



Environmental Defense Fund
Comments on the Draft Risk Evaluation of 1,4-dioxane
Docket ID: EPA-HQ-OPPT-2019-0238

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Environmental Defense Fund (EDF) appreciates the opportunity to provide comments to the Environmental Protection Agency (EPA) on the draft risk evaluation for 1,4-dioxane being prepared under section 6(b)(4) of the Toxic Substances Control Act (TSCA) as amended by the Lautenberg Act, enacted on June 22, 2016.¹ We request that our comments also be provided to the SACC for its review and consideration.

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

Summary

In its draft risk evaluation for 1,4-dioxane, the Environmental Protection Agency (EPA) has grossly understated the risks that workers and the environment face from exposure to the chemical. EPA has also abdicated its responsibility under the Toxic Substances Control Act (TSCA) to identify and evaluate the risks the chemical presents to consumers and the general population by excluding from its risk evaluation conditions of use and exposures that are known or reasonably foreseen. EPA has not met its mandatory duty under TSCA to identify and evaluate the risks to vulnerable subpopulations, falsely asserting there is no evidence that certain subpopulations are or may be more susceptible to adverse effects from exposure to the chemical. EPA has utterly failed to utilize the enhanced authorities Congress granted it in 2016 to ensure that it has or obtains robust information on 1,4-dioxane's uses, hazards and exposures, resulting

¹ U.S. EPA, *Draft Risk Evaluation for 1,4-Dioxane CASRN: 123-91-1* (June 2019), <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0238-0011>. All further citations to the draft risk evaluation for 1,4-dioxane consist of only a page number in parentheses.

in serious information and analytic gaps and deficiencies that severely undermine the scientific quality of its risk evaluation.

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

Among the major concerns addressed in Part I of these comments are the following:

Cross-cutting concerns

- EPA has ignored evidence that some subpopulations are or may be more susceptible to 1,4-dioxane exposures than the general population (see section 1.A).
- EPA has distorted OSHA requirements and over-relied on personal protective equipment and safety data sheets, ignoring their real-world limitations (see section 1.B).
- EPA has, without scientific basis, sought to sow doubt on the use of a linear, non-threshold model for 1,4-dioxane's carcinogenicity, an approach that reflects longstanding agency policy and consensus in the scientific community (see section 1.D).
- EPA has distorted the nature of information that companies submitted to the European Chemicals Agency and has relied on industry-prepared summaries without access to the full studies (see sec. 1.E.i and ii).
- EPA has dismissed the liver tumors observed in female mice in the key oral cancer study it uses to extrapolate dermal cancer risks. Its insufficient rationale ignores the IRIS program's basis for including these tumors and its determination that they are the most sensitive endpoint, which has been affirmed through peer review. As a result, cancer risk is significantly understated, a concern also noted by the New Jersey Department of Environmental Protection (see section 1.E.iv.)

Unwarranted exclusions of conditions of use and exposure pathways

- EPA has excluded *all* exposures and risks to consumers (and to workers from at least one use), based on 1,4-dioxane's presence in such products as a byproduct rather than being intentionally used, a distinction without any basis in science (see section 2.A).
- EPA has excluded from its risk evaluation all general population exposures to 1,4 dioxane, based on EPA's unsupported assertion that existing regulatory programs under other statutes EPA administers have addressed or are in the process of addressing potential risks of 1,4-dioxane in all media pathways (see section 2.B).
- EPA has ignored 1,4-dioxane's use and disposal in hydraulic fracturing fluids and produced wastewater and the resultant risks to the environment, the general population and workers (see section 2.D).

Need to adopt a linear, no-threshold approach for 1,4-dioxane's carcinogenicity

- EPA must adopt a linear, no-threshold approach for 1,4-dioxane's carcinogenicity, based on available evidence, scientific rebuttals of a cytotoxic mode of action, and policies firmly rooted in scientific and health-protective principles (see section 3).

Serious data gaps

- EPA's determination of no unreasonable risk to the environment is not supported by sufficient evidence (see section 4.A).
- EPA's occupational exposure assessment is not supported by sufficient inhalation or dermal exposure data (see section 4.B).

Analytic gaps and deficiencies

- EPA's disregard of environmental monitoring data led to an overreliance on predictive modeling lacking adequate uncertainty analysis (see section 5.A).
- EPA has failed to consider numerous environmental releases and has inappropriately dismissed sediment, groundwater and biosolids exposure pathways (see section 5.A).
- EPA has failed to apply all necessary uncertainty factors in calculating the benchmark margins of exposure, resulting in inaccurate risk characterizations (see section 5.B.i).
- EPA has failed to identify and analyze risks to people living in proximity to conditions of use and sources of contamination and environmental release (see section 5.B.ii).
- EPA fails to consider combined exposures to workers from different routes and sources and has omitted a number of workplace-related exposure scenarios (see section 5.B.iii and iv).
- EPA's assessments of worker and occupational non-user (ONU) dermal and inhalation risks suffer from numerous flaws (see section 5.B.v-viii).

Understating risks to workers

- EPA has significantly understated the extent of risks to workers it has identified (see section 6).

Flaws in EPA's unreasonable risk definition and determinations

- EPA's "expectation" of compliance with existing laws and standards as a basis for not finding unreasonable risk is unwarranted (see section 7.A).
- EPA finds no unreasonable risk even when the high-end risk exceeds relevant benchmarks, an approach that is not adequately protective (see section 7.B).
- EPA's allowance of a 1 in 10,000 cancer risk for workers is a major and unwarranted deviation from longstanding agency policy and practice to regulate upon finding cancer risks on the order of 1 in 1 million (see section 7.C).
- EPA's characterizations of its dermal risk analysis in its risk determinations are misleading and flawed (see section 7.E).

Flaws in EPA’s systematic review

- EPA’s systematic review to support the risk evaluation is flawed and not reflective of best practices and results in an arbitrary and capricious analysis (see section 8).

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PART II

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 i. *The plain text requires EPA to consider all hazards, exposures, and conditions of use.*98

 ii. *TSCA’s overall structure requires EPA to consider all hazards, exposures, and conditions of use.*101

 iii. *TSCA’s purpose, as well as basic logical reasoning and the best available science, require EPA to consider all hazards, exposures, and conditions of use to assess a chemical substance as a whole.*102

 iv. *The legislative history requires EPA to integrate a chemical’s exposure and hazard information and nothing suggests that EPA can ignore existing exposures and hazards.*102

 v. *EPA has inappropriately excluded all consumer uses and all contamination of industrial, commercial and consumer products.*103

 vi. *EPA has failed to identify certain conditions of use identified in the 2002 EU Risk Assessment for 1,4-dioxane.*105

 B. EPA’s own risk evaluation rule requires that EPA consider all relevant hazards and all exposures under the conditions of use within the risk evaluation.106

 C. The draft risk evaluation is incoherent and arbitrary and capricious because of EPA’s approach to hazard, exposure, and conditions of use.107

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PART I

1. Broad/cross-cutting concerns

A. Insufficient consideration of susceptible populations

In several key sections of the draft, EPA dismisses the potential for some worker subpopulations to be more susceptible to 1,4-dioxane, inappropriately asserting there are none (pp. 21, 150). For example, the Agency states that “the results of the available human health data for all routes of exposure evaluated (i.e., dermal and inhalation) indicate that there is no evidence of increased susceptibility for any single group relative to the general population” (p. 21). Furthermore, the Agency makes the unsupported and clearly erroneous assumption that all workers are “healthy” in its risk characterization (p. 132).

Yet, as EPA acknowledges elsewhere in the draft (p. 108) but fails to address in its analysis of risks, there may be numerous such worker subpopulations, including those with pre-existing conditions that affect the liver or impair metabolism (e.g., nonalcoholic fatty liver disease, which is estimated by the Mayo Clinic to impact between 80-100 million individuals in the United States),² or that affect the kidneys, upper respiratory system, or other organs targeted by 1,4-dioxane. Individuals with elevated alcohol intake may also exhibit increased liver sensitivity, yet EPA does not consider this sizeable subpopulation. Additionally, as the Agency acknowledges (p. 108):

variations in CYP enzyme expression may contribute to susceptibility because multiple CYP enzymes are involved in metabolism of 1,4-dioxane, including CYP2E1. There are large variations in CYP2E1 expression and functionality in humans (Ligocka et al., 2003) and similar variation in other CYPs involved in 1,4-dioxane metabolism are possible.

EPA also acknowledges that the database for potential reproductive and developmental toxicity of 1,4-dioxane is deficient (p. 108), and hence that “it is not known whether or not pregnant women in the workplace may be at greater risk from exposure.” Yet in section 5.3.4 (p. 150), EPA states that it “did not include women of reproductive age or pregnant women who may work with 1,4-dioxane or children ages 16-21 because the acute effects on liver enzymes and CNS effects are not expected to preferentially affect women or developing children.” Here, EPA makes an inappropriate leap to claim that a lack of data is equivalent to lack of risk. Pregnant women are expressly identified as a potentially exposed or susceptible subpopulation under

² NONALCOHOLIC FATTY LIVER DISEASE, <https://www.mayoclinic.org/diseases-conditions/nonalcoholic-fatty-liver-disease/symptoms-causes/syc-20354567> (last visited Jul. 19, 2019).

TSCA, yet EPA ignores any potential concern and takes no steps to fill this crucial data gap. (See “key data gaps” below.)

Part I sec. 5.B. of these comments discusses other serious omissions in EPA’s evaluation of exposures and risks to potentially exposed or susceptible subpopulations.

B. Overreliance on personal protective equipment and safety data sheets and overstatements of OSHA requirements

EPA’s risk determinations heavily rely on an assumption that all workers at all points in the value chain and lifecycle of 1,4-dioxane will always use personal protective equipment (gloves and respirators) and that it will be universally effective:

EPA expects there is compliance with federal and state laws, such as worker protection standards, unless case-specific facts indicate otherwise, and therefore existing OSHA regulations for worker protection and hazard communication will result in use of appropriate PPE consistent with the applicable SDSs in a manner adequate to protect workers (p. 175).

In addition to grossly distorting OSHA authorities and requirements (see below), EPA has provided no data or analysis whatsoever to support these sweeping assumptions. OSHA itself has highlighted the major limitations of reliance on PPE with regard to both extent of use and effectiveness, as has EPA in the recent past. These issues are discussed in detail in previous EDF comments, which are incorporated here by reference.³

In a few places in the draft, EPA acknowledges some of the limitations of PPE (pp. 48, 75), and the preferability of other options higher up in the industrial hygiene hierarchy of controls (pp. 48, 52, 74). But when it comes to determining risk, those limitations and preferences fall away and EPA exclusively relies on “expected” use of PPE to mitigate the risks it has identified.

EPA repeatedly overstates or distorts OSHA’s authorities and requirements, claiming that OSHA requires employers to provide PPE (p. 48), implying that OSHA requires the use of respirators for 1,4-dioxane (p. 52), and implying that OSHA’s requirement for safety data sheets (SDSs) is sufficient to ensure use of protective measures such as PPE by all downstream users of 1,4-dioxane (p. 60). In fact, OSHA authorities and requirements are quite limited and leave most of

³ See, e.g., EDF Comments on TSCA Review and Scoping for First 10 Chemicals under the Lautenberg Act at 6 (Mar. 15, 2017), <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0736-0046>; EDF Comments on Significant New Uses of Chemical Substances; Updates to the Hazard Communication Program and Regulatory Framework; Minor Amendments to Reporting Requirements for Premanufacture Notices (Nov. 21, 2016), <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2014-0650-0052>.

their applicability to be decided by employers, not OSHA. Among other things, OSHA regulations do not require that persons comply with SDSs. EDF has described these limitations in detail in a recent series of posts to our EDF Health blog.⁴ EDF incorporates those by reference. EDF also incorporates by reference the comments submitted by Jonathan Kalmuss-Katz and Randy Rabinowitz.⁵

Even if compliance with SDSs *were* mandatory, reliance on them would still be insufficient to ensure use of protective measures by all downstream users. Significant evidence demonstrates that SDSs are often of insufficient quality to be useful and are frequently not understood. Nicol et al. (2008) conducted a systematic search of the literature and identified serious problems with the use of SDSs as hazard communication tools: they are often inaccurate, incomplete, and too technical for workers to understand.⁶ The 2012 OSHA Hazard Communication Standard corroborates these findings.⁷ For example, the Standard reports that “several studies show that employees do not understand approximately one-third of the safety and health information listed on SDSs prepared in accordance with the current standard” and that “[s]tudies also report that roughly 40% of persons reviewing SDSs found them difficult to understand.”⁸

Furthermore, studies conducted by Eastlake et al. (2012) and Dodson et al. (2019), which examined SDSs for engineered nanomaterials developed after the 2012 update to the OSHA Hazard Communication Standard, demonstrate that SDSs often contain insufficient information to adequately communicate health hazards.^{9,10} For example, Hodson et al. (2019) found that of 67 SDSs evaluated, 35.8% were determined to be unreliable based on the Klimisch criteria and 79% “need significant improvement” based on the Eastlake et al. (2012) ranking scheme. The authors concluded “the quality of information on many [nanomaterial SDSs] still cannot be

⁴ See Appendix I.

⁵ Comments submitted by Jonathan Kalmuss-Katz, Staff Attorney, Earthjustice and Randy Rabinowitz, Executive Director, Occupational Safety & Health (OSH) Law Project <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0238-0021>.

⁶ Anne-Marie Nicol, et al., *Accuracy, comprehensibility, and use of material safety data sheets: A review*, 51:11 AM. J. OF INDUS. MED. 861-76 (Jul. 2008), <https://onlinelibrary.wiley.com/doi/abs/10.1002/ajim.20613>.

⁷ 77 Fed. Reg. 17574, 17593-95, 17603 (Mar. 26, 2019).

⁸ *Id.* at 17603; see also OSHA, *Inspection Procedures for the Hazard Communication Standard (HCS 2012)* (Jul. 2015), https://www.osha.gov/OshDoc/Directive_pdf/CPL_02-02-079.pdf.

⁹ Adrienne Eastlake, et al., *A critical evaluation of material safety data sheets (MSDSs) for engineered nanomaterial*, 19:5 J. OF CHEM. HEALTH & SAFETY 1-8 (Sept.-Oct. 2012), <https://www.ncbi.nlm.nih.gov/pubmed/26766894>.

¹⁰ Laura Hodson, et al., *An evaluation of engineered nanomaterial safety data sheets for safety and health information post implementation of the revised hazard communication standard*, 26:2 J. OF CHEM. HEALTH & SAFETY 12-18 (Mar.-Apr. 2019), <https://www.ncbi.nlm.nih.gov/pubmed/30906483>.

relied upon to offer adequate information on the inherent health and safety hazards, including handling and storage of engineered nanomaterials.”

EPA’s reliance on OSHA also runs contrary to TSCA §§ 6 and 9. 15 U.S.C. § 2608(a)(1). TSCA § 6(b) contemplates that EPA will prepare risk evaluations without considering the risk management options; Congress specifically bifurcated the analysis, directing EPA to prepare a comprehensive risk evaluation *without* consideration of non-risk factors under TSCA § 6(b) and then to consider risk management at a separate step governed by TSCA § 6(a) and 6(c). 15 U.S.C. § 2605.

Similarly, TSCA § 9 describes how TSCA interacts with other laws, and TSCA § 9 specifically contemplates that EPA will prepare its risk evaluations without relying on other laws and then will consider the ability of other agencies to address risk *after* EPA has completed the risk evaluation. *See* 15 U.S.C. § 2608. And EPA may refer a risk to another agency only upon finding “that such risk may be prevented or reduced to a sufficient extent by action taken under a Federal law” under TSCA § 9(a). *See* 15 U.S.C. § 2608(a). This finding requires that EPA have completed the risk evaluation identifying the risk to be prevented or reduced. Thus, TSCA does not provide for EPA to consider whether OSHA could address the risks presented by a chemical until *after* EPA has completed the risk evaluation. *See id.* Moreover, the Courts have specifically admonished agencies to implement their own statutory schemes, rather than attempt to defer their duties to other agencies. *See Environmental Defense Fund, Inc. v. United States Dep’t of Health, Education & Welfare*, 428 F.2d 1083, 1088 (D.C. Cir. 1970) (“For either department to relinquish its responsibility would destroy the regulatory scheme enacted by Congress.”).

C. Evidence of bias

Throughout the draft, EPA treats differently its determinations of unreasonable risk vs. no unreasonable risk in a consistently skewed manner. Three illustrations of this bias follow:

- EPA’s risk determination summary (pp. 21-22) uses direct, unqualified language whenever EPA is asserting it found no unreasonable risk for certain conditions of use. In contrast, EPA’s statements describing where it found risk are heavily caveated and tentative. For example, on p. 22 EPA states (emphases added) that it

has preliminarily concluded that the aforementioned conditions of use present an unreasonable risk of injury to health, as set forth in the risk determination section of this draft risk evaluation. This draft document’s preliminarily [sic] determination of unreasonable risk does not mean that this is EPA’s final conclusion. EPA will consider further input through scientific and public review.

No such caveats accompany the statements of no unreasonable risk in the preceding and following paragraphs of that key section.

- In the summary table (Table 6-1, pp. 157-175) that provides EPA’s risk determinations, each of the “Risk Considerations” sections repeatedly emphasizes those factors EPA believes overestimate the risk. In contrast, EPA relegates discussion of any factors that could lead to underestimation to less prominent sections of the draft. For example, on p. 55 (emphasis added) EPA acknowledges that the manufacturing worker exposure data on which it relies

mostly lacked specific descriptions of worker tasks, exposure sources, and possible engineering controls to provide context. EPA assumed that the 2016 BASF data are PBZ measurements relevant to worker activities and are also 8-hour TWA measurements. *This assumption could underestimate exposures.* The sampling rate was missing for some of the 2016 data, so EPA assumed the same sampling rate was applied for other data in the set. It is uncertain to what extent the limited monitoring data used to estimate inhalation exposures for this scenario that could be representative of occupational exposures in other manufacturing facilities of 1,4-dioxane.

None of these factors is mentioned in either the “Assumptions and Key Sources of Uncertainty” section (pp. 145-150) or in the final summary risk determinations in Table 6-1.

- EPA states that the degree of confidence or uncertainty in the data it has will be a factor in making its risk determinations (pp. 152, 154), but never explains how this will factor in or which way it will cut, which renders its application wholly arbitrary. We have one indication, however: On p. 169 EPA invokes the poor quality of its data as a basis for concluding there is no unreasonable risk to ONUs. EPA took no steps to require the development of better data; instead it opted to equate this lack of sufficient data with affirmative evidence of no unreasonable risk.

It also appears significant that the only place in the entire draft risk evaluation where EPA uses boldfaced text to emphasize its conclusion is on p. 101, to highlight that existing data do “**not** support a mutagenic mode of action hypothesis at low doses in vivo” (emphasis in original). This boldfacing of a statement unsupported by scientific consensus (see Part I sec. 1.D.), is further evidence of the Agency’s bias.

D. Inconsistencies with Agency guidelines

In several instances, EPA inappropriately dismisses key data relevant to genotoxicity. For example, when discussing the Itoh and Hattori (2019) publication, EPA reports that the authors discounted the statistically significant increase in micronucleated immature erythrocytes (MNIE) because these changes were within the historical control range. However, this decision contradicts EPA Cancer Guidelines,¹¹ which states that:

the standard for determining statistical significance of tumor incidence comes from a comparison of tumors in dosed animals with those in concurrent control animals...Generally speaking, statistically significant increases in tumor should not be discounted simply because incidence rates in the treated groups are within the range of historical controls or because incidence rates in concurrent controls are somewhat lower than average.

EPA's Benchmark Dose Guidance¹² also provides relevant commentary:

Typically, all endpoints within a study that a risk assessor has judged to be relevant to the exposure should be considered from modeling. This will help ensure that no endpoints with the potential of having the most sensitive effect for risk assessment applications, usually having the lowest BMDL, are excluded from the analysis.

EPA also downplays its own cancer risk assessment guidance regarding when a linear no-threshold model should be used: "In the absence of other information about MOA [mode of action], *EPA often takes* the health protective approach of assuming a linear no-threshold risk model consistent with a mutagenic mode of action" (p. 98, emphasis added). This approach is inconsistent with the Agency's cancer guidelines, which direct the agency to use the default linear approach in the absence of an alternative known MOA.¹³

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.

¹¹ U.S. EPA, *Guidelines for Carcinogen Risk Assessment* at pp. 2-20-2-21 (Mar. 2015), https://www.epa.gov/sites/production/files/2013-09/documents/cancer_guidelines_final_3-25-05.pdf.

¹² U.S. EPA, *Benchmark Dose Technical Guidance* at pp. 14-15 (June 2012), https://www.epa.gov/sites/production/files/2015-01/documents/benchmark_dose_guidance.pdf.

¹³ U.S. EPA, *Guidelines for Carcinogen Risk Assessment* at p. 3-21 (Mar. 2015), https://www.epa.gov/sites/production/files/2013-09/documents/cancer_guidelines_final_3-25-05.pdf.

By contrast, in this risk evaluation, EPA continues to develop and present the threshold non-linear model in tandem with the default linear no-threshold model despite the scientific consensus otherwise that there is insufficient evidence to support the threshold approach.¹⁴

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

E. Lack of transparency

- i. EPA must accurately describe its citations to dossiers submitted to the European Chemicals Agency as containing only study summaries, not full study reports, that are prepared by the registrant companies, not by ECHA.*

EPA also cites and uses data obtained from “ECHA dossiers” – which are the registration dossiers submitted by companies, pursuant to the European Union’s Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) Regulation, to the European Chemicals Agency (ECHA) (p. 39, 211, 223). EPA even decided that studies cited in these ECHA dossiers would bypass the data screening step and move directly to the data evaluation step (pp. 39, 42). However, the ECHA dossiers do not contain full study reports, only study summaries that are prepared and submitted by companies to ECHA, and there is no indication that the dossiers have been independently evaluated by ECHA or other government authorities in the EU.¹⁵ A

¹⁴ See, e.g., Mich. Dep’t of Env’tl. Quality, Toxics Steering Group, 1,4-Dioxane Subcomm., Review of a 1,4-Dioxane Presentation by Michael Dourson, Ph.D. on October 8, 2013 (Feb. 2015), https://www.michigan.gov/documents/deq/deq-aqd-toxics-14-DioxaneTSG_Report_2015_487415_7.pdf; N.J. Dep’t of Env’tl. Protection, Response to Public Input on Draft Interim Ground Water Quality Criteria and Draft Interim Practical Quantitation Levels for Eleven Chemicals at pp. 11-17, <https://www.state.nj.us/dep/dsr/supportdocs/11-chemicals-response.pdf>; U.S. EPA, IRIS, *Toxicological Review of 1,4-Dioxane (With Inhalation Update)* at p. 137 (Sept. 2013), https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0326tr.pdf.

¹⁵ See DOSSIER EVALUATION STATUS, https://echa.europa.eu/information-on-chemicals/dossier-evaluation-status?p_p_id=disslists_WAR_disslistsportlet&p_p_lifecycle=1&p_p_state=normal&p_p_mode=view&p_p_col_id=column-1&p_p_col_pos=3&p_p_col_count=

disclaimer at the bottom of each dossier entry states: “This information has not been reviewed or verified by the Agency or any other authority. The content is subject to change without prior notice.”¹⁶

In citing information available through ECHA, EPA must clearly distinguish between industry data that have not been evaluated, industry data that have been evaluated by ECHA or other government authorities in the EU, and information that ECHA has itself developed or provided.

EPA also needs to distinguish between cases where only summaries are available and not full study reports, and to make clear that those summaries are prepared by the REACH registrants and not by ECHA or other government authorities in the EU. EPA’s HERO entries for references in the draft risk evaluation that cite the REACH dossier for 1,4-dioxane erroneously state that the author of the study summaries and underlying studies is ECHA itself, when in fact it is the registrant. Examples of such erroneous entries include:

- https://hero.epa.gov/hero/index.cfm/reference/details/reference_id/3809089 (linked to on p. 211 of the draft risk evaluation as “ECHA, 1996”) and
- https://hero.epa.gov/hero/index.cfm/reference/details/reference_id/3809095 (linked to on p. 223 of the draft risk evaluation as “ECHA, 2014”).

While in this draft risk evaluation EPA has obscured the scope and authorship of the data provided in the ECHA database, in another more recently released draft risk evaluation, for 1-bromopropane, EPA has acknowledged that ECHA provides only study summaries, not full study reports.¹⁷

ii. EPA must obtain and make public the full studies.

From the draft risk evaluation, it is not at all clear that EPA has access to the full study reports for all of the studies it cites. While in this draft risk evaluation EPA has obscured whether it has obtained full study reports, in another more recently released draft risk evaluation, for 1-

[4&_disslists_WAR_disslistsportlet_javax.portlet.action=searchDissLists](#) (ECHA’s database has no dossier evaluation status for 1,4-dioxane) (last visited Aug. 19, 2019).

¹⁶ See, e.g., 1,4-DIOXANE WATER SOLUBILITY, <https://echa.europa.eu/registration-dossier/-/registered-dossier/15842/4/9/?documentUUID=1445d971-5945-4b59-891d-03a672763eb3> (last visited Aug. 19, 2019).

¹⁷ U.S. EPA, *Draft Risk Evaluation for 1-Bromopropane* pp. 47, 138 (August 2019), https://www.epa.gov/sites/production/files/2019-08/documents/01_1-bp_draft_risk_evaluation_hero_links_external.pdf.

bromopropane, EPA acknowledged that ECHA provides only study summaries and that EPA has not been able to obtain the full study reports.¹⁸

EPA needs to ensure it has obtained copies of the full studies, including those for which it cites ECHA as the source; as noted above, ECHA dossiers do not contain full study reports, only study summaries prepared by the registrants. EPA should request that submitters of information to EPA always provide copies of full studies, as well as underlying data whenever reasonably available or obtainable. Setting aside concerns about partiality, EPA needs the underlying data to ascertain the accuracy of the information and associated statements or conclusions, as well as to determine how much confidence or uncertainty applies to a particular submission.

EPA also needs to make copies of full studies on which it relies available to the public, including those to which it refers in the draft risk evaluation as identified in the ECHA Database or in REACH dossiers. EPA cites ECHA as a source and links to the dossier for 1,4-dioxane that contains registrants submissions to ECHA under REACH (*see, e.g.*, pp. 211, 223). However, only the study summaries are available through ECHA's dossiers, not the full studies. As EDF has explained in prior comments, there are numerous reasons that it is important that the public have access to full studies and the underlying information, not simply robust or other study summaries.¹⁹ Without access to full studies, the public will be challenged or unable to assess and comment on the quality of the studies used by the agency, including the extent to which the requirements of section 26(h) and 26(i) are met. Even the best study summaries are incomplete descriptions that do not allow for an independent examination of study quality and conclusions reached by authors. Common examples of such conclusions include, "findings were not statistically significant," "findings are within the range of historical controls," and "effects observed were non-linear [and therefore biologically questionable or irrelevant]." Divorced from the details of the actual design and results of a study, it is impossible to evaluate the appropriateness of such conclusions. It is important that EPA obtain the full studies, both so that EPA staff have access and so that EPA can make them publicly available. EPA should make such information public and easily searchable through online portals such as the Health and Environmental Research Online (HERO) database. EDF incorporates and reiterates the numerous points made in support of public access to the full studies here. *Id.* These points also support the importance of EPA obtaining the full studies.

¹⁸ U.S. EPA, *Draft Risk Evaluation for 1-Bromopropane* pp. 47, 138 (August 2019), https://www.epa.gov/sites/production/files/2019-08/documents/01.1-bp_draft_risk_evaluation_hero_links_external.pdf.

¹⁹ *See, e.g.*, EDF Comments on Procedures for Chemical Risk Evaluation under the Amended Toxic Substances Control Act at p.37, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0654-0074>.

iii. Missing citations, sources, & tables

Throughout the draft risk evaluation, EPA cites sources that are not publicly accessible, including a number of key EPA documents. They are not in the docket, the HERO entries for them lack hyperlinks, and we often have been unable to locate them through internet searches. Five examples:

- (1) Bronaugh 1982, which, as indicated in HERO,²⁰ is a chapter of a book about cosmetics that EPA cites 16 times as the basis for its skin absorption estimates. EPA has not provided access to it through HERO or in response to a request we made on July 10th, 2019. After several unsuccessful follow-up requests, EDF purchased the book.
- (2) US EPA, 2018a, which EPA used to estimate exposures to 1,4-dioxane in spray foam applications in the absence of any monitoring data (p. 68).
- (3) McConnell, 2013, a technical report which EPA uses to describe cytotoxicity as a potential MOA of liver toxicity and cancer.
- (4) BASF 2016, BASF 2018(a), and BASF 2018(b). BASF 2016 includes no link in HERO.²¹ While the BASF 2018(a) source includes a link, it is routed to an “error” in regulations.gov. For BASF 2018(b), HERO provides a link to a BASF webpage that requires a login to access the referenced 1,4 dioxane Safety Data Sheet.
- (5) JBRC, 1998, a 2-year animal study conducted in Japan, which EPA cites over 30 times.

EPA also omits a table explaining the calculations for section 4.2.6.2.5: Chronic Non-Cancer POD for Dermal Exposures extrapolated from Chronic Inhalation Studies (p. 117). This table should be included to ensure transparency.

EPA’s failure to provide all relevant materials violates the requirements of public notice and comment codified in TSCA § 6(b)(4)(H). 15 U.S.C. § 2605(b)(4)(H). By requiring notice-and-comment at the draft risk evaluation stage, Congress meant to ensure a meaningful notice-and-comment opportunity as embodied by the procedural requirements of the Administrative Procedure Act (APA). “Under APA notice and comment requirements, ‘[a]mong the information that must be revealed for public evaluation are the “technical studies and data” upon which the agency relies [in its rulemaking].’” *Am. Radio Relay League, Inc. v. FCC*, 524 F.3d 227, 236 (D.C. Cir. 2008) (quoting *Chamber of Commerce v. SEC*, 443 F.3d 890, 899 (D.C. Cir. 2006)). “[T]he court explained long ago that ‘[i]n order to allow for useful criticism, it is

²⁰ HERO, https://hero.epa.gov/hero/index.cfm/reference/details/reference_id/196146 (last visited Aug. 19, 2019).

²¹ Although there is a data table provided on pp. 255-56 that appears to summarize the monitoring data from BASF, EPA has nevertheless failed to provide access to the underlying report that explains the methodology.

especially important for the agency to identify and make available technical studies and data that it has employed in reaching the decisions to propose particular rules.” *Id.* (quoting *Conn. Light & Power Co. v. Nuclear Regulatory Comm’n*, 673 F.2d 525, 530 (D.C. Cir. 1982)). “It would appear to be a fairly obvious proposition that studies upon which an agency relies in promulgating a rule must be made available during the rulemaking in order to afford interested persons meaningful notice and an opportunity for comment.” *Id.* at 237. Here, EPA has failed to provide certain studies and data, and thus has deprived the public with an opportunity to review and comment on those materials.

iv. Insufficient justifications for key decisions

Table 4-12 (p. 126) contains the oral and associated dermal cancer slope factors (CSF) that EPA considers for its risk characterization. Included in this table are data for male and female rats as well as male mice from Kano et al. (2009). However, missing from this table is any mention of the hepatocellular tumors observed in female mice in the Kano et al. (2009) study. This omission is highly problematic, given that in the 2013 IRIS assessment,²² EPA selected this as the most sensitive endpoint and the basis for the oral CSF.

In the current risk evaluation, EPA seeks to justify its decision to omit the female mouse liver tumors by stating that “female mouse hepatocellular carcinoma data from Kano et al. (2009) were not modeled due to the difficulties that were previously noted in the US EPA (2013c) IRIS assessment” (p. 334). However, EPA fails to mention that IRIS was able to resolve this issue by “[applying] other BMD models...to the female mouse liver tumor dataset to achieve an adequate fit.”²³ Overall, EPA has not provided sufficient justification for its decision to drop the female mouse liver data, which had previously been identified by the IRIS program (and supported by internal and external peer-reviewers) as the most sensitive endpoint and the basis for the oral CSF.

We note that similar concerns about this omission were raised by the New Jersey Department of Environmental Protection in its July 9, 2019 comments on the draft risk evaluation.²⁴

This decision is highly consequential. In the 2013 IRIS assessment, EPA estimates an oral CSF of 0.1 (mg/kg/day), based on these female mouse liver tumor data. By contrast, in this risk evaluation, EPA estimates an oral CSF of 0.021 (mg/kg/day), based on combined tumors in male rats (Table 4-12) – *approximately 5-fold less protective*.

²² U.S. EPA, IRIS, *Toxicological Review of 1,4-Dioxane (With Inhalation Update)* (Sept. 2013), https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0326tr.pdf.

²³ U.S. EPA, IRIS, *Toxicological Review of 1,4-Dioxane (With Inhalation Update)* at p. 138 (Sept. 2013), https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0326tr.pdf.

²⁴ N.J. Dep’t of Env’tl. Protection Comment on Draft Risk Evaluation for 1,4-Dioxane, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0238-0014>.

EPA's Integrated Risk Information System (IRIS) has developed hazard and dose-response values for 1,4-dioxane through a rigorous process that included peer review. EPA should not lightly disregard this valuable work, and should continue to rely on these values as it has in the past. Should it choose not to, EPA must identify and explain any decision to deviate from these values, as well as the scientific basis for such deviation. Any such differences must be based on compelling scientific evidence and explicitly interrogated through the peer review process.

In several other sections of the document, EPA does not provide sufficient support for key analytical and modeling decisions it has made. For example, on pages 332-333, the Agency indicates that distinct p-value thresholds were used to judge goodness-of-fit for cancer ($\alpha=0.05$) vs. non-cancer ($\alpha=0.1$) models. EPA Benchmark Dose Modeling Guidelines "recommend that $\alpha=0.1$ be used to compute the critical value for goodness of fit...[except] when there is a priori reason to prefer a specific model."²⁵ In this risk evaluation, however, EPA has provided no justification for choosing an alternative threshold for the cancer model.

2. Exclusions of conditions of use and exposures

A. Exclusion of exposures when 1,4-dioxane is present as a byproduct

EPA has excluded *all* exposures and risks to consumers (and to workers from at least one industrial use: closed system functional fluids), based on 1,4-dioxane's presence in such products as a byproduct rather than being intentionally used. This distinction has no basis in TSCA, which never differentiates between the intentional and byproduct presence of a chemical. TSCA requires EPA to evaluate the risks of all known and reasonably foreseen uses of a chemical, which clearly encompass byproducts. Nor does the distinction have any basis in science, as byproducts can expose people and the environment just as surely as intentionally used chemicals. EPA's exclusion will result in a deficient and erroneous evaluation and determination of the chemical's risks.

Instead EPA asserts it can and will evaluate the risks of 1,4-dioxane in the risk evaluations for the ethoxylated chemicals that give rise to it as a byproduct (p. 28). But there are dozens or hundreds of such chemicals used in dozens or hundreds of types of consumer, commercial and industrial products, as EPA described in its scope document:²⁶

1,4-Dioxane may be produced as a reaction by-product, particularly in chemicals which are produced by ethoxylation. These include alkyl ether sulphates (AES,

²⁵ U.S. EPA, *Benchmark Dose Technical Guidance* at p. 33 (June 2012), https://www.epa.gov/sites/production/files/2015-01/documents/benchmark_dose_guidance.pdf.

²⁶ U.S. EPA, *Scope of the Risk Evaluation for 1,4-Dioxane* at p. 21 (June 2017), <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0723-0049>.

anionic surfactants) and other ethoxylated substances, such as alkyl, alkylphenol and fatty amine ethoxylates; polyethylene glycols and their esters; and sorbitan ester ethoxylates. Therefore, 1,4-dioxane may be present at residual concentrations in commercial and consumer products that contain ethoxylated chemicals. Examples of products potentially containing 1,4-dioxane as a residual contaminant are paints, coatings, lacquers, ethylene glycol-based antifreeze coolants, spray polyurethane foam, household detergents, cosmetics/toiletries, textile dyes, pharmaceuticals, foods, agricultural and veterinary products.

EPA's approach means not only that this chemical's risks will not be evaluated for many years, but that a full picture of its risks from all exposure sources will still be lacking if its evaluation is broken into dozens or hundreds of small pieces -- clearly not what Congress intended.

i. EPA must not agree to recent industry requests on byproducts without certain conditions.

EDF is aware of a recent comment submitted to EPA by the American Cleaning Institute and the Grocery Manufacturers Association²⁷ calling on EPA to include 1,4-dioxane's presence as a byproduct as a condition of use in its risk evaluation. This request was made despite the fact that these same interest groups had urged EPA to exclude 1,4-dioxane as a byproduct in earlier rounds of comments on documents leading to this draft risk evaluation.²⁸

If EPA decides to grant this industry request, which has arrived long after EPA initiated the risk evaluation process, EPA should do so only subject to the following five conditions:

- EPA needs to use its information authorities to require the submission and development of relevant information on the presence of 1,4-dioxane as a byproduct in industrial, commercial and consumer materials and products, as input into a revised draft risk evaluation.
- EPA must promptly make all such information it receives public, subject only to redactions of information claimed confidential by the submitters that EPA determines

²⁷ See <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0238-0030>.

²⁸ See, e.g., ACI Comment on 1,4-dioxane TSCA designation as one of the first 10 chemicals to undergo risk evaluation under TSCA, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0723-0005>; P&G Comment on 1,4-dioxane TSCA designation, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0723-0008>; and Household & Commercial Products Association on 1,4-dioxane problem formulation, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2018-0210-0101>; see also ACC Comment on Initial TSCA designations, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0723-0022> (requesting byproduct exclusions generally); Consumer Specialty Products Association Comment on Initial TSCA chemical designations, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0723-0013> (requesting trace levels exclusion generally).

meet all applicable requirements of TSCA section 14. It should be noted that much of the relevant information will constitute health and safety information that is not eligible for protection under section 14 and must be made public.

- EPA needs to carefully and thoroughly develop and fully integrate an analysis of the potential exposures and risks arising from the presence of 1,4-dioxane as a byproduct into all aspects of its risk evaluation, given that inclusion of the presence of 1,4-dioxane as a byproduct will affect all of the exposure and risk estimates EPA has examined in the current draft risk evaluation.
- EPA must publish a revised draft risk evaluation for public comment, providing the public with ample time to review the new draft and develop meaningful comments.
- EPA must subject its revised draft risk evaluation to full peer review by the Scientific Advisory Committee on Chemicals (SACC), providing the committee with ample time to review the new draft and develop meaningful comments.

ii. EPA should consider the presentations from two meetings by California Department of Toxic Substances Control (DTSC) regarding 1,4-dioxane in personal care and cleaning products.

By failing to consider 1,4-dioxane's presence as a byproduct in the draft risk evaluation, EPA has also failed to consider reasonably available information about exposures to 1,4-dioxane. Presentations at two recent meetings held by California DTSC summarized some of the relevant information regarding exposures to 1,4-dioxane as a result of its presence as a byproduct in personal care and cleaning products.²⁹ The presentations from June 28, 2019, dealt with the potential adverse impacts from the presence of 1,4-dioxane in consumer products, and the presentations on August 21, 2019, addressed issues with lowering the amount of 1,4-dioxane in consumer products. Although many of the presentations are relevant, we summarize below just a few of the points that make clear the impacts of exposure via byproducts to the general population and potentially exposed or susceptible subpopulations. EPA has chosen to entirely ignore:

- **Ground water contamination:** “Nearly all households using domestic wells are also on septic tanks, which do not effectively remove 1,4-dioxane. There have been numerous instances of 1,4-dioxane contamination of domestic wells emanating from septic tank effluent.”³⁰ The presenter noted that in California alone 1.2 million people may be exposed through this route. According to the U.S. Geological Survey (USGS), “[s]eptic tanks serve primarily as settling chambers removing solids from the sewage ***. In sand

²⁹ All of the slideshows are available here: <https://dtsc.ca.gov/scp/1-4-dioxane/>.

³⁰ Thomas Mohr, *1,4-Dioxane Removal from Personal Care Products – Opportunities, Benefits, and Costs* slide 3 (Aug. 2019), https://dtsc.ca.gov/wp-content/uploads/sites/31/2019/08/Tom-Mohr_14DX_Removal_PCPs_Opportunities.pdf.

and gravel aquifers characterized by large pore sizes that allow for relatively easy and rapid transport of water and contaminants, concentrated plumes of dissolved constituents from septic systems can occur in the shallow part of the aquifer and can affect the quality of drinking water withdrawn from domestic wells.”³¹

- **Surface water and/or ground water contamination:** Non-consumer surfactant uses, such as car washes and industrial laundries, may be a significant source of 1,4-dioxane.³² In particular one presenter cited a report from New Hampshire that found 1,4-dioxane in car wash soap concentrate at 760 mg/kg.³³ Other presenters also indicated that industrial and commercial surfactants are more concentrated, potentially increasing exposures to the environment if the surfactants are released.³⁴
- **Surface water contamination:** “In densely populated areas [waste water treatment plant] discharges to surface water often exceed 20%.”³⁵ Many of the utilities in California noted that 1,4-dioxane was present in their facilities and was not removed with treatment.³⁶

B. Exclusions based on other statutes

Referencing its earlier problem formulation, EPA has excluded from its risk evaluation all general population exposures to 1,4-dioxane, based on EPA’s assertion -- unsupported by any actual data or analysis -- that “the existing regulatory programs and associated analytical processes [under the other statutes EPA administers] have addressed or are in the process of addressing potential risks of 1,4-dioxane that may be present in various media pathways (e.g., air, water, land) for the general population” (p. 28; see also pp. 40, 156).

³¹ Ingrid M. Verstraeten, et al., *Is Septic Waste Affecting Drinking Water From Shallow Domestic Wells Along the Platte River in Eastern Nebraska?* (2004), <https://pubs.usgs.gov/fs/fs07203/>.

³² Thomas Mohr, *1,4-Dioxane Removal from Personal Care Products – Opportunities, Benefits, and Costs* slide 7 (Aug. 2019), https://dtsc.ca.gov/wp-content/uploads/sites/31/2019/08/Tom-Mohr_14DX_Removal_PCPs_Opportunities.pdf.

³³ *Id.*

³⁴ See also NH Environmental Fact Sheet: 1,4-Dioxane and Drinking Water p. 2 (2019), <https://www.des.nh.gov/organization/commissioner/pip/factsheets/dwgb/documents/dwgb-3-24.pdf>; Ann Ropeik, *State Data May Tie North Hampton Car Wash to Contaminants in Public Well* (June 2018), <https://www.nhpr.org/post/state-data-may-tie-north-hampton-car-wash-contaminants-public-well#stream/0>.

³⁵ Thorsten Reemtsa, *Persistent and Mobile Chemicals in the Water Cycle* (June 2019), <https://dtsc.ca.gov/wp-content/uploads/sites/31/2019/07/Reemtsma-Presentation.pdf>.

³⁶ Ann Heil, *1,4-dioxane at LACSD* (June 2019), <https://dtsc.ca.gov/wp-content/uploads/sites/31/2019/07/Heil-Presentation.pdf>; Mark Kawamoto, *OCSD and 1,4-Dioxane* (June 2019), <https://dtsc.ca.gov/wp-content/uploads/sites/31/2019/07/Kawamoto-Presentation.pdf>.

Aside from the absent legal basis, these exclusions present significant health concerns. For example, in the problem formulation for 1,4-dioxane (pp. 43-44), EPA explicitly relies on the Clean Water Act (CWA) and the Safe Drinking Water Act (SDWA) to dismiss the need to assess exposures to 1,4-dioxane in water. Yet under the CWA EPA has not to date recommended a human health water quality criterion for 1,4-dioxane, and its process and timeline for doing so are highly uncertain. In the absence of a recommended criterion from EPA, only a single state (CO) has adopted a health-based water quality criterion for 1,4-dioxane. EPA's reliance on the SDWA is also unwarranted because 1,4-dioxane has only been listed on the Contaminant Candidate List (CCL). The CCL is a list of *unregulated* contaminants that are known or anticipated to occur in public water systems and that EPA indicates *may* need regulation.³⁷ In order to establish an enforceable limit, EPA would have to undertake a number of procedures that have not been undertaken for 1,4-dioxane.³⁸

The exposures EPA is ignoring are far from trivial. Based on the most recent data from EPA's Toxics Release Inventory (TRI) and National Emissions Inventory (NEI), despite any regulations under other laws facilities release nearly 640,000 pounds annually of 1,4-dioxane to air, water and land. EPA's approach effectively reduces this quantity to zero.

Moreover, 1,4-dioxane is widely detected in public water systems (PWS). In the Third Unregulated Contaminant Monitoring Rule (UCMR3),³⁹ 1,4-dioxane was found in 21% of the 4864 PWS, and was in exceedance of EPA's own health-based reference value of 0.35 ug/L (corresponding to a cancer risk level of one in one million) in 341 (6.9%) of the PWS sampled. Not only are elevated levels in drinking water of concern; so is exposure by inhalation when people use water at elevated temperatures (e.g., while cooking, bathing or showering).⁴⁰

³⁷ 42 U.S.C. § 300g-1(b)(1)(B)(i); *see also* BASIC INFORMATION ON THE CCL AND REGULATORY DETERMINATION, <https://www.epa.gov/ccl/basic-information-ccl-and-regulatory-determination> (last visited Jul. 19, 2019).

³⁸ 42 U.S.C. § 300g-1(b)(1)(B)(ii), (E); *see also* BASIC INFORMATION ON THE CCL AND REGULATORY DETERMINATION, <https://www.epa.gov/ccl/basic-information-ccl-and-regulatory-determination> (last visited Jul. 19, 2019). EPA has yet to make a regulatory determination on whether even to initiate a rulemaking, let alone initiate the rulemaking and propose and finalize a rule.

³⁹ *See* U.S. EPA, *The Third Unregulated Contaminant Monitoring Rule (UCMR 3): Data Summary* (June 2017), <https://www.epa.gov/sites/production/files/2017-02/documents/ucmr3-data-summary-january-2017.pdf>.

⁴⁰ EPA cited this exposure in its problem formulation (p. 31), but made no mention of it in the draft risk evaluation.

C. Collapse of varied uses into a single category/single scenario

EPA lumps together a highly diverse set of uses as “industrial uses” (p. 58). They encompass a huge array of sectors, from textiles to agricultural chemicals to pharmaceuticals, and very different functional uses, from solvent to catalyst to intermediate to wetting agent. EPA asserts without providing any support that all such operations “are expected” to be similar.

Beyond this, EPA has used single scenarios to represent each of the following activities despite their varied nature: all processing scenarios other than repackaging (p. 163); all intermediate use scenarios (p. 165); all open system functional fluid use scenarios (p. 166); all laboratory chemicals use scenarios (p. 168); and all disposal scenarios (p. 175). EPA has provided no data or analysis to demonstrate that these scenarios are representative of other scenarios within a grouping or otherwise ensure a health-protective approach.

By using a single scenario for varied activities, EPA may significantly understate the risk presented for two reasons. First, EPA may not have selected the highest risk scenario to represent the activities. EPA has presented no evidence or analysis establishing that it selected the highest exposure/highest risk circumstances for each of these scenarios. Second, EPA has not accounted for the possibility that the same worker or occupational non-user might be subject to more than one activity within a given scenario or more than one exposure scenario, and hence experience a combined exposure that carries more risk than EPA assumed. As a result, EPA may significantly underestimate the risk. By failing to adequately account for the risk, EPA overlooks an important aspect of the problem and engages in an arbitrary and capricious analysis.

D. Failure to consider 1,4-dioxane’s use and disposal in hydraulic fracturing fluids and produced wastewater as conditions of use

- i. Use and disposal of 1,4-dioxane in hydraulic fracturing fluids and produced wastewater is known and reasonably foreseen.*

Under TSCA, EPA must conduct risk evaluations to determine whether “a chemical substance presents an unreasonable risk *** under the conditions of use.” 15 U.S.C. § 2605(b)(4)(A). “Conditions of use” are defined as “the circumstances, as determined by the Administrator, under which a chemical substance is intended, known, or reasonably foreseen to be manufactured, processed, distributed in commerce, used, or disposed of.” 15 U.S.C. § 2602(4). As explained in greater detail in Part II sec. 1 of these comments, TSCA was intended to cover *all* conditions of use of a chemical substance; EPA cannot pick and choose which conditions of use to include or ignore. Congress consistently used the phrase “a chemical substance” to describe the object risk evaluations. 15 U.S.C. § 2605(b)(1)-(4), (i). This language requires EPA to consider *all* hazards and exposures that contribute to the total risk presented by the chemical substance as a whole.

Yet EPA has failed to identify as conditions of use, or even acknowledge, the known and reasonably foreseen use and disposal of 1,4-dioxane in hydraulic fracturing fluids and produced wastewater.

In EPA's own report on the impacts of hydraulic fracturing on drinking water sources in the U.S., EPA identified 1,4-dioxane as an ingredient in hydraulic fracturing fluids and as detectable in wastewater (also referred to as "produced water")⁴¹ from hydraulic fracturing operations.⁴² EPA's analysis, which examined data from 2013 to 2015, cited three references that identified 1,4-dioxane as an ingredient in hydraulic fracturing fluids.⁴³ Therefore, based on EPA's own analysis, use and disposal of 1,4-dioxane in hydraulic fracturing fluids and produced wastewater are known and reasonably foreseen conditions of use of 1,4-dioxane. EPA's failure to identify and evaluate these conditions of use is arbitrary and capricious, as discussed at greater length in Part II sec. 1.

Additionally, according to FracFocus,⁴⁴ 1,4-dioxane has been reported more than 500 times as present in hydraulic fracturing fluids between January 1, 2016 and April 30, 2019.⁴⁵ These reports show that, at a minimum, the chemical is being used in 11 states:⁴⁶

⁴¹ EPA has defined "produced water" as "the fluid brought up from the hydrocarbon-bearing strata during the extraction of oil and gas, and includes, where present, formation water, injection water, and any chemicals added downhole or during the oil/water separation process." 40 C.F.R. § 435.33(v).

⁴² U.S. EPA, *Hydraulic Fracturing for Oil and Gas: Impacts from the Hydraulic Fracturing Water Cycle on Drinking Water Resources in the United States (Final Report)* at app. H p. H-9 (2016), http://ofmpub.epa.gov/eims/eimscomm.getfile?p_download_id=530160.

⁴³ *Id.* (citing references "2, 3, 4": Colborn, T., et al., *Natural gas operations from a public health perspective*, 17 HUMAN ECOL. RISK ASSESSMENT 1039-1056 (2011), <http://dx.doi.org/10.1080/10807039.2011.605662>; N.Y. State Dep't of Env'tl. Conservation, *Revised draft supplemental generic environmental impact statement (SGEIS) on the oil, gas and solution mining regulatory program* at p. 5-55, 5-78 (2011), <http://www.dec.ny.gov/data/dmn/rdsgeisfull0911.pdf>; U.S. EPA, *Data received from oil and gas exploration and production companies* (2013), <https://www.regulations.gov/docket?D=EPA-HQ-ORD-2010-0674>).

⁴⁴ FracFocus provides the public access to information on chemicals reported by companies as being used for hydraulic fracturing, see <https://fracfocus.org/welcome>.

⁴⁵ See FRACFOCUS CHEMICAL DISCLOSURE REGISTRY, <https://fracfocus.org/data-download> (last visited Aug. 7, 2019); 1,4-Dioxane Reports from FracFocus, attached as Appendix II.

⁴⁶ *Id.*

State	No. of Reports (1/1/2016 – 4/30/2019)
Colorado	28
Montana	4
New Mexico	30
North Dakota	69
Ohio	5
Oklahoma	54
Pennsylvania	19
Texas	264
Utah	14
Wyoming	50

Moreover, according to the reports submitted to FracFocus, 1,4-dioxane is not just present as an impurity or by-product.⁴⁷ Companies reported over 400 instances where 1,4-dioxane was used as an ingredient, representing 77% of the reported cases in this time period.⁴⁸

Numerous publications have also reported that 1,4-dioxane is present in produced wastewater.⁴⁹ Each of these publications reports that 1,4-dioxane was present at detectable concentrations, even

⁴⁷ EDF objects to EPA’s exclusion of 1,4-dioxane’s presence as a byproduct from the risk evaluation, as explained at Part I sec. 2.A. & Part II sec. I.A.v. To the extent EPA chooses to illegally ignore 1,4-dioxane’s presence as a byproduct, that provides no basis for EPA not to include 1,4-dioxane’s presence as an intentionally added substance.

⁴⁸ See 1,4-Dioxane Reports from FracFocus, attached as Appendix II (number calculated by filtering the “Ingredient Name” column, which identified the chemical as an “impurity” for certain reports); see also Hydraulic Fracturing Fluid Product Component Information Disclosures attached as Appendix III (downloaded from FracFocus, one shows 1,4-dioxane as an ingredient and the other indicates it is present as an impurity).

⁴⁹ See, e.g., Yaal Lester, et al., *Characterization of hydraulic fracturing flowback water in Colorado: Implications for water treatment*, 512 SCIENCE OF THE TOTAL ENV’T 637–644 (Apr. 2015), <https://doi.org/10.1016/j.scitotenv.2015.01.043>; Jhih-Shyang Shih, et al., *Characterization and Analysis of Liquid Waste from Marcellus Shale Gas Development*, 49 ENVTL. SCIENCE & TECHNOLOGY 9557-9565 (Jul. 2015), in *Supporting information*, available at <https://doi.org/10.1021/acs.est.5b01780>; Tom Hayes & Blaine Severin, *Barnett and Appalachian Shale Water Management and Reuse Technologies* (2012), https://edx.netl.doe.gov/dataset/barnett-and-appalachian-shale-water-management-and-reuse-technologies/resource_download/d167805d-9a16-40b8-b3fb-123ac3edab20; Tom Hayes, *Sampling and Analysis of Water Streams Associated with the Development of Marcellus Shale Gas* (2009), <https://www.water-research.net/naturalgasPA/pdf/MSCommission-Report.pdf>.

though quantification of constituents of concern in oil and gas wastewater is notoriously difficult.⁵⁰

It is important to note that in some of these cases where 1,4-dioxane has been detected in the produced wastewater (by state) it has not been reported to FracFocus. This suggests that EPA cannot base its consideration of 1,4-dioxane's presence in hydraulic fracturing fluids and produced wastewater solely on the information submitted to FracFocus, which is subject to trade secret laws.⁵¹

ii. EPA has failed to consider reasonably available information.

TSCA requires EPA to consider all “reasonably available” information relating to a chemical substance when conducting a risk evaluation. 15 U.S.C. § 2625(k). EPA’s rules define “reasonably available information” as “information that EPA possesses or can reasonably generate, obtain and synthesize for use ***.” 40 C.F.R. § 702.3, 702.33. EPA has disregarded numerous sources that are directly relevant to 1,4-dioxane’s use and disposal in hydraulic fracturing fluids and produced wastewater, and that are reasonably available. These include:

- EPA’s 2016 report on the effects of hydraulic fracturing on drinking water;⁵²
- FracFocus reports;⁵³
- Scholarly articles available on ingredients in produced water;
- Data available from state governments that do not mandate submission to FracFocus but still mandate some ingredient disclosure;⁵⁴ and

⁵⁰ Karl Oetjen, et al., *Emerging analytical methods for the characterization and quantification of organic contaminants in flowback and produced water*, 15 TRENDS IN ENVTL. ANALYTICAL CHEMISTRY 12-23 (2017),

<https://www.sciencedirect.com/science/article/pii/S2214158817300314>.

⁵¹ Cloelle Danforth, et al., *Alternative Management of Oil and Gas Produced Water Requires More Research on its Hazards and Risks*, INTEGRATED ENVTL. ASSESSMENT & MGMT. (2019), <https://setac.onlinelibrary.wiley.com/doi/abs/10.1002/ieam.4160>.

⁵² U.S. EPA, *Hydraulic Fracturing for Oil and Gas: Impacts from the Hydraulic Fracturing Water Cycle on Drinking Water Resources in the United States* (2016), http://ofmpub.epa.gov/eims/eimscomm.getfile?p_download_id=530159.

⁵³ FRACFOCUS CHEMICAL DISCLOSURE REGISTRY, <https://fracfocus.org/data-download> (last visited Aug. 7, 2019). At least 26 states, and the Bureau of Land Management, use FracFocus. See FRACFOCUS REPORTING STATES, http://fracfocus.org/sites/default/files/fracfocus_reporting_states_2-7-18-01_1.png (last visited Aug. 8, 2019). However, this data set is not necessarily complete and EPA should not treat it as such. Many states that are considered FracFocus “partners” do not *mandate* the use of FracFocus for chemical disclosure.

⁵⁴ There are a number of states that produce oil and natural gas that do not require reporting to FracFocus. See CRUDE OIL PRODUCTION,

- Information that is in the possession of companies that use 1,4-dioxane in their hydraulic fracturing operations but are not otherwise required to report its use, including information that companies would likely claim as confidential business information. This information is reasonably available because EPA could obtain this information utilizing its information authorities under TSCA.

These sources of information are all reasonably available. EPA’s failure to consider this reasonably available information is arbitrary and capricious.

iii. EPA cannot rely on its other statutory authorities to ignore these conditions of use.

Although EPA has not yet addressed these conditions of use, and has not yet provided any rationale for ignoring them, EDF preemptively cautions EPA that it cannot rely on other statutes having “adequately addressed” these conditions of use in order to ignore them.⁵⁵ Oil and gas operations have notoriously received broad exemptions under numerous environmental statutes; the following are just a few examples of the types of exemptions:

- Safe Drinking Water Act: EPA “may not prescribe requirements [that] interfere with or impede – (A) the underground injection of brine or other fluids which are brought to the surface in connection with oil or natural gas production, or 2) any underground injection for secondary or tertiary recovery of oil or natural gas, unless requirements are *essential* to assure that underground sources of drinking water will not be endangered by such injection.” 42 U.S.C. § 300h(b)(2) (emphasis added).
- Clean Water Act: National Pollution Discharge Elimination System (NPDES) permits cannot be required for stormwater discharges from “oil and gas exploration, production, processing or treatment operations, or transmission facilities ***.” 33 U.S.C. § 1342(l)(2). “Oil and gas exploration, production, processing or treatment operations, or transmission facilities” is defined to include “*all* field activities or operations associated with exploration, production, processing, or treatment operations, or transmission facilities, including activities necessary to prepare a site for drilling and for the movement

https://www.eia.gov/dnav/pet/pet_crd_crpdn_adc_mdbl_a.htm (last visited Aug. 8, 2019); NATURAL GAS ANNUAL SUPPLY & DISPOSITION BY STATE, https://www.eia.gov/dnav/ng/ng_sum_snd_a_EPG0_FPD_Mmcf_a.htm (last visited Aug. 8, 2019). Despite not requiring submission of data to FracFocus, some states nevertheless mandate some level of reporting. *See, e.g.*, Reporting of hydraulic fracturing information, 312 Ind. Admin. Code 29-22-8 (requiring disclosure of the additive trade name, type/purpose, MSDS, additive max volume % mass and % volume of total); Ill. Admin. Code tit. 62, § 245.700-.730 (requiring disclosure of chemicals intentionally added).

⁵⁵ EDF objects to EPA’s exclusion of exposure pathways for 1,4-dioxane based on EPA’s authority to regulate it under other statutes. For the reasons articulated in Part I sec. 2.b. and Part II sec. 5, such exclusions violate the law. As explained here, such exclusions would be inappropriate for hydraulic fracturing fluids and produced wastewater for additional reasons.

and placement of drilling equipment, whether or not such field activities or operations may be considered to be construction activity.” 33 U.S.C. § 1362(24) (emphasis added).

- Resource Conservation and Recovery Act: Generally, “drilling fluids, produced waters, and other wastes associated with the exploration, development, or production of crude oil or natural gas” are exempt from being listed as hazardous wastes under Subtitle C. *See* 42 U.S.C. § 6921(b)(2)(A).

Based on these exemptions and others, EPA would have absolutely no basis to assume that 1,4-dioxane in hydraulic fracturing fluids or produced wastewater is “adequately addressed” by any statute.

Moreover, such conspicuous regulatory gaps are precisely one of the reasons TSCA was adopted in the first place. When TSCA was originally signed into the law, President Gerald Ford noted that:

The bill *closes a gap* in our current array of laws to protect the health of our people and the environment. The Clean Air Act and the Water Pollution Control Act protect the air and water from toxic contaminants. The Food and Drug Act and the Safe Drinking Water Act are used to protect the food we eat and the water we drink against hazardous contaminants. Other provisions of existing laws protect the health and the environment against other polluting contaminants such as pesticides and radiation. However, *none of the existing statutes provide comprehensive protection*. This bill provides broad discretionary authority to protect the health and environment.⁵⁶

In sum, in addition to the many reasons why EPA cannot rely on its other statutory authorities to ignore environmental releases, EPA especially cannot do so in the case of hydraulic fracturing fluids and produced water because the oil and gas industry has received numerous exemptions under those other statutes. Therefore, TSCA’s regulatory authorities, which have no such exemptions, must be used to address those gaps by first assessing the risks posed by 1,4-dioxane through these conditions of use.

iv. EPA must address these conditions of use as they pertain to exposures of the environment, the general population and workers.

1,4-dioxane’s use and disposal in hydraulic fracturing fluids and produced wastewater has the potential to impact workers, tribal communities, the general population, and the environment. EPA’s own report on the impacts of hydraulic fracturing on drinking water states that:

⁵⁶ Statement of the President on Signing S. 3149 Into Law (Oct. 12, 1976), <https://ia801702.us.archive.org/18/items/leehisto00unit/leehisto00unit.pdf> (emphases added).

some chemicals in the hydraulic fracturing water cycle are of more concern than others because they are more likely to move with water (e.g., spilled hydraulic fracturing fluid) to drinking water resources, persist in the environment (e.g., chemicals that do not degrade), and/or affect human health.⁵⁷

These specific concerns underscore the urgency of evaluating 1,4-dioxane's use in hydraulic fracturing fluids; the physical/chemical properties EPA relies on throughout the draft risk evaluation indicate that 1,4-dioxane will persist primarily in the aqueous phase, and therefore will likely be associated with wastewater.

1,4-dioxane ranked as of high concern in the analysis EPA conducted for its 2016 report. Of the 1084 chemicals EPA identified as used in hydraulic fracturing, and of the 599 chemicals identified in produced water, 98 (9%) and 120 (20%) had either chronic oral reference values (RfVs) or oral slope factors (OSF) for cancer, respectively.⁵⁸ From this subset of chemicals, EPA conducted a multi-criteria decision analysis (MCDA) to assign chemicals in hydraulic fracturing fluids and produced water with scores based on toxicity, occurrence, and physicochemical properties. As described in the report, the MCDA scores “provide a preliminary evaluation of hazard potential, and serve as a qualitative metric for making comparison between chemicals when exposure assessment data is limited or unavailable.”⁵⁹ Because of 1,4-dioxane's presence in both hydraulic fracturing fluids and produced water, EPA included it in its ranking of chemicals of concern for Cancer based on the MCDA scores (see: Table A⁶⁰ and Table 2⁶¹).

In light of this preliminary analysis of 1,4-dioxane, it is clear that 1,4-dioxane's presence in hydraulic fracturing poses potential risks.

EPA must also take into consideration the potentially exposed or susceptible subpopulations that are exposed to 1,4-dioxane through these conditions of use. EDF urges EPA to consider the

⁵⁷ U.S. EPA, *Hydraulic Fracturing for Oil and Gas: Impacts from the Hydraulic Fracturing Water Cycle on Drinking Water Resources in the United States, Executive Summary* at p. 38 (2016), http://ofmpub.epa.gov/eims/eimscomm.getfile?p_download_id=530285.

⁵⁸ U.S. EPA, *Hydraulic Fracturing for Oil and Gas: Impacts from the Hydraulic Fracturing Water Cycle on Drinking Water Resources in the United States* at p. 9-79 (2016), http://ofmpub.epa.gov/eims/eimscomm.getfile?p_download_id=530159.

⁵⁹ *Id.* at 9-52.

⁶⁰ U.S. EPA, *Hydraulic Fracturing for Oil and Gas: Impacts from the Hydraulic Fracturing Water Cycle on Drinking Water Resources in the United States* at p. 9-71 (2016), http://ofmpub.epa.gov/eims/eimscomm.getfile?p_download_id=530159.

⁶¹ *Id.* at 9-77.

exposures workers face, both to the hydraulic fracturing fluids and the produced wastewater.⁶² EPA should also consider the disproportionate exposures to hydraulic fracturing fluids and produced wastewater of tribal communities.⁶³

3. EPA must adopt a linear, no-threshold approach for 1,4-dioxane’s carcinogenicity.

Where there is evidence that a chemical or other agent may act through a genotoxic or mutagenic mode of action (MOA), a non-threshold, linear extrapolation approach for cancer dose-response modeling is employed because the MOA “may involve a single direct reaction, specifically, a single hit in a single target (Kirsch-Volders et al., 2000). Thus there would always be some risk, even at very low doses.”⁶⁴

A. A mutagenic MOA for 1,4-dioxane remains plausible, despite EPA’s attempts to downplay it.

i. General support for mutagenic/genotoxic MOA

Tables H.1.5 and H.1.7 list studies that report evidence for a wide-range of mutagenic/genotoxic events following 1,4-dioxane treatment, including but not limited to: meiotic nondisjunction (Munoz and Barnett (2002)); micronucleus formation (i.e. clastogenic activity) (Mirkova (1994); Morita and Hayashi (1998); Roy et al. (2005); Itoh and Hattori (2019)); point mutations (Gi et al (2018)); single-strand breaks (Sina et al. (1983); Kitchin and Brown (1990)); and replicative DNA synthesis (Miyagawa et al. (1999)).

While there are some inconsistencies among the available studies, the body of evidence supports what several SAAC panel members emphasized during their July 2019 public meeting: that it is inappropriate for the Agency to dismiss the potential for a genotoxic MOA. SAAC members also urged EPA to adopt a broad definition of mutagenicity that incorporates these diverse effects, which are also strongly linked to cancer, rather than a more narrow focus on point mutations only.

Additional relevant evidence for genotoxicity highlighted during the SAAC peer review meeting relates to the nasal tumors seen in numerous studies. One member noted that observed incidence

⁶² See, e.g., Bevin Durant, et al., *Assessing dermal exposure risk to workers from flowback water during shale gas hydraulic fracturing activity*, 34 J. OF NATURAL GAS SCIENCE & ENG’G 969–978 (Aug. 2016),

<https://www.sciencedirect.com/science/article/abs/pii/S1875510016305236?via%3Dihub>.

⁶³ See, e.g., Jill E. Johnston, et al., *Wastewater Disposal Wells, Fracking, and Environmental Injustice in Southern Texas*, 106 AM. J. OF PUBLIC HEALTH 550-56 (Feb. 2016),

<https://ajph.aphapublications.org/doi/pdf/10.2105/AJPH.2015.303000>.

⁶⁴ Zoë Gillespi, et al., *Risk assessment approaches for carcinogenic food contaminants*, 1:1 INT’L FOOD RISK ANALYSIS J. 1-18 (2011), <https://hrcak.srce.hr/file/107066>.

of rare nasal tumor types following 1,4-dioxane exposure is likely the result of a genotoxic or mutagenic MOA. He noted that all other nasal carcinogens have such a MOA and that the types of rare tumors linked to this chemical would be unlikely to be attributed to a cytotoxic MOA. In addition, he argued that evidence for a genotoxic or mutagenic MOA in one organ (i.e., the olfactory system) should create a strong presumption that the same MOA is operating in other organs (i.e., the liver), absent compelling counter-evidence.

ii. Potential explanations for discordance between in vivo & in vitro results

Some challenges to a genotoxicity/mutagenicity MOA for 1,4-dioxane have pointed to the discordance between the results of *in vitro* and *in vivo* studies. Briefly, most (but not all) *in vitro* studies are negative with respect to genotoxicity/mutagenicity, while a number of *in vivo* studies are positive and cancer bioassays provide evidence of tumor formation. This discrepancy is not surprising, given the substantial differences between reductionist *in vitro* test systems and whole animal *in vivo* models. One highly plausible explanation for the discordance between these results is the role of metabolism, which fully functions only in the *in vivo* models.

More broadly, there are numerous examples of chemicals for which standard *in vitro* genotoxic assessments have failed to accurately classify human and/or animal genotoxicity. Brambilla and Martelli (2004)⁶⁵ review several well-known chemicals – including polychlorinated biphenyls (PCBs) and trichloroethylene (TCE) – for which the standard *in vitro* genotoxic battery provided false-negative results. They suggest several reasons for false-negative results in *in vitro* systems, including the lack of or limited metabolic capacity and interspecies differences. Overall, it is quite possible, as was postulated for o-toluidine,⁶⁶ that 1,4-dioxane is a “compound with a wide range of effects on genetic material, but with a mode of action requiring conditions which are frequently not met in standard short-term tests.”

iii. EPA distortion of a high-quality independent scientific conclusion

Despite these data in support of genotoxic mechanisms, the Agency seems to intentionally distort independent scientific conclusions in favor of casting doubt on a genotoxic MOA. For example, based on the results of their *in vivo* gene mutation assay, Gi et al. (2018)⁶⁷ conclude that:

⁶⁵ Giovanni Brambilla & Antonietta Martelli, *Failure of the standard battery of short-term tests in detecting some rodent and human genotoxic carcinogens*, 196 TOXICOLOGY 1-19 (2004), <https://www.ncbi.nlm.nih.gov/pubmed/15036752>.

⁶⁶ Natalie Danford, *The genetic toxicology of ortho-toluidine*, 258:3 MUTATION RESEARCH/REVIEWS IN GENETIC TOXICOLOGY 207-236 (Nov. 1991), <https://www.sciencedirect.com/science/article/pii/016511109190010S?via%3Dihub>.

⁶⁷ Min Gi, et al., *In vivo positive mutagenicity of 1,4-dioxane and quantitative analysis of its mutagenicity and carcinogenicity in rats*, 92:10 ARCHIVES OF TOXICOLOGY 3207-221 (Oct. 2018), <https://link.springer.com/article/10.1007/s00204-018-2282-0>.

- “In the present study, neither significant changes in 8-OHdG levels nor necrosis were observed in rats treated with mutagenic or/and carcinogenic doses of 1,4-dioxane. This suggests that oxidative DNA damage and necrosis followed by regeneration do not appear to play a role in 1,4-dioxane-induced mutagenesis and carcinogenesis.”
- “Overall, there is no evidence for alternative non-genotoxic mechanisms that could contribute to the carcinogenic effects of 1,4-dioxane. Lack of non-genotoxic mechanisms provides indirect evidence supporting a mutagenic MOA of 1,4-dioxane.”
- “1,4-dioxane is a genotoxic hepatocarcinogen and induces hepatocarcinogenesis through a mutagenic MOA.”

Gi et al. 2018 is a very recent study that EPA ranks as high-quality and that SACC members characterized as conducted by one of the premier laboratories in the field. However, in its draft risk evaluation, EPA has mysteriously concluded that “the weight of scientific evidence supports that 1,4-dioxane is not mutagenic” (p. 96). This conclusion is in direct contradiction to the authors’ own conclusions and is based on a “weight of evidence” approach that EPA apparently utilized but has failed to appropriately explain. While EPA discounted the results of this study based on treatment duration (p. 96), a SACC member questioned the validity of EPA’s argument.

B. There are serious flaws in the arguments supporting a cytotoxicity MOA.

In certain sections of the draft risk evaluation, EPA recognizes what numerous state health agencies (see subsection iii below) as well as the agency itself have concluded: that “evidence is not sufficient to support a MOA of cytotoxicity followed by sustained cell proliferation as a required precursor to tumor formation related to the metabolic saturation and accumulation of the parent compound, 1,4-dioxane” (p. 101).

i. Tumor formation in the absence of cytotoxicity

Significant evidence indicates that 1,4-dioxane exposure leads to tumor formation *in the absence of cytotoxicity*. EPA has already summarized relevant data and drawn similar conclusions in this risk evaluation:

- “Liver tumors identified from rodent liver bioassays occurred in the absence of reported lesions related to cytotoxicity (Kano et al., 2008; JBRC, 1998; NCI, 1978), suggesting that cytotoxicity may not be a key event after 1,4-dioxane exposure leading to liver carcinogenesis.” (p.99)
- “The doses of 1,4-dioxane at which cytotoxicity and cell proliferation were observed were greater than the doses for tumor induction.” (p.99)

- “[H]epatocellular tumors were seen in multiple male or female experimental groups at doses without evidence of the reported toxic effects in the subchronic study.” (p.100)
- “Neither cellular swelling, necrosis, nor other hepatocellular pathological changes were reported in the 2-year mouse bioassay (M or F).” (p.100)
- “[AST and ALT] enzyme levels were not elevated in the low dose chronic bioassay groups where there was still an increase in hepatocellular tumors (statistically significant in females).” (p.100)
- “[T]he doses in hepatotoxicity studies where cytotoxicity and cell proliferation were observed were greater than cancer bioassay dose levels.” (p.101)

ii. *Tumor formation at low doses versus saturation*

Some commenters have suggested that 1,4-dioxane is linked to tumors only at high doses, which they argue would 1) indicate a central role for metabolic saturation followed by cytotoxicity rather than a genotoxic MOA, 2) reflect effects only seen at doses unrealistic for human exposures. There are several flaws with these arguments. First, 1,4-dioxane has, in fact, been linked to tumor formation at *low doses*. For example, as reported by Kano et al. (2009) but excluded from consideration by EPA (see Part I sec. 1.E.iv), female mice at the lowest dose level tested (66 mg/kg-day or 10 mg/kg-day [HED]) exhibited increased combined liver adenomas and carcinomas.⁶⁸

With regard to a potential threshold based on enzyme saturation, it is well documented that enzymatic metabolic activity varies across the population.⁶⁹ Therefore, it is inappropriate to assume that a possible threshold found in limited *in vivo* studies in laboratory animals or *in vitro* studies apply across the entire distribution of the human population. Even if there were a threshold seen in such studies based on metabolic saturation, EPA would need to consider variation in the human population and protect the most sensitive individuals, who may experience this purported “threshold” at lower doses.

⁶⁸ Hirokazu Kano, et al., *Carcinogenicity studies of 1,4-dioxane administered in drinking-water to rats and mice for 2 years*, 47:11 FOOD & CHEMICAL TOXICOLOGY 2776-84 (Nov. 2009), <https://www.sciencedirect.com/science/article/pii/S0278691509003895?via%3Dihub>; see also U.S. EPA, IRIS, *Toxicological Review of 1,4-Dioxane (With Inhalation Update)* (Sept. 2013), https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0326tr.pdf (considered by EPA in the IRIS review of 1,4-dioxane).

⁶⁹ Joseph McGraw & Donald Waller, *Cytochrome P450 variations in different ethnic populations*, 8:3 EXPERT OPINION ON DRUG METABOLISM & TOXICOLOGY 371-382 (2012), <https://www.ncbi.nlm.nih.gov/pubmed/22288606>; Sarah C. Preissner, et al., *Polymorphic cytochrome P450 enzymes (CYPs) and their role in personalized therapy*, 8:12 PLOS ONE (2013), <https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0082562>.

A related issue is that of combined exposures to multiple chemicals. The existing *in vivo* studies are conducted on 1,4-dioxane only. They do not consider the real-world scenario of exposures to multiple chemicals to which everyone is constantly exposed. Again, even if a threshold based on metabolic saturation was convincingly demonstrated in a controlled laboratory experiment, the threshold level would likely be overestimated, given that a human individual's metabolic system would likely become saturated at a lower level as a result of his/her system responding to numerous exogenous chemicals.

Finally, arguments attempting to discount experimental studies based on their use of high doses ignore the fact that toxicologists typically use high doses of chemicals in studies using laboratory animals, because a) we cannot for ethical reasons test chemicals directly on people, b) lab animals live far shorter lives than do humans, and c) it is too costly and impractical (and unethical) to use large enough numbers of lab animals in a study to model the human population. Such doses are used to ensure that a study will detect an effect, if one occurs, in a relatively short time and in a relatively small number of animals. While there are legitimate scientific debates as to whether such studies may actually obscure or miss effects that occur at low doses of exposure, arguments claiming high doses are “unrealistic” are at best overly simplistic and at worst do a disservice to the public discourse and reflect an underlying bias.

iii. Independent scientific reviews by state agencies have rejected the proposal of a threshold, non-mutagenic mode of action for 1,4-dioxane.

In section 4.2.4, EPA conducted a mode of action (MOA) analysis in which it compared evidence for a mutagenicity-based MOA – which is the MOA that EPA has assumed previously, including in its 2013 IRIS assessment⁷⁰ – versus a cytotoxicity-based MOA – espoused in industry-sponsored studies published by Dourson et al. (2014, 2017)⁷¹ under the auspices of Dourson's consulting firm Toxicology Excellence for Risk Assessment (TERA).

Dourson's proposed MOA has been presented to several state regulatory agencies, who have thoroughly rejected his contention that sufficient evidence exists to establish a non-mutagenic MOA for 1,4-dioxane. The table below shows that numerous states have recently adopted or

⁷⁰ U.S. EPA, IRIS, *Toxicological Review of 1,4-Dioxane (With Inhalation Update)* (Sept. 2013), https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0326tr.pdf.

⁷¹ Michael Dourson, et al., *Mode of action analysis for liver tumors from oral 1,4-dioxane exposures and evidence-based dose response assessment*, 68:3 REGULATORY TOXICOLOGY & PHARMACOLOGY 387-401 (2014), <http://www.sciencedirect.com/science/article/pii/S0273230014000129>; Michael Dourson, et al., *Update: Mode of action (MOA) for liver tumors induced by oral exposure to 1,4-dioxane*, 88 REGULATORY TOXICOLOGY & PHARMACOLOGY 45-55 (2017), <http://www.sciencedirect.com/science/article/pii/S0273230017300429>.

proposed standards or guidance values that are based on a mutagenic MOA and a linear no-threshold approach to dose-response modeling.

States with Water Quality Standards or Other Health Values Based on a Linear, No-threshold Approach					
State	Type of Value	Value	Year	Link to standard/guidance value	Link to rebuttal of threshold approach or support for linear, no-threshold approach
New Jersey	Groundwater Quality Criterion	0.4 ppb	2018 2015	https://bit.ly/2K70268	https://bit.ly/2K70268 https://www.state.nj.us/de/p/dsr/supportdocs/11-chemicals-response.pdf
Vermont	Groundwater Enforcement Standard	0.3 µg/L	2019	https://dec.vermont.gov/sites/dec/files/dwgwp/DW/2019.07.06%20-%20GWPRS.pdf	https://dec.vermont.gov/sites/dec/files/dwgwp/DW/Groundwater-Protection-Rule-and-Strategy-Responsiveness%20Summary.pdf
Wisconsin	Enforcement Standard Recommended Value	0.35 µg/L	2019	https://www.dhs.wisconsin.gov/publications/p02434v.pdf	Same document
Michigan	Residential Drinking Water Criterion	7.2 ppb	2017	https://www.michigan.gov/documents/deq/deq-rrd-2017-041EQFinalEnvironmentalContaminatioResponseActivityDioxane_604807_7.pdf	https://www.michigan.gov/documents/deq/deq-aqd-toxics-14-DioxaneTSG_Report_2015_487415_7.pdf
New York*	Recommended Maximum Contaminant Level	1 µg/L	2019	https://www.dos.ny.gov/info/register/2019/july24.pdf	https://players.brightcove.net/2886492229001/default_index.html?videoId=5981007496001

* The link in the rightmost column is to a webcast, the relevant portion of which starts at 1:18:45.

Scientists from at least two of these state agencies have provided extensive formal responses: Michigan’s Department of Environmental Quality in 2015;⁷² and New Jersey’s Department of

⁷² Mich. Dep’t of Env’tl. Quality, Toxics Steering Group, 1,4-Dioxane Subcomm., Review of a 1,4-Dioxane Presentation by Michael Dourson, Ph.D. on October 8, 2013 (Feb. 2015), https://www.michigan.gov/documents/deq/deq-aqd-toxics-14-DioxaneTSG_Report_2015_487415_7.pdf.

Environmental Protection in both 2015⁷³ and 2018.⁷⁴ MI DEQ's response and NJDEP's 2015 response specifically considered Dourson et al. 2014, while NJ DEP's 2017 response considered both Dourson et al. 2014 and Dourson et al. 2017. This section will present key excerpts from these critical technical reviews.⁷⁵

a. *Rebuttal of Dourson et al. 2014*

NJ DEP:

1. "Based on their interpretation of the dose-response for these non-neoplastic effects, Dourson et al. (2014) suggest that these events preceded and were causative to tumor formation. In male mice, a higher incidence and/or greater severity for all of these effects were observed in both the high and low dose group as compared to controls. *However, in female mice, the incidence and/or severity of glycogen depletion, necrosis, inflammation, and Kupffer cell hyperplasia was similar or greater in controls as compared to the low dose group, and was increased in the high dose group as compared to controls in the high dose group.*" [The appearance of these pathologies in the control as well as the low dose female mice group does not support the theory that the effects are linked to low dose exposures.]
2. "Dourson et al. (2014) suggest that 1,4-dioxane causes liver tumors in rats and mice through a pathway involving cytotoxicity (as indicated by hypertrophy and necrosis)

⁷³ N.J. Dep't of Env'tl. Protection, Response to Public Input on Draft Interim Ground Water Quality Criteria and Draft Interim Practical Quantitation Levels for Eleven Chemicals at pp. 11-17, <https://www.state.nj.us/dep/dsr/supportdocs/11-chemicals-response.pdf>.

⁷⁴ N.J. Dep't of Env'tl. Protection, Final Criteria: 50 N.J.R. 334(a), Response to Comment 39, (Jan. 16, 2018), <https://bit.ly/2K70268>.

⁷⁵ It should be noted that these rebuttals also apply directly to a recent proposal by Health Canada to adopt a threshold MOA for 1,4-dioxane (Health Canada, *1,4-dioxane in Drinking Water: Guideline Technical Document for Public Consultation* (2018), <https://www.canada.ca/content/dam/hc-sc/documents/programs/consultation-1-4-dioxane-drinking-water/pub-eng.pdf>), to which some industry stakeholders have recently pointed. It is clear that Dourson's papers are the basis for the Health Canada proposal. On page 36, the proposal states:

The proposed key events describing this MOA for 1,4-dioxane include: (1) accumulation of parent compound, (2) liver cell hypertrophy and necrosis, (3) DNA synthesis, (4) regenerative cell proliferation, and (5) promotion of endogenously initiated tumours. A thorough presentation and analysis of the data supporting these key events has been published by Dourson et al. (2014).

And on p. 38, the proposal states:

Further evidence for 1,4-dioxane's regenerative proliferation MOA has recently been provided by the Centre for Toxicology Excellence in Risk Assessment, which conducted a detailed MOA analysis for 1,4-dioxane-induced liver tumours for the Alliance for Risk Assessment (Dourson et al., 2014, 2017).

followed by regenerative hyperplasia, and that a threshold approach is therefore appropriate for risk assessment for this compound. *However, this conclusion is not supported by the data in female mice. In female mice, the incidence of liver tumors in the control and low dose groups were 0 and 44% respectively, while the incidence of necrosis and other non-neoplastic effects is similar or lower in the low dose group as compared to the controls. These data suggest that necrosis is not part of the sequence of events leading to tumor formation in the low dose female mice.*”

3. Dourson et al. (2014) state that the lower incidence of non-neoplastic changes in the female mice, as compared to the male mice, in NCI (1978) may be due to the fact that the low dose in females was lower (about half) than the low dose in males. Although non-neoplastic changes such as necrosis are not reported by Kano et al. (2009), it should be noted that the low dose in this study (66 mg/kg/day) was almost 6-fold lower than the low dose in NCI (1978) (380 mg/kg/day). *However, the tumor incidence in the low dose group (70% compared to 10% in controls) in Kano et al. (2009) is higher than at the much higher dose (380 mg/kg/day) in NCI (1978).* When considered as a whole, these findings do not support the conclusions of Dourson et al. (2014) that non-neoplastic changes both occur more frequently at higher doses and are necessary precursors to tumor formation.”
4. “It should be noted that, while a commenter states that WHO (2005) presents an approach based on a threshold for toxicity for regulation of 1,4-dioxane, the document actually presents risk-based water values based on both threshold and non-threshold (linear low-dose extrapolation) approaches.”

NJ DEP’s conclusion:

In conclusion, the data and explanation provided by Dourson et al. (2014) do not establish a firm or unique link to the proposed MOA of cytotoxicity followed by regenerative hyperplasia, and does not indicate that a threshold approach is appropriate for risk assessment for this compound. As such, the information provided by Dourson et al. (2014) does not invalidate the conclusion made by USEPA IRIS (2013) that the available information does not establish a plausible mode of action for 1,4-dioxane, and that the available data are not sufficient to establish significant biological support for a non-linear (threshold) mode of action. For these reasons, the approach used by USEPA IRIS (2013) which uses a linear low dose extrapolation to develop an oral cancer slope factor for 1,4-dioxane is appropriate.

MI DEQ:

1. “The subcommittee finds that the weight of evidence (WOE) supporting TERA’s proposal is *weak*.”
2. “TERA’s proposal that tumor formation is caused by cytotoxicity is not adequately supported by other studies.”

3. *“Only information related to the MOA for liver tumor formation was presented, i.e., nothing related to the MOA for the other tumor types observed in 1,4-dioxane-treated animals.”*
4. *“While some of the data presented is supportive of TERA's proposed MOA for 1,4-dioxane liver tumor formation, the NTP/NCI slide re-read for female mice is not supportive of TERA's MOA and remains unexplained (McConnell, 2013). In other studies, data for female rats or mice were either insufficient to support TERA's MOA or conflicting information was reported. In the two year drinking water study (Kano et al., 2009), a significant increased incidence of HCA and HCC [hepatoadenoma and hepatocarcinoma, respectively] was observed at the lowest dose of 66 mg/kg (Table 5). Information was presented for preneoplastic altered foci in rats, but not for mice. Based on the findings in Kano et al. (2009), a determination of preneoplastic lesions (e.g., cytotoxicity, inflammation or altered cell foci) in female mice at the low dose level cannot be made to support TERA's proposed MOA of cytotoxicity and inflammation as a precursor to tumor formation.”*
5. *“For the Dourson-proposed pathway to be supported, cytotoxicity needs to precede tumor formation for all exposed animals. The measure of cytotoxicity used in the re-read of the NCI male and female mouse liver slides was necrosis. Necrosis preceded liver tumor formation in both male mice and female mice. However, low dose female mice had similar levels of necrosis and inflammation as the female control mice and some female mice with tumors had no evidence of necrosis. The low dose group developed liver tumors, while the control mice did not. ... This indicates that necrosis may not have been a step in the process to tumor formation for 1,4-dioxane.”*
6. *“Additionally, the following considerations of the IRIS toxicity assessment for 1,4-dioxane should be noted:*
 - *The IRIS assessment has undergone two external peer reviews.*
 - *The role of cytotoxicity as a required precursor of neoplasia is supported by only one study.*
 - *In the majority of studies, the dose-response does not support cytotoxicity as a necessary precursor of neoplasia.*
 - *...*
 - *All of the tumor types induced by 1,4-dioxane are relevant to humans and there is no sufficient support for any one MOA.”*
7. *“The most compelling argument for retaining the U.S. EPA default assumption of linearity for 1,4-dioxane is the presence of multiple tumor types in rodent models, all of which are relevant to humans. TERA hypothesized an MOA for the liver tumors alone.”*

MI DEQ's conclusion:

Based on the 1,4-dioxane assessment considerations as noted in this report, the subcommittee concludes that the data regarding the carcinogenicity of 1,4-dioxane are not sufficient to deviate

from the U.S. EPA's default assumption of linearity, as described in the Cancer Guidelines (U.S. EPA, 2005).

b. Rebuttal of Dourson et al. 2017

NJ DEP:

1. Dourson et al. (2017) adjusted doses from sub-chronic studies by dividing them by a factor of 3 in an apparent attempt to compare them to values from chronic studies. "It is not appropriate to compare data from different studies for the purpose of attempting to define a quantitative relationship across studies by adjusting doses for effects in sub-chronic studies, as was done by Dourson et al. (2017)."
2. Dourson et al. (2017)'s claim of a chronology of the appearance of endpoints leading to liver tumors is *based on the appearance of liver pathology at different doses, not over time. "One cannot infer chronology from dose-response data only."*
3. Dourson et al. (2017) "back-projected" the chronology onto events and dose-response data under his hypothesized MOA, but they could alternatively be interpreted as independent effects the cause of which are unrelated and merely a function of dose.
4. Dourson et al. (2017)'s claim that the toxicity pathways for 1,4-dioxane is dependent on metabolic saturation kinetics – via which decreased metabolism at higher doses would lead to increased toxicity – is not supported by the exposure data from the two-year bioassay, which do not appear to show such a dose-response relationship. "Rather, the incidence of these [liver] effects increases linearly or positively exponentially over the whole range of doses used in the studies."
5. Dourson et al. (2017) note that tumors were found in the low-dose group in the mouse study (Kano et al. 2009), below the dose postulated to reflect saturation kinetics. This is evidence "*that tumor formation and non-tumor toxicity are decoupled.*"
6. In the NCI 1978 study, hyperplasia showed a dose-response that significantly differed from that for adenoma tumors. Relative to controls, the incidence of hyperplasia dropped while adenoma incidence increased. "*These data strongly suggest no significant linkage between hyperplasia and adenomas.*"
7. Dourson et al. (2017) relied on seven studies to argue that their chronology of events resulted in tumor formation. "[I]n order to use these data to make the case for a sequential mode of action leading to tumors, it is logical and necessary that the studies utilized should be those studies that demonstrated tumors. Otherwise, there is no basis for asserting that the sequence of toxicity ends in tumors." *However, only three of the seven studies provided tumor data.*
8. *In the two-year inhalation study (Kano et al., 2009), hyperplasia but not the postulated intermediate step of necrosis/inflammation was seen at 1,000 ppm.* Necrosis/inflammation is critical to Dourson et al.'s postulated scheme, "since hyperplasia in the absence of necrosis can be attributed to induction of growth factors."

In the two-year drinking water study (Kociba et al, 1971, 1974), hyperplasia/abnormal tumor foci were not observed at a dose that did produce tumors. “[C]ritical intermediate steps (effects) in this causal chain are missing even when subsequent steps are observed, including at doses identified by Dourson et al. (2017) as resulting in saturation kinetics.”

9. Kano et al. (2009) did observe all steps in the causal chain, including tumors, at the higher dose of 5,000 ppm. Kasai et al. (2009) also observed all steps in the causal chain, including tumors, at 1,250 ppm. *If tumor formation was only possible after all other steps had occurred, as postulated, in some studies all of the intermediate steps – but not tumor formation except at the highest dose – should have been observed. “The occurrence of all the steps, including the tumors at the same dose as in the high dose groups in Kano et al. (2009) and Kasai et al. (2009), provides no evidence for a mode of action that involves a stepwise schema.”*
10. The only carcinogen for which EPA has accepted a threshold, based on a cytotoxicity mode of action, is chloroform. In that case, actual temporal data from multiple studies show the chronology of steps resulting in tumors, with strong dose consistency between cytotoxicity and tumors. “In contrast, as noted above, this is not the case for 1,4-dioxane, for which the data supporting such a mode of action do not rise to the level of those supporting the mode of action for chloroform.”

NJ DEP’s conclusion:

In conclusion, the mode of action for 1,4-dioxane carcinogenicity remains unknown. The data and explanations provided by Dourson et al. (2017) do not establish a firm or unique link to the proposed mode of action, and they do not indicate that a threshold approach is appropriate for risk assessment for this compound. As such, the information provided by Dourson et al. (2017) does not invalidate the conclusion made by USEPA (2013b) that the available information does not establish a plausible mode of action for 1,4-dioxane, and that the available data are not sufficient to establish significant biological support for a non-linear (threshold) mode of action. For these reasons, the Department continues to believe that the risk assessment approach used by USEPA (2013b) which develops an oral cancer slope factor for 1,4-dioxane based on the USEPA default approach, linear low dose extrapolation, is appropriate.

C. The scientifically sound and health-protective approach is to use linear extrapolation in cancer dose-response modeling for 1,4-dioxane.

i. Justification based on existing guidance

The information presented above 1) demonstrates that evidence supports the potential for a genotoxic MOA, and 2) casts doubt on the plausibility of a cytotoxic MOA. To the extent the evidence is deemed insufficient to identify with certainty the MOA, there is longstanding EPA policy guidance as well as federal and state agency precedent supporting a default to a no-threshold, linear extrapolation method for cancer dose-response modeling.

(1) The Agency’s own 2005 cancer guidelines state that:

When the weight of evidence evaluation of all available data are insufficient to establish the mode of action for a tumor site and when scientifically plausible based on the available data, linear extrapolation is used as a default approach, because linear extrapolation generally is considered to be a health-protective approach.⁷⁶

(2) EPA’s 2013 IRIS assessment of 1,4-dioxane concluded that “the default linear extrapolation should be utilized to estimate the cancer risk estimates.”⁷⁷ EPA cannot deviate from its prior finding on this issue absent a compelling and well-reasoned explanation for any change in EPA’s position. *See FCC v. Fox TV Stations, Inc.*, 556 U.S. 502, 515 (2009).

(3) In 2015, the New Jersey (NJ) Department of Environmental Protection (DEP) stated that “the available data are not sufficient to establish significant biological support for a non-linear (threshold) mode of action.”⁷⁸

(4) In a 2015 report, the Michigan Department of Environmental Quality Toxics Steering Committee stated that “the currently available scientific information regarding the carcinogenicity of 1,4-dioxane ... are insufficient to deviate from the U.S. EPA’s default assumption of linearity for developing a cancer potency factor.”⁷⁹

(5) In 2018, the NJ DEP reiterated its conclusion that “the mode of action for 1,4-dioxane carcinogenicity remains unknown. The agency emphasized that previously published studies, including Dourson 2014 and 2017, “do not indicate that a threshold approach is appropriate for risk assessment for this compound” and “that the available data are not sufficient to establish significant biological support for a non-linear (threshold) mode of action.” Importantly, NJ DEP concludes that “the risk assessment approach used by

⁷⁶ U.S. EPA, *Guidelines for Carcinogen Risk Assessment* at pp. 3-21 (Mar. 2015), https://www.epa.gov/sites/production/files/2013-09/documents/cancer_guidelines_final_3-25-05.pdf.

⁷⁷ *See* U.S. EPA, IRIS, *Toxicological Review of 1,4-Dioxane (With Inhalation Update)* at p. 137 (Sept. 2013), https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0326tr.pdf.

⁷⁸ N.J. Dep’t of Env’tl. Protection, Response to Public Input on Draft Interim Ground Water Quality Criteria and Draft Interim Practical Quantitation Levels for Eleven Chemicals at pp. 11-17, <https://www.state.nj.us/dep/dsr/supportdocs/11-chemicals-response.pdf>.

⁷⁹ Mich. Dep’t of Env’tl. Quality, Toxics Steering Group, 1,4-Dioxane Subcomm., Review of a 1,4-Dioxane Presentation by Michael Dourson, Ph.D. on October 8, 2013 (Feb. 2015), https://www.michigan.gov/documents/deq/deq-aqd-toxics-14-DioxaneTSG_Report_2015_487415_7.pdf.

USEPA (2013b) which develops an oral cancer slope factor for 1,4-dioxane based on the USEPA default approach, linear low dose extrapolation, is appropriate.”⁸⁰

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

EPA must employ health-protective approaches to dose-response modeling, as described at length in the National Research Council’s report, *Science and Decisions: Advancing Risk Assessment*. In this report, the NRC specifically provides important perspective on the need to conduct a linear extrapolation at the population level, even where a threshold might theoretically exist. The authors state, for example, that:⁸¹

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

Overall, the NRC concluded that “***cancer and noncancer responses [to chemical exposures] be assumed to be linear as a default***.”⁸²

In their *State-of-the-science workshop report: issues and approaches in low-dose-response extrapolation for environmental health risk assessment*, White et al. (2009)⁸³ also highlight that:

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

⁸⁰ N.J. Dep’t of Env’tl. Protection, Final Criteria: 50 N.J.R. 334(a), Response to Comment 39, published in the *New Jersey Register*, January 16, 2018, available at <https://bit.ly/2K70268>.

⁸¹ NAT’L RESEARCH COUNCIL, *SCIENCE AND DECISIONS: ADVANCING RISK ASSESSMENT* at chp. 5, pp. 130-131 (2009), <https://www.ncbi.nlm.nih.gov/pubmed/25009905>.

⁸² *Id.* at chp. 5, p. 180.

⁸³ Ronald H. White, et al., *State-of-the-science workshop report: issues and approaches in low-dose-response extrapolation for environmental health risk assessment*, 117:2 ENVTL. HEALTH PERSPECTIVES 283-87 (2008), <https://www.ncbi.nlm.nih.gov/pubmed/19270800> (emphasis added).

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

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

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

4. Key data gaps

Under the 2016 reforms to TSCA, Congress enhanced EPA’s authority to require submission of existing information and development of new information, including on products and workplace exposures. Despite the major gaps identified below and others that EDF and other stakeholders have identified over the past 2.5 years, EPA made no effort whatsoever to use these authorities to obtain critical exposure information on 1,4-dioxane.

A. Environment

i. Dearth of environmental monitoring data

- EPA states that “recent monitoring data on ambient surface water levels indicate relatively low levels” (p. 213) but never provides any data.
- EPA states: “Limited sediment monitoring data for 1,4-dioxane that are available suggest that 1,4-dioxane is present in sediments” (pp. 131, 211). But no such data are presented in the draft risk evaluation or the preceding problem formulation document.

ii. Dearth of environmental fate data

EPA appears to have identified only one study providing measured values for environmental fate and transport of 1,4-dioxane: a microcosm study on soil biodegradation (p. 44). As a result, it relies on model estimate for all other fate and transport parameters.

iii. Dearth of ecotoxicity data

The only ecotoxicity data EPA has is for aquatic organisms (fish, algae, water flea); it lacks any such data for soil- or sediment dwelling organisms or terrestrial or avian species. Moreover, EPA has no aquatic chronic toxicity data except for fish. These concerns are elaborated on in subsection iv immediately below. Despite these major gaps, EPA repeatedly makes sweeping statements about the lack of any unreasonable risks to the environment as a whole (pp. 21, 156).

iv. Available information on potential ecological hazards is insufficient to adequately evaluate risks.

Information on potential ecological hazards in EPA's draft risk evaluation is limited to aquatic organisms based on: two short-term acute toxicity studies for aquatic plants; three acute toxicity studies to aquatic invertebrates; and four acute toxicity studies for fish. For chronic toxicity, EPA relied on two studies conducted on fish.

EPA cannot fully and sufficiently evaluate the potential ecotoxicity of 1,4-dioxane on the basis of these studies alone. EPA has not identified (or has ignored): 1) any study of toxicity to sediment-dwelling organisms, or 2) any studies of toxicity to terrestrial or avian organisms. Indeed, EPA notes this gap in the problem formulation for 1,4-dioxane on which it relies in the draft risk evaluation. However, it simply assumes, with no explanation or support, that: "While no ecotoxicity studies were available for sediment organisms, the toxicity of 1,4-dioxane to sediment invertebrates is expected to be similar to the toxicity to aquatic invertebrates" (Problem Formulation, p. 42).

EPA expanded on this assumption by concluding that "available hazard, fate and exposure characteristics (Sections 2.3.1 and 2.3.3) suggest that sediment organisms are not at risk from 1,4-dioxane exposures" (Problem Formulation, p. 34). This statement is problematic because (i) there are no actual ecotoxicity data for sediment organisms (Problem Formulation, p. 34), and (ii) there are sufficient data to suggest that these organisms are being exposed to 1,4-dioxane through exposure to sediment pore water (Problem Formulation, p. 28 and Part I sec. 5.A.i.c.1) of these comments.)

Additionally, EPA ignored the one study on terrestrial plants that it identified in its problem formulation (p. 34):

In one study, lettuce (*Actuca sativa*) were exposed to 1,4-dioxane in a germination/root elongation toxicity test for 3-days. An EC50 of 1,450 mg/L was reported for germination (Reynolds, 1989).

This single, short-term study is not sufficient to assess potential acute, let alone chronic, toxicity to terrestrial organisms that might be exposed to 1,4-dioxane through land-application of biosolids or sludge (see Part I sec. 5.A.i.c.2)) as well as through water sources.

As a result of having such limited ecotoxicity data, EPA cannot possibly sufficiently evaluate the ecological hazards and risks of 1,4-dioxane present in environmental releases (including those that EPA has largely or entirely ignored (see Part I sec. 5.A.i.c. iv, v). EPA also cannot evaluate relevant environmental hazards as required by 40 C.F.R. § 702.31(d)(3).

In sum, EPA's sweeping conclusion that 1,4-dioxane presents a low ecological hazard lacks substantial evidence in the record and does not reflect the best available science. In addition, given that EPA could have reasonably required the submission or generation of this information during the development of this draft risk evaluation, EPA has failed to consider reasonably available information about these hazards.

B. Human Health

There was considerable discussion during the 1,4-dioxane SACC Meeting on the serious data gaps in the risk evaluation. Among the many noted by the SACC are data gaps in dermal absorption data; extent of use of PPE and its efficacy; workplace monitoring data; exposures from certain conditions of use, like metal working; and data on central nervous system effects. One SACC member recommended that EPA use its authorities to gather additional data, or at least perform quantitative uncertainty analyses. SACC members gave specific examples of data that EPA could acquire before finalizing the risk evaluation. Among those were dermal absorption testing through use of NAMs and workplace exposure monitoring. One member noted that EPA could go into facilities and acquire monitoring data within 30 days, using modern techniques.

Below we provide further comment on some of the serious data gaps affecting EPA's assessment of human health risks in its draft risk evaluation.

i. Dearth of product/use concentration data

- Open system functional fluids: EPA claims it derived fluid concentrations from available SDSs (p. 62), but none of the relevant cited SDSs that are publicly accessible makes any mention of 1,4-dioxane as a constituent.
- Spray foam application: Only one of the several SDSs EPA cites for this use (p. 68) makes any mention of 1,4-dioxane as a constituent, so EPA's entire exposure analysis rests on this one source and value, precluding any ability to know whether EPA's analysis is at all representative of a large industry that entails, by EPA's estimate, nearly 180,000 workers (p. 68).

- EPA’s illegal decision to exclude exposures to 1,4-dioxane when present as a byproduct (see Part I sec. 2.A.) means it has failed to examine the concentrations of 1,4-dioxane in any consumer products. It cannot be assumed that such concentrations and resulting exposures are infrequent or insignificant. EDF located two recent reports from the Citizens Campaign for the Environment (CCE) that report on analyses of levels of 1,4-dioxane in consumer products that found detectable levels of 1-4,dioxane in dish soaps, laundry detergent, shampoos, and body washes, as well as a number of baby products.⁸⁴ The group’s 2018 report listed detectable levels of 1,4-dioxane in 23 out of 30 products tested, and its 2019 report analyzed 80 products and found that 65 of the products had detectable levels of 1,4-dioxane.

ii. *Limited, unrepresentative inhalation exposure data for workers*

- EPA’s sources of workplace exposure data are from selective, unrepresentative sources; lack critical detail on which processes, exposure sources and worker activities they represent; and are insufficient to understand the distribution of exposures in a given setting (pp. 55, 57, 60, 62, 65, 67, 69-74, 146-7).
- Industrial uses: EPA lumps together a highly diverse set of uses as “industrial uses” (p. 58). They encompass a huge array of sectors, from textiles to agricultural chemicals to pharmaceuticals, and very different functional uses, from solvent to catalyst to intermediate to wetting agent. EPA asserts without providing any support that all such operations “are expected” to be similar. EPA’s only source of worker exposure data for this broad swath of uses is an EU risk assessment that looked only at the pharma sector and use as a solvent (p. 59). That source provided no detail as to how the data were calculated or what percentile they represent.
- Open system functional fluids: EPA’s cited source (Burton and Driscoll 1997) is a NIOSH site report motivated by worker concern over fungi- and bacteria-contaminated synthetic metal-working fluids (MWF). It entailed no direct measurements of 1,4-dioxane, only synthetic MWF and it is not clear the fluids at this site even contained the chemical (p 61).

⁸⁴ Citizens Campaign for the Environment, *Shopping Safe: The 2018 Consumer Shopping Guide Protecting Your Household from 1,4-dioxane Exposure* (2018), <https://static1.squarespace.com/static/5b72eb5b8ab7222baffc8dbb/t/5c70869924a694a5a159cefe/1550878371100/DioxaneShoppersGuide.pdf>; and Citizens Campaign for the Environment, *Shopping Safe: The 2019 Consumer Shopping Guide Protecting Your Household from 1,4-dioxane Exposure* (2019), https://static1.squarespace.com/static/5b72eb5b8ab7222baffc8dbb/t/5c9a8745ee6eb01dd7c77d5e/1553631051532/FINAL319_+The+2019+Consumer+Shopping+Guide+.pdf.

- Spray foam application: EPA lacks any monitoring data (pp. 67-68, Table 3-17). EPA says it estimated values using an EPA exposure scenario document (USEPA 2018a) that is not publicly accessible (p. 68). EPA also employed modeling that it asserts is “conservative” because it assumes activities take place “indoors, without engineering controls, and in an open-system environment where vapors freely escape.” Yet all of these conditions may well characterize spray foam application, which takes place in myriad houses and other buildings
- Printing inks: EPA’s analysis of worker exposure to printing inks is based on a single air sample reported in a 2016 paper; despite the fact that the authors and other researchers note that the concentration could well be an underestimate (p. 70), EPA asserts it is likely an overestimate (p. 71).

iii. *Failure to adequately consider other authoritative sources of workplace inhalation exposure data*

a. *EPA references OSHA monitoring data, but does not incorporate them into its exposure assessment.*

- EPA’s 1,4-dioxane Problem Formulation refers to OSHA data collected between 2002 and 2016 as “key data.”⁸⁵
- However, EPA inappropriately excludes these data due merely to challenges it experienced in downloading the data from OSHA’s online platform. The data received a score of only 8 for Applicability because, according to EPA: “Looks like it should be an excel file with exposure data, but it’s all smooshed together in a text file and not useful.” That low score pulled the overall score assigned to the OSHA data into the Unacceptable range.⁸⁶ (see Part I sec. 8.C. in these comments for more discussion)
- Our own search of the OSHA Chemical Exposure Health Data yielded 475 air samples for 1,4-dioxane between 1987-2012.⁸⁷ The OSHA PEL for 1,4-dioxane

⁸⁵ U.S. EPA, *Problem Formulation of the Risk Evaluation for 1,4-Dioxane CASRN: 123-91-1* at pp. 30, 69 (May 2018), https://www.epa.gov/sites/production/files/2018-06/documents/14-dioxane_problem_formulation_5-31-18.pdf.

⁸⁶ U.S. EPA, *Risk Evaluation for 1,4-Dioxane Systematic Review Supplemental File: Data Quality Evaluation of Environmental Releases and Occupational Exposure Data* at p. 105 (June 2019), https://www.epa.gov/sites/production/files/2019-06/documents/4_14-d_supplemental_data_quality_evaluation_environmental_release_and_occupational_exposure_06272019.pdf.

⁸⁷ SAMPLE DATA SEARCH RESULT, https://www.osha.gov/pls/samp/sampling_search.search?establishment=&city=&state=--&zip=&startyear=&endyear=&sic=&naics=&substance=dioxane&imis=&beginresult=&endresult=&p_start=120&p_finish=140&p_sort=&p_desc=asc&p_direction=Prev&p_show=20 (last visited Jul. 19, 2019).

of 100 ppm (8-hour, TWA) was last updated in 1989 (p. 194), so workplace monitoring data after 1989 are likely relevant.

b. *EPA has excluded relevant data from the 2002 EU Risk Assessment.*⁸⁸

- For Industrial Uses, EPA excludes the highest exposure point (184 mg/m³) from the 2002 EU Risk Assessment, asserting but not adequately explaining why it considers the value “is likely an outlier” (p. 264).
- While it does not appear that EPA assessed cleaning agents and paint as end uses of 1,4-dioxane at all, the EU Risk Assessment did so and found the chemical’s use as a cleaning agent, in particular, to be a significant exposure source:
 - For 6-8-hour exposure, the EU Risk Assessment found the reasonable worst case to be 50 mg/m³ and the typical concentration to be 15 mg/m³, which are considerably higher than the Central Tendency ADCs and High-end ADCs EPA relies on for all of its exposure scenarios (see Table 5-5 on p. 137).
 - “Repeated-dose toxicity and carcinogenicity after combined (i.e. respiratory and dermal) exposure at the workplace cannot be excluded for the scenario ‘formulation’ and the subscenario ‘use in cleaning agents.’”⁸⁹

By excluding these data sources from its analysis, EPA has failed to consider reasonably available information in violation of TSCA § 26(k).

iv. *Reliance on extremely limited industry workplace inhalation data from a single site*

For its Manufacturing scenario, EPA chose to use only data it received from BASF, comprised of just 30 samples from a single manufacturing site in Zachary, Louisiana, which closed in 2018. In doing so, EPA has assumed these data to be representative of all U.S. manufacturing (see pp. 254-257).

- While the 2016 BASF data are summarized in Appendix G, EPA does not provide access to the original source; no link is provided in the HERO entry for this source.
- Further, EPA makes several assumptions about these data, which appear not to have been confirmed with BASF. While EPA first states: “Occupational exposures to 1,4-dioxane during manufacturing were estimated by evaluating full-shift, personal breathing zone (PBZ) monitoring data obtained by BASF during internal industrial hygiene (IH) studies,” (p. 54), it later states:

⁸⁸ European Chemicals Bureau, *European Union Risk Assessment Report: 1,4-dioxane* (2002), <https://echa.europa.eu/documents/10162/a4e83a6a-c421-4243-a8df-3e84893082aa>.

⁸⁹ *Id.* at 99.

- “EPA *assumed* that the 2016 BASF data are PBZ measurements relevant to worker activities and are also 8-hour TWA measurements” (p. 55, emphasis added), and
- “EPA *assumed* that these monitoring data were originated via PBZ measurements” (p. 254, emphasis added).
- These data have other serious limitations, as EPA acknowledges: “The data sets used mostly lacked specific descriptions of worker tasks, exposure sources, and possible engineering controls to provide context” (p. 55).
- EPA scored the 2016 BASF and 2017 BASF data as 1.3 and 1.7, respectively, in its systematic review.⁹⁰ Several questions arise:
 - 2016 BASF data: Why did EPA assign a score of 1 to “Sample Size” and included a note indicating “Representative sample size,” when the data set comprised only 28 samples from a single site? In the draft risk evaluation itself EPA acknowledges that these data are unlikely to be representative: “It is uncertain to what extent the limited monitoring data used to estimate inhalation exposures for this scenario that could be representative of occupational exposures in other manufacturing facilities of 1,4-dioxane” (p. 55).
 - 2017 BASF data: Why did EPA assign a score of 2 to “Sample Size,” when the data set comprised only four data points from a single site?

v. *Lack of dermal exposure data*

EPA has no data on dermal exposures or dermal absorption in humans. Instead – as the basis for its entire evaluation of dermal risks – EPA heavily relies on Bronaugh, 1982 (see p. 76 for the first of many citations to this source), which apparently reported the results of an *in vitro* assay using excised human skin.

EDF has serious concerns with EPA’s reliance on Bronaugh, 1982. As indicated in HERO,⁹¹ this source is a chapter of a book about cosmetics. However, it has not been made publicly available by EPA. EDF requested on July 10th, 2019, that EPA provide the source but the Agency has not done so. After several unsuccessful follow-up requests, EDF purchased the book.

Upon reading the book chapter, it became immediately clear that Bronaugh, 1982 is a secondary source, and cites data from studies without providing sufficient description of their methodology

⁹⁰ U.S. EPA, *Risk Evaluation for 1,4-Dioxane Systematic Review Supplemental File: Data Quality Evaluation of Environmental Releases and Occupational Exposure Data* at pp. 124-25 (June 2019), https://www.epa.gov/sites/production/files/2019-06/documents/4_14-d_supplemental_data_quality_evaluation_environmental_release_and_occupational_exposure_06272019.pdf.

⁹¹ https://hero.epa.gov/hero/index.cfm/reference/details/reference_id/196146.

and other details. Bronaugh provides the following citation for the skin penetration values on which EPA relies, which is not to a published paper but rather to a meeting *abstract*:

Reference 3. Bronaugh, R. L., Congdon, E. R., Stadtmauer, E. A., and Scheuplein, R.J.: Percutaneous absorption of 1,4-dioxane through excised human skin, Soc. Toxicol. Abstracts, March 1980, p. 49.

EDF attempted to determine whether the abstract was part of a published study by searching online using the reference above, without success. Thus, it appears that data on which EPA is relying is drawn from only an abstract submitted to a meeting, and any underlying study – if one exists at all – that appears to be unpublished and not peer reviewed. Further, it seems very unlikely that EPA itself has more than the book chapter, i.e., the original study.

Given all this, it is highly concerning that EPA not only relied so heavily on this source, but did not apply its systematic review approach to evaluate its quality. Had it done so, the source likely would not have scored well.

Of note, EPA reported that the dermal exposure to 1,4 dioxane in this experiment only lasted for 205 minutes (p. 83), rather than the standard 8- or 24-hour exposure period. As such, the study may have underestimated absorption because the experimental conditions would not have reached and reflected a steady state.

Furthermore, during the July 30th SACC meeting on 1,4-dioxane, one of the panel members, an expert on dermal exposure assessment, pointed to a number of concerns with a separate aspect of the same study described in the book chapter. It is unclear to EDF whether or how the results reported from that study, which indicates that 1,4-dioxane readily evaporates (90% evaporation), are incorporated into the draft risk evaluation. However, the SACC panel member raised two concerns with the experiment:

1. It used wax paper as the substrate, which is an inappropriate surrogate for skin. While the chemical may readily evaporate from wax paper, it is likely that some of the chemical will be absorbed into skin.
2. The test may well have taken place in a fume hood, which would result in an overestimation of the extent and rate of evaporation due to elevated air flow conditions in the hood.

The 90% evaporation rate reported by this study differs greatly from the 14% and 22% evaporation rates from the Kasting and Miller (2006) framework. These limitations further compromise our confidence in the Bronaugh book chapter.

EPA also relies on Marzulli et al., 1981, which examined absorption in adult rhesus monkeys. Yet, even beyond the differences between species, the vehicles employed in this study as a carrier for the chemical were methanol and skin lotion, and it is not clear how representative they are of absorption under the conditions of use relevant to this risk evaluation. Moreover, the authors describe their results as providing only “crude estimates.”

In addition to relying on these questionable studies, EPA appears to have ignored other relevant dermal absorption data. A 2013 study conducted by Dennerlein et al. assessed the dermal absorption of three industrial chemicals, including 1,4-dioxane following a four-hour exposure.⁹² While the study also had a short exposure period, the authors found that 1,4-dioxane had the highest percutaneous penetration when compared to the other two chemicals analyzed (anisole and cyclohexanone), with a penetration of 2,868.2 µg per 0.64 cm² of skin over four hours of exposure⁹³ and mean flux of 1,116.8–1,483.4 µg per cm² of skin per hour. It does not appear that EPA identified, evaluated or utilized this study, as it is not referenced in either the draft risk evaluation or the Systematic Review Supplemental File. EPA’s failure to identify this recent study suggests deficiencies in the systematic review process.

EPA argues that “only a fraction” of 1,4-dioxane on the skin will be absorbed due to its rapid evaporation (p. 75). To the extent this is the case, EPA does not appear to have accounted for the resulting inhalation exposure – or the potential for combined exposure pathways (see below - *Failure to consider combined exposure pathways for workers*) – to such a dermally exposed worker. Elsewhere in its draft, EPA notes that “if in aqueous solution, evaporation may be less likely” (p. 150); but it is not at all clear whether or how EPA factored variable rates of evaporation into its analysis of different conditions of use.

In the absence of any actual monitoring data, EPA is forced to make yet more assumptions to estimate dose. For example, EPA assumes, without any explanation or substantiation, that workers will experience only “one exposure event (applied dose) per work day” (p. 76). (Of note, one of the SACC panel members expressed that this assumption seemed “too optimistic” and “not conservative.”) As highlighted above, all of the Agency’s arguments remain speculative, at best, given the dearth of actual dermal exposure data on 1,4-dioxane. EPA could have easily filled these data gaps using its testing authority. One of the SACC panel members noted, for example, that EPA could still quickly and inexpensively acquire dermal exposure data, including through the use of appropriate NAMs (kinetic absorption through skin).

⁹² Kathrin Dennerlein, et al., *Studies on percutaneous penetration of chemicals – Impact of storage conditions for excised human skin*, 27:2 TOXICOLOGY IN VITRO 708-13 (2013), <https://www.ncbi.nlm.nih.gov/pubmed/23219852>.

⁹³ Bronaugh, 1982 used a 205-minute exposure, equivalent to 3.4 hours.

vi. *Lack of data on glove use and efficacy*

As noted by several SACC panel members, EPA does not appear to have any actual data on glove use, such as types used and frequency of use. Use of gloves is likely very context-specific. For example, one SACC member noted that it may be less likely for a worker to use gloves if 1,4-dioxane is diluted in a solution; in this case, evaporation would be slower and absorption would be modified by other constituents in the solution, leading to potentially higher dermal doses.

EPA also does not appear to have any data on the efficacy of gloves when they are used. While EPA notes some of the ways in which glove use can actually *increase* skin exposure through occlusion (Appendix G, section G.7.3, p. 292), the agency simply assumes fixed protection factors (PFs) of 5x, 10x, and 20x, which do not appear to be supported by any empirical data reflecting the complexities of gloves and glove use in the real world. (see further discussion in Part I sec. 7.E.)

vii. *Dearth of dermal toxicity data*

EPA has identified no acute or repeated dose, short-term, subchronic, or chronic studies that examined toxicity via dermal exposure (pp. 85, 90). As a result it relied on extrapolation from oral and inhalation toxicity studies; we discuss in Part I sec. 5.B.vi. of these comments the many concerns raised by this approach.

viii. *Lack of reproductive/developmental/neuro/immuno toxicity data*

The only current available developmental toxicity study is Giavini et al 1985 (p. 87), a short-term study that evaluated toxicity by the oral route of exposure. The figure below, from the 2012 ATSDR ToxProfile of 1,4-dioxane,⁹⁴ clearly shows the database deficiencies for reproductive, developmental, neurological, and immunological endpoints through one or more routes of exposure. However, the Agency has made no effort to use its authority to fill these data gaps. By failing to fill these gaps, EPA violates its duty to evaluate relevant human hazards under 40 C.F.R. § 702.31(d)(3).

⁹⁴ Agency for Toxic Substances & Disease Registry, *Toxicological Profile for 1,4 Dioxane* at p. 139 (Apr. 2012), <https://www.atsdr.cdc.gov/toxprofiles/tp187.pdf>.

	Systemic									
	Death	Acute	Intermediate	Chronic	Immunologic/Lymphoretic	Neurologic	Reproductive	Developmental	Genotoxic	Cancer
Inhalation	●	●	●	●		●				●
Oral	●	●	●	●		●		●	●	●
Dermal	●	●	●							●

Animal

● Existing Studies

5. Analytic gaps/deficiencies

In this section, we identify numerous instances of EPA failing to consider available information or engaging in irrational analyses. In the process, EPA violates its duties to rely on the “best available science” (TSCA section 26(h)) and to consider all “reasonably available information” on hazards and exposures (TSCA section 26(k)). EPA’s analyses are also arbitrary and capricious because they fail to consider important aspects of the problem and adopt conclusions that run counter to the evidence before the agency.

A. Environment

- i. *Disregard of environmental monitoring data led to an overreliance on predictive modeling.*
 - a. *Models are predictions and subject to uncertainty and variability, which must be considered and presented along with conclusions.*

In the Environmental Fate and Transport section of its draft risk evaluation, EPA identified and evaluated four sources of data through its systematic review process⁹⁵—in addition to “reasonably available information source[s] to determine environmental fate and transport” that were “not subject” to systematic review evaluation: two handbooks on predictive modeling of physico-chemical properties; EpiSuite; and a peer-reviewed study conducted in 2001 on the applicability of *Amycolata sp.* CB1190 (a bacterium) for bioremediation.

⁹⁵ U.S. EPA, *Risk Evaluation for 1,4-Dioxane, Systematic Review Supplemental File: Data Quality Evaluation of Environmental Fate and Transport Studies*, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0238-0004>

EPA's disregard of empirical data on the fate and transport of 1,4-dioxane in the environment (p. 43) led EPA to rely on a "qualitative assessment of the physical-chemical properties and fate of 1,4-dioxane in the environment for sediment and land-applied biosolids" (p. 20) and a screening-level assessment of risks to aquatic organisms (p. 148).

While models based on physico-chemical properties of chemicals can be useful for preliminary assessments of both hazard and exposure, they are inappropriate for assessing risk without a corresponding and supportive uncertainty analysis. This is because deviations in model assumptions can lead to high variability and error propagation in the model prediction.⁹⁶ For example, in the 2015 Work Plan Chemical Problem Formulation and Initial Assessment for 1,4-dioxane, the fugacity model in EpiSuite predicted that 1,4-dioxane would preferentially partition to soil (up to 56%).⁹⁷ This is in contrast to the conclusions of other risk assessments of 1,4-dioxane⁹⁸ — and to the conclusions in this draft risk evaluation, which are that "1,4-dioxane is not likely to accumulate in wastewater biosolids, sediment, soil, or biota, and is expected to largely remain in aqueous phases ***" (p. 46).

The variability in this model output (as predicted by the same program, for the same risk evaluation, conducted by the same Agency) speaks to the sensitivity of predictive modeling to underlying assumptions and inputs, which must be quantified prior to drawing conclusions about the lack of exposure or risk in the risk evaluation. The Guiding Principles for Monte Carlo Analysis,⁹⁹ describes, generally, a framework and the principles for characterizing uncertainty and variability in risk assessment. This document was developed in recognition of "[t]he importance of adequately characterizing variability and uncertainty in fate, transport, exposure and dose-response assessments for human health and ecological risk assessments," which "has been emphasized in several U.S. Environmental Protection Agency (EPA) documents and activities."¹⁰⁰

⁹⁶ Matthew MacLeod, et al., *Evaluating and expressing the propagation of uncertainty in chemical fate and bioaccumulation models*, 21(4) ENVTL. TOXICOLOGY & CHEMISTRY 700–709 (2002), [10.1002/etc.5620210403](https://doi.org/10.1002/etc.5620210403).

⁹⁷ U.S. EPA, *TSCA Work Plan Chemical Problem Formulation and Initial Assessment* p. 20 (2015), https://www.epa.gov/sites/production/files/2017-06/documents/14_dioxane_problem_formulation_and_initial_assessment.pdf.

⁹⁸ ECHA, *Risk Assessment Report: 1,4-dioxane* at p. 21 (2002), <https://echa.europa.eu/documents/10162/a4e83a6a-c421-4243-a8df-3e84893082aa>; ATSDR, *Toxicological Profile For 1,4-Dioxane* at p. 166 (2012), <https://www.atsdr.cdc.gov/toxprofiles/tp187.pdf>.

⁹⁹ U.S. EPA, *Guiding Principles for Monte Carlo Analysis* (1997), <https://www.epa.gov/sites/production/files/2014-11/documents/montecar.pdf>.

¹⁰⁰ GUIDING PRINCIPLES FOR MONTE CARLO ANALYSIS, <https://www.epa.gov/risk/guiding-principles-monte-carlo-analysis> (last visited Aug. 20, 2019).

Specifically, predictive modeling of soil partitioning is heavily dependent on one physico-chemical value—the organic carbon to water partition coefficient (K_{OC}). This value has itself been estimated, not measured, for 1,4-dioxane (pp. 45, 212).¹⁰¹ Furthermore, K_{OC} values are known to vary by much as 50 percent, depending on how they are estimated, including the properties of the soil used in testing or assumed.¹⁰²

For this draft risk evaluation, EPA reported two estimated K_{OC} values (0.4 and 1.23, reported as $\log K_{OC}$ or 2.5 and 17 L/kg, respectively) (p. 45, tbl. 3.1). These values differ by nearly seven-fold. Rather than completing an analysis on how this variability may affect the uncertainty in the outputs from its predictive model, the Agency instead simply used the lower, less conservative value, and on that basis concluded that “1,4-Dioxane is not expected to adsorb to soil and sediment due to its low partitioning to organic matter (estimated $\log K_{OC} = 0.4$)” (p. 212).

Considering uncertainty and variability in these models is important in this context because EPA used this conclusion to justify both 1) eliminating all environmental pathways from being analyzed in the Draft Risk Assessment, other than “ambient water exposure to aquatic vertebrates, invertebrates and aquatic plants, sediment and land-applied biosolids,” (Problem Formulation, p. 41) and 2) deciding it need not conduct any further analysis of the pathways it did retain in the risk evaluation. It did so without including any uncertainty analysis to understand the confidence that can be put into the models on which it relied.

EPA’s failure to address the uncertainty or variability underlying its entire aquatic exposure analysis is arbitrary because EPA has “entirely failed to consider an important aspect of the problem.” *Motor Vehicle Mfrs. Ass’n v. State Farm Mut. Auto. Ins. Co.*, 463 U.S. 29, 43 (1983). EPA’s environmental risk characterization also fails to account for uncertainty and variability as required by 40 C.F.R. § 702.43(b)(1). EPA has not integrated the information about uncertainty and variability into an overall characterization of the impact of uncertainty and variability on estimated risks. *See id.*

b. *EPA ignored relevant, reasonably available environmental data that could be used to support, or in place of, model predictions.*

EDF has discussed elsewhere in these comments EPA’s failure to identify and incorporate into its analysis 1,4-dioxane data from the Third Unregulated Contaminant Monitoring Rule

¹⁰¹ *See also* MACKAY D, HANDBOOK OF PHYSICAL-CHEMICAL PROPERTIES AND ENVIRONMENTAL FATE FOR ORGANIC CHEMICALS p. 2310 (2d ed. 2006) (“Sorption Partition Coefficient, $\log K_{OC}$: 1.23 (soil, estimated-KOW, Lyman et al. 1982).

¹⁰² *See, e.g.,* Lawrence A. LeBlanc, et al., *Occurrence, Distribution and Transport of Pesticides, Trace Elements and Selected Inorganic Constituents into the Salton Sea Basin, California, 2001–2002*, USGS p. 31 (2004), <https://pubs.usgs.gov/sir/2004/5117/>.

(“UCMR3”) (see Part I sec. 5.A.iv) and 1,4-dioxane’s presence in groundwater (see Part I sec. 5.A.v). In this section we discuss two additional relevant data sources EPA has also ignored that could support model predictions or be used to identify environmental exposure.

- 1) Site investigation reports that delineate 1,4-dioxane impact and predict its fate and transport.

There are important circumstances under which fugacity models cannot accurately predict fate and transport of a chemical such as 1,4-dioxane without empirical data or more extensive modeling. For example, depending on groundwater flow and hydrostatic conditions, there is some evidence that 1,4-dioxane when present in water as a contaminant can effectively be stored in place in the pore water and will persist there.¹⁰³ This is consistent with the conclusion in the ATSDR Toxicological Profile that “1,4-Dioxane is expected to persist in both water and soil.”¹⁰⁴

When chemicals persist in the environment, the chance for long-term exposure to human and ecological receptors increases.

EPA chose not to evaluate 272 on-topic studies it had identified as relevant to analyzing aquatic exposure because “EPA determined that no environmental pathways would be further analyzed (p. 213). Yet given potential model uncertainties, variabilities, and limitations, EPA still lacks – and needs – an empirical basis to draw sound conclusions about the chemical’s environmental fate and transport. Another potential source of empirical data EPA also did not evaluate are site investigation reports,¹⁰⁵ which evaluate 1,4-dioxane’s presence in the environment—primarily under corrective action programs—and would provide EPA with a more complete understanding of the actual fate and transport of 1,4-dioxane.

These site investigation reports are “reasonably available information” that EPA was required to consider in conducting a risk evaluation under TSCA. 15 U.S.C. § 2625(k). EPA’s failure to consider these reports is contrary to TSCA, and arbitrary and capricious. Similarly, EPA’s exclusion of 272 on-topic studies regarding aquatic exposure violate EPA’s duty to consider reasonably available information and is arbitrary and capricious.

¹⁰³ U.S. EPA, Office of Solid Waste & Emergency Response, *Treatment Technologies for 1,4-Dioxane: Fundamentals and Field Applications* (Dec. 2006), <https://clu-in.org/download/remed/542r06009.pdf>.

¹⁰⁴ *Id.* at p. 182.

¹⁰⁵ See, e.g., David T. Adamson, et al., *A Multisite Survey To Identify the Scale of the 1,4-Dioxane Problem at Contaminated Groundwater Sites*, 1(5) ENVTL. SCIENCE & TECHNOLOGY LETTERS 254–258 (2014), [10.1021/ez500092u](https://doi.org/10.1021/ez500092u); 1,4-DIOXANE: SITE CHARACTERIZATION AND ANALYTICAL METHODS, https://clu-in.org/contaminantfocus/default.focus/sec/14-dioxane/cat/site_characterization_and_analytical_methods/ (last visited Aug. 9, 2019).

- 2) STORET non-detection data points based on elevated method detection limits cannot be disregarded.

In its assessment of aquatic environmental exposures, EPA reviewed the STOrage and RETreival (STORET) and National Water Information System (NWIS) for the past ten years (p. 46). These data, along with E-FAST model outputs (0.006 µg/L to 11,500 µg/L) (pp. 46, 223), were compared against calculated acute and chronic aquatic concentrations of concern (COC, 247,200 µg/L and 14,500 µg/L, respectively). According to its review of surface water data reported to STORET and NWIS, EPA found that there was a “detection rate of approximately 6% for this media, with detections ranging from 0.568 to 100 µg/L.” However, the range EPA cites discarded analytical sample values with extremely high method detection limits (MDLs) in these databases.

MDLs describe the concentration below which the analytical lab cannot confidently measure and determine if the analyte concentration is greater than zero. Our review of the STORET and NWIS data between the years 2009-2019¹⁰⁶ revealed that there were 59 samples with MDLs greater than 100 ug/L. Of these, 34 were from surface water samples, some of which had MDLs as high as 28,000 ug/L—nearly double the chronic aquatic COC. EPA discarded all of these values – even though the “true” concentration of these surface water samples may be well above its chronic COCs for aquatic organisms.

Estimates of surface water concentrations of 1,4-dioxane must take MDLs, as well as other data sampling and quality concerns, into account when considering potential exposure. These data are “reasonably available information” that EPA was required to consider in conducting its risk evaluation for 1,4-dioxane. 15 U.S.C. § 2625(k). EPA’s failure to consider these data is contrary to TSCA, and arbitrary and capricious. EPA has also failed to comply with 40 C.F.R. § 702.43(b)(2)’s requirement that EPA include a “discussion of data quality (e.g., reliability, relevance, and whether methods employed to generate the information and reasonable for and consistent with the intended use of the information), as well as assumptions used.” *Id.*

c. EPA cannot ignore relevant environmental exposures pathways based on low partitioning to organic compounds

Even if EPA’s conclusion that 1,4-dioxane has “low sorption to soil, sediment, and suspended solids” (p. 45) is correct, it still cannot be assumed that, just because 1,4-dioxane does not partition strongly to organic material, there is no pathway for exposure via sediment or land-applied biosolids. The empirical fact that 1,4-dioxane is present in these environments must be considered.

¹⁰⁶ WATER QUALITY DATA, <https://www.waterqualitydata.us/portal/#sampleMedia=Other&characteristicName=1%2C4-Dioxane&startDateLo=01-01-2009&mimeType=csv> (last visited Aug. 9, 2019).

1) Sediment exposure pathways

Sediment-dwelling organisms often live in or are in contact with the pore water of sediment systems. Due to the fact that some of these organisms exist in the interstitial spaces in sediment and sand, they are even termed “interstitial fauna,”¹⁰⁷ and pore water can be a key route of exposure to these organisms.¹⁰⁸ In fact, partitioning from pore water to sediment has been shown to decrease the bioavailability of certain contaminants of concern to benthic organisms—meaning, the higher the concentration of the contaminant in the pore water, the more likely it is to cause toxicological effects.¹⁰⁹ Therefore, EPA cannot ignore an exposure pathway for sediment-dwelling organisms.

2) Land application of biosolids

EPA’s conclusion in the draft risk evaluation that exposures to 1,4-dioxane from biosolids are negligible is faulty for two reasons: (i) It is difficult to assess the environmental impact from land-applied biosolids without a mass balance; and (ii) EPA’s assumption that land-applied biosolids are only generated through wastewater treatment plants (WWTP) is incorrect.

- a) It is difficult to assess the environmental impact from land-applied biosolids without accounting for what is in the wastewater.

In its cursory analysis of exposure via biosolids, EPA asserted that “the exposures to surface water from biosolids are estimated to be low” (p. 131, 212). EPA also assumed, with no explanation, that 1,4-dioxane “is not likely to accumulate in wastewater biosolids ***” (p. 45). According to EPA, biosolids from WWTPs are between 70-95% water, which EPA assumes has the same concentration of 1,4-dioxane as is in the wastewater. This concentration can potentially be significant, as industrial wastewater can be directly or indirectly discharged into sewers or WWTPs.¹¹⁰ While water associated with biosolids only represents 2% of the total wastewater treated by the facility, some WWTPs in the United States treat hundreds of millions of gallons of

¹⁰⁷ Tom M. Fenchel, *The Ecology of Micro-and Meiobenthos*, 9 ANNUAL REVIEW OF ECOLOGY & SYSTEMATICS 99–121 (1978),

<https://www.annualreviews.org/doi/abs/10.1146/annurev.es.09.110178.000531>.

¹⁰⁸ See, e.g., Peter M. Chapman, et al., *Pore water testing and analysis: the good, the bad, and the ugly*, 44(5) MARINE POLLUTION BULLETIN 359–366 (2002), [10.1016/S0025-326X\(01\)00243-0](https://doi.org/10.1016/S0025-326X(01)00243-0).

¹⁰⁹ Martin T.K. Tsui & L.M. Chu, *Comparative Toxicity of Glyphosate-Based Herbicides: Aqueous and Sediment Porewater Exposures*, 46:3 ARCHIVES ENVTL. CONTAMINATION & TOXICOL. 316-23 (2004), <http://link.springer.com/10.1007/s00244-003-2307-3>.

¹¹⁰ Amie C. McElroy, et al., *1,4-Dioxane in drinking water: emerging for 40 years and still unregulated*, 7 CURRENT OPINION IN ENVTL. SCIENCE & HEALTH 117–125 (2019), <https://www.sciencedirect.com/science/article/pii/S2468584418300485?via%3Dihub>.

wastewater daily.¹¹¹ While 2% may seem insignificant, 2% of say, 300 million gallons, is 6 million gallons of water potentially associated with land-applied biosolids generated daily. Furthermore, this biosolids-associated water is applied directly to land where it will likely migrate either to surface water via runoff or infiltrate—largely undiluted—to groundwater. To accurately assess impacts to the environment from land-applied biosolids, a total accounting of the 1,4-dioxane in the biosolids should be developed, not simply dismissed completely because it represents a small portion of potentially larger whole.

- b) EPA's assumption that land-applied biosolids are only generated through WWTP facilities is incorrect.

Biosolids or sludge generated from manufacturing facilities have the potential to have extremely high concentrations of 1,4-dioxane, due to the high concentration in the associated wastewater stream. Publicly available data demonstrate 1,4-dioxane is present in biosolids, and that the levels are not low. An analysis conducted by Policy Watch found that 1,4-dioxane was present in sludge from a manufacturing facility in Fayetteville, NC at a concentration of 20,400 ug/kg.¹¹² In a follow-up analysis by the North Carolina Department of Environmental Quality, sludge samples contained levels of 1,4-dioxane as high as 138,000 ug/kg.¹¹³ Both analyses were conducted on samples from a facility that manufactures plastics, a condition of use of 1,4-dioxane (p. 30). EPA cannot legally ignore this reasonably available information establishing that exposures from biosolids can be significant.

This sludge can be treated prior to land application, but dewatering generally only removes approximately 30% of associated water (problem formulation, p. 42). Given the recalcitrance of 1,4-dioxane to conventional wastewater treatment and biodegradation (p. 45), it must be assumed that any 1,4-dioxane in the aqueous phase associated with sludge or biosolids will either volatilize, or more likely—due to predicted low volatility (p. 45)—remain in the wastewater, whether the effluent or the pore water accompanying biosolids.

Sludge, or biosolids, associated with disposed waste or wastewater treatment facilities other than WWTPs, which have been shown to be a significant source of 1,4-dioxane, must also be included in EPA's analysis of releases to land.

¹¹¹ <https://empoweringpumps.com/a-look-at-the-largest-wastewater-treatment-plant-worldwide/>

¹¹² Lisa Sorg, *PW special report: Unregulated, untested and unknown*, POLICY WATCH (Apr. 26, 2019), <http://www.ncpolicywatch.com/2019/04/25/pw-special-report-unregulated-untested-and-unknown/>.

¹¹³ Letter from Taylor Cannon, GEL Laboratories, LLC, to Mark Brantley, NC Dept Environmental Quality (Mar. 21, 2019) (providing the analytical results for samples taken from DAK Americas) (copy of the letter is with EDF).

EPA's dismissal of exposures to 1,4-dioxane in biosolids is especially alarming in light of the findings in a recent Office of Inspector General report that indicates EPA "lacks the data or risk assessment tools" to make determinations on the risk levels for pollutants found in biosolids.¹¹⁴ Moreover, according to OIG, "[t]he regulations for biosolids do not require the EPA to obtain the data necessary to complete risk assessments."¹¹⁵ With little known about the pollutants in biosolids, how is EPA's statement that "1,4-dioxane is not likely to accumulate in wastewater biosolids" supported by the best available science?

ii. Misuse of TRI data

To conduct its analysis of aquatic water pathways, EPA relied on the 2015 Toxics Release Inventory (TRI) value for releases to water, which it asserted amounted to 35,402 lbs (pp. 46, 214). There are a number of flaws in EPA's approach.

First, EPA's use of the value reported in 2015 of 35,402 lbs as the amount of releases to water ignores indirect discharges of 1,4-dioxane to water. Total water releases in 2015, according to TRI, amounted to 56,935 lbs. According to EPA's Enforcement and Compliance History Online (ECHO) portal,¹¹⁶ discharges to sewage treatment plants amounted to 24,815 lbs, which is the difference between the actual TRI value and that cited by EPA in the draft risk evaluation. EPA has provided no explanation for its decision to ignore the discharges to sewage treatment plants. It may be that EPA's decision was based on its expectation that such discharges include 1,4-dioxane that was present as a byproduct (e.g., household discharges containing the chemical from the use of cleaning products, etc.), which it has decided to exclude from this risk evaluation. (see Part I sec. 2.A. of these comments for a discussion of the serious concerns that exclusion raises.) However, excluding all indirect discharges would also exclude *industrial* discharges to sewage treatment plants that contain 1,4-dioxane that had been intentionally produced. As EPA has acknowledged, sewage treatment results in only low rates of removal of 1,4-dioxane (p. 45).

Second, EPA relied on outdated TRI data, choosing to use data from 2015 even though data from 2016 and 2017 are readily available. EPA provided a cursory explanation for why it rejected use of the more recent updated data: "[i]t is not expected that the incorporation of the more recent TRI reporting years would have altered the conclusions of the screening-level assessment" (p. 213). However, the releases to water of 1,4-dioxane reported in 2015 (56,935 lbs) are

¹¹⁴ U.S. OIG, *EPA Unable to Assess the Impact of Hundreds of Unregulated Pollutants in Land-Applied Biosolids on Human Health & the Environment* (2018), https://www.epa.gov/sites/production/files/2018-11/documents/epa_oig_20181115-19-p-0002.pdf.

¹¹⁵ *Id.*

¹¹⁶ See WATER POLLUTION SEARCH, <https://echo.epa.gov/trends/loading-tool/water-pollution-search/> (last visited Jul. 19, 2019).

significantly lower than the 2016 and 2017 reported releases, which are 222,991 lbs and 236,508 lbs respectively. As a result, EPA's analysis seriously underestimates the impacts from water releases of this chemical. As recent literature on 1,4-dioxane has indicated, not all releases of 1,4-dioxane are captured by TRI.¹¹⁷

EPA's use of outdated environmental data is contrary to TSCA's mandates to take into consideration all reasonably available information, "including exposure information," (TSCA section 26(k)) and to use the best available science (TSCA section 26(h)).

iii. Failure to consider air and land releases reported under TRI and NEI

EPA has also ignored the impact on the environment of air and land releases of 1,4-dioxane. These releases are substantial. In 2015, companies reported discharging 62,596 pounds to air and 577,400 pounds to land of 1,4-dioxane under the TRI.

While EPA included the 2015 TRI data in a table in Appendix E, (p. 214), it conducted no evaluation of these environmental exposures in this risk evaluation, effectively treating them as equal to zero.

EPA also failed to cite and evaluate the air emission values reported for 1,4-dioxane through the National Emissions Inventory (NEI), which are much higher than those reported under the TRI: 134,484 lbs.¹¹⁸ EPA's ignoring of this reasonably available information violates TSCA § 26(k) and the requirement that EPA consider the best available science under TSCA § 26(h).

iv. Failure to consider data from the Third Unregulated Contaminant Monitoring Rule

EPA has also failed to consider data from the Third Unregulated Contaminant Monitoring Rule ("UCMR3") (see Appendix IV). The UCMR3 includes comprehensive monitoring data for 1,4-dioxane conducted from 2013 to 2015.¹¹⁹ These data have been utilized frequently in scholarly literature to characterize 1,4-dioxane's presence in the environment.¹²⁰

¹¹⁷ Amie C. McElroy, et al., *1,4-Dioxane in drinking water: emerging for 40 years and still unregulated*, 7 ENVTL. SCIENCE & HEALTH p. 118 (2019), <https://doi.org/10.1016/j.coesh.2019.01.003> (noting that "important 1,4-dioxane releases that impacted North Carolina surface water [] were not captured by the TRI.").

¹¹⁸ 2014 NATIONAL EMISSIONS INVENTORY (NEI) DATA, <https://www.epa.gov/air-emissions-inventories/2014-national-emissions-inventory-nei-data> (last visited Jul. 18, 2019).

¹¹⁹ THIRD UNREGULATED CONTAMINANT MONITORING RULE, <https://www.epa.gov/dwucmr/third-unregulated-contaminant-monitoring-rule> (last visited Aug. 6, 2019).

¹²⁰ See, e.g., Krystal J. Godri Pollitt, et al., *1,4-Dioxane as an emerging water contaminant: State of the science and evaluation of research needs*, 690 SCIENCE OF THE TOTAL ENV'T 853-66 (2019), <https://www.sciencedirect.com/science/article/pii/S0048969719330165>; David

EPA must conduct risk evaluations under TSCA with consideration of all “reasonably available” information relating to a chemical substance. 15 U.S.C. § 2625(k). EPA’s rules further define “reasonably available information” as “information that EPA possesses or can reasonably generate, obtain and synthesize for use ***.” 40 C.F.R. § 702.3, 702.33. The UCMR3 data squarely fit within this definition, yet EPA has entirely failed to use it, or even reference it.¹²¹ 15 U.S.C. § 2625(k). The UCMR3 data were published on EPA’s website on January 2017; well before the scoping document for 1,4-dioxane was even released. EPA has possessed these data, in their final form, for over *two years* and there is no logical rationale for ignoring it. EPA’s decision not to use it is arbitrary and capricious.

According to comments made by EPA staff members at the SACC meeting, EPA argued that it cannot use the UCMR3 data because they cannot be attributed back to a particular source. This rationale provides no basis for ignoring these data. Nothing in TSCA allows EPA to ignore data simply because they have not been tied to a particular condition of use. To the contrary, TSCA instructs EPA to consider *all* reasonably available information, which is in direct conflict with what EPA has done in this case.

Additionally, EPA has not shown that the UCMR3 data do *not* relate to conditions of use. It is entirely likely that at least some of the data are related to the conditions of use EPA has identified in the draft risk evaluation. EPA has not attempted to explain why it believes the data are irrelevant to the conditions of use it has chosen to analyze. If EPA continues to ignore these data, at the very least it needs to provide this explanation in the final risk evaluation.

To the extent some of the data from the UCMR3 are unrelated to the limited conditions of use and exposure sources EPA chose to include in the risk evaluation, and which exclude numerous known sources of the chemical, that is a problem of EPA’s own creation and simply one more indication why EPA’s claimed discretion to apply such exclusions is contrary to TSCA. As discussed in Part II sec. 1, EPA must determine whether the chemical substance as a whole presents an unreasonable risk; therefore, reasonably available environmental monitoring data such as the UCMR3 data are directly relevant to whether *1,4-dioxane* presents an unreasonable risk.

Adamson, et al., *1,4-Dioxane drinking water occurrence data from the third unregulated contaminant monitoring rule*, SCIENCE OF THE TOTAL ENV’T 236-245 (2017), <https://www.sciencedirect.com/science/article/pii/S0048969717309221?via%3DIhub>.

¹²¹ EPA acknowledges the existence of the UCMR3 data in a table at the end of the draft risk evaluation that identifies the statutory authorities EPA has relied on to ignore all air, water, and land releases. See p. 193. A mere citation in an appendix does not constitute taking into consideration reasonably available information.

EPA's decision to exclude certain conditions of use and exposure sources does not validate its further decision to ignore directly relevant data. Even assuming EPA had the authority to apply such exclusions, the UCMR3 data constitute relevant exposure data that EPA must use to conduct a risk evaluation. If EPA were to determine that 1,4-dioxane poses an unreasonable risk based, in part, on the UCMR3 data, to the extent the source of that risk is a factor, that would only potentially be relevant at the risk management stage. In fact, section 6(a) specifically provides that EPA may find that risk results from any conditions of use or "any combination of such activities," 15 U.S.C. § 2605(a), and authorizing EPA to regulate risks even when they flow from conditions of use in "any" combination. EPA must apply regulatory "requirements to such a substance or mixture to the extent necessary so that the chemical substance no longer presents such risk." 15 U.S.C. § 2605(a) (emphasis added).

To conclude, the UCMR3 data are reasonably available information that EPA was required to consider in conducting the risk evaluation for 1,4-dioxane, and there is nothing in the statute that suggests EPA can ignore these data.

v. Failure to consider 1,4-dioxane's presence in groundwater

EPA has entirely ignored the presence of 1,4-dioxane in groundwater, despite reasonably available information that reveals numerous sites exist where this chemical is known to be present in groundwater,¹²² leading to greater potential exposures for the subpopulations living in proximity to these sites. By remaining entirely silent on the potential exposures to 1,4-dioxane from contaminated groundwater, EPA's draft risk evaluation is arbitrary and capricious. *See Ctr. for Biological Diversity v. United States BLM*, 698 F.3d 1101, 1124 (9th Cir. 2012) (concluding that it was arbitrary and capricious to entirely ignore the potential impact of groundwater withdrawals to a listed species). EPA also violates its duty to consider the subpopulations potentially suffering greater exposure from these sites.

1,4-dioxane has been identified as a pollutant at 37 Superfund sites.¹²³ However, it is important to note that this is likely a significant underestimation of the number of sites contaminated with 1,4-dioxane. First, according to numerous sources, "large groundwater contaminant plumes of

¹²² See, e.g., U.S. EPA, *Technical Fact Sheet – 1,4-Dioxane* (Jan. 2017), https://www.epa.gov/sites/production/files/2014-03/documents/ffro_factsheet_contaminant_14-dioxane_january2014_final.pdf (1,4-dioxane "has been found in groundwater at sites throughout the United States.").

¹²³ See Appendix V (spreadsheet developed from the National Institute of Health's Toxmap, which is available at <https://toxmap.nlm.nih.gov/toxmap/app/>); see also ATSDR, 2017 Substance Priority List, https://www.atsdr.cdc.gov/spl/resources/ATSDR_2017_Full_SPL_Spreadsheet.xlsx (indicating that there are 33 Superfund Sites, see tab titled "SPL data" and column titled "site frequency" for 1,4-dioxane frequency).

dioxane often form, usually *co-occurring with chlorinated-constituent plumes ***.*¹²⁴ In particular, 1,4-dioxane “is frequently present at sites where chlorinated solvents are detected primarily because of its widespread use as a stabilizer in 1,1,1-trichloroethane (TCA) formulations.”¹²⁵ However, 1,4-dioxane is not only found in groundwater as a co-contaminant with TCA. 1,4-dioxane “was not exclusively used to stabilize TCA (according to the U.S. Patent Literature) and may have been used to stabilize some formulations of other chlorinated solvents,” including trichloroethylene (TCE).¹²⁶

“[D]ioxane has yet to be identified at a large number of [Superfund] sites where it is likely to be present based on the presence of co-occurring chlorinated solvents.”¹²⁷ Based on data from ToxMap, 1,4-dioxane could be present as a co-contaminant at 482 contaminated sites that contain TCA,¹²⁸ and 412 contaminated sites containing TCE.¹²⁹ These site counts may be a more accurate representation of the number of sites contaminated with 1,4-dioxane than the number of identified Superfund sites.

¹²⁴ Ni Yan, et al., *Influence of Groundwater Constituents on 1,4-Dioxane Degradation by a Binary Oxidant System*, 227 WATER, AIR, & SOIL POLLUTION 436 (2016), <https://doi.org/10.1007/s11270-016-3146-y> (emphasis added); see also David T. Adamson, et al., *A multi-site survey to identify the scale of the 1,4-dioxane problem at contaminated groundwater sites*, 1(5) ENVTL. SCIENCE & TECHNOLOGY LETTERS 254–58 (2014), <https://pubs.acs.org/doi/10.1021/ez500092u>; Richard Anderson, et al., *Co-occurrence of 1,4-dioxane with trichloroethylene in chlorinated solvent groundwater plumes at US Air Force installations: fact or fiction*, 8(4) INTEGRATED ENVTL. ASSESSMENT & MGMT. 731–37 (2012), <https://www.ncbi.nlm.nih.gov/pubmed/22492728>.

¹²⁵ David T. Adamson, et al., *Evidence of 1, 4-dioxane attenuation at groundwater sites contaminated with chlorinated solvents and 1, 4-dioxane*, 46(11) ENVTL. SCIENCE & TECHNOLOGY, 6510–18 (2015), <https://pubs.acs.org/doi/pdf/10.1021/acs.est.5b00964?rand=nz7j4029>.

¹²⁶ *Id.*

¹²⁷ David T. Adamson, et al., *A multi-site survey to identify the scale of the 1,4-dioxane problem at contaminated groundwater sites*, 1(5) ENVTL. SCIENCE & TECHNOLOGY LETTERS 257 (2014), <https://pubs.acs.org/doi/10.1021/ez500092u>.

¹²⁸ TOXMAP, <https://toxmap.nlm.nih.gov/toxmap/app/> (last visited Aug. 9, 2019); see also ATSDR, 2017 Substance Priority List, https://www.atsdr.cdc.gov/spl/resources/ATSDR_2017_Full_SPL_Spreadsheet.xlsx (indicating that there are 792 Superfund Sites, see tab titled “SPL data” and column titled “site frequency” for TCA frequency).

¹²⁹ TOXMAP, <https://toxmap.nlm.nih.gov/toxmap/app/> (last visited Aug. 9, 2019); see also ATSDR, 2017 Substance Priority List, https://www.atsdr.cdc.gov/spl/resources/ATSDR_2017_Full_SPL_Spreadsheet.xlsx (indicating that there are 1,051 Superfund Sites, see tab titled “SPL data” and column titled “site frequency” for TCE frequency).

Additionally, 1,4-dioxane was historically not reported at Superfund sites because of a lack of adequate methodology for detecting and quantifying it.¹³⁰ EPA itself has said that it has not always looked for 1,4-dioxane in groundwater because the analytical methods were inadequate.¹³¹ Occasionally EPA has gone back and discovered 1,4-dioxane, but it is unclear how frequently this is done or the extent to which doing so is required.¹³²

EPA's sole reference in the draft risk evaluation to 1,4-dioxane's presence in groundwater states that:

[w]hile 1,4-dioxane is present in various environmental media such as *groundwater*, surface water, and air, EPA determined during problem formulation that no further analysis beyond what was presented in the problem formulation document would be done for those environmental exposure pathways in this draft risk evaluation. (p. 19, emphasis added)

In fact, the problem formulation did not actually analyze 1,4-dioxane in groundwater. *See* Problem Formulation, pp. 42-45. Although EPA very briefly states its assumption that *future* disposal of 1,4-dioxane will be addressed by the Resource Conservation and Recovery Act (RCRA), RCRA does not address the *ongoing* presence of 1,4-dioxane in groundwater.

vi. Failure to analyze exposures during distribution

EPA has conducted no analysis of releases or exposures occurring during distribution of 1,4-dioxane or products containing it, based on the unsupported assertion that “chemicals are packaged in closed-system containers during distribution in commerce and no exposures are expected” (pp. 28, 165).

¹³⁰ *See, e.g.* THOMAS K.G. MOHR, ENVIRONMENTAL INVESTIGATION AND REMEDIATION: 1,4-DIOXANE AND OTHER SOLVENT STABILIZERS (2016) (“Because analytical methods for 1,4-dioxane were not widely available before the late 1990s, many solvent contamination site investigations did not include testing for 1,4-dioxane.”).

¹³¹ U.S. EPA, Office of Solid Waste and Emergency Response, *Treatment Technologies for 1,4-Dioxane: Fundamentals and Field Applications* at p. ES-1 (Dec. 2006), <https://clu-in.org/download/remed/542r06009.pdf>. (“Groundwater investigations at solvent release sites have not typically included 1,4-dioxane as a target analyte because it was not detectable at low concentrations in a standard laboratory scan for volatile organic compounds.”)

¹³² *See, e.g.*, U.S. EPA, *First five-year review report for Tucson international airport area superfund site pima county, Arizona* at p. iv (2013), <https://semspub.epa.gov/work/HQ/181023.pdf> (“The Air Force modified their 1985 remedy with an Explanation of Significant Difference to address 1,4-dioxane and update the target cleanup levels but this is not included for review as there are no EPA decision documents associated with this contaminant.”).

In the problem formulation, EPA took a similar approach, stating: “During distribution, 1,4-dioxane is contained in closed systems (e.g. drums, pails, bottles) so *releases and exposures are not expected*” (p. 37, emphasis added). This blanket assertion too was made with absolutely no supporting analysis or data, either documenting the extent to which the identified “closed systems” are actually used, or the extent to which they are in fact “closed” and lead to no releases or exposures whatsoever, as EPA asserts. Even on their face, the examples raise many questions. For example: Are drums or bottles never open? How is a pail a “closed system”?

To the extent EPA relies on Department of Transportation (DOT) regulations to avoid analyzing exposures to 1,4-dioxane during distribution, any assumption that risks from those exposures are “adequately managed” is unfounded. While EPA refers to the DOT regulations at 49 C.F.R. § 171-177, EPA has made no attempt to explain or apparently even to discern what types of risks those regulations are intended to address, e.g., acute risks from emergency spills or risks from more routine, long-term exposures of workers engaged in distribution-related activities such as loading, unloading.

Those regulations were adopted pursuant to a mandate in the Hazardous Materials Transportation Act (HMTA) that DOT “prescribe regulations for the *safe* transportation, including security, of hazardous material in intrastate, interstate, and foreign commerce.” 49 U.S.C. § 5103(b)(1) (emphasis added). What it means to provide “safe transportation” is not defined in the statute, nor in the rules adopted by DOT. EPA has made no effort to demonstrate whether and if so, how, it has determined the regulations are protecting workers from unreasonable risk during distribution. Notably, the Material Transportation Bureau (MTB), in adopting the regulations, stated that the rules were adopted “primarily to ensure that hazardous wastes are properly identified to carriers and that they are delivered to predetermined designated facilities.” 45 Fed. Reg. 34,560, 34,569 (May 22, 1980) (emphasis added).

vii. Reliance on qualitative and screening-level environmental assessments

EPA has acknowledged that its evaluation of environmental exposures and risks is based only on a “qualitative assessment of the physical-chemical properties and fate of 1,4-dioxane in the environment for sediment and land-applied biosolids” (p. 20) and a screening-level assessment of risks to aquatic organisms (p. 148). These are terms EPA has developed to seek to justify conducting assessments in the absence of sufficient information on hazards, exposures and risks of 1,4-dioxane. These terms have no basis in TSCA itself, which requires EPA to conduct robust risk evaluations of chemicals that are based on the “best available science” (TSCA section 26(h)) and all “reasonably available information” on hazards and exposures (TSCA section 26(k)), the latter defined by EPA in its Risk Evaluation Rule as encompassing “information that EPA possesses or can reasonably generate, obtain, and synthesize for use in risk evaluations.”¹³³

¹³³ 40 C.F.R. § 702.33.

B. Human health

- i. *EPA fails to include all necessary uncertainty factors in calculating the benchmark margins of exposure, resulting in inaccurate risk characterizations*

In chemical risk assessment, uncertainty factors are default values applied in the characterization of chemical hazard and risk to account for uncertainties in the evidence base (e.g., using animal data to characterize effects in humans) and expected variability in the population (e.g., differences in underlying health conditions). As described in the National Academy of Sciences report, *Science and Decisions: Advancing Risk Assessment*:

[Uncertainty] factors are used to adjust for differences in individual human sensitivities, for humans' generally greater sensitivity than test animals' on a milligrams-per-kilogram basis, for the fact that chemicals typically induce harm at lower doses with longer exposures, and so on. At times, the factors have been termed safety factors, which is especially problematic given that they cover variability and uncertainty and are not meant as a guarantee of safety.¹³⁴

EPA fails to include necessary uncertainty factors in its calculations of benchmark margins of exposure (BMOE) for risks to workers of non-cancer effects from inhalation and dermal exposure. The BMOE that EPA derives is 30, resulting from the multiplication of two uncertainty factors—3 for interspecies variation (UF_A) and 10 for intraspecies variation (UF_H). However, at a minimum, EPA should have included an additional uncertainty factor for “the uncertainty associated with extrapolation from animal data when the database is incomplete.”^{135,136}

With regard to the database uncertainty factor, the EPA Risk Assessment Forum notes in its 2002 report, *A Review of the Reference Dose and Reference Concentration Processes*:

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,

¹³⁴ NAT'L RESEARCH COUNCIL, *SCIENCE AND DECISIONS: ADVANCING RISK ASSESSMENT* at chp. 5, p. 132 (2009), <https://www.ncbi.nlm.nih.gov/pubmed/25009905>.

¹³⁵ U.S. EPA Risk Assessment Forum, *A Review Of The Reference Dose And Reference Concentration Processes* p. 4-38 (Dec. 2002), <https://www.epa.gov/sites/production/files/2014-12/documents/rfd-final.pdf>.

¹³⁶ REFERENCE DOSE (RFD): DESCRIPTION AND USE IN HEALTH RISK ASSESSMENTS, <https://www.epa.gov/iris/reference-dose-rfd-description-and-use-health-risk-assessments#1.4> (last visited Jan. 14, 2019).

review of existing data may also suggest that a 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.¹³⁷

In addition, EPA's reliance on oral-to-dermal extrapolation for sub-chronic/chronic effects – necessitated by the total absence of dermal toxicity data – also introduces uncertainty that EPA has failed to account for.

The characterization of 1,4-dioxane's human health toxicity is clearly incomplete as discussed earlier extensively in Part I sec. 4.B.vii and viii. EPA has no dermal toxicity data at all and only a single short-term developmental toxicity study; hence, the Agency lacks any sub-chronic or chronic reproductive, developmental or neurotoxicity data. Thus, it is imperative that EPA apply an additional uncertainty factor of 10 to account for these data gaps.

EPA acknowledges some sources of uncertainty arising from its forced reliance on oral-to-dermal extrapolation for sub-chronic/chronic effects (p. 90):

No repeated-dose dermal toxicity studies were identified on 1,4-dioxane. However, the available data suggest that delivery of 1,4-dioxane via the inhalation- (i.e., pulmonary/systemic circulation) and oral- (i.e., portal circulation) routes of exposure results in comparable toxic endpoints. Since dermally absorbed compounds enter the systemic circulation, route-to-route extrapolations would generally be performed using the repeated-dose inhalation toxicity data. However, the inhalation studies were performed by whole body exposure, rather than nose only exposure, which may have led to additional dosing by the oral and dermal routes of exposure, due to deposition on fur and the grooming behavior of rodents. Therefore, EPA considered the oral studies more relevant in terms of actual dose received. The route-to-route extrapolations enabled EPA to estimate applied dermal PODs. It should be noted that EPA was unable to conclude with certainty that comparable toxic endpoints would be associated with the dermal route of exposure, considering the expected quantitative ADME [absorption, distribution, metabolism, and elimination] differences and the absence of an adequate PBPK [physiologically based pharmacokinetic] model. Notwithstanding these uncertainties, EPA considered this approach appropriate considering the comparable toxic endpoints identified in the available repeated-dose oral/inhalation toxicity studies and the uncertainty with the putative toxicant (i.e., 1,4-dioxane or a metabolite(s)).

¹³⁷ U.S. EPA Risk Assessment Forum, *A Review Of The Reference Dose And Reference Concentration Processes* p. 4-44 (Dec. 2002), <https://www.epa.gov/sites/production/files/2014-12/documents/rfd-final.pdf>.

Numerous sources of uncertainty are apparent in this description of the basis for EPA's decision to rely on oral-to-dermal extrapolation, all of which contribute substantial uncertainty to its risk calculations. Therefore, as is recommended for route-to-route extrapolation generally¹³⁸ and oral-to-dermal extrapolation specifically,¹³⁹ EPA should apply an additional uncertainty factor of 10 to account for these uncertainties.

Application of just one of these aforementioned uncertainty factors dramatically alters EPA's risk characterizations and determinations, negating or calling into question most or all of EPA's assertions that 1,4-dioxane does not present unreasonable risks to workers (or occupational non-users) under various conditions of use.

Beyond these uncertainty factors that should unquestionably be applied, there are additional relevant uncertainty factors that could also reasonably be applied, for example to account for EPA's failure to consider the increased susceptibility of certain subpopulations based on factors such as pre-existing health conditions and pregnancy (see Part I sec. 1.A.).

Overall, EPA's failure to adequately address uncertainty in its quantitative risk characterizations and determinations means that it cannot conclude that 1,4-dioxane does not present unreasonable risks under its conditions of use.

ii. EPA needs to analyze those potentially exposed or susceptible subpopulations that face greater risk due to greater exposure.

TSCA § 3(12) states that “the term ‘potentially exposed or susceptible subpopulation’ means a group of individuals within the general population identified by the Administrator who, due to either greater susceptibility *or greater exposure*, may be at greater risk than the general population of adverse health effects from exposure to a chemical substance or mixture, such as infants, children, pregnant women, workers, or the elderly.” 15 U.S.C. § 2602(12). In its draft risk evaluation, EPA erroneously limits its analysis to only half of this definition; EPA discusses whether persons might face greater susceptibility to 1,4-dioxane, but EPA does not consider

¹³⁸ See, e.g., Schröder, K., et al., *Evaluation of route-to-route extrapolation factors based on assessment of repeated dose toxicity studies compiled in the database RepDose®*, 261 TOXICOLOGY LETTERS 32-40 (Nov. 2016), <https://www.ncbi.nlm.nih.gov/pubmed/27553675>; Dankovic, D.A., et al., *The scientific basis of uncertainty factors used in setting occupational exposure limits*, 12 J. OF OCCUPATIONAL & ENVTL. HYGIENE 55-68 (Nov. 2015), <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4643360/>.

¹³⁹ Rennen, MA, et al., *Oral-to-inhalation route extrapolation in occupational health risk assessment: a critical assessment*, 39:1 REG. TOXICOLOGY & PHARMACOLOGY 5-11 (2004), <https://www.ncbi.nlm.nih.gov/pubmed/14746775>.

whether subpopulations may face a greater risk due to greater exposure. EPA must consider and analyze each of these types of subpopulations.

- a. *EPA needs to analyze the potentially exposed or susceptible subpopulations that faces greater exposure due to their proximity to conditions of use.*

EPA must identify those who face greater exposures due to their proximity to conditions of use as a “potentially exposed or susceptible subpopulation” since they are a “group of individuals within the general population identified by the Administrator who, due to *** greater exposure, *may* be at greater risk than the general population of adverse health effects from exposure to a chemical substance or mixture.” 15 U.S.C. § 2602(12) (emphasis added). Notably, in the problem formulation, EPA seemed to acknowledge that it should analyze these vulnerable subpopulations. *See, e.g.*, Problem Formulation for 1,4-Dioxane at p. 32 (“Other groups of individuals within the general population who may experience greater exposures due to their proximity to conditions of use identified in Section 2.2 that result in releases to the environment and subsequent exposures (e.g., individuals who live or work near manufacturing, processing, distribution, use or disposal sites).”).

But in the draft risk evaluation, EPA does not identify these populations as potentially exposed or susceptible subpopulations (pp. 151-152). EPA provides no analysis of whether those living in proximity to the conditions of use are at greater risk due to greater exposure. EPA should analyze these exposures and should analyze these *potentially* exposed subpopulations. EPA’s failure to consider this relevant aspect of the problem is arbitrary and capricious.

In order to accurately assess the exposure of these subpopulations, EPA should analyze the environmental pathways that lead to the exposure of these subpopulation. Thus, EPA should not exclude those pathways for the reasons given above, and in addition, EPA cannot rationally evaluate the greater exposure these subpopulations face without analyzing these pathways. EPA has provided no rationale explaining how it plans to accurately evaluate the risks faced by these subpopulations while ignoring these pathways of exposure. Instead, EPA simply fails to mention these subpopulations entirely, but ignoring these subpopulations violates EPA’s duty to consider potentially exposed or susceptible subpopulations.

As part of this analysis, EPA should identify people living near disposal sites as potentially exposed or susceptible subpopulations. These groups include (but are not limited to) those living near so-called “legacy” disposal sites. To be clear, many disposal sites are associated with activities that reflect ongoing or prospective manufacturing, processing, distribution, or use, so EPA must analyze those disposals and disposal sites even assuming EPA were correct about its asserted authority to ignore so-called legacy uses, associated disposal, and legacy disposal. But EPA should analyze all disposal sites and populations living in proximity to them; the

distinctions EPA has drawn between disposals find no basis in the statute, and as explained below, TSCA expressly requires EPA to consider disposal.

As EDF previously explained in its comments on the scopes, EPA cannot rationally exclude so-called legacy uses and associated disposals. EDF incorporates and reiterates those points here as well.¹⁴⁰ For the same reasons, EPA cannot rationally exclude so-called legacy disposals. Along with other petitioners, EDF has further developed these arguments in Briefs which are attached as Appendices VI and VII. EDF incorporates and reiterates those points here. *See* Appendix VI at 40-51; Appendix VII at 3-14.

In sum, a chemical's conditions of use include "the circumstances" under which the chemical is "known, or reasonably foreseen to be manufactured, processed, distributed in commerce, *used, or disposed of.*" 15 U.S.C. § 2602(4) (emphasis added). Because the definition uses a disjunctive "or" list, each lifecycle stage of a chemical, standing alone, is a condition of use, even if some of the chemical's lifecycle stages have been discontinued. *See, e.g., Horne v. Flores*, 557 U.S. 433, 454 (2009). So-called legacy disposals are "circumstances" under which a chemical is "known *** to be *** disposed of." 15 U.S.C. § 2602(4). As the Senate Report accompanying an early version of the amended TSCA acknowledged, "there may be exposures of concern from substances that are not currently or no longer in commerce, and the section provides EPA authority to prioritize inactive substances that meet certain criteria." S. Rep. No. 114-67, at 11. "Disposal" of a chemical substance (including products containing that substance) is not a one-time occurrence when the substance or product is buried or placed in a landfill or other waste facility, but remains ongoing after the initial act of discard. Moreover, even in its flawed risk evaluation rule, EPA stated that "EPA may consider background exposures from legacy use, associated disposal, and legacy disposal as part of an assessment of aggregate exposure or as a tool to evaluate the risk of exposures resulting from non-legacy uses." 82 Fed. Reg. at 33,730. Thus, even if EPA follows its illegal rule (which it should not—EPA should give full weight to the consideration of the exposures arising from these conditions of use), EPA should consider these exposures in assessing the combined exposure faced by subpopulations near disposal sites.

In addition, EPA should be analyzing communities who live or work near past manufacturing, processing, distribution, or use sites, even if those activities have ceased. The statute does not allow EPA to ignore conditions of use merely because they happened in the past, and in any event, the disposal at these sites remains ongoing at this time.

¹⁴⁰ EDF Comments on Ten Scopes under the Toxic Substances Control Act pp.8-9, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0743-0069>.

- b. EPA should identify people living in proximity to sources of contamination as potentially exposed or susceptible subpopulations.*

EPA should also analyze the potentially exposed or susceptible subpopulation of persons in proximity to contamination not necessarily linked to or able to be attributed to a specific condition of use. For example, for 1,4-dioxane, EPA has identified groundwater and surface water contamination that would lead to potential elevated exposures of people nearby (p. 19). TSCA defines the term “potentially exposed or susceptible subpopulation” to include “a group of individuals within the general population identified by the Administrator who, due to *** *greater exposure*, may be at greater risk than the general population of adverse health effects from exposure to a chemical substance or mixture.” 15 U.S.C. § 2602(12) (emphasis added). Thus, a subpopulation can qualify due solely to “greater exposure” to a chemical substance; the statute includes no text qualifying “greater exposure” requiring that the exposure be linked to a particular condition of use.

Thus, EPA needs to expand its list of subpopulations to include “other groups of individuals within the general population who may experience greater exposures due to their proximity to sources of contamination (e.g., contaminated groundwater) not necessarily linked to or able to be attributed to a specific condition of use.”

Reasonably available information reveals numerous sites where 1,4-dioxane is known to be present and thus where the subpopulations in their proximity may be at greater risk due to greater exposure. This information is presented and discussed in detail in Part I sec. 5.A. of these comments.

- c. Reasonably available information reveals that consumers, adult women who use multiple cosmetics and cleaning products, and workers using products may be at greater risk due to greater exposure.*

EPA also should include consumers and adult women who use multiple cosmetics and cleaning products as potentially exposed or susceptible subpopulations, given EPA’s prior identification of those subpopulations as particularly likely to be exposed; see Part I sec. 2.A. Workers likely to be using industrial or commercial products contaminated with 1,4-dioxane should also be identified.

- iii. Failure to consider combined exposure pathways for workers*

EPA never bothers to add up its calculated risks from the inhalation and dermal exposures it does consider – even though many workers could readily experience exposures by both routes, including over the same time period. EPA has acknowledged that dermal exposure results in systemic distribution of 1,4-dioxane (p. 90) just as do inhalation (and oral) exposures. Thus the

exposures from all contributing exposure routes would determine the effects associated with systemic circulation of 1,4-dioxane.

In fact, EPA never even acknowledges, let alone accounts for, the potential for simultaneous inhalation and dermal exposure. For example, in the context of estimating dermal exposure, the agency states that “only a fraction of 1,4-dioxane that contacts the skin will be absorbed as the chemical readily evaporates from the skin” (p. 75). Despite acknowledging the likelihood of evaporation, which would lead to increased concentration in the air in the immediate vicinity of the dermally exposed worker, EPA never considers the potential risk of combined exposures through both inhalation and dermal routes. This also means EPA ignores the potential for synergistic effects in scenarios with combined inhalation and oral exposures, a finding of Take et al. (2012) that EPA only briefly mentions elsewhere in the draft (p. 84).

Our concern was reinforced during the 1,4-dioxane SACC Meeting. For example, one SACC member recommended that EPA consider total daily intake by combining exposures from inhalation and dermal, as well as oral routes. The SACC member noted that workers often do not wash their hands before eating, leading to hand-to-mouth exposure. Yet EPA has assumed there are *no* oral exposures to workers – let alone combine such exposures with inhalation and dermal exposure (p. 19; also problem formulation, p. 31). As a result, EPA ignores the potential that such exposures, in conjunction with workplace inhalation exposures, could interact synergistically to increase systemic concentrations of 1,4-dioxane, as observed by Take et al. (2012). At a minimum, EDF urges the agency to combine the inhalation and dermal exposures to more accurately assess risk.

Furthermore, as raised by another SACC member, EPA has ignored all non-occupational baseline exposure to workers due to its exclusion of all other exposures via product use as well as environmental releases to air, water, and land. The SACC member argued that the agency at least needs to take into account these baseline exposures for workers, even if the agency does not consider them as arising from conditions of use it has included within the scope of the risk evaluation. In other words, even if the agency does not intend to regulate environmental releases through the air, water, and land (due to the mere existence of other statutes), it cannot ignore these real-world exposures when assessing the risk 1,4-dioxane presents to an individual (a worker, in this case).

iv. Workplace-related exposure scenarios not considered

During the 1,4 dioxane SACC Meeting, there was robust discussion regarding a number of exposure scenarios that the agency failed to consider. Among those discussed include:

- Exposures from spills in the workplace, especially considering inhalation exposure from evaporation;

- “Take home exposures,” whereby the family of a worker, including children, may be exposed via contact with the worker’s contaminated clothing or skin;
- Exposure of maintenance staff, especially those cleaning up spills and leaks; and
- Exposure of workers at small or medium facilities where assumptions of routine PPE use or other protections are even less likely to be valid.

EDF believes that each of these are known or reasonably foreseen conditions of use, and should be evaluated. With regards to the last of these, SACC members expressed concern that even if one assumes that PPE is typically used in larger, industrial facilities, smaller facilities are much less likely to consistently and appropriately require use of protective equipment and to have engineering controls, like closed systems. Workers at smaller facilities where use of PPE can clearly not be assumed should be considered a vulnerable subpopulation. For this subpopulation, EPA must determine risk based on exposures without use of PPE.

v. Unclear and insufficient consideration of risk to ONUs

For most conditions of use (e.g., import and repackaging, printing ink, disposal), EPA does not have any exposure data for occupational non-users (“ONUs”). As a result, it appears that EPA did not assess risk to ONUs at all for these conditions of use (see 5-6, p. 138 and Table 5-8, p. 141). Instead, for these conditions of use, EPA simply (and repeatedly) asserts that ONUs will have lower exposure: “The ONU exposures are anticipated to be lower than worker exposures, since ONUs do not typically directly handle the chemical” (see, e.g., p. 57). However, as pointed out by SACC members, the conclusion of lower exposure is likely only valid if the agency assumes ONUs also wear respirators or other PPE – an assumption a number of SACC members did not believe to be appropriate. Without data demonstrating that ONUs wear PPE, EPA must evaluate exposure in the absence of PPE when evaluating risk to this subpopulation. Furthermore, as described by an occupational exposure expert on the SACC, more information than EPA has provided on the specific workplace setting and surrounding environment is needed to draw any conclusion that ONUs have lower exposure. As noted by another SACC member, this is not a trivial issue given that there are more ONUs than direct users.

However, for one of the three conditions of use where EPA actually considered ONUs (Open System Functional Fluids, Spray Application, and Film Cement), it is difficult to discern whether or not EPA assumed use of respirators. For only this condition of use – Film Cement – EPA does present non-cancer risk estimates for this population with and without respirator use (see Table 5-6, p. 138). Here EPA found that the calculated MOE is below the benchmark MOE in the absence of respirator use (MOE = 17, benchmark MOE = 30). Despite this, EPA makes the false statement: “As shown in Table 5-6, all exposure scenarios for ONUs resulted in calculated MOEs that were greater than the benchmark MOE” (p. 138). For Film Cement, this statement only applies if respirator use is (inappropriately) assumed, yielding a MOE = 167 for a respirator of APF 10.

However, even in this case EPA still asserts there is no unreasonable risk, hand-waving away such a finding in the Risk Determinations section (p. 169) using an entirely different, if still suspect, rationale: Rather than invoke PPE use, EPA cites uncertainties in the underlying data and the “proximity” of the MOE to the benchmark MOE (see Part I sec. 7.D.). If the data EPA is relying on are insufficient, the agency should certainly not be making a “no unreasonable risk determination,” but rather using its authorities to obtain the required data.

In short, it does not appear that EPA conducted a robust analysis of the risk to ONUs.

vi. Dermal risk

For both the oral to dermal and inhalation to dermal extrapolations, EPA relies on the Bronaugh (1982) *in vitro* dermal absorption study to estimate dermal absorption. As highlighted in our comments elsewhere (Part I sec. 1.E.iii. and sec. 8.B.), this study is not publicly available and has not been subject to any quality review. Nevertheless, the Agency uses it in the calculation of applied human equivalent doses (HEDs), which are themselves used as a basis for reducing the interspecies uncertainty value from 10 to 3 (pp. 111, 118).

EPA has paid scant attention to the uncertainties that route-to-route extrapolations introduce. The source EPA cites for its approach to extrapolation (p. 150, citing USEPA 2004) recommends that, at a minimum, a thorough discussion of associated uncertainties be included when such extrapolation is used. As noted below, other authors have argued that an additional uncertainty factor, or an increase in the uncertainty factor for database insufficiencies, may be warranted.¹⁴¹ In this case, EPA has done neither.

a. Oral to dermal extrapolation

EDF has already discussed our serious concerns with EPA’s dismissal of the liver tumors observed in female mice in the key oral cancer study it uses to extrapolate dermal cancer risks (see Part I sec. 1.E.iv.).

EPA relied on oral-to-dermal extrapolation (p. 90) for sub-chronic/chronic non-cancer outcomes, with little acknowledgment of the substantial uncertainties associated with route-to-route extrapolation. The very guidance that EPA cites for its extrapolation protocol explicitly indicates

¹⁴¹ See, e.g., Schröder, K., et al., *Evaluation of route-to-route extrapolation factors based on assessment of repeated dose toxicity studies compiled in the database RepDose®*, 261 TOXICOLOGY LETTERS 32-40 (Nov. 2016), <https://www.ncbi.nlm.nih.gov/pubmed/27553675>; Dankovic, D.A., et al., *The scientific basis of uncertainty factors used in setting occupational exposure limits*, 12 J. OF OCCUPATIONAL & ENVTL. HYGIENE 55-68 (Nov. 2015), <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4643360/>.

the need for a thorough evaluation of uncertainty, including “a qualitative evaluation of key exposure variables and models, and their impact on the outcome.”¹⁴² Yet in this risk evaluation, EPA has provided only a single statement of uncertainty -- “oral to dermal route-to-route extrapolation assumes that the oral route of exposure is most relevant to dermal exposures” (p. 150) – which is far from sufficient. Some prior research even suggests the inclusion of additional uncertainty factors for route-to-route extrapolation may be appropriate.¹⁴³ This issue is discussed further in Pat I sec. 7.E. below.

b. Inhalation to dermal extrapolation

EPA appears to use inappropriate model inputs for the chronic non-cancer assessment for dermal exposures extrapolated from chronic inhalation studies (p. 117): The agency uses an inhalation rate of 1.25 m³/hr for their inhalation to dermal conversion. This does not match with the number in the EPA Exposure Factors Handbook¹⁴⁴ for average adult moderate activity level (Table 6-28 suggests 2.1 m³/hr). EPA should explain the rationale for this deviation.

vii. Inhalation risk

The Agency provides insufficient and/or irrelevant details for the chronic non-cancer inhalation risk estimates, obfuscating the modeling process. For example, in the discussion of Risk Characterization Assumptions and Uncertainties (p.150), EPA states that the “LOAEC was used with an uncertainty factor for LOAEC to NOAEC extrapolation.” However, where and how it did so is not explained clearly in earlier sections of the document (ex: Section 4.2.6.2.3, pp. 111-114), where text and tabular calculations are provided for this outcome. Why EPA included this text about the LOAEC to NOAEC extrapolation within the discussion of Risk Characterization Assumptions and Uncertainties (p. 150) is also unclear, given that EPA ultimately used BMD in these calculations. As such, the Agency should have instead or also provided a discussion of assumptions and uncertainties relevant to BMD.

On page 135, EPA claims that an APF=10 respirator is sufficient to eliminate even high-end inhalation non-cancer risk “during industrial use.” This is not accurate: EPA found that an APF=25 respirator is necessary to get the acute high-end MOE above the benchmark MOE

¹⁴² U.S. EPA, *Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment)* at sec. 5-2 (June 2004), https://www.epa.gov/sites/production/files/2015-09/documents/part_e_final_revision_10-03-07.pdf.

¹⁴³ Schröder, K., et al., *Evaluation of route-to-route extrapolation factors based on assessment of repeated dose toxicity studies compiled in the database RepDose®*, 261 TOXICOLOGY LETTERS 32-40 (Nov. 2016), <https://www.ncbi.nlm.nih.gov/pubmed/27553675>.

¹⁴⁴ U.S. EPA, *Exposure Factors Handbook: 2011 Edition* (2011), https://ofmpub.epa.gov/eims/eimscomm.getfile?p_download_id=522996.

(Table 5-4) and that even an APF=50 respirator is not sufficient to get the chronic high-end MOE above the benchmark MOE (Table 5-5). (The chronic finding is more accurately stated on p. 137.)

For the evaluation of acute/short term inhalation effects, EPA uses a LOAEC of 100 ppm from Mattie et al., 2012 to derive PODs for liver effects (which were assumed to be protective of acute effects to the nasal system, lungs, and brain). While this study is more recent than (and was not yet available) when ATSDR completed its ToxProfile in 2012, the experiments were conducted in rats. By contrast, ATSDR identified a NOAEL of 20 ppm from Ernstgard et al., 2006, which was conducted in humans and evaluated eye and respiratory irritation as well as pulmonary function. Importantly, this study does not require interspecies extrapolation. EPA's decision to utilize the Mattie et al., 2012 study results in a much higher and less protective POD(HEC) of 75 ppm compared to ATSDR's ultimate minimal risk level (MRL) of 2 ppm.

EPA's decision *not* to utilize the Ernstgard et al., 2006 study is not well justified. EPA merely states that "there were limitations with the human studies [ex: Ernstgard et al., 2006] that precluded their use for quantitative risk assessment, including for example, the absence of measures of systemic effects (e.g., serum chemistry panels)" (p. 87). This latter statement is not correct; in fact, Ernstgard et al. assessed inflammatory parameters, which reflect a systemic response, and reported no exposure-related changes at the identified NOAEL.¹⁴⁵ Most importantly, however, even if Ernstgard et al. had not evaluated systemic effects, there is absolutely no scientific basis for exclusion based on this rationale. Indeed, the National Research Council states that "the crucial toxic effect used is the one generally characterized by the lowest NOAEL. This approach is based on the assumption that if the critical toxic effect is prevented, then all toxic effects are prevented."¹⁴⁶ There is no requirement that the NOAEL be based on systemic effects. As such, EPA should use Ernstgard et al. 2006 as the basis for its acute/short term inhalation modeling.

viii. Failure to explain or justify assumption of one exposure event per day

In its dermal exposure assessment, EPA states that the "dose estimates assume one exposure event (applied dose) per work day" (p. 76). EPA provides no justification for this assumption, and the assumption seems unwarranted. What basis does EPA have for assuming that workers will only engage in one action per work day that could result in an exposure? Given the 8-hour work day and the repetition common in many jobs, it seems far more likely that workers would engage in activities that could result in an exposure multiple times a day. At a minimum, EPA

¹⁴⁵ L. Ernstgård, et al., *Acute effects of exposure to vapours of dioxane in humans*, 25:12 HUMAN & EXPERIMENTAL TOXICOLOGY 723-29 (2006), <https://www.ncbi.nlm.nih.gov/pubmed/17286150>.

¹⁴⁶ NAT'L RESEARCH COUNCIL, SCIENCE AND JUDGMENT IN RISK ASSESSMENT (1994), <https://www.nap.edu/catalog/2125/science-and-judgment-in-risk-assessment>.

must present some rationale for its assumption. This assumption seems likely to significantly underestimate the risk faced by workers.

6. Risk characterizations

Despite EPA's indications to the contrary and the numerous data gaps and deficiencies in its analysis identified above, EPA has found numerous, significant risks to workers. Summaries of these findings are provided below.

A. Inhalation risks

Acute (Table 5-4, p. 136):

- For 8 of 11 conditions of use (COUs), high-end MOEs are below EPA's benchmark MOE (300) and respirators are required to get above the benchmark (a respirator with an APF=50 is required for 2 COUs; APF=25 for 1 COU; APF=10 for 5 COUs).
- For 5 of 11 COUs, central tendency MOEs are below the benchmark MOE and respirators are required to get above the benchmark (APF=25 for 2 COUs; APF=10 for 3 COUs).

Chronic non-cancer (Table 5-5, p. 137):

- For 8 of 10 COUs, ***both central tendency and high-end MOEs are below the benchmark MOE (30)***.
 - For 5 of these, even an APF=50 isn't sufficient to get the high-end MOE above the benchmark.
 - For the other 3 COUs, respirators are required to get the high-end MOE above the benchmark (APF=50 for 1 COU; APF=10 for 2 COUs).
- For 1 of these, even an APF=50 isn't sufficient to get the central tendency MOE above the benchmark. For the other 7 COUs, respirators are required to get the central tendency MOE above the benchmark (APF=50 for 1 COU; APF=25 for 1 COU; APF=10 for 5 COUs).

Cancer (Table 5-7, p. 140):

EPA employed several devices to minimize the extent of inhalation cancer risk to worker that it found. First, it made an unsupported assumption of universal, effective use of respirators. See Part I sec. 1.B. and sec. 7.A. Second, it applied a very high cancer risk benchmark of 1 in 10,000 that is virtually unprecedented for EPA when administering its statutes and is wholly at odds with TSCA's mandate to protect workers as a "potentially exposed or susceptible

subpopulation.” See Part I sec. 7.C. Third, it only identified unreasonable risks when its central tendency as well as high-end estimates exceeded the benchmark. See Part I sec. 7.B.

Examining Table 5-7 on page 140, one can see that absent PPE, EPA’s inhalation cancer risk estimates are above its already excessive benchmark of 1 in 10,000 for 7 of its 10 scenarios under high-end exposures and 5 of the 10 scenarios under central tendency exposures. Even the assumed universal use of a high-efficiency respirator was not enough to reduce exposures below that high benchmark for the broad category of Industrial Use, where EPA found a risk of 2 in 10,000 even with a respirator with an APF of 50.

Had EPA used a more protective and typical cancer risk benchmark of 1 in 100,000, the table shows there would be excessive risk in *all ten scenarios for both central tendency and high-end exposures -- even with respirator use*.

Further details:

- *For all 10 COUs, inhalation cancer risk levels for workers are above 1 in 100,000 – even with respirator use – for both central tendency and high-end exposures.* Even for ONUs, the same is true for film cement use (Table 5-8, p. 141).
- For 7 of the 10 COUs, high-end cancer risk levels for workers are above 1 in 10,000.
 - For 1 of these, even an APF=50 isn’t sufficient to get the high-end cancer risk below this risk level.
 - For the other 6 COUs, respirators are necessary to get the high-end cancer risk levels below 1 in 10,000 (APF=50 for 2 COUs; APF=25 for 3 COUs; APF=10 for 1 COU).
- For 5 of 10 COUs, central tendency cancer risk levels are also above 1 in 10,000. For these, respirators are necessary to get the central tendency cancer risks risk levels below 1 in 10,000 (APF=25 for 1 COU; APF=10 for 4 COUs).

B. Dermal risks¹⁴⁷

Acute (Table 5-9, p. 142):

- For 8 of 10 COUs, high-end MOEs are below the benchmark MOE (300) and gloves with a protection factor of 5 (PF=5) are required to get above the benchmark.

¹⁴⁷ The tables presenting the dermal data need revision for accuracy and clarification:

- Table 5-9:
 - Columns showing the results for central tendency scenarios need to be added.
 - The values in the PF=5 column that are <3400 need to be boldfaced
- Tables 5-10 and 5-11: Columns showing the results for central tendency as well as high-end scenarios need to be added.

- For 4 of the 10 COUs, central tendency MOEs are below the benchmark MOE (based on the text just above the table; EPA has not provided the specific data for these exposures or what gloves are necessary to get above the benchmark).

Chronic non-cancer (Table 5-10, p. 144 – EPA doesn’t distinguish between high-end and central tendency exposures):

Examining this table, one can see that the estimated MOE is below the benchmark MOE of 30 in 8 of the 10 scenarios when gloves are *not* assumed to be worn – and in 5 scenarios even when a glove with a protection factor of 5 is assumed to be worn. Only by making the unsupported assumption of universal, effective use of gloves with a high PF was EPA able to claim these dermal risks are above its benchmark MOE.

Further details:

- For 9 of 11 COUs, the MOEs are below the benchmark MOE (30).
 - For 5 of these, even PF=5 gloves aren’t sufficient to get above the benchmark.
 - For the other 4 COUs, PF=5 gloves are required to get above the benchmark.

Cancer (Table 5-11, p. 145 – EPA doesn’t distinguish between high-end and central tendency exposures):

- *For 9 of 11 COUs, dermal cancer risk levels for workers are above 1 in 100,000 – even with PF=20 glove use.*
- *For 9 of 11 COUs, dermal cancer risk levels for workers are above 1 in 10,000 – even with PF=5 glove use.*
 - For 8 of these, PF=10 gloves still leave risk above 1 in 10,000, and
 - For 6 of these, even PF=20 gloves are not sufficient to get risk below 1 in 10,000.

C. Aggregate vs. sentinel exposures

EPA provides no significant discussion of its decision to rely on sentinel exposures instead of aggregate exposures, and EPA provides no support for the decision to consider the highest exposure to be sentinel (vs. the exposure of the longest duration/frequency, etc.) (p. 152). Nor does EPA provide any rationale for how its decision to conduct a sentinel exposure assessment comports with its collapsing of multiple uses and scenarios into single scenarios (see Part I sec. 2.C. of these comments): On what basis did EPA determine that its selected scenario is representative of all of the scenarios that were collapsed into it? And that exposures for that scenario were in fact the most significant? EPA has provided no such analysis in its draft risk evaluation.

In a risk evaluation, EPA must describe whether aggregate or sentinel exposures under the conditions of use were considered “and the basis for their consideration.” 40 C.F.R. § 702.43(a)(2). EPA has violated this duty by failing to articulate the basis for considering sentinel exposure.

In addition, EPA’s risk evaluation rule defines “sentinel exposure” to “mean[] the exposure from a single chemical substance that represents the plausible upper bound of exposure relative to all other exposures within a broad category of similar or related exposures.” 40 C.F.R. § 702.33. But EPA has failed to apply this definition throughout the draft risk evaluation. EPA did not establish that the exposures it analyzed represent the “plausible upper bound of exposure relative to all other exposures” within the relevant categories. This regulatory definition requires that, when EPA prepares a sentinel exposure for workers, EPA must identify or evaluate the worker whose exposure represents the upper bound of exposure. 82 Fed. Reg. 33,3726, 33,733 (July 20, 2017). EPA has not established that, for each category of worker, it actually identified and evaluated the worker whose exposure represents the upper bound of exposure.

7. Flaws in EPA’s unreasonable risk definition and determinations

A. Expectation of compliance with existing laws and standards

In reaching its unreasonable risks determinations, “EPA expects there is compliance with all federal and state laws, such as worker protection standards, unless case-specific facts indicate otherwise” (p. 175, FN 1). EPA goes on to conclude that “therefore existing OSHA regulations for worker protection and hazard communication will result in use of appropriate PPE consistent with the applicable SDSs in a manner adequate to protect workers.” As noted in Part I sec. 1.B. above, EPA mischaracterizes these OSHA regulations, which do *not* in fact require that persons comply with SDSs.

It is wholly inappropriate for EPA to simply assume either that there is universal compliance with laws and recommended standards, or that even when complied with, such requirements eliminate all risk such that EPA can ignore the contribution of such regulated activities to the overall risks posed by 1,4-dioxane. EPA has provided no analysis whatsoever of the degree of compliance with various requirements, including the extent to which they are effectively enforced. It has made no attempt to identify, let alone evaluate, the risks posed by the releases and exposures that continue to occur even in the presence of those requirements, and their contribution to the total exposure and risks. EPA has also failed to acknowledge that the other requirements derive from statutes that establish different criteria for establishing requirements to address human and environmental health risks. Many of these other statutes, for example, require EPA or other agencies to consider factors such as cost and feasibility when setting standards -- factors that TSCA explicitly forbids EPA from taking into account when assessing risks. TSCA section 6(b)(4)(A) states (emphasis added):

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

B. Allowance for exceedances for high-end risks when finding no unreasonable risk

EPA states that its “determination of unreasonable risk is likely to consider the risk estimates associated with the *central tendency exposure scenarios*” (p. 151, emphasis added). EPA also states (p. 152, emphasis added):

Where risks greater than the acceptable benchmarks are identified for high-end exposures, but *not for central tendency exposures*, and where EPA determines that a potentially exposed or susceptible subpopulation is not expected to be affected under the conditions of use, EPA may determine that while some risk exists, the risk is not unreasonable for the occupational conditions of use.

This is not theoretical: EPA has applied this approach to specific risk determinations in this risk evaluation; for examples, see pp. 160, 161.

Among other concerns, EPA’s approach is at odds with its obligation under TSCA to conduct risk evaluations that ensure protection of “potentially exposed or susceptible subpopulations,” which TSCA explicitly defines as including workers. Elsewhere, EPA represents its high-end estimates as “generally intended to cover the *most exposed* individuals or sub-populations,” while its central tendency estimates apply to the “average or typical exposure” that workers experience (p. 153). But TSCA does not allow EPA to protect only the “average or typically exposed” workers; in fact, when it comes to workers EPA is required to protect all of them.

Moreover, EPA stated that it would use sentinel exposure levels which it defines as “the exposure to a single chemical substance that represents the plausible upper bound of exposure relative to all other exposures within a broad category of similar or related exposures” (p. 152). How can EPA justify relying on the central tendency over the high-end exposure scenario when EPA has committed to using the “plausible upper bound of exposure” in its exposure assessments? EPA’s use of the central tendency exposure as determinative of unreasonable risk is inconsistent with EPA’s definition of sentinel exposure in its risk evaluation rule. *See* 40 C.F.R. § 702.33.

C. 1 in 10,000 cancer risk level deemed reasonable for workers

EPA has relied on NIOSH guidance in order to establish 1×10^{-4} as the cancer risk benchmark for workers (pp. 133, 153, 155). EPA cites the *Benzene* decision for support (p. 155, footnote 12), but that case pertained to how the standard for protection applied under OSHA was to be determined, not under TSCA. EPA's decision is wholly at odds with its own acknowledgment two pages earlier that other laws have standards that differ from TSCA's (p. 153, footnote 10).

EPA is required to protect workers, both generally and as a "potentially exposed or susceptible subpopulation," under TSCA, not under OSHA. The 2016 amendments to TSCA strengthened EPA's already-existing mandate to protect workers. TSCA's new definition of "potentially exposed or susceptible subpopulation" has no asterisk next to workers, and there is no basis in TSCA for EPA to provide less protection to workers than any other such subpopulation, let alone than the general population. Yet that is exactly what EPA has done here.

The 2016 amendments to TSCA also explicitly preclude EPA from considering feasibility or other non-risk factors when determining whether a chemical presents an "unreasonable risk," including to workers; see TSCA section 6(b)(4)(A). Yet EPA invokes standards under other statutes that lack this prohibition in an effort to claim precedent for its 1×10^{-4} benchmark (p. 155, footnote 11).

Indeed, EPA's reliance on the *Benzene* decision cannot be reconciled with the statutory differences between OSHA's standard and TSCA's unreasonable risk standard. In the *Benzene* case, the Court interpreted a provision of the OSH Act that defined standards as "reasonably necessary or appropriate to provide safe or healthful employment and places of employment," as requiring OSHA "to make a threshold finding that a place of employment is unsafe—in the sense that *significant* risks are present and can be eliminated or lessened by a change in practices." *Indus. Union Dep't, AFL-CIO v. API*, 448 U.S. 607, 642 (D.C. Cir. 1980) (emphasis added). The Court's interpretation turned on the statutory language of the OSH Act, the Act's structure, and its legislative history. But EPA can point to *no* statutory language in TSCA invoking this standard, EPA has pointed to no similarities between the two statute's structures, nor has EPA pointed to any legislative history suggesting that TSCA adopted the OSH Act's standard. Moreover, if Congress had intended to adopt the *Benzene* standard under TSCA, it would have required that EPA regulate "significant risks," not "unreasonable risks." Indeed, the significant differences between the language and structure of the two statutes strongly indicates that Congress meant to adopt a different standard in TSCA, not the standard articulated by the Court in the *Benzene* case.

Moreover, in implementing TSCA (even before the amendments) and its other environmental statutes, EPA has generally sought to reduce population risks from chemicals in commerce that are carcinogens to below about one case per one million people. See, for example, this EPA

statement from 1989: “EPA believes *** that it should reduce risks to less than 1×10^{-6} for as many exposed people as reasonably possible.” National Emission Standards for Hazardous Air Pollutants; Radionuclides, 54 Fed. Reg. 51,654, 51,686 (Dec. 15, 1989). Nor does EPA only apply this standard under the Clean Air Act. When setting Clean Water Act criteria, “EPA intends to use the 10^{-6} risk level, which the Agency believes reflects an appropriate risk for the general population. EPA’s program office guidance and regulatory actions have evolved in recent years to target a 10^{-6} risk level as an appropriate risk for the general population. EPA has recently reviewed the policies and regulatory language of other Agency mandates (e.g., the Clean Air Act Amendments of 1990, the Food Quality Protection Act) and believes the target of a 10^{-6} risk level is consistent with Agency-wide practice.”¹⁴⁸ When Congress amended TSCA to include the unreasonable risk standard, it did so knowing that agency practice was to regulate cancer risks at the 10^{-6} risk level. It should be presumed that Congress meant to adopt this risk standard when codifying the unreasonable risk standard.

In grasping for support for its approach in this risk evaluation by citing other mentions by EPA of the 1×10^{-4} risk level (p. 155, footnote 11), EPA blurs a critical distinction made when EPA has invoked the less stringent level of protection from cancer risks: the level set to reflect the maximum risk faced by any individual vs. the level set to protect a broader population. EPA invokes the “two-step approach” used under the Clean Air Act, where EPA includes a “limit on *maximum individual* lifetime [cancer] risk (MIR) of approximately 1 in 10 thousand” (p. 155 n. 11, citing 54 Fed. Reg. 38,045 (Sept. 14, 1989)) (emphasis added). But that is entirely different than the level set to protect the vast majority of the population in question.

More specifically, the two-step, risk-based decision framework for the National Emission Standard for Hazardous Air Pollutants (NESHAP) program is described as follows by EPA:

First, the rule sets an upper limit of acceptable risk at about a 1-in-10,000 (or 100-in-1 million) lifetime cancer risk for the *most exposed person*. As the rule explains, “The EPA will generally presume that if the risk to that individual [the Maximum Individual Risk] is no higher than approximately 1 in 10 thousand, that risk level is considered acceptable and EPA then considers the other health and risk factors to complete an overall judgment on acceptability.”

¹⁴⁸ U.S. EPA, Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (2000), p.2 8, <https://www.epa.gov/sites/production/files/2018-10/documents/methodology-wqc-protection-hh-2000.pdf>.

Second, the benzene rule set a target of protecting the *most people possible* to an individual lifetime risk level no higher than about *1-in-1 million*.¹⁴⁹

But in this risk evaluation, EPA has set a risk level for the entire worker population that is the same as the level EPA elsewhere set for the most exposed individual in a population. EPA then erroneously invokes this level repeatedly to find the majority of conditions of use of 1,4-dioxane to pose no risk to *any* workers, thereby subjecting many tens of thousands of workers to cancer risks that are as much as *two orders of magnitude higher* than warranted. This approach must be rejected on scientific as well as legal grounds.

D. Shifting the goalposts when risk values are only a little above acceptable benchmarks

EPA has instituted an approach under which it can still deem a risk to be reasonable even though it exceeds the applicable acceptable level, as long as it is “close” to the acceptable level. Specifically, EPA states that it can consider “the proximity of the calculated risk estimate to the benchmark to determine that this condition of use does not present an unreasonable risk” (p. 169). Indeed, EPA had applied this approach to multiple conditions of use (pp. 159-60, 160-61, 169). In these cases EPA found its estimated MOEs of 25 or 17, well below its benchmark MOE of 30, still do not constitute unreasonable risks because they are in “proximity” to the benchmark.

But EPA applies this in only one direction in the risk evaluation. Even where EPA’s estimated MOEs are only slightly greater than the benchmark MOE, EPA still finds no unreasonable risk. See, for example:

- Table 5-5, rightmost column for Film Cement: calculated MOE of 31 vs benchmark MOE of 30 is deemed not to represent unreasonable risk.
- Table 5-4, rightmost column for Industrial Use: calculated MOE of 338 vs benchmark MOE of 300 is deemed not to represent unreasonable risk.

EPA’s approach to unreasonable risk violates its duties under TSCA. TSCA § 6 requires that EPA “shall conduct risk evaluations pursuant to this paragraph to determine whether a chemical substance presents an unreasonable risk of injury to health or the environment.” 15 U.S.C. § 2605(b)(4)(A). Here, EPA’s analysis of certain conditions of use finds an unreasonable risk with the estimated MOEs falling below the benchmark MOE, and EPA then reverses that finding because the estimated MOEs are in “proximity” to EPA’s risk benchmark. By doing so, EPA

¹⁴⁹ WHAT DOES EPA BELIEVE CONSTITUTES AN ACCEPTABLE LEVEL OF RISK?, <https://www.epa.gov/national-air-toxics-assessment/nata-frequent-questions#risk2> (emphasis added) (last visited Jul. 19, 2019).

adopts a finding on unreasonable risk that runs counter to the evidence before the agency. EPA's own analysis establishes that a risk exists, and EPA has not explained how the MOEs being in "proximity" to the benchmark negates the finding of unreasonable risk. While EPA emphasizes that some uncertainties might overestimate the risk presented by these conditions of use, EPA fails to account for how these or other uncertainties might underestimate the risk. EPA's failure to adopt a final risk determination consistent with its factual findings is arbitrary and capricious.

E. Flaws in dermal exposure analysis and misleading characterizations of EPA's dermal risk analysis in its risk determinations

EPA repeatedly states in the Risk Determination section that the agency's approach to estimating dermal exposures "could overestimate risk." The draft risk evaluation states: "EPA chose to use 3.2%, the higher value, for the dermal absorption factor. The actual absorption could be ten-fold lower based on the Bronaugh *in vitro* study (Bronaugh, 1982). For this pathway, EPA expects that the risks are not underestimated." (For examples of this language, see pp. 158, 163).

i. Insufficiency and mischaracterization of glove modeling scenarios

EPA's *actual* analysis of what absorption values and assumptions were applied to which scenarios is far from clear and at the very least must be far more thoroughly explained. Based on our best effort to discern what EPA did, it appears the description just cited mischaracterizes EPA's actual analysis, which cannot be fairly characterized as an overestimation of exposure, for several reasons:

1. EPA states that it used the higher 3.2% absorption rate *only* in occluded scenarios, where gloves are worn: "[f]or quantifying potential dermal risks to workers, EPA used the measured absorption values of 0.3% for scenarios without gloves and 3.2% for scenarios with gloves to quantify the amount of the applied dermal dose that would be systemically available." (p. 76) This would be appropriate, and necessary, if EPA could reasonably rely on Bronaugh, 1982 (which EPA should not – see Part I sec. 4.B.v.), as Bronaugh found, according to EPA, that "[d]ermal penetration of 1,4-dioxane was 3.2% of the applied dose for the occluded condition, and 0.3% for unoccluded" (p. 83).

However, it is not at all clear that EPA implemented this approach. Its risk values for the scenarios without gloves are reduced exactly by the protection factor (PF) EPA assumed for the three with-gloves scenarios. See Table 5-10 on p. 144 and Table 5-11 on p. 145. This should not be the case if EPA applied different values for skin absorption for the no-gloves and gloves scenarios.

2. If EPA *did* use the lower 0.3% for scenarios where gloves are not worn (which it does not state it did in the Risk Determination sections), then the more conservative and health-protective approach would have been to also use the higher 3.2% absorption for the non-

occluded/no-glove scenario. Support for use of a higher rate is provided by the other study EPA cites, Marzulli et al., 1981, which found a dermal absorption rate of 2-3% in a *non-occluded* scenario (albeit after a 24-hour exposure). While EPA described the study (p. 83), it appears not to have chosen to use its higher absorption rate for non-occluded/no glove scenarios, based on the statement on p. 76 cited above. As a result, EPA's repeated assertion in the Risk Determination sections that its approach was conservative is questionable at best.

3. Elsewhere in the draft, EPA notes some of the numerous ways in which glove use can actually *increase* skin exposure through occlusion (p. 292): “[g]loves can prevent the evaporation of volatile chemicals from the skin. Chemicals trapped in the glove may be broadly distributed over the skin (increasing S in Equation G-13), 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.” One of the SAAC panel members highlighted this issue as well: in the real-world, an untrained user may contaminate a glove, leading to occlusion and higher exposure. Likewise, permeable gloves may enable the chemical to absorb through the glove, while preventing or slowing evaporation. Gloves can also increase skin temperature and humidity, which can increase absorption.

However, it does not appear that EPA's analysis accounted for such factors that could lead to increased skin absorption during use of gloves.

EPA appears to want to have it both ways: To acknowledge the limitations of gloves and their potential to increase skin absorption, but then to simply assume that gloves actually provide 5x, 10x or 20x levels of protection over no gloves without citing any evidence to support these values.

The issue of dermal absorption in the presence of gloves is clearly highly complex. EPA needs to provide a far more thorough analysis, including by expanding its glove and usage scenarios. The Agency should also provide a more detailed explanation than is currently provided. In doing so, it should also account for more recent data, such as Dennerlein et al., 2013 (see Part I sec. 4.B.v in these comments for further detail).

ii. Inaccurate modeling leads to underestimate of exposure

During the July 30th SACC meeting on 1,4-dioxane, one of the SACC panel members, an expert on dermal exposure assessment, expressed major concerns with EPA's dermal exposure estimates and ultimate conclusions. The SACC member suggested that dermal exposures should not be discounted on the basis of questionable, and not well described, experiments and assumptions. Among the issues he flagged are: reliance on Bronaugh, 1982 (see Part I sec. 4.B.v. above); EPA's extrapolation from inhalation to dermal risks without considering flux dynamics that are uniquely applicable to dermal absorption; EPA's assumption that time equals infinity in its dermal modeling (which overestimates evaporation and underestimates absorption);

crucial mistakes in the Agency's dermal dose equation calculations; and EPA's use of fixed glove PFs of 5x, 10x, and 20x.

Here we provide further details on the latter two of these issues:

- a) In the draft risk evaluation, EPA cites the fractional absorption potential for 1,4-dioxane to be 0.86 or 0.78, depending on the setting, based on Kasting and Miller (2006) (p. 76). These values represent the fractions of the chemical on the skin that are potentially systemically absorbed. However, on p. 76 the Agency erroneously claims that these values represent only absorption into the stratum corneum. As a result, EPA then erroneously further adjusts the 0.86 or 0.78 values by the 0.3% or 3.2% values based on Bronaugh, 1982. Through this inappropriate "double" adjustment, the Agency severely underestimates potential dermal absorption – by a factor of at least 31.¹⁵⁰
- b) The SACC member explained that the assumption that PFs can only range from 1x (no gloves) to 20x PF is erroneous, and that the range should include PFs below 1x. As described by the member, glove testing is typically conducted in a lab in ideal conditions – without an actual hand present. However, as noted above, in the real-world, an untrained user may contaminate a glove, leading to occlusion and higher exposure. Likewise, permeable gloves may enable the chemical to absorb through the glove, while preventing or slowing evaporation. Though EPA acknowledged these factors (as described in #3 above) it failed to incorporate them into its analysis when it assumed only positive PF values for gloves.

In the Risk Determination section, EPA ultimately only cites the risk estimates for the 20x PFs, ignoring the higher risks found with no gloves or lower PFs. The unstated but highly questionable premise seems to be that if the most protective gloves potentially available can reduce risk to below the benchmark, then there is no unreasonable risk. This approach will allow clear risks to occur whenever a worker uses anything less than the most protective gloves (or no gloves), a scenario quite likely – and certainly reasonably foreseen – to occur in the real world.

In sum, it is far from clear that EPA has, as it claims, overestimated risk from dermal exposure. If anything, it seems likely that the agency underestimated risk.

¹⁵⁰ We derived this value by multiplying the 0.86 fractional absorption value by 3.2% to derive the "double" adjusted value (i.e., $0.86 \times 0.032 = 0.0275$). The ratio of the unadjusted value of 0.86 to EPA's "double" adjusted value of 0.0275 is 31 (i.e., $0.86 / 0.0275 = 31.27$).

8. Systematic review issues

EDF has previously provided extensive comments on EPA's systematic review approach.¹⁵¹ We incorporate those comments by reference.

A. OPPT does not provide explanation nor empirical support for its revisions to the systematic review data quality criteria for epidemiological studies, and certain revisions make it more difficult for epidemiological studies to be scored overall as high quality.

- OPPT released an updated version of its systematic review data quality criteria for epidemiological studies, but did not provide any explanation for the numerous changes it made to these criteria. OPPT's scoring methodology is already at odds with best practices in systematic review (see Section 4 of EDF's previous comments on this issue),¹⁵² and the agency's decision to alter scoring criteria without providing any empirical rationale for the changes further underscores that the study quality evaluation strategy that OPPT developed is not evidence-based.
- At least six metrics in OPPT's updated epidemiological criteria can no longer receive a score of High. These changes preclude epidemiological studies from receiving High scores for all study metrics—this was previously possible. Notably, these types of revisions to the epi criteria—prohibiting a score of high for certain data quality metrics—did not occur for animal or in vitro studies where it remains possible to score High across every data quality metric. The effect is to diminish the contribution of epidemiological evidence relative to animal and in vitro studies.
- There were four instances where professional judgment was used to up/downgrade the overall study quality scores for animal toxicity studies (see Supplemental File for Animal and In Vitro studies¹⁵³). Importantly, the treatment of two of these studies (Kano 2008 and Argus 1965) highlights but one of many deeply flawed aspects of OPPT's systematic review methodology: if a single metric is assigned a score of Unacceptable the entire study is dismissed.

¹⁵¹ Environmental Defense Fund Comments on Application of Systematic Review in TSCA Risk Evaluations, submitted August 16, 2018, available at <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2018-0210-0077>.

¹⁵² Environmental Defense Fund Comments on Application of Systematic Review in TSCA Risk Evaluations, submitted August 16, 2018, available at <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2018-0210-0077>.

¹⁵³ U.S. EPA 2019, *Risk Evaluation for 1,4-Dioxane, Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies – Animal and In Vitro Studies*, https://www.epa.gov/sites/production/files/2019-06/documents/7_1-4-d_supplement_-_data_quality_evaluation_animal_and_in_vitro_tox_06272019.pdf.

B. OPPT’s dermal absorption analyses rely heavily on a single study that is not publicly available and was not evaluated using the agency’s systematic review process.

- Dermal absorption analyses in the draft risk evaluation for 1,4-dioxane hinge largely on what the agency refers to as the “Bronaugh in vitro study” (e.g., p. 110). This source (Bronaugh, 1982) does not appear to be publicly available. Moreover, the HERO page indicates that this “in vitro study” is in fact a book chapter rather than an actual scientific study document. The source is not in any of OPPT’s supplemental files containing the systematic review data quality evaluation sheets. Thus, OPPT’s entire characterization of a central human exposure consideration (dermal absorption) is drawn from a 1982 book chapter that is not publicly accessible, and for which there is no indication that an underlying study is available to assess through OPPT’s systematic review approach.
- OPPT relies on Bronaugh 1982 to derive toxicity values for multiple types of dermal hazard:
 - Acute/short-term POD for dermal exposures – sec. 4.2.6.2.2, p. 110-111
 - Chronic non-cancer POD for dermal exposures – sec. 4.2.6.2.5, p. 117-118
 - Chronic cancer unit risk for dermal exposures – sec. 4.2.6.2.7, p. 122-123

C. OPPT has inappropriately scored an occupational exposure study Unacceptable, removing critical data from consideration in the risk evaluation.

- OPPT gave a score of unacceptable to a workplace monitoring study (OSHA, 2016); chemical exposure health data (p. 105 of SR Supplemental File: Data Quality Evaluation of Environmental Releases and Occupational Exposure Data)
- Metric 3 (Applicability) has been scored Unacceptable, with the reviewer comment stating, “Looks like it should be an excel file with exposure data, but it’s all smooshed together in a text file and not useful” (p. 105).
 - The explanation provided in the reviewer comment is absurd and inappropriately results in a score of Unacceptable. In OPPT’s scoring guidelines for this study type (Application of SR in TSCA REs document, p. 76), the description for Unacceptable for the Applicability metric states, “The data are from an occupational or non-occupational scenario that does not apply to any occupational scenario within the scope of the risk evaluation.” Thus, a study should only be given a score of Unacceptable for this metric when the data are not within the scope of the evaluation. That is not the case here.

- To the extent OPPT had trouble accessing the data in a useable form, OPPT should have worked with OSHA to obtain the data, e.g., in an Excel sheet. The information is reasonably available and should have been considered by EPA in its analysis.

D. OPPT has again failed to define and explain its approach to evidence integration. Further, the approach taken to evidence integration in the draft 1,4-dioxane risk evaluation does not align with best practices as reflected and shared by leading systematic review methods for chemical assessment (e.g., OHAT, NavGuide, IRIS).

- As we have described in previous comments,¹⁵⁴ OPPT has not provided a pre-established methodology for its approach to evidence integration. This violates the agency’s own definition of weight of the scientific evidence; the final rule *Procedures for Chemical Risk Evaluation Under the Amended Toxic Substances Control Act* states that weight of the scientific evidence is:

“a systematic review method, applied in a manner suited to the nature of the evidence or decision, that uses a *pre-established protocol* to comprehensively, objectively, transparently, and consistently identify and evaluate each stream of evidence, including strengths, limitations, and relevance of each study and to integrate evidence as necessary and appropriate based upon strengths, limitations, and relevance.”

- Rather than providing a pre-established protocol for evidence integration, OPPT’s approach to evidence integration appears to be limited to the development of a “weight-of-the-scientific evidence narrative” (p. 82). This type of narrative approach is explicitly frowned upon in systematic review -- historically producing assessments of evidence that were inconsistent and lacked transparency -- and in large part motivated the inception of systematic review.

As noted in the 2014 National Academy of Sciences (NAS) report that reviewed EPA’s IRIS program:

¹⁵⁴ Environmental Defense Fund Comments on Application of Systematic Review in TSCA Risk Evaluations, submitted August 16, 2018, available at <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2018-0210-0077>; Environmental Defense Fund Comments on 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), submitted January 14, 2019, available at <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2018-0604-0013>.

Critical elements of conducting a systematic review include formulating the specific question that will be addressed (problem formulation) and *developing the protocol* that specifies the methods that will be used to address the question (protocol development).¹⁵⁵

After the systematic-review questions are specified, protocols for conducting the systematic reviews to address the questions should be developed. *A protocol makes the methods and the process of the review transparent, can provide the opportunity for peer review of the methods, and stands as a record of the review.* It also minimizes bias in evidence identification by ensuring that inclusion of studies in the review does not depend on the studies' findings. Any changes made after the protocol is in place should be transparent, and the rationale for each should be stated. EPA should include protocols for all systematic reviews conducted for a specific IRIS assessment as appendixes to the assessment.¹⁵⁶

EPA's IRIS program reflects this NAS recommendation by developing problem formulation and assessment protocols for each of its assessments.¹⁵⁷ OPPT needs to develop full protocols for each of its risk evaluation, and should consult with the IRIS program on how best to do so in consideration of requirements under TSCA.

E. OPPT's inconsistent application of its systematic review criteria results in an arbitrary and capricious analysis.

- As noted in Part 8.B, EPA relies on some sources without evaluating them under its systematic review process. But EPA then excludes some other sources on the basis of its systematic review process. EPA's inconsistent application of its systematic review process results in an arbitrary and capricious analysis since EPA includes and excludes sources based on EPA's decision about whether to apply the systematic review process, without explanation or justification.
- EPA has also not explained how it can exclude evidence from its systematic review process while still complying with its regulatory definition of "weight of the evidence" which states that evidence will be subject to systematic review.

¹⁵⁵ Nat'l Research Council, *Review of EPA's Integrated Risk Information System (IRIS) Process* (2014) at p. 5, <https://www.ncbi.nlm.nih.gov/books/NBK230060/> (emphasis added).

¹⁵⁶ *Id.* at 6 (emphases added).

¹⁵⁷ U.S. EPA, Office of Research & Dev., National Academy of Science Committee to Review Advances Made to the IRIS Program at slide 23 (Feb. 2018), <http://nas-sites.org/dels/files/2018/01/AdIRIS-15.pdf>.

PART II

EDF previously provided comments on the scope and problem formulation for 1,4-dioxane.¹⁵⁸ In those comments, EDF identified a variety of legal violations and other problems with EPA's approach to the 1,4-dioxane risk evaluation. Unfortunately, many of those same violations and problems appear in the draft risk evaluation, along with new ones. EDF incorporates and reiterates those earlier points here, as well as providing additional comments that address the new concerns.

Similarly, EDF has, as part of a broader coalition, filed an Opening Brief and Reply Brief explaining why the Risk Evaluation Rule is illegal and arbitrary and capricious. For these same reasons, it is illegal and arbitrary and capricious for EPA to follow the Rule in developing this risk evaluation. EDF incorporates and reiterates some of those points here as well. We attach those Briefs as Appendices VI and VII. EPA should fix all of these problems in its final risk evaluation.

1. TSCA requires EPA to analyze whether a chemical substance, as a whole, presents an unreasonable risk, and EPA does not have discretion to ignore conditions of use, exposures, or hazards.

In its prior scoping document and problem formulation for 1,4-dioxane,¹⁵⁹ EPA stated that it had authority to exclude conditions of use. In the draft risk evaluation, EPA again reiterated this assertion of authority and again stated that it has excluded all consumer uses from its analysis, on the theory that it can do so because 1,4-dioxane is present only as a byproduct in those consumer uses (p. 28). In our comments on the scope and the problem formulation, EDF explained that this approach is foreclosed under the statute, and EDF incorporates those arguments here.¹⁶⁰ Similarly, EDF incorporates the arguments presented in our Briefs attached as Appendix VI at 21-40 and Appendix VII at 14-31.

¹⁵⁸ EDF Comments on Ten Scopes under the Toxic Substances Control Act, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0743-0069>; EDF Comments on Problem Formulations for Risk Evaluations To Be Conducted Under Toxic Substances Control Act, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0723-0083>.

¹⁵⁹ U.S. EPA, Problem Formulation of the Risk Evaluation for 1,4-Dioxane (May 2018), p.18, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0723-0064> (hereinafter "Problem Formulation for 1,4-Dioxane").

¹⁶⁰ EDF Comments on Ten Scopes under the Toxic Substances Control Act, pp. 4-11, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0743-0069>; EDF Comments on Problem Formulations for Risk Evaluations To Be Conducted Under Toxic Substances Control Act, pp.13-20, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0723-0083>.

In the problem formulation, as well as in the draft risk evaluation (p. 28), EPA states that it will also exclude exposures under other conditions of use as well. Specifically, EPA ignores numerous exposure pathways that are allegedly addressed by other statutes (p. 156). In the draft risk evaluation, EPA acknowledges that these exposures flow from conditions of use, including “industrial releases to air, water or land; and other conditions of use” (p. 156). EPA also effectively ignores certain hazards by completely failing to analyze environmental hazards to sediment-dwelling, terrestrial, or avian organisms (pp. 41, 79-80). EPA also effectively ignores certain hazards by failing to analyze the unique hazards presented to potentially exposed or susceptible subpopulations (p. 108).

TSCA’s language and structure unambiguously foreclose EPA’s interpretation that it may ignore certain conditions of use, exposures, and hazards. EPA’s decision to disregard certain exposure pathways and hazards is also “arbitrary, capricious, [or] an abuse of discretion” under the APA, 5 U.S.C. § 706(2)(A), because it will lead EPA to consider “factors which Congress has not intended it to consider [and] entirely fail[] to consider an important aspect of the problem.” *Motor Vehicle Mfrs. Ass’n v. State Farm Mut. Auto. Ins. Co.*, 463 U.S. 29, 43 (1983). Moreover, as the draft risk evaluation itself reveals, this approach leads to irrational and arbitrary applications. Instead, EPA should be guided by the statutory language and consider all of the conditions of use, exposures, and hazards related to a chemical substance. EPA should evaluate all of the evidence of conditions of use, exposure, and hazard; it should not ignore evidence because of self-imposed blinders.

A. The plain text, overall structure, purpose, and legislative history of TSCA indicate that EPA has to determine whether a chemical substance presents an unreasonable risk comprehensively, considering all of its hazards, exposures, and conditions of use.

i. The plain text requires EPA to consider all hazards, exposures, and conditions of use.

Statutory interpretation should begin, as always, with the language of the statute. The plain language of the risk evaluation provision supports the interpretation that EPA must consider all hazards, exposures, and conditions of use as necessary “to determine whether a *chemical substance* presents an unreasonable risk.” 15 U.S.C. § 2605(b)(4)(A) (emphasis added). This directive expresses Congress’s clear intent that EPA evaluate the risks posed by “a chemical substance” as a whole. Congress consistently used the phrase “a chemical substance” to describe the object of priority designations and risk evaluations. 15 U.S.C. § 2605(b)(1)-(4), (i) (using the phrase 14 times). This language requires EPA to consider all hazards and exposures that contribute to the total risk presented by the chemical substance as a whole.

This whole-substance focus begins during prioritization. The definitions of high- and low-priority substances make clear that it is the “substance” that receives the designation, not selected

conditions of use, exposures, or hazards. *See id.* § 2605(b)(1)(B). The provision requiring EPA to select the first ten chemicals also directed that the risk evaluations be “conducted on 10 chemical substances drawn from the 2014 update of the TSCA Work Plan,” making the object of these risk evaluations the chemical substances as a whole. *Id.* § 2605(b)(2)(A). As EPA reasoned in the Prioritization Rule, “[t]he statute is clear that EPA is to designate the priority of the ‘chemical substance’—not a condition of use for a chemical substance.” 82 Fed. Reg. 33,753, 33,755 (July 20, 2017) (citing 15 U.S.C. § 2605(b)(1)(A)). Similarly, EPA must prioritize the whole chemical, and EPA is not directed to prioritize only certain hazards or exposures. Indeed, the prioritization process expressly “shall include a consideration of the hazard and exposure potential of a chemical substance,” without any basis for EPA to limit that consideration to only certain hazards or exposures. 15 U.S.C. § 2605(b)(1)(A).

EPA must also conduct risk evaluations on “a chemical substance” as a whole. For example, TSCA provides that “[u]pon designating a chemical substance as a high-priority substance, the Administrator shall initiate a risk evaluation on the *substance*.” 15 U.S.C. § 2605(b)(3)(A) (emphasis added). Similarly, the statute directs EPA to determine either that “a *chemical substance* presents” or “does not present an unreasonable risk.” *Id.* § 2605(i)(1)-(2) (emphasis added). Congress also uses the phrase “a chemical substance” or “chemical substances” in many other places in TSCA’s risk evaluation provisions. *See, e.g., id.* § 2605(b)(4)(G) (setting deadlines for completing evaluation for “a chemical substance”), (b)(2)(A), (b)(2)(B), (b)(3)(A), (c)(1).

The plain language of the risk evaluation provisions requires EPA to consider all available information about hazards, exposures, and conditions of use, without limitation. TSCA § 6(b)(4)(F)(i) expressly requires that EPA “integrate and assess available information on hazards and exposures for the conditions of use of the chemical substance.” 15 U.S.C. § 2605(b)(4)(F)(i). Thus, if there is “available information on hazards and exposures,” then EPA must integrate and assess that information as part of the risk evaluation. Similarly, TSCA § 6(b)(4)(F)(iv) requires that EPA “take into account, where relevant, the likely duration, intensity, frequency, and number of exposures under the conditions of use of the chemical substance.” *Id.* § 2605(b)(4)(F)(iv). This provision requires EPA to take into account exposures unless EPA can establish that they are irrelevant. Finally, TSCA § 6(b)(4)(F)(v) requires that EPA “describe the weight of the scientific evidence for the identified hazard and exposure.” *Id.* § 2605(b)(4)(F)(v).

All of these provisions direct EPA to consider a chemical’s hazards, exposures, and conditions of use, and none of them include any language providing EPA with any discretion to ignore any hazards, exposures, or conditions of use. While EPA previously articulated a legal theory (albeit flawed) for ignoring certain conditions of use, EPA has not pointed to any legal basis for

ignoring hazards or exposures under the conditions of use being analyzed in a risk evaluation. EPA has pointed to no textual basis for these exclusions.

Moreover, when EPA promulgates risk-management regulations under TSCA § 6(a):

[EPA] shall consider and publish a statement based on reasonably available information with respect to—

- (i) the effects of the chemical substance or mixture on health and the magnitude of the exposure of human beings to the chemical substance or mixture;
- (ii) the effects of the chemical substance or mixture on the environment and the magnitude of the exposure of the environment to such substance or mixture;

15 U.S.C. § 2605(c)(2)(A). In order to accurately draft this statement, EPA will have to have considered all of the hazards posed by a chemical (i.e., its effects on human health and the environment) as well as all exposures. EPA cannot accurately describe “the magnitude of the exposure of human beings to the chemical substance,” if EPA has ignored numerous exposures. 15 U.S.C. § 2605(c)(2)(A)(i). Similarly, EPA cannot accurately describe “the magnitude of the exposure of the environment” for chemicals, *id.* § 2605(c)(2)(A)(ii), if EPA has ignored the vast majority of environmental exposures, as EPA proposes to do. Congress specifically intended for EPA to “satisfy these requirements on the basis of the conclusions regarding the chemical’s health and environmental effects and exposures in the risk evaluation itself.” 114 Cong. Rec. S3517 (daily ed. June 7, 2016). Thus, EPA must evaluate all hazards and exposures in its risk evaluations.

Moreover, TSCA requires that EPA evaluate a chemical’s risk “without consideration of costs or other nonrisk factors.” 15 U.S.C. § 2605(b)(4)(A). By excluding certain hazards, exposures, and conditions of use for reasons that bear no relationship to risk, EPA is considering nonrisk factors. For example, by excluding exposures because they could be regulated under another statute, EPA is considering a nonrisk factor.

Textually, EPA’s approach also directly conflicts with TSCA § 26(k). 15 U.S.C. § 2625(k). TSCA § 26(k) requires EPA to “take into consideration information relating to a chemical substance or mixture, including hazard and exposure information, under the conditions of use, that is reasonably available to the Administrator.” *Id.* Congress included this provision to ensure that EPA could not ignore “reasonably available” “information relating to a chemical substance or mixture”; the purpose of this provision is to compel EPA to consider all reasonably available information. Congress also specified that EPA must consider the reasonably available “hazard and exposure information.” It would undermine this directive if EPA chooses to ignore certain hazards or exposures.

- ii. *TSCA's overall structure requires EPA to consider all hazards, exposures, and conditions of use.*

Moreover, EPA's pick-and-choose approach cannot be squared with the overall structure of TSCA.

As EPA reasoned in its proposed Risk Evaluation Rule, when discussing conditions of use, that TSCA "provides no criteria for EPA to apply" for selecting hazards, exposures, and conditions of use for analysis shows that the Agency does not have "license to choose" among those hazards, exposures, and conditions of use for analysis. 82 Fed. Reg. 7562, 7566 (Jan. 19, 2017). The precision with which Congress prescribed EPA's implementation of section 6 supports this reading. Section 6 lays out detailed directions for EPA. *See* 15 U.S.C. § 2605(b)(1)(A) (mandating considerations for priority designations), (b)(4)(D) (identifying risk factors to include in a risk evaluation's scope), (b)(4)(F)(i)-(v) (detailing requirements for conducting risk evaluations); *see also id.* § 2605(a) (specifying possible risk management measures). These provisions indicate that Congress did not mean to allow EPA to exclude hazards, exposures, or conditions of use from risk evaluation without any criteria or instruction. *Cf. NRDC, Inc. v. EPA*, 863 F.2d 1420, 1432 (9th Cir. 1988) (invalidating regulatory procedure that "is wholly silent as to what factors the agency is to consider in granting exceptions" and provides "no discernible standard [for] limit[ing] th[at] discretion").

Indeed, when Congress intended EPA to exercise discretion under TSCA, it said so explicitly. *See, e.g.*, 15 U.S.C. §§ 2613(f) (granting EPA "[d]iscretion" in handling claims to protect confidential information), 2608(a) (instructing EPA, if it "determines, in the Administrator's discretion," that an unreasonable risk may be prevented under a federal law administered by another agency, to notify the agency), 2608(b), 2605(b)(4)(E)(iv)(II). That Congress purposefully included the language of discretion "in one section of the statute but omit[ted] it in another section of the same Act" shows that Congress did not intend EPA to use discretion to pick and choose which hazards, exposures, and conditions of use to consider in prioritization and risk evaluation. *Hernandez v. Ashcroft*, 345 F.3d 824, 834 (9th Cir. 2003) (quoting *Andrieu v. Ashcroft*, 253 F.3d 477, 480 (9th Cir. 2001) (en banc)).

Implicitly recognizing that Congress did not grant EPA boundless discretion to exclude exposures, EPA suggests that it will "focus its analytical efforts on exposures that are likely to present the greatest concern." *See, e.g.*, Problem Formulation for 1,4-Dioxane at 11. But no language in TSCA limits EPA to this "greatest concern" or "greatest potential for risk" focus. Nor does EPA point to any statutory terms that even arguably supply such a limitation. Moreover, EPA cannot rely on this reasoning to justify its exclusions for 1,4-dioxane because EPA has excluded all consideration of 1,4-dioxane's exposures to the general population and consumers, as well as *unregulated* exposure pathways through water and drinking water; EPA

has presented no analysis showing that it has actually selected those exposures that present the greatest concern.

TSCA's provisions direct EPA to prepare risk evaluations and the related findings for "chemical substances," as a whole, not for specific or limited hazards, exposures, or conditions of use of those substances. For example, the risk management provision expressly requires EPA to address risks when the risks arise from combined sources of exposure. TSCA § 6(a) provides that: "If [EPA] determines in accordance with [the risk evaluation provision] that the manufacture, processing, distribution in commerce, use, or disposal of a chemical substance or mixture, or that any combination of such activities, presents an unreasonable risk of injury to health or the environment," then EPA must issue a risk management rule. 15 U.S.C. § 2605(a); *see also* 15 U.S.C. § 2608(a) (using same language in provision governing requests to other federal agencies to address risks). Thus, if exposures resulting from "any combination" of conditions of use present an unreasonable risk, EPA must issue a risk management rule. But EPA must analyze *all* of the exposures resulting from these activities to assess whether *any combination* presents such a risk.

iii. TSCA's purpose, as well as basic logical reasoning and the best available science, require EPA to consider all hazards, exposures, and conditions of use to assess a chemical substance as a whole.

The purpose of the risk evaluation is to analyze the risks of a substance based on an assessment of its hazards and exposures. Ignoring potential exposures and hazards at the outset undermines that purpose. And science and logic do not support EPA's exclusions. As explained below in Part II sec. 1.C and sec. 5, EPA's exclusions of certain exposures result in an incoherent draft risk evaluation where EPA acknowledges ample evidence of exposure, for example, in the monitoring data, but then refuses to look at those very exposures in its final analysis. Willfully ignoring these exposures at the outset is contrary to the purpose of TSCA's risk evaluations, as well as the law's requirement that EPA rely on the best available science. EPA is imposing blinders on its analysis by asserting authority to refuse to look at certain exposures, including known exposures, and the result is that EPA is overlooking exposures in the real world. This approach is both contrary to law and arbitrary and capricious.

iv. The legislative history requires EPA to integrate a chemical's exposure and hazard information and nothing suggests that EPA can ignore existing exposures and hazards.

Numerous statements in the legislative history reveal that Congress intended for EPA to assess "risk" based on "the integration of hazard and exposure information about a chemical." S. Rep. No. 114-67 at 17 (June 18, 2015); 161 Cong. Rec. H4551 at H4556 (daily ed. June 23, 2015) ("The risk evaluation itself only asks does the chemical present an unreasonable risk of injury to

health or the environment. That is a science question based on a combination of hazard and actual exposure.”). Senator Vitter described an accurate assessment of risk as turning on integrating exposure and hazard information. *See* 162 Cong. Rec. S3511 at S3519 (daily ed. June 7, 2016) (“Exposure *potential*, when integrated with the hazard *potential* of a chemical, determines a chemical’s potential for risk.”) (emphases added). Congress intended for EPA to integrate all available information about exposure and hazard when assessing risk, as reflected in this history and the text of TSCA.

No statement in the legislative history suggests that EPA may ignore exposures or hazards when assessing the risk presented by a chemical substance. In its Risk Evaluation Rule, EPA relied on a floor statement from a single Senator to justify its interpretation that it had discretion to choose the conditions of use for analysis. 40 Fed. Reg. at 33,728 (citing 114 Cong. Rec. S3519-20 (daily ed. June 7, 2016) (statement of Sen. Vitter)). As EDF has previously explained,¹⁶¹ the legislative history as a whole does not justify EPA’s approach to conditions of use, but here EPA has even less basis for its approach; EPA has not pointed to any statement in the legislative history supporting its approach of ignoring certain exposures or hazards.

v. EPA has inappropriately excluded all consumer uses and all contamination of industrial, commercial and consumer products.

In the draft risk evaluation, EPA states that: “[c]onsumer uses were not considered within scope of this risk evaluation per the problem formulation” (p. 28). In the problem formulation, EPA asserts that it “did not find evidence of any current consumer uses for 1,4-dioxane and is excluding consumer uses from the scope of the risk evaluation” and that “contamination of industrial, commercial and consumer products are *not intended conditions of use* for 1,4-dioxane and will not be evaluated.” U.S. EPA, Problem Formulation of the Risk Evaluation for 1,4-Dioxane at p. 18 (emphasis added) (May 2018), <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0723-0064>. For support, EPA cites its earlier scope document. *See* 1,4-Dioxane, Scope, Docket ID: EPA-HQ-OPPT-2016-0723.

In our comments on the scope document, EDF already commented at length on the illegality of these exclusions; those comments are incorporated here by reference.¹⁶² In brief, EPA’s position that it can ignore known and foreseeable conditions of use of a chemical violates the text of the law. “Conditions of use” expressly includes “the circumstances *** under which a chemical substance is intended, known, or reasonably foreseen to be to be manufactured, processed, distributed in commerce, used, or disposed of.” 15 U.S.C. § 2602(4). Congress expressly chose

¹⁶¹ EDF Comments on Ten Scopes under the Toxic Substances Control Act, pp.7-8, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0743-0069>.

¹⁶² EDF Comments on Ten Scopes under the Toxic Substances Control Act, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0743-0069>.

to define “conditions of use” broadly to include not only “intended,” but also “known” or “reasonably foreseen” manufacture, processing, distribution, use, and disposal. 15 U.S.C. § 2602(4). Disregarding chemical substances such as 1,4-dioxane that are present in products as impurities or byproducts because they are not “intended” essentially reads the other two scenarios out of the statute. This exclusion will result in a deficient and erroneous evaluation and determination of the chemical’s risks.

EPA’s scope document identified numerous products that “potentially contain[] 1,4-dioxane as a residual contaminant, including paints, coatings, lacquers, ethylene glycol-based antifreeze coolants, spray polyurethane foam, household detergents, cosmetics/toiletries, textile dyes, pharmaceuticals, foods, agricultural and veterinary products,” as well as “magnetic tape and adhesives.” See 1,4-Dioxane, Scope at p. 21, Docket ID: EPA-HQ-OPPT-2016-0723. And in EPA’s 2015 Problem Formulation and Initial Assessment for 1,4-dioxane, EPA stated:

EPA/OPPT concludes that exposure to consumers can result from the use of soaps and detergents and other products that contain 1,4-dioxane as a contaminant. Adult women who use multiple cosmetics and cleaning products are likely the most exposed population as determined in the Canada assessment.¹⁶³

Bizarrely, EPA nonetheless concludes in its problem formulation that it “did not find evidence of any current consumer uses for 1,4-dioxane.” Problem Formulation for 1,4-Dioxane at 18.¹⁶⁴ As discussed in detail in our earlier comments, these products are known and reasonably foreseen conditions of use leading to exposures to 1,4-dioxane, and EPA’s decision to ignore them when analyzing whether this chemical presents an unreasonable risk is arbitrary and capricious. EPA must analyze these conditions of use in the risk evaluation.

In the draft risk evaluation, EPA presents one additional rationale for excluding consumer uses. EPA states that its regulatory tools under TSCA § 6(a) are “better suited to addressing any unreasonable risks that might arise from these activities through regulation of the activities that generate 1,4-dioxane as an impurity or cause it to be present as a contaminant than addressing them through direct regulation of 1,4-dioxane” (p. 28). But this argument is irrational and illegal

¹⁶³ See U.S. EPA, *TSCA Work Plan Chemical Problem Formulation and Initial Assessment, 1,4-Dioxane, CASRN: 123-91-1* at 28 (Apr. 2015), https://www.epa.gov/sites/production/files/2017-06/documents/14_dioxane_problem_formulation_and_intial_assessment.pdf.

¹⁶⁴ Equally bizarrely, EPA states: “The 1,4-dioxane life cycle diagram (Figure 2-1) indicates that no uses of 1,4-dioxane were identified in consumer products. *EPA did not receive data, information or comments that informed a change was necessary to the scope.*” Problem Formulation for 1,4-Dioxane at p. 41 (emphasis added) This statement is, of course, demonstrably false. EPA’s own scope document identifies numerous uses of 1,4-dioxane in consumer products, as did EDF’s and, no doubt, many others’ comments.

because EPA has full authority under TSCA § 6(a) to directly regulate 1,4-dioxane as a byproduct (or impurity or contaminant).

EPA has full authority under TSCA § 6(a) to regulate 1,4-dioxane when it appears as an impurity, contaminant, or byproduct as long as EPA finds that it presents an unreasonable risk in a risk evaluation. 15 U.S.C. § 2605(a). Indeed, TSCA § 6(a) expressly requires that EPA “apply one or more of the following requirements to such substance or mixture to the extent necessary so that the chemical substance or mixture no longer presents such risk.” TSCA § 6(a). The requirements that EPA has authority to apply can all be applied to 1,4-dioxane as an impurity, byproduct, or contaminant. *See* 15 U.S.C. § 2605(a). For example, under TSCA § 6(a)(1), EPA may prohibit or limit the “manufacturing, processing, or distribution in commerce” of a chemical substance as a byproduct. 15 U.S.C. § 2605(a)(1). EPA has long recognized that the production and presence of a chemical as a byproduct counts as manufacturing the chemical for purposes of TSCA. *See, e.g.*, 40 C.F.R. § 710.3 (defining “manufacture for commercial purposes” to include production of byproducts regardless of whether they have separate commercial value).¹⁶⁵ And when a chemical substance is distributed in commerce as a byproduct, it is still distributed in commerce and can be regulated as such. Similarly, each other regulatory authority under TSCA § 6(a) may be applied when a chemical appears as an impurity, byproduct, or contaminant. All of these regulatory authorities can be exercised to directly regulate 1,4-dioxane as a byproduct, so EPA’s suggestion that its authorities are not suited to regulate 1,4-dioxane in such circumstances is simply irrational and incorrect.

vi. EPA has failed to identify certain conditions of use identified in the 2002 EU Risk Assessment for 1,4-dioxane.

In the 2002 EU Risk Assessment,¹⁶⁶ the Assessment found that there was a “deliberate use” of 1,4-dioxane as a solvent/reagent in a wide range of applications including “products like lacquers, varnishes, cleaning and detergent preparations, adhesives, cosmetics, and deodorant fumigants.”¹⁶⁷ The Assessment stated that 1,4-dioxane was “found in several products mainly solvents and cleaning agents,” and in a sealing compound available to consumers. In its problem formulation and draft risk evaluation, EPA appears to have overlooked some of these deliberate uses, even though they are known to have occurred and certainly qualify as reasonably

¹⁶⁵ Moreover, when EPA previously regulated Polychlorinated Biphenyls (PCBs), EPA stated that its regulations “applie[d] to all persons who manufacture, process, distribute in commerce, use, or dispose of PCBs or PCB Items.” 40 C.F.R. § 761.1(b)(1). EPA then clarified that this language also applied the regulations to “other chemical substances or combinations of substances, including impurities and byproducts and any byproduct, intermediate, or impurity manufactured at any point in a process.” *Id.*

¹⁶⁶ European Chemicals Bureau, *European Union Risk Assessment Report: 1,4-dioxane* (2002), <https://echa.europa.eu/documents/10162/a4e83a6a-c421-4243-a8df-3e84893082aa>.

¹⁶⁷ *Id.* at 49.

foreseeable. EPA must analyze these conditions of use to prepare a comprehensive risk evaluation.

B. EPA’s own risk evaluation rule requires that EPA consider all relevant hazards and all exposures under the conditions of use within the risk evaluation.

EDF disagrees with EPA’s final Risk Evaluation Rule for numerous reasons, as discussed in our prior comments and in litigation challenging that rule. EDF reiterates and incorporates those points here. *See* Appendix VI; Appendix VII. Nonetheless, even EPA’s final Risk Evaluation Rule requires EPA to consider all relevant hazards and exposures under the conditions of use within the risk evaluation. The Rule specifically requires that: “Relevant *potential* human and environmental hazards will be evaluated.” 40 C.F.R. § 702.41(d)(3) (emphasis). Thus, EPA must consider any relevant “potential” hazards when preparing a risk evaluation. *See also* 40 C.F.R. § 702.41(d)(2) (“The hazard assessment process will identify the types of hazards to health or the environment posed by the chemical substance under the condition(s) of use within the scope of the risk evaluation.”). The Rule also requires that: “[e]xposure information related to potential human health or ecological hazards of the chemical substance will be reviewed in a manner consistent with the description of best available science and weight of scientific evidence.” 40 C.F.R. § 702.41(e)(3). When preparing the risk characterization, EPA shall “[t]ake into account, where relevant, the likely duration, intensity, frequency, and number of exposures under the condition(s) of use of the chemical substance.” 40 C.F.R. § 702.43(a)(4). Thus, EPA must consider all hazards and all exposures under the conditions of use. None of these duties are qualified or provide an authority for EPA to exclude hazards or exposures from analysis.

Other provisions of the rule confirm this reading. EPA requires manufacturer requests for risk evaluations to “include or reference *all* available information on the health and environmental hazard(s) of the chemical substance, human and environmental exposure(s), and exposed population(s), as relevant to the circumstances identified in the request.” 40 C.F.R. § 702.37(b)(4) (emphasis added). Thus, manufacturers must submit all available information on hazard and exposure under the identified conditions of use because EPA must consider all hazards and exposures when preparing risk evaluations.

In the preamble to the rule, EPA commits to considering all hazards and exposures under the conditions of use:

The Administrator will consider relevant factors including, but not limited to: The effects of the chemical substance on health and human exposure to such substance under the conditions of use (including cancer and non-cancer risks); the effects of the chemical substance on the environment and environmental exposure under the conditions of use.

82 Fed. Reg. at 33,735. EPA thus committed to considering the “effects of the chemical substance on health and human exposure to such substance under the conditions of use.” *Id.* These commitments are not qualified or accompanied by any assertion of discretion to ignore effects or exposure information under the conditions of use. EPA cannot fulfill this duty without considering all the hazards and sources of human exposure under the conditions of use.

Similarly, in the preamble, EPA states that “[u]sing reasonably available information, exposures will be estimated (usually quantitatively) for the identified conditions of use.” 82 Fed. Reg. at 33,742. EPA cannot prepare an accurate quantitative estimate for exposure if EPA has excluded exposure pathways. “For environmental evaluations specifically, EPA plans to include a discussion of the nature and magnitude of the effects, the spatial and temporal patterns of the effects, [and] implications at the species, population, and community level.” 82 Fed. Reg. at 33,743. EPA cannot accurately discuss the magnitude of the effects on the environment or the spatial and temporal patterns of those effects if EPA ignores the vast majority of the environmental exposures, as EPA does in the problem formulation and draft risk evaluation. In addition, EPA completely fails to discuss the environmental effects on sediment-dwelling, terrestrial, and avian organisms; EPA instead limits its environmental analysis to aquatic organisms. Indeed, the draft risk evaluation does not include any discussion of “the spatial and temporal patterns of the effects, [and] implications at the species, population, and community level.” 82 Fed. Reg. at 33,743.

Moreover, in the preamble to the rule, while EPA went to great lengths to describe its alleged discretion to pick-and-choose conditions of use, EPA never stated that it had discretion to exclude hazards or exposures related to conditions of use within the risk evaluation. EPA’s failure to assert any discretion to exclude exposures and hazards reflects that EPA, in fact, lacks any such discretion. Similarly, in the preamble to the risk evaluation rule, EPA asserted that it had authority to ignore conditions of use under other agencies’ jurisdiction. 82 Fed. Reg. at 33,729 (July 20, 2017). This is incorrect, but EPA never asserted that it had authority to ignore exposures under EPA’s jurisdiction. Once again, EPA’s silence on this issue in its rule highlights that EPA could not justify such discretion. In sum, EPA’s arguments for excluding certain conditions of use cannot simply be extended mindlessly to exclude consideration of exposures and hazards. *See United States Sugar Corp. v. EPA*, 830 F.3d 579, 650 (D.C. Cir. 2016) (agency may not assume a rationale for one exemption identically applies elsewhere).

C. The draft risk evaluation is incoherent and arbitrary and capricious because of EPA’s approach to hazard, exposure, and conditions of use.

EPA’s illegal approach to exposures leads it to put “blindness” on regarding risks. The result is “arbitrary, capricious, [or] an abuse of discretion” under the APA, 5 U.S.C. § 706(2)(A), because it will lead EPA to have considered “factors which Congress has not intended it to consider [and]

entirely failed to consider an important aspect of the problem.” *State Farm*, 463 U.S. at 43. It also violates several provisions of TSCA § 26 because by ignoring uses, exposures, hazards, and related information, EPA will not be acting “consistent with the best available science,” EPA will not base decisions on “on the weight of the scientific evidence,” and EPA will not “take into consideration information relating to a chemical substance or mixture, including hazard and exposure information, under the conditions of use, that is reasonably available to the Administrator.” 15 U.S.C. § 2625(h), (i), (k). In addition, because EPA’s distinction is a false one untethered to the information, EPA seems to treat certain exposures inconsistently throughout the document.

For example, as detailed more below, early in the draft risk evaluation, EPA acknowledges that 1,4-dioxane exposures occur through numerous media (p. 19). But EPA then systematically excludes many of these pathways of exposure from its risk evaluation (p. 28). Thus, in the problem formulation, EPA (correctly) describes the factual reality that exposures to humans and the environment occur through these environmental pathways. But EPA then imposes blinders on its analysis by excluding these pathways from further consideration. This is the definition of arbitrary and capricious conduct.

EPA’s should change the final risk evaluation to assess the reasonably available information on hazards and exposures for 1,4-dioxane, and that information should inform EPA’s evaluation of the risks of the chemical. If there is a real-world or reasonably foreseen exposure or hazard, then EPA should not ignore it.

2. EPA should not refuse to further analyze exposure pathways on a cursory basis, and in any event, EPA still needs to consider those exposures when evaluating the combined exposures.

In the problem formulation for 1,4-dioxane, EPA insufficiently justified many of its decisions not to include known or potential exposures or conduct further analysis, and prematurely concluded various exposures present no significant risk. EPA’s 1,4-dioxane problem formulation contained many rushes to judgment, with EPA all but concluding there is no unreasonable risk from certain exposures, based on little analysis and with no indication that it intended to revisit those exposures or risks in combination with those it did intend to analyze further. The draft risk evaluation fails to provide the analysis missing from the problem formulation, and thus, these cursory analyses remain deeply flawed. We describe some of EPA’s flawed analyses below.

When EPA declines to analyze a pathway further, EPA must have developed and applied a sound, rational basis for assessing the exposure level, supported by scientific evidence. In addition, EPA cannot then effectively ignore the exposure. Rather, EPA still must consider how the exposure may combine with other sources of exposure, so EPA must actually assess the level

of exposure from the pathway individually and then consider how it combines with other sources of exposure. EPA has completely failed to consider these combined exposures in its draft risk evaluation.

Risks to aquatic invertebrates and aquatic plants: With little analysis and based on limited data, EPA asserted that “[m]easured and estimated levels of 1,4-dioxane in the environment are sufficiently below the acute and chronic aquatic COCs [concentrations of concern],” planned no further analysis, and implied it had concluded that any associated risks can be ignored. Problem Formulation for 1,4-dioxane at p. 41. Yet:

- EPA’s predicted concentrations in surface water for acute and chronic scenarios are up to 58% and 40% of the COCs, leaving little room for error.
- Elsewhere, EPA acknowledged “[T]here are relatively fewer data available on 1,4-dioxane levels in surface water,” (*id.* at 28), indicating a data gap that EPA apparently will do nothing to address.
- EPA implied that its calculations of COCs are conservative at least in part because of its use of assessments factors (*Id.* at 29, 70, 81). The use of such factors is not conservative: They account for *real-world sources of variability as well as database limitations*, and cannot be construed as “safety factors” that yield conservative estimates.¹⁶⁸ As EPA states: “The application of AFs [assessment factors] provides a lower bound effect level that would likely encompass more sensitive species not specifically represented by the available experimental data. AFs are also account for differences in inter- and intra-species variability, as well as laboratory-to-field variability.” (*Id.* at 70)
- EPA’s calculated acute COC was inconsistently reported. In the text, it is listed as 59,800 ppb (*Id.* at 35), while in Appendix C it is listed as 20,000 ppb (*Id.* at 70). EPA’s modeling of surface water concentrations includes assumptions that were not necessarily conservative, despite EPA’s claims to the contrary.

For example, EPA pointed to the surface water modeling assumption that “[w]astewater treatment removal is assumed to be 0% for this exercise” (*Id.* at 29); yet its own modeling of wastewater treatment removal efficiency using EPISuite STP module indicates removal rates will be very low, on the order of 2% (*Id.* at 24). Far from being a conservative assumption, this use of 0% was a reasonable conclusion based on the available data. Despite a promised “full table of results, see Appendix E” (*Id.* at 29), that table provided only EPA’s conclusions and none of its analysis.

¹⁶⁸ See Part II sec. 9 of these comments.

EPA's current analysis is incoherent and unexplained. EPA must obtain additional information on exposure through this pathway as well as hazard, and EPA must prepare a scientifically valid analysis of this pathway.

Risks to sediment organisms: In the problem formulation, EPA stated: "While no ecotoxicity studies were available for sediment organisms, the toxicity of 1,4-dioxane to sediment invertebrates is expected to be similar to the toxicity to aquatic invertebrates." (*Id.* at 42) EPA provided no basis for this assertion of expected similar toxicity. This is a clear data gap that EPA should have filled, or should now move to fill, rather than resort to such hand-waving to dismiss a potential risk it has not examined.

Occupational exposures: Occupational exposures appear to be the only human exposures to 1,4-dioxane EPA analyzed in the risk evaluation. But in the problem formulation, EPA ruled out numerous potential exposures to mist with no real analysis supporting its conclusion that mists would not present a route of exposure.

In the problem formulation, EPA stated: "EPA reviewed the potential for occupational exposures associated with subcategories of conditions of use where a mist may be generated. *EPA determined that most subcategories will not produce a mist during their typical use and, for these, EPA concludes that exposure to 1,4-dioxane would be negligible and does not plan further analysis.*" Problem Formulation for 1,4-dioxane at 37 (emphasis added) EPA appears to have conducted no analysis, at all, let alone any "further analysis." It provides no supporting analysis or data to support this sweeping assertion. Yet EPA has drawn an apparently final conclusion not to be revisited that exposure is "negligible." The draft risk evaluation does not appear to address mists for the conditions of use where EPA eliminated mists from consideration at the problem formulation stage. Thus, the missing analysis has still not been provided.

Indeed, even for those uses where EPA does consider mists in the risk evaluation, EPA's analysis for some of those uses provides no convincing analysis or evidence for finding that mists are "not expected" (p. 261). EPA did not assess mists for those uses, but once again, EPA has not provided any evidence or analysis supporting its conclusion that mists are unlikely to present a route of exposure.

EPA must analyze occupational exposures based on the best available science, and EPA must use its information authorities to obtain reasonably available information about these exposures.

General population exposures: In the problem formulation, EPA stated that it "does not expect to consider and analyze general population exposures in the risk evaluation for 1,4-dioxane." (p. 49) The draft risk evaluation in fact did not consider or analyze such exposures (p. 28). Yet its own analyses point to the clear potential for such exposures. Below are some examples.

EPA stated:

Indoor air exposures may occur from infiltration from ambient air or emissions from tap water during activities such as showering and bathing. Based on the relatively high water solubility and relatively low Henry's law constant for 1,4-dioxane, EPA expects that volatilization would be low for many indoor uses. However, increased water temperature during bathing and showering can increase volatilization.

Problem Formulation for 1,4-Dioxane at p. 31.

In addition to exposure to 1,4-dioxane contaminated tap water used for showering or bathing, use of products containing 1,4-dioxane especially in warm or hot water could also lead to exposures. This would include personal care products like shampoo or soap (some of which may not fall under TSCA jurisdiction) but also various cleaning, laundry or related products that could or would be used in hot or warm water. EPA must analyze exposures to the general population, including exposures through water and products.

EPA stated: "1,4-Dioxane has also been detected in landfill leachate (ATSDR, 2012)." (Problem Formulation, p. 28) Yet it stated its intent to wholly exclude such exposures based on presumed adequate management under federal or state law, absent any analysis demonstrating this. The fact that 1,4-dioxane is present in liquids leaching from landfills suggest that it may be coming from consumer products containing it – yet another demonstration of how arbitrary EPA's decision is to ignore exposures associated with consumer use of products containing the chemical.

3. EPA must analyze background exposures in the draft risk evaluation.

In the draft risk evaluation for 1,4-dioxane, EPA does not consider the background exposures that workers experience through consumer uses, drinking water, and other exposure pathways. EPA needs to include consideration of such exposures in its draft risk evaluation for the reasons articulated in Part II sec. 1. But the exclusion of these exposures also undermines EPA's analysis of those circumstances that EPA does analyze in the draft risk evaluation because it is the total level of exposure to a chemical that determines risk, and this includes exposures that are not generally attributable to any one use or source.

4. EPA should analyze past conditions of use because they are reasonably foreseen.

Past conditions of use are known to have occurred in the past and are certainly reasonably foreseen conditions of use, absent compelling evidence that they will not resume. As argued further in Part II sec. 1, EPA must consider all conditions of use when preparing a risk evaluation

under TSCA § 6, including so-called legacy uses, associated disposals, and legacy disposals. EDF has previously articulated these arguments and incorporates the arguments here.¹⁶⁹

In the problem formulation for 1,4-dioxane, EPA identified a past condition of use—use as a fuel or fuel additives—that it excluded from its risk evaluation. Problem Formulation for 1,4-Dioxane at 18. Past conditions of use that are not currently ongoing are “known” to have occurred in the past, and these conditions of use are definitely “reasonably foreseen.” 15 U.S.C. § 2602(4). Congress included “reasonably foreseen” circumstances within TSCA with the express goal of ensuring that EPA swept more broadly than known (or intended) uses; EPA cannot evade that duty by limiting its analysis to conditions of use with evidence of current, ongoing use—such an interpretation would effectively limit EPA’s analysis to “known” uses. While there may well be circumstances in which a use that is not currently occurring could be said to be not “reasonably foreseen” at this time, the term surely cannot be read in such a way that only uses that are known to be current are “reasonably foreseen” as that would read it out of existence and collapse the inquiry to one where a use must be “known” to be considered “reasonably foreseen.”

Reasonably foreseen is a term of art with a long history in the law; it is well established under the law that “[a] natural and probable consequence is a foreseeable consequence. But to be reasonably foreseeable [t]he consequence need not have been a strong probability; a possible consequence which might reasonably have been contemplated is enough.” *People v. Medina*, 209 P.3d 105, 110 (Cal. 2009) (internal citations and quotation marks omitted). Numerous courts have recognized that circumstances are reasonably foreseen when similar circumstances have occurred in the past. *See, e.g., McKown v. Simon Prop. Grp., Inc.*, 344 P.3d 661, 663 (Wash. 2015); *Burns v. Penn Cent. Co.*, 519 F.2d 512, 515 (2d Cir. 1975). The fact that these conditions of use occurred in the past establishes that they are reasonably foreseen.

It is hard to see how the mere cessation of use, particularly if it ceased recently, is by itself sufficient to render the use not “reasonably foreseen.” The concept of “reasonably foreseen” wraps in uses that have never before existed if there is a logical rationale for thinking that such a use could occur; if a use has actually occurred, but merely halted, it is clearly not speculation that the chemical substance being evaluated could be used in that way; it is only a question of how likely it is that the chemical could be used that way again. EPA, however, does not appear to have undertaken such analyses. Rather, in the problem formulation, the Agency seems to assume that because certain (unidentified) racing authorities had indicated that they had banned the use of 1,4-dioxane in competitions, it would never be used as a fuel additive. Problem Formulation for 1,4-Dioxane at 18. But this analysis is utterly unconvincing. The very fact that

¹⁶⁹ EDF Comments on Ten Scopes under the Toxic Substances Control Act, pp. 4-11 (Sept. 2017), <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0743-0069>; *see also* Appendix VI and Appendix VII.

racing authorities had to ban its use as a fuel or fuel additive indicates that the use is reasonably foreseeable, and of course, 1,4-dioxane could be used as a fuel or fuel additive in other circumstances even if it is banned in certain specific competitions. Moreover, these communications with the racing authorities do not appear to be publicly available for consideration by the public. Based on the statements in the problem formulation, EPA has not even established that 1,4-dioxane has been phased out of use in fuel, much less that it could not resume easily.

As EPA itself acknowledged in its recently proposed significant new use rule for certain uses of asbestos, absent a regulation governing the resumption of an old condition of use, “the importing or processing of” a chemical for a past use that is no longer ongoing “may begin at any time.” 83 Fed. Reg. at 26,927. Thus, the condition of use is reasonably foreseen absent a legal ban on it. Even if a chemical is no longer used for a particular condition of use, persons may resume past uses in response to economic, regulatory, or other changes. *If* a chemical had a particular condition of use in the past, EPA should analyze that condition of use absent compelling evidence that the use will not resume in the future. Here, there is evidence that 1,4-dioxane has been used in fuel, and EPA should analyze that condition of use unless there is compelling evidence that the use will not resume in the future.

5. EPA cannot ignore ongoing, real-world exposures because they are occurring despite another statute that could potentially cover those exposures.

As established above, EPA must assess all hazards and exposures when evaluating the risk presented by a chemical substance. For this same reason, EPA must consider all real-world, intended, and reasonably foreseen exposures that occur even if they fall under the jurisdiction of other statutes. In the problem formulation for 1,4-dioxane, EPA excluded numerous exposure “pathways under other environmental statutes, administered by EPA, which adequately assess and effectively manage exposures and for which long-standing regulatory and analytical processes already exist.” Problem Formulation for 1,4-Dioxane at 42. Specifically, EPA excluded the drinking water pathway, ambient water pathways, the ambient air pathway, and disposal pathways. Problem Formulation for 1,4-Dioxane at 42-46. In the draft risk evaluation, EPA continues to exclude these pathways (p. 156).

EPA’s approach is illegal and arbitrary and capricious for numerous reasons, including because TSCA requires EPA to analyze all exposures for the reasons discussed above. This approach also violates the text and structure of TSCA for additional reasons unique to this rationale for excluding exposures.

As discussed in more detail below, first and foremost this approach is factually and scientifically inaccurate. For numerous sources of exposure, EPA treats the overall exposure from a particular pathway as “zero” or non-existent despite the fact that the available evidence thoroughly

establishes that exposure is occurring at levels well above zero regardless of any actions taken under the other statutes EPA invokes. Thus, in reality, human beings and the environment are experiencing levels of exposure that EPA is willfully ignoring. EPA is choosing to adopt false factual assumptions, and “[r]eliance on facts that an agency knows are false at the time it relies on them is the essence of arbitrary and capricious decisionmaking.” *Animal Legal Def. Fund, Inc. v. Perdue*, 872 F.3d 602, 619 (D.C. Cir. 2017). This approach also violates the requirements to act “consistent with the best available science” and to “take into consideration information relating to a chemical substance or mixture, including hazard and exposure information, under the conditions of use, that is reasonably available to the Administrator.” 15 U.S.C. § 2625(h), (k). Thus, for example, in its problem formulation for 1,4-dioxane, EPA states that its inclusion criteria for data sources reporting environmental fate data expressly do not include consideration of vast swathes of data:

For 1,4-dioxane no exposure pathways to human and ecological receptors from consumer products, environmental releases, or waste streams associated with industrial and commercial activities will be further analyzed in risk evaluation. In the absence of exposure pathways for further analysis, environmental fate data will not be evaluated further. Therefore, no [Population, Exposure, Comparator, and Outcome] statement or fate data needs and associated processes, media and exposure pathways considered in the development of the environmental fate assessment for 1,4-dioxane will be presented.

Problem Formulation for 1,4-Dioxane at 90. The draft risk evaluation establishes how much evidence EPA excludes as a result of this approach. For example, for environmental fate and transport data sources, EPA excluded 2,939 of 2,940 references because “EPA determined during problem formulation that no environmental pathways would be further analyzed” (p. 38). But the problem formulation and draft risk evaluation do not establish that the regulation of 1,4-dioxane under other statutes will eliminate exposures, and in fact, the publicly available evidence all establish that exposures continue to occur in the real-world despite these statutes. EPA cannot ignore those exposures.

In addition, EPA must consider the possibility that these exposures, *combined with other sources of exposure*, could present an unreasonable risk. EPA’s decision to ignore exposures one-by-one rather than look at combined exposure is inherently inaccurate and will invariably lead to an underestimation of exposure and risk.

Furthermore, EPA has not established that these environmental statutes “adequately assess and effectively manage exposures” (p.156); Problem Formulation for 1,4-Dioxane at p. 37. EPA’s bald assertions to the contrary do not make it so. In particular, as detailed below, EPA has completely failed to regulate 1,4-dioxane in drinking water and ambient water, so EPA cannot rationally state that it has adequately assessed and effectively managed those exposures.

In any event, that is not the legally correct standard under TSCA. As explained below, EPA can only rely on statutory authorities other than TSCA in compliance with TSCA § 9 (notably, the TSCA § 9 process occurs after EPA has completed a comprehensive risk evaluation finding unreasonable risk). To comply with TSCA § 9, EPA must find that those authorities eliminate the risks EPA has previously identified or reduce them to a sufficient extent under TSCA § 9(b)(1), and TSCA requires that EPA reduce risk “to the extent necessary so that [the chemical] no longer presents [an unreasonable risk of injury to health or the environment].” *See* 15 U.S.C. §§ 2608(b)(1), 2605(a). In addition, under TSCA § 9(b)(2) EPA must consider “all relevant aspects of the risk” when deciding whether to regulate under TSCA or another statute. *Id.* § 2608(b)(2). EPA has not met any of these standards in the problem formulations, and EPA’s statements that the exposures are adequately assessed and effectively managed under other statutes are legally irrelevant (even if they were true).

When relying on these other statutory authorities, EPA merely provides a list of various regulatory standards and criteria that EPA indicates apply or could apply to certain sources of the chemicals. EPA provides no analysis whatsoever as to: the extent to which the standards or criteria cover the full range of exposure to the chemical through the pathway; the extent and magnitude of releases of the chemical allowed under each of the regulatory standards or criteria; or any other factors that would be necessary to analyze to determine the extent and nature of potential risk allowed under the standards. In particular, TSCA § 6(b)(4)(F)(iv) requires that, in conducting a risk evaluation, EPA evaluate “the likely duration, intensity, frequency, and number of exposures,” 15 U.S.C. § 2605(b)(4)(F)(iv), including exposures resulting from those allowable emissions, discharges, or releases. EPA needs to provide this analysis, and EPA cannot simply point to regulation under another statute to bypass the analysis. EPA has also not acknowledged, let alone analyzed, the overall risks to the general population or to vulnerable subpopulations due to the combination of exposures arising from the various sources for which standards exist, not to mention in combination with additional emission sources not subject to any standard. EPA has made no attempt to reconcile any such risk with that allowed under TSCA.

EPA offers only vague claims, such as that EPA “as appropriate, has reviewed, or is in the process of reviewing remaining risks.” Problem Formulation for 1,4-Dioxane at 43. No specifics as to the status of or timeline for such reviews have been provided, and no indication is made as to when and on what basis such reviews are deemed “appropriate.” Nor have the results of any such reviews, if they have been completed, been provided, let alone analyzed in the context of TSCA’s requirements.

EPA also justifies these exclusions on the theory that other exposures are “likely to represent the greatest areas of concern to EPA,” (p. 156). EPA provides no factual support for this assertion, and in the case of 1,4-dioxane, it is unsupported. EPA has excluded all exposures to the general population, despite exposures to 1,4-dioxane through drinking water and ambient water

that raise health concerns, according to EPA's own analyses. EPA's own problem formulation acknowledges that 6.9% of monitored water systems measured levels of 1,4-dioxane that correspond to a 1 in a million-lifetime cancer risk. Problem Formulation for 1,4-Dioxane at p.43. EPA has presented no reasoning or evidence that these exposures are not areas of great concern.

At a minimum, EPA has completely failed to establish that these statutes reduce exposure to zero. To the contrary, it is thoroughly clear that humans and the environment continue to experience significant exposures through the excluded pathways. To prepare a scientifically accurate risk evaluation, EPA must analyze the exposures through those pathways.

A. The text and overall structure of TSCA makes it clear that EPA has to analyze exposures, even if they have been or could be assessed under another statute.

In the problem formulation, EPA asserts that it has discretion to exclude "certain exposure pathways that fall under the jurisdiction of other EPA-administered statutes." *See, e.g.*, Problem Formulation for 1,4-Dioxane at p. 11. But EPA provides no textual basis for ignoring those exposures. Instead, in a footnote, EPA cites to its discussion regarding "conditions of use," but even assuming for the sake of argument that EPA has authority to exclude conditions of use, such power does not justify excluding exposures related to conditions of use still within the scope of the risk evaluation, as EPA proposes to do. Nothing in TSCA's risk evaluation provision authorizes EPA ignoring exposures because of other statutory authorities, and as explained above, EPA has to analyze all exposures including these exposures. And several other provisions of TSCA indicate that Congress intended for EPA to consider such exposures, except to the extent Congress explicitly provided otherwise.

First, Congress expressly excluded certain chemicals or uses of chemicals regulated under other statutes when it defined "chemical substance" in TSCA § 3(2). 15 U.S.C. § 2602(2)(B). For example, "chemical substance" does not include "any pesticide (as defined in the Federal Insecticide, Fungicide, and Rodenticide Act) when manufactured, processed, or distributed in commerce for use as a pesticide." *See id.* § 2602(2)(B)(ii). Thus, when Congress intended for EPA not to regulate certain exposures because they were regulated under other specific EPA-administered statutes, Congress expressly excluded those exposures. That Congress chose a limited, specific set of exclusions indicates that Congress did not intend for EPA generally to ignore other exposures where they fall under other federal regulatory schemes.

Second, in TSCA's risk evaluation provision, Congress specifically intended for EPA to "conduct risk evaluations *** to determine whether a chemical substance presents an unreasonable risk of injury to *** the environment," 15 U.S.C. § 2605(b)(4)(A), but EPA's approach has eliminated almost all analysis of environmental exposures. EPA has largely read the requirement to evaluate risks to the environment out of the statute, but this approach violates

a fundamental tenant of statutory interpretation. A. Scalia & B. Garner, *Reading Law: The Interpretation of Legal Texts* 174 (2012) (“If possible, every word and every provision is to be given effect *** None should needlessly be given an interpretation that causes it to duplicate another provision or to have no consequence.”). Moreover, Congress enacted this requirement that EPA analyze risks to the environment against the backdrop of the existing environmental statutes; if Congress had considered them per se sufficient, Congress would not have included this mandate in TSCA. But Congress did.

Third, Congress specifically directed EPA to analyze the risks of chemicals presented “under the conditions of use,” and Congress consciously decided to specify that “disposal” is a condition of use under TSCA. “Conditions of use” expressly includes “the circumstances *** under which a chemical substance is intended, known, or reasonably foreseen to be to be manufactured, processed, distributed in commerce, used, or *disposed of*.” 15 U.S.C. § 2602(4) (emphasis added). In the problem formulations, EPA systematically excludes exposures through disposal based on a variety of theories, and in doing so, EPA is ignoring Congress’s direction that it assess risks associated with the conditions of use, including disposal. Similarly, EPA is ignoring exposures from other conditions of use, such as “manufactur[ing],” “process[ing],” and potentially distribution in commerce, by for example ignoring the emissions from the manufacturing and processing facilities. Congress expressly included all of these circumstances within the definition of “conditions of use,” and EPA should not ignore the exposures resulting from them.

Fourth, TSCA § 9(b) provides that EPA “shall coordinate *actions* taken under [TSCA] with *actions* taken under other Federal laws administered in whole or in part by the Administrator.” 15 U.S.C. § 2608(b) (emphases added). While EPA is supposed to coordinate the “actions” under each statute, this provision does not contemplate EPA excluding exposures from the analyses prepared under TSCA. Indeed, the remaining language of TSCA § 9(b) highlights that Congress intended for EPA to prepare risk evaluations analyzing all exposures, including those that might be addressed under another authority.

Under TSCA § 9(b)(1), EPA can only choose to rely on other authorities “[i]f [EPA] determines that a risk to health or the environment associated with a chemical substance or mixture *could be eliminated or reduced to a sufficient extent* by actions taken under the authorities contained in such other Federal laws.” 15 U.S.C. § 2608(b)(1) (emphasis added). Thus, Congress provided a standard that EPA must meet before relying on other authorities: with respect to the “risk to health or the environment” presented by a chemical, the other authority must either “eliminate[]” that risk or “reduce [the risk] to a sufficient extent.” *Id.* Reduction in risk must be “sufficient” as defined by TSCA, and the word “extent” cross-references the basic standard set forth in section 6(a). *See* 15 U.S.C. § 2605(a). Section 6(a) provides that if EPA determines that a substance or mixture “presents an unreasonable risk of injury to health or the environment,” EPA

“shall” apply requirements to the “substance or mixture to the extent necessary so that the chemical substance or mixture no longer presents such risk.” *Id.* Thus, EPA may only rely on actions under another statute if those actions will reduce an identified risk “to the extent necessary so that [it] no longer presents [an unreasonable risk of injury to health or the environment].” EPA cannot assume that other statutes, with different standards, meet the requirements of TSCA.

TSCA requires that EPA eliminate the “unreasonable risk,” *id.* and that unreasonable risk of injury to health or the environment must be identified under TSCA § 6(b)(4)(A) “without consideration of costs or other nonrisk factors, including an unreasonable risk to a potentially exposed or susceptible subpopulation identified as relevant to the risk evaluation by the Administrator.” 15 U.S.C. § 2605(b)(4)(A). Thus, TSCA’s standard requires EPA to resolve risks identified without consideration of costs or other nonrisk factors, and EPA must specifically consider risks to vulnerable subpopulations. Generally speaking, the other EPA-administered statutes do not have this same standard. Some of these statutes allow consideration of nonrisk factors and do not explicitly require consideration of vulnerable subpopulations. EPA cannot simply assume that regulatory efforts that meet the requirements of those statutes will also meet TSCA’s requirement that EPA eliminate unreasonable risks. And Congress’s decision to enact the TSCA standard reflects that Congress wanted EPA, when implementing TSCA, to meet that standard; EPA cannot rely on its fulfillment of a different standard under a different statute to evade that duty.

Under TSCA § 9(b)(2) Congress directed EPA to consider certain factors to resolve overlaps in EPA’s statutory jurisdictions after completing the risk evaluation. Specifically, in determining whether to address a risk under TSCA or another statutory authority administered by EPA, EPA “shall consider, based on information reasonably available to the Administrator, all relevant aspects of the risk,” among other things. *Id.* § 2608(b)(2). Thus, EPA has to analyze “all relevant aspects of the risk” in its risk evaluations, *before* deciding whether to address particular risks through TSCA or another statutory authority. Congress would not have included this requirement if Congress had meant for EPA to simply defer to current regulatory approaches to those chemicals at the outset before conducting a risk evaluation.

Among other concerns, if EPA just ignores risks arising from exposures that fall within other statutes’ jurisdiction, then EPA will lack the information necessary to prepare the necessary analyses under TSCA § 9(b)(2). TSCA § 9(b) clearly contemplates that EPA will analyze all these exposures in risk evaluations and then meet its duties under TSCA § 9(b) based, in part, on the analyses prepared in the risk evaluations. As reflected in TSCA § 6, Congress expressly chose to separate risk evaluation and risk management into different procedural steps (with risk evaluation preceding risk management), to ensure that EPA provided a robust risk evaluation uncolored by nonrisk factors or other risk management concerns.

Notably, in its problem formulation, EPA makes no showing that its actions under other statutes reduce the risk “to the extent necessary so that [it] no longer presents [an unreasonable risk of injury to health or the environment],” and EPA does not present any actual analysis of “all relevant aspects of the risk” arising from the ignored exposures. So EPA has undisputedly failed to comply with TSCA § 9(b). Given that Congress expressly addressed the issue of overlapping regulatory jurisdictions in TSCA § 9, EPA cannot avoid those procedures by simply ignoring exposures that fall within another statute’s jurisdiction.

Furthermore, EPA is expressly required to evaluate exposures from combinations of activities, which it cannot do if it excludes some exposures at the outset that may be able to be addressed under another authority, particularly when any risk management under the other authority would not reduce exposure to zero.

B. EPA’s approach to the general population and subpopulations highlights that its decision to exclude exposures under other EPA-administered statutes is illegal and arbitrary and capricious.

- i. EPA must analyze whether 1,4-dioxane presents a risk to the general population because the record establishes that the general population is exposed to 1,4-dioxane.*

In its draft risk evaluation, EPA states that it will not analyze general population exposures for 1,4-dioxane because EPA considers its existing regulatory programs sufficient (p. 156). EPA’s approach is illegal for the reasons given above. In addition, the reasonably available information establishes that the general population experiences significant exposures to these chemicals, and it is irrational to ignore those exposures in light of this evidence.

In the problem formulation, EPA acknowledges that the general population may be exposed from inhalation of ambient air, through drinking water, and exposure during washing and bathing. Problem Formulation for 1,4-Dioxane at p. 31; see also Part II. sec. 2.

Given ample evidence that the general population in fact experiences exposures to these chemicals under EPA’s current regulatory regimes, it is arbitrary and capricious for EPA to adopt an approach to risk evaluation that disregards the risks presented to the general population.

- ii. EPA cannot accurately evaluate potentially exposed or susceptible subpopulations such as fenceline communities if EPA excludes the vast majority of exposure pathways leading to their greater exposure.*

In the problem formulation, EPA correctly recognized that a potentially exposed or susceptible subpopulation includes those “groups of individuals within the general population who may experience greater exposures due to their proximity to conditions of use identified in Section 2.2

that result in releases to the environment and subsequent exposures (e.g., individuals who live or work near manufacturing, processing, distribution, use or disposal sites).” *See, e.g.*, Problem for 1,4-Dioxane at p. 32. But EPA then ignores the vast majority of pathways that cause these groups to face greater exposures—such as through releases to air, water, and land. EPA provides no rational explanation for how it will accurately and effectively evaluate the actual risk faced by these subpopulations while ignoring these exposures. Moreover, EPA’s (correct) recognition that these groups face greater exposure highlights that it is irrational for EPA to ignore the pathways leading to these exposures.

As a result, in the draft risk evaluation, EPA entirely fails to analyze this potentially exposed or susceptible subpopulation (pp. 151-52). EPA limits its analysis of greater exposure to workers, and EPA completely ignores the greater exposure experienced by individuals living in proximity to conditions of use. As a result, EPA fails to consider an important aspect of the problem because EPA fails to analyze the risks posed to a potentially exposed or susceptible subpopulation that EPA previously acknowledged.

In addition, TSCA specifically requires that EPA protect these subpopulations because they face greater potential exposure. And, EPA’s existing regulations under other statutes, which may not have been developed with a focus on these particular subpopulations, may not always be “sufficient” under the TSCA standard.

C. EPA’s failure to regulate 1,4-dioxane in drinking water does not justify EPA’s decision to ignore exposures through drinking water; EPA should analyze the real-world exposures to 1,4-dioxane.

EPA is excluding exposures to 1,4-dioxane through drinking water on a particularly irrational and illegal basis. Specifically, EPA has not yet established any regulatory standard for 1,4-dioxane under the Safe Drinking Water Act (SDWA). Instead, it is on the Contaminant Candidate List, which EPA acknowledges “is a list of *unregulated* contaminants that are known or anticipated to occur in public water systems and that may require regulation.” Problem Formulation for 1,4-Dioxane at p. 43 (emphasis added). By EPA’s own acknowledgement, there are likely exposures to 1,4-dioxane through drinking water systems and it remains unregulated.

This approach is unreasonable for the reasons given above, but in addition, EPA does not even have the fig-leaf that 1,4-dioxane is regulated under SDWA. Numerous additional steps would need to be taken to actually regulate 1,4-dioxane under SDWA, which have not been taken. The vague statement that the chemical is “currently being evaluated”—with no specification of what outcomes may result or any timeline for further action toward regulation—provides no basis for EPA’s assertion that its risks are being “adequately assess[ed] and effectively manage[d].” Problem Formulation for 1,4-Dioxane at pp. 42-43. An agency cannot ignore ongoing, current exposures on the theory that the agency might regulate that exposure at some uncertain point in

the future. If a regulation is not legally in-place and in-force, EPA cannot rationally give it any weight. Among other things, it would be arbitrary and capricious to consider speculative future regulations that have not been promulgated through rulemaking and do not yet have legal effect.

EPA also cannot reasonably assume that it will know whether a final regulation will be finalized or, if so, the final regulation's conditions, until it has entered into and completed the notice-and-comment process for the regulation. *See Nat'l Rest. Ass'n v. Solis*, 870 F. Supp. 2d 42, 50 (D.D.C. 2012) (“[C]omments received by the agency are expected to shape the outcome of a final rule.”). “The whole rationale of notice and comment rests on the expectation that the final rules will be somewhat different and improved from the rules originally proposed by the agency.” *Trans-Pac. Freight Conf. of Japan/Korea v. Fed. Mar. Comm'n*, 650 F.2d 1235, 1249 (D.C. Cir. 1980). Thus, EPA cannot assume that any (entirely speculative) future regulation under SDWA would provide adequate protection.

Moreover, the data in the problem formulation establishes that exposures through drinking water to 1,4-dioxane are likely and a cause for concern. As a factual matter, these exposures are occurring and EPA must consider them; for EPA to treat these exposure levels as “zero” when they are known not to be does not comport with the best available science. Of the 4,915 water systems monitored, 1,077 systems had detections of 1,4-dioxane in at least one sample. Problem Formulation for 1,4-Dioxane at p. 43. “341 systems (6.9%) had results at or above 0.35 µg/L (which corresponds to a 1 in a million-lifetime cancer risk).” *Id.* “Reported levels of 1,4-dioxane in groundwater range from 3 to 31,000 µg/L (ATSDR, 2012; USGS, 2002).” *Id.* at 28. EPA also acknowledged that some studies report 1,4-dioxane in surface water, though data are more limited and further study of surface water levels seems appropriate. *Id.* To ignore drinking water exposure when 1,4-dioxane has often been reported at hazardous levels is fundamentally arbitrary and capricious and a threat to public health. Given evidence of real-world exposure, EPA must assess these exposures in its risk evaluation. EPA cannot rationally exclude them from analysis.

In seeking to justify its exclusion of drinking water pathways, EPA states:

EPA's Office of Water has established a Health Advisory level of 35 µg/L (which corresponds to a 1 in ten thousand lifetime cancer risk) for 1,4-Dioxane.

Problem Formulation for 1,4-Dioxane at p. 43.

This statement is highly misleading as it misconstrues the context and purpose of Health Advisories. Here is what the Office of Water's own Health Advisory 2018 compilation states:

HAs [Health Advisories] are intended to protect against *noncancer effects*. The 10^{-4} Cancer Risk level provides information concerning cancer effects.¹⁷⁰

EPA's own November 2017 Fact Sheet on 1,4-dioxane – never mentioned in the problem formulation – cites the 1,000-fold lower level of 0.35 ug/L that corresponds to EPA's typical cancer risk protection goal for general populations of 10^{-6} (a 1-in-one-million lifetime cancer risk). It also shows that most states have guidelines far below 35 ug/L.¹⁷¹ EPA uses this value to assert that there are no exceedances for 1,4-dioxane, but in fact, as EPA notes, nearly 7% of public water systems – serving 7 million Americans – exceed the risk level representing acceptable risk to the general population.

EPA's decision to ignore such clearly significant levels of exposure and risk, and to refuse to evaluate their contribution to the overall risks of this chemical, is unconscionable.

Even if EPA were to eventually develop a Maximum Contaminant Level (MCL) for 1,4-dioxane under SDWA, it would still be arbitrary and capricious and illegal to ignore drinking water exposures to 1,4-dioxane. First, to the extent available evidence reveals that exposure happens through a certain pathway, EPA must analyze that exposure. Analyzing exposure through drinking water is particularly important for EPA to obtain an accurate estimate of the exposure of infants and children, often a potentially exposed or susceptible subpopulation. *See, e.g.*, U.S. EPA, Problem Formulation of the Risk Evaluation for Perchloroethylene (Ethene, 1,1,2,2-Tetrachloro) (May 2018), p.48 <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0732-0080> (“Drinking water could be a significant source of perchloroethylene ingestion exposure for children, who drink roughly four times as much water as adults.”).

In addition, MCLs are only set at the level “feasible” which refers to both the ability to treat water to meet the MCL and the ability to monitor water quality at the MCL. Thus, MCLs are based on non-risk factors and do not necessarily eliminate exposures. Specifically, the contaminant level set under the SDWA considers “non-risk” factors, and the MCL is not sufficient to eliminate risks. While EPA must set a maximum contaminant level goal (MCLG) that is fully protective of health for drinking water contaminants, 42 U.S.C. § 300g-1(b)(1)(E); *see also* 42 U.S.C. § 300g-1(b)(4)(A), the MCLG is not the national drinking water standard. Rather, the agency must establish a maximum contaminant level (MCL) that is as close to the MCLG “as is feasible,” considering technological limitations and costs, and promulgate a national primary drinking water regulation (NPDWR) for the contaminant based on the MCL.

¹⁷⁰ U.S. EPA, *2018 Edition of the Drinking Water Standards and Health Advisories Tables* at p. iii (Mar. 2018), <https://www.epa.gov/sites/production/files/2018-03/documents/dwtable2018.pdf> (emphasis added).

¹⁷¹ U.S. EPA, *Technical Fact Sheet 1,4-dioxane* at 3-4 (Nov. 2017), https://www.epa.gov/sites/production/files/2014-03/documents/ffrro_factsheet_contaminant_14-dioxane_january2014_final.pdf.

42 U.S.C. § 300g-1(b)(4)(B). In other words, the contaminant level EPA actually sets for safe drinking water is less protective than the MCLG because it accounts for feasibility and costs, which are non-risk factors that EPA may not consider during the risk evaluation process.

In addition, the SDWA does not regulate all sources of drinking water. It is estimated that more than 13 million households rely on private wells for drinking water in the United States.^{172,173} The national drinking water standards established under the SDWA do not apply to private wells. *See* 42 U.S.C. § 300f(1) (a “primary drinking water regulation” only applies to “public water systems”); 42 U.S.C. § 300f(4)(A) (a “public water system” is a system that “has at least fifteen service connections or regularly serves at least twenty-five individuals”). Therefore, exposures in drinking water from private wells is not addressed by the SDWA and would need to be evaluated in the draft risk evaluation.

D. EPA’s failure to regulate 1,4-dioxane under the Clean Water Act does not justify EPA’s decision to ignore exposures through ambient water; EPA should analyze the real-world exposures.

EPA is excluding exposures to 1,4-dioxane through ambient water on an irrational and illegal basis. *See* Problem Formulation for 1,4-Dioxane at pp. 43-44. EPA discusses the issue of a water quality criterion for 1,4-dioxane, but EPA never acknowledges that it has not yet set a human health criterion for 1,4-dioxane.¹⁷⁴ Yet inexplicably, EPA excludes ambient water pathways from the risk evaluation of human health risks to the general population (p. 156). EPA has provided no justification for this exclusion. As EPA itself later admits in the problem formulation, only a single state has developed a water quality standard for human health for 1,4-dioxane. *See, e.g.*, Problem Formulation for 1,4-Dioxane at pp. 44 (“Currently, only one state (Colorado) includes human health criteria for 1,4-dioxane in their water quality standards.”). EPA’s failure to regulate 1,4-dioxane under the Clean Water Act cannot justify EPA’s decision to exclude this pathway, for reasons previously articulated in Part II sec. 5.A.

Moreover, the factual record establishes that 1,4-dioxane is present in water sources, and EPA should use its information authorities to obtain needed additional information about its presence in ambient water. EPA has evidence of 1,4-dioxane in drinking water supplies, as described above, and evidence of 1,4-dioxane in groundwater. Problem Formulation for 1,4-Dioxane at p.

¹⁷² PRIVATE DRINKING WATER WELLS, <https://www.epa.gov/privatewells> (last visited Jul. 31, 2018) (citing the US Census American Housing Survey 2015).

¹⁷³ An estimated 44.5 million people in the United States, or 14 percent of the population, provided their own water for domestic use in 2010. U.S. Geological Survey, *Estimated Use of Water in the United States in 2010* (2014), <https://pubs.usgs.gov/circ/1405/pdf/circ1405.pdf>.

¹⁷⁴ *See* NATIONAL RECOMMENDED WATER QUALITY CRITERIA - HUMAN HEALTH CRITERIA TABLE, <https://www.epa.gov/wqc/national-recommended-water-quality-criteria-human-health-criteria-table> (last visited Aug. 16, 2018).

28. EPA has acknowledged that it has “relatively fewer data available on 1,4-dioxane in surface water,” so EPA should use its information authorities to obtain more data. *Id.*

Even if EPA were to develop a water quality criterion for 1,4-dioxane, that would not justify ignoring 1,4-dioxane exposure through ambient water. Under the CWA, EPA establishes recommended water quality criteria, but not all states have updated their criteria to reflect the current CWA criteria. See 80 Fed. Reg. 36,986 (June 29, 2015). There is often significant variation between EPA’s recommended criteria and the criteria adopted by the states. Given that some states have water quality criteria that are significantly less protective than EPA’s recommendations, EPA cannot rely on its recommendations to assume that the risks are adequately managed, much less that they result in zero exposure.

E. Real-world exposures still occur through groundwater, and EPA cannot ignore those real-world exposures when assessing the risk presented by 1,4-dioxane, particularly since EPA cannot identify any statute that allegedly addresses groundwater.

In the problem formulation, EPA stated that: “Reported levels of 1,4-dioxane in groundwater range from 3 to 31,000 µg/L (ATSDR, 2012; USGS, 2002). Such instances of ground water contamination with 1,4-dioxane are documented in the states of California and Michigan. These data provide a basis for including groundwater in the scope of the 1,4-dioxane risk evaluation from manufacturing, processing, distribution and use unless otherwise regulated or managed.” Problem Formulation for 1,4-Dioxane at p.28. Thus, EPA’s problem formulation initially appeared to state that EPA would analyze exposure through groundwater.

But later in the problem formulation, EPA stated that it did not plan to further analyze background levels of exposure to groundwater. *Id.* at 47. And the draft risk evaluation does not include an analysis of groundwater exposure, stating that “[w]hile 1,4-dioxane is present in various environmental media such as groundwater, surface water, and air, EPA determined during problem formulation that no further analysis beyond what was presented in the problem formulation document would be done for those environmental exposure pathways in this draft risk evaluation” (p. 19).

EPA appears to be excluding groundwater from analysis for reasons similar to its reasons for ignoring all other environmental exposures, but EPA does not even identify a statute or regulation that allegedly “adequately assess[es] and effectively manage[s] these] exposures” (p. 156); Problem Formulation for 1,4-Dioxane at p. 37. EPA cannot rationally exclude groundwater exposure from its analysis when EPA cannot even identify a statute that allegedly will address this source of exposure.

Moreover, EPA's cursory analysis of biosolids highlights how arbitrary and capricious it is for EPA to exclude groundwater, ambient water, or air pathways. Basically, EPA dismisses biosolids exposure, in part, because 1,4-dioxane "is expected to be mobile in soil and to migrate to surface waters and groundwater or volatilize to air" (p. 131). In other words, when dismissing biosolids exposures, EPA emphasizes that 1,4-dioxane will migrate to water and air, but EPA then elsewhere refuses to analyze the water and air pathways for 1,4-dioxane. This inconsistent and incoherent approach is arbitrary and capricious and fails to reflect the best available science.

F. Real-world exposures still exist through the air pathway even though 1,4-dioxane is listed as a hazardous air pollutant; EPA should analyze the real-world exposures.

EPA excluded exposures 1-4 dioxane through the air pathway because it is listed as a hazardous air pollutant (HAP) under the Clean Air Act (CAA). Problem Formulation for 1,4-Dioxane at pp. 42-43. This approach is unreasonable for the reasons given above, but in addition, EPA has not made the necessary showing that the established HAPs eliminate any unreasonable risk and EPA has not assessed all relevant aspects of the risk. As EPA acknowledges in the problem formulation, the listing as a HAP leads to a technology-based standard for certain stationary sources. *See, e.g.*, Problem Formulation for 1,4-dioxane at p. 42. Such regulations do not necessarily eliminate exposures. Moreover, EPA is relying on "technology-based" standards, but under TSCA § 9, EPA can only rely on another statutory authority if it reduces exposures "to a sufficient extent" under TSCA, 15 U.S.C. § 2608(b)(1), and TSCA specifically requires that EPA eliminate the unreasonable risk, see 15 U.S.C. § 2605(a), without reference to technology. EPA cannot assume that other statutes, with different standards, meet the requirements of TSCA.

i. EPA's Clean Air Act authority is not a comprehensive substitute for TSCA.

EPA's mandate to control toxic air pollutants under the Clean Air Act (CAA) differs from TSCA's provisions applicable to the same substances and thus does not presumptively address the same scope of risks. EPA points to CAA Sections 111 and 112, 42 U.S.C. §§ 7411-12, as an adequate proxy for TSCA regulations that would address the "ambient air pathway" of exposure to toxic air pollutants covered under both statutes, yet the statutory structures that empower EPA to control these pollutants through CAA regulation are different from EPA's authority to regulate or even prohibit the production or use of these substances under TSCA.

CAA Sections 111 and 112 differ in scope and approach as compared to TSCA. EPA points to CAA Section 112 which requires EPA to promulgate regulations applicable to sources of listed hazardous air pollutants including 1,4-dioxane. Section 112 instructs EPA to list and regulate substances for which "emissions, ambient concentrations, bioaccumulation or deposition of the substance are known to cause or may reasonably be anticipated to cause adverse effects to human health or adverse environmental effects." 42 U.S.C. § 7412(b)(2). As EPA acknowledges, under the CAA "For stationary source categories emitting [Hazardous Air

Pollutants] HAP, the CAA requires issuance of technology-based standards and, if necessary, additions or revisions to address developments in practices, processes, and control technologies, and to ensure the standards adequately protect public health and the environment.” Problem Formulation for 1,4-Dioxane at p. 42. Under section 112(d)(1), EPA sets source-specific “standards for each category or subcategory of major sources and area sources of hazardous air pollutants listed.” 42 U.S.C. § 7412(d)(1). This source-specific regulatory scheme requires EPA to:

require the maximum degree of reduction in emissions of the hazardous air pollutants subject to this section (including a prohibition on such emissions, where achievable) that the Administrator, taking into consideration the cost of achieving such emission reduction, and any non-air quality health and environmental impacts and energy requirements, determines is achievable for new or existing sources in the category or subcategory to which such emission standard applies.

Id. § 7412(d)(2). This approach reflected in section 112 is distinct from TSCA which empowers EPA look at the risk posed by the chemical broadly without necessarily focusing on source-specific technology, costs of regulation, or what standards are “achievable” for each source category. Indeed, as explained previously, TSCA requires that EPA evaluate a chemical’s risk “without consideration of costs or other nonrisk factors.” 15 U.S.C. § 2605(b)(4)(A). In addition, TSCA requires EPA to consider the “conditions of use” of a chemical, with no distinction drawn between stationary sources and other sources. As a result, EPA cannot presumptively assume that section 112 regulation would necessarily address all the risks that TSCA requires the agency to identify and ameliorate.

Similarly, EPA points to CAA Section 111, 42 U.S.C. § 7411, as a basis for declining to evaluate risks associated with the ambient air pathway under TSCA. But, like section 112, section 111 differs in material respects from the approach embodied in TSCA. Section 111 requires EPA to set and periodically update standards of performance for categories of new stationary sources and existing stationary sources of pollution that cause or contribute “significantly, to air pollution which may reasonably be anticipated to endanger public health or welfare.” 42 U.S.C. § 7411(b). In setting “standard[s] of performance” for each source category or even sub-category of sources, EPA must select a standard that “reflects the degree of emission limitation achievable through the application of the best system of emission reduction which (taking into account the cost of achieving such reduction and any nonair quality health and environmental impact and energy requirements) the Administrator determines has been adequately demonstrated.” 42 U.S.C. § 7411(a)(1). TSCA’s regime likewise diverges from this approach in its focus on the risks posed by chemical substances and EPA actions that can ameliorate those risks.

In addition to these substantive differences, existing standards under sections 111 and 112 are subject to different procedural requirements. For example, the CAA’s source-specific standards

under Section 111 are structured around a series of 8-year intervals for review and Section 112’s list of substances is reviewed every 5 years, along with other periodic reviews called for under Section 112. EPA is also subject to a series of consent decrees for required reviews under Section 112(f)(2) and Section 112(d)(6), often setting longer timelines for new rulemaking. As a result, many of the category specific regulations under these provisions are in various stages of being updated. Accordingly, even if there were some substantive alignment between TSCA and the CAA provisions EPA cites—which is not the case, as we describe above—it would be manifestly arbitrary and capricious for the Agency to determine that CAA standards that have not been updated for many years, or even decades, presumptively discharge EPA’s present-day responsibility to assess the risks these chemicals pose under TSCA.

ii. The factual record establishes that there is exposure to 1,4-dioxane through ambient air.

First, the problem formulation itself establishes that exposures through air persist for 1,4-dioxane despite any regulation under the CAA, and it is arbitrary and capricious for EPA to ignore those exposures. For EPA to treat these exposure levels as “zero” when they are known not to be does not comport with the best available science.

EPA states that a total of 62,596 lbs of the chemical were released to the air in 2015 according to the EPA Toxics Release Inventory (TRI). Problem Formulation for 1,4-Dioxane at p. 26. Both indoor and outdoor monitoring detected 1,4-dioxane. *Id.* at 28. “Of a total of 1397 collected samples, there were 948 non-detects (68%) and 449 detections (32%), which ranged from 0.005 to 0.96 ppb.” *Id.*

Second, EPA should not limit its analysis of air emissions to TRI data. EPA should also consider the data available from the National Emissions Inventory (NEI), which tend to reveal significantly greater levels of air emissions of, and thus air pathway exposures to, 1,4-dioxane. EPA cannot reasonably ignore this available information about air emissions and resulting exposures to 1,4-dioxane. As revealed in the below chart, despite the Clean Air Act protections, there are significant annual emissions and thus exposures through the air pathway for these chemicals.

	TRI 2016			NEI 2014
Chemical	Fugitive Air Emissions (lbs)	Point Source Air Emissions (lbs)	TOTAL	(lbs)
1,4-Dioxane	10,522	45,210	55,732	134,484

EPA should analyze these exposures and the risks they present to both human health and the environment, including terrestrial species.

Moreover, EPA should be collecting and analyzing information about exposure levels through the ambient air pathway, particularly near sites where people may experience greater exposure due to their proximity to conditions of use or contamination sites. By excluding pathways such as the ambient air pathway, EPA will seriously underestimate the levels of exposure. In particular, EPA cannot adequately assess the risks faced by subpopulations consisting of people experiencing greater exposure due to their proximity to conditions of use without assessing pathways such as the ambient air pathway. EPA should use its information authorities to obtain additional information about exposure levels experienced by the subpopulations living near conditions of use.

iii. EPA's analysis of air emissions fails to provide the analyses or information necessary to assess the risk presented by 1,4-dioxane air emissions.

In seeking to justify its exclusion of exposures from air emission pathways, in the problem formulation EPA states:

1,4-Dioxane is a HAP. EPA has issued a number of technology-based standards for source categories that emit 1,4-dioxane to ambient air and, *as appropriate*, has reviewed, *or is in the process of reviewing remaining risks*. Because stationary source releases of 1,4-dioxane to ambient air are adequately assessed and any risks effectively managed when under the jurisdiction of the CAA, EPA does not plan to evaluate emission pathways to ambient air from commercial and industrial stationary sources or associated inhalation exposure of the general population or terrestrial species in this TSCA evaluation.

Problem Formulation for 1,4-dioxane at p. 43 (emphases added). In the Appendix to the problem formulation, EPA merely provides a list of technology-based standards for certain source categories. EPA provides no analysis whatsoever as to: the extent to which the standards cover the full range of stationary sources of this chemical; the extent and magnitude of releases of the chemical allowed under each of the standards; the duration, intensity, frequency, and number of exposures resulting from those allowable emissions (as required under TSCA section 6(b)(4)(F)(iv)); or any other factors that would be necessary to analyze and determine the extent and nature of potential risk allowed under the standards. EPA has not acknowledged, let alone analyzed, the overall risks to the general population or to vulnerable subpopulations due to the combination of exposures arising from the various sources for which standards exist, not to mention additional emission sources not subject to any standard. EPA has made no attempt to reconcile any such risk with that allowed under TSCA.

In the absence of such analyses, there is no basis whatsoever for EPA to assert that air releases of this chemical have been adequately assessed or that any risks have been effectively managed under TSCA's standards.

EPA offers only a vague claim that EPA "as appropriate, has reviewed, or is in the process of reviewing remaining risks." No specifics as to the status of or timeline for such reviews have been provided, and no indication is made as to when and on what basis such reviews are deemed "appropriate." Nor have the results of any such reviews that have been completed been provided, let alone analyzed in the context of TSCA's requirements.

Given evidence of real-world exposure through the air pathway, EPA must evaluate those exposures in its risk evaluation. In particular, EPA needs to consider whether these exposures combine with other sources of exposure in a manner that leads to an unreasonable risk, including to certain subpopulations. EPA cannot rationally exclude these exposures from its analysis.

G. Real-world exposures still occur through disposal pathways, and EPA cannot ignore those real-world exposures when assessing the risk presented by 1,4-dioxane.

In the problem formulation for 1,4-dioxane, EPA contends that due to regulation of disposal under the Resource Conservation and Recovery Act (RCRA), the Clean Air Act (CAA), the Safe Drinking Water Act (SDWA), and various state programs, EPA can ignore all exposures from all disposal-related pathways and associated activities (e.g., collection, processing, storage and transport). Problem Formulation for 1,4-Dioxane at pp. 44-45.

This approach is unreasonable for the reasons given above. EPA has not made the necessary showing that these regulations eliminate any unreasonable risk and EPA has not assessed all relevant aspects of the risk. Indeed, EPA has not even established or shown that these disposal regulations meet EPA's illegal standard that these regulations "adequately assess and effectively manage exposures." For example, EPA has not shown or established that disposal in a RCRA Subtitle C hazardous waste landfill or a RCRA Subtitle D non-hazardous waste landfill would actually reduce unreasonable risk to a sufficient extent. EPA's approach is also arbitrary and capricious for a variety of reasons.

While EPA invokes the standards for RCRA Subtitle C landfills as providing sufficient protection, not all disposal occurs in such landfills. Even for those chemicals regulated under RCRA, EPA acknowledges that disposal also occurs in Subtitle D municipal solid waste (MSW) landfills and industrial-non-hazardous and construction/demolition waste landfills (which are primarily regulated under state regulatory programs). These disposal approaches do not need to meet the requirements of Subtitle C landfills, thus EPA's invocation of the Subtitle C standards does not justify ignoring exposures from these disposals. While the purpose of RCRA subtitle C is at least to "protect human health and the environment," *see, e.g.*, 42 U.S.C. §§ 6922(a),

6924(a), subtitle D is intended “to assist in developing and encouraging methods for the disposal of solid waste which are environmentally sound and which maximize the utilization of valuable resources including energy and materials *** and to encourage resource conservation.” 42 U.S.C. § 6941. Therefore, EPA’s exclusions based on the regulations under subtitle D potentially raise even greater, unaddressed, public health concerns than EPA’s exclusions under subtitle C.

In addition, states impose varying requirements on such landfills under their delegated RCRA Subtitle D authorities. For example, EPA indicates that some state programs may not include requirements for liners to limit release of landfill leachate.

EPA itself has acknowledged that enforcement and regulation under RCRA is inconsistent, so EPA cannot simply assume that RCRA implementation provides a basis for ignoring exposures under TSCA. As the Office of Inspector General explained the challenges of the RCRA system:

The Hazardous and Solid Waste Amendments of 1984 (HSWA) amended RCRA and added provisions including land disposal restrictions, RCRA corrective action for solid waste management units and regulation of small-quantity generators. When the EPA creates new hazardous waste rules, it does so under the authority of either or both of these laws. Rules promulgated under HSWA authority are immediately effective in all states and are administered by the EPA until states become authorized for those rules. In contrast, *rules promulgated under RCRA authority (non-HSWA rules) cannot be enforced by the EPA in states with an authorized base program and do not go into effect until these states become authorized for the rules.*¹⁷⁵

According to the OIG, the fact that a number of rules are not yet adopted by the states and cannot be enforced by EPA “creates a regulatory gap and risk to human health and the environment, and an inconsistent regulatory landscape across the states.”¹⁷⁶ OIG’s report states that “there are almost 1,300 instances of required rules for which various state hazardous waste programs have not been authorized. Of the rules for which states have not received authorization, there are about 500 each of HSWA and non-HSWA rules, and about 300 rules that have components of both.”¹⁷⁷

¹⁷⁵ U.S. EPA, Office of Inspector General, *Incomplete Oversight of State Hazardous Waste Rule Authorization Creates Regulatory Gaps and Human Health and Environmental Risks* at 2 (Jul. 2018), https://www.epa.gov/sites/production/files/2018-07/documents/epa_oig_20180731-18-p-0227.pdf (emphasis added).

¹⁷⁶ *Id.* at 11.

¹⁷⁷ *Id.* at 12; see also AUTHORIZATION STATUS BY RULE, https://www.epa.gov/sites/production/files/2018-06/documents/authorization_status_by_rule.pdf

When states do not keep their hazardous waste programs up to date, it means citizens in different states are unevenly protected from hazardous waste-related risks. This is critical because “60,000 RCRA facilities exist in the United States, generating and managing 30 to 40 million tons of hazardous waste annually. Eighty percent of all U.S. citizens live within a 3-mile radius of a RCRA-regulated hazardous waste generator or treatment storage and disposal facility, and 50 percent of citizens live within a 1-mile radius.”¹⁷⁸ Therefore, EPA cannot rely on any assumption of consistent implementation and enforcement of RCRA to ensure that all exposures have been adequately managed.

Indeed, many of the problem formulations themselves establish that exposures from disposal persist for these chemicals despite RCRA regulations, and it is arbitrary and capricious for EPA to ignore those exposures. For EPA to treat these exposure levels as “zero” when they are known to exist does not comport with the best available science.

To be sure, EPA often appears to have less monitoring information that speaks to whether a particular exposure arises from disposal or some other source, and EPA also appears to have less monitoring information about 1,4-dioxane’s presence in soil, sediment, and leachate, than it does for its presence in water or air. As EDF has previously explained, EPA must consider “reasonably available” information, and thus EPA must both consider the information it already possesses and use its authorities under TSCA §§ 4 and 8 to obtain additional information. EDF incorporates and reiterates those points here as well.¹⁷⁹ EPA should use those authorities to obtain additional information about the exposures arising from disposal of 1,4-dioxane.

EPA cannot assume that exposure from disposal is zero just because it could be regulated under other authorities. For example, the problem formulation contains information suggesting that exposures may arise from disposal. In particular, EPA acknowledged that “1,4-Dioxane has also been detected in landfill leachate.” Problem Formulation for 1,4-Dioxane at p. 28.

H. EPA must analyze all the environmental risks presented by 1,4-dioxane through ambient water.

EPA recognizes that it must evaluate the risks to aquatic species arising from exposures through water for 1,4-dioxane (pp. 46, 131). But EPA did not analyze the risks to terrestrial species from

(last visited Aug. 10, 2018) (documenting for each state whether they have adopted the RCRA regulations).

¹⁷⁸ U.S. EPA, Office of Inspector General, *EPA Has Not Met Statutory Requirements for Hazardous Waste Treatment, Storage and Disposal Facility Inspections, but Inspection Rates Are High* at 1 (March 2016), <https://www.epa.gov/sites/production/files/2016-03/documents/20160311-16-p-0104.pdf>.

¹⁷⁹ EDF Comments on Ten Scopes under the Toxic Substances Control Act pp. 11-15, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0743-0069>.

exposure through ambient water, despite the fact that terrestrial species also can experience exposures through surface water. *But see* U.S. EPA, Problem Formulation for Cyclic Aliphatic Bromides Cluster (HBCD) (May 2018), p. 50 <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0735-0071> (“Aquatic and terrestrial ecological receptors may also be directly exposed due to proximity to surface water and sediment.”). EPA should have considered the risks presented to terrestrial ecological receptors as well as aquatic species. EPA provides no convincing explanation for excluding exposures to terrestrial organisms for 1,4-dioxane.

I. EPA cannot rely on its actions under other authorities when there are numerous problems with compliance, implementation, and enforcement under those authorities.

EPA cannot ignore exposure through these pathways for the reasons given above, but in addition, it is arbitrary and capricious for EPA to assume zero exposure through other pathways based on EPA-administered statutes when EPA has documented extensive problems with compliance, implementation, and enforcement of these statutes.

- i. EPA’s own analyses establish that State enforcement of these environmental statutes is inconsistent and often deficient.*

There are multiple EPA reports documenting enforcement problems with EPA’s environmental statutes.¹⁸⁰ Specifically, these reports have noted that “data quality, identification of violations, issuing enforcement penalties and other enforcement actions in a timely and appropriate manner, and general oversight issues” are all key issues impacting the enforcement of these statutes.¹⁸¹

Generally, EPA’s regional offices provide oversight to ensure that the state enforcement programs are following EPA’s guidance, policies, and regulations.¹⁸² Despite EPA oversight, which is a separate concern, state enforcement of these statutes has been found deficient in a number of cases. For instance:

¹⁸⁰ U.S. EPA, Office of Inspector General, *EPA Must Improve Oversight of State Enforcement* at App. B, p. 32-34 (Dec. 2011), <https://www.epa.gov/sites/production/files/2015-10/documents/20111209-12-p-0113.pdf> (identifying a long list of GAO and OIG reports documenting deficiencies in enforcement of environmental statutes).

¹⁸¹ *Id.* at 32.

¹⁸² U.S. Government Accountability Office, *EPA-State Enforcement Partnership Has Improved, but EPA’s Oversight Needs Further Enhancement* at 1 (Jul. 2007), <https://www.gao.gov/products/GAO-07-883>.

- According to a 2011 OIG report, **North Dakota** appears “philosophically opposed to taking enforcement action.”¹⁸³ For instance, during the entire period of the report (FYs 2003-2009), the state assessed no penalties against known CWA violators.¹⁸⁴
- In **Louisiana** multiple petitions have been filed by citizens to remove the state’s delegated authorities under the CWA, CAA, and RCRA.¹⁸⁵ The poor performance under these statutes was attributed to “a lack of resources, natural disasters, and a culture in which the state agency is *expected to protect industry*.”¹⁸⁶
- The **U.S Virgin Islands** “has not met program requirements for numerous activities related to implementing the Clean Air Act, Clean Water Act, Safe Drinking Water Act, and Underground Storage Tank/Leaking Underground Storage Tank programs. These activities included monitoring environmental conditions, conducting compliance inspections and enforcing program requirements.”¹⁸⁷

Notably, even where enforcement of these statutes has been consistently deficient, EPA has generally not de-authorized states. According to the 2011 OIG report, “the threat of EPA revoking a state’s authorization [is] moot because there is a general understanding that no EPA region has the resources to operate a state program. This reality undercuts EPA’s strongest tool for ensuring that authorized states adequately enforce environmental laws: de-authorization.”¹⁸⁸ Although EPA has taken steps in a number of cases to improve state programs, ultimately implementation and enforcement of these statutes remains deficient in a number of states, resulting in continued excessive exposure to chemicals through air, water, and land. These exposures must be assessed under TSCA.

Below are a few more specific examples, among many, of deficiencies under each of the statutes.

¹⁸³ U.S. EPA, Office of Inspector General, *EPA Must Improve Oversight of State Enforcement* at 17 (Dec. 2011), <https://www.epa.gov/sites/production/files/2015-10/documents/20111209-12-p-0113.pdf>.

¹⁸⁴ *Id.* at 15.

¹⁸⁵ *Id.* at 16.

¹⁸⁶ *Id.* (emphasis added).

¹⁸⁷ U.S. EPA, Office of Inspector General, *Conditions in the U.S. Virgin Islands Warrant EPA Withdrawing Approval and Taking Over Management of Some Environmental Programs and Improving Oversight of Others* (April 2015), <https://www.epa.gov/sites/production/files/2015-09/documents/20150417-15-p-0137.pdf>; U.S. EPA Region 2, *National Strategy Oversight Plan* at 3 (Mar. 2016), <https://www.documentcloud.org/documents/2992740-Region-2-State-Oversight-Plan-March-2016-v2.html>.

¹⁸⁸ U.S. EPA, Office of Inspector General, *EPA Must Improve Oversight of State Enforcement* at 17 (Dec. 2011), <https://www.epa.gov/sites/production/files/2015-10/documents/20111209-12-p-0113.pdf>.

Safe Drinking Water Act: As explained above, EPA has excluded exposures to drinking water for 1,4-dioxane based on the assumed effectiveness of state implementation and enforcement of the SDWA. A 2011 GAO report states that EPA often receives unreliable data from the states.¹⁸⁹ EPA relies on state data to determine whether there is compliance with the SDWA. Without reliable data EPA has no way to verify that the requirements of the SDWA are being met by the states.

Here is one example of deficient state enforcement of the SDWA:

- **Pennsylvania:** EPA sent a letter in December 2016 to the Pennsylvania Department of Environmental Protection, stating that the department lacks the necessary staff to enforce safe drinking water standards and that the lack of staff has caused the number of unaddressed Safe Drinking Water Act violations to nearly double in the past five years, from 4,298 to 7,922.¹⁹⁰

Clean Water Act: EPA has also excluded exposures to ambient water for 1,4-dioxane based on the assumed “effectiveness” of the CWA’s National Pollution Discharge Elimination System (NPDES) program and the water quality criteria process.

But over half of assessed U.S. river and stream miles violate state water quality standards.¹⁹¹ EPA’s own analysis, provided below, indicates that waters remained impaired throughout the United States, despite the CWA standards.

¹⁸⁹ U.S. Government Accountability Office, *Unreliable State Data Limit EPA’s Ability to Target Enforcement Priorities and Communicate Water Systems’ Performance* (June 2011), <https://www.gao.gov/products/GAO-11-381>.

¹⁹⁰ Letter from Jon M. Capacasa, Director, EPA Region III Water Protection Division, to Lisa D. Daniels Director, Pa. Dep’t of Env’tl. Prot. Bureau of Safe Drinking Water (Dec. 30, 2016), <https://drive.google.com/file/d/0B4Y3VQLxjKxObjZ0ZXISVDZvRWc/view>.

¹⁹¹ NATIONAL SUMMARY OF STATE INFORMATION, https://ofmpub.epa.gov/waters10/attains_nation_cy.control (last visited Jul. 31, 2018).

Assessed Water of the United States¹⁹²

	Size of Water							
	Rivers and Streams (Miles)	Lakes, Reservoirs, and Ponds (Acres)	Bays and Estuaries (Square Miles)	Coastal Shoreline (Miles)	Ocean and Near Coastal (Square Miles)	Wetlands (Acres)	Great Lakes Shoreline (Miles)	Great Lakes Open Water (Square Miles)
Good Waters	516,800	5,392,817	11,516	1,285	617	569,328	106	1
Threatened Waters	4,495	30,309						
Impaired Waters	586,910	13,158,111	44,619	3,330	6,218	665,979	4,354	39,230
Total Assessed Waters	1,108,205	18,581,237	56,135	4,615	6,836	1,235,307	4,460	39,231
Total Waters	3,533,205	41,666,049	87,791	58,618	54,120	107,700,000	5,202	196,343
Percent of Waters Assessed	31.4	44.6	63.9	7.9	12.6	1.1	85.7	20.0

EPA also publishes the Annual Noncompliance Report, which summarizes enforcement data for facilities with individual NPDES permits but that are not major dischargers.¹⁹³ According to the 2015 report, the percentage of facilities with formal enforcement actions compared to facilities with violations was merely 8.9% in 2015.¹⁹⁴ Below are a few examples of enforcement deficiencies:

- **Tennessee:** The Tennessee Department of Environment and Conservation neglected to timely penalize permit holders despite months of noncompliance, failed to assess appropriate fines, and did not report significant discharge violations from major facilities.¹⁹⁵
- **Alaska:** EPA regional directors told OIG that “when the region authorized the state to run the program, both the region and OECA officials were aware that the state lacked the capacity to be successful.”¹⁹⁶ EPA’s State Review Framework for Alaska revealed that, among other serious concerns, the state does not consistently take timely or appropriate enforcement actions, inspect permitted facilities anywhere close to state goals.¹⁹⁷

¹⁹² *Id.*

¹⁹³ U.S. EPA, Office of Enforcement and Compliance Assurance, *Annual Noncompliance Report (ANCR) Calendar Year 2015* (Aug. 2016), https://echo.epa.gov/system/files/2015_ANCR.pdf.

¹⁹⁴ *Id.* at 7.

¹⁹⁵ U.S. EPA Region 4, *State Review Framework Tennessee* at 28-35 (Sept. 2016), <http://www.documentcloud.org/documents/3173730-TN-Final-SRF-Report-9-29-16.html>.

¹⁹⁶ U.S. EPA, Office of Inspector, *EPA Must Improve Oversight of State Enforcement* at 16 (Dec. 2011), <https://www.epa.gov/office-inspector-general/report-epa-must-improve-oversight-state-enforcement>.

¹⁹⁷ U.S. EPA Region 10, *State Review Framework Alaska* at exec. summary (Dec. 2014), <https://www.epa.gov/sites/production/files/2015-01/documents/srf-rd3-rev-ak.pdf>.

- **Louisiana:** Louisiana reviewed the compliance status for less than 50% of individually-permitted non-major NPDES permittees from 2010-2015.¹⁹⁸

Clean Air Act: State performance also varies widely under the CAA. In 2011, the Office of the Inspector General examined the percentage of facilities inspected, the percentage of significant noncompliance or high priority violations identified per inspection, and the percentage of final actions with penalties for fiscal years 2003-2009 and found that performance varied significantly across the country, in this case “by almost 50 percentage points.”¹⁹⁹ Below are a few specific examples of insufficient state enforcement of the CAA:

- **Florida:** The Florida Department of Environmental Protection opened only 18 air enforcement cases in 2015, compared to a previous annual average of 93.²⁰⁰ Additionally, from 2013 to 2015 the state only filed one asbestos case, compared to a past annual average of 13.²⁰¹
- **North Carolina:** “CAA metric for assessed penalties dropped by 93% statewide from about \$235,000 in FY II to just under \$17,000 in FY 14. During the same period the number of facilities with informal and formal enforcement actions also dropped dramatically (52% and 79%, respectively).”²⁰²
- **Ohio:** The Region found that a number of High Priority Violations (HPV) are being resolved by the state through a permit modification/revision. EPA believes that HPV cases should be resolved through a formal enforcement action per the HPV policy, and the state disagrees.²⁰³
- **Texas:** “A review of five years of state records by the Environmental Integrity Project and Environment Texas shows that the state imposed penalties on *less than 3 percent* of the illegal pollution releases (588 out of 24,839) *reported by companies* during maintenance or malfunctions from 2011 through 2016, even though the incidents released

¹⁹⁸ U.S. EPA, Office of Enforcement and Compliance Assurance, *Annual Noncompliance Report (ANCR) Calendar Year 2015* at 8 (Aug. 2016), https://echo.epa.gov/system/files/2015_ANCR.pdf.

¹⁹⁹ U.S. EPA, Office of Inspector, *EPA Must Improve Oversight of State Enforcement* at 10 (Dec. 2011), <https://www.epa.gov/office-inspector-general/report-epa-must-improve-oversight-state-enforcement>.

²⁰⁰ Public Employees for Environmental Responsibility, *Report on Enforcement Efforts by the Florida Department of Environmental Protection* at 23 (Aug. 2016), https://www.peer.org/assets/docs/fl/8_18_16_DEP_Report_on_2015_Enforcement.pdf.

²⁰¹ *Id.*

²⁰² Letter from J. Scott Gordon, Director, EPA Region IV Office of Enforcement Coordination, to Donald R. van der Vaart, Secretary, N.C. Dep’t of Env’tl. Quality (May 9, 2016), <https://assets.documentcloud.org/documents/3114598/EPA-Region-4-Letter-to-NCDEQ.pdf>.

²⁰³ U.S. EPA Region 5, *State Review Framework Ohio* at 3, 38-39 (Aug. 2013), <https://www.epa.gov/sites/production/files/2014-05/documents/srf-rd2-rev-oh.pdf>.

more than 500 million pounds of air pollution.”²⁰⁴ Thus, 500 million pounds of illegal emissions were reported in Texas for 2011 through 2016: it would be irrational to assume that these emissions did not occur. Moreover, the state of Texas did not impose penalties for 97% of these illegal pollution releases reported by companies. Of course, not all violations are promptly or accurately reported by companies, so this number may actually overestimate the level of compliance and enforcement. With such lax enforcement, compliance levels are going to be low.

Resource Conservation and Recovery Act: As with the other statutes upon which EPA relies to avoid analyzing exposure pathways, there are serious state enforcement problems with RCRA. For example, Mississippi has not accurately identified and documented RCRA violations.²⁰⁵ Additionally, despite EPA guidance that states civil penalties should recoup at least the economic benefit the violator gained through noncompliance, the state does not routinely document or consider the economic benefit.²⁰⁶

ii. *Reduced EPA enforcement provides even less assurance that exposures through the excluded pathways are being effectively managed.*

Under the current Administration, enforcement of these environmental statutes has been significantly curbed. For instance, management at EPA has directed EPA investigators to seek authorization before asking companies to conduct testing or sampling under the CAA, RCRA, or the CWA.²⁰⁷ The memo also states that investigators need authorization if they do not have information specific to a company that it may have violated the law, or if state authorities objected to the tests.²⁰⁸

Additionally, in its proposed 2020 budget, the current Administration sought a 31 percent reduction in funding for EPA.²⁰⁹ This reduction would affect EPA’s enforcement budget and the resources available to ensure enforcement of the above the statutes. EPA cannot rely on its

²⁰⁴ Environmental Integrity Project, *Breakdowns in Enforcement Texas Rarely Penalizes Industry for Illegal air Pollution Released during Malfunction and Maintenance* at 1 (Jul. 2017), <https://www.environmentalintegrity.org/wp-content/uploads/2017/02/Breakdowns-in-Enforcement-Report.pdf> (emphases added).

²⁰⁵ U.S. EPA Region 4, *State Review of Framework Mississippi* at Executive Summary (Mar. 3, 2016), <https://www.epa.gov/sites/production/files/2016-03/documents/srf-rd3-rev-ms.pdf>.

²⁰⁶ *Id.* at 24.

²⁰⁷ Memorandum from Susan Shinkman, Director, EPA Office of Civil Enforcement, to Regional Counsel, Regional Enforcement Directors and Coordinators, and OCE Division Directors (May 31, 2017), <https://www.documentcloud.org/documents/4324892-EPA-Clean-Air-Act-and-Its-Power-to-Request.html#document/p60/a392202>.

²⁰⁸ *Id.*

²⁰⁹ Office of Mgmt. & Budget, *A Budget for a Better America* at 93, <https://www.whitehouse.gov/wp-content/uploads/2019/03/budget-fy2020.pdf>.

actions under other authorities when EPA has itself taken steps to ensure that those authorities are not adequately addressing the risks presented.

* * * * *

In sum, EPA must analyze all exposures to 1,4-dioxane. EPA cannot legally ignore exposures that occur under other EPA-administered statutes, and treating exposures that are known to occur in the world as nonexistent is arbitrary and capricious. EPA must assess these exposures based on their real-world existence and consider how they may combine with other sources of exposure to accurately estimate the risks presented by 1,4-dioxane. Where EPA has inadequate information, EPA should use its information authorities to obtain more information about these exposures.

6. EPA needs to analyze potential exposures from distribution, as well as from known and reasonably foreseeable accidental exposures.

EPA's analysis of distribution was inadequate in the draft risk evaluation and problem formulation. In the draft risk evaluation, EPA stated that: "No further evaluation of distribution of 1,4-dioxane was included in this risk evaluation because chemicals are packaged in closed-system containers during distribution in commerce and no exposures are expected." (p. 28). In the problem formulation, EPA stated that: "During distribution, 1,4-dioxane is contained in closed systems (e.g. drums, pails, bottles) so releases and exposures are not expected." Problem Formulation for 1,4-Dioxane at p. 37. This blanket assertion is made with absolutely no supporting analysis or data, either documenting the extent to which the identified "closed systems" are actually used, or the extent to which they are in fact "closed" and lead to no releases or exposure whatsoever, as EPA asserts. Even on their face, the examples raise many questions. For example: Are drums or bottles never open? How is a pail a "closed system"?

Neither the problem formulation nor the draft risk evaluation provide evidence or support for EPA's assertion that 1,4-dioxane is always distributed in closed systems, and indeed, a "pail" is hardly a closed system. EPA has provided no evidence that exposures and releases during distribution will be nonexistent.

The draft risk evaluation and problem formulation also give no attention to potential releases and exposures resulting from accidental releases. EDF does not suggest that EPA needs to consider every possible scenario, but the risk of accidental releases and exposures is very real and certainly "reasonably foreseen" in many respects, and EPA has authority to mandate steps to reduce those risks. For example, as and after Hurricane Harvey passed through Houston, over 40

sites released toxic chemicals into the environment.²¹⁰ Given the known accidental releases, the huge number of petrochemical plants and refineries in the Houston area, and the likelihood that flooding there may become more common in light of climate change, such events are clearly reasonably foreseen and hence EPA needs to give more consideration to the potential for accidental releases.

7. EPA must consider “reasonably available” information, and thus EPA must use its authorities under TSCA §§ 4 and 8 to obtain additional information.

TSCA orders EPA to consider “available” and “reasonably available” information in crafting a risk evaluation, 15 U.S.C. §§ 2605(b)(4)(F)(i), 2625(k), and under the new risk evaluation rule, EPA defined “[r]easonably available information” to mean “information that EPA possesses or can reasonably generate, obtain, and synthesize for use in risk evaluations, considering the deadlines specified in TSCA section 6(b)(4)(G) for completing such evaluation.” 40 C.F.R. § 702.33, promulgated at 82 Fed. Reg. 33,748 (July 20, 2017). Thus, under its own rule, EPA has to consider information that it “can reasonably generate, obtain, and synthesize.”

In our prior comments on the scope document and problem formulation, EDF expanded on EPA’s duties to use its authorities under TSCA §§ 4 and 8 to obtain additional information about 1,4-dioxane, and EDF incorporates those arguments here.²¹¹ In response to EDF’s comment on the scope, EPA acknowledged its duty to consider “reasonably available information” and EPA described its efforts to gather information up to that point.²¹² While EPA detailed its “data gathering activities,” EPA has not established that these activities resulted in EPA obtaining all the reasonably available information that EPA could “generate, obtain, and synthesize” if EPA also used its authorities under TSCA §§ 4 and 8 to obtain additional information. Thus, EPA has not established that it has or will obtain all reasonably available information.

In particular, EDF’s prior comments established that relying solely on voluntary requests for information, may result in limited, biased, inaccurate, or incomplete information on the

²¹⁰ See, e.g., *More Than 40 Sites Released Hazardous Pollutants Because of Hurricane Harvey*, N.Y. TIMES (Sept. 8, 2017), https://www.nytimes.com/interactive/2017/09/08/us/houston-hurricane-harvey-hazardous-chemicals.html?_r=0.

²¹¹ EDF Comments on Ten Scopes under the Toxic Substances Control Act at pp. 11-16, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0743-0069>; EDF Comments on Problem Formulations for Risk Evaluations To Be Conducted Under Toxic Substances Control Act at pp.57-62, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0723-0083>.

²¹² EPA’s Responses to Public Comments Received on the Scope Documents for the First Ten Chemicals for Risk Evaluation under TSCA at pp.10-14, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0725-0051>.

chemicals. EDF incorporates those arguments here.²¹³ EPA's response to this comment was that "EPA has not indicated it would rely solely on voluntary requests for information."²¹⁴ Thus, EPA appears to recognize that voluntary requests standing alone are insufficient. Despite that acknowledgement, EPA still has not relied on its available authorities to obtain additional information. EDF again urges EPA to do so.

EPA's primary response to EDF's request that EPA consider all reasonably available information appears to be that the information EPA currently has is "adequate."²¹⁵ But, as a general matter, EPA has to consider all reasonably available information; TSCA does not authorize EPA to stop its analysis on the basis that EPA believes its current information is adequate. And as explained more below, it is clear that the information is not yet adequate to meet EPA's obligations under TSCA.

A. Relying on voluntary requests for information will result in limited, biased, inaccurate, or incomplete information on the chemicals.

In the 1,4-dioxane problem formulation, EPA stated that "EPA encourages submission of additional existing data, such as full study reports or workplace monitoring from industry sources, that may be relevant for refining conditions of use, exposures, hazards and potentially exposed or susceptible subpopulations during the risk evaluation. EPA will continue to consider new information submitted by the public." Problem Formulation for 1,4-Dioxane at p. 47. With this language EPA seemed to acknowledge the serious data gaps it faced; yet despite clear authority to require workplace monitoring by industry and to obtain full study reports using its existing authorities, EPA resorted merely to encouraging their submission.

As detailed in our comments in Part I sec. 4, many key data gaps remain regarding 1,4-dioxane in the draft risk evaluation. EPA has insufficient information about: environmental monitoring data, environmental fate data, ecotoxicity data, concentrations in products/uses, inhalation exposure data for workers, dermal exposure data, dermal toxicity data, and reproductive/developmental/neurodevelopmental data. Thus, EPA's reliance on voluntary submissions has failed to produce the information necessary for a robust and accurate risk evaluation.

²¹³ EDF Comments on Ten Scopes under the Toxic Substances Control Act at pp. 16-20, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0743-0069>; EDF Comments on Problem Formulations for Risk Evaluations To Be Conducted Under Toxic Substances Control Act at pp. 58-61, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0723-0083>.

²¹⁴ EPA's Responses to Public Comments Received on the Scope Documents for the First Ten Chemicals for Risk Evaluation under TSCA at p.13, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0725-0051>.

²¹⁵ *See id.* at pp. 13, 10-14.

Rather than relying solely on voluntary submissions—an approach that has proven insufficient in the past—EPA should use its information authorities to obtain necessary information on conditions of use, exposures, hazards, and potentially exposed or susceptible subpopulations.

There are several obvious problems and limitations with this voluntary approach which EPA has still not addressed.

First, a voluntary call is much less likely to produce all of the necessary information than rules mandating that affected parties provide the requested information. If manufacturers and processors are legally required to provide the information, that legal obligation provides a strong incentive for them to develop or obtain and submit all relevant information. Absent that incentive, some companies may choose to focus time and attention on other matters.

Second, EPA has provided no empirical evidence establishing that this voluntary approach will result in EPA obtaining all “reasonably available” information. Unless EPA has some empirical basis for stating that the voluntary approach will allow EPA to obtain all reasonably available information that it can obtain under its legal authorities, EPA must rely on its existing authorities to obtain a complete set of information.

Third, manufacturers and processors of these chemicals have a vested interest in EPA finding that the chemicals do not present an unreasonable risk. A no-unreasonable-risk finding reduces the likelihood of government regulation, including potential restrictions on risky chemicals, and it may reduce any stigma they may otherwise face in the marketplace. The financial costs of regulation may ultimately be very high for some specific firms and individuals, and even if not, many firms and individuals may believe that the costs of regulation will be high. These companies have a “financial interest” in the outcome of these proceedings, and they are not impartial. *See, e.g.*, 28 U.S.C. § 455(b)(4) (requiring Judges to disqualify themselves in proceedings where they have a financial interest). Because of this reality and appearance of partiality, relying solely on voluntary measures decreases the credibility of this risk evaluation.

Relying solely on voluntary presentation of information raises the concern that the companies or trade associations may present an incomplete or skewed picture. Companies and trade associations may choose to “cherry pick” information and provide only the information that paints 1,4-dioxane in a favorable light. They may provide only summaries of information that reflect conscious and subconscious judgment calls that result in unduly favorable conclusions; and without access to the full information neither EPA nor the public can independently assess such conclusions. They may choose not to review records robustly when the review may disclose unfavorable information. They may seek to put their best foot forward and describe the

ideal scenario of use and safety measures. Or, if they have unfavorable information, they may choose not to provide any information at all and simply not participate in these proceedings.

EPA cannot simply assume that members of the regulated community will voluntarily disclose unfavorable or complete information about their practices and products. *See* The Federalist No. 51 (James Madison) (“If men were angels, no government would be necessary. *** [E]xperience has taught mankind the necessity of auxiliary precautions.”); *Williams v. Pennsylvania*, 136 S. Ct. 1899, 1905-06 (2016) (“Bias is easy to attribute to others and difficult to discern in oneself. *** This objective risk of bias is reflected in the due process maxim that ‘no man can be a judge in his own case and no man is permitted to try cases where he has an interest in the outcome.’”). Here, manufacturers and processors obviously have an interest in the outcome, and EPA must craft its procedures and approaches with that reality in mind. Requiring the submission of information is the safest approach to ensuring that these parties provide all relevant information, and that is in turn crucial to establishing and demonstrating the credibility of this process.

The problem with EPA’s voluntary approach is readily apparent from the draft risk evaluation. EPA did not receive data on many occupational exposures, requiring EPA to rely on models instead of actual data. Moreover, even when EPA received data, it was generally limited and unrepresentative. For example, EPA relied heavily upon data received from BASF for a single manufacturing site in Zachary, Louisiana. EPA treats this information as representative of manufacturing in general, but as the only data submitted for this condition of use, one could rationally assume that it may reflect the most favorable, lowest exposure scenario. Rather than treat it as representative, EPA should have assessed this data as representing the “best case” scenario for manufacturing.

If EPA acts under TSCA §§ 8(a), (c), and (d), the regulations impose some requirements that will help ensure the accuracy and completeness of the information. First, EPA can require that certain information and underlying information be provided in full, which ensures completeness. In addition, a § 8(d) rule requires that people engage in an adequate search of records. 40 C.F.R. § 716.25. Second, submitters must file certification statements by authorized officials that certify that the submitted information has been submitted in compliance with the requirements of this process. *See, e.g.*, 40 C.F.R. § 711.15(b)(1). Third, submitters often must retain records of required submissions for a period of five years, and the retention of records can help encourage accurate reporting since those records would be available should a submission later be investigated. *See, e.g.*, 40 C.F.R. § 711.25. None of these features apply to the voluntary requests for information EPA has indicated it is relying on.

B. EPA cannot rationally rely on unvetted industry submissions, and to the extent EPA relies on voluntary submissions from industry, EPA must take numerous additional steps to increase their reliability and transparency.

In the draft risk evaluation, EPA uncritically relies on industry submissions, and this reliance does not constitute the best available science. For example, EPA relies on monitoring data submitted by BASF (p. 54), but EPA itself acknowledges that the data lacked crucial information necessary to understand the data (p. 54), much less assess its reliability. From these records, it is not possible for the public to even begin to assess the accuracy of EPA's conclusions based on these data.

To the extent it relies on voluntary submissions from industry, EPA needs to take additional steps to better ensure that the voluntary information it receives is accurate and complete. EPA would need to develop a far more rigorous and structured process than it currently has. For example, EPA's submission process does not appear to require anyone to certify that the information in their submissions is accurate or complete to the best of their knowledge. EPA should consider approaches for vetting statements and assertions, particularly when made by entities with a financial interest in the outcome of these risk evaluations.

C. The draft risk evaluation and these comments identify numerous information gaps that EPA needs to fill using its information authorities.

Throughout these comments, EDF points to information gaps that EPA should fill with its information authorities. The draft risk evaluation also identifies various data gaps; for example, EPA states that “[t]here are data gaps for 1,4-dioxane inhalation and dermal exposure in humans and 1,4-dioxane dermal exposure in animals leading to carcinogenic effects” (p. 107). Another acknowledged data gap appears in EPA's discussion of potentially exposed or susceptible subpopulations, where EPA acknowledges that “Information on induction of liver enzymes, genetic polymorphisms and gender differences was inadequate to quantitatively assess toxicokinetic or toxicodynamic differences in 1,4-dioxane hazard between animals and humans and the potential variability in human susceptibility.” (p. 151). EPA should use its information authorities to develop information to allow an analysis of susceptibility. As a general matter, EPA should use its information authorities to fill the gaps identified in the draft risk evaluation and these comments.

In particular, our comments identify numerous data gaps in Part I sec. 4. These data gaps include an absence of sufficient environmental monitoring data; environmental fate data; ecotoxicity data; product/use and concentration data; inhalation exposure data; dermal exposure data; dermal toxicity data; and reproductive/developmental/neurodevelopmental toxicity data. To prepare an adequate risk evaluation, EPA should use its information authorities to fill these data gaps.

D. EPA cannot assume that an absence of evidence about particular hazards or exposures provides evidence of that those hazards or exposures are absent.

When a data gap exists, EPA cannot rationally assume that the absence of evidence regarding a particular hazard or exposure establishes that the hazard or exposure is not present. As just one example, EPA acknowledges that the database for potential reproductive and developmental toxicity of 1,4-dioxane is deficient (p. 108), and hence that “it is not known whether or not pregnant women in the workplace may be at greater risk from exposure.” Yet in section 5.3.4 (p. 150), EPA states that it “did not include women of reproductive age or pregnant women who may work with 1,4-dioxane or children ages 16-21 because the acute effects on liver enzymes and CNS effects are not expected to preferentially affect women or developing children.” Here, EPA makes an inappropriate leap to claim that a lack of data is equivalent to lack of risk. When EPA has failed to collect information about particular hazards or exposures, it is arbitrary and capricious to assume that the lack of information establishes that the particular hazard or exposure does not exist. In addition, such assumptions violate EPA’s duty to consider all reasonably available information, which EPA could generate to fill these data gaps, as well as EPA’s duty to use the best available science.

E. EPA needs to implement the requirements of TSCA § 14 when reviewing materials for the risk evaluations.

EPA has an affirmative obligation to review at least 25% of non-chemical identity confidentiality claims under TSCA, 15 U.S.C. § 2613(g), and EPA has stated that it is implementing that obligation by “review[ing] every fourth submission received that contains non-chemical identity [confidential business information (CBI)] claims.”²¹⁶ Thus, on balance, EPA should be reviewing all confidentiality claims asserted in at least approximately one-fourth of the information submissions it receives. Those claims must be substantiated at the time of submission. EPA must complete reviews of confidentiality claims within 90 days of receipt of the claims, and if EPA denies a claim, EPA must disclose the information that had been claimed confidential 30 days after notifying the claimant of the denial, absent a challenge to the denial in district court. 15 U.S.C. § 2613(g)(1)(A), (g)(2)(B).

In addition, TSCA requires disclosure of “any health and safety study which is submitted under [TSCA] with respect to any chemical substance or mixture which, on the date on which such study is to be disclosed has been offered for commercial distribution.” 15 U.S.C. § 2613(b)(2)(A). TSCA also requires disclosure of “any information reported to, or *otherwise obtained by*, [EPA] from a health and safety study which relates to [such] a chemical substance. . .” *Id.* § 2613(b)(2)(B) (emphases added). Thus, any health and safety studies and related

²¹⁶ EPA REVIEW AND DETERMINATION OF CBI CLAIMS UNDER TSCA, <https://www.epa.gov/tsca-cbi/epa-review-and-determination-cbi-claims-under-tsca> (last visited Jan. 18, 2018).

information on 1,4-dioxane must be disclosed. TSCA defines “health and safety study” to mean “any study of any effect of a chemical substance or mixture on health or the environment or on both, including underlying information and epidemiological studies, studies of occupational exposure to a chemical substance or mixture, toxicological, clinical, and ecological studies of a chemical substance or mixture, and any test performed pursuant to this Act.” *Id.* § 2602(8). EPA has provided further details on this expansive definition of “health and safety study,” explaining that it encompasses, among other things, “[a]ny data that bear on the effects of a chemical substance on health or the environment” and “[a]ny assessments of risk to health and the environment resulting from the manufacture, processing, distribution in commerce, use, or disposal of the chemical substance.” 40 C.F.R. § 720.3(k). Thus, any health and safety study or other information on health or environmental effects or any assessment of risk EPA prepared must be disclosed. The only exception from that disclosure requirement is for “information *** that discloses processes used in the manufacturing or processing of a chemical substance or mixture or, in the case of a mixture, the portion of the mixture comprised by any of the chemical substances in the mixture.” 15 U.S.C. § 2613(b)(2).

In developing this draft risk evaluation, a large fraction of the information EPA relied upon constituted health and safety studies. All such information not subject to the two narrow exceptions needs to be made public.

8. EPA needs to ensure that environmental justice is appropriately considered, analyzed, and addressed in the risk evaluation.

Environmental justice is “the fair treatment and meaningful involvement of all people regardless of race, color, national origin, or income with respect to the development, implementation and enforcement of environmental laws, regulations and policies.”²¹⁷ According to EPA, providing “[f]air treatment” will ensure that “no group of people should bear a disproportionate share of the negative environmental consequences resulting from industrial, governmental and commercial operations or policies.”²¹⁸ EPA has committed to integrate environmental justice into “everything” the agency does in order to “reduce[] disparities in the nation’s most overburdened communities.”²¹⁹

Despite this commitment, and EPA’s obligations to comply with Executive Order 12898 (see below), EPA has not incorporated environmental justice considerations into the draft risk evaluation. In addition, EPA does not appear to have undertaken any outreach oriented towards ensuring the meaningful involvement of environmental justice communities in the risk

²¹⁷ EJ 2020 GLOSSARY, <https://www.epa.gov/environmentaljustice/ej-2020-glossary>.

²¹⁸ *Id.*

²¹⁹ U.S. EPA, *EJ 2020 Action Agenda* at 1 (2016), https://www.epa.gov/sites/production/files/2016-05/documents/052216_ej_2020_strategic_plan_final_0.pdf.

evaluation process. EPA must address environmental justice in the risk evaluation, both by incorporating an analysis into the evaluation and ensuring meaningful involvement by environmental justice communities in the development of the risk evaluation.

A. The risk evaluation is subject to Executive Order 12898.

Executive Order 12898 directed federal agencies to identify and address “disproportionately high and adverse human health or environmental effects of its programs, policies, and activities on minority populations and low-income populations.” Exec. Order No. 12898, 59 Fed. Reg. 7629 (Feb. 16, 1994). EPA must comply with this duty in the Executive Order. *See Sherley v. Sebelius*, 689 F.3d 776, 784 (D.C. Cir. 2012) (“[A]s an agency under the direction of the executive branch, it must implement the President’s policy directives to the extent permitted by law.”). The Executive Order applies, by its own terms, to all “programs, policies, and activities” of a federal agency, and EPA’s preparation of the risk evaluation undoubtedly fall within this capacious definition, qualifying as “activities” of EPA, carried out as part of its “programs” and pursuant to its “policies.” As an agency action that may affect the level of protection provided to human health or the environment, this risk evaluation under TSCA must address environmental justice communities.²²⁰ EPA’s own guidance on considering environmental justice defines “agency action” to include risk assessments.²²¹ EPA has articulated no theory for why the Executive Order would not apply to the risk evaluation.

Yet EPA has failed to mention, let alone adequately address, Executive Order 12898 or “environmental justice” in the draft risk evaluation. Failure to do so violates EPA’s obligations under the Executive Order.

Notably, EPA has stated that the identification of potentially exposed or susceptible subpopulations under TSCA would “carry[] out the spirit” of Executive Order 12898.²²² EPA’s implication that the act of merely identifying “potentially exposed or susceptible subpopulations,” standing alone, is sufficient to comply with the Executive Order, is plainly incorrect. The Executive Order specifically states that EPA must consider the disparate impacts of pollution on “minority populations and low-income populations.”²²³ The failure to do so in

²²⁰ *See* U.S. EPA, *EPA’s Action Development Process Interim Guidance on Considering Environmental Justice During the Development of an Action* at 18 (Jul. 2010), <https://www.epa.gov/sites/production/files/2015-03/documents/considering-ej-in-rulemaking-guide-07-2010.pdf>.

²²¹ *Id.* at 1.

²²² U.S. EPA, Risk Evaluation Rule Response to Comments at 1, <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0654-0109>.

²²³ Exec. Order No. 12898; *see also* U.S. Office of Inspector General, *EPA Needs to Consistently Implement the Intent of the Executive Order on Environmental Justice* at 9-10 (Mar. 2004), <https://www.epa.gov/sites/production/files/2015-10/documents/20040301-2004-p-00007.pdf>

the draft risk evaluation, in particular by failing to consider minority, low-income, and indigenous communities when identifying potentially exposed or susceptible populations, does not “carry out the spirit,” or the letter, of the Executive Order. EPA must prepare an actual environmental justice analysis to comply with the Executive Order.

B. EPA’s exclusions in the draft risk evaluation violate the Executive Order by underestimating the risks faced by environmental justice communities.

EPA’s decision to exclude environmental releases covered by other statutes because those statutes “adequately address” risk fails to acknowledge that other statutes have historically failed to consider environmental justice communities in permitting and enforcement. The National Environmental Justice Advisory Council (NEJAC), a federal advisory committee to EPA, has stated that:

Environmental protection in this country has grown by individual pieces of legislation, developed to address a particular environmental media or a pressing problem like abandoned toxic sites. Environmental law has not evolved from a master game plan or unifying vision. As a result, the statutes *have gaps in coverage* and do not assure compatible controls of environmental releases to all media from all sources.²²⁴

Those gaps in coverage were often a result of controlling pollution solely “through technology-based regulation or an individual chemical-by-chemical approach.”²²⁵ The Lautenberg Act’s unique emphasis on protecting “potentially exposed or susceptible subpopulations” recognized, in part, that the historical regulation of pollutants resulted in some subpopulations, including low-income, minority, and indigenous communities, being disproportionately impacted by chemical contamination.

In addition to the general gaps in coverage, environmental justice communities are often disproportionately exposed to sources of chemical contamination. For instance, a report by the General Accounting Office revealed that:

- three-quarters of hazardous waste landfill sites in eight southeastern states were located in communities whose residents were primarily poor and African-American or Latino, and

(explaining that the intent of the Executive Order, in part, was to place EPA’s focus on minority and low-income communities).

²²⁴ National Environmental Justice Advisory Council, Cumulative Risks/Impacts Work Group, *Ensuring Risk Reduction in Communities with Multiple Stressors: Environmental Justice and cumulative Risks/Impacts* at 7 (Dec. 2004), <https://www.epa.gov/sites/production/files/2015-04/documents/ensuringriskreductionnejac.pdf> (emphasis added).

²²⁵ *Id.* at 11.

- race and ethnicity were the most significant factors in deciding where to place landfills, waste and environmentally hazardous facilities.²²⁶

EPA's exclusion from the draft risk evaluation of exposure pathways resulting from environmental releases fails to recognize that environmental justice communities have not historically been protected by other environmental statutes and are often disproportionately exposed to chemical substances through disposal and other conditions of use. These exclusions will result in unfair treatment to environmental justice communities by ensuring that they will continue to "bear a disproportionate share of the negative environmental consequences resulting from industrial, governmental and commercial operations or policies."²²⁷

Moreover, EPA's exclusions of exposure pathways linked to disposal sites and legacy use, associated disposal, and legacy disposal will specifically underestimate the exposures of environmental justice communities. In fact, NEJAC has previously informed EPA of this exact concern:

It is particularly important to recognize historical exposures in communities and tribes suffering environmental injustice. In some cases, community members were exposed to pollutants for many years in the past from facilities that are *no longer functioning or in business*. These past exposures could act to increase the body burden of a subpopulation so that vulnerable individuals start off at a higher dose. Even if the dose-response curves among the subpopulation are the same as the general population, starting off at a higher point on this curve puts the members of the vulnerable subpopulation at greater risk for exposure to the same amount of a compound than the general population. This fact is highly pertinent to the historical legacy of racial and economic discrimination, and the relationship of vulnerability to health disparities.²²⁸

Failing to consider exposures linked to disposal, legacy uses, associated disposal, and legacy disposal systematically underestimates the background level of exposures faced by many environmental justice communities. In order to determine whether those communities will face

²²⁶ General Accounting Office, *Siting Hazardous Waste Landfills and Their Correlation with Race and Economic Status of Surrounding Communities* at 13-21 (1983), <https://www.gao.gov/products/RCED-83-168>.

²²⁷ EJ 2020 GLOSSARY, <https://www.epa.gov/environmentaljustice/ej-2020-glossary>.

²²⁸ National Environmental Justice Advisory Council, Cumulative Risks/Impacts Work Group, *Ensuring Risk Reduction in Communities with Multiple Stressors: Environmental Justice and cumulative Risks/Impacts* at 24 (Dec. 2004), <https://www.epa.gov/sites/production/files/2015-04/documents/ensuringriskreductionnejac.pdf> (emphasis added).

an unreasonable risk of injury from 1,4-dioxane, EPA must consider exposures from disposal, legacy uses, associated disposal, and legacy disposal.

9. Assessment factors do not lead to conservative calculations; in fact, assessment factors account for real-world sources of variability as well as database limitations.

In the problem formulation, EPA often stated that it used a “conservative approach” and “conservative assumptions” when assessing aquatic environmental exposures. *See, e.g.*, Problem Formulation for 1,4-Dioxane at p. 29. These statements at least in part appear based on EPA’s use of assessment factors (AFs) in developing the concentrations of concern (COCs). In fact, AFs account for real-world sources of variability as well as database limitations, and cannot be construed as “safety factors” that yield conservative estimates. As EPA acknowledges: “The application of AFs provides a lower bound effect level that would likely encompass more sensitive species not specifically represented by the available experimental data. AFs are also account for differences in inter- and intra-species variability, as well as laboratory-to-field variability” (p. 223).

The National Academy of Sciences, in its 2009 report titled *Science and Decisions: Advancing Risk Assessment* has this to say on this subject, albeit in the context of human rather than environmental health:

Another problem *** is that the term *uncertainty factors* is applied to the adjustments made to calculate the RfD [reference dose, derived from, e.g., a no-effect level] to address species differences, human variability, data gaps, study duration, and other issues. The term engenders misunderstanding: groups unfamiliar with the underlying logic and science of RfD derivation can take it to mean that the factors are simply added on for safety or because of a lack of knowledge or confidence in the process. That may lead some to think that the true behavior of the phenomenon being described may be best reflected in the unadjusted value and that these factors create an RfD that is highly conservative. But the factors are used to adjust for differences in individual human sensitivities, for humans’ generally greater sensitivity than test animals’ on a milligrams-per-kilogram basis, for the fact that chemicals typically induce harm at lower doses with longer exposures, and so on. At times, the factors have been termed *safety factors*, which is especially problematic given that they cover variability and uncertainty and are not meant as a guarantee of safety.²²⁹

²²⁹ NAT’L RESEARCH COUNCIL, *SCIENCE AND DECISIONS: ADVANCING RISK ASSESSMENT* at chp. 5, p. 132 (2009), <https://www.ncbi.nlm.nih.gov/pubmed/25009905> (emphases in original).

In evaluating risks, EPA should recognize that AFs ensure greater accuracy and do not provide a safety factor rendering the evaluation “conservative.”

10. EPA’s discussion of its systematic review methodology is insufficiently explained and suggests that EPA is taking an approach to the evidence that violates TSCA §§ 26(i) and 26(h).

In the problem formulation, EPA stated that it will rely on data and studies that meet the “systematic review” data quality criteria.

Human health hazards from acute and chronic exposures will be identified by evaluating the human and animal data that meet the systematic review data quality criteria described in the Application of Systematic Review in TSCA Risk Evaluations document (U.S. EPA, 2018). *** Hazards identified by studies *meeting data quality criteria* will be grouped by routes of exposure relevant to humans (oral, dermal, inhalation) and by cancer and noncancer endpoints.

Problem Formulation for 1,4-Dioxane at p. 51. EPA has not explained, either here or in its OCSP Systematic Review document, what it means for data or studies to “meet the systematic review data quality criteria.” EPA must do so.

Moreover, this language suggests EPA will apply its data quality criteria in a black-or-white manner: a study is either in or out. How is this consistent with the statute’s requirement that EPA take a weight-of-evidence approach? How is it consistent with the scientific standards in TSCA section 26(h), which require EPA to consider the “extent” or “degree” to which various factors characterize information, methods, models, etc. – which does not support the black-or-white approach EPA appears to intend to apply. EDF has previously explained that TSCA §§ 26(h) and 26(i) contemplate EPA weighing various information, see Appendix VI at 55-57, and EPA should implement those requirements consistent with that approach.

11. EPA’s description of systematic review is scientifically flawed and needs extensive revision to align with best practices and leading systematic review approaches.

EPA’s description of systematic review in the problem formulation is wholly deficient. Specifically, EPA describes systematic review as follows: “EPA/OPPT generally applies a systematic review process and workflow that includes: (1) data collection, (2) data evaluation and (3) data integration of the scientific data used in risk evaluations developed under TSCA.” Problem Formulation for 1,4-Dioxane at p. 14.

A. EPA fails to address protocol development, which is a fundamental component of systematic review.

A major deficiency in this description of EPA’s systematic review approach, and in its related OCSPP Systematic Review document, is the complete absence of protocol development—a fundamental component of systematic review.

As noted in the 2014 National Academy of Sciences (NAS) report that reviewed EPA’s IRIS program:

Critical elements of conducting a systematic review include formulating the specific question that will be addressed (problem formulation) and *developing the protocol* that specifies the methods that will be used to address the question (protocol development).²³⁰

After the systematic-review questions are specified, protocols for conducting the systematic reviews to address the questions should be developed. *A protocol makes the methods and the process of the review transparent, can provide the opportunity for peer review of the methods, and stands as a record of the review.* It also minimizes bias in evidence identification by ensuring that inclusion of studies in the review does not depend on the studies’ findings. Any changes made after the protocol is in place should be transparent, and the rationale for each should be stated. EPA should include protocols for all systematic reviews conducted for a specific IRIS assessment as appendixes to the assessment.²³¹

EPA’s IRIS program reflects this NAS recommendation by developing problem formulation and assessment protocols for each of its assessments.²³² OCSPP needs to develop full protocols for each of its risk evaluation, and should consult with the IRIS program on how best to do so in consideration of requirements under TSCA.

B. EPA fails to describe its approach to evidence integration (weight of evidence) despite claims that it has done so in the problem formulation.

EPA has also failed to describe its approach to evidence integration at all. In multiple instances, EPA points to its OCSPP Systematic Review document as providing more information on how it plans to conduct evidence integration. For example, EPA states:

²³⁰ Nat’l Research Council, *Review of EPA’s Integrated Risk Information System (IRIS) Process* at p. 5 (2014), <https://www.ncbi.nlm.nih.gov/books/NBK230060/> (emphasis added).

²³¹ *Id.* at 6 (emphases added).

²³² U.S. EPA, Office of Research & Dev., National Academy of Science Committee to Review Advances Made to the IRIS Program at slide 23 (Feb. 2018), <http://nas-sites.org/dels/files/2018/01/AdIRIS-15.pdf>.

Evaluate the weight of the evidence for consumer exposures. EPA will rely on the weight of the scientific evidence when evaluating and integrating data related to consumer exposure. The weight of the evidence may include qualitative and quantitative sources of information. The data integration strategy will be designed to be fit-for-purpose in which EPA will use systematic review methods to assemble the relevant data, evaluate the data for quality and relevance, including strengths and limitations, followed by synthesis and integration of the evidence. *Refer to the Application of Systematic Review in TSCA Risk Evaluations (U.S. EPA, 2018) document for more information on the general process for data integration.*

Problem Formulation for 1,4-Dioxane at p. 49.

In fact, EPA has not described its approach to data (evidence) integration in any of its problem formulations, nor in its OCSPP Systematic Review document. Indeed, OCSPP has not described its approach to evidence integration anywhere. Instead, it appears that EPA intends to do so in each individual draft chemical risk evaluation and in the absence of a protocol established up front. This approach is hugely problematic, lending itself to bias and inconsistency in how EPA conducts weight of evidence across risk evaluations. EPA should describe its general approach to evidence integration in a revised systematic review methodology document and then incorporate that into specific protocols it develops for each risk evaluation (see EDF's comments on EPA's OCSPP Systematic Review document).

* * * * *

More broadly, in revising its approach to conducting systematic review, we recommend that OCSPP consult with IRIS, the National Toxicology Program's Office Health Assessment and Translation, and other leading experts on the application of systematic review for chemical assessment, as discussed further in EDF's comments on EPA's OCSPP Systematic Review document.²³³

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EDF appreciates the opportunity to provide comments and EPA's consideration of them.

²³³ EDF Comments on Application of Systematic Review in TSCA Risk Evaluations (Aug. 16, 2018), <https://www.regulations.gov/docket?D=EPA-HQ-OPPT-2018-0210>.