Response to Comments

Draft MCL Determination for Trichloroethylene (Oct 2013)
Comments Received from Peer Review Panel: December 6, 2013

Response Document Prepared: January 7, 2014
By
The Connecticut Department of Public Health
Occupational and Environmental Health Assessment

A peer review panel was convened in October 2013 to review the Draft MCL Determination for TCE document prepared by the Connecticut Department of Public Health dated October 2013. The panel had a conference call on November 18th and then submitted formal comments by December 6th 2013. These comments have been incorporated into the document or otherwise addressed as detailed below. The members of the peer review panel were as follows:

Michael Hutcheson, Massachusetts Dept of Environmental Protection
Mark Smith, Massachusetts Dept of Environmental Protection
Kate Sande, Minnesota Dept of Health
Gloria Post, New Jersey Dept of Environmental Protection
John Budroe, California OEHHA
Pam Wadman, Maine Dept of Health

Comments from Michael Hutcheson, MassDEP

TO: Gary Ginsberg, CT DPH
FROM: Michael Hucheson, MassDEP
DATE: December 2, 2013
RE: Review Comments on Draft Maximum Contaminant Level (MCL) Determination for Trichloroethylene by Connecticut Department of Public Health. October 2013

GENERAL COMMENTS

The overall document is technically well done. The points made are well supported with technical evaluations. A nice balance has been struck where lengthy reviews by other groups were summarized to cover certain topics such as the overall toxicology of TCE.

I was glad to see incorporation of the inhalation and dermal exposures associated with use of TCE-containing tap water in the home. The case was well made and the relative levels of exposure by the various routes are in agreement with my appreciation of the literature.

SPECIFIC COMMENTS

1. It would help to have more background and introduction to how the proposed MCL of 1 ug TCE/L was chosen and derived. Right now at the bottom of p. 2 there is reference to the fact that it was a follow-up to an Action Level for private wells. This expanded information should be towards the front of the document likely in the "Background" section. Questions that come to my mind about that value that I would hope to see some information on are: was it derived using standard drinking water guidance methods; were age-dependent adjustment factors used for cancer potency estimates and for early life stage exposure parameters; was it based on just ingestion, or were non-ingestion uses factored in and how?

DPH Response: the new Background section provides additional perspective on the relationship between the Action Level and the MCL and the new Executive Summary outlines the basis for the MCL; since the MCL determination updates and supercedes the AL determination, there is no reason in the Background section to provide a detailed description of the Action Level.

2. Unit conversion factors are missing from a number of equations where units are going between ug and mg or visa versa. Would make things more complete for the less technical reader trying to follow the equations.

DPH Response: DPH has gone through all equations in the document to make them clearer and more uniform with terms better described and units made more consistent.

3. I found a few of the tables and equations confusing in terms of following how or where various parameters in the tables were derived. For the equations, you might first show

the expression with the variables defined and then have the numerical values under them, or show the equation with variable abbreviations, followed by the expression with the values being used followed by a listing of the variable acronyms being defined. To help set off the equations and associated material from the text, I suggest indenting that material and perhaps showing the equations in italics or offsetting them somehow. For the tables, I particularly had a hard time following Table 4 and the material below it. Column head for the 4th column would seem to be just "Dose" since it is listing the dose for that exposure route. I found it confusing to have the table and then the second analysis in the 5 lines together under the table using the information from the table to derive a total exposure. After further reading, I think I see that the first 3 of these lines are more calculations following on from the table, whereas the last 2 lines are text conclusions from the information presented in the table and the 3 lines above. I was getting confused thinking that the statements in the last 2 lines were assumptions built into the table and lines above, not conclusions. I suggest separating the content of these 2 lines from the material that precedes it. I think that it would be clearer if you renamed and restructured the table to show TOTAL household TCE exposure by adding one line in the table for the ingestion exposure. You can then show sums across routes to show Total Daily Exposure. Footnotes can be used to explain assumptions.

DPH Response: Figures and Tables have been redone for consistency and transparency. Table 4 has been better explained in text and modified to separate out exposure routes with the derivation of dermal fraction now described in text rather than table footnote.

4. Summary of California's cancer potency estimates on p. 11-12. Is there a benefit to or need to summarize the basis of California's older CPFs when their current value is the EPA value? Probably half this section is devoted to describing the basis of their older values.

DPH Response: the California potency descriptions and differences relative to USEPA remain as the CTDPH process is to make an independent evaluation of non-IRIS values especially where they are substantially different from IRIS values, which is the case with the California values. A statement has been added to this section from John Budroe's comments stating the California perspective that while the Proposition 65 program has formally adopted the IRIS slope factor that other programs have yet to do so (opportunity has not yet arisen for the drinking water section). This statement makes it less counterintuitive to include the details of California derivations that they appear to be moving away from.

Shorter comments are shown in the text of the draft document provided to us for review.

DPH Response: Comments noted on the document itself in track change mode have been addressed.

Comments from Mark Smith, MassDEP

TO: Gary Ginsberg, CT DPH
FROM: C. Mark Smith, MassDEP
DATE: December 6, 2013
RE: Review Comments on Draft Maximum Contaminant Level (MCL) Determination for Trichloroethylene by the Connecticut Department of Public Health. October 2013

Summary

This is an excellent assessment that provides a compelling justification for lowering the CT MCL for TCE. Specific comments, including a few suggestions are noted below.

Specific Comments/Suggestions

1. An executive summary should be added. This should briefly note something like the following: Based on the most up-to-date toxicity information from US EPA, the current MCL is associated with cancer risks that could exceed 1E-06 using standard exposure assumptions for adults and considering only direct water ingestion. The MCL associated with a 10E-6 risk level using the standard exposure assumptions and the most up-to-date US EPA cancer value would be 0.67 ug/L. Consideration of inhalation and dermal exposures, as well as the potential for increased cancer vulnerability of children, which is appropriate in light of TCE's chemical and toxicological characteristics, would increase the estimated risks and lead to a target drinking water level well below 0.67 ug/L. In light of these risk estimates and feasibility considerations a revision of the CT MCL from 5 to 1 ug/L is recommended. This value will substantially address both cancer and non cancer risks attributable to TCE in drinking water and has been demonstrated to be both feasible and practical based on the fact that NJ has successfully implemented an MCL of 1 ug/L for many years."

DPH Response: An executive summary along the lines described above has been added to the document.

2. The documents consideration of inhalation and dermal exposures attributable to use of TCE contaminated water in the home is appropriate for this contaminant. The estimate for dermal exposure appears less well supported compared to that for inhalation.

DPH Response: Additional data and citations have been used to support the dermal uptake fraction.

3. Consideration of childhood vulnerability and risk is also appropriate. Although the case for such an adjustment is strongest for the kidney cancer endpoint, a case can be made for also considering this adjustment for the liver cancer and non-hodgkins lymphoma endpoints, as a mutagenic MOA is possible for these as well. A range of values should be presented with the preferred value noted.

DPH Response: the implications of having the children's ADAFs apply to cancer endpoints in addition to renal cancer is now shown in Table 5 and associated text.

4. To shorten the document the material regarding the CA cancer potency derivations could be truncated to just note they have accepted the latest US EPA values.

DPH Response: see answer to Michael Hutcheson comment #4 above.

5. The discussion of cardiac developmental risks could be strengthened. For example on page 15 the following additions/clarifications are recommended: 1) It should be noted that US EPA concluded "….that TCE exposure poses a potential hazard for congenital malformations, including cardiac defects, in offspring ."; 2) US EPA's determination that the epidemiology studies, while not conclusive, were relatively consistent with respect to this endpoint should be noted ("The epidemiological studies, while individually limited, as a whole show relatively consistent elevations, some of which were statistically significant."); 3) Regarding the animal studies, it should be more clearly noted that US EPA concluded that results from the key rat studies were appropriate for use in assessing ingestion and inhalation developmental risks. 4) A statement that CT DPH concurs with these determinations by US EPA would strengthen the subsequent assessments, including the benefits assessment.

DPH Response: the recommended changes have been made to this section.

6. The basis of the recommended MCL revision to 1 ug/L should be more clearly and prominently explained. The risk estimates derived in the assessment could support MCL values below 1 ug/L if based on risk considerations alone. The selection of 1 ug/L seems to be based more on the fact that it is feasible and implementable as evidenced by its successful use by NJ, rather than because of the uncertainty in the analysis (a statement that could be deleted).

DPH Response: Additional statements have been added at several points of the MCL derivation section to make it clear that the MCL is not completely risk-based but is a policy decision to use precedent and adopt a value used elsewhere.

7. On page 27 regarding the cardiac risks, although the exposure duration of concern during pregnancy is not precisely known, it is likely to be more on the order of days to weeks rather than weeks to months.

DPH Response: this wording has been changed as suggested.

8. An additional approach to the benefits assessment for the developmental endpoint that may be worth considering would be to use US EPA's PBPK derived HED_{99}/HEC_{99} as a conservative (e.g. it doesn't account for pharmacodynamic uncertainty) estimate of risk i.e. 21 ug/m^3 presents at least a 1% excess risk in the 1% of the population that are "high metabolizers", or at least a 10^{-4} excess risk in the overall population. Based on a quick back of the envelope calculation, I think this would yield a benefit attributable to cases

prevented that is about equal to the estimated treatment costs, which would be a very conservative "floor" estimate for the benefits.

DPH Response: This is an interesting approach. However, it involves an extrapolation from rats to humans, with the point of departure for cardiac defects being a 1% increase in rats. This increase in rats relative to the rat background of cardiac malformation is less informative for human risk assessment than the epidemiology data from Endicott NY; those data provide an odds ratio that can be converted to population incidence and the number of extra cases due to TCE that is directly useful in benefit assessment. The rat data are supportive of the human epidemiology in terms of cardiac defects as an important endpoint from TCE exposure in humans.

9. I think the $100,000 CDC cost figure is only for health care costs and does not account for other costs associated with CHD (e.g. cost of premature death; etc.), so is likely to be a significant underestimate. Suggest that this be noted.

DPH Response: this possibility has been noted in the text.

Comments from Pamela Wadman, Maine DHHS

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December 4, 2013

Maine CDC appreciated the opportunity to review the Connecticut Department of Public Health Draft Maximum Contaminant Level (MCL) Determination for TCE dated October 2013. The document provides a logical approach and support for the proposed MCL and will be a useful asset for the risk assessment community. We respectfully submit the following comments, which are the opinion of the primary reviewer, Pam Wadman, and do not necessarily represent the position of the Maine CDC.

General Comments

Although we realize this document has not been finalized to the point of formatting, to make it reader friendly, the following presentation issues should be addressed.

- The document would benefit from a table of contents.

DPH Response: this has been added as suggested.

- The document would benefit from a page with definition of acronyms and units, I believe the most widely recognized presentation of units for RfD is mg/kg-day, and CSF is (mg/kd-day)-1

DPH Response: a glossary of abbreviations has been added as suggested.

- Is it CTDPH, CT DPH or DPH (each used multiple times)? Is it OEHHA or Cal OEHHA?

DPH Response: these abbreviations have been made consistent throughout the text.

- If possible, tables should not be split across pages, if they are the column headers should be repeated and the table labeled "(continued)".

DPH Response: this has been addressed in final formatting so that tables are not split across pages.

- Equations should be numbered and presented first as terms with a "Where:" that defines the terms with units, followed by the actual calculation with numbers and units.

DPH Response: Equations have been reformatted as suggested.

- Equations should be presented for every number that is derived.

DPH Response: additional equations have been presented to better describe key quantitative aspects of the assessment.

- Terms used for derived numbers should be consistent throughout document e.g. Table 4 vs. .Table 7.
- Please eliminate the comma between first author and "et. al"(occurs 5 times).

Specific comments

DPH Response: Specific editorial comments are addressed in the text as called out below. Two comments that triggered more substantiative changes are numbers 16 and 25 below. See DPH responses to these comments.

1. Page number 2, 4th paragraph, last sentence typo. Suggest changing "CTDEEP" to "CTDEP"
2. Page number 4, Table 1. Suggest changing "(fem)" to "(female)"
3. Page number 10, 1st paragraph 1st sentence. Suggest changing "which" to "that"
4. Page number 10, 2nd paragraph 1st sentence. Suggest changing "file" to "profile"
5. Page number 11, 1st paragraph 3rd sentence. Suggest changing "adopts" to "adopted"
6. Page number 13, 1st paragraph 1st sentence. Suggest changing "dosimetry" to "dosiometric"
7. Page number 14, 3rd paragraph last sentence. Suggest changing "PPARalpha" to "peroxisome proliferator-activated receptor alpha".
8. Page number 14, 3rd paragraph last sentence. A sentence about what the PPAR-alpha up-regulation in rat liver means to human health might be useful here.
9. Page number 16, Table 3. Please add the abbreviations used to the table footnotes.
10. Page number 16, Table 3. "LOAEL = 1.4 mg/L" - this appears to be a serum concentration, not a dose.
11. Page number 18, Table 3. The Intra-human UF should be TK (toxicokinetic).
12. Page number 18, Equation 1. Please see general comments about presentation of equations and calculations.
13. Page number 19, 4th paragraph 3rd sentence. The term "small microenvvironment" is redundant.
14. Page number 19, 2nd paragraph 3rd sentence. Suggest changing "Jo, et al. (1990), " to "Jo et al., (1990a)"
15. Page number 19, 2nd paragraph 4th sentence. Please present or describe how the 6.3 mid-range estimate was derived. (The median of the ratios of shower air to water formaldehyde concentrations as presented in Jo et al. 1990a.)
16. Page number 19, 2nd paragraph sentence. Why choose chloroform when there is TCE data? Why not cite the works of McKone et al.?

DPH Response: CT DPH conducted a search for studies by Dr. McKone on TCE bathing and showering exposure. A paper by McKone and Knezovich (1991) was the only one found. Rather

than show actual shower stall air concentrations, it provided data on transfer efficiency of TCE from shower water to air. Their result is consistent with what had already been cited in the DPH document for chloroform further supporting our use of the more extensive chloroform database. The text now provides this information.

17. Page number 20, Use of consistant terminology would simplify for the reader, for example "Whole house indoor air" is also termed "Household water uses". Please consider separating equations with headings, and presenting equations, followed by input parameters with units, and the actual calculation resulting in 1.35 ug/m3.
18. Page number 20, Table 4. This table is confusing, the source of the numbers is not clear. It would be helpful to use terms consistent with page 19, "Whole house indoor air" and "Household water use" should be the same. Where is the shower dermal calculation presented? For the showering dermal entry, "Dermal 48% of total uptake", where is total uptake presented? Total uptake in shower? How does dermal uptake get converted to an inhaled dose?
19. Page number 20, Table 4. "Total Daily Exposure" is termed "Household TWA Air Conc" on page 27, suggest choosing one term for each parameter.
20. Page number 20, Table 4. The peak concentration in air is calculated from the water concentration air/water ratio from Jo 1990a, 6.3 , for a water concentration of 5 ug/L the air concentration is 31.5 ug/m3. Please show the calculation.
21. Page number 20, Table 4. Please show the calculation of inhaled dose.
22. Page number , 1st paragraph 1st sentence. "The inhalation contribution is calculated as follows" is confusing because it is directly followed by the ingestion contribution calculation. Suggest rewording to something like "The relative contributions of inhalation and oral exposures are presented below."
23. Page number 20, 2nd paragraph. Instead of "Inhalation/dermal:" the terms should be consistent with the label in Table 4 "Total Daily Exposure to air 24hr TWA", or the terminology used on page 27.
24. Page number 20, 3rd paragraph. This paragraph is not clear. Where do these water equivalents come from? What do they mean?
25. Page number 20, 4th paragraph. As we discussed via email, this risk calculation is over simplified. The ILCR of 2.9e-5 was calculated using an oral CSF, but much of the exposure is inhalation. According to my calculations, using the ADAF adjusted URi (presented in IRIS, 4.1E-6 (ug/m3)-1)), the total 70 year ADAF adjusted oral and inhalation risk will be 1.6E-5. Suggest

striking the latter part of this sentence and thus emphasizing the increased dose without mentioning the risk (for simplification).

DPH Response: This comment is accurate in that the initial draft used the oral cancer slope to calculate the risk from oral dermal and inhalation. Given that the inhalation slope on IRIS is lower than the oral slope, separating the pathway exposures in risk calculations does make a difference (less risk) than just using the oral slope. The new draft does contain this separation (oral and dermal exposures multiplied by the oral slope, inhalation exposure multiplied by the inhalation slope) and this yields the cumulative cancer risk cited above in PW's comment. This change in cumulative cancer risk is carried through all subsequent sections and while it lowers the risk 43% this does not change the overall conclusions or the outcome of the MCL derivation.

26. Page number 20, 4th paragraph. It would be helpful to define the 4 fold increase as the "inhalation exposure factor" referred to on page 26.
27. Page number 20, 4th paragraph, 1st sentence. To the "dose associated with current MCL 4 fold to 5.8e-4" please add units (mg/kd-day).
28. Page number 21, 2nd paragraph 3rd sentence. The "between 3 and 15 years" should be "between 3 and 16 years", USEPA 2005, page 33,"For exposures between 2 and <16 years of age (i.e., spanning a 14-year time interval from a child's second birthday up until their sixteenth birthday), a 3-fold adjustment." Likewise for the calculations, as per EPA 2005, children's additional risk should be calculated for the interval of 3-16 years (14 years). Adult risk is calculated for an exposure duration of 54 years.
29. Page number 21, 3rd paragraph. Suggest introducing the calculation with a header "Calculation of increased lifetime cancer risk considering mutagenic mechanism of action."
30. Page number 22, Table 5. Please identify "IRIS slope with child factor" (kidney) with a footnote "IRIS slope x increased total risk across life stages (page 20)"
31. Page number 22, 2nd paragraph 1st sentence. For clarity, suggest changing "Application of the slope modified for children" to "Application of the combined IRIS oral slope factor modified for children". Using this approach, shouldn't the child specific inhalation unit risk be calculated as well? The 27 % increase in the combined oral slope factor cannot be applied to the inhalation portion of risk presented on page 20. For full lifetime exposure to a constant exposure level, the ADAF-adjusted inhalation unit risk estimate for TCE is 4.8 × 10-6 per µg/m3 (U.S. EPA (2011). Also, it would be helpful to present and define the "children's vulnerability factor" of 1.27, mentioned on page 26, in this paragraph.

32. Page number 22, 4th paragraph. Suggest changing "Exposure Dose" to "Ingestion Exposure Dose"
33. Page number 25, 3rd paragraph 5th sentence. Suggest changing "significant" to "considerable".
34. Page number 25, paragraph 5 (equation). Does the average daily dose (1 mg/kg-day) belong in this equation? I believe the "1000 ug/kg/d/mg-kg-d" should be termed a conversion factor (CF) and the units should be ug/mg. Please present the formula for the Risk-based target = (TR x BW x CF)/ (CPF x IR).
35. Page number 26, 2nd paragraph. The cancer risk-based target is calculated with the oral CPF, we can't mix inhalation and ingestion exposures into one risk calculation using an oral CSF. This argument needs to be reworked. In addition, it will be much clearer for the reader, if consistent terminology is used for terms that are present multiple times in the document. When stating "derived above" – a page or equation number would be helpful.
36. Page number 27, 6th paragraph 1st sentence. The RSC has been previously defined, suggest dropping the last half of the sentence and the definition of RSC.
37. Page number 27, Table 6 paragraph. Suggest presenting the Household TWA in a column before the Peak Air Concentrations, so that ratio ranges in column 5 appear logical, low to high.
38. Page number 27, Table 7. Footnote 3, suggest adding "as derived on page 26".
39. Page number 28, 1st paragraph 3rd sentence. This doesn't make sense to me, the proposed MCL is based on non-cancer, the cancer based targets are below the non-cancer target. The proposed MCL is not protective of potential cancer risks at a 10E-6 level.
40. Page number 29, 2nd paragraph 2nd sentence. Suggest changing " indoor air concentrations that within" to "indoor air concentrations that are within".
41. Page number 29, 3rd paragraph. Is this a comprehensive listing of other state's work? ATSDR profule (1997) lists AZ and NH in addition to FL and MN.
42. Page number 33, 1st paragraph 2nd sentence typo. Suggest changing 140 ug/mn3 to 140 ug/m3
43. References. Please use consistent format, put the year in parentheses, or not. Please add a space before the entry for Pastino et al., 2000

References missing:

ATSDR 2013

CTDPH 2003

Ginsberg 2003

Jo 1990 references need to be qualified with a and b

MADEP 2013

Minnesota DPH

NRC 2006

USEPA Children's Exposure Factor Handbook

DPH Response: these references and other new references stemming from this round of comment/revision have been added to the updated reference list.

Comments of John Budroe, California OEHHA

General Comments

The October 2013 draft Maximum Contaminant Level (MCL) Determination for Trichloroethylene is a well written document incorporating the most recently available human health risk assessment information. The MCL document uses cancer potency factors and non-cancer RfC and RfD values developed in the 2011 US EPA IRIS risk assessment document for trichloroethylene (TCE). That document was comprehensive, included the most recent scientific data available for TCE toxicity, was transparent in the derivation of its risk assessment factors and received both internal and external peer review. The use of those US EPA risk assessment values for the development of a TCE MCL was entirely appropriate.

Specific Comments

p.9, para 3: The evidence of formation of genotoxic metabolites of TCE should be considered to be applicable to mode of action determinations of any of the tumor types associated with TCE exposure in either animals or humans.

DPH Response: this possibility has been considered in light of USEPA/IRIS stating that the renal endpoint is the only recommended for the application of ADAFs. The implications of the mutagenic MOA being relevant to all tumor endpoints is now considered in Table 5 and related text.

p. 14, para 1: The IRIS TCE cancer potency factor has been adopted by the Proposition 65 program of the California Office of Environmental Health Hazard Assessment (OEHHA) for the purpose of developing a No Significant Risk Level for Proposition 65 purposes. However, it should be noted that the OEHHA drinking water Public Health Goal (PHG) program has not adopted the 2011 US EPA IRIS human health risk assessment values, and the PHG value adopted for TCE in 2009 by OEHHA has not been changed. The PHG program may reevaluate the PHG value for TCE in the future.

DPH Response: A sentence to this effect has been added to the relevant section.

p. 21, para 2 (Consideration of Children's Cancer Risk): The TCE mutagenicity data suggests a mutagenic MOA for all types of TCE-induced cancer, indicating that it would be appropriate to apply age-specific adjustment factors to cancer risk for all tumor types associated with TCE exposure, including liver and lymphatic tumors.

DPH Response: This comment is similar to a previous comment from this reviewer and is addressed above.

p. 26, para 3 (Non-Cancer Endpoints): OEHHA is not currently using the TCE RfD developed in the US EPA IRIS 2011 TCE document for any programmatic purpose.

DPH Response: The OEHHA reference in this sentence has been deleted.

Comments of Kate Sande, Minnesota Dept of Health

Comments from: Kate Sande
Minnesota Department of Health

I. Introductions/purpose

The introduction does a good job of laying out the purpose of your MCL document. I do suggest that the last paragraph in the introduction be moved to be the first paragraph in the section; or maybe it could become a summary that precedes the background section. If you do create a summary, you could also reiterate that Connecticut did not conduct an independent review of the TCE toxicity studies but instead relied upon the EPA IRIS Toxicological Review for TCE as the starting point for your assessment and MCL development process.

DPH Response: An executive summary was added so there is no need to rearrange the introduction as the summary paragraph referred to in this comment is captured in the ES. We do not state that CT did not perform an independent review since we did evaluate USEPA IRIS in relation to other sources of toxicology information (e.g., California, New Jersey, ATSDR) to determine which toxicology values to use.

II. Comments on overall writing, clarity, scope, timing/rationale

The document is well done and overall, the writing is clear. A table of contents and a short summary section up front could help add clarity about the scope of the document. The timing and rationale are appropriate.

An acronym summary would be helpful.

DPH Response: Executive Summary, Table of Contents and Glossary of abbreviations have been added as suggested.

III. Comments on toxicology review

The toxicology review is a clear and provides a concise summary of the key points for a very complex document (the IRIS TCE tox review).

It would be helpful if there are separate paragraphs summarizing the key findings for each of the three oral candidate critical studies selected by the EPA that provided the basis for the non-cancer RfDs. I suggest including information about the study design and life stage that was evaluated in each study. For example, you do describe that mice in the Peden-Adams study were exposed in utero and postnatally, which is useful. I would provide that kind of information for all three critical studies. I think it would also be helpful to explicitly state that the exposure occurred during critical windows of immune system development for the Peden-Adams study (I think you already provide this kind of information for the Johnson study). And it might be useful to explain that animals in the Keil study were exposed to TCE when they were older and their immune systems were more mature.

I think it would also be useful to provide a brief explanation about how the immune effects observed in mice in the Peden-Adams and Keil studies might translate to effects in humans (such as autoimmunity). You may do this in another part of the document but I can't recall for sure.

You did a great job describing the Johnson study and its limitations. One of the things that we (MDH) struggled with was acknowledging the Johnson study and the body of evidence supporting the risk of cardiac malformations posed by TCE exposure while highlighting the uncertainty we had about the levels of exposure that pose a true risk for heart defects.

Table 3: An RfC was developed based on the Keil study, not the Peden-Adams study (I made this edit in the document using track changes).

DPH Response: added information was provided for the mouse immunotoxicity studies that were used by USEPA in RfD derivation and Table 3 was corrected as pointed out in this comment.

IV. Comments on exposure (inhalation) assessment and children's risk

I suggest including a more information about where the adjustment factors came from that were used for the cancer risk scenario.

It would be useful to include more scenarios that evaluate non-cancer risks for vulnerable populations and that reflect the life stages that were evaluated in the critical studies - pregnant women and infants and not just adult males. I suggest showing the derivation of guidance values using a pregnant woman's water intake rate and an infant/child's water intake rate, both of which may be higher than the intake rate based on a 70 kg adult that drinks 2 Liters of water per day.

Examples
$95^{th}/90^{th}$ percentile drinking water intake rates for a bottle fed infant 1-3 months old:

0.25 L/kg-d / 0.17 mg/kg-d
-6-10 times higher than the default intake (0.029 L/kg-d)
(based on Table 3-9 from the 2008 EPA Children's Exposure Factors Handbook)

95th/90th percentile drinking water intake rates for a bottle fed infant 6-12 months old:
0.15 L/kg-d / 0.12 L/kg-d
-5 times higher than the default intake (0.029 L/kg-d)
(based on Table 3-9 from the 2008 EPA Children's Exposure Factors Handbook)

The 95th percentile time weighted average drinking water intake rate for a pregnant woman used by the Minnesota Department of Health is: 0.043 mg/kg-d
-1.5 times higher than the default intake (0.029 L/kg-d)

DPH Response: The document has been updated to make equations and derived adjustment factors more explicit. The document already includes the potential for postnatal drinking water ingestion rate/body weight being a relevant calculation and discusses this target scenario as an uncertainty. Given that the timing of the TCE effect on immune system development is unknown, DPH did not choose to use the more conservative (early life based) exposure calculation. Regarding pregnancy water consumption rates, this is now reviewed but DPH retains the 2 L/day, 70 kg body weight default calculation as the main approach as explained in the text.

V. Comments on assessment of current MCL risks

Again, I suggest that you include a discussion about the non-cancer risks for exposure scenarios that address vulnerable populations (pregnant women, infants, and children) in this section in addition to discussing the risks based on the default scenario (70 kg and 2 L water/day).

It would be helpful if you included more background about the decision to use 0.2 for the RSC. This is the same RSC that MDH used for all of the durations we evaluated and it was used because it is our default RSC for a volatile chemical (and based on the EPA RSC decision tree).

DPH Response: Regarding the other scenarios, see response to previous comment. Regarding more background on the RSC, this is a very commonly used value and so further explanation is not considered necessary.

VI. Comments on calculation of draft MCL

An additional discussion is needed about the decision to use 1 ug/L versus the risk based values that are derived in the MCL document.

I suggest that the selection of 1 ug/L be explicitly described as a policy decision that is supported by the cancer and non-cancer based guidance values you derived. Further discussion about the MCLG of 0 ug/L would support your decision and can help justify the decision to lower the MCL from 5 ug/L to 1 ug/L. Additionally, a discussion about the MCLG

provides an opportunity to remind public water systems that 0 ug/L is still an appropriate goal for carcinogens.

DPH Response: This advice is been taken with the determination of 1 ug/L now described in both section heading and text as a policy decision. The MCLG is used as additional justification as suggested.

VII. Comments on benefits assessment

This section was well done. I made some minor edits in the document itself using track changes. I do not have additional substantial comments about the benefits assessment as this is outside my knowledge base.

Comments of Gloria Post, New Jersey Dept Environmental Protection

Gary,

Attached is the Draft CT TCE MCL document with my suggested edits and comments in Tracked Changes.

Although I have many comments (both of a substantive and editorial nature), the overall basis and presentation of the proposed TCE MCL is sound in my opinion. The risk analysis presented in the document is very thorough and technically sound. Specifically, the presentation of exposure and risks from ingestion alone, ingestion + dermal/inhalation for cancer and non-cancer endpoints, cancer risks with and without consideration of additional exposure and risks for children, and cancer risks for 30 vs. 70 years is very complete and useful. Also, the consideration of risks of potential developmental effects at the maximum quarterly exceedance of the MCL based on running annual average as is very helpful and important.

My main comments are:

1. In general, the language in the document should be carefully reviewed and revised where necessary to make sure that the information and conclusions are presented clearly and precisely. There are many instances where this is not the case, in my opinion, although the intended meaning is clear to someone familiar with the topic.

A few examples where more precise language is suggested (not a complete list) are:
--An MCL cannot exceed a Reference Dose. Rather, the exposure from drinking water at the MCL can exceed a Reference Dose.
--The "Risk-based MCL" (called MCLG by USEPA, Health-based MCL by NJ, or more generally "Health-based Goal" or "Health-based Criterion") needs to be distinguished from the final regulatory MCL when mentioned.
--The basis for CT's use of 1 x 10-6 as the cancer risk level needs to be stated. This risk level is not necessarily generally accepted as the de minimus risk level; some states use 1 x 10-5 or other levels.

--The analytical basis for the MCL (the PQL) is not the level to which the chemical can be detected, but rather the level to which it can be reliably measured. There are many documents on approaches to derive the PQL from the Detection Level. (I can provide citations later if you would like.)

DPH Response: Detailed comments provided in track changes in the text have been reviewed and incorporated to improve clarity and precision, although this document was not intended for something other than a technical audience. A new section has been added that describes the basis for CT's use of 1E-06 as de minimus risk. The description of the PQL has been changed to be more precise as suggested.

2. As you know, the NJ MCL of 1 ug/L was developed over 25 years ago based on the PQL achievable at that time. From the discussions on our conference call, I understand the rationale for proposing 1 ug/L instead of 0.5 ug/L, the currently achievable PQL. However, I am not sure that the rationale as presented in the document (use of a very old MCL based on outdated PQL) will be apparent and logical to the readers.

DPH Response: The MCL determination of 1 ug/L is now described as a policy decision based upon risk-based considerations, detectability and prior precedent that demonstrates feasibility. This will hopefully clarify that it is not a technical decision based narrowly on the current PQL.

3. I am not sure about the rationale for the cost-benefit analysis. I made some comments in the document itself about this. As I am not an expert on this topic, I suggest that you ask someone with expertise in this field to look at my comments/questions and see what they think.

DPH Response: The comments made in the text regarding benefit analysis have been incorporated. In addition, other text comments have been incorporated to improve clarity and to add missing references.

Thank you for the opportunity to review this important document. As mentioned in several places, I would be glad to provide additional citations if you would like. Please let me know if you have any questions about my comments or need additional information.

Best regards,
Gloria

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