MARCH 20, 2019

RE: Comments on Proposed Changes to Acceptable Source Impact Levels (ASILs)

Dear Ms. Guilfoil:

NCASI greatly appreciates the opportunity to provide input on the Proposed “WAC 173-460-150 Draft Table of ASIL, SQER and de minimis emission values.” NCASI conducts research on environmental topics relevant to the forest products industry. Over its 75-year history, it has conducted studies in a variety of areas related to air emissions and worked extensively in developing emissions data used in multiple National Emissions Standards for Hazardous Air Pollutants (NESHAP) rulemakings affecting this industry. NCASI also assisted EPA during development and implementation of the 2011 Pulp and Paper Information Collection Request (ICR), which was used by EPA as part of the Residual Risk and Technology Review (RTR) of the pulping, bleaching, and wastewater MACT (“Subpart S”) and the pulp mill chemical recovery NESHAP (“Subpart MM”). NCASI has assisted EPA in the development of the various iterations of the industrial boiler and process heater NESHAP (“Subpart DDDDD”) and also has extensive experience assisting the forest products industry and regulators in many states with development and implementation of science-based solutions to environmental issues, including occupational and community health risk assessments for substances of interest to the industry, such as hydrogen sulfide, formaldehyde, chloroform, and particulate matter. We draw from our experience and long history of involvement in these areas in providing input to Washington Department of Ecology (Ecology) on the proposed changes to the ASIL values in Table 150.

Our specific technical input can be summarized as follows:

1. There is a lack of evidence that the proposed use of an Age Dependent Adjustment Factor (ADAF) will provide health benefits to susceptible populations.
2. There appears to be a technical error in the proposed ASIL value or averaging period for mercury.

3. Clarity will be needed for implementation of the mercury ASIL.

Detailed technical discussion of this input is attached to this communication. NCASI would like to thank you for the opportunity to provide input on these rule changes and your consideration of the issues included herein. Please feel free to contact me at (813) 734-4385 or gjohnson@ncasi.org if you have questions or need more information.

Sincerely,

[Signature]

Giffe Johnson, PhD
Principal Scientist
DETAILED TECHNICAL COMMENTS

1.0 There is a lack of evidence that the proposed use of an Age Dependent Adjustment Factor (ADAF) will provide health benefits to susceptible populations.

EPA provided guidance for Age Dependent Adjustment Factors (ADAFs) for cancer slope factors to adjust carcinogenic potency during early life stages for substances considered to be ‘linear’ carcinogens in the document Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens, on which Ecology bases proposed changes to some ASILs. The agency’s purpose in proposing these changes is ostensibly to increase protection against cancer from exposure to carcinogens at earlier life stages. However, these life stage-based adjustments are (1) associated with substantial uncertainty; (2) being applied to standards that already contain multiple conservative assumptions; and therefore (3) unlikely to confer any additional public health benefit if implemented in their proposed form. The impact of these proposed changes is that many ASIL values may be substantially reduced, potentially impacting dischargers and government agencies that manage discharges, without clear evidence that a public health benefit will result. The scientific basis of Washington’s ASIL values would be strengthened if the agency were to reevaluate and revise its implementation of ADAFs and life stage susceptibility assumptions to be consistent with the current state of scientific evidence regarding early exposure to carcinogens, as well as the substantial limitations found in the EPA guidance document.

1.1 ADAF adjustment

In the proposed ASIL values for several linear, mutagenic carcinogens, including chromium VI and multiple polycyclic aromatic hydrocarbons (PAHs), Ecology proposes to multiply the cancer slope factor (CSF) by a factor of 10 for ages birth to 2 years, and to multiply the CSF by a factor of 3 for ages 2 to less than 16 years. These adjustments are weighted by the time spent in the age range of interest. The justification for increasing the CSF during earlier life stages is the hypothesis that certain types of modes of action for carcinogens have greater impact if they occur at an earlier life stage. For example, it is suggested that mutagenic modes of carcinogenesis may have a greater impact with early life stage exposure because a mutated parent cell may produce a greater number of daughter cells that inherit the mutation due to the rapid proliferation of cells that takes place at an earlier life stage. EPA acknowledges, however, that the scientific underpinings of ADAFs are not well characterized, and recommends them at least partially on the basis of policy rather than science (underlined for emphasis):

The Agency has also carefully considered both the advantages and disadvantages to extending the default potency adjustment factors to carcinogenic chemicals for which the mode of action remains unknown. It is the Agency’s long-standing science policy position that use of the linear low-dose extrapolation approach (without further adjustment) provides adequate public health conservatism in the absence of chemical-specific data indicating differential early-life susceptibility. At the present time, therefore, EPA is recommending these age-dependent adjustment factors only for carcinogens acting through
a mutagenic mode of action based on a combination of analysis of available data and the above-mentioned science policy position. (USEPA 2005, p 35)

Not all carcinogens with a mutagenic mode of action have been demonstrated to confer an exceptional early life stage risk and the degree of impact is poorly characterized. In addition, EPA notes that the linear extrapolation method provides adequate public health conservatism, unadjusted for ADAFs, because of the extremely low risk levels addressed by this approach. Without specific data that early life stage exposure for a substance (at environmentally relevant levels) is having an impact on cancer risk, ADAF application is not likely to confer any public health benefit.

1.2 Impact of ADAFs

By weighting the slope factor for ages 0 to <2 yr by a factor of 10 and weighting the slope factor for ages 2 to <16 yr by a factor of 3, resultant ASILs decrease by approximately 40% compared to the unweighted adult algorithm. Again, this reduction in the standard would apply to all ‘linear’ carcinogens, characterized by a mutagenic mode of action, and result in lowering the ASIL for at least eight substances.

While the authors of the Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens put forth some plausible hypotheses to suggest that early life stage exposure may increase risk of developing cancer, the data to support them are limited. This is noted in the EPA document:

The relative rarity in the incidence of childhood cancers and a lack of animal testing guidelines with perinatal exposure impede a full assessment of children’s cancer risks from exposure to chemicals in the environment. Unequivocal evidence of childhood cancer in humans occurring from chemical exposures is limited. (USEPA 2005, p 2)

Not only is the underlying data to support or quantify an increased risk of cancer associated with early life stage exposure limited, but at the exposure levels being regulated by the ASIL they are wholly absent. No studies provide direct evidence of any risk at such exposure levels, much less those that characterize differences between early life stage exposure risk and lifetime average exposure risk. It is at these exposure levels that the convention of linear extrapolation requires the disclaimer that the true value of the risk is unknown and may be as low as zero. The intent of the linear extrapolation method is to use an upper bound estimate of dose response (where actual data may exist from animal studies) drawn down to an extremely low acceptable risk (such as 1 in 1,000,000 where no actual data exist) such that the risk from exposure is undetectable, or possibly zero. Assuming that the mechanisms that produce susceptibility at much higher exposures in animals for early life stage cancer risk also exist at exposures orders of magnitude lower is a policy decision, not a science-based decision, as data at these low exposure levels do not exist to support such a decision. Applying additional adjustments to the cancer slope factor at these exposure levels has not been demonstrated to result in any public health benefit.
Again, the idea that early life stage exposure confers additional risk for the development of cancer remains a hypothesis. In the Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens the authors offer two plausible mechanisms for any observations of increased risk from early life stage exposure (underlined for emphasis):

While the induction of cancer by ionizing radiation and the induction of cancer by chemical mutagens are not identical processes, both involve direct damage to DNA as critical causal steps in the process. In both cases, the impacts of early exposure can be greater than the impacts of later exposures, probably due to some combination of early-life stage susceptibility and the longer periods for observation of effects. (USEPA 2005, p 24)

As noted in the EPA document, most animal studies to evaluate lifetime cancer risk begin after the animals reach sexual maturity, reducing total lifetime exposure to a suspected carcinogen by that amount of time. The authors of the EPA document offer this limited exposure time (i.e., less than a full lifetime due to lack of early stage exposure) as a potential source for an increase in cancer risk from early stage exposure. However, it is important to note that in the traditional risk assessment process for carcinogens, exposures are assumed to be persistent over a 70-year lifetime. This means that even though some exposure period is lost during typical lifetime testing in animals, that exposure is built back into the risk assessment model. Any further adjustment of the model because of this potential mechanism is redundant and not likely to confer additional public health benefit.

In addition, the traditional linear extrapolation method for conducting risk assessment for carcinogens uses an upper bound estimate of the potency of the carcinogen (e.g., the cancer slope factor). This upper bound estimate is purposefully conservative in order to ensure protection for susceptible populations. The result is that risk is always overestimated rather than underestimated with this method, and the degree of overestimation increases as the exposure level decreases. Because of the existing conservatism in the linear extrapolation method used to develop cancer slope factors, modest increases in assumed potency from ADAFs (at higher exposure levels in animal studies) are not likely to confer additional public health benefit at exposures related to the policy-dictated risk management levels of 1 in 100,000 and 1 in 1,000,000, which occur at orders of magnitude lower exposures.

It is also important to consider these proposed changes within the broader context of the conservative assumptions that already exist throughout the ASIL development process. Collectively, using multiple conservative assumptions results in an ASIL that may be far more protective than necessary to meet the risk management goal used to derive it. This phenomenon of greater conservatism embodied by the whole rather than the conservatism of each individual part is referred to as “compounded conservatism.” In the ASIL derivation process, compounded conservatism plays a role both in determination of individual factors of the derivation equations (i.e., in toxicity factors and explicit and implicit exposure elements) and in the equations’ use of multiple factors, most based on upper bound limits and/or conservative assumptions. Given both the inherent conservatism in the linear extrapolation model for evaluating the risk of carcinogens
and the other conservative assumptions used in the ASIL process at large, it is unlikely that the use of ADAFs will confer any additional benefit to public health in the ASIL values.


2.0 There appears to be a technical error in the proposed ASIL value or averaging period for mercury.

It appears that WAC proposes to adjust the mercury ASIL to 0.03 µg/m³, equal to the value selected by the California Office of Environmental Health Hazard Assessment (OEHHA) for chronic inhalation risk. However, WAC does not propose to adjust the averaging period for the mercury ASIL. This presents a mismatch between a concentration representing a chronic (i.e., yearly) exposure and an averaging period more closely related to an acute exposure (i.e., 24 hour). If WAC is to use the OEHHA values for mercury exposure, it would be more correct to either use the OEHHA acute value of 0.6 µg/m³ or to adjust the averaging time to yearly.

3.0 Clarity will be needed for implementation of the mercury ASIL.

In addition to the issue described above, there is an implementation issue with the mercury ASIL. The draft of Table 150 lists “Mercury, CAS # 7439-97-6”; this is the CAS # for elemental mercury (i.e., not oxidized or organic bound). Previous versions of Table 150 have this entry listed as “Mercury, Elemental.” The focus on elemental mercury as a key risk driver is reasonable, and care should be taken that oxidized forms of mercury are not subjected to an ASIL developed for elemental mercury. This could be addressed by changing the draft of Table 150 to read “Mercury, Elemental” or through implementation guidance.