

# Air Toxics Science Advisory Committee

## Meeting minutes

March 18, 2015 ATSAC Meeting



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### Introduction

The fourth meeting of the 2014/2015 ATSAC took place in Conference EQC-A at DEQ HQ, 811 SW Sixth Avenue, Portland OR 97204, from 9 am to Noon. Five of the six current ATSAC members were present, and provided a quorum: Bill Lambert, Dean Atkinson, Bruce Hope, Max Hueftle, and David Farrer. Kent Norville could not attend this meeting.

### Presentation: 1999 Emissions Inventory vs. 2011 Emissions Inventory Data Comparison (Mr. Chris Swab, DEQ)

Chris Swab, DEQ Senior Emissions Inventory Analyst, gave a presentation of the results of the work requested of him by the ATSAC at the February 2015 ATSAC meeting. At that time, ATSAC requested that Swab present a summary of increased and decreased emissions in chemicals from 1999 to 2011, as available from the Emissions Inventory (also commonly referred to as the EI) and identify chemicals which appear to have dropped off the list or been added to it.

Acetaldehyde and Formaldehyde emissions show big increases from 1999 to 2011. The noticeable increase in butadiene emissions is due (82%) to wildfires and prescribed burning, and the presence of related biogenic organic materials. Plus, the extraordinarily large Biscuit-area fires increased emissions in this category in 2002, while only mid-size fires occurred in 2011. Also, there was consistent wildfire activity from 2005-2008. Wildfires contributed to benzene changes, as well. More than half of the 2011 formaldehyde emissions are attributable to biogenic sources.

Polycyclic aromatic hydrocarbons (commonly referred to as PAHs) are a subcategory of organic materials. It should be noted that both the 1999 and 2011 EI datasets represent snapshots in time, with the 2011 snapshot providing better details than the 1999 snapshot due to improved monitoring and interpretation protocols.

Swab talked about compounds measured in air (such as diesel exhaust) that do not have assigned Chemical Abstract Service Registry Numbers; it is difficult to extract data from the EI for a chemical that had no CASRN number. Swab used the current Oregon Ambient Benchmark Concentration list and the list of chemicals in Hope's 1999 report to choose the chemicals he compared for this presentation. The Oregon Ambient Benchmark Concentrations are most typically referred to as ABCs.

The PAH/polycyclic organic matter emissions have decreased significantly from 1999 to 2011. Part of this difference is due to double-counting in 1999 based on the way the categories were parsed. Another example of a changed category is the category for nickel and nickel compounds, since only the "Nickel compounds" category was available in 1999; nickel categories are different in the 2011 data, so it is difficult to directly compare EI nickel results from 1999 to those in 2011.

Air toxics contributing to cancer risk in Oregon were discussed. The EI database only provides emissions data; data from the National Air Toxics Assessment (also referred to as NATA) becomes available sometime after the EI results are published, and assigns modeled cancer risk

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811 SW 6<sup>th</sup> Avenue  
Portland, OR 97204  
Phone: 503-229-6458  
800-452-4011  
Fax: 503-229-5850  
Contact: Sue MacMillan  
[Macmillan.susan@deq.state.or.us](mailto:Macmillan.susan@deq.state.or.us)

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values to the air data. Currently, 2011 NATA data is not yet available, and so we cannot yet discuss the cancer risks related to the 2011 EI data.

Quinoline and lindane present in 1999, but not in 2011. Swab made some guesses as to why these two chemicals didn't appear in 2011, including the possibility that lower emissions of these chemicals occurred in 2011. Also, pulp and paper activities in Oregon have dwindled; pulp and paper had been a major source of lindane previously. However, although lindane is known to be persistent in soil and the environment, it may finally have broken down enough by 2011 so as not to be volatilizing to the air anymore.

Swab also gave a breakdown of the types of sources that emitted the typical 15 PAHs in 2011. Regarding the category of diesel particulate matter have a particle diameter of 2.5 micrometers or less (commonly referred to as PM2.5), there was a change in methodology because different models were used between 1999 and 2011. Swab also gave a breakdown of the subsets of on-road and non-road chemical emissions of diesel PM. Construction and mining operations are the biggest contributors to non-road emissions of diesel PM.

Swab stated that he hasn't yet had time to determine whether some chemicals are no longer of concern, or whether there are some new ones. However, it was then suggested by the ATSAC that any further drill-down by on this data is probably not the most efficient use of Swab's time. The ATSAC chair praised Swab for the complex work he had to do in order to give today's presentation.

The ATSAC wants to make sure we look at all chemicals that we need to (for example, start with 151 chemicals, screen down to the 50 most relevant due to high emissions, then rank this smaller group according to related toxicity data; finally, then, the ATSAC would review perhaps a total of 25 to 30 compounds in total.

Swab stated that we need 2011 NATA data as well as the currently-available 2011 EI data to be able rank emitted chemicals of concern more accurately. He pointed out that in 1999, both the EI and NATA data were available; right now, all we have for 2011 is EI data.

EPA has told Lane Regional Air Protection Agency that Oregon's EI contains one of the more complete data sets they've seen.

Swab stated that his presentation regarding comparison of 1999 EI data to 2011 EI data is valuable because it indicates *why* a chemical might have dropped off the list. His intent was to give the ATSAC a good overview of the 1999 data in comparison to the 2011 EI data. This comparison also identifies items where over-estimation of emissions are suspected or known. An ATSAC member mentioned that he just wanted to try to identify chemicals dropping off the list versus chemicals that may have appeared since 1999, and stated that the ATSAC will need to do this at some point.

The ATSAC would like to close this current EI information loop by documenting the results. To address this task, the committee asked Swab to write a memo summarizing the work he did to create today's presentation. As with all work products, the ATSAC and DEQ will need to document for the public record our rationale for decisions made. Note, among other things, that the way 1999 was captured and interpreted was a lot different from how the EI data was gathered in 2011, and is akin to comparing apples to oranges. No real discernable pattern of change is observable between chemicals emitted in 1999 and chemicals emitted in 2011. But the ATSAC can still use the 2011 EI data in a limited and cautious way until the NATA data comes out. The

ATSAC noted that when the committee discusses PAHs, they'll want to hear from Swab again. It was cautioned to be careful about putting too much emphasis on wildfires and prescribed burning percentages in regard to proportions of a particular chemical emitted, because this



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811 SW 6<sup>th</sup> Avenue  
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Phone: 503-229-6458  
800-452-4011

Fax: 503-229-5850

Contact: Sue MacMillan  
[Macmillan.susan@deq.state.or.us](mailto:Macmillan.susan@deq.state.or.us)

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information is related to a statewide estimate, and regional and small area emissions inventories will vary.

LRAPA compared 2005 NATA data to on-the-ground monitoring data collected by LRAPA in 2005, and found that at that time, the NATA modeling under-predicted the impact of metals by a factor of 2.

**Presentation: Partially-Completed Screening Review of Chemicals with ABCs which have not yet been Reviewed by the ATSAC (Sue MacMillan, DEQ)**

Sue MacMillan, DEQ Air Toxics Science and Policy Analyst and the agency lead for the ATSAC, presented a draft summary of her review of the current list of 52 compounds with ABCs to determine the ones that require review of newly available information that may impact the choice of concentration level for certain ABCs.

MacMillan presented color-coded text of her screening of a portion of the chemicals, rather than a summary table, which she will create over the next month. She chose one chemical as an example, to demonstrate how she came to the conclusion that the chemical may not need ATSAC review. She emphasized that in the case of each chemical she screened, the final decision will be the ATSAC's, not hers.

The ATSAC asked MacMillan what she means when she states that there is no new toxicity information for a chemical. Does she mean that no new studies have come out, or that no new interpretations of a former study have been made available? Or, that someone did a review of existing information, but no changes were suggested?

She explained that she had not delved into the toxicity information that deeply, but can do so. She pointed out that she did provide related dates for the toxicity information she had reviewed, and also explained in the text the basis of the toxicity information in her screening.

A committee member asked MacMillan if her screening check determined whether actual new toxicity information had become available, or has a former study just been re-analyzed (for example, were uncertainty factors added). The committee emphasized that EPA does a toxicity review of a chemical as part of IRIS process, and also provides the agency's "draft" opinions, in some cases, as a Provisional Peer Reviewed Toxicity Value. In regard to EPA Regional Screening Levels, one should note which toxicity values EPA used to come up with their Regional Screening Levels, because none of the toxicity values referenced in the related tables are original values; they have come from other recognized peer-reviewed sources.

MacMillan stated that she was familiar with this information, and then pointed out that EPA Regional Screening Levels are typically about three times higher than the toxicity value on which they're based, due to exposure parameters used in concert with toxicity information.

A committee member emphasized that Provisional Peer-Reviewed Toxicity Values are true EPA toxicity values; and that EPA does this type of review when there are no other peer-reviewed toxicity values available from other sources.

MacMillan stated that she only used the EPA Regional Screening Levels as a rough check against the toxicity values being reviewed, since these EPA levels are not part of the recognized source hierarchy used by the ATSAC. She plans to create a table summarizing all of her screening check information, and additional technical details of the kind just discussed. She will then submit the draft summary table to the ATSAC for review. Her goal is to pinpoint the chemicals for which new toxicity or other information has become available since 2010, and assist the ATSAC on concentrating their review time on those chemicals selected by ATSAC for evaluation. For example, MacMillan would like the ATSAC to review 1,3-Butadiene simply because of the increase in emissions from 1999 to 2011, as presented by Swab.



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811 SW 6<sup>th</sup> Avenue  
Portland, OR 97204  
Phone: 503-229-6458  
800-452-4011  
Fax: 503-229-5850  
Contact: Sue MacMillan  
[Macmillan.susan@deq.state.or.us](mailto:Macmillan.susan@deq.state.or.us)

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A committee member discussed the fact that even though many of the chemical increases from 1999 to 2011 are due to wildfires and prescribed burning, the Committee will need to look at the toxicity information for these chemicals anyway. MacMillan confirmed that the DEQ will need ATSAC recommendations for ABCs based on toxicity information alone; it will be the responsibility of DEQ to decide what to do about the feasibility or utility of using ABC values in their regulatory programs.

A committee member emphasized that these emissions are based on a state-wide area; so wildfires and burning are a kind of a regional “background” issue we will have to assume is always part of the Oregon emissions data.

Ms. MacMillan explained that although she has yet to provide the ATSAC with a complete summary table of her screening check of the ABCs, that she nonetheless was able to identify a few chemicals for which new toxicity information has become available, and thus definitely require review by the ATSAC, including:

- 1.) Methylene Chloride
- 2.) Perchloroethylene/tetrachloroethylene
- 3.) Toluene
- 4.) Acrolein
- 5.) Carbon Tetrachloride
- 6.) Chloroform
- 7.) 1,3-Butadiene (based on its EI increase from 1999 to 2011)

In regard to benzene and vinyl chloride, MacMillan’s initial check of the toxicity information available for these two chemicals indicated that there doesn’t appear to be any new toxicity information, so perhaps the ATSAC doesn’t need to review these two chemicals. But she pointed out that these are two highly visible and controversial chemicals, so maybe the ATSAC should review them on that basis.

A committee member cautioned Ms. MacMillan to be very careful in her screening documentation to identify not only toxicity changes which could make the ABCs more stringent, but also consider and provide any new toxicity information that indicates the ABCs should be less stringent.

MacMillan pointed out that phosgene and fluorides other than hydrogen fluoride are two new chemicals/chemical groups that almost certainly will need to be reviewed by the ATSAC, based on new emissions information that’s become available recently. A committee member pointed out that fluorides disperse quickly in the atmosphere are thus not likely present at concentrations that will result in adverse exposure to human receptors, and thought this is a consideration that the ATSAC needs to be aware of. MacMillan acknowledged these facts, but said that DEQ will still need an ABC for fluorides, and then the Agency will decide later how to facilitate use of the ABC for fluorides. The DEQ will still need a toxicity-based value from the ATSAC. On the next ABC table published, Ms. MacMillan would like to include a new column for “Additional Information”, so that DEQ can educate the public on how ABC decisions were made.

The committee recommended that this additional information should include details such as, for example, what form of the chemical we believe people will actually be exposed to, and explain that in some cases a very stringent ABC was chosen to protect people against, say, the most toxic form of the chemical in question, although that most toxic form is unlikely to be present at exposure concentrations that will actually cause harm. Other considerations to provide as additional information, if relevant to a chemical’s ABC: aerosol versus particulate form of a chemical, soluble versus insoluble forms, distance to human receptors, etc.



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811 SW 6<sup>th</sup> Avenue  
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Phone: 503-229-6458  
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MacMillan then asked the committee if there had been additional ABC rulemaking work done after 2006? In other words, at what point did the previous ATSAC say “we’re done” in regard to review of toxicity information at that time?

Committee members said that the bulk of the work of identifying ABCs was completed in 2006, when rulemaking occurred: but the ATSAC performed review of new toxicity information that became available post-2006 for a few chemicals, and retained or revised 2006 ABCs for these few chemicals on that basis in 2009, the results of which were documented in 2010. Mercury, manganese, lead, and formaldehyde were some (if not all) of the compounds that were re-reviewed after 2006, because new toxicity information became available and the ATSAC was still in session; probably a total of six or seven compounds were reviewed post-2006.

Hope, who was the DEQ lead for the former ATSAC, does not know for sure if these post-2006 changes were reflected in rulemaking or not. Lambert stated that the 2010 ABCs are current through 2009; he remembers appearing before the EQC in regard to the 2006 rulemaking, but does not think he appeared again post-2006. He wondered if this information didn’t somehow move through on paper to the EQC. MacMillan committed to finding out whether the post-2006 ABC changes were made part of the rule.

Hueftle suggested that, as part of the ABC chemical ranking process, we might want to consider comparing air monitoring data to 2011 NATA results, to see if they match; in other words, ground-truth the model’s results. It should be remembered that for the 2005 NATA data that LRAPA reviewed, NATA was under-predicting metals by a factor of 2 to 10 times, based on LRAPA ground-truthing of monitoring data available at that time.

MacMillan acknowledged this issue, and said that the ATSAC can do some review work before the 2011 NATA comes out that won’t compromise review decisions. For example, we already know that the ATSAC is going to have to review PAHs and then diesel.

Lambert requested that MacMillan continue to add to and archive her color-coded notes as she creates the related summary table. MacMillan agreed and requested that the ATSAC serve as peer reviewers of the correctness and accuracy of the summary table information, as it will not have been peer-reviewed previously. The ATSAC agreed.

Hope cautioned MacMillan to try to keep the various toxicity values in the same units as those used for ABCs, i.e., micrograms per cubic meter.

### **ATSAC Administrative Item**

Four ATSAC members approved February ATSAC meeting minutes, while David Farrer abstained due to his absence from the February 2015 ATSAC meeting.

### **Break**

### **Discussion of Review of Toxicity Data for Cadmium, Manganese, and Nickel Compounds**

**Cadmium:** Dean Atkinson and David Farrer, reviewers.

Farrer presented information for cadmium. Cadmium is a heavy metal used in batteries and pigments, and the stabilization of plastics. The emission rate of 0.1 tons/year was obtained from the 2011 Air Emissions Inventory Pivot Table provided by MacMillan. Much of the cadmium seen in the Portland metro area comes from residential heating, and also from the burning of natural gas. Cadmium detections are higher than predicted from the Oregon monitoring and modeling; it’s not known why this is occurring. However, Farrer doesn’t think that cadmium levels had exceeded the ABC for cadmium very often. Cancers of the respiratory tract drive cadmium cancer numbers; for the non-cancer effects of cadmium, the kidney is the target organ.



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811 SW 6<sup>th</sup> Avenue  
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Phone: 503-229-6458  
800-452-4011  
Fax: 503-229-5850  
Contact: Sue MacMillan  
[Macmillan.susan@deq.state.or.us](mailto:Macmillan.susan@deq.state.or.us)

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The half-life of cadmium in the body is long, due to ongoing cycling of cadmium in the system, sometimes for years in the kidney. The half-life of a chemical in the body is defined as the time required for the concentration of that chemical in the body to decrease by half. Cadmium can also cause bone problems, probably due to kidney impacts; specifically bone mineralization, or brittle bones. The human populations most sensitive to cadmium exposure are people with preexisting kidney dysfunction, and people with diabetes.

The current ABC for cadmium is based on the U.S. Environmental Protection Agency Integrated Risk Information System (commonly referred to as IRIS) Inhalation Unit Risk for lung cancer; OEHHA has a different cancer value for cadmium, but that value hasn't changed since the former 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. The Agency of Toxic Substances and Disease Registry and EPA are identical in their recommendations.

Based on the ATSAC's policy to use the higher of two values when two similar studies are compared, and on the fact that the results of the two studies are within an order-of-magnitude of each other, ATSAC should retain the current ABC of  $6 \times 10^{-4}$  micrograms per cubic meter for cadmium.

The cadmium study done by Michael Boone in 1989 documented a cancer outcome for cadmium exposure and also looked at urinary biomarkers related to non-cancer effects, but the cancer effects were the driver in this case. The results are related to the National Institute for Occupational Safety and Health studies, which assessed highly exposed worker groups. More-current European and Chinese studies replicate Michael Boone's work.

The article referring to the exposure of Native Americans to cadmium, which was provided by MacMillan to the ATSAC as a review item, also supports the fact that cadmium causes cancer.

Also, the retention of cadmium via the inhalation pathway is higher than that which occurs for the oral pathway. Thus, the carcinogenicity evidence for cadmium is very strong. Oregon's cadmium benchmark is in line with this information.

In 1999, IRIS published a toxicological review which was dated *after* the IRIS Inhalation Unit Risk value was established, and the review recommended an Inhalation Unit Risk value nearly identical to that used by the California Office of Environmental Health Hazard Assessment, also referred to as OEHHA. The air concentration associated with a one-in-a-million risk resulting from the proposed Inhalation Unit Risk value matched the OEHHA number, which was 0.2 nanograms per cubic meter, rather than 0.6 nanograms per cubic meter. But for some reason, no IRIS change occurred. It is unknown why EPA didn't publish the recommended lower value.

Thus, the ATSAC might want to watch for a change in the IRIS Inhalation Unit Risk based on the 1999 external peer-reviewed draft of the IRIS toxicological review. This is just a heads-up to the committee that a revision might occur.

**ATSAC VOTE:** Retain current ABC for Cadmium of  $6 \times 10^{-4}$  micrograms per cubic meter. Unanimous agreement.

**Manganese:** Bill Lambert and Kent Norville, reviewers.

Lambert presented the manganese toxicity information, as Knorville was not in attendance today. The original ABC for manganese of 0.2 micrograms per cubic meter was set in 2006, and revised to 0.09 micrograms per cubic meter 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. Manganese is used as alloy in steel production and in the manufacturing of dry-cell batteries.

*Clinical disorders:* The central nervous system and the lungs are affected by exposure to manganese. But the central nervous system is the primary target, and is related to the



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811 SW 6<sup>th</sup> Avenue  
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Phone: 503-229-6458  
800-452-4011  
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development of Parkinson's disease. Chronic exposure to excessive manganese levels can lead to a variety of psychiatric and motor disturbances, termed manganism. It is still observed in occupational settings among workers exposed via welding fumes. Exposure causes palsy, tremor. Also, there are documented changes in neurological function that occur over weeks to months in test animals exposed to very low manganese levels.

*Epidemiology* studies are particularly informative. Though these types of cadmium studies, lowest-observed-adverse-effect levels for cadmium were obtained, based on a study of workers exposed to high levels of manganese.

In summary, there is a "three-legged stool" of evidence supporting the toxicity of manganese: In-vitro studies, in-vivo studies, and epidemiological studies.

One complication must be noted. Manganese is an essential nutrient and a trace element. Therefore, there is a graphically depicted J-shaped curve of adverse effects at too-low and too-high exposures. Americans are receiving anywhere from 0.03 to 3.5 milligrams of cadmium per day in their diet, so deficiencies aren't observed in the clinical setting. The main concern with cadmium exposure should be related to chronic over-exposure to manganese; this type of exposure is what the ATSA should focus on.

EPA critical studies for cadmium include two studies and an additional analysis performed by Roels 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 cadmium: 1) Look at occupational epidemiology results first, then extrapolate down to 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.

The Manganese Information Group [MIG] (files received by MacMillan on 3-20-15 and then forwarded to the committee) takes the latter perspective, arguing that the use of epidemiology studies is not the best approach and that the uncertainty factors are needlessly conservative. In his estimation, Lambert found that MIG, while using the PBPK model, was making equally large assumptions in regard to their own arguments. Their use of PBPK modeling does not serve to advance our understanding of the mechanism of toxicity of manganese, but rather presents arguments against the use of a particular approach. In his opinion, the limitations of the available epidemiologic data support the application of uncertainty factors, which are conservative. Note that the MIG is funded by industry, and therefore their consultant's modeling results should be considered within the context of this conflict of interest.

Dr. Peter Spencer, who is a scientist at Oregon Health Sciences University. Published a book entitled "Experimental and Clinical Neurotoxicology". Lambert quoted from this book: "The target site in brain (palladium) exhibits selective vulnerability to manganese. It's proposed that this vulnerability is related to theories of oxidative stress on neuronal tissues." This potential mechanism is not discussed anywhere in the MIG consultant report, which Lambert sees as a severe weakness. So, in Lambert's opinion, the ATSA 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.

In 1992, the related lowest-observed-adverse-effect level for cadmium was estimated as 0.05 micrograms per cubic meter. In 1997, the same authors came up with a different estimate of 0.34



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micrograms per cubic meter. Since then the median concentration of the two studies, 0.15 micrograms per cubic meter, is the value that has been relied upon to represent a lowest-observed-adverse-effect level related to manganese exposure effects of short-term memory, hand tremor, and reaction time.

In 2006, ATSAC set the ABC for manganese at 0.2 micrograms per cubic meter; then ATSAC revised it in 2009 to 0.09 micrograms per cubic meter in order to be consistent with the OHHEA Reference Exposure Level, which specifically applied an uncertainty factor to protect children. A committee member asked if OEHHA had used uncertainty factors to come up with new value, or had the OEHHA used a benchmark dose protocol? After some discussion, it was agreed that the toxicological Point of Departure was the lowest-observed-adverse-effect level, not a no-observed-adverse-effect level. The ATSAC chair told the committee that it should rely on the three studies by Roels; it's very limited information, but it's what's available. And because the information is limited, application of uncertainty factors is warranted.

It was stated that the Agency for Toxic Substances and Disease Registry (typically referred to as ATSDR) used the same basic study, but used modeling to determine that an uncertainty factor of 10 for individual variability is sufficient to cover the additional risk to children. Also, an additional uncertainty factor of 10 was applied due to a limited data base. This ATSDR review and the related Toxicity Profile became available in 2012; this information was not available to the ATSAC in 2010.

After some discussion, the committee agreed, that in regard to the MIG report, it is basically talking about the same acknowledged IQ deficit as we have for lead, and the precise mechanism for its effect on the brain is not yet understood, but we rely on epidemiological data to set our standards. The MIG model is inherently limited by its lack of inclusion of a biological mechanism. Another weakness of the MIG report is their focus on brain effects from air levels, because it is not always true that the site of highest concentrations of chemical exposure is the relevant biological site of damage.

ATSAC VOTE: Retain current ABC of 0.09 micrograms per cubic meter. Unanimous agreement.

**Nickel and nickel compounds:** Max Hueftle and Bruce Hope, reviewers.

Hueftle discussed the characteristics of nickel and nickel compounds. Nickel is highly resistant to corrosion, and is used in metal alloy production, plating, cast iron. Some forms of nickel are essential nutrients. Food intake is a major source of nickel exposure, with a typical ingestion rate of 100 to 300 micrograms per day of nickel. In 2005, ATSAC adopted a single ABC of 0.004 micrograms per meter nickel; comments in 2006 facilitated the adoption of ABCs for various nickel compounds. The Portland Air Toxics Study, or PATS, estimated that in 2017, 2.6 tons/year of nickel will be emitted in the Portland metro area. The major anthropogenic source of nickel in the U.S. is the use of fossil fuel. But in PATS, point sources were identified as the primary source of nickel emissions.

DEQ doesn't speciate nickel when it analyzes air samples, but dispersion modeling can be done to estimate amounts of nickel species. As mentioned before, the 2005 NATA data for nickel were 2 to 10 times lower than nickel concentrations monitored by the Lane Regional Air Protection Authority.

Nickel refinery dust, which is assumed to be made up primarily of nickel subsulfide, was the form of nickel originally considered to be carcinogenic; additional nickel compounds are now considered to be carcinogenic, as well.



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Nickel compounds can be separated into two general categories: (1) those that are insoluble in water, are thought to be carcinogenic, are generally emitted as particulates primarily from smelting, refining, and metals processing operations, and include nickel refinery dust, nickel subsulfide, nickel sulfide, and nickel oxide; and (2) those nickel compounds that are soluble in water, are generally emitted as aerosols primarily from nickel plating operations, and include nickel sulfate, nickel chloride, and several other nickel compounds. Oregon's only nickel smelter, originally located near Riddle, Oregon, closed in the 1990s. Current industrial sources are likely to be metals processing operations or plating operations. Nickel is also emitted as a result of fuel combustion, is present in soil, and can be leached from cookware; it doesn't only come from industrial sources.

The Hanna Mine on Nickel Mountain outside Riddle was Oregon's only nickel mine and smelter. The mine closed sometime in the 1980s, but the smelter seems to have struggled on as Glenbook Mining into the 1990s using ore imported from New Caledonia. A United Kingdom-based mining company has recently proposed opening two new nickel mines in Curry County amid huge controversy; so the story of nickel emissions in Oregon may not be done yet. There is no record of an EPA Superfund listing for the Hanna Mine or smelter.

The committee asked: What is nickel subsulfide? A committee member responded that nickel subsulfide is any sulfide in which the number of sulfur atoms is fewer than the number of cation elements. Thus, the chemical formula for nickel subsulfide is Ni<sub>3</sub>S<sub>2</sub>, which is a nonstoichiometric form of nickel sulfide.

It was 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 micrograms per meter, 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 micrograms per cubic meter, 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.



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Solutions, Technical  
Services**

811 SW 6<sup>th</sup> Avenue  
Portland, OR 97204  
Phone: 503-229-6458  
800-452-4011

Fax: 503-229-5850  
Contact: Sue MacMillan  
Macmillan.susan@deq.state  
.or.us

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- (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.

Oregon would need to speciate nickel into the various nickel compounds in order to enable comparison of these new ABCs to appropriate types of nickel data. Other jurisdictions have generated values in the 0.004-to-0.01 microgram-per-cubic-meter range, depending on the nickel compounds in question. Texas has come up with slightly higher values, for example 0.07 micrograms per cubic meter for nickel sulfate. The Ontario Ministry of the Environment in Canada utilizes a value of 0.02 micrograms per cubic meter for all nickel compounds in PM10-size fractions. Washington's Acceptable Source Impact Level is 0.002 micrograms per cubic meter, which applies to all nickel compounds. The committee wondered what kind of nickel data is available to compare to these ABCs. Need to check with Chris Swab. You wouldn't apply the 0.004 micrograms per cubic meter value to nickel plating operations, because you'd be dealing with mainly aerosols in that case. Conversely, you wouldn't apply the aerosol-related value of 0.01 micrograms per cubic meter to operations that generate mainly particulates.

Although they're not doing it currently, the DEQ air laboratory would be able to speciate nickel present in air samples, although this would be expensive. However, if a problem was first identified via the less-expensive protocol of dispersion modeling, then the monitoring and analysis of nickel species in air wouldn't necessarily be needed. Thus, based on the modeling results, you could choose to decide whether or not to speciate nickel air monitoring data.

ATSAC VOTE: Create two new ABCs for nickel: 0.004 micrograms per cubic meter for insoluble nickel compounds, and 0.01 micrograms per cubic meter for soluble nickel compounds. Drop nickel refinery dust category altogether. Unanimous agreement.

However, a few days after the March 2015 ATSC meeting, additional information on nickel compounds was provided by Bruce Hope to the ATSAC. This additional information was discussed in the open ATSAC meeting in April 2015, and was then used to slightly revise the chemical species assigned to the insoluble and soluble nickel groups.

### **ATSAC Identifies Compounds for Review at April ATSAC Meeting**

Assignments for compounds to be reviewed for April 22, 2015 ATSAC meeting:

1. Methylene chloride: David, Max
2. Tetrachloroethylene/Perchloroethylene: Bill, Max
3. Toluene: Bruce, Dean
4. Acrolein: Dean, Bruce

Sue MacMillan and Bill Lambert will assign a chemical to Kent Norville and to the new ATSAC member for review at April ATSAC meeting. The committee will use the entire duration of the April ATSAC meeting to conduct chemical reviews.

Lambert discussed status of pending approval for a new member of ATSAC. Next ATSAC meeting will occur on April 22, not April 15. Typically, the monthly ATSAC meeting is held on the third Wednesday of the month.

MacMillan agreed to email all members the toxicity summary sheets used today to discuss the review chemical, and also Chris Swab's Powerpoint slides from his presentation.

### **Comments from Audience**



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811 SW 6<sup>th</sup> Avenue  
Portland, OR 97204  
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800-452-4011  
Fax: 503-229-5850  
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[Macmillan.susan@deq.state.or.us](mailto:Macmillan.susan@deq.state.or.us)

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Gregg Lande: EI and modeling, and then air monitoring, are two ways that DEQ determines risk. What forms of nickel are actually measured by DEQ? Does this mean that facilities will have to report soluble and insoluble forms of nickel, in order to eventually get to the risk determinations through comparison to ABCs for insoluble and soluble nickel compounds?

Committee response: Good points; this issue can be addressed as we prepare the related guidance. If total nickel exceeds the lowest benchmark(s), *then* you could decide to speciate/monitor various nickel compounds. Same approach could be used with cobalt.



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