

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

Comments of Safer Chemicals Healthy Families, Environmental Health Strategy Center, Earthjustice and Natural Resources Defense Council on EPA's Draft Risk Evaluation for Trichloroethylene under Section 6(b) of TSCA

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Safer Chemicals Healthy Families (SCHF), Environmental Health Strategy Center, Earthjustice and Natural Resources Defense Council submit these comments on the Environmental Protection Agency (EPA) draft risk evaluation for trichloroethylene (TCE) under section 6(b) of the Toxic Substances Control Act (TSCA).¹ Our organizations are committed to assuring the safety of chemicals used in our homes, workplaces and the many products to which our families and children are exposed each day. We took a leadership role during the TSCA legislative process, advocating the most protective and effective legislation possible to reduce the risks of toxic chemicals in use today.

These comments identify serious flaws in the TCE Risk Evaluation for consideration by EPA's Science Advisory Committee on Chemicals (SAAC) during its upcoming meeting on March 24-27, 2020. TCE is a high exposure/high hazard chemical with several known health effects that have long been of deep concern to state and federal agencies and the public. The draft evaluation determines that virtually every existing condition of use of TCE presents unreasonable risks to workers and users of consumer products. While these findings are alarming, however, they fail to reflect the full seriousness of TCE's risks to health and the true size of the population at risk. Because of its serious understatement of exposure and risk, the EPA evaluation is insufficiently protective and, if used as the basis for risk management, will leave large segments of the US population exposed to unsafe levels of TCE.

Because states will be pre-empted by TSCA from adopting additional risk management measures to address TCE once EPA's actions are complete, it is critical for EPA to fully account for all TCE pathways of exposure and conditions of use, accurately and fully identify all health endpoints contributing to TCE's risks, and ensure that its risk evaluation and risk management actions protect vulnerable populations.

We focus in these comments on three aspects of the draft evaluation that greatly understate TCE's risks. These are far from the only deficiencies in the draft risk evaluation, and we intend to submit additional comments during the pending comment period. We encourage the SACC to consider all submitted comments when preparing its report.

Failure to Base Unreasonable Risk Determinations on Evidence of Fetal Heart Defects

¹ 85 Federal Register 11079 (February 26, 2020); Draft Toxic Substances Control Act (TSCA) Risk Evaluation for Trichloroethylene (TCE Risk Evaluation), https://www.epa.gov/sites/production/files/2020-02/documents/1_draft_risk_evaluation_for_trichloroethylene_tce_public.pdf

In past assessments and rulemakings under TSCA, EPA has consistently concluded that the weight of the scientific evidence supports the link between TCE and fetal heart malformations and that, as the most sensitive endpoint, these effects should drive risk determinations for acute and chronic TCE exposure. As originally drafted by EPA career scientists, the draft risk evaluation reaffirmed this approach. However, a recent investigative report has now revealed that, after the draft was submitted for interagency review, the White House directed EPA not to use fetal heart defects to determine unreasonable risk.² As a result, the draft evaluation was revised to state that “there are uncertainties which decrease EPA’s confidence in this endpoint” and therefore EPA will now use “immunosuppression and autoimmunity as the key endpoints for determining whether or not a condition of use presents unreasonable risks.”

EPA’s dose response analysis of acute exposure scenarios shows that the HEC₉₉ for immune system effects is 470 times higher than the HEC₉₉ for heart malformations. Thus, for consumers and workers, the Margins of Exposure (MOEs) are over two orders of magnitude lower for heart defects than immune effects. This means that exposure limits based only on immune effects would be unprotective for women of childbearing age and their offspring.

There is no credible scientific justification for ignoring evidence of fetal heart defects in evaluating TCE’s risks to health:

- EPA has repeatedly found that the "weight of evidence" (WOE) demonstrates that TCE causes fetal heart malformations, the available data are sufficient for dose-response assessment, and these data provide a sound basis for determining risks to consumers and workers. While the Agency now asserts (at the direction of the White House) that unspecified “uncertainties” weaken its “confidence” in the heart defect evidence, the entirety of the risk evaluation shows the exact opposite -- that this evidence is strong and reliable.
- The only change in circumstance since EPA’s earlier TCE assessments is a recent study by the Halogenated Solvents Industry Alliance (HSIA) that purports to find that TCE does not cause heart malformations. However, the draft evaluation concludes that this study’s “methodology was likely of reduced sensitivity” and did “not sufficiently examine the complete range of potential cardiac defects.” Moreover, for the narrow category of cardiac defects it addressed, the HSIA study in fact found a dose-related increase in heart malformations.
- The TCE draft selects immune effects as a “representative endpoint” that should drive determinations of unreasonable risks to the exclusion of other more sensitive endpoints. Under this unprecedented approach, sensitive endpoints supported by the weight of the evidence

² Elizabeth Shogren, *EPA scientists found a toxic chemical damages fetal hearts. The Trump White House rewrote their assessment*, Reveal/Center for Investigative Reporting, February 28, 2010 (Reveal Report) <https://www.revealnews.org/article/epa-scientists-found-a-toxic-chemical-damages-fetal-hearts-the-trump-white-house-rewrote-their-assessment/>. A copy of this article is attached to these comments. It would be instructive for SACC to compare the draft evaluation submitted for interagency review with the current public comment version.

could be ignored on the ground that the data for less sensitive endpoints warrant greater “confidence.” This violates the long-standing public health policy that risk managers should protect against the most sensitive health endpoints adequately demonstrated by the available science. Until now, EPA has consistently followed this principle.

- While TCE’s immune effects are serious and should be included in the TCE evaluation, the implication that the data supporting them are significantly more “certain” than the evidence of heart defects is an after-the-fact invention of the White House with no support elsewhere in the draft evaluation. It is clear from the evaluation that EPA career scientists had “high confidence” in *all* the endpoints selected as Points of Departure (PODs) and drew no distinction between immune effects and fetal heart defects based on relative degrees of “certainty.”

Failure to Address the Contribution of Air, Water and Soil Contamination to the Risks Faced by the General Populations and Vulnerable Subpopulations

Like previous evaluations, the draft ignores the human health implications of TCE releases to the environment. In fact, TCE air emissions and contaminated groundwater, drinking water and soil are pervasive across the US and contribute significantly to overall TCE exposure. Each of these pathways is alone responsible for cancer and non-cancer risks to large segments of the population that exceed EPA benchmarks. Moreover, some subpopulations are exposed by multiple pathways simultaneously – i.e. individuals who breath TCE in indoor and outdoor air, consume contaminated drinking water and live near TCE-contaminated Superfund sites. Because TCE exposure levels are higher for these subpopulations than for the general population, they face elevated risks of TCE-related health effects that the draft evaluation ignores. Indeed, even for the limited populations (workers and users of consumer products) that the draft evaluation addresses, EPA significantly understates risks by ignoring exposure to TCE in air, water and soil.

A comprehensive risk evaluation as required under TSCA would identify and quantify these subpopulations, estimate total exposure from all sources and characterize the increased risk resulting from concurrent exposure pathways. However, because of its narrow scope, the draft TCE evaluation fails to provide this analysis and therefore presents a limited and incomplete picture of TCE’s risks to the public. The SACC should recommend that EPA revise the draft TCE evaluation so it accounts for *all* sources of exposure and risk and provides a comprehensive understanding of TCE’s dangers to public health.

Unwarranted Reliance on Personal Protective Equipment (PPE) in Determining TCE Risks to Workers

As in previous risk evaluations, EPA’s risk determinations for workers exposed to TCE calculate MOEs assuming both the use of respirators and gloves and the absence of protective equipment. Even for scenarios where workers consistently and reliably use PPE, EPA concludes that MOEs are below “benchmarks” for all conditions of use. However, while unacceptably low even with PPE use, EPA’s MOEs are significantly lower for “no PPE” scenarios.

As the SAAC has repeatedly underscored and EPA's draft evaluations recognize, an expectation of universal PPE use is in fact contrary to the realities of workplace practice and sound principles of worker protection. For this reason, the "no PPE" scenario is the only defensible baseline for determining current risk levels for exposed workers and then defining the additional worker protections necessary to eliminate unreasonable risk. SACC should thus recommend that the final TCE evaluation base determinations of unreasonable risk solely on the "no PPE" scenario.

I. EPA's Unreasonable Risk Determination for TCE Should be Based on Cardiac Malformations as the Most Sensitive Endpoint Supported by the Weight of the Evidence

EPA's 2011 IRIS³ and 2014 Workplan⁴ assessments concluded that the weight of the scientific evidence supports the link between TCE and fetal heart malformations and that, as the most sensitive endpoint, these effects should drive risk determinations for acute and chronic TCE exposure. These conclusions formed the basis for EPA's proposals in late 2016 and early 2017 to ban vapor and aerosol degreasing and spot removal uses of TCE under section 6 of TSCA.⁵

EPA again relied on the evidence of fetal heart defects in the draft TSCA risk evaluation it submitted to the White House for interagency review in December 2019. According to a recent report by the Center for Investigative Reporting, this draft stated as follows:⁶

"EPA identifies developmental cardiac malformations as the driver end point for the conditions of use that EPA has preliminarily determined present unreasonable risk. This is the effect that is most sensitive, and it is expected that addressing risks for this effect would address identified risks."

However, the draft that EPA released for public comment and peer review on February 21 omits this statement and no longer bases EPA's determination of unreasonable risk on fetal heart defects. Instead, it claims that "there are uncertainties which decrease EPA's confidence in this endpoint" and therefore EPA will now use "immunosuppression and autoimmunity as the key endpoints for determining whether or not a condition of use presents unreasonable risks."⁷ As the Center for Investigative Reporting found and EPA has now admitted,⁸ this reversal of EPA's longstanding position occurred at the express direction of the White House Executive Office of the President, which instructed EPA career scientists to

³ EPA, Toxicological review of trichloroethylene (CASRN 79-01-6) in support of summary information on the Integrated Risk Information System (IRIS) (IRIS Report). (EPA/635/R-09/011F), September 2011 https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0199tr/0199tr.pdf

⁴ EPA, TSCA Work Plan Chemical Risk Assessment Trichloroethylene: Degreasing, Spot Cleaning and Arts & Crafts Uses, June 2014 (Work Plan Assessment), https://www.epa.gov/sites/production/files/2015-09/documents/tce_opptworkplanchemra_final_062414.pdf

⁵ 81 Fed. Reg. 91592 (Dec. 16, 2016) (proposed TSCA ban on TCE aerosol degreasing and spot removal uses); 82 FR 7432 (Jan. 19, 2017) (proposed TSCA ban on TCE use for vapor degreasing).

⁶ Reveal Report, note 2.

⁷ TCE Draft Evaluation at 377.

⁸ INSIDE EPA, *EPA Defends Its Process For Crafting Public Draft TCE Risk Evaluation*, March 2, 2020, <https://insideepa.com/daily-news/epa-defends-its-process-crafting-public-draft-tce-risk-evaluation>

rewrite the draft to cast doubt on the evidence of cardiac defects and to shift the basis of its risk determinations to less sensitive endpoints.

The revised draft developed at White House direction asserts that despite these changes, its unreasonable risk determinations remain the same for most TCE conditions of use, implying that the exclusion of fetal heart defects from these determinations is inconsequential from a public health perspective:⁹

“For the majority of the occupational and consumer conditions of use, unreasonable risk determinations were consistent whether based on congenital heart defects (an endpoint for which EPA has lower confidence) or immunosuppression and autoimmunity endpoints.”

This is highly misleading. While the evaluation concludes that immune-related effects do present unreasonable risks for nearly all conditions of use, these effects occur at significantly higher dose levels than heart malformations. Thus, a significant and unreasonable risk will still exist if EPA bases exposure limits on the less sensitive immune endpoints. For example, EPA’s dose response analysis of acute exposure scenarios shows that the HEC₉₉ for immune system effects is 470 times higher than the HEC₉₉ for heart malformations.¹⁰ Thus, for consumers and workers, the Margins of Exposure (MOEs) are over two orders of magnitude lower for heart defects than immune effects. This means that exposure limits based on the immune effects would be unprotective for women of childbearing age and their offspring, for whom heart defects can cause serious health impairments and death in utero, during childhood and later in life.

As shown below, there is no credible justification for ignoring fetal heart defects and the serious dangers they pose to pregnant women exposed to extremely low levels of TCE:

- EPA has repeatedly found – and the draft evaluation reaffirms -- that the "weight of evidence" (WOE) demonstrates that TCE causes fetal heart malformations, the available data are sufficient for dose-response assessment and there is a sound basis for using MOEs for these effects for determinations of unreasonable risk. While the Agency now asserts (at the direction of the White House) that unspecified “uncertainties” weaken its “confidence” in the heart defect evidence, the entirety of the risk evaluation shows the exact opposite -- that this evidence is strong and reliable.
- The only change in circumstance since EPA’s earlier TCE assessments is a recent study by the Halogenated Solvents Industry Alliance (HSIA), representing TCE manufacturers, that purports to find that TCE does not cause heart malformations.¹¹ However, the draft evaluation contains a lengthy critique of the HSIA study which concludes that its “methodology was likely of reduced sensitivity” and did “not sufficiently examine the complete range of potential cardiac defects.”¹²

⁹ TCE Risk Evaluation at 377.

¹⁰ Id, at 252.

¹¹ Halogenated Solvents Industry Alliance, An Oral (Drinking Water) Study of the Effects of Trichloroethylene (TCE) on Fetal Heart Development in Sprague Dawley Rats, Charles River Laboratories Ashland, February 25, 2019 (HSIA Study), [file:///C:/Users/Owner/Downloads/EPA-HQ-OPPT-2016-0737-0120%20\(1\).pdf](file:///C:/Users/Owner/Downloads/EPA-HQ-OPPT-2016-0737-0120%20(1).pdf)

¹² TCE Risk Evaluation at 222-23.

For this reason and because of other flaws, EPA found that the HSIA study did not “sway the weight of evidence for the endpoint.” Thus, while White House reviewers may have viewed the HSIA study as a new source of “uncertainty,” the EPA scientific review concluded that it did not materially alter previous EPA assessments of the strength of the data. In fact, for the narrow category of cardiac defects it addressed, the HSIA study found a dose-related increase in heart malformations remarkably similar to the increase reported in the (Johnson et al 2003) study that HSIA has sought to discredit.

- To justify disregarding fetal heart defects, the TCE draft selects immune effects as a “representative endpoint” that should drive determinations of unreasonable risks to the exclusion of other more sensitive endpoints. This approach would allow the Agency to disregard the endpoints of greatest concern based on subjective and scientifically dubious judgements of the relative “certainty” of different bodies of evidence. Sensitive endpoints supported by the weight of the evidence could thus be ignored on the ground that the data for other endpoints warrant greater “confidence.” This violates the long-standing public health policy that risk managers should protect against the most sensitive health effects adequately demonstrated by the available science. Until now, EPA has consistently followed this principle. Nothing in TSCA provides any basis for a different approach. Indeed, the law requires EPA to assure that its risk evaluations address all unreasonable risks to “potentially exposed or susceptible subpopulations.” Fetuses exposed to TCE at levels that can cause life-threatening heart defects in utero or after birth fall squarely within the vulnerable populations that EPA must protect under TSCA.
- While TCE’s immune effects are serious and should be included in the TCE evaluation, the implication that the data supporting them are significantly more “certain” than the evidence of heart defects is an after-the-fact invention of the White House with no support elsewhere in the draft evaluation. The evaluation repeatedly states that EPA has “high confidence” in *all* the endpoints selected as Points of Departure (PODs). While the draft evaluation (presumably at White House direction) cites factors that purportedly warrant greater reliance on the immune endpoints, this comparison is unconvincing when the strengths and limitations of the two bodies of evidence are objectively evaluated. Thus, there is simply no basis to claim that the immune effects data provide sufficient “certainty” for a determination of unreasonable risk but the heart defect data do not.

A. EPA Has Repeatedly Determined that the Weight of Evidence Demonstrates the Link Between TCE and Fetal Heart Defects

IRIS Assessment. The 2011 IRIS assessment of TCE relied on the fetal cardiac effects demonstrated in Johnson et al (2003) to derive an RFC and RFD for TCE, finding “that the most sensitive developmental effect by far was heart malformations in the rat reported by Johnson et al. (2003), . . . [and that]

although this study has important limitations, the overall weight of evidence supports an effect of TCE on cardiac development.”¹³

The Johnson data were derived from a 6-year academic research program and consolidated data from several cohorts. Control data were combined from 6 independent cohort experiments.¹⁴ The study administered 0 ppb, 2.5 ppb, 250 ppb, 1.5 ppm, and 1100 ppm of TCE to pregnant Sprague-Dawley rats via drinking water for the entire duration of pregnancy. On the last day of pregnancy, dams were euthanized, and the heart and great vessels of fetuses were examined for abnormalities. The study reported statistically significant increases in the incidence of a broad array of severe cardiac defects at multiple dose levels

The cardiac malformations reported by Johnson et al were also observed in studies of other species. Evaluating the animal data as a whole, IRIS concluded that:¹⁵

“The animal data provide strong, but not unequivocal, evidence of the potential for TCE-induced cardiac malformations following oral exposures during gestation. Strengths of the evidence are the duplication of the adverse response in several studies from the same laboratory group, detection of treatment-related cardiac defects in both mammalian and avian species (i.e., rat and chicken), general cross-study consistency in the positive association of increased cardiac malformations with test species (i.e., rat), route of administration (i.e., oral), and the methodologies used in cardiac morphological evaluation (i.e., fresh dissection of fetal hearts). Furthermore, when differences in response are observed across studies, they can generally be attributed to obvious methodological differences, and a number of in vivo and in vitro studies demonstrate a consistent and biologically plausible mode of action for one type of malformation observed.”

IRIS also found that epidemiology studies provided evidence of TCE-related cardiac effects in humans:

“[T]wo well-conducted studies by ATSDR (2008b, 2006a) clearly demonstrated an elevation in cardiac defects. It could be surmised that the identified cardiac defects were detected because they were severe, and that additional cases with less severe cardiac anomalies may have gone undetected.”¹⁶

Finally, IRIS cited mechanistic data from in vitro studies as further confirmation of human and animal data:¹⁷

¹³ IRIS Assessment at 5-45.

¹⁴ Johnson PD, Goldberg SJ, Mays MZ, Dawson BV. Threshold of trichloroethylene contamination in maternal drinking waters affecting fetal heart development in the rat. *Environ Health Perspect.* 2003 Mar;111(3):289-92. Erratum in: *Environ Health Perspect.* 2014 Apr;122(4):A94.

¹⁵ IRIS Assessment at 4-565

¹⁶ *Id.*

¹⁷ *Id.* at 4-564

“Thus, in summary, a number of studies have been conducted in an attempt to characterize the mode of action for TCE-induced cardiac defects. A major research focus has been on disruptions in cardiac valve formation, using avian in ovo and in vitro studies. These studies demonstrated treatment-related alterations in endothelial cushion development that could plausibly be associated with defects involving septal and valvular morphogenesis in rodents and chickens.”

Summarizing its weight of evidence assessment, IRIS indicated that, “based on weakly suggestive, but overall consistent, epidemiologic data, in combination with evidence from experimental animal and mechanistic studies, it can be concluded that TCE exposure poses a potential hazard for congenital malformations, including cardiac defects, in offspring.”¹⁸

The TCE IRIS assessment underwent several levels of peer review, including agency review, science consultation on the draft assessment with other federal agencies and the Executive Office of the President, public comment, external peer review by the EPA’s Science Advisory Board (SAB) in 2002, scientific consultation by the U.S. National Academy of Sciences (NAS) in 2006,¹⁹ external peer review of the revised draft assessment by the EPA’s SAB in January 2011,²⁰ and final internal agency review and EPA-led science discussion on the final draft.

2014 TSCA Workplan Assessment. EPA’s 2014 Workplan risk assessment likewise determined risks of acute TCE exposure based “on the most health protective endpoint (i.e., fetal cardiac malformations; Johnson et al., 2003) representing the most sensitive human population (i.e., adult women of child-bearing age and fetus > 16 yrs).”²¹ These risks were of particular concern for acute exposure “based on U.S. EPA’s policy that a single exposure of a chemical within a critical window of fetal development may produce adverse developmental effects.” The assessment found that “TCE-induced fetal cardiac malformations are biologically plausible based on the weight of evidence analysis presented in the TCE IRIS assessment, which considered human and animal findings as well as mechanistic data.”²² Updating the IRIS review of the weight of evidence in light of additional information about the Johnson studies, EPA found that a “recent erratum (Johnson, 2014) and subsequent evaluation of the developmental toxicity data reaffirmed that the Johnson et al. studies are adequate to use in hazard identification and dose-response assessment” and that despite their limitations, “there is insufficient reason to dismiss

¹⁸ Id. at 6-11

¹⁹ NAS report, “Assessing the human health risks of trichloroethylene: Key scientific issues (2006)”: http://www.nap.edu/catalog.php?record_id=11707.

²⁰ EPA’s SAB peer review report for the 2009 EPA’s Draft Assessment entitled “Toxicological Review of Trichloroethylene”: [http://yosemite.epa.gov/sab/sabproduct.nsf/c91996cd39a82f648525742400690127/B73D5D39A8F184BD85257817004A1988/\\$File/EPA-SAB-11-002-unsigned.pdf](http://yosemite.epa.gov/sab/sabproduct.nsf/c91996cd39a82f648525742400690127/B73D5D39A8F184BD85257817004A1988/$File/EPA-SAB-11-002-unsigned.pdf).

²¹ Work Plan Assessment at 104.

²² Id at 21.

their findings, especially when the findings are analyzed in combination with the remaining body of human, animal and mechanistic evidence.”²³

2016 WOE Assessment. In 2016, several EPA scientists published an updated weight of evidence (WOE) review of the available scientific literature on TCE-related developmental cardiac defects, reporting on the quality, strengths, and limitations of the available studies (Makris et al 2016).²⁴ Their updated review and assessment confirmed earlier EPA determinations that the weight of the evidence demonstrated the relationship between fetal heart defects and TCE exposure and that the Johnson studies, augmented by detailed additional information about study design and conduct, were sufficient for dose-response analysis and determinations of risk.

The authors conducted an in-depth examination of the Johnson study, which concluded that:

“On the whole, the Johnson et al. study is considered suitable for use in deriving a POD for the following reasons. The study has an appropriate design. It was conducted by a relevant route of exposure (drinking water), covered the entire period of gestation which subsumes the developmental window for the initiation of cardiac defects, and tested multiple exposure levels.”

Responding to criticisms of the Johnson and Dawson studies, the authors found that a “number of potential concerns associated with these studies were dispelled, e.g., that inadequate or inappropriate cardiac evaluation methods were used, control animals were not on study concurrently with treated animals, fetuses were not randomly assigned to evaluations, cardiac examinations were conducted with knowledge of treatment group, and statistical analysis of cardiac malformation data was inappropriate.”

Based on a detailed methodological comparison of Johnson/Dawson and negative animal studies, Makris et al reached “the conclusion that differences in study methods (e.g., route of exposure, vehicle, animal source or strain, or other factors) may have contributed to differences in the detection of cardiac malformations.”

Makris et al added that “further support [for relying on the Johnson study] was derived from the finding of a robust, statistically significant dose-response relationship.” As they explained:

“Confidence that data from Johnson et al. [51] represent a real response is supported by the increasing trend in response (Fig. 6), and the observations of higher percentages of cardiac malformations elicited by higher doses (500 mg/kg-day and higher) in studies of rats exposed to TCE metabolites, TCA and DCA [27,79,78]. The highest dose in the Johnson et al. [51] study lies at the lower end of doses that elicited substantial responses in these other studies. *Thus, a hypothesis that the Johnson data represent a false positive or an anomalous dose-response*

²³ Id at 98.

²⁴ Makris SL, Scott CS, Fox J, Knudsen TB, Hotchkiss AK, Arzuaga X, Euling SY, Powers CM, Jinot J, Hogan KA, Abbott BD, Hunter ES 3rd, Narotsky MG. A systematic evaluation of the potential effects of trichloroethylene exposure on cardiac development. *Reprod Toxicol.* 2016 Oct;65:321-358. doi:10.1016/j.reprotox.2016.08.014. Review. PubMed PMID: 27575429.

pattern seems implausible, based on trend tests and comparison with studies that used higher doses” (emphasis added).

Makris et al also found that concerns about variability among litters were resolved in the method for data analysis:

“The possibility of increased variability among litters due to temporal drift and perhaps other factors across time (overdispersion), was dealt with by using a standard method for clustered data. The dose-response trend was found to be highly significant after adjusting for adjusting for overdispersion. Because the maximal observed response was 10%, models with plateaus of less than 100% were investigated and were found to not substantially change the general conclusions and results. Confidence in the dose-response relationship is supported by the increasing trend in response and by metabolite studies that demonstrate findings at higher dose levels.”

Overall, like the IRIS and Work Plan assessments, the Makris et al review determined that, “[d]espite the recognized uncertainties and limitations in the TCE database, the evidence supports a conclusion that TCE has the potential to cause cardiac defects in humans when exposure occurs at sufficient doses during a sensitive period of fetal development. This conclusion is warranted by the data that demonstrate or suggest a potential hazard to cardiac development, including epidemiological studies, developmental toxicology studies in rodents with TCE and its metabolites (DCA and TCA), avian in ovo studies, in vitro assays, and mechanistic data that form the basis of a preliminary conceptual model of an AOP for valvulo-septal defects resulting from TCE exposures.”²⁵

B. Despite White House Intervention, the Draft Evaluation Reaffirms the Weight of Evidence for TCE-Related Cardiac Defects

Even with the changes demanded by the White House, the draft TCE evaluation presents a strong case for the sufficiency of the evidence of TCE-related cardiac effects.

Both the body of the risk evaluation and Appendix G provide a detailed analysis of the weight of evidence for congenital heart defects. Based on scoring of all relevant studies and integration of data across lines of evidence, EPA summarized the database as follows:²⁶

“In summary, the database contains a large and diverse set of studies pertinent to assessing congenital heart defects from TCE exposure (overall relevance was rated as ++). Well-designed, conducted and reported studies were located for all categories, although the epidemiology studies were limited to ecological or case-control study designs with high potential for misclassification of exposure and many of the in vivo animal studies contained at least one major limitation (overall reliability rating of +/-++). The integrated strength area score was (+),

²⁵ An industry sponsored WOE review, Wikoff et al 2018, reached a different conclusion using a Risk Of Bias assessment for internal study validity but, as noted in the draft evaluation, this review focused only on animal and epidemiological data. TCE Risk Evaluation at 222.

²⁶ Id at 620.

indicating a suggestive positive association of TCE with congenital cardiac defects. The epidemiology studies as a group provide suggestive evidence for an effect of TCE on cardiac defects in humans (summary score of +). Oral in vivo studies provided ambiguous to weakly positive (0/+) results for TCE itself, but positive results for its TCA and DCA metabolites (+), while inhalation studies contributed negative evidence (-). Mechanistic studies provided solid, consistent supporting information for effects of TCE and metabolites on cardiac development and precursor effects (summary score of ++).”

EPA then concluded that:²⁷

“Overall, the database is both reliable and relevant and provides positive overall evidence that TCE may produce cardiac defects in humans (based on positive evidence from epidemiology studies, mixed evidence from animal toxicity studies, and stronger positive evidence from mechanistic studies).”

As EPA indicated, “[t]he fetal cardiac defects reported in (Dawson et al., 1993) and (Johnson et al., 2003) were identified as the most sensitive endpoint within the developmental toxicity domain and across all of the health effects domains evaluated in the TCE IRIS assessment.”²⁸ EPA noted that these studies were rated “medium” for data quality in its TSCA systematic review, which incorporated all available information on the two studies, including subsequent errata and communications to EPA. As EPA explained, “[w]hile the original publications had extensive data and methodology reporting issues, many of the data quality concerns from the original study were mitigated by the information provided in these updates.”²⁹

Of the two studies, EPA “decided to utilize (Johnson et al., 2003) for dose-response analysis, which has increased statistical sensitivity from the additional two dose levels and allowed a nested design for BMD modeling analysis in order to account for litter effects.”³⁰ Johnson was suitable for dose response assessment, according to EPA, because it “reported a statistically and biologically significant increase in the formation of heart defects at the 0.048 mg/kg-bw/day and higher dose levels (concentrations of 0, 0.00045, 0.048, kg-bw/day) measured on both an individual fetus basis and a litter basis.”³¹

²⁷ Id at 621.

²⁸ Id. at 232.

²⁹ Id., at 232-3. According to the draft, these “updates provided the following information which was lacking in the initial publications:

- 1) Individual fetal cardiac malformation data for each litter
- 2) Individual maternal terminal body weight data
- 3) Detailed description of fetal evaluation procedures including:
 - methods used to blind fetal examiners to treatment group
 - protocol for unanimous confirmation of any observed cardiac defects by the three principle investigators
- 4) Additional information on animal husbandry and randomized group assignment of dams to study group
- 5) Transparency regarding experimental variables across the dates of the experiments.”

³⁰ Id. at 233.

³¹ Id. at 237.

Using additional information reported by the study authors, EPA reevaluated the BMR used in the 2014 risk assessment using biological and statistical factors, concluding "that the biological severity of the effect, potentially lethal heart defects, strongly supported a BMR of 1%." Compared to the 2014 assessment, EPA concluded that "the p- value of = 0.661 from the updated BMDS nested model run (Appendix N) is significantly improved, demonstrating strong model fit and confirming the 2011 conclusion that the modeling results for cardiac malformation data are appropriate for reference value derivation."³²

C. The HSIA Study Does Not Rebut the Johnson Study and In Fact Provides Additional Evidence of the Link between TCE Exposure and Fetal Heart Defects

Since EPA's 2011, 2014 and 2016 WOE reviews of the evidence for fetal heart defects, the only new information to become available is the 2019 HSIA-sponsored drinking water study of TCE's effects on fetal heart development in Sprague Dawley rats.³³ The stated purpose of this study was to replicate the fetal malformations observed in the Johnson and Dawson studies. The study authors reported that the study was negative. However, Appendix G of EPA's draft evaluation includes a detailed review of the HSIA study which concludes that, because of its severe limitations, the study did not negate the earlier findings of TCE-induced heart defects and thus did not warrant any change in the Agency's previous WOE determinations for this endpoint.

As EPA notes, the "Johnson study clearly shows greater incidences of cardiac defects at 0.25 ppm, 1.5 ppm, and 1100 ppm compared to the same or similar doses" in the HSIA study. However, "VSDs, and specifically only membranous VSDs, were the only type of heart malformation identified" in the HSIA study, whereas "the Johnson study identified a broad variety of defects in exposed fetuses."³⁴ The explanation of this discrepancy, according to EPA, is that the HSIA study [was] insufficiently sensitive to non-VSD defects.³⁵ After conducting a detailed analysis of studies on other chemicals finding atrial and valve fetal heart defects (including RA, the positive control in the HSIA study), EPA found that:³⁶

"In the Johnson study, the materials and methods section described examination of the internal structure of the heart for all fetuses. The dissection methodology allows detailed examination of the atrial septum. In contrast, the [HSIA] study states that the fetal evaluation methods were conducted according to Stuckhardt and Poppe (1984), which does not include examination of atrial septal defects. Therefore, the methodology used by the [HSIA] study was likely to miss this important category of cardiac malformations."

³² Id. at 236-237.

³³ See note 11 supra.

³⁴ TCE Risk Evaluation at 601.

³⁵ Id. at 604.

³⁶ Id. at 607-608.

EPA thus concluded that the HSIA study “insufficiently replicates the methodology of ([Johnson et al., 2003](#)), and the results do not entirely contradict the conclusions of that study.”³⁷

Even in its identification and analysis of VSDs, EPA found that the HSIA study was highly flawed. According to EPA, the HSIA study discounted the <1mm VSDs induced by TCE because “... similar to humans, small spontaneous interventricular septal defects in rats close postnatally and hence should not be considered adverse.”³⁸ On this premise, the study authors claimed that “the interventricular septal defects observed in the TCE-treated groups were considered to be spontaneous background occurrences and unrelated to TCE exposure.” However, EPA did not accept this characterization, emphasizing that “one cannot rule out the possibility that any VSD may be a potential adverse effect of chemical exposure.” It added that “even if a membranous VSD is able to spontaneously close, there are likely functional impacts of that closer, resulting in an adverse health effect.”³⁹

EPA also found that HSIA’s efforts to dismiss the increase in VSDs in treated animals as “spontaneous” and “unrelated to TCE exposure” was “confounding and internally inconsistent . . . because the vast majority (92%) of VSDs observed in the RA-treated positive control group were also <1mm.” As EPA explained, “[i]f VSDs <1mm are truly non-adverse, then this positive control data provides additional indication that the study is insufficiently sensitive for detecting adverse cardiac defects.”⁴⁰

Equally important, the ventricular septal defects (VSDs) observed in treated animals showed a startling trend of increasing VSD with increasing dose and the VSD incidences at different dose levels were very close to those in the Johnson study. As EPA compared VSDs in the two studies:⁴¹

“In fact, the [HSIA] study (2019) observed a similar percentage of VSDs as (Johnson et al., 2003). Considering total VSDs, 3.5% of fetuses showed a VSD in [HSIA] vs 3.8% in Johnson at the highest dose, with 1.5% in [HSIA] vs 2.2% in Johnson at 1.5ppm. When considering only membranous VSDs (the only type observed in the [HSIA] study), observed incidences were actually higher in [HSIA] at the highest dose (3.5% vs 2.86%).”

HSIA’s convoluted efforts to establish that the dose-related VSD increases in its study were not statistically significant when compared to controls should receive little weight in assessing the study results. The unit of analysis in their statistical analysis is the litter, but with only 20 litters, the analysis is likely to be statistically underpowered. Typically, one would conduct statistical analyses using both the litter and the individual fetus, but this does not appear to have been done. In addition, the use of two-sided tests is inappropriate; such tests presume the treatment is like a pharmaceutical drug that could be either harmful or beneficial. Instead, HSIA and EPA should have used a one-sided test since the only possible test hypotheses are either no effect or adverse effects, not benefit (no one has seriously

³⁷ Id. at 222.

³⁸ Id. at 609.

³⁹ Id. at 610.

⁴⁰ Id.

⁴¹ Id. at 222.

proposed that TCE causes any benefits for fetal development). Had HSIA used the more appropriate one-sided statistical test, it would have doubled the statistical power, and likely would have resulted in a study outcome showing statistically significant harmful effects of the treatment. Thus, analyzing the VSDs on an individual animal basis through the Cochran Armitage trend test, the one-sided p-value is 0.0196, which is highly significant. EPA should provide this analysis in Appendix G.

An additional important problem is the HSIA's use of historical control data for some endpoints but not others, with no real rationale provided. The seemingly arbitrary oscillation between using within-study and historical controls casts doubt on the rigor and consistency of the statistical analysis, making it appear instead to be manipulated and biased to dismiss evidence of harm.

Finally, as the EPA guidelines for carcinogenicity risk assessment advise,⁴² observation of a dose-response trend may be sufficient to identify compound-related adverse effects in the absence of statistical significance, particularly when the adverse endpoint is permanent, serious, and possibly life-threatening. An important new paper published [this month in Nature](#), one of the world's most prestigious and highly ranked scientific journals, signed by over 800 supporters, argues that over-reliance on statistical significance to deny or disregard an adverse effect is a misuse of statistics and puts the public health at risk:

"Let's be clear about what must stop: we should never conclude there is 'no difference' or 'no association' just because a P value is larger than a threshold such as 0.05 or, equivalently, because a confidence interval includes zero. Neither should we conclude that two studies conflict because one had a statistically significant result and the other did not. These errors waste research efforts and misinform policy decisions" (Nature 2019).

This recommendation is of particular relevance to the dose-related VSD increases seen in the HSIA study, which represent a permanent and potentially fatal effect that mirrored similar dose-related cardiac defects seen in the Johnson study.

In short, even with its flaws, the HSIA study provides evidence of a link between TCE exposure and fetal heart defects, adding to the overall weight of evidence for this endpoint.

D. The White House-Imposed Rationale for Disregarding the Heart Defects Is Contrary to Sound Science and Accepted Policies and Principles of Risk Assessment

At the direction of the White House, the revised risk evaluation claims that "[w]hile congenital heart defects were the most sensitive endpoint for TCE, for the purpose of the draft risk determination, there are uncertainties which decrease EPA's confidence in this endpoint."⁴³ Nowhere, however, does EPA identify these "uncertainties" or describe why they "decrease confidence" in the heart defect endpoint.

EPA instead relies on general "scientific principles" required under TSCA that supposedly cast doubt on the finding of cardiac defects:

⁴² EPA, *Guidelines for Carcinogen Risk Assessment*, March 2005, https://www.epa.gov/sites/production/files/2013-09/documents/cancer_guidelines_final_3-25-05.pdf.

⁴³ TCE Risk Evaluation, at 377.

Section 26 of TSCA requires that EPA make decisions consistent with the “best available science.” Section 26 also requires other scientific considerations including consideration of the “extent of independent verification” and “weight of the scientific evidence.” As described in EPA’s framework rule for risk evaluation [82 FR 33726] weight of the scientific evidence includes consideration of the “strengths, limitations and relevance of the information.”

In fact, these are the very “principles” that EPA scientists used in evaluating the database on congenital heart effects. The Agency conducted an analysis of the “weight of the scientific evidence” (more detailed than for any other non-cancer endpoint) and examined the “strengths, limitations and relevance” of each study and the overall evidence. The EPA analysis demonstrated that the finding of TCE-related cardiac effects was not limited to animal studies but “independently verified” in epidemiology and mechanistic studies and that “the database is both reliable and relevant and provides positive overall evidence that TCE may produce cardiac defects in humans.” The analysis thus conformed to the definition of “weight of the evidence” in EPA risk evaluation regulations, which calls for the Agency to “comprehensively, objectively, transparently, and consistently, identify and evaluate each stream of evidence, including strengths, limitations, and relevance of each study and to integrate evidence as necessary and appropriate based upon strengths, limitations, and relevance.” 40 CFR §702.33

Nowhere did the EPA WOE analysis express a lack of “confidence” in the heart defect data. In fact, in multiple assessments, it found that the data as a whole provide a strong basis for determining unreasonable risk.

EPA’s analysis also incorporated the “best available science” as defined in its regulations. Id. The WOE assessment for TCE relied on data “that is reliable and unbiased” and derived from “studies conducted in accordance with sound and objective science practices.” For example, the key study, Johnson et al, that EPA used for dose-response analysis was screened for data quality using EPA’s TSCA systematic review protocol and scored as “medium” and therefore acceptable for inclusion in the risk evaluation. It also was ranked ++ for strength in EPA’s more detailed weight of evidence analysis, in contrast to the HSIA study, which was ranked as 0/- for this metric.

EPA scientists have now reached the same conclusions in four separate WOE assessments over the last nine years and these conclusions have been reviewed both by the EPA SAB and NAS. For the White House to disavow a decade of scientific work on the basis of nebulous “uncertainties” is the exact opposite of the “best available science” that EPA is obligated to use under TSCA.

To justify disregarding the cardiac malformations, the White House directed EPA to apply the novel approach of selecting a single “representative endpoint” to determine unreasonable risk and then ignoring more sensitive endpoints that present greater risks. Applying this concept, EPA chose immunotoxicity over heart defects as its “representative endpoint” for TCE.⁴⁴ This approach is without

⁴⁴ TCE risk evaluation, at 257

precedent in previous EPA risk evaluations under TSCA or other laws and is contrary to sound public health protection policy. As many examples demonstrate, risk assessors and risk managers have always based determinations of risk and related exposure limits on the most sensitive endpoint for which there is sufficient scientific evidence.⁴⁵ This ensures that at risk populations receive adequate protection from adverse effects. Otherwise, exposure limits will be too high to prevent harm and unreasonable risks will remain unaddressed.

These are particularly important considerations for congenital heart defects. As EPA underscored in its 2016 proposal to ban TCE use in aerosol degreasing, “TCE may cause fetal cardiac malformations that begin in utero. In addition, fetal death, possibly resulting from cardiac malformation, can be caused by exposure to TCE. Cardiac malformations can be irreversible and impact a person’s health for a lifetime.”⁴⁶ EPA elaborated that:⁴⁷

“Cardiac defects, which can result from very low level exposure to TCE, affect the structural development of a baby’s heart and how it works. The defects impact how blood flows through the heart and out to the rest of the body. The impact can be mild (such as a small hole in the heart) or severe (such as missing or poorly formed septal wall and valves of the heart). While diagnosis for some cardiac defects can occur during pregnancy, for other cardiac defects, detection may not occur until after birth or later in life, during childhood or adulthood. These cardiac defects can be occult or life- threatening with the most severe cases causing early mortality and morbidity.”

The occurrence of cardiac defects in the population of newborns is significant. According to the 2016 proposal:⁴⁸

“Nearly 1% or about 40,000 births per year in the United States are affected by cardiac defects (Ref. 46). About 25% of those infants with a cardiac defect have a critical defect. Infants with critical cardiac defects generally need surgery or other procedures in their first year of life. Some estimates put the total number of individuals (infants, children, adolescents, and adults) living with cardiac defects at 2 million.”

⁴⁵ For example, the EPA risk assessment guidelines for developmental toxicity state that “[t]he most sensitive developmental effect (i.e., the critical effect) from the most appropriate and/or sensitive mammalian species is used for determining the NOAEL, LOAEL, or the benchmark dose.” EPA, *Guidelines for Developmental Toxicity Risk Assessment*, December 1991, at 42, https://www.epa.gov/sites/production/files/2014-11/documents/dev_tox.pdf.

⁴⁶ 81 Fed. Reg. 91612

⁴⁷ 81 Fed. Reg. 91613

⁴⁸ *Id.*

EPA is simply wrong that its “representative endpoint” of immune effects “would address other identified risks.”⁴⁹ The Agency’s dose response analysis for the four acute endpoints it assessed is as follows:⁵⁰

2030 **Table 3-7: Dose-response analysis of selected studies considered for acute exposure scenarios**

| Target Organ/System | Species | Duration | POD Type ¹ (applied dose) | Effect | Dose Metric | HEC ₅₀ (ppm) | HEC ₉₉ (ppm) | HED ₅₀ (mg/kg) | HED ₉₉ (mg/kg) | Uncertainty Factors (UFs) ² | Reference | Data Quality ³ |
|-----------------------|-----------------|---|--|----------------------------|------------------|-------------------------|-------------------------|---------------------------|---------------------------|--|------------------------------|---------------------------|
| Developmental Effects | Rat (female) | Gestational days 6 to 15 | BMDL ₀₁ = 32.2 mg/kg-bw/day | Increased resorptions | TotMetab BW34 | 57 | 23 | 29 | 28 | UFS=1; UFA= 3; UFH=3; UFL=1; Total UF=10 | (Narotsky et al., 1995) | High (1.3) |
| | Rat (female) | 22 days throughout gestation (gestational days 0 to 22) | BMDL ₀₁ = 0.0207 mg/kg-bw/day | Congenital heart defects | TotOx Metab BW34 | 0.012 | 0.0037 | 0.0058 | 0.0052 | UFS=1; UFA= 3; UFH=3; UFL=1; Total UF=10 | (Johnson et al., 2003) | Medium (1.9) |
| | Rat (male pups) | Postnatal days 10 to 16 | LOAEL = 50 mg/kg-bw/day | Decreased rearing activity | TotMetab BW34 | 8 | 3 | 4.2 | 4.1 | UFS=1; UFA= 3; UFH=3; UFL=10; Total UF=100 | (Fredriksson et al., 1993) | Medium (1.7) |
| Immune System | Rat (female) | 3hr/day, single dose; followed by respiratory infection | BMDL ₀₁ = 13.9 ppm | Immuno-suppression | N/A ⁴ | N/A ⁴ | 1.74 ⁴ | N/A ⁴ | 2.74 ^{4,5} | UFS=1; UFA= 3; UFH=10; UFL=1; Total UF=30 | (Selgrade and Gilmour, 2010) | High (1.6) |

¹ POD type can be NOAEL, LOAEL, or BMDL. EPA adjusted all values to continuous exposure.
² UFS=subchronic to chronic UF; UFA=interspecies UF; UFH=intraspecies UF; UFL=LOAEL to NOAEL UF.
³ See [Data Quality Evaluation of Human Health Hazard Studies. Docket: EPA-HQ-OPPT-2019-0500] for full evaluation by metric.
⁴ Data from Selgrade and Gilmour, 2010) was not subject to PBPK modeling due to uncertainty concerning the most appropriate dose metric. The BMDL value adjusted for a 24hr exposure will be used as the POD for occupational risk estimates, while the 3hr value will be used for consumer risk estimates. This value is presented in the HEC₉₉ column but does not represent any particular percentile since it was not PBPK-modeled.
⁵ A dermal HED was obtained through route-to-route extrapolation using breathing rate and body weight data on male CD-1 mice (insufficient female data was reasonably available) from (U.S. EPA, 1988) and allometric scaling based on (U.S. EPA, 2011d) using a dosimetric adjustment factor of 0.14 for mice.

Thus, the acute HEC₉₉ for immune system effects is 470 times higher than the acute HEC₉₉ for heart malformations. This significant disparity translates into large differences in the acute MOEs for the two endpoints. For example, EPA calculated acute inhalation MOEs (high-end exposure/no PPE) for workers in batch open top vapor degreasing operations of .000014 for heart defects but 0.67 for immune effects.⁵¹ Both MOEs are far below the benchmark MOEs for these endpoints but the MOE for heart defects is over two orders of magnitude below the MOE for immune effects.⁵²

Accordingly, the large number of pregnant women exposed to TCE would be unprotected from fetal heart defects in their offspring by an exposure limit based only on immunotoxicity. This outcome would be directly contrary to EPA’s obligation in section 6(b)(4)(A) of TSCA to determine whether TCE “presents an unreasonable risk to a potentially exposed or susceptible subpopulation.”⁵³ Section 3(12) of TSCA states explicitly that such populations include “infants, children [and] pregnant women” yet EPA’s approach would deny them the special protection that TSCA requires.

⁴⁹ TCE Risk Evaluation at 377.

⁵⁰ Id at 252.

⁵¹ Id. at 545.

⁵² For chronic risks, the differences in the MOEs for the two endpoints are less dramatic but the MOE for heart defects is still generally an order of magnitude lower than the MOE for immune effects. Thus, the HEC₉₉ for chronic autoimmunity was 0.033 ppm as compared to 0.0037 ppm for heart malformations. Id. at 253.

⁵³ Once EPA identifies an unreasonable risk to a potentially exposed or susceptible subpopulation, EPA must take regulatory action under section 6(a) of TSCA “necessary so that the chemical no longer presents such risk.”

E. The White House-Dictated Comparison of the Relative Strength of the Evidence for Heart Defects and Immune Effects Is Misleading and Contrary to the Evaluation as a Whole

While TCE's immune effects are serious and should be included in the TCE evaluation, the claim (apparently added to the evaluation at White House direction) that the data supporting them are significantly more "certain" than the evidence of heart defects is incorrect and based on a selective and misleading comparison of the WOE for the two endpoints.

According to EPA, "the POD for mortality due to immunosuppression from (Selgrade and Gilmour, 2010) is considered to be the most robust and best representative POD for acute non- cancer scenarios."⁵⁴ EPA claims that:

"Considerations for selection of this study and the High confidence rating include the following:

- 1) The study scored a High in data quality evaluation
- 2) The study used a broad dose range, with several concentrations above and below the LOAEL
- 3) The response data followed a consistent dose-response curve
- 4) The data is based on an acute exposure study so there is no uncertainty resulting from extrapolating from a repeated-dose study
- 5) The study demonstrated multiple assays supporting the apical outcome e endpoint is severe"

However, several of these factors also apply to the heart defect database. Heart malformations are an extremely "severe" effect; the Johnson study used a "broad dose range"; the "dose response curve" in Johnson was clear and consistent; and while Johnson was a repeated dose study, EPA's longstanding policy is that a single exposure to a chemical within a critical window of fetal development can cause adverse effects.⁵⁵ Finally, the slightly different quality scores of the two studies – "medium" for Johnson and "high" for Selgrade and Gilmour (2010) – are unimportant compared to their strength in demonstrating adverse effects and the overall WOE supporting their findings.

Moreover, uncertainty factors (UF) for immune effects in the IRIS assessment and draft risk evaluation were actually *higher* than for the fetal heart malformations. In the TSCA evaluation, the UF for fetal heart defects based on Johnson et al was 10. However, for acute immunosuppression effects based on Selgrade, the UF was 30 "because the data was not subject to PBPK modeling and therefore a HEC99/HED99 value was not applied which would have accounted for human toxicokinetic variability."⁵⁶

⁵⁴ TCE Risk Evaluation, at 257.

⁵⁵ Thus, the EPA risk assessment guidelines for developmental toxicity state (at 38) that. "for developmental toxic effects, a primary assumption is that a single exposure at a critical time in development may produce an adverse developmental effect, i.e., repeated exposure is not a necessary prerequisite for developmental toxicity to be manifested. In most cases, however, the data available for developmental toxicity risk assessment are from studies using exposures over several days of development, and the NOAEL, LOAEL, and/or benchmark dose is most often based on a daily dose, e.g., mg/kg-day. Usually, the daily dose is not adjusted for duration of exposure because appropriate pharmacokinetic data are not available."

⁵⁶ Id. at 239. EPA also assigned a UF of 30 to the Keil et al study it relied on to determine the POD for chronic autoimmune effects (id at 245), lower than the IRIS UF of 100 but higher than the UF of 10 for the Johnson study.

IRIS also pointed to “notable uncertainty in the [BMR] modeling” for immune effects.⁵⁷ EPA expressed similar concerns about the Selgrade study in its draft evaluation, observing that a “reliable BMDL could not be obtained from the percentage infected data because BMDs and BMDLs from all models were well below the lowest data point and cannot be considered reliable.”⁵⁸

The draft evaluation underscores that the EPA scientists had *high confidence* in *all* the endpoints selected as PODs for calculating MOEs:⁵⁹

“There is high confidence in the database for human health hazard. All studies considered for dose-response analysis scored either Medium or High in data quality evaluation and were determined to be highly relevant to the pertinent health outcome. EPA selected the best representative study for each identified endpoint from among a broad selection of studies, taking into account factors such as data quality evaluation score, species, exposure duration, dose range, cumulative uncertainty factor, and relevance.”

These descriptions of the human health database are directly at odds with eleventh hour White House efforts to pick one “representative endpoint” and exclude others that are more sensitive. Since EPA scientists rejected any differentiation between the endpoints it chose as PODs and had “high confidence” in all of them, it is indefensible for the White House to now force EPA to conclude that the immune effects data provide sufficient “certainty” for a determination of unreasonable risk but the heart defect data do not.

In sum, the SACC should recommend that EPA revise the draft risk evaluation to use the heart defect data for addressing TCE’s acute and chronic risks to human health and, as the most sensitive endpoint, the key driver for determining whether TCE presents an unreasonable risk of injury under TSCA.

II. The Draft Evaluation Ignores Significant Environmental Releases of TCE That Present Serious Health Risks

Like previous evaluations, the EPA draft lacks any assessment of risks to the general population from TCE’s presence in air, water and soil. Few chemicals are as ubiquitous in the environment as TCE and, because of its many adverse health effects, its widespread distribution presents a significant threat to communities across the US. EPA’s failure to account for environmental pathways of exposure is a major shortcoming of its draft evaluation and results in a dramatic underestimate of the exposed population and the level of risk it faces.

In light of these higher UFs, EPA’s claim that it has greater confidence in in Keil et al because of reduced uncertainty (id. at 257) is not credible.

⁵⁷ IRIS Assessment at 5-22

⁵⁸ TCE Risk Evaluation at 238.

⁵⁹ Id at 254 (emphasis added). EPA also emphasized that “[t]here is high overall confidence in the database, weight of evidence, and dose-response for chronic non- cancer endpoints” and that “there is strong WOE in support of all health effects.” Id. at 257.

A. EPA's Exclusion of Environmental Releases Will Result in An Incomplete Risk Evaluation and Disregards Previous SACC Recommendations

As in other evaluations, EPA declined to address environmental releases of TCE because “those exposure pathways are covered under the jurisdiction of other environmental statutes, administered by EPA, which adequately assess and effectively manage exposures, i.e., CAA, SDWA, CWA, and RCRA.”⁶⁰ This exclusion defeats the central TSCA goal of providing a comprehensive picture of a chemical's risks to humans and the environment. Congress wanted EPA to examine the combined impact of all sources and pathways of exposure on affected populations and provided no exemption for environmental releases that might be subject to other environmental laws. Moreover, as TCE illustrates, other laws are not adequately addressing the contribution of air, soil and drinking water to total risk. If these pathways are ignored under TSCA, the result will likely be an incomplete understanding of TCE's risks and inadequate protection of health and the environment.

The SACC has repeatedly raised concerns about EPA's failure to consider environmental pathways of human exposure. Thus, in its review of the 1,4-dioxane draft risk evaluation, the SACC said:⁶¹

“Exposure scenarios that include consumers are important given the known presence of 1,4-Dioxane in plastics, other commercially available products, surface water, drinking water, groundwater, and in sediments. The Committee also had concerns that the omission of these multiple routes of exposure puts workers who inhale or ingest 1,4-Dioxane outside the workplace at even greater risk.”

The SACC added that:⁶²

“The Committee discussed that if each program office of the EPA says others are assessing the risks and thus not including them in their assessment, the U.S. public will be left with no overall IRIS assessment of risks. If risks have been assessed by other program offices of EPA then the Agency should present them as part of the underlying data to support this TSCA Evaluation—if not, the Agency must gather the data for an assessment or include an assessment based on the assumption of near-worst-case exposures.”

The SACC underscored that “[g]eneral human population and biota exposure must be assessed for inhalation, ingestion, and dermal routes [and that] [d]ifferent sub-populations may have different extents of exposure, but each route must be assessed.”⁶³ EPA's narrower approach, it said, “strayed from basic risk assessment principles by omitting well known exposure routes such as water

⁶⁰ TCE Risk Evaluation at 34.

⁶¹ 1,4-Dioxane and HBCD SACC Report, at 18.

⁶² Id.

⁶³ Id.

consumption by all occupationally and non-occupationally-exposed humans as well as similar exposures to other biological receptors.”⁶⁴

The SACC review of the 1-BP draft risk evaluation similarly took EPA to task for failing to consider air emissions and other environmental releases:⁶⁵

“The lack of consideration for general population exposures excludes a vast extent of the US population (workers, consumers, school children, and other populations) who are exposed to 1-BP, perhaps on a daily basis. The lack of consideration of the general population exposure is concerning given the strong evidence of widespread exposure to a chemical that may be 1-BP based (from biomonitoring data).”

The SACC report for the methylene chloride evaluation raised similar concerns:⁶⁶

“Several Committee members expressed concern that large quantities of methylene chloride are volatilized to ambient air from diverse and disperse uses and that there is no COU that provides a basis for setting any limit on these emissions. While EPA asserts that the Clean Air Act (CAA) can be used to control these emissions, Committee members thought the CAA would address only a fraction of total emissions, i.e. only from Major Sources as defined by the 1990 CAA Amendments.”

The Report added that:⁶⁷

“Concern was expressed that many of the methylene chloride releases to the environment are unaccounted for, and the Committee recommended EPA consider using a mass-balance approach to match amount manufactured/imported with amounts used in products, recycled or disposed, and released to the environment. . . . Discharges to air, ground water, soils and sediments are not considered.”

The SACC expressed concern that “readers of this Evaluation receive a partial picture of risks, finding for example, that recycling and proper disposal present the only environmental hazards under TSCA” and that “this incomplete picture of risks may be used to promote improper releases and disposal of methylene chloride.”⁶⁸

For TCE, like several other chemicals EPA is evaluating, the exclusion of environmental release pathways is not merely a theoretical concern. There is considerable evidence of TCE’s ubiquitous presence in air,

⁶⁴ Id.

⁶⁵ SACC 1-BP Report at 17.

⁶⁶ SACC Methylene Chloride Report at 75.

⁶⁷ Id at 15.

⁶⁸ Id.

soil and drinking water at levels that likely harm human health and contribute to ozone depletion and climate change.

B. Air Emissions of TCE Are Substantial and Are Harmful to Human Health

Like other halogenated solvents, TCE is highly volatile at ambient temperatures and, according to ATSDR, most of the TCE “used in the United States is released into the atmosphere by evaporation.”⁶⁹ Toxic Release Inventory (TRI) reporting indicates that 1,886,809 pounds (855.8 metric tons) of TCE were released to the atmosphere from 154 domestic manufacturing and processing facilities in 2017.⁷⁰ TRI requirements apply to a narrow subset of facilities that release chemicals to the environment and thus understate total TCE emissions. The 2011 EPA National Emission Inventory (NEI) estimated US TCE emissions of 3,250 tons – or 7,150,000 pounds.⁷¹

TCE has been detected in the air throughout the United States. Atmospheric levels are highest in areas concentrated with industry and population, and lower in remote and rural regions.⁷² According to IRIS, “[t]he most recent data (2006) come from 258 monitors located in 37 states. The means for these monitors range from 0.03 to 7.73 $\mu\text{g}/\text{m}^3$ and have an overall average of 0.23 $\mu\text{g}/\text{m}^3$.”⁷³ As IRIS has summarized the data:

Table 2-6. TCE ambient air monitoring data ($\mu\text{g}/\text{m}^3$)

| Yr | Number of monitors | Number of states | Mean | Standard deviation | Median | Range |
|------|--------------------|------------------|------|--------------------|--------|------------|
| 1999 | 162 | 20 | 0.30 | 0.53 | 0.16 | 0.01–4.38 |
| 2000 | 187 | 28 | 0.34 | 0.75 | 0.16 | 0.01–7.39 |
| 2001 | 204 | 31 | 0.25 | 0.92 | 0.13 | 0.01–12.90 |
| 2002 | 259 | 41 | 0.37 | 1.26 | 0.13 | 0.01–18.44 |
| 2003 | 248 | 41 | 0.35 | 0.64 | 0.16 | 0.02–6.92 |
| 2004 | 256 | 37 | 0.32 | 0.75 | 0.13 | 0.00–5.78 |
| 2005 | 313 | 38 | 0.43 | 1.05 | 0.14 | 0.00–6.64 |
| 2006 | 258 | 37 | 0.23 | 0.55 | 0.13 | 0.03–7.73 |

Source: EPA’s Air Quality System database at the AirData Web site:

<http://www.epa.gov/air/data/index.html>.

⁶⁹ ATSDR, *Toxicological Profile for Trichloroethylene* June 2019 (ToxProfile) at 305, <file:///C:/Users/Owner/Downloads/ATSDR%20TCE.pdf>.

⁷⁰ Id at 307.

⁷¹ EPA, Technology transfer network. Clearinghouse for lower in remote and rural regions. Inventories & emissions factors. National Emissions Inventory (NEI) air pollutant emissions trends data. U.S. Environmental Protection Agency. <http://www3.epa.gov/ttnchie1/trends/>. December 7, 2015.

⁷² IRIS Assessment at 2-6/2-7.

⁷³ Id at 2.8.

Table 2-7. Mean TCE air levels across monitors by land setting and use (1985–1998)

| | Rural | Suburban | Urban | Agricultural | Commercial | Forest | Industrial | Mobile | Residential |
|---|-------|----------|-------|--------------|------------|--------|------------|--------|-------------|
| Mean concentration ($\mu\text{g}/\text{m}^3$) | 0.42 | 1.26 | 1.61 | 1.08 | 1.84 | 0.1 | 1.54 | 1.5 | 0.89 |
| N | 93 | 500 | 558 | 31 | 430 | 17 | 186 | 39 | 450 |

Source: EPA’s Air Quality System database at the AirData Web site:

<http://www.epa.gov/air/data/index.html>.

These ambient levels are of health concern based on EPA’s assessment of TCE’s health effects. For example, IRIS has determined the following cancer risk levels (70 year lifetime exposure) for different TCE ambient air concentrations:⁷⁴

| | |
|----------------------|------------------------------|
| E-4 (1 in 10,000) | 20 $\mu\text{g}/\text{m}^3$ |
| E-5 (1 in 100,000) | 2 $\mu\text{g}/\text{m}^3$ |
| E-6 (1 in 1,000,000) | 0.2 $\mu\text{g}/\text{m}^3$ |

Thus, mean TCE levels in ambient air for all locations except forests would present lifetime cancer risks above 1 in 1 million, EPA’s benchmark for determining unreasonable cancer risks for non-worker population. Risks for higher levels within the range measured would exceed 1 in 100,000.

Similarly, mean ambient air levels in most locations (which range between 0.89 and 1.6 $\mu\text{g}/\text{m}^3$) would be very close to the IRIS non-cancer RfC of 0.0004 ppm (0.4 ppb or 2 $\mu\text{g}/\text{m}^3$), which IRIS describes as having “robust support [from] . . . estimates for multiple effects from multiple studies.”⁷⁵ For individuals exposed to ambient TCE levels near the higher end of the reported range, the RfC would be exceeded.

Thus, large segments of the US population are likely exposed to TCE levels in air that present unreasonable risks of cancer and non-cancer effects.

C. Indoor Air Levels of TCE are Significantly Greater than Ambient Levels and Pose Greater Risks

According to IRIS, “TCE can be released to indoor air from use of consumer products that contain it (i.e.,

⁷⁴ IRIS Chemical Assessment Summary for TCE (IRIS Summary) at 44, https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0199_summary.pdf#nameddest=rfd.

⁷⁵ IRIS Assessment at 5-97. The IRIS RfC is similarly to the risk determination methodology EPA’s draft evaluation uses for fetal heart defects. The chronic HED₉₉ for these effects is 0.0037 ppm which, when reduced to reflect EPA’s UF of 10, would result in a concentration very close to the RfD. TCE Risk Evaluation at 280

adhesives and tapes), vapor intrusion (migration of volatile chemicals from the subsurface into overlying buildings) and volatilization from the water supply.”⁷⁶ Consistently, measured indoor levels have been shown to be higher than outdoor levels. IRIS summarizes a number of key studies as follows:⁷⁷

- The 1987 EPA Total Exposure Assessment Methodology study ([Wallace, 1987](#)) showed that the ratio of indoor to outdoor TCE concentrations for residences in Greensboro, NC, was about 5:1.
- In two homes using well water with TCE levels averaging 22–128 µg/L, the TCE levels in bathroom air ranged from <500–40,000 µg/m³ when the shower ran <30 minutes ([Andelman, 1985](#)).
- Shah and Singh ([1988](#)) report an average indoor level of 7.2 µg/m³ based on over 2,000 measurements made in residences and workplaces during 1981–1984 from various locations across the United States.
- Hers et al. ([2001](#)) provides a summary of indoor air TCE measurements at locations in United States, Canada, and Europe with a range of <1–165 µg/m³.
- Sapkota et al. ([2005](#)) measured TCE levels inside and outside of the Baltimore Harbor Tunnel toll booths during the summer of 2001. Mean TCE levels were 3.11 µg/m³ indoors and 0.08 µg/m³ outdoors based on measurements on 7 days. The authors speculated that indoor sources, possibly dry cleaning residues on uniforms, were the primary source of the indoor TCE.
- Sexton et al. ([2005](#)) measured TCE levels inside and outside residences in Minneapolis/St. Paul metropolitan area. Two day samples were collected over three seasons in 1999. Mean TCE levels were 0.5 µg/m³ indoors (*n* = 292), 0.2 µg/m³ outdoors (*n* = 132) and 1.0 µg/m³ based on personal sampling (*n* = 288).
- Zhu et al. ([2005](#)) measured TCE levels inside and outside of residences in Ottawa, Canada. Seventy-five homes were randomly selected and measurements were made during the winter of 2002/2003. TCE was above detection limits in the indoor air of 33% of the residences and in the outdoor air of 19% of the residences. The mean levels were 0.06 µg/m³ indoors and 0.08 µg/m³ outdoors. Given the high frequency of nondetects, a more meaningful comparison can be made on basis of the 75th percentiles: 0.08 µg/m³ indoors and 0.01 µg/m³ outdoors.

These reported levels would in most cases exceed a 1 in 1 million cancer risk and, at the higher end of the reported range, would exceed the IRIS RfC as well.

The contribution to TCE indoor levels of volatilization of contaminated drinking water is well-documented. According to ATSDR, “In two homes (using well water containing the relatively high level of 40,000 ppb trichloroethylene), a running shower was found to elevate trichloroethylene levels in bathroom air from <0.5 to 81 mg/m³ (93–15,072 ppb) in <30 minutes (Andelman 1985a).”⁷⁸ ATSDR also reports that “[t]he transfer of trichloroethylene from shower water to air in one study had a mean efficiency of 61%, which was independent of water temperature (McKone and Knezovich 1991) [and] the study authors concluded that showering for 10 minutes in water contaminated with

⁷⁶ IRIS Assessment at 2-10.

⁷⁷ Id.

⁷⁸ ToxProfile at 335.

trichloroethylene could result in a daily exposure by inhalation comparable to that expected by drinking contaminated tap water.”⁷⁹

Although the draft risk evaluation examines exposure levels for specific TCE-containing consumer products, it does not look more broadly at indoor TCE air concentrations to which consumers are exposed, and as a result, overlooks the combined contributions to exposure of product use and other indoor exposure pathways like volatilization of TCE from contaminated water and intrusion of TCE vapors from contaminated soil and groundwater. Thus, it underestimates TCE risks in the indoor environment. Equally important, EPA’s risk evaluation assumes that consumers only have acute exposure to TCE. However, the evidence of ongoing TCE concentrations in indoor air indicates that chronic exposure is also occurring and therefore consumers are at risk for cancer and other chronic health effects that EPA fails to address.

D. TCE Is Pervasive in Surface Water, Groundwater and Drinking Water at Levels of Health Concern

IRIS describes the presence of TCE in surface water as follows:⁸⁰

“According to IARC ([1995a](#)), the reported median concentrations of TCE in 1983–1984 were 0.5 µg/L in industrial effluents and 0.1 µg/L in ambient water. Results from an analysis of the EPA STORET Data Base (1980–1982) showed that TCE was detected in 28% of 9,295 surface water reporting stations nationwide ([ATSDR, 1997c](#)). A more recent search of the STORET database for TCE measurements nationwide during 2008 in streams, rivers and lakes indicated three detects (0.03–0.04 µg/L) out of 150 samples (STORET Database, <http://www.epa.gov/storet/dbtop.html>).”

According to ATSDR, “[a] summary of U.S. groundwater analyses from both federal and state studies reported that trichloroethylene was the most frequently detected organic solvent and the one present in the highest concentration.”⁸¹ As ATSDR notes, TCE “was detected in 388 of 669 groundwater samples collected in New Jersey from 1977 to 1979, with a maximum concentration of 635 ppb . . . Maximum concentrations ranging from 900 to 27,300 ppb trichloroethylene were found in contaminated wells from four states (Pennsylvania, New York, Massachusetts, and New Jersey).”⁸²

In light of the widespread presence of TCE in groundwater, it is not surprising that TCE is a common contaminant in drinking water. According to IRIS, “[i]t has been estimated that between 9 and 34% of

⁷⁹ Id. at 342.

⁸⁰ IRIS Assessment at 2-12.

⁸¹ ToxProfile at 330. The draft risk evaluation describes surface water monitoring data for 2013-2017 from STORET at 93-94. The average detection frequency for this period was 3.04% and the average TCE concentration was 0.33 µg/L.

⁸² Id.

the drinking water supply sources tested in the United States may have some TCE contamination.”⁸³ As ATSDR describes, drinking water monitoring conducted by or for EPA has consistently detected TCE in public water systems (PWSs) across the US.⁸⁴

“The EPA (2011d) released the results of its second 6-year review of 69 regulated contaminants in public water systems (PWS) located across the United States. . . . During 2005, trichloroethylene was detected in 2,292 out of 46,937 samples (4.9%) collected from groundwater supplied PWS and 1,874 out of 12,705 samples (14.8%) collected from surface water supplied PWS. The median, 95th percentile, and maximum concentrations of the positive samples were 1.1, 13.0, and 159 ppb, respectively, in groundwater supplied PWS and 1.6, 28.0, and 50.0 ppb, respectively, in the surface water supplied PWS. . . . The EPA Groundwater Supply Survey of finished water from 945 drinking water systems nationwide using groundwater sources found trichloroethylene in 91 water systems (detection limit 0.2 ppb); the median level of the positive samples was approximately 1 µg/L (ppb), with a single maximum level of 130 µg/L (ppb) (Westrick et al. 1984).”

ATSDR reports similar findings in other studies:⁸⁵

“Williams et al. (2002) reported annual levels of trichloroethylene measured in 3,447–4,226 California drinking water sources between 1995 and 2001. Trichloroethylene was detected in 9.6–11.7% of the sources over the time period with an average detected concentration ranging from 14.2 to 20.7 µg/L (ppb). . . . Drinking water supplies at Camp Lejeune have been shown to be heavily contaminated with trichloroethylene and other chlorinated solvents due to handling and disposal practices of an off-site dry cleaning facility (ATSDR 2017b). Water samples obtained from the Hadnot Point Water Treatment plant at Camp Lejeune had levels of trichloroethylene of up to 1,400 µg/L in 1982 (ATSDR 2017b).”

In 1987, EPA set a National Primary Drinking Water Regulation (NPDWR) for TCE which establishes a maximum contaminant level goal (MCLG) of zero and an enforceable maximum contaminant level (MCL) of 5 ug/L (5 ppb).⁸⁶ Based on the monitoring data presented above, exceedances of the MCL (in some cases by an order of magnitude or more) have been recorded in several PWSs. Moreover, the current MCL is not health protective in light of current science. The IRIS assessment for TCE determines that drinking water exposures over a lifetime to 0.5 ug/L – a tenth of the MCL – pose a cancer risk of 1 in a million.⁸⁷ Similarly, the IRIS non-cancer RfD is 0.0005 mg/kg/day (0.5 ug/L or 0.5 ppb).⁸⁸

⁸³ IRIS Assessment at 2-12.

⁸⁴ ToxProfile at 328.

⁸⁵ Id.

⁸⁶ 52 Federal Register 25690 (July 8, 1987).

⁸⁷ IRIS Summary at 39

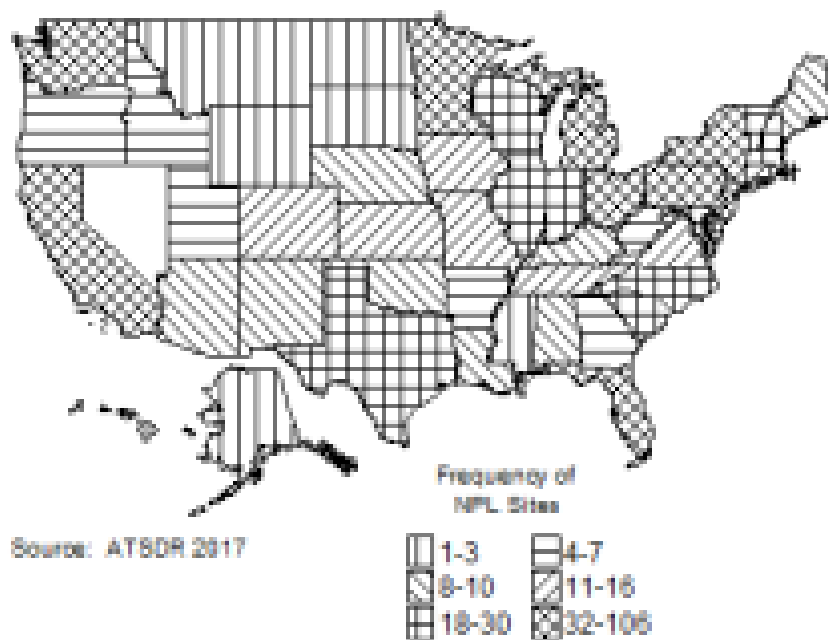
⁸⁸ IRIS Assessment at 5-101.

Based on EPA-mandated drinking water monitoring, the Environmental Working Group (EWG) has determined that 149 PWSs in 30 states have detected TCE levels in drinking water above health guidelines and that these utilities serve 2.6 million people.⁸⁹ **Cancer and non-cancer risks to this subpopulation exceed EPA benchmarks for unreasonable risk, even without considering the volatilization of household water during showering and other daily activities and resulting in TCE inhalation exposure.**

E. TCE Is Frequently Found at Contaminated Sites, Resulting in Contamination of Groundwater and Release of TCE Vapors into Ambient Air and Buildings

TCE is a significant concern at contaminated sites within the purview of the EPA Superfund program. ATSDR reports that TCE “has been identified in at least 1,051 of the 1,854 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL).”⁹⁰ ATSDR depicts the geographic distribution of these sites as follows:

Frequency of NPL Sites with Trichloroethylene Contamination



Volatilization of TCE from contaminated soils is relatively rapid and may lead to elevated ambient air levels in communities near NPL sites. ATSDR notes that “[r]elease of trichloroethylene also occurs at treatment and disposal sites,” including “through volatilization and air-stripping procedures” at water treatment facilities and “gaseous emissions from landfills.”⁹¹ According to ATSDR, TCE’s mobility in soil is

⁸⁹ <https://www.ewg.org/tapwater/contaminant.php?contamcode=2984>. EWG used a health guideline of 0.4 ppb for TCE, which the state of Minnesota has set as a health risk limit.

⁹⁰ ToxProfile at 305.

⁹¹ Id. at 314.

well-documented,⁹² and it readily leaches to the subsurface and to groundwater. The presence of TCE in leachate from active and inactive landfills is considered an important pathway for groundwater contamination and is linked to TCE-contaminated groundwater at many NPL sites.⁹³

TCE vapor intrusion is a serious concern at contaminated sites near residences or commercial buildings. As described by the State of Minnesota:⁹⁴

“TCE can evaporate from the polluted soil and groundwater and rise toward the ground surface. If these TCE vapors come to a basement as they travel to the surface, they may enter through cracks in the foundation, around pipes, or through a sump or drain system. In this way, the vapors enter buildings and contaminate indoor air. This process, when pollution moves from air spaces in soil to indoor air, is called vapor intrusion.”

ATSDR describes vapor intrusion as a “notable exposure route” and cites several studies which attributed elevated TCE indoor air levels to vapor intrusion from TCE-contaminated cleanup sites or groundwater.⁹⁵

F. By Failing to Account for Environmental Pathways, the Draft Evaluation Disregards Large at Risk Subpopulations and Greatly Understates Risks to Workers and Users of Consumer Products

This brief survey of TCE releases to air, water and soil demonstrates the important contribution of TCE air emissions and contaminated groundwater, drinking water and soil to overall TCE exposure. Each of these pathways is alone responsible for cancer and non-cancer risks to large segments of the population that exceed EPA benchmarks. Moreover, some subpopulations are exposed by multiple pathways simultaneously – i.e. individuals who breath TCE in indoor and outdoor air, consume contaminated drinking water and live near TCE-contaminated NPL sites. Because TCE exposure levels are higher for these subpopulations than the general population, they face elevated risks of TCE-related health effects like cancer, fetal heart malformations and immunotoxicity. A comprehensive risk evaluation as required by TSCA would identify and quantify these subpopulations, estimate total exposure from all sources and characterize the increased risk resulting from concurrent exposure pathways. However, because of its narrow scope, the draft TCE evaluation fails to provide this analysis and therefore presents a limited and incomplete picture of TCE’s risks to the public.

The draft evaluation even understates risks to the population groups – workers and users of consumer products – that it does address. These groups also are exposed to TCE in air, water and soil in addition to the pathways that EPA addresses. For example, workers in vapor degreasing operations may live in

⁹² Id. at 317

⁹³ Id. at 330.

⁹⁴ <https://www.health.state.mn.us/communities/environment/hazardous/topics/tce.html>.

⁹⁵ ToxProfile. at 327, 341.

industrialized areas with high ambient air levels and one or more Superfund sites and consume TCE-contaminated drinking water. In the aggregate, TCE exposure by these workers would be significantly greater than exposure in the workplace alone and health risks (which are already alarmingly high for worker activities) would be correspondingly higher. This would likewise be true of users of consumer products who have concurrent exposure to TCE air emissions, contaminated drinking water and elevated indoor air levels due to vapor intrusion. EPA's MOEs for consumer product use (while themselves significantly below benchmark MOEs) would be reduced further if other contributors to consumer exposure are taken into account. Moreover, since exposure to TCE in ambient air and contaminated drinking water is continuous, EPA could not limit its evaluation to acute risks to consumers, as it does in its draft evaluation. Instead, it would need to address long-term exposure scenarios and determine risks for chronic endpoints like cancer, liver and kidney toxicity, and developmental and immunotoxicity related to repeated dose exposure.⁹⁶

EPA's claim that other programs are effectively protecting against TCE environmental releases and obviate the need to evaluate them under TSCA is a red herring. In fact, the EPA media-specific programs responsible for air, water and waste are not examining TCE's cross-media risks and could not do so since they lack authority over multiple environmental pathways. Moreover, distracted by other priorities, these programs are in many cases not even effectively addressing TCE risks within their areas of responsibility. For example, the TCE drinking water MCL is over 30 years old but there are no plans to update it in light of the many TCE health concerns that have come to light in the intervening years.

TSCA is the only law administered by EPA that provides comprehensive authority to examine chemical risks across all pathways of exposure. It is clear that Congress viewed this unique strength of TSCA as an essential tool in protecting against the cross-media effects of chemicals like TCE on human health and the environment. The SACC should recommend that EPA revise the draft TCE evaluation so it accounts for all sources of exposure and risk and provides a complete understanding of how TCE endangers public health.

III. EPA's Unreasonable Risk Determinations for Workers Should Not Assume They Will be Protected by PPE

As in previous risk evaluations, EPA's risk determinations for workers exposed to TCE calculate MOEs assuming both the use of respirators and gloves and the absence of protective equipment. Even for scenarios where workers consistently and reliably use PPE, EPA concludes that MOEs are below "benchmarks" for all conditions of use and that these conditions present unreasonable risks of injury to workers. However, while unacceptably low even with PPE use, EPA's MOEs are significantly lower for "no PPE" scenarios. For example, for batch open top vapor degreasing operations, the "no PPE" acute inhalation MOE for fetal heart defects (high-end exposure) is 1.4E-04 but 7.1E-03 assuming use of

⁹⁶ Even without considering these sources of exposure, the draft evaluation understates risks to consumers by failing to account for concurrent use of multiple consumer products, repeated consumer product use over time and exposure by dermal and inhalation routes simultaneously.

respirators with an APF of 50. Similarly, for the “no PPE” scenario, the lifetime cancer risk for this condition of use is 0.20 but 4.0E-03 for the respirator (APF = 50) scenario.⁹⁷

Thus, how much risk workers currently face – and how much risk reduction is necessary to fully protect them under TSCA section 6 – depend on whether PPE are now in widespread use and effectively controlling exposure. However, as the SAAC has repeatedly underscored and EPA’s draft evaluations recognize, an expectation of universal PPE use is not supported by evidence and is in fact contrary to the realities of workplace practice and sound principles of worker protection. For this reason, the “no PPE” scenario is the only defensible baseline for determining current risk levels for exposed workers and then defining the additional worker protections necessary to eliminate unreasonable risk. **SACC should recommend that the final TCE evaluation base determinations of risk solely on the “no PPE” scenario.**

A. The SACC Has Repeatedly Raised Serious Concerns About EPA’s Undue Reliance on PPE to Determine the Absence of Unreasonable Risk

In each of its reviews of draft evaluations, the SACC has repeatedly raised concerns about EPA’s undue reliance on PPE for determinations of unreasonable risk. In its report on the PV29 draft, the SACC noted that “the analysis in the Evaluation does not discuss or account for the fact that downstream commercial users may be oblivious to chemical risks and lack even rudimentary industrial hygiene measures.”⁹⁸ Similarly, in reviewing the 1,4-dioxane evaluation, the SACC concluded that the “consensus of the Committee believes that PPE may not be consistently and properly worn, as EPA assumed”⁹⁹ and noted that “[g]love use should not always be assumed to be protective” and, if worn improperly, gloves “could actually lead to higher exposures.”¹⁰⁰ As it concluded, “8-hour use of PPE should not be used in the risk characterization of inhaled 1,4-Dioxane. Risk estimates should be presented without the use of PPE as reasonable worst case.”¹⁰¹

In the case of HBCD, the SACC noted that “it was unreasonable to assume workers would wear PPE for entire 8-hour shifts due to underlying medical conditions, facial hair, discomfort, and other issues” and added that:¹⁰²

“[M]any members of the Committee believed EPA should place more emphasis on the limited likelihood that respiratory protection will be adopted without specific occupational exposure guidelines for HBCD . . . Dust exposures in the construction trades (especially residential

⁹⁷ TCE Risk Evaluation at 286.

⁹⁸ SACC Report on PV29 at 37.

⁹⁹ These “heightened exposures” could occur as a result of “contamination of the interior of the glove” (if workers were not properly trained in glove use and replacement) or by “acting as a reservoir” for contaminants (if the gloves were not impermeable). Such occlusion (greater penetration of the skin where contaminants build up inside the glove because it is permeable) would result in *greater* dermal exposure than in the “no glove” scenario.

¹⁰⁰ SACC Report on 1,4-dioxane and HBCD, at 55.

¹⁰¹ *Id.* at 53.

¹⁰² *Id.* at 118.

construction) continue to represent an occupational health concern because of the many small-to-medium size operators and the use of temporary (and, not infrequently, undocumented) workers. Workers in these small-to-medium enterprises may not be likely to adopt personal protective equipment (PPE) controls, so EPA's characterization of reasonable risk relying on use of PPE is not sufficiently supported by the practical realities of many workplaces."

The SACC report on 1-BP provides further amplification of these concerns:¹⁰³

"One member noted that the Committee has now received public testimony from two former highly distinguished Occupational Safety and Health Administration (OSHA) administrators expressing concerns regarding EPA's reliance upon non-regulatory guidance and PPE to reduce risks to reasonable levels. Persons familiar with PPE use realize that nominal protection factors may not be achieved in actual practice. The most recent of these comments also noted that compounds with high vapor pressures (such as 1-BP) may "breakthrough" cartridge type respirators in time frames much shorter than a work shift. Since respirators do not have real-time indicators of remaining capacity, respiratory protection failure is more likely for high vapor pressure compounds. 1-Bromopropane also is known to penetrate many glove types. This increases the likelihood of failure to select an appropriate glove."

The SACC concluded that EPA "[a]ssumptions about PPE use are likely unrealistic for many of the scenarios and so the determination of whether a condition of use results in an acceptable or unacceptable risk should be based on no PPE use, with the possible exception of in a manufacturing facility."¹⁰⁴

The SACC report on the methylene chloride risk evaluation reinforced these points, stating that "[m]ost Committee members agreed that EPA's assumptions of PPE use likely do not reflect actual conditions in most workplaces."¹⁰⁵ SACC added that:¹⁰⁶

"The Agency's reliance on appropriate use of personal protective equipment (PPE), including both respirators and gloves, is not supported by current research literature or industrial hygiene practice. The mere presence of a regulation requiring respirators does not mean that they are used or used effectively. Inadequacies in respirator programs are documented. Respirators require multiple respiratory protection (RP) compliance factors in order to perform as certified. Brent et al. (2005) used data from the NIOSH and Bureau of Labor Statistics (BLS) joint survey on Respirator Usage in Private Sector Firms, (BLS, 2001) to examine the adequacy of respirator protection programs in private industries. They found "large percentages of establishments requiring respirator use [under OSHA or the Mine Safety and Health Administration (MSHA)

¹⁰³ SACC Report on 1-BP, at 30-31.

¹⁰⁴ Id at 66.

¹⁰⁵ SACC Report on methylene chloride, at 17.

¹⁰⁶ Id at 36.

regulations] had indicators of potentially inadequate respirator programs.” Later, Janssen et al. (2014) reported that ‘APFs do not apply to RPD used in the absence of a fully compliant RP program; less than the expected level of protection is anticipated in these situations.’ Moving beyond program elements, the frequency of proper use of gloves and respirators is largely unknown.”

B. There is Compelling Evidence that TCE-Exposed Workers are not Meaningfully Protected by PPE

Most worker exposure to TCE is in small, poorly controlled operations. For example, nearly all vapor degreasing occurs in “open-top” degreasers, estimated by EPA to number between 2,600 and 6,000. Batch systems with enclosed or closed-loop operations are considerably less common, numbering around 120 according to EPA. EPA estimates that there are 150 in-line systems currently using TCE. The Agency projects that there are approximately 40,800 to 102,000 persons (workers and occupational bystanders) exposed to TCE from open-top degreasing operations, and an additional 2,040 and 2,550 persons exposed from closed-loop and in-line systems, respectively.¹⁰⁷

The current OSHA time-weighted average 8-hour Permissible Exposure Limit (PEL) for TCE is 100 parts per million (ppm), three orders of magnitude higher than the level that current TCE health effects data warrant. The PEL was adopted in 1971 and has never been updated. OSHA has no plans to revise the TCE PEL. In the absence of a meaningful OSHA limit on workplace exposure, it is inconceivable that OSHA is enforcing – or employers are systematically implementing – the stringent PPE requirements that would be necessary for the substantial reductions in worker exposure necessary to achieve safe levels.

Moreover, consistent PPE use requires effective warnings and product labels but, in its proposal to ban vapor degreasing, EPA concluded that worker comprehension of warnings and labels would be poor:¹⁰⁸

“EPA found that presenting information about TCE on a label would not adequately address the identified unreasonable risks because the nature of the information the user would need to read, understand, and act upon is extremely complex. It would be challenging to most users to follow or convey the complex product label instructions required to explain how to reduce exposures to the extremely low levels needed to minimize the risk from TCE. Rather than a simple message, the label would need to explain a variety of inter-related factors, including but not limited to the use of local exhaust ventilation, respirators and assigned protection factor for the user and bystanders, and time periods during pregnancy with susceptibility of the developing fetus to acute developmental effects, as well as effects to bystanders. *It is unlikely that label language changes for this use will result in widespread, consistent, and successful adoption of risk reduction measures by users and owners.*”

¹⁰⁷ 82 Fed. Reg. 7442.

¹⁰⁸ 82 Fed. Reg. 7441. (emphasis added)

These conclusions are particularly compelling in light of the nature of the TCE-exposed worker population. Many TCE-using operations are small shops that lack effective worker training and hazard communication programs. Their employees may be part-time and/or short duration workers who are unlikely to study product warnings and labeling (and may not even understand English). Occupational bystanders – a group at serious risk from TCE use – may not even come into contact with warnings and labels because they are not handling TCE directly.

EPA's TCE degreasing proposal also concluded that respirators could not be relied upon to protect TCE-exposed workers because "there are many documented limitations to successful implementation." As EPA elaborated:¹⁰⁹

"Not all workers can wear respirators. Individuals with impaired lung function, due to asthma, emphysema, or chronic obstructive pulmonary disease for example, may be physically unable to wear a respirator. Determination of adequate fit and annual fit testing is required for a tight fitting full-face piece respirator to provide the required protection. Also, difficulties associated with selection, fit, and use often render them ineffective in actual application, preventing the assurance of consistent and reliable protection, regardless of the assigned capabilities of the respirator. Individuals who cannot get a good face piece fit, including those individuals whose beards or sideburns interfere with the face piece seal, would be unable to wear tight fitting respirators. In addition, respirators may also present communication problems, vision problems, worker fatigue and reduced work efficiency (63 FR 1156, January 8, 1998). According to OSHA, 'improperly selected respirators may afford no protection at all (for example, use of a dust mask against airborne vapors), may be so uncomfortable as to be intolerable to the wearer, or may hinder vision, communication, hearing, or movement and thus pose a risk to the wearer's safety or health. (63 FR 1189-1190).'"

Adding to these limitations is the difficulty of implementing an effective respirator program in the small establishments where much TCE use and exposure occur. The OSHA respiratory protection standard (29 CFR 1910.134) contains numerous elements, *e.g.*, for program administration; worksite-specific procedures; respirator selection; employee training; fit testing; medical evaluation; and respirator cleaning, maintenance, and repair. These requirements would be beyond the resources or expertise of, say, a small machine shop or metal plater, which would likely lack any previous experience with respirator programs. The difficulty of compliance would be magnified by the nature of the workforce in these shops, which is likely to have high turnover and many part-time employees with little or no industrial hygiene sophistication. Training these workers to use respirators conscientiously would be a huge challenge. And given the number and nature of the businesses involved, meaningful oversight by OSHA would likely be non-existent.

¹⁰⁹ 82 Fed. Reg. 7445

The draft TCE risk evaluation explains the well-established “hierarchy of controls” for protecting workers as follows:¹¹⁰

“OSHA and NIOSH recommend that employers utilize the hierarchy of controls to address hazardous exposures in the workplace. The hierarchy of controls strategy outlines, in descending order of priority, the use of elimination, substitution, engineering controls, administrative controls, and lastly personal protective equipment (PPE). The hierarchy of controls prioritizes the most effective measures first which is to eliminate or substitute the harmful chemical (e.g., use a different process, substitute with a less hazardous material), thereby preventing or reducing exposure potential. Following elimination and substitution, the hierarchy recommends engineering controls to isolate employees from the hazard, followed by administrative controls, or changes in work practices to reduce exposure potential (e.g., source enclosure, local exhaust ventilation systems) . . . As the last means of control, the use of personal protective equipment (e.g., respirators, gloves) is recommended, when the other control cannot reduce workplace exposure to an acceptable level.”

Consistent with the hierarchy of controls and SACC recommendations, EPA’s risk determinations for TCE should assume no PPE use. How to then eliminate TCE’s unreasonable risks to workers should be decided in the later TSCA risk management phase and PPE should be considered as a last resort, only after other means of control such as engineering controls have been shown to be inadequate.

Conclusion

We appreciate this opportunity to comment to the SACC on the draft TCE risk evaluation. Please contact SCHF counsel Bob Sussman with any questions at bobsussman1@comcast.net.

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¹¹⁰ TCE Risk Evaluation at 119.

