Environmental Defense Fund
Comments on the Toxic Substances Control Act (TSCA)
Draft Risk Evaluation of Trichloroethylene
Docket ID: EPA-HQ-OPPT-2019-0500

Submitted April 27, 2020

Environmental Defense Fund (EDF) appreciates the opportunity to provide comments on the Environmental Protection Agency's (EPA) draft risk evaluation for trichloroethylene (TCE) being prepared under section 6(b)(4) of the Toxic Substances Control Act (TSCA) as amended by the Lautenberg Act, enacted on June 22, 2016.[1]

As a result of EPA's decision not to respond to the comments provided on the TCE problem formulation, EDF's comments are submitted in two parts. Part I addresses comments that have been developed since the draft risk evaluation was published, and Part II includes comments that were provided on the problem formulation[2] but that remain an issue in the draft risk evaluation. In a few instances EDF's comments are repeated in both Part I and Part II; this is a function of EPA's failure to respond to the comments initially provided on the problem formulation.

Summary[3]

In this draft risk evaluation, EPA has grossly understated the risks from exposure to TCE. EPA did more frequently identify unreasonable risks than in draft risk evaluations for other chemicals released in the past year, making the deficiencies harder to readily discern. EPA has employed a host of unwarranted and unsupported assumptions and methodological approaches that lead it to either avoid identifying unreasonable risk when it should have, or to understate the extent and magnitude of the unreasonable risks it did identify. These assumptions and approaches are not mere policy choices; they have serious scientific and public health consequences (see section

[1] U.S. EPA, *Draft Risk Evaluation for Trichloroethylene CASRN: 79-01-6* (February 2020), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0500-0002. Further citations in these comments of the draft risk evaluation consist of only a page number in parentheses.
[2] EDF's comments on the TCE Problem Formulation are herein incorporated by reference available at https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0737-0100.
[3] Section references in this summary are to Part I of these comments unless otherwise noted.

1.C. of these comments for more discussion of this issue). Below we summarize some of the major concerns addressed in these comments.

Exclusion of known uses and exposures: Once again, EPA has abdicated its responsibility under TSCA to identify and evaluate the risks the chemical presents to the general population, by excluding from its risk evaluation conditions of use and exposures that are known or reasonably foreseen, including exposures from releases of TCE to air, water, and land. EPA has also failed to consider exposure to background levels of TCE. See section 2 of these comments.

Insufficient consideration of vulnerable subpopulations: EPA has not met its mandatory duty under TSCA to thoroughly identify and evaluate the risks to vulnerable subpopulations. These include, in addition to workers: subpopulations that are more susceptible to TCE exposure, including pregnant women and the developing fetus and diseased subpopulations including those managing kidney and liver disease; as well as consumers and others who may be at risk of cancer from acute exposures. See sections 1.A., 4.A., and 4.D of these comments.

Failure to protect against the most sensitive endpoint, congenital heart defects[4]: EPA's reliance on immune-related endpoints instead of congenital heart defects for its determinations of acute and chronic risk deviates from scientific best practices, defies requirements under the law, ignores longstanding Agency policy, and is not sufficiently protective of public health and vulnerable subpopulations. See sections 4.C., D., E., G., and H. of these comments and Appendix 1.[5]

Underestimation of occupational risks: Of particular concern is the extent to which EPA has underestimated occupational risks. EDF has analyzed each of the individual risk estimates EPA has made in this draft risk evaluation, which is presented in sections 5.A. and 7.A. of these comments. EDF's analyses identify and quantify several major ways in which EPA has underestimated occupational risks, including through: EPA's unsupported assumptions regarding worker use of personal protective equipment in many scenarios; its use of a cancer risk level for workers that fails to protect them as a vulnerable subpopulation as required by TSCA; its failure to consider combined exposures of workers from multiple sources; and its failure to identify unreasonable risks for the most highly exposed (and hence especially vulnerable) occupational

[4] Various terms are used by EPA and across the scientific literature to refer to congenital heart defects linked to TCE exposure, including "fetal heart malformations," "fetal cardiac defects," and others. For consistency, EDF will use the term "congenital heart defects" throughout these comments except where quoting from others.

[5] Appendix 1, EDF comment "Peer reviewers of EPA's TCE report must affirm that the key risk is fetal heart damage," is also available at https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0500-0071.

non-users (ONUs). EPA's exposure assessment has underestimated occupational exposures. See sections 1.B., 5.A., 5.B., and 7.A. of these comments.

Dismissal of risk by invoking uncertainty: EPA invokes uncertainty as an unwarranted basis for ignoring risks it has identified to the environment and to ONUs, and for not accounting for combined exposures to TCE. See sections 5.B.i., 5.E.i., 7.A.iii., and 7.B. of these comments.

Failure to adequately evaluate environmental risks of TCE release and exposure: Ignoring numerous studies on TCE in the environment, EPA has over-relied on predictions from physical-chemical properties and unwarranted assumptions to disregard or underestimate environmental risks, including to aquatic, sediment, and terrestrial organisms. EPA has also ignored available data on environmental releases. See section 6 of these comments.

Use of a flawed systematic review approach: EPA has continued to use its flawed TSCA systematic review approach that inappropriately downgrades epidemiological evidence, fails to provide and utilize a pre-established methodology, and selectively includes or excludes studies in a manner that reveals inconsistency and bias. See section 8 of these comments.

As discussed in these comments, these deficiencies severely compromise EPA's risk determinations for individual conditions of use of TCE presented in Table 5-1 and section 5.3 of the draft risk evaluation. These factors have led EPA to systematically underestimate the risks of TCE arising from individual conditions of use, including risks to human health and specifically to vulnerable subpopulations, as well as to the environment.

Moreover, TSCA requires EPA to make a determination as to whether TCE, as a whole, presents an unreasonable risk of injury to health or the environment (see Part II, section 1 of these comments). The extent and magnitude of the flaws in this draft risk evaluation, and the resulting underestimation of risk, mean that EPA has clearly *not* provided support for any assertion that TCE, across all of its conditions of use, does not present unreasonable risk. Indeed, EPA's determinations that many conditions of use of the chemical do present unreasonable risk can only support a conclusion that the chemical as a whole presents unreasonable risk. The flaws we have identified make clear that EPA has significantly understated the extent and magnitude of the chemical's unreasonable risk overall as well as for specific conditions of use.

Part I of these comments first provides some broad, cross-cutting concerns about the draft risk evaluation as a whole and then presents additional comments in the approximate order of the scoping, risk evaluation and risk determination processes. The order of the comments does not imply relative importance.

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PART I[6]

1. Broad/cross-cutting concerns

A. EPA has given insufficient consideration to vulnerable subpopulations.

EPA has failed to sufficiently identify and address potential hazards, exposures, and risks to several vulnerable subpopulations. Our concerns are detailed in the following sections of these comments:

- Workers and ONUs: Sections 1.A., 1.B., 2.F., 5.A., 5.B., 5.C., 5.D., 5.E., 7.A.
- Consumers: Sections 2.A., 2.E., 4.A., 5.B., 5.E.
- Pregnant women, infants and children: Sections 1.A., 4.B., 4.C., 4.D, 4.E., 4.G.
- Health-compromised or genetically susceptible subpopulations: Sections 1.A., 4.B., 4.I.
- People in proximity to conditions of use or sources of contamination: Sections 2.B., 2.C., 2.D., 2.E.

The remainder of this subsection identifies additional ways not discussed elsewhere in which EPA has given insufficient consideration to vulnerable subpopulations.

i. Insufficient consideration of the unique susceptibility of pregnant women and the developing fetus

It is important to quantify the prevalence of pregnant women and their fetuses in the population in order to understand the magnitude of the subpopulation at risk: there are approximately 4 million births in the U.S. every year (and 1 in 100 of these babies has a congenital heart defect).[7]

a. Failure to account for women of childbearing age in dermal risk estimates

On p. 352 of the draft risk evaluation, EPA states:

> Dermal risk estimates were calculated for both average workers and women of childbearing age [Occupational Risk Estimate Calculator. Docket # EPA-HQ-OPPT-2019-0500], based on differences in delivered dose accounting for differing body weight and hand size. Exposures differ by only ~10% between these groups, so this difference is relatively insignificant considering the magnitude of risk estimates relative to the benchmark MOE. Accordingly, the risk

[6] Section cross-references in Part I to sections in Part II specify they refer to Part II, while section cross-references internal to Part I do not specify they refer to Part I.

[7] CDC webpage, "Data and Statistics on Congenital Heart Defects." [accessed 2020 Apr 27]. https://www.cdc.gov/ncbddd/heartdefects/data.html.

characterization section only presents dermal risk estimates for average adult workers (Section 4.2.2).

Under TSCA, EPA has a mandate to protect vulnerable populations, including women of childbearing age. Thus, the Agency must use exposure values applicable to subpopulations with elevated exposure, even if EPA believes the values would not significantly affect the overall risk conclusion. To ignore these data is counter to the law by failing to identify the actual risks to potentially exposed or susceptible subpopulations; their omission also makes it more likely EPA will fail to identify an unreasonable risk where it should have, and even where it identifies such a risk, will fail to adequate address that risk in subsequent regulation under TSCA section 6. EPA's practice of choosing to ignore risks it deems "relatively insignificant" also fails to consider the contribution of such risks to overall risks faced by individuals or subpopulations in light of additional exposures they experience.

b. Insufficient acknowledgement of the importance of potential transfer through the blood-brain barrier

EPA appropriately acknowledges that TCE can be found in many tissues including the brain (p. 203); however, EPA fails to explicitly highlight that TCE can readily cross the blood-brain barrier. For example, the 2019 ATSDR ToxProfile for TCE states: "Trichloroethylene crosses the blood-brain barrier, and the extent of transfer could possibly be greater in young children, although trichloroethylene is expected to readily cross the blood-brain barrier in all age groups."[8] This is essential to emphasize given the evidence for neurotoxicity, including developmental neurotoxicity (see, for example, sections 3.2.3.1.3 and 3.2.3.1.6 of the draft risk evaluation).

c. Limited evaluation of developmental toxicity

EPA only conducted a very limited evaluation of developmental toxicity endpoints in this draft risk evaluation. During the March 24-27, 2020, virtual SACC meeting to peer-review the TCE draft risk evaluation (hereafter "TCE SACC meeting"), several panelists noted that additional review on this topic must be conducted.

For example, the only developmental immunotoxicity study that EPA identified was Peden-Adams et al. 2006. However, this study was deemed "Low" quality according to the OPPT TSCA systematic review method and a POD was not derived. As discussed in section 8 of these comments, the TSCA systematic review method is problematic and any conclusions of study quality based on the method is suspect and should be carefully scrutinized. For example, the IRIS toxicology review of TCE used Peden-Adams et al. 2006 to support the derivation of the

[8] Agency for Toxic Substances and Disease Registry (ATSDR). 2019. Toxicological profile for Trichloroethylene (TCE). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, p. 261.

TCE RfD, indicating that "[f]or adult and developmental immunological effects, there is high confidence in the evidence of immunotoxic hazard from TCE."[9]

Given that 1) immunotoxicity served in this draft as the representative endpoint for both acute and chronic non-cancer POD modeling, and 2) the developmental period is known to be particularly sensitive to toxic chemical exposures, SACC panelists urged EPA to make additional effort to find other data relevant to developmental immunotoxicity. Similarly, the SACC asked EPA to improve its review of developmental neurotoxicity endpoints, including by incorporating the missing references that the panelists provided.

ii. Insufficient acknowledgment of variability in human susceptibility

a. Failure to highlight key groups of susceptible individuals

In section 3.2.5.2, EPA acknowledges a wide variety of potentially exposed or susceptible subpopulations. In addition to those EPA lists, EPA should explicitly acknowledge the following additional groups that represent key susceptible subpopulations:[10]

- Individuals with compromised liver or kidney function: In the U.S. there are more than 4.5 million adults living with chronic liver disease[11] and more than 6 million adults with diagnosed kidney disease.[12] This burden of disease will increase in coming years; the U.S. prevalence of liver and kidney disease are projected to increase from 2020 to 2030.[13,14]

[9] Integrated Risk Information System (IRIS). 2011. Trichloroethylene (CASRN 79-01-6). https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0199_summary.pdf#nameddest=rfd.

[10] ATSDR Toxicological Profile for TCE, p. 271.

[11] CDC National Center for Health Statistics. 2019. Chronic Liver Disease and Cirrhosis. [accessed 2020 Apr 22]. https://www.cdc.gov/nchs/fastats/liver-disease.htm.

[12] CDC National Center for Health Statistics. 2020. Kidney Disease. [accessed 2020 Apr 22]. https://www.cdc.gov/nchs/fastats/kidney-disease.htm.

[13] Estes C, Razavi H, Loomba R, Younossi Z, Sanyal AJ. Modeling the epidemic of nonalcoholic fatty liver disease demonstrates an exponential increase in burden of disease. Hepatology. 2018 Jan;67(1):123-133.

[14] CDC Chronic Kidney Disease Surveillance System—United States. Projected Prevalence of CKD in 2020 and 2030 in Adults Aged 65 Years or Older. [accessed 2020 Apr 22]. https://nccd.cdc.gov/CKD/detail.aspx?QNum=Q678.

- Individuals with cardiac arrhythmias: Between 3 and 6 million individuals in the U.S. have atrial fibrillation, the most common type of heart arrhythmia.[15]
- Individuals with obesity: TCE distributes in the body particularly to body fat and liver. The prevalence of obesity is expected to increase to about 50% of the U.S. population by 2030.[16,17]
- Individuals with multiple chronic conditions: Among U.S. adults aged 18 and over, about 26% (more than 50 million people) have two or more chronic conditions such as heart, kidney and liver disease.[18]
- Individuals co-exposed to chemicals that interact with TCE metabolism, including:
    - Chlorinated hydrocarbons, which commonly co-occur in drinking water: According to the U.S. Geological Survey, chlorinated solvents are "one of the two most frequently detected VOC groups in the nation's aquifers."[19]
    - Phenobarbital, a medication used to treat epilepsy: In the U.S., there are more than 3 million adults with epilepsy.[20]
    - Ethanol: More than 14 million Americans have alcohol use disorder.[21,22]

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[15] CDC National Center for Chronic Disease Prevention and Health Promotion, Division for Heart Disease and Stroke Prevention. 2019. Atrial Fibrillation | cdc.gov. Centers for Disease Control and Prevention. [accessed 2020 Apr 22]. https://www.cdc.gov/heartdisease/atrial_fibrillation.htm.

[16] Agency for Toxic Substances and Disease Registry (ATSDR). Trichloroethylene (TCE) Toxicity: What Is the Biological Fate of Trichloroethylene in the Body? [accessed 2020 Apr 22]. https://www.atsdr.cdc.gov/csem/csem.asp?csem=15&po=9

[17] Ward ZJ, Bleich SN, Cradock AL, Barrett JL, Giles CM, Flax C, Long MW, Gortmaker SL. Projected U.S. State-Level Prevalence of Adult Obesity and Severe Obesity. N Engl J Med. 2019 Dec 19;381(25):2440-2450.

[18] Ward BW, Schiller JS, Goodman RA. Multiple chronic conditions among US adults: a 2012 update. Prev Chronic Dis. 2014 Apr 17;11:E62. doi: 10.5888/pcd11.130389

[19] Zogorski, John S. *The quality of our nation's waters: Volatile organic compounds in the nation's ground water and drinking-water supply wells*. Vol. 1292. Geological Survey (USGS), 2006.

[20] CDC. Epilepsy Data and Statistics. [accessed 2020 Apr 22]. https://www.cdc.gov/epilepsy/data/index.html.

[21] Müller, G., Spassowski, M. & Henschler, D. Metabolism of trichloroethylene in man. Arch Toxicol 33, 173–189 (1975).

[22] National Institute on Alcohol Abuse and Alcoholism (NIAAA). 2019. Alcohol Facts and Statistics. [accessed 2020 Apr 22]. https://www.niaaa.nih.gov/publications/brochures-and-fact-sheets/alcohol-facts-and-statistics.

b. Lack of detail regarding the extent of genetic variation in key metabolic pathways

EPA appropriately acknowledges that "[s]ignificant variability may exist in human susceptibility to TCE toxicity given the existence of CYP isoforms and the variability in CYP-mediated TCE oxidation" (p. 204). Yet, given the implications of this variability for considering risks to potentially exposed or susceptible subpopulations, further information on and analysis of the potential variability in CYP oxidation across the human population should be provided. For example, in the 2019 ATDSR ToxProfile, that Agency clearly stated that "human variability in metabolism of trichloroethylene via cytochrome P450-dependent pathways was within a 10-fold range."[23] Such quantitative details are crucial for understanding the true impact of these variations.

iii. Failure to consider workers with compromised health

EPA's Risk Estimation Approach for Human Health Risk only considers *healthy* female and male workers (see footnote 1, Table 4-4, p. 277). Assuming that all workers are "healthy" is erroneous and counter to the mandate of TSCA, which directs EPA to protect the most vulnerable subpopulations.

iv. Insufficient consideration of potential elevated respiration rates in exposed workers

On p. 352 of the draft risk evaluation, the Agency states:

> EPA expects that variability in human physiological factors (e.g., breathing rate, body weight, tidal voume [*sic*]) which may affect internal delivered concentration or dose is sufficiently accounted for in the PBPK model, although some differences among lifestages or between working and at-rest individuals may not have been accounted for. The use of HEC/HED99 values is expected to account for the vast majority of physiological differences among individuals."

Based on this paragraph, it is unclear whether the PBPK model sufficiently addresses potential elevated respiratory rates in workers. EPA states that it "expects" its model has accounted for variability in basic "physiological factors (e.g. breathing rate)" but also that "some differences… between working and at-rest individuals may not have been accounted for." Workers are a crucial vulnerable subpopulation with respect to TCE, and therefore EPA must fully and accurately characterize and account for potential elevated respiratory rates among active workers. This point was reinforced by a peer reviewer during the TCE SACC meeting, who

[23] Agency for Toxic Substances and Disease Registry (ATSDR). 2019. Toxicological profile for Trichloroethylene (TCE). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. P. 207.

indicated that EPA should not use a resting breathing rate for workers but rather an exercise breathing rate or at least something in between the two.

In the recent National Academies of Sciences, Engineering, and Medicine (NASEM) *Review of DOD's Approach to Deriving an Occupational Exposure Level for Trichloroethylene*, the NAS highlights a similar concern:[24]

> Lastly, all PBPK-based derivations of HECs were performed using resting ventilation and associated cardiac output physiological profiles. This may be appropriate for clerical or other office workers (e.g., vapor intrusion within an office building) but for other DOD occupations where ventilation and cardiac output are elevated by more strenuous exertion for extended durations, the resulting HECs may not be sufficiently protective. If such workplace exposure cases are considered relevant to DOD, the committee recommends incorporating exercise (work) physiology and realistic durations from actual job profiles into PBPK simulations for selected end points most likely to drive the OEL.

In the draft risk evaluation, EPA is clearly charged with examining the risks to workers in addition to clerical or other office workers. If EPA did use resting cardiac profiles, this analysis must be enhanced to provide more realistic estimates of exposure levels for active workers. If EPA used respiration rates appropriate for active workers, this should be more clearly communicated.

B. EPA has overrelied on personal protective equipment and the adequacy of OSHA requirements.

EPA's risk determinations heavily rely on assumptions that workers, at many or most points in the value chain and lifecycle of TCE, will use personal protective equipment (PPE) (gloves and respirators) and that it will be universally effective:

> EPA expects there is compliance with federal and state laws, such as worker protection standards, unless case-specific facts indicate otherwise, and therefore existing OSHA regulations for worker protection and hazard communication will result in use of appropriate PPE consistent with the applicable SDSs. (p. 377)

Section 5.A. of these comments provides an analysis EDF conducted of the extent and impact of this over-reliance on PPE.

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[24] National Academies of Sciences, Engineering, and Medicine. 2019. *Review of DOD's Approach to Deriving an Occupational Exposure Level for Trichloroethylene*. Washington, DC: The National Academies Press. https://doi.org/10.17226/25610.

EPA makes clear that its risk determinations "incorporate consideration of expected PPE (frequently estimated to be a respirator of APF 25 or 50 and gloves with PF 5 – 20)" (p. 35).[25] While EPA does still find unreasonable risk for a significant majority of conditions of use (COUs) – due to the extremely high toxicity of TCE – through its PPE assumptions the Agency has dramatically underestimated the risk, which likely will have implications for the risk management stage (see sections 5.A. and 7.A.i. for further discussion).

EPA's assumptions about PPE use are wholly unsupported and unwarranted. EPA has provided no data or analysis whatsoever to support these sweeping assumptions. In previous draft risk evaluations, the Agency has made it clear that it does not have any actual data on respirator or glove use, including data on types used, frequency of use, or prevalence of respiratory protection programs. For TCE, EPA has made some similar statements but buried them in the Supplemental File: Environmental Releases and Occupational Exposure:[26]

> The complexity and burden of wearing respirators increases with increasing APF. The APFs are not to be assumed to be interchangeable for any conditions of use, any workplace, or any worker or ONU. The use of a respirator not necessarily would resolve inhalation exposures since it cannot be assumed that employers have or will implement comprehensive respiratory protection programs for their employees. (p. 32, supplemental file)
>
> Data about the frequency of effective glove use – that is, the proper use of effective gloves – is very limited in industrial settings. Initial literature review suggests that there is unlikely to be sufficient data to justify a specific probability distribution for effective glove use for a chemical or industry. (p. 223, supplemental file)

Instead, EPA simply assumed without evidence that respirators or glove use would result in various levels of protection based on different, purely hypothetical PPE scenarios. EPA then found unreasonable risk only where either: a) the most stringent PPE it could assume was insufficient to mitigate the risk, or b) EPA could not possibly justify any assumption that PPE would be used:

> For workers (which are one example of PESS), an unreasonable risk may be indicated when risks are not adequately addressed through expected use of

[25] Note that we found only a single instance where EPA relied on a protection factor (PF) of 5 for workers (for toner aid; p. 367); otherwise EPA always assumed a PF of 10 or 20.
[26] Supplemental File: Environmental Releases and Occupational Exposure. Available at: https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0500-0020.

> workplace practices and exposure controls, including engineering controls or use of personal protective equipment (PPE). (p. 374)

There is considerable evidence of major real-world limitations of PPE, with regards to both the extent of use and effectiveness. In fact, OSHA has highlighted the major limitations of reliance on PPE, as has EPA in the recent past. For example, in 2016 OSHA informed EPA that respirators are the "least satisfactory approach to exposure control," providing the following explanation:

> [T]o be effective, respirators must be individually selected, fitted and periodically refitted, conscientiously and properly worn, regularly maintained, and replaced as necessary. The absence of any one of these conditions can reduce or eliminate the protection the respirator provides.
>
> Respirator effectiveness ultimately relies on the practices of individual workers who must wear them. … Furthermore, respirators can impose substantial physiological burdens on workers, including the burden imposed by the weight of the respirator; increased breathing resistance during operation; limitations on auditory, visual, and olfactory sensations; and isolation from the workplace environment.[27]

It is also important to recognize that reliance on PPE as a primary measure to protect workers is counter to OSHA's Industrial Hygiene Hierarchy of Controls (HOC), a long-standing principle that prioritizes measures to eliminate or reduce the presence of a hazard in occupational settings (e.g., substitution/use of less toxic chemicals and institution of engineering controls) over measures that shift burdens onto the workers themselves, such as through reliance on PPE and warning labels. The HOC exemplifies the best available science for creating safe, healthful workplace environments.

EDF previously commented on the serious limitations of labeling and PPE, as well as the importance of adherence to the industrial HOC to limit workplace exposures.[28] EDF

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[27] Comment Letter from David Michaels, PhD, MPH, Assistant Secretary, U.S. Dep't of Labor, Occupational Safety & Health Admin., to James J. Jones, Assistant Administrator, U.S. EPA, Office of Chem. Safety & Pollution Prevention (Oct. 25, 2016), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2014-0650-0041.

[28] *See, e.g.*, EDF Comments on TSCA Review and Scoping for First 10 Chemicals under the Lautenberg Act at 6 (Mar. 15, 2017), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0736-0046; EDF Comments on Significant New Uses of Chemical Substances; Updates to the Hazard Communication Program and Regulatory Framework; Minor Amendments to Reporting Requirements for Premanufacture Notices (Nov. 21, 2016), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2014-0650-0052.

incorporates and reiterates the points made in those comments here. Of note, during the TCE SACC meeting, one peer reviewer repeatedly mentioned that EPA's approach inappropriately ignores the HOC, jumping straight to PPE. The reviewer suggested that measures higher up the HOC, including whether the chemical is needed at all as well as protection afforded by engineering controls, should be considered first.

It would be wholly inappropriate for EPA to assume that there is compliance with OSHA's Permissible Exposure Limit (PEL) and that such compliance would be at all health-protective. The PEL, set at 100 ppm, was adopted by OSHA nearly 50 years ago in 1971, and, as EPA acknowledges (p. 468), OSHA itself "has recognized that many of its permissible exposure limits (PELs) are outdated and inadequate for ensuring protection of worker health" and recommends the use of the 2 ppm NIOSH Recommended Exposure Limit (REL). As a further indication of the inadequacy of OSHA's PEL, in the course of developing proposed rules that would have banned particularly high-risk uses of TCE (proposals that have since been abandoned), EPA developed a recommendation for an Existing Chemical Concentration Limit, or "ECEL"[29] of *1 ppb* (8-hour time weighted average) as a more current benchmark for workplace exposures.

Despite the severe problems with the OSHA PEL, EPA still seems to assume compliance even with this overly lax standard and ignore real-world workplace monitoring data points it has obtained that are above 100 ppm. EPA did so in its "PEL-capped" analysis, where the Agency excluded data samples that were above the PEL (see section 5.D. below). Ironically, notwithstanding its assumption to the contrary (p. 377), EPA's analysis demonstrates that non-compliance has in fact occurred: real-world exposure monitoring data include samples at levels that exceed the PEL.

Moreover, EPA invokes the OSHA PPE standard at 29 CFR § 1910.134 throughout the draft risk evaluation (e.g., pp. 54, 61, 62). However, this standard is rendered unprotective by the outdated TCE PEL: OSHA cannot require use of respirators as long as TCE levels do not exceed the 100 ppm PEL. Beyond this, the OSHA respiratory protection standard requires any employers who do provide respirators to their employees to create an entire program, including fit testing and medical exams.[30] Therefore, there is a strong disincentive for employers to provide such PPE if it is not required because workplace levels do not exceed 100 ppm.

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[29] U.S. EPA, Recommendation for an Existing Chemical Exposure Concentration Limit (ECEL) for Occupational Use of Trichloroethylene (TCE) and Sampling and Analysis Methods for TCE (Sept. 2016), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0163-0039.

[30] OSHA, 29 CFR 1910.134, available: https://www.osha.gov/pls/oshaweb/owadisp.show_document?p_id=12716&p_table=standards, and "Comment submitted by David Michaels, Department of Environmental and Occupational

EPA also mischaracterizes OSHA regulations with regards to SDSs. OSHA regulations do not in fact require that persons comply with SDSs, but rather provide broad discretion to employers themselves to decide whether any PPE is needed. In our comments on the 1-Bromopropane Draft Risk Evaluation,[31] EDF commented extensively on why it is inappropriate for EPA to assume that workers will be aware of, understand and follow SDS recommendations. In short, not only do OSHA regulations *not* require that persons comply with SDSs, but even if compliance with SDSs were mandatory, reliance on them would still be insufficient to ensure protection given that SDSs are often inaccurate, incomplete, and too technical for many workers to understand. We incorporate our previous comments herein by reference. EDF has further described these limitations in detail in a 2019 series of posts to our EDF Health blog.[32] EDF incorporates those posts by reference as well. We also incorporate by reference the comments submitted to EPA by Jonathan Kalmuss-Katz and Randy Rabinowitz on the 1,4-Dioxane Draft Risk Evaluation.[33]

Furthermore, OSHA's database of inspections demonstrates significant noncompliance with OSHA respiratory protection requirements such as those that apply to TCE. For example, in both fiscal years 2018 and 2019, OSHA cited nearly 3,000 violations each year of the respiratory protection standard, identified in nearly 1,300 separate inspections each year.[34] Violations of the respiratory standard were the 4th most common type of violation in OSHA inspections in fiscal year 2018, exceeded only by those for two categories of physical hazard and the Hazard Communication Standard.[35]

Health, Milken Institute School of Public Health, The George Washington University." https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0500-0061.

[31] Environmental Defense Fund Comments on the Draft Risk Evaluation of 1-Bromopropane, (Oct. 11, 2019), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0235-0047.

[32] See Appendix 2 of these comments.

[33] Comments submitted by Jonathan Kalmuss-Katz, Staff Attorney, Earthjustice and Randy Rabinowitz, Executive Director, Occupational Safety & Health (OSH) Law Project, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0238-0021.

[34] In FY 2018, OSHA reported 2,892 violations identified in 1,281 separate inspections. In FY 2019, OSHA reported 2,932 violations identified in 1,289 separate inspections. The FY 2019 statistics are available at U.S. Department of Labor, Occupational Safety and Health Administration, Industry Profile for OSHA Standard 19100134, https://www.osha.gov/pls/imis/industryprofile.stand?p_esize=&p_stand=19100134&p_state=FEFederal&p_type=5 (last accessed April 24, 2020). The FY 2018 statistics were recorded by EDF earlier and have since been replaced with the FY 2019 data and appear not to be accessible anymore.

[35] U.S. Department of Labor, Occupational Safety and Health Administration, Top 1- Most Frequently Cited Standards, https://www.osha.gov/top10citedstandards (last accessed April 24, 2020).

In comments submitted to the TCE docket on April 9, 2020, Cal/OSHA highlighted OSHA inspection data specific to Automotive Body, Paint, and Interior Repair and Maintenance (NAICS Code 81121), noting that it was the second most cited industry classification in respiratory protection inspections (40 inspections, 107 citations) in 2018, and that many of the occupational exposure scenarios covered by the TCE risk evaluation consist of industries similar to NAICS Code 81121.[36]

In the same comments, Cal/OSHA raised concerns with EPA's assumption of 100% compliance with and effectiveness of PPE workplace programs.[37] Cal/OSHA summarized data from a recent study on workplace safety practices in the auto collision industry,[38] and recommended that EPA discern two points from the study:

> First, employer adherence to required safety programs is rarely 100%; even with active intervention from a third-party, required safety programs were in place in only 73% of the workplaces in the study. Second, the declines in respiratory protection and RtK [Right to Know] training tracks what is found by OSHA – these two programs are typically the most cited violations in OSHA investigations.

Of note, the SACC Peer Review Report on Methylene Chloride recommended that EPA incorporate data from the NIOSH and Bureau of Labor Statistics (BLS) joint survey on "Respirator Usage in Private Sector Firms," which provides industry-based estimates of respirator program effectiveness, and discussed in considerable detail these data as well as additional data from other published literature sources.[39] EPA summarized the findings of the

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[36] Comments submitted by Eric Berg, Deputy Chief, Cal/OSHA Research and Standards. April 19, 2020. Available: https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0500-0080.

[37] Ibid.

[38] Bejan A, Parker DL, Brosseau LM, Xi M , Skan M. 2015. Two-Year Follow-up of the Collision Auto Repair Safety Study (CARSS) Ann Occup Hyg. 59(5): 534–546. Available: https://europepmc.org/article/med/25539646.

[39] Meeting Minutes and Final Report for the TSCA Science Advisory Committee on Chemicals for Methylene Chloride, pp. 36-37:

> 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

joint NIOSH/BLS survey in the draft carbon tetrachloride risk evaluation, explaining that the survey found of establishments that required respirator use: 59% provided training to workers on respirator use; 34% had a written respiratory protection program; 47% performed an assessment of the employees' medical fitness to wear respirators; and 24% included air sampling to determine respirator selection. The draft carbon tetrachloride risk evaluation concluded, based on these data, "the likelihood of respirator use may not be widespread."[40] In contrast, EPA has completely failed to acknowledge or incorporate these data into the TCE risk evaluation.

Even when respirators and gloves *are* used, workers may still be exposed to TCE. Organic solvents like TCE may breakthrough the carbon or other medium in organic vapor cartridge respirators, and this can occur without providing any indication to the user that the respirator is no longer functioning.[41]

> 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) 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. The Committee suggested that the NIOSH BLS respirator usage survey can be used to provide industry-based estimates of respirator program effectiveness, which could then be employed to set the best Assigned Protection Factor (APF) for an industry. One Committee member indicated that the high-end exposure scenarios do not include protection factors derived from assumed respirator use. …
>
> The EPA should present and/or reference the literature reviewed and should be clear when they believe that PPE will be used within an industry and present the appropriate justification. The EPA should indicate when/if the assessment of PPE use was made based on professional judgment."

[40] EPA, 2020. Draft Risk Evaluation for Carbon Tetrachloride, p. 63. Available: https://www.epa.gov/sites/production/files/2020-01/documents/1_ccl4_draft_risk_evaluation_draft_public_updated.pdf.

[41] United States Air Force AFIOH, 2004. "Estimating Organic Vapor Cartridge Service Life," p. 29. Available at: https://apps.dtic.mil/dtic/tr/fulltext/u2/a439710.pdf.

Gloves may also experience chemical breakthrough and provide limited protection from TCE exposure (see p. 221 of Supplemental File: Environmental Releases and Occupational Exposure Assessment[42]). Protection also varies greatly with different glove materials, which EPA alludes to by acknowledging specific glove material types recommended by SDSs (p. 116 of the supplemental file). Yet, the Agency cites no data on actual use of such gloves versus less effective or ineffective alternatives, and instead simply assumes default glove protection factors (PFs) and disregards the potential for occlusion to increase exposure (see section 5.B.iv. for further discussion of EPA's apparent failure to make risk determinations based on heightened exposure due to occlusion).

In a few places in the draft, EPA very briefly acknowledges some of the limitations of PPE and acknowledges the preferability of other options higher up in the industrial hygiene hierarchy of controls (e.g., pp. 119-120). But when it comes to determining risk, those limitations and preferences fall away and EPA exclusively relies on "expected" use of PPE to understate the extent and magnitude of the risks it has identified (see section 5.A. of these comments for an analysis documenting the extent of EPA's reliance).

As further addressed in subsection C. below, EPA's reliance on PPE is not merely a policy determination. It is a huge assumption that dramatically alters the magnitude of risk presented in EPA's risk characterizations for TCE. EPA's reliance on PPE is also a key driver of a large fraction of EPA's risk determinations for workers even though EPA has no actual data on the extent or effectiveness of PPE use. EPA's reliance on PPE leads the Agency to understate the extent and magnitude of the risk where it does identify unreasonable risk. See section 5.A. of these comments. EPA's failure to provide *any* supporting data that PPE is universally used and effective, as assumed in its risk determinations for many conditions of use (COUs), is a glaring flaw in this draft risk evaluation.

Section 5.A. of these comments presents an analysis showing that, for many of the endpoints and exposure routes under the conditions of use of TCE, EPA found that they present no unreasonable risk only by assuming that workers wear effective PPE to protect against inhalation or dermal exposures. For most of those conditions of use where EPA did identify unreasonable risk, it was compelled to do so because even the most stringent level of respiratory or dermal PPE EPA examined and assumed would be used was insufficient to eliminate that risk.

[42] Supplemental File: Environmental Releases and Occupational Exposure. Available at: https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0500-0020.

C. EPA's "policy" determinations have serious scientific and public health consequences.

EPA has publicly stated that a number of Agency decisions made in this and other draft risk evaluations – which EDF, other stakeholders, and the SACC have criticized heavily – are in the realm of policy, implying that they are somehow off-limits. These include:

- EPA's decision to exclude all general population risks arising from exposures from releases to land, air, and water based on the assumption that other statutes adequately address the exposures;
- EPA's decision to assume that PPE is always used and effective under many conditions of use; and
- EPA's decision to use a benchmark cancer risk level of 1 x 10^{-4} to define unreasonable risk to workers.

All three decisions, among others, have major direct scientific and public health consequences, as they clearly lead to underestimations of risk – to the environment, the general population, workers, and vulnerable subpopulations. In several final peer review reports, including those for 1,4-dioxane, 1-BP, and methylene chloride,[43] the SACC was critical of these Agency decisions.

All three of EPA's decisions described above represent assumptions that EPA has not verified or adequately explained, and that introduce major uncertainty into its risk evaluation that EPA has not analyzed, despite the fact that they dramatically affect EPA's characterization of exposure, hazard, and risk. Each of these assumptions is addressed below.

First, the statutory-based exclusions. As described in section 2.B. and Part II, section 5 of these comments, EPA has asserted that exposures to the general population are "adequately managed" without any analysis whatsoever of the standards under the other statutes, including standards that are not strictly health based, unlike TSCA's standard. EPA devoted a few scant paragraphs to justify its decision to eliminate entire pathways, and provided no data or analysis of the exposures and risks that remain and their contribution to total exposure and risk.

[43] *See* SACC Final Report/Meeting Minutes for 1,4-Dioxane, transmitted Oct. 31, 2019, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0237-0064; Meeting Minutes and Final Report for the SACC on 1-Bromopropane, transmitted Dec. 12, 2019, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0235-0061; Meeting Minutes and Final Report for the SACC on Methylene Chloride, transmitted March 2, 2020, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0437-0080.

EPA has failed to provide any scientific rationale for this assumption, and the SACC has been charged with commenting precisely on the adequacy of the support EPA has provided for just such assumptions.

Second, as discussed in further detail in sections 1.B. and 7.A.i. of these comments, EPA frequently assumes PPE is used and effective in order to find no unreasonable risk to workers, even though EPA has stated elsewhere that it does not have data on the extent of use and effectiveness of gloves or existence of comprehensive respiratory protection programs.

EPA should provide any feedback it has received from OSHA and NIOSH on its assumption regarding PPE use, and more generally, any input they have provided EPA regarding the extent and sufficiency of OSHA's authorities.

Third, EPA's unprecedented use of 1 in 10,000 as the cancer risk benchmark for workers also clearly underestimates risk, and flies in the face of EPA's longstanding policy "that it should reduce risks to less than 1 x 10^{-6} for as many exposed people as reasonably possible." National Emission Standards for Hazardous Air Pollutants; Radionuclides, 54 Fed. Reg. 51,654, 51,686 (Dec. 15, 1989). Workers are specifically identified under TSCA as a vulnerable subpopulation warranting special protection. See section 7.A.ii. for more detail on this issue.

In sum, EPA's sweeping assumptions regarding exposures from environmental releases, PPE use, and adequacy of reliance on a less protective cancer risk benchmark for workers have direct impacts on the scientific integrity of EPA's implementation of TSCA.

D. EPA's risk evaluation lacks an adequate mass balance.

As discussed by the SACC in its peer-review reports,[44] EPA's draft risk evaluations have failed to account for a chemical substance's presence and flow at the different stages of its lifecycle. In the case of TCE, over 170 million pounds of TCE are manufactured in or imported into the United States annually (p. 28), yet only about 2.2 million pounds of TCE were identified as released to the air, water and land;[45] the draft risk evaluation does not make clear where the rest

[44] SACC July 2019 Meeting Minutes and Final Report on 1,4-dioxane and HBCD at p. 44, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0238-0063; 1-BP TSCA SACC Meeting Minutes Final Report at p. 61, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0235-0061; SACC NMP Meeting Minutes and Final Report 3/5/2020, at pp. 19-20, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0236-0066; MeCl Meeting Minutes Final Report 03/02/2020 at pp. 22, 25, 27, 75, 77, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0437-0080.

[45] U.S. EPA, 2018 TRI Data, https://www.epa.gov/toxics-release-inventory-tri-program/tri-data-and-tools.

of it goes. In order to provide transparency, SACC members recommended that EPA should develop and present a mass balance for each chemical subject to a risk evaluation. EDF concurs and recommends that the Agency should provide a mass balance for TCE.

While the term "mass balance" can mean different things,[46] it is appropriate to look at the definition under the Emergency Planning and Community Right-to-Know Act (EPCRA), under which EPA must collect release data on chemicals through the Toxics Release Inventory (TRI). According to EPCRA, mass balance is "an accumulation of the annual quantities of chemicals transported to a facility, produced at a facility, consumed at a facility, used at a facility, accumulated at a facility, released from a facility, and transported from a facility as a waste or as a commercial product or byproduct or component of a commercial product or byproduct." 42 U.S.C. § 11023(l)(4). While EPA relies on the CDR and TRI to compile some estimates of these values, there are limitations on both of those reporting schemes that result in an incomplete picture of the chemical's lifecycle.

As reported by the National Research Council (NRC):

> Congress was aware that the toxic chemical release estimates reported under [TRI] might not accurately reflect the amounts actually released from reporting facilities (U.S. Congress, House, 1986). This potential inaccuracy is based on the provision that quantities of chemical releases can be obtained from theoretical calculations, engineering estimates, or by subtracting mass balance quantities (e.g., chemical quantity purchased minus the quantity contained in the product) rather than from measurements of actual releases."[47]

In order to conduct a robust and transparent risk evaluation on TCE, and more generally on chemical substances, EPA must provide significantly more detail about the chemical's lifecycle by conducting a mass balance analysis. Section 26(h)(3) requires the Administrator to consider the "degree or clarity and completeness with which [ ] data *** are documented," and without a mass balance analysis EPA has not reached any reasonable degree of clarity. 15 U.S.C. § 2625(h)(3). The failure to prepare an adequate mass balance is arbitrary and capricious because EPA has "entirely failed to consider an important aspect of the problem. *Motor Vehicle Mfrs. Ass'n v. State Farm Mut. Auto. Ins. Co.*, 463 U.S. 29, 43 (1983).

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[46] National Research Council, *Tracking Toxic Substances at Industrial Facilities: Engineering Mass Balance Versus Materials Accounting* p.2 (1990), https://www.nap.edu/read/1415/chapter/2.

[47] National Research Council, *Tracking Toxic Substances at Industrial Facilities: Engineering Mass Balance Versus Materials Accounting* (1990), https://www.nap.edu/read/1415/chapter/6.

2. EPA has inappropriately or illegally excluded conditions of use and exposures.

A. EPA failed to analyze certain reasonably foreseen conditions of use.

In the problem formulation, EPA excluded "paints and coatings for consumer use" on the basis of its assertion that TCE is no longer used in such products. Problem Formulation at p.20. EPA notably continues to analyze these conditions of use in the industrial and commercial context. *See* draft risk evaluation at p.50. During the TCE SACC meeting, an EPA representative explained that the Agency's exclusion of "paints and coatings for consumer use" is due to the Agency's promulgation of a "significant new use rule" (SNUR) on certain consumer uses of TCE in 2016.[48] EPA should nevertheless analyze the consumer uses in these circumstances because (as discussed in more detail below) the existence of a SNUR is insufficient to conclude that these uses will not occur or are not "reasonably foreseeable." Moreover, TCE's availability for use in the industrial and commercial context makes it at least reasonably foreseen that consumers may obtain TCE-containing products and be exposed.

While compelling evidence of both the absence of consumer products and the inaccessibility of commercial products to consumers could establish that these circumstances are not "known" conditions of use, EPA has failed to addressed whether "paints and coatings" are "reasonably foreseen" conditions of use for consumers. *See* 15 U.S.C. § 2602(4) ("The term 'conditions of use' means the circumstances, as determined by the Administrator, under which a chemical substance is intended, known, or *reasonably foreseen* to be manufactured, processed, distributed in commerce, used, or disposed of.") (emphasis added).

Congress included "reasonably foreseen" circumstances within TSCA with the express goal of ensuring that EPA swept more broadly than known (or intended) uses; EPA cannot evade that duty by limiting its analysis to conditions of use with evidence of current, ongoing use—such an interpretation would effectively limit EPA's analysis to "known" uses. While there may well be circumstances in which a use that is not currently occurring could be said to be not "reasonably foreseen" at this time, the term surely cannot be read in such a way that only uses that are known to be current are "reasonably foreseen" as that would read the term out of existence and collapse the inquiry to one where a use must be "known" to be considered "reasonably foreseen."

Reasonably foreseen is a term of art with a long history in the law; it is well established under the law that "[a] natural and probable consequence is a foreseeable consequence. But to be reasonably foreseeable [t]he consequence need not have been a strong probability; a possible consequence which might reasonably have been contemplated is enough." *People v. Medina*, 209 P.3d 105, 110 (Cal. 2009) (internal citations and quotation marks omitted). When a

[48] 81 Federal Register 20,535. April 8, 2016. Available: https://www.regulations.gov/document?D=EPA-HQ-OPPT-2014-0697-0014.

chemical is used in a commercial setting and where "there is no evidence to show that the manufacturer, wholesaler, or retailer of a hazardous substance sought to limit sales of the product to industrial or professional users, it is reasonably foreseeable that household consumers will have access to the product." *Canty v. Ever-Last Supply Co.*, 296 N.J. Super. 68, 78-79 (N.J. Super. Ct. 1996). Even where a product is "labeled for industrial use," it may be reasonably foreseeable that the product may ultimately be used by a consumer. *See No. 98-1979 v. Sunnyside Corp.*, 2000 Wisc. App. LEXIS 118, *12 (Ct. of Appeals WI, Feb. 15, 2000).

As noted above, during the TCE SACC meeting, an EPA representative explained that the Agency excluded the "paints and coatings for consumer use" because of its promulgation of a SNUR in 2016. The representative's comments implied that the 2016 SNUR *prohibits* consumer use of TCE in paints and coatings. However, this SNUR does not place any restrictions on such use; it only requires advance notification to and review by EPA of such use, and any actual restriction would require further Agency action subsequent to that review. Short of a ban on consumer uses promulgated under section 6 of TSCA, there is no assurance that EPA will not at some point allow such use and hence exposure is still "reasonably foreseen," even with a SNUR in place. Furthermore, even if a ban on TCE's use in such consumer products were in place, absent specific steps to ensure that consumers cannot gain access to products intended for industrial or commercial uses, such use would still be "reasonably foreseen."

Finally, non-occupational bystanders may be exposed to industrial or commercial uses of paints and coatings containing TCE during regular use, e.g., during painting of residential spaces or houses or other buildings. A peer reviewer at the TCE SACC meeting raised this concern, asking what types of industrial and commercial paints contain TCE and noting that certain types – like those for use on automobiles or aircraft – may be not be problematic, but commercial use of house paint would certainly lead to bystander exposures.

EPA's invoking of its 2016 SNUR applicable to certain consumer uses of TCE is far from sufficient to exclude consumer and bystander exposures to TCE-containing paint and coatings as reasonably foreseen. Therefore, EPA must evaluate these conditions of use in the final risk evaluation.

B. EPA has inappropriately excluded exposures based on other statutes.

Referencing its earlier problem formulation, EPA has excluded from its risk evaluation all general population exposures to TCE, based on EPA's assertion – unsupported by any actual data or analysis – that the exposure pathways for the general population are "adequately assess[ed] and effectively manage[d]" under other environmental statutes (p. 35). Specifically, EPA states:

> Exposure pathways to the general population are covered by other statutes and consist of: the ambient air pathway (i.e., TCE is listed as a HAP in the Clean Air Act (CAA)), the drinking water pathway (i.e., National Primary Drinking Water Regulations (NPDWRs) are promulgated for TCE under the Safe Drinking Water Act), ambient water pathways (i.e., TCE is a priority pollutant with recommended water quality criteria for protection of human health under the CWA), the biosolids pathway (i.e., the biosolids pathway for TCE is currently being addressed in the CWA regulatory analytical process), disposal pathways (TCE disposal is managed and prevented from further environmental release by RCRA and SDWA regulations). As described above, other environmental statutes administered by EPA adequately assess and effectively manage these exposures. Therefore, EPA did not evaluate hazards or exposures to the general population in this risk evaluation, and there is no risk determination for the general population (p. 379; see also 35, 276).

EPA therefore excluded from the draft risk evaluation all general population exposure through air, water, and land to TCE.

Aside from the absent legal basis, these exclusions present significant health concerns. For example, in the problem formulation for TCE (p. 54), EPA explicitly relies on the Clean Air Act (CAA) to dismiss the need to assess exposures to TCE from air emissions. TCE is regulated as a hazardous air pollutant (HAP) under the CAA, but the standards under the CAA for HAPs are set for individual source categories, meaning that the exposures to TCE from all sources in combination are never considered. In a recent proposed rule under the CAA for a source category for another chemical, EPA has stated that:

> Although we are interested in placing source category and facility-wide HAP risk in the context of total HAP risk from all sources combined in the vicinity of each source, we are concerned about the uncertainties of doing so. Estimates of total HAP risk from emission sources other than those that we have studied in depth during this RTR review would have significantly greater associated uncertainties than the source category or facility-wide estimates. Such aggregate or cumulative assessments would compound those uncertainties, making the assessments too unreliable.

National Emission Standards for Hazardous Air Pollutants: Rubber Tire Manufacturing Residual Risk and Technology Review, 84 Fed. Reg. 58,268, 58,273 (proposed Oct. 30, 2019). This explanation of EPA's approach to assessments under the CAA makes clear that EPA does not look at overall risk from a chemical substance. Therefore, EPA's approach to this and its other draft risk evaluations under TSCA ensures that EPA *never* evaluates, and the public never finds

out, the risk from all air emissions of TCE or any other chemical substance. The SACC has previously noted the flaws in this approach to EPA.[49]

In addition, even by its own account, EPA's CAA regulations do not eliminate risk to exposed populations. For example, when EPA promulgated a regulation "to limit emissions of methylene chloride (MC), trichloroethylene (TCE) and perchloroethylene (PCE) from facilities engaged in halogenated solvent cleaning," EPA did not eliminate cancer risks. National Air Emission Standards for Hazardous Air Pollutants: Halogenated Solvent Cleaning, 72 Fed. Reg. 25,138 (May 3, 2007). Instead, EPA adopted standards that it acknowledged would leave the maximum individual risk of cancer at "between 20 and 50-in-a-million and the total number of people with risks greater than 1-in-a-million would *** be *** between 500,000 and 1,000,000." *Id.* at 25,148. Thus, by EPA's own account, its CAA regulation of TCE did not eliminate all risk from just these facilities, much less consider how exposure to TCE from the regulated facilities might combine with exposures from other facilities and sources to increase overall risk.

EPA has also failed to acknowledge that the requirements to address human and environmental health risks it relies on derive from statutes that establish criteria different than those under TSCA.[50] Many of these other statutes, for example, require EPA or other agencies to consider factors such as cost and feasibility when setting standards -- factors that TSCA explicitly forbids EPA from taking into account when assessing risks. TSCA section 6(b)(4)(A) states (emphasis added):

> The Administrator shall conduct risk evaluations pursuant to this paragraph to determine whether a chemical substance presents an unreasonable risk of injury to health or the environment, *without consideration of costs or other nonrisk factors*, including an unreasonable risk to a potentially exposed or susceptible subpopulation identified as relevant to the risk evaluation by the Administrator, under the conditions of use.

Furthermore, as noted by peer reviewers at the TCE SACC meeting, the SDWA does not regulate all sources of drinking water. It is estimated that more than 13 million households rely

[49] SACC July 2019 Meeting Minutes and Final Report Docket (for 1,4-dioxane), p. 18 (Oct. 31, 2019), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0238-0063.

[50] EDF has addressed these limitations in greater detail in prior comments. *See, e.g.*, EDF Comment on the 1,4-dioxane Draft Risk Evaluation pp. 113-31, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0238-0058.

on private wells for drinking water in the United States.[51,52] The national drinking water standards established under the SDWA do not apply to private wells. *See* 42 U.S.C. § 300f(1) (a "primary drinking water regulation" only applies to "public water systems"); 42 U.S.C. § 300f(4)(A) (a "public water system" is a system that "has at least fifteen service connections or regularly serves at least twenty-five individuals"). Therefore, exposures in drinking water from private wells is not addressed by the SDWA and needs to be evaluated in the risk evaluation.

The releases and exposures EPA is ignoring are far from trivial. Based on the most recent data from EPA's Toxics Release Inventory (TRI) and National Emissions Inventory (NEI), despite existing regulations under other laws, facilities release 2 to 3 million pounds annually of TCE to air, water and land. EPA's approach effectively reduces this quantity to zero.

C. EPA's failure to consider general population exposures to TCE ignores numerous major exposure pathways.

EPA's decision to exclude any consideration of general population exposure to TCE in the draft risk evaluation ignores the exposures of millions of Americans to one of the most pervasive and toxic chemical pollutants in our environment. This omission also ignores exposures to the most vulnerable subpopulations, the developing fetus, infants, and children. These omissions violates the intent of the Lautenberg Act's 2016 amendments to TSCA and are contrary to the core mission of EPA to protect public health.

EPA and the Centers for Disease Control/Agency for Toxic Substances and Disease Registry (CDC/ATSDR) have documented the following key exposure pathways:[53]

Outdoor Air: EPA, CDC/ATSDR and most states have documented TCE concentrations in ambient air, with elevated levels around sources and in highly populated areas.

Indoor Air and Vapor Intrusion: TCE is pervasive in indoor air at concentrations documented to be several times higher than outdoor levels due to consumer products, vapor intrusion from subsurface contamination, and volatilization from contaminated drinking water.

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[51] PRIVATE DRINKING WATER WELLS, https://www.epa.gov/privatewells (last visited Jul. 31, 2018) (citing the US Census American Housing Survey 2015).

[52] An estimated 44.5 million people in the United States, or 14 percent of the population, provided their own water for domestic use in 2010. U.S. Geological Survey, *Estimated Use of Water in the United States in 2010* (2014), https://pubs.usgs.gov/circ/1405/pdf/circ1405.pdf.

[53] The information presented in this subsection is drawn from EPA's (2011) Toxicological Review of Trichloroethylene, available at https://www.epa.gov/iris/supporting-documents-trichloroethylene.

Groundwater and Drinking Water Wells: CDC/ATSDR has reported that TCE is the most frequently detected chemical contaminant in groundwater. Shallow private wells used for drinking water and irrigation are particularly vulnerable. As previously noted in these comments (see subsection B.), these wells are not monitored nor covered by the Safe Drinking Water Act.

Food: TCE has been found in a wide variety of foods. The FDA Total Diet Program has measured TCE throughout the food supply including butter, cheese, fruits, and cereal and grain products.

Breast Milk and Formula: TCE has been detected in breast milk in the general population. Formula fed infants are also vulnerable because of the pervasive contamination of drinking water and their high ingestion rate.

D. EPA needs to analyze those potentially exposed or susceptible subpopulations that face greater exposure due to their proximity to conditions of use.

TSCA § 3(12) states that "the term 'potentially exposed or susceptible subpopulation' means a group of individuals within the general population identified by the Administrator who, due to either greater susceptibility or greater exposure, may be at greater risk than the general population of adverse health effects from exposure to a chemical substance or mixture, such as infants, children, pregnant women, workers, or the elderly." 15 U.S.C. § 2602(12). In its draft risk evaluation, EPA erroneously limits its analysis to only half of this definition; EPA discusses whether persons might face greater susceptibility to TCE, but, outside of its consideration of worker and consumer exposures, EPA does not consider whether subpopulations may face a greater risk due to greater exposure. EPA must consider and analyze each of these types of subpopulations.

EPA must identify those who face greater exposures due to their proximity to conditions of use as a "potentially exposed or susceptible subpopulation" since they are a "group of individuals within the general population identified by the Administrator who, due to *** greater exposure, *may* be at greater risk than the general population of adverse health effects from exposure to a chemical substance or mixture." 15 U.S.C. § 2602(12) (emphasis added). Notably, in the problem formulations, EPA seemed to acknowledge that it should analyze these vulnerable subpopulations. *See, e.g.*, Problem Formulation for TCE at pp. 38-39 ("Other groups of individuals within the general population who may experience greater exposures due to their proximity to conditions of use that result in releases to the environment and subsequent exposures (e.g., individuals who live or work near manufacturing, processing, use or disposal sites).").

In the draft risk evaluation, EPA in a few places makes passing reference to such exposures. For example, it notes that "[e]xposures of TCE would be expected to be higher amongst groups

living near industrial facilities" (p. 186). And EPA acknowledges that it has underestimated consumer exposure due to its approach:

> Background levels of TCE in indoor and outdoor air are not considered or aggregated in this assessment; therefore, there is a potential for underestimating consumer inhalation exposures, particularly for populations living near a facility emitting TCE… . (p. 177)

But EPA does not identify these subpopulations as potentially exposed or susceptible subpopulations, or provide any analysis of the extent to which these or other subpopulations living in proximity to the conditions of use are at greater risk due to greater exposure.

EPA has failed to provide any factual justification for excluding such exposures. EPA should analyze these exposures and should analyze the associated risks to these *potentially* exposed subpopulations. EPA's failure to consider this relevant aspect of the problem is arbitrary and capricious.

In order to accurately assess the exposure of these subpopulations, EPA should analyze the environmental pathways that lead to their exposure. Thus, EPA should not exclude those pathways for the reasons given above, and in addition, EPA cannot rationally evaluate the greater exposure these subpopulations face without analyzing these pathways. Ignoring these subpopulations violates EPA's duty to consider potentially exposed or susceptible subpopulations.

As part of this analysis, EPA should identify people living near all disposal sites as potentially exposed or susceptible subpopulations. These groups include (but are not limited to) those living near Superfund sites.[54] To be clear, many disposal sites are associated with activities that reflect ongoing or prospective manufacturing, processing, distribution, or use, so EPA must also analyze those disposals and disposal sites and subpopulations living in proximity to them.

E. EPA has failed to consider exposure to background levels of TCE.

EPA states (p. 177):

> Background levels of TCE in indoor and outdoor air are not considered or aggregated in this assessment; therefore, there is a potential for underestimating consumer inhalation exposures, particularly for populations living near a facility emitting TCE or living in a home with other sources of TCE, such as TCE-containing products stored in the home.

[54] *See* Appendix 3 for a list of the 731 active Superfund sites containing TCE.

EPA must justify this decision, given that subpopulations living in proximity to such exposure sources represent potentially exposed or susceptible subpopulations that must be considered under TSCA (see subsection D.).

Should the Agency nevertheless decide to proceed with this exclusion, at a minimum EPA must quantify the number of people expected to experience substantial exposures to background concentrations of TCE in indoor and outdoor air to allow the public to understand the magnitude of the exposures being omitted as a result of the Agency's decision. Furthermore, EPA should conduct sensitivity analyses to quantify the potential extent of underestimation due to excluding these background exposures.

F. EPA excluded a number of workplace-related exposure scenarios.

EPA excluded a number of reasonably foreseen conditions of use in the workplace that should have been evaluated. During the SACC meeting to peer-review the 1,4-dioxane draft risk evaluation, concerns were raised about a number of exposure scenarios that the Agency failed to consider, each of which equally applies to TCE. Among those discussed are:

- Exposures from spills in the workplace, especially considering the potential for inhalation exposure from evaporation for maintenance workers cleaning up spills and leaks;[55]
- "Take-home exposures," whereby the family of a worker, including children, may be exposed via contact with the worker's contaminated clothing or skin; and
- Exposures of workers at small or medium facilities where assumptions of routine PPE use or other protections are even less likely to be valid.

With regards to the last point, as documented in section 5.A. of these comments, while we recognize that EPA did not assume use of respirators by workers under five conditions of use (COUs) presenting occupational exposure potential, EPA still assumed universal use and effectiveness of respirators in all of the other such COUs. SACC members peer-reviewing 1,4-dioxane expressed concern that even if one assumes that PPE is typically used in larger, industrial facilities, smaller facilities are much less likely to require routine and effective use of protective equipment or to employ engineering controls, like closed systems. Workers at any facility – whether small, medium, or large – where use of effective PPE cannot be thoroughly documented should be considered vulnerable subpopulations and the risk they face be

[55] While the Conceptual Model on page 57 indicates that fugitive emissions were considered (which the Agency defines as "those that are not stack emissions, and include fugitive equipment leaks from valves, pump seals, flanges, compressors, sampling connections and open-ended lines; evaporative losses from surface impoundment and ***spills***; and releases from building ventilation systems," emphasis added), it is unclear whether or how EPA actually addressed spills in the draft risk evaluation.

specifically assessed. For these subpopulations, EPA must determine risk based on exposures without assuming any use of PPE.

"Conditions of use" are broadly defined under TSCA to mean "the circumstances, as determined by the Administrator, under which a chemical substance is intended, known, or *reasonably foreseen* to be manufactured, processed, distributed in commerce, used, or disposed of." 15 U.S.C. § 2602(4) (emphasis added). Each of the circumstances described above—spills, take home exposures, and exposures without appropriate PPE—is a "reasonably foreseen" aspect of the circumstances under which methylene chloride is manufactured, processed, distributed, used, or disposed of.[56] It is well established under the law that "[a] natural and probable consequence is a foreseeable consequence. But to be reasonably foreseeable [t]he consequence need not have been a strong probability; a possible consequence which might reasonably have been contemplated is enough." *People v. Medina*, 46 Cal. 4th 913, 920 (Cal. 2009) (internal citations and quotation marks omitted).

"Reasonably foreseen" is a term of art with a long history in the law, and EPA should turn to the ample precedent interpreting this language to inform implementation of this legal requirement. Spills and leaks are undoubtedly reasonably foreseeable, and indeed, when preparing environmental impact statements (EISs) for federal projects, the federal government regularly analyzes the potential for spills and leaks because they are reasonably foreseen aspects of such projects. *See, e.g.*, *Sierra Club v. Clinton*, 689 F. Supp. 2d 1123, 1139 (D. Minn. 2010) (describing analysis of potential for leaks and spills).

And in the tort context, courts have found that spills and leaks can be reasonably foreseen. *See, e.g.*, *Monroe v. Safeway, Inc.*, 2004 Wash. App. LEXIS 672, *6 (Ct. Appeals Wash. 2004); *Ceasar v. Wal-Mart Stores, Inc.*, 787 So. 2d 582, 588 (Ct. Appeals. La. 2001) (finding spill reasonably foreseeable); *Lumbermens Mut. Cas. Co. v. Banco Espanol De Credito*, 2006 U.S. Dist. LEXIS 75728, *25 (S.D.N.Y. Oct. 13, 2006) (describing leak as reasonably foreseeable); *Goehler v. Wal-Mart Stores, Inc.*, 2000 U.S. App. LEXIS 20932, *1-2 (4th Cir. Aug. 17, 2000) (same).

Take-home exposure and exposure of persons not using PPE are equally reasonably foreseen.

[56] During the SACC meeting on methylene chloride, EPA's Dr. Barone stated that "spills are not a condition of use." We disagree; spills constitute a "reasonably foreseen *** circumstance under which a chemical is manufactured, processed, distributed, used, or disposed of" and lead to exposures that need to be considered in a risk evaluation.

3. EPA is justified in adopting a linear, no-threshold approach for TCE's carcinogenicity.

A. There is strong support for TCE's cancer classification and a mutagenic mode of action for kidney cancer.

In the draft risk evaluation, EPA correctly concludes that TCE is linked to non-Hodgkin's lymphoma, kidney, and liver cancer (see section 3.2.3.2, pp. 218-219 and section 3.2.4.2, pp. 225-226).

EPA's decision to affirm TCE's carcinogenicity and carry forward cancer hazard for dose-response modeling is wholly consistent with numerous other classifications:

- The International Agency for Research on Cancer (IARC) has stated that TCE is "*carcinogenic to humans (Group 1)*," based on sufficient evidence in both humans and experimental animals.[57]
- The National Toxicology Program's (NTP) *Report on Carcinogens* concluded that TCE is "*known to be a human carcinogen* based on sufficient evidence of carcinogenicity from humans."[58]
- EPA's IRIS program has classified TCE as "carcinogenic to humans by all routes of exposure."[59]

The Agency's conclusion in the draft risk evaluation is also aligned with the EPA's 2014 Work Plan Chemical Risk Assessment of TCE as well as the recent 2019 ATSDR Toxicological Profile for TCE.[60]

Moreover, EPA appropriately concludes that TCE is genotoxic, stating "there is sufficient evidence that TCE-induced kidney cancer operates primarily through a mutagenic mode of action" (p. 30). EPA also states (p. 227):

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[57] IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. "Trichloroethylene, Tetrachloroethylene, and Some Other Chlorinated Agents." *IARC monographs on the evaluation of carcinogenic risks to humans* 106 (2014).

[58] Natl'l Toxicology Program, *Report on Carcinogens Monograph for Trichloroethylene*, https://ntp.niehs.nih.gov/ntp/roc/monographs/finaltce_508.pdf

[59] EPA IRIS website, https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0199tr/0199tr.pdf.

[60] Agency for Toxic Substances and Disease Registry (ATSDR). 2019. Toxicological Profile for Trichloroethylene (TCE). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, available at https://www.atsdr.cdc.gov/ToxProfiles/tp.asp?id=173&tid=30.

> The predominant mode of action (MOA) for kidney carcinogenicity involves a genotoxic mechanism through formation of reactive GSH metabolites (e.g., DCVC, DCVG). This MOA is well-supported, as toxicokinetic data indicates that these metabolites are present in both human blood and urine, and these metabolites have been shown to be genotoxic both in vitro and in animal studies demonstrating kidney specific genotoxicity (U.S. EPA, 2011e).

Importantly, the Agency also states (p. 256) that while there is some evidence for the involvement of cytotoxicity and regenerative proliferation, these processes do not have:

> the extent of support as for a mutagenic mode of action. In particular, data linking TCE-induced proliferation to increased mutation or clonal expansion are lacking, as are data informing the quantitative contribution of cytotoxicity. Because any possible involvement of a cytotoxicity mode of action would be additional to mutagenicity, the dose-response relationship would nonetheless be expected to be linear at low doses. Therefore, the additional involvement of a cytotoxicity mode of action does not provide evidence against the use of linear extrapolation from the POD.

This conclusion regarding a lack of evidence for alternative MOAs is also consistent with other findings of authoritative agencies. For example, IARC determined that "[t]he data supporting the non-genotoxic mechanisms of kidney carcinogenesis were limited."[61]

Overall, given the strong evidence for TCE's carcinogenicity and mutagenicity, we strongly support the Agency's decision to adhere to the *EPA Guidelines for Carcinogen Risk Assessment* and use the approach of linear non-threshold extrapolation in the cancer risk modeling for TCE.

B. The scientifically sound and health-protective approach is to use linear extrapolation in cancer dose-response modeling for TCE.

i. Justification based on existing guidance

The information presented above: 1) demonstrates that the evidence supports a genotoxic MOA for TCE, and 2) casts doubt on the plausibility of alternative MOAs. Even were the evidence deemed insufficient to identify with certainty a genotoxic MOA, there is longstanding EPA policy guidance and precedent supporting a default to a no-threshold, linear extrapolation method for cancer dose-response modeling.

[61] IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. "Trichloroethylene, Tetrachloroethylene, and Some Other Chlorinated Agents." *IARC monographs on the evaluation of carcinogenic risks to humans* 106 (2014).

The Agency's own 2005 cancer guidelines state that:

> When the weight of evidence evaluation of all available data are insufficient to establish the mode of action for a tumor site and when scientifically plausible based on the available data, linear extrapolation is used as a default approach, because linear extrapolation generally is considered to be a health-protective approach.[62]

EPA must follow its guidance documents in preparing the final risk evaluation. "An agency may not … depart from a prior policy *sub silentio* or simply disregard rules that are still on the books." *FCC v. Fox TV Stations, Inc.*, 556 U.S. 502, 515 (2009). EPA's guidance documents reflect the considered judgment of the Agency on major factual issues, and an Agency may not lightly adopt new policies reflecting contradictory factual findings without providing a detailed justification for the shift in position. *Id.* Moreover, EPA's Risk Evaluation Rule provides that "EPA guidance will be used, as applicable where it represents the best available science appropriate for the particular risk evaluation." 40 C.F.R. § 702.41(a)(2). Thus, EPA must use its guidance in this risk evaluation unless EPA can establish that the guidance does not represent the best available science appropriate for this particular risk evaluation.

ii. Justification based on human population variability and other real-world considerations to protect public health

EPA must employ health-protective approaches to dose-response modeling, as described at length in the National Research Council's report, *Science and Decisions: Advancing Risk Assessment.* In this report, the NRC committee specifically provides important perspective on the need to conduct a linear extrapolation at the population level, even where a threshold might theoretically exist. The authors state, for example, that: [63]

- "Human variability with respect to the individual thresholds for a nongenotoxic cancer mechanism can result in linear dose-response relationships in the population."
- "In the laboratory, nonlinear dose-response processes … may be found to cause cancer in test animals. However, given the high prevalence of these background processes, given cancer as an end point, and given the multitude of chemical exposures and high variability in human susceptibility, the results may still be manifested as low-dose linear dose-response relationships in the human population."

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[62] U.S. EPA, *Guidelines for Carcinogen Risk Assessment* at pp. 3-21 (Mar. 2015), https://www.epa.gov/sites/production/files/2013-09/documents/cancer_guidelines_final_3-25-05.pdf.

[63] NAT'L RESEARCH COUNCIL, SCIENCE AND DECISIONS: ADVANCING RISK ASSESSMENT at chp. 5, pp. 130-131 (2009), https://www.ncbi.nlm.nih.gov/pubmed/25009905.

Overall, the NRC report recommended that "cancer and noncancer responses [to chemical exposures] be assumed to be linear as a default."[64] The NRC committee called for a unified approach using linear extrapolation to account for both background exposures and the wide range of variability in individual susceptibility. It argued that this approach also improves the risk characterization and ultimately risk management decisions by providing quantification of excess population risk rather than a margin of exposure.

In their *State-of-the-science workshop report: issues and approaches in low-dose-response extrapolation for environmental health risk assessment*, White et al. 2009[65] also highlight that (emphasis added):

> At the human population level *** biological and statistical attributes tend to *smooth and linearize the dose-response relationship, obscuring thresholds that might exist for individuals*. Most notable of these attributes are population variability, additivity to preexisting disease or disease processes, and background exposure-induced disease processes.

The 2016 amendments to TSCA made explicit and strengthened EPA's obligation to consider risks to and protect subpopulations that may be more exposed or more susceptible to the effects of chemical exposure than the general population. To meet this statutory requirement, EPA must use a linear non-threshold modeling approach.

In summary, given 1) existing Agency guidance, 2) the many sources of variability in the human population, 3) TSCA's mandate to protect "potentially exposed or susceptible subpopulations," and 4) the clear presence of individuals with preexisting health conditions, metabolic or genetic variability, or other factors that make them more susceptible to methylene chloride exposure (see, for example, pp. 275, 386), the use of the linear extrapolation is the only appropriate option for cancer dose-response modeling. EPA also must use this approach to cancer dose-response modeling to comply with EPA's duty to consider the "best available science" under TSCA § 26(h).

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[64] *Ibid.* at chp. 5, p. 180.

[65] Ronald H. White, et al., *State-of-the-science workshop report: issues and approaches in low-dose–response extrapolation for environmental health risk assessment*, 117:2 ENVTL. HEALTH PERSPECTIVES p. 284 (2009), https://www.ncbi.nlm.nih.gov/pubmed/19270800.

4. EPA's human health hazard assessment raises significant questions and concerns, while exhibiting positive improvements in a few areas.

A. EPA has failed to include any estimate of acute cancer risks.

Despite EPA's acknowledgment that the weight of the scientific evidence indicates TCE is a mutagenic carcinogen and that linear extrapolation is warranted (p. 30), the Agency has chosen not to estimate cancer risks based on acute exposures. As an explanation, EPA states that the "extrapolation of lifetime theoretical excess cancer risks to single exposures has great uncertainties" and that "the relationship between a single short-term exposure to TCE and the induction of cancer in humans has not been establish in the current scientific literature (p. 251).

However, the National Research Council (NRC) states (emphasis added):[66]

> Guidance on the development of short-term exposure levels, published by the NRC, identified cancer as one of the potential adverse health effects that might be associated with short-term inhalation exposures to certain chemical substances (NRC 1993a). That guidance document discusses and recommends specific risk-assessment methods for known genotoxic carcinogens and for carcinogens whose mechanisms are not well understood. As a first approximation, *the default approach involves linear low-dose extrapolation from an upper confidence limit on theoretical excess risk*. Further, the NRC guidance states that *the determination of short-term exposure levels will require the translation of risks estimated from continuous long-term exposures to risks associated with short-term exposures*. Conceptually, the approach recommended for genotoxic carcinogens adopted the method developed by Crump and Howe (1984) for applying the linearized multistage model to assessing carcinogenic risks based on exposures of short duration.

Later in the same document (p. 118), the NRC summarizes: "Guidance published by the NRC (1993a) states that the setting of AEGLs (CEELs) [acute exposure guideline levels (for what are termed "community emergency exposure levels")] should involve linear low-dose extrapolation from an upper confidence limit on excess risk for genotoxic carcinogens."

As stated in this NRC report, the decision to conduct such extrapolation and modeling should be based on the "sound biological and statistical principles." EDF is concerned that EPA did not

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[66] Nat'l Research Council, *Standard operating procedures for developing acute exposure guideline levels for hazardous chemicals*, pp. 111-112 (2001), https://www.epa.gov/aegl/standing-operating-procedures-developing-acute-exposure-guideline-levels-aegls-hazardous.

sufficiently consider such principles related to mode-of-action in deciding not to model acute cancer risk based on chronic exposure data. In particular, given that 1) the Agency recognizes that "there is sufficient evidence that TCE-induced kidney cancer operates primarily through a mutagenic mode of action" (p. 233) and 2) a mutagenic MOA suggests a role for "a single direct reaction, specifically, a single hit in a single target (Kirsch-Volders et al., 2000),"[67] a linear low-dose extrapolation from chronic to acute exposures would be the appropriate approach to take for TCE.

It is possible, though, that even a linear extrapolation from chronic cancer bioassays may underestimate the cancer risk of short-term exposures. Halmes et al. 2000 lends support to the potential for short-term exposures to result in similar or higher cancer risks than even chronic lifetime exposures.[68] The study used NTP data where both shorter term and full lifetime studies had been conducted.

EPA's current approach assumes acute exposures to TCE, including to consumers, pose *zero* cancer risk – an assumption that is clearly not warranted based on the weight of the evidence. EPA needs to apply an extrapolation that provides a scientifically sound estimate for cancer risk from acute and short-term exposures to TCE. As EDF stated in our comments on EPA's problem formulations (p. 81):

> EPA must closely examine any effect it believes to arise only from chronic exposures to determine whether in fact this is true across the diverse human population, including where potentially exposed or susceptible subpopulations may be at increased risk for effects after shorter periods of exposure compared to the general population.

B. Several critical toxicokinetic issues are raised but not all are sufficiently addressed in the draft risk evaluation.

The draft risk evaluation includes several toxicokinetic considerations that bear directly on characterizations and estimates of TCE risk, identified and discussed below.

i. Absorption via the inhalation and dermal routes

EPA appropriately assumes 100% absorption of TCE via the inhalation and occluded dermal routes. The 2011 IRIS toxicological review of TCE provides extensive scientific support and

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[67] Zoë Gillespi, et al., *Risk assessment approaches for carcinogenic food contaminants*, 1:1 INT'L FOOD RISK ANALYSIS J. 1-18 (2011), https://hrcak.srce.hr/file/107066.

[68] N. Christine Halmes, et al., *Reevaluating cancer risk estimates for short-term exposure scenarios*, 58:1 TOXICOLOGICAL SCIENCES 32-42 (2000), https://academic.oup.com/toxsci/article/58/1/32/1658920/#24341943.

discussion for these assumptions (see sections 3.1.2 and 3.1.3 of the IRIS toxicological review).[69]

ii. Important metabolic differences across the human population

EPA acknowledges important pathways—cytochrome P450 (CYP) oxidation pathway and glutathione (GSH) conjugation pathway—that are involved in TCE metabolism and lead to the generation of known toxic metabolites including dichloroacetic acid (DCA) and trichloroacetic acid (TCA) (pp. 204-207). The Agency further acknowledges variability across the human population with regard to these pathways:

> Significant variability may exist in human susceptibility to TCE given the existence of CYP isoforms and the variability in CYP-mediated TCE oxidation. (p. 204)
>
> Compared to the CYP oxidation pathway, there appear to be more significant sex and species differences in TCE metabolism via the GSH pathway. (p. 205)
>
> Significant variability in human susceptibility to TCE toxicity may result from differences in metabolic potential, given the existence of CYP isoforms and the variability in CYP-mediated TCE oxidation. Increased enzymatic activity of cytochrome P450 2E1 (CYP2E1) and glutathione-S-transferase (GST) polymorphisms may influence TCE susceptibility due to effects on the production of toxic metabolites. (p. 234)

EPA's PBPK model attempts to account for these metabolic differences (see for example section 3.2.2.1, pp. 206-209). However, acknowledged data gaps introduce uncertainty regarding the extent to which the PBPK model sufficiently addresses these important variabilities that in part contribute to differential susceptibility across individuals:

> In general, an attempt was made to use tissue-specific dose-metrics representing particular pathways or metabolites identified from reasonably available data on the role of metabolism in toxicity for each endpoint….The selection was limited to dose metrics for which uncertainty and variability could be adequately characterized by the PBPK model. For most endpoints, sufficient information on the role of metabolites or mode of action was not available to identify likely relevant dose metrics… (p. 206)

[69] EPA, IRIS Toxicological Review of TCE (2011) available at https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0199tr/0199tr.pdf.

EPA should more fully address the extent to which the PBPK model addresses the acknowledged uncertainty and whether it does so in a manner that is health-protective, including specifically for susceptible populations.

iii. Incorporation of pregnancy in the PBPK model

EPA states that "[f]or developmental toxicity endpoints, the TCE PBPK model did not incorporate a pregnancy model to estimate the internal dose of TCE in the developing fetus" (p 207). In the recent NASEM *Review of the Department of Defense's Approach to Deriving an Occupational Exposure Level for Trichloroethylene*, the review committee raised a concern on the same topic:[70]

> Specifically, the PBPK model does not have a component designed for pregnant or lactating animals, which is a potential limitation for internal dose metric determinations for pregnant or nursing mothers exposed to TCE in the workplace. PBPK models have been developed for pregnant rats, lactating rats, and nursing pups (e.g., Fisher et al. 1989, 1990). DOD should improve transparency in its choice of the PBPK model by discussing the reasons for, and potential impact of, not accounting for anatomic, physiologic, and metabolic changes that vary between species during pregnancy and nursing of infants.

At a minimum, EPA should explicitly discuss, with supporting evidence, the implications of deriving points of departure and risk estimates using a PBPK model that lacks a pregnancy component. As part of this discussion, EPA should describe how the protection of vulnerable populations, including the developing fetus, is ensured given EPA's reliance on an existing PBPK model that does not incorporate a pregnancy component.

C. EPA's weight of evidence approach for congenital heart defects raises questions.

i. EPA's weight of evidence (WOE) criteria raises concerns.

In Appendix G.2 (p. 611), EPA indicates its selection of the Risk Assessment Forum's Weight of Evidence in Ecological Assessment approach to apply to the evidence base for congenital heart defects. EPA uses applies this structured approach only for the congenital heart defect endpoint and uses a narrative summary in developing a weight of the scientific evidence for all other endpoints. As discussed in EDF's previously submitted comments on the TSCA systematic

[70] National Academies of Sciences, Engineering, and Medicine. 2019. *Review of DOD's Approach to Deriving an Occupational Exposure Level for Trichloroethylene*. Washington, DC: The National Academies Press, p. 43. https://doi.org/10.17226/25610.

review method,[71] EPA has yet to develop or articulate an approach to evidence integration and as such introduces risks for bias and inconsistency within and across risk evaluations.

For the TCE draft risk evaluation, EPA explains it has chosen a particular, structured approach to weight of the scientific evidence for congenital heart defects because of "conflicting results of the previous WOE assessments" (p. 223), including a problematic Wikoff et al. 2018 publication (see subsection D.vii, below). It then explains that "[a]fter reviewing a sampling of recent literature on systematic approaches to performing weight-of-evidence evaluation, EPA adopted the methodology described in [Weight of Evidence in Ecological Assessment. Risk Assessment Forum. EPA/100/R16/00] which advocates presenting the evidence on a semiqualitative scale…." (p. 223). EPA's explanation for the selection of this particular methodology is virtually non-existent and raises many questions – including whether and when the Agency will apply this method in future risk evaluations, and the extent to which EPA considered more prominent GRADE-based structured frameworks for evidence integration used by analogous chemical assessment approaches (i.e., National Toxicology Program OHAT health effects evaluations, UCSF Navigation Guide, EPA IRIS assessments). Setting aside these larger issues, the methodology EPA has chosen raises concerns.

The description of the methodology cites three main areas as the basis for scoring of evidence: reliability (quality), relevance, and strength. EPA highlights the strength criterion as a distinguishing feature of this approach, and further explains in a parenthetical that the strength of a given piece of evidence corresponds to its "magnitude, dose-response, etc." We are concerned with the inclusion of effect "magnitude" as a criterion for consideration, as "magnitude" could be interpreted either as the fraction of the affected population, or the effect size of the change in a measure of outcome. No definition is provided by EPA. Either way, an effect with a small "magnitude" either may affect a considerable fraction of the exposed population or could be sufficiently severe to warrant concern. Accordingly, we would advise caution in discounting evidence from well-designed, relevant studies with a small magnitude.

ii. EPA inappropriately combined exposure routes in its evidence integration for congenital heart defects endpoint

In the weight of evidence analyses for congenital heart defects presented in Appendix G.2, in the section for *in vivo* studies, EPA jointly considered the evidence for oral and inhalation studies in animals (pp. 620-621). When considered independently, the oral studies had an integrated area score of (+), whereas the inhalation studies had an integrated area score of (-). Taken together, EPA assigned the *in vivo* studies via all routes a (0), which impacts the overall evidence integration for the endpoint (the quantitative nature of its impact is unclear for this semi-

[71] EDF Comment on EPA OPPT's Application of Systematic Review in TSCA Risk Evaluations, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2018-0210-0077.

quantitative integration approach). It is not appropriate to consider the oral and inhalation routes together in this circumstance. Given potential differences in toxicokinetics and metabolism across routes, it is certainly plausible that oral exposures are associated with the endpoint while inhalation exposures are not. With this in mind, EPA should have additionally conducted WOE analyses separately by route. Had the Agency done this, the *in vivo* toxicity studies score would have been higher, which would have likely increased the overall Integrated Area Score and summary score. In the revised version of this draft risk evaluation, EPA should evaluate these data separately by route.

D. EPA's reliance on immune-related endpoints, instead of congenital heart defects, for its determinations of acute and chronic risk deviates from scientific best practices, defies requirements under the law, and is not sufficiently protective of public health.

In contrast to previous Agency assessments of TCE that characterized the chemical's risks in whole[72] or in part[73] based on congenital heart defects, the most sensitive endpoint, EPA has instead based its determinations of acute and chronic unreasonable risks on immune-related endpoints.

Notably, EPA agrees the scientific evidence supports TCE-induced congenital heart defects (p. 225):

> Overall, an association between increased congenital cardiac defects and TCE exposure is supported by the weight of the evidence, in agreement with previous EPA analyses (U.S. EPA, 2014b; Makris et al., 2016). Therefore, this endpoint was carried forward for dose-response analysis.

Thus, EPA's decision to make non-cancer risk determinations based on immune-related endpoint is counter to the preponderance of scientific evidence demonstrating TCE induces congenital heart defects. EPA's decision to base risk determinations on immune-related endpoints reflects is directly at odds with decades of scientific policy and practice, statutory requirements to protect

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[72] TSCA Work Plan Chemical Risk Assessment. Trichloroethylene: Degreasing, Spot Cleaning and Arts & Crafts Uses. 2014. Available at: https://www.epa.gov/sites/production/files/2014-11/documents/tce_opptworkplanchemra_final_062414.pdf.

[73] 2011 EPA IRIS Assessment. The IRIS RfD for TCE was derived as the midpoint among candidate RfDs for three other endpoints: decreased thymus weight at 4.8x10^-4 mg/kg-day (Keil et al. 2009), developmental immunotoxicity at 3.7x10^-4 mg/kg-day (Peden-Adams et al. 2006), and congenital heart defects at 5.1x10^-4 (Johnson et al. 2003). Similarly, the IRIS RfC for TCE was derived as the midpoint among candidate RfCs for two other endpoints: decreased thymus weight at 1.9x10^-3 mg/m^3 (Keil et al. 2009) and congenital heart defects at 2.1 mg/m^3 (Johnson et al. 2003).

potentially exposed and susceptible subpopulations and the Agency's mission to protect human health (see subsection G. below).

The rationale EPA provides for making risk determinations based on immune-related endpoints raises significant concerns. In section 3.2.6.4, EPA defends its choice of the selected acute non-cancer endpoint, "mortality due to immunosuppression" as observed in Selgrade and Gilmour 2010, and its selected chronic non-cancer endpoint, "autoimmunity" as observed in Keil et al. 2009, based on its rating of the referenced studies as "High" quality per the TSCA systematic review method, whereas EPA rated the study used in previous Agency assessments to derive a point of departure (POD) and make risk determinations,[74] Johnson et al. 2003, as "Medium" quality.

However, these ratings are based on the fundamentally flawed OPPT TSCA systematic review method that is currently, after long delays on EPA's part, undergoing peer review by the National Academies of Science, Engineering, and Medicine (NASEM).[75] Those flaws include the lack of any empirical support for the scoring system devised, the use of numerical scores to characterize study quality as a general matter, and the lack of a defined procedure for data integration among others (see section 8 of these comments and EDF's previously submitted comments on the TSCA systematic review method[76]). It is also worth noting that the earlier Dawson et al. 1993 study that reported on two TCE dose groups that were included in the Johnson et al. 2003 study had initially received a rating of High, but that rating was downgraded to Medium based on the study evaluator's professional judgment.[77]

EPA's scientifically unsupported and contradictory decision results in EPA relying its risk determinations on risk estimates across various TCE exposure scenarios that are orders of magnitude more lax than those risks estimates associated with the most sensitive endpoint, congenital heart defects.

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[74] TSCA Work Plan Chemical Risk Assessment. Trichloroethylene: Degreasing, Spot Cleaning and Arts & Crafts Uses. 2014. Available at: https://www.epa.gov/sites/production/files/2014-11/documents/tce_opptworkplanchemra_final_062414.pdf.

[75] Webpage for NASEM peer review of EPA OPPT TSCA systematic review method available at https://www.nationalacademies.org/our-work/review-of-epas-tsca-systematic-review-guidance-document.

[76] EDF Comment on EPA OPPT's Application of Systematic Review in TSCA Risk Evaluations, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2018-0210-0077.

[77] EPA, Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies – Animal and Mechanistic Data, February 2020, p. 231, available at https://www.epa.gov/sites/production/files/2020-02/documents/14_tce-data_quality_evaluation_of_human_health_hazard_studies_-_animal_and_mechanistic_data.pdf.

The comments that follow address multiple facets of EPA's TCE hazard characterization as it relates to congenital heart defects, and EPA's unprecedented and unsupported decision not to use this endpoint to reach determinations of risk, which ultimately leaves vulnerable Americans unprotected from the harms arising from TCE exposure.

i. Weight of the evidence supports TCE-induced congenital heart defects.

Under TSCA as amended by the Lautenberg Act, EPA is required to apply a weight of the scientific evidence approach to the evaluation of chemical risks. Multiple lines of evidence support the finding that congenital heart defects result from gestational exposure to TCE, including data from epidemiological, *in vivo*, and *in vitro* studies.

The 2011 EPA IRIS TCE assessment derived reference dose (RfD) and reference concentration (RfC) values in part based on congenital heart defects.[78,79] Following the IRIS assessment, EPA scientists conducted an additional review of TCE-induced cardiac malformations, partly due to the realization that short-term or peak exposures to TCE gestationally could result in adverse fetal outcomes. This review by Makris et al. was published in a peer-reviewed journal in 2016.[80] As with the IRIS assessment, Makris et al. applied a systematic approach to rigorously identify and evaluate the literature bearing on congenital heart defects resulting from gestational exposures to TCE, and applied a weight of the scientific evidence approach in drawing conclusions. The literature search spanned epidemiological, *in vivo*, and *in vitro* data. Drawing from this broad evidence base, the 2016 review affirmed the earlier IRIS determination that congenital heart defects occur following *in utero* exposure to TCE.

While defined MOAs are not required for hazard identification (see subsection D.iii., below), it should be noted that Makris et al. developed a preliminary Adverse Outcome Pathway (AOP) providing biological support for TCE-induced cardiac effects, specifically valvulo-septal defects, following developmental exposure. Comparing knockout mice with abnormalities in epithelial-

[78] EPA IRIS Assessment for Trichloroethylene. 2011. Available at: https://cfpub.epa.gov/ncea/iris2/chemicalLanding.cfm?substance_nmbr=199#tab-1

[79] It is worth noting that the IRIS RfD for TCE was derived as the midpoint among candidate RfDs for three other endpoints: decreased thymus weight at 4.8x10^-4 mg/kg-day (Keil et al. 2009), developmental immunotoxicity at 3.7x10^-4 mg/kg-day (Peden-Adams et al. 2006), and congenital heart defects at 5.1x10^-4 (Johnson et al. 2003). Similarly, the IRIS RfC for TCE was derived as the midpoint among candidate RfCs for two other endpoints: decreased thymus weight at 1.9x10^-3 mg/m^3 (Keil et al. 2009) and congenital heart defects at 2.1 mg/m^3 (Johnson et al. 2003).

[80] Makris, Susan L. "The systematic review of TCE cardiac defects (Makris et al. 2016)." *Reproductive toxicology (Elmsford, NY)* 71 (2017): 124.

mesencyhmal transition (EMT) – a key process underpinning valvulo-septal morphogenesis – yielded identification of phenotypes similar to those observed in avian TCE studies examining developmental exposure. Makris et al. indicate that additional mechanistic support for EMT disruption in TCE-induced cardiac malformation is provided by studies that reveal TCE associated "inhibition of cell-cell separation and mesenchymal formation, alterations in mesenchymal cell migration, and alterations in endocardial proliferation patterns."[81]

Based on their review of the evidence base regarding TCE-induced congenital heart defects, Makris et al. conclude:

> Despite 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 dose 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 result from TCE exposures. Limitations within the database that increase the uncertainties regarding this conclusion are acknowledged.[82]

Support for TCE-induced congenital heart defects based on weight of evidence considerations has also been provided by:

- the EPA Science Advisory Board (SAB) in its review of the IRIS TCE toxicological review,[83]
- an EPA TCE Developmental Cardiac Toxicity Assessment Update ("Update") following the publication of the IRIS toxicological review,[84] and

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[81] Makris, Susan L., et al. "A systematic evaluation of the potential effects of trichloroethylene exposure on cardiac development." *Reproductive Toxicology* 65 (2016): 321-358.
[82] Makris, Susan L., et al. "A systematic evaluation of the potential effects of trichloroethylene exposure on cardiac development." *Reproductive Toxicology* 65 (2016): 321-358. p. 345.
[83] EPA Scientific Advisory Board. Review of EPA's Draft Assessment entitled "*Toxicological Review of Trichloroethylene*" (October 2009) https://yosemite.epa.gov/sab/sabproduct.nsf/02ad90b136fc21ef85256eba00436459/B73D5D39A8F184BD85257817004A1988/$File/EPA-SAB-11-002-unsigned.pdf
[84] EPA. TCE Developmental Cardiac Toxicity Assessment Update. 2014. https://www.regulations.gov/document?D=EPA-HQ-OPPT-2012-0723-0045.

- EPA's response for a Request for Correction submitted by the Halogenated Solvents Industry Alliance regarding raising concerns regarding the Agency's reliance on Johnson et al. 2003.[85]

For example, the EPA SAB noted in its review, "The Panel found that the draft document adequately synthesizes the available scientific information to support a conclusion that TCE poses a potential human health hazard for non-cancer toxicity, including effects on…the developing fetus."[86]

The Update stated:

> The majority of the team members agreed that the overall evidence in the TCE database supports a conclusion that TCE is likely to cause cardiac defects at sufficient doses when exposure occurs during a sensitive period of fetal development. This conclusion was based upon the data that demonstrate or suggest a potential hazard to cardiac development, including epidemiology 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 proposed AOP.[87]

Finally, while much attention and debate has focused on *in vitro* and *in vivo* studies, it is important to note that additional support comes from the epidemiologic database. Throughout the history of EPA, human epidemiological studies have served as the "gold standard" for assessing population risks and guiding the Agency's efforts to protect public health and the environment. Epidemiologic studies provide information critical to understanding the causes of disease, factors influencing population susceptibility, and the actual levels of exposure at which health effects occur. Integration of evidence from epidemiologic, *in vivo* and *in vitro* studies can reduce uncertainties associated with each study design and allows for stronger scientific conclusions about risks.[88]

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[85] EPA. Response to Request for Correction by Halogenated Solvents Industry Alliance. 2015. https://www.epa.gov/sites/production/files/2015-10/documents/14001-response.pdf.

[86] EPA Scientific Advisory Board. Review of EPA's Draft Assessment entitled "*Toxicological Review of Trichloroethylene*" (October 2009) https://yosemite.epa.gov/sab/sabproduct.nsf/02ad90b136fc21ef85256eba00436459/B73D5D39A8F184BD85257817004A1988/$File/EPA-SAB-11-002-unsigned.pdf.

[87] EPA. TCE Developmental Cardiac Toxicity Assessment Update. 2014. P. 7. https://www.regulations.gov/document?D=EPA-HQ-OPPT-2012-0723-0045.

[88] Deener, K.C., et al., *Epidemiology: a foundation of environmental decision making*, 28:6 J. OF EXPOSURE SCIENCE & ENVTL. EPIDEMIOLOGY p. 515-521 (2018), https://www.ncbi.nlm.nih.gov/pubmed/30185947.

As one peer reviewer noted during the TCE SACC meeting, consistent epidemiological evidence exists for congenital heart defects and other birth defects and growth measures. Over several decades, research in the general population has found associations between a variety of TCE exposure metrics and some of the most severe and most common birth defects (central nervous system and neural tube defects, congenital heart defects, oral clefts) and growth measures such as small for gestational age and term low birth weight. These studies involved numerous communities (Woburn, MA, Endicott, NY, northern NJ, Camp Lejeune, NC, Milwaukee, WI and Tuscon, AZ), state registry studies (MA, TX), as well as a national birth defects prevention study.[89,90,91,92,93,94,95,96,97,98,99]

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[89] Bove, Frank J., et al. "Public drinking water contamination and birth outcomes." *American Journal of Epidemiology* 141.9 (1995): 850-862.

[90] Bove, Frank J. "Public drinking water contamination and birthweight, prematurity, fetal deaths, and birth defects." *Toxicology and industrial health* 12.2 (1996): 255-266.

[91] Brender, Jean D., et al. "Maternal residential proximity to chlorinated solvent emissions and birth defects in offspring: a case–control study." *Environmental Health* 13.1 (2014): 96.

[92] Forand, Steven P., Elizabeth L. Lewis-Michl, and Marta I. Gomez. "Adverse birth outcomes and maternal exposure to trichloroethylene and tetrachloroethylene through soil vapor intrusion in New York State." *Environmental health perspectives* 120.4 (2012): 616-621.

[93] Gilboa, Suzanne M., et al. "Association between maternal occupational exposure to organic solvents and congenital heart defects, National Birth Defects Prevention Study, 1997–2002." *Occup Environ Med* 69.9 (2012): 628-635.

[94] Goldberg, Stanley J., et al. "An association of human congenital cardiac malformations and drinking water contaminants." *Journal of the American College of Cardiology* 16.1 (1990): 155-164.

[95] Lagakos, Steven W., Barbara J. Wessen, and Marvin Zelen. "An analysis of contaminated well water and health effects in Woburn, Massachusetts." *Journal of the American Statistical Association* 81.395 (1986): 583-596.

[96] Yauck, Jennifer S., et al. "Proximity of residence to trichloroethylene-emitting sites and increased risk of offspring congenital heart defects among older women." *Birth Defects Research Part A: Clinical and Molecular Teratology* 70.10 (2004): 808-814.

[97] Wright JM, Evans A, Kaufman JA, Rivera-Nunez Z, Narotsky MG. Disinfection By-Product Exposures and the Risk of Specific Cardiac Birth Defects. Environ Health Perspect 2017 February 01; 125(2): 269-277.

[98] Ruckart PZ, Bove FJ, Maslia M. Evaluation of exposure to contaminated drinking water and specific birth defects and childhood cancers at Marine Corps Base Camp Lejeune, North Carolina: a case-control study. Environ Health 2013 December 04; 12: 104-104.

[99] Ruckart PZ, Bove FJ, Maslia M. Evaluation of contaminated drinking water and preterm birth, small for gestational age, and birth weight at Marine Corps Base Camp Lejeune, North Carolina: a cross-sectional study. Environ Health 2014 November 20; 13: 99-99.

No study is perfect and not every study reports all outcomes; but the consistency of developmental effects seen across this diversity of communities and types of studies gives substantial weight to this body of evidence. EPA must not "miss the forest for the trees." EPA must protect susceptible populations with dose-response data from the most sensitive developmental endpoint.

ii. Developing a POD based on the Selgrade & Gilmour study is not protective of public health and deviates from requirements of TSCA.

a. A POD based on Selgrade & Gilmour does not protect public health.

EPA uses "mortality due to immunosuppression" (p. 257) from Selgrade and Gilmour (2010), instead of congenital heart defects, as the representative acute non-cancer endpoint for POD modeling.

During the TCE SACC meeting, the panelists highlighted that using mortality to derive the POD results in an underestimation of sublethal effects. There are expected to be toxic effects on the immune system below the level that causes death. As such, this mortality endpoint is not expected to be sufficiently protective against more sensitive, sublethal endpoints across the population. More broadly, as summarized in subsection G. below, Agency guidance directs EPA to choose the most sensitive endpoint which is congenital heart defects in the case of TCE.

b. EPA deviates from TSCA's section 26 requirements in applying an inconsistent level of scrutiny to endpoints in support of decision-making regarding a POD.

On p. 377 of the draft risk evaluation, EPA justifies its selection of immunosuppression by noting that "public health is best served when EPA relies upon the highest quality information for which EPA has the greatest confidence." In the same paragraph, the Agency notes that "Section 26 requires that EPA make decisions consistent with the 'best available science,'" and, "Section 26 also requires other scientific considerations including consideration of the 'extent of independent verification' and 'weight of the scientific evidence.'"

In this context, EPA has strayed from the requirements of Section 26 of TSCA in relying on the quality of design for Selgrade and Gilmour (2010) as the basis for selecting immunosuppression. In the draft risk evaluation, EPA provides an extensive consideration of the weight of evidence supporting the congenital heart defects endpoint; so extensive, in fact, that it included a full, formal weight-of-evidence analysis as a 21-page appendix. In contrast, the immunosuppression endpoint does not receive anything close to a similar level of scrutiny: EPA provides less than a page of consideration in the weight-of-evidence section of the draft risk evaluation. The intensity of scrutiny placed on the Johnson et al. 2003 study in the draft risk evaluation is wholly inconsistent with the depth of examination of any other study in the database. As discussed by one peer reviewer during the TCE SACC meeting, had the same level of scrutiny been applied to

other studies, it is likely that additional concerns with them would have been identified. The narrow focus on congenital heart defects likely came at the expense of inadequate consideration of other endpoints, including that of immunosuppression

EPA's inequitable scrutiny of endpoints mirrors the errors in the US Department of Defense's (DOD) approach to developing an occupational exposure level for TCE, a flaw that was highlighted by the National Academies of Sciences, Engineering, and Medicine (NASEM) in a 2019 report:

> DOD gave special attention to evaluating the evidence on congenital heart defects, with particular scrutiny of a single study. The committee is aware that the data on this end point has been controversial, but found the emphasis on one study to be contrary to systematic review best practices. Importantly, DOD's study applicability tool does not seem to have been applied to this study, as it did not receive an applicability score, which indicates that the study was excluded from consideration earlier in the process. The basis for singling out one study for exclusion appears arbitrary, is not transparent, and is inconsistent with the process of how other studies were evaluated.[100]

With regard to the independent verification requirements of Section 26 of TSCA, there is as much evidence of independent verification for congenital heart defects as there is for immune effects, so EPA's rejection of congenital heart defects on this basis is inconsistent and not supportable. For example, the Agency notes on p. 220 of the draft risk evaluation that "[t]here are no other reasonably available studies that examined respiratory immunotoxicity, however this endpoint is consistent with other data on immunosuppression," although no supporting evidence is provided to bolster this point. In contrast, in the case of congenital heart defects endpoint, by EPA's own admission (p. 222), even the Halogenated Solvents Industry Alliance (HSIA)-funded Charles River Laboratories 2019 study provided a partial replication of and results consistent with the specific heart defects (ventricular septal defects) examined in both the Charles River and Johnson et al. 2003 studies. This, considered alongside positive epidemiologic and mechanistic evidence (as displayed in the weight of evidence summary presented in Table 3-6), makes a strong case for reproducibility and an integrated body of evidence that paints a consistent picture for congenital heart defects as a real, sensitive endpoint, protection against which is likely to be protective for other TCE-induced endpoints.

[100] National Academies of Sciences, Engineering, and Medicine. 2019. Review of DOD's approach to deriving an occupational exposure level for trichloroethylene, p. 4. Available: https://www.nap.edu/catalog/25610/review-of-dods-approach-to-deriving-an-occupational-exposure-level-for-trichloroethylene 17 April 2020].

This point was highlighted by several panelists during the TCE SACC meeting. Reviewers discussed the fact that conclusions from Selgrade & Gilmour represent data from a single study in a single species and that EPA provided no discussion of the weight of evidence of acute exposure to TCE and mortality due to immunosuppression. This imbalance is highly concerning. All endpoints should be evaluated based on the same standards and criteria.

iii. A conclusive Mode of Action (MOA) is not required for use of congenital heart defects in hazard identification.

We strongly support EPA's conclusion in the draft risk evaluation that "evidence of a single dominant MOA is not required in order for the data to support a plausible mechanism of TCE-induced congenital heart defects," particularly given that "teratogens may function through a multitude of pathways, often resulting in a constellation of effects" (p. 224). As discussed in the 2014 NAS *Review of EPA's Integrated Risk Information System (IRIS) Process*:[101]

> Organizing evidence around mechanism for chemicals on which only some human or animal data are available, however, seems inappropriate. Consider the Food and Drug Administration (FDA) and drug safety. If FDA were required to organize drug safety around mechanism, it would be nearly impossible to regulate many important drugs because the mechanism is often not understood, even for drugs that have been studied extensively. For example, it is known that estrogen plus progestin therapy causes myocardial infarctions on the basis of randomized clinical trials even though the mechanism is not understood (Rossouw et al. 2002). Randomized clinical trials are so successful partly because they bypass the need for mechanistic information and provide an indication of efficacy. Similarly, epidemiologic studies that identify unintended effects are often credible because explanations of an observed association other than a causal effect are implausible. For example, the associations between statins and muscle damage and between thalidomide and birth defects are widely accepted as causal; mechanistic information played a minor role in the determination, if any. The history of science is replete with solid causal conclusions in advance of solid mechanistic understanding.

In the same way, the specification of MOAs for TCE-induced congenital heart defects is not required to make this hazard determination nor to conduct subsequent dose-response analysis and point of departure derivation for this endpoint. The weight of the scientific evidence for congenital heart defects is robust, with corroborating data across mechanistic, animal, and human

[101] National Research Council. 2014. *Review of EPA's Integrated Risk Information System (IRIS) Process*. Washington, DC: The National Academies Press, p. 90. https://doi.org/10.17226/18764.

studies. A requirement that a MOA must be defined to legitimize this evidence is both unscientific and unprotective of public health.

In this case, it is important also to note that strong mechanistic support for TCE-induced congenital heart defects is available, as described by Makris et al. 2016 (see subsection D.i.) and others, some of whom have specifically found evidence of non-monotonicity.[102,103] Further relevant information is provided in subsection D.iv. below.

iv. Additional mechanistic support exists for TCE-induced congenital heart defects.

EPA appropriately conducted an additional literature search to supplement the database of information on congenital heart defects (p. 223). However, in addition to the Harris et al. 2018 mechanistic study identified by the Agency, there are other relevant studies supporting a mechanistic linkage between TCE and congenital heart defects that EPA should have considered and must consider prior to finalizing the TCE risk evaluation:

> Caldwell, Patricia T., et al. "Gene expression profiling in the fetal cardiac tissue after folate and low-dose trichloroethylene exposure." Birth Defects Research Part A: Clinical and Molecular Teratology 88.2 (2010): 111-127.
>
> Selmin O.I., Makwana O., Runyan R.B. (2014) "Environmental Sensitivity to Trichloroethylene (TCE) in the Developing Heart." In: Gilbert K., Blossom S. (eds) Trichloroethylene: Toxicity and Health Risks. Molecular and Integrative Toxicology. Springer, London.
>
> Jin, Hongmei, et al. "AHR-mediated oxidative stress contributes to the cardiac developmental toxicity of trichloroethylene in zebrafish embryos." Journal of hazardous materials 385 (2020): 121521.
>
> Chen, Sheri, et al. "HNF4a transcription is a target of trichloroethylene toxicity in the embryonic mouse heart." *Environmental Science: Processes & Impacts* (2020).

[102] Runyan, R.B., Selmin, O.I., Smith, S.M. and Freeman, J.L. (2019), Letter to the Editor. Birth Defects Research, 111: 1234-1236. doi:10.1002/bdr2.1573.

[103] Chen, Sheri, et al. "HNF4a transcription is a target of trichloroethylene toxicity in the embryonic mouse heart." *Environmental Science: Processes & Impacts* (2020).

v. EPA has repeatedly examined TCE-induced congenital heart defects and the use of Johnson et al. 2003 specifically for determining TCE hazard and risk, concluding the evidence to be scientifically robust and Johnson et al. 2003 to be appropriate for the derivation of toxicity values and risk estimates.

In 2014, EPA's Office of Chemical Safety and Pollution Prevention (OCSPP) conducted a Work Plan Chemical Risk Assessment of TCE focused on the chemical's use in degreasing, spot cleaning, and arts and crafts. The assessment relied on Johnson et al. 2003 to derive toxicity values for the congenital heart defect endpoint. EPA's IRIS assessment also used Johnson et al. 2003 to support derivation of a reference dose (RfD) and reference concentration (RfC) for TCE.

Industry groups have repeatedly challenged EPA's use of Johnson et al. 2003, pointing to certain limitations in study design and reporting, several of which were addressed by the study's authors in errata published since the original publication. Limitations of the Johnson et al. 2003 study have been repeatedly discussed and addressed by the Agency in a number of TCE assessment--related documents:

- IRIS Toxicological Review of TCE (2011)[104]
- TCE Developmental Cardiac Toxicity Assessment Update ("Update") (2014)[105]
- TCE work plan risk assessment (2014)[106]
- TCE work plan risk assessment response to comments (2014)[107]
- Response to HSIA response for correction (2015)[108]
- Response to HSIA response for correction (2016)[109]
- Response to HSIA request for reconsideration (2016)[110]

[104] EPA. IRIS Toxicological Review of TCE. 2011. https://www.epa.gov/iris/supporting-documents-trichloroethylene.

[105] EPA. TCE Developmental Cardiac Toxicity Assessment Update. 2014. https://www.regulations.gov/document?D=EPA-HQ-OPPT-2012-0723-0045.

[106] EPA. TCE Work Plan Chemical Risk Assessment. 2014. https://www.epa.gov/sites/production/files/2014-11/documents/tce_opptworkplanchemra_final_062414.pdf.

[107] EPA. TCE Work Plan Chemical Risk Assessment response to comments. 2014. https://www.regulations.gov/document?D=EPA-HQ-OPPT-2012-0723-0039.

[108] EPA. Response to Halogenated Solvents Industry Association (HSIA) response for correction. 2015. https://www.epa.gov/sites/production/files/2015-10/documents/14001-response.pdf.

[109] EPA. Response to HSIA response for correction. 2016. https://www.epa.gov/sites/production/files/2016-12/documents/epa_response_to_rfc_16001.pdf.

[110] EPA. Response to HSIA request for reconsideration. 2016. https://www.epa.gov/quality/epa-response-rfr-14001a-issued-02262016-0.

Throughout the course of developing prior TCE assessments, EPA extensively and repeatedly reviewed, discussed, and resolved limitations associated with the Johnson et al. 2003 study, in each case concluding that that the study is sufficient for hazard identification and dose-response analysis. For example, in the Update, the Agency notes:

> On the whole, a majority of the team members agreed that Johnson et al. (2003) is suitable for use in deriving a point of departure. The study has an appropriate design for dose-response analysis in terms of route, duration, and number dose groups. Additionally, this judgment also took into consideration the strengths and limitations of the study and uncertainties identified in the weight of evidence analysis. Additional support was derived from the finding of a robust, statistically significant dose-response relationships [*sic*] not only for the dataset as a whole, but also for various subsets of the dataset. Although some concern was raised regarding the plateau in the Johnson et al. (2003) response, its biological plausibility could not be ruled out based on examination of historical developmental toxicity datasets.[111]

The earlier peer review by EPA's Scientific Advisory Board of the IRIS TCE toxicological review drew a similar conclusion:

> The report [IRIS Assessment] explains logically why the Johnson et al. (2003) study was used to derive some reference points. Some recent publications confirm and reinforce the results obtained in the Johnson et al. (2003) study and could be cited to make a stronger argument.[112]

In sum, the science issues associated with the Johnson et al. 2003 study have been amply vetted and peer-reviewed and should be considered resolved. See Appendix 4 for a brief history of peer reviews associated with previous EPA assessments of TCE: EPA 2014 TCE work plan assessment and 2011 IRIS Toxicological Review of TCE.

Furthermore, during the TCE SACC meeting, several SACC members countered potential criticisms of key aspects of the Johnson et al. study, arguing that they did not invalidate overall study conclusions. For example, with respect to the fact that the study

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[111] EPA. TCE Developmental Cardiac Toxicity Assessment Update. 2014. https://www.regulations.gov/document?D=EPA-HQ-OPPT-2012-0723-0045

[112] EPA Scientific Advisory Board. Review of EPA's Draft Assessment entitled "*Toxicological Review of Trichloroethylene*" (October 2009) https://yosemite.epa.gov/sab/sabproduct.nsf/02ad90b136fc21ef85256eba00436459/B73D5D39A8F184BD85257817004A1988/$File/EPA-SAB-11-002-unsigned.pdf

combined experimental data across several years, one SACC member noted that this practice (i.e., pooling) is common and well-accepted in epidemiological studies. Another SACC member indicated that observing the same effects several years apart is similar to replication and therefore should be viewed as a strength. Historically, another area of industry concern about the Johnson et al. 2003 study was the use of tap water as the negative control in one period of the experiment but the use of distilled water in another period. However, one SACC member stated that this difference does not matter unless it can be demonstrated that one of these two types of water is directly causing the cardiac effects, which is highly unlikely. Likewise, another SACC member highlighted that there is no plausible mechanism by which tap water or distilled water would be responsible for cardiac defects.

Finally, it is important to recognize that Johnson et al. 2003 is wholly consistent with the findings of many other studies—including human, *in vitro* and *in vivo* studies—that also indicate congenital heart defects resulting from TCE exposure; these were extensively reviewed by Makris et al. 2016 and recently revisited by Runyan et al. 2019. These findings are also addressed throughout section 4 of these comments. The Agency must continue to assess and determine risks for this endpoint in light of the entire body of scientific evidence as required by its own regulations for risk evaluation.

vi. DeSesso et al. 2019 does not negate the body of evidence supporting TCE-induced cardiac malformations and itself presents methodological shortcoming and unsupported conclusions.

DeSesso et al. 2019, a publication based on the Charles River Laboratories (2019) study sponsored by the American Chemistry Council and the Halogenated Solvents Industry Alliance, attempts to refute the evidence for TCE-induced congenital heart defects by critiquing Johnson et al. 2003. Specifically, DeSesso et al. 2019 reviews the Charles River Laboratories rodent developmental toxicity study of TCE delivered via drinking water—the same general study design as Johnson et al. 2003—where the authors report that no congenital heart defects were observed, and also critiques a single *in ovo* study by Boyer, Finch, and Runyan 2000.

Beyond ignoring the much broader and extensive literature dealing with TCE-induced congenital heart defects, the Charles River Laboratories study has multiple shortcomings such that it cannot be used as a basis to negate the findings of Johnson et al. 2003. EPA's draft risk evaluation appropriately notes:

> While the results of the Charles River study (2019) results appear to contradict the results observed by (Johnson et al., 2003) and (Dawson et al., 1993), EPA concludes that that Charles River study methodology was likely of reduced

> sensitivity and therefore does not entirely replicate the study conditions of those earlier studies. (p. 222)

It is also worth emphasizing that while DeSesso et al. states that no congenital heart defects were observed, the Charles River Laboratories study (2019) did in fact identify cardiac effects; the study authors ignore them by erroneously deeming the observed effects to be insignificant (see subsection D.vi.e. below).

a. DeSesso et al. 2019 ignores the weight of the scientific evidence supporting congenital heart defects by focusing exclusively on a single study, Johnson et al. 2003.

DeSesso et al. 2019 singularly focuses on refuting the findings of Johnson et al. 2003 to argue that developmental exposure to TCE does not induce congenital heart defects, noting that:

> TCE is also listed as a high priority chemical for evaluating the human health risks to workers and the general population under the ... Frank F. Lautenberg Chemical Safety for the 21st Century Act. EPA is required by Congress to complete this assessment by the end of 2019. Hence, there is an important need to resolve the reproducibility of the Johnson et al. (2003) study.[113]

This focus is incredibly myopic and at odds with TSCA and EPA's own regulation that requires that the Agency apply a weight of the scientific evidence approach to evaluating chemical risks.[114] Specifically, EPA's risk evaluation rule states that "[Weight of the scientific evidence] starts with assembling the relevant information, evaluating the information for quality and relevance, and synthesizing and integrating the different lines of evidence to support conclusions."[115] DeSesso et al. 2019 is far from anything resembling what would meet EPA's requirements under TSCA to comprehensively evaluate the evidence regarding TCE and congenital heart defects.

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[113] DeSesso, John M., et al. "Trichloroethylene in drinking water throughout gestation did not produce congenital heart defects in Sprague Dawley rats." *Birth defects research* 111.16 (2019): 1217-1233.

[114] EPA, Procedures for Chemical Risk Evaluation Under the Amended Toxic Substances Control Act, Final Rule, p. 33750, available at https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0654-0108 and codified at 40 C.F.R. 702.41(a)(4) "Evaluation requirements," https://www.law.cornell.edu/cfr/text/40/702.41.

[115] Ibid at 33733.

b. The heart dissection method used in DeSesso et al. 2019 is insufficiently sensitive to detect cardiac malformations.

With regard to DeSesso et al. 2019, the draft risk evaluation notes:

> [T]he methodology and positive control data indicate that the Charles River study (2019) was primarily focused on ventricular septal defects (VSDs) and therefore did not sufficiently examine the complete range of potential cardiac defects. The Johnson study (2003) specifically described assessment of valves and observed both valve and atrial defects using their laboratory dissection and examination methodology. In contrast, while the Stuckhardt and Poppe dissection method (1984) used by the Charles River study did not report valve defects in any TCE group or the RA positive control even though many other published reports have identified valve defects following administration of TCE or RA. Additionally, the Stuckhardt and Poppe method (1984) does not include examination of the heart for atrial septal defects, and the Charles River study did not report any atrial septal defects in either the RA positive control group or the TCE groups. In fact, the Charles River study (2019) observed a similar percentage of VSDs as (Johnson et al. 2003)….As further indication of the potentially limited sensitivity of (Charles Rivers Laboratories, 2019), the defects observed from exposure to the retinoic acid (RA) positive control were also somewhat limited compared to the broader RA literature (which did identify atrial septal defects). (pp. 222-223)

As recognized by EPA, the concerns about the sensitivity of the Charles River Laboratories study mean both that the study's assertions of the absence of congenital heart defects following TCE exposure are not supported, and that the study cannot be used to negate the findings of Johnson et al. 2003, which has been extensively and repeatedly peer-reviewed by EPA and peer review panels such as the Agency's Scientific Advisory Board.

c. DeSesso et al. 2019's differential administration of test substance and positive control contradicts the applicable test guidelines.

The DeSesso et al. study was purportedly conducted in accordance with the OECD Test Guideline 414,[116] among others. This OECD Test Guideline specifically states that "[a]nimals in the control group(s) should be handled in an identical manner to test group animals" (p. 4). However, in DeSesso et al., retinoic acid (the positive control) was administered in a completely different manner than was TCE. Retinoic acid was delivered by daily oral gavage, while TCE was administered through drinking water. Furthermore, retinoic acid was only administered

[116] OECD Guidelines for the Testing of Chemicals. Test No. 414: Prenatal Developmental Toxicity Study. https://www.oecd-ilibrary.org/environment/test-no-414-prenatal-development-toxicity-study_9789264070820-en.

during gestational days (GD) 6-15, while TCE was administered during GD 1-21. These differences introduce call into question the experimental design and could compromise the validity and utility of the study's positive control.

d. There is a discrepancy between incidence of congenital heart defects in DeSesso et al. control animals and historical control data.

The DeSesso et al. study reports that 2.4% of control fetuses developed ventricular septal defects (VSDs). However, in Appendix 8 of the original report upon which this publication is based, the incidence of various interventricular septal defects in the historical control database is recorded as 0.01% with a maximum mean incidence of 0.26%.[117] DeSesso et al. comment on these differences, noting that "[t]he mean litter proportion of VSDs in the control group was more than ninefold higher than the maximum mean value for this parameter in the historical controls. The extreme discrepancy between the Charles River Laboratory concurrent and historic control incidence data is surprising and concerning. During the TCE SACC meeting, several panelists highlighted that this observation suggests that the animals used by DeSesso et al. represent an anomalous population. Overall, this inconsistency increases skepticism about the applicability and conclusions of this study and indicates that the findings should be interpreted with extreme caution.

e. All ventricular septal defects (VSDs) are relevant.

DeSesso et al. attempt to downplay the significance of the small VSDs (<1 mm) that were observed in their study, claiming that "small VSDs which close spontaneously...should be considered normal developmental delay."[118] In Appendix G, EPA appropriately points out concerns with regard to this statement, namely that:

> [t]his claim is confounding and internally inconsistent however, because the vast majority (92%) of VSDs observed in the RA-treated positive control group were also <1 mm. If VSDs <1 mm are truly non-adverse, then this positive control data provides additional indication that the study is insufficiently sensitive for detecting adverse cardiac effects… (p. 609)

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[117] Coder, P. S. (2019) Final Report. An Oral (Drinking Water) Study of the Effects of Trichloroethylene (TCE) on Fetal Heart Development in Sprague Dawley Rats. Charles River Laboratories, Laboratory Project ID 00459506.
Available at: https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0737-0120.

[118] DeSesso, John M., et al. "Trichloroethylene in drinking water throughout gestation did not produce congenital heart defects in Sprague Dawley rats." Birth defects research 111.16 (2019): 1217-1233.

Moreover, the claim that small VSDs are not adverse is not supported by the epidemiological literature on congenital heart malformations. First, recent studies indicate that even small VSDs that do not require surgery can result in significant morbidity leading to medical complications over the course of life.[119,120,121,122,123] As stated by one group of study authors, the "data show that the clinical course of isolated, small restrictive VSDs cannot be assumed to be benign."[124] More broadly, these conclusions regarding the potential for lifelong cardiac complications are relevant for numerous categories/types of lower-complexity congenital heart disease.[125] Furthermore, it should also be noted that growing evidence indicates that individuals who undergo surgery for VSD closure experience significant long-term complications, particularly related to reduced cardiac function.[126,127,128,129]

Overall, the evidence does not support DeSesso et al.'s assertion that small VSDs do not have clinical significance. Data clearly indicate that even small cardiac malformations can lead to lifelong - and potentially costly - morbidity.

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[119] Goldberg, Jason F. "Long-term Follow-up of "Simple" Lesions—Atrial Septal Defect, Ventricular Septal Defect, and Coarctation of the Aorta." Congenital heart disease 10.5 (2015): 466-474.

[120] Gabriel HM, Heger M, Innerhofer P, et al. Long-term outcome of patients with ventricular septal defect considered not to require surgical closure during childhood. J Am Coll Cardiol. 2002; 39: 1066–1071.

[121] Karonis, Theodoros, et al. "Clinical course and potential complications of small ventricular septal defects in adulthood: Late development of left ventricular dysfunction justifies lifelong care." International journal of cardiology 208 (2016): 102-106.

[122] Neumayer, U., S. Stone, and J. Somerville. "Small ventricular septal defects in adults." European heart journal 19.10 (1998): 1573-1582.

[123] Soufflet, Veerle, et al. "Behavior of unrepaired perimembranous ventricular septal defect in young adults." The American journal of cardiology 105.3 (2010): 404-407.

[124] Karonis et al, 2016, op. cit.

[125] Saha, Priyanka, et al. "Substantial Cardiovascular Morbidity in Adults With Lower-Complexity Congenital Heart Disease." Circulation 139.16 (2019): 1889-1899.

[126] Goldberg, 2015, op. cit.

[127] Menting ME, Cuypers JA, Opić P, et al. The unnatural history of the ventricular septal defect: outcome up to 40 years after surgical closure. J Am Coll Cardiol. 2015; 65: 1941–1951.

[128] Heiberg J, Petersen AK, Laustsen S, Hjortdal VE. Abnormal ventilatory response to exercise in young adults operated for ventricular septal defect in early childhood: a long-term follow-up. Int J Cardiol. 2015;194: 2–6.

[129] Nederend, Ineke, et al. "Long-term follow-up after ventricular septal defect repair in children: cardiac autonomic control, cardiac function and exercise capacity." European Journal of Cardio-Thoracic Surgery 53.5 (2017): 1082-1088.

f. The oral gavage study by Fisher et al. 2001 has critical shortcomings not acknowledged in DeSesso et al.

DeSesso et al. repeatedly point to the Fisher et al. 2001 study to support an assertion that TCE does not cause congenital heart defects. However, the Fisher study has serious shortcomings in both its methodology and its characterization of findings that significantly reduce confidence in its conclusion that TCE does not cause congenital heart defects. In the draft risk evaluation, EPA identifies several of these deficiencies:

> [W]hile (Fisher et al., 2001) did not report statistically-significant increases in combined cardiac and cardiovascular effects, there was a very high background incidence of cardiovascular defects in soybean oil-control rats and the authors did observe a 19% increase in cardiac-specific defects (per litter, significance not calculated) following TCE treatment compared to controls. (p. 216-217)

> Additionally, the other oral TCE study (Fisher et al., 2001), which did not identify a statistically significant increase in cardiac defects following TCE administration at a high dose via gavage, identified a significant number of additional defects that match those identified in (Johnson et al., 2003) and (Dawson et al., 1993) (including atrial septal and valve defects). (p. 223)

> The score was downgraded for (Fisher et al., 2001) because only a single dose group was used and the negative control for TCE demonstrated a very elevated prevalence of heart and cardiovascular defects. (p. 615)

> The (Fisher et al., 2001) study (also reviewed separately for TCE administration) only showed a small, non-statistically significant increase in cardiac defects for both TCA and DCA, but the single dose level used in these studies was too low to rule out effects at higher doses based on results of the other studies. (p. 616)

During the TCE SACC meeting, EPA staff involved in the draft risk evaluation reiterated many of these points, including that the incidence of cardiac effects in the negative control (soybean oil) was very high—36% of pups had cardiac defects—and that the incidence of cardiac defects with the TCE treatment group was 55%, which according to the study authors yielded a non-statistically significant effect. EPA staff also noted that Fisher did not perform statistical analyses for all the observed effects including a pooled analysis of combined effects.

g. Flaws in DeSesso et al. 2019 were highlighted in a letter to the editor by Runyan et al 2019.

In response to DeSesso et al. 2019, Runyan et al. – whose four authors are all experts in developmental toxicology and cardiac malformations – published a letter to the editor highlighting the following:

> Since Johnson et al. (2003), we have gained substantial understanding of cardiac morphogenesis and the critical contributions of both function and gene expression. Measures of these latter elements provide nuanced understanding of how toxicants disturb cardiac structure and functionality. DeSesso et al. utilizes a static assessment methodology that captures only a subset of dysmorphologies and does not evaluate actual function. We argue that their data is insufficient to overcome a substantial literature showing the sensitivity of the developing heart to environmentally relevant TCE exposures. Their conclusion that ingestion of TCE in drinking water at less than 1,000 ppm does not cause heart defects is not supported by their data.[130]

The authors further indicate that the Johnson et al. 2003 itself likely missed cardiac-related effects of TCE at low levels of exposure, attributed to extensive evidence of non-monotonicity at levels of exposure below 1,000 ppm:

> The study [DeSesso et al. 2019] design ignores the many studies published in the last 18 years that show TCE toxicity at exposures lower than 1,000 ppm, as well as evidence that TCE exhibits nonmonotonic effects. Our laboratories have shown that low level exposures in the 10–100 ppb range inhibit developmental mechanisms, alter gene transcription and produce changes in cardiac output. These effects occur with exposures below the range tested by DeSesso et al.[131]

The authors' letter is provided as Appendix 5 to our comments. Table 1 of the letter illustrates the extensive study database demonstrating effects of TCE below 1000 ppm.

Overall, the Runyan et al. 2019 letter lends further support to an already significant body of evidence that TCE exposure causes congenital heart defects, likely at levels of exposure below even what Johnson et al. 2003 suggests.

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[130] Runyan, R.B., Selmin, O.I., Smith, S.M. and Freeman, J.L. (2019), Letter to the Editor. Birth Defects Research, 111: 1234-1236. doi:10.1002/bdr2.1573.

[131] Ibid.

h. Conflicts of interest are evident in DeSesso et al.

Given the well-documented association between research sponsorship and study findings,[132,133,134] it should be noted that the DeSesso et al. 2019 study was commissioned and supported by the Halogenated Solvents Industry Alliance (HSIA) and the American Chemistry Council (ACC).[135] These groups represent companies that have direct and substantial financial interests in the continued production and use of TCE as well as with respect to potential liability associated with releases and exposures to TCE, including from contaminated sites. Risk of bias from conflict of interest is an important consideration in systematic review of the environmental health field and it should be – but has not been – considered by OPPT.[136]

vii. EPA missed key flaws in Wikoff et al. 2018 that should have reduced EPA's confidence in the conclusions of that review.

EPA makes references to the conflicting results of two prior WOE analyses (Makris et al. 2016 and Wikoff et al. 2018) as a rationale for conducting a new WOE analysis in the draft risk evaluation. Our examination of the Wikoff et al. 2018 review identified numerous, significant flaws that adversely impact confidence in its conclusions.

a. Creation of "sub-domains"

Wikoff et al. 2018 adapts the Office of Health Assessment and Translation (OHAT) Risk of Bias (RoB) rating tool for human and animal studies to assess the internal validity of experimental animal and human evidence linking maternal exposure to TCE to congenital heart defects. The studies examined by the authors include those previously examined in Makris et al. 2016 and additional studies published after Makris' review window (2010 – 2015) and additional studies they identified through reference lists from existing papers and reviews. The authors state that, following guidance presented in the OHAT handbook, they have modified the OHAT framework

[132] Lee Friedman & Michael Friedman, Financial conflicts of interest and study results in environmental and occupational health research, 58:3 J. OF OCCUPATIONAL & ENVTL. MEDICINE 238-247 (2016).

[133] Lisa Bero, et al., The relationship between study sponsorship, risks of bias, and research outcomes in atrazine exposure studies conducted in non-human animals: Systematic review and meta-analysis, 92 ENV'T INT'L 597-604 (2016).

[134] Jenny White & Lisa Bero, Corporate manipulation of research: strategies are similar across five industries, 21 STAN. L. & POL'Y REV. 105 (2010).

[135] DeSesso et al. 2019, op. cit.

[136] Tracey Woodruff et al., An evidence-based medicine methodology to bridge the gap between clinical and environmental health sciences, 30:5 HEALTH AFFAIRS 931-937 (2011); see also EDF comments on TSCA systematic review, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2018-0210-0077.

to tailor it to the specific research hypothesis under study. Specifically, Wikoff et al. took some of the 11 research questions/domains from OHAT and created "subdomains" that split out the combined criteria into multiple, separate considerations. For example, in OHAT, Question 1, "[w]as administered dose or exposure level adequately randomized?", was broken into two separate subdomains, Question 1a ("Adequate randomization of animals to control or exposure/dose groups") and Question 1b ("Were all study groups (control and exposed) investigated concurrently?"). While both concepts are jointly considered in OHAT guidance, separating them into multiple questions creates additional opportunities to highlight shortcomings of individual studies. It is not clear whether the subdomains are quantitatively considered equivalent to domains (this is not clearly described in the study), but the visual effect on RoB heatmaps is that studies that perform poorly on individual subdomains appear to be of even lower quality than they would be if subdomains were retained as single domains per the OHAT RoB rating tool.

It is also worth examining the choices made with respect to deconstruction of domains into subdomains. Curiously, the Johnson et al. 2003 study, which has been the study used by EPA for quantitative dose-response assessment (and was defended in Makris et al. 2016 as the best choice for this purpose) performs especially poorly according to the rating scheme fabricated by Wikoff et al. and the associated heat maps. It would seem that Wikoff et al. worked backwards from shortcomings in conduct/presentation of the Johnson et al. 2003 study to put more emphasis on the elements of the OHAT framework that would devalue that study and cause it to be discarded.

b. Problems with "key" criteria and their application to the Johnson et al. 2003 study

Key Criterion 5b:[137] For the Johnson study, Wikoff et al. selects a bias rating that means: "Probably High - indirect evidence that non-treatment-related experimental conditions were not comparable between study groups." There is no evidence presented to support this rating. The authors in the 2005 erratum to the Johnson et al. 2003 study say, "The control "sets" were statistically analyzed comparing the data to each other before being combined. The authors opine that the control values were statistically consistent across and throughout all the treatment groups. Using the control data in a cumulative manner increased the generalizability of the data, which purports to demonstrate the background rate and variability around rate estimates."[138]

[137] Criterion 5b in Wikoff et al. is as follows: "Question 5b—Were non-treatment-related experimental conditions the same for all study groups (control and exposed)?" (p. 127).
[138] Johnson, Paula D., et al. "Threshold of trichloroethylene contamination in maternal drinking waters affecting fetal heart development in the rat." *Environmental health perspectives* 111.3 (2003): 289-292. Erratum-ibid: https://ehp.niehs.nih.gov/doi/10.1289/ehp.113-1253738.

Key Criterion 9a:[139] Wikoff et al. make a determination regarding the acceptable methods for assessment of congenital heart defect outcomes:

> Given the minute size of the fetal heart in rodents and other small animal species, and the sensitivity of this organ tissue, CHDs [congenital heart defects] have been commonly identified by using 1 of 2 common and acceptable fetal dissection techniques [reviewed in Tyl and Marr[27]]: the fresh in situ microdissection technique[28,29] and the fixation, serial sectioning technique.[30]

The authors state that use of these specific methods results in a low risk of bias. Johnson et al. 2003 applied a different cardiac evaluation method and therefore received a "probably high" risk of bias rating for this criterion. However, in the draft risk evaluation, Table Apx G-5 (p. 603-604), EPA presents a comparison of the types of cardiac malformations observed under the methods employed by each study, noting that the Johnson study was capable of detecting a variety of septal and valve defects, as well as atrial, ventricular, and other miscellaneous abnormalities (many of which were not observable using the methods employed by the 2019 Charles River study). EPA further elaborates in Appendix G.1.2.3 key differences in the dissection methods between Johnson et al. 2003 and the 2019 Charles River study that would lead to superior sensitivity of Johnson et al. 2003 in the detection of cardiac malformations (p. 607-609). On these bases, it appears entirely unreasonable that Wikoff et al. 2018 rated Johnson et al. 2003 as probably high bias for outcome assessment.

c. Risk of bias tables not available

The completed risk of bias tables were not available from the Wikoff study. This lack of transparency prevents EPA and the public from examining the bases and justifications for the specific ratings for each study.

viii. The Urban et al. 2020 systematic evaluation of mechanistic data is flawed and does not negate the strong body of mechanistic data supporting the link between TCE and congenital heart defects.

Urban et al. recently published a purported systematic evaluation of the mechanistic data on TCE and congenital heart defects.[140] The authors concluded that the totality of these data are not

[139] Criterion 9a in Wikoff et al. is as follows: "Question 9a—Is there confidence in the outcome assessment method?"

[140] Urban, Jonathan D., et al. "Systematic Evaluation of Mechanistic Data in Assessing In Utero Exposures to Trichloroethylene and Development of Congenital Heart Defects." *Toxicology* (2020): 152427.

supportive of the potential for TCE to cause congenital heart defects in humans. However, a closer look at this study reveals serious concerns and flaws that reduce its validity.

a. Flaws in systematic evaluation approach

1. Misappropriation of the NTP-OHAT method

According to the methods section of Urban et al., the authors generally followed the National Toxicology Program (NTP)- Office of Health Assessment and Translation (OHAT) approach to evaluate the mechanistic data:

> This evaluation of mechanistic data also follows the general systematic review concepts from the NTP-OHAT guidance, as well as their recommendations for mechanistic data. This approach was selected because it is the only finalized approach issued to date which includes mechanistic data (others, such as Navigation Guide, do not include formal mechanistic data considerations). Because NTP-OHAT does not include methods for assessing the validity of in vitro studies, other approaches were utilized for critical appraisal of individual studies. Also, because NTP-OHAT does not provide guidance specific to synthesizing and integrating mechanistic evidence, multiple approaches based on established concepts (e.g., AOP) were applied herein.[141]

The description provided here raises serious concerns, including a suggestion that the NTP-OHAT systematic review method was applied.[142] However, the NTP-OHAT method for evaluating a study's internal validity (risk of bias) does not address mechanistic studies, nor does it have a formal, structured approach to evidence integration for mechanistic data as it does for animal and human studies. The absence of these elements from the NTP-OHAT method in no way represents an oversight of the method, which has undergone extensive review. Rather, it reflects the fact that empirically based methods for the consideration of mechanistic data in systematic review are not yet established; this is still an active area of research. Indeed, the NTP-OHAT method indicates: "New methods are needed for evidence-based evaluation of nonhuman toxicological studies, including mechanistic studies."[143] As recently as 2018 and 2019, the National Academies of Sciences, Engineering, and Medicine (NASEM) held two workshops to discuss the state of the science regarding the inclusion of mechanistic data in

[141] Ibid.

[142] National Toxicology Program (NTP). Handbook for Conducting a Literature-Based Health Assessment Using OHAT Approach for Systematic Review and Evidence Integration. 2019. https://ntp.niehs.nih.gov/ntp/ohat/pubs/handbookmarch2019_508.pdf.

[143] Ibid. p. 2.

systematic review—a reflection of the evolving nature of this topic.[144,145] Furthermore, the NTP-OHAT method explicitly indicates that mechanistic data are not required to reach hazard conclusions, and describes its use as follows: "The NTP does not require mechanistic or mode-of-action data in order to reach hazard identification conclusions, although when available, this and other relevant supporting types of evidence may be used to raise (or lower) the category of the hazard identification conclusion."[146]

Taken together, Urban et al.'s assertion that they modeled their evaluation on NTP-OHAT methods is disingenuous and misleading, and the implication that whatever methodology they applied would be generally perceived as scientifically valid is unsupported.

2. Reliance on flawed TSCA systematic review method

Urban et al. relied on the deeply flawed TSCA systematic review scoring method for evaluating study quality and to support integration of evidence across identified mechanistic studies:

> Study quality was evaluated using the USEPA's Office of Pollution Prevention and Toxics (USEPA OPPT) draft systematic review study quality tool (United States Environmental Protection Agency (USEPA,2018b) …. Studies/assays determined to be "unacceptable" are considered unreliable for risk assessment under the OPPT guidelines. However, none of the mechanistic datasets were discarded or disqualified from the current evaluation, regardless of the quality rating results; rather, the data quality scoring was used as one of several elements considered as part of synthesis and integration.[147]

As discussion in section 8 of these comments, the TSCA systematic review method is problematic and raises concerns that have spurred a peer review by NASEM that is now

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[144] National Academies of Sciences, Engineering, and Medicine (NASEM), Workshop on Strategies and Tools for Conducting Systematic Reviews of Mechanistic Data to Support Chemical Assessments, Dec. 10-11, 2018, *see* http://dels.nas.edu/Upcoming-Workshop/Strategies-Tools-Conducting-Systematic/AUTO-5-32-82-N.

[145] National Academies of Sciences, Engineering, and Medicine (NASEM), Workshop on Evidence Integration, Jun. 3-4, 2019, *see* http://dels.nas.edu/Upcoming-Event/Evidence-Integration-Workshop/AUTO-0-96-15-Q.

[146] National Toxicology Program (NTP). Handbook for Conducting a Literature-Based Health Assessment Using OHAT Approach for Systematic Review and Evidence Integration. 2019. https://ntp.niehs.nih.gov/ntp/ohat/pubs/handbookmarch2019_508.pdf.

[147] Urban, Jonathan D., et al. "Systematic Evaluation of Mechanistic Data in Assessing In Utero Exposures to Trichloroethylene and Development of Congenital Heart Defects." *Toxicology* (2020): 152427. p. 3.

ongoing.[148] Indeed, Urban et al. illustrates some of these concerns when describing how many of the mechanistic studies are excluded when applying EPA's data quality criteria for mechanistic studies:

> Application of the OPPT study quality metrics (United States Environmental Protection Agency (USEPA, 2018b) to the mechanistic evidence base demonstrated that the majority of experimental datasets (approximately 70 %) were assigned a score=4 for at least one of the OPPT study quality metrics, indicating these data sets are unreliable for risk assessment (Supplemental Table 2).[149]

As discussed in subsection D.viii.b. below, these exclusions are often unwarranted.

The use of the flawed TSCA systematic review method by Urban et al. 2020 calls into serious question any conclusions drawn from its characterization of the mechanistic evidence base for TCE-induced congenital heart defects.

b. Inappropriate criteria for rejection of mechanistic data

In recent comments submitted to the docket, Dr. Raymond Runyan, an expert in developmental cardiac toxicity, indicates that Urban et al. used inappropriate criteria to reject 16 studies that provide mechanistic support for the link between TCE and congenital heart defects:[150]

> Urban et al. raise issues of substance preparation and storage, data analysis and testing for potential cytotoxicity as the primary reasons for rejection of 16 studies that provide mechanistic support for the link that they challenge. My laboratory and those of my colleagues understand that TCE is volatile, degrades quickly and partitions into headspace. We have long used freshly opened bottles from reputable suppliers, stored TCE under argon gas, replaced exposure media frequently and used glass or stainless steel to avoid loss into plastic containers. We viewed this as a convention in this field that did not require specification. Our recent publications now include this information and we would have been happy to inform Urban et al. of this had we been contacted. Urban et al. also argued that our recent study by Harris et al. 2018 should be downgraded because

[148] National Academies of Sciences, Engineering, and Medicine (NASEM), Review of EPA's TSCA Systematic Review Guidance Document, see project page at https://www.nationalacademies.org/our-work/review-of-epas-tsca-systematic-review-guidance-document.

[149] Urban et al., op. cit. p. 4.

[150] Comment submitted by Raymond Runyan, Professor of Cellular and Molecular Medicine, University of Arizona, p. 1. https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0500-0066.

> they would have preferred to see an ANOVA analysis rather than T tests used in the statistical analysis. Our statisticians argued that we were not attempting to utilize multiple interdependent measures together and that an ANOVA was not necessary for our analysis. Several of these studies were disqualified because we failed to test for cytotoxicity. I note that the vast majority of our studies have been conducted at concentrations of 1ppm or less and there is no evidence of cytotoxicity in the heart at these concentrations in the 20 years we have been looking at the problem. Such toxicity would be recognized both from observations of function (myocardial contraction) and the quality of RNA extracted from the hearts. Thus, disqualification of studies for a lack of toxicity data is inappropriate.

It is particularly concerning, as highlighted by Dr. Runyan, that no attempt was made to contact authors of the disqualified studies, many of whom likely would have been able to provide the missing information. Indeed, the NTP-OHAT method that Urban et al. purport to have modeled their study after indicates:

> OHAT will attempt to contact authors of included studies to obtain missing information considered important for evaluating risk of bias. The product of the evaluation (e.g., monograph, report, or publication) will note that an attempt to contact study authors was unsuccessful if study researchers do not respond to an email or phone request within one month of the attempt to contact. If additional data or information are acquired from study authors, risk of bias judgments will be modified to reflect the updated study information.[151]

One could reasonably posit that the Urban et al. had a desired conclusion in mind when undertaking a review of mechanistic data regarding TCE-induced congenital heart defects, given that all of the studies disqualified based on these inappropriate criteria provided mechanistic evidence of the linkage between TCE and congenital heart defects.

[151] National Toxicology Program (NTP). Handbook for Conducting a Literature-Based Health Assessment Using OHAT Approach for Systematic Review and Evidence Integration. 2019. P. 41. https://ntp.niehs.nih.gov/ntp/ohat/pubs/handbookmarch2019_508.pdf.

c. Conflict of interest in Urban et al. 2020

As noted for DeSesso et al. 2019 above, there are well-documented association between research sponsorship and study findings.[152,153,154] The Urban et al. 2020 study was supported by the American Chemistry Council (ACC).[155] This group represents companies that have direct and substantial financial interests in the continued production and use of TCE as well as with respect to potential liability associated with releases of and exposures to TCE, including from contaminated sites. As a general matter, risk of bias from conflict of interest is an important consideration in conducting systematic reviews and it should be – but has not been – considered by OPPT.[156]

ix. Use of the congenital heart defects endpoint protects against other endpoints, whereas use of the immunosuppression POD from Selgrade and Gilmour (2010) does not.

On page 377, the Agency states, without any evidence, that "*EPA has the most confidence in [immunosuppression and autoimmunity] and it is expected that addressing risk for these effects would address other identified risks.*" The use of these immune endpoints for POD derivation and subsequent risk determination would not protect against a constellation of more sensitive health effects that have been demonstrated in the literature to occur close to the range of the POD for congenital heart defects derived from the Johnson et al. 2003 study. In contrast, existing evidence and Agency precedent indicate that using the Johnson et al. 2003 study for POD derivation is well justified and would protect against these numerous additional effects.

As noted in the IRIS 2011 Toxicological Review of Trichloroethylene, the POD from the Johnson et al. 2003 study supported an RfD (0.0005 mg/kg/day) within 20% of candidate RfDs derived for other critical effects (developmental immunotoxicity and decreased thymus weights)

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[152] Lee Friedman & Michael Friedman, Financial conflicts of interest and study results in environmental and occupational health research, 58:3 J. OF OCCUPATIONAL & ENVTL. MEDICINE 238-247 (2016).

[153] Lisa Bero, et al., The relationship between study sponsorship, risks of bias, and research outcomes in atrazine exposure studies conducted in non-human animals: Systematic review and meta-analysis, 92 ENV'T INT'L 597-604 (2016).

[154] Jenny White & Lisa Bero, Corporate manipulation of research: strategies are similar across five industries, 21 STAN. L. & POL'Y REV. 105 (2010).

[155] DeSesso et al. 2019, op. cit.

[156] Tracey Woodruff et al., An evidence-based medicine methodology to bridge the gap between clinical and environmental health sciences, 30:5 HEALTH AFFAIRS 931-937 (2011); see also EDF comments on TSCA systematic review, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2018-0210-0077.

from studies in mice, and within a factor of 2 of candidate RfDs for kidney effects (toxic nephropathy and increased kidney weight) in rats. Ultimately in that assessment, EPA based its RfD on multiple effects (heart malformations, adult immunological effects, developmental immunological effects, and kidney effects) from studies of multiple species, all via the oral route. The assessment noted "*there is strong, robust support for an RfD of 0.0005 mg/kg/day provided by the concordance of estimates derived from multiple effects from multiple studies.*"[157]

E. EPA should include congenital heart defects in the assessment of acute risks from TCE exposure.

EPA appropriately recognizes that developmental toxicity studies are relevant for evaluating acute exposure scenarios. EPA notes:

> Although developmental studies typically involve multiple exposures, they are considered relevant for evaluating single exposures because evidence indicates that certain developmental effects may result from a single exposure during a critical window of development (Davis et al., 2009; Van Raaij et al., 2003). This is consistent with EPA's Guidelines for Reproductive Toxicity Risk Assessment (U.S. EPA, 1996) and Guidelines for Developmental Toxicity Risk Assessment (U.S. EPA, 1991), which state that repeated exposure is not a necessary prerequisite for the manifestation of developmental toxicity. This is a health protective assumption. (p. 221)

In its determinations of acute risks resulting from TCE exposure, however, EPA chose to rely on its margin of exposures (MOE) values based on immunosuppression as measured by mortality: "Mortality was selected as the most statistically sensitive endpoint due to a larger numbers of mice per exposure group and more dose groups…." (p. 231).

EPA's decision is flawed and contradicts long-standing Agency policy and previous EPA assessments of TCE that require basing risk assessment on the most sensitive endpoint.

As described in EPA 2014 TCE work plan assessment and in the TSCA section 6 proposed rules for the use of TCE in vapor degreasing, aerosol degreasing, and in spot cleaning in dry cleaning facilities, the Agency relied on developmental endpoints for assessing and addressing the health risks of TCE resulting from acute exposure.

For example, EPA states in section 2.7.2 of the 2014 TCE Work Plan Chemical Risk Assessment:

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[157] EPA. IRIS Toxicological Review of TCE. 2011. https://www.epa.gov/iris/supporting-documents-trichloroethylene, p. 5-101.

> The acute inhalation risk assessment used developmental toxicity data to evaluate the acute risks for the TSCA TCE use scenarios. As indicated previously, EPA's policy supports the use of developmental studies to evaluate the risks of acute exposures. This policy is based on the presumption that a single exposure of a chemical at a critical window of fetal development, as in the case of cardiac development, may produce adverse developmental effects (EPA, 1991).[158]

EPA elaborated extensively on the use of developmental toxicity to assess acute risks of TCE in its response to comments associated with the 2014 Work Plan Chemical Risk Assessment for TCE. Specifically, the Agency stated:

> EPA/OPPT used developmental toxicity data to evaluate the non-cancer risks of acute exposures based on EPA's long standing policy that a single exposure within a critical window of development may induce developmental effects, as discussed in the EPA's Guidelines for Developmental Toxicity Risk Assessment (http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=23162). EPA/OPPT acknowledges that this is a health-protective policy that may overestimate the acute risks.
>
> Developmental effects, including fetal cardiac defects, may occur following maternal exposure to TCE. Chick embryo and oral developmental studies, including those reported by the Johnson et al. studies (see list of references below), have reported cardiac malformations after exposure to TCE. The incidence of congenital cardiac malformation has been replicated in several studies from the same laboratory group and has been shown to be TCE-related. Moreover, studies with TCE metabolites have also induced cardiac defects in developmental oral toxicity studies.
>
> 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 (Appendix N). As explained in the TCE IRIS assessment, while the Johnson et al. studies have limitations, there is insufficient reason to dismiss their findings, especially when the findings are analyzed in combination with human, animal and mechanistic evidence. A summary of the weight of evidence supporting TCE-related fetal cardiac defects is provided in section 2.6.2.3.6 and Appendix N of the final TCE OPPT risk assessment. The comprehensive WOE evaluation of the developmental toxicity

[158] EPA. TCE Work Plan Chemical Risk Assessment. 2014. https://www.epa.gov/sites/production/files/2015-09/documents/tce_opptworkplanchemra_final_062414.pdf, p. 104.

> data, including fetal cardiac teratogenesis, is discussed in the TCE IRIS assessment and expanded in this assessment (Appendix N).[159]

Further, as elaborated on in section 4.D.v. of these comments, EPA has previously determined and explained that Johnson et al. 2003 is appropriate for deriving a point of departure. In this draft risk evaluation, however, rather than abide by health-protective Agency policy and precedents from previous Agency assessments—which have undergone extensive peer review (see Appendix 4)—the Agency has decided to rely on immunosuppression as measured by death in mice as a basis for its determinations for acute risks resulting from TCE exposure. The effect of this decision to lower the risk estimates by several orders of magnitude across multiple conditions of use.

EPA's decision not to take a health-protective approach to assessing acute TCE risks is at odds with TSCA's requirement to protect potentially exposed or susceptible subpopulations, which explicitly include pregnant women and children.

F. There are inconsistencies in the use of study quality for decision-making across the draft risk evaluation.

While EPA erroneously prioritizes study quality above all else in selecting the immune endpoints as the basis for its determinations of acute and chronic non-cancer risks, the Agency's flawed approach is inconsistent with its decision-making in other areas of the risk evaluation. For example, when selecting *between* studies of the same endpoint, EPA seems to have no hesitation in choosing medium quality studies. For the liver toxicity endpoint (Table 3-8, p. 240), the Agency reviews both high and medium quality studies and chooses to advance a medium quality study to represent liver toxicity. Similarly, for the reproductive toxicity endpoint (Table 3-12, p. 248), EPA reviews both high and medium quality studies and chooses to advance a medium quality study to represent reproductive toxicity.

These examples clearly indicate that EPA uses study quality in a selective, biased and inconsistent way when it comes to congenital heart defects versus other endpoints. If the Agency sees fit to advance medium quality studies for an endpoint also having high quality studies, it has no reason to reject that approach when it comes to acute and chronic non-cancer endpoints that will drive its risk determinations.

On page 235 of the draft risk evaluation, EPA presents six criteria for evaluation of candidate health domains, studies, and PODs (data quality evaluation score, species, exposure duration,

[159] EPA. TCE Work Plan Chemical Risk Assessment response to comments. 2014. https://www.epa.gov/sites/production/files/2015-09/documents/tce_response_to_comments_final_062414.pdf, p. 17-18.

dose range, cumulative uncertainty factor, and relevance to the endpoint of interest and human exposure scenarios). While the Agency addresses some of these criteria in its justification for selecting the immunosuppression endpoint from the Selgrade and Gilmour (2010) study, it does not present a comparable evaluation for other candidate health domains, studies, or PODs against these stated criteria. Without such a comparison, EPA does not adequately justify its endpoint/POD selection, especially given the substantial difference in sensitivity between the congenital heart defect and immunosuppression endpoints. Further, on page 377, EPA justifies its selection of the immunosuppression endpoint solely on the basis of the quality of the study, ignoring the remainder of its own criteria even for this single endpoint.

As a general matter, in its decision-making, EPA would be best served by considering both the weight of the evidence supporting the endpoint and its sensitivity together. In the case of TCE, congenital heart defects and immunosuppression were both determined by the Agency to be of adequate quality to move forward for dose-response modeling; subsequent to this point in the process, endpoint sensitivity should be expected to drive POD selection in order to best protect public health.

G. Scientific, policy and legal arguments indicate EPA must use congenital heart defects to determine risks from TCE.

i. EPA's decision to ignore the most sensitive endpoint when reaching risk determinations represents a deeply troubling break with decades of Agency scientific policy and practice designed to protect public health.

In section 5.2, Risk Determinations for TCE, EPA indicates that it has based its determinations of unreasonable risk on immune-related endpoints. Specifically, EPA indicates that it "[i]s relying upon immunosuppression for acute inhalation and dermal exposures, and autoimmunity for chronic inhalation and dermal exposures" (p. 377). It is worth emphasizing that nothing short of death is the effective endpoint EPA used to derive the POD from the immunosuppression study by Selgrade and Gilmour (2010). Based on a comparison of the $BMDL_{01}$s for these endpoints, the result of this choice is a significant increase in the POD, indicating that the selected endpoint is orders of magnitude less sensitive than the congenital heart defects endpoint.

With this decision, EPA has chosen not to protect against the most sensitive endpoint, congenital heart defects, for which there is strong scientific support. Indeed, EPA presents a rigorous case for the congenital heart defects endpoint throughout the draft risk evaluation, considering multiple lines of evidence that converge into an integrated strength area score of (+) (see Weight of Evidence analysis and summary presented in Appendix G.2). The Agency highlights the robust evidence base multiple times throughout the draft risk evaluation:

> Overall, an association between increased congenital heart defects and TCE exposure is supported by the weight of the evidence, in agreement with previous

> EPA analyses (U.S. EPA, 2014b; Makris et al., 2016). Therefore, this endpoint was carried forward to dose-response analysis. (p. 225).

and

> 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). (p. 621)

In comparison, the overall study database for the immunotoxicity and autoimmunity endpoint is more limited than for the congenital heart defects endpoint. While Selgrade and Gilmour (2010) may have been assigned a high-quality rating as an individual study, this endpoint is orders of magnitude less sensitive and is not supported by the same, rigorous weight of evidence analysis to which the congenital heart defects endpoint was subjected.

Furthermore, it is inappropriate to use study quality as the sole basis for endpoint and study selection. Study quality is *an* appropriate consideration of the adequacy of published research to serve as the basis for dose-response analyses. After inappropriate studies are eliminated as candidates for dose-response analyses, other considerations, such as sensitivity, should form the basis for endpoint selection for dose-response analysis.

Instead of carrying the most sensitive endpoint, congenital heart defects, through to be the basis for its risk determinations, EPA erroneously asserts, in defiance of public health protection principles and statutory requirements under TSCA to explicitly protect potentially exposed or susceptible subpopulations (see subsection G.ii. below), that "[n]either the statute nor the framework rule require that EPA choose the lowest number and EPA believes that public health is best served when EPA relies upon the highest quality information for which EPA has the greatest confidence." (p. 377) ***In fact, public health is best served when public health is best protected.***

EPA's decision to ignore strong scientific evidence that indicates TCE induces congenital heart defects at levels of exposure far lower than those associated with the immune-related effects EPA has chosen to use for its risk determinations is not only scientifically unsupported, it is contrary to the Agency's core mission to protect public health. EPA is failing to protect against the most sensitive endpoint, as supported by the weight of the scientific evidence, and is failing to protect a critical susceptible subpopulation: pregnant women and the developing fetus.

EPA's choice also contradicts previous Agency assessments of TCE and existing Agency guidance to use the most sensitive endpoint and protect the most sensitive group (emphases added):

- EPA, *Guidelines for Developmental Toxicity Risk Assessment*: "The risk characterization of an agent should be based on data from the most appropriate species, or, if such information is not available, on the most sensitive species tested. *It should also be based on the most sensitive indicator of toxicity*, whether maternal, paternal, or developmental, when such data are available, and should be considered in relationship to other forms of toxicity."[160]
- EPA Risk Assessment Task Force, *Staff Paper on Risk Assessment Principles and Practices*: "Combined with UFs and other upper-bound estimates, *basing cancer and non-cancer risks on the most sensitive animal data* gives reasonable assurance that the potential for harm will not be underestimated, most likely even when some toxicity endpoints have not been evaluated."[161]
- EPA, *A Review of the Reference Dose and Reference Concentration Processes*: "[T]he 'critical effect' is used as the basis for the POD, and various UFs are applied to the dose at the critical effect to derive the RfD or the RfC. *The critical effect is defined as 'the first adverse effect, or its known precursor, that occurs to the most sensitive species as the dose rate of an agent increases'* (U.S. EPA, 2002c). The underlying assumption is that if the RfD or the RfC is derived to prevent the critical effect from occurring, then no other effects of concern will occur…."[162]
- EPA's policy on evaluating risk to children: "It is the policy of the U.S. Environmental Protection Agency (EPA) to *consider the risks to infants and children* consistently and explicitly as a part of risk assessments generated during its decision making process, including the setting of standards to protect public health and the environment. To the degree permitted by available data in each case, the Agency will develop a separate assessment of risks to infants and children or state clearly why this is not done - for example, a demonstration that infants and children are not expected to be exposed to the stressor under examination."[163]

The National Academy of Sciences has also reiterated the need to protect the most sensitive subpopulations and to protect against the most sensitive endpoints (emphases added):

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[160] EPA, Guidelines for Developmental Toxicity Risk Assessment. https://www.epa.gov/sites/production/files/2014-11/documents/dev_tox.pdf, p. 48.

[161] EPA. Office of the Science Advisor Staff Paper Risk Assessment Principles and Practices 2004 https://www.worldcat.org/title/examination-of-epa-risk-assessment-principles-and-practices-staff-paper-prepared-for-the-us-environmental-protection-agency-by-members-of-the-risk-assessment-task-force/oclc/55850621, pp. 57-58.

[162] A Review of the Reference Dose and Reference Concentration Processes. https://www.epa.gov/sites/production/files/2014-12/documents/rfd-final.pdf, p. 4-22.

[163] EPA's policy on evaluating risk to children. https://www.epa.gov/children/epas-policy-evaluating-risk-children, p. 1.

- National Academy of Sciences, *Science and Decisions: Advancing Risk Assessment*:
    - "In addition to characterizing the full population at risk, attention should be directed to *vulnerable individuals and subpopulations that may be particularly susceptible* or more highly exposed."[164]

- National Academy of Sciences, *Science and Judgment*:
    - "The *most sensitive end point of toxicity* should continue to be used for establishing the reference dose."[165]
    - "*The critical toxic effect used is the one generally characterized by the lowest NOAEL*. This approach is based on the assumption that if the critical toxic effect is prevented, then all toxic effects are prevented."[166]

These excerpts represent a non-exhaustive list of direction to Agency to protect sensitive subpopulations, and to protect against the most sensitive endpoint. EPA's proposed risk determinations fail on both accounts.

These excerpts also make clear that if EPA protects against the most sensitive endpoint, it will also generally protect against other effects. In contrast, EPA asserts without providing a shred of evidence that "it is expected that addressing risks for these [immune system] effects would address other identified risks." (p. 377) EPA should be ashamed of itself.

ii. TSCA's requirement that EPA assess risks to susceptible subpopulations demands that EPA base its risk determinations on the endpoint—congenital heart effects—that specifically impacts pregnant women, infants, and children.

TSCA section 6(b)(4)(A) states (emphasis added):

> The Administrator shall conduct risk evaluations pursuant to this paragraph to determine whether a chemical substance presents an unreasonable risk of injury to health or the environment, without consideration of costs or other nonrisk factors, including *an unreasonable risk to a potentially exposed or susceptible subpopulation* identified as relevant to the risk evaluation by the Administrator, under the conditions of use.

[164] NAS, Science and Decisions: Advancing Risk Assessment. https://www.nap.edu/catalog/12209/science-and-decisions-advancing-risk-assessment, p. 120.
[165] NAS, Science and Judgment. https://www.nap.edu/catalog/2125/science-and-judgment-in-risk-assessment, p. 142.
[166] Ibid. p. 145.

In turn, TSCA section 3(12) defines "potentially exposed or susceptible subpopulation" as (emphases added):

> a group of individuals within the general population identified by the Administrator who, due to either *greater susceptibility* or greater exposure, may be at greater risk than the general population of adverse health effects from exposure to a chemical substance or mixture, such as *infants, children, pregnant women*, workers, or the elderly.

EPA acknowledges that "congenital heart defects were the most sensitive endpoint for TCE" (p. 377; Table 4-5 on p. 280), and this endpoint is directly relevant to the potentially exposed or susceptible subpopulations of pregnant women, infants, and children identified under TSCA. Yet EPA fails to rely on this endpoint in its risk determinations and instead relies on a different, far less sensitive endpoint that is not the most relevant to those subpopulations. In making this switch in endpoints, EPA is not only making risk determinations based on a far more lax risk benchmark; it is also failing to carry out its mandate under TSCA section 6(b)(4)(A) to conduct a risk evaluation "to determine whether a chemical substance presents … *an unreasonable risk to a potentially exposed or susceptible subpopulation*." (emphasis added) EPA cannot adequately identify or protect against risks specific to pregnant women (and their developing fetuses) or infants or children by selecting immune effects as the basis for its risk determinations. EPA must develop risk determinations that address the endpoint—congenital heart defects—that specifically impacts pregnant women, infants, and children.

iii. TSCA's requirement that EPA assess risks using the best available science demands that EPA base it risk determinations on congenital heart defects.

The best available science provides evidence of congenital heart defects. EPA itself scored both the Dawson et al. 1993 and Johnson et al. 2003 studies as "a Medium in data quality evaluation," (p. 222) and EPA has repeatedly relied on Medium studies in its draft risk evaluations to date, including this one (see subsections D.v., F., and I.i.). And in EPA's own words: "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)." (p. 621). To the extent EPA made a scientific finding on congenital heart defects in this draft risk evaluation, EPA has found that they do occur and that the evidence indicates that they are the most sensitive endpoint for TCE. EPA did *not* find that contrary studies on congenital heart defects were stronger or more reliable than the Johnson et al. 2003 study.

Despite these scientific findings on congenital heart defects, EPA chooses to ignore this endpoint in its final analysis, by adopting risk estimates and determinations that do not account for this endpoint. EPA seeks to justify this decision by invoking the "best available science" (p. 377),

but in doing so, EPA relies on nonscientific and illogical reasoning. Specifically, EPA states that it has greater confidence in other endpoints—immunosuppression for acute inhalation and dermal exposures, and autoimmunity for chronic inhalation and dermal exposures. (p. 377). EPA then uses this asserted greater confidence to rely on these endpoints for its risk estimates and determination, but nothing about the availability of data in which it says it has greater confidence for these other endpoints undermines the evidence for congenital heart defects. In other words, EPA dismisses the evidence on congenital heart defects by pointing to evidence that has no bearing whatsoever on congenital heart defects.

The "best available science" on congenital heart defects supports that they occur and supports use of the Johnson et al. study. EPA tries to dismiss these findings based on alleged uncertainty, but "[q]uite simply, the [Agency] cannot demand a greater level of scientific certainty than has been achieved in the field to date." *Defenders of Wildlife v. Jewell*, 176 F. Supp. 3d 975, 1003 (D. Mont. 2016); *Survivors v. United States DOI,* 321 F. Supp. 3d 1011, 1040 (N.D. Cal. 2018) ("[W]here superior information is not readily available, the 'best available science' requirement of the ESA does not 'insist on perfection' and does not require the 'the best scientific data possible.'") (quoting *San Luis & Delta-Mendota Water Auth. v. Jewell*, 747 F.3d 581, 602 (9th Cir. 2014)); *NRDC v. Evans*, 364 F. Supp. 2d 1083, 1131 (N. Cal. 2003) ("[An] interpretation of the [best available science] requirement to provide 'the best scientific data available' to exclude highly relevant research because its methodology—like most studies—can be criticized effectively eviscerates the requirement to use the best available science and rewrites the standard to perfect science.").

When the available evidence supports an effect, the Agency must give weight to that effect. *See Defenders of Wildlife v. Jewell*, 176 F. Supp. 3d 975, 1003 (D. Mont. 2016). Under the "best available science" standard, the Agency "cannot ignore available biological information." *Conner v. Burford*, 848 F.2d 1441, 1454 (9th Cir. 1988). It also "prohibits [an Agency] from disregarding available scientific evidence that is in some way better than the evidence [it] relies on.'" *San Luis & Delta-Mendota Water Auth. v. Jewell*, 747 F.3d 581, 602 (9th Cir. 2014) (quoting *Kern County Farm Bureau v. Allen*, 450 F.3d 1072, 1080 (9th Cir. 2006)). It is undisputed that, *with respect to congenital heart defects*, the Johnson et al. 2003 study is better evidence than the studies of immunosuppression and autoimmunity, which did not analyze congenital heart defects at all. The problem with EPA's analysis is that it dismisses the best available science on congenital heart defects by pointing to studies that do not analyze or consider that endpoint. Even if the studies on immunosuppression and autoimmunity are higher quality, they do not assess congenital heart defects and thus do not speak to this effect.

To draw an analogy to listing species under the Endangered Species Act—another context where the federal government must use the best available science—EPA's analysis is the equivalent of dismissing habitat destruction despite a finding that habitat is likely to be destroyed simply

because the Agency has even greater confidence that the species is also threatened by disease. But the logical and scientific approach is to consider both effects, in conjunction, and that is how the agencies tasked with listing species have historically considered such effects. In the context of risk evaluations for chemicals under TSCA, one accounts for multiple effects by selecting the most sensitive endpoint for risk characterization. By addressing the risks for these effects, one also is able to "address other identified risks"—which is allegedly EPA's goal. (p. 377). In contrast, EPA's approach of selecting the endpoint with the greatest confidence *does* not "address other identified risks"—it leaves risks from congenital heart defects insufficiently addressed, as indicated by the lower levels of exposure to TCE that cause those defects relative to exposures required to cause the immune effects.

H. Developments during the course of the preparation of the draft risk evaluation and its peer review raise serious questions about EPA's failure to use congenital heart defects as the key driver of TCE's risks.

i. The draft risk evaluation was subject to political interference by the Trump White House.

Late in the process of drafting the TCE risk evaluation, political appointees in the Trump White House intervened and forced EPA career staff to make fundamental changes to the draft before it was released to the public. The changes were those sought by the appointees as well as apparently by other federal entities, such as the Department of Defense, that have vested interests due to their use of TCE and responsibility for exposures associated with those uses or with contaminated sites or their cleanup.

This interference only came to light as a result of an in-depth investigation conducted by Elizabeth Shogren of the Center for Investigative Reporting and published in Reveal News on February 20, 2020.[167] The investigation found that the draft of the risk evaluation completed by EPA in December 2019 and sent to the White House for review utilized as the key health endpoint for is risk determinations congenital heart defects; this health endpoint has been used repeatedly by EPA in prior assessments and has been subject to numerous rounds of peer review and public comment.[168]

[167] Shogren, Elizabeth. "EPA scientists found a toxic chemical damages fetal hearts. The Trump White House rewrote their assessment." Reveal News, February 28, 2020, available at https://www.revealnews.org/article/epa-scientists-found-a-toxic-chemical-damages-fetal-hearts-the-trump-white-house-rewrote-their-assessment/.

[168] For a list of the prior EPA assessments utilizing congenital heart defects as the key risk driver, and peer and public reviews to which they were subject, see Appendix 3.

The draft of the risk evaluation released to the public on February 21, 2020 and also provided at that time to the SACC for peer review, however, had been drastically modified by replacing the congenital heart defects endpoint as the key risk driver with immune endpoints that EPA deemed arise only at much higher levels of TCE exposure.

The initial Reveal News article included some key excerpts from the December 2019 draft risk evaluation. Subsequently, Reveal News has made public a full copy of that draft.[169]

At the TCE SACC meeting, a member of the review panel noted her concern with the prospect of political interference with the draft risk evaluation by the Trump White House and called for the SACC to investigate the matter further in the course of its peer review.

EDF believes that this sorry episode heavily taints the scientific integrity and credibility of EPA's draft risk evaluation. As we have made clear in other sections of these comments (including subsections D. and G., above), the decision not to rely on congenital heart defects as the key driver for EPA's determinations of TCE's acute and chronic risks deviates from scientific best practices, defies requirements under the law, and is not sufficiently protective of public health – particularly the health of especially vulnerable subpopulations.

ii. The SACC peer review panel lacked critical expertise in pediatric health and heart development.

Despite requests by a number of SACC members to postpone the peer review in the face of the unfolding COVID-19 crisis, EPA elected to proceed with a virtual meeting of the peer review panel for the draft TCE risk evaluation. The virtual meeting went forward as originally scheduled on March 24-27, 2020.

At least two members of the peer review panel were not able to attend any of the meeting, and several other members were able to attend only portions of the meeting - at least in some cases due to their responsibilities as members of the public health community responding to the COVID-19 crisis.[170] One of the members unable to attend at all is one of the few members of the panel with expertise in pediatric environmental health.

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[169] The December 2019 draft is available via a link provided in Shogren, Elizabeth, "EPA science panel plows ahead with toxic chemical's review, despite coronavirus crisis," Reveal News, March 26, 2020. The link to the December 2019 draft (note that the file is extremely large) is: https://www.documentcloud.org/documents/6819132-Evaluation.html.

[170] EDF submitted a comment to the docket for the SACC peer review of the TCE draft risk evaluation documenting the absence of two panel members because their absence was not noted to other members or the public during the course of the meeting. See https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0500-0070.

Given the longstanding, central debate over congenital heart defects as a key risk driver for TCE, it is particularly concerning that the SACC peer review panel lacked anyone with specific expertise in heart development. Numerous researchers are active in this area but to our knowledge none were contacted about inclusion in this panel. This is a serious omission that taints the strength of the peer review of the draft.

iii. Key questions were left unanswered at the SACC peer review meeting of the TCE draft risk evaluation.

The discussion of the congenital heart defects issue at the TCE SACC meeting clearly indicated there was not consensus among its members over EPA's eleventh-hour decision to jettison its reliance on that health endpoint as the key driver for TCE's risks. Some members stated that they believe the developmental toxicity of TCE exposure is a critical endpoint supported by the weight of scientific evidence, even while acknowledging that some uncertainty exists in the available literature that poses challenges for moving from hazard identification to dose-response modeling. Some members also noted the extreme nature of the scrutiny paid to studies identifying congenital heart defects in comparison to that applied to the immune studies or other studies on which EPA relied (see subsection D.ii.b. above) as well as changes made in response to political interference (see subsection H.i. above).

Other members supported EPA's decision, arguing that the congenital heart defects endpoint was an outlier, emphasizing what they saw as flaws in the design of studies identifying that endpoint and placing greater weight on studies sponsored by the chemical industry that failed to replicate those effects.

In response to this discussion, EPA career staff posed some key questions to the SACC: How can EPA protect against risks of a health endpoint that the weight of evidence indicates is real and that some studies show occurs at very low doses, if there is a view by some that the data are not ideal for dose-response modeling? How would reliance instead on a non-developmental endpoint that shows effects only at higher doses fulfill EPA's responsibility under TSCA to identity and protect against risks to the most vulnerable subpopulation?

For the reasons provided elsewhere in these comments (see subsections D. and G.), EDF strongly believes that the evidence for congenital heart defects is both compelling and amendable to dose-response modeling. EDF also believes EPA must rely on this endpoint in order to ensure it is in fact protecting the most vulnerable subpopulation from the risks of TCE exposure.

I. EPA's chronic non-cancer PODs and related calculations depart from prior assessment decisions and are not sufficiently protective of public health.

i. EPA dismissed the NTP study of kidney toxicity without sufficient justification.

In this draft risk evaluation, EPA selects Maltoni et al. 1986 as the representative study for the kidney toxicity endpoint. This is a departure from the 2014 Work Plan Assessment, in which the NTP 1988 study was selected because it provided the lowest POD. It should be noted that both studies were rated as "Medium" quality in the draft risk evaluation. Yet, as demonstrated in Table 3-9, selecting the Maltoni et al. 1986 study results in an HEC_{99} that is nearly five times higher than the HEC_{99} derived from the NTP study (Maltoni et al. HEC_{99} = 0.025 ppm; NTP HEC_{99} = 0.0056 ppm).

EPA seeks to justify this decision because the "elevated doses in (NTP, 1988) resulted in massive nephrotoxicity and introduce large uncertainty in BMD modeling the effects at low doses well below the tested doses with a BMR well below the observed effect incidence in the study" (p. 242). These issues with the NTP 1988 study were directly addressed in the 2011 IRIS assessment and deemed not to represent a concern sufficient to warrant not relying on the NTP study: "This BMR required substantial extrapolation below the observed responses (about 60%); however, the response level seemed warranted for this type of effect and the ratio of the BMD to the BMDL was not large (1.56)."[171] Furthermore, the 2011 IRIS assessment highlights that the "effect observed in NTP (1988) is more severe."[172]

Therefore, given that 1) the NTP study provides the *lowest* HEC_{99} on the *most severe* kidney toxicity endpoint and 2) modeling challenges did not present concerns in prior assessments, EPA should select the POD from the NTP 1988 study rather than the Maltoni et al. 1986 study to represent the kidney toxicity endpoint.

ii. EPA dismissed key immunotoxicity endpoints without sufficient justification.

In another departure from the 2014 Work Plan Assessment, the Agency decided not to consider decreased thymus weight and cellularity (observed in Keil et al. 2009) in the risk estimation process for immunotoxicity because it deemed these endpoints to be "insufficiently adverse compared to other endpoints" (p. 245). However, the 2011 IRIS Assessment takes a clear and distinct position on this, stating "[d]ecreased thymus weight reported at relatively low exposures in nonautoimmune-prone mice is a *clear indicator of immunotoxicity* (Keil et al. 2009), and is

[171] EPA IRIS Assessment for Trichloroethylene. 2011. Available at: https://cfpub.epa.gov/ncea/iris2/chemicalLanding.cfm?substance_nmbr=199#tab-1, p. 5-20.
[172] Ibid.

therefore considered a candidate critical effect" (emphasis added).[173] As such, EPA has not provided sufficient justification for dismissing this endpoint from the risk evaluation process and should not do so.

The implications of this decision – in conjunction with the Agency's dismissal of the Johnson et al. 2003 study as a representative chronic non-cancer study (see subsection D. above) – are highly consequential. Utilizing the alternative endpoint of autoimmunity from Keil et al. 2009 results in an approximately 9-fold underestimation of risk compared to what would have been calculated using the Johnson study.

iii. An inappropriate uncertainty factor (UF) value was utilized for chronic non-cancer POD modeling based on the autoimmunity endpoint from Keil et al. 2009.

EPA uses the autoimmunity endpoint from Keil et al. 2009, rather than decreased thymus weight and cellularity from the same study (see subsection I. ii. above), as the representative chronic non-cancer endpoint for POD modeling.

During the TCE SACC meeting, several panelists criticized EPA's decision to use a value of "3", rather than the default of "10," as the LOAEL to NOAEL UF (UF_L) for the Keil et al. autoimmunity endpoint. EPA justifies this decision by stating that "the observed effect ….is considered an early, subclinical or pre-clinical early marker of disease" (p. 245). However, SACC panelists asserted that autoimmunity (i.e., changes in antibody levels) should itself be considered a relevant immune effect rather than only a precursor or subclinical marker. This scenario could be viewed as analogous to considering liver enzyme changes as a marker of liver toxicity.

When revising the draft risk evaluation, EPA should use a UF_L of 10 for the Keil et al. 2009 autoimmunity endpoint.

J. EPA needs to apply an uncertainty factor (UF) to account for lack of dermal toxicity data.

The draft risk evaluation states (p. 279): "EPA used a previously developed peer-reviewed PBPK model in order to obtain both HECs and HEDs from animal toxicological studies involving either oral or inhalation administration of TCE. The PBPK model does not account for dermal exposure, so EPA relied on traditional route-to-route extrapolation from oral HED values."

[173] EPA IRIS Assessment for Trichloroethylene. 2011. Available at: https://cfpub.epa.gov/ncea/iris2/chemicalLanding.cfm?substance_nmbr=199#tab-1, p. 5-27

As EDF has commented on for prior draft risk evaluations that have taken a similar approach, EPA's decision to rely on inhalation-to-dermal extrapolation contributes substantial uncertainty to its risk calculations. Therefore, as is recommended for route-to-route extrapolation generally,[174,175] EPA should apply an additional uncertainty factor of 10 to account for these uncertainties.

K. EPA should migrate to a unified approach to presenting dose-specific population risks for both cancer and noncancer endpoints.

More broadly, EPA must employ health-protective approaches to dose-response modeling, as described at length in the National Academy of Sciences (NAS) report, *Science and Decisions: Advancing Risk Assessment*.[176] Among other recommendations, the NAS argued that "***cancer and noncancer responses [to chemical exposures] be assumed to be linear as a default ***."[177]

The MOE approach presented in the TCE draft risk evaluation provides a bright-line, yes/no approach to risk and fails to provide a measure of population risk at a given exposure level. This approach limits the assessment's utility for risk managers, particularly when comparing options for substitution and conducting risk-benefit comparisons.

The National Academy of Sciences (NAS) Committee on Improving Risk Analysis Approaches Used by the U.S. EPA concluded "separation of cancer and noncancer outcomes in dose response analysis is artificial because noncancer endpoints can occur without a threshold or low dose nonlinearity at the population level."[178] The Committee further stated that background exposures and underlying disease processes can contribute to background risk and lead to linearity at population doses of concern.

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[174] *See, e.g.*, Schröder, K. et al., *Evaluation of route-to-route extrapolation factors based on assessment of repeated dose toxicity studies compiled in the database RepDose®*, 261 TOXICOLOGY LETTERS 32-40 (Nov. 2016), https://www.ncbi.nlm.nih.gov/pubmed/27553675; Dankovic, D.A. et al., *The scientific basis of uncertainty factors used in setting occupational exposure limits*, 12 J. OF OCCUPATIONAL & ENVTL. HYGIENE 55-68 (Nov. 2015), https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4643360/.

[175] Rennen, MA et al., *Oral-to-inhalation route extrapolation in occupational health risk assessment: a critical assessment*, 39:1 REG. TOXICOLOGY & PHARMACOLOGY 5-11 (2004), https://www.ncbi.nlm.nih.gov/pubmed/14746775.

[176] NAT'L RESEARCH COUNCIL, SCIENCE AND DECISIONS: ADVANCING RISK ASSESSMENT (2009), https://www.ncbi.nlm.nih.gov/books/NBK214630/.

[177] *Id.* at chp. 5, p. 180.

[178] *Id.* at chp. 5, p. 177.

This issue was also highlighted in the 2019 NASEM *Review of DOD's Approach to Deriving an Occupational Exposure Level for Trichloroethylene:*

> The committee recommendation, consistent with NRC (2009a), is to include all health end points within a unified framework for dose-response assessment.[179]

EPA should implement the recommendations repeatedly made by the NAS and develop a unified approach to presenting dose-specific population risks for both cancer and noncancer endpoints.

5. EPA's human exposure assessment raises significant questions and concerns.

A. EPA's unwarranted assumption of respirator and glove use obscures the full extent of unreasonable risk to workers posed by exposure to TCE.

i. Context and summary

EPA presents its occupational risk determinations in Section 5.3 (pp. 383-420). While EPA has determined that each condition of use relevant to workers presented an unreasonable risk overall, it still dramatically understates the extent of actual unreasonable inhalation and dermal risks to workers.

As discussed elsewhere in the comments (section 1.B.), EPA has adopted a flawed assumption – absent any empirical evidence to support it – that workers under most industrial/commercial conditions of use of TCE will always wear fully effective personal protective equipment (PPE).

EPA's application of this assumption to workers under the various conditions of use of TCE dramatically altered its final risk estimates for workers – both in cases where EPA did find a condition of use presented an unreasonable risk overall and in cases where it did not.
In many cases, EPA has used its assumption of PPE to assert that a risk estimate that it found exceeded its risk benchmark in the absence of PPE no longer represents an unreasonable risk. In other cases, EPA found that even with PPE use, a risk estimate still exceeded its benchmark; in those cases EPA presented the risk estimate with PPE as the driver of its determination, thereby understating the magnitude of the actual risk to workers.

To characterize the impact of EPA's PPE assumption, EDF undertook an analysis of the risk estimates summarized in EPA's Table 4-54 (pp. 358-369); this table shows: 1) the risk estimates EPA calculated *before* it applied its assumption regarding PPE use; 2) whether EPA's assumption of PPE could make enough risk go away so that EPA could claim that risk estimate

[179] National Academies of Sciences, Engineering, and Medicine. 2019. *Review of DOD's Approach to Deriving an Occupational Exposure Level for Trichloroethylene*. Washington, DC: The National Academies Press. https://doi.org/10.17226/25610.

did not represent an unreasonable risk; 3) if so, what degree of PPE efficiency EPA had to assume would be used; and 4) the risk estimates EPA calculated *after* it applied its assumption regarding PPE use. EDF's analysis is provided in the attached Excel file submitted as Appendix 6 along with these comments.

Our examination revealed the following:

- There are only two kinds of scenarios under which EPA did find that a risk estimate for a given condition of use represented an unreasonable occupational risk:
    - **Scenarios where the risk estimate EPA calculated is so high that it could not make it go away even *after* assuming that workers would always use the most protective PPE that EPA considered**. This would often require use of a highly efficient (and highly cumbersome) respirator with an "assigned protection factor" (APF) of 50, i.e., one that reduces air concentrations by 50-fold, or gloves providing a 10- or 20-fold protection factor (PF).
    - **Scenarios where EPA could not plausibly assume *any* use of respirators**. This applies to EPA's risk determinations for workers under five COUs[180] and for occupational non-users (ONUs) under all conditions of use.
- For nearly all conditions of use where EPA found that its risk estimates for acute, chronic or cancer risks to workers did not represent an unreasonable risk, in order to reach that finding, **EPA had to assume that all of the workers were using respirators or gloves, or both**.
- Even where EPA did find unreasonable risk to workers, **EPA has grossly understated both the extent and magnitude of those risks by assuming use of PPE**.

Put another way: For every one of the conditions of use where EPA could assume PPE might plausibly be used by some workers, EPA either:

- avoided identifying its risk estimates as representing an unreasonable risk ***only*** by assuming universal, effective use of respirators, gloves, or both; or

[180] For these five COUs EPA still assumed all workers would wear gloves. The COUs are:

- Industrial/Commercial Use – Cleaning and furniture care products – Carpet cleaner; wipe cleaning
- Industrial/Commercial Use – Laundry and dishwashing products – Spot remover
- Industrial/Commercial Use – Ink, toner, and colorant products – Toner aid
- Industrial/Commercial Use – Apparel and footwear care products – Shoe polish
- Industrial/Commercial Use – Hoof polishes; gun scrubber; pepper spray; other miscellaneous industrial and commercial uses

- found its risk estimates to represent unreasonable risk even with the use of such respirators or gloves – but, by relying on the risk estimates calculated assuming the PPE, thereby grossly understated both the extent and magnitude of the risk.

Consider this example for EPA's "domestic manufacturing" condition of use, as shown in Table 4-54 (p. 358):

- Absent PPE, all 12 of EPA's risk estimates for workers exceeded its benchmarks and represented unreasonable risks: 3 endpoints (acute, chronic, and cancer risks) x 2 exposures routes (dermal and inhalation) x 2 exposure levels (high-end and central tendency) = 12 risk estimates; all are boldfaced/shaded in the "no PPE" columns.
- After applying its PPE assumptions:
    - Four of these 12 risk estimates are no longer deemed unreasonable (not boldfaced/shaded).
    - The other eight risk estimates are still unreasonable (boldfaced/shaded) – *but* the magnitude of each of these has been adjusted by a factor representing the level of protection assumed to have been provided by the specified PPE. These adjusted risk estimates are the ones identified by EPA in its final risk determination for this condition of use on p. 383 of the draft risk evaluation.

EPA's unwarranted approach raises major concerns. First, risk estimates that are not carried over into the final risk determinations may not subsequently be regulated, forgoing EPA's only opportunity to ensure that PPE it assumed is actually used and workers are protected.

Second and equally or more important in this draft risk evaluation, even though EPA does find all occupational conditions of use do present unreasonable risk to workers, by carrying over into its final risk determinations risk estimates that are understated because of its PPE assumptions, any subsequent regulation EPA promulgates under TSCA will be under-protective of workers.

The magnitude of this underestimation is very large (see subsection A.ii. below and the "Averages" tab of the Excel file submitted as Appendix 6 along with these comments for details):

- For ***acute risks*** there is a 16-fold underestimation overall; 50-fold for inhalation exposure, and 14-fold for dermal exposure.
- For ***chronic risks*** there is a 34-fold underestimation overall; 49-fold for inhalation exposure, and 18-fold for dermal exposure.
- For ***cancer risks*** there is a 23-fold underestimation overall; 50-fold for inhalation exposure, and 16-fold for dermal exposure.

ii. Detailed analysis of the effect of EPA's assumed routine use of PPE on estimates of worker risks

Based on Table 4-54, EPA estimated worker risks for 102 combinations of industrial/commercial COUs, exposure routes (inhalation or dermal) and exposure levels (high-end or central tendency). For all but seven (i.e., 95) for acute risk as well as five (i.e., 97) for chronic and cancer risks of these cases, EPA found excessive risk in the absence of PPE. Then in most of these cases, EPA assumed routine use of PPE. Our analysis of these cases found the following:

ACUTE TOXICITY: EPA identified excessive risk to workers in 95 cases in the absence of PPE.

- For 44 of the 95 cases, EPA identified no unreasonable risk *only* after assuming the use of PPE.
  - For 23 of those cases EPA assumed use of a respirator:
    - with an APF of 50 for 12 cases; and
    - with an APF of 10 for the other 11 cases.
  - For the other 21 of those cases EPA assumed use of gloves:
    - with a PF of 20 for 3 cases;
    - with a PF of 10 for 17 cases; and
    - with a PF of 5 for 1 case.
- For 5 of the 95 cases, EPA identified unreasonable risk without assuming the use of a respirator.
- For the other 46 of the 95 cases, EPA identified unreasonable risk *even after* assuming the use of PPE (either respirators or gloves).
  - For the 13 cases involving inhalation exposure, even EPA's assumed use of a respirator with an APF of 50 was not enough to eliminate the unreasonable risk.
  - For the other 33 cases involving dermal exposure, even EPA's assumed use of gloves with a PF of 20 (22 cases) or PF of 10 (11 cases) was not enough to eliminate the unreasonable risk.

  However, EPA's assumption of PPE use greatly underestimates the magnitude of that unreasonable risk. Averaging the calculated MOEs with and without PPE across these 46 combinations of industrial/commercial COUs, exposure routes (inhalation or dermal) and exposure levels (high-end or central tendency), EPA's PPE assumption yielded MOEs for acute toxicity that were higher than those without PPE:
  - by 16-fold overall;
  - by 50-fold for inhalation exposure; and
  - by 14-fold for dermal exposure.

CHRONIC TOXICITY: EPA identified excessive risk to workers in 97 cases in the absence of PPE.

- For 1 of the 97 cases, EPA identified no unreasonable risk *only* after assuming the use of a respirator with an APF of 50.
- For another 6 of the 97 cases, EPA identified unreasonable risk without assuming the use of a respirator.
- For the other 90 of the 97 cases, EPA identified unreasonable risk *even after* assuming the use of PPE (either respirators or gloves).
    - For the 36 cases involving inhalation exposure, even EPA's assumed use of a respirator with an APF of 50 was not enough to eliminate the unreasonable risk.
    - For the 54 cases involving dermal exposure, even EPA's assumed use of gloves with a PF of 20 (42 cases) or PF of 10 (12 cases) was not enough to eliminate the unreasonable risk.

  However, EPA's assumption of PPE use greatly underestimates the magnitude of that unreasonable risk. Averaging the calculated MOEs with and without PPE across these 90 combinations of industrial/commercial COUs, exposure routes (inhalation or dermal) and exposure levels (high-end or central tendency), EPA's PPE assumption yielded MOEs for chronic toxicity that were higher than those without PPE:
    - by 34-fold overall;
    - by 49-fold for inhalation exposure; and
    - by 18-fold for dermal exposure.

CANCER (Even using EPA's overly lax cancer risk benchmark; see section 7.A.ii.): EPA identified excessive risk to workers in 97 cases in the absence of PPE.

- For 12 of the 97 cases, EPA identified no unreasonable risk *only* after assuming the use of a respirator with an APF of 50 (8 cases) or APF of 10 (4 cases).
- For another 6 of the 97 cases, EPA identified unreasonable risk without assuming the use of a respirator.
- For the other 79 of the 97 cases, EPA identified unreasonable risk *even after* assuming the use of PPE (either respirators or gloves).
    - For the 25 cases involving inhalation exposure, even EPA's assumed use of a respirator with an APF of 50 was not enough to eliminate the unreasonable risk.
    - For the 54 cases involving dermal exposure, even EPA's assumed use of gloves with a PF of 20 (42 cases) or PF of 10 (12 cases) was not enough to eliminate the unreasonable risk.

  However, EPA's assumption of PPE use greatly underestimates the magnitude of that unreasonable risk. Averaging the calculated MOEs with and without PPE across these 79 combinations of industrial/commercial COUs, exposure routes (inhalation or dermal) and

exposure levels (high-end or central tendency), EPA's PPE assumption yielded cancer risk estimates that were lower than those without PPE:

- by 23-fold overall;
- by 50-fold for inhalation exposure; and
- by 16-fold for dermal exposure.

B. EPA has underestimated occupational and consumer exposures.

i. EPA has failed to consider combined exposure from multiple pathways.

EPA never considers the combined risks from the inhalation and dermal exposures it calculates – even though many workers and consumers could readily experience exposures by both routes, including over the same time period. For example, in the context of estimating dermal exposure, the Agency describes TCE's rapid evaporation:

> Instantaneous exposures to skin are expected to evaporate before significant dermal absorption occurs based on TCE's physical chemical properties which include the vapor pressure, water solubility and log Kow. (p. 137)

Such rapid evaporation from the skin would lead to increased concentration in the air in the immediate vicinity of the dermally exposed worker. Because both inhalation and dermal exposure result in systemic distribution of TCE, (e.g., EPA states that "[r]egardless of the route of exposure, TCE is widely distributed throughout the body," p. 203) it is essential to evaluate exposures from both of these routes in combination, including simultaneously, to assess total body burden and the associated effects.

EPA does acknowledge in the draft risk evaluation that workers and consumers may experience both inhalation and dermal exposures simultaneously:

> Inhalation and dermal exposures are assumed to occur simultaneously for workers and consumers. (p. 33)

> Dermal exposures to liquid TCE are expected to be concurrent with inhalation exposures. (p. 137)

However, EPA quickly dismisses with insufficient justification an additivity approach to assess overall exposure:

> EPA chose not to employ simple additivity of exposure pathways at this time within a condition of use because of the uncertainties present in the current exposure estimation procedures, which may may [*sic*] lead to an underestimate or overestimate of the actual total exposure. (pp. 33-34)

EPA's decision not to apply an additivity approach because of "uncertainties" will necessarily result in an underestimate of exposure – something EPA acknowledged in its draft methylene chloride risk evaluation[181] but has failed to do here.

Our concern is reinforced by the comments of several SACC members made during peer review meetings, who have repeatedly called on the Agency to combine the inhalation and dermal exposures. The SACC expanded on this concern in several of its reports on earlier draft evaluations. For example:

- 1-Bromopropane SACC Meeting Minutes and Final Report: The SACC stated, "In addition, inclusion of an estimate of combined oral and dermal exposure would be welcome."[182]
- Methylene Chloride SACC Meeting Minutes and Final Report: In responses to Charge Question 6.2 on uncertainties and assumptions not adequately presented, the SACC asked EPA: "Effects of simultaneous dermal and inhalation exposures: Inhalation and dermal exposure to methylene chloride can occur simultaneously. Are effects simply additive (an undiscussed assumption)?"[183]

During the TCE SACC meeting many panelists reiterated this concern. In response to Question 6.3, one peer reviewer specifically indicated that they did not believe it appropriate to ignore exposures in a risk assessment based on statutory arguments.

Another concern raised by a SACC member during the 1,4 dioxane peer review is salient for TCE as well: EPA has ignored all non-occupational baseline exposures workers experience, due to its exclusion of all exposures via environmental releases to air, water, and land. The SACC member argued that the Agency at least needs to take these into account as baseline exposures for workers, even if the Agency persists in not considering them as arising from conditions of use it has included within the scope of the risk evaluation. In other words, even if the Agency does not intend to assess risks from environmental releases of TCE through the air, water, and land (due to its assertions as to the adequacy of actions taken under other statutes), EPA cannot ignore these real-world exposures when assessing the risk TCE presents to an individual.

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[181] Draft Risk Evaluation for Methylene Chloride, p. 387. Available: https://www.epa.gov/sites/production/files/2019-10/documents/1_methylene_chloride_risk_evaluation_peer_review_draft_heronet_public.pdf.

[182] 1-Bromopropane SACC Meeting Minutes and Final Report, December 2019, available: https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0235-0061, p. 13.

[183] Methylene Chloride SACC Meeting Minutes and Final Report, March 2020, available: https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0437-0080, p. 69.

ii. EPA may have underestimated exposure to ONUs.

We support EPA's decision to assume that occupational non-users (ONUs) will not wear respirators. Beyond the concerns we raised earlier with assumptions that workers handling a chemical will consistently wear PPE and that it will be universally effective, it would be even more unrealistic to assume that ONUs would wear any PPE. This point was raised repeatedly by SACC members during their 1,4-dioxane peer review meeting.

Nevertheless, EPA may still have underestimated exposure to ONUs in several ways. First, as discussed in detail in section 7.A.iii., EPA assumes central tendency exposures for ONUs in any case where it does not have monitoring data or modeling specific to ONUs.

Second, where EPA does have data to estimate exposure of ONUs specifically, the Agency assumes that they are only present in the "far field zone" – i.e., outside of the "near field" workers' zone (p. 127). However, ONUs may not stay within the "far field zone." Several SACC members raised this concern during the 1-BP peer review meeting. For example, a SACC member with industrial hygiene experience noted that workers and ONUs may regularly pass into each other's space, e.g., to communicate or otherwise interact. EPA fails to recognize this. Under section 2.3.1.3 ("Assumptions and Key Sources of Uncertainty for Occupational Exposures"), EPA describes the potential for its method to overestimate exposure to workers – but fails to acknowledge that the converse is true, i.e., the method may underestimate exposure to ONUs.[184]

iii. EPA has failed to explain or justify its assumption of one exposure event per day.

In its dermal exposure assessment, EPA assumes one exposure event per day for both workers (pp. 101, 117) and consumers (p. 140). EPA provides little justification for these assumptions.

Yet, for workers, given the typical 8-hour (or longer) work day and the repetition common in many jobs, it seems far more likely that workers would regularly engage in activities that could result in multiple exposure events per day. While EPA recognizes this under its section on assumptions and uncertainties (p. 128), it fails to acknowledge that this assumption will underestimate exposure (as the Agency has done for other chemicals, e.g., methylene chloride

[184] "Exposures are calculated by assuming workers spend the entire activity duration in their respective exposure zones (i.e., the worker in the near-field and the occupational non-user in the far-field). Since vapor degreasing and cold cleaning involve automated processes, a worker may actually walk away from the near-field during part of the process and return when it is time to unload the degreaser. As such, assuming the worker is exposed at the near-field concentration for the entire activity duration may overestimate exposure." P. 127.

draft risk evaluation, pp. 165, 375). EPA has not, but must, account for this underestimation and at a minimum provide an uncertainty analysis.

With regards to consumers, EPA also assumes a single exposure event per day, although it does take into account varying durations of exposure (p. 349). This assumption is particularly problematic for "do-it-yourselfers," which EPA acknowledged may be exposed more than once per day: "EPA assumes that a consumer product would be used only once per day. This is a reasonable assumption for most scenarios, but a Do-It-Yourself- (DIY-) type user could potentially use the same product multiple times in one day" (p. 178). Yet EPA fails to actually address this scenario in calculating exposure and risk estimates.

EPA also fails to assess any chronic exposures to consumers despite acknowledging in several places in the draft risk evaluation they are expected to occur:

> Although high-end frequencies of consumer use are up to 50 times per year, reasonably available toxicological data is based on either single or continuous TCE exposure and it is unknown whether these use patterns are expected to be clustered or intermittent (e.g. one time per week). There is uncertainty regarding the extrapolation from continuous studies in animals to the case of repeated, intermittent human exposures. Therefore, EPA cannot fully rule out that consumers at the high-end frequency of use could possibly be at risk for chronic hazard effects, however it is expected to be unlikely. (p. 136)

> [C]hronic exposures were not evaluated for TCE-containing consumer products. However, it is possible that there would be concern for chronic exposure effects for use frequencies greater than intermittent. For example, daily or DIY-type uses of consumer products could constitute a short-term chronic exposure scenario or repeated-acute exposure scenario that is not captured in this evaluation. Identified chronic non-cancer and cancer hazard endpoints (Section 3.2) are unlikely to present for these populations based on reasonably available information, however the possibility cannot be ruled out. For the vast majority of the consumer population which are only exposed through short-term, occasional use of TCE products, only acute exposure is applicable. (p. 178)

While chronic exposure may not be typical for consumers, EPA's failure to assess DIY users as a "potentially exposed or susceptible subpopulation" is troubling. This is especially the case given that EPA implies that it considered DIY users as a sentinel exposure under section 4.4.2 of the draft risk evaluation:

> In terms of this risk evaluation, EPA considered sentinel exposures by considering risks to populations who may have upper bound exposures – for example, workers and ONUs who perform activities with higher exposure potential, or consumers

> who have higher exposure potential (e.g., ***those involved with do-it-yourself projects***) or certain physical factors like body weight or skin surface area exposed. EPA characterized high-end exposures in evaluating exposure using both monitoring data and modeling approaches. (p. 353, emphasis added)

EPA's assumptions about consumer exposure are likely to significantly underestimate the risks they face, which EPA recognizes under section 2.3.2.8 of the draft risk evaluation.[185] At the very least, EPA needs to conduct a sensitivity analysis regarding these assumptions in the context of this risk evaluation, which is different than the sensitivity analysis EPA indicates was done on the model itself (p. 516).

During the TCE SACC meeting, there appeared to be a consensus among panelists assigned to Charge Questions 4.7 and 4.8 that EPA should estimate chronic exposure to consumers. Their rationale included that consumers are likely to use more than one product containing TCE and that actual exposures may be more frequent than EPA assumes in the model based on a single type of product and single exposure scenario at a time. One peer reviewer provided an apt example: Products containing TCE and stored in a garage or elsewhere in the home may continue emitting TCE at a low level – leading to chronic exposures. Finally, the same peer reviewer repeatedly noted that the WESTAT survey data EPA utilizes for consumer products and use patterns may be outdated, and unlikely to represent the full range of high frequency users.

iv. EPA's assessment of dermal exposure likely underestimates exposure due to its crude assumptions about glove use and efficacy.

As noted above, section 1.B., EPA does not have any actual data on glove use and efficacy, which is necessary to accurately assess dermal exposure. While EPA skirts around this issue in the draft risk evaluation itself, the Supplemental File: Environmental Releases and Occupational Exposure states:

> Data about the frequency of effective glove use – that is, the proper use of effective gloves – is very limited in industrial settings. Initial literature review suggests that there is unlikely to be sufficient data to justify a specific probability distribution for effective glove use for a chemical or industry. (p. 223 of supplement)

[185] "However, ease of access to products on-line or in big box stores (like home improvement stores), readily accessible how-to videos, and a consumer movement toward more do-it-yourself projects with products containing the chemical of concern could impact the representativeness of the consumer use patterns described within the Westat Survey and may lead to an underestimate of overall consumer exposure." P. 180.

EPA recognizes the potential for occlusion, whereby glove use can *increase* skin exposure, in both the draft risk evaluation (e.g., "Dermal exposure may be significant in cases of occluded exposure," p. 116) and the Supplemental File: Environmental Releases and Occupational Exposures (e.g., "Many gloves do not resist the penetration of low molecular weight chemicals… Wearing gloves which are internally contaminated can lead to increased systemic absorption," p. 221). The Agency says that it calculated exposures under occluded scenarios:

> EPA also estimated central tendency and high-end dermal retained doses for occluded scenarios for OESs [occupational exposure scenarios] where occlusion was reasonably expected to occur. Occluded scenarios are generally expected where workers come into contact with bulk liquid TCE during use in open systems (e.g., during solvent changeout in vapor degreasing) and not expected in closed-type systems (e.g., during connection/ disconnection of hoses used in loading of bulk containers in manufacturing). (p. 102)

These exposure estimates are reflected in Table 2-15 (p.106); notably, EPA found exposures that are *7.6-12.2 times higher* than the no-glove scenarios.[186]

However, it appears that the exposure estimates under occluded conditions are not actually incorporated at all into the ultimate risk estimates and risk determinations for the occupational scenarios. For example, when one compares Table 2-15 to Tables 4-6 through 4-27, the occluded exposure scenarios appear to disappear from the risk estimates shown in the latter tables. Likewise, occluded scenarios do not appear in the Supplemental Information File: Risk Calculator for Occupational Exposures (e.g., see tab "RR" in EPA's "TCE-Risk Calculator for Occupational Exposures" spreadsheet).[187] If EPA did in fact incorporate occlusion into its ultimate risk estimates and risk determinations, it needs to be far clearer on how it did so.

Instead, the Agency simply uses default glove protection factors, ignoring the elevated dermal exposures of workers in occluded scenarios. More specifically, the Agency assumes fixed protection factors (PFs) of 5x, 10x, and 20x. While EPA has now described somewhat more detailed scenarios for each assumed PF (see Table 2-20, p. 117), they do not appear to be supported by any empirical data that account for the complexities of glove use in the real world. EPA skirts around the issue in this draft risk evaluation, but previously acknowledged in the draft

[186] For most COUs, EPA calculated an occluded exposure of 2,247 mg/day compared to 184.36 mg/day for high end exposure without gloves (2,247/184.36 = **12.19**). For Commercial Printing and Copying, EPA calculated an occluded exposure of 786 mg/day compared to 101.3 mg/day for high end exposure without gloves (786/101.3 = **7.59**).

[187] TCE-Risk Calculator for Occupational Exposures, Excel spreadsheet, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0500-0021.

methylene chloride risk evaluation that the glove protection factors are "'what-if' assumptions and are uncertain" (methylene chloride draft risk evaluation, p. 166).[188] Further, the Agency fails to acknowledge the uncertainties and deficiencies in its glove use assumptions in the Risk Determination section of this draft risk evaluation.

During both the 1,4-dioxane and 1-BP SAAC peer review meetings, a SACC member who is a dermal exposure expert expressed his concern with EPA's approach, which EPA has repeated for each chemical, including TCE. He noted that glove testing is typically conducted in a lab under ideal conditions – without an actual human hand present. However, in the real world, an insufficiently trained or attentive user may contaminate a glove, leading to occlusion and higher exposure. Likewise, permeable gloves may enable the chemical to be absorbed through the glove, while preventing or slowing evaporation. Gloves can also increase skin temperature and humidity, which can increase absorption. Therefore, the assumption that PFs can only range as low as 1x (no gloves) is erroneous; rather, the range should include PFs below 1x.

With regard to consumers, while EPA considered scenarios with impeded evaporation (e.g., rag soaked with TCE), it appears that EPA did not assume any use of gloves. While it is a reasonable assumption that many consumers would not seek out, purchase, and wear gloves capable of protecting the user from TCE, it is also reasonable to assume that some consumers may well use insufficiently protective gloves that allow TCE to permeate through the material, such as simple latex gloves that are readily found in home improvement stores that sell TCE-containing consumer products. But EPA fails to consider improper glove use and its potential to lead to occlusion and, thus, potentially higher exposure than the no-gloves/soaked rag assumption on which EPA relies (e.g., greater surface area exposed, longer duration of exposure). While EPA argues that it may have overestimated consumer dermal exposure from use of a soaked rag (p. 178), EPA's assumptions for soaked rags likely do not overestimate the exposure duration or surface area exposed when occlusion occurs during a consumer's use of gloves.

With regard to workers, EPA appears to want to have it both ways: To acknowledge the limitations of gloves and their potential to increase skin absorption, but then to simply assume that gloves actually provide 5x, 10x or 20x levels of protection over no gloves – regardless of the potential for occlusion in the workplace – without citing any evidence to support these values. As described in subsection A.ii., in 21 cases EPA found that central-tendency dermal exposures did not present unreasonable acute risks only by assuming workers in those scenarios always wore gloves that consistently provided the assumed PF. In these cases, the unstated, but highly questionable, premise seems to be that if gloves potentially available can be assumed to provide

[188] Draft Risk Evaluation for Methylene Chloride, October 2019, available here: https://www.epa.gov/sites/production/files/2019-10/documents/1_methylene_chloride_risk_evaluation_peer_review_draft_heronet_public.pdf.

a PF that reduces risk to below the benchmark, then EPA can conclude there is no unreasonable risk. This approach will allow clear risks to occur whenever a worker uses anything less than the assumed gloves (or no gloves), or when there is occlusion; these scenarios are quite likely – and certainly reasonably foreseen – to occur in the real world.

During the TCE SACC meeting, numerous additional concerns with the dermal exposure assessments were raised, including that EPA:

- failed to consider exposure via dermal vapor. While this may not constitute a major exposure route for TCE, EPA needs to conduct the analysis to determine whether or not it can be considered negligible.
- relies upon data that does not account for the potential impact of skin damage. Exposure to neat TCE could cause damage to skin, especially with chronic exposures, which in turn can allow for higher dermal penetration of the compound. While human data may not be available, dermal penetration from damaged skin increases ~25x, according to one peer reviewer.
- EPA should present fractional absorption and applied flux assumptions side by side.
- EPA does not provide a clear justification for the consumer model's assumption of only 10% of hand surface area being exposed.

v. EPA may underestimate dermal exposure based on absorption assumptions

EPA assumed a dermal absorption rate of 8% in industrial settings and 13% in commercial settings based on the Kasting and Miller, 2006 model and the following assumptions:

> The steady state fractional absorption (fabs) for TCE is estimated to be 0.08 in industrial facilities with higher indoor wind flows or 0.13 in commercial facilities with lower indoor wind speeds based on a theoretical framework provided by Kasting and Miller (2006) (Kasting and Miller, 2006), meaning approximately 8 or 13 percent of the applied dose is absorbed through the skin following exposure, from industrial and commercial settings, respectively. (p. 117)

However, elsewhere EPA indicates that dermal absorption is rapid:

> Rapid absorption through the skin has been shown by both vapor and liquid TCE contact with the skin. In several human volunteer studies, both TCE liquid and vapors were shown to be well absorbed in humans via the dermal route. Dermal absorption was rapid following exposures of between 20 and 30 minutes, with peak TCE levels in expired air occurring within 15 minutes (liquid) and 30 minutes (vapor) (U.S. EPA, 2011e). Dermal exposure to TCE disrupts the stratum corneum, impacting the barrier function of skin and promoting its own absorption.

> Therefore, absorption may increase at a greater than linear rate due to increasing epidermal disruption over time (ATSDR, 2019). (p. 203)

It is unclear whether EPA considered this latter research when setting the fractional absorption rates of 8% and 13%. If not, the Kasting and Miller, 2006 model may underestimate dermal exposure from TCE, given the cited human and excised skin tissue studies specific to TCE.

C. EPA's workplace exposure monitoring data present several concerns.

i. EPA inappropriately relies solely on occupational exposure data from the Halogenated Solvents Industry Alliance for three conditions of use.

For the "Manufacturing," "Processing as a Reactant," and "Other Industrial Uses" conditions of use (COU), EPA relies solely on data voluntarily submitted by the Halogenated Solvents Industry Alliance (HSIA) to the Agency in 2018 (collected in 2016).[189] For the latter two COUs, EPA uses HSIA's manufacturing data as surrogate monitoring data.

HSIA is the main trade association for manufacturers of TCE, and, as such, it has a strong vested interest in EPA finding the chemical present as low a risk as possible. This vested interest calls into question the reliability and completeness of the data voluntarily submitted by HSIA. We have previously commented extensively on the shortcomings of EPA's reliance on data voluntarily submitted by industry; see, for example, our comments to the Agency on the draft methylene chloride risk evaluation; we incorporate those comments by reference.[190]

In its systematic review process, EPA rated the HSIA data as 1.6, or "High."[191] In doing so, EPA made some questionable decisions. First, EPA assigned the data a score of "1" for Geographic Scope because the data come from U.S. facilities. However, the data represent only one manufacturing facility ("Company B," see p. 698); it is highly unlikely that workplace monitoring data from a single manufacturing site are representative of the entire country.[192]

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[189] Halogenated Solvents Industry Alliance Problem Formulation Comments on TCE (Aug. 2018), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0737-0103.

[190] See EDF Comments on Draft Risk Evaluation of Methylene Chloride, pp. 147-149. December 30, 2019. Available at: https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0437-0042.

[191] This rating applies to the HSIA data dated 2018, the review of which appears on p. 228 of Systematic Review Supplemental File: Data Quality Evaluation of Environmental Release and Occupational Exposure Data, at https://www.epa.gov/sites/production/files/2020-02/documents/6_tce-data_quality_evaluation_of_environmental_releases_and_occupational_exposure_data.pdf.

[192] EPA's 2016 Chemical Data Reporting data (*see* 2016 CHEMICAL DATA REPORTING RESULTS, https://www.epa.gov/chemical-data-reporting/2016-chemical-data-reporting-results (last visited

Second, EPA scored the HSIA data a "1" for "Sample Size," even though the dataset is only comprised of a mere 16 samples.

Third, as EPA acknowledges, HSIA has not provided a standard description of the methods used to collect the data or to analyze the samples. EPA assigned the 2018 data a "3" for Methodology explaining that "no method provided by the HSIA Industry organization." However, EPA's approach to weighting criteria, which is inconsistent with best practices in systematic reviews, results in the "Low" Methodology score having little impact on the overall score.

Fourth, and more broadly, EPA OPPT's systematic review protocol does not take into consideration the potential for bias based on the data source. EPA provides insufficient justification for its exclusive reliance upon this potentially biased data without independent validation and quality assurance reporting. Shockingly, EPA raises potential bias concerns with OSHA data (see subsection C.ii. below), while failing to acknowledge the significant potential bias in the HSIA data.

While there may be a role for the data submitted by HSIA in the risk evaluation, it is inappropriate for EPA to rely solely on these data for several COUs. EPA has not adequately compared HSIA's data to that available through OSHA; see further discussion below.

ii. EPA appears to have ignored OSHA data and dismisses it as "biased"

OSHA has collected a significant amount of data on TCE exposure since the mid-1980s. Our own search using the OSHA Chemical Exposure Health Data tool[193] yielded 3,225 air samples for TCE dated as recently as December 2018. However, it appears that EPA only relied on OSHA data for a single condition of use (Metalworking Fluids, 3 data points) and incorporated OSHA data into an additional two conditions of use (Adhesives, Sealants, Paints, and Coatings as well as Spot Cleaning and Wipe Cleaning, <8 data points). It is unclear why the other OSHA

March 11, 2020) identified by name and location three U.S. facilities manufacturing TCE, but also listed records for two additional facilities for which the submitter had claimed as confidential business information (CBI) whether the facility produced or imported methylene chloride. In addition, EPA has withheld that information for one additional facility. Moreover, there may be additional facilities that did not report, given that only companies that manufacture or imported 25,000 pounds or more at a site during the reporting period were required to report information under the CDR rule. So it is not possible to discern from these data how many more than three facilities manufacture TCE in the U.S.

[193] CHEMICAL EXPOSURE HEALTH DATA, https://www.osha.gov/opengov/healthsamples.html (last visited March 11, 2020).

data – which are not even mentioned in the systematic review supplemental file on environmental releases and occupational exposure[194] – have not been incorporated. OSHA similarly has collected significant data on methylene chloride; for that chemical, Dr. Adam Finkel, a former OSHA official, submitted public comments to the Agency in 2017 including the OSHA dataset from 1984-2016.[195] While EPA did not utilize those data to their full extent,[196] the Agency did incorporate them to a significant extent into its draft methylene chloride risk evaluation. In contrast, here it appears that EPA has simply ignored the bulk of the OSHA data.

In finalizing the risk evaluation, EPA must acquire all of the relevant OSHA data on TCE in order to comply with its requirements to consider reasonably available information and the best available science, in accordance with TSCA section 26.

Furthermore, EPA inappropriately singles out OSHA data as potentially biased in the draft TCE risk evaluation:

> Some data sources may be inherently biased. For example, bias may be present if exposure monitoring was conducted to address concerns regarding adverse human health effects reported following exposures during use. Similarly, OSHA CEHD are obtained from OSHA inspections, which may be the result of worker complaints, and may provide exposure results that may generally exceed the industry average. (p. 126)

EPA's decision to highlight potential bias in OSHA data – while neglecting to mention potential bias in the industry data on which it relies – is unjustified and likely inaccurate. For example, Dr. Finkel debunked this argument in 2017 comments he submitted to the Agency:

> As an industrial hygienist and former OSHA Regional Administrator, I find the notion that OSHA air sampling data are biased upwards to be facile. OSHA receives very few employee complaints about health issues (as opposed to safety

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[194] Systematic Review Supplemental File: Data Quality Evaluation of Environmental Releases and Occupational Exposure Data. Available at: https://www.epa.gov/sites/production/files/2020-02/documents/6_tce-data_quality_evaluation_of_environmental_releases_and_occupational_exposure_data.pdf.

[195] Comment by Dr. Adam M. Finkel on Regulation of Certain Uses under Toxic Substances Control Act: Methylene Chloride and N-Methylpyrrolidone at 3 (May 19, 2017), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0231-0536.

[196] See EDF Comments on Draft Risk Evaluation of Methylene Chloride, pp. 53-55. December 30, 2019. Available at: https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0437-0042.

hazards), and inspections within the sectors it targets for inspection are (by law) random, not aimed at likely violators. Counteracting the tendency (if it exists) for OSHA to gravitate towards less-compliant facilities is the strong downward bias inherent in the fact that OSHA does not tend to inspect very small (10 or fewer employees) establishments in proportion to their abundance in the economy; these facilities tend to have higher chemical exposures, and they are especially abundant in the paint/coating SICs.[197]

During the TCE SACC meeting, several reviewers questioned EPA's sparse use of the OSHA data and EPA's assertion that such data are not representative. One peer reviewer questioned whether the OSHA data are at least as representative as the single-site data EPA used (presumably referring to the HSIA data described above). He and another peer reviewer suggested that EPA consider a composite data analysis – combining the OSHA data and the HSIA data – to increase the confidence compared to relying on data from a single study/site.

iii. There are apparent errors in EPA's characterization of exposure monitoring systematic review rankings.

EPA appears to have mischaracterized its own systematic review data quality rankings in the draft risk evaluation for several exposure monitoring data sources. While we have not checked each instance, we have identified and raise here the following errors in Table 2-26:

- **Batch Open-Top Vapor Degreasing**
  - EPA states on p. 129: "These monitoring data include 123 data points from 16 sources, and the data quality ratings from systematic review for these data were ***medium***." (emphasis added)
  - Based on the description on p. 705, the referenced data sources appear to be from these 10 studies: Daniels et al. 1988, Ruhe et al. 1981, Barsan, 1991, Ruhe, 1982, Rosensteel and Lucas, 1975, Seitz and Driscoll, 1989, Gorman et al. 1984, Gilles et al. 1977, Vandervort and Polakoff, 1973, and Lewis, 1980.
  - In the document "Systematic Review Supplemental File: Data Quality Evaluation of Environmental Releases and Occupational Exposure Data," all 10 studies received an overall quality determination of ***high***, not medium.

[197] Comment by Dr. Adam M. Finkel on Regulation of Certain Uses under Toxic Substances Control Act: Methylene Chloride and N-Methylpyrrolidone at 4 (May 19, 2017), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0231-0536.

- **Spot Cleaning and Wipe Cleaning**
    - EPA states on p. 133: "These monitoring data include 8 data points from 2 sources, and the data quality ratings from systematic review for these data were ***medium***." (emphasis added)
    - Based on the description on p. 732, the referenced data sources appear to be Burton and Monesterskey, 1996 and NIOSH, 1997.
    - In the document "Systematic Review Supplemental File: Data Quality Evaluation of Environmental Releases and Occupational Exposure Data," both Burton and Monesterskey, 1996 and NIOSH, 1997 received an overall quality determination of ***high*** (1.6 and 1.4, respectively; see p. 159 and 172 of the systematic review supplemental file), not medium.

- **Commercial Printing and Copying**:
    - EPA states on p. 134: "These monitoring data include ***20*** data points from 1 source, and the data quality ratings from systematic review for these data were ***medium***." (emphasis added)
    - Based on the description on p. 737, the referenced data source appears to be Finely and Page, 2005.
    - In the document, "Systematic Review Supplemental File: Data Quality Evaluation of Environmental Releases and Occupational Exposure Data," Finely and Page, 2005 received an overall quality determination of ***high*** (1.6) and had 23 samples (see p. 126 of the systematic review supplemental file), not medium.

These apparent errors call into question EPA's ultimate "overall confidence" ratings for the inhalation exposure estimates presented in Table 2-26 (pp. 128-134).

iv. EPA should acquire additional occupational exposure data from government partners and companies.

During the TCE SACC meeting, several reviewers raised concern over EPA's lack of sufficient occupational exposure data – especially with regards to ONUs – and suggested EPA undertake a more concerted effort to acquire data from OSHA, NIOSH, and companies to fill these gaps. For example, one peer reviewer suggested that OSHA or NIOSH inspection data could be helpful in understanding where ONUs are located in facilities, helping to refine the near field versus far field assumptions. The peer reviewer indicated that, if these agencies do not currently have applicable data, EPA could request that they collect such data moving forward.

Similarly, another peer reviewer noted that the same data gap issues have arisen in multiple draft risk evaluations and will continue to arise unless addressed; he suggested that EPA begin looking forward to the next 20 chemicals slated for risk evaluations to proactively fill data gaps by better collaborating with NIOSH and OSHA.

D. EPA's "PEL-capped" analysis is inappropriate.

In the draft risk evaluation, EPA conducted a "PEL-capped" analysis, whereby the Agency calculated exposure estimates only using data points below the OSHA PEL of 100 ppm. EPA found:

> a reduction of the high-end acute exposure estimate from 25.92ppm *[sic]* to 19.23 ppm and the central tendency acute exposure estimate from 4.60 ppm to 4.26 ppm. Chronic high-end and central tendency exposures are reduced from 17.75 ppm and 3.15 ppm to 13.17 ppm and 2.92 ppm, respectively. Lifetime exposures are reduced from 9.10 ppm and 1.25 ppm to 6.75 ppm and 1.15 ppm, respectively. The reduced exposures do not significantly affect the risk estimates, since exposures were only reduced by up to ~30%. (p. 288)

It appears that EPA ultimately did not incorporate this analysis into the final risk estimates because "MOEs remains orders of magnitude below the benchmark MOE" (p. 288). The implication is that if EPA were to have found that the PEL-capped analysis impacted the risk determinations, it may have relied upon this approach.

Presumably EPA pursued this analysis under an assumption of compliance with OSHA's PEL standard. EPA's approach is flawed. First, EPA must utilize the full dataset, regardless of whether data points are above or below the PEL. Second, if anything, the data indicate exactly the opposite of what EPA assumes: the existence of real-world exposure monitoring data above the PEL demonstrate that non-compliance is both known to occur and is reasonably foreseeable. It is inappropriate for EPA to consider excluding data points collected in the real world on the basis of its flawed assumption of universal compliance with regulatory requirements.

E. EPA did not rely on either an aggregate or sentinel exposure assessment.

"In conducting a risk evaluation ***, [EPA] shall—describe whether aggregate or sentinel exposures to a chemical substance under the conditions of use were considered, and the basis for that consideration." 15 U.S.C. § 2605(b)(4)(F)(ii). As explained below, EPA did not prepare aggregate exposure assessments, and it did not establish that it prepared adequate sentinel exposure assessments, in its risk evaluation. EPA has failed to explain how its decision to rely on other exposure assessments can be reconciled with TSCA § 6(b)(4)(F)(ii).

i. EPA did not perform an aggregate exposure assessment.

EPA's regulations define "[a]ggregate exposure [as] the combined exposures to an individual from a single chemical substance across multiple routes and across multiple pathways." 40

C.F.R. § 702.33. In the draft risk evaluation, EPA did not actually prepare an aggregate exposure assessment. EPA states that:

> aggregating dermal and inhalation exposure for risk characterization was not appropriate due to uncertainties in quantifying the relative contribution of dermal vs inhalation exposure, since dermally applied dose could evaporate and then be inhaled. Aggregating exposures from multiple routes could therefore inappropriately overestimate total exposure, as simply adding exposures from different routes without an available PBPK model for those routes would compound uncertainties. (pp. 352-353)

However, EPA's statement does not explain why a worker's inhalation of part of a dermally applied dose would lead to an overestimate of the total dose experienced by the individual, or could not be accounted for in an aggregate exposure assessment. Notably, EPA does not dispute that failing to aggregate inhalation and dermal exposures may lead to an underestimate of exposure as it ignores the reality that exposure from dermal and inhalation routes would be combined. Thus, EPA underestimates exposure. EPA then invokes uncertainty as its excuse for that underestimation. To the extent there are uncertainties in an aggregating analysis, such uncertainties do not support assuming exposure is less than the sum of the exposures; by not combining the exposures it is far more likely that EPA is underestimating the exposure than overestimating it. Uncertainty does not justify ignoring the fact that these exposures are actually experienced in combination.

Notably, EPA did not merely fail to combine exposures between inhalation and dermal exposure pathways; EPA also failed to combine any exposures from multiple conditions of use. Instead, EPA looked at each condition of use separately: "EPA also did not consider aggregate exposure among individuals who may be exposed both in an occupational and consumer context because there is insufficient information reasonably available as to the likelihood of this scenario or the relative distribution of exposures from each pathway." (p. 353). EPA could have used its information authorities to gain more information about these scenarios, and in any event, it is reasonably foreseeable that a person who uses TCE in an occupational context would also use it as a consumer. It is also reasonably foreseeable that a worker or consumer might use or otherwise be exposed to TCE in more than one use over time; EPA did not address the potential for multiple exposures to the same individual worker or consumer. Thus, EPA failed to assess "the combined exposures to an individual from a single chemical substance across multiple routes and across multiple pathways." 40 C.F.R. § 702.33.

To accurately assess overall exposure to TCE, EPA should prepare an exposure assessment that actually looks at "the combined exposures to an individual from a single chemical substance across multiple routes and across multiple pathways." 40 C.F.R. § 702.33. Such an exposure

assessment should combine exposures from both the inhalation and dermal pathways, and EPA should also consider the scenarios where individuals are exposed via multiple conditions of use.

EPA has not justified its decision to forego an aggregate exposure assessment beyond invoking "uncertainty," which is not a justification for underestimating the overall exposure to TCE.

ii. EPA did not establish that its so-called sentinel exposure assessments actually reflect "the plausible upper bound of exposure," as required by EPA's regulation, and EPA did not rely on sentinel assessments in its risk characterizations.

EPA's regulations describe "[s]entinel exposure [as] the exposure from a single chemical substance that represents the plausible upper bound of exposure relative to all other exposures within a broad category of similar or related exposures." 40 C.F.R. § 702.33. In the draft risk evaluation, EPA stated:

> In terms of this risk evaluation, EPA considered sentinel exposures by considering risks to populations who may have upper bound exposures – for example, workers and ONUs who perform activities with higher exposure potential, or consumers who have higher exposure potential (e.g., those involved with do-it-yourself projects) or certain physical factors like body weight or skin surface area exposed. EPA characterized high-end exposures in evaluating exposure using both monitoring data and modeling approaches. Where statistical data are reasonably available, EPA typically uses the 95th percentile value of the reasonably available dataset to characterize high-end exposure for a given condition of use. For consumer and bystander exposures, EPA characterized sentinel exposure through a "high-intensity use" category based on both product and user-specific factors. (p. 353).

However, EPA did not establish that the exposures it analyzed represent the "plausible upper bound of exposure relative to all other exposures" within the relevant categories. This regulatory definition requires that, when EPA prepares a sentinel exposure assessment for workers or consumers, EPA must identify or evaluate the worker or consumer whose exposure represents the upper bound of exposure. 82 Fed. Reg. 33,726, 33,733 (July 20, 2017). EPA has not established that, for each category of exposure, it actually identified and evaluated the worker or consumer whose exposure represents the plausible upper bound of exposure.

Perhaps most importantly, EPA has not stated whether, in identifying sentinel exposures for workers, EPA assumed use of PPE To accurately assess "the plausible upper bound of exposure," EPA should consider exposures without any PPE unless EPA can establish that PPE

is *always and effectively* used for the particular condition of use. As discussed in section 1.B., EPA does not have data sufficient to establish this.

Notably, it is clear that in making its risk determinations, EPA assumed PPE use. *Compare* p. 383 (using values with PPE assumption), *with* p.358 (Chart providing values for both no PPE and with assumption of PPE). See also subsection A. of this section of these comments. Therefore, as a practical matter, EPA did not rely on sentinel exposures—the "plausible upper bound of exposure relative to all other exposures"—in developing its risk characterizations.

* * * * *

Thus, EPA's risk characterizations did not rely on either aggregate or sentinel exposure assessments. EPA has not explained how its approach is consistent with TSCA § 6(b)(4)(F)(ii). To accurately assess the total risk presented by TCE, EPA needs to consider combined exposures, including those faced by the most exposed individuals. In EDF's view, EPA should prepare an actual aggregate exposure assessment.

6. EPA's environmental assessment raises a number of questions and concerns.

A. EPA's approach and methodology for assessing environmental exposure ignore or over-simplify fate characteristics and ignore key data.

In its problem formulation for TCE, EPA states:

> TCE is widely detected in a number of environmental media. While the primary fate of TCE released to surface waters or surface soils is volatilization, TCE is more persistent in air and ground water, where it is commonly detected through national and state-level monitoring efforts. TCE is *frequently* found at Superfund sites as a contaminant in *soil* and ground water. (p. 33, emphases added)

Despite acknowledging TCE's documented persistence in environmental media other than surface water, EPA dismisses these potential impacts outright, simply because they are not the "primary fate" of TCE. By considering only water releases, EPA ignored the tens of thousands of pounds of TCE released through land disposal. Updated TRI data from 2018 show the total land disposal to be greater than 217,000 pounds, including 57,185 pounds to RCRA Subtitle C landfills, 2,935 to "other" landfills, and finally "other land disposal" of TCE, which amounted to nearly 157,000 pounds. [198] The release of TCE via "other land disposal" appears to be from a

[198] US EPA. (2020). TRI Explorer (2018 National Analysis Dataset, released November 12, 2019). Retrieved from https://enviro.epa.gov/triexplorer/. Accessed April 16, 2020; "Other land disposal" defined as: Other land disposal is the disposal of the toxic chemical to land at the

facility that seems to have been discharging TCE to land for a number of years.[199] It is unclear how this facility is permitted for such a discharge.

EPA has given TRI and DMR data a "medium" confidence rating due to potential underreporting because of limitations to the reporting requirements under these programs (p. 77). Hence, the data cited above likely understate the extent of discharges of TCE to the environment and cannot be ignored by EPA in its risk evaluation.

EPA itself has previously highlighted the environmental concern from TSCA-uses of TCE, stating in its 2014 work plan risk assessment:

> The absence of an environmental risk assessment of the TCE TSCA uses should not be construed as saying that the fate and transport properties of TCE suggest that water and soil contamination is likely low or do not pose an environmental concern.*** While the primary concern with this contamination has been human health, there is potential for TCE exposures to ecological receptors in some cases.[200]

TCE is a well-studied chemical with a long history of documented environmental impact. For EPA now to dismiss environmental impacts to soil and sediment based on predicted environmental partitioning does not represent consideration of the best available science or reasonably available information.

i. Partitioning coefficients do not account for an inherently non-equilibrated system.

Physical-chemical properties of a chemical can describe its ultimate environmental fate characteristics. EPA used measured properties and EPISuite to predict a number of important environmental fate characteristics based on those properties, which it then coupled with assumptions about particular conditions of use to justify disregarding pathways of exposure to sediment and terrestrial organisms.

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facility that does not fall into one of the other on-site land release categories found in Sections 5.5.1 through 5.5.3 on the TRI Form R. Other disposal includes such activities as placement in waste piles and spills or leaks. Data from Section 5.5.4 on the TRI Form R.

[199] *Id*. See Other Land Disposal (On-Site) for the US DOE Waste Isolation Pilot Plant in Carlsbad, NM; see also USEPA ECHO Detailed Facility Report (2020) https://echo.epa.gov/detailed-facility-report?fid=110060818735#overEnvirofactsReport.

[200] US EPA (2014). TSCA work plan chemical risk assessment. Trichloroethylene: Degreasing, spot cleaning and arts & crafts uses. (740-R1-4002). Washington, DC: Environmental Protection Agency, Office of Chemical Safety and Pollution Prevention. P. 30.

Importantly, partition coefficients assume chemical equilibrium has been established. However, chemicals of concern can occur in high concentrations in different environmental compartments prior to reaching equilibrium. When considering an open, multi-media system, a better approach for approximation might be the Level III Fugacity model, which predicts that 9.9% of TCE will be distributed to soil, 36.8% to air, 53% to water, and the remainder (0.26%) to sediment, as calculated using EPISuite 4.11. A 10% percent distribution to soil cannot be dismissed as *de minimis*.

Outlined below are examples of how using estimated or even measured partition coefficients to calculate residence in soil, soil-vapor, and water may lead to erroneous conclusions.

a. Reliance on physical-chemical parameters can lead to underestimation of TCE partitioned to soil.

EPA reported that the organic carbon:water partition coefficient (Log K_{OC}) for TCE ranged between 1.8 and 2.17, which generally suggests that soil and sediment sorption of TCE is low. Other sources cited by EPA indicate a moderately higher Log K_{OC} of 2.4, while still others have measured Log K_{OC} at above 3.4,[201] and note that in practice, "[m]easured partition coefficients, however, *may be considerably higher than calculated values*, especially at lower aqueous concentrations.[202] As noted by peer reviewers at the TCE SACC meeting, TCE partitioning in the environment is affected by more than just organic carbon, and there are numerous sorption studies for TCE. One such study, conducted at the Savannah River Site, notes that measured soil distribution coefficient values for TCE were "60 to 100 times higher than those estimated based on [sediment organic fraction] and K_{OC}."[203] Hence the predicted value EPA relies on for TCE associated with soil could well significantly *underestimate* what is actually present.

b. The high volatility of TCE leads to air exposure through releases to soil and water, not just through direct emissions to ambient air.

EPA acknowledges that TCE is expected to volatilize to air, based on physical-chemical properties (p. 275) and the STP model in EPISuite, which predicted 80% removal via volatilization (p. 70). When TCE moves to the atmosphere, it's half-life through degradation by

[201] Grathwohl P. 1990. Influence of organic matter from soils and sediments from various origins on the sorption of some chlorinated aliphatic hydrocarbons: Implications on KOC correlations. Environmental Science and Technology, 24(11):1687–1693.
[202] US EPA. (1992). TCE Removal from Contaminated Soil and Ground Water. (EPA/540/S-92/002). Washington, DC: Office of Solid Waste and Emergency Response.
[203] Riley RG, Szecsody JE, Mitroshkov AV, Brown CF. PNNL (2006). Desorption Behavior of Trichloroethene and Tetrachloroethene in U.S. Department of Energy Savannah River Site Unconfined Aquifer Sediments. (PNNL-15884). P. 16.

reactants in the atmosphere is nearly two weeks,[204] which has led EPA to conclude that "long range transport is possible" (See Problem Formulation, p. 30). The logical conclusion is that land-applied TCE and TCE-contaminated wastewater sent to treatment facilities are likely an important source of air-exposures of TCE, which EPA has not addressed.

Furthermore, it is important to note that this type of degradation will only occur in the atmosphere. However, migration of TCE in soil does not always result in volatilization to the atmosphere. EPA notes that, "[o]nce in soil, TCE can become associated with soil pore water, enter the gas phase because of its Henry's Law constant, or exist as a nonaqueous phase liquid (NAPL). It is possible that upward or downward movement of TCE can occur in each of these three phases***."[205]

TCE present in soil vapor (a well-documented phenomenon primarily recognized through soil vapor intrusion into indoor air[206]) will not degrade via atmospheric reactions. EPA has disregarded impacts from such exposure to terrestrial organisms whose habitat exists in the vadose zone. Fossorial and semi-fossorial organisms (those that burrow) or have an "increased exposure potential from inhalation at site contaminated with volatile chemicals in the subsurface." [207] EPA has ignored these sources of environmental exposure to such organisms.

c. The physical-chemical properties of TCE will lead to longer half-lives in water than predicted by the EPISuite volatilization module, which likely biases predictions of concentrations in surface water to be artificially low.

In its draft risk evaluation, EPA reports the modeled volatilization half-life of TCE in a model river will be 1.2 hours and the half-life in a model lake will be 110 hours (p. 259). Importantly, TCE is a dense non-aqueous phase liquid (DNAPL). In its 2014 TCE work plan risk assessment, EPA notes that (emphasis added):

[204] TCE has an estimated atmospheric half-life of about 13 days (using Version 4.10 of EpiSuite, EPA, 2012b).

[205] US EPA (2014). TSCA work plan chemical risk assessment. Trichloroethylene: Degreasing, spot cleaning and arts & crafts uses. (740-R1-4002). Washington, DC: Environmental Protection Agency, Office of Chemical Safety and Pollution Prevention. Pp. 158-159.

[206] See: https://www.epa.gov/vaporintrusion/vapor-intrusion-superfund-sites#tri

[207] Gallegos P, Lutz J, Markwiese J, Ryti R, Mirenda R. 2007. Wildlife ecological screening levels for inhalation of volatile organic chemicals. Environmental Toxicology and Chemistry. 26(6):1299–1303. doi:10.1897/06-233R.1.

> Volatilization from water surfaces will be an important fate process based upon TCE's measured Henry's Law constant. However, its density may cause it to sink in the water column, potentially *increasing the aquatic residence time of TCE.*[208]

The TCE work plan risk assessment further notes that the "[v]olatilization half-lives in an experimental *field* mesocosm consisting of seawater, planktonic, and microbial communities ranged from 10.7 to 28 days," contrasting those values to values measured "half-lives of evaporation from *laboratory* water surfaces (distilled water) [that] have been reported to be on the order of several minutes to hours, depending upon the turbulence." (p. 157; emphases added) This suggests that the volatilization half-life used by EPA in this evaluation is too low. Even considering less-turbulent water bodies (lakes), the half-life reported by EPA is one-half to one-fifth the value of that found in natural conditions.

The density of TCE, coupled with its relatively low solubility, indicates that sampling surface water using grab samples at the tops of water columns will bias the analysis, resulting in artificially low environmental concentrations. Hence, such an approach to sampling may not represent the actual concentrations of TCE found in surface water.

ii. EPA has ignored STORET data available for evaluating sediment impacts.

Importantly, sampling only surface water overlooks a potentially more likely environmental compartment for a chemical that is denser than water. As a DNAPL, TCE is likely to be present in the sediment, at the bottom of a water column.

In its problem formulation EPA noted that the STORET database would be examined for recent data on TCE levels in sediment (p. 34). However, these data are absent from the draft risk evaluation. Instead EPA states that it "included a *qualitative* assessment describing trichloroethylene exposure from sediments for aquatic organisms" because TCE "is not expected to accumulate in sediments" (p. 31, emphasis added).

We reviewed data reported in the National Water Quality Monitoring Council database of Water Quality Data[209] for TCE in sediment (above detection) in the last 10 years. We applied the same qualifiers for sediment that EPA used for surface water (as described in section 2.2.4.2 of the draft risk evaluation); we did not exclude sites with "known contamination," nor did we include

[208] US EPA. (2014). TSCA work plan chemical risk assessment. Trichloroethylene: Degreasing, spot cleaning and arts & crafts uses. (740-R1-4002). Washington, DC: Environmental Protection Agency, Office of Chemical Safety and Pollution Prevention. P. 157 (emphasis added).
[209] National Water Quality Monitoring Council, Water Quality Data. https://www.waterqualitydata.us/portal/ (last accessed March 13, 2020).

samples below detection. This analysis resulted 21 quantifiable analyses of TCE in sediment; the maximum detected concentration was 26,000 ug/kg.

EPA overlooked these data, which are environmentally relevant and describe measured impacts to environmental systems simply because of its assertion that TCE "is not expected to accumulate in sediments" (p. 31).

iii. EPA has overlooked important considerations for the anaerobic biodegradation of TCE.

EPA has concluded, based on its review of test data that the rate of anaerobic biodegradation is "'fast'" (p. 70). We do not disagree that under ideal conditions with correct microbial consortia that carry the metabolic capability to reductively dehalogenate TCE to ethene,[210] this conclusion is valid; however, there are important caveats to this conclusion that must be considered. EPA goes on to acknowledge that there is inherent variability in the reported biodegradation rates that arises from "methodology, interlaboratory variability and variability due to factors such as the specific microbial populations used, water, soil and sediment chemistry, oxygen concentration/redox potential, of the collected samples used in the study, temperature and test substance concentration." Yet EPA still concludes that the "weight of evidence shows *** the anaerobic biodegradation in anaerobic condition is fast" (p.71).

Biologically mediated processes that transform compounds cannot be assumed to lead to complete removal of a compound. Notably, under anaerobic conditions, TCE biologically degrades via sequential removal of chloride ions first to cis-dichloroethene, and next to vinyl chloride, which is itself a potent carcinogen. Vinyl chloride degradation to ethane (under anaerobic conditions) is often the rate-limiting step in this transformation, as it is mediated by a select group of microorganisms.[211] As the rate-limiting step, there are many documented cases of stalled TCE-degradation, which has led to elevated vinyl chloride concentrations in the environment[212] – arguably a condition as bad as or worse than TCE alone.

Where TCE is discharged into the environment, simply reporting standard biodegradation rates can obscure important impacts due to transformation processes.

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[210] Duhamel M, Mo K, Edwards EA. 2004. Characterization of a Highly Enriched Dehalococcoides-Containing Culture That Grows on Vinyl Chloride and Trichloroethene. Applied and Environmental Microbiology. 5538-5545. DOI: 10.1128/AEM.70.9.5538-5545.2004.

[211] *Id.*

[212] Stroo HF, Ward CH. 2010. In Situ Remediation of Chlorinated Solvent Plumes. Springer Science & Business Media.

B. Over-reliance on qualitative assessments of TCE partitioning in the environment means EPA has failed to adequately address risks to terrestrial and sediment-dwelling organisms.

Despite having environmental monitoring data that indicate that TCE is present in air, soil and sediment and will likely expose terrestrial and sediment-dwelling organisms, EPA instead relied exclusively on qualitative and screening-level assessments to minimize such impacts.

i. TCE exposures to terrestrial organisms can occur through multiple pathways of exposure.

EPA has ignored important pathways of TCE exposure to terrestrial organisms, justifying its lack of a quantitative assessment of exposures to terrestrial organisms because "TCE is not expected to partition to soil but is expected to volatilize to air, based on its physical-chemical properties" (p. 275). This is despite finding potential hazard based on reviewed data (p. 275 and in the Problem Formulation, pp. 40-41).

Importantly, this statement ignores entirely TCE exposures to terrestrial organisms through air, which is a primary pathway of exposure to TCE. EPA dismisses exposure to terrestrial organisms from the ambient air pathway based on the unsupported argument that such exposures are adequately managed by the Clean Air Act (p. 276); see section 2.B. for our comments on the many concerns with this argument.

Additionally, EPA is ignoring exposures to terrestrial organisms that may occur from contaminated water and soil. EPA must comprehensively consider all routes of exposure to terrestrial organisms in its risk evaluation of TCE given its widespread detection throughout the environment including at contaminated sites.

In addition to the fact that nearly two million pounds[213] of TCE are released annually into the air, due to its volatility, disposal to water and land may also create a route of exposure to organisms living at the water-atmosphere or water-soil interface (e.g., amphibians, birds and shorebirds, and burrowing organisms). These organisms may be significantly impacted by TCE exposure.

Additionally, EPA dismissed potential exposure based on land-applied biosolids (p. 90), stating that:

> [any] TCE present in the water portion of biosolids following wastewater treatment and land application would be expected to rapidly volatilize into air. To further support this analysis, TCE was not detected in EPA's Targeted National Sewage Sludge Survey (TNSSS), nor was it reported in biosolids during EPA's

[213] 2018 TRI Data, https://www.epa.gov/toxics-release-inventory-tri-program/tri-data-and-tools.

> Biennial Reviews for Biosolids, a robust biennial literature review conducted by EPA's Office of Water (U.S. EPA, 2019d). (p.70)

However, our review of the cited document[214] as well as the TNSSS Sampling and Analysis Technical Report[215] did not indicate that TCE was included in the sample analysis, which calls into question use of the biennial review as support for EPA's conclusions. Interestingly, TCE *has* been detected in biosolids at concentrations as high as 8,770 ug/kg.[216]

EPA's dismissal of exposures to TCE in biosolids is alarming in light of the findings of a recent Office of Inspector General (OIG) report that indicates EPA "lacks the data or risk assessment tools" to make determinations on the risk levels for pollutants found in biosolids.[217] Moreover, according to the OIG, "[t]he regulations for biosolids do not require the EPA to obtain the data necessary to complete risk assessments."[218]

EPA needs to provide a rational and clear analysis based on the best available science and reasonably available information to support its conclusions, and at this point, it has failed to do so.

ii. Impacts on sediment dwelling organisms need to be evaluated.

For sediment-dwelling organisms, EPA stated in its problem formulation:

> No data on the toxicity to sediment organisms (e.g. *Lumbriculus variegatus, Hyalella azteca, Chironomus riparius*) were found; however, *** TCE is not expected to partition to sediment, based on physical chemical properties. (p.41)

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[214] U.S. EPA. (2019). Biennial Review of 40 CFR Part 503 As Required Under the Clean Water Act 3574 Section 405(d)(2)(C). Reporting Period 2016–2017. EPA-822R18003

[215] US EPA. (2009). Targeted National Sewage Sludge Survey Sampling and Analysis Technical Report. EPA-822-R-08-016.

[216] US EPA. (1992). Statistical Support Documentation for the 40 CFR, Part 503: Final Standards for the Use or Disposal of Sewage Sludge, Vol. 1. https://www.epa.gov/sites/production/files/2015-04/documents/statistics_1992_support_document_-_biosolids_vol_i.pdf. P. 91, Table 7-20.

[217] U.S. OIG, *EPA Unable to Assess the Impact of Hundreds of Unregulated Pollutants in Land-Applied Biosolids on Human Health & the Environment* (2018), https://www.epa.gov/sites/production/files/2018-11/documents/_epaoig_20181115-19-p-0002.pdf.

[218] *Id.*

Absence of hazard data does not equate to absence of hazard. A cursory review of the literature identified a study that found sensitivity of nematodes (sediment-dwelling organisms) to TCE at concentrations of 1 ug/ml (or 1000 ppb).[219] At 30 mg/L, the researchers reported a significant reduction in the nematode maturity index, described as an index of diversity based on trophic groupings in nematodes in riparian soil microcosms. As noted previously (subsection A.ii.), TCE has been measured in the sediment at concentrations of up to 26,000 ug/kg (or 26,000 ppb). Therefore, dismissing sediment exposure as a potential impact is clearly unwarranted.

The scope of the draft risk evaluation limited the COUs included to those with applicable occupational exposure scenarios (OES). EPA then appears to have illogically limited its evaluation of risks to *environmental* receptors to just these COUs (p. 46). As a result, it is likely that some environmental receptors potentially impacted by TCE discharges have been ignored because those discharges are not associated with a specific COU chosen based on worker exposure potential. Ignoring TCE-impacted sediment data illustrates this point. Furthermore, EPA disregarded data associated with contaminated sites from its water monitoring data ("Data Filtering and Cleansing," p. 89) and excluded monitoring data potentially impacted by Superfund sites in its watershed analysis ("Geospatial Analysis Approach," p. 89).

While EPA acknowledges that TCE has been measured in sediments, it immediately dismisses these data, asserting that this detection is likely for TCE present in pore water (p 275); on this basis, EPA does not address risk to sediment-dwelling organisms.

While evidence indicates EPA's assertion is not supported, even if TCE were only associated with pore water, sediment-dwelling organisms (also called "interstitial fauna")[220] often live in or are in contact with the pore water of sediment systems. Given that some of these organisms exist in the interstitial spaces in sediment and sand (they are even termed “interstitial fauna”) pore water can be a key route of exposure to these organisms. Therefore, EPA cannot ignore this exposure pathway for sediment-dwelling organisms.

C. EPA cannot ignore environmental releases of a chemical because it cannot attribute each release to a particular condition of use.

EPA has indicated that “only a few USGS-NWIS and STORET monitoring stations aligned with the watersheds of the TCE-releasing facilities *identified under the scope* of this assessment, and

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[219] Fuller ME, Scow KM, Lau S, Ferris H. 1997. Trichloroethylene (TCE) and toluene effects on the structure and function of the soil community. Soil Biology and Biochemistry. 29(1):75–89. doi:10.1016/S0038-0717(96)00247-7.

[220] Tom M. Fenchel. 1978. The Ecology of Micro-and Meiobenthos. Annual Review of Ecology & Systematics. 9: 99–121. https://www.annualreviews.org/doi/abs/10.1146/annurev.es.09.110178.000531.

the co-located monitoring stations had samples with concentrations below the detection limit; therefore, *no direct correlation* can be made between them" (p. 98, emphases added). This language suggests EPA may believe it must be able to attribute every environmental release of a chemical to a particular condition of use or facility in order to consider its risks in a risk evaluation. This is not the case.

Nothing in TSCA allows EPA to ignore data simply because they have not been tied to a particular condition of use, let alone a particular facility. EPA must conduct risk evaluations under TSCA that consider all "reasonably available" information relating to a chemical substance, including information that may not be tied to specific conditions of use. 15 U.S.C. § 2625(k). EPA's rules further define "reasonably available information" as "information that EPA possesses or can reasonably generate, obtain and synthesize for use ***." 40 C.F.R. §§ 702.3, 702.33.

Data that cannot be attributed to specific conditions of use are still relevant to determining whether *the chemical substance* presents an unreasonable risk, and as such must be considered by EPA. EPA cannot ignore data simply because it has not determined or even cannot determine how much of the exposure is attributable to a particular condition of use. Such a consideration may be more relevant at any subsequent risk management stage, when EPA may need to understand the extent to which specific measures will reduce exposure and risk. But that future need provides no basis for EPA to ignore risk-relevant information at the risk evaluation stage.

D. EPA's analysis of aquatic risks may underestimate the risk.

i. EPA's concentration of concern (COC) for algae does not pass muster.

As explained more in section 7.B., EPA's own analyses showed that TCE presents an unreasonable risk to aquatic organisms (pp. 354, 378-379). Specifically, EPA found that releases from certain disposal and recycling facilities would result in surface water concentrations well above the concentrations of concern (COC) for TCE (pp. 260-263). But if anything, EPA's analysis may have underestimated the risk from these releases especially for algae.

EPA calculated a COC for algae of 52,000 ppb (52 mg/L) using species sensitivity distribution (SSD), justifying it as being representative for algae species "as a whole" (p. 194). EPA determined that "as a whole" in this case constitutes nine species of algae. Yet algae are an incredibly diverse (and poorly defined) group of organisms that represent 15 phyla and 54 classes; estimates of total species of algae are between 72,000 and 1 million.[221]

[221] Guiry MD. 2012. How many species of algae are there? Journal of Phycology. 48(5):1057–1063. doi:10.1111/j.1529-8817.2012.01222.x.

To conclude that a COC of 52 mg/L is protective of algae "as a whole," based on only nine species, with a concentration that is over 17,000 times higher[222] than the COC EPA derived for the most sensitive species of algae identified for the draft risk evaluation is indefensible. Instead, EPA should use the most sensitive species as its indicator organism to develop appropriately protective COCs.

Using the far more appropriate COC of 3 ppb, EPA identified risks from exposure to TCE to the most sensitive algae specie at 521 facilities (p. 354); nevertheless, EPA dismissed these RQs as actually showing no risk for "algae species as a whole" based on its questionably calculated COC (pp. 378-379).

ii. EPA based its exposure estimates on unreliable surface water concentrations uncertain calculations.

As discussed previously (see subsection C.), not only did EPA ignore environmental impacts to surface water from TCE discharges, the existing surface water data may not be representative of TCE concentrations. EPA acknowledges the limitations of data in the USGS-NWIS and STORET databases, stating "the monitoring studies used to collect the data were not specifically designed to evaluate TCE distribution across the US," and "it is unclear whether the data are representative of other locations in the US" (p. 98). EPA goes on to note that these data " cannot be interpreted as reflecting concentrations downstream of direct release sites, which could be higher than reported measured levels" (p. 98).

When calculating surface water release estimates, EPA correctly states that "release estimates serve as the key inputs into the exposure mode and are therefore a key component of the overall aquatic exposure scenario confidence" (p. 98). Based on available data, and other considerations relating to the estimation of rates of discharges from various facilities – including outdated stream flow data in EFAST, some of which are decades old[223] – EPA was over-generous in assigning a "moderate" confidence in wastewater discharge estimates (p. 98).

Furthermore, EPA applied a wastewater treatment removal rate of 81% to all indirect releases, as well as to direct releases from WWTPs (p. 85 and footnote b of Table 4-1). EPA did not establish that this assumed removal actually occurs, so EPA may be underestimating the total risk presented by releases from these facilities.

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[222] See p. 199. The algal COC derived by EPA for TCE is 3 ppb; the algal HC_{05} (Hazardous Concentration threshold for 5% of species) derived by EPA is 52,000 ppb, a 17,000-fold difference.

[223] See p. 98. Despite having access to newer hydrological data, EPA used the stream flow data in EFAST, which are 15 to 30 years old.

7. EPA's unreasonable risk definition and risk determinations are severely flawed.

A. EPA grossly underestimates occupational risk, leading to 'no unreasonable risk' findings or understatements of the extent and magnitude of the unreasonable risks it does find.

EPA underestimates occupational risks in three major ways in its draft risk evaluation:

1. EPA assumes that workers will wear fully effective personal protective equipment (respirators and gloves) in most scenarios and relies on that assumption to avoid finding that its risk estimates represent unreasonable risk or to understate the extent and magnitude of the risk. See section 5.A. and subsection A.i. below for the details.
2. EPA finds a cancer risk to workers unreasonable only if it exceeds a level of 1 in 10,000 – which is as much as 100 times higher a risk than warrants regulation under TSCA to protect workers and other vulnerable subpopulations. See subsection A.ii. below for the details.
3. For ONUs EPA has failed to identify unreasonable risks for the most highly exposed, and hence most vulnerable, basing its ONU risk determinations exclusively on central tendency exposure estimates. See subsection A.iii. below for the details.

The effect of each of these decisions is to underestimate occupational risk – ultimately either leading EPA to determine "no unreasonable risk" or to grossly understate the extent and magnitude of the unreasonable risks it does find. Below we discuss each of these issues in further detail.

i. By assuming use of PPE, EPA conflates risk evaluation and risk management and significantly understates risk.

TSCA intentionally divides risk evaluation and risk management into two distinct processes, whereby regulatory measures are to be considered *after* EPA finds an unreasonable risk. However, by choosing to make risk determinations based on an assumption of PPE, EPA conflates risk evaluation and risk management and leads EPA either not to find an exposure presents unreasonable risk, or more commonly, to underestimate the extent and magnitude of TCE's risk under many scenarios (see section 5.A.). EPA's failure to make an unreasonable risk determination based on its PPE assumption could potentially deny itself the opportunity to impose mandatory requirements sufficient to control workplace exposures.

For example, Table 4-54 (pp. 358-369) demonstrates that for non-cancer risk from acute dermal exposure, EPA has actually found excessive risk in the absence of glove use in ***every*** occupational scenario it examined. EPA had to assume use of gloves in order not to find excessive risk under most central tendency exposure scenarios. Thus, when it comes to the risk determinations, EPA makes almost no unreasonable risk determinations based on central

tendency dermal exposures, invoking PPE (section 5.3 of the draft risk evaluation). EPA's failure to identify that central tendency dermal exposures can lead to unreasonable risk in the absence of PPE could constrain its authority to require that the gloves it assumed are used will actually be used.

See EDF's further critique of EPA's assumption of PPE use in the workplace in sections 1.B. and 5.A.

ii. EPA's use of a 1 in 10,000 cancer risk level as reasonable for workers is deeply flawed.

a. EPA's approach must be rejected on scientific as well as legal grounds.

EPA has proposed to establish 1 x 10^{-4} as the cancer risk benchmark for workers (p. 376). EPA cites NIOSH guidance and the *Benzene* decision for support (p. 376, footnote 22), but that guidance and that case pertain to how the standard for health protection is applied under OSHA, not under TSCA. EPA's decision is wholly at odds with its own acknowledgment two pages earlier that other laws have standards that differ from TSCA's (p. 374, footnote 20).

EPA is required to protect workers, both generally and as a "potentially exposed or susceptible subpopulation," under TSCA, not under OSHA. The 2016 amendments to TSCA strengthened EPA's already-existing mandate to protect workers. TSCA's new definition of "potentially exposed or susceptible subpopulation" has no asterisk next to workers, and there is no basis in TSCA for EPA to provide less protection to workers than any other such subpopulation, let alone than the general population. Yet that is exactly what EPA has done here.

The 2016 amendments to TSCA also explicitly preclude EPA from considering feasibility or other non-risk factors when determining whether a chemical presents an "unreasonable risk," including to workers; see TSCA section 6(b)(4)(A). Yet EPA invokes standards under other statutes that lack this prohibition in an effort to claim precedent for its 1 x 10^{-4} benchmark (p. 376, footnote 21).

Indeed, EPA's reliance on the *Benzene* decision cannot be reconciled with the statutory differences between OSHA's standard and TSCA's unreasonable risk standard. In the *Benzene* case, the Court interpreted a provision of the OSH Act that defined standards as "reasonably necessary or appropriate to provide safe or healthful employment and places of employment," as requiring OSHA "to make a threshold finding that a place of employment is unsafe—in the sense that *significant* risks are present and can be eliminated or lessened by a change in practices." *Indus. Union Dep't, AFL-CIO v. API*, 448 U.S. 607, 642 (D.C. Cir. 1980) (emphasis added). The Court's interpretation turned on the statutory language of the OSH Act, the Act's structure, and its legislative history. But EPA can point to *no* statutory language in TSCA invoking this

standard, EPA has pointed to no similarities between the two statute's structures, nor has EPA pointed to any legislative history suggesting that TSCA adopted the OSH Act's standard. Moreover, if Congress had intended to adopt the *Benzene* standard under TSCA, it would have required that EPA regulate "significant risks," not "unreasonable risks." Indeed, the significant differences between the language and structure of the two statutes strongly indicates that Congress meant to adopt a different standard in TSCA, not the standard articulated by the Court in the *Benzene* case.

Moreover, in implementing TSCA (even before the amendments) and its other environmental statutes, EPA has generally sought to reduce population risks from chemicals in commerce that are carcinogens to below about one case per one million people. See, for example, this EPA statement from 1989: "EPA believes *** that it should reduce risks to less than 1 x 10^{-6} for as many exposed people as reasonably possible." National Emission Standards for Hazardous Air Pollutants; Radionuclides, 54 Fed. Reg. 51,654, 51,686 (Dec. 15, 1989). Nor does EPA only apply this standard under the Clean Air Act. When setting Clean Water Act criteria, "EPA intends to use the 10^{-6} risk level, which the Agency believes reflects an appropriate risk for the general population. EPA's program office guidance and regulatory actions have evolved in recent years to target a 10^{-6} risk level as an appropriate risk for the general population. EPA has recently reviewed the policies and regulatory language of other Agency mandates (e.g., the Clean Air Act Amendments of 1990, the Food Quality Protection Act) and believes the target of a 10^{-6} risk level is consistent with Agency-wide practice."[224] When Congress amended TSCA to include the unreasonable risk standard, it did so knowing that Agency practice was to regulate cancer risks at the 10^{-6} risk level. It should be presumed that Congress meant to adopt this risk standard when codifying the unreasonable risk standard.

In grasping for support for its approach in this risk evaluation by citing other mentions by EPA of the 1 x 10^{-4} risk level (p. 376, footnote 21), EPA blurs a critical distinction made when EPA has invoked the less stringent level of protection from cancer risks: the level set to reflect the maximum risk faced by any individual vs. the level set to protect a broader population. EPA invokes the "two-step approach" used under the Clean Air Act, where EPA includes a "limit on *maximum individual* lifetime [cancer] risk (MIR) of approximately 1 in 10 thousand" (p. 426 n. 22, citing 54 Fed. Reg. 38,045 (Sept. 14, 1989)) (emphasis added). But that is entirely different than the level set to protect the vast majority of the population in question.

More specifically, the two-step, risk-based decision framework for the National Emission Standard for Hazardous Air Pollutants (NESHAP) program is described as follows by EPA:

[224] U.S. EPA, *Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health* p. 2-6 (2000), https://www.epa.gov/sites/production/files/2018-10/documents/methodology-wqc-protection-hh-2000.pdf.

> First, the rule sets an upper limit of acceptable risk at about a 1-in-10,000 (or 100-in-1 million) lifetime cancer risk for the *most exposed person*. As the rule explains, "The EPA will generally presume that if the risk to that individual [the Maximum Individual Risk] is no higher than approximately 1 in 10 thousand, that risk level is considered acceptable and EPA then considers the other health and risk factors to complete an overall judgment on acceptability."
>
> Second, the benzene rule set a target of protecting the *most people possible* to an individual lifetime risk level no higher than about *1-in-1 million*.[225]

But in this draft risk evaluation, EPA has set a risk level for the *entire* worker population that is the same as the level EPA elsewhere set for the most exposed individual in a population. EPA then erroneously invokes this level repeatedly to find a number of conditions of use of methylene chloride to pose no risk to *any* workers, thereby subjecting many tens of thousands of workers to cancer risks that are as much as *two orders of magnitude higher* than warranted. This approach must be rejected on scientific as well as legal grounds.

b. EPA's approach leads to a major understatement of the extent of unreasonable risk workers and ONUs face from TCE exposure.

EPA's occupational risk estimates were significantly impacted by EPA's selection of 10^{-4} as the cancer risk benchmark. The impact is less than in draft risk evaluations for other chemicals only because EPA's identified cancer risk even exceeds its lax benchmark for most scenarios it examined. Nonetheless, EPA failed to identify the cancer risk as unreasonable in a number of cases, and it of course effectively understates the magnitude of the cancer risk even where it identified it as unreasonable.

To determine how large the impact is, EDF examined EPA's cancer risk estimates to workers for each of the combinations of industrial/commercial COUs, exposure routes (inhalation or dermal) and exposure levels (high-end or central tendency) presented in Table 4-54. Our analysis is provided in the Excel file submitted as Appendix 6 along with these comments.

While EPA applied a 10^{-4} cancer risk benchmark to these estimates, EDF looked at whether they exceed a cancer risk benchmark of 10^{-5} or 10^{-6} and should have at least potentially been identified as presenting an unreasonable risk to workers.

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[225] WHAT DOES EPA BELIEVE CONSTITUTES AN ACCEPTABLE LEVEL OF RISK?, https://www.epa.gov/national-air-toxics-assessment/nata-frequent-questions#risk2 (emphasis added) (last visited Nov. 26, 2019).

In the 91 cases where EPA assumed use of respirators, EPA identified 79 as exceeding its 10^{-4} cancer risk benchmark. Had EPA used a benchmark of 10^{-5} or 10^{-6}, EPA would have identified as unreasonable an additional 11 and 12 of the 91 cases, respectively. Equally important, even for those cases EPA identified as presenting unreasonable risk, use of the more appropriate benchmark would have established the need to reduce exposure to TCE to at least a 10-fold lower level by subsequent regulation of TCE to eliminate the unreasonable risk EPA has identified.

iii. EPA's assumption that ONUs are never exposed at levels above the central tendency estimates it derives for workers is flawed.

For COUs where EPA states it has no basis to distinguish between worker and ONU exposures, it reports for ONUs only the risk associated with its central tendency estimate for workers and provides no estimate of high-end risk for ONUs. These cases are those where the "population" column in Table 4-54 identifies the population as "ONU (upper limit)." EPA then determines ONUs face an unreasonable risk only if its central tendency risk estimate for workers (carried over to ONUs) exceeds its benchmark.

EPA states:

> For some conditions of use, EPA did not separately calculate risk estimates for ONUs and workers. For these conditions of use, there is uncertainty in the ONU risk estimates since the data or modeling did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency risk estimate when determining ONU risk for those conditions of use for which ONU exposures were not separately estimated. (p. 35)

Among other concerns, EPA has provided no empirical basis at all for its therefore-arbitrary assumption that ONUs will never be exposed at levels higher than the central tendency exposure workers experience. EPA's approach is at odds with its obligation under TSCA to conduct risk evaluations that ensure protection of "potentially exposed or susceptible subpopulations," which TSCA explicitly defines as including workers. EPA represents its high-end estimates as "generally intended to cover individuals or sub-populations with greater exposure," while its central tendency estimates apply to the "average or typical exposure" that people experience (p. 375). TSCA would not permit EPA to protect against only the "average or typical exposure;" in fact, when it comes to workers, ONUs, and other "potentially exposed or susceptible subpopulations," EPA is required to protect all of them.

iv. EPA has dismissed unreasonable risk based on biased assessment of exposure estimates.

Epidemiological and exposure assessment studies have the potential to either under- or over-estimate exposure, depending on the methods and approaches utilized. For this reason, it is important to consider data from the entire body of evidence rather than any particular study alone. Yet, in attempting to downplay its unreasonable risk determinations for TCE, the Agency has chosen only to emphasize the potential for data sources to *overestimate* exposure. EPA states:

> Additionally, some data sources may be inherently biased. For example, bias may be present if exposure monitoring was conducted to address concerns regarding adverse human health effects reported following exposures during use. These sources may cause exposures to be overestimated. (p. 348)

This one-sided statement, emphasizing only those factors tending to overestimate exposure while ignoring the potential for similar factors to underestimate exposures, highlights the Agency's bias evident in this draft risk evaluation.

B. EPA cannot reasonably dismiss its findings of environmental risk merely by invoking uncertainty.

For environmental risk, EPA's own analyses showed that TCE presents an unreasonable risk to aquatic organisms (pp.354, 378-379), but EPA dismisses this unreasonable risk by invoking "uncertainty" (p. 379), which is reflective of EPA limiting its analysis to only a "qualitative consideration of the physical-chemical and fate characteristics" as well as conditions of use (pp. 31, 378-379). Beyond this weak assertion and the accompanying distortion of its own findings with respect to environmental risk, EPA provides no basis for its dismissal of identified risks.

EPA used a Risk Quotient (RQ) to compare environmental concentration to the effect level to characterize the risk to aquatic organisms. (p. 31). Under this approach, "[i]f the RQ is greater than 1, the exposure is greater than the effect concentration and there is potential for risk presumed." (p. 376). Risks to the most sensitive species of algae were identified near 521 facilities (with 20 days or more of exceedances for 461 of these facilities, and more than 100 days exceedances for 10 facilities).

Thus, based on EPA's own analyses, EPA found risks to aquatic organisms from 521 facilities (p. 354), with the RQ exceeding 1 (in one case exceeding the COC by 1,000-fold) (p. 261), but EPA dismissed this risk merely by invoking uncertainty and relying on a dubiously calculated COC for algae (see section 6.D.i.). This approach is arbitrary and capricious because EPA refuses to accept the outcomes of its own analyses, and EPA's conclusions run contrary to the

evidence before the Agency. Based on the analysis presented in the draft risk evaluation, EPA should find an unreasonable risk to the environment presented by certain conditions of use.

In summarizing its risk conclusions, EPA states that: "Risk to the most sensitive species of algae were identified near 521 facilities (with 20 days or more of exceedances for 461 of these facilities, and more than 100 days exceedances for 10 facilities)." (p. 354). But EPA then does not make risk findings. Instead—in addition to the wholesale dismissal of potential risks to the most sensitive algae species—in the risk characterization section, EPA states that:

> For aquatic organisms like aquatic invertebrates and fish, one facility had an acute RQ greater than 1 (RQ = 3.11) *** Another facility had an acute RQ of 0.94 indicating some uncertainty about whether it would also pose risks to aquatic organisms from acute exposures. *** Both facilities had chronic RQs greater than 1, exceeding the chronic COC of 788 ppb for 20 days. *** Monitored data from literature showed some exceedances of the algae COC of 3 ppb in ambient water; however, the data show no exceedances of the algae COC of 52,000 ppb. Therefore, EPA did not identify risks for acute or chronic exposure durations in ambient water for areas where monitored data were reasonably available. Given the uncertainties in the modeling data and exceedance of the acute RQ for only one data point and of the chronic RQ for only two out of 70 facilities modeled, EPA does not consider these risks unreasonable (see Section 4.5.2). (pp. 378-379)

EPA essentially acknowledges that it did find unreasonable risk for some conditions of use, and EPA then dismisses that risk on the basis of "uncertainties in the data" and on selective monitoring data that exclude contaminated environments and ranged across five orders of magnitude (above 3 ppb but below 52,000 ppb).[226] Notably, EPA provides no cogent explanation of what uncertainties exist in the data. Moreover, to the extent there are uncertainties in EPA's analysis, such uncertainties counsel in favor of a finding of unreasonable risk – EPA could as easily be underestimating the risk presented by these conditions of use as overestimating them. Uncertainty increases the chances of an unreasonable risk; it does not diminish them. Uncertainty, standing alone, does not justify a finding of no unreasonable risk when EPA's own analyses support a finding of unreasonable risk.

[226] For context, other TCE toxicity thresholds that fall between 3 ppb and 52,000 ppb (52 ppm) include the LC50 for fish (between 28 and 66.8 mg/L) and acute toxicity for aquatic invertebrates (7.8 mg/L) see Table 3.1 p. 193). Hence concentrations that would not exceed EPA's asserted algae COC of 52,000 ppb would be *highly lethal to fish* and *highly acutely toxic to aquatic invertebrates*.

To be clear, for one of these facilities, the exceedances EPA calculated were far in excess of the relevant concentration of concern. That facility, according to EPA, had:

> a chronic RQs of 3.81 with 20 days of exceedance, and an algae COCs representing the most sensitive species of algae of 1,000 with 20 days of exceedance. In other words, the surface water concentration modeled for this facility was 3.81 times higher than the COC for chronic exposures, and 1,000 times higher than the COC for the most sensitive species of algae. Assuming 260 days of releases from the facility, the algae RQ representing the most sensitive species was 56.33 with 350 days of exceedance. (pp. 260-261).

C. EPA's analysis of distribution in commerce should be clarified and made explicit.

In the draft risk evaluation, EPA states that "distribution in commerce" "presents an unreasonable risk of injury to health (workers and occupational non-users)," (p. 391), but the draft risk evaluation does not describe the analysis supporting this finding. EPA states that a "quantitative evaluation of the distribution of TCE was not included in the risk evaluation because exposures and releases from distribution were considered within each condition of use." (p. 391). In truth, EPA did not prepare even a *qualitative* evaluation of distribution in commerce of TCE. Based on our search of the draft risk evaluation and supplemental documents, nowhere does it appear EPA actually analyzed distribution in commerce, and EPA should clarify how it analyzed this condition of use and provide the basis for its finding of unreasonable risk.

EPA states that it analyzed distribution in commerce when analyzing the other conditions of use. (pp. 391, 48). But when examining EPA's analysis of various conditions of use—for example, Occupational Exposures—EPA does not appear to have actually analyzed the distribution in commerce of TCE as it relates to these other conditions of use. (pp. 100-107). We could not find any discussion in the analysis of the other conditions of the use that actually addressed distribution in commerce as an aspect of those conditions of use.

Nonetheless, EPA finds that distribution in commerce presents an unreasonable risk. (p. 391). This finding makes sense in light of EPA's conclusion that the other conditions of use present an unreasonable risk (pp. 379-383). If EPA really did analyze distribution of commerce when analyzing these other conditions of use, then the finding of unreasonable risk on these other conditions of use would seem to extend to distribution in commerce. Nonetheless, EPA should clarify how it analyzed distribution and the basis for its finding of unreasonable risk.

In addition, the draft risk evaluation and problem formulation give no attention to potential releases and exposures resulting from accidental releases. EDF does not suggest that EPA needs to consider every possible scenario, but the risk of accidental releases and exposures is very real

and certainly "reasonably foreseen" in many respects. For example, as and after Hurricane Harvey passed through Houston, over 40 sites released toxic chemicals into the environment.[227] Given the known accidental releases, the huge number of petrochemical plants and refineries in the Houston area, and the likelihood that flooding there may become more common in light of climate change, such events are clearly reasonably foreseen and hence EPA needs to give more consideration to the potential for accidental releases.

D. EPA should have banned uses of TCE in vapor degreasing, aerosol degreasing and spot cleaning in dry cleaning facilities.

In December 2016, EPA proposed to ban the use of trichloroethylene (TCE) in aerosol degreasing and spot cleaning in dry cleaning facilities, after finding excessive risks to workers, occupational bystanders, and consumers.[228] In January 2017, the Agency proposed a second ban on the use of TCE in vapor degreasing after finding excessive risks to workers.[229]

EPA's 2014 TCE Work Plan risk assessment[230] and supplemental technical reports make clear that these uses of TCE present an unreasonable risk. These documents reflected input from numerous and extensive peer reviews, incorporated the best available science, and applied a weight-of-the-scientific-evidence approach. EPA's decision to forgo a ban on these uses of TCE in favor of reevaluating their risks in the current risk evaluation was unnecessary and inappropriate. It will delay by years – if not deny – critical actions needed to protect workers and consumers from the dangers posed by these uses of TCE. EDF incorporates by reference its comments on EPA's proposed rules to ban TCE for use in vapor degreasing, aerosol degreasing,

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[227] *See, e.g.*, *More Than 40 Sites Released Hazardous Pollutants Because of Hurricane Harvey*, N.Y. TIMES (Sept. 8, 2017), https://www.nytimes.com/interactive/2017/09/08/us/houston-hurricane-harvey-harzardous-chemicals.html?_r=0.

[228] USEPA, Regulation of Certain Uses under Toxic Substances Control Act: Trichloroethylene, proposed rule, 81 FR 91592 (Dec. 16, 2016), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0163-0001.

[229] USEPA, Regulation of Certain Uses under Toxic Substances Control Act: Trichloroethylene; Vapor Degreasing, proposed rule, 82 FR 7432 (Jan. 19, 2017), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0387-0001.

[230] U.S. EPA, TSCA Work Plan Chemical Risk Assessment Trichloroethylene: Degreasing, Spot Cleaning and Arts & Crafts Uses (2014), https://www.epa.gov/assessing-and-managing-chemicals-under-tsca/tsca-work-plan-chemical-risk-assessment.

and spot cleaning in dry cleaning facilities.[231,232] EPA should promptly act to finalize these bans even as it proceeds to finalize its risk evaluation focusing on risks from other conditions of use of TCE and exposures that would remain after banning these conditions of use due, for example, to allowed exemptions.

8. Systematic review issues

A. OPPT provides neither explanation nor empirical support for its revisions to the systematic review data quality criteria for epidemiological studies, which make it difficult for epidemiological studies to be scored as overall as high quality.

EPA applied its Office of Pollution Prevention and Toxics' (OPPT) updated data quality criteria to epidemiological studies in this draft risk evaluation. The completed data quality evaluation for these studies was provided in the Systematic Review Supplemental File.[233] OPPT provides neither an explanation nor empirical support for its revisions to the systematic review data quality criteria for epidemiological studies, and certain revisions make it more difficult for epidemiological studies to be scored overall as high quality. OPPT's scoring methodology is already at odds with best practices in systematic review, see our earlier comments on OPPT's Application of Systematic Review in TSCA Risk Evaluations,[234] and the Agency's decision to alter scoring criteria without providing any empirical rationale for the changes further underscores that the study quality evaluation strategy that OPPT developed is not evidence-based.

Further, at least six metrics in OPPT's updated epidemiological criteria can no longer receive a score of High, including Metric 5 (Exposure Levels) and Metric 15 (Statistical Models). Since these individual metrics can at best be rated as Medium (a change from the earlier epidemiological criteria), epidemiological studies are thus less likely to be considered high quality overall and as a result may be given more limited consideration than other types of

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[231] EDF Comments on Trichloroethylene (TCE); Regulation of Use in Vapor Degreasing Under TSCA Section 6(a) (May 19, 2017), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0387-0696.

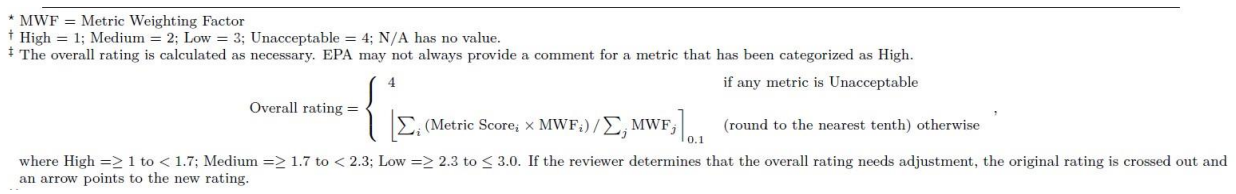
[232] EDF Comments on Trichloroethylene; Regulation of Certain Uses Under TSCA § 6(a) (Mar. 16, 2017), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0163-0172.

[233] U.S. EPA, *Systematic Review Supplemental File: TCE Data Quality Evaluation of Human Health Hazard Studies – Epidemiological Studies CASRN: 75-09-2* p. 5 (Oct. 2019), https://www.epa.gov/sites/production/files/2020-02/documents/15_tce-data_quality_evaluation_of_human_health_hazard_studies_-_epidemiological_data.pdf.

[234] EDF Comment on OPPT's Application of Systematic Review in TSCA Risk Evaluations (Aug. 16, 2018), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2018-0210-0077.

evidence (animal and *in vitro* studies), where it is remains possible to score High across every data quality metric.

In addition to issues with individual scoring criteria, the scheme used to calculate the overall rating for a particular study is not clearly presented in either the updated criteria document or the completed evaluation. The following equation is presented for calculating the overall rating:[235]



The subscripts of *i* and *j* are not defined, and the final subscript of *0.1* is not explained. From this description, it is not possible to see how OPPT calculated its overall ratings for these studies.

Given the concerns related to the appropriateness of the OPPT tool for epidemiological studies and the effect of its application in the context of TCE, the Agency should consider other study evaluation tools that are more appropriate for the consideration of the quality of observational epidemiologic studies. Examples include the Conducting Systematic Reviews and Meta-Analyses of Observational Studies of Etiology (COSMOS-E) tool[236] and the Navigation Guide.[237]

B. OPPT fails to address protocol development, which is a fundamental component of systematic review.

A major deficiency in OPPT's systematic review approach, and in its related OPPT Systematic Review document, is the complete absence of protocol development—a fundamental component of systematic review.

[235] U.S. EPA, *Systematic Review Supplemental File: TCE Data Quality Evaluation of Human Health Hazard Studies – Epidemiological Studies CASRN: 75-09-2* p. 5 (Oct. 2019), https://www.epa.gov/sites/production/files/2020-02/documents/15_tce-data_quality_evaluation_of_human_health_hazard_studies_-_epidemiological_data.pdf.
[236] Dekkers, Olaf M., et al. "COSMOS-E: guidance on conducting systematic reviews and meta-analyses of observational studies of etiology." *PLoS medicine* 16.2 (2019).
[237] Woodruff, Tracey J., and Patrice Sutton. "The Navigation Guide systematic review methodology: a rigorous and transparent method for translating environmental health science into better health outcomes." *Environmental health perspectives* 122.10 (2014): 1007-1014.

As noted in the 2014 National Academy of Sciences (NAS) report that reviewed EPA's IRIS program:

> Critical elements of conducting a systematic review include formulating the specific question that will be addressed (problem formulation) and *developing the protocol* that specifies the methods that will be used to address the question (protocol development).[238]
>
> After the systematic-review questions are specified, protocols for conducting the systematic reviews to address the questions should be developed. *A protocol makes the methods and the process of the review transparent, can provide the opportunity for peer review of the methods, and stands as a record of the review*. It also minimizes bias in evidence identification by ensuring that inclusion of studies in the review does not depend on the studies' findings. Any changes made after the protocol is in place should be transparent, and the rationale for each should be stated. EPA should include protocols for all systematic reviews conducted for a specific IRIS assessment as appendixes to the assessment.[239]

EPA's IRIS program reflects this NAS recommendation by developing problem formulation and assessment protocols for each of its assessments.[240] OPPT needs to develop full protocols for each of its risk evaluations, and should consult with the IRIS program on how best to do so in consideration of requirements under TSCA.

The absence of a protocol violates the Agency's own definition of weight of the scientific evidence: EPA's final rule *Procedures for Chemical Risk Evaluation Under the Amended Toxic Substances Control Act* states that weight of the scientific evidence is:

> a systematic review method, applied in a manner suited to the nature of the evidence or decision, that uses a *pre-established protocol* to comprehensively, objectively, transparently, and consistently identify and evaluate each stream of evidence, including strengths, limitations, and relevance of each study and to

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[238] Nat'l Research Council, *Review of EPA's Integrated Risk Information System (IRIS) Process* at p. 5 (2014), https://www.ncbi.nlm.nih.gov/books/NBK230060/ (emphasis added).
[239] *Id.* at 6 (emphases added).
[240] U.S. EPA, Office of Research & Dev., *National Academy of Science Committee to Review Advances Made to the IRIS Program* at slide 23 (Feb. 2018), http://nas-sites.org/dels/files/2018/01/AdIRIS-15.pdf.

integrate evidence as necessary and appropriate based upon strengths, limitations, and relevance.[241]

C. OPPT's approach taken to evidence integration in the draft TCE risk evaluation is inconsistent and raises concerns.

See our comments in section 4.C.i. above.

D. EPA's selective inclusion of studies otherwise excluded as part of its systematic review process raises concern around inconsistency and bias.

At various points in the draft risk evaluation, EPA chooses to include studies that were otherwise excluded through the Agency's systematic review process, and in doing so raises concerns about inconsistency and bias.

For example, in Section 3.2.1 (Approach and Methodology description for the evaluating Human Health Hazards), EPA notes that "[i]nformation from studies that were rated unacceptable were only discussed on a case-by-case basis for hazard ID and weight-of-scientific-evidence assessment but were not considered for dose-response analysis." (p. 202) Setting aside its significant flaws (see subsections A. and B., above), OPPT's TSCA Systematic Review Method leads EPA to rate a study as unacceptable when one or more data quality metrics for that study are scored as unacceptable. A metric score of unacceptable means that "[s]erious flaws are noted in the domain metric that consequently make the data/information source unusable." Per the TSCA Systematic Review Method studies rated unacceptable, are "...disqualified from further consideration…."

In the context of the draft risk evaluation, EPA fails to identify which "unacceptable" studies were referenced for hazard identification and weight-of-the-scientific-evidence assessment, for which endpoints, and on what basis. Absent any explanation, let alone guidance, for when and how "unacceptable" studies may be considered during risk evaluation, EPA's use ad hoc use of unacceptable studies introduces significant risk for arbitrary, biased, and inconsistent treatment of scientific evidence.

Similarly, footnote 15 in section 3.2.1 (p. 202) indicates that "some of the studies that were excluded based on the PECO statement were considered later during the systematic review process as needed. For example, EPA reviewed mode of action information to qualitatively support the health hazard assessment." While referencing mechanistic information during hazard

[241] EPA, Procedures for Chemical Risk Evaluation Under the Amended Toxic Substances Control Act, Final Rule, p. 33748, available at https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0654-0108 and codified at 40 C.F.R. 702.33 Definition of "Weight of scientific evidence," https://www.law.cornell.edu/cfr/text/40/702.33.

identification is reasonable, EPA's use of studies here that are otherwise excluded through the PECO statement again raises concern that EPA has introduced bias and inconsistency in the risk evaluation process.

EPA should develop general guidance for when these allowances may be considered, and clearly identify, with supporting justification, those specific instances where studies excluded during systematic review or other processes can be referenced and relied on in developing the risk evaluation.

E. EPA's draft risk evaluation appears to be missing key studies.

Comments submitted to the docket raise concerns regarding the absence or dismissal of studies that are relevant or potentially relevant to EPA's draft risk evaluation. For example, comments submitted by Dr. Ivan Rusyn note:

> This section [kidney toxicity, section 3.1.3.1.2] states that the "EPA did not identify new [*sic*] any repeat-dose experimental studies in animals or human epidemiological studies that would contribute significant additional hazard information for this endpoint." This is unfortunate as there are additional informative studies. For example, a study by Yoo et al., 2015 (HERO ID 2799570; PMID: 25424545; PMCID: PMC4281933) examined TCE metabolite levels and toxicity phenotypes in kidneys in mice of various strains." [242]

Panel members at the TCE SACC meeting raised additional concerns around the absence of studies on specific topic areas. For example, one panelist noted the absence of sufficient information on female reproductive toxicity. Another panelist indicated that TCE-induced occupational dermatitis is prevalent but that much of the relevant literature is published in a foreign language. EPA should include relevant studies published in languages other than English when pertinent to a risk evaluation and employ necessary resources to have them translated to ensure these studies are captured.

More broadly, in revising its approach to conducting systematic review, we recommend that OCSPP consult with IRIS, the National Toxicology Program's Office Health Assessment and Translation, and other leading experts on the application of systematic review for chemical assessment, as discussed further in EDF's comments on EPA's OCSPP Systematic Review

[242] Comment submitted by Dr. Ivan Rusyn, MD, PhD, .https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0500-0062.

document.[243] It is also vital that OCSPP's approach be subject to review by the National Academy of Sciences, which appears finally to be happening.

PART II[244]

EDF previously provided comments on the scope and problem formulation for the TCE risk evaluation.[245] In those comments, EDF identified a variety of legal violations and other problems with EPA's approach to the risk evaluation. Unfortunately, many of those same violations and problems appear in the draft risk evaluation, along with new ones. EDF incorporates and reiterates those earlier points here, as well as providing additional comments that address the new concerns.

Similarly, EDF, as part of a broader coalition, filed an Opening Brief and Reply Brief in a lawsuit, *Safer Chems. v. United States EPA*, explaining why EPA must prepare comprehensive risk evaluations considering all conditions of use, including evaluating how exposures from those conditions of use may combine, when evaluating the risks to a chemical.[246] For these same reasons, EPA must comprehensively evaluate TCE, including all its conditions of use and how exposures from those uses combine. EDF incorporates and reiterates those points here as well. We attach those Briefs as Appendices 7 and 8. EPA should fix all of these problems in its final risk evaluation.

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[243] EDF Comments on Application of Systematic Review in TSCA Risk Evaluations (Aug. 16, 2018), https://www.regulations.gov/docket?D=EPA-HQ-OPPT-2018-0210.

[244] Section cross-references in Part II to sections in Part I specify they refer to Part I, while section cross-references internal to Part II do not specify they refer to Part II.

[245] EDF Comments on Ten Scopes under the Toxic Substances Control Act, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0742-0072; EDF Comments on Problem Formulations for Risk Evaluations To Be Conducted Under Toxic Substances Control Act, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0742-0101.

[246] The Ninth Circuit Court's Opinion in that case was issued in November, 2019, and is discussed further in section 1 below.

1. TSCA requires EPA to analyze whether a chemical substance, as a whole, presents an unreasonable risk, and EPA does not have discretion to ignore conditions of use, exposures, or hazards.

In its prior scoping document and problem formulation for TCE,[247] EPA stated that it had authority to exclude conditions of use. In the draft risk evaluation, EPA again relied on this assertion of authority. In our comments on the scope and the problem formulation, EDF explained that this approach is foreclosed under the statute, and EDF incorporates those arguments here.[248] Similarly, EDF incorporates the arguments presented in our Briefs attached as Appendix 7 at 21-40 and Appendix 8 at 14-31. Moreover, as explained more below, this approach is also foreclosed by EPA's regulations, as interpreted by the Ninth Circuit in *Safer Chems. v. United States EPA*, 2019 U.S. App. LEXIS 33976, *39-40 (9th Cir. Nov. 14, 2019).

In the problem formulation, as well as in the draft risk evaluation (p. 35), EPA states that it will also exclude hazards and exposures under some conditions of use as well. Specifically, in the draft risk evaluation EPA states that it will exclude all general population exposures to TCE through groundwater, drinking water, surface water, air, and land (pp. 35, 379), and as a result the draft risk evaluation contains no analysis of risks to the general population (p. 379). EPA acknowledges that these exposures flow from conditions of use, including "industrial and/or commercial uses; industrial releases to air, water or land; and other conditions of use" (p. 35). EPA also effectively ignores certain hazards by completely failing to provide any quantitative analysis of environmental hazards to sediment-dwelling, terrestrial, or avian organisms (p. 29) (limiting such analysis to aquatic hazards). EPA also effectively ignores other hazards by failing to analyze: (1) cancer risks from acute exposures (p. 251) and (2) the unique hazards presented to certain potentially exposed or susceptible subpopulations in its risk analysis; *see* Part I, sections 1.A. and 4.A.

TSCA's language and structure unambiguously foreclose EPA's interpretation. EPA's decision to disregard certain exposure pathways and hazards is also "arbitrary, capricious, [or] an abuse of discretion" under the APA, 5 U.S.C. § 706(2)(A), because it will lead EPA to consider "factors which Congress has not intended it to consider [and] entirely fail[] to consider an important aspect of the problem." *Motor Vehicle Mfrs. Ass'n v. State Farm Mut. Auto. Ins. Co.*, 463 U.S. 29, 43 (1983). Moreover, as the draft risk evaluation itself reveals, this approach leads to

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[247] U.S. EPA, Problem Formulation of the Risk Evaluation for Trichloroethylene (May 2018), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0737-0083 (hereinafter, "Problem Formulation for TCE").

[248] EDF Comments on Ten Scopes under the Toxic Substances Control Act, pp. 4-11, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0742-0072; EDF Comments on Problem Formulations for Risk Evaluations To Be Conducted Under Toxic Substances Control Act, pp.13-20, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0742-0101.

irrational and arbitrary applications. Instead, EPA should be guided by the statutory language and consider all of the conditions of use, exposures, and hazards related to a chemical substance. EPA should evaluate all of the evidence of conditions of use, exposure, and hazard; not ignore evidence because of self-imposed blinders.

A. The plain text, overall structure, purpose, and legislative history of TSCA indicate that EPA has to determine whether a chemical substance presents an unreasonable risk comprehensively, considering all of its hazards, exposures, and conditions of use.

i. The plain text requires EPA to consider all hazards, exposures, and conditions of use.

Statutory interpretation should begin, as always, with the language of the statute. The plain language of the risk evaluation provision supports the interpretation that EPA must consider all hazards, exposures, and conditions of use as necessary "to determine whether a *chemical substance* presents an unreasonable risk." 15 U.S.C. § 2605(b)(4)(A) (emphasis added). This directive expresses Congress's clear intent that EPA evaluate the risks posed by "a chemical substance" as a whole. Congress consistently used the phrase "a chemical substance" to describe the object of priority designations and risk evaluations. 15 U.S.C. § 2605(b)(1)-(4), (i) (using the phrase 14 times). This language requires EPA to consider all hazards and exposures that contribute to the total risk presented by the chemical substance as a whole.

This whole-substance focus begins during prioritization. The definitions of high- and low-priority substances make clear that it is the "substance" that receives the designation, not selected conditions of use, exposures, or hazards. *See id.* § 2605(b)(1)(B). The provision requiring EPA to select the first ten chemicals also directed that the risk evaluations be "conducted on 10 chemical substances drawn from the 2014 update of the TSCA Work Plan," making the object of these risk evaluations the chemical substances as a whole. *Id.* § 2605(b)(2)(A). As EPA reasoned in the Prioritization Rule, "[t]he statute is clear that EPA is to designate the priority of the 'chemical substance'—not a condition of use for a chemical substance." 82 Fed. Reg. 33,753, 33,755 (July 20, 2017) (citing 15 U.S.C. § 2605(b)(1)(A)). Similarly, EPA must prioritize the whole chemical, and EPA is not directed to prioritize only certain hazards or exposures. Indeed, the prioritization process expressly "shall include a consideration of the hazard and exposure potential of a chemical substance," without any basis for EPA to limit that consideration to only certain hazards or exposures. 15 U.S.C. § 2605(b)(1)(A).

EPA must also conduct risk evaluations on "a chemical substance" as a whole. For example, TSCA provides that "[u]pon designating a chemical substance as a high-priority substance, the Administrator shall initiate a risk evaluation on the *substance*." 15 U.S.C. § 2605(b)(3)(A) (emphasis added). Similarly, the statute directs EPA to determine either that "a *chemical substance* presents" or "does not present an unreasonable risk." *Id.* § 2605(i)(1)-(2) (emphasis added). Congress also uses the phrase "a chemical substance" or "chemical substances" in many

other places in TSCA's risk evaluation provisions. *See, e.g.*, *id.* § 2605(b)(4)(G) (setting deadlines for completing evaluation for "a chemical substance"), (b)(2)(A), (b)(2)(B), (b)(3)(A), (c)(1).

The plain language of the risk evaluation provisions requires EPA to consider all available information about hazards, exposures, and conditions of use, without limitation. TSCA § 6(b)(4)(F)(i) expressly requires that EPA "integrate and assess available information on hazards and exposures for the conditions of use of the chemical substance." 15 U.S.C. § 2605(b)(4)(F)(i). Thus, if there is "available information on hazards and exposures," then EPA must integrate and assess that information as part of the risk evaluation. Similarly, TSCA § 6(b)(4)(F)(iv) requires that EPA "take into account, where relevant, the likely duration, intensity, frequency, and number of exposures under the conditions of use of the chemical substance." *Id.* § 2605(b)(4)(F)(iv). This provision requires EPA to take into account exposures unless EPA can establish that they are irrelevant. Finally, TSCA § 6(b)(4)(F)(v) requires that EPA "describe the weight of the scientific evidence for the identified hazard and exposure." *Id*. § 2605(b)(4)(F)(v).

All of these provisions direct EPA to consider a chemical's hazards, exposures, and conditions of use, and none of them include any language providing EPA with any discretion to ignore any hazards, exposures, or conditions of use. While EPA previously articulated a legal theory (albeit flawed) for ignoring certain conditions of use, EPA has not pointed to any legal basis for ignoring hazards or exposures under the conditions of use being analyzed in a risk evaluation. EPA has pointed to no textual basis for these exclusions.

Moreover, when EPA promulgates risk-management regulations under TSCA § 6(a):

> [EPA] shall consider and publish a statement based on reasonably available information with respect to—
>
> (i) the effects of the chemical substance or mixture on health and the magnitude of the exposure of human beings to the chemical substance or mixture;
>
> (ii) the effects of the chemical substance or mixture on the environment and the magnitude of the exposure of the environment to such substance or mixture;

15 U.S.C. § 2605(c)(2)(A). In order to accurately draft this statement, EPA will have to have considered all of the hazards posed by a chemical (i.e., its effects on human health and the environment) as well as all exposures. EPA cannot accurately describe "the magnitude of the exposure of human beings to the chemical substance," if EPA has ignored numerous exposures. 15 U.S.C. § 2605(c)(2)(A)(i). Similarly, EPA cannot accurately describe "the magnitude of the exposure of the environment" for chemicals, *id.* § 2605(c)(2)(A)(ii), if EPA has ignored the vast majority of environmental exposures, as EPA proposes to do. Congress specifically intended for EPA to "satisfy these requirements on the basis of the conclusions regarding the chemical's

health and environmental effects and exposures in the risk evaluation itself." 114 Cong. Rec. S3517 (daily ed. June 7, 2016). Thus, EPA must evaluate all hazards and exposures in its risk evaluations.

Moreover, TSCA requires that EPA evaluate a chemical's risk "without consideration of costs or other nonrisk factors." 15 U.S.C. § 2605(b)(4)(A). By excluding certain hazards, exposures, and conditions of use for reasons that bear no relationship to risk, EPA is considering nonrisk factors. For example, by excluding exposures because they could be regulated under another statute, EPA is considering a nonrisk factor.

Textually, EPA's approach also directly conflicts with TSCA § 26(k). 15 U.S.C. § 2625(k). TSCA § 26(k) requires EPA to "take into consideration information relating to a chemical substance or mixture, including hazard and exposure information, under the conditions of use, that is reasonably available to the Administrator." *Id.* Congress included this provision to ensure that EPA could not ignore "reasonably available" "information relating to a chemical substance or mixture"; the purpose of this provision is to compel EPA to consider all reasonably available information. Congress also specified that EPA must consider the reasonably available "hazard and exposure information." It would undermine this directive if EPA chooses to ignore certain hazards or exposures.

ii. TSCA's overall structure requires EPA to consider all hazards, exposures, and conditions of use.

Moreover, EPA's pick-and-choose approach cannot be squared with the overall structure of TSCA.

As EPA reasoned in its proposed Risk Evaluation Rule, when discussing conditions of use, that TSCA "provides no criteria for EPA to apply" for selecting hazards, exposures, and conditions of use for analysis shows that the Agency does not have "license to choose" among those hazards, exposures, and conditions of use for analysis. 82 Fed. Reg. 7562, 7566 (Jan. 19, 2017). The precision with which Congress prescribed EPA's implementation of section 6 supports this reading. Section 6 lays out detailed directions for EPA. *See* 15 U.S.C. § 2605(b)(1)(A) (mandating considerations for priority designations), (b)(4)(D) (identifying risk factors to include in a risk evaluation's scope), (b)(4)(F)(i)-(v) (detailing requirements for conducting risk evaluations); *see also id.* § 2605(a) (specifying possible risk management measures). These provisions indicate that Congress did not mean to allow EPA to exclude hazards, exposures, or conditions of use from risk evaluation without any criteria or instruction. *Cf. NRDC, Inc. v. EPA*, 863 F.2d 1420, 1432 (9th Cir. 1988) (invalidating regulatory procedure that "is wholly silent as to what factors the agency is to consider in granting exceptions" and provides "no discernible standard [for] limit[ing] th[at] discretion").

Indeed, when Congress intended EPA to exercise discretion under TSCA, it said so explicitly. *See, e.g.*, 15 U.S.C. §§ 2613(f) (granting EPA "[d]iscretion" in handling claims to protect

confidential information), 2608(a) (instructing EPA, if it "determines, in the Administrator's discretion," that an unreasonable risk may be prevented under a federal law administered by another Agency, to notify the Agency), 2608(b), 2605(b)(4)(E)(iv)(II). That Congress purposefully included the language of discretion "in one section of the statute but omit[ted] it in another section of the same Act" shows that Congress did not intend EPA to use discretion to pick and choose which hazards, exposures, and conditions of use to consider in prioritization and risk evaluation. *Hernandez v. Ashcroft*, 345 F.3d 824, 834 (9th Cir. 2003) (quoting *Andreiu v. Ashcroft*, 253 F.3d 477, 480 (9th Cir. 2001) (en banc)).

Implicitly recognizing that Congress did not grant EPA boundless discretion to exclude exposures, EPA suggests that it will "focus on those exposure pathways associated with TSCA uses that are not subject to the regulatory regimes [such as the Clean Air Act] because these pathways are likely to represent the greatest areas of concern to EPA." (p. 33). But no language in TSCA limits EPA to this "greatest areas of concern" focus. Nor does EPA point to any statutory terms that even arguably supply such a limitation.

TSCA's provisions direct EPA to prepare risk evaluations and the related findings for "chemical substances," as a whole, not for specific or limited hazards, exposures, or conditions of use of those substances. For example, the risk management provision expressly requires EPA to address risks when the risks arise from combined sources of exposure. TSCA § 6(a) provides that: "If [EPA] determines in accordance with [the risk evaluation provision] that the manufacture, processing, distribution in commerce, use, or disposal of a chemical substance or mixture, or that any combination of such activities, presents an unreasonable risk of injury to health or the environment," then EPA must issue a risk management rule. 15 U.S.C. § 2605(a); *see also* 15 U.S.C. § 2608(a) (using same language in provision governing requests to other federal agencies to address risks). Thus, if exposures resulting from "any combination" of conditions of use present an unreasonable risk, EPA must issue a risk management rule. But EPA must analyze *all* of the exposures resulting from these activities to assess whether *any combination* presents such a risk.

iii. TSCA's purpose, as well as basic logical reasoning and the best available science, require EPA to consider all hazards, exposures, and conditions of use to assess a chemical substance as *a whole*.

The purpose of the risk evaluation is to analyze the risks of a substance based on an assessment of its hazards and exposures. Ignoring potential exposures and hazards at the outset undermines that purpose. And science and logic do not support EPA's exclusions. As explained below in sections 1.C. and 5, EPA's exclusions of certain exposures result in an incoherent draft risk evaluation where EPA acknowledges ample evidence of exposure, for example, in the monitoring data, but then refuses to look at those very exposures in its final analysis. Willfully ignoring these exposures at the outset is contrary to the purpose of TSCA's risk evaluations, as well as the law's requirement that EPA rely on the best available science. EPA is imposing

blinders on its analysis by asserting authority to refuse to look at certain exposures, including known exposures, and the result is that EPA is overlooking exposures in the real world. This approach is both contrary to law and arbitrary and capricious.

iv. The legislative history requires EPA to integrate a chemical's exposure and hazard information and nothing suggests that EPA can ignore existing exposures and hazards.

Numerous statements in the legislative history reveal that Congress intended for EPA to assess "risk" based on "the integration of hazard and exposure information about a chemical." S. Rep. No. 114-67 at 17 (June 18, 2015); 161 Cong. Rec. H4551 at H4556 (daily ed. June 23, 2015) ("The risk evaluation itself only asks does the chemical present an unreasonable risk of injury to health or the environment. That is a science question based on a combination of hazard and actual exposure."). Senator Vitter described an accurate assessment of risk as turning on integrating exposure and hazard information. *See* 162 Cong. Rec. S3511 at S3519 (daily ed. June 7, 2016) ("Exposure *potential*, when integrated with the hazard *potential* of a chemical, determines a chemical's potential for risk.") (emphases added). Congress intended for EPA to integrate all available information about exposure and hazard when assessing risk, as reflected in this history and the text of TSCA.

No statement in the legislative history suggests that EPA may ignore exposures or hazards when assessing the risk presented by a chemical substance. In the preamble to the Risk Evaluation Rule, EPA relied on a floor statement from a single Senator to justify its interpretation that it had discretion to choose the conditions of use for analysis. 40 Fed. Reg. at 33,728 (citing 114 Cong. Rec. S3519-20 (daily ed. June 7, 2016) (statement of Sen. Vitter)). As EDF has previously explained,[249] the legislative history as a whole does not justify EPA's approach to conditions of use, but here EPA has even less basis for its approach; EPA has not pointed to any statement in the legislative history supporting its approach of ignoring certain exposures or hazards.

B. Properly interpreted, EPA's own risk evaluation rule requires that EPA consider all relevant hazards, all exposures, and all conditions of use.

Properly interpreted, EPA's own risk evaluation rule does not give EPA discretion to exclude any hazards, exposures, or conditions of use.

i. EPA's Risk Evaluation Rule does not give EPA discretion to exclude conditions of use.

EPA's Risk Evaluation Rule does not grant EPA discretion to exclude conditions of use. While the preamble to the Rule asserted that EPA had discretion to exclude conditions of use, no

[249] EDF Comments on Ten Scopes under the Toxic Substances Control Act, pp.7-8, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0742-0072.

provision of the Rule actually granted EPA such discretion, and the Rule read as a whole undermines EPA's claims to discretion. Specifically, the Rule provides that the scope of the risk evaluation will include: "The condition(s) of use, as determined by the Administrator, that the EPA plans to consider in the risk evaluation," 40 C.F.R. § 702.41(c)(1), and the Rule then often refers to EPA analyzing the hazards and exposures "for the conditions of use within the scope of the risk evaluation," 40 C.F.R. § 702.43(a)(1). As the Ninth Circuit has interpreted the language of the Rule:

> The phrase "the conditions of use within the scope of" an evaluation simply refers to the conditions of use that are applicable to any particular substance—and that therefore are included in the scope of that substance's evaluation—without excluding any conditions of use in forming that list. Likewise, the phrase that refers to the conditions of use "that the EPA plans to consider" simply refers to the Agency's role in determining what the conditions of use are for a particular substance.

Safer Chems. v. United States EPA, 2019 U.S. App. LEXIS 33976, *38-39. Therefore, the Court found that the relevant provisions "unambiguously do not grant EPA the discretion" to pick-and-choose conditions of use for inclusion in a risk evaluation. *Id.* at *40. Notably, the Court explained that "because the scope provisions are not ambiguous on their face, reference to the preamble discussion would be improper." *Id.* This ruling renders EPA's assertion of discretion to exclude conditions of use in the preamble meaningless.

The Court's ruling in this case turned on the Court's finding the regulations unambiguous on this issue; thus the ruling cannot be dismissed as mere *dicta*. The Court unequivocally found that the regulations were "unambiguous" on this point, leaving EPA no discretion to interpret the regulations in a different way. Thus, EPA must interpret the regulations as requiring EPA to analyze all conditions of use.

ii. EPA's Risk Evaluation Rule does not give EPA discretion to exclude any hazards and exposures under the conditions of use within the risk evaluation.

In addition, EPA's final Risk Evaluation Rule requires EPA to consider all relevant hazards and exposures under the conditions of use within the risk evaluation. The Rule specifically requires that: "Relevant *potential* human and environmental hazards will be evaluated." 40 C.F.R. § 702.41(d)(3) (emphasis). Thus, EPA must consider any relevant "potential" hazards when preparing a risk evaluation. *See also* 40 C.F.R. § 702.41(d)(2) ("The hazard assessment process will identify the types of hazards to health or the environment posed by the chemical substance under the condition(s) of use within the scope of the risk evaluation."). The Rule also requires that: "[e]xposure information related to potential human health or ecological hazards of the chemical substance will be reviewed in a manner consistent with the description of best available science and weight of scientific evidence." 40 C.F.R. § 702.41(e)(3). When preparing the risk

characterization, EPA shall "[t]ake into account, where relevant, the likely duration, intensity, frequency, and number of exposures under the condition(s) of use of the chemical substance." 40 C.F.R. § 702.43(a)(4). Thus, EPA must consider all hazards and all exposures under the conditions of use. None of these duties are qualified or provide an authority for EPA to exclude hazards or exposures from analysis.

Other provisions of the rule confirm this reading. EPA requires manufacturer requests for risk evaluations to "include or reference *all* available information on the health and environmental hazard(s) of the chemical substance, human and environmental exposure(s), and exposed population(s), as relevant to the circumstances identified in the request." 40 C.F.R. § 702.37(b)(4) (emphasis added). Thus, manufacturers must submit all available information on hazard and exposure under the identified conditions of use because EPA must consider all hazards and exposures when preparing risk evaluations.

In the preamble to the rule, EPA commits to considering all hazards and exposures under the conditions of use:

> The Administrator will consider relevant factors including, but not limited to: The effects of the chemical substance on health and human exposure to such substance under the conditions of use (including cancer and non-cancer risks); the effects of the chemical substance on the environment and environmental exposure under the conditions of use.

82 Fed. Reg. at 33,735. EPA thus committed to considering the "effects of the chemical substance on health and human exposure to such substance under the conditions of use." *Id.* These commitments are not qualified or accompanied by any assertion of discretion to ignore effects or exposure information under the conditions of use. EPA cannot fulfill this duty without considering all the hazards and sources of human exposure under the conditions of use.

Similarly, in the preamble, EPA states that "[u]sing reasonably available information, exposures will be estimated (usually quantitatively) for the identified conditions of use." 82 Fed. Reg. at 33,742. EPA cannot prepare an accurate quantitative estimate for exposure if EPA has excluded exposure pathways. "For environmental evaluations specifically, EPA plans to include a discussion of the nature and magnitude of the effects, the spatial and temporal patterns of the effects, [and] implications at the species, population, and community level." 82 Fed. Reg. at 33,743. EPA cannot accurately discuss the magnitude of the effects on the environment or the spatial and temporal patterns of those effects if EPA ignores the vast majority of the environmental exposures, as EPA proposes to do.

Moreover, in the preamble to the rule, while EPA went to great lengths to describe its alleged discretion to pick-and-choose conditions of use (which discretion is lacks under the Rule for the reasons given above), EPA never stated that it had discretion to exclude hazards or exposures related to conditions of use within the risk evaluation. EPA's failure to assert any discretion to

exclude exposures and hazards reflects that EPA, in fact, lacks any such discretion. Similarly, in the preamble to the risk evaluation rule, EPA asserted that it had authority to ignore conditions of use under other agencies' jurisdiction. 82 Fed. Reg. at 33,729 (July 20, 2017). This is incorrect, but EPA never asserted that it had authority to ignore exposures under EPA's jurisdiction. Once again, EPA's silence on this issue in its rule highlights that EPA could not justify such discretion. In sum, EPA's arguments for excluding certain conditions of use cannot simply be extended mindlessly to exclude consideration of exposures and hazards. *See United States Sugar Corp. v. EPA*, 830 F.3d 579, 650 (D.C. Cir. 2016) (Agency may not assume a rationale for one exemption identically applies elsewhere).

C. The draft risk evaluation is incoherent and arbitrary and capricious because of EPA's approach to certain hazards, exposures, and conditions of use.

EPA's illegal approach to exposures leads it to put "blinders" on regarding risks. The result is "arbitrary, capricious, [or] an abuse of discretion" under the APA, 5 U.S.C. § 706(2)(A), because it will lead EPA to have considered "factors which Congress has not intended it to consider [and] entirely failed to consider an important aspect of the problem." *State Farm*, 463 U.S. at 43. It also violates several provisions of TSCA § 26 because by ignoring uses, exposures, hazards, and related information, EPA will not be acting "consistent with the best available science," EPA will not base decisions on "on the weight of the scientific evidence," and EPA will not "take into consideration information relating to a chemical substance or mixture, including hazard and exposure information, under the conditions of use, that is reasonably available to the Administrator." 15 U.S.C. § 2625(h), (i), (k). In addition, because EPA's distinction is a false one untethered to the information, EPA seems to treat certain exposures inconsistently throughout the documents.

For example, as detailed more below, at multiple points in the draft risk evaluation, EPA acknowledges that TCE exposures occur through numerous media (pp. 29, 56, 99). But EPA then systematically excludes many of these pathways of exposure from its risk evaluation (pp. 35, 379), imposing arbitrary blinders on its analysis despite the factual evidence before it. This is the definition of arbitrary and capricious conduct.

EPA should change the final risk evaluation to assess the reasonably available information on all hazards and exposures for TCE, and that information should inform EPA's evaluation of the risks of this chemical. If there is a real-world or reasonably foreseen exposure or hazard, then EPA cannot ignore it.

2. EPA should not refuse to further analyze exposure pathways on a cursory basis, and in any event, EPA still needs to consider those exposures when evaluating combined exposures.

In the problem formulation for TCE, EPA insufficiently justified many of its decisions not to include known or potential exposures or conduct further analysis, and prematurely concluded

various exposures present no significant risk. EPA's problem formulation contained many rushes to judgment, with EPA all but concluding there is no unreasonable risk from certain exposures, based on little analysis and with no indication that it intended to revisit those exposures or risks in combination with those it did intend to analyze further. The draft risk evaluation fails to provide the analysis missing from the problem formulation, and thus, these cursory analyses remain deeply flawed. We describe some of EPA's flawed analyses below in section 3.

When EPA declines to analyze a pathway further, EPA must have developed and applied a sound, rational basis for assessing the exposure level, supported by scientific evidence. In addition, EPA cannot then effectively ignore the exposure. Rather, EPA still must consider how the exposure may combine with other sources of exposure, so EPA must actually assess the level of exposure from the pathway individually and then consider how it combines with other sources of exposure.

EDF has previously argued that EPA must consider how exposures combine to result in total exposure in our Briefs attached as Appendix 7 at 39-40 and Appendix 8 at 27-31. We incorporate those arguments by reference here.

3. EPA excludes certain exposure pathways based on insufficient evidence or illogical rationales.

EPA has made a number of inappropriate decisions not to further analyze specific exposure pathways or receptors with little or no explanation. The exclusions violate EPA's duty to analyze all exposure pathways, and they are also arbitrary and capricious for failing to consider an important aspect of the problem.

A. EPA ignores certain exposure pathways to humans with little analysis or basis.

EPA excludes the oral exposure route. (pp. 57-58). For consumers, EPA acknowledges the potential for exposure via hand-to-mouth patterns" of behaviors but waves it off by stating that "based on physical chemical properties, mists of TCE are expected to be rapidly absorbed in the respiratory tract or evaporate upon being introduced into the respiratory tract." Problem Formulation for TCE at p. 37. The oral exposure pathway for workers is ignored altogether. (p. 57; Problem Formulation for TCE at p. 36). The absence of any analysis renders these decisions arbitrary and capricious. The draft risk evaluation does not provide the missing analysis.

EPA excludes exposure to consumers from disposal. EPA's rationale, with no supporting data or analysis, is that the Agency "does not expect exposure to consumers from disposal of consumer products. It is anticipated that most products will be disposed of in original containers, particularly those products that are purchased as aerosol cans." Problem Formulation for TCE at p. 37. However, EPA then goes on to state that "liquid products may be recaptured in an alternate container following use (refrigerant flush or coin cleaning)," Problem Formulation for

TCE at p. 37, providing a plausible mechanism by which consumers may be exposed during disposal.

B. EPA excluded exposures to terrestrial species on a cursory basis.

EPA ignores important pathways of TCE exposure to terrestrial organisms based on unclear and apparently non-conservative assumptions. EPA's analysis does not reflect the best available science and is arbitrary and capricious. See Part I, section 6.B.

4. EPA must analyze background exposures in the risk evaluation.

In the draft risk evaluation for TCE, EPA does not consider the background exposures that workers and consumers experience through ambient air, drinking water, and other exposure pathways. See Part I, section 2.E. EPA needs to include consideration of such exposures in its draft risk evaluation for the reasons articulated in section 1. But the exclusion of these exposures also undermines EPA's analysis of those circumstances that EPA does analyze in the draft risk evaluation because it is the total level of exposure to a chemical that determines risk, and this includes exposures that are not generally attributable to any one use or source.

5. EPA cannot ignore ongoing, real-world exposures because they are occurring despite another EPA-administered statute that could potentially cover those exposures.

As established above, EPA must assess all hazards and exposures when evaluating the risk presented by a chemical substance. For this same reason, EPA must consider all real-world, intended, and reasonably foreseen exposures that occur even if they fall under the jurisdiction of other EPA-administered statutes. In the draft risk evaluation, EPA stated that it excluded exposure pathways when they were covered by other EPA-administered statutes. (pp. 35, 379). Specifically, EPA asserted that:

> Exposure pathways to the general population are covered by other statutes and consist of: the ambient air pathway (i.e., TCE is listed as a HAP in the Clean Air Act (CAA)), the drinking water pathway (i.e., National Primary Drinking Water Regulations (NPDWRs) are promulgated for TCE under the Safe Drinking Water Act), ambient water pathways (i.e., TCE is a priority pollutant with recommended water quality criteria for protection of human health under the CWA), the biosolids pathway (i.e., the biosolids pathway for TCE is currently being addressed in the CWA regulatory analytical process), disposal pathways (TCE disposal is managed and prevented from further environmental release by RCRA and SDWA regulations). As described above, other environmental statutes administered by EPA adequately assess and effectively manage these exposures. EPA believes that the TSCA risk evaluation should focus on those exposure pathways associated with TSCA conditions of use that are not subject to the regulatory regimes discussed above because those pathways are likely to represent

> the greatest areas of concern to EPA. Therefore, EPA did not evaluate hazards or exposures to the general population in this risk evaluation, and there is no risk determination for the general population (U.S. EPA, 2018d).

This approach is illegal and arbitrary and capricious for numerous reasons, including because TSCA requires EPA to analyze all exposures for the reasons discussed above. This approach also violates the text and structure of TSCA for additional reasons unique to this rationale for excluding exposures.

As discussed in more detail below, first and foremost this approach is factually and scientifically inaccurate. For numerous sources of exposure, EPA treats the overall exposure from a particular pathway as "zero" or non-existent despite the fact that the available evidence thoroughly establishes that exposure is occurring at levels well above zero regardless of any actions taken under the other statutes EPA invokes. Thus, in reality, human beings and the environment are experiencing levels of exposure that EPA is willfully ignoring. EPA is choosing to adopt false factual assumptions, and "[r]eliance on facts that an agency knows are false at the time it relies on them is the essence of arbitrary and capricious decisionmaking." *Animal Legal Def. Fund, Inc. v. Perdue*, 872 F.3d 602, 619 (D.C. Cir. 2017). This approach also violates the requirements to act "consistent with the best available science" and to "take into consideration information relating to a chemical substance or mixture, including hazard and exposure information, under the conditions of use, that is reasonably available to the Administrator." 15 U.S.C. § 2625(h), (k). The draft risk evaluation does not establish that the regulation of these chemical substances under other statutes will eliminate exposures, and in fact, the problem formulation, draft risk evaluation and publicly available evidence establish that exposures continue to occur in the real-world despite these statutes. EPA cannot ignore those exposures.

In addition, EPA must consider the possibility that these exposures, *combined with other sources of exposure*, could present an unreasonable risk. EPA's decision to ignore exposures one-by-one rather than look at combined exposure is inherently inaccurate and will invariably lead to an underestimation of exposure and risk.

Furthermore, EPA has not established that these environmental statutes "adequately assess and effectively manage exposures." EPA's bald assertions to the contrary do not make it so. In any event, that is not the legally correct standard under TSCA. As explained below, EPA can only rely on statutory authorities other than TSCA in compliance with TSCA § 9 (notably, the TSCA § 9 process occurs *after* EPA has completed a comprehensive risk evaluation finding unreasonable risk). To comply with TSCA § 9, EPA must find that those authorities eliminate the risks EPA has previously identified or reduce them to a sufficient extent under TSCA § 9(b)(1), and TSCA requires that EPA reduce risk "to the extent necessary so that [the chemical] no longer presents [an unreasonable risk of injury to health or the environment]." *See* 15 U.S.C. §§ 2608(b)(1), 2605(a). In addition, under TSCA § 9(b)(2) EPA must consider "all relevant aspects of the risk" when deciding whether to regulate under TSCA or another statute.

Id. § 2608(b)(2). EPA has not met any of these standards in the draft risk evaluation, and EPA's statements that the exposures are adequately assessed and effectively managed under other statutes are legally irrelevant (even if they were true).

When relying on these other statutory authorities, EPA merely provides a list of various statutes or regulatory standards and criteria that EPA indicates apply or could apply to certain sources of TCE (*see, e.g.*, Table_Apx A-1, pp. 464-468). EPA provides no analysis whatsoever as to: the extent to which the standards or criteria cover the full range of exposure to the chemical through the pathway; the extent and magnitude of releases of the chemical allowed under each of the regulatory standards or criteria; or any other factors that would be necessary to analyze to determine the extent and nature of potential risk allowed under the standards. In particular, TSCA § 6(b)(4)(F)(iv) requires that, in conducting a risk evaluation, EPA evaluate "the likely duration, intensity, frequency, and number of exposures," 15 U.S.C. § 2605(b)(4)(F)(iv), including exposures resulting from those allowable emissions, discharges, or releases. EPA needs to provide this analysis, and EPA cannot simply point to regulation under another statute to bypass the analysis. EPA has also not acknowledged, let alone analyzed, the overall risks to the general population or to vulnerable subpopulations due to the combination of exposures arising from the various sources for which standards exist, not to mention in combination with additional emission sources not subject to any standard. EPA has made no attempt to reconcile any such risk with that allowed under TSCA.

At a minimum, EPA has completely failed to establish that these statutes reduce exposure to zero. To the contrary, it is thoroughly clear that humans and the environment continue to experience significant exposures through the excluded pathways. To prepare a scientifically accurate risk evaluation, EPA must analyze the exposures through those pathways.

A. The text and overall structure of TSCA makes it clear that EPA has to analyze exposures, even if they have been or could be assessed under another statute.

In the problem formulation, to which EPA refers in the draft risk evaluation for support (p. 35), EPA asserts that it has discretion to exclude "certain exposure pathways that fall under the jurisdiction of other EPA-administered statutes." *See, e.g.*, Problem Formulation for TCE at p. 13. But EPA provides no textual basis for ignoring those exposures. Instead, in a footnote, EPA cites to its discussion regarding "conditions of use," but even assuming for the sake of argument that EPA has authority to exclude conditions of use, such power does not justify excluding exposures related to conditions of use still within the scope of the risk evaluation, as EPA proposes to do. Nothing in TSCA's risk evaluation provision authorizes EPA ignoring exposures because of other statutory authorities, and as explained above, EPA has to analyze all exposures including these exposures. And several other provisions of TSCA indicate that Congress intended for EPA to consider such exposures, except to the extent Congress explicitly provided otherwise.

First, Congress expressly excluded certain chemicals or uses of chemicals regulated under other statutes when it defined "chemical substance" in TSCA § 3(2). 15 U.S.C. § 2602(2)(B). For example, "chemical substance" does not include "any pesticide (as defined in the Federal Insecticide, Fungicide, and Rodenticide Act) when manufactured, processed, or distributed in commerce for use as a pesticide." *See id*. § 2602(2)(B)(ii). Thus, when Congress intended for EPA not to regulate certain exposures because they were regulated under other specific EPA-administered statutes, Congress expressly excluded those exposures. That Congress chose a limited, specific set of exclusions indicates that Congress did not intend for EPA generally to ignore other exposures where they fall under other federal regulatory schemes.

Second, in TSCA's risk evaluation provision, Congress specifically intended for EPA to "conduct risk evaluations *** to determine whether a chemical substance presents an unreasonable risk of injury to *** the environment," 15 U.S.C. § 2605(b)(4)(A), but EPA's approach has eliminated almost all analysis of environmental exposures. EPA has largely read the requirement to evaluate risks to the environment out of the statute, but this approach violates a fundamental tenant of statutory interpretation. A. SCALIA & B. GARNER, READING LAW: THE INTERPRETATION OF LEGAL TEXTS 174 (2012) ("If possible, every word and every provision is to be given effect *** None should needlessly be given an interpretation that causes it to duplicate another provision or to have no consequence."). Moreover, Congress enacted this requirement that EPA analyze risks to the environment against the backdrop of the existing environmental statutes; if Congress had considered them per se sufficient, Congress would not have included this mandate in TSCA. But Congress did.

Third, Congress specifically directed EPA to analyze the risks of chemicals presented "under the conditions of use," and Congress consciously decided to specify that "disposal" is a condition of use under TSCA. "Conditions of use" expressly includes "the circumstances *** under which a chemical substance is intended, known, or reasonably foreseen to be to be manufactured, processed, distributed in commerce, used, or *disposed of*." 15 U.S.C. § 2602(4) (emphasis added). In the problem formulation, EPA systematically excludes exposures through disposal based on a variety of theories (Problem Formulation for DCM at pp. 55-57), and in doing so, EPA is ignoring Congress's direction that it assess risks associated with the conditions of use, including disposal. Similarly, EPA is ignoring exposures from other conditions of use, such as "manufactur[ing]," "process[ing]," and potentially distribution in commerce, by for example ignoring the emissions from the manufacturing and processing facilities. Congress expressly included all of these circumstances within the definition of "conditions of use," and EPA should not ignore the exposures resulting from them.

Fourth, TSCA § 9(b) provides that EPA "shall coordinate *actions* taken under [TSCA] with *actions* taken under other Federal laws administered in whole or in part by the Administrator." 15 U.S.C. § 2608(b) (emphases added). While EPA is supposed to coordinate the "actions" under each statute, this provision does not contemplate EPA excluding exposures from the analyses prepared under TSCA. Indeed, the remaining language of TSCA § 9(b) highlights that

Congress intended for EPA to prepare risk evaluations analyzing all exposures, including those that might be addressed under another authority.

Under TSCA § 9(b)(1), EPA can only choose to rely on other authorities "[i]f [EPA] determines that a risk to health or the environment associated with a chemical substance or mixture *could be eliminated or reduced to a sufficient extent* by actions taken under the authorities contained in such other Federal laws." 15 U.S.C. § 2608(b)(1) (emphasis added). Thus, Congress provided a standard that EPA must meet before relying on other authorities: with respect to the "risk to health or the environment" presented by a chemical, the other authority must either "eliminate[]" that risk or "reduce [the risk] to a sufficient extent." *Id.* Reduction in risk must be "sufficient" as defined by TSCA, and the word "extent" cross-references the basic standard set forth in section 6(a). *See* 15 U.S.C. § 2605(a). Section 6(a) provides that if EPA determines that a substance or mixture "presents an unreasonable risk of injury to health or the environment," EPA "shall" apply requirements to the "substance or mixture to the extent necessary so that the chemical substance or mixture no longer presents such risk." *Id.* Thus, EPA may only rely on actions under another statute if those actions will reduce an identified risk "to the extent necessary so that [it] no longer presents [an unreasonable risk of injury to health or the environment]." EPA cannot assume that other statutes, with different standards, meet the requirements of TSCA.

TSCA requires that EPA eliminate the "unreasonable risk," *id.* and that unreasonable risk of injury to health or the environment must be identified under TSCA § 6(b)(4)(A) "without consideration of costs or other nonrisk factors, including an unreasonable risk to a potentially exposed or susceptible subpopulation identified as relevant to the risk evaluation by the Administrator." 15 U.S.C. § 2605(b)(4)(A). Thus, TSCA's standard requires EPA to resolve risks identified without consideration of costs or other nonrisk factors, and EPA must specifically consider risks to vulnerable subpopulations. Generally speaking, the other EPA-administered statutes do not have this same standard. Some of these statutes allow consideration of nonrisk factors and do not explicitly require consideration of vulnerable subpopulations. EPA cannot simply assume that regulatory efforts that meet the requirements of those statutes will also meet TSCA's requirement that EPA eliminate unreasonable risks. And Congress's decision to enact the TSCA standard reflects that Congress wanted EPA, when implementing TSCA, to meet that standard; EPA cannot rely on its fulfillment of a different standard under a different statute to evade that duty.

Under TSCA § 9(b)(2) Congress directed EPA to consider certain factors to resolve overlaps in EPA's statutory jurisdictions after completing the risk evaluation. Specifically, in determining whether to address a risk under TSCA or another statutory authority administered by EPA, EPA "shall consider, based on information reasonably available to the Administrator, all relevant aspects of the risk," among other things. *Id.* § 2608(b)(2). Thus, EPA has to analyze "all relevant aspects of the risk" in its risk evaluations, *before* deciding whether to address particular risks through TSCA or another statutory authority. Congress would not have included this

requirement if Congress had meant for EPA to simply defer to current regulatory approaches to those chemicals at the outset before conducting a risk evaluation.

Among other concerns, if EPA just ignores risks arising from exposures that fall within other statutes' jurisdiction, then EPA will lack the information necessary to prepare the necessary analyses under TSCA § 9(b)(2). TSCA § 9(b) clearly contemplates that EPA will analyze all these exposures in risk evaluations and then meet its duties under TSCA § 9(b) based, in part, on the analyses prepared in the risk evaluations. As reflected in TSCA § 6, Congress expressly chose to separate risk evaluation and risk management into different procedural steps (with risk evaluation preceding risk management), to ensure that EPA provided a robust risk evaluation uncolored by nonrisk factors or other risk management concerns.

Notably, in the draft risk evaluation, EPA makes no showing that its actions under other statutes reduce the risk "to the extent necessary so that [it] no longer presents [an unreasonable risk of injury to health or the environment]," and EPA does not present any actual analysis of "all relevant aspects of the risk" arising from the ignored exposures. So EPA has undisputedly failed to comply with TSCA § 9(b). Given that Congress expressly addressed the issue of overlapping regulatory jurisdictions in TSCA § 9, EPA cannot avoid those procedures by simply ignoring exposures that fall within another statute's jurisdiction.

Furthermore, EPA is expressly required to evaluate exposures from combinations of activities, which it cannot do if it excludes some exposures at the outset that may be able to be addressed under another authority, particularly when any risk management under the other authority would not reduce exposure to zero.

B. EPA's approach to the general population and subpopulations highlights that its decision to exclude exposures under other EPA-administered statutes is illegal and arbitrary and capricious.

i. EPA must analyze whether TCE presents a risk to the general population because the record establishes that the general population is exposed to it.

EPA states that it will not analyze general population exposures to TCE because EPA considers its existing regulatory programs sufficient. (pp. 35, 379; Problem Formulation for TCE at p. 62). EPA's approach is illegal for the reasons given above. In addition, the reasonably available information establishes that the general population experiences significant exposures to TCE, and it is irrational to ignore those exposures in light of this evidence.

In particular, the most recent TRI data for TCE establishes that TCE is released to air, water, and land in significant quantities:

Table A: Toxics Release Inventory Data for TCE, 2018[250]

Chemical Substance	Air (lb)	Water (lb)	Land (lb)	Total (lb)
TCE	1,935,303	20	248,209	2,183,532

Moreover, the Problem Formulation for TCE states that: "Exposure may occur through inhalation, oral and dermal pathways, due to trichloroethylene's widespread presence in a variety of environmental media. Exposures to the general population may occur from industrial and/or commercial uses; industrial releases to air, water or land; and other conditions of use." Problem Formulation for TCE at pp. 10-11. The Problem Formulation details the presence of TCE in various environmental media, including soil and groundwater, ambient and indoor air, and drinking water systems." *Id*. at 33-34.

Given ample evidence that the general population in fact experiences exposures to TCE under EPA's current regulatory regimes, it is arbitrary and capricious for EPA to adopt an approach to risk evaluation that disregards the risks presented to the general population.

ii. EPA cannot accurately evaluate potentially exposed or susceptible subpopulations such as fenceline communities if EPA excludes the vast majority of exposure pathways leading to their greater exposure.

In the problem formulation for TCE, EPA correctly recognized that a potentially exposed or susceptible subpopulation includes those "groups of individuals within the general population who may experience greater exposures due to their proximity to conditions of use that result in releases to the environment and subsequent exposures (e.g., individuals who live or work near manufacturing, processing, use or disposal sites)." *See, e.g*., Problem Formulation for TCE at pp. 38-39. But in the draft risk evaluation EPA ignores the vast majority of pathways that cause these groups to face greater exposures—such as through releases to air, water, and land (pp. 35, 379). EPA provides no rational explanation for how it will accurately and effectively evaluate the actual risk faced by these subpopulations while ignoring these exposures. Moreover, EPA's (correct) recognition that these groups face greater exposure highlights that it is irrational for EPA to ignore the pathways leading to these exposures.

As a result, in the draft risk evaluation, EPA largely fails to analyze this potentially exposed or susceptible subpopulation (p. 352). EPA limits its analysis of greater exposure to workers and

[250] 2018 TRI PRELIMINARY DATASET, https://www.epa.gov/toxics-release-inventory-tri-program/2018-tri-preliminary-dataset (last visited April 7, 2020).

consumers, and EPA largely ignores the greater exposure experienced by individuals living in proximity to conditions of use. As a result, EPA fails to consider an important aspect of the problem because EPA fails to analyze the risks posed to a potentially exposed or susceptible subpopulation that EPA previously acknowledged.

In addition, TSCA specifically requires that EPA protect these subpopulations because they face greater exposure. And, EPA's existing regulations under other statutes, which may not have been developed with a focus on these particular subpopulations, may not always be "sufficient" under the TSCA standard.

C. The listing of TCE as a hazardous air pollutant does not result in zero exposures to it through the air pathway; EPA should analyze the real-world exposures.

EPA excluded exposures to TCE through the air pathway because it is listed as a hazardous air pollutant (HAP) under the Clean Air Act (CAA). Problem Formulation for TCE at p. 54. This approach is unreasonable for the reasons given above, but in addition, EPA has not made the necessary showing that the established HAPs eliminate any unreasonable risk and EPA has not assessed all relevant aspects of the risk. As EPA acknowledges in the problem formulation, the listing as a HAP leads to a technology-based standard for certain stationary sources. *See, e.g.*, Problem Formulation for TCE at p. 54. Such regulations do not necessarily eliminate exposures. Moreover, EPA is relying on "technology-based" standards, but under TSCA § 9, EPA can only rely on another statutory authority if it reduces exposures "to a sufficient extent" under TSCA, 15 U.S.C. § 2608(b)(1), and TSCA specifically requires that EPA eliminate the unreasonable risk, see 15 U.S.C. § 2605(a), without reference to technology. EPA cannot assume that other statutes, with different standards, meet the requirements of TSCA.

i. EPA's Clean Air Act authority is not a comprehensive substitute for TSCA.

EPA's mandate to control toxic air pollutants under the Clean Air Act (CAA) differs from TSCA's provisions applicable to the same substances and thus does not presumptively address the same scope of risks. EPA points to CAA Sections 111 and 112, 42 U.S.C. §§ 7411-12, as an adequate proxy for TSCA regulations that would address the "ambient air pathway" of exposure to toxic air pollutants covered under both statutes, yet the statutory structures that empower EPA to control these pollutants through CAA regulation are different from EPA's authority to regulate or even prohibit the production or use of these substances under TSCA.

CAA Sections 111 and 112 differ in scope and approach as compared to TSCA. EPA points to CAA Section 112 which requires EPA to promulgate regulations applicable to sources of listed hazardous air pollutants including TCE. Section 112 instructs EPA to list and regulate substances for which "emissions, ambient concentrations, bioaccumulation or deposition of the substance are known to cause or may reasonably be anticipated to cause adverse effects to human health or adverse environmental effects." 42 U.S.C. § 7412(b)(2). As EPA acknowledges, under the CAA "For stationary source categories emitting [Hazardous Air

Pollutants] HAP, the CAA requires issuance of technology-based standards and, if necessary, additions or revisions to address developments in practices, processes, and control technologies, and to ensure the standards adequately protect public health and the environment." Problem Formulation for DCM at p. 54. Under section 112(d)(1), EPA sets source-specific "standards for each category or subcategory of major sources and area sources of hazardous air pollutants listed." 42 U.S.C. § 7412(d)(1). This source-specific regulatory scheme requires EPA to:

> require the maximum degree of reduction in emissions of the hazardous air pollutants subject to this section (including a prohibition on such emissions, where achievable) that the Administrator, taking into consideration the cost of achieving such emission reduction, and any non-air quality health and environmental impacts and energy requirements, determines is achievable for new or existing sources in the category or subcategory to which such emission standard applies.

Id. § 7412(d)(2). This approach reflected in section 112 is distinct from TSCA which empowers EPA look at the risk posed by the chemical broadly without necessarily focusing on source-specific technology, costs of regulation, or what standards are "achievable" for each source category. Indeed, as explained previously, TSCA requires that EPA evaluate a chemical's risk "without consideration of costs or other nonrisk factors." 15 U.S.C. § 2605(b)(4)(A). In addition, TSCA requires EPA to consider the "conditions of use" of a chemical, with no distinction drawn between stationary sources and other sources. As a result, EPA cannot presumptively assume that section 112 regulation would necessarily address all the risks that TSCA requires the Agency to identify and ameliorate.

Similarly, EPA points to CAA Section 111, 42 U.S.C. § 7411, as a basis for declining to evaluate risks associated with the ambient air pathway under TSCA. But, like section 112, section 111 differs in material respects from the approach embodied in TSCA. Section 111 requires EPA to set and periodically update standards of performance for categories of new stationary sources and existing stationary sources of pollution that cause or contribute "significantly, to air pollution which may reasonably be anticipated to endanger public health or welfare." 42 U.S.C. § 7411(b). In setting "standard[s] of performance" for each source category or even sub-category of sources, EPA must select a standard that "reflects the degree of emission limitation achievable through the application of the best system of emission reduction which (taking into account the cost of achieving such reduction and any nonair quality health and environmental impact and energy requirements) the Administrator determines has been adequately demonstrated." 42 U.S.C. § 7411(a)(1). TSCA's regime likewise diverges from this approach in its focus on the risks posed by chemical substances and EPA actions that can ameliorate those risks.

In addition to these substantive differences, existing standards under sections 111 and 112 are subject to different procedural requirements. For example, the CAA's source-specific standards under Section 111 are structured around a series of 8-year intervals for review and Section 112's

list of substances is reviewed every 5 years, along with other periodic reviews called for under Section 112. EPA is also subject to a series of consent decrees for required reviews under Section 112(f)(2) and Section 112(d)(6), often setting longer timelines for new rulemaking. As a result, many of the category specific regulations under these provisions are in various stages of being updated. Accordingly, even if there were some substantive alignment between TSCA and the CAA provisions EPA cites—which is not the case, as we describe above—it would be manifestly arbitrary and capricious for the Agency to determine that CAA standards that have not been updated for many years, or even decades, presumptively discharge EPA's present-day responsibility to assess the risks TCE poses under TSCA.

ii. The available evidence establishes that there is exposure through ambient air.

Indeed, the problem formulation itself establishes that exposures through air persist for TCE despite any regulation under the CAA, and it is arbitrary and capricious for EPA to ignore those exposures. For EPA to treat these exposure levels as "zero" when they are known not to be does not comport with the best available science. In the problem formulation for TCE, EPA states that a total of 1,880,569 lbs of the chemical were released to the air in 2015 according to TRI. Problem Formulation for TCE at p. 32. "TCE has been detected in ambient air across the United States,." *Id*. at 33. EPA reports: "A summary of the ambient air monitoring data for TCE (i.e., measured data) in the United States from 1999 to 2006 suggests that TCE levels in ambient air have remained fairly constant in ambient air for the United States since 1999, with an approximate mean value of 0.23 μg/m3." *Id*.

The problem formulation identifies other evidence that TCE contamination in the air is widespread:

- "Degradation by reactants in the atmosphere has a half-life of several days meaning that long range transport is possible." Problem Formulation for TCE at p. 30.
- "Of the nearly 2 million pounds of total disposal or other releases, there were stack and fugitive air releases, water releases, Class I underground injection, releases to Resource Conservation and Recovery Act (RCRA) Subtitle C landfills and other land disposal, and other releases. Of these releases, 96% were released to air." Problem Formulation for TCE at p. 32.
- "EPA expects that exposure via inhalation will be the most significant route of exposure for consumer exposure scenarios." Problem Formulation for TCE at p. 36.
- "Based on TRI data and TCE physical-chemistry and fate properties, it is expected that inhalation represents the primary route of exposure for the general population from ongoing industrial and/or commercial activities." Problem Formulation for TCE at p. 37.

Additional information sources reveal that exposures through ambient air are occurring, and these additional information sources indicate that EPA's current analyses underestimate the

exposure level through this pathway. EPA should not limit its analysis of air emissions to TRI data. EPA should also consider the data available from the National Emissions Inventory (NEI), which tend to reveal significantly greater levels of air emissions of, and thus air pathway exposures to, TCE. EPA cannot reasonably ignore this available information about air emissions and resulting exposures of TCE. As revealed in the chart below, despite the Clean Air Act protections, there are significant annual emissions and thus exposures through the air pathway for TCE.

Table B: National Emissions Inventory Data for TCE

Chemical Substance	2014 NEI (lb)	2017 NEI (lb)
TCE	12,191,695	2,740,336

EPA should analyze these exposures and the risks they present to both human health and the environment, including terrestrial species. With more than 12 million lbs of TCE emitted to the air in 2014, it is absurd to treat the overall exposure through this pathway as if it were "zero."

Moreover, EPA should be collecting and analyzing information about exposure levels through the ambient air pathway, particularly near sites where people may experience greater exposure due to their proximity to conditions of use or contamination sites. By excluding pathways such as the ambient air pathway, EPA will seriously underestimate the levels of exposure.

In addition, EPA cannot adequately assess the risks faced by subpopulations consisting of people experiencing greater exposure due to their proximity to conditions of use without assessing pathways such as the ambient air pathway. If EPA ignores the ambient air pathway, EPA will completely ignore exposure levels at locations across the country. EPA should use its information authorities to obtain additional information about exposure levels experienced by the subpopulations living near conditions of use.

* * * * *

Given evidence of real-world exposure through the air pathway, EPA must evaluate those exposures in its risk evaluations. In particular, EPA needs to consider whether these exposures combine with other sources of exposure in a manner that leads to an unreasonable risk, including to certain subpopulations. EPA cannot rationally exclude these exposures from its analysis.

D. Real-world exposures still occur through drinking water, and EPA cannot ignore those real-world exposures when assessing the risk presented by TCE.

EPA decided to ignore all exposures through drinking water for TCE. (pp. 35, 39). The systematic decision to ignore all exposures through this pathway is arbitrary and capricious

because the available evidence reveals that exposures do occur through this pathway. Analyzing exposure through drinking water is also particularly important for EPA to obtain an accurate estimate of the exposure of infants and children, often a potentially exposed or susceptible subpopulation.[251]

The existence of a Maximum Contaminant Level does not result in zero exposures to TCE through drinking water; EPA should analyze the real-world exposures. In the draft risk evaluation, EPA did not analyze exposures to TCE through drinking water, and in the problem formulation, EPA justified this exclusion because EPA has set an enforceable Maximum Contaminant Level (MCL) under the Safe Drinking Water Act (SDWA). Problem Formulation for TCE at p. 54.

This approach is unreasonable for the reasons given above, but in addition, EPA has not made the necessary showing that the established MCL of 0.005 mg/L eliminates any unreasonable risk and EPA has not assessed all relevant aspects of the risk. As EPA itself acknowledges in the problem formulation, the MCL is only set at the level "feasible" which "refers to both the ability to treat water to meet the MCL and the ability to monitor water quality at the MCL." *See, e.g.*, Problem Formulation for TCE at p. 54. Thus, the MCL is based on non-risk factors and does not necessarily eliminate exposures.

Specifically, the contaminant level set under the SDWA considers "non-risk" factors, and the MCL is not sufficient to eliminate risks. While EPA must set a maximum contaminant level goal (MCLG) that is fully protective of health for drinking water contaminants, 42 U.S.C. § 300g-1(b)(1)(E); *see also* 42 U.S.C. § 300g-1(b)(4)(A), the MCLG is not the national drinking water standard. Rather, the Agency must establish a maximum contaminant level (MCL) that is as close to the MCLG "as is feasible," considering technological limitations and costs, and promulgate a national primary drinking water regulation (NPDWR) for the contaminant based on the MCL. 42 U.S.C. § 300g-1(b)(4)(B). In other words, the contaminant level EPA actually sets for safe drinking water is less protective than the MCLG because it accounts for feasibility and costs, which are non-risk factors that EPA may not consider during the risk evaluation process.

Notably, the MCLG for TCE is zero, indicating that in order to avoid adverse effects on human health from drinking water EPA believes that TCE should not be in drinking water at any level. Because the MCL for TCE is higher, EPA must, among other things, address in the risk evaluation the risks posed by ongoing exposure to TCE at levels in drinking water below the MCL.

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[251] *See, e.g.*, U.S. EPA, Problem Formulation of the Risk Evaluation for Perchloroethylene (Ethene, 1,1,2,2-Tetrachloro) (May 2018), p.48 https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0732-0080 ("Drinking water could be a significant source of perchloroethylene ingestion exposure for children, who drink roughly four times as much water as adults.").

In addition, the SDWA does not regulate all sources of drinking water. It is estimated that more than 13 million households rely on private wells for drinking water in the United States.[252,253] The national drinking water standards established under the SDWA do not apply to private wells. *See* 42 U.S.C. § 300f(1) (a "primary drinking water regulation" only applies to "public water systems"); 42 U.S.C. § 300f(4)(A) (a "public water system" is a system that "has at least fifteen service connections or regularly serves at least twenty-five individuals"). Therefore, exposures to TCE in drinking water from private wells is not addressed by the SDWA and need to be evaluated in the risk evaluation.

Moreover, the problem formulation itself establishes that exposures through drinking water persist for TCE despite any regulations under the SDWA, and it is arbitrary and capricious for EPA to ignore those exposures. For EPA to treat these exposure levels as "zero" when they are known not to be does not comport with the best available science. In the problem formulation, EPA reported that TCE has been detected in ground water and surface water used for drinking water. Problem Formulation for TCE at p. 34. "TCE is one of the most frequently detected organic solvents in U.S. ground water. … Between 1985 and 2001, the detection frequency of TCE was 2.6%, with a median concentration of 0.15 µg/m3. … TCE has been detected in drinking water systems through national and state-wide monitoring efforts.." *Id*. The problem formulation also states that: "The general population may ingest TCE via contaminated drinking water and other ingested media. It is anticipated that ingestion of drinking water containing TCE, for on-going TSCA uses, represents the primary route of oral exposure for this chemical. TCE has been detected in national-scale drinking water monitoring datasets (i.e., EPA's Six-Year Review 3) and is released to surface water from ongoing TSCA uses and activities. The primary oral exposure route for TCE is expected to be via drinking water." *Id.* at p. 38.

Given evidence of real-world exposure, EPA must assess those exposures in its risk evaluation. EPA cannot rationally exclude them from analysis.

E. Real-world exposures still occur through ambient water, and EPA cannot ignore those real-world exposures when assessing the risk to human health presented by TCE.

EPA decided to effectively ignore all risks to human health arising from exposures through ambient water for TCE. The systematic decision to ignore the exposures through this pathway is arbitrary and capricious because the available evidence reveals that exposures do occur through this pathway. In the problem formulation, when discussing its approach to assessing risk to

[252] PRIVATE DRINKING WATER WELLS, https://www.epa.gov/privatewells (last visited Jul. 31, 2018) (citing the US Census American Housing Survey 2015).

[253] An estimated 44.5 million people in the United States, or 14 percent of the population, provided their own water for domestic use in 2010. U.S. Geological Survey, *Estimated Use of Water in the United States in 2010* (2014), https://pubs.usgs.gov/circ/1405/pdf/circ1405.pdf.

human health, EPA states it will exclude exposures to TCE through ambient water because, under the Clean Water Act (CWA), EPA has recommended water quality criteria for protection of human health which are available for adoption into state water quality standards and to permitting authorities. Problem Formulation for TCE at p. 55. But the existence of a recommended water quality criterion for human health does not result in zero exposures to TCE through ambient water; EPA should analyze the real-world exposures.

EPA's approach is unreasonable for the reasons give above, but in addition, EPA has not made the necessary showing that the recommended water quality criteria it has set eliminate any unreasonable risk and EPA has not assessed all relevant aspects of the risk. Indeed, EPA has not even established or shown that these recommended water quality criteria meet EPA's illegal standard that these criteria "adequately assess and effectively manage exposures."

i. EPA has not addressed several reasons that its Clean Water Act authority is not a comprehensive substitute for action under TSCA.

Under the Clean Water Act (CWA), EPA establishes recommended water quality criteria, but not all states have updated their criteria to reflect the current CWA criteria. *See* 80 Fed. Reg. 36,986 (June 29, 2015). There is often significant variation between EPA's recommended criteria (shown in the table below) and the criteria adopted by the states.

Table C: EPA's National Recommended Water Quality Criteria for TCE:[254]

Chemical Name	Human Health Criterion for w+o (µg/L)	Human Health Criterion for o (µg/L)
Trichloroethylene	0.6	7

For example, Maryland has set its human health criteria for TCE at higher levels than the current EPA recommended water quality criteria.[255] Other examples of states adopting less stringent standards are available. Given that some states have water quality criteria that are significantly

[254] There are two sets of human health criteria: (1) exposure through organisms only (o), and (2) exposure to water and organisms (w+o). NATIONAL RECOMMENDED WATER QUALITY CRITERIA - HUMAN HEALTH CRITERIA TABLE, https://www.epa.gov/wqc/national-recommended-water-quality-criteria-human-health-criteria-table.

[255] NUMERICAL CRITERIA FOR TOXIC SUBSTANCES IN SURFACE WATERS, http://www.dsd.state.md.us/comar/comarhtml/26/26.08.02.03-2.htm (last visited Apr. 8, 2020).

less protective than EPA's recommendations, EPA cannot rely on its recommendations to assume that the risks are adequately managed, much less that they result in zero exposure.

EPA has also not assessed whether the established criteria, which EPA set and were adopted to varying extents by states in the past, reflect the current best available science regarding the risk presented by TCE. For example, EPA acknowledges that EPA may need to update its water quality criteria for TCE. Problem Formulation for TCE at p. 55.

Moreover, while EPA relies on the CWA to dismiss the ambient water pathway for human health, EPA never acknowledges the ongoing uncertainty surrounding the definition of "waters of the United States"[256] regulated under the CWA. EPA itself has stated that since the Supreme Court's decision in *Rapanos v. United States*, 547 U.S. 715 (2006), there has been uncertainty regarding the regulatory reach of the CWA. The EPA Office of Inspector General has stated that "*Rapanos* has created a lot of uncertainty with regards to EPA's compliance and enforcement activities. Processing enforcement cases where there is a jurisdictional issue has become very difficult."[257] EPA cannot assume that all ambient water is adequately managed under the CWA when EPA itself expresses ongoing uncertainty over the jurisdictional reach of the CWA.

Indeed, EPA has asserted that *Solid Waste Agency of Northern Cook County v. Army Corps of Engineers*, 531 U.S. 159 (2001), "squarely eliminate[d] CWA jurisdiction over isolated waters that are intrastate and non-navigable, where the sole basis for asserting CWA jurisdiction is the actual or potential use of the waters as habitat for migratory birds that cross state lines in their migrations." Advance Notice of Proposed Rulemaking on the Clean Water Act Regulatory Definition of "Waters of the United States," 68 Fed. Reg. 1991, 1996 (Jan. 15, 2003). Therefore, it makes even less sense that EPA would assume that the CWA will ensure that all ambient waters are adequately managed.

Furthermore, EPA cannot assume that the CWA has adequately managed the discharge of all TCE because there are recognized lapses in the regulatory process. EPA's Office of Inspector General has reported that:

> Management controls put in place by the EPA to regulate and control hazardous chemical discharges from sewage treatment plants to water resources have limited effectiveness. The EPA regulates hazardous chemical discharges to and from sewage treatment plants, but these regulations are not effective in controlling the

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[256] EPA's main webpage summarizes the ongoing litigation regarding the 2015 regulation that finalized a definition of "waters of the United States." *See* ABOUT WATERS OF THE UNITED STATES, https://www.epa.gov/wotus-rule/about-waters-united-states (last visited Aug. 11, 2018).

[257] U.S. EPA, Office of Inspector General, *Congressionally Requested Report on Comments Related to Effects of Jurisdictional Uncertainty on Clean water Act Implementation* (Apr. 2009), https://www.epa.gov/sites/production/files/2015-11/documents/20090430-09-n-0149.pdf.

> discharge of hundreds of hazardous chemicals to surface waters such as lakes and streams. Sewage treatment plant staff do not monitor for hazardous chemicals discharged by industrial users.[258]

At the time of the report by the Inspector General, there was no database of the information submitted by dischargers, nor was a compilation of the information available to officials in the regions or states that were interviewed.

Considering the documented lack of awareness regarding chemical discharges into and out of wastewater treatment plants, and EPA's own acknowledged failure to regulate discharges through this pathway, EPA should commit to analyzing any exposures through this pathway in its risk evaluations.

In sum, EPA has failed to analyze numerous aspects of its exercise of its CWA authority that amply demonstrate that EPA cannot dismiss the entire ambient water pathway simply because EPA has established water quality criteria. EPA must analyze the ambient water pathway in the risk evaluations.

ii. The problem formulation and draft risk evaluation contain information establishing that there is exposure through ambient water.

In any event, the recommended water quality criteria clearly do not eliminate exposures. In the problem formulation, EPA acknowledges that TCE is detected in surface water. Problem Formulation for TCE at p. 34. In the draft risk evaluation, EPA describes monitoring data and published literature showing that TCE is present in surface water. (pp. 71-72, 88-90). EPA's own modeling shows that TCE is present in surface water at significant concentrations. (pp. 74, 84-88). EPA cannot assume that TCE has nonexistent exposure through ambient water when the data show it is present.

EPA should look to the real-world exposures to TCE to assess its risk. The draft risk evaluation presents some of the monitoring results for TCE in surface water, but it then fails to consider how these exposures might impact human health. EPA should examine and summarize that exposure information when evaluating the risks presented by TCE; if that information is insufficient, EPA should use its authorities to require the development of additional needed information.

[258] U.S. EPA, Office of Inspector General, *More Action is Needed to Protect Water Resources from Unmonitored Hazardous Chemicals* at 3 (Sept. 2014), https://www.epa.gov/sites/production/files/2015-09/documents/20140929-14-p-0363.pdf.

F. Real-world exposures still occur through disposal pathways, and EPA cannot ignore those real-world exposures when assessing the risk presented by TCE.

In the problem formulation for TCE, EPA contends that due to regulation of disposal under the Resource Conservation and Recovery Act (RCRA), the Clean Air Act (CAA), the Safe Drinking Water Act (SDWA), and various state programs, EPA can ignore all exposures from all disposal-related pathways and associated activities (e.g., collection, processing, storage and transport). Problem Formulation for TCE at pp. 55-57. Similarly, the draft risk evaluation excludes exposures through various media pathways on the theory that they are adequately managed under these statutes (pp. 35, 379). While the draft risk evaluation does analyze "Process Solvent Recycling and Worker Handling of Wastes," EPA limited that analysis to the exposures of workers and ONUs. (pp. 51-52, 320). EPA also excluded from the draft risk evaluation all ambient air, land disposal, and waste incineration pathways and their potential exposures to the general population (pp. 35, 379).

This approach is unreasonable for the reasons given above. EPA has not made the necessary showing that these regulations eliminate any unreasonable risk and EPA has not assessed all relevant aspects of the risk. Indeed, EPA has not even established or shown that these disposal regulations meet EPA's illegal standard that these regulations "adequately assess and effectively manage exposures." For example, EPA has not shown or established that disposal in a RCRA Subtitle C hazardous waste landfill or a RCRA Subtitle D non-hazardous waste landfill would actually reduce unreasonable risk to a sufficient extent. EPA's approach is also arbitrary and capricious for a variety of reasons.

While EPA invokes the standards for RCRA Subtitle C landfills as providing sufficient protection, not all disposal occurs in such landfills. For example, EPA acknowledges that in 2015 TCE was disposed in "land disposal" facilities other than RCRA Subtitle C landfills. Problem Formulation for DCM at p. 32. While the amount not disposed in Subtitle C landfills that year was quite small, EPA stated:

> While most TCE going to land disposal went to Subtitle C Hazardous Waste Landfills in 2015, in past years, the TRI data show TCE going to other types of land disposal as well. In 2014, 12,600 pounds was transferred for off-site land treatment, and in both 2013 and 2014 over 11,000 pounds were transferred to off-site landfills other than RCRA subtitle C landfills. From 2012 through 2014, 24,000 pounds to over 100,000 pounds of TCE were released on-site to other land disposal. (p. 32).

EPA cannot rely on regulations that do not apply to protect against risks.

Even for those chemicals regulated under RCRA, EPA acknowledges that disposal also occurs in Subtitle D municipal solid waste (MSW) landfills and industrial-non-hazardous and construction/demolition waste landfills (which are primarily regulated under state regulatory

programs). These disposal approaches do not need to meet the requirements of Subtitle C landfills, thus EPA's invocation of the Subtitle C standards does not justify ignoring exposures from these disposals. While the purpose of RCRA subtitle C is at least to "protect human health and the environment," *see, e.g.*, 42 U.S.C. §§ 6922(a), 6924(a), subtitle D is intended "to assist in developing and encouraging methods for the disposal of solid waste which are environmentally sound and which maximize the utilization of valuable resources including energy and materials *** and to encourage resource conservation." 42 U.S.C. § 6941. Therefore, EPA's exclusions based on the regulations under subtitle D potentially raise even greater, unaddressed, public health concerns than EPA's exclusions under subtitle C.

In addition, states impose varying requirements on such landfills under their delegated RCRA Subtitle D authorities. For example, EPA indicates that some state programs may not include requirements for liners to limit release of landfill leachate.

EPA itself has acknowledged that enforcement and regulation under RCRA is inconsistent, so EPA cannot simply assume that RCRA implementation provides a basis for ignoring exposures under TSCA. As the Office of Inspector General explained the challenges of the RCRA system:

> The Hazardous and Solid Waste Amendments of 1984 (HSWA) amended RCRA and added provisions including land disposal restrictions, RCRA corrective action for solid waste management units and regulation of small-quantity generators. When the EPA creates new hazardous waste rules, it does so under the authority of either or both of these laws. Rules promulgated under HSWA authority are immediately effective in all states and are administered by the EPA until states become authorized for those rules. In contrast, *rules promulgated under RCRA authority (non-HSWA rules) cannot be enforced by the EPA in states with an authorized base program and do not go into effect until these states become authorized for the rules*.[259]

According to the OIG, the fact that a number of rules are not yet adopted by the states and cannot be enforced by EPA "creates a regulatory gap and risk to human health and the environment, and an inconsistent regulatory landscape across the states."[260] OIG's report states that "there are almost 1,300 instances of required rules for which various state hazardous waste programs have not been authorized. Of the rules for which states have not received authorization, there are

[259] U.S. EPA, Office of Inspector General, *Incomplete Oversight of State Hazardous Waste Rule Authorization Creates Regulatory Gaps and Human Health and Environmental Risks* at 2 (Jul. 2018), https://www.epa.gov/sites/production/files/2018-07/documents/_epaoig_20180731-18-p-0227.pdf (emphasis added).
[260] *Id.* at 11.

about 500 each of HSWA and non-HSWA rules, and about 300 rules that have components of both."[261]

When states do not keep their hazardous waste programs up to date, it means citizens in different states are unevenly protected from hazardous waste-related risks. This is critical because "60,000 RCRA facilities exist in the United States, generating and managing 30 to 40 million tons of hazardous waste annually. Eighty percent of all U.S. citizens live within a 3-mile radius of a RCRA-regulated hazardous waste generator or treatment storage and disposal facility, and 50 percent of citizens live within a 1-mile radius."[262] Therefore, EPA cannot rely on any assumption of consistent implementation and enforcement of RCRA to ensure that all exposures have been adequately managed.

EPA cannot assume that exposure from disposal is zero just because it could be regulated under other authorities. EPA appears to have made no significant effort to identify data on the amounts of TCE in soil or sediment, available for example in EPA's STORET database, which it used to obtain surface water data on TCE. This is despite the fact that EPA does mention in passing that "[l]imited sediment monitoring data for TCE that are available suggest that TCE is present in sediments." (p. 275) In contrast, EPA did conduct such searches and located substantial amounts of data for another chemical undergoing risk evaluation. The problem formulation for methylene chloride acknowledges that various studies and databases provide hundreds of measurements of methylene chloride in soil and sediment. Problem Formulation for DCM at p. 36. EPA also notes in that problem formulation: "In a literature review of various VOC concentrations found in landfill leachates, Klett et al. (2005) found methylene chloride ranged in concentration from 1.0 – 58,200 µg/L. Staples et al. (1985) reported that methylene chloride was found in 20% of sediment samples in the STORET database." *Id*. at 36-7. There is every reason to believe that analogous data for TCE would have been located had EPA conducted the same kinds of searches it did for methylene chloride.

Exposures from disposal no doubt persist for TCE despite RCRA regulations, and it is arbitrary and capricious for EPA to ignore those exposures. For EPA to treat these exposure levels as "zero" when they exist does not comport with the best available science.

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[261] *Id*. at 12; *see also* AUTHORIZATION STATUS BY RULE, https://www.epa.gov/sites/production/files/2018-06/documents/authorization_status_by_rule.pdf (last visited Aug. 10, 2018) (documenting for each state whether they have adopted the RCRA regulations).

[262] U.S. EPA, Office of Inspector General, *EPA Has Not Met Statutory Requirements for Hazardous Waste Treatment, Storage and Disposal Facility Inspections, but Inspection Rates Are High* at 1 (March 2016), https://www.epa.gov/sites/production/files/2016-03/documents/20160311-16-p-0104.pdf.

To be sure, EPA often appears to have less monitoring information that speaks to whether a particular exposure arises from disposal or some other source, and, based on what it found for methylene chloride, EPA likely has less monitoring information about TCE's presence in soil, sediment, and leachate than it does for its presence in water or air. As EDF has previously explained, EPA must consider "reasonably available" information, and thus EPA must both consider the information it already possesses and use its authorities under TSCA §§ 4 and 8 to obtain additional information. EDF incorporates and reiterates those points here as well.[263] EPA should use those authorities to obtain additional information about the exposures arising from disposal for TCE.

G. EPA should obtain some actual monitoring data to confirm its biosolids predictions for TCE, and to the extent EPA excludes biosolids on the theory that the chemical will instead enter other pathways, EPA must consider those exposure pathways.

In the problem formulation for TCE, EPA states that the chemical is expected to either enter the aqueous component and/or volatilize to air, and thus EPA asserts it can ignore the biosolids exposure pathway. *See, e.g.*, Problem Formulation for TCE at p. 53. In the draft risk evaluation, EPA stated that: "The conclusions of the problem formulation were that no further analysis is necessary in the risk evaluation for sediment, soil and land-applied biosolid pathways leading to exposure to terrestrial and aquatic organisms and for water pathways leading to exposure to terrestrial organisms. Further analysis was not conducted for biosolid, soil and sediment pathways, and for water pathways of exposure to terrestrial organisms, based on a qualitative assessment of the physical-chemical properties and fate of TCE in the environment and a quantitative comparison of hazards and exposures for aquatic and terrestrial organisms." (p. 39). EPA did not conduct a significant analysis of biosolids in the draft risk evaluation; EPA instead dismissed this pathway on the basis of physical-chemical and fate properties of TCE.

EPA should obtain some monitoring data to confirm these analyses, but in any event, EPA cannot rationalize ignoring exposures from biosolids on the basis that TCE will enter the water and air and then also choose to ignore the exposure pathways through water and air. For example, in the problem formulation, EPA stated that: "Any TCE present in the water portion of biosolids following wastewater treatment and land application would be expected to rapidly volatilize into air. Furthermore, TCE is not anticipated to remain in soil, as it is expected to either volatilize into air or migrate through soil into groundwater." Problem Formulation for TCE at p. 53. EPA's justification for ignoring the biosolids pathways for TCE highlights that EPA's decision to ignore other pathways is particularly arbitrary and capricious.

[263] EDF Comments on Ten Scopes under the Toxic Substances Control Act pp.11-15, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0742-0072.

H. EPA must analyze all the environmental risks presented by TCE through ambient water.

In both the problem formulation and the draft risk evaluation, EPA recognizes that it must evaluate the risks to aquatic species arising from exposures through water for TCE. Problem Formulation for TCE at p. 53. But in the draft risk evaluation, EPA did not analyze the risks to terrestrial or sediment-dwelling species from exposure through ambient water for TCE, despite the fact that terrestrial and sediment-dwelling species also can experience exposures through surface water. (p. 29). When EPA evaluates the risks presented by exposure through ambient water, EPA must consider the risks presented to terrestrial and sediment-dwelling ecological receptors as well as aquatic species.

EPA provides no convincing explanation for excluding exposures to terrestrial or sediment-dwelling organisms for TCE. In the problem formulation for TCE, EPA stated that, despite the fact that its "[r]eview of hazard data for terrestrial organisms shows that there is likely to be hazard," it will not further analyze exposure to terrestrial or sediment-dwelling organisms through water, sediment, or migration from biosolids via soil deposition, based on TCE's physical-chemical properties. Problem Formulation for TCE, Appendix E, p. 202. In the draft risk evaluation, EPA states that: "Review of hazard data for terrestrial organisms shows potential hazard; however, physical-chemical properties do not support an exposure pathway through water and soil pathways to terrestrial organisms." (p. 275). Therefore, once again EPA dismisses a potential risk on the basis of physical-chemical properties, but EPA does not present or analyze data confirming this analysis. EPA also again dismisses most of these risks by arguing that TCE will volatilize to air, but EPA then dismisses the air pathway by relying on the CAA. (p. 276). Given EPA's repeated finding that TCE will be present in the air pathway, EPA should analyze the real-world exposures through that pathway.

I. EPA cannot rely on its actions under other authorities when there are numerous problems with compliance, implementation, and enforcement under those authorities.

EPA cannot ignore exposure through these pathways for the reasons given above, but in addition, it is arbitrary and capricious for EPA to assume zero exposure through other pathways based on EPA-administered statutes when EPA has documented extensive problems with compliance, implementation, and enforcement of these statutes.

i. EPA's own analyses establish that State enforcement of these environmental statutes is inconsistent and often deficient.

There are multiple EPA reports documenting enforcement problems with EPA's environmental statutes.[264] Specifically, these reports have noted that "data quality, identification of violations, issuing enforcement penalties and other enforcement actions in a timely and appropriate manner, and general oversight issues" are all key issues impacting the enforcement of these statutes.[265]

Generally, EPA's regional offices provide oversight to ensure that the state enforcement programs are following EPA's guidance, policies, and regulations.[266] Despite EPA oversight, which is a separate concern, state enforcement of these statutes has been found deficient in a number of cases. For instance:

- According to a 2011 OIG report, **North Dakota** appears "philosophically opposed to taking enforcement action."[267] For instance, during the entire period of the report (FYs 2003-2009), the state assessed no penalties against known CWA violators.[268]
- In **Louisiana** multiple petitions have been filed by citizens to remove the state's delegated authorities under the CWA, CAA, and RCRA.[269] The poor performance under these statues was attributed to "a lack of resources, natural disasters, and a culture in which the state agency is *expected to protect industry*."[270]
- The **U.S Virgin Islands** "has not met program requirements for numerous activities related to implementing the Clean Air Act, Clean Water Act, Safe Drinking Water Act, and Underground Storage Tank/Leaking Underground Storage Tank programs. These

[264] U.S. EPA, Office of Inspector General, *EPA Must Improve Oversight of State Enforcement* at App. B, p. 32-34 (Dec. 2011), https://www.epa.gov/sites/production/files/2015-10/documents/20111209-12-p-0113.pdf (identifying a long list of GAO and OIG reports documenting deficiencies in enforcement of environmental statutes).
[265] *Id.* at 32.
[266] U.S. Government Accountability Office, *EPA-State Enforcement Partnership Has Improved, but EPA's Oversight Needs Further Enhancement* at 1 (Jul. 2007), https://www.gao.gov/products/GAO-07-883.
[267] U.S. EPA, Office of Inspector General, *EPA Must Improve Oversight of State Enforcement* at 17 (Dec. 2011), https://www.epa.gov/sites/production/files/2015-10/documents/20111209-12-p-0113.pdf.
[268] *Id.* at 15.
[269] *Id.* at 16.
[270] *Id.* (emphasis added).

activities included monitoring environmental conditions, conducting compliance inspections and enforcing program requirements."[271]

Notably, even where enforcement of these statutes has been consistently deficient, EPA has generally not de-authorized states. According to the 2011 OIG report, "the threat of EPA revoking a state's authorization [is] moot because there is a general understanding that no EPA region has the resources to operate a state program. This reality undercuts EPA's strongest tool for ensuring that authorized states adequately enforce environmental laws: de-authorization."[272] Although EPA has taken steps in a number of cases to improve state programs, ultimately implementation and enforcement of these statutes remains deficient in a number of states, resulting in continued excessive exposure to these chemicals through air, water, and land. These exposures must be assessed under TSCA.

Below are a few more specific examples, among many, of deficiencies under each of the statutes.

Safe Drinking Water Act: As explained above, EPA has excluded exposures to drinking water for TCE based on the assumed effectiveness of state implementation and enforcement of the SDWA. A 2011 GAO report states that EPA often receives unreliable data from the states.[273] EPA relies on state data to determine whether there is compliance with the SDWA. Without reliable data EPA has no way to verify that the requirements of the SDWA are being met by the states.

Here is one example of deficient state enforcement of the SDWA:

- **Pennsylvania**: EPA sent a letter in December 2016 to the Pennsylvania Department of Environmental Protection, stating that the department lacks the necessary staff to enforce safe drinking water standards and that the lack of staff has caused the number of

[271] U.S. EPA, Office of Inspector General, *Conditions in the U.S. Virgin Islands Warrant EPA Withdrawing Approval and Taking Over Management of Some Environmental Programs and Improving Oversight of Others* (April 2015), https://www.epa.gov/sites/production/files/2015-09/documents/20150417-15-p-0137.pdf; U.S. EPA Region 2, *National Strategy Oversight Plan* at 3 (Mar. 2016), https://www.documentcloud.org/documents/2992740-Region-2-State-Oversight-Plan-March-2016-v2.html.

[272] U.S. EPA, Office of Inspector General, *EPA Must Improve Oversight of State Enforcement* at 17 (Dec. 2011), https://www.epa.gov/sites/production/files/2015-10/documents/20111209-12-p-0113.pdf.

[273] U.S. Government Accountability Office, *Unreliable State Data Limit EPA's Ability to Target Enforcement Priorities and Communicate Water Systems' Performance* (June 2011), https://www.gao.gov/products/GAO-11-381.

unaddressed Safe Drinking Water Act violations to nearly double in the past five years, from 4,298 to 7,922.[274]

Clean Water Act: EPA has also excluded exposures to ambient water for TCE based on the assumed "effectiveness" of the CWA's National Pollution Discharge Elimination System (NPDES) program and the water quality criteria process.

But over half of assessed U.S. river and stream miles violate state water quality standards.[275] EPA's own analysis, provided below, indicates that waters remained impaired throughout the United States, despite the CWA standards.

Table D: Assessed Water of the United States[276]

	Size of Water							
	Rivers and Streams (Miles)	Lakes, Reservoirs, and Ponds (Acres)	Bays and Estuaries (Square Miles)	Coastal Shoreline (Miles)	Ocean and Near Coastal (Square Miles)	Wetlands (Acres)	Great Lakes Shoreline (Miles)	Great Lakes Open Water (Square Miles)
Good Waters	516,800	5,392,817	11,516	1,285	617	569,328	106	1
Threatened Waters	4,495	30,309						
Impaired Waters	586,910	13,158,111	44,619	3,330	6,218	665,979	4,354	39,230
Total Assessed Waters	1,108,205	18,581,237	56,135	4,615	6,836	1,235,307	4,460	39,231
Total Waters	3,533,205	41,666,049	87,791	58,618	54,120	107,700,000	5,202	196,343
Percent of Waters Assessed	31.4	44.6	63.9	7.9	12.6	1.1	85.7	20.0

EPA also publishes the Annual Noncompliance Report, which summarizes enforcement data for facilities with individual NPDES permits but that are not major dischargers.[277] According to the 2015 report, the percentage of facilities with formal enforcement actions compared to facilities with violations was merely 8.9% in 2015.[278] Below are a few examples of enforcement deficiencies:

- **Tennessee**: The Tennessee Department of Environment and Conservation neglected to timely penalize permit holders despite months of noncompliance, failed to assess

[274] Letter from Jon M. Capacasa, Director, EPA Region III Water Protection Division, to Lisa D. Daniels Director, Pa. Dep't of Envtl. Prot. Bureau of Safe Drinking Water (Dec. 30, 2016), https://drive.google.com/file/d/0B4Y3VQLxjkxObjZ0ZXlSVDZvRWc/view.
[275] NATIONAL SUMMARY OF STATE INFORMATION, https://ofmpub.epa.gov/waters10/attains_nation_cy.control (last visited Jul. 31, 2018).
[276] *Id.*
[277] U.S. EPA, Office of Enforcement and Compliance Assurance, *Annual Noncompliance Report (ANCR) Calendar Year 2015* (Aug. 2016), https://echo.epa.gov/system/files/2015_ANCR.pdf.
[278] *Id*. at 7.

appropriate fines, and did not report significant discharge violations from major facilities.[279]

- **Alaska**: EPA regional directors told OIG that "when the region authorized the state to run the program, both the region and OECA officials were aware that the state lacked the capacity to be successful."[280] EPA's State Review Framework for Alaska revealed that, among other serious concerns, the state does not consistently take timely or appropriate enforcement actions, inspect permitted facilities anywhere close to state goals.[281]
- **Louisiana**: Louisiana reviewed the compliance status for less than 50% of individually permitted non-major NPDES permittees from 2010-2015.[282]

Clean Air Act: State performance also varies widely under the CAA. In 2011, the Office of the Inspector General examined the percentage of facilities inspected, the percentage of significant noncompliance or high priority violations identified per inspection, and the percentage of final actions with penalties for fiscal years 2003-2009 and found that performance varied significantly across the country, in this case "by almost 50 percentage points."[283] Below are a few specific examples of insufficient state enforcement of the CAA:

- **Florida**: The Florida Department of Environmental Protection opened only 18 air enforcement cases in 2015, compared to a previous annual average of 93.[284] Additionally, from 2013 to 2015 the state only filed one asbestos case, compared to a past annual average of 13.[285]
- **North Carolina**: "CAA metric for assessed penalties dropped by 93% statewide from about $235.000 in FY II to just under $17,000 in FY 14. During the same period the

[279] U.S. EPA Region 4, *State Review Framework Tennessee* at 28-35 (Sept. 2016), http://www.documentcloud.org/documents/3173730-TN-Final-SRF-Report-9-29-16.html.
[280] U.S. EPA, Office of Inspector, *EPA Must Improve Oversight of State Enforcement* at 16 (Dec. 2011), https://www.epa.gov/office-inspector-general/report-epa-must-improve-oversight-state-enforcement.
[281] U.S. EPA Region 10, *State Review Framework Alaska* at exec. summary (Dec. 2014), https://www.epa.gov/sites/production/files/2015-01/documents/srf-rd3-rev-ak.pdf.
[282] U.S. EPA, Office of Enforcement and Compliance Assurance, *Annual Noncompliance Report (ANCR) Calendar Year 2015* at 8 (Aug. 2016), https://echo.epa.gov/system/files/2015_ANCR.pdf.
[283] U.S. EPA, Office of Inspector, *EPA Must Improve Oversight of State Enforcement* at 10 (Dec. 2011), https://www.epa.gov/office-inspector-general/report-epa-must-improve-oversight-state-enforcement.
[284] Public Employees for Environmental Responsibility, *Report on Enforcement Efforts by the Florida Department of Environmental Protection* at 23 (Aug. 2016), https://www.peer.org/assets/docs/fl/8_18_16_DEP_Report_on_2015_Enforcement.pdf.
[285] *Id.*

number of facilities with informal and formal enforcement actions also dropped dramatically (52% and 79%, respectively)."[286]

- **Ohio**: The Region found that a number of High Priority Violations (HPV) are being resolved by the state through a permit modification/revision. EPA believes that HPV cases should be resolved through a formal enforcement action per the HPV policy, and the state disagrees.[287]

Resource Conservation and Recovery Act: As with the other statutes upon which EPA relies to avoid analyzing exposure pathways, there are serious state enforcement problems with RCRA. For example, Mississippi has not accurately identified and documented RCRA violations.[288] Additionally, despite EPA guidance that states civil penalties should recoup at least the economic benefit the violator gained through noncompliance, the state does not routinely document or consider the economic benefit.[289]

ii. Reduced EPA enforcement provides even less assurance that exposures through the excluded pathways are being effectively managed.

Under the current Administration, enforcement of these environmental statutes has been significantly curbed. For instance, management at EPA has directed EPA investigators to seek authorization before asking companies to conduct testing or sampling under the CAA, RCRA, or the CWA.[290] The memo also states that investigators need authorization if they do not have information specific to a company that it may have violated the law, or if state authorities objected to the tests.[291]

[286] Letter from J. Scott Gordon, Director, EPA Region IV Office of Enforcement Coordination, to Donald R. van der Vaart, Secretary, N.C. Dep't of Envtl. Quality (May 9, 2016), https://assets.documentcloud.org/documents/3114598/EPA-Region-4-Letter-to-NCDEQ.pdf.
[287] U.S. EPA Region 5, *State Review Framework Ohio* at 3, 38-39 (Aug. 2013), https://www.epa.gov/sites/production/files/2014-05/documents/srf-rd2-rev-oh.pdf.
[288] U.S. EPA Region 4, *State Review of Framework Mississippi* at Executive Summary (Mar. 3, 2016), https://www.epa.gov/sites/production/files/2016-03/documents/srf-rd3-rev-ms.pdf.
[289] *Id.* at 24.
[290] Memorandum from Susan Shinkman, Director, EPA Office of Civil Enforcement, to Regional Counsel, Regional Enforcement Directors and Coordinators, and OCE Division Directors (May 31, 2017), https://www.documentcloud.org/documents/4324892-EPA-Clean-Air-Act-and-Its-Power-to-Request.html#document/p60/a392202.
[291] *Id.*

Additionally, in its proposed 2020 budget, the current Administration sought a 31 percent reduction in funding for EPA.[292] This reduction would affect EPA's enforcement budget and the resources available to ensure enforcement of the above the statutes. EPA cannot rely on its actions under other authorities when EPA has itself taken steps to ensure that those authorities are not adequately addressing the risks presented.

* * * * *

In sum, EPA must analyze all exposures to TCE. EPA cannot legally ignore exposures that occur under other EPA-administered statutes, and treating exposures that are known to occur in the world as nonexistent is arbitrary and capricious. EPA must assess these exposures based on their real-world existence and consider how they may combine with other sources of exposure to accurately estimate the risks presented by this chemical substance. Where EPA has inadequate information, EPA should use its information authorities to obtain more information about these exposures.

6. EPA needs to analyze potential exposures from distribution, as well as from known and reasonably foreseeable accidental exposures.

EPA's analysis of distribution was inadequate in the draft risk evaluation and problem formulation. In the draft risk evaluation, EPA stated that: "Activities related to distribution (e.g., loading and unloading) will be considered throughout the TCE life cycle, rather than using a single distribution scenario." (p. 55). It appears from this statement that EPA assumes that exposure from distribution occurs only during loading and unloading. It is not clear how, if at all, EPA considered exposures from loading and unloading under individual conditions of use, as it presents no specific analysis of these activities in the context of the various conditions of use. Moreover, EPA does not appear to address at all exposures from distribution aside from those arising from loading and unloading. Does EPA simply assume that all distribution occurs through so-called "closed systems" which lead to no releases or exposure whatsoever?

Neither the problem formulation nor the draft risk evaluation provides evidence or support for any assumption that TCE is always distributed in closed systems leading to no releases or exposures. EPA has provided no evidence that exposures and releases during distribution will be nonexistent.

The problem formulation and draft risk evaluation give no attention to potential releases and exposures resulting from accidental releases. EDF does not suggest that EPA needs to consider every possible scenario, but the risk of accidental releases and exposures is very real and certainly "reasonably foreseen" in many respects, and EPA has authority to mandate steps to reduce those risks. For example, as and after Hurricane Harvey passed through Houston, over 40

[292] Office of Mgmt. & Budget, *A Budget for a Better America* at 93, https://www.whitehouse.gov/wp-content/uploads/2019/03/budget-fy2020.pdf.

sites released toxic chemicals into the environment.[293] Given the known accidental releases, the huge number of petrochemical plants and refineries in the Houston area, and the likelihood that flooding there may become more common in light of climate change, such events are clearly reasonably foreseen and hence EPA needs to give more consideration to the potential for accidental releases.

7. EPA must consider "reasonably available" information, and thus EPA must use its authorities under TSCA §§ 4 and 8 to obtain additional information.

TSCA orders EPA to consider "available" and "reasonably available" information in crafting a risk evaluation, 15 U.S.C. §§ 2605(b)(4)(F)(i), 2625(k), and under the new risk evaluation rule, EPA defined "[r]easonably available information" to mean "information that EPA possesses or can reasonably generate, obtain, and synthesize for use in risk evaluations, considering the deadlines specified in TSCA section 6(b)(4)(G) for completing such evaluation." 40 C.F.R. § 702.33, promulgated at 82 Fed. Reg. 33,748 (July 20, 2017). Thus, under its own rule, EPA has to consider information that it "can reasonably generate, obtain, and synthesize."

In our prior comments on the scope documents, EDF expanded on EPA's duties to use its authorities under TSCA §§ 4 and 8 to obtain additional information about TCE, and EDF incorporates those arguments here.[294] In response to EDF's comment, EPA acknowledged its duty to consider "reasonably available information" and EPA described its efforts to gather information up to this point.[295] While EPA details its "data gathering activities," EPA has not established that these activities will result in EPA obtaining all the reasonably available information that EPA could "generate, obtain, and synthesize" if EPA also used its authorities under TSCA §§ 4 and 8 to obtain additional information. Thus, EPA has not established that it will obtain all reasonably available information.

In particular, EDF's prior comments established that relying solely on voluntary requests for information, may result in limited, biased, inaccurate, or incomplete information on methylene chloride. EDF incorporates those arguments here.[296] EPA's response to this comment was that

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[293] *See, e.g., More Than 40 Sites Released Hazardous Pollutants Because of Hurricane Harvey*, N.Y. TIMES (Sept. 8, 2017), https://www.nytimes.com/interactive/2017/09/08/us/houston-hurricane-harvey-harzardous-chemicals.html?_r=0.

[294] EDF Comments on Ten Scopes under the Toxic Substances Control Act at pp. 11-16, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0742-0072.

[295] EPA's Responses to Public Comments Received on the Scope Documents for the First Ten Chemicals for Risk Evaluation under TSCA at pp.10-14, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0725-0051.

[296] EDF Comments on Ten Scopes under the Toxic Substances Control Act at pp. 16-20, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0742-0072.

"EPA has not indicated it would rely solely on voluntary requests for information."[297] Thus, EPA appears to recognize that voluntary requests standing alone are insufficient. Despite that acknowledgement, EPA still has not relied on its available authorities to obtain additional information. EDF urges EPA to do so.

EPA's primary response to EDF's request that EPA consider all reasonably available information appears to be that the information EPA currently has is "adequate."[298] But, as a general matter, EPA has to consider all reasonably available information; TSCA does not authorize EPA to stop its analysis on the basis that EPA believes its current information is adequate. And as explained more below, it is clear that the information is not yet adequate to meet EPA's obligations under TSCA.

A. Relying on voluntary requests for information will result in limited, biased, inaccurate, or incomplete information on TCE.

In the problem formulation, EPA stated that: "*EPA encourages submission of additional existing data, such as full study reports or workplace monitoring from industry sources*, that may be relevant for refining conditions of use, exposures, hazards and potentially exposed or susceptible subpopulations during the risk evaluation. EPA will continue to consider new information submitted by the public." Problem Formulation for TCE at p. 58 (emphasis added).

With this language EPA seems to acknowledge the serious data gaps it faces; yet despite clear authority to require workplace monitoring by industry and to obtain full study reports using its existing authorities, EPA resorts merely to encouraging their submission.

Rather than relying solely on voluntary submissions—an approach that has proven insufficient in the past—EPA should use its information authorities to obtain necessary information on conditions of use, exposures, hazards, and potentially exposed or susceptible subpopulations.

There are several obvious problems and limitations with this voluntary approach which EPA has still not addressed.

First, a voluntary call is much less likely to produce all of the necessary information than rules mandating that affected parties provide the requested information. If manufacturers and processors are legally required to provide the information, that legal obligation provides a strong incentive for them to develop or obtain and submit all relevant information. Absent that incentive, some companies may choose to focus time and attention on other matters.

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[297] EPA's Responses to Public Comments Received on the Scope Documents for the First Ten Chemicals for Risk Evaluation under TSCA at p.13, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0725-0051.

[298] *See id.* at pp. 13, 10-14.

Second, EPA has provided no empirical evidence establishing that this voluntary approach will result in EPA obtaining all "reasonably available" information. Unless EPA has some empirical basis for stating that the voluntary approach will allow EPA to obtain all reasonably available information that it can obtain under its legal authorities, EPA must rely on its existing authorities to obtain a complete set of information.

Third, manufacturers and processors of TCE have a vested interest in EPA finding that TCE does not present an unreasonable risk. A no-unreasonable-risk finding reduces the likelihood of government regulation, including potential restrictions on TCE, and it may reduce any stigma they may otherwise face in the marketplace. The financial costs of regulation may ultimately be very high for some specific firms and individuals, and even if not, many firms and individuals may believe that the costs of regulation will be high. These companies have a "financial interest" in the outcome of these proceedings, and they are not impartial. *See, e.g*., 28 U.S.C. § 455(b)(4) (requiring Judges to disqualify themselves in proceedings where they have a financial interest). Because of this reality and appearance of partiality, relying solely on voluntary measures decreases the credibility of this risk evaluation.

Relying solely on voluntary presentation of information raises the concern that the companies or trade associations may present an incomplete or skewed picture. Companies and trade associations may choose to "cherry pick" information and provide only the information that paints TCE in a favorable light. They may provide only summaries of information that reflect conscious and subconscious judgment calls that result in unduly favorable conclusions; and without access to the full information neither EPA nor the public can independently assess such conclusions. They may choose not to review records robustly when the review may disclose unfavorable information. They may seek to put their best foot forward and describe the ideal scenario of use and safety measures. Or, if they have unfavorable information, they may choose not to provide any information at all and simply not participate in these proceedings.

EPA cannot simply assume that members of the regulated community will voluntarily disclose unfavorable or complete information about their practices and products. *See* THE FEDERALIST NO. 51 (James Madison) ("If men were angels, no government would be necessary. *** [E]xperience has taught mankind the necessity of auxiliary precautions."); *Williams v. Pennsylvania*, 136 S. Ct. 1899, 1905-06 (2016) ("Bias is easy to attribute to others and difficult to discern in oneself. *** This objective risk of bias is reflected in the due process maxim that 'no man can be a judge in his own case and no man is permitted to try cases where he has an interest in the outcome.'"). Here, manufacturers and processors obviously have an interest in the outcome, and EPA must craft its procedures and approaches with that reality in mind. Requiring the submission of information is the safest approach to ensuring that these parties provide all relevant information, and that is in turn crucial to establishing and demonstrating the credibility of this process.

If EPA acts under TSCA §§ 8(a), (c), and (d), the regulations impose some requirements that will help ensure the accuracy and completeness of the information. First, EPA can require that certain information and underlying information be provided in full, which ensures completeness. In addition, a § 8(d) rule requires that people engage in an adequate search of records. 40 C.F.R. § 716.25. Second, submitters must file certification statements by authorized officials that certify that the submitted information has been submitted in compliance with the requirements of this process. *See, e.g*., 40 C.F.R. § 711.15(b)(1). Third, submitters often must retain records of required submissions for a period of five years, and the retention of records can help encourage accurate reporting since those records would be available should a submission latter be investigated. *See, e.g*., 40 C.F.R. § 711.25. None of these features apply to the voluntary requests for information EPA has indicated it is relying on.

B. EPA cannot rationally rely on unvetted industry submissions, and to the extent EPA relies on voluntary submissions from industry, EPA must take numerous additional steps to increase their reliability and transparency.

In the problem formulation and draft risk evaluation, EPA uncritically relies on industry submissions, and this reliance does not constitute the best available science. To the extent it relies on voluntary submissions from industry, EPA needs to take additional steps to better ensure that the voluntary information it receives is accurate and complete. EPA would need to develop a far more rigorous and structured process than it currently has. For example, EPA's submission process does not appear to require anyone to certify that the information in their submissions is accurate or complete to the best of their knowledge. EPA should consider approaches for vetting statements and assertions, particularly when made by entities with a financial interest in the outcome of these risk evaluations.

C. The draft risk evaluation and these comments identify numerous information gaps that EPA needs to fill using its information authorities.

Throughout these comments, EDF points to information gaps that EPA should fill using its information authorities. Yet the problem formulation and draft risk evaluation do not adequately address these data gaps – let alone describe a strategy for acquiring the needed information.

The draft risk evaluation identifies data gaps that EPA should have addressed earlier in this process. To cite some examples:

- Information on activities where ONUs may be present are insufficient to determine their exposures. (pp. 32, 33, 35, 101, 105, 107, 281, 348, 350, 351, 356, 378, 384, 385, 386, 387, 389, 390, 391, 394, 401, 405, 420).
- For numerous conditions of use, EPA lacked adequate monitoring data for TCE. (pp. 30, 32-33, 101, 103, 126, 181, 275, 293, 302, 309, 386, 388, 389, 391, 395, 397, 398, 399, 402, 405, 408, 409, 416, 417, 419, 420, 701, 703, 707, 715, 716, 718, 720, 722, 723, 724, 725, 730, 732, 734, 736, 737, 738, 739).

As a general matter, EPA should use its information authorities to fill the gaps identified in the draft risk evaluation and these comments.

D. EPA's problem formulation also revealed numerous data gaps, yet EPA failed to address any of them.

Notably, many data gaps were evident at the problem formulation stage, and EPA failed to address these data gaps despite its information authorities under TSCA as reformed in 2016. In this subsection we discuss examples of the many data gaps apparent from the Problem Formulation and EPA's resort to insufficient approaches to work around the gaps without actually filling them. EPA should have used its information authorities to fill these data gaps, and EPA still could do so in preparing a final risk evaluation.

While EPA expressly acknowledged few data gaps in the Problem Formulation, the existence of exposure information gaps is implied through EPA's plan to use surrogate data and models rather than using its information authorities to obtain exposure information on TCE.

Where EPA did acknowledge information gaps, it failed to set forth a plan to use its TSCA section 4 and 8 information authorities to fill such gaps. Here are two examples of unfilled data gaps in the Problem Formulation, which were identified in our comments on the problem formulation and are in addition to the exposure data gaps identified in subsection C. above:

- TCE levels in soil: On page 34 of the Problem Formulation EPA states: "Compared with other environmental media, there is a relative lack of nationally representative monitoring data on levels of TCE in ambient soil." EPA has identified an information gap that it should have filled using its information authorities under TSCA, but it did not do so.

E. Dermal exposure modeling inadequacies:

In describing its analysis plan for occupational exposure on page 64, EPA states: "EPA anticipates that existing EPA/OPPT dermal exposure models would not be suitable for quantifying dermal exposure to highly volatile chemicals such as TCE." The draft risk evaluation does not acknowledge this earlier concern or make clear whether or how it was addressed. If EPA does not have sufficient information on dermal exposure whether through measured or modeled data, it should have used its authorities to obtain them. EPA cannot assume that an absence of evidence about particular hazards or exposures provides evidence of that those hazards or exposures are absent.

When a data gap exists, EPA cannot rationally assume that the absence of evidence regarding a particular hazard or exposure establishes that the hazard or exposure is not present. When EPA has failed to collect information about particular hazards or exposures, it is arbitrary and capricious to assume that the lack of information establishes that the particular hazard or

exposure does not exist. In addition, such assumptions violate EPA's duty to consider all reasonably available information, which EPA could generate to fill these data gaps, as well as EPA's duty to use the best available science.

8. EPA needs to implement the requirements of TSCA § 14 when reviewing materials for the risk evaluation.

EPA has an affirmative obligation to review at least 25% of non-chemical identity confidentiality claims under TSCA, 15 U.S.C. § 2613(g), and EPA has stated that it is implementing that obligation by "review[ing] every fourth submission received that contains non-chemical identity [confidential business information (CBI)] claims."[299] Thus, on balance, EPA should be reviewing all confidentiality claims asserted in at least approximately one-fourth of the information submissions it receives. Those claims must be substantiated at the time of submission. EPA must complete reviews of confidentiality claims within 90 days of receipt of the claims, and if EPA denies a claim, EPA must disclose the information that had been claimed confidential 30 days after notifying the claimant of the denial, absent a challenge to the denial in district court. 15 U.S.C. § 2613(g)(1)(A), (g)(2)(B).

In addition, TSCA requires disclosure of "any health and safety study which is submitted under [TSCA] with respect to *** any chemical substance or mixture which, on the date on which such study is to be disclosed has been offered for commercial distribution." 15 U.S.C. § 2613(b)(2)(A). TSCA also requires disclosure of "any information reported to, or *otherwise obtained by*, [EPA] *from* a health and safety study which relates to [such] a chemical substance ***." *Id*. § 2613(b)(2)(B) (emphases added). Thus, any health and safety studies and related information on TCE must be disclosed. TSCA defines "health and safety study" to mean "any study of any effect of a chemical substance or mixture on health or the environment or on both, including underlying information and epidemiological studies, studies of occupational exposure to a chemical substance or mixture, toxicological, clinical, and ecological studies of a chemical substance or mixture, and any test performed pursuant to this Act." *Id*. § 2602(8). EPA has provided further details on this expansive definition of "health and safety study," explaining that it encompasses, among other things, "[a]ny data that bear on the effects of a chemical substance on health or the environment" and "[a]ny assessments of risk to health and the environment resulting from the manufacture, processing, distribution in commerce, use, or disposal of the chemical substance." 40 C.F.R. § 720.3(k). Thus, any health and safety study or other information on health or environmental effects or any assessment of risk EPA prepared must be disclosed. The only exception from that disclosure requirement is for "information *** that discloses processes used in the manufacturing or processing of a chemical substance or mixture

[299] EPA REVIEW AND DETERMINATION OF CBI CLAIMS UNDER TSCA, https://www.epa.gov/tsca-cbi/epa-review-and-determination-cbi-claims-under-tsca (last visited Jan. 18, 2018).

or, in the case of a mixture, the portion of the mixture comprised by any of the chemical substances in the mixture." 15 U.S.C. § 2613(b)(2).

In developing this draft risk evaluation, a large fraction of the information EPA relied upon constituted health and safety studies. All such information not subject to the two narrow exceptions needs to be made public.

9. EPA needs to accurately identify the relevant potentially exposed or susceptible subpopulations.

A. EPA should identify people living near conditions of use, including disposal sites, as potentially exposed or susceptible subpopulations.

In the problem formulation, EPA correctly recognized that potentially exposed or susceptible subpopulations included "groups of individuals within the general population who may experience greater exposures due to their proximity to conditions of use that result in releases to the environment and subsequent exposures (e.g., individuals who live or work near manufacturing, processing, use or disposal sites)." Problem Formulation for TCE at pp. 38-39. Such subpopulations fall neatly within TSCA's definition of the term "potentially exposed or susceptible subpopulation" to include "a group of individuals within the general population identified by the Administrator who, due to *** *greater exposure*, may be at greater risk than the general population of adverse health effects from exposure to a chemical substance or mixture." 15 U.S.C. § 2602(12) (emphasis added).

Despite recognizing that EPA should be analyzing such subpopulations in the problem formulation, EPA did not analyze these potentially exposed and susceptible subpopulations in the draft risk evaluation. (p. 352). EPA should analyze these subpopulations in the final risk evaluation.

EPA should also identify people living near disposal sites as potentially exposed or susceptible subpopulations. As explained below, TSCA expressly requires EPA to consider disposal.

TSCA expressly defines "conditions of use" to include "the circumstances" under which the chemical is "known, or reasonably foreseen to be *** disposed of." 15 U.S.C. § 2602(4). Thus, EPA must analyze the exposures arising from the activities associated with disposal of a chemical substance. EPA must also identify those who face greater exposures due to their proximity to disposal sites as a "potentially exposed or susceptible subpopulation" since they are a "group of individuals within the general population identified by the Administrator who, due to *** greater exposure, may be at greater risk than the general population of adverse health effects from exposure to a chemical substance or mixture." 15 U.S.C. § 2602(12). In addition, as EDF previously explained in its comments on the scopes, EPA cannot rationally exclude so-called legacy uses and associated disposals. EDF incorporates and reiterates those points here as

well.[300] The Ninth Circuit has ruled that EPA does not have authority to exclude such conditions of use from its analysis, so EPA must include them. *See Safer Chems. v. United States EPA*, 2019 U.S. App. LEXIS 33976, *50-53 (9th Cir. Nov. 14, 2019). And the Court recognized that EPA must analyze disposals that are "in fact ongoing" when analyzing disposal. *Id.* at 56.

Problematically, EPA also excluded the pathways leading to this exposure from further analysis. *E.g.*, *compare* Problem Formulation for TCE at p. 38 (recognizing subpopulation), *with* draft risk evaluation at pp. 35, 379 (excluding pathways allegedly covered by other statutes from analysis). As EDF previously explained, this approach is irrational and incoherent. *See* above in section 5.B.ii. EPA should not exclude those pathways for the reasons given above, and in addition, EPA cannot rationally evaluate the greater exposure these subpopulations face without analyzing these pathways. EPA has provided no rationale explaining how it plans to accurately evaluate the risks faced by these subpopulations while ignoring these pathways of exposure.

In addition, EPA should be analyzing communities who live or work near past manufacturing, processing, distribution, or use sites, even if those activities have ceased. The statute does not allow EPA to ignore conditions of use merely because they happened in the past, and in any event, the disposal at these sites remains ongoing at this time.

B. EPA should identify people living in proximity to sources of contamination as potentially exposed or susceptible subpopulations.

In addition to subpopulations living in proximity to conditions of use, EPA should analyze subpopulations to those in proximity to sources of contamination not necessarily linked to or able to be attributed to a specific condition of use. TSCA defines the term "potentially exposed or susceptible subpopulation" to include "a group of individuals within the general population identified by the Administrator who, due to *** *greater exposure*, may be at greater risk than the general population of adverse health effects from exposure to a chemical substance or mixture." 15 U.S.C. § 2602(12) (emphasis added). Thus, a subpopulation can qualify due solely to "greater exposure" to a chemical substance; the statute includes no text qualifying "greater exposure" requiring that the exposure be linked to a particular condition of use.

Thus, EPA needs to expand its analysis of vulnerable subpopulations to include other groups of individuals within the general population who may experience greater exposures due to their proximity to sources of contamination (e.g., contaminated groundwater) not necessarily linked to or able to be attributed to a specific condition of use.

Reasonably available information reveals numerous sites where TCE is known to be present and thus where the subpopulations in their proximity may be at greater risk due to greater exposure.

[300] EDF Comments on Ten Scopes under the Toxic Substances Control Act pp.8-9, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0742-0072.

We attach a list of some of the known sites contaminated with TCE so that EPA can analyze the subpopulations potentially suffering greater exposure from these sites. *See* Table E below and Appendix 3.

Table E: Superfund Sites with TCE

Chemical Substance	Number of Superfund Sites with the Chemical Substance
Trichloroethylene	731

10. EPA needs to ensure that environmental justice is appropriately considered, analyzed, and addressed in the risk evaluation.

Environmental justice is "the fair treatment and meaningful involvement of all people regardless of race, color, national origin, or income with respect to the development, implementation and enforcement of environmental laws, regulations and policies."[301] According to EPA, providing "[f]air treatment" will ensure that "no group of people should bear a disproportionate share of the negative environmental consequences resulting from industrial, governmental and commercial operations or policies."[302] EPA has committed to integrate environmental justice into "everything" the Agency does in order to "reduce[ ] disparities in the nation's most overburdened communities."[303]

Despite this commitment, and EPA's obligations to comply with Executive Order 12898 (see below), EPA has not incorporated environmental justice considerations into the draft risk evaluation. In addition, EPA does not appear to have undertaken any outreach oriented towards ensuring the meaningful involvement of environmental justice communities in the risk evaluation process. EPA must address environmental justice in the risk evaluation, both by incorporating an analysis into the evaluation and ensuring meaningful involvement by environmental justice communities in the development of the risk evaluation.

A. The risk evaluation is subject to Executive Order 12898.

Executive Order 12898 directed federal agencies to identify and address "disproportionately high and adverse human health or environmental effects of its programs, policies, and activities on minority populations and low-income populations." Exec. Order No. 12898, 59 Fed. Reg. 7629

[301] EJ 2020 GLOSSARY, https://www.epa.gov/environmentaljustice/ej-2020-glossary.
[302] *Id.*
[303] U.S. EPA, *EJ 2020 Action Agenda* at 1 (2016), https://www.epa.gov/sites/production/files/2016-05/documents/052216_ej_2020_strategic_plan_final_0.pdf.

(Feb. 16, 1994). EPA must comply with this duty in the Executive Order. *See Sherley v. Sebelius*, 689 F.3d 776, 784 (D.C. Cir. 2012) ("[A]s an agency under the direction of the executive branch, it must implement the President's policy directives to the extent permitted by law."). The Executive Order applies, by its own terms, to all "programs, policies, and activities" of a federal Agency, and EPA's preparation of the risk evaluation undoubtedly falls within this capacious definition, qualifying as an "activit[y]" of EPA, carried out as part of its "programs" and pursuant to its "policies." As Agency actions that may affect the level of protection provided to human health or the environment, the risk evaluation under TSCA must address environmental justice communities.[304] EPA's own guidance on considering environmental justice defines "Agency action" to include risk assessments.[305] EPA has articulated no theory for why the Executive Order would not apply to the risk evaluation.

Yet EPA has failed to mention, let alone adequately address, Executive Order 12898 or "environmental justice" in the draft risk evaluation. Failure to do so violates EPA's obligations under the Executive Order.

Notably, EPA has stated that the identification of potentially exposed or susceptible subpopulations under TSCA would "carry[ ] out the spirit" of Executive Order 12898.[306] EPA's implication that the act of merely identifying "potentially exposed or susceptible subpopulations," standing alone, is sufficient to comply with the Executive Order, is plainly incorrect. The Executive Order specifically states that EPA must consider the disparate impacts of pollution on "minority populations and low-income populations."[307] The failure to do so in the draft risk evaluation, in particular by failing to consider minority, low-income, and indigenous communities when identifying potentially exposed or susceptible populations, does not "carry out the spirit," or the letter, of the Executive Order. EPA must prepare an actual environmental justice analysis to comply with the Executive Order.

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[304] *See* U.S. EPA, *EPA's Action Development Process Interim Guidance on Considering Environmental Justice During the Development of an Action* at 18 (Jul. 2010), https://www.epa.gov/sites/production/files/2015-03/documents/considering-ej-in-rulemaking-guide-07-2010.pdf.

[305] *Id.* at 1.

[306] U.S. EPA, Risk Evaluation Rule Response to Comments at 1, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0654-0109.

[307] Exec. Order No. 12898; *see also* U.S. Office of Inspector General, *EPA Needs to Consistently Implement the Intent of the Executive Order on Environmental Justice* at 9-10 (Mar. 2004), https://www.epa.gov/sites/production/files/2015-10/documents/20040301-2004-p-00007.pdf (explaining that the intent of the Executive Order, in part, was to place EPA's focus on minority and low-income communities).

B. EPA's exclusions in the draft risk evaluation violate the Executive Order by underestimating the risks faced by environmental justice communities.

EPA's decision to exclude environmental releases covered by other statutes because those statutes "adequately address" risk fails to acknowledge that other statutes have historically failed to consider environmental justice communities in permitting and enforcement. The National Environmental Justice Advisory Council (NEJAC), a federal advisory committee to EPA, has stated that:

> Environmental protection in this country has grown by individual pieces of legislation, developed to address a particular environmental media or a pressing problem like abandoned toxic sites. Environmental law has not evolved from a master game plan or unifying vision. As a result, the statutes *have gaps in coverage* and do not assure compatible controls of environmental releases to all media from all sources.[308]

Those gaps in coverage were often a result of controlling pollution solely "through technology-based regulation or an individual chemical-by-chemical approach."[309] The Lautenberg Act's unique emphasis on protecting "potentially exposed or susceptible subpopulations" recognized, in part, that the historical regulation of pollutants resulted in some subpopulations, including low-income, minority, and indigenous communities, being disproportionately impacted by chemical contamination.

In addition to the general gaps in coverage, environmental justice communities are often disproportionately exposed to sources of chemical contamination. For instance, a report by the General Accounting Office revealed that:

- three-quarters of hazardous waste landfill sites in eight southeastern states were located in communities whose residents were primarily poor and African-American or Latino, and
- race and ethnicity were the most significant factors in deciding where to place landfills, waste and environmentally hazardous facilities.[310]

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[308] National Environmental Justice Advisory Council, Cumulative Risks/Impacts Work Group, *Ensuring Risk Reduction in Communities with Multiple Stressors: Environmental Justice and cumulative Risks/Impacts* at 7 (Dec. 2004), https://www.epa.gov/sites/production/files/2015-04/documents/ensuringriskreducationnejac.pdf (emphasis added).

[309] *Id.* at 11.

[310] General Accounting Office, *Siting Hazardous Waste Landfills and Their Correlation with Race and Economic Status of Surrounding Communities* at 13-21 (1983), https://www.gao.gov/products/RCED-83-168.

EPA's exclusion from the draft risk evaluation of exposure pathways resulting from environmental releases fails to recognize that environmental justice communities have not historically been protected by other environmental statutes and are often disproportionately exposed to chemical substances through disposal and other conditions of use. These exclusions will result in unfair treatment to environmental justice communities by ensuring that they will continue to "bear a disproportionate share of the negative environmental consequences resulting from industrial, governmental and commercial operations or policies."[311]

Moreover, EPA's exclusions of exposure pathways linked to disposal sites and legacy use, associated disposal, and disposal that is in fact ongoing will specifically underestimate the exposures of environmental justice communities. In fact, NEJAC has previously informed EPA of this exact concern:

> It is particularly important to recognize historical exposures in communities and tribes suffering environmental injustice. In some cases, community members were exposed to pollutants for many years in the past from facilities that are *no longer functioning or in business*. These past exposures could act to increase the body burden of a subpopulation so that vulnerable individuals start off at a higher dose. Even if the dose-response curves among the subpopulation are the same as the general population, starting off at a higher point on this curve puts the members of the vulnerable subpopulation at greater risk for exposure to the same amount of a compound than the general population. This fact is highly pertinent to the historical legacy of racial and economic discrimination, and the relationship of vulnerability to health disparities.[312]

Failing to consider exposures linked to disposal, legacy uses, associated disposal, and disposal that is in fact ongoing systematically underestimates the background level of exposures faced by many environmental justice communities. In order to determine whether those communities will face an unreasonable risk of injury from TCE, EPA must consider exposures from disposal, legacy uses, associated disposal, and disposal that is in fact ongoing.

11. Assessment factors do not lead to conservative calculations; in fact, assessment factors account for real-world sources of variability as well as database limitations.

EPA's use of assessment factors (AFs) in developing the concentrations of concern (COCs) account for real-world sources of variability as well as database limitations, and cannot be

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[311] EJ 2020 GLOSSARY, https://www.epa.gov/environmentaljustice/ej-2020-glossary.

[312] National Environmental Justice Advisory Council, Cumulative Risks/Impacts Work Group, *Ensuring Risk Reduction in Communities with Multiple Stressors: Environmental Justice and cumulative Risks/Impacts* at 24 (Dec. 2004), https://www.epa.gov/sites/production/files/2015-04/documents/ensuringriskreducationnejac.pdf (emphasis added).

construed as "safety factors" that yield conservative estimates. As EPA acknowledges: "The application of AFs provides a lower bound effect level that would likely encompass more sensitive species not specifically represented by the available experimental data. AFs can also account for differences in inter- and intra-species variability, as well as laboratory-to-field variability. These AFs are dependent on the availability of datasets that can be used to characterize relative sensitivities across multiple species within a given taxa or species group." (p. 198).

The National Academy of Sciences, in its 2009 report titled *Science and Decisions: Advancing Risk Assessment* has this to say on this subject, albeit in the context of human rather than environmental health:

> Another problem *** is that the term *uncertainty factors* is applied to the adjustments made to calculate the RfD [reference dose, derived from, e.g., a no-effect level] to address species differences, human variability, data gaps, study duration, and other issues. The term engenders misunderstanding: groups unfamiliar with the underlying logic and science of RfD derivation can take it to mean that the factors are simply added on for safety or because of a lack of knowledge or confidence in the process. That may lead some to think that the true behavior of the phenomenon being described may be best reflected in the unadjusted value and that these factors create an RfD that is highly conservative. But the factors are used to adjust for differences in individual human sensitivities, for humans' generally greater sensitivity than test animals' on a milligrams-per-kilogram basis, for the fact that chemicals typically induce harm at lower doses with longer exposures, and so on. At times, the factors have been termed *safety factors*, which is especially problematic given that they cover variability and uncertainty and are not meant as a guarantee of safety.[313]

In evaluating risks, EPA should recognize that AFs ensure greater accuracy and do not provide a safety factor rendering the evaluation "conservative."

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EDF appreciates the opportunity to provide comments and EPA's consideration of them.

[313] NAT'L RESEARCH COUNCIL, SCIENCE AND DECISIONS: ADVANCING RISK ASSESSMENT at chp. 5, p. 132 (2009), https://www.ncbi.nlm.nih.gov/pubmed/25009905 (emphases in original).