provided	1998	Sensitive nature of subclinical endpoint UF = 3 Database uncertainty UF = 3 Total UF = 10	Kreiss et al., 1996	Beryllium sensitization and progression to CBD	24-hr AAL is applicable to Intermediate exposures (but used EPA RfC) [1]
	20	MIDEQ, 24-hr ITSL	Humans	LOEAL (based on Occupational exposure level in a residential community near a beryllium plant)	Beryllium emitted from ceramics plant near residential community	Not provided	2015	EPA RfC had: LOEAL UF = 10 Database UF = 3 Total UFs = 30	Kreiss et al., 1996 (also Elsenbud et al., 1949)	Beryllium sensitization ; progression to chronic beryllium disease (CBD)	24-hr ITSL, (but used EPA RfC as basis) [2]

Chemical	Value (ng/m^3)	Source	Species	Point of Departure	Chemical form	Exposure duration	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
	20 (short-term, health-based ESL value used for air permitting purposes --No AMCV value provided)	TCEQ AMCV, short-term, health-based, Interim ESL value	Humans	TLV, OSHA is basis of short term health-based ESL	NA	NA	2003	NA	NA	Intended to minimize the likelihood of developing acute and chronic beryllium disease (CBD) and potential lung cancer.	Short-term health-based AMCVs and ESLs are typically used to evaluate 1-hr exposures

*Oregon ABC value available for beryllium is 0.0004 ug/m3, which is based on cancer effects (1998 USEPA IRIS URE of 3 x 10-6 per ug/m3).

No OEHHA acute or 8-hr RELs, no inhalation ATSDR MRLs, no ERPGs, no AEGLs, no NJDEP short-term RfCs, no MDOH acute or subchronic HRVs or HBVs, no NYSDEC SGCs.

1 = RIDEM Air Toxics Guideline (revised Sept. 2008) states that "In keeping with recommendations from the RI Dept. of Health, OAR applied a 24-hr averaging time for some of the RfCs and RfDs listed in EPA's IRIS database. Although RfCs and RfDs are generally understood to be chronic exposure guidelines, the EPA air program has not assigned a specific averaging period to these benchmarks, and it is the belief of RI DOH and OAR that using the RfCs and RfDs as annual average benchmarks would not be appropriately protective in all cases."

2 = The MIDEQ ITSL was originally established with an averaging time set at 24 hrs, which is the default averaging time per AQD Rule 232(2). Although EPA's RfC values are established to target chronic exposure durations, the 24 hr averaging time for beryllium will be retained by the RIDEQ Air Quality Division at this time, because it has been noted by EPA that "the onset of CBD [chronic beryllium disease] from initial exposure (latency) ranges from a few months to an average of 23.7 years (Kreiss et al., 1993); however, at the present time there is no clear relationship between duration of exposure and the development of the disease" (EPA, 1998; Kreiss et al., 1996).

Proposed 24-hour screening number:

20 ng/m3 used by Rhode Island, Michigan, and Texas.

Rationale

The short-term toxicity values used by RI, MI, and TX and proposed for use in Oregon are all identical to the EPA IRIS chronic RfC. MI's program pointed out that there is no specifically quantified minimum exposure period to trigger the sensitization that the IRIS RfC is based on. Once acquired, this sensitization can be debilitating. Based on the severity of the endpoint and the uncertainty around minimum necessary exposures, OHA/DEQ toxicologists consider this chronic RfC to be an appropriate 24-hour screening level as well.

Beryllium Background Information

Description and uses

Beryllium is a metallic element mined as bertrandite and beryl mineral ores. It is also naturally emitted to the air by windblown dusts and volcanic particles. The major anthropogenic emission include the combustion of coal and fuel oil. It is also used in the space, aircraft, and nuclear industries due to its lightness. The Cal OEHHA Appendix D.3. (from OEHHA, 2008) lists four compounds under the heading of "Beryllium and beryllium compounds": beryllium oxide, beryllium hydroxide, beryllium sulfate, and metallic beryllium (2001 Chronic Toxicity Summary)..

Tons per year emitted to Oregon's Air

From regulated sources: 0.07

Modeled air data for Beryllium

The Portland Air Toxics Assessment did not evaluate beryllium.

Health Effects

The respiratory tract in the major target organ system in humans after inhalation exposure to beryllium. Symptoms of chronic beryllium disease (CBD) are shortness of breath upon exertion, cough, fatigue, chest pain, anorexia, and overall weakness.

Sensitive populations

It is logical to assume that people with pre-existing lung/pulmonary conditions are more vulnerable to the toxicity of beryllium through inhalation exposure.

Chemical	Value (ng/m^3)	Source	Species	Point of Departure	Form of Compound used	Duration of Exposure	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
Cadmium **CASRN: 7440-43-9**	30	ATSDR	Rat	LOAEL	Cadmium oxide	6.2 hours/day 5 days/week 2 weeks	2012	10 for LOAEL to NOAEL 3 for animals to humans with dosimetric adjustment 10 for human variability Total 300	NTP 1995	Early signs of lung inflammation	Acute (2 weeks or less)
	10	ATSDR	Human	UCLD10	Cadmium oxide and Cadmium sulfide	Meta-analysis of general population epi studies (long term)	2012	3 for individual variability 3 modifying factor for diabetics who are especially sensitive Total 10	Buchet 1990 Jarup 2000 Suwazono 2006	Renal Toxicity in humans	Chronic
	20	OEHHA	Human	NOAEL	Total Cadmium Compounds		2001	3 for subchronic to chronic extrapolation 10 for individual variability Total 30	Lauwerys 1974	Human workers, renal (proteinuria) and respiratory effects (decreased FVC, FEV, and peak flow rate)	Chronic
	550 (proposed)	TCEQ	Rat	LOAEL	Cadmium oxide	6.2 hours/day	2016	3 for animals to humans	NTP 1995	Early signs of lung inflammation*	Acute (24-hours)

Chemical	Value (ng/m^3)	Source	Species	Point of Departure	Form of Compound used	Duration of Exposure	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
						5 days/week 2 weeks		10 for human variability 10 for LOAEL to NOAEL Total 300		(differs from ATSDR value in exposure duration adjustment; based on same tox study)	
	41000	EPA (AEGL-1)	Rat	LOAEL	Cadmium Chloride Cadmium Oxide	2 hours	2010	3 for animal to human 3 for human variability Total 10	Takenaka et al. 2004	Respiratory irritation and lung inflammation	Acute (8-hour)

*Current Oregon ABC is 6E-4 µg/m3 from the IRIS URE

Proposed 24-hour screening number

30 ng/m^3 ATSDR Acute MRL

Rationale

ATSDR values have undergone significant review and applying the 2 week averaging time to a 24-hour monitoring period offers another degree of health protectiveness. ATSDR values are derived with the health of general public in mind and are designed for exposure periods lasting "up to 14 days" which can include a single 24-hour period. The Texas 24-hour screening level uses the same basic study as the ATSDR acute MRL but makes a time adjustment from the study exposure period to 24-hours. This practice removes a degree of health protectiveness that is inappropriate given that ATSDR's intent is for their acute MRL to be applied to any exposure period less than 14 days, which already includes a 24-hour exposure period. EPA's AEGL averaging time of 8 hours is too short, and there is an inadequate margin of safety.

Cadmium background

Description and uses

Cadmium is a heavy metal found in the earth's crust. It is used in batteries, pigments, coatings and platings, stabilizers for plastics, and photovoltaic devices. Cadmium is also released when wood is burned.

Tons per year emitted to Oregon's Air

From regulated sources: 0.1

More from residential heating (burning wood and natural gas both add cadmium to air. Note that cadmium is present in cigarette smoke, and smokers get much higher doses of inhaled cadmium than non-smokers.

Modeled air data for Cadmium

The Portland Air Toxics Assessment found that most instances of cadmiums exceedence of the ABC were due to point sources. Overall, there are relatively few locations in the Portland metro area modeled to be over the ABC. Monitored results in Portland (North Roselawn) have occasionally shown higher levels of cadmium than the model predicted at that location, but rarely exceed ABC.

Health Effects

This risk driving health outcome for inhaled cadmium is cancers of the respiratory tract, including the trachea, lungs, and bronchi. Non-cancer target organs are the kidney and bones. Cadmium has a relatively long half-life (years) and recycles within the kidney and liver as it is actively resorbed after being filtered out. This repeated cycling through the kidney not only prolongs its stay in the body but is probably the main reason that the kidney bears the brunt of damage caused by cadmium. Non-cancer inhalation exposure also causes respiratory inflammation, which is the basis of the ATSDR acute MRL that is proposed for use as Oregon's 24-hour screening level.

Sensitive populations

Anyone with pre-existing kidney dysfunction would be more sensitive to cadmium than average. Some studies indicate that people with diabetes are also more sensitive to the effects of cadmium on the kidneys. Individuals already exposed to cadmium via cigarette smoking may also be at increased risk.

Chemical	Value (ng/m^3)	Source	Species	Point of Departure	Chemical form	Exposure duration	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
Cobalt **CASRN:** 7440-48-4	100	ATSDR chronic MRL, 2016	Human	NOAEL (0.0053 mg/m3)	Metallic cobalt	Not clearly stated; may be two to five workshops of about 8 hrs each	2004	Human variability UF = 10 Total UFs = 10	Nemery et al., 1992	Respiratory effects; eye, nose, and throat irritation	Chronic
	200	MIDEQ, ITSL (but 8-hr ITSL based on OEL) [1]	Human	ACGIH TLV is basis	Not readily available	Not readily available	1995	Not readily available	ACGIH, 1995	Adverse respiratory effects, asthma, changes in pulmonary function; cobalt asthma	8-hr ITSL
	200	TCEQ, AMCV short-term health-based, Interim	Human	ACGIH TLV is basis of short term health-based ESL	Not currently available; should be available sometime after Aug. 19, 2016	Not currently available; should be available sometime after Aug. 19, 2016	2003	Not currently available; should be available sometime after Aug. 19, 2016	Not currently available; should be available sometime after Aug. 19, 2016	Intended to minimize the potential for developing asthma, pulmonary function alterations, and myocardial effects.	1 hr

Chemical	Value (ng/m^3)	Source	Species	Point of Departure	Chemical form	Exposure duration	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
	20	EPA provisional peer-reviewed subchronic toxicity value, 2008 (Chronic inhalation PPRTV is 0.006 ug/m3)	Humans	NOAEL of 5.3 ug/m3 (when adjusted for continuous occupational exposure, was 1.9 ug/m3)	Metallic cobalt	Not clearly stated; may be two to five workshops of about 8 hrs each	2008	Inter-individual variability (including sensitive subgroups) UF =10 Database insufficiencies UF = 10 Total UFs = 100 [A]	Nemery et al., 1992	Respiratory tract is critical target; Dose-response relationship on lung function, correlated with urinary cobalt levels, after adjusting for effects of smoking and gender. For critical study, decreased pulmonary function and respiratory tract irritation were ID'd as co-critical effects for derivation of subchronic PPRTV.	Subchronic

*Oregon ABC value available for cobalt compounds is 0.1 ug/m3, based on 2001 ATSDR MRL.

No OEHHA acute or 8-hr RELs, no ERPGs, no AEGLs, no inhalation acute or intermediate ATSDR MRLs, no NJDEP short-term RfCs, no MDOH acute or subchronic HRVs or HBVs, no NYSDEC SGCs.

1 =from Michigan Dept. of Natural Resources Interoffice Communication dated May 15, 1995: Initial Threshold Screening Level for Cobalt. In regard to choosing basis for ITSL derivation: "…a draft documentation of the recently revised ACGIH TLV for cobalt, 0.02 mg/m3, is available. While extensively documented and obviously based upon health effects, the TLV documentation addresses the critical effect (Cobalt asthma) only cursorily as one of a number of possible health effects. Nonetheless, considerable experience with occupational exposure to cobalt is reflected in the TLV, an ITSL derived the TLV would be more than an order of magnitude below the lowest cobalt concentration linked specifically with cobalt asthma in Kusaka's work (7 ug/m3). This would allow for more than a ten-fold uncertainty factor to account for humans presumably more sensitive than healthy workers, and for variations in duration of exposure. Considering the low prevalence of cobalt asthma in even heavily exposed populations, the extreme variability in the exposure concentrations and durations reported to provoke clinical asthma, and the previously mentioned uncertainties associated with the LOAELs for cobalt asthma currently available, use of the TLV as an Occupational Exposure Limit (OEL) to drive the derivation of an ITSL seems a reasonable course of action". Per ITSL derivation protocol, then, the resulting ITSL is 0.2 ug/m3.

A = For the PPRTV value, which is based on the Nemery et al.,1992 study, EPA says: "The composite UF of 100 is composed of two uncertainty factors: 10 for database insufficiencies and 10 for inter-individual variability. Nemery et al. (1992) did not report exposure duration for any worker in this study; an assumption is made that worker exposure was at least of subchronic duration. A factor of 10 was applied to account for database insufficiencies due to the lack of inhalation developmental toxicity studies and a multi-generation reproduction study. A factor of 10 was applied to account for human variability, including sensitive subgroups. Individuals with underlying respiratory diseases (asthma, chronic obstructive pulmonary disease) may be more sensitive to the respiratory effects of inhaled cobalt. This subchronic p-RfC may not be protective for people with hypersensitivity to cobalt."

Proposed 24-hour screening number:

100 ng/m^3 ATSDR chronic MRL and Oregon ambient benchmark concentration

Rationale

No true acute toxicity values were available. MI and TX both had developed short-term toxicity values based on adaptation of occupational exposure limits, but they are very similar to the chronic ATSDR value. There is a chronic EPA PPRTV, but OHA/DEQ staff are hesitant to adopt a 24-hour screening level that is lower than the annual ambient benchmark concentration. Instead, OHA/DEQ propose to simply apply the ambient benchmark concentration to a 24-hour averaging time as well. Also because cobalt can cause an asthma like syndrome that may not require long-term exposure to develop.

Cobalt Background Information

Description and uses

Sources of cobalt in air are both natural and anthropogenic. Natural sources include wind-blown continental dust, seawater spray, volcanoes, forest fires, and continental and marine biogenic emissions; these sources contribute slightly more cobalt emissions than do anthropogenic sources. The burning of fossil fuel and sewage sludge, phosphate fertilizers, mining and smelting of cobalt-containing ores, processing of cobalt-containing alloys, and industries that use cobalt compounds also produce emissions of cobalt. Cobalt is emitted to the air in particulate form. Generally, it is assumed that chemical form of cobalt originating from anthropogenic sources is cobalt oxide; also, cobalt my be released into the atmosphere as its arsenide or sulfide during ore extraction processes.

Tons per year emitted to Oregon's Air

From regulated sources: 0.29

Modeled air data for Cobalt

Not readily available.

Health Effects

Cobalt is most detrimental to lungs and pulmonary system. Cobalt asthma is a recognized condition, although there is uncertainty surrounding which chemical form of cobalt causes asthma. It should be noted that studies have shown that humans require a much longer clearance time to get rid of cobalt in the lungs than do typical study animals, such as mice. Therefore, toxicity information obtained from animal inhalation studies should be used with caution if trying to identify related protective levels for cobalt in regard to human exposure. Kusaka (1986) stated that the theoretically most sensitive workers had a latency of 20 years between the first exposure to hard metal (cobalt) and the actual development of clinical cobalt asthma.

Sensitive populations

People with pre-existing asthma are likely to be much more vulnerable to the toxicity of cobalt that occurs through inhalation.

Chemical	Value (ng/m^3)	Source	Form of hex chrome	Species	Point of Departure	Exposure Duration	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
Hexavalent Chromium **CASRN: 18540-29-9**	5	ATSDR (MRL)	Dissolved hexavalent chromium aerosol mist (Chromic acid)	Human	LOAEL	Epi study in workers exposed from 1 month to 20 years	2012	10 for LOAEL to NOAEL 10 for human variability Total 100	Lindberg 1983	Nasal irritation; mucosal atrophy, decreased lung function as measured by spirometry	Intermediate (15-364 days)
	300	ATSDR (MRL)	Sodium dichromate particulate	Rat	BMCL	22 hours/day 7 days/week 90 days	2012	3 Animal to human 10 human variability Total 30	Glaser 1990	Alterations in lactate dehydrogenase levels in bronchiolar lavage fluid	Intermediate (15-364 days)
	8	EPA IRIS (RfC)	Dissolved hexavalent chromium aerosol mist (Chromic acid)	Humans	LOAEL	Epi study in workers exposed from 1 month to 20 years	1998	3 for LOAEL to NOAEL 3 for subchronic to chronic 10 for individual variability Total 90	Lindberg 1983	Nasal septum atrophy	Chronic
	100	EPA IRIS (RfC)	Sodium dichromate particulate	Rat	BMCL		1998	3 for animal to human 10 subchronic to chronic 10 human variability	Glaser 1990 Malsch 1994	Alterations in lactate dehydrogenase levels in bronchiolar lavage fluid	Chronic

Chemical	Value (ng/m^3)	Source	Form of hex chrome	Species	Point of Departure	Exposure Duration	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
								Total 300			
	2	OEHHA (REL)	Dissolved hexavalent chromium aerosol mist (Chromic acid)	Humans	LOAEL		1999	3 LOAEL to NOAEL 10 for subchronic to chronic 10 for human variability Total 300	Lindberg 1983	Nasal atrophy, ulcerations, and septal perforations; transient deficits in lung function	Chronic
	200	OEHHA (REL)	Sodium dichromate particulate	Rat	BMCL		1999	3 for rats to humans 3 for subchronic to chronic 10 for individual variability Total 100	Glaser 1990	Bronchiolar hyperplasia	Chronic
	1,300	TCEQ AMCV, short-term, health-based, Interim value	Particulate compounds	Rat	BMCL10	22 hours/day 7 days/week 30 days	2014	Human equivalent dose adjustment 3 for interspecies	Glaser 1990	Increased relative lung weight	24 hours

Chemical	Value (ng/m^3)	Source	Form of hex chrome	Species	Point of Departure	Exposure Duration	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
								10 for individual variability Total 30			
	100	TCEQ AMCV, short-term, health-based, Interim value	Chromic acid mist	Humans	NOAEL	Epi study in workers exposed from 1 month to 20 years	2014	10 for Individual variability Total 10	Lindberg 1983	Upper respiratory irritation	24 hours

*Current Oregon ABC is 0.08 ng/m^3 from the IRIS URE

Proposed interim/provisional 24-hour screening value

5 ng/m^3 based on ATSDR intermediate MRL

Rationale

ATSDR's intermediate MRL is based on a study in workers exposed to chromic acid mist occupationally. Workers in the study had been working there from 1 month to 20 years. The study does not make clear whether symptoms developed earlier than 1 month for the workers who had been there less time. OHA/DEQ consider this intermediate MRL appropriate for use as a 24-hour screening level. Particulate salts of hexavalent chromium are considerably less toxic than chromic acid mist, and ATSDR's intermediate MRL for sodium dichromate would be appropriate for use if it were known that this was the form of hexavalent chromium present. However, DEQ's monitoring equipment does not differentiate chromic acid mists from particulate salts, so OHA/DEQ propose using the intermediate MRL for chromic acid (the more toxic form) assuming that total hexavalent chromium is 100% chromic acid mist out of an abundance of caution. In situations where the form of hexavalent chromium present is known, OHA/DEQ propose using the intermediate MRL from ATSDR that is most appropriate as a 24-hour screening level.

Hexavalent Chromium background

Description and uses

Hexavalent chromium is a compound used in metal plating, hide tanning, and colored art glass manufacture. It is also a common component of cement powder from some sources. Hexavalent chromium is also a component of diesel exhaust.

Health Effects

Hexavalent chromium has been shown to increase the risk of respiratory tract and lung cancers when exposure is prolonged. It is carcinogenic by a mutagenic mode of action such that early life exposures may pose greater cancer risks than later life exposures. For short-term inhalation exposure, the health effects of hexavalent chromium are most severe when in the chromic acid mist form, in which case it causes severe respiratory tract irritation, nasal septum atrophy, and ulceration at high concentration. Particulate salt forms are less potent, but follow a similar pattern of respiratory irritation and lung inflammation.

Sensitive populations

People with pre-existing asthma or respiratory disease and children are at greatest risk

Chemical	Value ($\mu g/m^3$)	Source	Species	Point of Departure	Form of Compound used	Duration of Exposure	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
Hydrogen Sulfide CASRN: 7783-06-4	97.57	ATSDR	human (asthmatic)	LOAEL	hydrogen sulfide	30 min	2014	3 for use of minimal LOAEL, 3 for human variability, 3 for databas deficiencies	Jappinen 1990	impaired lung function in people with asthma	acute
	27.88	ATSDR	rat	NOAEL	hydrogen sulfide	6 hours/day, 7 days/week for 10 weeks	2014	3 for species extrapolation, 10 for human variability	Brenneman 2000	nasal cavity lesions	intermediate
	2	EPA IRIS (RfC)	rat	NOAEL	hydrogen sulfide	6 hr/day, 7 days/week, for 10 weeks	2003	3 for interspecies extrapolation, 10 for sensitive populations, 10 for subchronic exposure	Brenneman 2000	nasal cavity lesions	chronic
	42	OEHHA REL	human	mean odor threshold	hydrogen sulfide	individuals were each exposed to increasing concentrations of H2S until his or her odor threshold	1999	none	CA Department of Public Health, 1969; CARB, 1984; Reynolds and Kamper, 1985;	odors, headaches, nausea	acute (1 hr)

Chemical	Value ($\mu g/m^3$)	Source	Species	Point of Departure	Form of Compound used	Duration of Exposure	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
						was reached			Amoore, 1985		
Hydrogen Sulfide CASRN: 7783-06-4	10	OEHHA REL	mouse	NOAEL	hydrogen sulfide	6 hours/day, 5 days/week, 90 days	2000	3 for subchronic, 3 for interspecies variability, 10 for intraspecies variability	Chemical Industry Institute of Toxicology 1983	respiratory system (inflammatory changes in nasal mucosa)	chronic
	462	EPA AEGL-1	human	LOAEL	hydrogen sulfide	30 min	2010	3 for interindividual variability	Jappinen 1990	headache in humans with asthma	8hr
	7	Ontario (based on EPA RfD)	rat	EPA RfD (just modified subcrhonic uncertainty factors)	hydrogen sulfide	6 hr/day, 7 days/week, for 10 weeks	2007	3 for interspecies extrapolation, 10 for sensitive populations, 3 for subchronic exposure	Brenneman 2000	Nasal lesions	24hr
	10	Ontario					2013			health and odor	30min
Hydrogen Sulfide	2	MI ITSL (EPA RfC)	rat	NOAEL	hydrogen sulfide	6 hr/day, 7 days/wee	2012	3 for interspecies extrapolation, 10 for	Brenneman 2000	nasal cavity lesions	annual

Chemical	Value ($\mu g/m^3$)	Source	Species	Point of Departure	Form of Compound used	Duration of Exposure	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
CASRN: 7783-06-4						k, for 10 weeks		sensitive populations, 10 for subchronic exposure			
	100	MI ITSL (ATSDR MEL)	human	LOAEL	hydrogen sulfide	30 min	2012	3 for use of minimal LOAEL, 3 for human variability, 3 for databased deficiencies	Jappinen 1990	impaired lung function in people with asthma	24hr
	30	Rhode Island AAL (based on ATSDR intermediate MRL)	rat	NOAEL	hydrogen sulfide	6 hours/day, 7 days/week for 10 weeks	2008	3 for species extrapolation, 10 for human variability	Brenneman 2000	nasal cavity lesions	24hr
	10	Rhode Island AAL (OEHHA REL)	mouse	NOAEL	hydrogen sulfide	6 hours/day, 5 days/week, 90 days	2000	3 for subchronic, 3 for interspecies variability, 10 for intraspecies variability	Chemical Industry Institute of Toxicology 1983	respiratory system (inflammatory changes in nasal mucosa)	annual
Hydrogen Sulfide CASRN: 7783-06-4	10	MN HRV (based on IRIS	rat	NOAEL	hydrogen sulfide	6 hr/day, 7 days/week, for 10 weeks	2001	100 (removed subchronic factor used by EPA as	Brenneman 2000	nasal cavity lesions	subchronic

Chemical	Value (µg/m^3)	Source	Species	Point of Departure	Form of Compound used	Duration of Exposure	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
		database)						this is a subchronic HRV)			
	2	New Jersey (based on IRIS RfC)	rat	NOAEL	hydrogen sulfide	6 hr/day, 7 days/week, for 10 weeks	2011	3 for interspecies extrapolation, 10 for sensitive populations, 10 for subchronic exposure	Brenneman 2000	nasal cavity lesions	annual
	42	New Jersey (based on OEHHA)	human	mean odor threshold	hydrogen sulfide	individuals were each exposed to increasing concentrations of H2S until his or her odor threshold was reached	2011	none	California State Department of Public Health, 1969; CARB, 1984; Reynolds and Kamper, 1985; Amoore, 1985	odors, headaches, nausea	1 hr
	1,400	ACGIH (TLV)									8hr

Current OR ABC= 2µg/m3 (EPA IRIS RfC)

EPA AEGL Level of Odor Awareness = 0.01 ppm; ATSDR cites odor threshold between 0.0005 to 0.3 ppm

Proposed 24-hour screening number

98 µg/m^3 (rounded from ATSDR acute MRL 97.57)

Rationale

Where available, we use the acute MRL set by ATSDR for our 24 hour screening values. The acute MRL for hydrogen sulfide is based on an acute non occupational human study of people with asthma. The value is therefore designed to be protective of short-term exposures in a particularly sensitive population. Though odor detection thresholds for hydrogen sulfide are lower than the acute MRL, our 24 hr screening concentrations are intended to be protective of health effects as opposed to odor detection.

Hydrogen Sulfide background

Description and uses

Hydrogen sulfide is a colorless gas with a sulfer "rotten egg" smell. It occurs naturally and can be present in petroleum, natural gas, sewage treatment plants, manure operations, and pulp and paper mills. While most hydrogen sulfide in the air is released from natural sources like volcanoes, swamps and bogs, it is also emitted from industrial sources such as petroleum refineries, paper mills, manure and waste water treatment facilities. Hydrogen sulfide air concentrations from natural sources range between 0.00011 and 0.00033 ppm. In urban areas, the air concentrations are generally less than 0.001 ppm.

Health Effects

Inhalation is the primary route of exposure to hydrogen sulfide. In occupational settings, high exposures to hydrogen sulfide have been associated with effects on the respiratory tract (irritation to nose and throat, difficulty breathing for some asthmatics) and nervous system (headache, poor memory, tiredness, balance problems).

Sensitive populations

People with asthma may be particularly sensitive to the respiratory effects of hydrogen sulfide. Little is known about the sensitivity of children to the effects of hydrogen sulfide. In animal studies, exposure to low concentrations of hydrogen sulfide does not cause birth defects.

Chemical	Value ($\mu g/m^3$)	Source	Species	Point of Departure	Form of Compound used	Duration of Exposure	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
MnO2 **CASRN: 7440-43-9**	0.09	CA OEHHA REL	human	BMCL 05 (probit)	MnO2	Occupational Epi study, exposure reported as years worked on the job (0.2-17.7 years)	2008	10 for toxicokinetic (children), 10 for toxicodynamics (children), √10 for subchronic to chronic	Roels et al., 1992	nervous system; visual reaction time, eye-hand coordination, hand steadiness	chronic
	0.17	CA OEHHA REL	human	BMCL 05 (probit)	MnO2	Occupational Epi study, exposure reported as years worked on the job (0.2-17.7 years)	2008	10 for toxicokinetic (children), 10 for toxicodynamics (children), √10 for subchronic to chronic	Roels et al., 1992	nervous system; visual reaction time, eye-hand coordination, hand steadiness	8hr
	0.30	ATSDR	human	BMCL 10 (logistic model)	Mn02 (airborne manganese concentration)	Occupational Epi study, exposure reported as years worked on the job (0.2-17.7 years)	2012	10 for human variability, 10 for limitations in dataset (lack of data on exposure to soluble forms)	Roels et al., 1992	nervous system; incidence of abnormal hand eye coordination	chronic (annual)

Chemical	Value (µg/m^3)	Source	Species	Point of Departure	Form of Compound used	Duration of Exposure	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
	0.3	Michigan (ITSL; ATSDR MRL)	human	BMCL	MnO2	Occupational Epi study, exposure reported as years worked on the job (0.2-17.7 years)	2012	10 for human variability, 10 for limitations in dataset (lack of data on exposure to soluble forms)	Roels et al., 1992	nervous system	chronic (annual)
	0.17	New Jersey (based on OEHHA REL)	human	BMCL 05 (probit)	MnO2	Occupational Epi study, exposure reported as years worked on the job (0.2-17.7 years)	2011	10 for toxicokinetic (children), 10 for toxicodynamics (children), √10 for subchronic to chronic	Roels et al., 1992	nervous system; visual reaction time, eye-hand coordination, hand steadiness	8hr
	0.15	WHO	human	BMDL 5	MnO2	Occupational Epi study, exposure reported as years worked on the job (0.2-17.7 years)	2000	10 for interindividual variation, 5 for developmental effects	Roels et al., 1992	Neurological effects	Chronic (annual)

Chemical	Value ($\mu g/m^3$)	Source	Species	Point of Departure	Form of Compound used	Duration of Exposure	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
	200	ACGIH TLV	human							Central nervous system impairment	8hr
	0.1	Ontario Air Standards (AAQC)	Human	BMCL 05	Manganese and manganese compounds in the PM2.5 size fraction	Occupational Epi study, exposure reported as years worked on the job (0.2-17.7 years)	2011	10 for human variability, 3 for database limitations, 3 for vulnerability of the developing nervous system, 3 for subchronic extrapolation	Roels 1992	Neurological effects	24hr
	0.2	Ontario Air Standards (AAQC)	human	BMCL 05	Manganese and manganese compounds in the PM10 size fraction	Occupational Epi study, exposure reported as years worked on the job (0.2-17.7 years)	2011	10 for human variability, 3 for database limitations, 3 for vulnerability of the	Roels 1992	Neurological effects	24hr
	0.4	Ontario Air Standards	human	BMCL 05	Total suspended particulate	Occupational Epi study, exposure reported as years worked on the job	2011	10 for human variability, 3 for database limitations, 3 for vulnerability of the	Roels 1992	Neurological effects	24hr

Chemical	Value ($\mu g/m^3$)	Source	Species	Point of Departure	Form of Compound used	Duration of Exposure	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
						(0.2-17.7 years)					
Manganese PM 3.5 CASRN: 7440-43-9	0.05	HealthCanada (RfC)	Human	BMC	Inhaled Manganese PM 3.5	Occupational Epi study, reported as work history average respirable manganese and average respirable manganese over the last 5 years	2010	10 interindividual variability, 10 for limitations in the data	Lucchini 1999	Neurological effects	chronic
Manganese and Manganese compounds CASRN: 7440-43-9	0.05	EPA IRIS (RfC)	human	LOAEL	MnO2 (1992), MnO2, Mn3O4, Mn salts (1987)	Occupational Epi study, exposure reported as years worked on the job (0.2-17.7 years)	1993	10 for use of LOAEL, 10 for human variability, 10 for data limitations	Roels et al., 1992; Roels et al., 1987	nervous system	chronic (annual)
Manganese and Manganese compounds	0.05	Rhode Island (IRIS RfC)	human	LOAEL	MnO2 (1992), MnO2, Mn3O4,	Occupational Epi study, exposure		10 for use of LOAEL, 10 for human variability, 10	Roels et al., 1992; Roels	nervous system	24hr

Chemical	Value ($\mu g/m^3$)	Source	Species	Point of Departure	Form of Compound used	Duration of Exposure	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
CASRN: 7440-43-9					Mn salts (1987)	reported as years worked on the job (0.2-17.7 years)		for data limitations	et al., 1987		
	0.2	Minnesota (modified from IRIS RfC)	Human	BMC	MnO2	Occupational Epi study, exposure reported as years worked on the job (0.2-17.7 years)	2001	10 for sensitive individuals, 3 for database deficiencies, 3 for extrapolation to lifetime exposure	Roels 1992	Neurological effects	chronic
	0.05	New Jersey (based on IRIS RfC)	human	LOAEL	MnO2 (1992), MnO2, Mn3O4, Mn salts (1987)	Occupational Epi study, exposure reported as years worked on the job (0.2-17.7 years)	2011	10 for use of LOAEL, 10 for human variability, 10 for data limitations	Roels et al., 1992; Roels et al., 1987	nervous system	chronic

Proposed 24-hour screening number

0.09 µg/m3, based on the OEHHA chronic REL

Current OR ABC= 0.09 (based on the OEHHA chronic REL)

Rationale

Almost all guidelines are derived from the same long-term occupational exposure study and most agencies conclude that chronic exposure limits will be sufficiently protective of short term exposures. OEHHA uses the most conservative approach to interpreting this data by using a $BMCL_{05}$ (whereas ATSDR uses the $BMCL_{10}$) and incorporating uncertainty factors for human variability for both toxicokinetics and toxicodynamics. Oregon has already adopted the OEHHA chronic REL as the Ambient Benchmark Concentration. Because there is some potential for developmental neurotoxicity (for which relatively short-term exposures may be important), we will use the conservative chronic REL as our 24hr screening level as well. OEHHA's acute REL relies on the same analysis and is simply adjusted to a shorter averaging time. We avoid using short-term values that are derived from arbitrary time adjustments of chronic values.

EPA and ATSDR do not have short term exposure guidelines and simply rely on the chronic guidelines. Given a lack of data to support short-term guidelines, they assume that chronic guidelines are health protective. The EPA IRIS is scheduled to be updated. The original analysis relies on the LOAEL as opposed to the benchmark which mean it incorporates additional uncertainty. The more recent 2012 ATSDR assessment uses a BMDL approach that allows it to remove some uncertainty.

It is important to be aware that the Roels 1992 study underlying the ATSDR MRL and OEHHA REL is limited in that it focused only on MnO2 as opposed to a more comprehensive set of manganese compounds.

Other sources searched:

MO, NJ, EPA AEGL, NY SGC, Texas (only interim short-term AMCVs exist). The ACGIH TLV is behind a paywall and is not worth tracking down as it is an occupational standard that would not be the most health protective.

Manganese background (from ATSDR profile, OEHHA, and MI)

Description and uses

Manganese is a trace metal and essential nutrient occurring in food, soil, air, and water. It is typically found in complex with other elements, including oxygen, sulfur and chlorine. It is used in fertilizer, paints, fireworks, batteries, medical imaging, and cosmetics.

Health Effects

Manganese is most acutely toxic when exposure occurs via inhalation. In occupational epidemiological studies, exposure to manganese via inhalation is associated with neurological effects. Specifically, it is associated with Manganism, a Parkinson's-like syndrome characterized by impaired motor skills and tremors. Occupational exposures have also been linked to respiratory effects, impotence, and loss of libido.

Sensitive populations

The elderly, particularly those with asymptomatic pre-parkinsonism, may be particularly vulnerable to the neurotoxic effects of manganese. Liver disease or iron deficiency may increase susceptibility. Occupational exposures can result in particularly high exposures. The ATSDR toxicology profile for manganese also cites some laboratory animal evidence that developing fetuses and young children may be particularly susceptible to neurotoxic effects of manganese, though insufficient data is available on neurodevelopmental endpoints.

Chemical	Value (µg/m³)	Source	Species	Point of Departure	Chemical form	Exposure duration	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
Methyl ethyl ketone (aka 2-Butanone) **CASRN: 78-93-3**	13,000	OEHHA 2016	Human	LOAEL	MEK	2 hrs	1999	LOAEL UF = 6 Intraspecies UF = 10 Total UF = 60	Nakaaki, 1974	Eye, nose, throat irritation. Targets: Eyes, respiratory system.	1 hr (acute)
	59,000	AEGL-1 March 2016	Human	NOAEL	MEK		?	Total UF = 1; no modifying factor	Dick et al. 1992; Muttray et al. 2002; Seeber et al. 2002; Shibata et al. 2002) NOAEL for subjective symptoms in humans	Sensory irritation and neurobehavioral effects	10 min, 30 min, 1 hr, 4 r. 8 hr
	5,000	MIDEQ ITSL (24-hr)	Mouse	EPA RfC, IRIS 2003	MEK	7 hrs/day on gestation days 6 through 15	2003	Intraspecies variability UF = 10 Database deficiencies UF = 10 Interspecies extrapolation UF = 3 Total UF = 300	Schwetz et al., 1991	Developmental effects (skeletal variations)	24 hr
	13,000	NJDEP short-term RfC	Human		MEK		?				1 hr

Chemical	Value (µg/m³)	Source	Species	Point of Departure	Chemical form	Exposure duration	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
		(Aug. 2011)		Cal acute REL 2008							
	13,000	RIDEM AALs 2008 (based on acute CAL 2008)	Human	Cal acute REL 2008	MEK		?			Eye and respiratory inflammation	1 hr (Acute)
	5,000	RIDEM AAL	Mouse	EPA RfC, IRIS 2003	MEK	7 hrs/day on gestation days 6 through 15	2003	Intraspecies variability UF = 10 Database deficiencies UF = 10 Interspecies extrapolation UF = 3 Total UF = 300	Schwetz et al., 1991	Developmental effects (skeletal variations)	24-hr (RfC-based) Intermediate (RIDEM term)
	10,000	MDOH short-term HRV	Human	Cal acute REL, LOAEL of 800 mg/m3						Eye, respiratory system irritation	Acute, 1-hr
	13,000	NYSDEC SGC		Derived by NSYDEC, likely							1 hr

Chemical	Value (µg/m³)	Source	Species	Point of Departure	Chemical form	Exposure duration	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
				based on similarity to a surrogate chemical							
	59,000	TCEQ AMCV short-term health-based	Human	NOAEL of 200 ppm (free-standing NOAEL)	MEK	4 hrs		Intraspecies UF = 10 Total UF = 10	Dick et al., 1992	Neurological effects; sensory irritation in human volunteers	1 hr

*No Oregon ABC value available for methyl ethyl ketone.

No ERPGs or ATSDR MRLs available for MEK.

Proposed 24-hour screening number

5,000 µg/m^3 based on EPA RfC for chronic exposure and RI and MI short-term values.

Rationale

EPA chronic RfC; The unadjusted chronic non-cancer value is appropriate for use as a 24-hour screening level given that the critical endpoint is developmental toxicity. The 2003 update of the EPA RfC used a different method and newer data than the previous RfC values and is based on developmental effects in mice. Both Michigan DEQ and Rhode Island DEM use this value for 24-hr exposure durations.

Methyl ethyl ketone Background Information

Description and uses

Methyl ethyl ketone (MEK) is a solvent often found in mixtures with acetone, ethyl acetate, n-hexane, toluene, or alcohols, and has applications in the surface coating industry and in the dewaxing of lubricating oils. MEK is also used in the manufacture of colorless synthetic resins, artificial leather, rubbers, lacquers, varnishes, and glues. MEK is also sometimes referred to as 2-butanone.

Tons per year emitted to Oregon's Air

From regulated sources: NA

Modeled air data for Methyl ethyl ketone

Not available.

Health Effects

Symptoms of acute MEK exposure include irritation of the eyes, nose, and throat. Occupational workers exposed to MEK have complained of mild neurologic effects including headaches, dizziness, and nausea – but these were related to exposure to multiple solvents, not just MEK alone.

Sensitive populations

People with preexisting eye, neurologic, skin, or respiratory conditions may be more sensitive to the toxic effects of MEK.

Chemical	Value ($\mu g/m^3$)	Source	Species	Point of Departure	Exposure Duration	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
Naphthalene **CASRN: 91-20-3**	3.7	ATSDR (MRL)	Rat	LOAEL		2005	10 for LOAEL to NOAEL 3 for animals to humans with dosimetric adjustment 10 for human variability Total 300	Abdo, 2001 NTP 1992 NTP 2000	Nonneoplastic nasal lesions	Chronic
	3	EPA IRIS (RfC)	Mouse	LOAEL		1998	10 for LOAEL to NOAEL 10 for mice to humans 10 for individual variability 3 for database limitations Total 3000	NTP 1992	Nonneoplastic lesions of the olfactory and respiratory epithelium (nasal lesions)	Chronic
	9	OEHHA (REL)	Mouse	LOAEL		2000	10 for LOAEL to NOAEL 10 for mice to humans 10 for individual variability Total 1000	NTP 1992	Nonneoplastic lesions of the olfactory and respiratory epithelium (nasal lesions)	Chronic

Chemical	Value ($\mu g/m^3$)	Source	Species	Point of Departure	Exposure Duration	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
	500 (interim)	TCEQ (Ambient Monitoring Comparison Value)	Rat	NIOSH REL		2015	100 (Texas standard practice to divide occupational values by 100 to derive interim acute values)	NIOSH REL	Intended to minimize eye and respiratory tract irritation and ocular toxicity that could include cataract formation, optical neuritis and retinal degeneration.	Acute
	200	Minnesota (HBV)	Rat	NOAEL		2004	10 for animal to human 10 for human variability 10 for database limitations Total 1000	Buckpitt 1982	Respiratory cell swelling and sloughing. Also consistent with odor threshold related nausea and vomiting in humans. This value is also consistent with reported health effects in humans related odor perception	Acute (1-hour)

Chemical	Value (µg/m^3)	Source	Species	Point of Departure	Exposure Duration	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
	7900	New York (SGC)	Human	ACGIH (STEL)		2014	10 (New York standard practice is to divide ACGIH STELs by 10 to derive short-term guideline concentrations)	ACGIH	Unknown (no access to ACGIH)	Acute
	130000	IDLH/10*	Human	NIOSH (IDLH)		2014	One of EPA's acute screening levels. It is one tenth the NIOSH immediately dangerous to life and health level	NIOSH	Respiratory and ocular irritation; nausea, vomiting	Acute (1 hour)
	---	EPA (AEGL-1)	---	---		---	---	---	No AEGL-1 available for naphthalene	
	186	New Hampshire (24-Hour AAL)	Unknown	ACGIH (TLV)			ACGIH divided by 100 and divided by a time factor of 2.8 as per New Hampshire regulations	ACGIH	Ocular irritation	Acute

Chemical	Value (µg/m^3)	Source	Species	Point of Departure	Exposure Duration	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
	----	Washington (ASIL)							Cancer Based	
	520	Michigan (second ITSL)	Unknown	ACGIH (TLV)			ACGIH TLV divided by 100	ACGIH	Ocular irritation	Acute (24-hour)
	---	STAR							Long-term cancer toxicity values only	

*Current Oregon ABC is 0.03 µg/m3 from the IRIS URE

Proposed interim/provisional 24-hour screening value

200 µg/m^3 Minnesota short term Health Based Value

Rationale

The Minnesota acute health based value (HBV) is the lowest (most health protective) of the acute values available and is not adapted from an occupational guideline. This value meets Oregon criteria for a 24-hour screening value in that it is not based on a cancer endpoint. Minnesota's HBV is based on respiratory cell sloughing and swelling in a mouse study. It is also consistent with anecdotal health effects reported by people in communities relative to the odor threshold.

New Hampshire, Michigan, and Texas all used the same occupational exposure limit derived by the American College of Governmental Industrial Hygienists (ACGIH) to derive acute toxicity values. The ACGIH threshold limit value (TLV) is 52 mg/m3. Michigan and Texas both applied an uncertainty factor of 100 to extrapolate from occupational settings to the general public including sensitive populations. Texas rounded to the nearest significant figure (520 to 500 µg/m3) while Michigan did not. In addition to the same uncertainty factor of 100, New Hampshire applied an additional modifying factor of 2.8, which New Hampshire applies to all air toxics that it designates as "toxicity class I." Naphthalene meets New Hampshire's criteria for toxicity class I. While New Hampshire's 24-hour ambient air level (AAL) is lower than Minnesota's HBV, it may have a greater level of uncertainty, along with all other values derived from occupational limits. New Hampshire's 24-hour AAL is also within rounding distance of Minnesota's HBV (186 vs. 200 µg/m3).

The first three values in the table established by the Agency for Toxic Substances and Disease Registry (ATSDR), the U.S. Environmental Protection Agency (EPA), and California's Office of Environmental Health Hazard Assessment (Cal OEHHA) are designed to protect against non-cancer health effects over long-term/chronic exposures. Oregon already has an established ambient benchmark concentration that is appropriately based on long-term cancer risk for chronic exposures. The purpose of this effort was to consider

appropriate benchmark concentrations for short-term exposures for measuring likelihood of immediate or acute health effects. The 3 chronic values were listed as a point of reference.

Naphthalene background

Description and uses

Naphthalene is the lightest and most volatile of the polycyclic aromatic hydrocarbons and is the active ingredient in mothballs. It is a component of many petroleum products and combustion, and is especially high in some creosote formulations.

Health Effects

This risk driving health outcome for inhaled naphthalene in the short-term is respiratory irritation, including nasal lesions. High concentrations can lead to hemolytic anemia especially in people with a deficiency in the enzyme glucose-6-phosphate dehydrogenase. Chronic exposure can increase risk of cancers of the nasal and respiratory tract. Inhaled naphthalene is rapidly metabolized and dose not accumulate to an appreciable extent.

Sensitive populations

Anyone with a deficiency in the glucose-6-phosphate dehydrogenase enzyme is especially vulnerable to the hemolytic anemia effects of naphthalene. This enzyme deficiency is especially common in people of African, Middle Eastern, and Mediterranean descent.

Nickel

Chemical	Value (ng/m^3)	Source	Species		Point of Departure	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time	Chemical form used in critical study	Exposure duration
Nickel CASRN: 7440-02-0	200	ATSDR intermediate MRL (March 2016)	Rat		NOAEL of 0.06 mg Ni/m3	2005	3 (extrapolation from animals to humans per dosimetric adjustment) 10 (human variability) Total UF = 30	NTP 1996c	Chronic active inflammation respiratory	Intermediate (> 14 days, < 1 yr)	$NiSO_4$ (nickel sulfate)	13 weeks
	90	ATSDR chronic MRL (March 2016)	Rats		NOAEL of 0.03 mg Ni/m3	2005	3 (extrapolation from animals to humans per dosimetric adjustment) 10 (human variability) Total UF = 30	NTP 1996c	Chronic active inflammation respiratory and lung fibrosis	Chronic	$NiSO_4$ (nickel sulfate)	2 years
	200	OEHHA table, acute REL (March 2016)	Mouse		BMDL	2012	BMR UF = √10 Interspecies UF = 10 Intraspecies UF (√10PD * 10PK) Total UF = 1,000	Graham et al., 1978	Immunotoxicity, pneumotoxicity	1 hour (Acute inhalation REL)	$NiCL_2$	2 hr
	60	OEHHA table, 8-hour	Rat		NOAEL	2012	Interspecies UF = √10	NTP 1994c	Respiratory, immune systems	8-hr inhalation REL	$NiSO_4$	Several intervals, up to 2 years: 6.2

Nickel

Chemical	Value (ng/m^3)	Source	Species		Point of Departure	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time	Chemical form used in critical study	Exposure duration
		REL (March 2016)					Intraspecies UF = 30 Total UF = 100					hrs/day, 5 days/wk, for 16 days to 24 months
	14	OEHHA table, chronic REL (March 2016)	Rat		BMDL $_{05}$	2012	Interspecies UF = √10 Intraspecies UF = 30 Total UF = 100	NTP 1994a and NTP 1994c	Respiratory system; hematologic system	Chronic inhalation REL	$NiSO_4$	Several intervals, up to 2 years: 6.2 hrs/day, 5 days/wk, 104 wks
	6,000	NJ DEP short-term RfC (Cal 2008)		Please refer to information for OEHHA acute REL.						1 hr	Please refer to information for OEHHA acute REL.	
	200	RI DEM AAL (based on ASTDR intermediate MRL)		Please refer to information for ATSDR intermediate MRL.						Intermediate – 24 hrs	Please refer to information for ATSDR intermediate MRL.	
	6,000	RI DEM AAL (based on Cal acute REL)		Please refer to information for OEHHA acute REL.						Acute – 1 hr	Please refer to information for OEHHA acute REL.	
	11,000	MDOH acute HRV	**?**		LOAEL (0.067 mg/m3)	2016	UF = 6	**?**	Irritation of respiratory system	Acute – 1 hr	**?**	**?**
	200	NYSDEC STG			Derived by NYSDEC, likely based on similarity							

Nickel

Chemical	Value (ng/m^3)	Source	Species		Point of Departure	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time	Chemical form used in critical study	Exposure duration
					to a surrogate compound							
	1,100	TCEQ short-term health-based AMCV, Final	Humans		LOAEL	TCEQ DSD 2013	Intraspecies UF = 1 LOAEL UF = 10 Incomplete database UF = 3 Total UF = 30	Cirla et al. 1985	Bronchial constriction in human volunteers with occupational asthma	AMCVs, ReVs are generally used for 1-hr exposure, unless specifically noted otherwise	Nickel sulfate	30 min
Nickel carbonyl CASRN: 13463-3-3	31,400	AEGL-2 (no AEGL-1 values available)	Mice *		NOAEL (2.17 ppm for 30 minutes)	2016	Interspecies UF = 3 Intraspecies UF = 3 Total UFs = 9 MF = 3	Kincaid et al. 1953	Pulmonary damage	8 hr	Nickel carbonyl	*[[10 min, 30 min, 1 hr, 4 hr, 8 hr respectively per the five ppm concentrations]]*

- From AEGLs for Selected Airborne Chemicals, Vol. 6: "30-minute exposure to 2.17 ppm nickel carbonyl was considered a reasonable estimate of an exposure that might cause pulmonary damage in the mouse (the most sensitive species tested), but not result in irreversible adverse effects".

BMDL – lower confidence limit of Benchmark Dose

No MIDEQ ITSL for nickel

Current Oregon ABC (non-cancer) for nickel includes:

Nickel refinery dust, no CAS, 0.004 ug/m3 from 1991 IRIS URE of 2.4 x 10-4 per ug/m3 for nickel refinery dust.

Nickel subsulfide (cancer), CAS 12035-72-2, 0.002 ug/m3 from 1991 IRIS URE of 4.8 x 10-4 per ug/m3 for nickel subsulfide.

Nickel compounds (soluble), non-cancer, CAS – multiple numbers, 0.05 ug/m3, which equals the 2000 OEHHA REL.

Recommendation from 2015 ATSAC:

Nickel compounds (soluble), primarily non-cancer, CAS – multiple numbers, 0.01 ug/m3, from the OEHHA 2011 Reference Exposure Level

Nickel compounds (insoluble), primarily cancer, CAS – multiple numbers, 0.004 ug/m3, from newer OEHHA 2011 value for nickel subsulfide.

Proposed interim/provisional 24-hour screening value

Nickel

200 ng/m^3, based on ATSDR intermediate MRL and OEHHA acute REL

Rationale

ATSDR values have undergone significant review and applying the 2 week averaging time to a 24-hour monitoring period offers another degree of health protectiveness. This value is also consistent with the acute REL from OEHHA which is derived from a different critical study. RI DEM also uses this as a 24-hr Ambient Air Level. This value reflects respiratory effects and immune responses in rats and mice.

Nickel Background Information

Description and uses

Nickel is an abundant natural element. Nickel compounds are used for nickel plating and in nickel alloys, to color ceramics, to make certain batteries, and as catalysts for chemical reactions. It is released into the atmosphere by industries that make or use nickel; oil- and coal-burning power plants, and trash incinerators. Nickel carbonyl is formed by the reaction of carbon monoxide with metallic nickel, and is used in processes like nickel refining. However, in air, nickel carbonyl rapidly decomposes to metallic nickel and carbon monoxide. Nickel sulfate hexahydrate is used in nickel electro plating, as a mordant in dyeing and printing textiles, as a blackening agent for zinc and brass, and in the manufacture of organic nickel salts.

Tons per year emitted to Oregon's Air

From regulated sources: 1.5

Modeled Air Data for Nickel

The Portland Air Toxics Assessment found that nickel emissions in the Portland metro area were related to a localized impact areas and likely to industry operations.

Health Effects

People sensitive to nickel may experience asthma attacks following exposure to nickel. Chronic bronchitis and reduced lung function have been noted in workers at nickel refineries and processing plants. Lungs and the pulmonary system are the primary targets, but nickel also causes adverse health effects to the stomach, blood, liver, kidneys, immune system, and reproductive system in rodents, and includes developmental effects. It does not appear that children are more vulnerable to nickel exposure than adults are.

Sensitive Populations

None were identified in the literature reviewed.

Chemical	Value (µg/m³)	Source	Species	Point of Departure	Chemical form	Exposure duration	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
Selenium **CASRN: 7782-49-2**	5	OEHHA, 2016 (flammable gas)			*HYDROGEN SELENIDE*						
	20	OEHHA 2016	Humans	LOAEL	Selenium dioxide	Workers exposed to fumes and dust underwent a 5-year study	2001	Intraspecies UF = 3 Total UF = 3	Yang et al., 1989 Value extrapolated from oral value of 0.005 mg/kg-day)	Hair and nail loss, discoloration and decay of teeth, and central nervous system disturbances (pain and numbness in extremities)	Chronic value only; no acute or 8-hr RELs available.
	Only an oral value of 0.005 mg/kg/day is available; when extrapolated is 20 µg/m3	ATSDR, 2016	Humans	LOAEL	Selenium dioxide	Workers exposed to fumes and dust underwent a 5-year study	2001	Total = 3	Yang et al., 1989 Value extrapolated from oral value of 0.005 mg/kg-day)	Hair and nail loss, discoloration and decay of teeth, and central nervous system disturbances (pain and	Chronic value only; no acute or 8-hr RELs available.

Chemical	Value (µg/m³)	Source	Species	Point of Departure	Chemical form	Exposure duration	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
										numbnes s in extremiti es)	
	2	MIDEQ ITSLs	Human	An OEL value of 0.2 mg/m3 developed by both NIOSH and ACGIH were considere d. Based on rule, the ACGIH TLV was used. MIDEQ protocol then divides this number by 100. See notes 1, 2, and 3.	NA	NA	2001	NA	ACGIH informa tion, 2001	Respirat ory tract injuries	MIDEQ: 8-hr averaging time

Chemical	Value (µg/m³)	Source	Species	Point of Departure	Chemical form	Exposure duration	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
	20	RIDEM STVs, Chronic (from Cal 2008)	Human	Cal REL 2008, based on Oral toxicity						Cardiovascular, nervous systems	Only a chronic AAL is available.
	20	NJDEP (from Cal 2008)	NA	Cal REL 2008, based on Oral toxicity	NA	NA	2011	NA	NA	NA	NA
	2	TCEQ AMCV, short-term, health-based, Interim value	Human	NC effects from occupational exposure	NA	NA	2003	NA	NA	Intended to minimize the potential for eye and upper respiratory tract irritation, as well as systemic effects such as headache, garlic odor of the breath, metallic taste,	NA

Chemical	Value (µg/m³)	Source	Species	Point of Departure	Chemical form	Exposure duration	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
										and skin rashes.	

*No Oregon ABC value available for selenium.

No AEGLs, ERPGs, or Minnesota DOH HRVs or HBVs available for selenium.

Note 1: From MIDEQ: "The combined ambient impact of all selenium and inorganic selenium compounds with CAS # 7446-08-4, 7746-34-6, 7783-00-8, 10102-18-8, and 13410-01-0 cannot exceed 2 ug/m3 (8-hr averaging time)."

Note 2: The ITSL for selenium is 2 µg/m3 based on an 8-hr averaging time. This screening level does not apply to organic forms of selenium.

Note 3: For more-detailed information, see MIDEQ Interoffice Communication: Selenium and Inorganic Selenium Compounds File (CAS# 7782-49-2); Subject: Screening Level for Selenium and Inorganic Selenium Compounds; Date: March 12, 2015.

Proposed 24-hour screening number

2,000 ng/m^3, based on Michigan and Texas short-term toxicity values.

Rationale

Both Michigan and Texas short-term toxicity values are derived (according to statutes in each state) by dividing occupational exposure limits by a factor of 100. State agency toxicologists preferred not to use adjusted occupational values, but in this case, the only other option was a chronic inhalation value that California OEHHA had extrapolated from a study where the actual exposure was via the oral route. This adjusted occupational value used by Michigan and Texas is lower (i.e. more health protective/conservative) than OEHHA's route-to-route extrapolated value. New Hampshire does have a tox value of 710 ng/m3, which is also extrapolated from occupation exposure limits by dividing by the same factor of 100 and an additional factor of 2.8 reserved for "high toxicity" compounds. OHA/DEQ toxicologists do not consider this additional factor justified in the case of selenium, which is an essential nutrient. In addition, the 710 ng/m3 is significantly out of step with short-term toxicity values used by other agencies and is not based on any additional toxicological science.

Selenium Background Information

Description and uses

Selenium is an essential trace element in humans and other species. Selenium compounds are used in the glass industry as decolorizing agents and in the rubber industry as vulcanizing agents, and also as part of photographic and xerographic toning baths, insecticides, and photoelectric cells. Selenious acid is a component of gun-cleaning chemicals; selenium sulfide is used in shampoos as an anti-dandruff agent. The most widely used selenium compound in industry is selenium dioxide, which catalyzes reactions of organic compounds and is produced by the oxidation of selenium with nitric acid followed by evaporation or by burning selenium in oxygen. Largest anthropogenic sources of atmospheric selenium are from combustion of fossil fuels and the production/refining of copper; particulates are the primary expected form of the compound.

Tons per year emitted to Oregon's Air

From regulated sources: 1.3

Health Effects

Acute occupational exposure to selenium dioxide resulted in bronchospasm, irritation of the upper respiratory passages, violent coughing, and gagging with nausea and vomiting.

Sensitive populations

None identified in literature that was reviewed.

Chemical	Value (µg/m^3)	Source	Species	Point of Departure	Form of Compound used	Duration of Exposure	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
Styrene CASRN 100-42-5	21,300	ATSDR	human	NOAEL	styrene	four 15-minute exposures to peak concentrations of 49 ppm, 49 ppm, or 49 ppm with four 15-minute exposures to peak concentrations of 98 ppm for 6 hours	2010	10 for human variability	Ska 2003	neurotoxicity	acute (14 days or less)
	1000	EPA IRIS (RfC)	human	NOAEL	dose-response was characterized based on urine concentrations of mandelic acid and phenylglyoxylic acid levels normalized to creatinine	occupational exposure (mean 8.6 years)	1992	3 for data inadequacy, 3 for lack of information on chronic studies, 3 for intraspecies variability	Mutti 1984	CNS effects	chronic

Chemical	Value (µg/m^3)	Source	Species	Point of Departure	Form of Compound used	Duration of Exposure	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
	1000	MI ITSL (EPA RfC)	human	NOAEL	dose-response was characterized based on urine concentrations of mandelic acid and phenylglyoxylic acid levels normalized to creatinine	occupational exposure (mean 8.6 years)	1992	3 for data inadequacy, 3 for lack of information on chronic studies, 3 for intraspecies variability	Mutti 1984	CNS effects	chornic (annual)
Styrene CASRN 100-42-5	21,000	OEHHA REL	human	NOAEL	styrene	1 hour	1999	10 for intraspecies variability	Stewart 1968	respiratory system	acute (1hr)
	900	OEHHA REL	human	BMC	dose-response was characterized based on urine concentrations of mandelic acid and phenylglyoxylic acid levels normalized	occupational exposure (mean 8.6 years)	2000	3 for intraspecies variability	Mutti 1984	CNS effects	chronic

Chemical	Value (µg/m^3)	Source	Species	Point of Departure	Form of Compound used	Duration of Exposure	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
					to creatinine						
	1000	Rhode Island AAL (EPA RfC)	human	NOAEL	dose-response was characterized based on urine concentrations of mandelic acid and phenylglyoxylic acid levels normalized to creatinine	occupational exposure (mean 8.6 years)	2008	3 for data inadequacy, 3 for lack of information on chronic studies, 3 for intraspecies variability	Mutti 1984	CNS effects	Acute (24hr)
Styrene CASRN 100-42-5	21,000	MN HRV (based on acute CA REL)	human	NOAEL	styrene	1 hour	2001	10 for intraspecies variability	Stewart 1968	eye and respiratory system irritant	acute
	1000	New Jersey (based on IRIS RfC)	human	NOAEL	dose-response was characterized based on urine concentrations of	occupational exposure (mean 8.6 years)	2011	3 for data inadequacy, 3 for lack of information on chronic studies, 3 for intraspecies variability	Mutti 1984	CNS effects	chronic

Chemical	Value (µg/m^3)	Source	Species	Point of Departure	Form of Compound used	Duration of Exposure	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
					mandelic acid and phenylglyoxylic acid levels normalized to creatinine						
	21,000	New Jersey (based on CA REL)	human	NOAEL	styrene	1 hour	2011	10 for intraspecies variability	Stewart 1968	eye and respiratory system irritant	1 hr
Styrene CASRN 100-42-5	22,000	Texas AMCV (ReV)	human	NOAEL	styrene	1 hour	2015	10 for human variability	Stewart 1968	eye, nose and throat irritation, nausea, impairment of coordination and balance	Acute
	17,000	NYSDEC SGC (based on ACGIH STEL)		ACGIH TLV							Acute
	70	WHO								Odor threshold	30 min

Chemical	Value (µg/m^3)	Source	Species	Point of Departure	Form of Compound used	Duration of Exposure	Date	Uncertainty Factors	Critical study	Endpoint	Recommended Averaging Time
	260	WHO	Human	LOAEL	styrene	Variable (occupational)		10 for interindividual variability, 10 for use of LOAEL	Harkonen 1978, Lindstrom 1992, Mutti 1984	Reduced visuomotor accuracy and verbal learning skills in humans	weekly
	554 mg/m3	EPA AEGL-1					2007				8hr
	400	Ontario AQCC (adjusted from WHO)								Odor/health	24hr

Proposed 24-hour screening number

21,000 µg /m^3 ATSDR acute MRL

Rationale

The proposed screening level concentration is consistent with the ATSDR acute MRL, and acute standards adopted by several states including the CA OEHHA acute (1 hr) REL, Minnesota, New Jersey and Texas. The ATSDR acute MRL is based on controlled short-term non-occupational human exposure studies. EPA IRIS only provides a chronic exposure RfC. The only states with more conservative acute exposure guidelines are New York (which is based on the ACGIH TLV) and Rhode Island (which just uses the chronic RfC as a 24hr standard). An argument could be made for using the more protect WHO weekly standard, but documentation of this standard is fairly limited. The stricter standard used by Ontario was originally based on odor thresholds but now is attributed to potential health effects (based on an adaptation of the WHO standard for weekly averaging times).

Styrene background

Description and uses

Styrene is used in manufacturing plastics, synthetic rubber, polystyrene, and propylene oxide. It is found in a range of consumer products including packing materials, electrical insulation, home insulation, fiberglass, disposable cups/plates, and carpet backing. In addition to industrial sources and consumer products, people are exposure to styrene from

cigarette smoke, photocopiers, and automobile exhaust. Styrene is usually broken down relatively quickly in air (1-2 days). Background levels in outdoor air range from 0.06–4.6 parts per billion (ppb).

Health Effects

The nervous system is the primary target of inhaled styrene. Effects of exposure in occupational settings include delayed response time, tiredness, concentration problems, balance problems, and color vision impairment. In animals very high exposures have resulted in hearing loss. IARC has classified styrene as a possible carcinogen.

Sensitive populations

Workers exposed to high levels of noise may be more susceptible to the potential effects of styrene on hearing loss. There is very little information on the potential impacts of styrene on young children. Based on occupational epidemiology studies and laboratory animals, there is no strong evidence for an increased risk of birth defects.

List of Acronyms in Proposed 24-Hour Screening Level Documents

AAL – Rhode Island Acceptable Ambient Levels

ABC – Oregon Ambient Benchmark Concentration OHA – Oregon Health Authority

ACGIH – American College of Governmental Industrial Hygienists

AEGL – EPA's Acute Environmental Guideline Level

AMCV – TCEQ's Ambient Monitoring Comparison Value

ASIL – Washington Acceptable Source Impact Level

ATSAC – Air Toxics Science Advisory Committee

ATSDR – Agency for Toxic Substances and Disease Registry

BMCL – Benchmark Concentration Level

CASRN – Chemical Abstract Services Registry Number

DEQ- Oregon Department of Environmental Quality

DOH – Minnesota Department of Health

EPA – United States Environmental Protection Agency

ERPGs – California Emergency Response Planning Guidelines

ESL – TCEQ Environmental Screening Level

HBV – Minnesota Health Based Value (have not gone through public vetting)

HRV – Minnesota Health Risk Value (have gone through public vetting)

IRIS – EPA's Integrated Risk Information System

ITSL – Michigan's Initial Threshold Screening Level

LOAEL – Lowest Observable Adverse Effect Level

MIDEQ – Michigan Department of Environmental Quality

MRL – ATSDR's Minimal Risk Level

NAAQS – National Ambient Air Quality Standard

NJDEP – New Jersey Department of Environmental Protection

NOAEL- Now Observable Adverse Effect Level

NYSDEC – New York State Department of Environmental Conservation

OAR – RIDEM Office of Air Resources

OEHHA – California Office of Environmental Health Hazard Assessment

OEL – Occupational Exposure Limit

PPRTV – EPA's Provisional Peer Review Toxicity Value

REL – California's Reference Exposure Level

ReV – TCEQ inhalation Reverence Value

RfC – EPA's Inhalation Reference Concentration

RIDEM – Rhode Island Department of Environmental Management

SGC – New York Short-term Guideline Concentration

STAR – Louisville Kentucky's regional air authority's air toxics program (Strategic Toxic Air Reductions) program

TCEQ – Texas Commission on Environmental Quality

TLV – ACGIH Threshold Limit Value

UCLD10 – ATSDR Lower 95^{th} confidence limit on urinary cadmium level that would result in a 10% increase in the prevalence of β2-microglobulin proteinuria

UF – Uncertainty Factor