UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

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

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

Executive Summary

PCE is a high exposure/high hazard solvent with several known chronic health effects that have long been of concern to state and federal agencies, members of the military, labor unions, and the general public. These effects include neurotoxicity, reproductive and developmental effects, liver and kidney toxicity and cancer. Human studies of short-term exposure also document an association between PCE and serious vision impairments in both occupational and residential settings.

According to the draft evaluation, as many as 120,000 commercial and industrial sites across the US manufacture, process or use PCE.[2] The total number of exposed workers at these sites is in the range of 750,000.[3] EPA does not estimate the number of exposed consumers but this population includes a sizable number of Americans who use PCE-containing products and/or are exposed to PCE in indoor or outdoor air, through drinking water or because of proximity to contaminated sites and facilities where PCE is manufactured, processed or used.

EPA's draft evaluation determines that nearly all conditions of use of PCE present unreasonable risks to workers and users of consumer products. While these findings are alarming, they fail to reflect the full seriousness of PCE's risks to health and the true extent of the population at risk. Because of its serious understatement of exposure and risk, the EPA evaluation is insufficiently protective and, if used as the

[1] 85 Federal Register 26464 (May 4, 2020); Draft Toxic Substances Control Act (TSCA) Risk Evaluation for Perchloroethylene (PCE) (Draft Evaluation), https://www.epa.gov/sites/production/files/2020-04/documents/draft_risk_evaluation_for_perchloroethylene_public.pdf

[2] Draft Evaluation at 72-73.

[3] EPA provides estimates of the number of exposed workers and occupational non-users (ONUs) on a use-by-use basis throughout the draft evaluation. Our tabulation of these individual estimates yields a total of around 750,000.

basis for risk management, will leave large segments of the US population exposed to unsafe levels of PCE.[4]

PCE is one of a group of large volume solvents – including trichloroethylene (TCE), methylene chloride and carbon tetrachloride – on which EPA is now conducting or will conduct risk evaluations under TSCA. The four draft evaluations completed to date confirm that these solvents have similar molecular structures and metabolites, common health effects like cancer, and overlapping conditions of use that often result in co-exposure by many workers and consumers. EPA has been addressing each solvent in isolation, but it is likely that their cumulative effects on health and the environment are markedly greater than the individual EPA evaluations suggest. This understatement of cumulative risk should be an important consideration when weighing options for risk management.

Historically, PCE's most prominent use has been in dry cleaning and spot cleaning. EPA and state agencies have devoted considerable attention to this use because it poses cancer and other risks not only to dry cleaning workers but to many segments of the general population, including people who wear dry cleaned clothing, live near, next to, or above dry cleaners, are in the families of dry cleaning employees, or use coin operated dry cleaning facilities. Previous assessments have shown that millions of people in these categories are exposed to lifetime cancer risks greater than EPA's TSCA benchmark of one in one million. However, despite the efforts of regulators, the dry-cleaning industry remains heavily dependent on PCE. According to the draft problem formulation, PCE use in dry-cleaning machines has declined modestly from 83 percent in 1991 to 60 percent in 2017.[5] EPA estimates that, assuming a 60 percent market share, PCE is now used at 12,822 commercial and 12 industrial dry cleaners employing a total of 57,000 workers.[6] *The ongoing TSCA evaluation highlights the continued pervasiveness of PCE in this industry in the face of large risks to health and the need for EPA to use its TSCA authorities to phase out this unsafe PCE use along with vapor degreasing and other dangerous uses.*

We focus in these comments on several aspects of the draft evaluation that greatly understate PCE's risks. Our concerns are summarized below.

- **Failure to Address the Contribution of Environmental Release Pathways to Risks to the General Populations and Vulnerable Subpopulations (pp. 7-22)**

Like previous evaluations, the draft ignores the human health implications of PCE releases to the environment. This omission violates TSCA's requirement to assess all pathways of exposure and departs from repeated recommendations of EPA's Science Advisory Committee on Chemicals (SAAC). PCE air emissions and contaminated groundwater, drinking water and soil are pervasive across the US and contribute significantly to overall PCE exposure. Dry cleaning releases account for a significant portion of emissions and result in elevated levels of PCE in outdoor and indoor air that affect a large

[4] Not only will EPA risk management measures be insufficient but states who believe the risks are greater than EPA has determined will be preempted from imposing more protective requirements under section 18 of TSCA.
[5] EPA, *Problem Formulation for the Risk Evaluation of Perchloroethylene,* May 2018 (PCE Problem Formulation) at 22, https://www.epa.gov/sites/production/files/2018-06/documents/perc_problem_formulation_5-31-2018v3.pdf
[6] Draft Evaluation at 157.

subpopulation. These emissions – and higher concentrations of PCE in indoor and outdoor air generally – are responsible for cancer risks that exceed the EPA TSCA lifetime benchmark of one in one million. Moreover, large subpopulations are exposed to PCE by multiple pathways simultaneously – i.e. by breathing PCE in indoor and outdoor air, consuming contaminated drinking water and living near PCE-contaminated Superfund sites or manufacturing or processing sites. Because PCE exposure levels are higher for these subpopulations than for the general population and they face elevated health risks, they constitute Potentially Exposed or Susceptible Subpopulations (PESSs) for which EPA must make specific determinations of unreasonable risk under TSCA. Indeed, even for the specific subpopulations (workers and users of consumer products) that the draft evaluation addresses, EPA significantly understates risks by ignoring exposure across routes and pathways of exposure, including in air, water and soil.

A comprehensive risk evaluation accounting for all PCE conditions of use and pathways of exposure is required under TSCA and EPA's regulations. EPA must estimate total exposure from all known and reasonably foreseen conditions of use and characterize the increased risk resulting from all concurrent exposure pathways. However, because of its narrow scope, the draft PCE evaluation presents a limited and incomplete picture of PCE's risks to the public. EPA must revise the draft PCE evaluation so it addresses *all* sources of exposure and risk.

- **Failure to Address the Risks of Chronic PCE Exposure by Consumers (pp. 22-26)**

The draft evaluation only addresses risks to consumers from acute exposure to PCE and thus does not examine chronic health effects linked to PCE, including cancer, developmental and reproductive toxicity, neurotoxicity and liver and kidney toxicity. This creates the incorrect impression that consumers are not at risk for these serious effects. However, multiple lines of evidence demonstrate that consumers have long-term PCE exposure. Numerous measurements of indoor air concentrations of PCE (some at extremely high levels) indicate that consumer exposure to PCE is not episodic but continuous. Consumers using contaminated drinking water are likewise exposed to PCE on an ongoing basis. There is also extensive evidence of the presence of PCE in human blood, urine and breath samples and in human breast milk, again consistent with long-term continuous exposure.

Focusing only on individual consumer products, EPA claims that "consumer exposure scenarios are expected to be intermittent and it is unlikely that the expected use patterns would cumulatively" result in repeated exposure. However, most PCE-containing consumer products are used regularly by hobbyists, household cleaners, home renovators, artists, and do-it-yourself vehicle mechanics. Moreover, while EPA's draft assumes use of a single product type during a day, many consumers likely use different PCE-containing products on the same day or over time. In its report on the trichloroethylene (TCE) evaluation, the SACC "disagreed with EPA's decision not to characterize chronic risks for consumers," indicating that "[s]everal Committee members suggested that some consumers are likely to be exposed more frequently and more pervasively to emissions from [consumer] products" than EPA assumed and pointing to the widespread presence of TCE in indoor air as evidence of such continuous exposure. Thus, even apart from the extensive evidence that all consumers have chronic

exposure, intensive users of PCE-containing consumer products are plainly exposed to PCE on a recurring basis. Because these users comprise a PESS under TSCA, EPA must directly address whether they are at risk of chronic health effects and how large that risk is.

- **Failure to Combine Exposures and Risks Across Routes and Pathways of Exposure and Conditions of Use (pp. 26-29)**

Like past evaluations, the PCE draft does not combine dermal and inhalation exposures even though these two routes occur simultaneously for both workers and consumers. The PCE evaluation also fails to address combined exposures across multiple pathways and conditions of use. This results in a considerable underestimation of risk because overall exposure to PCE may derive from its presence in the workplace, consumer products, ambient and indoor air, drinking water and waste at contaminated sites. For example, job-related PCE exposures may be magnified by consumer product use and environmental sources of exposure. Families of workers may also have "take home" exposures, i.e. elevated air levels in residences because of the worker's contaminated clothing or skin (a known occurrence for families of dry-cleaning workers). Subpopulations with elevated exposure to PCE from multiple routes and pathways are PESSs under TSCA and evaluating known, intended or reasonably foreseen combinations of exposures is a necessary step in adequately protecting them from unreasonable risks.

- **Failure to Account for Subpopulations with Greater Susceptibility to PCE's Health Effects and to Apply Sufficient Uncertainty Factors (UFs) (pp. 29-35)**

EPA has also identified numerous subpopulations with increased susceptibility to PCE but has failed to make determinations of unreasonable risk specific to these PESSs. Thus, the draft evaluation does not address how much more susceptible these subpopulations are to PCE and provide non-cancer Margins of Exposure (MOEs) and cancer risk estimates that account for the greater likelihood of harm. Without such an analysis, EPA cannot address whether risks to the PESSs (as opposed to average workers and consumers) are unreasonable and quantify the additional increment of risk to which these subpopulations are exposed.

The default 10X factor for intraspecies variability does not adequately account for the increased risk to susceptible subpopulations, as EPA itself acknowledges, and must be increased. Consistent with IRIS, EPA must also apply an additional 10X UF for data-base deficiencies. Together, these two adjustments would result in a significant increase in estimated risks.

- **Correct Determination that PCE is a Non-Threshold Carcinogen but Understatement of Cancer Risk to Humans (pp. 35-43)**

PCE is universally recognized to be a probable human carcinogen based on extensive evidence of multiple tumor types in animal and human epidemiological studies. Like the IRIS assessment, the draft evaluation correctly determines that, in accordance with EPA's cancer guidelines, PCE should be treated as a non-threshold carcinogen and that hypothesized modes of action (MOAs) that assume a threshold are unsupported. We agree with EPA that linear extrapolation is the correct approach to estimate cancer risk using the liver tumor findings in animal studies.

However, we believe that EPA has not adequately accounted for extensive epidemiological data showing PCE's carcinogenicity in humans. These studies indicate an association between PCE exposure and non-Hodgkins lymphoma (NHL), multiple myeloma (MM), and bladder, esophagus, lung, liver, cervical, and breast cancer. Looking at each endpoint in isolation, EPA concludes that "[e]pidemiological studies provide suggestive evidence for an association between PCE exposure and tumor development in humans." However, a stronger classification is warranted when considering the total weight of evidence linking both drinking water and inhalation exposures of PCE to numerous cancer types in multiple well-designed and well-conducted epidemiological studies across several population cohorts. Consistent with EPA's guidelines for cancer risk assessment, we recommend that EPA classify PCE as "carcinogenic to humans" rather than simply "likely to be carcinogenic."

We are also concerned that EPA's risk evaluation fails to account for acute cancer risks to workers and consumers and recommend that EPA follow the recommendations of the National Research Council (NRC) in determining acute cancer risks.

- **Failure to Use a Protective Benchmark for Unreasonable Cancer Risk to Workers (pp. 43-45)**

As in earlier evaluations, EPA has used a cancer risk of 1×10^{-4} as the benchmark for determining whether PCE presents an unreasonable risk to workers. This contrasts with the more protective benchmark of 1×10^{-6} that EPA has used for consumers. The SACC has stated that EPA has not provided an "adequate explanation and justification" for applying a less stringent risk standard to workers than other subpopulations. In fact, workers are specifically identified as a PESS in section 3(12) of the law. Thus, there is no basis for affording them *less* protection than other subpopulations by denying them the benefit of well-established EPA benchmarks for unacceptable cancer risk. In the final PCE evaluation, EPA should treat any increased cancer risk to workers exceeding 1×10^{-6} as unreasonable, thereby triggering risk management under section 6 of TSCA.

- **Failure to Model Realistic Dermal Exposure Scenarios (pp. 45-50)**

EPA appropriately developed exposure and risk estimates for dermal as well as inhalation routes of exposure. However, EPA's estimates of dermal exposure by workers rest on questionable assumptions and likely understate the magnitude of PCE exposure by this route. EPA should model a broader range of dermal contact scenarios based on its own analysis of variations in dermal exposure conditions and base risk estimates on multiple dermal exposure events per day. It should also estimate increases in exposure and risk where occlusion results in higher skin absorption of PCE during glove use.

While finding significant risks from dermal exposure to several consumer products, EPA has arbitrarily failed to address dermal exposure risks from many others. EPA has not explained why it believes there is no dermal exposure to these products and this conclusion would be inconsistent with realistic use scenarios and EPA's approach to assessing dermal exposure by workers. Moreover, where EPA has estimated dermal exposures for consumer products, the MOEs are often quite low, suggesting that incremental dermal exposure from other consumer products could well contribute meaningfully to overall risk and affect whether it is unreasonable.

- **Unwarranted Reliance on Personal Protective Equipment (PPE) in Determining PCE Risks to Workers (pp. 50-54)**

As in previous risk evaluations, EPA's determinations of unreasonable risk assume that workers will be protected from PCE exposure by using respirators and gloves. However, as the SAAC has repeatedly underscored, an expectation of universal PPE use is in fact contrary to the realities of workplace practice and sound principles of worker protection. None of EPA's draft evaluations have provided any evidence that PPE are in widespread use and effectively controlling exposure in workplaces where the subject chemicals are manufactured, processed and used. For this reason, the "no PPE" scenario is the only defensible basis for determining whether PCE presents an unreasonable risk to exposed workers. The requirements necessary to eliminate this unreasonable risk should be decided in the later TSCA risk management phase. At this point, under the well-established hierarchy of controls, PPE should be considered as a last resort, only after other means of risk elimination such as chemical substitution and engineering controls have been shown to be inadequate.

- **Use of the Poorly Defined and Unrealistic Category of Occupational Non-Users (ONUs) For Workplace Exposure Assessment (pp. 55-57)**

Like previous draft evaluations, the PCE evaluation differentiates between directly exposed workers and the amorphous category of "occupational non-users" (ONUs). This is a false dichotomy, and inconsistent with the state of the science for industrial exposure assessment. Instead, experts make a more meaningful distinction between near-field and far-field exposure and differentiate among jobs by whether they may be near or far from the source of exposure. Consistent with this approach, EPA should replace the broad ONU category with more refined groupings of near- and far-field workers and, within each grouping, conduct a more detailed exposure analysis which reflects job responsibilities and exposure scenarios specific to different types of workers and chemicals.

- **Understatement of PCE's Risks to the Environment (pp. 57-59)**

Throughout the draft risk evaluation, EPA repeatedly underestimates PCE's ecological risks.
First, EPA violates fundamental risk assessment principles by making use-by-use determinations of unreasonable environmental risk. These piecemeal ecological risk determinations understate the effects of PCE on the environment since they fail to address scenarios where facilities discharge PCE to the same water body at the same time. Second, EPA selects ecological concentrations of concern (COCs) that, according to EPA's own calculations, leave the most sensitive species subject to unreasonable risk. Third, EPA does not establish that any of its Assessment Factors used to calculate ecological risks are sufficient to address the uncertainty acknowledged in its environmental risk evaluation. Finally, EPA ignores its own risk calculations to conclude that multiple conditions of use with RQs far above 1 nonetheless present no unreasonable risk.

Although EPA has correctly determined that PCE presents an unreasonable risk to the environment, it must address these concerns so that its final evaluation accurately reflects the full magnitude of PCE's harmful ecological impacts.

- **Failure to Consider the Risks Associated with PCE's Known Degradation Products (p. 59)**

EPA acknowledges that "PCE biodegradation products include potentially hazardous substances including trichloroethylene, cis-1,2 dichloroethene and vinyl chloride." However, EPA fails to consider the known risks associated with PCE degradation in its draft risk evaluation.

- **Continued Reliance on the Flawed TSCA Systematic Review Method (pp. 59-63)**

The TSCA systematic review protocol used in the PCE and preceding nine draft risk evaluations is deeply flawed and has compromised their quality, validity, and protectiveness. The SACC has raised numerous concerns about the TSCA protocol, and it is now undergoing review by the National Academy of Sciences (NAS). Given the many concerns that have been raised and lack of a completed peer review, EPA should abandon the TSCA protocol and instead apply one of the established methodologies for systematic review that are consistent with the definition developed by the Institute of Medicine (IOM), such as the National Toxicology Program (NTP) OHAT method or the Navigation Guide Systematic Review Method developed by the University of California San Francisco. These methodologies embody recognized principles of systematic review and have been endorsed by NAS and other peer review bodies.

- **Failure to Make a Comprehensive Assessment of "Unreasonable Risk" (p. 63)**

TSCA mandates that EPA determine whether "the chemical substance" presents unreasonable risk, but EPA has evaluated each condition of use in isolation, avoiding assessment of the total risk posed by PCE. EPA must examine the combination of all conditions of use to total risk and exposure and cannot determine unreasonable risk for each condition of use in isolation.

I. By Excluding General Population Exposure, the Draft Evaluation Overlooks Significant Contributors to Human Health Risk

Like previous evaluations, the EPA draft lacks any assessment of risks to the general population from PCE's presence in air, drinking water and soil. This omission violates TSCA's mandate to comprehensively assess risk from all pathways of exposure and conflicts with repeated recommendations by the SACC. Few chemicals are as ubiquitous in the environment as PCE. Because of its many adverse health effects, its widespread distribution in environmental media presents significant health risks to large segments of the population. Of particular concern are subpopulations with elevated exposures because of proximity to dry cleaning operations – including consumers who patronize dry cleaners or use do-it-yourself cleaners, families of dry-cleaning employees, residents of apartments near, next to, or above dry cleaners and occupants of nearby homes and businesses. Although these groups comprise Potentially Exposed or Susceptible Subpopulations (PESS) under TSCA, they are nowhere addressed in the draft evaluation. This is a serious shortcoming which has the effect of dramatically underestimating the size of PCE-exposed population and overlooking significant contributors to risk.

A. TSCA Requires Risk Evaluations to Address All Pathways of Exposure

Risk evaluations under section 6(b)(4)(A) must determine "whether a chemical substance presents an unreasonable risk of injury to health or the environment" This requirement cannot be met without examining all sources of exposure that contribute to health and environmental risk. Section 6(b)(4)(A) provides that a risk evaluation must determine the substance's risks under "the conditions of use." This broad term spans the entire life cycle of a chemical and is defined under section 3(4) to mean "the circumstances . . . under which a chemical substance is intended, known or reasonably foreseen to be manufactured, processed, distributed in commerce, used or disposed of." Moreover, TSCA section 6(a) requires EPA to take into account "any combination of such activities." These "circumstances" clearly include environmental releases that result in pathways of human exposure, whether or not they might be controlled under other environmental laws.

If Congress had intended a blanket exemption for environmental releases from risk evaluations under section 6(b), it surely would have said so explicitly. But not only is there no such exemption in the law, but its legislative history and structure demonstrate that Congress intended TSCA to provide a comprehensive framework for identifying and managing chemical risks, including those that derive from environmental exposure pathways that are subject to other environmental laws.

When it enacted TSCA in 1976, Congress recognized that then-existing environmental laws were "clearly inadequate" to address the "serious risks of harm" to public health from toxic chemicals. H.R. Rep. No. 94-1341, at 7 (1976); see S. Rep. No. 94-698, at 3 ("[W]e have become literally surrounded by a manmade chemical environment. … [T]oo frequently, we have discovered that certain of these chemicals present lethal health and environmental dangers."). While other federal environmental laws focused on specific media, such as air or water, none gave EPA authority to "look comprehensively" at the hazards of a chemical "in total." S. Rep. No. 94-698, at 2. Congress designed TSCA to fill these "regulatory gaps," S. Rep. No. 94-698, at 1, through a comprehensive approach to chemical risk management that considered "the full extent of human or environmental exposure," H.R. Rep. No. 94-1341, at 6.

In amending TSCA in 2016, Congress sought to promote "effective implementation" of the 1976 law's objectives. See S. Rep. No. 114-67, 114th Cong., 1st Sess. (2015) at 2. Thus, it affirmed that the intent of the original law—to give EPA "authority to look at the hazards [of chemicals] in total," S. Rep. No. 94-698, at 2—remained "intact." S. Rep. No. 114-67, at 7. Indeed, in a statement accompanying the law's passage, its Senate Democratic sponsors underscored that, with the expanded authorities conferred by Congress, TSCA should not be "construed as a 'gap filler' statutory authority of last resort" but "as the primary statute for the regulation of toxic substances."[7] Excluding from risk evaluations all pathways of chemical exposure through air, water and soil would be directly contrary to these Congressional expectations.

EPA's position that other environmental laws should displace TSCA risk evaluations for *all* chemicals arbitrarily assumes that these laws provide equivalent protection of public health and the environment and that there is no added benefit in addressing environmental pathways of exposure under TSCA. But in reality, these other laws vary greatly in the degree of protection they afford against chemical risks and

[7] Congressional Record – Senate 3517 (June 7, 2016).

the extent of their application to unsafe chemicals. In many cases, other laws do not regulate the entire universe of polluting sources. They may also impose controls based not on of risk but on other considerations like cost or available technology. Moreover, the Clean Air Act (CAA), Safe Drinking Water Act (SDWA), Clean Water Act (CWA) and Resource Conservation and Recovery Act (RCRA) are specific to individual media; they do not contemplate or authorize an examination of exposure and risk across media, a responsibility that Congress only conferred on EPA under TSCA. In addition, other EPA authorities are struggling with their workloads and resources and may simply lack the bandwidth to tackle serious chemical risks that do not represent immediate priorities. These limitations are precisely why Congress gave EPA comprehensive authority over chemical risks under TSCA in 1976 and strengthened that authority in 2016.

In the 1976 law, Congress recognized the need to coordinate use of TSCA with implementation of other environmental laws. However, it chose to do so *not* by excluding environmental releases from the purview of TSCA – the approach EPA is arbitrarily pursuing now. Instead, it established a framework for determining, on a case-by-case basis, whether the risks of particular chemicals are best addressed under these laws or under TSCA. Thus, section 9(b)(1) of TSCA provides that EPA may use TSCA regulatory authorities if it "determines, in [its] discretion, that it is in the public interest to protect against [a particular] risk by action taken under this Act" but should use other environmental laws if it determines that "a risk to health or the environment . . . could be reduced to a sufficient extent by actions taken under" these laws.

In 2016, Congress underscored the chemical-specific focus of this analysis by revising section 9(b)(2) so that, in deciding whether to regulate under TSCA or another law, EPA must "consider . . . all relevant aspects of the risk" in question and make a "comparison of the estimated costs and efficiencies" of addressing the risk under TSCA and other laws. Commenting on this language, the law's Senate Democratic sponsors explained that it allowed EPA to regulate under other laws in lieu of TSCA only where the "Administrator has already determined 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 additional actions taken under other EPA authorities."[8]

This approach presupposes that EPA has already used the TSCA risk evaluation process to identify the risks of a chemical and the exposure pathways contributing to those risks and thus has an informed basis to determine whether they "could be eliminated or reduced to a sufficient extent" under another law. However, If EPA has not examined the specific pathways of environmental exposure and their contribution to total risk under TSCA, then it cannot conduct the analysis that section 9(b) requires because it will be unable to evaluate the relative strengths of using TSCA or another law to eliminate the risk. By presuming that other laws are *always* superior to TSCA in identifying and reducing the risks of chemicals in environmental media, EPA's blanket exclusion of environmental releases thus turns section 9(b) on its head.

B. SAAC Reports Strongly Recommend that EPA Address Environmental Pathways of Exposure

[8] Congressional Record – Senate 3517 (June 7, 2016).

As in previous evaluations, EPA has defended the exclusion of general population risk and environmental pathways of exposure on the ground that:[9]

> . . . 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.

This approach is contrary to TSCA, which was clearly intended to provide for a comprehensive examination of risks from all pathways, including those subject to media-specific environmental laws. Moreover, EPA's assumption that environmental pathways of exposure are of lesser concern ignores the significance of these pathways for chemicals like PCE and the importance of accounting for all sources of exposure so that human health risks are not understated.

The SACC has repeatedly raised concerns about EPA's failure to consider environmental pathways of human exposure. In its review of the 1,4-dioxane draft risk evaluation, for example, the SACC said:[10]

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

The SACC added that:[11]

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

The SACC underscored that "[g]eneral human population and biota exposure must be assessed for inhalation, ingestion, and dermal routes [and that] [d]ifferent sub-populations may have different extents of exposure, but each route must be assessed."[12] EPA's narrower approach, it said, "strayed

[9] Draft evaluation at 460.
[10] 1,4-Dioxane and HBCD SACC Report, at 18.
[11] Id.
[12] Id.

from basic risk assessment principles by omitting well known exposure routes such as water consumption by all occupationally and non-occupationally-exposed humans as well as similar exposures to other biological receptors."[13]

The SACC review of the 1-BP draft risk evaluation similarly took EPA to task for failing to consider air emissions and other environmental releases: [14]

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

The SACC report for the methylene chloride evaluation raised similar concerns:[15]

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

The Report added that:[16]

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

The SACC expressed concern that "readers of this Evaluation receive a partial picture of risks, finding for example, that recycling and proper disposal present the only environmental hazards under TSCA" and that "this incomplete picture of risks may be used to promote improper releases and disposal of methylene chloride."[17] The SACC's concerns are based on its expert assessment of the "best available science," which EPA is required to employ in its risk evaluations.

[13] Id.
[14] SACC 1-BP Report at 17.
[15] SACC Methylene Chloride Report at 75.
[16] Id at 15.
[17] Id.

The PCE draft is yet another example of a risk evaluation approach that the SACC has repeatedly and sharply questioned. As described below, the exclusion of PCE environmental pathways is a significant concern because the evidence indicates that these pathways are significant contributors to PCE's risks to human health.

C. The Presence of PCE in Indoor and Outdoor Air is Widespread and Substantial

Because of PCE's volatility and widespread use in open processes, air emissions are a major source of exposure. ATSDR indicates that PCE air releases of 667,902 pounds (~303 metric tons) were reported for the Toxic Release Inventory (TRI) in 2016.[18] As ATSDR notes, the universe of facilities subject to TRI reporting is limited and reported air releases underrepresent the actual total. EPA's National Emission Inventory (NEI) database is more comprehensive. ATSDR reports that, according to the NEI, 2008 and 2011 PCE emissions were 5,318 and 11,138 metric tons, respectively, with the biggest source being PCE's use as a dry-cleaning solvent.[19] Emissions reflected in TRI reporting and the NEI data-base are generally from facilities in concentrated areas that may have multiple sources of PCE as well as other chemicals of concern. Not surprisingly, communities in these areas bear higher pollution burdens than the general population and are therefore PESSs requiring special consideration and protection under TSCA.

ATSDR provides this overview of PCE levels in ambient air:[20]

> Outdoor (ambient) air monitoring studies in the United States have shown tetrachloroethylene concentrations of 400–2,100 ng/m3 (0.059–0.31 ppb) in Portland, Oregon, in 1984 (Ligocki et al. 1985), 5.2 µg/m3 (0.77 ppb) in Philadelphia, Pennsylvania, in 1983–1984 (Sullivan et al. 1985), 0.24–0.46 ppb in three New Jersey cities during the summer of 1981 and the winter of 1982 (Harkov et al. 1984), and 0.29–0.59 ppb in seven cities in 1980–1981 (Singh et al. 1982).

Citing data from the EPA Air Quality System (AQS) database, ATSDR indicates that, "in general, the average concentration of tetrachloroethylene in outdoor air is <1 µg/m3 (0.15 ppb) for the majority of the U.S. locations sampled; however, several 24-hour average values exceeded 1 µg/m3."[21]

The draft risk evaluation acknowledges that "[c]oncentrations of volatile organic compounds, such as PCE, are often higher in indoor air than outdoor air" and summarizes available evidence of PCE indoor air levels as follows:[22]

> EPA identified 19 acceptable studies from the United States and Canada deemed to be in the scope of this risk assessment, which monitored residential or commercial indoor air for PCE

[18] Agency for Toxic Substances and Disease Control, *Toxicological Profile for Tetrachloroethylene,* June 2019 (ToxProfile)
[19] ToxProfile at 266.
[20] Id at 282.
[21] Id. at 283.
[22] Draft Evaluation at 200.

concentrations, for a total of 3172 measured samples. Identified studies were conducted between the years 1980 and 2013. The detection frequency of PCE in the identified studies ranged from 30% to 100% detection, with a median of 95% detection (with 4 studies not reporting detection frequency). Measured PCE concentrations in indoor air ranged from non-detects (detection limits varied) to 94985 ug/m3, with reported central tendency (mean) values ranging from 0.2 ug/m3 to 58348 ug/m3. The maximum air concentration of PCE was measured in a do-it-yourself laundry facility with coin-operated dry-cleaning machines (Howie 1981).

The draft evaluation further notes that, of the studies measuring PCE air concentrations in homes in the United States and Canada, "[c]oncentrations ranged from non-detect (limits varied) to 171 µg/m3. The highest concentration was from the Canadian study (Chan et al. 1990), which sampled air concentration in Canadian residences. The next highest concentration was 78 µg/m3, collected from inner-city homes in New York, New York (Sax et al. 2004). Maximum concentrations of approximately 30 µg/m3 were detected in garages in Boston, Massachusetts (Dodson et al. 2008) and in living areas of industrial, urban, and suburban homes in Michigan (Jia et al. 2008a)."[23]

The evaluation makes no effort to examine the health significance of these indoor levels of PCE. However, the EPA IRIS Assessment provides useful context by identifying air concentrations for different levels of cancer risk:[24]

Air Concentrations at Specified Risk Levels:
Risk Level Lower Bound on Concentration Estimate

E-4 (1 in 10,000) 400 µg/m3
E-5 (1 in 100,000) 40 µg/m3
E-6 (1 in 1,000,000) 4 µg/m3

Although indoor and outdoor PCE levels vary over a wide range, the higher concentrations measured in the above studies present lifetime cancer risks – without considering other sources of exposure – that exceed EPA's 1 X 10^{-6} threshold for unreasonable cancer risk to the general population under TSCA.

D. Dry Cleaners Account for a Significant Portion of General Population Exposure to PCE

As several studies show, higher PCE levels in indoor and ambient air are correlated with elevated exposures from dry-cleaning operations. These exposures take several forms.

- *Families of dry-cleaning workers have elevated PCE exposures.* According to ATSDR, "[i]ndoor air of apartments where dry cleaners lived was about 0.04 ppm compared to 0.003 ppm in the apartments of the controls (Aggazzotti et al. 1994a), indicating that dry cleaners serve as a

[23] Id at 200-201.
[24] EPA, Integrated Risk Information System (IRIS), Chemical Assessment Summary for Tetrachloroethylene (IRIS Summary), 2012, at 34

source of exposure for their families. Breath concentrations of tetrachloroethylene in dry cleaners, family members, and controls were 0.65, 0.05, and 0.001 ppm, respectively (Aggazzotti et al. 1994b)."[25]

- *Members of the public who patronize dry cleaning establishments or pass them on the street have significant PCE exposures.* For example, ATSDR reported that, in "another locality in France, the highest measured concentration of tetrachloroethylene (678 µg/m3; 100 ppb) was found in front of a dry-cleaning shop in the indoor air of a shopping center."[26]

- *Members of the public who use self-service, coin operated laundromats have high PCE exposures.* As cited by ATSDR, a "survey of 15 coin-operated dry cleaning establishments in Hamburg, Germany, showed indoor air concentrations of tetrachloroethylene between 3.1 and 331 mg/m3 (457 and 48,812 ppb) and a concentration of 4.5 mg/m3 (664 ppb) in one building 7.5 months after removal of dry-cleaning machines, indicating that tetrachloroethylene may be absorbed by building materials and then slowly released into the air over time (Gulyas and Hemmerling 1990)."[27] According to the draft evaluation, Howie (1981) measured indoor air PCE concentrations in coin-operated dry cleaning facilities in the United States (6 facilities). PCE was detected in 100% of collected samples, with air concentration range from 508 to 94984 µg/m3.[28] A large number of consumers may frequent coin-operated dry cleaners and likely have high PCE exposures while cleaning their clothes and other belongings.[29]

- *Apartments above dry cleaners can have high PCE concentrations.* According to ATSDR, it "was found that the mean 48-hour average concentration in residences above cleaners that adhered to EPA's regulations was 0.57 mg/m3, while the concentration was 2.1 mg/m3 with cleaners that partially followed EPA's regulations and 2.7 mg/m3 with cleaners with no documentation of adherence to the rules (Garetano and Gochfield 2000). In an older study, elevated levels of tetrachloroethylene were also found in apartments above dry-cleaning facilities (Schreiber et al. 1993). Tetrachloroethylene concentrations ranged from 0.04 to 8.1 ppm in six apartments above dry cleaning facilities when measurements were completed from 7 a.m. to 7 p.m., and from 0.01 to 5.4 ppm when measured from 7 p.m. to 7 a.m."[30] ATSDR also referenced a study of indoor air in Paris in which "[a]nnual levels ranged from 0.6 to 124.2 µg/m3 (0.09–18.3 ppb) in residential homes that were in close proximity to dry cleaning facilities."[31]

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[25] ToxProfile at 291.
[26] Id. at 286.
[27] Id. at 293-294.
[28] Draft Evaluation at 244.
[29] EPA ignores this scenario on the basis that it was not able to "determine if coin operated dry cleaning machines were still in use in the United States." Id. There is no indication of what EPA's inquiries entailed but given the historical operation of many coin-operated dry cleaners, this exposure pathway should have been addressed in the draft evaluation unless EPA could conclusively demonstrate that it no longer exists.
[30] ToxProfile at 298.
[31] Id. at 286. The area source regulations that EPA promulgated under the CAA in 2006 set a goal of closing all PCE-using dry cleaners below residential apartments by the end of 2020.
https://www.epa.gov/sites/production/files/2015-06/documents/fact_sheet_dry_cleaning_july2006.pdf

- *Dry cleaned garments and other fabrics stored in homes release PCE, exposing family members and visitors.* For example, ATSDR references a study that "showed that the storage of newly dry-cleaned garments in a residential closet resulted in tetrachloroethylene levels of 0.5–2.9 mg/m3 (74–428 ppb) in the closet after 1 day, followed by a rapid decline to 0.5 mg/m3 (74 ppb), which persisted for several days (Tichenor et al. 1990)."[32]

The draft EPA evaluation addresses the last scenario[33] but ignores the other four even though they result in significant acute and chronic exposures.

To put these exposures in context, it is useful to examine a 2005 risk assessment by EPA's Office of Air and Radiation (OAR), which calculates cancer risks of PCE dry cleaner emissions to the general population and persons co-residing with dry cleaning facilities. Using Unit Risk Estimates (UREs) of the State of California and EPA's TSCA office, EPA determined the number of people in the US with cancer risks of 1 in one million or more: [34]

Table 14: NATA-Derived Population Risk for Free-Standing Area Source PCE Dry Cleaners

Maximum Individual Cancer Risk (per million)

Dose-Response Value	**100 in a million**	**10 in a million**	**1 in a million**
OPPTS	0	0	970,000
Cal EPA	0	400,000	56,000,000

Even with application of emissions controls, EPA found that the cancer risks remained significant:[35]

[32] Draft Evaluation at 293.
[33] The draft evaluation estimates consumer exposures and risks for contact with PCE-treated articles at pp. 233-43 and 398-399, concluding that acute neurotoxicity risks from dermal exposure (but not inhalation) are below the benchmark Margin of Exposure (MOE) and hence unreasonable.
[34] EPA Office of Air Quality Planning and Standards (OAQPS), *Perchloroethylene Dry Cleaners Refined Human Health Risk Characterization*, November 2005 at 20.
[35] Id., at 22.

Table 19: Estimated Maximum Individual Cancer Risk for Area Sources by Machine Type and Control Option using the Cal EPA URE

Machine Type	***Control Option and Maximum Individual Cancer Risk (per million)***		
	GACT-level	Leak Detection and Repair	LDAR+Secondary Controls
Transfer	340	280	160
Vented	310	250	160
Refrigerated Condenser	220	170	160
Refrigerated Condenser and Carbon Adsorber	190	160	--

Importantly, these risk estimates only reflect the impacts of releases to air from dry cleaners. Other emission sources like open-top vapor degreasing add to ambient levels of PCE and, combined with dry cleaning sources, could result in a larger cancer risk to the general population.

EPA also estimated the inhalation individual cancer risks posed by dry cleaners co-located with residences, assuming lifetime exposures at 5th percentile, median, geometric mean, 95th percentile and maximum measured indoor PCE concentrations:[36]

Table 20: Summary of Co-Residential Area Source Inhalation Cancer Risk

	Estimated Lifetime Cancer Risk (per million)	
Distribution of Exposure	Cal EPA URE	OPPTS URE
Lower 5th Percentile[49]	30	4
Median	100	10
Geometric Mean	200	20
Upper 95th Percentile	4,100	500
Maximum	30,000	4,000

Although these estimates may overstate risks and do not necessarily reflect current conditions,[37] they clearly illustrate how EPA's exclusion of dry cleaner emissions from its draft removes significant subpopulations with elevated cancer risk from the PCE evaluation.

[36] Id., at 23.

[37] For example, the Cal EPA URE is considerably larger than the Inhalation Unit Risk (IUR) used in the 2012 EPA IRIS Assessment and the TSCA draft risk evaluation.

E. The Draft Risk Evaluation Fails to Consider Exposure to PCE In Drinking Water

The problem formulation for EPA's risk evaluation indicates that PCE "is a common contaminant in municipal drinking water supplies and ground water, with some of the highest measured concentrations in ground water occurring near perchloroethylene contaminated sites."[38] ATSDR comments that PCE "has been detected in most drinking water, groundwater, surface water, and rainwater supplies" and that "[t]ap water may be an important source of exposure . . . Three percent of the water supply systems that use well water contain ≥0.5 µg/L (≥0.5 ppb) tetrachloroethylene (WHO 2003)."[39]

In addition to groundwater contamination from dry cleaning and industrial sites, PCE "can be released into drinking water by leaching from liners in pipes, as in the case of contaminated water in New England." As ATSDR explains: [40]

> The liners were installed to asbestos cement pipes to take away a foul taste in the water (Larson et al. 1983). They were comprised of vinyl plastic and tetrachloroethylene. The manufacturers expected tetrachloroethylene to volatilize from the pipe after they administered the compound; however, it stayed in the coating and was found to progressively leach into the drinking water (Aschengrau et al. 2003). Tetrachloroethylene was present at concentrations ranging from 1.5 to 7,750 µg/L in Cape Cod, Massachusetts, and was reduced to 40 µg/L after bleeding and flushing the pipes (Aschengrau et al. 2012).

The pervasive presence of PCE in drinking water is well-documented. As ATSDR has summarized the results of drinking water monitoring:[41]

> Williams et al. (2002) reported annual levels of tetrachloroethylene measured in 3,422–4,218 California drinking water sources between 1995 and 2001. Approximately 10–13% of the sampled drinking water sources contained detectable levels over this 7-year period. The average annual detected concentration of tetrachloroethylene ranged from 17.0 µg/L (2000) to 28.0 µg/L (1998).
>
> Tetrachloroethylene and several other VOCs have been detected at high levels in drinking water at the Camp Lejeune, Marine Corps Base in North Carolina (ATSDR 1998, 2013). Tetrachloroethylene levels in tap water were shown to range from <1 to 215 µg/L (ppb), and groundwater levels as high as 170,000 µg/L (ppb) were observed in 1985. A recent historical reconstruction study of this site, which applied additional modeling methods, reported a maximum monthly average concentration of 183 ppb (Maslia et al. 2016).
>
> Tetrachloroethylene was monitored in a comprehensive survey conducted by the USGS of VOCs in private and public groundwater wells used for drinking water (USGS 2006). Tetrachloroethylene was identified in approximately 4% of 3,498 aquifer samples at a median

[38] PCE Problem Formulation at 41.
[39] ToxProfile at 271.
[40] Id at 271-272.
[41] Id. at 288.

concentration of 0.090 µg/L for the samples having positive detections. The percentage of samples exceeding the 5 µg/L MCL was 0.70% (USGS 2006). In an analysis of domestic groundwater wells, the median concentration of tetrachloroethylene was reported as 0.058 µg/L for samples having positive detections.

The mean detected concentration of tetrachloroethylene in the drinking water of California was 3–6 times higher than the MCL of 5 µg/L from 1995 to 2000 (Williams et al. 2002).

While detected levels of PCE vary over a wide range, some exceed the EPA Maximum Contaminant Level (MCL) for PCE of 5 µg/L (ppb) by a significant margin. EPA's own review of monitoring by drinking water systems confirms the prevalence of MCL violations. According to the PCE problem formulation, "EPA's Second Six-Year Review Contaminant Occurrence Data . . . showed perchloroethylene occurrence in 2.5% of roughly 50,000 public water systems, with thirty-six states reporting drinking water systems with at least one detection above the maximum contaminant level (MCL: 5 µg/L)."[42] Using EPA's occurrence data, EWG reported the presence of PCE in water supply systems serving 24 million people in 47 states.[43]

The PCE MCL was set in 1991 based on existing limits of detection and is likely not protective in light of the new information on and analysis of PCE's health effects.[44]

As ATSDR notes, "[s]howering or bathing with contaminated water can also result in tetrachloroethylene exposure." In one paper, Rao and Brown (1993) described a combined PBPK exposure model that estimates brain and blood levels of tetrachloroethylene following a 15-minute shower or 30-minute bath with water containing 1 mg tetrachloroethylene/L. Based on the model, "Rao and Brown (1993) estimated that shower air would contain an average of 1 ppm and that the air above the bathtub would contain an average of 0.725 ppm if the water contained 1 mg tetrachloroethylene/L."[45] Lower levels of PCE in the water used for showering or bathing would result in lower concentrations in air, but inhalation exposure could still be significant.

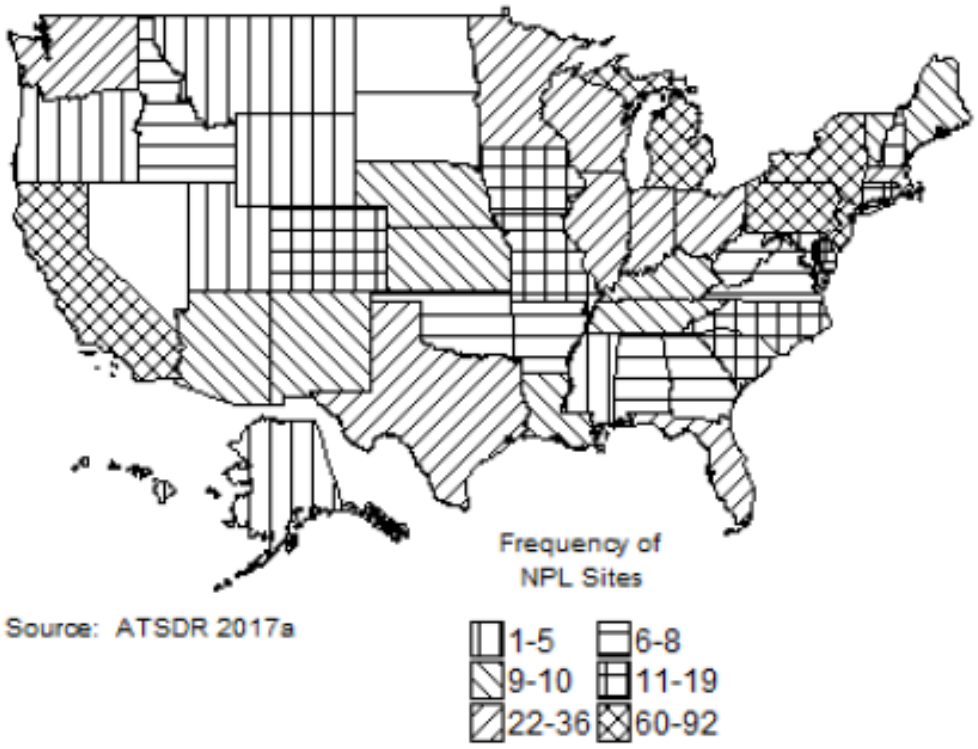
F. PCE Is Frequently Found at Contaminated Sites, Resulting in Contamination of Groundwater and Release of PCE Vapors into Ambient Air and Buildings

PCE is a significant concern at contaminated sites within the purview of the EPA Superfund program. ATSDR reports that PCE "in at least 949 of the 1,854 hazardous waste sites that have been proposed for

[42] PCE Problem Formulation at 41.
[43] https://www.ewg.org/tapwater/contaminant.php?contamcode=2987
[44] For example, In August 2001, the California Office of Environmental Health Hazard Assessment (OEHHA) established a Public Health Goal (PHG) of 0.06 µg/L for PCE in drinking water. The PHG was based on cancer studies in laboratory animals and designed to limit the lifetime cancer risk of drinking water consumers to no more than 1 X 10^{-4}. Office of Environmental Health Hazard Assessment California Environmental Protection Agency, *Public Health Goal for Tetrachloroethylene in Drinking Water,* August 2001, at https://oehha.ca.gov/media/downloads/water/chemicals/phg/pceaug2001.pdf.
[45] ToxProfile at 263.

inclusion on the EPA National Priorities List (NPL)."[46] ATSDR depicts the geographic distribution of these sites as follows:

Figure 6-1. Frequency of NPL Sites with Tetrachloroethylene Contamination



Only a small minority of contaminated sites are listed or proposed for listing on the NPL. There are undoubtedly far more sites with PCE contamination than those identified in the figure above.

Contaminated sites are often the result of spills and leaks from dry cleaning facilities and industrial operations such as degreasing. Once mixed with soil, PCE does not readily volatilize but may instead be rapidly transported into groundwater by leaching through soil fissures.[47] ATSDR notes that "a considerable number of monitoring studies have detected tetrachloroethylene in groundwater," reflecting its mobility in soil.[48] According to EPA's problem formulation, "[h]istoric industrial, commercial, and military use of perchloroethylene, including unregulated or improper disposal of perchloroethylene wastes . . . have resulted in location-specific soil and groundwater contamination."[49]

[46] ToxProfile at 305.
[47] Id. at 279.
[48] Id. at 277.
[49] PCE Problem Formulation at 42.

Leaching of PCE to groundwater at contaminated sites is a major pathway for drinking water contamination, as discussed above.

A large volume of PCE-containing waste from ongoing industrial activities is actively managed at production and use sites as well as landfills and waste treatment facilities. As described in the PCE problem formulation, TRI reports for 2015 showed that "27 facilities reported a total of 65 million pounds of perchloroethylene waste managed. Of this total, roughly 46 million pounds were recycled, 2.3 million pounds were recovered for energy, 15 million pounds were treated and 1.18 million pounds were released into the environment."[50] Since TRI reporting requirements do not apply to many facilities, the actual volume of PCE-containing waste at industrial sites, treatment facilities and landfills is likely much greater. This PCE may volatilize and contribute to ambient air exposure or be spilled and leaked, creating pathways for contamination of soil, surface water and groundwater.

Like trichloroethylene (TCE), carbon tetrachloride (CCl4) and other volatile organics found at contaminated sites, PCE when vaporized is known to "intrude" into nearby buildings, contributing to elevated levels in indoor air inhaled by occupants. PCE vapor intrusion has been reported at a number of contaminated sites. ATSDR underscores the significance of this pathway for human exposure:

> The concept of vapor intrusion was introduced in the late 1990s. It was previously thought that contaminated water was a threat only when the groundwater was used as drinking water. In 1979, 4,100 gallons of 1,1,1-trichloroethlyene were spilled in the Village of Endicott, New York. Tetrachloroethylene was one of the many chemicals found in the groundwater analysis after the spill; however, the compound was not present because of the spill, but rather from previous spills and releases.

EPA has repeatedly acknowledged the risks associated with vapor intrusion of volatile solvents like PCE and has published guidance governing the calculation of vapor intrusion risks.[51]

G. EPA Fails to Consider Exposure to PCE-Containing Biosolids

PCE is "released to surface water and land in sewage sludge."[52] However, EPA asserts that "risks would not be evaluated for land-applied biosolids because PCE is currently being addressed in the Clean Water Act (CWA) regulatory analytical process."[53] In particular, EPA alleges that because "PCE has been identified in biosolids biennial reviews under the CWA,"[54] biosolid exposures can be excluded from the TSCA risk evaluation. This assertion is incorrect.

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[50] Id at 37.
[51] *See* EPA, *OSWER Technical Guide for Assessing and Mitigating the Vapor Intrusion Pathway from Subsurface Vapor Sources to Indoor Air* (June 2015) ("EPA Vapor Intrusion Guidance"), https://www.epa.gov/sites/production/files/2015-09/documents/oswer-vapor-intrusion-technical-guide-final.pdf
[52] ToxProfile at 300.
[53] Draft Evaluation at 38.
[54] Id. at 460.

As described above, TSCA does not authorize EPA to ignore the exposure pathways associated with a chemical's conditions of use merely because such exposures are, or may be, regulated under other laws as well. Moreover, as EPA admits, the CWA does not regulate PCE levels in biosolids. Instead, EPA solely claims that PCE "has been identified in biosolid biennial reviews."[55] The mention of PCE in a biennial review does have any regulatory significance; instead, biennial reviews are used to identify chemicals in biosolids that may warrant further research to determine whether or not to regulate them. EPA fails to mention that PCE was first included in a CWA biennial review in 2005, and EPA has not taken or proposed any measure to regulate PCE in biosolids in the 15 years since then.[56] This inaction is not unique to PCE; according to EPA's Inspector General, "[i]n over 20 years, no new pollutants have been regulated" under the Clean Water Act's biosolids authority.[57] EPA's claim that "PCE is currently being addressed in the Clean Water Act regulatory … process" is simply incorrect.

H. By Failing to Account for Environmental Pathways, EPA Disregards Subpopulations with Higher PCE Exposures and Elevated Risks

This survey of PCE environmental releases demonstrates the important contribution of PCE air emissions and contaminated groundwater, drinking water and soil to overall PCE exposure. Some of these pathways are alone responsible for cancer and non-cancer risks to large segments of the population. As discussed above, risks to residents of areas with elevated air concentrations from dry cleaners or vapor degreasing operations exceed EPA unreasonable risk benchmarks even without considering other sources of exposure. This same is true for consumers of drinking water containing high PCE levels.

Moreover, in reality, PCE exposure occurs by multiple pathways simultaneously for many subpopulations, and, depending on the circumstances, can greatly exceed general population exposure levels. An example is an urban neighborhood that is in close proximity to dry cleaners, high-emitting industrial facilities and NPL sites and whose residents consume PCE-contaminated drinking water. Individuals living in these communities would inhale elevated PCE levels in indoor and outside air, ingest additional PCE in drinking water and inhale PCE volatilized during bathing and showering. The higher exposure levels from these multiple sources would make the community a PESS, for which EPA must make a specific unreasonable risk determination under TSCA.

Some community members might also work in PCE processing or manufacturing facilities and/or use PCE-containing consumer products, adding to environmentally related exposures and thus increasing likely risks. This subset of the community would also comprise a PESS which requires a specific assessment of unreasonable risk. For both PESSs, the combination of exposure sources would likely result in MOEs well below benchmark MOEs for non-cancer endpoints and cancer risks far above 1×10^{-6}.

A comprehensive risk evaluation as required by TSCA would identify these PESSs, estimate total exposure from all sources and characterize the increased risk resulting from concurrent exposure

[55] Id.

[56] https://www.epa.gov/sites/production/files/2019-06/documents/2016-2017-biosolids-biennial-review.pdf

[57] https://www.epa.gov/sites/production/files/2018-11/documents/_epaoig_20181115-19-p-0002.pdf

pathways. However, because of its narrow scope, the draft TCE evaluation fails to provide this analysis and therefore presents an unrepresentative and incomplete picture of PCE's risks to the public.

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

TSCA is the only law administered by EPA that provides a mandate and comprehensive authority to examine chemical risks from all conditions of use, and thus across all pathways of exposure. It is clear that Congress viewed this unique strength of TSCA as an essential tool in protecting against the cross-media effects of chemicals like PCE on human health and the environment. EPA is obligated to use this tool as Congress directed.

II. EPA Fails to Address the Risks of Chronic PCE Exposure by Consumers

The draft EPA evaluation only addresses acute neurotoxic risks of PCE to consumers. No risks to consumers are addressed from chronic health effects linked to PCE, including cancer, developmental and reproductive toxicity, neurotoxicity and liver and kidney toxicity. These chronic endpoints are only evaluated for workers. This approach creates the misleading impression that consumers are not at risk for serious chronic health effects – an impression contradicted by multiple lines of evidence.

A. Consumers Have Long-term Chronic Exposure to PCE

Multiple measurements of ubiquitous indoor air concentrations of PCE (some at extremely high levels) indicate that consumer exposure to PCE is not episodic but continuous. Along similar lines, consumers using contaminated drinking water are also exposed to PCE on an ongoing basis, adding to exposures to PCE from indoor air.

Another line of evidence is the presence of PCE in human blood, urine and breath samples in multiple studies described in the draft risk evaluation.[58] The most comprehensive source of data on PCE levels in blood is the National Health and Nutrition Examination Survey (NHANES) conducted by CDC's National Center for Health Statistics (NCHS). According to EPA, at the 95th percentile, blood concentrations ranged from 9.4E-02 µg/L (2007-2008) to 1.9E-01 µg/L (2001-2002). For 1999-2004 (n=2577), the mean sample concentration was 8.1E-02 µg/L, and the median sample concentration was 3.4E-02 µg/L.

The risk evaluation also cites a study (Sexton et al. 2005) that measured concentrations of PCE in whole blood from 150 children from two poor, minority neighborhoods in Minneapolis, Minnesota in four periods during 2000-2001. PCE was detected in 37 to 63% of the samples, with concentrations ranging

[58] Draft Evaluation at 107.

from 2.0E-02 – 3.0E-02 ng/mL (10th percentile) to 0.1-0.8 ng/mL (99th percentile). The draft evaluation also reports that blood samples were collected as part of the National Human Exposure Assessment Survey (NHEXAS) Phase I conducted by EPA (Clayton et al. 1999). Samples were collected from 147 people in six states (IL, IN, OH, MI, MN, and WI) in 1995-1997. PCE was detected in 37% of the samples, with a mean of 0.2 ng/mL, a 50th percentile of 5.0E-02 ng/mL, and a 90th percentile of 0.1 ng/mL. Summarizing these data, EPA states that "PCE concentrations in blood were similar between the NHANES, SHIELD, and NHEXAS surveys conducted between 1995 and 2016."

In addition to blood samples, NHANES collected urine samples from males and females ages 6+ years for the PCE metabolite N-Acetyl-S-(trichlorovinyl)-L-cysteine. Although for the survey years 2011-2012 all samples measured were below the detection limit of 3.0 µg/L, EPA indicates that "the NHANES urine metabolite data for PCE was also used in a 2015 study analyzing the reported data to develop means and other descriptive statistics (Jain, 2015)." In that paper, "[t]he mean concentration for male children was reported as 6.9 ng/mL and 6.4 ng/mL for female children. The 95% confidence interval around the mean was reported as 5.8 to 8.4 ng/mL for male children and 5.2 to 8.0 ng/mL for female children."

The draft evaluation also references PCE breath samples collected as part of the Total Exposure Assessment Methodology (TEAM) Study (Wallace 1987). As described by EPA,[59] arithmetic means for PCE measured in these samples ranged from 8.3 to 13 µg/m3, with detection in 58 to 100% of samples. Another study of breathing zone PCE concentrations, likewise described by EPA, was conducted by NHANES and produced the following results:[60]

> The highest concentration was observed in NHANES survey data from 1999-2000 (Jia et al. 2008a). The study notes that two participants had exposure to highly elevated levels of PCE; one participant spent more time than usual at work/school and the other participant worked with paint thinners, brush cleaners, or strippers as well as glues, adhesives, hobbies or crafts, and also reported having new carpet installed in the past 6 months. The 95th percentile concentration for the NHANES study was 18.5 µg/m3.

Finally, PCE has been found in human breast milk. As noted by ATSDR, PCE "was present at unspecified levels in seven of eight samples of mother's milk from four urban areas in the United States (Pellizzari et al. 1982)" and a "woman in Halifax, Nova Scotia, who visited her husband daily at the dry cleaning plant where he worked, was found to have [PCE] present in her breast milk."[61] According to ATSDR, using a PBPK model, "Schreiber (1993) predicted that for women exposed under occupational conditions, breast milk concentrations would range from 857 to 8,440 µg/L. The exposure scenarios for the low concentrations were 8 hours at about 6 ppm (exposure concentration of counter workers, pressers, and

[59] Id at 107-108.
[60] Id at 204.
[61] ToxProfile at 296.

seamstresses) and 16 hours at 0.004 ppm (residential background), and for the high concentration, exposure scenarios were 8 hours at 50 ppm and 16 hours at 0.004 ppm (residential background)."[62]

The consistent detection of PCE in human blood, urine, breath, and breast milk is incompatible with the assumption that consumer exposure is short-term and episodic. Instead, it provides strong evidence of continuous exposure to PCE by consumers, probably from multiple sources. Reinforcing this conclusion is the relatively short elimination half-life of PCE: according to the draft risk evaluation, "[h]alf-life of PCE from blood-rich tissues, muscle, and adipose tissue is 12-16 hours, 30-40 hours, and 55-65 hours, respectively."[63] Moreover, PCE is not persistent and has a low potential for bioaccumulation. [64]

B. Many PCE-Containing Consumer Products are Used Repeatedly and Concurrently with Other Products and Result in Chronic Exposure

Without considering the evidence of chronic exposure described above, the draft risk evaluation claims that "consumer exposure scenarios are expected to be intermittent and it is unlikely that the expected use patterns would cumulatively" result in repeated, ongoing exposure.[65] EPA's assertion that PCE exposure is "sparse and intermittent . . .for the vast majority of users" is based on unrealistic assumptions about real-world use of PCE-containing consumer products.

The draft evaluation identifies 24 separate categories of PCE-containing products for which EPA developed 15 different use scenarios.[66] In addition, EPA examined PCE exposure from dry-cleaned articles in homes. Some of these products (degreasers, adhesives, sealants, aerosol coatings and primers) would be expected to be used regularly by hobbyists, artists who work at home or home renovators. Others (stain removers, mold cleaners, carpet cleaners, marble and stone cleaners) would be applied frequently during normal household cleaning and maintenance. A large category of products (lubricants and greases, parts cleaners, engine degreasers and brake cleaners) would be used frequently by consumers who maintain and repair their own or friends' vehicles. And many consumers regularly patronize dry cleaners and bring dry cleaned clothing home, resulting in repeated residential releases of PCE.

Moreover, EPA's draft evaluates exposures on a product-specific basis and assumes use of a single product type during a day, not multiple products. This is unrealistic: many consumers likely use different

[62] Id. EPA does not discuss the presence of PCE in human breast milk in its draft risk evaluation. However, as ATSDR's summary of relevant studies indicates, PCE in human breast milk not only demonstrates ongoing exposure by mothers but is a source of exposure by infants who are breast feeding. Infants are a PESS under TSCA, so EPA should be examining the risks of PCE to infants through the pathway of the breast milk of their mothers.
[63] Draft Evaluation at 260.
[64] Id. at 252. Even if PCE had a much longer elimination half-life and was bioaccumulative, this would not negate a finding of chronic exposure, although it could mean that PCE is building up in the body as a result of recurring short-term exposure events.
[65] Id at 136.
[66] Id. at 386-398.

PCE-containing products on the same day or over time. To ignore this scenario is to overlook the additional consumer exposure resulting from multiple product use.

EPA itself expresses doubts about its consumer use scenarios, noting that "there is uncertainty whether chronic risks may be of concern for consumers at the very high end of the range for frequency of use, especially if a product is used several days consecutively."[67] EPA also acknowledges that:[68]

> [T]here is a growing consumer practice to complete projects or activities as do it yourselfers. Do it yourself activities could lead to an increased frequency of product use as well as using more than one product containing a chemical of concern within a given day. These and other factors associated with do it yourself activities could result in underestimating consumer exposure concentrations modeled in this evaluation for the do it yourself consumer.

In its report on the TCE evaluation, the SACC "disagreed with EPA's decision not to characterize chronic risks for consumers." As it explained:[69]

> Several Committee members suggested that some consumers are likely to be exposed more frequently and more pervasively to emissions from these products than indicated by the Westat survey data (U.S. EPA, 1987). Firstly, certain high-exposed consumers (hobbyists, home businesses, etc.) are likely to use more than one trichloroethylene-containing product on the same day and/or multiple and consecutive days. Secondly, the Westat survey was unlikely to capture the true distribution of use frequency for high-end users (i.e., oversampling these subpopulations would have been required to obtain a reliable estimate of use patterns for these individuals). Thirdly, it is likely that contributions to indoor air concentrations (and, therefore, exposures) persist for longer periods of time than assumed by EPA from sources such as carpet spot cleaners and fabric sprays (see also, for example, Doucette et al., 2018; Gorder and Dettenmaier, 2011)"

Even if EPA were correct that chronic consumer exposures only occur "at the very high end of use frequency," this would not justify ignoring chronic risks to consumers. Heavy users of PCE-containing consumer products would qualify as a PESS and under TSCA EPA must address risks to such high-exposure groups and determine if they are unreasonable. Treating these groups as irrelevant, as EPA has done, violates TSCA. Moreover, while EPA has no evidence to justify concluding that chronic consumer exposure is rare and infrequent, it has extensive evidence that such exposure is ongoing and continuous. A glaring disconnect in EPA's draft evaluation is that it acknowledges and discusses the presence of measurable PCE levels in indoor air, human blood, urine and breast milk and personal breathing zones but ignores this information in developing consumer exposure scenarios, which are based entirely on modeling

[67] Id at 402.
[68] Id at 245.
[69] TSCA Science Advisory Committee on Chemicals Meeting Minutes and Final Report No. 2020-4 Peer Review for EPA Draft Risk Evaluation for Trichloroethylene (TCE), June 1, 2020, at 58.

of isolated releases from individual products and not on the best evidence of cumulative exposure by consumers.

C. EPA's Claimed Inability to Evaluate Chronic Health Risks to Consumers Is a Failure to Use the Best Available Science as Required by TSCA

EPA also asserts that it cannot in any case account for chronic consumer exposure because "it is unknown how the available toxicological data relates to the human exposures expected in consumer exposure scenarios" and "[t]here is uncertainty regarding the extrapolation from continuous studies in animals to the case of repeated, intermittent human exposures."[70] This is a feeble excuse for failing to address health risks to consumers that are plainly of concern. It is typical for chemical use scenarios to involve repeated but not continuous exposure. Risk assessors have previously had no trouble using repeated dose toxicity studies to estimate the long-term health risks of these scenarios. indeed, PCE industrial and commercial use scenarios likely involve fluctuations in exposure over time based on worker practices and job responsibilities. Nonetheless, EPA estimates chronic health risks for these use scenarios in its draft evaluation. Its failure to develop similar risk estimates for chronically exposed consumers constitutes a failure to use the "best available science" required under TSCA.

EPA could construct chronic exposure scenarios for PCE-exposed consumers on the basis of central tendency and upper bound PCE concentrations in indoor air and personal breathing zones. It could also undertake PBPK modeling using biomonitoring studies showing PCE levels in blood and urine. These methods would allow for a calculation of steady-state PCE exposures that account for day-to-day variations in exposure, much as EPA does in estimating worker exposures and risks. EPA could also modify representative steady-state exposure calculations to account for high-end PESS exposure scenarios, such as intensive and recurring consumer product use, proximity to dry cleaners or high-emitting industrial or commercial facilities, vapor intrusion from contaminated sites, or families with dry cleaning workers who expose other family members to PCE.

EPA must estimate risks to PCE-exposed consumers from the chronic health endpoints of cancer, neurotoxicity, developmental and reproductive toxicity, and kidney and liver effects that the draft evaluation attributes to PCE. Failure to address these risks would be a departure from the best available science and a glaring gap in public health protection that defeats the goals and requirements of TSCA.

III. EPA Has Failed to Combine Risks Across Routes and Pathways of Exposure and Conditions of Use

A. EPA Should Combine Exposures Across Dermal and Inhalation Routes

Like past evaluations, the PCE draft does not combine dermal and inhalation exposure to derive composite risk estimates even though these two routes of exposure occur simultaneously for workers and consumers

[70] Id at 386.

in most PCE use scenarios. Since inhalation and dermal risks are significant in their own right for most PCE conditions of use, the failure to combine exposure across these routes results in a significant understatement of risk.

The PCE evaluation explains that:[71]

> Exposures to PCE were evaluated by inhalation and dermal routes separately. Inhalation and dermal exposures are assumed to occur simultaneously for workers and consumers. EPA chose not to utilize additivity of exposure pathways at this time within a condition of use because of the uncertainties present in the current exposure estimation procedures and this may lead to an underestimate of exposure.

EPA offered a different and conflicting explanation in the draft TCE evaluation:[72]

> In this risk evaluation, EPA determined 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.

EPA's claim that aggregating dermal and inhalation exposure could "inappropriately overestimate total exposure" is puzzling and counter-intuitive; in its draft evaluation for methylene chloride, EPA in fact said that failure to combine the two routes "may lead to an underestimate of exposure."[73] EPA's apparent concern is that combining exposures from the two routes could result in double-counting dermal exposures because a large portion of these exposures are not absorbed through the skin but volatilized and inhaled. However, elsewhere in its evaluation, EPA has based estimates of dermal risk on the percentage of PCE absorbed through the skin. Any amount of PCE absorbed through the skin will necessarily not be inhaled and thus not be counted in modeling or measuring inhalation exposure. The more realistic concern is not that combining these concurrent sources of exposure will overstate risk but that failing to combine exposure across dermal and inhalation routes will unjustifiably lower estimates of risk.

In its report on the draft evaluation for 1-bromopropane (1-BP), the SACC recommended that EPA estimate "cumulative exposures, which involves both dermal and inhalation contact with 1-BP" because "dermal exposure to 1-BP would most likely correspond with simultaneous inhalation exposure" and "vapor and dermal exposures are not separable."[74] EPA should similarly use combined dermal and inhalation exposures to determine PCE's risks in its final evaluation.

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[71] Id. at 32. EPA does not explain what it means by "the uncertainties present in the current exposure estimation procedures" or why these "uncertainties" would preclude combining dermal and inhalation exposures.
[72] TCE Risk Evaluation at 352-353.
[73] Methylene Chloride Risk Evaluation at 304.
[74] TSCA Science Advisory Committee on Chemicals Meeting Minutes and Final Report No. 2019-03,

B. EPA Should Combine Exposure Across Pathways

As discussed above, exposure to PCE results from its presence in ambient and indoor air, in drinking water, and at waste management facilities and contaminated sites. For many subpopulations, these sources of exposure are additive and should be considered in combination when determining overall risk. EPA's decision to ignore the contribution of environmental pathways of exposure to overall risk not only violates TSCA but results in a fragmented and incomplete understanding of PCE's human health impacts. No individual environmental law enables EPA to combine exposure across environmental media and, if TSCA is not used for this purpose, the cross-media risks of chemicals like PCE will not be addressed.

Combining exposures across pathways is not only important for the general population but is a necessary step in adequately protecting PCE-exposed workers and users of PCE-containing consumer products. For workers, job-related PCE exposures will be magnified by environmental sources of exposure and, in many cases, residential exposures. For example, in the home environment, workers may frequently use PCE-containing household products, such as degreasers, spot removers, adhesives and sealants. Workers may also do weekend work or have a side business using the same skills – and the same PCE-containing products – as during their weekday work. They may have additional exposure to PCE when frequenting dry cleaners, storing and wearing dry-cleaned garments or living above dry cleaning establishments. Families of workers may also have "take home" exposures, i.e. contact with the worker's contaminated clothing or skin. As noted above, for example, it is well-documented that PCE levels are elevated in the homes of dry-cleaning employees.

For these subpopulations, PCE-related risks would be a function of the total contribution of each activity and pathway to total exposure. However, the draft evaluation looks at each exposure pathway in isolation from others, thus ignoring the large number of people with concurrent exposure to PCE in the workplace, from the ambient environment and at home. In its TCE evaluation, EPA defended failing to "consider aggregate exposure among individuals who may be exposed both in an occupational and consumer context" on the basis that "there is insufficient information reasonably available as to the likelihood of this scenario or the relative distribution of exposures from each pathway."[75] However, lack of perfect information cannot excuse ignoring risk pathways of obvious concern. EPA could make reasonable assumptions about the number of people with concurrent workplace and consumer exposure to PCE and develop a range of exposure scenarios for these overlapping populations based on its exposure assessments for different industrial and commercial uses and consumer products. These scenarios would enable EPA to identify subpopulations with elevated risks because of the convergence of multiple exposure pathways and estimate the impact of these pathways on overall risk. In this way, EPA would meet its obligation under TSCA to define PESS and protect them from unreasonable risks.

Peer Review for the United States Environmental Protection Agency (EPA) Draft Risk Evaluation for 1-Bromopropane (1-BP) (SACC Report on 1-BP), December 12, 2019, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0235-0061, at 47, 73.

[75] Draft TCE Evaluation, at 353.

The SACC report on the 1-BP evaluation indicates that:

> The Committee found that the draft risk evaluation failed to consider cumulative or aggregate exposures. It was pointed out that a worker who is occupationally exposed may also be exposed through other conditions of use in the home. Yet, these exposures are decoupled in the draft risk evaluation. The Committee was concerned that 1-BP off-gassing from insulation in home and schools is inadequately assessed, thereby underestimating exposures.[76]

TSCA requires EPA to consider all exposures associated with a chemical's known, intended and reasonably foreseen conditions of use.[77] It also requires EPA to separately evaluate whether there is unreasonable risks to subpopulations that face greater exposures than the general public, including people who are exposed to PCE by multiple routes, both on the job and at home and from the ambient environment.[78] EPA must include this analysis in its final PCE evaluation.

IV. EPA Fails to Account for Subpopulations with Greater Susceptibility to PCE's Health Effects and Applies Inadequate Uncertainty Factors to Protect these PESSs

A. The Draft Evaluation Does Not Evaluate the Degree of Increased Risk to PESSs and Fails to Determine Whether They are at Unreasonable Risk as Required by TSCA

PESSs are defined in section 3(12) of TSCA as groups within the general population who are at greater risk because of higher levels of exposure or greater susceptibility. In addition to the many subpopulations with elevated exposures to PCE as described above, EPA fails to account for subpopulations that are more likely to be harmed by PCE exposure because they are more susceptible to its adverse health effects.

Infants are identified in TSCA as a possible PESS and they and fetuses are susceptible subpopulations for PCE. The PCE IRIS assessment indicates that "In utero, lipophilic substances are known to cross the placental barrier" and "[t]here is biological plausibility of transfer of [PCE] across the human placental barrier as [PCE] has been measured in fetal blood and amniotic fluid in rodents."[79] In human and animal studies, PCE has caused "implantation losses, increased incidence of total malformations, decreased fetal weight, increased incidence of skeletal retardations or delayed ossification, and/or decreased postnatal survival."[80] IRIS also indicates that, "[f]or some infants the primary route of exposure may be through breast milk ingestion . . . while for other infants the dose received through ingestion of breast milk will become insignificant when compared with inhalation exposure."[81] According to the draft risk evaluation, "infants fed by formula may also experience increased PCE exposure if PCE is present in

[76] SACC Report on 1-BP Evaluation at 16.
[77] 15 U.S.C. §§ 2602(4), 2606(b)(2).
[78] 15 U.S.C. §§ 2602(12), 2606(b)(2).
[79] IRIS, Toxicological Review of Tetrachloroethylene (Perchloroethylene), February 2012, (IRIS Assessment) at 4-409.
[80] Draft Evaluation, at 268-69.
[81] IRIS Assessment at 6-14.

drinking water supplies."[82] IRIS indicates that the "neurological effects of tetrachloroethylene may constitute the most sensitive endpoints of concern for noncancer effects, and limited data show that early life-stages may be more susceptible to visual deficits than are adults."[83]

The draft evaluation identifies several other subpopulations with greater susceptibility to PCE:[84]

> Factors affecting susceptibility examined in the available studies on PCE include lifestage, biological sex, genetic polymorphisms, race/ethnicity, preexisting health status, lifestyle factors, and nutrition status. PCE is lipophilic and accumulates in fatty fluids and tissues in the human body (Section 0). Additionally, the PCE half-life is substantially higher in adipose tissue compared to others (55-65 hours in adipose, <12-40 hours in others, see Section 3.2.2.1.3). Subpopulations that may have higher body fat composition, and therefore may be more highly exposed to sustained internal PCE concentrations/doses, include pubescent and adult women (including women of child-bearing age) as well as any individual with an elevated body-mass-index. Based on evidence of developmental toxicity from PCE exposure, pregnant women, the developing fetus and newborn infants are all considered highly susceptible subpopulations, and therefore women of childbearing age are susceptible by proxy. Effects on male fertility are more likely to present in older men, while kidney and liver effects are of most concern to subpopulations with pre-existing liver or kidney dysfunction. The partitioning of PCE to fatty tissue is of particular concern for those with fatty liver disease. Neurological endpoints are primarily related to visual function, pattern recognition, and memory. Therefore, subpopulations with poor vision or neurocognitive deficiencies may be especially susceptible to these hazards.

Under TSCA, identifying PESSs for a chemical undergoing a risk evaluation is only the first step. EPA's responsibility to evaluate the chemical's risks includes a determination whether it presents "an unreasonable risk to a potentially exposed or susceptible subpopulation."[85] EPA has not made this determination for any of the many subpopulations it has identified with greater susceptibility to PCE. For example, it has not analyzed how much *more susceptible* these subpopulations are to PCE than the general population and adjusted non-cancer MOEs and cancer risk estimates to account for the greater likelihood of harm. Without such an analysis, EPA cannot address whether risks to the PESSs (as opposed to average workers and consumers) are unreasonable and quantify the additional increment of risk to which these subpopulations are exposed.

B. The Standard 10X UF for Intraspecies Variability Is Not Adequately Protective of PESSs

As in prior evaluations, EPA has attempted to account for the enhanced susceptibility of PESSs by applying a default intraspecies uncertainty/variability factor (UF) of 10. As the Agency explains: "EPA identified lifestage, biological sex, genetic polymorphisms, race/ethnicity, preexisting health status, and lifestyle factors and nutrition status as factors affecting biological susceptibility" and concluded that

[82] Draft Evaluation at 248.
[83] IRIS Assessment at 6-14.
[84] Draft Evaluation at 300.
[85] 15 USC § 2605(b)(4)(A).

"most but not all of these factors are expected to be covered by the inclusion of a 10x UFH."[86] However, this UF is customarily used by EPA to account for normal expected variations in sensitivity within the healthy population.[87] Thus, EPA guidance provides that "a 10-fold factor may sometimes be too small because of factors that can influence large differences in susceptibility, such as genetic polymorphisms."[88] In cases where risks are more than 10 times greater for susceptible subgroups than healthy adults, a larger UF would be warranted.

Since EPA has not analyzed how much more susceptible the PESSs might be to PCE, it has no basis to conclude that the 10X UF will be adequately protective. Indeed, according to EPA, "variability in CYP metabolic capacity," which can render certain subpopulations more susceptible to PCE's liver effects, "is generally believed to vary by approximately 10-fold among all humans" and "individual variations in in vitro CYP2E1 activity as high as 20-50 fold have also been reported."[89] This one genetic risk factor, therefore, has the potential to exceed the 10-fold intra-species uncertainty factor applied by EPA, without beginning to account for other sources of susceptibility. EPA itself admits that it "was unable to directly account for all possible PESS considerations and subpopulations in the risk estimates." As a result, it "is unknown whether the 10x UF to account for human variability will cover the full breadth of human responses, and subpopulations with particular disease states or genetic predispositions may fall outside of the range covered by this UF."[90] Given the requirement in TSCA to make specific determinations of unreasonable risk for PESSs, EPA must separately evaluate risks to known PESS or apply an uncertainty factor that accounts for the specific risks faced by those populations, as opposed to a default value that may leave many PESS underprotected.

For example, EPA argues that differences in response to PCE across life-stages are accommodated in the draft evaluation because "variability in human physiological factors (e.g., breathing rate, body weight, tidal volume) which may affect internal delivered concentration or dose is sufficiently accounted for through the use of a 10x UF for human intraspecies variability."[91] However, the Agency itself admits that "some differences among lifestages or between working and at-rest individuals may not have been accounted for by this value. [92] Moreover, the greater susceptibility of, say, infants as compared to healthy adults may involve not simply pharmacokinetic differences that affect internal dose but biological factors as well. Thus, as noted above, IRIS pointed out that some evidence shows that infants are particularly sensitive to PCE's neurotoxic effects and may experience more severe effects at equivalent internal doses. Similarly, EPA found "evidence of both male and female reproductive effects

[86] Draft Evaluation at 402.
[87] For instance, in its draft Pigment Violet 29 risk evaluation EPA used an intraspecies UF of 10 despite finding "no evidence of increased susceptibility for any single group relative to the general population." Draft Risk Evaluation for C.I. Pigment Violet 29 (Anthra[2,1,9-def:6,5,10-d'e'f']diisoquinoline- 1,3,8,10(2H,9H)-tetrone) (Nov. 2018), found at https://www.epa.gov/sites/production/files/2018-11/documents/draft_pv29_risk_evaluation_public.pdf (PV29 Risk Evaluation).
[88] EPA-630-P02-002F, A Review of the Reference Dose and Reference Concentration Processes, at 4-44(Dec. 2002) https://www.epa.gov/risk/review-reference-dose-and-reference-concentration-processes-document. (RD and RC Review).
[89] Draft Evaluation at 300.
[90] Id. at 402.
[91] Id at 401-402
[92] Id at 402.

in animals as well as associations between exposure and female reproductive in humans along with indications of developmental effects in both study types, both reproductive and developmental toxicity following PCE exposure."[93] For both endpoints, the data demonstrate the unique susceptibility to harm from early-life exposure to PCE in pregnant women and children.

Guidance issued by the California Environmental Protection Agency (Cal EPA) provides a precedent for basing intraspecies UFs on differential susceptibilities to carcinogens and non-carcinogens. [94] Cal EPA conducted a literature review to derive age and life stage adjustment values for carcinogens which include the prenatal period[95] and also increased the default intraspecies UF for non-carcinogens to 30 and 100 for specific endpoints such as asthma or neurotoxicity. [96] This is particularly salient as the most sensitive endpoint for PCE is neurotoxicity.

To provide adequate protection to PESSs, a UF beyond the default intraspecies 10X factor should be applied, as EPA has previously done for other susceptible groups such as infants and children.[97] Determination of an appropriate intra-species UF will require further analysis of the particular susceptibilities of the PESSs for PCE, but we recommend applying an additional UF of at least 10X, as Congress mandated for children exposed to pesticides under the Food Quality Protection Act. [98]

C. Consistent with IRIS, EPA Must Apply an Additional 10X UF for Data-base Deficiencies

EPA guidance calls for application of a UF where the absence of adequate data creates uncertainty in determining a chemical's health effects:[99]

> The database UF is intended to account for the potential for deriving an underprotective RfD/RfC as a result of an incomplete characterization of the chemical's toxicity. In addition to identifying toxicity information that is lacking, review of existing data may also suggest that a

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[93] Risk Evaluation at 293. EPA's conclusions are strongly supported by the epidemiology evidence from the Aschengrau et al studies of the Cape Cod cohort of over 1,300 exposed and 772 nonexposed individuals with PCE contaminated drinking water. These studies reported an over 3-fold elevated risk of neural tube defects (OR 3.5, 95% CI 0.8-14.0), and oral cleft defects (OR 3.2, 95% CI 0.7-15.0). ToxProfile at 176. Both of these congenital abnormalities can be life threatening, or lead to lifetime adverse health effects.

[94] OEHHA. In Utero and Early Life Susceptibility to Carcinogens: [Internet]. 2009. Available from: https://oehha.ca.gov/media/downloads/crnr/appendixjearly.pdf

[95] California EPA 2009. Cal EPA 2009. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. http://oehha.ca.gov/media/downloads/crnr/tsdcancerpotency.pdf

[96] Cal EPA 2008. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Technical Support Document For the Derivation of Noncancer Reference Exposure Levels http://oehha.ca.gov/media/downloads/crnr/noncancertsdfinal.pdf

[97] EPA, Consideration of the FQPA Safety Factor and Other Uncertainty Factors In Cumulative Risk Assessment of Chemicals Sharing a Common Mechanism of Toxicity, February 28, 2002, available at https://www.epa.gov/sites/production/files/2015-07/documents/apps-10x-sf-for-cra.pdf; Assessing susceptibility from early-life exposure to carcinogens. Environ Health Perspect. 2005;113(9):1125-33. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1280390/

[98] https://www.epa.gov/sites/production/files/2015-07/documents/determ.pdf

[99] RD and RC Review at 4-44

> lower reference value might result if additional data were available. Consequently, in deciding to apply this factor to account for deficiencies in the available data set and in identifying its magnitude, the assessor should consider both the data lacking and the data available for particular organ systems as well as life stages.

The size of this UF can vary between 3 and 10. EPA guidance advises that "the size of the database factor to be applied will depend on other information in the database and on how much impact the missing data may have on determining the toxicity of a chemical and, consequently, the POD."[100]

None of the 10 initial TSCA risk evaluations have applied a UF for data-base deficiencies although it is standard practice in IRIS assessments and the EPA guidance calling for this UF is agency-wide in application. The decision of the TSCA program to deviate from EPA guidance has never been explained or justified and is particularly troubling since at the same time EPA has failed to use its streamlined testing authority under amended TSCA to fill data-gaps for PCE and other risk evaluation chemicals.

EPA has consistently recognized that, despite data demonstrating adverse effects for several endpoints, critical gaps exist in understanding of PCE's human health effects. These data-gaps are called out in the 2012 IRIS assessment and TSCA risk evaluation, but the latter fails to recognize the implications of these uncertainties for EPA's determinations of risk and to include a UF to account for them.

EPA explains in the draft risk evaluation that: [101]

> While there was some indication of specific endpoints related to immunotoxicity or blood effects, EPA determined that the database was not fully consistent and there was an absence of adequate quantitative information available to conclude that the domains supported dose-response analysis (Section 0). There is uncertainty whether the PODs for other endpoints carried forward are sufficiently protective of any potential immune or hematological effects that were not accounted for in this risk evaluation.

EPA reiterates that it "can also not rule out that certain subpopulations, whether due to very elevated exposure or biological susceptibility, may be at risk for hazards that were not fully supported by the weight of evidence or could not be quantified (e.g. immune and blood effects)." However, to minimize this concern, "EPA assumes that these effects are likely to occur at a higher dose than more sensitive endpoints that were accounted for by risk estimates."[102] This assumption is pure guesswork. EPA cannot assess the levels at which PCE is immunotoxic without adequate data for this endpoint, and its recent draft evaluation on Trichloroethylene (TCE), which is from the same chemical family as PCE and has common metabolites, identified immunotoxicity as one of two highly sensitive endpoints,

IRIS also underscored the absence of adequate immunotoxicity data for PCE but, in contrast to the draft evaluation, concluded that this data gap (along with others) made it impossible "to adequately characterize the hazard and dose response in the human population."[103] IRIS pointed to "uncertainties

[100] Id. at 4-45.
[101] Draft Evaluation at 315.
[102] Id. at 403.
[103] IRIS Assessment at 5-18.

associated with database deficiencies on neurological, developmental, and immunological effects." For neurotoxicity endpoints, it commented that:[104]

> [D]ata characterizing dose-response relationships and chronic visuospatial functional deficits and the cognitive effects of tetrachloroethylene exposure under controlled laboratory conditions are lacking. Data from acute studies in animals suggest that cognitive function is affected by exposure to tetrachloroethylene. These studies do not address the exposure-response relationship for subchronic and chronic tetrachloroethylene exposures on cognitive functional deficits observed in humans. There is also a lack of cognitive testing following exposures of longer than acute duration, including during development. . . . [T]here has been a limited evaluation of effects of chronic exposure to tetrachloroethylene on visual function in rodents, with the exception of the evoked potential studies by Mattsson et al. (1998). These types of studies could help determine whether there are both peripheral and central effects of tetrachloroethylene exposure on visual perception, and they could be used as an animal model to better define the exposure-response relationships in humans.

IRIS identified similar gaps for hematological and immunotoxicity endpoints:[105]

> Finally, additional data are needed to assess the potential hematological and immunological effects of tetrachloroethylene. In humans, Emara et al., (2010) reported changes in various standard hematological measures in subjects with mean tetrachloroethylene blood levels of 1.685 mg/L. The limited laboratory animal studies of hematological toxicity demonstrated an effect of tetrachloroethylene exposure on red blood cells (decreased RBCs, or decreased erythrocyte colony- forming units, with reversible hemolytic anemia observed in female mice exposed to low drinking water levels (0.05 mg/kg-day) of tetrachloroethylene beginning at 2 weeks of age in one series of studies. Ebrahim et al. (2001) also observed decreased hemoglobin, platelet counts, and packed cell volume, and increased WBC counts. Although additional corroborating studies are lacking, the observation of an effect at a low exposure level raises additional concern about hematological and immunological effects. The fact that other solvents [e.g., toluene, and the structurally similar solvent trichloroethylene) have been associated with immunotoxicity contributes further concern about this gap in the database for tetrachloroethylene.

As a result of these gaps, IRIS applied a data-base uncertainty UF of 10. Because the draft TSCA evaluation applies no UF for these uncertainties, *the IRIS RFCs are an order of magnitude lower than the corresponding PODs used in the evaluation to calculate MOEs*. This difference obviously has important implications for whether the MOEs for PCE's non-cancer health effects are below benchmark MOEs and what assumed levels of exposure by workers and consumers would be deemed to lack adverse health effects when setting PCE exposure limits.

[104] Id. (citations omitted).
[105] Id., at 5-19 (citations omitted).

Addition of the UFs recommended above would result in benchmark MOEs significantly higher than those in the draft evaluation, which are already well above the calculated MOEs for nearly all conditions of use.

V. EPA Properly Applied a Linear Model to Estimate Cancer Risk based on Rodent Liver Tumors but Failed to Recognize the Strength of the Epidemiological Studies Demonstrating Multiple Tumors in Humans

A. Consistent with IRIS, EPA Used a Linear Extrapolation Model to Estimate Risks from Liver Tumors Observed in Animal Studies

The draft evaluation concludes that, under EPA's guidelines or carcinogenicity risk assessment, "PCE is considered 'likely to be carcinogenic in humans' by all routes of exposure based on conclusive evidence in animals and suggestive evidence in humans."[106] As EPA described the animal data:[107]

> There is conclusive evidence of the carcinogenicity of PCE, administered by ingestion or inhalation, in rats and mice. The most notable findings were statistically significant increases in the incidence of liver tumors (hepatocellular adenomas and/or carcinomas) in male and female B6C3F1 and Crj:BDF1 mice exposed by inhalation (JISA 1993; NTP 1986a) and male and female B6C3F1 mice exposed by ingestion (NCI 1977). Significant increases were also observed in the incidences of mononuclear cell leukemia (MCL) in male and female rats (F344/N and/or F344/DuCrj) exposed to PCE by inhalation (JISA 1993; NTP 1986a). Additional findings potentially related to treatment included increases in testicular interstitial cell tumors and renal tubular adenomas and adenocarcinomas in male F344/N rats exposed by inhalation (NTP 1986a), brain gliomas in male and female F344/N rats exposed by inhalation (NTP 1986a), hemangiosarcomas/ hemangiomas in male Crj:BDF1 mice exposed by inhalation (JISA 1993), and adenomas of the Harderian gland in male Crj:BDF1 mice exposed by inhalation (JISA 1993).

As its Point for Departure (POD) for risk estimates, EPA relied on the "hepatocellular tumors, the tumor type that was observed in all three animal bioassays and was the basis of the cancer slope factors in the EPA IRIS Assessment for PCE."[108] To calculate the cancer slope factor, EPA "modeled the 1993 JISA bioassay data for male and female mice using the dose metrics of total liver oxidative metabolism, PCE AUC, and TCA AUC in blood."[109]

EPA's 2005 Guidelines for Carcinogen Risk Assessment[110] emphasize the high level of evidence necessary to depart from the presumption of linearity for carcinogens:

> Elucidation of a mode of action for a particular cancer response in animals or humans is a data-rich determination. *Significant information should be developed to ensure that a scientifically*

[106] Draft Evaluation, at 294.
[107] Id. at 295.
[108] Id at 303.
[109] Id. at 306.
[110] EPA, Guidelines for Carcinogen Risk Assessment (Cancer Guidelines), March 2005 at 84-85, https://www.epa.gov/sites/production/files/2013-09/documents/cancer_guidelines_final_3-25-05.pdf

> *justifiable mode of action underlies the process leading to cancer at a given site. In the absence of sufficiently, scientifically justifiable mode of action information, EPA generally takes public health protective, default positions* regarding the interpretation of toxicologic and epidemiologic data animal tumor findings are judged to be relevant to humans, and cancer risks are assumed to conform with low dose linearity" (emphasis added) (1-10 through 1-11).

The Guidelines add 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. Nonlinear *approaches generally should not be used in cases where the mode of action has not been ascertained.* (emphasis added) (3-21). A nonlinear approach should be selected when there are sufficient data to ascertain the mode of action and conclude that it is not linear at low doses and the agent does not demonstrate mutagenic or other activity consistent with linearity at low doses (3-22).

EPA correctly applied these principles in the draft evaluation to conclude that PCE should be considered a non-threshold, linear carcinogen.

Although some have suggested that the mouse liver tumors should be discounted as not relevant to humans or should be modeled assuming a threshold MOA, EPA examined and rejected these claims, as IRIS did in its 2012 TCE assessment:[111]

> Overall, the reasonably available evidence for all three tumor sites likely supports a complex MOA, with multiple contributing mechanisms of varying significance. There is evidence of kidney and liver-specific genotoxicity from PCE metabolites and evidence of PCE genotoxicity in humans from epidemiological studies. Induction of other non-genotoxic mechanisms including cytotoxicity and PPARα activation are supported by various evidence, however there is insufficient causal link between these pathways and tumorigenesis. Induction of these pathways is often at doses higher than which have been shown to promote tumorigenesis, and the effects are not consistent across sex, dose, and time relative to the results of cancer bioassays. While α-2u-globulin-based kidney toxicity in male rats is not relevant to humans and the PPARα pathway is of reduced significant in humans, the reasonably available data does not support a clear indication that these are major contributors to the tumorigenesis observed in animal cancer bioassays. Therefore, animal carcinogenicity data is considered relevant to humans.

Thus, in accordance with its cancer risk assessment guidelines, EPA concluded that "[t]he evidence for at least a significant contribution of a genotoxic MOA supports use of the low-dose linear assumption, while other mechanisms are not well-enough supported to suggest a potential threshold approach."[112]

[111] Id. at 292.
[112] Id.

A critical element in EPA's dose-response analyses and risk determinations for both cancer and non-cancer endpoints is its utilization of a 2011 physiologically-based pharmacokinetic (PBPK) model. We strongly support use of the model. Importantly, the SACC in its discussions did not raise any concerns that the model is incorrect, or that any additional changes would significantly alter the model output. The PBPK model is the best available, is a good fit for purpose, is published, and continues to be regarded as a high-quality model. SACC member Professor Zhoumeng Lin – who has extensive professional experience with PBPK models and their application to computational toxicology -- thoroughly reviewed the model, its application to PCE, and the underlying code. He praised the Chiu and Ginsberg (2011) model, noting that it is well documented, the structure is properly justified, and it is calibrated.[113] Dr. Lin concluded that, "the quality is really good among the published models", and supporting its use by EPA. [114] Comments submitted to this docket from the Chiu and Ginsberg (2011) model's main author, Dr. Chiu, point out that the model has very little uncertainty in the areas critical to its use by EPA in this PCE assessment, and that it is essentially a deterministic model as recommended by the SACC. [115] Consistent with SACC expert Dr. Lin and others, we support EPA's use of this model in the IRIS assessment and current TSCA PCE risk evaluation.

In sum, EPA's estimate of cancer risk by applying a linear non-threshold model to the PCE rodent liver tumors and the 2011 PBPK model was correct and should be retained in the final risk evaluation.

B. EPA Should Upgrade Its Cancer Classification for PCE to 'Carcinogenic to Humans' Based on Epidemiology Data

In its draft evaluation, EPA concludes that "[e]pidemiological studies provide suggestive evidence for an association between PCE exposure and tumor development in humans." The Agency identifies "tumor types in humans with varying degrees of supporting evidence for an association with PCE exposure includ[ing] NHL, MM, and bladder, esophagus, lung, liver, cervical, and breast cancer" based on studies reviewed in the 2012 IRIS assessment and newer studies.[116] Many of these tumors are terrifyingly swift and deadly, such as lung and esophageal cancers (each has a 5 year survival rate of only 20%), and all of them impose significant financial and quality of life costs to patients and their loved ones.[117]

Given the epidemiological evidence linking both drinking water and inhalation exposures of PCE to so many cancer types, EPA should classify PCE as "carcinogenic to human" rather than the weaker classification of "likely to be carcinogenic." Taken as a whole, these data provide "convincing epidemiologic evidence of a causal association between human exposure and cancer" under EPA's descriptor of "carcinogenic to humans" in its Cancer Guidelines.[118]

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[113] Notes from J. Sass, NRDC, of the SACC public meeting, May 28, 2020.
[114] SACC Weighs Urging EPA To Redo Key Pieces Of Draft Perc Evaluation. Inside EPA, May 29, 2020. Maria Hegstad.
[115] Public Comment by Professor Weihsueh Chiu, PhD., regarding Meetings: Toxic Substances Control Act Science Advisory Committee on Chemicals; Perchloroethylene, Draft TSCA Risk Evaluation (Docket ID: EPA-HQ-OPPT-2019-0502). June 30, 2020
[116] Draft Evaluation, Appendix F.
[117] NIH SEER database for Cancer Stat Facts. https://seer.cancer.gov/statfacts/
[118] Cancer Guidelines, at 2-54.

At least one SACC member, Dr. Calvin Willhite, a toxicologist retired from California's Department of Toxic Substance Control, "recommended EPA review its 2005 Cancer Risk Assessment Guidelines' criteria for cancer classification and clarify why it reaches the conclusion that perc is likely to be carcinogenic to humans. Willhite had earlier suggested that EPA should upgrade the classification to carcinogenic" to humans.[119] We agree with Dr. Willhite and show below that a more definitive and protective classification of PCE as "carcinogenic to humans"' is consistent with the evidence from the epidemiology database.

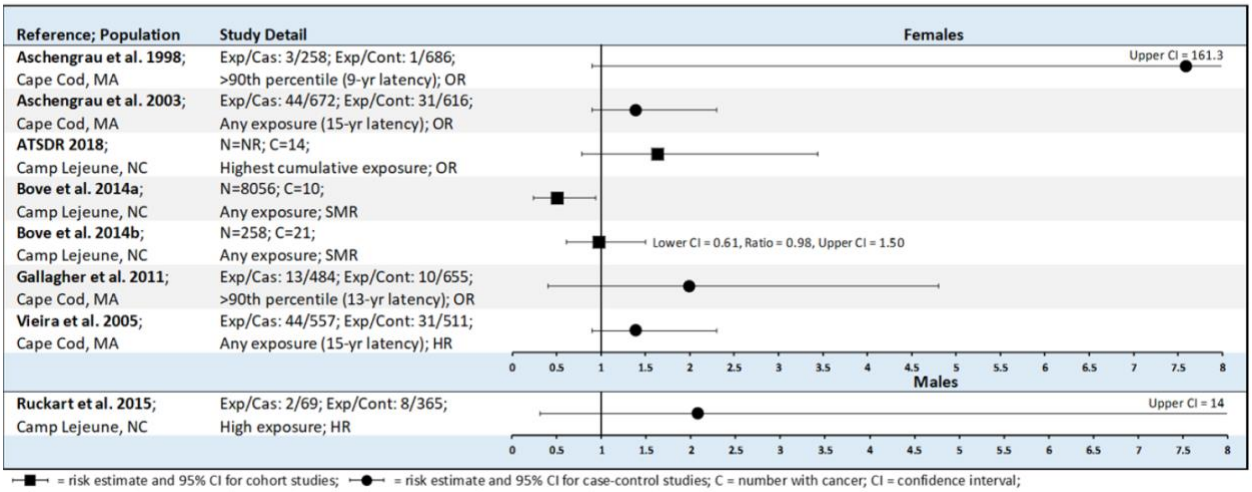
Breast cancer risks – evidence from drinking water and inhalation studies. EPA in 2012 considered the breast cancer risks to be "suggestive but limited evidence" of an association with PCE exposure based on studies available at that time. Unfortunately, although its draft risk evaluation references the same studies as in the ATSDR report – and finds them to be informative, well conducted studies -- EPA's conclusions have not been updated to reflect the strength of the evidence.[120]

Among the strongest of the oral PCE exposure data are the retrospective cohort studies conducted with residents of Cape Cod, Massachusetts who were exposed to PCE-contaminated drinking water over 15 years from leaching from the lining of vinyl-lined asbestos-cement water supply pipes (Aschengrau et al. 1998, 2003, 2008, 2009, 2011, 2012; Getz et al. 2012; Janulewicz et al. 2008, 2012; 2013; Paulu et al. 1999; Vieira et al. 2005). PCE levels in the water in 1980 – when the problem was first discovered – were as high as 7,750 parts per billion (ppb), far higher than the enforceable 5 ppb drinking water maximum contaminant limit (MCL), and the MCL goal (MCLG) of 0 ppb for PCE. Exposure to other water contaminants was considered by the study authors to be rare, limiting confounding by co-exposures. These characteristics – high PCE levels and little or no co-exposures – make the exposure metrics, and thus the studies as a whole, highly reliable and an important piece of evidence linking PCE to cancer in human populations.

ATSDR (2019) provides an excellent summary of the breast cancer data from the most reliable PCE drinking water studies available, in the 'whisker plot' figure (shown below, excerpted from ATSDR Figure 3-17). All the studies with risk estimates (the median point, like the 'cat nose') to the right of the "1" value are depicting a positive link to cancer, with the 95% confidence intervals shown by the horizontal lines (the whiskers of the cat).

[119] SACC Weighs Urging EPA To Redo Key Pieces Of Draft Perc Evaluation. Inside EPA, May 29, 2020. Maria Hegstad.
[120] Draft Evaluation at 611.

Figure 3-17. Summary of Epidemiological Studies Evaluating Associations between Oral Tetrachloroethylene and Breast Cancer



= risk estimate and 95% CI for cohort studies; = risk estimate and 95% CI for case-control studies; C = number with cancer; CI = confidence interval; Exp/Cas = number of exposed cases/number of cases; Exp/Cont = number of exposed controls/number of controls; HR = hazard ratio; N = population size; OR = odds ratio

As shown in the above Figure 3-17 from ATSDR, among the 8 studies depicted, only the Bove et al 2014 mortality studies are not reporting elevated cancer risks. This is likely because these studies are of Camp Lejeune populations, whose drinking water included TCE levels that were 100-times higher than those of PCE. The short follow up time of these studies (only 6% of the military personnel and 10% of civilian employees in the study have thus far died) is likely not enough to tease out the deaths attributable to PCE from those caused by TCE,.[121]

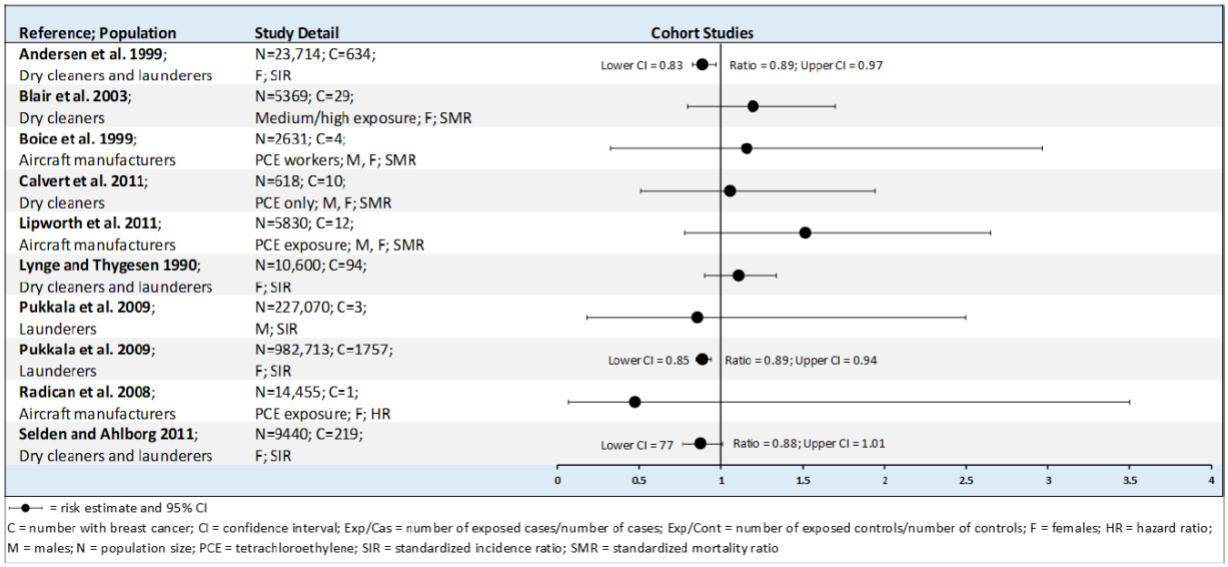
EPA devotes two pages to a lengthy discussion of the Gallagher et al (2011) study, which applies an updated exposure analysis to the Cape Cod cohort, concluding that the study, "suggests a modest association between high drinking water exposure to PCE and breast cancer risk in women."[122] It is unclear why EPA does not consider a 'modest' link to cancer as evidence of a link to cancer – carcinogenicity should be evaluated separately from potency. The EPA Cancer Guidelines do not require a high potency to be a known carcinogen: "A modest risk, however, does not preclude a causal association and may reflect a lower level of exposure, an agent of lower potency, or a common disease with a high background level."[123] Many chemicals, including PCE, may have a more 'modest' potency estimates, but should still be characterized as known to cause cancer in humans.

In addition to drinking water exposures linked to breast cancer, there is also epidemiology evidence linking inhalation exposures to breast cancer. In its public meeting, many SACC members emphasized in public discussions "that EPA needs to review a dozen epidemiology studies of dry cleaning, electrical and aerospace workers exposed to perc, that have been published since the eight-year-old IRIS publication."[124] These important published studies, including both cohort and case-control design

[121] Draft Evaluation at 606.
[122] Id at 616.
[123] Cancer Guidelines, at 2-13.
[124] SACC Weighs Urging EPA To Redo Key Pieces of Draft Perc Evaluation. Inside EPA, May 29, 2020. Maria Hegstad.

studies, were well-conducted and provide evidence of adequate statistical power in relevant human populations for PCE-linked elevated risks of many cancer types, including breast cancer. The studies are described in Figure 3-10 below, excerpted from ATSDR).

Figure 3-10. Summary of Epidemiological Studies Evaluating Associations between Inhaled Tetrachloroethylene and Breast Cancer



C = number with breast cancer; CI = confidence interval; Exp/Cas = number of exposed cases/number of cases; Exp/Cont = number of exposed controls/number of controls; F = females; HR = hazard ratio; M = males; N = population size; PCE = tetrachloroethylene; SIR = standardized incidence ratio; SMR = standardized mortality ratio

Although EPA continues to characterize the breast cancer data as "suggestive but limited," a description it used for several cancer endpoints individually, a different picture emerges when findings for multiple cancer endpoints are combined. This integrated approach demonstrates a more definite overall link between PCE exposure and cancer, as discussed below.

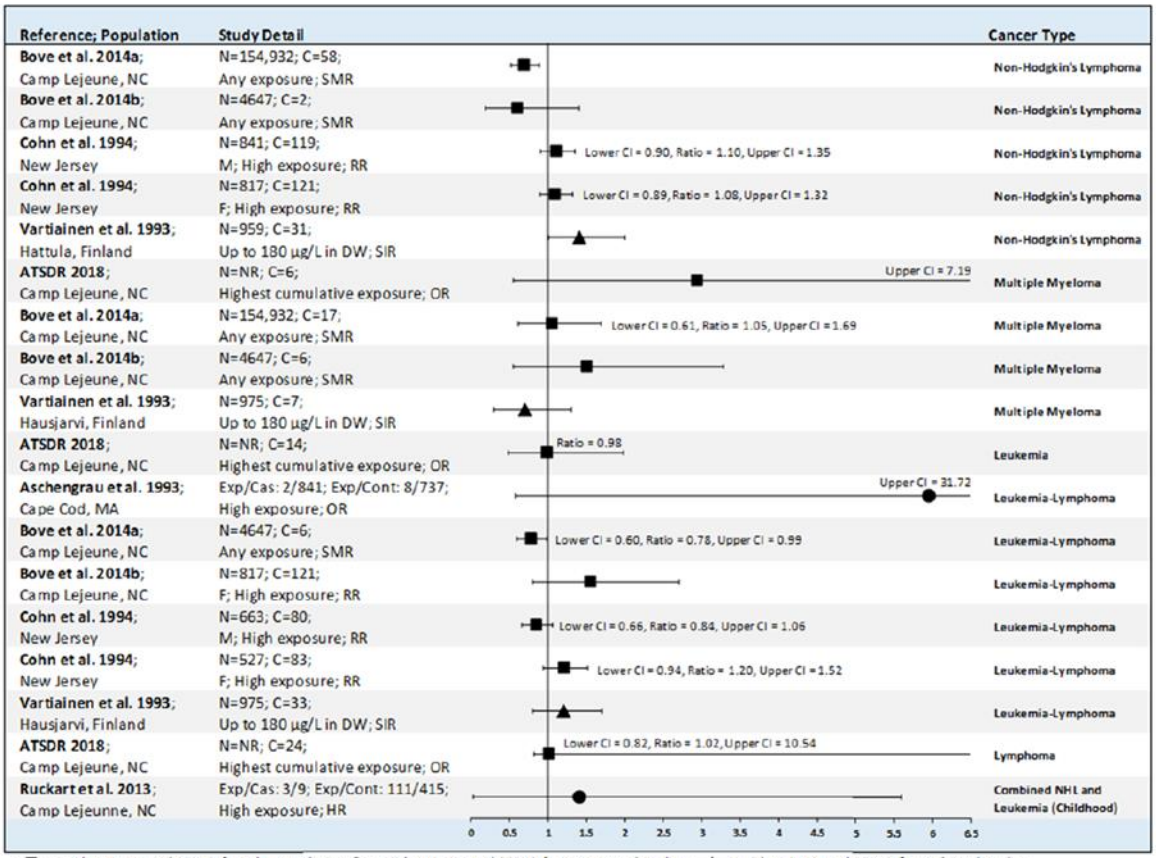
Hematopoietic cancer risks – evidence from drinking water and inhalation studies. EPA should be evaluating the hematopoietic cancers together – as ATSDR has done (see Figure 3-18 below, excerpted from the ATSDR ToxProfile) -- rather than slicing-and-dicing the evidence, which obscures the strength of the overall evidence for hematopoietic cancers in human populations. Had EPA done this, it would present a more accurate characterization of the overall epidemiologic evidence of elevated risk for hematopoietic cancers – from both oral and inhalation – in PCE exposed communities.

For example, in 2012, EPA concluded from the studies available at that time that there was an association between multiple myeloma and PCE, but in its current evaluation, EPA notes more recent studies that do not report a link.[125] EPA also concluded in 2012 that NHL was linked to PCE exposures, but in this current risk evaluation EPA dismisses those conclusions by citing to more recent studies.[126] This is inappropriate – epidemiological studies are designed to 'bias to the null', making it harder to find a true effect that exists. Further, it is widely recognized that the most common study limitations will all

[125] Draft evaluation at 606.
[126] Id.

bias to the null (for example, nondifferential exposure misclassification, inadequate follow up, lost cases, low statistical power such as from small studies, and use of 5% probability levels to minimize chances of false positives, etc). [127] Thus, results in well-conducted studies of adequate statistical power and adequate follow up time should not be cast into doubt by newer studies that fail to find an effect – there is no statute of limitations on the truth. Importantly, drinking water studies that EPA classified as medium or high quality do identify an elevated risk for Non-Hodgkin's Lymphoma (NHL), multiple myeloma, and leukemia-lymphoma associated with PCE exposure from drinking water, as shown below by ATSDR (Figure 3-18 excerpted from the ATSDR ToxProfile):

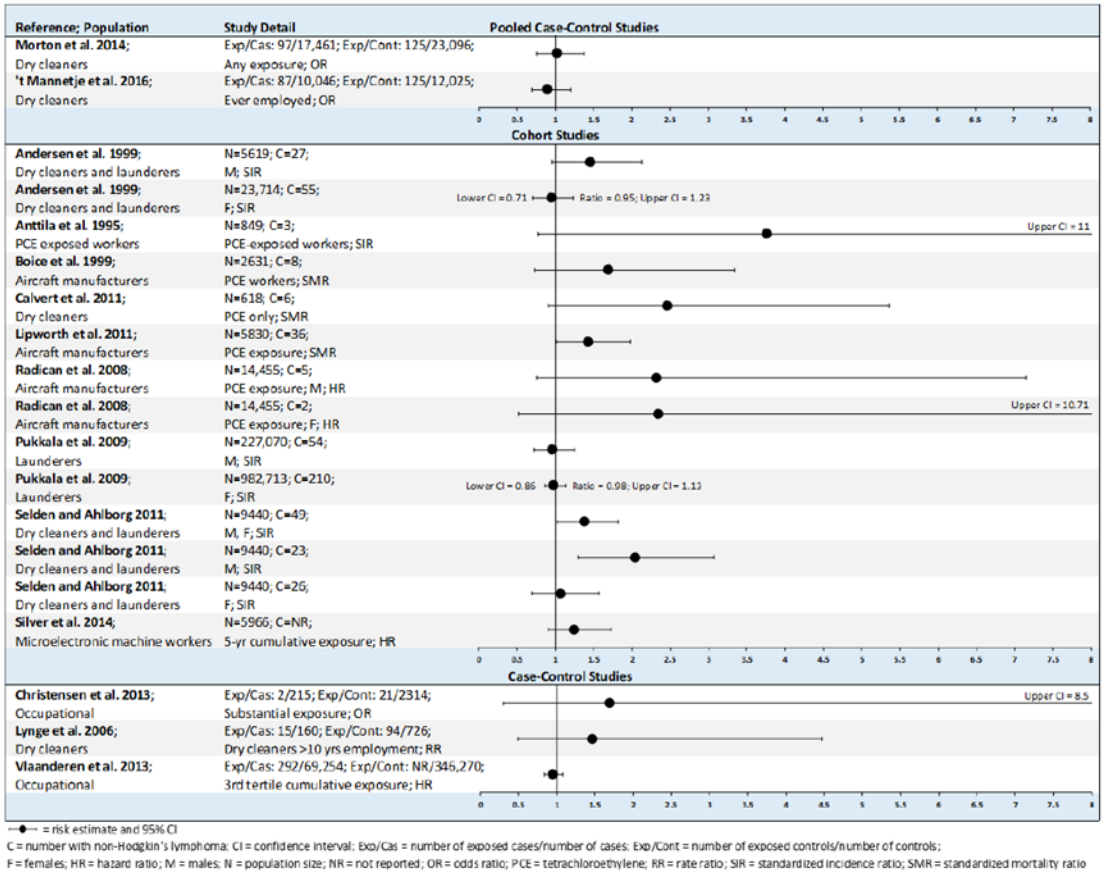
Figure 3-18. Summary of Epidemiological Studies Evaluating Associations between Oral Tetrachloroethylene and Hematopoietic Cancers



= risk estimate and 95% CI for cohort studies; = risk estimate and 95% CI for case-control studies; = risk estimate and 95% CI for ecological studies;
C = number with cancer; CI = confidence interval; DW = drinking water; Exp/Cas = number of exposed cases/number of cases; Exp/Cont = number of exposed controls/number of controls; F = females; HR = hazard ratio; M = males; N = population size; OR = odds ratio; RR = rate ratio; SIR = standardized incidence ratio; SMR = standardized mortality ratio

In addition to the above oral exposure studies, the workplace studies of inhalation exposures that the SACC recommended that EPA review also report an elevated risk of NHL in most studies (see Figure 3-3 below, excerpted from the ATSDR ToxProfile):

Figure 3-3. Summary of Epidemiological Studies Evaluating Associations between Inhaled Tetrachloroethylene and Non-Hodgkin's Lymphoma



In sum, the large number of positive epidemiological findings for hematopoietic cancers, combined with similar findings for breast cancers, demonstrate a sufficient weight of evidence to warrant classifying PCE as carcinogenic in humans. EPA should include this classification in its final risk evaluation.

C. EPA's Risk Evaluation Should Account Acute as Well as Chronic Cancer Risks

It is widely recognized that genotoxic carcinogens like PCE can induce cancer following a limited acute exposure event and that methods to estimate such risks are available. As stated in a 2011 National Research Council (NRC) report:[128]

> 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

[128] NRC, *Standard Operating Procedures for Developing Acute Exposure Guideline Levels for Hazardous Chemicals*, pp. 111-112 (2001), available at https://www.epa.gov/aegl/standing-operating-procedures-developing-acute-exposure-guideline-levels-aegls-hazardous

> 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.

Thus, there exists a recognized methodology for extrapolating from findings of carcinogenicity in long-term studies to exposures of short duration. In its draft TCE risk evaluation, EPA acknowledged the possibility of calculating acute cancer risks but declined to calculate such risk due to "uncertainties" in the NRC methodology.[129] **Rather than summarily dismissing acute cancer risks because they are harder to estimate, an approach not countenanced by TSCA, EPA should quantify these risks using the framework outlined by NRC, which reflects the best available science.**

VI. EPA Should Use a Benchmark of 1 x 10^{-6} to Determine Whether Cancer Risks to Workers and Consumers are Unreasonable under TSCA

As with earlier evaluations, EPA continues to use a cancer risk of 1 x 10^{-4} as the benchmark for determining unreasonable risk to workers. Using this benchmark results in a significantly smaller number of worker exposure scenarios that present unreasonable risks than under cancer risk levels of 1 x 10^{-5} and 1 X 10^{-6}. The SACC has previously stated that EPA has not provided "adequate explanation and justification" for this reduced threshold[130] and the PCE draft evaluation also fails to justify EPA's approach.

The draft PCE evaluation describes how EPA has previously approached cancer risks under the laws it administers as follows:[131]

> Standard cancer benchmarks used by EPA and other regulatory agencies are an increased cancer risk above benchmarks ranging from 1 in 1,000,000 to 1 in 10,000 (i.e., 1x10-6 to 1x10-4) depending on the subpopulation exposed. Generally, EPA considers 1 x 10-6 to 1x 10-4 as the appropriate benchmark for the general population, consumer users, and non-occupational PESS.

Thus, as EPA notes, in applying CAA "residual risk" standards for air toxics, it uses a two-step approach that includes a "presumptive limit on maximum individual lifetime [cancer] risk (MIR) of approximately 1 in 10 thousand" and consideration of whether emissions standards provide an ample margin of safety to protect public health "in consideration of all health information, including the number of persons at risk levels higher than approximately 1 in 1 million, as well as other relevant

[129] TCE Risk Evaluation at 251 (discussing NRC methodology).
[130] SACC 1,4-Dioxane and HBCD Report at 23.
[131] Risk Evaluation at 457.

factors."[132] EPA likewise uses a risk range of 1 x 10^{-4} to 1 x 10^{-6} to set cleanup goals at CERCLA hazardous waste sites.[133] In fact, EPA has used a 1 x 10^{-6} cancer standard to evaluate risk and determine CERCLA remedies at sites where carcinogens are present. [134]

Despite reserving discretion to make case-by-case decisions within this range, however, EPA has identified 1 x 10^{-6} as its goal for public health protection. Thus, in its air toxics standard for radionuclides, EPA stressed that it "should reduce risks to less than 1 x 10-6 for as many exposed people as reasonably possible."[135] Similarly, in guidance for setting health-based water quality criteria under the Clean Water Act (CWA), EPA explained that it:[136]

> 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.

In the CERCLA program, EPA guidance provides that, while "remedies should reduce the risks from carcinogenic contaminants such that the excess cumulative individual lifetime cancer risk for site-related exposures falls between 10-4 and 10-6," the Agency "has expressed a preference for cleanups achieving the more protective end of the risk range (i.e., 10-6)."[137]

However, EPA's recent draft risk evaluations deviate from this approach for worker exposures, maintaining that risks smaller than 1 x 10^{-4} will be considered "reasonable" under TSCA because, "consistent with case law and 2017 NIOSH guidance," this risk level applies to "industrial and commercial work environments subject to Occupational Safety and Health Act (OSHA) requirements."[138]

OSHA precedent does not control decision-making under TSCA, a separate law with different purposes and wording. The cancer risk threshold applied by NIOSH and OSHA is rooted in the Supreme Court's *Benzene* decision, which interpreted the OSH Act as requiring "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 (1980) (emphasis added). The Court grounded this interpretation in an examination of the language, structure and legislative history of the OSH Act. TSCA, by contrast, is anchored in the concept of "unreasonable risk" (a term that implies a lower risk threshold than the OSH Act concept of

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[132] 54 Fed. Reg. 38044, 38045 (September 14, 1989).
[133] EPA, *Rules of Thumb For Superfund Remedy Selection*, August 1997, found at https://semspub.epa.gov/work/HQ/174931.pdf. (CERCLA Guidance).
[134] *See* Record of Decision, Bofors Nobel Superfund Site at 12 (Sept. 1990) (methylene chloride).
[135] 54 Fed. Reg. 51654, 51686 (Dec. 15, 1989).
[136] 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.
[137] CERCLA Guidance at 9.
[138] Risk Evaluation at 457.

"significant risk"). No provision of TSCA provides that workers should receive less protection than other exposed subpopulations or that well-established EPA benchmarks for unacceptable cancer risks would be inapplicable to workers. Indeed, workers are specifically identified as a "potentially exposed or susceptible subpopulation" that EPA is required to protect in section 3(12) of TSCA, indicating that Congress was particularly concerned by the levels of toxic chemicals in the workplace and the special vulnerability of some employee populations to their adverse health effects. Moreover, contrary to EPA's claims, NIOSH does not recommend that workers be left exposed to a 1 in 10,000 risk of cancer. Instead, the NIOSH guidance cited by EPA states "for most carcinogens, there is no known safe level of exposure … [and] NIOSH will continue to recommend that employers reduce worker exposure to occupational carcinogens as much as possible through the hierarchy of controls, most importantly elimination or substitution of other chemicals that are known to be less hazardous …"[139]

In contrast to the OSH Act, TSCA provides protections to workers not just from chemical exposure in the workplace but from air emissions and other environmental releases as well as exposures to consumer products. As discussed above, while draft EPA risk evaluations have assessed worker exposure in isolation from other pathways, this approach understates risks; instead, EPA should combine exposures from all relevant pathways and determine an aggregate risk reflecting the contribution of each source. This is a further reason why setting a higher cancer risk threshold for workers than other populations is unjustified under TSCA.

EPA must apply to workers the same benchmarks for determining unreasonable cancer risks that it uses for other populations. For all exposed populations, EPA should consider any increased cancer risk exceeding 1 x 10^{-6} to be unreasonable and to require action under TSCA.

VII. EPA Has Failed to Model Realistic Dermal Exposure Scenarios

PCE is a volatile liquid and both inhalation and dermal exposures are expected during manufacturing, processing, use and disposal. Accordingly, EPA developed exposure and risk estimates for dermal as well as inhalation exposures for both workers and consumers. However, the methodologies EPA used to evaluate dermal exposure for these two populations were different and resulted in differing estimates of dermal absorption rates. EPA does not explain its rationale for using different methodologies for workers and consumers and their underlying assumptions seem conflicting. For workers, EPA has understated the magnitude of PCE dermal exposure. For consumers, EPA's approach is more realistic, but it is of concern that EPA assumes no dermal exposure for half of the consumer uses it addresses.

A. EPA's Dermal Exposure Scenarios for Workers Understate Dermal Absorption

Because "[d]ermal exposure data was not readily available for the conditions of use in the assessment," EPA used modeling techniques to estimate dermal exposure. For workers, "[d]ermal exposures are assessed using the Dermal Exposure to Volatile Liquids Model, which relies on the theoretical framework presented by Kasting and Miller (2006) to estimate the fractional absorption in accounting

[139] Christine Whittaker et al., NIOSH, Current Intelligence Bull. 68, NIOSH Chemical Carcinogen Policy 20 (July 2017), https://www.cdc.gov/niosh/docs/2017-100/pdf/2017-100.pdf.

for chemical volatilization.[140] This "model determines a dermal potential dose rate based on an assumed amount of liquid on skin during one contact event per day and the steady-state fractional absorption for PCE" using the Kasting formula.[141] Applying the model, EPA estimated "that 13 to 19 percent of the applied dose is absorbed through the skin" following exposure. [142]

For industrial and commercial uses of PCE, EPA then used this dermal exposure rate to estimate workers' dermal exposure with and without gloves. For the glove use estimates, EPA developed four different hypothetical glove protection factors (PFs) (also used in several earlier risk evaluations). Commercial and industrial PCE uses were assigned to six different "bins" corresponding to maximum possible exposure concentration and the likely level of exposure resulting from the use.[143] For each bin, EPA applied the four PFs to estimate differences in exposure based the effectiveness of glove use.

As EPA itself acknowledged, several of the steps in this analysis were based on debatable assumptions and could well underestimation of dermal exposure.

Higher Dermal Penetration Scenarios. EPA recognized that its dermal exposure "model assumes a fixed fractional absorption of the applied dose; however, fractional absorption may be dependent on skin loading conditions."[144] Thus, EPA acknowledged that its assumption of rapid volatilization of PCE after skin contact did not hold true in all worker operations:[145]

> Dermal absorption of PCE depends on the type and duration of exposure. Where exposure is non-occluded, only a fraction of PCE that comes into contact with the skin will be absorbed as the chemical readily evaporates from the skin. However, dermal exposure may be significant in cases of occluded exposure, repeated contacts, or dermal immersion. For example, work activities with a high degree of splash potential may result in PCE liquids trapped inside the gloves, inhibiting the evaporation of PCE and increasing the exposure duration.

EPA expanded on this point in its draft evaluation for carbon tetrachloride [146]

> Due to increased area of contact and reduced skin barrier properties, repeated skin contact with chemicals could have even higher than expected exposure if evaporation of the chemical occurs and the concentration of chemical in contact with the skin increases. In the workplace the wearing of gloves could have important consequences for dermal uptake. If the worker is handling a chemical without any gloves, a splash of the liquid or immersion of the hand in the chemical may overwhelm the skin contamination layer so that the liquid chemical essentially

[140] Draft Evaluation at 192.
[141] Id. at 132.
[142] Id. at 192.
[143] Id. at 192-94.
[144] Id. at 199.
[145] Id. at 191.
[146] Carbon tetrachloride risk evaluation at 92

> comprises the skin contamination layer. If the material is undiluted, then uptake could proceed rapidly as there will be a large concentration difference between the skin contamination layer and the peripheral blood supply.

However, these higher exposure scenarios are not hypothetical but can be expected to occur regularly in workplaces. Thus, EPA should have developed additional risk and exposure estimates reflecting the higher levels of dermal absorption likely under reasonably foreseen conditions of use.

EPA's assumption of low dermal absorption based on rapid PCE volatilization is also open to question. In its TCE evaluation, EPA admitted that its absorption rate modeling was uncertain because "there is a large standard deviation experimental measurement, which is indicative of the difficulty in spreading a small, rapidly evaporating dose of TCE evenly over the skin surface."[147] Moreover, EPA elsewhere cited data showing that TCE dermal absorption can in fact be rapid:[148]

> 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).

ATSDR has discussed similar dermal absorption studies for PCE.[149] However, they are not addressed in the draft PCE evaluation.

Multiple Dermal Exposure Events. in its carbon tetrachloride risk evaluation, EPA admitted that its dermal "model assumes one exposure event per day, which likely underestimates exposure as workers often come into repeat contact with the chemical throughout their workday."[150] The PCE evaluation likewise recognizes that its dermal absorption model "assumes a single exposure event per day . . . and does not address variability in exposure duration and frequency."[151] Despite acknowledging this limitation, EPA did not model any repeat contact scenarios for PCE involving higher levels of dermal exposure. In its review of the draft risk evaluation for methylene chloride, the SACC similarly "was concerned with the assumption of only a single dermal exposure per day and thought that this assumption results in an underestimation of potential exposures."[152] Indeed, in its methylene chloride

[147] TCE risk evaluation at 117.
[148] Id. at 203.
[149] ToxProfile, at 184-86.
[150] Carbon tetrachloride risk evaluation, at 168.
[151] Draft Evaluation at 199.
[152] SAAC, *Peer Review for EPA Draft Risk Evaluation for Methylene Chloride*. December 3-4, 2019, at 33, file:///C:/Users/Owner/Downloads/EPA-HQ-OPPT-2019-0437-0080%20(6).pdf

evaluation, EPA acknowledged that, "[f]or workplace exposures inhalation and dermal exposures are assumed to occur simultaneously i.e. both occur at the start of the task and continue through the end of the task, shift, or work day."[153] Similarly, EPA should base dermal exposure scenarios in the final PCE evaluation on an assumption of ongoing exposure by this route throughout the work day, not a single exposure event.

Glove Protection Assumptions. EPA's assumption that gloves will provide any level of protection from dermal absorption is highly speculative. In the *Supplemental File: Environmental Releases and Occupational Exposure* for its PCE evaluation, EPA acknowledges that:[154]

> "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. Instead, the impact of effective glove use should be explored by considering different percentages of effectiveness (e.g., 25% vs. 50% effectiveness)."

Thus, EPA admits that "[g]love protection factors are presented as what-if scenarios to show the potential effect of glove use on exposure levels. EPA does not know the actual frequency, type, and effectiveness of glove use in specific workplaces with PCE conditions of use." [155] Even where gloves are used, their effectiveness is not assured. As the PCE Supplement recognizes,[156] some glove types may lack impermeability for specific chemicals and even protective glove types will fail to fully prevent exposure if not properly maintained and replaced.

Moreover, it is well-known that glove use can *increase* skin absorption under some circumstances. As the PCE Supplement notes, "[g]loves can prevent the evaporation of volatile chemicals from the skin, resulting in occlusion. Chemicals trapped in the glove may be broadly distributed over the skin .. . , or if not distributed within the glove, the chemical mass concentration on the skin at the site of contamination may be maintained for prolonged periods of time."[157] As EPA noted in the TCE evaluation, "[d]ermal exposure may be significant in cases of occluded exposure," exceeding absorption levels where not gloves are used.[158] EPA recognizes that occlusion is an expected occurrence for several PCE conditions of use:[159]

> EPA expects occlusion to be a reasonable occurrence at sites where workers may come in contact with bulk liquid chemical and handle the chemical in open systems. This includes conditions of use such as vapor degreasing, cold cleaning, and dry cleaning where workers are

[153] Methylene Chloride Risk Evaluation at 387.
[154] *Supplemental File: Environmental Releases and Occupational Exposure* (Supplement) at 297.
[155] Draft Evaluation at 192.
[156] Supplement at 297-298.
[157] Id. at 296.
[158] TCE Risk Evaluation at 116.
[159] Supplement at 297.

> expected to handle bulk chemical during cleanout of spent solvent and addition of fresh solvent to equipment. Similarly, occlusion may occur at coating or adhesive application sites when workers replenish application equipment with liquid coatings or adhesives.

The Supplement discusses various methodologies for estimating the increase in dermal absorption due to occlusion but states that, rather than making these calculations, EPA "addresses the occlusion scenario in combination with other glove contamination and permeation factors through the use of a protection factor."[160] However, this only compounds uncertainties because EPA's PFs are purely hypothetical and in any case do not address occlusion scenarios, which result in *more* dermal absorption than in the absence of gloves.[161]

B. EPA Used More Realistic Dermal Absorption Scenarios for Some Consumer Uses but Unreasonably Assumed an Absence of Dermal Exposure for other Uses

Unlike its dermal exposure estimates for workers, EPA's estimates for consumers assumed that certain conditions of use involve limited evaporation of PCE from dermal surfaces and significant levels of absorption:[162]

> PCE is absorbed dermally, and exposure magnitude depends on exposure characteristics such as skin surface area, product volume, chemical loading and weight fraction, and exposure duration. PCE is a volatile solvent, expected to evaporate from skin quickly. However, there are certain consumer use scenarios for which product evaporation may be limited, for example due to immersion of hands into a reservoir of cleaning solvent (reasonable given that consumers are not assumed to use PPE, as well as the nature of PCE containing products and uses), the wearing of recently dry cleaned fabrics, or handling/wiping using a solvent soaked rag. Consumer uses analyzed for dermal exposure with impeded evaporation include immersive parts cleaning, aerosol degreasers, liquid stone and marble polishes, liquid sealants, liquid paint primers and the wearing of recently dry-cleaned articles

To determine the rate of absorption, EPA used a different model for consumers than it used for workers and its consumer permeability method accounted for product-specific low evaporation use scenarios:[163]

> Dermal exposure to PCE from consumer product use was estimated using CEM's permeability method (P_DER2b). The permeability method is based on the ability of a chemical to penetrate

[160] Id. at 296.
[161] EPA's failure to account for increased absorption from occlusion in the PCE evaluation differs from its approach in the TCE evaluation, where EPA in fact modeled the effect of occlusion on dermal exposure and "estimated central tendency and high-end dermal retained doses for occluded scenarios for OESs where occlusion was reasonably expected to occur." TCE Risk Evaluation at 102. As Table 2-15 shows, occlusion greatly increases dermal absorption: with occlusion, exposures are 7.6-12.2 times higher than in the no-glove scenarios. Id. at 106.
[162] Draft Evaluation at 208.
[163] Id. at 210.

the skin layer once contact occurs. The model assumes a constant supply of chemical, directly in contact with the skin, throughout the exposure duration. Evaporative loss of PCE from the skin during product use is expected to be considerable, except in cases where the nature of use limits evaporation, such as from the use of a solvent soaked rag, or immersion of hands in a container of PCE based cleaner. Only product use scenarios where a reasonable assumption could be made for limited evaporation from skin were assessed for dermal exposure. A chemical-specific skin permeability coefficient of 1.8x10-2 cm/hr was used for permeability estimates (Nakai et al. 1999).

For those consumer products assessed for dermal exposure, several MOEs were extremely small, indicating a high level of dermal risk. For example, the dermal MOE for high-intensity adult users of aerosol brake cleaners was 7.2E-02[164], considerably smaller than the acute dermal MOEs for commercial aerosol degreasers and lubricants,[165] which would likely be used in the same way.

Considering the large dermal risks for the consumer products that EPA does assess, its decision to assume an absence of dermal exposure for the remaining PCE-containing products is unwarranted. These products (such as caulks, sealants and column adhesives) plainly have the potential for dermal exposure although evaporative losses may be greater than for the products EPA assesses. Since EPA itself acknowledges that a "key uncertainty for the dermal estimates is the accuracy of the assumption of which COUs are likely to result in exposure with impeded evaporation,"[166] the best course is to estimate dermal exposures and risks for *all* TCE-containing consumer products.[167]

In sum, EPA should (1) model a broader range of worker dermal absorption scenarios based on its own analysis of variations in workplace dermal exposure conditions, (2) base risk estimates for workers on multiple dermal exposure events per day, (3) recognize that gloves can increase dermal absorption if occlusion occurs and account for this increase in exposure, and (4) assess dermal exposures and risks for all PCE-containing consumer products.

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

As in previous risk evaluations, EPA's risk determinations for workers exposed to PCE calculate MOEs assuming both the use of respirators and gloves and the absence of protective equipment. Even for scenarios where workers consistently and reliably use PPE, MOEs are below "benchmarks" for most conditions of use and endpoints. This is not the case in all instances, however. Moreover, EPA's MOEs

[164] Id. at 388.
[165] Id. at 380. The MOEs for these commercial PCE uses are nonetheless well below benchmark MOEs.
[166] Id. at 402.
[167] Remarkably, the draft TCE evaluation claimed that dermal exposure during use of TCE-containing consumer products "is unlikely to contribute significantly to overall exposure." TCE Risk Evaluation at 137. This is a more extreme approach than EPA has used for PCE even though the two chemicals are similar in rate of volatilization and have overlapping conditions of use.

are significantly lower for the "no PPE" scenarios. Basing determinations of unreasonable risks on these scenarios will therefore result in more comprehensive risk management restrictions and more protective exposure limits.

As with previous chemicals, the PCE evaluation provides no evidence that PPE are in widespread use and effectively controlling exposure in workplaces where PCE is manufactured, processed and used. Moreover, as the SAAC has repeatedly underscored and EPA itself has recognized, the assumption of universal PPE use is contrary to the realities of workplace practice and sound principles of worker protection. For this reason, the "no PPE" scenario is the only defensible basis for determining whether PCE presents an unreasonable risk to exposed workers.

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

In each of its reviews of draft evaluations, the SACC has repeatedly raised concerns about EPA's undue reliance on PPE for determinations of unreasonable risk. In its report on the PV29 draft, the SACC noted that "the analysis in the Evaluation does not discuss or account for the fact that downstream commercial users may be oblivious to chemical risks and lack even rudimentary industrial hygiene measures."[168] Similarly, in reviewing the 1,4-dioxane evaluation, the SACC concluded that the "consensus of the Committee believes that PPE may not be consistently and properly worn, as EPA assumed"[169] and noted that "[g]love use should not always be assumed to be protective" and, if worn improperly, gloves "could actually lead to higher exposures."[170] As it concluded, "8-hour use of PPE should not be used in the risk characterization of inhaled 1,4-Dioxane. Risk estimates should be presented without the use of PPE as reasonable worst case."[171]

In the case of HBCD, the SACC noted that "it was unreasonable to assume workers would wear PPE for entire 8-hour shifts due to underlying medical conditions, facial hair, discomfort, and other issues" and added that:[172]

> [M]any members of the Committee believed EPA should place more emphasis on the limited likelihood that respiratory protection will be adopted without specific occupational exposure guidelines for HBCD . . . Dust exposures in the construction trades (especially residential construction) continue to represent an occupational health concern because of the many small-to-medium size operators and the use of temporary (and, not infrequently, undocumented) workers. Workers in these small-to-medium enterprises may not be likely to adopt personal protective equipment (PPE) controls, so EPA's characterization of reasonable risk relying on use of PPE is not sufficiently supported by the practical realities of many workplaces.

[168] SACC Report on PV29 at 37.

[169] According to the SACC, these "heightened exposures" could occur as a result of "contamination of the interior of the glove" (if workers were not properly trained in glove use and replacement) or by "acting as a reservoir" for contaminants (if the gloves were not impermeable). Such occlusion (greater penetration of the skin where contaminants build up inside the glove) is discussed in Part V above.

[170] SACC Report on 1,4-dioxane and HBCD, at 55.

[171] Id. at 53.

[172] Id at 118.

The SACC report on 1-BP provides further amplification of these concerns:[173]

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

The SACC concluded that EPA's "[a]ssumptions about PPE use are likely unrealistic for many of the scenarios and so the determination of whether a condition of use results in an acceptable or unacceptable risk should be based on no PPE use, with the possible exception of in a manufacturing facility."[174]

The SACC report on the methylene chloride risk evaluation reinforced these points, stating that "[m]ost Committee members agreed that EPA's assumptions of PPE use likely do not reflect actual conditions in most workplaces."[175] The SACC added that:[176]

> The Agency's reliance on appropriate use of personal protective equipment (PPE), including both respirators and gloves, is not supported by current research literature or industrial hygiene practice. The mere presence of a regulation requiring respirators does not mean that they are used or used effectively. Inadequacies in respirator programs are documented. Respirators require multiple respiratory protection (RP) compliance factors in order to perform as certified. Brent et al. (2005) used data from the NIOSH and Bureau of Labor Statistics (BLS) joint survey on Respirator Usage in Private Sector Firms, (BLS, 2001) to examine the adequacy of respirator protection programs in private industries. They found "large percentages of establishments requiring respirator use [under OSHA or the Mine Safety and Health Administration (MSHA) regulations] had indicators of potentially inadequate respirator programs." Later, Janssen et al. (2014) reported that 'APFs do not apply to RPD used in the absence of a fully compliant RP program; less than the expected level of protection is anticipated in these situations.' Moving beyond program elements, the frequency of proper use of gloves and respirators is largely unknown.

B. There is No Evidence that PCE-Exposed Workers are Meaningfully Protected by PPE

The draft PCE evaluation provides no data documenting ongoing PPE use by PCE-exposed workers. However, in a departure from some previous evaluations, it divides PCE conditions of use into two

[173] SACC Report on 1-BP, at 30-31.
[174] Id at 66.
[175] SACC Report on methylene chloride, at 17.
[176] Id at 36.

categories: (1) those where respirator use is "plausible" and workers "may use" respirators; and (2) those with "no respirator use."[177] While some industrial and commercial activities are likely carried out without respirators, viewing respirator use as "plausible" for other activities is a far cry from demonstrating that respirators are consistently and reliably protecting workers. For example, EPA classifies open-top degreasing as a PCE use where workers "may use" respirators. But EPA also finds that, at the 50th percentile use level, 4,942 sites are using PCE in open-top vapor degreasing operations and that these operations employ a total of 54,000 exposed workers and ONU.[178] Most of the facilities where open-top degreasing is performed are small businesses which lack extensive industrial hygiene programs that focus on working training in proper respirator use and adequate fit testing.

In predicating its unreasonable risk determinations on PPE use, "EPA assumes that workers will responsibly wear gloves and respirators and that employers implement a continuing, effective respiratory protection program according to the requirements of OSHA's Respiratory Protection Standard."[179] This assumption is not credible. The OSHA Respiratory Protection Standard (29 CFR 1910.134) contains numerous elements, *e.g.,* for program administration; worksite-specific procedures; respirator selection; employee training; fit testing; medical evaluation; and respirator cleaning, maintenance, and repair. As SACC has noted, the NIOSH and Bureau of Labor Statistics report on respirator use cited in the PCE evaluation[180] found that many establishments where respirators were required by law "had indicators of potentially inadequate respirator programs", including multiple failures to implement requirements of the OSHA RPS. The small businesses where most PCE use occurs are, if anything, likely to be even less diligent in complying with respiratory protection protocols.

The current OSHA time-weighted average 8-hour Permissible Exposure Limit (PEL) for PCE is 100 parts per million (ppm), three orders of magnitude higher than the level that current TCE health effects data would warrant. The PEL was adopted in 1970 and has never been updated. It lacks specific implementation measures, including PPE requirements, typical in more recent standards. In the absence of a health-protective OSHA limit on workplace exposure, it is inconceivable that OSHA is enforcing – or employers are systematically implementing – the stringent PPE requirements that would be necessary for the substantial reductions in worker exposure required to achieve safe levels of PCE in the workplace.

According to the draft PCE evaluation, "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."[181] However, neither the OSHA standard for PCE nor other OSHA regulations call for employers to implement PPE or other measures sufficient to eliminate the unreasonable risks to workers demonstrated in EPA's draft evaluation in the absence of respirator and glove use. Even in the highly unlikely event that industry SDSs recommended

[177] Draft Evaluation at 334-36.
[178] Id. at 144.
[179] Id. at 401.
[180] Supplement at 33-34.
[181] Draft Evaluation at 458.

comprehensive PPE programs, OSHA hazard communication regulations do not require employers to follow SDS recommendations, and the preamble to these regulations expressly state that "there is no requirement for employers to implement the recommended controls."[182] Moreover, OSHA regulations give employers wide latitude to interpret evidence of workplace risks and to select worker protection measures they deem appropriate. Thus, OSHA's PPE standard requires employers to assess the hazards workers face but to provide PPE only when the employer deems such measures "necessary." [183] EPA may be correct in "expecting" compliance with OSHA regulations, but it's plainly incorrect that these regulations compel employers to use PPE to eliminate unreasonable risks that fall below the OSHA PEL.

In fact, established OSHA policy is to rely principally on engineering controls and other control strategies to address chemical risks, with PPE as a backstop if these measures are infeasible. As EPA itself explains in the draft evaluation:[184]

> OSHA and NIOSH recommend employers utilize the hierarchy of controls to address hazardous exposures in the workplace. The hierarchy of controls strategy outlines, in descending order of priority, the use of elimination, substitution, engineering controls, administrative controls, and lastly personal protective equipment (PPE). The hierarchy of controls prioritizes the most effective measures first which is to eliminate or substitute the harmful chemical (e.g., use a different process, substitute with a less hazardous material), thereby preventing or reducing exposure potential. Following elimination and substitution, the hierarchy recommends engineering controls to isolate employees from the hazard (e.g., source enclosure, local exhaust ventilation systems), followed by administrative controls (e.g. do not open machine doors when running), or changes in work practices (e.g., maintenance plan to check equipment to insure no leaks) to reduce exposure potential. Administrative controls are policies and procedures instituted and overseen by the employer to limit worker exposures. As the last means of control, the use of personal protective equipment (e.g., respirators, gloves) is recommended, when the other control measures cannot reduce workplace exposure to an acceptable level.

Thus, the SACC review of the HBCD evaluation stressed that "[m]any Committee members were concerned with the reliance on PPE or engineering controls to reduce risk, as that is contrary to the hierarchy of controls."[185]

Consistent with the hierarchy of controls and the SACC's repeated recommendations, EPA's unreasonable risk determinations for PCE should assume no PPE use. The requirements necessary to eliminate these unreasonable risks should be decided in the later TSCA risk management phase. At this point, PPE should be considered as a last resort, only after other means of control such as chemical substitution and engineering controls have been shown to be inadequate.

[182] Hazard Communication, 77 Fed. Reg. 17574, 17693 (Mar. 26, 2012).
[183] 29 C.F.R. § 1910.132(a).
[184] Draft Evaluation at 131.
[185] SACC Report on 1,4-dioxane and HBCD, at 73.

IX. EPA Should Abandon the Poorly Defined Category of Occupational Non-Users (ONUs) In Favor of a More Realistic Framework for Exposure Analysis

Like previous draft evaluations, the PCE evaluation differentiates between directly exposed workers and the category of "occupational non-users" (ONUs). EPA defines occupational users as workers that directly handle a chemical and occupational non-users (ONUs) as workers who do not directly handle the chemical but perform work in an area where PCE is present.[186] The draft evaluation provides few details on the job responsibilities and activities of ONUs. Nonetheless, EPA takes the approach that "[w]hile the difference between the exposures of ONUs and the exposures of workers directly handling PCE generally cannot be quantified, ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical."[187] Thus, EPA arbitrarily assumed "the ONU exposures to be equal to the central tendency risk estimates for workers when determining ONU risk attributable to inhalation."[188] EPA also claimed, without justification, that "dermal exposures are not expected because ONUs do not typically directly handle PCE, nor they are in the immediate proximity of PCE."[189] As a result of this approach, "EPA determined that most applicable conditions of use do not present unreasonable risks" to ONUs.[190]

EPA's methodology of differentiating among workers based on whether the worker's job description includes direct contact with the chemical and assuming that all workers without such contact have lower exposures is a false dichotomy with no basis in accepted methodologies for industrial exposure assessment. In fact, the term "ONU" or "occupational non-user" does not appear on a search of PubMed – the NIH medical library of over 10,000 scientific journals – or on a 'google' search, other than in EPA TSCA documents.

Instead, the distinction experts use is between near-field and far-field exposure, and they differentiate among jobs by whether they may be near or far from the source of exposure.[191] This distinction allows the assessor to evaluate exposures by grouping workers into near and far field categories (see citations for examples).[192] The near-field/far-field distinction is the state of the science because it has logic –

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[186] Draft Evaluation at 29, FN1.
[187] Id. at 31.
[188] Id.
[189] Id.
[190] Id. at 34.
[191] LeBlanc M, Allen JG, Herrick RF, Stewart JH. Comparison of the near field/far field model and the advanced reach tool (ART) model V1.5: exposure estimates to benzene during parts washing with mineral spirits. Int J Hyg Environ Health. 2018 Mar;221(2):231-238. doi: 10.1016/j.ijheh.2017.10.016. Epub 2017 Oct 31.
Keil CB, Nicas M. Predicting room vapor concentrations due to spills of organic solvents. AIHA J (Fairfax, Va). 2003 Jul-Aug;64(4):445-54. PubMed PMID: 12908858.
Lee EG, Lamb J, Savic N, Basinas I, Gasic B, Jung C, Kashon ML, Kim J, Tischer M, van Tongeren M, Vernez D, Harper M. Evaluation of Exposure Assessment Tools under REACH: Part I-Tier 1 Tools. Ann Work Expo Health. 2019 Feb 16;63(2):218-229. doi: 10.1093/annweh/wxy091.
[192] LeBlanc M, Allen JG, Herrick RF, Stewart JH. Comparison of the near field/far field model and the advanced reach tool (ART) model V1.5: exposure estimates to benzene during parts washing with mineral spirits. Int J Hyg Environ Health. 2018 Mar;221(2):231-238. doi: 10.1016/j.ijheh.2017.10.016. Epub 2017 Oct 31.
Keil CB, Nicas M. Predicting room vapor concentrations due to spills of organic solvents. AIHA J (Fairfax, Va). 2003 Jul-Aug;64(4):445-54. PubMed PMID: 12908858.

workers whose job brings them near to the chemical are considered to share the same exposures as other near-field workers, whether or not they are specifically tasked with directly contacting the material.

In fact, workers tasked with directly working with the chemical are often not the highest exposed, because they are the most protected, working in a fume hood or behind a shield, or with proper fitted and functioning PPE. It may be the other workers in the near-field that are not necessarily tasked with directly contacting the chemical that may be at increased risk – workers that EPA classifies as ONUs. For example, janitorial staff that clean up spills, workers that repair leaks, lab workers in neighboring stations, administrative staff in nearby open offices, truck drivers that transport the chemical if there is an accidental spill or leak, etc. EPA does not expect these workers to handle the chemical as part of the normal course of their workday, but the reality – which EPA ignores – is that they perform work in an area near where the chemical is present. That is, their exposure is that of 'near-field workers', but EPA wrongly classifies them in its ONU category, to which EPA assigns 'far-field' exposures. Using this classification, EPA then applies risk-lowering assumptions – i.e. that there is no dermal exposure and that central tendency exposure estimates should be used for risk determinations – that are likely incorrect for many near-field workers.

The SACC recognized this in its report on the methylene chloride evaluation: "The Agency should consider exploring different categories of ONUs (e.g., workers who do not handle methylene chloride directly, but whose job requires them to be in the same area as users; cleaning staff that can be exposed after hours to residues present in the work area, or office/managerial workers that could be incidentally exposed when visiting a work area but are not at risk from exposure routinely) because their potential exposure risk likely varies."[193] The SACC further explained that:[194]

> ONUs are likely a heterogeneous population of workers, and some could be exposed more than just occasionally to high concentrations. This possibility should be included explicitly as a source of uncertainty. As recommended earlier, EPA should consider the different categories of ONUs potentially at risk.

Real world examples of near-field workers who experience elevated exposures to solvents (wrongly classified by EPA as 'far-field' ONUs) are provided in the comments of the Toxics Use Reduction Institute (TURI) on the methylene chloride evaluation. For example: [195]

> … occupational non-users can have levels of exposure similar to that of occupational users. Concerns about this category of workers are exacerbated by the fact that they may work in

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Lee EG, Lamb J, Savic N, Basinas I, Gasic B, Jung C, Kashon ML, Kim J, Tischer M, van Tongeren M, Vernez D, Harper M. Evaluation of Exposure Assessment Tools under REACH: Part I-Tier 1 Tools. Ann Work Expo Health. 2019 Feb 16;63(2):218-229. doi:
10.1093/annweh/wxy091.

[193] SACC report on methylene chloride risk evaluation, at. 31

[194] Id at 44.

[195] Comments of the Massachusetts Toxics Use Reduction Institute (TURI) on EPA's Draft Toxic Substances Control Act (TSCA) Risk Evaluation: Methylene chloride. December 2019. Docket Number EPA-HQ-OPPT-2019-0437-0070. Available at https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0437-0070

close proximity to methylene chloride yet may not be provided with personal protective equipment (PPE). For example, at one of the furniture refinishing facilities visited by program staff, paint stripping was performed in an open room with work areas separated by plastic lining dividers. In addition, a break room was located in close proximity to the workstations.

TURI program staff also emphasize that they "have observed that an individual using methylene chloride directly may be equipped with PPE, while an individual doing another task, such as sanding, may be standing close to the methylene chloride user separated only by a plastic barrier. This individual generally lacks respiratory protection."[196] TURI program staff warn that they have "also heard anecdotally about methylene chloride being used outside a fume hood in research and educational environments."[197]

TURI's observations are relevant to PCE and several other chemicals.

Thus, a simplistic categorization of all non-production workers as ONUs who have uniformly lower levels of exposure is unjustified and understates risks to many workers. EPA should replace this broad category with more refined groupings of near- and far-field workers and, within each grouping, conduct a more detailed exposure analysis which reflects job responsibilities and exposure scenarios specific to different types of workers and chemicals. Implementing this approach for PCE will require EPA to undertake additional outreach to obtain "reasonably available" information – as required by TSCA -- about real world near and far-field exposure scenarios for this substance.

X. EPA's Determinations of PCE's Risks to the Environment Are Flawed and Understated

Throughout the draft risk evaluation, EPA repeatedly underestimates PCE's ecological risks.

First, as it did its evaluation of human health risks, EPA violates TSCA and fundamental risk assessment principles by making use-by-use determinations of unreasonable environmental risk. As explained below, TSCA requires EPA to evaluate the risks presented by "a chemical substance" as a whole, under all of its conditions of use.[198] Moreover, EPA's piecemeal ecological risk determinations understate the effects of PCE on the environment, since if two facilities discharge PCE to the same water body at the same time, EPA may never evaluate the combined impacts on the fish, algae, and other species that are exposed to PCE from both sources. In the draft risk evaluation, EPA references direct PCE discharges from an Occidental Chemical Plant in Geismar, LA (condition of use: manufacturing) and a Honeywell Plant in Geismar, LA (condition of use: processing as a reactant) but does not discuss whether those facilities discharge to the same water bodies and, if so, what the effects of those combined discharges would be.[199] EPA also identifies five different facilities discharging PCE to the Cherry Creek-South Platte River in Colorado, but does not calculate the total risk to the species in that river from their combined

[196] Id.
[197] Id.
[198] 15 U.S.C. 2605(b)(4)(A).
[199] Draft Evaluation at 405-406, 408-409.

discharges.[200] Accordingly, EPA has not evaluated the total risks posed by "the chemical substance," as required by TSCA.

Second, EPA selects ecological concentrations of concern (COCs) that, according to EPA's own calculations, leave the most sensitive species subject to unreasonable risk. Instead of using the NOAEL or LC50 from the most sensitive species, EPA averages NOAELs and LC50s across studies of different species and uses the geometric mean as the COC. For acute impacts to fish, EPA reports an LC50 of 4.82 mg/L for *O. mykiss* (rainbow trout), but selects a COC of 12 mg/L because some other fish species are more tolerant of PCE.[201] In its comments on the methylene chloride risk evaluation, the SACC advised EPA that "dose response curves differ from species-to-species hence small changes in dose may be more impactful for one species than another. As such, it is incorrect to use the geometric mean of LC50 values from multiple species as the measure of lethality … The Committee suggests calculating LC01 values for all species and using the lowest value as the POD."[202] Likewise, instead of relying on the geometric mean of different species' LC50 values here, EPA should use the LC01 for the most sensitive species to determine the PODs for PCE as well.

To measure chronic aquatic toxicity, EPA relies on a 32-day toxicity study on exposure of *Pimphales promelas* (fathead minnow). The study reported "NOAEL - LOAEL values of 0.5 - 1.4 mg/l, respectively, based on growth and mortality of [fathead minnow] exposure to PCE."[203] Instead of relying on the lowest NOAEL, however, EPA took the geometric mean of those values, without any evidence that COC is protective of the most sensitive effect.

EPA also fails to adequately account for uncertainty and inter- and intra-species variability in its ecological risk evaluation. EPA used an assessment factor (AF) in its calculations of acute aquatic risks, and an AF of 10 in its calculations of chronic risks and risks to algae. However, EPA does not establish that any of those AFs are sufficient to address the uncertainty in its environmental risk evaluation. EPA acknowledges that "algae species tend to vary widely in their sensitivity to chemical pollutants, and data were only available for three algal species and may not represent the most sensitive species at a given site."[204] Moreover, EPA's use of the geometric mean of different LC50 values increases the likelihood that its COCs are not adequately protective of all species, and thus warrants a greater AF than the default value used by EPA. In its report on the methylene chloride risk evaluation, the SACC recommended that EPA "[d]evelop LC01 values for test species and select the lowest value for use in hazard quotient (HQ) determination" or, if that is not deemed feasible, to "apply an assessment factor of 100."[205] That recommendation is equally applicable to PCE.

[200] Id. 586.
[201] Id. at 250.
[202] SACC Report on Methylene Chloride Risk Evaluation at 28.
[203] Draft Evaluation at 251.
[204] Id at 255.
[205] SACC Report on Methylene Chloride Risk Evaluation at 29.

Finally, EPA ignores its own risk calculations to conclude that multiple conditions of use with RQs above 1 nonetheless present no unreasonable risk. For the manufacturing of PCE, repackaging/importing, and incorporation of PCE into formulations, EPA calculated unreasonable risks from PCE, with RQs up to **1,453** and up to **299 days** of exceedance per year.[206] Yet, for all of those conditions of use, EPA "does not consider these risks to be unreasonable."[207]

For some conditions of use, EPA's sole explanation for this drastic departure from its own risk calculations is unspecified "uncertainties in the data."[208] Any such uncertainties should result in a more conservative risk characterization, not the wholesale disregard of high ecological risks. For others, EPA notes that some of the greatest dischargers do not have NPDES permits and argues that "lack of a NPDES permit increases the uncertainty in the surface water release estimate for a facility."[209] Lack of a NPDES permits also increases the likelihood of excessive PCE releases, since there is no regulatory mechanism to hold the discharger accountable and readily enforce effluent limitations. EPA's decision to discount its own risk evaluations and to determinations of no unreasonable risk despite RQs of nearly 1,500 does not reflect of the "best available science."

Although EPA has correctly determined that PCE presents an unreasonable risk to the environment, it must address these concerns so that its final evaluation accurate reflects the full magnitude of PCE's harmful ecological impacts as required under TSCA.

XI. EPA Fails to Consider the Risks Associated with PCE's Known Degradation Products

EPA acknowledges that "PCE biodegradation products include potentially hazardous substances including trichloroethylene, cis-1,2 dichloroethene and vinyl chloride."[210] However, EPA fails to consider the known risks associated with PCE degradation in its draft risk evaluation. This oversight is particularly striking given that EPA recently conducted a TSCA risk evaluation for one of those degradation products (TCE), and it failed to consider PCE degradation as a source of TCE in that risk evaluation as well. EPA has established that PCE degradation is a major source of TCE at Superfund sites and elsewhere. Yet EPA has not considered the risks associated with those exposures in either its draft risk evaluation for the parent chemical (PCE) or the degradation product (TCE). Instead, EPA pretends as if those exposures and risks – which are directly attributable to PCE's known, intended and reasonable foreseen use and disposal – do not exist. EPA should account for these risks in the final PCE risk evaluation.

XII. EPA Must Abandon its Flawed TSCA Systematic Review Protocol and Apply Scientifically Valid and Peer Reviewed Systematic Review Methodologies

[206] Draft Evaluation at 474-478.
[207] Id.
[208] Id. at 474.
[209] Id. at 479.
[210] Id. at 62.

As in previous evaluations, EPA is using "systematic review" criteria developed by the TSCA program[211] to evaluate the quality of available data on PCE. Our organizations have previously commented that the TSCA method represents a deeply flawed and unscientific approach to systematic review that will compromise the quality, validity and protectiveness of the 10 risk evaluations.[212] These concerns were summarized in a recent peer-reviewed commentary published in the *American Journal of Public Health*.[213]

A. The TSCA Systematic Review Method Is Deeply Flawed

"Systematic review" is a well-established approach for evaluating and integrating scientific evidence to arrive at judgments about hazard, exposure and risk. The EPA framework risk evaluation rule recognizes the need for a systematic review process in determining chemical risks under TSCA.[214] However, the TSCA protocol departs radically from accepted scientific principles for systematic review adopted by the IOM,[215] [i]the NTP,[216] and EPA's Integrated Risk Information System (IRIS)[217] and endorsed by the NAS[218] and other peer review bodies.

The TSCA approach applies a rigid scoring system to grade the "quality" of studies on chemicals. This system could result in many studies being arbitrarily classified as "poor" or "unacceptable" based on a small number of reporting or methodology limitations that do not negate their overall value for assessing health and environmental risks. The consequence will be that important evidence of public health impacts -- particularly epidemiological studies demonstrating harm in human populations – will be either disregarded or given limited weight in risk evaluations. Other systematic review methodologies

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[211] 83 Fed. Reg. 26998 (June 11, 2018); Application of Systematic Review in TSCA Risk Evaluations, available at https://www.epa.gov/sites/production/files/2018-06/documents/final_application_of_sr_in_tsca_05-31-18.pdf

[212] Comments of Safer Chemicals Healthy Families et al. on Application of Systematic Review in Risk Evaluations under Section 6 of the Amended Toxic Substances Control Act, August 16, 2018, Docket ID EPA-HQ-OPPT-2018-0210. We incorporate these comments by reference.

[213] Singla V, Sutton P, Woodruff TW. (2019) The Environmental Protection Agency Toxic Substances Control Act Systematic Review Method May Curtail Science Used to Inform Policies, With Profound Implications for Public Health. Am J Public Health. doi: 10.2105/AJPH.2019.305068

[214] 82 Fed. Reg. 33726 33734 (July 20, 2017).

[215] Institute of Medicine. Finding What Works in Health Care. Standards for Systematic Review. Washington, D.C.: The National Academies Press.; 2011.

[216] National Toxicology Program. Handbook for Conducting a Literature-Based Health Assessment Using OHAT Approach for Systematic Review and Evidence Integration. In: U.S. Department of Health and Human Services, editor.: Office of Health Assessment and Translation, Division of National Toxicology Program, National Institute of Environmental Health Sciences; 2015.

[217] National Research Council. Review of EPA's Integrated Risk Information System (IRIS) Process. Washington, DC: National Academies Press; 2014.

[218] National Research Council. Review of EPA's Integrated Risk Information System (IRIS) Process. Washington, DC: National Academies Press; 2014; National Research Council. Review of the Environmental Protection Agency's State-of-the-Science Evaluation of Non Monotonic Dose–Response Relationships as They Apply to Endocrine Disruptors. Washington, DC: National Academies Press; 2014; National Academies of Sciences, Engineering, and, Medicine. Application of Systematic Review Methods in an Overall Strategy for Evaluating Low-Dose Toxicity from Endocrine Active Chemicals. Washington, DC: 2017.

do not use numerical scoring systems for assessing study quality and the NAS recommends strongly against such scoring.

The TSCA approach also focuses on one limited aspect of systematic review – study quality – but fails to address other critical elements that the Agency itself recognizes are essential for science-based risk judgments. EPA's July 2017 risk evaluation framework rule defines systematic review as a comprehensive, consistent and transparent process to "identify and evaluate each stream of evidence" and "to integrate evidence as necessary and appropriate based on strengths, limitations, and relevance."[219] Yet the TSCA document lacks any protocol for these important tasks. Experts agree that systematic review methods need to be established in advance of individual evaluations to eliminate the potential for bias and to assure that evidence reviews are conducted using consistent, well-defined criteria. EPA's failure to take this necessary step *before conducting risk evaluation*s has severely compromised the scientific validity of the 10 initial TSCA risk evaluations.

Recent draft risk evaluations have also been based on a "hierarchy of preferences," a new concept that was not part of the original TSCA systematic review document and has likewise not been subject to peer review or public comment. The 1-BP evaluation briefly explains this approach as follows:[220]

"EPA's approach uses a hierarchy of preferences that guide decisions about what types of data/information are included for further analysis, synthesis and integration into the environmental release and occupational exposure assessments. EPA prefers using data with the highest rated quality among those in the higher level of the hierarchy of preferences (i.e. data>modeling>occupational exposure limits or release limits)."

EPA does not explain why some types of studies should receive preference over others in determining the weight of evidence for a particular endpoint and on what basis these studies should be assigned to a "higher level." Thus, there are no objective criteria for determining which evidence to rely on and which to exclude, undermining transparency and consistency in the systematic review process and encouraging subjective judgments.

B. The SACC Has Expressed Numerous Concerns about the TSCA Systematic Review Method

In its peer review of the Draft Risk Evaluation of PV29, the EPA SACC highlighted the following areas of concern with the TSCA systematic review method:

- "The Agency rationale for developing the TSCA SR should include a comparison to other SR approaches and describe the rationale for major differences." [221]
- "The Committee discussed the need to publish peer reviewed pre-established protocols for each of the Agency's reviews prior to performing the actual risk assessment. The protocol for PV29 was

[219] 40 C.F.R. 704.33.
[220] Draft Risk Evaluation for 1-Bromopropane, August 2019, at 45, available at https://www.epa.gov/sites/production/files/2019-08/documents/01._1bp_draft_risk_evaluation_hero_links_external.pdf.
[221] PV 29 SACC Report at 26.

created concurrently with the review, which is contrary to best practices for systematic reviews."[222]

- "The Committee noted that the TSCA SR weighted scoring system may be inappropriate if there is disagreement in the weighting of different metrics. For example, a certain study characteristic that may be a "fatal flaw" would be weighted equally to other more minor elements. The "Agency should provide justification for using a weighted scoring system and the rationale for the specific metrics used for differential weighting in its evaluation of studies."[223]
- "Regarding data integration, the Committee discussed the benefits of including a more thorough and inclusive data integration discussion in the TSCA SR for PV29 … there is a need in the Evaluation for a thorough description and outline for how all evidence and data are integrated into a final weight of evidence conclusion"[224]

These concerns were forcefully underscored in the SACC review of the 1,4-dioxane risk evaluation:[225]

"Committee members did not find the systematic review to be a transparent and objective method to gather the relevant scientific information, score its quality, and integrate the information. Several Committee members brought up examples of references that were not in the systematic review bibliography and/or not considered in the Data Quality evaluation step, but which were used at different stages in the Evaluation. Several Committee members found that it was difficult to determine whether the relevant information was properly evaluated and considered in the Evaluation."

The SACC "noted problems with both the systematic review design and consistent implementation of its protocols," elaborating that:[226]

> Signs that the systematic review design has issues include the need for "backward reference searching" or "targeted supplemental searches," which shouldn't be required if the initial search finds all the relevant references. Similarly, the Committee noted a high fraction of studies where the initial quality score was later changed, indicating that the data quality evaluation protocol is not clearly defined and possibly inconsistently implemented by different reviewers. The automated gray literature search found mostly several off-topic documents and also missed other useful documents.

The SACC report further indicated that "[s]everal Committee members recommended simplifying the scoring system or adopting an existing peer-reviewed method, such as the method used by the National Toxicology Program's Center for the Evaluation of Risks to Human Reproduction (NTP-CERHR)."[227]

[222] Id. at 27.
[223] Id. at 26-7.
[224] Id. at 27.
[225] 1,4-Dioxane and HBCD SACC Report, at 30.
[226] Id. at 31.
[227] Id.

Thus far, the serious issues and concerns raised repeatedly by the SACC have not been addressed by EPA in its most recent draft evaluations. At a minimum, EPA's final risk evaluations must respond fully to the SACC's comments.

The SACC and others have raised more far-reaching concerns about the scientific validity and underpinnings of the TSCA systematic review method. Belatedly, EPA is finally following through on its commitment to seek an NAS review of its method, a course that the SACC has repeatedly recommended and to which EPA agreed nearly a year ago. In the face of the serious concerns of SACC and others and the ongoing NAS review, EPA should stop using the TSCA systematic review protocol. Instead, it must apply one of the established methodologies for systematic review that are consistent with the definition developed by the Institute of Medicine (IOM), such as the National Toxicology Program (NTP) OHAT method or the Navigation Guide Systematic Review Method developed by the University of California San Francisco. These methodologies embody recognized principles of systematic review and have been endorsed by NAS and other peer review bodies.

XIII. EPA's Determinations that Individual Conditions of Use of PCE Pose "No Unreasonable Risk" Violate TSCA

EPA's draft risk evaluation proposes to determine that certain individual conditions of use of PCE pose no unreasonable risk of injury to human health.[228] This "use-by-use" approach to risk determinations is unlawful and threatens to prevent EPA from eliminating the unreasonable risks posed by PCE. TSCA commands that EPA determine "whether" "a chemical substance"—not particular uses of a chemical substance—presents an unreasonable risk in a single, comprehensive determination. 15 U.S.C. § 2605(b)(4)(A); *see id*. § 2605(a) (requiring risk-management rule if "any combination of" a chemical's conditions of use presents "an unreasonable risk").[229] TSCA section 6(b)'s requirement that EPA determine "whether" the substance poses an unreasonable risk "indicates a binary choice." *SAS Inst. Inc. v. Iancu*, 138 S. Ct. 1348, 1355-56 (2018). This holistic risk determination for PCE must reflect EPA's evaluation of all of PCE's conditions of use considered in combination, and EPA must "integrate and assess available information on hazards and exposures" for all of PCE's uses, including where relevant "the likely duration, intensity, frequency, and number of exposures under the conditions of use of the chemical substance." 15 U.S.C. § 2605(b)(4)(F)(i), (iv). Piecemeal determinations that isolated conditions of use of PCE pose "no unreasonable risk" violate TSCA's plain language.

EPA must revise its risk evaluation for PCE to make a single risk determination for the chemical substance as a whole. Based on EPA's findings that nearly all conditions of use present unreasonable risks to health, EPA must conclude under TSCA section 6(b)(4)(A) that PCE presents an unreasonable risk to human health.

Conclusion

[228] Id. at 35.

[229] To the extent that EPA's regulations purport to allow this "use-by-use" approach to risk determinations, *see* 40 C.F.R. §§ 702.41(a)(9), 702.47, 702.49(d), the regulations are unlawful and violate TSCA.

We appreciate this opportunity to comment on the draft PCE risk evaluation.

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