Kirk Hanawalt
President, ENTEK Manufacturing

February 12, 2018

Members of the committee:

Thank you for allowing me to speak with you today. My name is Kirk Hanawalt. I am here on behalf of the ENTEK family of Companies where I have worked for the past 23 years. I am a Chemical Engineer by training and have served ENTEK as the Director of Environmental Compliance and Vice President of Manufacturing. I became President of manufacturing and one of the owners of ENTEK in 2012.

ENTEK was a Lebanon, Oregon start-up business with a handful of employees in 1987. Today ENTEK employs 400 associates at our Lebanon plant. ENTEK also has facilities in Newcastle, England, and Jakarta, Indonesia bringing our global employment to over 700 associates.

ENTEK makes two kinds of separators for batteries. One separator we make is a component inside batteries for automobiles, trucks, golf carts, forklifts, and backup power supplies. We have developed technology used world-wide to enable the start-stop automobiles that assist in reducing carbon emissions. ENTEK exports 95% of these separators outside the borders of Oregon.

ENTEK is the sole remaining US-owned producer of separators for lithium-ion batteries. Lithium batteries are used for personal devices such as cell phones and hybrid and fully electric vehicles. We export all our lithium-ion separators to Asia and Europe.

ENTEK has been a regulated source from day one of our operations in Oregon, initially with a State of Oregon air permit and later with a Title V permit overseen by Oregon DEQ. ENTEK's current Title V permit was issued on February 22, 2011 and expired on November 1, 2015. We have twice submitted our application for renewal, but DEQ's staffing in the air permitting division is under-strength and the department has not been able to process ENTEK's renewal. Fortunately, ENTEK can operate under the expired permit until DEQ writes a new one.

My reason for being here today is to communicate ENTEK's concerns with the draft Cleaner Air Oregon rules.

The Proposed Rules Require Complex and Expensive Analyses by Permittees and DEQ

In 2017, DEQ sent a list of 633 chemicals to all Title V sources in the state and required these sources to submit figures for past actual usage and future estimated usage for each of the 633 chemicals on the list. I supervised a Chemical Engineer who spent 6 full-time months on this effort making a detailed review of over 1,500 Safety Data Sheets (SDS) for every substance ENTEK purchases to see if one of the 633 chemicals on DEQ's list appeared in the Safety Data Sheet. We then had to obtain purchasing and inventory records for each substance where we found one or more of the 633 chemicals.

At the end of this task, most of the chemicals on the DEQ list of 633 we identified in products ENTEK purchases were emitted in quantities less than 1 pound per year and fell below the reporting limit. We submitted a list of about 20 chemicals out of the 633 on the DEQ list where we identified yearly emissions greater than 1 pound per year.

ENTEK is a mature company with a Safety Data Sheet database system, manufacturing resource planning software to track purchases, inventory management systems to track usage, and the resources to hire the technical specialists able to use these systems to determine which of the 633 chemicals on DEQ's list we use and in what quantities we use them.

When I joined ENTEK we lacked the resources to do this work properly and had we been required to make this submission in 1995 or 2005, it would have taken far more hours to do the work and the results would been much lower in quality. As difficult as this task was for ENTEK as an existing source with well-developed systems, I can't imagine how a new source could know a priori which of the 633 chemicals on DEQ's list they will emit and in what quantities.

Two chemicals made up more than 99% of ENTEK's yearly emissions for Cleaner Air Oregon: isopropyl alcohol (IPA) and trichloroethylene (TCE). ENTEK's use of these substances is regulated by our Title V permit and we keep monthly records of our usage of these two chemicals and submit mass balances yearly as part of our Title V permit reporting requirements.

Cleaner Air Oregon requires a source to computer model emissions for any chemical on the DEQ list of 633 that the source emits. In ENTEK's case, DEQ had previously requested that we model our emissions of trichloroethylene and share the results with the agency. We did this in 2017, so we know what modelling involves.

I have a Ph.D. in Chemical Engineering and use computers every day in my work, but the modelling software requires specialized training to use and I haven't been able to figure it out. ENTEK hired CH2MHill and we provided them with hourly estimates of emissions from our plant site for an entire year. CH2MHill combined our hourly emissions data with three years of local weather data and an hourly estimate of atmospheric turbulence made from cloud cover and temperature gradient data from the Salem Airport to generate the model results. We submitted the results to DEQ's air modeler who is, to my knowledge, the only air modeler on the DEQ staff. Ten months and many thousands of dollars in modelling costs later, we have received no feedback from the agency on our modelling.

ENTEK just submitted its Title V reporting for 2017, so we know that we emitted 23% of our permitted volatile organics, which includes trichloroethylene and isopropyl alcohol. ENTEK operates a carbon bed solvent recovery system at 99.7% recovery efficiency to ensure we comply with our Title V permit. Determining compliance with the draft Cleaner Air Oregon rules is very difficult.

DEQ proposes to use EPA's Integrated Risk Information System (IRIS) values to set risk levels. EPA's IRIS values for TCE are not based on observations of inhalation exposure and health outcomes in humans: these are based on animal studies where the animals drink TCE-laced water. These results have then been extrapolated to account for inhalation versus ingestion, rat versus human, and additional margins of safety.

The EPA IRIS value for TCE of 2 micrograms per cubic meter is extremely conservative, probably unreasonably so. In the case of TCE, this would set the risk levels at 26871 times lower than the most conservative workplace exposure levels. Here's an analogy to understand how tiny the figure of 2 micrograms per cubic meter is: 1 penny in $26,871,165.

	TCE Concentration
OSHA, 8-hr TWA	537,423 $\mu g/m^3$
NIOSH, 8-Hr TWA	134,356 $\mu g/m^3$
ACGIH, 8-hr TWA	53,742 $\mu g/m^3$
EPA IRIS	2 $\mu g/m^3$

ACGIH is an organization of industrial hygienists with the most conservative recommendation for workplace exposure to TCE. ACGIH recommends 53,742 micrograms per cubic meter for an 8-hr working day. These values are based on human exposure to TCE that have been published in peer-reviewed scientific literature.

One of the key studies that EPA used to set the IRIS values for TCE is a study in rats can't be replicated despite multiple attempts to do so ***(references attached***). The inability to replicate a study invalidates that study's results according to the scientific method.

I had the opportunity to discuss this point with an Oregon Health Authority (OHA) toxicologist and asked how OHA would evaluate the EPA IRIS values for TCE and other chemicals when issues like this arise. The answer was that he lacks the resources to conduct an independent evaluation and would adopt whatever was in the IRIS database. In the case of TCE, we know that even EPA is evaluating its current recommendations.

I'd like to think that the EPA's work has been and always will be driven solely by science but having observed some abrupt policy shifts over the past 13 months I have come to believe that other considerations play a role in that agency's output.

Having decided on a risk level there is still the question of using the modelling data to show compliance with the proposed Cleaner Air Oregon rules. This requires determining where, when and for how long people are likely to be exposed; the age and health of the people exposed, and an allowance for uncertainty in the modelling results. All of these points will arise over and over again for the sources trying to comply with the draft Cleaner Air Oregon Rules.

As of today, I can't say for certain that ENTEK would be compliant with the Cleaner Air Oregon rules because, even if the hazard index for existing sources in the draft rules is adopted, there are too many additional considerations requiring specific technical expertise to make the determination. As an engineer with advanced training, I find this situation frustrating. As an executive and owner in a company likely to be regulated under Cleaner Air Oregon if these rules become law, I find this deeply unsettling.

Staffing and Budget

DEQ is under-staffed and behind in its current workload in air permitting under Title V. Cleaner Air Oregon will require additional resources in DEQ and OHA for permit writing, air modelling, toxicology, data science, and administration. Hiring and training the large number of technical and administrative staff would be difficult under the best of circumstances, let alone in the current full-employment environment.

Sources falling under the Cleaner Air Oregon rules will be spending hundreds of thousands of dollars for legal and technical services to demonstrate compliance with the new rules and will be assessed higher fees to pay for it all. This will be a windfall for law firms and consultancies based in Portland, Salem and Eugene, but will take money out of the rural communities where many of the businesses subject to the Cleaner Air Oregon rules are located.

Policymaking Should be Done by the Legislature not the Agencies

ENTEK believes that rulemaking with such far-reaching impacts on residents, communities, and businesses in the State of Oregon should be made by the Oregon legislature, not directly by the agencies. DEQ is under-staffed and behind in its current workload; it lacks the resources and ability to evaluate the impacts of its rule-making on all stakeholders in the State of Oregon.

More importantly, having DEQ write the rules it administers politicizes the agency. This will undermine the agency's effectiveness as an environmental regulator and will make its relationships with regulated sources more contentious and less productive.

Give Title V a Chance

The circumstances that led to the drive for Cleaner Air Oregon had something to do with lead and one or more regulated sources. Lead is one of the hazardous air pollutants named and regulated under Title V. ENTEK strongly believes that DEQ should request and the legislature should make available the necessary resources to fully staff and oversee the existing air permitting process under Title V before launching a new and complex program that will require even more resources that DEQ does not have and is unlikely to acquire.

Summary

I'd like to say that ENTEK has valued the work and input of the DEQ permit writers who have overseen our company's operations. Mr. Fritz Skirvin, Mr. Jim Boylan, and Ms. Karen White-Fallon have been consummate professionals and have made ENTEK a better company. We are proud of our environmental record and look forward to many more years of operations in the State of Oregon.

ENTEK's concern with the draft Cleaner Air Oregon rules are that DEQ lacks the resources to manage the program effectively; that demonstrating compliance with the draft rules will be very expensive and time consuming; the real public health benefits of the program will be minimal, especially in low population density rural areas; the fees required to support the program will be high; and having DEQ write and administer the rules politicizes the agency to the detriment of its ability to serve the interests of all stakeholders.

Thank you for the opportunity to express ENTEK's concerns.

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Letter to the editor

Review of TCE cardiac defects data by Makris et al. is not systematic

To the Editor

We have read with dismay the recent article by Makris and colleagues [1]. Their contribution provides little new insight into the controversy surrounding the potential for trichloroethylene (TCE) to cause developmental effects at environmentally relevant exposures. After describing the strengths and weaknesses of the available *in-vitro*, animal, and human studies, the authors fail to factor this information into their weight-of-evidence (WOE) determination – a critical part of any systematic approach. Additionally, laboratory studies that followed guidelines for developmental toxicity safety studies (*e.g.*, [2], [3]) are not incorporated into the WOE determination, while those with well documented issues (*e.g.*, [4]) are.

Makris et al. reiterate the suggestion that "differences in route of administration" explain the positive results in the Johnson et al. drinking water studies when compared to negative findings reported in the oral gavage study by Fisher et al. [2] and the inhalation study by Carney et al. [3]. However, in a contradictory manner, they offer the results of an epidemiology study based on estimated *inhalational* exposure to TCE vapors by residents of Endicott, NY [5] as "clear evidence" of an association. Makris et al. also fail to adjust their findings for the significant risk of bias associated with the analysis in the Endicott study [6]. Concerns about model overfitting and the potential that this could bias the result toward a positive finding, while not addressed by Forand et al., are discussed in an earlier analysis of the same data [7].

The lack of a predominant type of cardiac defect in the Johnson et al. rat studies and human epidemiology studies highlighted in Makris et al. makes the exploration of mechanistic concepts by the authors largely academic. Their discussion of adverse outcome pathways (AOPs) focuses on results reported in avian models, which differ greatly in mode and duration of exposure compared to mammals. While suggesting that similarities may exist in valvuloseptal development between species, the authors do not attempt to explain how these AOPs contribute to the diversity of cardiac defects reported in the animal studies of Johnson et al. and in several human populations studied involving putative TCE exposures.

Perhaps most concerning is the authors' statement that "[d]esigning and conducting an exact replica of the Johnson et al. study might be very difficult, if not impossible." Reproducibility of study results is one of the central tenets of the Bradford Hill criteria that form the basis of EPA's evidence-integration framework, not to mention being a pillar of scientific enquiry. A more fruitful approach to the reproducibility question would be to consider why the results of Johnson et al. are so different from those of two EPA Guideline/GLP-compliant studies. Instead, the authors dismiss the Guideline studies because they "do not replicate all aspects" of Johnson et al. rather than discussing how the differences in study design and execution might explain the drastically different outcomes.

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Review and Recommendations for TCE Short-Term Action Levels in Indoor Air

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Reviewed by Jenny Phillips, DABT, TRC Companies, Inc.

Confusion and misinterpretation is the state of the 2014 regulatory environment as it pertains to short-term trichloroethylene (TCE) action levels for indoor air. The issue starts with comprehension of the United States Environmental Protection Agency's (EPA's) TCE toxicity profile, released in September 2011 (EPA, 2011), that lowered the non-cancer inhalation toxicity value (reference concentration [RfC]) from 10 micrograms per cubic meter (ug/m^3) to 2 ug/m^3, which is equivalent to a reduction from 1.9 parts per billion by volume (ppbv) to 0.4 ppbv. This decrease is equivalent to a 5-fold increase in noncancer risk.

The basis of the 2 ug/m^3 (0.4 ppbv) is a controversial study (Johnson et al., 2003), where fetal heart malformations were observed during the 21-day gestational period of the Sprague-Dawley rat based on drinking water (oral) exposure. The concern of this study is that the critical effect occurred from *in utero* exposure (Johnson et al, 2003), which could translate to human cardiac development.

There are several weaknesses with the Johnson 2003 study. Most notably, the fetal heart malformation results could not be replicated in other studies, including one study where TCE was administered via inhalation (Carney et al., 2006), and another study that Johnson collaborated on where TCE was administered via oral dosing (Fisher et al., 2001).

The inability of either study to replicate fetal heart malformation effects, specifically the Carney et al, 2006 inhalation study, introduces significant uncertainty with the Johnson et al., 2003 findings. Study results varied widely, and were not uniformly distributed in the Johnson et al., 2003 study, which infers low confidence in the study itself (Alliance for Risk Assessment, 2013). The questionable toxicity study lends to low confidence in determination of short-term action levels for use in industrial settings. This paper provides a review of a wider literature base on the topic and suggests alternate short-term action levels protective of human health, until such time that EPA Headquarters finalizes their assessment on this topic.

1.0 LITERATURE REVIEW OF TCE TOXICITY

1.1 EPA Toxicity Profile (September 2011)

As mentioned above, EPA's toxicity profile was released in September 2011, with the Johnson et al., 2003 toxicity study as the cornerstone of the new inhalation RfC. EPA incorporated a substantial factor of safety in the derivation of the RfC from the results of Johnson et al. 2003 study, resulting in a very conservative value. The derivation of the inhalation RfC is based on an oral-to-inhalation extrapolated, 99th percentile Human Equivalent Concentration (HEC_{99}) to the rat internal Benchmark Dose-low (BMDL) associated with a 1% extra risk to each rat pup (BMDL01) (Integrated Risk Information System [IRIS], 2014). This means that the oral dose administered to the rats during the study was extrapolated to an absorbed or internal dose using a physiologically based pharmacokinetic (PBPK) model. The modeled internal dose was then used for input into a second model to determine the 95% lower confidence bound

of a dose protective of 99% of the dosed population (i.e. BMDL01). The rat dose was then converted to the HEC_{99} using a third model to account for exposure to humans (Alliance for Risk Assessment, 2013). This $HEC_{99,BMDL01}$ concentration of 21 ug/m^3 (4 ppbv) protective of lifetime continuous exposure, is then divided by an additional uncertainty factor of 10, to arrive at EPA's inhalation RfC (2 ug/m^3 or 0.4 ppbv) (IRIS, 2014).

The toxicity profile for TCE is presented on EPA's IRIS website (IRIS, 2014) and states that "the RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a *continuous inhalation exposure* to the human population (including sensitive subgroups) that is likely to be without an appreciable *risk of deleterious effects during a lifetime*". Therefore, the intent of the inhalation RfC is to protect against lifetime risk from continuous inhalation exposure, which is not representative of short-term exposure or conditions requiring immediate action (e.g., removal of indoor workers).

1.2 ATSDR Studies

The mission statement of the United States Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR) is to "serve the public through responsive public health actions to promote healthy and safe environments and prevent harmful exposures" (ATSDR, 2009). As noted on their website, http://www.atsdr.cdc.gov/about/mission_vision_goals.html, ATSDR's goals include:

- Protecting the public from environmental hazards and toxic exposures, as well as promoting healthy environments;
- Advancing the science of environmental public health, through:
    - Collection, analysis, and data summarization on environmental exposures and health; and
    - Conducting research to identify associations between environmental exposures and health risks.
- Educating communities, partners and policy makers about environmental health risk and protective measures; and
- Providing unique scientific and technical expertise to advance public health science and practice, through:
    - Collaborative laboratory research that yields:
        - Critical population-level data;
        - A greater understanding of adverse health outcomes; and
        - Information to evaluate public health interventions.

Based on the mission and goals of the ATSDR, evaluating short-term exposure and health effects of TCE is within their authority. In 2013, ATSDR released a Health Consultation study regarding the Millsboro TCE Site, located in Millsboro, Delaware. At this site, drinking water was contaminated with TCE from October 2004 for approximately one year, at which point, a water treatment filter was installed to remove the TCE from the drinking water. The purpose of the ATSDR Health Consultation was to determine whether or not TCE exposure during that timeframe was a human health concern (ATSDR, 2013a).

In order to evaluate the inhalation exposure pathway, ATSDR assumed that residents would be exposed to TCE in groundwater that volatized during showering or other household uses such as dishwashing or laundry, during a time period of no more than one year. ATSDR's Health Consultation indicated that "*there is no suitable comparison values for TCE that represent the (one-year) timeframe in which the Millsboro residents were exposed...EPA's reference dose and reference concentration are both intended for comparison to chronic or longer duration exposure scenarios. ATSDR used the Human Equivalent Concentration (HEC_{99}) for inhalation during showering. The HEC is the concentration derived from animal studies that takes into account the physiologic and pharmacokinetic differences in animal models*

and man." Note that this HEC_{99} of 21 ug/m^3 (4 ppbv) is the same value that was derived from the Johnson et al., 2003 study (despite the uncertainty surrounding the study) and was used to compare against calculated 24-hour average indoor air concentrations of TCE to determine whether health effects were a concern (ATSDR, 2013a). This study evaluated residential exposure and determined a maximum allowable 24-hour (continuous) indoor air concentration of 21 ug/m^3 (4 ppbv) for TCE. Therefore, using the same concentration of TCE (21 ug/m^3 or 4 ppbv) is protective of lesser-exposed receptors, such as an indoor worker, for an intermediate period of time (e.g., 8 –hour workdays, five days per week for over one year).

In 2013, ATSDR also released a Public Health Assessment regarding the Pohatcong Valley Groundwater Contamination Superfund Site, located in Warren County, New Jersey. At this site, the public was exposed to drinking water contaminated with TCE from 1972 to 1981, when treatment systems were put into place (ATSDR, 2013b). Similar to the Millsboro Site, the Pohatcong Site assumed that residents would be exposed to TCE in groundwater that volatilized during showering. Therefore, ATSDR calculated time-weighted average (TWA) indoor air concentrations based on showering activities and compared them to the 21 ug/m^3 (4 ppbv) HEC_{99} (the Lowest Observed Adverse Effect Level, or LOAEL) of the Johnson et al, 2003 study (ATSDR, 2013b), despite the uncertainty surrounding this study. Similar to the Millsboro study, this risk assessment indicates that 21 ug/m^3 (4 ppbv) is a reasonable, allowable TWA indoor air concentration for residents over a period of approximately 10 years. Therefore, a TCE concentration of 21 ug/m^3 (4 ppbv) would also be protective of lesser-exposed receptors, such as an indoor worker.

2.0 REGULATORY REVIEW

Given the uncertainty surrounding the Johnson et al., 2003 study and EPA's route extrapolation to arrive at an inhalation RfC of 2 ug/m^3 (0.4 ppbv) for chronic exposure, this paper explores alternate short-term action levels protective of intermediate exposure periods similar to the ATSDR Millsboro study, as well as conditions requiring immediate action/removal. The remainder of this section presents a regulatory review of how risk-based indoor air levels for TCE have been determined.

2.1 Risk-Based Remediation Goal (RBRG) vs. Removal Action Level (RAL)

The terms "Risk-Based Remediation Goal" (RBRG) and "Removal Action Level" (RAL) are used frequently among regulatory agencies when identifying concentrations protective of indoor air exposure. The RBRG is protective of long-term, chronic indoor air exposure, while a RAL is focused on short-term conditions requiring immediate action. The noncancer Hazard Quotient (HQ), which is a ratio of the exposure level to a screening value, is lower when calculating an RBRG (which assumes an HQ of 1.0 per EPA's *Risk Assessment Guidance for Superfund, Volume I. Human Health Evaluation Manual, Part B: Development of Risk-Based Preliminary Remediation Goals* (EPA, 1991)) versus calculating an RAL (which assumes an HQ of 3.0, per EPA's *Revised Superfund Removal Action Levels memorandum* (EPA, 2008). The intention of the 3-fold increase in HQ for RAL development is to allow a cushion between long-term health protectiveness and short-term immediate action. Therefore, for purposes of setting a short-term immediate action level for TCE, an HQ of 3.0 is appropriate as recommended by EPA (EPA, 2008). Screening levels based on ATSDR's Millsboro and Pohatcong Valley findings, as well as published EPA values, are summarized in Table 1.

TABLE 1: Summary of Risk-Based TCE Indoor Air Levels

Source	Screening Levels and RALs	Basis for Concentration*
Intermediate Residential Exposure (ATSDR, 2013a; 2013b)	TCE = 21 ug/m^3 (4 ppbv)	Based on HEC_{99}, assumed protective of intermediate (1 year) residential exposure
EPA Indoor Worker Regional Screening Level (RSL) (EPA, 2014a)	TCE = 8.8 ug/m^3 (1.6 ppbv)	Based on long-term worker exposure (8-hour workday, 250 days per year for 25 years), inhalation RfC (2 ug/m^3 or 0.4 ppbv), HQ = 1.0
EPA Region 9 RAL (EPA, 2012a)	TCE = 15 ug/m^3 (2.8 ppbv)	Based on acute (short-term) 10-hr workday, inhalation RfC (2 ug/m^3 or 0.4 ppbv), HQ = 3.0
EPA Region 10 Short-Term Concentration (EPA, 2012b)	TCE = 8.4 ug/m^3 (1.6 ppbv)	Based on 21-day exposure period, inhalation RfC (2 ug/m^3 or 0.4 ppbv), HQ = 1.0**
EPA Region 9 Accelerated Response Action Level (EPA, 2014b)	TCE = 8 ug/m^3 (1.5 ppbv) (8-hour workday); TCE = 7 ug/m^3 (1.3 ppbv) (10-hour workday)	Based on short-term commercial/industrial exposure, inhalation RfC (2 ug/m^3 or 0.4 ppbv), HQ = 1.0**
EPA Region 9 Urgent Response Action Level (EPA, 2014b)	TCE = 24 ug/m^3 (4.5 ppbv) (8-hour workday); TCE = 21 ug/m^3 (4 ppbv) (10-hour workday)	Based on short-term commercial/industrial exposure, inhalation RfC (2 ug/m^3 or 0.4 ppbv), HQ = 3.0

* Both the HEC_{99} and RfC used to determine screening levels and RALs were calculated using the Johnson et al., 2003 study. However, as described above, these inhalation-based values are extrapolated from an oral exposure study. Furthermore, the Johnson study results varied widely, indicating a high degree of uncertainty. Finally, no other study has been able to replicate the toxicological, critical effects observed in the Johnson study.

** HQ of 1.0 is not consistent with EPA (2008) HQ of 3.0 for short-term exposure.

3.0 RECOMMENDATIONS FOR SHORT-TERM ACTION LEVELS IN INDOOR AIR

Based on a review of available indoor air levels protective of an indoor worker, it is apparent that the Johnson et al., 2003 study and the HEC_{99} value of 21 ug/m^3 (4 ppbv) are used as either the ultimate goal (in the case of the two ATSDR studies) or the basis of the inhalation RfC (2 ug/m^3 or 0.4 ppbv), despite the uncertainty surrounding the Johnson study. The variability in the indoor air levels presented in Table 1 is due to different HQ values. Using an HQ of 3.0 is consistent with approved methodologies per EPA (2008) guidance when setting RALs, similar to the methodology EPA Region 9 uses to calculate their 2014 Urgent Response Action Levels and 2012 Remedial Action Level for TCE. Note, EPA Region 9's July 2014 Accelerated Response Action Level and EPA Region 10's 2012 Short-Term Concentration are both based on an HQ of 1.0, which is not consistent with the standard approach for setting a RAL. The use of an HQ = 1.0 is considered appropriate when evaluating long-term, chronic exposure; thus, the EPA Indoor Worker RSL is correctly derived using an HQ = 1.0.

Based on this review, two short-term TCE action levels protective of an indoor worker are recommended:

- ATSDR's use of the HEC_{99} (21 ug/m^3 or 4 ppbv) for intermediate residential exposure, which would also be protective of short-term worker exposure for intermediate periods (e.g., 8 hour workdays, five days per week for over one year) (ATSDR, 2013a: 2013b):

- Adjusting the HQ from 1.0 to 3.0 for EPA's indoor worker RSL of 8.8 ug/m^3 (1.6 ppbv) to calculate an indoor worker RAL of 26.4 ug/m^3 (4.9 ppbv) (8.8 ug/m^3 x 3.0, or 1.6 ppbv x 3.0) representative of short-term conditions requiring immediate action, consistent with EPA Region 9 Urgent Response Action Level (EPA, 2014b).

Both of these recommended values (21 and 26.4 ug/m^3, or 4 to 4.9 ppbv) are similar, which lends confidence in using this concentration range for short-term indoor air action levels for TCE, instead of relying on risk-based concentrations protective of chronic, long-term inhalation exposure, which are not representative of intermediate periods up to one year or for conditions requiring immediate action/removal. Regardless of which concentration is chosen, the lack of inhalation studies producing fetal heart malformations should be revisited by EPA prior to setting a policy decision on indoor air action levels for TCE.

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