Environmental Defense Fund
Comments on the Draft Risk Evaluation of 1-Bromopropane
Docket ID: EPA-HQ-OPPT-2019-0235

Submitted October 11, 2019

Environmental Defense Fund (EDF) appreciates the opportunity to provide comments to the Environmental Protection Agency (EPA) on the draft risk evaluation for 1-bromopropane (1-BP) 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 Science Advisory Committee on Chemicals (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; this is 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-BP, the Environmental Protection Agency (EPA) has grossly understated the risks 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 thoroughly identify and evaluate the risks to vulnerable subpopulations. 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-BP's uses, hazards and exposures, resulting in serious information and analytic gaps and deficiencies that severely undermine the scientific quality of its risk evaluation.

[1] U.S. EPA, *Draft Risk Evaluation for 1-Bromopropane CASRN: 106-94-5* (August 2019), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0235-0022. Further citations in these comments of the draft risk evaluation for 1-bromopropane consist of only a page number in parentheses.

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 failed to sufficiently identify and address potential hazards, exposures, and risks of 1-BP to several vulnerable subpopulations (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 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 section 1.C.i and ii). EPA must obtain and make public the full studies.
- EPA's risk evaluation lacks an adequate mass balance to account for the lifecycle of 1-BP (see section 1.C.iii).

Unwarranted exclusions of conditions of use and exposure pathways

- EPA has excluded or overlooked specific conditions of use without providing adequate documentation or rationale (see section 2.A).
- EPA has excluded from its risk evaluation all general population exposures to 1-BP from releases to air and land, 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-BP in these media pathways (see section 2.B).
- EPA has collapsed into a single category a highly diverse set of industrial/commercial uses that encompass a huge array of sectors, from hobby materials to construction materials to laboratory chemicals, and very different functional uses (see section 2.C).
- EPA has ignored 1-BP's use in spray foam blowing and as a flame retardant without providing any rationale (see section 2.D).

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

- EPA must maintain its adoption of a linear, no-threshold approach for 1-BP's carcinogenicity, based on: decisions by other authoritative bodies; available evidence; scientific rebuttals of a postulated nonmutagenic mode of action and a flawed study questioning the relevance of mouse lung tumors to humans; and policies firmly rooted in scientific and health-protective principles (see section 3.A.-E).
- EPA should migrate to a unified approach to presenting dose-specific population risks for both cancer and noncancer endpoints (see section 3.F).

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 use information, dermal toxicity data, or inhalation or dermal exposure data (see section 4.B).

Analytic gaps and deficiencies

Environment

- EPA's disregard of environmental monitoring data led to an overreliance on predictive modeling lacking adequate uncertainty analysis (see section 5.A.i).
- EPA has inappropriately dismissed sediment and terrestrial toxicity and exposure pathways (see section 5.A.ii).
- EPA over-relies on limited and incomplete TRI data to exclude or dismiss the significance of numerous releases and exposure pathways (see section 5.A.iii).

Human Health

- EPA has illegitimately dismissed human studies of 1-BP exposure (see section 5.B.i).
- EPA has excluded without justification identified hazards of 1-BP from its quantitative risk characterization (see section 5.B.ii).
- 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.iii).
- EPA has failed to consider cancer risk from acute exposure scenarios (see section 5.B.iv).
- 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.v).
- 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.vi and vii).
- EPA may have underestimated risks to occupational non-users (ONUs) and has inadequately addressed uncertainties in its dermal risk estimates (see section 5.B.viii and ix).
- EPA appears to have ignored a significant source of data on inhalation exposure (see section 5.B.x).
- EPA has failed to explain or justify its assumption of one exposure event per day (see section 5.B.xi).
- EPA has failed to address exposure and risk to children, a susceptible subpopulation, from 1-BP's use in dry cleaning (see section 5.B.xii).
- EPA has inadequately assessed consumer exposures to insulation (see section 5.B.xiii).

Understating risks to workers

- EPA has significantly understated both the extent of its unwarranted assumption of PPE use by workers and the risks to workers it has identified (see section 6.A).

- EPA's assessment of dermal risk likely underestimates exposure due to its crude assumptions about glove use and efficacy (see section 6.B).
- EPA's approaches to both aggregate and sentinel exposures are flawed (see section 6.C).

Flaws (and one bright spot) 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 appropriately found unreasonable risk when high-end risk exceeds relevant benchmarks, an approach that is needed to be 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 assumptions of PPE use and a 1-in-10,000 acceptable risk levels for workers conflates risk evaluation and risk management and significantly understates risk (see section 7.D).
- 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

- OPPT does not provide explanation or 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 (see section 8.A).
- OPPT's approach taken to evidence integration in the draft 1-BP 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) (see section 8.B).
- OPPT's inconsistent application of its systematic review criteria results in an arbitrary and capricious analysis (see section 8.C).
- EPA inappropriately excluded from systematic review several studies because they were in other languages and EPA had failed to request them (see section 8.D).

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PART I

PART I

1. Broad/cross-cutting concerns

A. Insufficient consideration of susceptible subpopulations

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

- Workers: Sections 1.B., 2.C., 5.B.vi.-xi., 6.C., 7.A., 7.C, 7D.
- Consumers: Sections 2.C., 2.D., 4.B.iii., 5.B.ii., 5.B.xii.
- Children: Section 5.B.xii.
- People in proximity to conditions of use or sources of contamination: Section 5.B.v.

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 workers, at all points in the value chain and lifecycle of 1-BP, will always use personal protective equipment (PPE) (gloves and respirators) and that it will be universally effective:

> EPA expects there is compliance with federal and state laws, such as worker protection standards, unless case-specific facts indicate otherwise, and therefore existing OSHA regulations for worker protection and hazard communication will result in use of appropriate PPE consistent with the applicable SDSs in a manner adequate to protect them (p. 289).

These assumptions are wholly unwarranted. In addition to grossly distorting OSHA authorities and requirements (see below), EPA has provided no data or analysis whatsoever to support these sweeping assumptions. Rather, the agency makes clear that it does not have any actual data on respirators or gloves, such as types used and frequency, by stating elsewhere in the draft risk evaluation that:

> • "Few literature sources indicate the use of respirators in 1-BP conditions of use ***;" (p. 57) and
> • "EPA does not know the actual frequency, type, and effectiveness of glove use in specific workplaces with 1-BP conditions of use." (p. 108)

There is considerable evidence of major real world limitations of PPE, with regards to both the extent of use and effectiveness. In fact, OSHA has highlighted the major limitations of reliance

on PPE, as has EPA in the recent past. These issues are discussed in detail in previous EDF comments, which are incorporated here by reference.[2]

For 1-BP in particular, any OSHA requirement for employers to provide respiratory protection from 1-BP exposure will apply only extremely rarely for many reasons, including the fact that no OSHA permissible exposure limit (PEL) exists for 1-BP. EPA distorts the relevant OSHA requirements when it invokes OSHA's Respiratory Protection Standard at 29 CFR § 1910.134 (p. 57) – which only applies for chemicals with an OSHA PEL. Dr. Finkel's comments on 1-BP submitted for consideration by the SACC discuss this issue further; we urge EPA to carefully consider those comments.[3]

Second and more broadly, even where OSHA respiratory protection requirements do apply to a chemical, OSHA's database of inspections demonstrates significant noncompliance with those requirements. In fiscal year 2018 alone, OSHA cited 2,892 violations of the respiratory protection standard identified in 1,281 separate inspections.[4] Violations of the respiratory standard were the 4th most common type of violation in OSHA inspections that year, exceeded only by those for two categories of physical hazard and the Hazard Communication Standard.[5]

Two CDC reports – both cited by EPA in another context but ignored in its discussion of PPE – serve to further illustrate the serious limitations of relying on PPE to protect workers from 1-BP exposure. First, a 2008 MMWR report[6] linked neurological illness in two workers in the electronics and dry cleaning industries to 1-BP exposure. Both workers presented with

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[2] *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.

[3] Comment submitted by Adam M. Finkel to SACC on 1-BP draft risk evaluation, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0235-0026.

[4] U.S. Department of Labor, Occupational Safety and Health Administration, Industry Profile for OSHA Standard 19100134, https://www.osha.gov/pls/imis/industryprofile.stand?p_esize=&p_stand=19100134&p_state=FEFederal&p_type=5 (last visited Sept. 9, 2019). These FY 2018 statistics have been replaced with the FY 2019 data and appear not to be currently accessible anymore.

[5] U.S. Department of Labor, Occupational Safety and Health Administration, Top 1- Most Frequently Cited Standards, https://www.osha.gov/top10citedstandards (last visited Sept. 9, 2019).

[6] U.S. Center for Disease Control and Prevention, *Neurologic Illness Associated with Occupational Exposure to the Solvent 1-Bromopropane --- New Jersey and Pennsylvania, 2007—2008*, MORBIDITY AND MORTALITY WEEKLY REPORT (Dec. 5, 2008), https://www.cdc.gov/mmwr/preview/mmwrhtml/mm5748a2.htm.

neurological symptoms such as dizziness, nausea, and confusion, and visited the emergency department. One was hospitalized. The investigation demonstrated that both workers did not, or did not typically, wear PPE while handling 1-BP. Second, a 2010 CDC and NIOSH report evaluated 1-BP use in four New Jersey dry cleaning facilities.[7] Among other findings, the authors reported that "[r]espirators, gloves, and eye protection were not being used" and "[r]espirators were not equipped with the correct cartridges for 1-BP."

Even when respirators and gloves *are* used, workers may still be exposed to 1-BP. In his comments, Dr. Adam Finkel, former Director of OSHA Standards, raised a critical issue with respirators specifically in the context of 1-BP exposure.[8] He describes that organic solvents like 1-BP may breakthrough the carbon or other medium in organic vapor cartridges, and a SACC member during the September 10-12, 2019, peer review meeting noted further that this can occur without providing any indication to the user that the respirator is no longer functioning. Dr. Finkel calculated that breakthrough would occur for 1-BP within 117 minutes – less than two hours. This is the same issue known to occur with methylene chloride, which EPA has acknowledged necessitates use of air-supplied respirators.[9] Gloves may also provide limited protection from 1-BP exposure. EPA also acknowledges on p. 106 of the draft risk evaluation, citing a 2013 OSHA Hazard Alert, that "1-BP easily travels through most glove materials. Recommended glove materials for protection against 1-BP are supported polyvinyl alcohol or multiple-layer laminates." Despite acknowledging this critical issue, the agency simple uses default glove protection factors (PFs) and excludes dermal exposure in occluded scenarios (see Part I, sec. 6.B. for further discussion).

In a few places in the draft, EPA acknowledges some of the limitations of PPE (p. 57, 206), and the preferability of other options higher up in the industrial hygiene hierarchy of controls (p. 57). 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 (see Part I, sec. 6.A. of these comments describing the extent of EPA's reliance). To do so, EPA unrealistically assumes that "workers are properly trained and fitted on respirator use, and that they wear respirators for the entire duration of the work activity where there is potential exposure to 1-BP." (p. 24) As just one example, EPA finds no unreasonable risk for non-cancer acute inhalation occupational use of 1-BP in manufacturing – despite the fact that its MOE for high-end exposure is substantially *lower* than its benchmark MOE (63 and 100, respectively) – only by assuming

[7] National Institute for Occupational Safety and Health, Evaluation of 1-Bromopropane Use in Four New Jersey Commercial Dry Cleaning Facilities (July 2010), https://www.cdc.gov/niosh/hhe/reports/pdfs/2008-0175-3111.pdf

[8] Comment submitted by Adam M. Finkel to SACC on 1-BP draft risk evaluation, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0235-0026.

[9] 82 Fed. Reg. 7,464, 7,474 (Jan. 19, 2017), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0231-0001.

universal and effective use of a respirator with an assigned protection factor (APF) of 10 (see Table 4-6, p. 195).

EPA also repeatedly overstates or distorts OSHA's authorities and requirements, claiming that OSHA requires employers to provide PPE (p. 289), implying that OSHA requires the use of respirators for 1-BP (p. 57), 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-BP (p. 289). In fact, OSHA authorities and requirements are quite limited and leave most of 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.[10] EDF incorporates those posts by reference. EDF also incorporates by reference the comments submitted to EPA by Jonathan Kalmuss-Katz and Randy Rabinowitz.[11]

Even if compliance with SDSs *were* mandatory, reliance on them would still be insufficient to ensure use of protective measures by all downstream processors and 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.[12] The 2012 OSHA Hazard Communication Standard corroborates these findings.[13] 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."[14]

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

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[10] *See* Appendix A.
[11] 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.
[12] 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.
[13] 77 Fed. Reg. 17574, 17593-95, 17603 (Mar. 26, 2019).
[14] *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.

to adequately communicate health hazards.[15,16] 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 relied upon to offer adequate information on the inherent health and safety hazards, including handling and storage of engineered nanomaterials."

EPA's reliance on PPE is not merely a policy determination. It is a huge assumption that dramatically alters EPA's risk characterization for 1-BP. EPA's reliance on PPE is the foundation of EPA's many no-unreasonable-risk determinations for workers even though EPA has acknowledged, for example, that "[f]ew literature sources indicate the use of respirators in 1-BP conditions of use" (p. 57) and "EPA *does not know* the actual frequency, type, and effectiveness of glove use in specific workplaces with 1-BP conditions of use" (p. 108, emphasis added). EPA's reliance on PPE also leads the agency to understate the magnitude of risk even where it does identify unreasonable risk. *See* Part I, sec. 6.A. of these comments. EPA's failure to provide *any* supporting data that PPE is universally used and effective, as assumed in its risk determinations, is a glaring flaw in this draft risk evaluation.

In its risk determinations, EPA has masked the extent of its reliance on PPE, by failing to be clear about when PPE (as well as engineering controls) are and are not assumed. This point was repeatedly made by SACC members during its September 10-12, 2019, peer review meeting on 1-BP.

Part I, section 6.A. of these comments presents an analysis showing that, for virtually every condition of use of 1-BP where PPE might plausibly be used, EPA found no unreasonable risk only by assuming that workers wear PPE to protect against both inhalation and dermal exposures. For those conditions of use where EPA did identify unreasonable risk, it was compelled to do so because even the most stringent level of PPE protection EPA examined and assumed would be used was insufficient to eliminate that risk.

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[15] 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.

[16] 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.

C. Lack of transparency

i. EPA lacks access to full studies and relies only on summaries, prepared by industry, of limited aquatic toxicity testing to conclude 1-BP presents no unreasonable risks to the entire environment.

a. EPA has mischaracterized data it cites as sourced from ECHA.

EPA 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). On p. 43 of its draft risk evaluation, EPA claims ECHA dossiers are existing chemical assessments equivalent to EPA and ATSDR governmental assessments:

> Examples of existing assessments are EPA's chemical assessments (e.g. previous work plan risk assessments, problem formulation documents), ATSDR's Toxicological Profiles, EPA's IRIS assessments and *ECHA's dossiers*.

But in fact ECHA dossiers are not assessments and are not government documents. They are compilations of *industry* information submitted to ECHA that have *not* been evaluated for quality or reliability by ECHA or any other governmental entity. For EPA to equate them with EPA and ATSDR assessments is simply wrong.

ECHA's posting of the dossiers themselves makes clear the lack of government review. At the bottom of each page of each dossier[17] is the following statement (emphasis added):

> Information on Registered Substances comes from registration dossiers which have been assigned a registration number. The assignment of a registration number does however not guarantee that the information in the dossier is correct or that the dossier is compliant with Regulation (EC) No 1907/2006 (the REACH Regulation). This information has not been reviewed or verified by the Agency or any other authority.

EPA exacerbates the mischaracterization through its text references to the industry's dossiers. EPA typically cites the dossiers posted on the ECHA website through a hyperlinked reference in the text of its documents that reads "ECHA, [date]." Clicking on that link takes the reader to EPA's entry for that source in its Health & Environmental Research Online (HERO) data

[17] *See* 1-BROMOPROPANE, https://echa.europa.eu/de/registration-dossier/-/registered-dossier/15004 (last visited Oct. 9, 2019).

system. Those HERO entries then prominently list the "European Chemicals Agency" as the reference's author.[18]

Such text citations and HERO entries are false and highly misleading. All of these documents were prepared by the industry registrants, not ECHA. And the information has not been evaluated by ECHA or any other governmental entity.

While some chemicals do eventually undergo a "substance evaluation" by government authorities under REACH, 1-BP has not. Chemicals that an EU authority "has evaluated or will evaluate [] over the coming years" are listed on the ECHA website.[19] Of the chemicals for which EPA has released draft risk evaluations, only Pigment Violet 29 (CAS # 81-33-4) is listed, and that is because it has been identified by the EU as a "suspected PBT [persistent, bioaccumulative and toxic] or vPvB" [very persistent, very bioaccumulative] chemical and as a result is scheduled to undergo a substance evaluation in 2021.[20]

b. EPA lacks access to the full studies in the ECHA dossier and did not subject them to systematic review.

The ECHA dossiers EPA has cited contain only summaries of studies, not the studies themselves, and those summaries were prepared by the registrant, not ECHA.

EPA's Charge Question 5.1 to the SACC[21] states that (emphasis added):

> Only a few environmental test data endpoints (including ECHA) are available in the public domain for 1-BP. Most are from the ECHA website. *EPA attempted to obtain the full ECHA studies with no success.* Since the studies were in French and Japanese (and no U.S.A. sponsor), EPA decided not to make further attempts to find the studies. Given that the ECHA environmental test data results are in the public domain, EPA decided to use the experimental data.[22]

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[18] As an example, see this EPA HERO citation to the industry registrant's 1-Bromopropane dossier: https://hero.epa.gov/hero/index.cfm/reference/details/reference_id/3827329.
[19] *See* SUBSTANCE EVALUATION – CORAP, https://echa.europa.eu/information-on-chemicals/evaluation/community-rolling-action-plan/corap-table (last visited Oct. 9, 2019).
[20] *See* Richard Denison, *EPA says PV29 is perfectly safe. The EU, citing concerns and a dearth of data, begs to differ*, EDF BLOG POST (May 7, 2019), http://blogs.edf.org/health/2019/05/07/epa-says-pv29-is-perfectly-safe-the-eu-citing-concerns-and-a-dearth-of-data-begs-to-differ/.
[21] *See* EPA Scientific Advisory Committee on Chemicals Charge to the Panel – 1-Bromopropane (1-BP) CASRN: 106-94-5 p. 6 (July 2019), https://www.epa.gov/sites/production/files/2019-08/documents/02._1-bp_draft_re_charge_questions_8_09_2019.pdf.
[22] *Id.*

As we have described in previous comments, EPA needs access to 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. 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. We incorporate our previous comments by reference.[23]

EPA itself has forthrightly stated this very need. In a request EPA sent to industry requesting full studies on Pigment Violet 29, obtained through a FOIA request made by EDF and other groups,[24] EPA states (emphasis added):

> [S]ummary study results do not provide sufficient information upon which the hazard(s) and risk(s) from manufacture, distribution in commerce, processing, use, or disposal of this substance or any combination of such activities on health or the environment can reasonably be determined or predicted. *** *EPA needs to review the full study reports to confirm the information in the summaries meets the scientific standards set forth in TSCA section 26.*

One other reason why EPA (as well as public and peer reviewer) access to full studies is essential: As EDF uncovered recently, industry registrants can change – and have changed – their ECHA study summaries at will without any requirement that they even give notice of having done so.[25]

Despite this, EPA indicates it used the ECHA summaries to characterize environmental hazards (p. 138). Not only is EPA relying only on summaries and lacks access to the full studies, it is

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[23] *See, e.g.*, EDF Problem Formulation Comments at p. 61, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0741-0085; 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.

[24] Letter from Maria Doa, U.S. EPA, Director of Chemical Control Division, to Martijn Schoonenberg, Sun Chemical Group p. 1 (Sept. 15, 2017) http://blogs.edf.org/health/files/2019/09/Response-1-and-6.-Letter-to-Sun-Chemical-Group-Cooperatief-U.A._PV29.9-15-17_Redacted.pdf.

[25] Richard Denison, *Industry deletions in PV29 study summaries should raise alarm bells on both sides of the Atlantic*, EDF BLOG POST (May 2, 2019), http://blogs.edf.org/health/2019/05/02/industry-deletions-in-pv29-study-summaries-should-raise-alarm-bells-on-both-sides-of-the-atlantic/.

relying on industry-prepared summaries that it cannot independently verify and that no government entity has evaluated for quality or accuracy. EPA indicates on p. 138 that the ECHA summaries were not subjected to systematic review, ironically invoking its lack of access to the full studies as an excuse.

Furthermore, it appears that the ECHA study summaries completely bypassed the data screening step of the literature search process (p. 44):

> These are key and supporting studies from existing assessments (e.g., EPA IRIS assessments, ATSDR assessments, ECHA dossiers) that were considered highly relevant for the TSCA risk evaluation. These studies bypassed the data screening step and moved directly to the data evaluation step.

To the extent that the screening step is more than a screen for relevance and implies any sort of quality or reliability evaluation, bypassing the screening step for ECHA dossiers is totally inappropriate.

But none of this stopped EPA from using the summaries in its analysis. On the following pages, EPA describes the results of these studies reported in the summaries, even while indicating that, because it could not obtain the full studies, they were not reviewed for study quality. These studies comprise nearly all of the acute aquatic toxicity data EPA has.

c. EPA has limited data and over-relies on ECHA study summaries.

EPA proceeds to use the results of the ECHA-sourced studies it has never obtained to draw conclusions about environmental risk.

With regards to acute aquatic toxicity, EPA states on p. 141 (emphasis added):

> As a result, *only a single acute fish toxicity study* identified during the literature search process (Geiger et al., 1988) has been evaluated according to the systematic review criteria in *The Application of Systematic Review in TSCA Risk Evaluations* (U.S. EPA, 2018a). Although full studies summarized in ECHA have not been evaluated for data quality, according to the systematic review criteria in *The Application of Systematic Review in TSCA Risk Evaluations*, a qualitative consideration of the results of these summaries indicates that the hazard conclusions of these summaries are consistent with the results of the fish study that was reviewed for data quality. All studies indicate that 1-BP presents a low or moderate hazard to aquatic environmental receptors. As a result, the environmental hazards are primarily described using the acute fish study, which was rated as high confidence (Geiger et al., 1988) (EPA, 2019l). This study

> constitutes the best available data to assess the environmental hazards of 1-BP. In an effort to utilize all available data characterizing the environmental hazards of 1-BP, the data presented in the ECHA study summaries were considered to contextualize and characterize the potential hazards and risks of 1-BP to aquatic receptors.

With respect to chronic aquatic hazard, EPA states on pp. 139-140 (emphasis added):

> As *no data were available* to characterize the hazards of chronic exposure to aquatic species, EPA estimated hazards from chronic exposure using an acute-to-chronic ratio (ACR). The most sensitive species following acute exposure, which in this case were freshwater fish, with a 96-hr LC50 of 67.3 mg/L (the value (Geiger et al., 1988) and 24.3 mg/L (ECHA, 2017) (EPA, 2019d) were divided by an ACR of 10 to estimate chronic values (ChV) for fish. This results in a fish chronic value (ChV) of 67.3 mg/L/ 10= 6.73 and 24.3 mg/L/10= 2.43 mg/L, respectively. This approach was also used for aquatic invertebrates, where the 48-hr LC50 of 99.3 mg/L is divided by an acute-to-chronic ratio (ACR) of 10 to obtain a chronic value (ChV) for aquatic invertebrates. This results in a ChV of 99.3 mg/L/10= 9.93 mg/L. (emphasis added).

Both the fish 24.3 mg/L and invertebrate 99.3 mg/L values are those reported by industry in the ECHA dossier. EPA relies on both values. EDF previously criticized EPA's resorting to use of an ACR in our comments on the Problem Formulation for 1-BP (see end of section 23 on p. 97). That critique remains relevant, and EPA has never responded to this point. Specifically, EPA provides no justification for its application of an 'acute-to-chronic ratio' or its specific value of 10, nor does it provide even a citation to the use of such values in other contexts. Even a cursory search of the literature indicates that an ACR of at least 100 may be needed to be sufficiently protective.[26]

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[26] *See* Martin May, et al., *Evaluation of acute-to-chronic ratios of fish and Daphnia to predict acceptable no-effect levels*, 28:1 ENVTL. SCIENCES EUROPE 16 (2016), https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5044967/; Jan Ahlers, et al., *Acute to chronic ratios in aquatic toxicity - Variation across trophic levels and relationship with chemical structure*, 25:11 ENVTL. TOXICOLOGY & CHEMISTRY (Dec. 2009), https://setac.onlinelibrary.wiley.com/doi/abs/10.1897/05-701R.1 ("For fish, daphnids, and algae, acute to chronic ratios (ACRs) have been determined from experimental data regarding new and existing chemicals. Only test results in accord with the European Union Technical Guidance Document (TGD) and validated by authorities were considered. Whereas the median ACRs of 10.5 (fish), 7.0 (daphnids), and 5.4 (algae) are well below the ACR safety factor of 100 as implied by the TGD, individual ACRs vary considerably and go up to 4400. The results suggest that a safety factor of 100 is not protective for all chemicals and trophic levels. Neither a correlation between ACR and baseline toxicity as modeled through the logarithmic octanol-water partition coefficient nor an ACR correlation across trophic levels exists. Narcosis is associated

Note that:

- EPA uses the ECHA value of 24.3 mg/L for acute fish toxicity as part of the basis for its fish chronic calculation even though that value has not been subject to any quality review because EPA lacks the full study.
- EPA characterizes the only other value it has, from Geiger et al., as the value for the "most sensitive species following acute exposure, which in this case were freshwater fish" (p. 140). That value is higher than the value from the ECHA-sourced study, however.
- EPA also uses the ECHA value of 99.3 mg/L for acute invertebrates (first cited on p. 139) as the sole basis for its invertebrate chronic calculation even though that value has not been subject to any quality review because EPA lacks the full study.

EPA also has no toxicity data for sediment or terrestrial organisms and no monitoring data (pp. 140, 188, 146). On p. 47, EPA states: "During problem formulation, EPA made refinements to the conceptual models resulting in the elimination of the terrestrial exposure pathway from further analysis. Thus, environmental hazard data sources on terrestrial organisms were considered out of scope and excluded from data quality evaluation." Instead, EPA resorts to vague arguments invoking the 1-BP's physical-chemical properties to argue there will be little or no *exposure* (pp. 23, 140-141, 186).

Based only on the above very limited data and questionable analysis, EPA nevertheless draws a sweeping conclusion about the entire environment: "As a result, EPA determined that 1-BP does not present unreasonable risk to the environment under the identified conditions of use." (p. 23) EPA's analysis is wholly insufficient to establish that 1-BP does not present an unreasonable risk to the environment.

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

In its draft risk evaluation, EPA acknowledged that ECHA provides only study summaries and that EPA has not obtained the full study reports.[27]

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with a preference for a low ACR; nevertheless, low ACRs are frequently obtained for nonnarcotics. Analysis of chemical structures led to the derivation of structural alerts to identify compounds with a significantly increased potential for a high ACR, which may prove to be useful in setting test priorities. At present, however, life-cycle tests are the only way to conservatively predict long-term toxicity.") (emphases added).

[27] 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.

EPA not only needs to obtain copies of the full studies, it also needs to make full copies of these and other studies on which it relies available to the public. 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.[28] 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.

Our earlier comment about the need for EPA access to full studies applies equally to public access: 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.

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 full studies here. *Id*.

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

As discussed by multiple SACC members, EPA's draft risk evaluation has failed to account for 1-bromopropane's presence and flow at the different stages of its lifecycle. The SACC members emphasized that over 25 million pounds of 1-BP are manufactured in or imported into the United States annually (p. 19), yet less than one million pounds of 1-BP were identified as released to the air and only five pounds to surface water; the draft risk evaluation does not make clear where the rest of it goes. In order to provide transparency, SACC members recommended, and EDF agrees, that EPA should show a mass balance for 1-bromopropane. While the term "mass balance" can mean different things,[29] it is appropriate to look at the definition under the Emergency Planning and Community Right-to-Know Act (EPCRA), under which EPA must collect release data on chemicals through the Toxics Release Inventory (TRI). According to EPCRA, mass balance is "an accumulation of the annual quantities of chemicals transported to a facility, produced at a facility, consumed at a facility, used at a facility, accumulated at a facility, released from a facility, and transported from a facility as a waste or as a commercial product or

[28] *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.

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

byproduct or component of a commercial product or byproduct." 42 U.S.C. § 11023(l)(4). While EPA relies on the CDR and TRI to compile some estimates of these values, there are limitations on both of those reporting schemes that result in an incomplete picture of the chemical's lifecycle.

As reported by the National Research Council (NRC):

> Congress was aware that the toxic chemical release estimates reported under [TRI] might not accurately reflect the amounts actually released from reporting facilities (U.S. Congress, House, 1986). This potential inaccuracy is based on the provision that quantities of chemical releases can be obtained from theoretical calculations, engineering estimates, or by subtracting mass balance quantities (e.g., chemical quantity purchased minus the quantity contained in the product) rather than from measurements of actual releases."[30]

In order to conduct a robust and transparent risk evaluation on 1-BP, and more generally on chemical substances, EPA must provide significantly more detail about the chemical's lifecycle by conducting a mass balance analysis. Section 26(h)(3) requires the Administrator to consider the "degree or clarity and completeness with which [ ] data *** are documented," and without a mass balance analysis EPA has not reached any reasonable degree of clarity. 15 U.S.C. § 2625(h)(3).

2. Exclusions of conditions of use and exposures

A. Exclusions or overlooking of specific conditions of use, including consumer uses

At the problem formulation step, EDF criticized the basis of EPA's exclusions from the scope of its risk evaluation. Those exclusions have been retained in the draft risk evaluation.

EPA has excluded multiple conditions of use with no legal basis and limited factual support. On page 19 of the draft risk evaluation, EPA states those conditions of use include:

> non-pesticidal agricultural products (since 1-BP is only used in the processing of such products and is not present in the final non-pesticidal agricultural products), adhesives in consumer products (except as an adhesive accelerant for arts and crafts), and engine degreasing or brake cleaning in consumer products (since these are only commercial products) (p. 19).

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

EDF has previously commented on the limited factual basis for these exclusions, which is addressed in Part II, sec. 1.D. EDF has also previously explained why EPA's pick-and-choose approach to conditions of use is contrary to the law, which is addressed in Part II, sec. 1.A.

B. Exclusions based on other statutes

Referencing its earlier problem formulation, EPA has excluded from its risk evaluation certain general population exposures to 1-BP, based on EPA's assertion – unsupported by any actual data or analysis – that "other environmental statutes administered by EPA would adequately assess and would effectively manage these exposures" (p. 258; see also pp. 27, 38). EPA has excluded two exposure pathways on this basis:

- Air pathway –EPA asserted that 1-BP would be adequately assessed under the Clean Air Act (CAA) because at some point in the future 1-bp will be listed as a Hazardous Air Pollutant (HAP) (p. 258).
- Disposal pathway – In the problem formulation, EPA stated that, under the Resource Conservation and Recovery Act (RCRA), exposures to the general population would be "adequately assess[ed] and effectively manage[d]" (Problem Formulation, p. 54).

Aside from the absent legal basis, discussed at Part II, sec. 5, these exclusions present significant health concerns for the general population.

As EPA explained in the Problem Formulation for 1-BP:

> 1-BP migration to groundwater from RCRA Subtitle C landfills or RCRA Subtitle D municipal landfills regulated by the state/local jurisdictions to groundwater *will likely be mitigated* by landfill design (double liner, leachate capture for RCRA Subtitle C landfills and single liner for RCRA Subtitle D municipal landfills) and requirements to adsorb liquids onto solid adsorbent and containerize prior to disposal. Problem Formulation, p. 32 (emphasis added).

Reflected perhaps in the conditional language it uses ("will likely be mitigated"), EPA neither provides nor cites any data or analysis to support this sweeping assertion. Where authority is or can be delegated to states, as is the case with RCRA, differential state enforcement of laws and regulations can mean that the actual extent of protection from risks can vary greatly. *See* Part II, sec. 5.E. Also, a 2011 report from EPA's Office of the Inspector General extensively

documented insufficient EPA oversight of state enforcement as well as large state-to-state variations.[31]

Later in the problem formulation, EPA drops even the conditional language and asserts unequivocally as to the adequacy of existing disposal regulations – yet still fails to provide any supporting data or analysis. For example, on p. 34 EPA stated without qualification: "EPA will not further analyze releases to hazardous waste landfills because these types of landfill mitigate exposure to the wastes."

Additionally, in its zeal to rely on RCRA to exclude all disposal-related exposure pathways, EPA glossed over important distinctions in the problem formulation in how hazardous wastes are identified under RCRA. In its general introductory discussion of applicable regulations, EPA states:

> Some industrial and commercial users use 1-BP as a general degreaser because chlorinated solvents are listed hazardous wastes under RCRA, whereas 1-BP is not, and therefore waste containing 1-BP may not be hazardous depending on the characteristics of the overall waste stream (Problem Formulation, p. 20).

Later, however, when seeking to justify its exclusions, EPA gets rather more definitive:

> Solid wastes containing 1-BP may be regulated as a hazardous waste under the RCRA waste code D001 (ignitable liquids, 40 CFR 261.21) (Problem Formulation, p. 32).

And still later it gets even more definitive (and more inaccurate):

> 1-BP is regulated as a hazardous waste, waste code D001 (ignitable liquids, 40CFR 261.21) (Problem Formulation, p. 54).

Finally, buried in an appendix, EPA acknowledges:

> Currently, 1-BP is not regulated under federal regulations as a hazardous waste (Problem Formulation, p. 92).

[31] U.S. EPA, Office of Inspector General, *EPA Must Improve Oversight of State Enforcement* (Dec. 2011), https://www.epa.gov/sites/production/files/2015-10/documents/20111209-12-p-0113.pdf.

1-BP is not in fact listed as a hazardous waste under RCRA and would only be identified as one if it was disposed of in high enough concentrations to meet the characteristic of "ignitability."[32] Yet EPA has repeatedly and inaccurately invoked disposal of 1-BP as subject to RCRA hazardous waste regulations.

Additionally, 1-BP is not listed as a Hazardous Air Pollutant (HAP) under the Clean Air Act; EPA was petitioned to list it *ten years ago* and EPA staff under the last Administration preliminarily recommended that the petition be granted. In the draft risk evaluation EPA says that a final decision is expected from the air office by the end of this year; however, that decision date is not mandated and whether the decision will be to grant or deny the petition is not known at this point. In EPA's semi-annual regulatory agenda of federal agency action, EPA has described finalizing the HAP listing of 1-BP as a "long-term action" with no deadline, which calls further into question EPA's intention.[33]

The exposures EPA is ignoring are far from trivial. Based on the most recent data from EPA's Toxics Release Inventory (TRI), despite any regulations under other laws, facilities reported in 2018 releasing nearly 1,000,000 pounds of 1-BP to the air and land. EPA's approach effectively reduces this quantity to zero.

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

EPA lumps together a highly diverse set of industrial/commercial uses that encompass a huge array of sectors, from hobby materials to construction materials to laboratory chemicals, and very different functional uses, from solvent to adhesive to functional fluid (pp. 278-79).

For EPA's dermal exposure estimates, EPA collapsed 12 use subcategories into a single bin: "Bin 5" or "Aerosol Spray Degreaser/Cleaner, Other Aerosol and Non-aerosol Uses" (p. 234). EPA has proposed a single determination of no unreasonable risk for all of these uses, even though this bin covers an expanse of uses without any rationale provided by EPA in its description for collapsing them into one:

> Bin 5 covers aerosol uses, where workers are likely to have direct dermal contact with film applied to substrate and incidental deposition of aerosol to skin. This bin

[32] *See* DEFINING HAZARDOUS WASTE: LISTED, CHARACTERISTIC AND MIXED RADIOLOGICAL WASTES, https://www.epa.gov/hw/defining-hazardous-waste-listed-characteristic-and-mixed-radiological-wastes (last visited Sept. 25, 2019).
[33] U.S. OMB, *Petition to Add n-Propyl Bromide to the List of Hazardous Air Pollutants*, https://www.reginfo.gov/public/do/eAgendaViewRule?pubId=201904&RIN=2060-AS26 (last visited Sept. 25, 2019).

also covers miscellaneous non-aerosol applications that are typically niche uses of 1-BP (p. 107).

EPA was even less transparent in its analysis of non-cancer risk from inhalation exposures. For all of the other industrial/commercial conditions of use, EPA has provided tables of its inhalation assessments based on both modeling and monitoring data (pp. 207-225). EPA does not include this broad category of uses in any of these tables. Yet in EPA's risk determination table, a section on these uses states that EPA expects "[d]evelopmental adverse effects resulting from acute and chronic *inhalation exposures*. Cancer resulting from chronic inhalation exposures" (p. 278, emphasis added). The same margins of exposure (MOE) that EPA states apply to these uses can only be found in the "aerosol spray degreaser/cleaner" table, a separate condition of use (p. 218). EPA briefly states in the risk determination section that "[f]or conditions of use where 1-BP is used in an aerosol application, the exposure levels may be as high as those presented for aerosol spray degreaser and cleaner. Actual exposure levels for each condition of use will likely vary depending on the use volume, engineering control, and PPE" (p. 279). EPA provides no additional explanation, but based on this statement and the MOEs, it appears that EPA did no separate inhalation analysis for this broad category of uses and instead relied on the analysis for aerosol spray degreaser. EPA needs to provide far greater transparency as to what analysis and assumptions it applied to this broad category of uses.

In addition to the lack of transparency, 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 poses 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. EPA has failed to include use of 1-BP in spray foam insulation and as a flame retardant as conditions of use.

EPA has failed to address two conditions of use of 1-BP, use in spray foam blowing and as a flame retardant, both of which were identified to EPA as conditions of use early on in the development process for the risk evaluation.[34] EPA has not provided any rationale for excluding these conditions of use. Notably, multiple SACC members discussed the likelihood of 1-BP's presence in spray foam insulation.

[34] Comment by Enviro Tech International, Inc. on the Scoping Efforts for 1-BP (Sept. 2017), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0741-0054.

3. EPA must adopt a linear, no-threshold approach for 1-BP's carcinogenicity.

A. There is strong support for 1-BP's cancer classification.

EPA quite tentatively states (p. 163): "Evidence from chronic cancer bioassays in rats and mice *suggests* that 1-BP *may pose a carcinogenic hazard to humans*," citing IARC, 2018 for support. IARC has classified 1-BP as "possibly carcinogenic to humans (Group 2B)."[35] However, other authoritative bodies not cited by EPA here, including EPA itself, have drawn more definitive conclusions:

- The National Toxicology Program's (NTP) *Report on Carcinogens* concluded in 2013 that 1-BP is "reasonably anticipated to be a human carcinogen."[36]
- The Agency for Toxic Substances and Disease Registry (ATSDR) confirmed this classification in its 2017 profile:

> The potential carcinogenicity of 1-bromopropane has been examined in bioassays in rats and mice (Morgan et al. 2011; NTP 2011). In both bioassays, animals were exposed 6 hours/day, 5 days/week for up to 105 weeks. Rats were exposed to 0, 125, 250, or 500 ppm 1-bromopropane vapors, while mice were exposed to 0, 62.5, 125, 250, or 500 ppm 1-bromopropane vapors. 1-Bromopropane was a multisite carcinogen in rats, significantly increasing the incidence of large intestine adenomas in females (500 ppm), skin keratoacanthoma in males (≥250 ppm), skin keratoacanthoma, basal cell adenoma, or squamous cell carcinoma in males (≥125 ppm), malignant mesothelioma in males (500 ppm), and pancreatic islet adenoma in males (≥125 ppm). In mice, exposure to 1-bromopropane significantly increased the incidence of combined alveolar/bronchiolar adenoma or carcinoma in females (≥62.5 ppm).[37]

- EPA's own 2016 draft risk assessment for 1-BP, based on a weight-of-evidence analysis, concluded:

> Following EPA's Guidelines for Carcinogen Risk Assessment, *overall, the totality of the available data/information and the weight of evidence*

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[35] https://monographs.iarc.fr/wp-content/uploads/2018/06/mono115.pdf, p. 67.
[36] Natl'l Toxicology Program, *Report on carcinogens Monograph for 1-bromopropane* at 49 (Sept. 2013), https://ntp.niehs.nih.gov/ntp/roc/thirteenth/monographs_final/1bromopropane_508.pdf.
[37] Agency for Toxic Substances & Disease Registry, *Toxicological profile for 1-bromopropane* at pp. 77-8 (Aug. 2017), https://www.atsdr.cdc.gov/ToxProfiles/tp209.pdf.

> support a justifiable basis to conclude a probable mutagenic mode of action for 1-BP carcinogenesis. 1-BP may be considered to be "*Likely to be Carcinogenic in Human* [sic]".[38]

In its draft risk evaluation, EPA also states (p. 163): "The exact mechanism/mode of action of 1-BP carcinogenesis is not clearly understood." However, for many chemicals the biological processes underlying observed effects are often not well understood but that serves as no basis to reject the observed effects. This is the case even for pharmaceuticals available on the market today. The National Research Council wrote in its 2014 report, *Review of EPA's Integrated Risk Information System (IRIS) Process*, that "if FDA were required to organize drug safety around mechanism, it would be nearly impossible to regulate many important drugs because the mechanism is often not understood, even for drugs that have been studied extensively."[39] Indeed, an earlier 2010 *Nature Medicine* editorial noted:

> It is true that we use many highly prescribed drugs without a clear idea of how they work—which targets they hit, what processes they alter and which of these actions are required for therapeutic efficacy. For instance, lithium, used to treat bipolar disorder, modulates many molecular targets, but which—or how many—of these are required for its beneficial effects is uncertain.[40]

Therefore, regardless of the postulated mechanism, it is essential that EPA describe the cancer classification of 1-BP as more definitive than as currently stated in the draft risk evaluation. EPA has a strong foundation in scientific evidence and guidance to retain and move forward with the conclusion it reached in 2016, that 1-BP is "likely to be carcinogenic in humans."

B. There is significant evidence for a mutagenic/genotoxic MOA for 1-BP.

EPA cites significant information that supports a mutagenic/genotoxic MOA for 1-BP. Specifically, on pp. 158-159, pp. 387-399, and pp. 404-406 of the draft risk evaluation, EPA reviews relevant evidence from studies employing the Ames test; mammalian cell and tissue studies; and structure activity relationship evaluations. Of particular importance in evaluating the *in vivo* data, as noted by a member of the SAAC during the September public meeting, is that

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[38] U.S. EPA, *TSCA work plan chemical risk assessment: Peer review draft 1-bromopropane: (n-Propyl bromide) Spray Adhesives, Dry Cleaning, and Degreasing Uses CASRN: 106-94-5* at 95 (2016), https://www.epa.gov/sites/production/files/2016-03/documents/1-bp_report_and_appendices_final.pdf (first emphasis added).

[39] Nat'l Research Council, *Review of EPA's Integrated Risk Information System (IRIS) Process* at chp. 6, p. 90 (2014), https://www.ncbi.nlm.nih.gov/books/NBK230065/.

[40] Editorial, *Mechanism Matters*, 16:4 Nature Med. 347 (Apr. 2010), https://www.nature.com/articles/nm0410-347.pdf.

the observed tumors develop at the site of exposure (ex: lungs, large intestines, and skin), which is consistent with the behavior of direct-acting agents. Therefore, we strongly support the agency's decision to adhere to the *EPA Guidelines for Carcinogen Risk Assessment* and use the default approach of linear non-threshold extrapolation in the cancer risk modeling for 1-BP.

In addition to these data reviewed by the agency, a new study provides additional support for 1-BP's potential mutagenic/genotoxic activity. Nepal et al. (2019) document dose- and time-dependent formation of DNA adducts (specifically, N^7-propyl guanine adducts) in response to 1-BP treatment in male Sprague-Dawley rats.[41] The authors suggest, therefore, that "this chemical may be acting as a direct alkylating agent" leading to mutations (i.e., in lay terms, that 1-BP interacts directly with DNA and chemically attaches an alkyl group to it, yielding a mutation.[42]

Not only does the evidence indicate that 1-BP itself is likely to be mutagenic/genotoxic, but there is also substantial evidence to indicate that metabolic activation of 1-BP leads to production of mutagenic intermediates. These data are reviewed by EPA on p. 400 (among other locations) of the draft risk evaluation. The agency notes that:

> [m]etabolic pathways by which propylene oxide may be generated from 1-BP are shown in Jones and Walsh (1979), NTP (2013b), and IARC (2018), and a pathway by which glycidol may be generated from 1-BP is shown in IARC (2018). Epoxide intermediates such as propylene oxide and glycidol are expected to have more mutagenic activity than 1-BP (IARC, 2018, 2000, 1994).

C. A recent paper cited by the American Chemistry Council (ACC) does not support a non-mutagenic threshold MOA for cancer.

Comments ACC provided to EPA for consideration by the SACC[43] cited a study (Stelljes et al., 2019[44]) that asserts 1-BP acts through a non-mutagenic/non-genotoxic MOA. We have identified a number of concerns with this study that indicate its utility in understanding the MOA of 1-BP may be limited and/or its results misleading. Many of these concerns are methodological and overlap with those raised by EPA on pp. 401-403 regarding prior similar

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[41] Mahesh Raj Nepal, et al., *Identification of DNA and glutathione adducts in male Sprague–Dawley rats exposed to 1-bromopropane*, 82:8 J. OF TOXICOLOGY & ENVTL. HEALTH 1-12 (2019, https://www.ncbi.nlm.nih.gov/pubmed/31140386.

[42] *Id.*

[43] Comment by ACC, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0235-0025.

[44] Mark Stelljes, et al., *28-Day somatic gene mutation study of 1-bromopropane in female Big Blue B6C3F1 mice via whole-body inhalation: Support for a carcinogenic threshold*, 104 REGUL. TOXICOL. PHARMACOL. 1-7 (2019), https://www.ncbi.nlm.nih.gov/pubmed/30779931.

reports – Young (2016) and Weinberg (2016) – also funded by solvent manufacturers but which EPA has not made publicly available.[45] Specifically, we note that:

- The OECD test guidelines for Transgenic Rodent Somatic and Germ Cell Mutation Assays (Test No. 488) specify that because "mutations accumulate with each treatment, a repeated-dose regimen is necessary, with *daily* treatments for a period of 28 days."[46] Stelljes et al. (2019) administered the test chemical for only 5 days/week for 28 days. This study's failure to adhere to fundamental OECD guidelines regarding dosing procedures calls its results into question.

- Stelljes et al. (2019) evaluated effects only in female mice. As noted by the agency on p. 401 of the draft risk evaluation:

> it is possible that males may also be sensitive to mutagenicity/ carcinogenicity from exposure to 1-BP. Indeed, the NTP (2011b) 2-year inhalation study found statistically significant increases in tumor incidence not only in female B6C3F1 mice but also in both male and female F344/N rats.

  Furthermore, OECD test guidelines state that: "Male animals should normally be used. There may be cases where testing females alone would be justified; for example, when testing human female-specific drugs, or when investigating female-specific metabolism."[47] 1-BP does not match either of these example exceptions, and therefore it is inappropriate to have excluded male animals.

- Stelljes et al. (2019) asserted that the maximum tolerated dose (MTD) was 250 ppm. But the authors indicated that at that dose "[t]here were no differences in body weights, food intake, or organ weights in any of the study groups."[48] As EPA noted in

[45] Based on overlapping authorship, sponsorship, and similarities in methods, there is reason to believe that these three publications may in fact derive from the same or closely related studies.
[46] OECD Guidelines for the Testing of Chemicals: Test No. 488: Transgenic Rodent Somatic and Germ Cell Gene Mutation Assays p. 1 (2013), https://read.oecd-ilibrary.org/environment/test-no-488-transgenic-rodent-somatic-and-germ-cell-gene-mutation-assays_9789264203907-en#page1 (emphasis added).
[47] OECD Guidelines for the Testing of Chemicals: Test No. 488: Transgenic Rodent Somatic and Germ Cell Gene Mutation Assays p. 5 (2013), https://read.oecd-ilibrary.org/environment/test-no-488-transgenic-rodent-somatic-and-germ-cell-gene-mutation-assays_9789264203907-en#page5.
[48] Mark Stelljes, et al., *28-Day somatic gene mutation study of 1-bromopropane in female Big Blue B6C3F1 mice via whole-body inhalation: Support for a carcinogenic threshold*, 104 REGUL. TOXICOL. PHARMACOL. 3 (2019), https://www.ncbi.nlm.nih.gov/pubmed/30779931.

describing concerns about Young et al. (2016) on p. 401, because there were "no treatment-related effects on survival, clinical observations, body weight, food consumption, or organ weights, the highest 1-BP test concentration was not an MTD and the study needs to be repeated to include a top concentration at the MTD."

- Stelljes et al. (2019) evaluated mutagenicity only in lung, liver, and colon tissue, as well as germ cells. Again, as noted by EPA on p. 402 of the draft risk evaluation:

> [t]he NTP (2011b) 2-year study for 1-BP included neoplasm findings for skin, large intestine, lung, and pancreas. Before concluding that a mutagenic mode of action of 1-BP is not operable for all target sites, additional target sites, including skin, pancreas, and intestines at a minimum, would need to be assessed for 1-BP.

- Stelljes et al. (2019) used a sampling time of 3 days for lung, liver, and colon tissues. However, OECD states that:

> The sampling time is a critical variable because it is determined by the period needed for mutations to be fixed. This period is tissue-specific and appears to be related to the turnover time of the cell population, with bone marrow and intestine being rapid responders and the liver being much slower. … If slowly proliferating tissues are of particular importance, then a later sampling time of 28 days following the 28 day administration period may be more appropriate (16) (29).[49]

Given this study's focus on the liver, a slowly proliferating tissue, a longer sampling period is indeed warranted.

Additionally, we echo EPA's overarching concerns about the Big Blue ® assay (pp. 402-403), of which Stelljes et al. (2019) is another example. EPA states that:

> the Big Blue ® assay lacks a body of data on mutagenic and carcinogenic chemicals with structural similar to 1-BP… To enhance confidence that the methods used for 1-BP testing in the Big Blue ® assays are sufficient to prevent false negative mutagenicity findings, mutagenicity data from Big Blue ® assays of rats and mice are needed from independent testing of bromoethane (and other known mutagenic carcinogens with structural similarity to 1-BP) or these 1-BP

[49] OECD Guidelines for the Testing of Chemicals: Test No. 488: Transgenic Rodent Somatic and Germ Cell Gene Mutation Assays p. 7 (2013), https://read.oecd-ilibrary.org/environment/test-no-488-transgenic-rodent-somatic-and-germ-cell-gene-mutation-assays_9789264203907-en#page7.

> analogs could be included as potentially-positive controls in additional Big Blue® studies of 1-BP. If mutagenic and carcinogenic structural analogs of 1-BP are not mutagenic in Big Blue® rodent assays, it can be concluded that these assays are not suitable for assessing the mutagenicity of 1-BP.

Finally, it should be noted that Stelljes et al. (2019) was entirely funded by EnviroTech International, which describes itself as follows:

> Enviro Tech International is a top supplier of stabilized n-propyl bromide [or nPB, a synonym for 1-bromopropane] and fluorinated solvents for industrial parts cleaning applications. Since creating the nPB market in 1994, we've grown our small company from the ground up to become a formidable and trusted force in the industry.[50]

In fact, Enviro Tech has been the industry-leading provider of solvents, including 1-BP, for over twenty years.[51]

D. EPA should reject Albemarle's argument that mouse lung tumors are not relevant to humans.

One public commenter, Albemarle,[52] has argued that mouse lung tumors, which form the basis for the IUR and dermal slope factor in this draft risk evaluation, are of limited relevance to humans given purported differential expression of metabolizing enzymes in the mouse lung. As support, Albemarle cited a review article (Smith et al., 2018[53]) the lead author of which is employed by Albemarle.

However, in the US EPA's 2014 State-of-the-Science Workshop on Chemically-Induced Mouse Lung Tumors: Applications of Human Health Assessments Summary Report, Dr. Ron Melnick's presentation is described as providing a number of points of rebuttal to this argument. He first noted:

> In the absence of convincing data to the contrary, the International Agency for Research on Cancer (IARC), the US National Toxicology Program (NTP), and

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[50] ENVIRO TECH COMPANY, https://www.envirotechint.com/company/ (last visited Oct. 9, 2019).
[51] *Id.*
[52] Comment by Albemarle, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0235-0029.
[53] Carr J. Smith, et al., *Bronchioloalveolar lung tumors induced in "mice only" by non-genotoxic chemicals are not useful for quantitative assessment of pulmonary adenocarcinoma risk in humans*, 2 TOXICOLOGY RESEARCH & APPLICATION 1-24. (2018), https://journals.sagepub.com/doi/full/10.1177/2397847318816617.

> the US EPA consider animal tumor findings relevant to evaluations of human risk. Countering this basic public health perspective requires sufficient and valid evidence for a species-specific cancer response.[54]

According to the summary report, Dr. Melnick goes on to describe key criteria that must be met before deviating from this default assumption – explained below for CYP2F2 but equally relevant in the context of CYP2E1, the primary metabolizing enzyme for 1-BP:

> To establish the CYP2F2 MOA for each particular chemical, at least three fundamental issues need to be thoroughly addressed: 1) demonstration that CYP2F2-mediated metabolites are the determinants of the mouse lung tumor response, 2) demonstration that these reactive metabolites are produced by CYP2F2 only in the mouse lung and not systemically distributed (or are not distributed from other tissues in sufficient quantity to cause cytotoxicity), and 3) demonstration that the relationship between hypothesized essential precursor events (cytotoxicity and sustained regenerative hyperplasia) in the mouse lung and the tumor response in that organ is consistent (i.e., that tumors do not occur at exposure levels for which cytotoxicity does not occur), since genotoxicity produced by non-CYP2F2-mediated metabolites could be carcinogenic for some CYP2F2 substrates and not for others.[55]

In the context of 1-BP and CYP2E1, these criteria have not been met with sufficient confidence to warrant deviation from the default assumption that observed animal tumors are relevant to humans. For example, with regard to the first criterion, CYP2E1-mediated metabolites are *not* the sole determinants of lung tumor response. The argument discounting the human relevance of mouse lung tumors hinges on the theory that these tumors arise only in response to elevated quantities of toxic metabolites produced in mouse lung Clara cells compared to humans. However, as noted by EPA in the draft risk evaluation, "data suggest that 1-BP may be a direct-acting mutagen since similar responses were observed both with and without metabolic activation" (p. 387). As such, the relative difference in the quantities of metabolic enzymes between mice and humans and their contributions to carcinogenicity are not the sole considerations in understanding tumor formation; EPA must also consider the potential for 1-BP to be a direct-acting mutagen, which is not a species-specific event nor related to CYP2E1 levels.

Even if EPA were to focus only on the pathway to carcinogenicity via toxic metabolites, it would still be inappropriate to discount human lung tumors due to the relative increased enzyme

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[54] U.S. EPA, *Summary Report: State-of-the-Science Workshop on Chemically-Induced Mouse Lung Tumors: Applications to Human Health Assessments* p. 37 (Dec. 2014), https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=291094&CFID=67867665&CFTOKEN=37343828.

[55] *Id.*

expression in mouse Clara cells compared to those in average humans. Throughout the draft risk evaluation, EPA acknowledges that there are several factors and/conditions that could up-regulate CYP2E1 in individuals across the human population, such as alcoholism and diabetes. These individuals with increased CYP2E1 expression would have increased production of toxic metabolites, suggesting that a mouse model with increased production of toxic metabolites would still be relevant to consider in order to meet TSCA's mandate that EPA evaluate the risks of chemicals to potentially exposed or susceptible subpopulations. *See* 15 U.S.C. § 2605(b)(4)(A). Furthermore, with regard to Dr. Melnick's second criterion, it is unlikely that these CYP2E1-related toxic metabolites would be present only in the lungs, given that this enzyme is expressed in other organs such as the liver.[56]

The third criterion noted by Dr. Melnick relates to the proposed MOA of the chemical in question. The assertion that mouse lung tumors are formed through a cytotoxic rather than genotoxic pathway is a central line of argument put forth in the Smith et al. (2018) publication authored by Albemarle.[57] Under this line of reasoning, the observed pulmonary tumors are due to Clara cell metabolism-derived cytotoxic metabolites that stimulate cellular proliferation leading to cancer. However, as described throughout the draft risk evaluation and highlighted in Part I, sec. 3.B. above, there is substantial evidence to indicate that 1-BP acts through a genotoxic/mutagenic MOA. As such, Dr. Melnick's final criterion is also not met and arguments against the relevance of mouse lung tumors to humans, which rely on this requirement, can be dismissed.

Finally, Smith et al. (2018), which suggests that mouse lung tumors are not relevant to humans, provides only a general rather than a chemical-specific discussion. These non-specific arguments cannot be automatically assumed to apply to 1-BP. In fact, EPA has previously stated that there is no overarching, cross-chemical consensus on this issue. Specifically, in the January 2017 Federal Register notice that EPA published for public comment regarding petitions to list 1-BP as a HAP,[58] the agency stated (emphasis added):

> Albemarle disputed the use of the alveolar/bronchiolar adenomas in the cancer assessment, suggesting a lack of human relevance of these mouse tumors. While

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[56] *See* CYP2E1 CYTOCHROME P450 FAMILY 2 SUBFAMILY E MEMBER 1 [HOMO SAPIENS (HUMAN)], https://www.ncbi.nlm.nih.gov/gene/1571 (last visited Oct. 9, 2019); CYP2E1, https://www.proteinatlas.org/ENSG00000130649-CYP2E1/tissue (last visited Oct. 9, 2019).

[57] Carr J. Smith, et al., *Bronchioloalveolar lung tumors induced in "mice only" by non-genotoxic chemicals are not useful for quantitative assessment of pulmonary adenocarcinoma risk in humans*, 2 TOXICOLOGY RESEARCH & APPLICATION 1-24. (2018), https://journals.sagepub.com/doi/full/10.1177/2397847318816617.

[58] 82 Fed. Reg. 2,354, 2,360 (Jan. 9, 2017), https://www.regulations.gov/document?D=EPA-HQ-OAR-2014-0471-0062.

this topic has been debated in the scientific literature and was the topic of a technical workshop convened by the EPA (U.S. EPA, 2014), *there is no cross-chemical consensus on the human relevance of mouse lung tumors; each chemical will need to be judged separately regarding relevance*. Furthermore, the NTP conclusions, supported by the EPA, do not rely solely on the lung tumor data, but rather on the totality of the available information. The commenter also claimed that the EPA has not considered potential uncertainties in the mutagenicity, genotoxicity, and carcinogenicity data for nPB. The NTP review, however, assessed available mutagenicity data in its review. This took into account reports of mutations in bacterial and mammalian cells and limited data on DNA damage in nPB-exposed workers. Furthermore, it is noted that metabolic pathways are similar in humans and experimental animals, and several metabolites of nPB have been identified as mutagens and are known to cause DNA damage. Results from some of these in vitro assays are mixed, and confounding factors may include the volatility of nPB or active metabolites. Finally, the commenter provided a summary of an unpublished study they commissioned showing negative results in the Ames assay; however, the EPA is not persuaded, and these results do not change the conclusion regarding the mutagenicity of nPB and its metabolites.

In sum, there is a strong scientific basis for EPA to maintain the use of mouse lung tumors as the basis for the IUR and dermal slope factors for 1-BP.

E. The scientifically sound and health-protective approach is to use linear extrapolation in cancer dose-response modeling for 1-BP.

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. Even were the evidence deemed insufficient to identify with certainty a genotoxic MOA, there is longstanding EPA policy guidance and precedent supporting a default to a no-threshold, linear extrapolation method for cancer dose-response modeling.

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.[59]

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

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

EPA must employ health-protective approaches to dose-response modeling, as described at length in the National Research Council's report, *Science and Decisions: Advancing Risk Assessment.* In this report, the NRC committee specifically provides important perspective on the need to conduct a linear extrapolation at the population level, even where a threshold might theoretically exist. The authors state, for example, that: [60]

- "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 report concluded that "***cancer and noncancer responses [to chemical exposures] be assumed to be linear as a default****."[61] The NRC committee called for a unified approach using linear extrapolation to account for both background exposures and the wide range of variability in individual susceptibility. It argued that this approach also improves the

[59] 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.
[60] 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.
[61] *Id.* at chp. 5, p. 180.

risk characterization and ultimately risk management decisions by providing quantification of excess population risk rather than a margin of exposure.

In their *State-of-the-science workshop report: issues and approaches in low-dose-response extrapolation for environmental health risk assessment*, White et al. (2009)[62] 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.

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

In summary, given 1) existing agency guidance, 2) the many sources of variability in the human population, 3) TSCA's mandate to protect "potentially exposed or susceptible subpopulations," and 4) the clear presence of individuals with preexisting health conditions, metabolic or genetic variability, or other factors that make them more susceptible to 1-bromopropane exposure (see, for example, pp. 150-151, 181, 192), 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).

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

More broadly, EPA must employ health-protective approaches to dose-response modeling, as described at length in the National Academy of Sciences (NAS) report, *Science and Decisions: Advancing Risk Assessment*.[63] Among other recommendations, the NAS argued that "***cancer and noncancer responses [to chemical exposures] be assumed to be linear as a default ***."[64]

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[62] 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).

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

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

The MOE approach presented in the 1-BP risk evaluation provides a bright-line, yes/no approach to risk and fails to provide a measure of population risk at a given exposure level. This approach limits the assessment's utility for risk managers, particularly when comparing options for substitution and conducting risk-benefit comparisons.

The National Academy of Sciences (NAS) Committee on Improving Risk Analysis Approaches Used by the U.S. EPA concluded "separation of cancer and noncancer outcomes in dose response analysis is artificial because noncancer endpoints can occur without a threshold or low dose nonlinearity at the population level."[65] The Committee further stated that background exposures and underlying disease processes can contribute to background risk and lead to linearity at population doses of concern. *See* Part I, sec. 3.E.ii.

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

4. Key data gaps

A. Environment

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

EPA has relied solely on acute aquatic toxicity data, therefore EPA's draft risk evaluation has cannot establish that 1-BP presents no acute aquatic hazard or risk, chronic aquatic hazard or risk, or no environmental hazard or risk, generally. EPA decisions under TSCA must be supported by substantial evidence, 15 U.S.C. § 2618(c)(1)(B)(II), and here EPA does not have sufficient evidence to support these conclusions.

ii. Dearth of ecotoxicity data and lack of environmental monitoring data

EPA has evaluated the potential risk to the environment and ecological receptors based on few or no toxicity data, as discussed previously in these comments (*see* Part I, sec. 1.C.i.c). There was only a single acute toxicity study that EPA had access to and reviewed (as well as a handful of industry summaries in unevaluated ECHA dossiers), and no toxicity data for terrestrial, sediment, or avian organisms.

Furthermore, EPA has determined there is no risk from these exposure pathways, despite having no environmental monitoring data for confirmation (see p. 34 of the problem formulation, noting that "[e]nvironmental monitoring data were not identified in the 2016 Draft Risk Assessment"),

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

instead relying on qualitative and screening level assessments. These deficiencies are discussed more below and in Part I, sec. 5.A.

a. Aquatic pathways

Notably, EPA's derivation of its chronic COC is based on no actual chronic toxicity data. EPA states:

> Since there are *no long-term chronic studies for 1-BP*, the fish *** ChV is then divided by an AF (10) to obtain a chronic COC. (p. 138)

EPA should have identified this as a data gap and taken steps to address it. EPA provides no justification for its application of an "acute-to-chronic ratio" or its specific value of 10, nor does it provide even a citation to the use of such values in other contexts (*see* Part I, sec. 1.C.i.c).

b. Terrestrial pathways

EPA states:

> [A]lthough terrestrial exposure pathways are included in the scope of the problem formulation, no further analyses of hazards to terrestrial receptors is necessary as a result of a consideration of the conditions of use, and physical chemical properties of 1-BP *** [N]o further analysis of hazards to aquatic or terrestrial receptors was carried out as part of this evaluation under the conditions of use of this assessment. (p. 138)

As described above, Part I, sec. 1.C.i.c., this wholesale dismissal of risk to terrestrial receptors based on presumed lack of exposure—in turn based solely on physical-chemical properties and assumptions about exposures from conditions of use and not on environmental monitoring data—ignores TRI disposal data, uses over-simplified reasoning to describe fate and transport, and is based in unsubstantiated data. Rather, it appears to be a pretext for EPA to ignore the fact that it lacks toxicity data, as was noted in the problem formulation:

> During data screening, there were no available sediment, soil, nor avian toxicity studies found in the scientific literature for 1-BP. The toxicity of 1-BP is expected to be low *based on the lack of on-topic environmental hazard data for 1-BP to sediment and terrestrial organisms in the published literature* and the physical/chemical/fate properties (relatively high volatility (Henry's Law constant of $7.3X10^{-3}$ atm-m^3/mole), high water solubility (2.4 g/L), and low log Koc (1.6) suggesting that 1-BP will only be present at low concentrations in these environmental compartments. (Problem Formulation, p. 41) (emphasis added)

Bizarrely, in the draft risk evaluation EPA states:

> During problem formulation, EPA made refinements to the conceptual models *resulting in the elimination of the terrestrial exposure pathway from further analysis*. Thus, environmental hazard data sources on terrestrial organisms were *considered out of scope and excluded from data quality evaluation*. (p. 47, note to Figure 1-9; emphases added)

Furthermore, all mention of avian toxicity studies was dropped from the draft risk evaluation. EPA acknowledges that "[b]ased on [the] estimated half-life in air, long range transport via the atmosphere is possible" (p. 51). Given the habitat of birds, 1-BP's residence time in air, and potential for long-range transport , these organisms seem at risk of exposure. It is irresponsible of the agency to ignore these and other ecological receptors.

Most bizarrely, these data gaps and exclusions did not prevent EPA from definitively asserting, for each and every condition of use of 1-BP, that the chemical does not present an unreasonable risk to terrestrial organisms (pp. 260-288).

c. Sediment exposure pathways

EPA states:

> 1-BP is expected to be present at low concentrations in*** the sediment compartment of aquatic ecosystems so no further analysis of hazards to these environmental receptors is necessary. (p. 141)

EPA additionally asserts:

> 1-BP in sediment is expected to be in the pore water rather than adsorbed to the sediment solids based on a high water solubility (2.4 g/L) and low log Koc (1.6). (p. 188)

It is important to note that sediment-dwelling organisms live in or are in contact with the pore water of sediment systems, and therefore, this can be a key route of exposure.[66] Furthermore, higher concentrations of certain contaminants of concern in pore water can increase bioavailability to benthic organisms—meaning, the higher the concentration of the contaminant in the pore water, the more likely it is to cause toxicological effects to act.[67] Therefore, EPA cannot ignore or fail to analyze exposure of sediment-dwelling organisms.

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[66] *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.
[67] 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.

iii. Assessment factors do not lead to conservative calculations; in fact, assessment factors account for real-world sources of variability as well as database limitations.

EPA contends that it "utilized a conservative screening level approach to calculate risk to the aquatic organisms." (p. 141). The agency asserts that its assumption that wastewater treatment removal is 0% is conservative. However, this is not the case. EPA itself notes that "reported releases likely already account for wastewater treatment, which means any removal has already been accounted for." (Problem Formulation, p. 35, reiterated in draft risk evaluation, p. 187)

Moreover, the agency also asserts that its concentrations of concern (COCs) for aquatic effects are "conservative."

Discussing its acute and chronic COC:

> [H]azard thresholds, known as Concentrations of Concern (COCs) were calculated to provide a *conservative* estimate for a screening level comparison with estimated surface water concentrations to identify potential concerns to aquatic species. (p. 141, emphasis added)

EPA implies that its calculations of COCs are conservative at least in part because of its use of assessments factors. 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. 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. 328)

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.[68]

In evaluating risks, EPA should recognize that AFs ensure greater accuracy and do not provide a safety factor rendering the evaluation "conservative."

B. Human health

i. Dearth of product/use concentration data

For consumer uses, EPA relied on results from the Westat Survey for a number of model parameters, including the duration of use, the mass of the product, and the room of use (p. 117). EPA rated this source as high quality by EPA in its systematic review. However, EPA directed the survey more than 30 years ago, in 1987. As noted by SACC members, consumer use patterns have changed significantly since 1987. For instance, a survey from 1987 would not accurately reflect the amount of time spent using consumer electronics today. There are also significantly more do-it-yourself (DIY) consumers today, with innumerable products specifically produced for and marketed to them. The widespread availability of products online is another major shift that could also increase overall consumer exposure as compared to 1987. EPA noted many of these uncertainties, yet nevertheless determined that the survey was still representative of current consumer uses, and relied on it, declaring it to be of "medium uncertainty" without specifying how it reached that ranking (p. 133).

EPA must conduct a new survey to determine consumer use patterns for 1-BP, as well as other chemicals for which it is or will be conducting risk evaluations. In addition to satisfying the data needs for the first ten chemical substances, EPA will also have a constant need for robust consumer use data in its future risk evaluations under section 6. In order to conduct risk evaluations based on the best available science, EPA needs much more recent data on consumer uses.

ii. EPA has failed to consider the continuous nature of off-gassing from consumer products containing 1-BP.

SACC members advised EPA that consumers may also be exposed to 1-BP from the off-gassing of stored consumer products. The failure to consider these consumer exposures is especially

[68] 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).

notable in light of EPA's failure to consider chronic exposures from any consumer uses. EPA simply assumed that there are no consumer exposures "considered chronic in nature" (p. 205).

Continuous off-gassing from stored consumer products would result in chronic exposures to 1-BP, however. EPA itself warns that consumers should "[t]hrow away partially full containers of old or unneeded chemicals safely *** [b]ecause gases can leak even from closed containers ***"[69] – even as it proceeds to ignore such exposures.

The failure to analyze indoor, chronic exposures to 1-BP is critical because, according to EPA:

- "Americans, on average, spend approximately 90 percent of their time indoors, where the concentrations of some pollutants are often 2 to 5 times higher than typical outdoor concentrations."
- "People who are often *most susceptible* to the adverse effects of pollution (e.g., the very young, older adults, people with cardiovascular or respiratory disease) tend to spend even more time indoors."
- "Indoor concentrations of some pollutants have increased in recent decades due to such factors as energy-efficient building construction ***."[70]

Additionally, based on EPA's Office of Research and Development's Total Exposure Assessment Methodology (TEAM) Study:

> Concentrations of many VOCs are consistently higher indoors than outdoors. [The] study by the EPA, covering six communities in various parts of the United States, found indoor levels up to ten times higher than those outdoors — even in locations with significant outdoor air pollution sources, such as petrochemical plants.[71]

In the final draft risk evaluation EPA must consider chronic, consumer exposures to 1-BP, including from stored consumer products.

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[69] VOLATILE ORGANIC COMPOUNDS' IMPACT ON INDOOR AIR QUALITY, https://www.epa.gov/indoor-air-quality-iaq/volatile-organic-compounds-impact-indoor-air-quality (last visited Sept. 30, 2019).
[70] INDOOR AIR QUALITY, https://www.epa.gov/report-environment/indoor-air-quality (last visited Sept. 30, 2019) (emphasis added).
[71] American Lung Association, U.S. EPA, U.S. CPSC, & American Medical Association, *Indoor Air Pollution: An Introduction for Health Professionals* p. 13 (2015), https://www.epa.gov/sites/production/files/2015-01/documents/indoor_air_pollution.pdf.

iii. EPA relies on a loading & unloading model to estimate inhalation exposures despite that model failing to account for the relevant process activities.

In the draft risk evaluation, EPA repeatedly uses its *Tank Truck and Railcar Loading and Unloading Release and Inhalation Exposure Model* (pp. 56, 62, 64, 67, 68-69, 102), but for most of the conditions of use modeled this way, EPA admits the model does not accurately reflect the exposures for the conditions of use. Specifically, according to EPA, the model would be appropriate "to estimate worker exposure during container and truck unloading activities that occur at industrial facilities" (p. 56), but "the model does not account for other potential sources of exposure at industrial facilities, such as sampling, equipment cleaning, and *other process activities*" (p. 62) (emphasis added).

Given the reality of exposures from other process activities, why does EPA consider this model accurate and use it to model: (i) processing as a reactant; (ii) processing – incorporation into articles; (iii) repackaging; and (iv) disposal and recycling (pp. 64, 67, 69, 102)? Each of these conditions of use appear to involve activities beyond simply loading and unloading, but the model does not account for the exposures from those activities. It appears that EPA is limiting its modeling to the loading and unloading aspects of these conditions of use and ignoring the rest of the circumstances making up these conditions of use. It is arbitrary and capricious for EPA to use a model that fundamentally does not apply when attempting to model exposures from these conditions of use.

The model appears to contain other flaws as well, some of which are acknowledged by EPA. For example, it assumes that only one container is loaded per day, when the conditions of use being analyzed might well involve more than one loading activity per day (pp. 56, 62, 64, 67, 68-69, 102). For any such case, the model underestimates worker exposure (pp. 62, 64, 67, 68-69, 102). The model also calculates the quantities of released air using dimensions that "may not be representative of the whole range of loading equipment used at industrial facilities handling 1-BP" (p. 244). The model estimates fugitive emissions based on factors where their applicability to 1-BP, and the accuracy of EPA's assumptions, are not known (p. 244). The model assumes the use of a vapor balance system to minimize fugitive emissions, but EPA does not know whether these systems are used by all facilities that handle 1-BP (p. 244).

Given the numerous unknowns and the fact that this model is not designed to model exposures from these conditions of use, EPA cannot justify its statement that it has a medium level of confidence in the assessed exposures (pp. 62, 64, 67, 69, 102). Rather than rely on a model that EPA admits does not accurately reflect these conditions of use, EPA should use its information authorities to obtain actual monitoring information for these conditions of use. Alternatively, EPA should develop models that actually model the relevant conditions of use. But EPA cannot simply accept a readily available model and use it regardless of its applicability.

iv. Dearth of dermal toxicity data

EPA appears to have identified no acute or repeated dose studies that examined toxicity via dermal exposure (pp. 144, 153, 184). As a result it relied on extrapolation from inhalation toxicity studies; we discuss in Part I, sec. 5.B.ix.a of these comments the concerns raised by this approach.

5. Analytic gaps/deficiencies

A. Environment

i. EPA over-relies on models and unevaluated data for physical/chemical properties

a. EPA must evaluate the original data used to generate properties, not just simply cite books.

As described in the problem formulation, "physical-chemical properties influence environmental behavior and the toxic properties of a chemical" (Problem Formulation, p. 17). Consequently, it is important not only to be judicious in sourcing these values, but also to justify reliance on those sources and address any uncertainty associated with them. The values presented in the problem formulation (Table 2-1), which are the same values used in the draft risk evaluation (Table 1-1, p. 28), either were sourced from textbooks or were estimated using EPISuite. The values sourced from textbooks[72] are not original data; therefore, the quality of the studies (or models) and the underlying data must be evaluated before they are used in a risk evaluation.

For example, the water solubility value (which is variously described as being "high" (pp. 23, 140, 186, 188, 246, 249, 258), "moderate" (p. 51), and "low" (p. 337) is sourced from Yalkowsky et al. 2010; however, that textbook in turn references a study conducted in 1917 (Horiba 1917),[73] which, in turn, is actually referencing data from 1906. Yalkowsky et al. noted in their data evaluation that the purity of solute, equilibrium time/agitation, and analysis were all not provided by Horiba, which indicates these data are not reliable. Given the importance EPA has placed on water solubility in determining risk, this value must be scrutinized before being used to dismiss hazard, exposure potential, or risk.

[72] *See* EPA's references at pp. 290-309: Boublík et al. 1984, Hansch 1995, Haynes and Lide 2010, O'Neil 2013, Patty et al. 1963, Yalkowsky et al. 2010.
[73] Shinkichi Horiba, *Studies of solution. I. The change of molecular solution volumes in solutions, in* MEMOIRS OF THE COLLEGE OF SCIENCE, KYOTO IMPERIAL UNIVERSITY, 1-43, (1917), https://babel.hathitrust.org/cgi/pt?id=nyp.33433109947840&view=1up&seq=7.

b. Models used to derive physical-chemical properties lack performance transparency.

Physical-chemical property models in EPISuite lack transparency in performance and applicability. According to the EPA in this draft risk evaluation, "a full discussion of the performance of the individual property estimation methods used in EPISuite is available in the EPI Suite™ help files" (p. 51). However, the property model performance estimations are only presented in terms of overall performance and do not describe whether or not the model is applicable for any specific chemical.[74] For example, the accuracy and domain section in the help file for KOWWIN (the Log Octanol-Water Partition Coefficient Program) describes the domain for the model as having "no universally accepted definition..." and that the user "may wish to consider the possibility" that property estimates are less accurate for compounds with molecular weights higher or lower than those used in the training set. Similar disclaimer statements are found in each program that uses quantitative structure property relationships (QSPR). These examples illustrate that this, and other, EPISuite property models lack transparency as to their appropriateness for application to 1-BP.

A poignant example of an inappropriate application of EPISuite is for the chemical Pigment Violet 29 (PV29), despite its molecular weight being within the values used for the training set. When analyzed in EPISuite, the water solubility of PV29 is given as both 5.85 $x10^{-3}$ mg/L and 0.169 mg/L, as derived by two different property models, with no indication as to which—or if either—model is appropriate.

The newer QSPR model, EPA's OPEn structure-activity/property Relationship App (OPERA), includes the reporting of a chemical-specific applicability domain, and was built using a newer database of physical-chemical parameters.[75] It is unclear why EPA did not use this newer, more transparent model for its estimation of physical-chemical properties. For example, the Henry's Law constant for 1-BP estimated in EPISuite is 7.3 x 10^{-3} versus 2.28 x 10^{-3} (atm-m^3/mole) in OPERA, a difference of nearly three-fold. This means that 1-BP may be somewhat less volatile than EPA assumed in the draft risk evaluation.

[74] *See* Kamel Mansouri, et al., *OPERA models for predicting physicochemical properties and environmental fate endpoints*, 10 J. OF CHEMINFORMATICS (2018), https://doi.org/10.1186/s13321-018-0263-1 ("The classical approach of comparing models by global R^2/Q^2 fitting performance may or may not reflect higher predictive ability, especially when dealing with different sizes of datasets, for example. Therefore, comparisons of model fit should be local and specific, not based on overall statistics.").
[75] *Id*.

ii. EPA's approach and methodology for assessing environmental exposure ignores or over-simplifies fate characteristics and ignores key data.

As discussed above, physical-chemical properties of a chemical help describe its environmental fate characteristics. EPA used EPISuite to predict a number of important environmental fate characteristics, which it then coupled with assumptions about particular conditions of use to justify disregarding pathways of exposure to sediment and terrestrial organisms:

> EPA integrated relevant pathways of environmental exposure with *available hazard data to estimate risk to terrestrial environmental receptors*. A qualitative consideration of the high volatility, high water solubility and low Log Koc of 1-BP, as well as a consideration of the conditions of use of this assessment suggest that 1-BP will only be present at low concentrations in the sediment and terrestrial environmental compartments, so these pathways were not further analyzed in this risk evaluation since risk from these exposure pathways are not expected. (p. 23, emphasis added)

The dismissal of these pathways is problematic, for four reasons. First, EPA has no hazard data for terrestrial organisms. In the Problem Formulation, EPA notes "there were no available sediment, soil, nor avian toxicity studies found in the scientific literature for 1-BP" (Problem Formulation, p. 41).

Second, EPA has not adequately supported the sources, and therefore the values, for the physical-chemical properties it relies on to reach this conclusion, as described above in Part I, sec. 5.A.i. For example, EPA's draft risk evaluation repeatedly invokes 1-BP's "high" water solubility as a primary basis for concluding the chemical poses little hazard, exposure potential, or risk (pp. 23, 140, 186, 188, 246, 249, 258), despite the dubious nature of its source discussed earlier.

Third, partition coefficients assume chemical equilibrium. However exposure to chemicals of concern can occur in high concentrations in different environmental compartments prior to reaching equilibrium. Additionally, when considering an open, multi-media system, a better approximation might be the Level III Fugacity model, which predicts 10% of 1-BP will be distributed to soil, 44.7% to air, 45.2% to water, and the remainder (0.1%) to sediment, as calculated using EPISuite 4.11. A 10% percent distribution to soil cannot be automatically dismissed as *de minimis*.

Finally, in evaluating the various conditions of use, EPA failed to consider air and land releases reported under TRI, as described in more detail in Part I, sec. 5.A.iii.a. below.

iii. EPA over-relies on limited and incomplete data, including from TRI, to exclude or dismiss the significance of numerous exposure pathways.

In its problem formulation, EPA made extensive use of the limited 2016 data on 1-BP reported under the Toxics Release Inventory (TRI). The conclusions drawn by EPA with respect to potential environmental impacts based in these inadequate data were not revisited in the draft risk evaluation; moreover, the agency also ignored additional categories of TRI releases reported for 1-BP in 2017 that had not been reported in 2016, including nearly 8,000 pounds of 1-BP reported as "other land disposal," which is described as "such activities as placement in waste piles and spills or leaks"[76] and approximately 14,500 lbs. to "other off-site management."[77]

It should be noted that 1-BP was only recently added to the TRI and the 2016 data on which EPA so heavily relies were from the first year the chemical was required to be reported. That may help explain why a TRI report for 1-BP was received from only about 40% of facilities (55 of 140 facilities) expected to report the chemical, a fact EPA discusses in the problem formulation, (p. 32) but then largely ignores when citing TRI data as the basis for excluding exposure pathways or asserting low release or exposure to 1-BP. In fact, the gap between reported and actual releases may be even worse than that: EPA's summary of TRI data in Table 2-6 on page 33 of the problem formulation shows that very few facilities (often only one) reported any releases at all to various media or waste management facilities, suggesting that there may be more facilities that did not report.

EPA's decision to make sweeping exclusions of exposure pathways or assume negligible releases and exposures based on TRI data alone is troubling, given EPA's own speculation as to why such a large gap exists between the number of TRI reports it received vs. what was expected:

> The difference in estimated versus actual reporting facilities could be due to several factors such as, 1) facilities could be moving away from using 1-BP; 2) *some facilities may not yet be aware of the reporting requirements since this is the first year of reporting*; 3) *facilities could be below the threshold for reporting*. Facilities are required to report if they manufacture (including import) or process

[76] TRI METADATA OTHER DISPOSAL, https://iaspub.epa.gov/triexplorer/trimetadata.show?p_title=Other+Disposal (last visited Oct. 10, 2019).
[77] TRI METADATA TRANSFER TO DISPOSAL-OTHER OFF-SITE MANAGEMENT, https://iaspub.epa.gov/triexplorer/trimetadata.show?p_title=Transfer+to+Disposal-Other+Off-Site+Management (last visited Oct. 10, 2019) (defined as "[c]hemicals in waste sent to sites where the waste is managed by techniques not specifically listed" elsewhere in TRI).

> more than 25,000 pounds of 1-BP, or if they otherwise use more than 10,000 pounds of 1-BP. (Problem Formulation , pp. 32-3, emphasis added)

Beyond this paragraph, EPA never grapples with the enormous uncertainty and likely unreliability of the TRI data on which it so heavily relies, which is further explored below.

a. Exclusion of exposures from disposal pathways based on inadequate TRI data

The agency acknowledges that disposal can result in environmental releases and lead to exposure pathways, but then dismisses those pathways based on an unsupported assumption that such releases are subject to RCRA subtitle C requirements. EPA states in the draft risk evaluation that "manufacturing, processing, use *and disposal* of 1-BP can result in releases to the environment" (p. 52).

EPA relies heavily on 2016 TRI data to justify its exclusion of disposal pathways from the 1-BP risk evaluation. For example, EPA states:

> Table 2-6 shows TRI reports approximately 58,000 pounds of disposal *to a single RCRA Subtitle C landfill*. EPA will not further analyze releases to hazardous waste landfills because these types of landfill mitigate exposure to the wastes. TRI also reports approximately 90,000 pounds of 1-BP transferred to other off-site landfills [3 in total]. Further review of TRI data indicated that all reported transfers "other off-site landfills" were to facilities permitted to manage RCRA regulated waste. (Problem Formulation, p. 34, emphasis added)

EPA has not provided to the public its "further review of TRI data." Especially given the inadequacy of the TRI data the agency is relying upon, it is certainly plausible that significantly more 1-BP than cited above is not subject to RCRA subtitle C requirements.

In fact, 1-BP disposals reported to land under the 2017 TRI included: 69,952 lb. to off-site *non-hazardous waste landfills*. They also included two additional categories of TRI releases reported for 1-BP in 2017 that had not been reported in 2016: 7,885 lb. to "other" off-site land disposal, and approximately 14,500 lbs. to "other off-site management." None of these can be assumed to be subject to RCRA subtitle C requirements. As discussed above, Part I, sec. 2.B., 1-BP is not listed as a hazardous waste under RCRA.

b. Dismissal of terrestrial exposures based on inadequate data

The agency dismisses terrestrial routes of exposure because, due to its "high volatility (Henry's Law constant of $7.3X10^{-3}$ atm-m^3/mole), high water solubility (2.4 g/L), and low log Koc (1.6) it is expected that that 1-BP will only be present in terrestrial environmental compartments as a

vapor." Even assuming that expectation is accurate, the extent to which the terrestrial environment and its receptors are impacted by interstitial vapor is completely ignored.

Furthermore, as discussed previously, Part I, sec. 5.A.ii., this conclusion assumes chemical equilibrium. Given the large amounts of 1-BP that are being disposed of in non-hazardous waste landfills and through "other" means of land disposal, the potential for terrestrial organisms to be impacted by these releases is real.

Confusingly, EPA then goes on to dismiss airborne exposures because "[n]o specific conditions of use (i.e. systematic application to land) were identified that resulted in systematic, significant airborne exposures that overlap with terrestrial habitats." However, the agency found that "TRI data *** show air a primary medium of environmental release" (Problem Formulation, p. 39, appendix C) and that "long range transport via the atmosphere is possible" (draft risk evaluation, p. 51).

Given the paucity of environmental monitoring data for 1-BP, the agency should consider environmental fate and transport of similar halogenated organic solvents, such as TCE and perchloroethylene, which have demonstrated impacts on terrestrial organisms through air exposure pathways.[78]

c. Assumed low releases to surface water and low exposures via drinking water

EPA reports that there are no water monitoring data for 1-BP (Problem Formulation, p. 34). Despite their limitations, EPA relies nearly exclusively on TRI data to argue that it need not further analyze exposures via surface water and effectively can conclude such exposures are safe.

First, EPA appears to accept without question the reliability of TRI water release data, even though "[i]n the 2016 TRI, only 1 facility out of 55 reported releases to water." (Problem Formulation, p. 34) EPA uses the data from this one facility to conclude that this particular discharge was safe: "This facility reported 5 lbs of direct surface water discharge; assuming the

[78] *See* Hans Back & Peter Süsser, *Concentrations of volatile chlorinated hydrocarbons and trichloroacetic acid in earthworms*, 24:12 SOIL BIOLOGY & BIOCHEMISTRY 1745–1748 (1992), 10.1016/0038-0717(92)90181-V ("The field study revealed a contamination of the earthworms with volatile chlorinated hydrocarbons (VCH), especially with TCE *** These data suggest that the main contamination of the worms with TCE does not arise from the refuse tip but is airborne and comes possibly from the adjacent chemical plant."); *see also* Ludwig Weissflog et al., *Trichloroacetic acid in the vegetation of polluted and remote areas of both hemispheres—Part I. Its formation, uptake and geographical distribution*, 35:26 ATMOSPHERIC ENVIRONMENT 4511–4521 (2001), 10.1016/S1352-2310(01)00226-6 ("TCA in the pine needles can be explained *** by the direct formation of TCA by plants due to the ubiquitous spread of TECE [perchloroethene] and TCE.")

release occurred over a single day, the surface water concentration in reported receiving waters is well below the COC [concentration of concern] based on EPA's preliminary calculations." (Problem Formulation, p. 34)

Then EPA uses those single-facility data to model surface water concentrations *in general*: "EPA used the reported releases from EPA's Toxics Release Inventory (TRI) to predict surface water concentrations near reported facilities for this Problem Formulation." (Problem Formulation, p. 35) EPA then definitively concludes, based on the TRI data from this one facility, that "*releases to water are very low*." (Problem Formulation, p. 35, emphasis added). These calculations are reported again in the draft risk evaluation (Appendix G).

Building from there, EPA uses an analysis based on the limited TRI data, without any qualification, to assert *all drinking water* exposures are also low:

> Recent TRI reporting indicated 0 pounds released to POTWs and 5 pounds released directly to water in 2016. EPA pretreatment regulations for industrial users discharging wastewater to POTWs *are expected* to limit the discharge of 1-BP to POTWs and ultimately to surface water (see Section 2.3.4). Waste disposal practices and 1-BP's rapid volatilization from water *are expected* to mitigate drinking water exposure potential and there is no data of 1-BP found in US drinking water." (Problem Formulation, p. 39, emphases added)

EPA's reliance on extremely limited TRI data, coupled with unsupported "expectations" that discharges and exposures will be minimal, is capped off here with an outlandish assumption that the lack of monitoring data for 1-BP means it must not be present. Has the chemical even been looked for in drinking water? No data on that question are cited by EPA.

EPA cannot equate a lack of evidence of 1-BP's presence in water with evidence of its absence, but that is precisely what EPA appears to be doing here.

The last step in EPA's construction of its house of cards comes on page 68 of the problem formulation:

> Environmental hazards will not be further analyzed *because exposure analysis* conducted using physical and chemical properties, fate information and *TRI environmental releases for 1-BP* show that ecological receptors are not significantly exposed to TSCA-related environmental releases of this chemical.

EPA makes a wholly exposure-based argument for its decision not to even consider the environmental hazards the chemical may present via water exposures, an approach industry interests have long advocated for, but one which fails to constitute sound science. (See Part I,

sec. 1.C.i.c. for discussion of additional problems with EPA's calculations of its concentrations of concern for aquatic species.)

Rather than constructing such a tenuous line of argument to compensate for the lack of any water monitoring data for 1-BP, EPA should use its clear TSCA authority under section 4 to require the development of the data.

More broadly, EPA cannot justify its heavy reliance on TRI data without resolving the discrepancies discussed earlier that cast serious doubt on the completeness and accuracy of these data.

B. Human health

i. EPA has dismissed available human studies on illegitimate grounds.

In the draft risk evaluation for 1-BP, the agency selected the Honma et al. (2003) rat study for derivation of its point of departure (POD) for dose-response assessment for 1-BP. While the agency acknowledged that available occupational epidemiologic studies "provided evidence of neurotoxicity in 1-BP-exposed workers, it asserted that these studies had "several methodological limitations" (p. 157) and chose not to use them for POD derivation. The decision to dismiss the human epidemiologic evidence has direct implications for assessing population risks, as it is likely that use of human epidemiologic data from the study database would have resulted in identification of a POD considerably lower than that derived by EPA using Honma et al. (2003).

Below, we first discuss the value of epidemiology in risk assessment and then document flaws in EPA's process leading to its dismissal of available epidemiologic evidence.

a. Epidemiology is a foundation of environmental public health decision-making at EPA and beyond.

Throughout the history of EPA, human epidemiological studies have served as the "gold standard" for assessing population risks and guiding the Agency's efforts to protect public health and the environment. Epidemiologic studies provide information critical to understanding the causes of disease, factors influencing population susceptibility, and the actual levels of exposure at which health effects occur. Integration of evidence from epidemiologic, *in vivo* and *in vitro* studies can reduce uncertainties associated with each study design and allows for stronger scientific conclusions about risks.[79]

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

In setting aside the epidemiological data in the draft risk evaluation for 1-BP, EPA is ignoring the most important and relevant data for dose-response assessment. The value of epidemiological data for human health risk assessment has been stated and reinforced by EPA[80] and others over many years. For example:

- EPA's 2005 Guidelines for Carcinogen Risk Assessment
  - "Data from epidemiologic studies are generally preferred for characterizing human cancer hazard and risk."[81]
  - "Epidemiologic data are extremely valuable in risk assessment because they provide direct evidence on whether a substance is likely to produce cancer in humans, thereby avoiding issues such as: species-to-species inference, extrapolation to exposures relevant to people, effects of concomitant exposures due to lifestyles."[82]

- EPA's 1991 Guidelines for Developmental Toxicity Risk Assessment
  - "Since the purpose of risk assessment is to make inferences about potential risks to human health, the most appropriate data to be used are those deriving from studies of humans."[83]

- ATSDR's 2005 Public Health Assessment Guidance Manual (Update)
  - "Clearly, a study based on human data holds the greatest weight in describing relationships between a particular exposure and a human health effect. Fewer uncertainties exist about potential outcomes documented in well-designed epidemiologic studies."[84]

- Members of the risk assessment research community:
  - "Epidemiology is essential to our understanding of the role of environmental exposures in human disease. After all, it is only by studying the human population that we will understand the complex interactions of the environment,

[80] As explained above in Part I, sec. 3.E.i, EPA must comply with its guidance documents in preparing the final risk evaluation
[81] U.S. EPA, *Guidelines for Carcinogen Risk Assessment* pp. 1-11 (2005), https://www.epa.gov/risk/guidelines-carcinogen-risk-assessment.
[82] *Id.* at 2-3.
[83] U.S. EPA, *Guidelines for Developmental Toxicity Risk Assessment* p. 1 (1991), https://www.epa.gov/risk/guidelines-developmental-toxicity-risk-assessment.
[84] Agency for Toxic Substances and Disease Registry, *Public Health Assessment Guidance Manual (Update)* (2005), https://www.atsdr.cdc.gov/hac/phamanual/pdfs/phagm_final1-27-05.pdf.

social factors, heredity, and behavior that determine individual and population health."[85]

Therefore, any exclusion of the human epidemiological evidence in the 1-BP risk evaluation must be clearly justified, including a presentation of the impacts of this exclusion on PODs and associated measures of margins of exposure (MOE), and its public health implications.

b. Problems with the epidemiologic study evaluation tool and its application.

EPA applied its Office of Pollution Prevention and Toxics' (OPPT) updated data quality criteria for epidemiologic studies to the three occupational studies.[86] The completed data quality evaluation for these studies was provided in the Systematic Review Supplemental File.[87] In this document (p. 9), EPA assigns the Li et al. (2010) study an Overall Quality Determination of 2.4, which corresponds to a "Low" rating. We have identified several problems with EPA's approach to evaluating the epidemiologic evidence, both with the employed tool itself and with the effect of applying that tool to the human epidemiological data.

With regard to the revised tool, EPA provides neither an explanation nor empirical support for its revisions to the systematic review data quality criteria for epidemiological studies, and certain revisions make it more difficult for epidemiological studies to be scored overall as high quality. EPA's scoring methodology is already at odds with best practices in systematic review,[88] and the agency's decision to alter scoring criteria without providing any empirical rationale for the changes further underscores that the methodology is not evidence-based.

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[85] Keeve E. Nachman, et al., *Leveraging epidemiology to improve risk assessment*, 4 THE OPEN EPIDEMIOLOGY J. p. 25 (2011), https://pdfs.semanticscholar.org/2655/e207ac111f37db20ab0bc92296f06fd88f6f.pdf.

[86] *See* Gaku Ichihara, et al., *Neurologic Abnormalities in Workers of a 1-Bromopropane Factory*, 112:13 ENVTL. HEALTH PERSPECTIVES pp. 1319-1325 (2004), https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1247523/; Li, W., et al., *Dose-Dependent Neurologic Abnormalities in Workers Exposed to 1-Bromopropane*, 52:8 J. OF OCCUPATIONAL & ENVTL. MEDICINE pp. 769-777 (2010), https://www.ncbi.nlm.nih.gov/pubmed/20657306; Toraason, M., et al., *DNA damage in leukocytes of workers occupationally exposed to 1-bromopropane*, 603:1 MUTATION RESEARCH/GENETIC TOXICOLOGY & ENVTL. MUTAGENESIS pp. 1-14 (2006), https://www.ncbi.nlm.nih.gov/pubmed/16412685.

[87] U.S. EPA, Risk Evaluation for 1-Bromopropane (n-Propyl Bromide) CASRN: 106-94-5 Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies – Epidemiologic Studies (July 2019), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0235-0012.

[88] *See* EDF's earlier comment at https://www.regulations.gov/document?D=EPA-HQ-OPPT-2018-0210-0077.

Further, at least six metrics in EPA'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 (which was previously possible). These types of revisions were made only to the epidemiologic evaluation criteria; similar modifications were not made to the criteria used for animal or in vitro studies, where it remains possible for them to score "High" across every data quality metric. The effect of this in practice is likely to diminish the contribution of epidemiological studies relative to *in vivo* and *in vitro* experimental studies.

In addition to concerns with individual scoring criteria, the scheme used to calculate the overall rating for a particular study is not clearly presented in either the updated criteria document or the risk evaluation and its supporting files. The following equation is presented for calculating the overall rating:[89]

* MWF = Metric Weighting Factor
† High = 1; Medium = 2; Low = 3; Unacceptable = 4; N/A has no value.
‡ The overall rating is calculated as necessary. EPA may not always provide a comment for a metric that has been categorized as High.

$$\text{Overall rating} = \begin{cases} 4 & \text{if any metric is Unacceptable} \\ \left[\sum_i (\text{Metric Score}_i \times \text{MWF}_i) / \sum_j \text{MWF}_j\right]_{0.1} & \text{(round to the nearest tenth) otherwise} \end{cases},$$

where High =≥ 1 to < 1.7; Medium =≥ 1.7 to < 2.3; Low =≥ 2.3 to ≤ 3.0. If the reviewer determines that the overall rating needs adjustment, the original rating is crossed out and an arrow points to the new rating.

The subscripts *i* and *j* are not defined, and the subscript of *0.1* is not explained. From this equation, it is not possible to see how EPA calculated its overall rating of 2.4 for Li et al. (2010).

Given the concerns related to the appropriateness of the OPPT tool for epidemiologic studies, the agency needs to consider use of other study evaluation tools that are available and are more appropriate for assessing the quality of observational epidemiologic studies. One recent example is the Conducting Systematic Reviews and Meta-Analyses of Observational Studies of Etiology (COSMOS-E) tool,[90] and there are others such as the Navigation Guide.[91]

[89] USEPA Risk Evaluation for 1-Bromopropane (n-Propyl Bromide) CASRN: 106-94-5 Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies – Epidemiologic Studies, July 2019 Draft, pp. 4, 10 and 14, available at https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0235-0012.
[90] Olaf M. Dekkers, et al., *COSMOS-E: Guidance on conducting systematic reviews and meta-analyses of observational studies of etiology*, 16:2 PLOS MEDICINE (2019), https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6383865/.
[91] Tracey Woodruff & P. Sutton, *The Navigation Guide Systematic Review Methodology: A Rigorous and Transparent Method for Translating Environmental Health Science into Better Health Outcomes*, 122:10 ENVTL. HEALTH PERSPECTIVES pp. 1007-14 (2014), https://www.ncbi.nlm.nih.gov/pubmed/24968373.

c. Flaws in EPA OPPT's choice of a toxicologically-based POD when viable epidemiologic evidence was discarded.

Despite evidence from human epidemiological studies of effects at lower levels of exposure, EPA has selected a point of departure (POD) for chronic, non-carcinogenic effects of 1-BP of 18.2 ppm (HEC=25 ppm) based on neurological effects in a rat study (Honma et al., 2003). EPA states that the human epidemiological studies have "several methodological limitations" (p. 156) that are discussed in Appendix I.4; however, EPA does not provide a clear rationale for the exclusion/dismissal of these studies.

There are three human epidemiological studies on the health effects of 1-BP – Ichihara et al. (2014), Li et al. (2010) and Toraason et al. (2006) – discussed in Appendix I.4. Ichihara et al. (2004) examined neurological abnormalities in 23 female workers at a 1-bromopropane factory in China. Compared to age-matched controls, exposed workers showed significant differences on tests of vibration sense, distal latency, nerve conduction velocity, and several neurobehavioral measures. Li et al. (2010) conducted a follow-up study to Ichihara et al. (2004)[92] by examining workers from two additional 1-bromopropane factories.[93] Exposure to 1-BP was associated with dose-dependent decreases in vibration sense, red blood cells, and sensory nerve conduction velocity. Toraason et al. (2006) investigated DNA damage in peripheral leukocytes of workers exposed to 1-BP.[94] Results of *in vivo* and *in vitro* tests provided evidence of an association between 1-BP exposure and DNA damage.

Although several limitations of the three human epidemiological studies are presented in the Draft Risk Evaluation, EPA does not provide a convincing argument for outright exclusion of these studies. For example, EPA states that Li et al. (2010) did not report or control for BMI, a known confounder for vibration sense, in their analyses. In fact, Li et al. (2010) found significant associations between 1-BP and decreased vibration sense after controlling for height and weight in multiple regression analyses. Moreover, EPA's discussion of the studies in Appendix I.4 does not appear to be consistent with the confidence ratings presented in the Supplemental File. No serious flaws that would make the data unacceptable for use in the human health hazard assessment were identified by the OPPT tool for any of the three epidemiological studies. Despite the fact that Ichihara et al. (2004) was assigned a "Medium"

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[92] Gaku Ichihara, et al., *Neurologic Abnormalities in Workers of a 1-Bromopropane Factory*, 112:13 ENVTL. HEALTH PERSPECTIVES pp. 1319-1325 (2004), https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1247523/.

[93] Li, W., et al., *Dose-Dependent Neurologic Abnormalities in Workers Exposed to 1-Bromopropane*, 52:8 J. OF OCCUPATIONAL & ENVTL. MEDICINE pp. 769-777 (2010), https://www.ncbi.nlm.nih.gov/pubmed/20657306.

[94] Toraason, M., et al., *DNA damage in leukocytes of workers occupationally exposed to 1-bromopropane*, 603:1 MUTATION RESEARCH/GENETIC TOXICOLOGY & ENVTL. MUTAGENESIS pp. 1-14 (2006), https://www.ncbi.nlm.nih.gov/pubmed/16412685.

confidence rating, EPA claims that it is "difficult to interpret the results of the study" based on the small sample size (p. 347). EPA should provide a clear justification before excluding/dismissing evidence from human epidemiological studies on the health effects for 1-BP.

The rationale presented by EPA for assigning ratings to certain metrics in the Supplemental File also appears to be inadequate or flawed. To give one example, in the evaluation of Li et al. (2010), EPA states that Metric 7 (Outcome measurement or characterization) was assigned a "Low" rating due to 1) a failure to collect individual skin temperatures at the test site based on standard methods, and 2) the inherent imprecision of the vibration sense outcome measurement based on the sensitivity of the subject relative to the examiner and the evidence of high variability in vibration sense reported in women. EPA fails to recognize that the vibration sense endpoint would not have been affected by skin temperature (which only would have impacted tests of nerve conduction velocity). In addition, EPA did not report that examiners were blinded to the exposure groups, and that the differences in vibration sense remained significant after adjustment for examiner and subject characteristics. Further, as noted by the Agency for Toxic Disease Substances and Registry (ATSDR) in its 1-BP evaluation, Li et al.'s use of a 128 Hz tuning fork will likely *underestimate* the presence of dysfunction as compared to what they likely would have measured had they used the gold standard for vibration sense (a neurothesiometer).[95]

EPA does not acknowledge that there is precedent from ATSDR for using data from Li et al. (2010) to set health-based standards for chronic inhalation exposures to 1-BP.[96] ATSDR (2017) acknowledged the limitations of Li et al. (2010) and rated confidence in the study as low but noted that the neurological effects observed are consistent with evidence from numerous studies of other 1-BP exposures. The endpoint of decreased vibration sense, which showed a dose-response relationship with 1-BP in Li et al. (2010), has also been reported in five case reports of human exposure, as reported in ATSDR 2017.[97]

In its draft risk evaluation, EPA focuses its criticisms on Li et al. (2010) without considering the broader availability of evidence from human studies of 1-BP. A more holistic consideration of the combined database of case reports and occupational epidemiologic studies may have increased EPA's confidence in Li et al. (2010).

[95] Agency for Toxic Substances and Disease Registry, *Toxicological Profile for 1-Bromopropane* p. 222 (2017), https://www.atsdr.cdc.gov/ToxProfiles/tp.asp?id=1471&tid=285.
[96] *Id.*
[97] *Id.* at p. A-15.

d. The effect of POD selection can significantly impact the resulting risk metric: a comparison of approaches in the ATSDR Toxicological Profile and EPA Draft Risk Evaluation for 1-BP.

The recent work on 1-BP by ATSDR demonstrates the importance of including epidemiological data in the Draft Risk Evaluation. The following comparison shows that EPA's evaluation of chronic risks of neurological effects is not adequately protective of human health. Because ATSDR derived a chronic inhalation MRL, similar to a reference concentration (RfC), the following table applies the information in the draft risk evaluation to calculate a risk metric equivalent that can be compared to the MRL. Note: EPA did not derive a risk metric, instead performing a MOE evaluation with a Benchmark MOE=100 to account for interspecies (animal to human) and intra-species (within human) differences. In the table below we present the information from the draft risk evaluation that can be used to derive a risk metric to enable a comparison to the ATSDR approach:

Table: Comparison of approaches by ATSDR (2017) and EPA (2019) for neurological effects from chronic inhalation of 1-BP

	ATSDR Toxicological Profile (2017)	**EPA Draft Risk Evaluation (2019)**
Key Study	Li et al. 2010 (human)	Honma et al. 2003 (rat)
Endpoint	Mild neurological impairment in females (increased vibration sense threshold)	Decreased time hanging from a suspended bar (traction time)
POD	"minimal LOAEL" of 1.28 ppm adjusted for continuous exposure, yielded a point of departure of: POD = 0.46 ppm	$BMCL_{1SD}$ = 18.2 converted to a human equivalent concentration of: HEC = 25 ppm
UFs	30 (3 for minimal LOAEL, 10 for human variability)	100 (10 for UF_A, 10 for UF_H) (Benchmark MOE)
Risk metric	MRL = 0.02 ppm	0.25 ppm

By using the human epidemiology study (Li et al. 2010), ATSDR derived an MRL that is more protective of human health by a factor of 10 (one order of magnitude).

In our comments on the draft risk evaluation for 1,4-dioxane,[98] we highlighted similar concerns regarding EPA's failure to use a human study, Ernstgard et al. (2006) previously used by ATSDR, a decision that resulted in use of a less protective POD. Together, the examples serve more broadly to highlight the inappropriateness of EPA's dismissal of human epidemiological data.

ii. EPA has excluded without justification identified hazards of 1-BP from its quantitative risk characterization.

In the Problem Formulation for 1-BP, EPA states:

> For the 2016 Draft Risk Assessment (U.S. EPA, 2016b) on 1-BP, EPA evaluated studies for the following non-cancer hazards: acute toxicity (acute lethality at high concentrations only), blood toxicity, immunotoxicity, cardiovascular toxicity, liver toxicity, kidney toxicity, reproductive toxicity, developmental toxicity, and neurotoxicity. A comprehensive summary of all endpoints considered can be found in the 2016 Draft Risk Assessment. *Five health hazards were used for quantitative risk characterization* and will be evaluated using our systematic review approach. (p. 43, *emphasis added*)

In EDF's comments on the 1-BP Problem Formulation, we raised concerns about EPA's decision to focus on only five endpoints and exclude other endpoints for 1-BP (p. 92). Yet, EPA has largely repeated its unjustified action in this draft risk evaluation. On p. 163, EPA states that:

> EPA considered adverse effects for 1-BP across organ systems and a comprehensive summary table is in Appendix I (Table_Apx I-2). *The full list of effects was screened to those that are relevant, sensitive and found in multiple studies* which include the following types of effects: liver toxicity, kidney toxicity, immunotoxicity, developmental/reproductive toxicity, neurotoxicity, and cancer as described above. In general, adverse effects were observed in all of these systems in rats exposed to 1-BP by inhalation in the range of 100 – 1000 ppm (LOAELs) (emphasis added).

The agency's rationale for focusing on certain endpoints appears to be based on what it deems "relevant, sensitive, and found in multiple studies." However, EPA provides no description of what these critically important terms "relevant" and "sensitive" actually mean in this context. This lack of transparency on such a central element of the risk evaluation process is highly problematic. EPA must provide a full explanation of the process used in this step of the risk evaluation.

[98] EDF Comments on the Draft Risk Evaluation of 1,4-dioxane p. 81 (Aug. 30, 2019), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0238-0058.

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

EPA should have included an additional uncertainty factor for "the uncertainty associated with extrapolation from animal data when the database is incomplete."[99] 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, 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.[100]

In addition, EPA's reliance on inhalation-to-dermal extrapolation for sub-chronic/chronic effects – necessitated by the dearth of dermal toxicity data – also introduces uncertainty that EPA has failed to account for. This is discussed further in Part I, section 5.B.ix.a. below.

iv. EPA fails to consider cancer risk from acute exposure scenarios.

Despite EPA's acknowledgment that the weight of the scientific evidence indicates 1-BP is a mutagenic carcinogen and that linear extrapolation is warranted (pp. 159, 163), the agency has chosen not to estimate cancer risks based on acute exposures for 1-BP. It provides the following rationale on p. 180:

> EPA did not use the IUR or dermal slope factor to calculate the theoretical cancer risk associated with a single (acute) inhalation/or dermal exposure to 1-BP. Published methodology for extrapolating cancer risks from chronic to short-term

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[99] 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; 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).

[100] 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.

> exposures to mutagenic carcinogens caveat that extrapolation of lifetime theoretical extra cancer risks to single exposures has great uncertainties (NRC, 2001). … Thus, EPA risk evaluation for 1-BP does not estimate extra cancer risks for acute exposures because the relationship between a single short-term exposure to 1-BP and the induction of cancer in humans has not been established in the current scientific literature.

However, the same NRC document cited by the agency above goes on to provide additional relevant information on this subject (pp. 111-112; emphasis added)[101]:

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

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

As stated in this NRC report, the decision to conduct such extrapolation and modeling should be based on the "sound biological and statistical principles." EDF is concerned that EPA did not sufficiently consider such principles related to mode-of-action in arriving at its decision not to model acute cancer risk based on chronic exposure data. In particular, given that 1) the agency recognizes that "[f]ollowing EPA's *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 2005a), the overall weight of the scientific evidence supports a mutagenic MOA for 1-BP induced carcinogenicity" (p. 159), and 2) a mutagenic MOA suggests a role for "a single direct

[101] National Research Council. *Standing operating procedures for developing acute exposure guideline levels for hazardous chemicals*. National Academies Press, 2001.

reaction, specifically, a single hit in a single target (Kirsch-Volders et al., 2000),[102]" a linear low-dose extrapolation from chronic to acute exposures would be the appropriate approach to take for 1-BP. It is possible, though, that even a linear extrapolation from chronic cancer bioassays may underestimate cancer risk due to short-term exposures. Halmes et al., 2000 lends supports to the possibility that short-term exposures can result in similar or higher cancer risks than even chronic lifetime exposures.[103] The study used NTP data where both shorter term and full lifetime studies had been conducted.

EPA's current approach assumes acute exposures to 1-BP, including to consumers, pose *zero* cancer risk – an assumption that is clearly not warranted based on the weight of the evidence. EPA needs to apply an extrapolation that provides a scientifically sound estimate for cancer risk from acute and short-term exposures to 1-BP. As stated in our problem formulation comments (p. 81):

> EPA must closely examine any effect it believes to arise only from chronic exposures to determine whether in fact this is true across the diverse human population, including where potentially exposed or susceptible subpopulations may be at increased risk for effects after shorter periods of exposure compared to the general population.
>
> *v. EPA needs to analyze those potentially exposed or susceptible subpopulations that face greater exposure due to their proximity to conditions of use.*

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

EPA must identify those who face greater exposures due to their proximity to conditions of use as a "potentially exposed or susceptible subpopulation" since they are a "group of individuals

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[102] 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.
[103] N. Christine Halmes, et al., *Reevaluating cancer risk estimates for short-term exposure scenarios*, 58:1 TOXICOLOGICAL SCIENCES 32-42 (2000), https://academic.oup.com/toxsci/article/58/1/32/1658920/#24341943.

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-BP at p. 40 ("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, use or disposal sites).").

But in the draft risk evaluation, EPA does not identify these populations as potentially exposed or susceptible subpopulations (p. 135). 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.

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.

vi. EPA has failed to consider workers' combined exposure from multiple pathways.

EPA never considers the combined calculated risks from the inhalation and dermal exposures – even though many workers could readily experience exposures by both routes, including over the same time period. For example, in the context of estimating dermal exposure, the agency states that "only a fraction of 1-BP that comes into contact with the skin will be absorbed as the chemical readily evaporates from the skin" (p. 106), which would lead to increased concentration in the air in the immediate vicinity of the dermally exposed worker. Because both inhalation and dermal exposure result in systemic distribution of 1-BP,[104] it is essential to evaluate both of these routes in combination, including simultaneously, to assess total body burden and the associated effects.

EPA does acknowledge in the draft risk evaluation that workers and consumers may experience both inhalation and dermal exposures – and even implies that the agency aggregated these exposures:

[104] *See* NIOSH, *Skin Notation Profiles: 1-Bromopropane* p. 2 (August 2017), https://www.cdc.gov/niosh/docs/2017-187/2017-187.pdf?id=10.26616/NIOSHPUB2017187; and 1-BP draft risk evaluation, p. 145.

> As part of this risk evaluation, EPA considered aggregate exposures by evaluating exposure and risk from both the inhalation and dermal routes for workers and consumers in scenarios where such exposures are expected. EPA expects workers to be exposed via both inhalation of 1-BP vapor and dermal contact with liquid containing 1-BP. Similarly, EPA expects certain consumer users to be exposed via both the inhalation of 1-BP vapor and dermal contact with liquid containing 1-BP.

However, based on our reading of the draft risk evaluation, we see no evidence that EPA added together the risks from dermal and inhalation exposures, as implied above. For example, all of the unreasonable risk determinations (Table 5-1) are based on *either* dermal risk or inhalation risk drivers. Merely assessing both routes of exposure – but not adding them together – does not constitute an aggregate exposure assessment. See also Part I, sec. 6.C.

Our concern was reinforced during the 1-BP SACC peer review meeting, as several SACC members indicated that the agency should combine the inhalation and dermal exposures. Another concern raised by a SACC member is salient here as well: EPA has ignored all non-occupational baseline exposures workers experience, due to its exclusion of all exposures via environmental releases to air, water, and land. The SACC member argued that the agency at least needs to take these into account as baseline exposures for workers, even if the agency 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-BP presents to an individual.

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

EPA excluded a number of reasonably foreseen conditions of use in the workplace that should have been evaluated. During the SACC meeting to peer-review the 1,4-dioxane draft risk evaluation, there was robust discussion regarding a number of exposure scenarios that the agency failed to consider, each of which equally applies to 1-BP. Among those discussed are:

- Exposures from spills in the workplace, especially considering the potential for 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;
- Exposures of maintenance staff, especially those cleaning up spills and leaks; and
- Exposures of workers at small or medium facilities where assumptions of routine PPE use or other protections are even less likely to be valid.

With regards to the last point, there are some scenarios in which EPA did not assume use of respirators for workers (e.g., cleaning and furniture care for: dry cleaning; spot cleaner and stain remover; and liquid cleaner, arts/crafts/auto care). While far from clear in the draft risk evaluation, EPA indicated at the 1-BP SACC meeting that the agency based these decisions on SDSs and a review of industrial hygiene practices in the literature. While there is logic behind the assumption that larger industrial facilities are more likely to have a better industrial hygiene program entailing protective equipment and engineering controls (a point that members of the 1,4-dioxane SACC made), EPA should be cautious in assuming that all larger facilities have robust industrial hygiene programs. For example, a 1-BP SACC member with industrial hygiene expertise indicated that he has personally been to large industrial facilities with poor industrial hygiene programs. He specifically pointed to a 2008 MMWR which found significant 1-BP exposure as a result of a poor industrial hygiene program at a large circuit board manufacturing company.[105] Workers at any facility – whether small, medium, or large – where use of effective PPE cannot be convincingly documented to apply should be considered vulnerable subpopulations. For these subpopulations, EPA must determine risk based on exposures without assuming any use of PPE.

"Conditions of use" are broadly defined to mean "the circumstances, as determined by the Administrator, under which a chemical substance is intended, known, or *reasonably foreseen* to be manufactured, processed, distributed in commerce, used, or disposed of." 15 U.S.C. § 2602(4) (emphasis added). Each of the circumstances described above—spills, take home exposures, exposures to maintenance staff, and exposures without appropriate PPE—is a "reasonably foreseen" aspect of the circumstances under which 1-BP is manufactured, processed, distributed, used, or disposed of. It is well established under the law that "[a] natural and probable consequence is a foreseeable consequence. But to be reasonably foreseeable [t]he consequence need not have been a strong probability; a possible consequence which might reasonably have been contemplated is enough." *People v. Medina*, 46 Cal. 4th 913, 920 (Cal. 2009) (internal citations and quotation marks omitted).

Reasonably foreseen is a term of art with a long history in the law, and EPA should turn to the ample precedent interpreting this language to inform implementation of this legal requirement. Spills and leaks are undoubtedly reasonably foreseeable, and indeed, when preparing environmental impact statements (EISs) for federal projects, the federal government regularly analyzes the potential for spills and leaks because they are reasonably foreseen aspects of such

[105] U.S. Center for Disease Control and Prevention, *Neurologic Illness Associated with Occupational Exposure to the Solvent 1-Bromopropane --- New Jersey and Pennsylvania, 2007—2008*, MORBIDITY AND MORTALITY WEEKLY REPORT (Dec. 5, 2008), https://www.cdc.gov/mmwr/preview/mmwrhtml/mm5748a2.htm.

projects. *See, e.g., Sierra Club v. Clinton*, 689 F. Supp. 2d 1123, 1139 (D. Minn. 2010) (describing analysis of potential for leaks and spills).

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

Take home exposure, maintenance staff exposure, and exposure of persons not using PPE are equally reasonably foreseen.

viii. EPA may have underestimated risk to ONUs.

EPA states: "Respirator use for occupational non-users was not evaluated because they do not directly handle 1-BP and are unlikely to wear respirators." (p. 24)

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

Nevertheless, EPA may still have underestimated exposure to ONUs in several ways. First, ONUs may not stay within their "far field zone" – i.e., outside of the "near field" workers' zone – as EPA assumes. Several 1-BP SACC members raised this concern. For example, one member indicated that ONUs may not have exposures distinct from workers in smaller dry cleaning operations. The same is likely true for other industries with relatively small work spaces. Another SACC member with industrial hygiene experience noted that workers and ONUs may regularly pass into each other's space, e.g, to communicate or otherwise socialize. EPA appears to acknowledge but fails to account for this: "The model assumes the occupational non-user spends their time entirely in the far-field. In reality, it is reasonably foreseeable that these employees will occasionally perform activities in the near-field, thereby having a higher level of exposure" (p. 91).

Second, in several scenarios EPA assumed exposure to ONUs would be negligible, apparently without any data to support the assumption. For example:

- Non-cancer acute and chronic inhalation risks to ONUs were not considered for Import, Repackaging, Processing as a Reactant, and Processing – Incorporation into Articles and Disposal because, as "the model assumes tank truck and railcar loading/unloading occurs outdoors, EPA expects ONU exposure to be negligible due to airborne concentration dilution in ambient air." (pp. 195, 204, 208, 225) No supporting data or analysis were provided.
- Non-cancer acute and chronic inhalation risks to ONUs were not considered for Manufacturing. This is EPA's basis for this decision: "Exposure monitoring was not performed for ONUs at this manufacturing facility. Based on the process and work activity description, exposure to ONU is expected to be negligible." (pp. 195, 207) Again, no supporting data or analysis were provided.

Third, it does not appear that EPA evaluated dermal risk for ONUs at all. While the draft risk evaluation indicates that EPA has done so on p. 20, elsewhere EPA suggests it did not: "Dermal exposure to liquid is not expected for occupational non-users, as they do not directly handle 1-BP" (p. 107). Moreover, none of the dermal risk estimate tables include a column for ONUs, as is done for the inhalation risk tables.

ix. EPA has inadequately addressed the uncertainties in its dermal risk estimates.

a. Inhalation to dermal extrapolation

EPA defined dermal HEDs by extrapolating from inhalation PODs. EPA's decision to rely on inhalation-to-dermal extrapolation contributes substantial uncertainty to its risk calculations. Therefore, as is recommended for route-to-route extrapolation generally.[106,107] EPA should apply an additional uncertainty factor of 10 to account for these uncertainties.

b. Dermal exposure to vapors or mists

EPA has not considered dermal exposure from vapor or mist deposition, despite the fact that the agency stated in the 1-BP Problem Formulation that "[d]ermal exposure may occur via vapor/mist deposition onto skin or via direct liquid contact during use, particularly in occluded scenarios." (p. 38 of Problem Formulation, emphasis added) EPA has not provided a

[106] *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/.

[107] 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.

justification for this decision or addressed the uncertainty it imparts to its dermal exposure assessment and associated risk estimates.

x. EPA appears to have ignored a significant source of data on inhalation exposure.

EPA has relied on occupational exposure data it received from OSHA, but has failed to make it public.[108] Because the data set is not publicly available, it is not possible to determine whether it is or includes the dataset EPA received two years ago from Dr. Adam Finkel in his comments urging EPA to list 1-BP as a HAP.[109] In his comment on the HAP petition, Dr. Finkel wrote that:

> in prior comments [I showed] that the average 1-BP concentration OSHA has found in workplace air sampling was about 59 ppm; for these comments, I updated the exposure data I received in a 2007 FOIA case with newer data now available on the OSHA website (I have merged the two data sets as a single Excel file, and uploaded it with these comments). Combining both datasets yields 261 separate air samples for 1-BP taken between 1998 and 2015. The average concentration is now just under 30 ppm (see Cell S264 of the spreadsheet), but ranged up to more than 422 ppm (Cell S267). More than 14 percent of all the samples (Cell S266) exceeded the 62.5 ppm rodent bioassay dose.

EPA did not mention in the draft risk evaluation whether it considered the data submitted by Dr. Finkel. However, EPA utilized OSHA 2019 data for two conditions of use in the draft risk evaluation: vapor degreasing and spot cleaning (pp. 71, 94).

The table with the monitoring table for vapor degreasers listed 6.70 ppm as the 50th percentile and 49.4 as the 95th percentile 8-hour TWA (p. 71). The table with the monitoring table for spot cleaners listed 0.90 ppm as the 50th percentile and 4.73 as the 95th percentile 8-hour TWA (p. 94). These values are significantly lower than the average and high-end concentrations identified in the data from Dr. Finkel.

Although it is not clear whether Dr. Finkel's OSHA data only pertained to vapor degreasing and spot cleaning, none of the values reported for monitored worker exposures EPA relied on for a number of other conditions of use (*see* pp. 60, 62, 63, and 66) came close to the levels of 1-BP identified in Dr. Finkel's OSHA data.

[108] The link for OSHA, 2019 goes to HERO, where no data is available: https://hero.epa.gov/hero/index.cfm/reference/details/reference_id/5018565.
[109] Attachment 2: OSHA 1bp samples through 2015, https://www.regulations.gov/document?D=EPA-HQ-OAR-2014-0471-0084.

EPA must explain the discrepancies between the monitoring data submitted by Dr. Finkel and the monitored worker exposure values EPA relied on in the draft risk evaluation. Moreover, EPA must make the OSHA 2019 dataset it used publicly available (see Part II, sec. 9).

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

In its dermal exposure assessment, EPA assumes one exposure event per day for both workers (p. 107) and consumers (except for insulation) (p. 131). EPA provides little justification for these assumptions.

Yet, given the typical 8-hour (or longer) work day and the repetition common in many jobs, it seems far more likely that workers would regularly engage in activities that could result in multiple exposure events per day. EPA seems to recognize this when it states:

> In addition, the underlying EPA dermal model assumes one exposure event per day, which likely underestimates exposure as workers often come into repeat contact with the chemical throughout their work day. Based on the uncertainties described above, EPA has a medium level of confidence in the assessed baseline exposure. (p. 108)

But EPA then fails to account for this underestimation or providing any sort of uncertainty analysis.

With regards to consumers, EPA not only assumes a single exposure event per day but also assumes that exposure will never be chronic in nature. We have discussed our concerns with this assumption in Part I, sec. 4.B.ii.

EPA at least recognizes the uncertainty with its once-per-day approach given that "do-it-yourselfers" may be exposed more frequently:

> This assumption may result in underestimating the exposure of certain consumer users, in particular those consumers who may be do-it-yourselfers who may use products more frequently or may use more than one product within a single day. There is a medium uncertainty associated with this assumption because of the possible of underestimating exposure of frequent use or multi-product users. (p. 130)

As EPA has acknowledged, the assumption of a single exposure event a day seems likely to significantly underestimate the risk faced by workers and by at least some consumers. This calls into question its ultimate risk calculations. At the very least, EPA needs to conduct a sensitivity analysis regarding this assumption.

xii. EPA has failed to address 1-BP exposure and risk to children, a susceptible subpopulation, from dry cleaning.

EPA has not calculated risk for children exposed to 1-BP from dry cleaners. By not doing so, EPA has ignored the recommendations from the Chemical Safety Advisory Committee (CSAC). In a cursory fashion, EPA dismissed the risk to this susceptible subpopulation based on two highly flawed assumptions: 1) acute health effects are not applicable to children, and 2) chronic exposures are not relevant to children. EPA's limited consideration of children under this condition of use lacks transparency and is contrary to the emphasis TSCA places on EPA's need to ensure protection of susceptible subpopulations.

a. EPA's decision to ignore risks to children is inconsistent with the recommendations of the CSAC.

EPA's failure to consider children's exposures from dry cleaning operations is contrary to the recommendations EPA received from the CSAC in its review of the 2016 Draft Risk Assessment for 1-BP:

> The Committee noted that the problem formulation and scope should be expanded to include chronic exposures and risk to the general population, including infants and children, from operations that use 1-BP for degreasing or dry-cleaning, or other emissive sources.[110]

EPA even noted the CSAC's recommendation in the current draft risk evaluation:

> EPA also considers exposure to children who may be present at the workplace, such as small family-owned dry cleaners, an occupational exposure scenario *recommended for assessment from the peer review* of the 2016 Draft Risk Assessment of 1-BP (p. 52, emphasis added).

Despite this recommendation, EPA failed to conduct a formal risk evaluation for such children. EPA also failed to make clear that it did not adopt the recommendations of the CSAC, and instead chose not to assess children's risks from dry cleaning operations.

Moreover, EPA appears to have artificially distinguished between children who are bystanders *within* the dry cleaning operation and children who are in the general population that live in

[110] Memorandum, Steven M. Knott, Designated Federal Officer, to Wendy Cleland Hamnett, Director Office of Pollution Prevention and Toxics, Transmittal of Minutes of the May 24-25, 20 16 Chemical Safety Advisory Committee (CSAC) Meeting Regarding the Draft Risk Assessment for TSCA Work Plan Chemical 1-Bromopropane (CASRN-1 06-94-5) pp. 23-24 (Aug. 22, 2016), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2015-0805-0028 (hereinafter "CSAC Report on 1-BP Risk Assessment").

homes co-located with the dry cleaning facilities – without directly explaining this in the draft risk evaluation. Instead, the draft provides no analysis of the general population and thus does not even acknowledge that children living in co-located homes are likely to be exposed to 1-BP. As EPA has noted, however, 1-BP is highly volatile and these children will likely be exposed via vapor intrusion. The CSAC previously recommended consideration of these exposures to EPA:

> 1-BP is a high production volume chemical and very volatile. Clearly because 1-BP is highly volatile, like perchloroethylene, it will escape from dry cleaning, degreasing, and other emissive operations. Many of the engineering controls described in the document involve venting 1-BP vapors to the outside air. *** Thus, the Committee found that exclusion of chronic exposure of the general public near facilities using 1- BP is a *major limitation* of this risk assessment.[111]

Unlike EPA's mandates under the Clean Air Act which EPA now relies on to ignore general population air exposures to 1-BP (see Part I, sec. 2.B.), under TSCA EPA is tasked with specifically considering whether a chemical substance poses an unreasonable risk to susceptible subpopulations, including children. EPA's failure to consider children's exposures to dry cleaning emissions when they live in co-located buildings is contrary to EPA's mandate under TSCA.

b. EPA failed to calculate acute risk.

While EPA calculates acute *exposure* to children at family dry cleaners (see Part I, sec. 5.B.xii.c. below), it stops short of actually calculating the associated risk. EPA's rationale is as follows:

> For acute exposure scenarios, EPA did not assess risks to children who may be present in in the workplace (e.g., dry cleaners) due to the uncertainties in extrapolating these specific developmental effects for this lifestage. (p. 194)

Instead, EPA assumes its risk estimates for pregnant women are protective of all lifestages: "Therefore EPA assumed that margins of exposure for pregnant women would also be protective of other lifestages" (p. 247-248).

However, this assumption is in direct contradiction to the recommendations of the CSAC in 2016 provided in its comments on the agency's 2016 1-BP Work Plan Risk Assessment. The CSAC specifically recommended that the agency *not* consider estimates for pregnant women to be protective of children:

> While the Agency states that they focused on exposures to pregnant women and that this may be protective of other populations, it should NOT be assumed that

[111] *Id.* at p. 21 (emphasis added); *see also id.* at pp. 12, 18.

> estimates based on pregnant women would also be protective of young children (children are still developing, have higher inhalation rates, larger skin surface area, different body weights, etc. compared to adults which would impact exposure doses estimated). In addition, as indicated by the Agency, there are 'postnatal exposure studies showing adverse developmental effects that manifest at various stages of development, and span multiple generations (WIL 2001).' Thus, at the very least the Agency should report this as a limitation of this risk assessment and indicate that exposures to children in these facilities could be higher than those estimated for the reasons stated above."[112]

Based on the CSAC's recommendation, EPA should have evaluated risk to children based on developmental effects. One potential option would be to derive a developmental toxicity POD for children based on the referenced postnatal exposure studies. (Note that while the "WIL Research (2001)" is also referenced in the draft risk evaluation, a link to the study is not provided in the HERO database; it is therefore challenging for EDF to confirm whether this approach is possible.)

However, even if EPA had a robust scientific rationale to support its assertion that there are too many uncertainties to extrapolate developmental effects to children, it would not be a sufficient rationale to ignore this susceptible subpopulation altogether. The agency should have, at the very least, evaluated the risk to children based on other endpoints. 1-BP results in a host of health effects following acute exposure, ranging from systemic toxicity to neurotoxicity, and the agency should at least have estimated risk based on the next most sensitive endpoint. Because children typically are subject to a higher internal dose from the same level of exposure, even use of less sensitive endpoints may have identified very real risks to children from 1-BP exposure. EPA acknowledged this option on page 248, but apparently chose not to conduct these calculations.

c. EPA's acute exposure assessment of children is inadequate.

Setting aside the fact that EPA did not utilize the results of its acute exposure assessment for children at dry cleaning facilities, it is important to note the inadequacies of the approach it took. EPA had no actual exposure data for this population, but instead relied exclusively on models. Given that the CSAC recommended in 2016 that this subpopulation needs to be evaluated, the agency has had plenty of time to acquire the needed monitoring data.

Further, the exposure assessment is likely to have underestimated exposure to children at family-owned dry cleaning facilities, for a number of reasons.

[112] CSAC Report on 1-BP Risk Assessment, pp. 28-29.

First, EPA assumes only four hours of exposure: "[b]ecause many dry cleaners are family owned and operated, EPA assumed children may be present for a four-hour period (3 – 7pm) after school" (p. 91). This assumption ignores several obvious realities:

- Not all children attend school: What about children that are unable to or too young to attend school (e.g., infants - 5 years of age), or are home-schooled?
- Children are not at school every day: What about exposures over the weekend or over the summer months?
- The day does not end at 7 pm: What about exposures that may occur in the evening or overnight?

Second, it is unclear from both the draft risk evaluation and the Supplemental Information on Occupational Exposure Assessment[113] whether exposure factors for children were incorporated into the model. Both documents simply state that children, "may be exposed at similar levels as occupational non-users" (draft risk evaluation, p. 91). This statement suggests that considerations such as increased breathing rate, mouthing behaviors, or dermal permeability in children were not taken into account. Based on EPA's assumption of four hours of exposure after school, EPA appears to have completely excluded infants and pre-school children – the very population that would have the highest internal dose – and potentially the greatest susceptibility – from the same level of exposure.

Third, it is worth noting that the CSAC recommended EPA consider children in dry cleaning facilities not only as bystanders, but also as workers.[114] The CSAC wrote in 2016:

> The Committee also noted that estimating exposures to children ("workers" and "bystanders") in dry cleaning facilities could be considered. It is plausible that in family-owned/operated dry cleaning facilities children under 16 could be helping/working (or could be "bystanders" while they wait for their parents to finish their job) and potentially be exposed to 1-bromopropane[.]

It is easy to envision scenarios in which older children help their parents in the workplace, or younger infants are carried by an adult worker.

d. EPA failed to calculate children's chronic exposure and risk.

EPA ignores chronic risk to children, its rationale being that chronic exposure to children is "unlikely":

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[113] U.S. EPA, 1-BP Supplemental File Supplemental Information on Occupational Exposure Assessment p. 28-29 (Aug. 2019), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0235-0014.

[114] CSAC Report on 1-BP Risk Assessment, p. 28.

> EPA did not calculate chronic and cancer risks for children at dry cleaners because EPA believes exposure to children at workplaces are unlikely to be chronic in nature. (p. 92)

EPA provides no data or evidence to support its sweeping conclusion that children at dry cleaning facilities would not be exposed chronically. This assumption is contrary to the logical conclusion that some children at family-owned dry cleaning facilities would be exposed day in and day out throughout their entire childhood – particularly those living in co-located facilities. This readily foreseen scenario would lead to years of exposure, which fits squarely within the various definitions of chronic exposure used by the EPA.

The Exposure Factors Handbook defines chronic exposure as, "[r]epeated exposure by the oral, dermal, or inhalation route for more than approximately 10% of the life span in humans (more than approximately 90 days to 2 years in typically used laboratory animal species)."[115] Assuming a child of a family with a dry cleaning facility is exposed until the age of 18 years, they would be exposed for well over 10% of their life assuming they reach an average age of an American (78.7 years). The definition in EPA's Benchmark Dose Software (BMDS) glossary of terms for chronic exposure indicates that exposures as short as six months would fit the definition: "[l]ong-term exposure usually lasting 6 months to a lifetime."[116]

In short, EPA completely ignores chronic risks to children with a flawed and unsupported assertion that children in dry cleaning facilities would not be exposed on a chronic basis – and despite the CSAC's explicit recommendation that the agency consider risks to children in just such scenarios (see Part I, sec. 5.B.xii.a. above).

e. EPA fails to consider children's exposure during spot cleaning.

While EPA recognizes that children may be present in dry cleaning facilities, it fails to consider all conditions of use relevant in this setting. Specifically, exposure to children during spot cleaning, an industrial/commercial condition of use also associated with dry cleaners, is completely ignored. EPA should also consider children as bystanders for this condition of use.

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[115] U.S. EPA, EXPOSURE FACTORS HANDBOOK p. 1427 (2011 ed.), https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=236252.
[116] BENCHMARK DOSE SOFTWARE (BMDS) GLOSSARY OF TERMS, https://ofmpub.epa.gov/sor_internet/registry/termreg/searchandretrieve/glossariesandkeywordlists/search.do?details=&vocabName=BMDS%20Glossary (last visited Sept. 27, 2019).

f. EPA's treatment of risks to children lacks transparency.

In addition to the numerous substantive issues identified above, EPA's limited consideration of risks to children lacks transparency. In numerous places throughout the draft risk evaluation, it appears that EPA did not consider infants or children as vulnerable subpopulations for any industrial/commercial conditions of use. For example, both the Executive Summary and Section 2.4 on Potentially Exposed or Susceptible Subpopulations state, without mention of children:

> [f]or occupational exposures, EPA assessed exposures to workers and ONUs from all 1-BP conditions of use" (p. 22, p. 135).

In both of these sections, EPA only refers to considering infants and children in the context of consumer exposures (p. 23, p. 137).

Yet hidden on p. 52 is the first reference to EPA's analysis of children's exposures from dry cleaning: "EPA also considers exposure to children who may be present at the workplace, such as small family-owned dry cleaners" (p. 52). Rather than provide an analysis, however, EPA then disregards *all* children's chronic exposures at dry cleaning facilities, noting it in a cursory footnote in the middle of the draft risk evaluation (p. 92) – and provides only limited additional rationale for dismissing acute exposures (p. 185 and p. 194). Relegating the entire analysis for children's chronic exposures to dry cleaning operations to a footnote lacks transparency, and must be corrected in the final risk evaluation.

xiii. EPA has inadequately assessed consumer exposures to insulation.

EPA's analysis of consumer exposures to insulation raises a number of concerns: (1) EPA did not conduct a chronic exposure analysis; (2) EPA assumed insulation is not present in living areas; and (3) EPA failed to consider exposures associated with insulation in basements.

First, EPA failed to conduct an analysis of chronic consumer exposures to 1-BP in residential insulation. EPA acknowledged that, "[u]nlike other 1-BP sources (e.g., liquid and aerosol sprays) summarized above, which cause short-term, high-level exposures (evaluated for acute exposure only), [ ] insulation causes long-term, low-level exposure after the initial spike in concentration upon installation" (p. 130). EPA then claimed to capture that risk by calculating acute inhalation exposures.

However, according to EPA's Exposure Factors Handbook chronic exposure is defined as, "[r]epeated exposure by the oral, dermal, or inhalation route for more than approximately 10% of the life span in humans (more than approximately 90 days to 2 years in typically used laboratory

animal species)."[117] And EPA's Benchmark Dose Software (BMDS) glossary of terms defines chronic exposure as "[l]ong-term exposure usually lasting 6 months to a lifetime."[118] Based on EPA's graph on p. 131, 1-BP from use in insulation is expected to be present in living areas at above 2 µg/m3 for almost 150 days, and will persist in the living area well beyond 400 days. Considering these concentrations of 1-BP in the living area, it is unclear, and EPA must explain, why EPA did not consider insulation exposures to consumers as chronic in nature.

EPA also assumed that living areas are not insulated (p. 121), which resulted in EPA calculating very high margins of exposure for consumer exposures in living areas (p. 205, Table 4-26). There are a number of concerns with this assumption, however:

- EPA assumes that living areas do not have insulation based on a study of *spray* insulation (p. 121).[119] Currently, EPA's condition of use for 1-BP is only for rigid board insulation, so it remains unclear how relevant use patterns for spray foam insulation would be to the use of insulated boards (p. 122).
- The U.S. Department of Energy specifically recommends that a home should be insulated from "the roof down to its foundation."[120]

EPA has provided no rationale for assuming insulation is not used in the living area of a home, an analysis it must conduct for the final risk evaluation.

Lastly, the SACC noted that EPA did not analyze consumer exposures in houses with basements containing insulation made with 1-BP. According to the National Association of Homebuilders (based on data collected by the US Census Bureau), the majority of *new* homes in the U.S. are built with basements.[121] It appears that EPA relied on the same spray foam insulation study – which it has not made publicly available – to select the three zones it considered (p. 121), as it provided no additional rationale. EPA must include an analysis of basements, *as a living area*,

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[117] U.S. EPA, EXPOSURE FACTORS HANDBOOK p. 1427 (2011 ed.), https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=236252.
[118] BENCHMARK DOSE SOFTWARE (BMDS) GLOSSARY OF TERMS, https://ofmpub.epa.gov/sor_internet/registry/termreg/searchandretrieve/glossariesandkeywordlists/search.do?details=&vocabName=BMDS%20Glossary (last visited Sept. 27, 2019).
[119] Notably, this study is not publicly available, EPA cites to a page that indicates the study is available for $89: https://www.astm.org/DIGITAL_LIBRARY/STP/SOURCE_PAGES/STP1589.htm.
[120] Department of Energy, Where to Insulate in a Home, https://www.energy.gov/energysaver/weatherize/insulation/where-insulate-home (last visited Sept. 30, 2019).
[121] Natalia Siniavskaia, *What Foundations are Built Across the Nation?*, NAHB Blog (Oct. 2014), http://eyeonhousing.org/2014/10/what-foundations-are-built-across-the-nation/.

with 1-BP insulation in the final risk evaluation, or provide a convincing rationale for not doing so.

6. Risk characterizations

A. EPA's unwarranted assumption of PPE use obscures the full extent of unreasonable risk posed by 1-BP.

EPA's presentation of its risk determinations in Table 5-1 (pp. 260-289) dramatically understates the extent of actual unreasonable risk it has identified.

As discussed elsewhere in the comments (Part I, secs. 1.B., 7.A.), EPA has adopted a legally and scientifically flawed assumption – absent any empirical evidence to support it – that workers throughout chemical supply chains will always wear effective personal protective equipment (PPE).

EPA's application of this assumption to various subpopulations under the various conditions of use is largely masked by its presentation, but our analysis demonstrates that this assumption is the key driver of EPA's risk determinations – both in cases where EPA did find unreasonable risk and in cases where it did not.

By poring through the dozens of detailed tables in the bowels of its draft risk evaluation, we were able to discern the levels of risk EPA found for various conditions of use of 1-BP. These tables show: 1) the risk levels EPA calculated *before* it applied its assumption regarding PPE use; then 2) whether EPA's assumption of PPE could make enough risk go away so that EPA could claim there is no unreasonable risk; and 3) if so, what degree of efficiency of respirators or gloves EPA had to assume would be used.

Our examination revealed the following:

- There are only two kinds of scenarios under which EPA did find unreasonable risk:
    - **Scenarios where the risks EPA calculated are so high that it could not make them go away even *after* assuming that workers would always use the most protective level of PPE that EPA considered**. For inhalation exposures, this would require use of a highly efficient (and highly cumbersome) respirator with an "assigned protection factor" (APF) of 50, i.e., one that reduces air concentrations by 50-fold. For dermal exposures, this would require use of highly impermeable gloves that EPA assumed would provide a "protection factor" (PF) of 20, i.e., they would reduce skin contact concentrations by 20-fold.
    - **Scenarios where EPA could not plausibly assume *any* use of PPE by the exposed persons**. These include consumers and bystanders; workers and

bystanders at businesses like dry cleaners; and so-called "occupational non-users" – workers not directly handling the chemical.

- With one exception, for all conditions of use where EPA found there was not unreasonable risk, in order to reach that finding, **EPA had to assume that all workers were using *both* respirators and gloves**. In other words, any worker not using both a respirator and gloves would face an unreasonable risk from inhalation or dermal exposure, based on EPA's own analysis.
    - The exception is consumer exposure to installed insulation, where bizarrely EPA only considered acute, not chronic, exposure.

EPA's presentations of its risk determinations in Table 5-1 identify either the "exposure scenario with highest risk estimate" or the "unreasonable risk driver," i.e., the exposure and endpoint that EPA identified as posing the highest risk. Where EPA could assume that PPE would be used and be sufficient to reduce that risk to below its assumed "safe" level, EPA found there was no unreasonable risk; where even that assumption did not suffice, EPA was compelled to find there was unreasonable risk.

But EPA's presentations obscure an important fact: For those conditions of use where PPE might plausibly be used, in virtually every case there were *additional* exposures that also yielded risks exceeding EPA's "safe" levels unless EPA's also assumed PPE use to address them.

More specifically:

- Wherever EPA found that use of a respirator was sufficient to reduce the highest identified risk to a level it deemed "reasonable," buried in the details is the fact that it *also* had to assume gloves would be used in order to sufficiently reduce other identified risks that exceeded its "safe" levels. And the converse was true: Where a dermal exposure posed the highest risk, there were excessive inhalation risks as well; only by assuming use of both respirators and gloves could EPA find there was no unreasonable risk.
- Where EPA found that use of a respirator was not sufficient to reduce the highest identified risk to a level it deemed "reasonable," it found unreasonable risk. But buried in the details in those cases as well is the fact that it *also* had to assume gloves would be used in order to sufficiently reduce other identified risks that exceeded its "safe" levels. And the converse was true: Where a dermal exposure posed the highest risk, even if assumed PPE use could suffice to address that risk, there were excessive inhalation risks as well; only by assuming use of a respirator could EPA find the inhalation exposure did not present an unreasonable risk.

The table below shows the full extent to which EPA's risk determinations are dependent on its assumptions about PPE use: For every condition of use where PPE might plausibly be used,

EPA avoided identifying an unreasonable risk from inhalation or dermal exposure, or both, ***only*** by assuming the use of PPE.

For *both* the "unreasonable risk" and "no unreasonable risk" determinations, EPA's unwarranted approach raises major policy concerns. If EPA's PPE assumptions erase unreasonable risks, then EPA will not regulate the chemical under TSCA and will forgo its only opportunity to ensure that PPE is actually used. If EPA does find unreasonable risk even with its PPE assumptions, by understating the magnitude of that risk, any subsequent regulation EPA promulgates under TSCA will be under-protective.

As described elsewhere in these comments (Part I, secs. 1.B., 7.A.), not only does EPA lack empirical data on the extent and effectiveness of PPE use; it relies instead on assertions volunteered by companies making or using the chemical – and even this anecdotal, undocumented information is germane to only a small subset of its conditions of use. EPA also grossly misrepresents the authority of the Occupational Health and Safety Administration (OSHA) and falsely implies that PPE use is mandatory under OSHA regulations when it is not. EPA also ignores numerous reports – specific to 1-BP – of PPE being used improperly or not at all, in some cases resulting in severe worker injuries.

Table: Summary Analysis of PPE Dependencies for 1-BP Risk Determinations

Page	Condition of use	Risk driver(s) identified by EPA	EPA's Determination[122]	Is it dependent on PPE?[123]
260	Domestic manufacture	Cancer (chronic), non-cancer (devtox, acute) via inhalation	No UR	Inhalation: Dependent on PPE (APF=10), Dermal: Dependent on PPE (PF=5)
261	Import	Cancer (chronic) via dermal	No UR	Dermal: Dependent on PPE (PF=5), Inhalation: Dependent on PPE (APF=10)
262	Processing as a reactant	Cancer (chronic) via dermal	No UR	Dermal: Dependent on PPE (PF=5), Inhalation: Dependent on PPE (APF=10)
263	Processing into formulation, mixture or reaction product	Cancer (chronic) via inhalation, devtox (acute, chronic) via inhalation	UR	Inhalation: PPE (APF=50) not sufficient, Dermal: Dependent on PPE (PF=5)
264	Processing into articles	Cancer (chronic) via dermal	No UR	Dermal: Dependent on PPE (PF=5), Inhalation: Dependent on PPE (APF=10)
265	Processing repackaging	Cancer (chronic) via dermal	No UR	Dermal: Dependent on PPE (PF=5), Inhalation: Dependent on PPE (APF=10)

[122] UR = "unreasonable risk"

[123] The first listed PPE in this column was flagged in EPA's risk determination tables as necessary to support its finding based on the risk driver it identified (*see* column 3). However, EPA also assumed the use of additional PPE in order to address other excessive risks it identified beyond the risk driver; these are also indicated in this column.

Page	Condition of use	Risk driver(s) identified by EPA	EPA's Determination[122]	Is it dependent on PPE?[123]
266	Processing recycling	Cancer (chronic) via dermal	No UR	Dermal: Dependent on PPE (PF=5), Inhalation: Dependent on PPE (APF=10)
267	Distribution	N/A (considered in individual COUs)	No UR	N/A
267	Ind/comm use: solvent, vapor degreaser open/inline	Devtox (acute) via inhalation, cancer (chronic) via inhalation	UR	Inhalation: PPE (APF=50) not sufficient, Dermal: Dependent on PPE (PF=5)
269	Ind/comm use: solvent, vapor degreaser closed loop	Devtox (acute) via inhalation, cancer (chronic) via inhalation	UR for ONUs only	Inhalation: Respirators not assumed for ONUs; for workers, Dependent on PPE (APF=10), Dermal: for workers, Dependent on PPE (PF=5)
270	Ind/comm use: solvent, cold cleaning	Devtox (acute/chronic) via inhalation, cancer (chronic) via inhalation	UR for workers and ONUs	Inhalation: PPE (APF=50) not sufficient, Dermal: Dependent on PPE (PF=5)
271	Ind/comm use: solvent, aerosol spray degreaser	Devtox (acute/chronic) via inhalation, cancer (chronic) via inhalation	UR for workers and ONUs	Inhalation: PPE (APF=50) not sufficient, Dermal: Dependent on PPE (PF=5)
273	Ind/comm use: adhesives & sealants spray for foam	Devtox (acute and chronic) via inhalation, cancer (chronic) via inhalation	UR for workers and ONUs	Inhalation: PPE (APF=50) not sufficient, Dermal: Dependent on PPE (PF=5)
275	Ind/comm use: cleaning & furniture care dry cleaning	Devtox (acute/chronic) via inhalation, cancer (chronic) via inhalation	UR for workers and ONUs	Inhalation: Respirators not assumed for workers or ONUs, Dermal: Dependent on PPE (PF=5)
276	Ind/comm use: cleaning & furniture care spot cleaner stain remover	Devtox (acute/chronic) via inhalation, cancer (chronic) via inhalation	UR for workers and ONUs	Inhalation: Respirators not assumed for workers or ONUs, Dermal: Dependent on PPE (PF=5)
278	Ind/comm use: cleaning & furniture care liquid cleaner, arts/crafts, etc.	Devtox (acute/chronic) via inhalation, cancer (chronic) via inhalation	UR for workers and ONUs	Inhalation: Respirators not assumed for workers or ONUs, Dermal: Dependent on PPE (PF=5)
280	Cons. use: solvent aerosol spray	Devtox (acute) via inhalation	UR for consumers and bystanders	N/A (dermal risk not addressed)

Page	Condition of use	Risk driver(s) identified by EPA	EPA's Determination[122]	Is it dependent on PPE?[123]
281	Cons. use: spot cleaner stain remover	Devtox (acute) via inhalation	UR for consumers and bystanders	N/A (dermal risk not addressed)
281	Cons. use: liquid cleaner	Devtox (acute) via inhalation	UR for consumers and bystanders	N/A (dermal risk also deemed excessive)
283	Cons. use: liquid spray/ aerosol cleaner	Devtox (acute) via inhalation	UR for consumers and bystanders	N/A (dermal risk not addressed)
284	Cons. use: arts/crafts, adhesive accelerant	Devtox (acute) via inhalation	UR for consumers and bystanders	N/A (dermal risk not addressed)
285	Cons. use: automotive care refrigerant flush	Devtox (acute) via inhalation and dermal	UR for consumers and bystanders	N/A
286	Cons. use: anti-adhesive agents - mold cleaning and release product	Devtox (acute) via inhalation	UR for consumers and bystanders	N/A (dermal risk not addressed)
287	Cons. use: Building/construction materials	Devtox (acute) via inhalation	No UR for consumers and bystanders	N/A (dermal risk not addressed)
288	Disposal	Cancer (chronic) via dermal	No UR	Dermal: Dependent on PPE (PF=5), Inhalation: Dependent on PPE (APF=10)

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

As noted above, Part I, section 1.B., EPA does not appear to have data on glove use and efficacy that is necessary to estimate dermal exposure. EPA states:

> EPA does not know the actual frequency, type, and effectiveness of glove use in specific workplaces with 1-BP conditions of use. (p.108)

Also as noted above, EPA acknowledges in the draft risk evaluation that gloves are likely to provide only limited protection from 1-BP, citing a 2013 OSHA Hazard Alert:

> 1-BP easily travels through most glove materials. Recommended glove materials for protection against 1-BP are supported polyvinyl alcohol or multiple-layer laminates. (p. 106)

Furthermore, EPA acknowledges that use of gloves can actually *increase* skin exposure through occlusion. For example, EPA states:

> Where exposure is non-occluded, only a fraction of 1-BP that comes into contact with the skin will be absorbed as the chemical readily evaporates from the skin. However, dermal exposure may be significant in cases of occluded exposure, repeated contacts, or dermal immersion. For example, work activities with a high degree of splash potential may result in 1-BP liquids trapped inside the gloves, inhibiting the evaporation of 1-BP and increasing the exposure duration. (p. 106)

Despite acknowledging all of these critical issues, the agency simply uses default glove protection factors and ignores dermal exposure in occluded scenarios for workers. The agency assumes fixed protection factors (PFs) of 5x, 10x, and 20x, which do not appear to be supported by any empirical data that account for the complexities of glove use in the real world. As noted in Part I, section 6.A., the vast majority of "no unreasonable risk determinations" rely on a glove protection factor of 5x for workers.

During the 1,4-dioxane SAAC peer review meeting, one SACC member who is a dermal exposure expert expressed this concern. He noted that glove testing is typically conducted in a lab under ideal conditions – without an actual human hand present. However, in the real world, an insufficiently trained or attentive user may contaminate a glove, leading to occlusion and higher exposure. Likewise, permeable gloves may enable the chemical to absorb through the glove, while preventing or slowing evaporation. Gloves can also increase skin temperature and humidity, which can increase absorption. Therefore, the assumption that PFs can only range as low as 1x (no gloves) is erroneous; rather, the range should include PFs below 1x. Notably, this same SACC member stated that EPA's glove protection factor assumptions may be even less supportable in the case of 1-BP, given that "only a relatively sophisticated glove will stop 1-BP."

EPA's apparent decision to ultimately ignore risk from occluded scenarios in the workplace is particularly troubling. EPA states that it considered occluded scenarios for workers (p. 107) and it appears that the agency did in fact calculate occluded dermal risk for several conditions of use. EPA's 1-BP Supplemental File Occupational Risk Calculator[124] shows that these risks are actually quite large: For example, the dermal cancer risk for Degreasing and Cold Cleaning is $8.3x10^{-2}$ – or a cancer risk level of nearly *1 in 10* workers. Yet it appears that the risk estimates under occluded conditions are not actually incorporated into the Risk Characterization and Risk Determinations in the draft risk evaluation at all. For example, when one compares Tables 4-48 through 4-53 in the draft risk evaluation (pp. 232-237) to the corresponding tables in the Supplemental File Occupational Risk Calculator (under the dermal tab), they are identical with the exception that all of the columns for occluded exposures have been removed in the versions of the tables in the draft risk evaluation.

[124] 1-BP Supplemental File Occupational Risk Calculator, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0235-0020 (Dermal tab in excel file).

EPA appears to want to have it both ways: To acknowledge the limitations of gloves and their potential to increase skin absorption, but then to simply assume that gloves actually provide 5x, 10x or 20x levels of protection over no gloves – regardless of the potential for occlusion – without citing any evidence to support these values. Further, the agency fails to acknowledge all the uncertainties and deficiencies with its glove use assumptions in the Risk Determination section of the evaluation. 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 EPA can conclude 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), or when there is occlusion, scenarios quite likely – and certainly reasonably foreseen – to occur in the real world.

C. EPA's approaches to both aggregate and sentinel exposures are flawed.

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 aggregate and sentinel exposure; indeed, EPA's discussion of these exposure approaches leaves it unclear whether EPA believes that it relied on aggregate exposure, sentinel exposure, or both.

EPA asserts that it "considered aggregate exposures by evaluating exposure and risk from both inhalation and dermal routes for workers and consumers in scenarios where such exposures are expected" (p. 245). But while EPA considered both inhalation and dermal routes, there is no evidence in the draft risk evaluation that EPA "combined" these exposures when assessing the risks each condition of use presented. *But see* 40 C.F.R. § 702.33 (defining "[a]ggregate exposure" to "mean[] the *combined* exposures to an individual from a single chemical substance across multiple routes and across multiple pathways.") (emphasis added). Instead, in the risk characterization section of the draft risk evaluation, EPA analyzes risk from inhalation exposures and then separately analyzes risk from dermal exposures; EPA never provides any description or analysis that *combines* these exposures to assess total exposure and determine whether it presents a risk. Therefore, it appears that EPA never performed an actual aggregate exposure assessment.

EPA's failure to combine the exposures also affects its tables setting forth its risk determinations (Table 5-1, pp. 260-289). For several conditions of use, EPA identifies the "exposure scenario with [the] highest risk estimate" as "chronic inhalation exposure" (p. 260), "acute inhalation exposure" (p. 287), or "chronic dermal exposure" (pp. 261-62, 264-66, 288). But for many of these conditions of use, it is likely that the exposure scenario with the highest risk estimate would actually be inhalation and dermal exposure "combined." But EPA never analyzes the exposures from these routes in combination or explains why they should not be combined.

This approach has real consequences for EPA's analyses, particularly when combined with EPA's (indefensible) assumption of PPE use and efficacy. For example, for manufacturing, EPA finds that the exposure scenario with the *highest* risk estimate is that of workers faced with developmental adverse effects from acute inhalation exposure and cancer resulting from chronic inhalation exposure (p. 260). EPA then assumes away those risks from inhalation based on an assumption that PPE (respirators) will eliminate the risks (p. 260). But EPA *also* found that dermal exposures posed risks greater than its benchmark, and then also assumes away those risks from dermal exposure, based on another assumption that PPE (gloves) will also eliminate the risks (p. 237). However, throughout all of this, EPA never addresses the potential for the risks from inhalation combined with the risks from dermal exposure to present an unreasonable risk – even with its dual unfounded assumptions of universal, effective use of PPE. In sum, even assuming PPE is used, EPA has provided no basis whatsoever for concluding that the combined risks from dermal and inhalation exposures will be addressed.

EPA also asserts that it "considered sentinel exposures to populations who may have upper bound exposures *** EPA characterized high-end exposures in evaluating exposure using both monitoring data and modeling approaches" (p. 254). 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. EPA has not established that its "high-end" exposure assessment represents the "plausible upper bound of exposure relative to all other exposures" within the relevant categories. The regulatory definition requires that, when EPA identifies 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. EPA relies on an unsupportable expectation of compliance with existing laws and standards.

In reaching its unreasonable risk determinations, "EPA expects there is compliance with federal and state laws, such as worker protection standards, unless case-specific facts indicate otherwise" (p. 289, n. 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." (p. 289, n. 1). As noted in Part I, section 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 standards, or that even when complied with, such requirements eliminate all risk such that EPA can ignore the contribution of remaining risks from such regulated activities to the overall risks posed by 1-BP. 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 account for 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 requirements it relies on derive from statutes that establish criteria different than those under TSCA 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. EPA appropriately acknowledged that exceedances for high-end risks should result in a finding of no unreasonable risk.

In its draft risk evaluation for 1,4-dioxane, EPA unjustifiably decided that even if it found excessive risks in some cases for high-end exposures, it still determined that the risk was not unreasonable as long as the risks of central tendency exposures did not exceed its benchmarks. It appears EPA has not applied this flawed approach in the 1-BP draft risk evaluation, and EPA must ensure that it does not do so in the final risk evaluation.

Among other concerns, EPA's approach for 1,4-dioxane is at odds with its obligation under TSCA to conduct risk evaluations that ensure protection of "potentially exposed or susceptible subpopulations," which TSCA explicitly defines as including workers. EPA represents its high-end estimates as "generally intended to cover individuals or sub-populations with greater exposure," while its central tendency estimates apply to the "average or typical exposure" that people experience (p. 256). TSCA would not permit EPA to protect against only the "average or typical exposure;" in fact, when it comes to workers and other "potentially exposed or susceptible subpopulations," EPA is required to protect all of them.

Moreover, EPA stated that it considered sentinel exposures, 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. 254). In its 1,4-dioxane draft risk evaluation, EPA took the wholly unjustifiable approach of finding a risk to be unreasonable only if the risk from *both* the high-end and the central tendency exposures exceeded its acceptable risk levels. In contrast, with 1-BP EPA takes the far more justifiable approach of finding a risk to be unreasonable even when the risks from *only* the high-end exposure exceed its acceptable risk levels. That approach is necessary to ensure that those experiencing high-end, i.e., sentinel, exposures will always be protected. For EPA not to do so would be inconsistent with its own definition of sentinel exposure in the risk evaluation rule. *See* 40 C.F.R. § 702.33.

C. EPA's use of a 1 in 10,000 cancer risk level as reasonable for workers is deeply flawed.

EPA has relied on NIOSH guidance in order to establish 1×10^{-4} as the cancer risk benchmark for workers (p. 257). While EPA does not cite the *Benzene* decision for support as it did in the 1,4-dioxane draft risk evaluation, EPA still cites "case law" and the NIOSH chemical carcinogen policy – both of which derive from the OSH Act – as the rationale for using this very high cancer risk benchmark. EPA is effectively still relying on the *Benzene* decision, which was reached in a case that pertained to how the standard for protection applied under OSHA, not under TSCA. EPA's decision is wholly at odds with its own acknowledgment two pages earlier that other laws have standards that differ from TSCA's (p. 255, n. 28).

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. 257, n. 29).

Indeed, EPA's approach 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 OSHA that defined standards as "reasonably necessary or appropriate to provide safe or healthful employment and places of employment," as requiring OSHA "to make a threshold finding that a place of employment is unsafe—in the sense that *significant* risks are

present and can be eliminated or lessened by a change in practices." *Indus. Union Dep't, AFL-CIO v. API*, 448 U.S. 607, 642 (D.C. Cir. 1980) (emphasis added). The Court's interpretation turned on the statutory language of the OSH Act, the Act's structure, and its legislative history. But EPA can point to no statutory language in TSCA invoking this standard, EPA has pointed to no similarities between the two statute's structures, nor has EPA pointed to any legislative history suggesting that TSCA adopted the OSH Act's standard. Moreover, if Congress had intended to adopt the *Benzene* standard under TSCA, it would have required that EPA regulate "significant risks," not "unreasonable risks." Indeed, the significant differences between the language and structure of the two statutes strongly indicates that Congress meant to adopt a different standard in TSCA, not the standard articulated by the Court in the *Benzene* case.

Moreover, in implementing TSCA (even before the amendments) and its other environmental statutes, EPA has generally sought to reduce population risks from chemicals in commerce that are carcinogens to below about one case per one million people. See, for example, this EPA statement from 1989: "EPA believes *** that it should reduce risks to less than 1 x 10-6 for as many exposed people as reasonably possible." National Emission Standards for Hazardous Air Pollutants; Radionuclides, 54 Fed. Reg. 51,654, 51,686 (Dec. 15, 1989). Nor does EPA only apply this standard under the Clean Air Act. When setting Clean Water Act criteria, "EPA intends to use the 10-6 risk level, which the Agency believes reflects an appropriate risk for the general population. EPA's program office guidance and regulatory actions have evolved in recent years to target a 10^{-6} risk level as an appropriate risk for the general population. EPA has recently reviewed the policies and regulatory language of other Agency mandates (e.g., the Clean Air Act Amendments of 1990, the Food Quality Protection Act) and believes the target of a 10^{-6} risk level is consistent with Agency-wide practice."[125] When Congress amended TSCA to retain, with significant contextual modifications, the unreasonable risk standard, it did so knowing that agency practice was to regulate cancer risks at the 10^{-6} risk level. It should be presumed that Congress meant to adopt this risk standard when codifying the unreasonable risk standard.

In grasping for support for its approach in this risk evaluation by citing other mentions by EPA of the 1 x 10^{-4} risk level (p. 257, n. 29), EPA blurs a critical distinction made on the rare occasions 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.

[125] 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.

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."
>
> 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*.[126]

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 a number of conditions of use of 1-BP 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. EPA's assumption of PPE use, together with its use of a 1-in-10,000 acceptable risk levels for workers, conflates risk evaluation and risk management and significantly understates risk.

TSCA intentionally divides risk evaluation and risk management into two distinct processes, whereby regulatory measures are to be considered *after* EPA finds an unreasonable risk. However, by choosing to make risk determinations based on an assumption of universal, effective use of PPE, EPA conflates risk evaluation and risk management and leads EPA either not to find unreasonable risk or to underestimate the magnitude of that risk in a number of scenarios – thereby denying itself the authority to impose mandatory requirements sufficient to control workplace exposures (see Part I, sec. 6.A.).

For example, Table 4-53 (p. 237) demonstrates that for cancer risk from dermal exposure, EPA has actually found excessive risk in ***every*** occupational scenario it examined – even using its very permissive 1 in 10,000 benchmark (see slide 1 below). Yet, when it comes to the risk determinations, EPA finds no unreasonable risk in several of these scenarios, including manufacturing (import) by stating "there is no unreasonable risk when PPE (gloves PF=5) are

[126] 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 Sept. 30, 2019).

used." (p. 261) EPA's failure to make an unreasonable risk determination will mean it will then lack any authority to require that the gloves it assumed will be used are actually used.

Slide 1: Assumption of PPE

Table 4-53. Cancer Risk Estimates for Dermal Exposure Following Occupational Use of 1-BP

Category	Dermal Slope Factor (mg/kg-day)$^{-1}$	No Gloves (PF = 1)	Protective Gloves (PF = 5)	Protective Gloves (PF = 10)	Protective Gloves (PF = 20)	Benchmark
Bin 1: Manufacture, Import, Processing, and Disposal	0.006	1.38E-04	2.77E-05	1.38E-05	6.91E-06	1E-04
Bin 2: Vapor Degreaser, Cold Cleaner		1.34E-04	2.68E-05	1.34E-05	6.71E-06	1E-04
Bin 3: Spray Adhesives		1.11E-04	2.21E-05	1.11E-05	N/A	1E-04
Bin 4: Dry Cleaning, Spot Cleaning		1.30E-04	2.60E-05	1.30E-05	N/A	1E-04
Bin 5: Aerosol Spray Degreaser/Cleaner, Other Aerosol and Non-aerosol Uses		1.38E-04	2.77E-05	1.38E-05	N/A	1E-04

(Page 237)

Manufacturing (import) risk determination:

- "**Does not present an unreasonable risk** of injury to health (workers and occupational non-users"
- Risk estimate: Workers: Dermal: 1.38E-04 for workers using no PPE (Table 4-53). Note: **There is no unreasonable risk when PPE (gloves PF=5) are used**.

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This same example demonstrates the ramifications of EPA's decision to understate risk by using the 1 in 10,000 benchmark. If EPA had used, say, even 1 in 100,000, the table shows it would have found excessive risk in every occupational scenario even assuming gloves with a PF up to 10. See slide 2 below (blue circles).

Using EPA's longstanding policy of aiming to reduce risks for as many exposed people as possible to less than 1 in a million, EPA would have found excessive risk in ***every single*** occupational scenario – even if gloves with a PF=20 were used. See slide 2 below (purple circles).

Slide 2: 1 x 10^{-6} Benchmark

Table 4-53. Cancer Risk Estimates for Dermal Exposure Following Occupational Use of 1-BP

Category	Dermal Slope Factor $(mg/kg\text{-}day)^{-1}$	No Gloves (PF = 1)	Protective Gloves (PF = 5)	Protective Gloves (PF = 10)	Protective Gloves (PF = 20)	Benchmark
Bin 1: Manufacture, Import, Processing, and Disposal	0.006	1.38E-04	2.77E-05	1.38E-05	6.91E-06	1E-04
Bin 2: Vapor Degreaser, Cold Cleaner		1.34E-04	2.68E-05	1.34E-05	6.71E-06	1E-04
Bin 3: Spray Adhesives		1.11E-04	2.21E-05	1.11E-05	N/A	1E-04
Bin 4: Dry Cleaning, Spot Cleaning		1.30E-04	2.60E-05	1.30E-05	N/A	1E-04
Bin 5: Aerosol Spray Degreaser/Cleaner, Other Aerosol and Non-aerosol Uses		1.38E-04	2.77E-05	1.38E-05	N/A	1E-04

These examples illustrate how hard EPA has had to work to avoided finding excessive cancer risk from dermal exposure in the occupational setting. In the draft risk evaluation, EPA has used these tactics so that it does not make a single unreasonable risk determination based on cancer risk from dermal exposure in the workplace.

8. Systematic review issues

A. OPPT provides neither explanation nor empirical support for its revisions to the systematic review data quality criteria for epidemiological studies, which make it impossible for epidemiological studies to be scored overall as high quality.

See our comments on this concern in Part I, sec. 5.B.i.b.

B. OPPT's approach taken to evidence integration in the draft 1-BP 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,[127] OPPT has not provided a pre-established methodology for its approach to evidence integration. This violates the agency's own

[127] Environmental Defense Fund Comments on Application of Systematic Review in TSCA Risk Evaluations (Aug. 16, 2018), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2018-

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 "narrative" (p. 159). 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:

> 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).[128]
>
> 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.[129]

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) (Jan. 14, 2019), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2018-0604-0013.

[128] 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).

[129] *Id.* at 6 (emphases added).

EPA's IRIS program reflects this NAS recommendation by developing problem formulation and assessment protocols for each of its assessments.[130] 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.

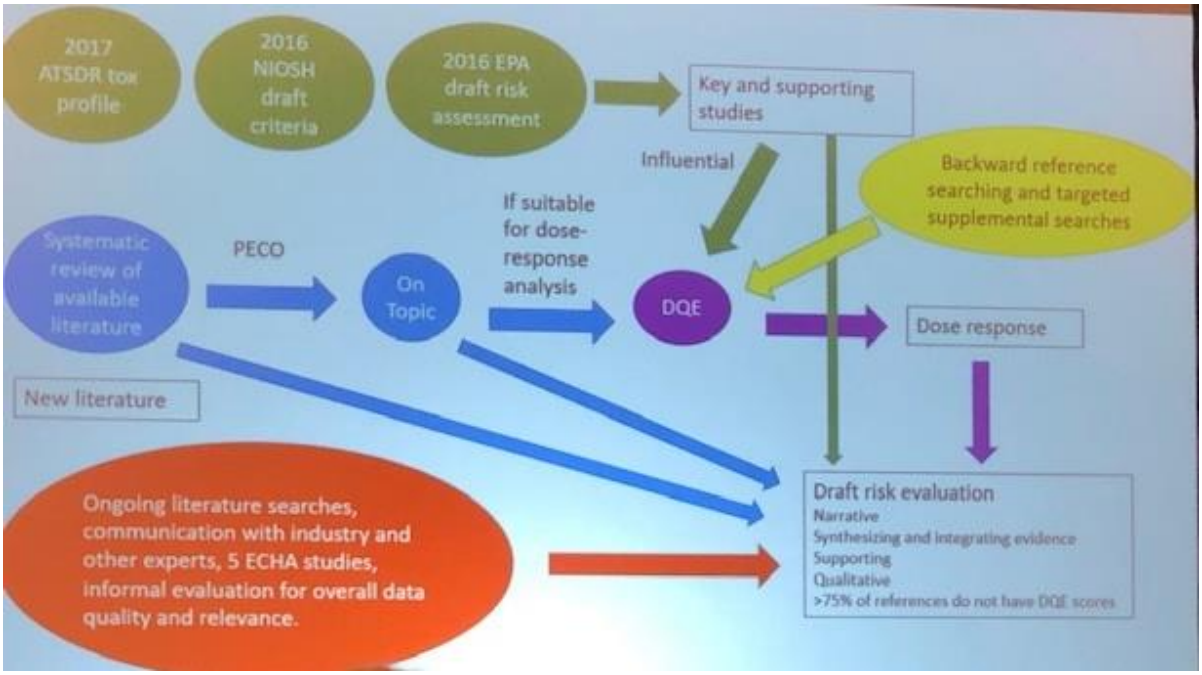
C. OPPT's inconsistent application of its systematic review criteria results in an arbitrary and capricious analysis.

EPA relies on some sources—for example, the summaries of studies available via ECHA (p. 138)—without evaluating them under its systematic review process. But EPA then excludes some other sources on the basis of its systematic review process. *See* pp.44-48 (charts showing EPA excluding some studies based on "unacceptable" findings through evaluation criteria). 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] scientific evidence" which states that evidence will be subject to systematic review. 40 C.F.R. § 702.33.

Only a quarter of the studies EPA relied on in the draft risk evaluation underwent data quality evaluation according to comments from one SACC member provided at the September 10-13, 2019, peer review meeting, who did a detailed review of EPA's application of the systematic review framework. This is despite the fact that EPA's stated goal at the SACC meeting was to ensure that *all* studies relied on the draft risk evaluation undergo data quality evaluation. The SACC member also provided a valuable flowchart of EPA's incorporation of evidence into the draft risk evaluation, which highlighted the volume of evidence that did not undergo data quality evaluation.

[130] 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.



D. EPA inappropriately excluded from systematic review several studies because they were in other languages and EPA had failed to request them.

EPA charged the SACC members with consideration of EPA's treatment of two studies available in foreign languages:

> Only a few environmental test data endpoints (including ECHA) are available in the public domain for 1-BP. Most are from the ECHA website. EPA attempted to obtain the full ECHA studies with no success. Since the studies were in French and Japanese (and no U.S.A. sponsor), EPA decided not to make further attempts to find the studies. Given that the ECHA environmental test data results are in the public domain, EPA decided to use the experimental data. Please comment on the reasonableness of this approach for the environmental hazard assessment of 1-BP.[131]

During the SACC meeting on 1-BP SACC members asked EPA about its policy regarding the treatment of studies that had not been translated into English. EPA responded that it was not EPA policy to exclude studies in foreign languages. EPA indicated that, based on the systematic review framework, EPA chose to focus on English reported studies first. Neither of these

[131] U.S. EPA, *EPA Scientific Advisory Committee On Chemicals Charge To The Panel – 1-Bromopropane (1-BP) CASRN: 106-94-5* at p. 6 (2019), https://www.epa.gov/sites/production/files/2019-08/documents/02._1-bp_draft_re_charge_questions_8_09_2019.pdf.

statements appear to be supported by EPA's systematic review framework or EPA's application of systematic review, which EPA must clarify to the SACC.

It is correct that EPA's "Application of Systematic Review" document does not ***directly*** address criteria for evaluating studies based on the language of the study. However, the systematic review document states, "[a] study is *disqualified* from further consideration if the confidence level of one or more metrics is rated as Unacceptable [score of 4]."[132]

Under the framework, EPA must evaluate each data source using four "data quality evaluation domains" which include: (1) reliability; (2) representativeness; (3) accessibility/clarity; (4) and variability and uncertainty.[133] EPA has defined the "accessibility/clarity" domain to simply mean whether "[t]he data and supporting information are accessible and clearly documented."[134]

In practice, it appears that EPA will disqualify studies in other languages as "inaccessible." For instance, in a supplemental file for the 1,4-dioxane draft risk evaluation, EPA graded a study as "unacceptable" on the basis that "[m]ost of paper is not in English; therefore, needed metadata are not provided."[135] EPA goes on to explain that "[c]onsistent with our Application of Systematic Review in TSCA Risk Evaluations document, if a metric for a data source receives a score of Unacceptable (score = 4), EPA will determine the study to be unacceptable. In this case, two of the metrics were rated as unacceptable. As such, the study is considered unacceptable and the score is presented solely to increase transparency."[136]

The treatment of these studies is concerning, as previously expressed by commenters on EPA's systematic review framework:

> The exclusive reliance on English-language studies may lead to under-representation of the entire body of available evidence, and studies have also suggested that language bias might lead to erroneous conclusions (46). Furthermore, when considering the inclusion or update of an existing systematic review, studies have found that language-inclusive systematic reviews (including studies in languages other than English) were of the highest quality, compared

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[132] U.S. EPA, *Application of Systematic Review In TSCA Risk Evaluations* p. 33 (May 2018), https://www.epa.gov/sites/production/files/2018-06/documents/final_application_of_sr_in_tsca_05-31-18.pdf (emphasis added).
[133] *Id.* at 66.
[134] *Id.*
[135] U.S. EPA, *Risk Evaluation for 1,4-Dioxane Systematic Review Supplemental File: Data Quality Evaluation of Environmental Releases and Occupational Exposure Data* p. 63 (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.
[136] *Id.* at 64.

> with other types of reviews (47). Online translation tools are readily available to allow screeners to quickly evaluate study abstracts for relevance, and therefore we recommend EPA to incorporate non-English language studies in their screening and not simply exclude in advance these potentially relevant papers.[137]

While it does not appear that EPA outright excluded any studies relevant to 1-BP based on its systematic review criteria, it is just as deeply concerning that EPA made no attempt to access the studies on 1-BP in French and Japanese based on a very similar rationale. EPA needs to clarify its position on foreign language studies to the SACC, as well as revisit its policy on labeling these studies as "inaccessible" and therefore disqualified from consideration. Considering the numerous data gaps on chemicals that EPA is currently reviewing and the dearth of data on most chemicals EPA will be reviewing in the future, the policy ensures EPA will not be relying on all reasonably available information as required by TSCA.

PART II

EDF previously provided comments on the scope and problem formulation for 1-BP.[138] In those comments, EDF identified a variety of legal violations and other problems with EPA's approach to the 1-BP 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 B and C. EPA should fix all of these problems in its final risk evaluation.

[137] Comments from Academics, Scientists and Clinicians on: The Application of Systematic Review in TSCA Risk Evaluations (Aug. 16, 2018), https://www.endocrine.org/-/media/endosociety/files/advocacy-and-outreach/society-letters/2018/2018-08-16-systematic-review-tsca-evaluations-ucsf-prhe-comments-epa.pdf?la=en.

[138] EDF Comments on Ten Scopes under the Toxic Substances Control Act, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0741-0059; 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-2016-0741-0085.

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-BP,[139] EPA stated that it had authority to exclude conditions of use. In the draft risk evaluation, EPA again relied on this assertion of authority. In our comments on the scope and the problem formulation, EDF explained that this approach is foreclosed under the statute, and EDF incorporates those arguments here.[140] Similarly, EDF incorporates the arguments presented in our Briefs attached as Appendix B at 21-40 and Appendix C at 14-31.

In the problem formulation, as well as in the draft risk evaluation (p. 27), EPA states that it will also exclude hazards and exposures under the condition of use as well. Specifically, in the draft risk evaluation EPA states that it will exclude all general population exposures to 1-BP through air, surface water, drinking water, and sediments (pp. 27, 258), and as a result the draft risk evaluation contains no analysis of risks to the general population (p. 259). EPA acknowledges that these exposures flow from conditions of use, including "industrial and/or commercial uses; industrial releases to air, water or land; and other conditions of use" (p. 259). EPA also effectively ignores certain hazards by completely failing to analyze environmental hazards to sediment-dwelling, terrestrial, or avian organisms (p. 138). EPA also effectively ignores certain hazards by failing to analyze: (1) cancer risks from acute exposures and (2) the unique hazards presented to certain potentially exposed or susceptible subpopulations in its risk analysis (p. 194, 253).

TSCA's language and structure unambiguously foreclose EPA's interpretation. EPA's decision to disregard certain exposure pathways and hazards is also "arbitrary, capricious, [or] an abuse of discretion" under the APA, 5 U.S.C. § 706(2)(A), because it will lead EPA to consider "factors which Congress has not intended it to consider [and] entirely fail[] to consider an important aspect of the problem." *Motor Vehicle Mfrs. Ass'n v. State Farm Mut. Auto. Ins. Co*., 463 U.S. 29, 43 (1983). Moreover, as the draft risk evaluation itself reveals, this approach leads to 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.

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[139] U.S. EPA, Problem Formulation of the Risk Evaluation for 1-Bromopropane (May 2018), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0741-0067 (hereinafter "Problem Formulation for 1-BP").

[140] EDF Comments on Ten Scopes under the Toxic Substances Control Act, pp. 4-11, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0741-0059; 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-2016-0741-0085.

EPA should evaluate all of the evidence of conditions of use, exposure, and hazard; not ignore evidence because of self-imposed blinders.

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

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

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

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

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

deadlines for completing evaluation for "a chemical substance"), (b)(2)(A), (b)(2)(B), (b)(3)(A), (c)(1).

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

All of these provisions direct EPA to consider a chemical's hazards, exposures, and conditions of use, and none of them include any language providing EPA with any discretion to ignore any hazards, exposures, or conditions of use. While EPA previously articulated a legal theory (albeit flawed) for ignoring certain conditions of use, EPA has not pointed to any legal basis for ignoring hazards or exposures under the conditions of use being analyzed in a risk evaluation. EPA has pointed to no textual basis for these exclusions.

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

> [EPA] shall consider and publish a statement based on reasonably available information with respect to—
> (i) the effects of the chemical substance or mixture on health and the magnitude of the exposure of human beings to the chemical substance or mixture;
> (ii) the effects of the chemical substance or mixture on the environment and the magnitude of the exposure of the environment to such substance or mixture;

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

S3517 (daily ed. June 7, 2016). Thus, EPA must evaluate all hazards and exposures in its risk evaluations.

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

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

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

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

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

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

Implicitly recognizing that Congress did not grant EPA boundless discretion to exclude exposures, EPA suggests that it will "focus its analytical efforts on exposures that are likely to present the greatest concern." *See, e.g.*, Problem Formulation for 1-BP at 13. 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.

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, sections 1.C. and 5, EPA's exclusions of certain exposures result in an incoherent draft risk evaluation where EPA acknowledges 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,[141] the legislative history as a whole does not justify EPA's approach to conditions of use, but here EPA has even less basis for its approach; EPA has not pointed to any statement in the legislative history supporting its approach of ignoring certain exposures or hazards.

B. 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 B & C. 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

[141] EDF Comments on Ten Scopes under the Toxic Substances Control Act, pp.7-8, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0741-0059.

hazards will be evaluated." 40 C.F.R. § 702.41(d)(3) (emphasis). Thus, EPA must consider any relevant "potential" hazards when preparing a risk evaluation. *See also* 40 C.F.R. § 702.41(d)(2) ("The hazard assessment process will identify the types of hazards to health or the environment posed by the chemical substance under the condition(s) of use within the scope of the risk evaluation."). The Rule also requires that: "[e]xposure information related to potential human health or ecological hazards of the chemical substance will be reviewed in a manner consistent with the description of best available science and weight of scientific evidence." 40 C.F.R. § 702.41(e)(3). When preparing the risk characterization, EPA shall "[t]ake into account, where relevant, the likely duration, intensity, frequency, and number of exposures under the condition(s) of use of the chemical substance." 40 C.F.R. § 702.43(a)(4). Thus, EPA must consider all hazards and all exposures under the conditions of use. None of these duties are qualified or provide an authority for EPA to exclude hazards or exposures from analysis.

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

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

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

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

Similarly, in the preamble, EPA states that "[u]sing reasonably available information, exposures will be estimated (usually quantitatively) for the identified conditions of use." 82 Fed. Reg. at 33,742. EPA cannot prepare an accurate quantitative estimate for exposure if EPA has excluded exposure pathways. "For environmental evaluations specifically, EPA plans to include a discussion of the nature and magnitude of the effects, the spatial and temporal patterns of the

effects, [and] implications at the species, population, and community level." 82 Fed. Reg. at 33,743. EPA cannot accurately discuss the magnitude of the effects on the environment or the spatial and temporal patterns of those effects if EPA ignores the vast majority of the environmental exposures, as EPA proposes to do.

Moreover, in the preamble to the rule, while EPA went to great lengths to describe its alleged discretion to pick-and-choose conditions of use, 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 "blinders" on regarding risks. The result is "arbitrary, capricious, [or] an abuse of discretion" under the APA, 5 U.S.C. § 706(2)(A), because it will lead EPA to have considered "factors which Congress has not intended it to consider [and] entirely failed to consider an important aspect of the problem." *State Farm*, 463 U.S. at 43. It also violates several provisions of TSCA § 26 because by ignoring uses, exposures, hazards, and related information, EPA will not be acting "consistent with the best available science," EPA will not base decisions on "on the weight of the scientific evidence," and EPA will not "take into consideration information relating to a chemical substance or mixture, including hazard and exposure information, under the conditions of use, that is reasonably available to the Administrator." 15 U.S.C. § 2625(h), (*i*), (k). In addition, because EPA's distinction is a false one untethered to the information, EPA seems to treat certain exposures inconsistently throughout the document.

For example, as detailed more below, early as well as later in the draft risk evaluation, EPA acknowledges that 1-BP exposures occur through numerous media (pp. 21, 258-59). But EPA then systematically excludes many of these pathways of exposure from its risk evaluation (pp. 27, 258-59), imposing arbitrary blinders on its analysis despite the factual evidence before it. This is the definition of arbitrary and capricious conduct.

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

D. EPA has provided insufficient justification for its exclusion of certain activities from the risk evaluation based on not being conditions of use or not being expected to occur.

In the problem formulation, EPA planned to exclude certain uses of 1-bromopropane (1-BP) from the risk evaluation by concluding the activities should not be considered conditions of use. EPA continued to exclude these activities in the draft risk evaluation (p. 19):

Agricultural non-pesticidal industrial/commercial/consumer use: EPA provides only a single statement with no relevant reference as the basis for this exclusion:

> Based on information available to EPA, EPA determined that 1-BP is not used in agricultural products (non-pesticidal), only in the processing of such products.

U.S. EPA, Problem Formulation of the Risk Evaluation for 1-Bromopropane at p. 19 (May 2018), https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0741-0067. The only source EPA cites (in Table 2-2) is the data EPA collected in 2016 under the Chemical Data Reporting (CDR) rule. This source indicates that a company in fact reported domestic manufacture of 1-BP for "industrial processing *and use*" in the "Pesticide, fertilizer, and other agricultural chemical manufacturing" sector, and further reported it accounted for 25% of its total production. Several questions remain:

First, EPA provides no indication of how it determined that 1-BP is "not used in agricultural products (non-pesticidal), only in the processing of such products." As written, this stands as a mere assertion by EPA. EPA has not provided additional information supporting this assertion in the draft risk evaluation.

Second, the "use" activity reported in the CDR is clearly a "condition of use" of 1-BP, which is reported as being manufactured for this very purpose, and EPA has no basis to exclude such a condition of use.

Third, while the CDR data indicate the chemical is an intermediate and processed as a reactant, EPA has not provided any data demonstrating unreacted 1-BP is not present in the final product as a residual.

Fourth, EPA has not clearly stated what specific activities are encompassed by its reference to 1-BP's use in the "processing *of* [agricultural products (non-pesticidal)]," which appears to be distinct from its use as a reactant to *make* such products. If EPA's reference to "processing" in this sector exclusively refers to 1-BP's use as a reactant, EPA needs to make that clear and document how it knows that is the only way in which 1-BP is "processed" in this sector, especially in light of the CDR's reference to its processing *and use* in the sector. If EPA's reference to "processing *of* agricultural products (non-pesticidal)" refers to and is intended to encompass activities beyond use as a reactant, these should be specified and the associated potential releases and exposures analyzed

Finally, as EPA well knows, CDR reporting is subject to numerous limitations, including volume thresholds and reporting exemptions that preclude EPA from relying solely on it to conclude manufacturing or processing for a particular use is not occurring.

EPA has provided an inadequate rationale for excluding this condition of use; EPA should analyze this condition of use in the final risk evaluation or provide further analysis establishing the basis for its exclusion and that EPA has considered all existing risks from the activities that are intended, known, or reasonably foreseen for 1-BP.

Consumer use of adhesives (except as an adhesive accelerant for arts and crafts), engine degreasing, and brake cleaning: EPA's only rationale for these exclusions is as follows:

> A review of the use of 1-BP as a solvent in adhesives, engine degreasers, and in brake cleaners showed that these uses of 1-BP are not consumer uses, except as an adhesive accelerant in arts and crafts. In all other uses of 1-BP as an adhesive, 1-BP-containing adhesives are sold through wholesale channels for commercial and industrial uses, and usually in amounts larger than consumers could use. 1-BP has never been advertised (or used) as a consumer brake cleaner or engine degreaser. ... Also, consumers will avoid the use of 1-BP as an engine degreaser or brake cleaner because 1-BP is expensive. In general, heavy duty degreasers containing 1-BP are twice the cost of other heavy duty degreasers and five times the cost of other available consumer brake cleaners. (Problem Formulation for 1-BP at pp. 19-20)

EPA's problem formulation fails to provide adequate support for these exclusions. The draft risk evaluation adopts these exclusions but does not provide further analysis supporting them (p. 19). First, no sources or supporting data are cited or provided. In the accompanying Table 2-2, the only sources EPA lists, purportedly to support these exclusions, in fact do the opposite.

- For adhesives, EPA cites two sources: First, its 2016 draft Work Plan Risk Assessment for 1-BP, which was in large part driven by concerns over just such consumer uses. Second, EPA also cites a March 2017 letter submitted to EPA by EnviroTech, which clarifies that 1-BP is used as a carrier for adhesives, but does

not address the assertions about consumer use, wholesale vs. retail sales, or advertising that EPA makes.[142]

- For brake cleaners or engine degreasers, EPA cites only its own 2017 use document, "Preliminary Information on Manufacturing, Processing, Distribution, Use, and Disposal: 1-Bromopropane," ("Use document") which prominently identifies the very uses EPA now plans to exclude.

Based on EDF's own search of the docket, we located a document posted by EPA but not cited in the problem formulation for brake cleaners or engine degreasers.[143] The document, dated February 2018, purports to support EPA's assertion that 1-BP-containing brake cleaners and engine degreasers are not used by consumers. It consists of two short paragraphs of "analysis" based on a single company's "product guide." The analysis makes numerous assumptions and leaps of logic in its effort to sweepingly conclude that consumers never purchase and use 1-BP-containing brake cleaners or engine degreasers, largely built on questionable notions of consumers' preferences and knowledge.

It is indeed worth highlighting that even EPA states: "It should be noted that some consumers may purchase and use products primarily intended for commercial use." (p. 49) Yet EPA plans to omit such uses entirely.

Second, EPA's own current problem formulation contradicts itself. On p. 10 EPA states:

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[142] The Enviro Tech letter does state, however:

> The use of nPB [n-propyl bromide, a synonym for 1-BP] in the Adhesive sector has a *sad history of over-exposure of workers*. In June, 2007, USEPA proposed to find nPB as unacceptable for use in the Adhesive, Coatings and Inks sector. Enviro Tech, along with the vast majority of our competitors and suppliers, have publically supported this proposed SNAP rule [issued under EPA's Significant New Alternatives Policy (SNAP) program, which identifies substitutes to ozone-depleting chemicals]. Unfortunately, USEPA has seen fit, without further comment, to leave the rule as only proposed by *not issuing a final rule for ten years*. After discussing the health effects of nPB in over 35 pages of text in the rule and proposed rules published ion [*sic*] 2007, we cannot understand why the USEPA would leave a rule in limbo for ten years, despite having the support of the industry that would be regulated by that rule. USEPA immediately issuing a final rule under SNAP would address an important concern shard by the industry and USEPA as noted in its TSCA documents on nPB. (p. 3, emphases added)

This excerpt is telling in that it notes that adhesive use of 1-BP remains a major concern and has not been addressed through existing regulatory authorities.

[143] *See* EPA-HQ-OPPT-2016-0741-0065.

> Consumers and bystanders may be exposed to 1-BP from various consumer uses such as *aerosol and spray adhesives*, aerosol spot removers and aerosol *cleaning and degreasing products*. For 1-BP, EPA considers workers, occupational non-users, consumers, bystanders, and certain other groups of individuals who may experience greater exposures than the general population due to proximity to conditions of use to be potentially exposed or susceptible subpopulations. (p. 10, emphases added)

Third, even if EPA has evidence these uses are not currently ongoing, on what basis can it conclude the uses are not "reasonably foreseen"? Such uses have not been banned (that could be done through a rulemaking pursuant to the current risk evaluation). Nor is there any serious structural, economic or technical rationale EPA has provided for why they could not resume. As discussed in detail earlier in the comments (see Part I, sec. 4.A.), EPA must assume that past uses, absent a regulatory ban, are reasonably foreseen and include them in its risk evaluations.

Interestingly, EPA itself makes an argument for the potential for a different use of 1-BP to return or increase. It does so when discussing, in this same section of the problem formulation, the use of 1-BP in dry cleaning:

> EPA currently believes that few dry cleaners use 1-BP as a dry cleaning solvent. *** However, *the use of 1-BP in the dry cleaning industry remains a reasonably foreseen condition of use.* EPA is currently evaluating tetrachloroethylene (perc) under TSCA, and if EPA were to restrict the use of perc in dry cleaning, many dry cleaners might use 1-BP in their machines *absent regulatory restrictions from doing so*." (p. 20, emphases added)

This logic – that other events could later alter the extent of use of a chemical – is among the reasons why Congress required EPA to include "reasonably foreseen" conditions of use in its risk evaluations under TSCA. The same logic should have been extended to other uses EPA intends to exclude altogether.

In sum, EPA has provided inadequate and contradictory reasons for excluding the consumer uses of 1-BP as a solvent in adhesives, engine degreasers, and in brake cleaners. EPA should analyze these conditions of use in the final risk evaluation.

Consumer disposal of consumer products: EPA plans not to analyze this activity based on an unsupported assumption that exposure from this activity is not expected. EPA states:

> EPA does not expect exposure to consumers from disposal of consumer products. It is anticipated that most products will be disposed of in original containers,

> particularly those products that are purchased as aerosol cans. Liquid products may be recaptured in an alternate container following use (refrigerant flush or coin cleaning). (p. 39, repeated verbatim on p. 50)

EPA provides no evidence to support its expectation and anticipation. In addition, in the last sentence EPA also contradicts itself about the potential for consumer exposure; the uses it identifies as potentially involving "recapture[] in an alternative container following use" – refrigerant flush and coin cleaning – are both listed as consumer uses in Table 2-3. Consumer collection and disposal of spent 1-BP after these uses, even if done in a different container, may well lead to consumer exposures. EPA should analyze the potential exposure to consumers from disposal of consumer products.

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-BP, 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-BP problem formulation contained many rushes to judgment, with EPA all but concluding there is no unreasonable risk from certain exposures, based on little analysis and with no indication that it intended to revisit those exposures or risks in combination with those it did intend to analyze further. The draft risk evaluation fails to provide the analysis missing from the problem formulation, and thus, these cursory analyses remain deeply flawed. We describe some of EPA's flawed analyses below in Part II, sections 4, 6, and 7.

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.

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

In the draft risk evaluation for 1-BP, EPA does not consider the background exposures that workers and consumers experience through ambient air, 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 needs actual data to analyze exposure through water and biosolids.

A. EPA needs to obtain reasonable available information on the potential exposure to 1-BP through water exposure.

In the problem formulation, EPA included the water pathway within the risk evaluation but has also insisted that it will perform no further analysis. *See* Problem Formulation for 1-BP at p. 53. Instead, EPA provided less than a page's worth of analysis of this entire pathway, and the resulting analysis largely fails to establish that EPA has sound reasons for failing to analyze this exposure pathway further. Unfortunately, in the draft risk evaluation, EPA did not revisit this analysis or provide any further data. EPA has the authority to obtain information about the presence of 1-BP in drinking water, and EPA should collect the information necessary to prepare an adequate risk evaluation.

EPA acknowledges that it has no data to justify these aspects of its analysis. *See* Problem Formulation for 1-BP at p. 53 ("[T]here is no data of 1-BP found in US drinking water."). While EPA relies on the physical-chemical properties of 1-BP to estimate that concentrations in water are low, EPA has not established that these concentrations and exposures will not be significant, particularly in conjunction with other exposure pathways. EPA should use its available information authorities to fill these information gaps rather than assume "zero" exposure, particularly since EPA's analyses at best establish that the exposure levels may be low, not nonexistent.

B. EPA should obtain actual monitoring data to confirm its biosolids predictions for 1-BP, and to the extent EPA excludes biosolids on the theory that 1-BP will instead enter other pathways, EPA must consider those exposure pathways.

In the 1-BP problem formulation, EPA states that 1-BP is expected to enter the aqueous component and volatilize to air, and thus asserts EPA can ignore the biosolids exposure pathway. *See, e.g.*, Problem Formulation for 1-BP at pp. 53-54. EPA should obtain some monitoring data to confirm these analyses, but in any event, EPA cannot rationalize ignoring exposures from biosolids on the basis that 1-BP will enter the water and air and then also choose to ignore the exposure pathways through water and air. EPA's justification for ignoring the biosolids pathways for 1-BP highlights that EPA's decision to ignore other pathways is particularly arbitrary and capricious.

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

As established above, EPA must assess all hazards and exposures when evaluating the risk presented by a chemical substance. For this same reason, EPA must consider all real-world, intended, and reasonably foreseen exposures that occur even if they fall under the jurisdiction of other EPA-administered statutes. In the problem formulation for 1-BP, EPA excluded from the risk evaluation "pathways under programs of 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-BP p. 54, 13, 45-46. Specifically, EPA excluded the disposal pathway on this basis. Then, in the draft risk evaluation, EPA *also* excluded the ambient air pathway. (pp. 27, 258-59). Notably, here EPA directly contradicts the problem formulation, which stated that EPA would consider the ambient air pathway. Problem Formulation for 1-BP p.52.

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). The draft risk evaluation does not establish that the regulation of 1-BP under other statutes will eliminate exposures, and in fact, publicly available evidence establishes 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[] risks." (p. 27). EPA's bald assertions to the contrary do not make it so. In any event, that is not the legally correct standard under TSCA. As explained below, EPA can only rely on statutory authorities other than TSCA in compliance with TSCA § 9 (notably, the TSCA § 9 process occurs after EPA has completed a comprehensive risk evaluation finding unreasonable risk). To comply with TSCA § 9, EPA must find that those authorities eliminate the risks EPA has previously identified or reduce them to a sufficient extent under TSCA § 9(b)(1), and TSCA requires that EPA reduce risk "to the extent necessary so that [the chemical] no longer presents [an unreasonable risk of injury to health or the environment]." *See* 15 U.S.C. §§ 2608(b)(1), 2605(a). In addition, under TSCA § 9(b)(2) EPA must consider "all relevant aspects of the risk" when deciding whether to regulate under TSCA or another statute. *Id*. § 2608(b)(2). EPA has not met any of these standards in the draft risk evaluation, and EPA's statements that the exposures are adequately assessed and effectively managed under other statutes are legally irrelevant (even if they were true).

When relying on these other statutory authorities, EPA merely provides a list of various regulatory standards and criteria that EPA indicates apply or could apply to certain sources of 1-BP (*see e.g.*, p.27; Problem Formulation for 1-BP pp.54-55). EPA provides no analysis whatsoever as to: the extent to which the standards or criteria cover the full range of exposure to the chemical through the pathway; the extent and magnitude of releases of the chemical allowed under each of the regulatory standards or criteria; or any other factors that would be necessary to analyze to determine the extent and nature of potential risk allowed under the standards. In particular, TSCA § 6(b)(4)(F)(iv) requires that, in conducting a risk evaluation, EPA evaluate "the likely duration, intensity, frequency, and number of exposures," 15 U.S.C. § 2605(b)(4)(F)(iv), including exposures resulting from those allowable emissions, discharges, or releases. EPA needs to provide this analysis, and EPA cannot simply point to regulation under another statute to bypass the analysis. EPA has also not acknowledged, let alone analyzed, the overall risks to the general population or to vulnerable subpopulations due to the combination of exposures arising from the various sources for which standards exist, not to mention in combination with additional emission sources not subject to any standard. EPA has made no attempt to reconcile any such risk with that allowed under TSCA.

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

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

In the problem formulation, 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-BP at p. 13. But EPA provides no textual basis for ignoring those exposures. Instead, in a footnote, EPA cites to its discussion regarding "conditions of use," but even assuming for the sake of argument that EPA has authority to exclude conditions of use, such power does not justify excluding exposures related to conditions of use still within the scope of the risk evaluation, as EPA proposes to do. Nothing in TSCA's risk evaluation provision authorizes EPA ignoring exposures because of other statutory authorities, and as explained above, EPA has to analyze all exposures including these exposures. And several other provisions of TSCA indicate that Congress intended for EPA to consider such exposures, except to the extent Congress explicitly provided otherwise.

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

Second, in TSCA's risk evaluation provision, Congress specifically intended for EPA to "conduct risk evaluations *** to determine whether a chemical substance presents an unreasonable risk of injury to *** the environment," 15 U.S.C. § 2605(b)(4)(A), but EPA's approach has eliminated almost all analysis of environmental exposures. EPA has largely read the requirement to evaluate risks to the environment out of the statute, but this approach violates a fundamental tenant of statutory interpretation. A. SCALIA & B. GARNER, READING LAW: THE INTERPRETATION OF LEGAL TEXTS 174 (2012) ("If possible, every word and every provision is to be given effect *** None should needlessly be given an interpretation that causes it to duplicate another provision or to have no consequence."). Moreover, Congress enacted this requirement that EPA analyze risks to the environment against the backdrop of the existing environmental statutes; if Congress had considered them per se sufficient, Congress would not have included this mandate in TSCA. But Congress did.

Third, Congress specifically directed EPA to analyze the risks of chemicals presented "under the conditions of use," and Congress consciously decided to specify that "disposal" is a condition of use under TSCA. "Conditions of use" expressly includes "the circumstances *** under which a

chemical substance is intended, known, or reasonably foreseen to be to be manufactured, processed, distributed in commerce, used, or *disposed of*." 15 U.S.C. § 2602(4) (emphasis added). In the problem formulations, EPA systematically excludes exposures through disposal based on a variety of theories (*see, e.g.*, Problem Formulation for 1-BP at pp. 54-55), 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 and draft risk evaluation, EPA makes no showing that its actions under other statutes reduce the risk "to the extent necessary so that [it] no longer presents [an unreasonable risk of injury to health or the environment]," and EPA does not present any actual analysis of "all relevant aspects of the risk" arising from the ignored exposures. So EPA has undisputedly failed to comply with TSCA § 9(b). Given that Congress expressly addressed the issue of overlapping regulatory jurisdictions in TSCA § 9, EPA cannot avoid those procedures by simply ignoring exposures that fall within another statute's jurisdiction.

Furthermore, EPA is expressly required to evaluate exposures from combinations of activities, which it cannot do if it excludes some exposures at the outset that may be able to be addressed

under another authority, particularly when any risk management under the other authority would not reduce exposure to zero.

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

i. EPA must analyze whether 1-BP presents a risk to the general population because reasonably available information establishes that the general population is exposed to 1-BP.

In its draft risk evaluation, EPA states that it will not analyze general population exposures for 1-BP because (1) EPA considers its existing regulatory programs sufficient (pp. 158-59) and (2) EPA considers a cursory analysis of exposure through water and land adequate to dismiss risks from that exposure (Problem Formulation for 1-BP at pp. 53-54). 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 particular, the most recent TRI data for 1-BP establishes that 1-BP is released to air and water in significant quantities:

Toxics Release Inventory: 2018[144]

Chemical Substance	Air (lb)	Water (lb)	Land (lb)	Total (lb)
1-bromopropane	777,574.85	1.00	171,310.00	948,885.85

EPA relies on the outdated data from 2016 in its Problem Formulation for 1-BP at p. 33, but EPA should use the most up-to-date data available, particularly given that the significantly larger quantities reported in 2018 may effect EPA's ultimate analysis. The new TRI data are certainly reasonable available information under TSCA § 26(k). *See* 15 U.S.C. § 2625(k).

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.

[144] 2018 TRI PRELIMINARY DATASET, https://www.epa.gov/toxics-release-inventory-tri-program/2018-tri-preliminary-dataset (last visited Sept. 23, 2019).

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

In the problem formulation for 1-BP, EPA correctly recognized that potentially exposed or susceptible subpopulations included 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, use or disposal sites)." Problem for 1-BP at p. 40. But in the draft risk evaluation, EPA excludes the primary pathway that would lead to exposures for these susceptible subpopulations—the ambient air pathway (*See* pp. 27, 258-59). EPA provides no rational explanation for how it will accurately and effectively evaluate the actual risk faced by these subpopulations while ignoring these exposures. Moreover, EPA's (correct) recognition that these groups face greater exposure highlights that it is irrational for EPA to ignore the pathways leading to these exposures.

As a result, in the draft risk evaluation, EPA largely fails to analyze this potentially exposed or susceptible subpopulation (pp. 135-37). EPA limits its analysis of greater exposure to workers, occupational non-users, and consumers, and EPA largely ignores the greater exposure experienced by individuals living in proximity to conditions of use. As a result, EPA fails to consider an important aspect of the problem because EPA fails to analyze the risks posed to a potentially exposed or susceptible subpopulation that EPA previously acknowledged.

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

C. The *potential* listing of 1-BP as a hazardous air pollutant does not reduce exposures at all, much less to resulting to zero exposures through the air pathway; EPA should analyze the real-world exposures.

In the draft risk evaluation, EPA excluded exposures to 1-BP through the air pathway because EPA *may* list it as a hazardous air pollutant (HAP) under the Clean Air Act (CAA) (p.27). But EPA has not yet taken final action to list 1-BP as a HAP, and EPA cannot rationally rely on a regulatory approach that it has not yet adopted. Additional steps would need to be taken to actually regulate 1-BP under CAA, which have not been taken. The vague statement that the EPA "intends to finalize an action before the end of 2019"—with no specification of what outcomes may result—provides no basis for EPA's assertion that 1-BP's risks are being "adequately assess[ed] and effectively manage[d]." (p.27). 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 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 CAA would provide adequate protection.

In any event, even assuming that EPA does list 1-BP as a HAP, EPA has not made the necessary showing that an established HAP would eliminate any unreasonable risk and EPA has not assessed all relevant aspects of the risk. As EPA acknowledges, the listing as a HAP leads to a technology-based standard for certain stationary sources (p. 27). 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 Section 112, 42 U.S.C. §§ 7412, (p. 27, 258) 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 the 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 *potentially* 1-BP. 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." U.S. EPA, Problem Formulation of the Risk Evaluation for Perchloroethylene (Ethene, 1,1,2,2-Tetrachloro) (May 2018), p.59 https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0732-0080. 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, CAA Section 111, 42 U.S.C. § 7411, 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.

ii. Reasonably available information establishes that there is exposure through ambient air.

The problem formulation for 1-BP itself establishes that exposures through air persist for 1-BP, 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. For example: EPA states that 626,660 pounds for 1-BP were released to the air in the 2016 reporting year according to the EPA Toxics Release Inventory (TRI). Problem Formulation for 1-BP at p. 32-33.

Using the more up-to-date data from the 2018 TRI reporting, 777,574.85 lbs were reported released to the air.[145] EPA cannot ignore this reasonable available information establishing that exposures through ambient air are occurring.

Moreover, in its request for public comment on listing 1-BP as a HAP, EPA found "that there is adequate evidence to support a determination that emissions and ambient concentrations of nPB may reasonably be anticipated to cause adverse health effects." 82 Fed. Reg. 2354, 2354 (Jan. 9, 2017). This finding would support a finding that current emissions of 1-BP present an unreasonable risk to human health and thus merit regulation under TSCA.

Given evidence of real-world exposure through the air pathway, EPA must evaluate those exposures in its final 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.

D. Real-world exposures still occur through disposal pathways, and EPA cannot ignore those real-world exposures when assessing the risk presented by 1-BP.

In the problem formulation for 1-BP, EPA contends that due to regulation of disposal under the Resource Conservation and Recovery Act (RCRA), EPA can ignore all exposures from all disposal-related pathways and associated activities (e.g., collection, processing, storage and transport). Problem Formulation for 1-BP at pp. 54-55. 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

[145] 2018 TRI PRELIMINARY DATASET, https://www.epa.gov/toxics-release-inventory-tri-program/2018-tri-preliminary-dataset (last visited Sept. 23, 2019).

not shown or established that disposal in a RCRA Subtitle C hazardous waste landfill or a RCRA Subtitle D municipal solid 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 chemicals allegedly managed under RCRA can be or are disposed of in non-hazardous waste landfills. For example, EPA's TRI reporting on 1-BP showed that most of the releases to the land were to "other off-site landfills," not RCRA Subtitle C landfills. Problem Formulation for 1-BP at p. 34. EPA cannot rely on regulations that do not apply to protect against risks.

Even for those chemicals regulated under RCRA, EPA acknowledges that disposal also occurs in Subtitle D municipal solid waste (MSW) landfills and industrial-non-hazardous and construction/demolition waste landfills (which are primarily regulated under state regulatory programs). These disposal approaches do not need to meet the requirements of Subtitle C landfills, thus EPA's invocation of the Subtitle C standards does not justify ignoring exposures from these disposals. While the purpose of RCRA subtitle C is at least to "protect human health and the environment," *see, e.g.*, 42 U.S.C. §§ 6922(a), 6924(a), subtitle D is intended "to assist in developing and encouraging methods for the disposal of solid waste which are environmentally sound and which maximize the utilization of valuable resources including energy and materials *** and to encourage resource conservation." 42 U.S.C. § 6941. Therefore, EPA's exclusions based on the regulations under subtitle D potentially raise even greater, unaddressed, public health concerns than EPA's exclusions under subtitle C. In addition, states impose varying requirements on such landfills under their delegated RCRA Subtitle D authorities. For example, EPA indicates that some state programs may not include requirements for liners to limit release of landfill leachate.

EPA itself has acknowledged that enforcement and regulation under RCRA is inconsistent, so EPA cannot simply assume that RCRA implementation provides a basis for ignoring exposures under TSCA. As the Office of Inspector General explained the challenges of the RCRA system:

> The Hazardous and Solid Waste Amendments of 1984 (HSWA) amended RCRA and added provisions including land disposal restrictions, RCRA corrective action for solid waste management units and regulation of small-quantity generators. When the EPA creates new hazardous waste rules, it does so under the authority of either or both of these laws. Rules promulgated under HSWA authority are immediately effective in all states and are administered by the EPA until states become authorized for those rules. In contrast, *rules promulgated under RCRA authority (non-HSWA rules) cannot be enforced by the EPA in states with an*

> *authorized base program and do not go into effect until these states become authorized for the rules*.[146]

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."[147] 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."[148]

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."[149] 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 these chemicals' presence in soil, sediment, and leachate, than it

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[146] U.S. EPA, Office of Inspector General, *Incomplete Oversight of State Hazardous Waste Rule Authorization Creates Regulatory Gaps and Human Health and Environmental Risks* at 2 (Jul. 2018), https://www.epa.gov/sites/production/files/2018-07/documents/_epaoig_20180731-18-p-0227.pdf (emphasis added).
[147] *Id.* at 11.
[148] *Id*. at 12; *see also* AUTHORIZATION STATUS BY RULE, https://www.epa.gov/sites/production/files/2018-06/documents/authorization_status_by_rule.pdf (last visited Aug. 10, 2018) (documenting for each state whether they have adopted the RCRA regulations).
[149] 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.

does for their presence in water or air. *See, e.g.*, Problem Formulation for 1-BP at p. 34. 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.[150] EPA should use those authorities to obtain additional information about the exposures arising from disposal of 1-BP. EPA should analyze the exposures resulting from disposal of 1-BP based on real scientific evidence; it should not simply make unsupported assumptions.

EPA cannot assume that exposure from disposal is zero just because it could be regulated under other authorities.

E. 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.[151] 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.[152]

Generally, EPA's regional offices provide oversight to ensure that the state enforcement programs are following EPA's guidance, policies, and regulations.[153] Despite EPA oversight, which is a separate concern, state enforcement of these statutes has been found deficient in a number of cases. For instance:

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[150] EDF Comments on Ten Scopes under the Toxic Substances Control Act pp.11-15, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0741-0059.
[151] 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).
[152] *Id.* at 32.
[153] 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."[154] For instance, during the entire period of the report (FYs 2003-2009), the state assessed no penalties against known CWA violators.[155]
- In **Louisiana** multiple petitions have been filed by citizens to remove the state's delegated authorities under the CWA, CAA, and RCRA.[156] The poor performance under these statues was attributed to "a lack of resources, natural disasters, and a culture in which the state agency is *expected to protect industry*."[157]
- 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."[158]

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."[159] 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. While EPA has not relied on the SDWA or the CWA to justify exclusions of pathways for 1-BP,

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[154] 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.
[155] *Id.* at 15.
[156] *Id.* at 16.
[157] *Id.* (emphasis added).
[158] 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.
[159] 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.

EPA's deficient enforcement of these statutes provides further evidence that full compliance with the statutes that it implements can by no means be assumed.

Safe Drinking Water Act: EPA has excluded exposures to drinking water for several of the other first ten chemicals 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.[160] 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.[161]

Clean Water Act: EPA has also excluded exposures to ambient water for numerous chemicals 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.[162] EPA's own analysis, provided below, indicates that waters remained impaired throughout the United States, despite the CWA standards.

[160] 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.
[161] Letter from Jon M. Capacasa, Director, EPA Region III Water Protection Division, to Lisa D. Daniels Director, Pa. Dep't of Envtl. Prot. Bureau of Safe Drinking Water (Dec. 30, 2016), https://drive.google.com/file/d/0B4Y3VQLxjkxObjZ0ZXlSVDZvRWc/view.
[162] NATIONAL SUMMARY OF STATE INFORMATION, https://ofmpub.epa.gov/waters10/attains_nation_cy.control (last visited Jul. 31, 2018).

Assessed Water of the United States[163]

	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.[164] According to the 2015 report, the percentage of facilities with formal enforcement actions compared to facilities with violations was merely 8.9% in 2015.[165] 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.[166]
- **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."[167] 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.[168]

[163] *Id.*
[164] 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.
[165] *Id.* at 7.
[166] 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.
[167] 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.
[168] 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.[169]

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."[170] 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.[171] Additionally, from 2013 to 2015 the state only filed one asbestos case, compared to a past annual average of 13.[172]
- **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)."[173]
- **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.[174]

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.

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[169] 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.
[170] 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.
[171] 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.
[172] *Id.*
[173] Letter from J. Scott Gordon, Director, EPA Region IV Office of Enforcement Coordination, to Donald R. van der Vaart, Secretary, N.C. Dep't of Envtl. Quality (May 9, 2016), https://assets.documentcloud.org/documents/3114598/EPA-Region-4-Letter-to-NCDEQ.pdf.
[174] 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.

For example, Mississippi has not accurately identified and documented RCRA violations.[175] 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.[176]

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.[177] 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.[178]

Additionally, in its proposed 2020 budget, the current Administration sought a 31 percent reduction in funding for EPA.[179] This reduction would affect EPA's enforcement budget and the resources available to ensure enforcement of the above the statutes. EPA cannot rely on its actions under other authorities when EPA has itself taken steps to ensure that those authorities are not adequately addressing the risks presented.

EPA cannot rely on its actions under other authorities when EPA has itself taken steps to ensure that those authorities are not adequately addressing the risks presented.

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In sum, EPA must analyze all exposures to these chemicals. 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 these chemical substances.

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[175] 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.
[176] *Id.* at 24.
[177] 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.
[178] *Id.*
[179] Office of Mgmt. & Budget, *A Budget for a Better America* at 93, https://www.whitehouse.gov/wp-content/uploads/2019/03/budget-fy2020.pdf.

Where EPA has inadequate information, EPA should use its information authorities to obtain more information about these exposures.

6. EPA relies extensively on assumptions that are inconsistent or not supported with data, and on models that are not conservative, despite claims to the contrary.

Terrestrial environmental exposures: EPA states:

> *EPA does not plan to further analyze terrestrial exposures*, due to low expected toxicity (see Section 2.4.1) and low expected exposure based on the physical/chemical properties (e.g., high vapor pressure; see Section 2.1). (Problem Formulation for 1-BP at p. 35, emphases added)

Yet the cited section 2.4.1 provides no data that demonstrate low toxicity; rather, it cites an *absence* of toxicity data – a clear data gap EPA fails to identify or indicate whether or how it will address:

> During data screening, there were no available sediment, soil, nor avian toxicity studies found in the scientific literature for 1-BP. The toxicity of 1-BP is expected to be low *based on the lack of on-topic environmental hazard data for 1-BP to sediment and terrestrial organisms in the published literature* and the physical/chemical/fate properties (relatively high volatility (Henry's Law constant of 7.3X10-3 atm-m3/mole), high water solubility (2.4 g/L), and low log Koc (1.6) suggesting that 1-BP will only be present at low concentrations in these environmental compartments. (p. 41)

The physical/chemical/fate properties EPA cites may be germane to some sediment- or soil-dwelling organisms, but in no way rule out exposure of terrestrial organisms through inhalation – a pathway that EPA elsewhere acknowledges is quite relevant to the subset of terrestrial organisms otherwise known as humans. Moreover, EPA's effort to dismiss toxicity data gaps based on exposure arguments does not reflect sound science. Nor does EPA's equating a lack of on-topic hazard data with evidence of low toxicity.

EPA must use its information authorities to generate hazard data for sediment-dwelling and other terrestrial organisms. EPA has failed to address this data gap and analytical flaw in the draft risk evaluation (p. 21), and EPA needs to do so before the final risk evaluation.

Dermal exposure:

In discussing consumer exposures, EPA states:

> Dermal exposure may occur via vapor/mist deposition onto skin or via direct liquid contact during use, particularly in occluded scenarios. (Problem Formulation for 1-BP at p. 38)

This scenario is equally or more likely to apply in occupational settings, yet is not mentioned in that section of the problem formulation (section 2.3.5.1).

Later, in discussing the conceptual model for consumer activities, EPA states:

> Some products may be purchased and used as a liquid. For these uses, consumers may have dermal contact from occluded exposures such as holding a rag soaked in liquid 1-BP where limited evaporation rates and penetration may be expected to be higher in these scenarios. *EPA does not expect to further analyze dermal exposure to 1-BP vapor*, however EPA does expect to further analyze direct dermal contact with liquid 1-BP for consumers during the risk evaluation phase. (p. 49)

Yet just pages earlier (and cited just above), EPA had acknowledged the potential significance of dermal exposures to *vapor*, referring to "vapor trapped against skin by gloves or continued contact with a wet rag) or where there is greater potential for dermal penetration due to longer durations of exposure." (pp. 38-9) Why is EPA now stating it will ignore such exposures altogether?

EPA's apparent decision to exclude certain dermal exposures to 1-BP altogether, or to conclude with no further analysis that certain dermal exposures are negligible, is inconsistent with TSCA's mandate that EPA consider the combination of exposures to a chemical in assessing its risks, a requirement discussed earlier in these comments (see Part II, sec. 1.A.ii.). It is also not consistent with EPA's own problem formulation, where EPA states:

> Based on the physical-chemical properties and high evaporative losses compared to dermal absorption as described in Section 2.3.5.2, non-occluded dermal exposures are not expected to be the primary route of exposure for consumers, *although dermal exposures will contribute to the overall exposure*. (Problem Formulation for 1-BP at p. 49, emphasis added)

This logic on the need to look at all contributors to overall exposure applies to numerous pathway scenarios that EPA says it will not further analyze because inhalation is deemed the "major" exposure pathway. As discussed earlier in these comments (see Part I, sec. 1.A.ii.), TSCA includes nothing that allows EPA to limit itself only to assessing the "major source" of exposure to 1-BP or other chemicals. EPA must analyze all routes of dermal exposures to 1-BP, including how these exposures contribute to overall exposure.

Ingestion: EPA states:

> EPA does not plan to further analyze exposure to consumers via ingestion of 1-BP. Ingestion is not expected to be a primary route of exposure. Based on the vapor pressure, 1-BP will exist as a vapor/mist during use. (Problem Formulation for 1-BP at p. 38)

Yet as just noted, EPA acknowledges elsewhere that "[s]ome products may be purchased and used as a liquid." (Problem Formulation for 1-BP at p. 49) How can EPA wholly rule out ingestion, including by accident? In the draft risk evaluation, EPA performed no further analysis of ingestion (p. 40) and EPA failed to justify its decision to rule out ingestion as a potential route of exposure.

7. 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: "EPA will evaluate activities resulting in exposures associated with distribution in commerce (e.g. loading, unloading) throughout the various lifecycle stages and conditions of use (e.g. manufacturing, processing, industrial use, consumer use, disposal) rather than as a single distribution scenario." (p. 30). It appears from this statement that EPA assumes that exposure from distribution occurs only during loading and unloading. EPA does not appear to address the exposures from distribution aside from loading and unloading. Does EPA simply assume that all distribution occurs through so-called "closed systems" which lead to no releases or exposure whatsoever?

Neither the problem formulation nor the draft risk evaluation provide evidence or support for EPA's apparent assumption that 1-BP is always distributed in closed systems leading to no releases or exposures. EPA has provided no evidence that exposures and releases during distribution will be nonexistent.

The 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.[180] 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.

8. 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-BP, and EDF incorporates those arguments here.[181] 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 this point.[182] While EPA details 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.

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[180] *See, e.g.*, *More Than 40 Sites Released Hazardous Pollutants Because of Hurricane Harvey*, N.Y. TIMES (Sept. 8, 2017), https://www.nytimes.com/interactive/2017/09/08/us/houston-hurricane-harvey-harzardous-chemicals.html?_r=0.

[181] EDF Comments on Ten Scopes under the Toxic Substances Control Act at pp. 11-16, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0741-0059; 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-2016-0741-0085.

[182] 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.

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 chemicals. EDF incorporates those arguments here.[183] EPA's response to this comment was that "EPA has not indicated it would rely solely on voluntary requests for information."[184] Thus, EPA appears to recognize that voluntary requests standing alone are insufficient. Despite that acknowledgement, EPA still has not relied on its available authorities to obtain additional information. EDF urges EPA to do so.

EPA's primary response to EDF's request that EPA consider all reasonably available information appears to be that the information EPA currently has is "adequate."[185] 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 1-BP.

In the 1-BP 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-BP at p. 57 (emphasis added). With this language EPA seems 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 resorts merely to encouraging their submission.

As detailed later in this section and in our comments in Part I, sec. 4, many key data gaps remain regarding 1-BP in the draft risk evaluation. Thus, EPA's reliance on voluntary submissions has failed to produce the information necessary for a robust and accurate risk evaluation.

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[183] EDF Comments on Ten Scopes under the Toxic Substances Control Act at pp. 16-20, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0741-0059; 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-2016-0741-0085.

[184] 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.

[185] *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 the 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 their chemical in a favorable light. They may provide only summaries of information that reflect conscious and subconscious judgment calls that result in unduly favorable conclusions; and without access to the full information neither EPA nor the public can independently assess such conclusions. They may choose not to review records robustly when the review may disclose unfavorable information. They may seek to put their best foot forward and describe the ideal scenario of use and safety measures. Or, if they have unfavorable information, they may choose not to provide any information at all and simply not participate in these proceedings.

EPA cannot simply assume that members of the regulated community will voluntarily disclose unfavorable or complete information about their practices and products. *See* THE FEDERALIST NO. 51 (James Madison) ("If men were angels, no government would be necessary. *** [E]xperience has taught mankind the necessity of auxiliary precautions."); *Williams v. Pennsylvania*, 136 S. Ct. 1899, 1905-06 (2016) ("Bias is easy to attribute to others and difficult to discern in oneself. *** This objective risk of bias is reflected in the due process maxim that 'no man can be a judge in his own case and no man is permitted to try cases where he has an interest in the outcome.'"). Here, manufacturers and processors obviously have an interest in the outcome, and EPA must craft its procedures and approaches with that reality in mind. Requiring the submission of information is the safest approach to ensuring that these parties provide all relevant information, and that is in turn crucial to establishing and demonstrating the credibility of this process.

If EPA acts under TSCA §§ 8(a), (c), and (d), the regulations impose some requirements that will help ensure the accuracy and completeness of the information. First, EPA can require that certain information and underlying information be provided in full, which ensures completeness. In addition, a § 8(d) rule requires that people engage in an adequate search of records. 40 C.F.R. § 716.25. Second, submitters must file certification statements by authorized officials that certify that the submitted information has been submitted in compliance with the requirements of this process. *See, e.g.*, 40 C.F.R. § 711.15(b)(1). Third, submitters often must retain records of required submissions for a period of five years, and the retention of records can help encourage accurate reporting since those records would be available should a submission latter be investigated. *See, e.g.*, 40 C.F.R. § 711.25. None of these features apply to the voluntary requests for information EPA has indicated it is relying on.

B. EPA cannot rationally rely on unvetted industry submissions, and to the extent EPA relies on voluntary submissions from industry, EPA must take numerous additional steps to increase their reliability and transparency.

In the draft risk evaluation, EPA uncritically relies on industry submissions, and this reliance does not constitute the best available science. For example, EPA relies on a technical report entitled Stewart, 1998 (p. 83), but when one follows the link for the report, the report is not available and the title is described as "sanitized." From this information it appears that someone submitted this technical report with a confidentiality claim, but it is impossible to review the underlying study or assess its accuracy.

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:

- "Exposures for occupational non-users can vary substantially. Most data sources do *not sufficiently* describe the proximity of these employees to the 1-BP exposure source. As such, exposure levels for the "occupational non-user" category will have high variability depending on the specific work activity performed" (p. 240).
- "There are limited exposure monitoring data in literature for certain conditions of use or job categories. For example, very few data points are available for cold cleaning and for spot-cleaning" (p. 240).
- "[T]here are data gaps and uncertainties regarding the environmental hazards of 1-BP" (p. 246).
- "A lack of sediment and terrestrial toxicity test data creates some uncertainty associated with this assessment" (p. 246).
- "The lack of monitoring data to characterize the concentrations of 1-BP present in aquatic sediment represents an uncertainty regarding aquatic hazard" (p. 246).
- "While it is anticipated that there may be differential 1-BP metabolism based on lifestage; currently there are no data available, therefore the impact of this cannot be quantified. Similarly, while it is known that there may be genetic differences that influence CYP2E1 metabolic capacity, there may also be other metabolizing enzymes that are functional and impact vulnerability. There is insufficient data to quantify these differences for risk assessment purposes" (p. 248).

As a general matter, EPA should use its information authorities to fill the gaps identified in the draft risk evaluation and these comments.

D. EPA's problem formulation also revealed numerous data gaps, yet EPA failed to address any of them.

Notably, many data gaps were evident at the problem formulation stage, and EPA failed to address these data gaps despite its information authorities under TSCA as reformed in 2016. In this section we provide a list of examples of the many data gaps apparent from the Problem Formulation and EPA's resort to insufficient approaches to work around the gaps without actually filling them. EPA should have used its information authorities to fill these data gaps, and EPA still could do so in preparing a final risk evaluation.

1. EPA appears adamant on relying on models rather than requiring the development of information to fill gaps or resolve discrepancies and uncertainties in the available data – even where the models contribute to that uncertainty based on variable results. For example, EPA states:

> The EPI Suite™ module that predicts biodegradation rates ("BIOWIN" module) was run using default settings to estimate biodegradation rates of 1-BP under aerobic conditions. Three of the models built into the BIOWIN module (BIOWIN 2, 5 and 6) estimate that 1-BP will not rapidly biodegrade in aerobic environments, while a fourth (BIOWIN 1) estimates that 1-BP will rapidly biodegrade in aerobic environments. These results support the biodegradation data presented in the 1-BP Scope Document (EPA-HQ-OPPT-2016-0741-0049), which demonstrate a range of biodegradation rates under aerobic conditions. The model that estimates anaerobic biodegradation (BIOWIN 7) predicts that 1-BP will rapidly biodegrade under anaerobic conditions. Further, previous assessments of 1-BP found that biodegradation occurred over a range of rates from slow to rapid [Toxicological Profile for 1-Bromopropane; (ATSDR, 2017)]. (Problem Formulation for 1-BP at p. 30)

2. EPA states: "*No measured bioconcentration studies for 1-BP are available*. An estimated BCF of 11 and an estimated BAF of 12 suggest that bioconcentration and bioaccumulation potential in aquatic organisms is low (BCF and BAF <1,000)." (Problem Formulation for 1-BP at p. 31)

Rather than require such bioconcentration studies be performed, EPA relies on models without any characterization of the resulting uncertainty associated with the conclusions it draws. Yet existing models have often been criticized as unreliable and often under-predictive of bioconcentration and bioaccumulation potential.[186]

3. EPA states: "Currently, EPA is not aware of the presence of 1-BP in recycled articles." (Problem Formulation for 1-BP at p. 34) This clear data gap is used by EPA to suggest that exposures from recycling activities are not of concern. In reality, it simply means the question cannot be answered without addressing the data gap.

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[186] *See, e.g.,* Arnot, J.A. & Frank Gobas, *A review of bioconcentration factor (BCF) and bioaccumulation factor (BAF) assessments for organic chemicals in aquatic organisms,* 14 ENVIRON. REV. 257-297 (2006), http://rem-main.rem.sfu.ca/papers/gobas/A%20Review%20of%20Bioconcentration%20factor%20(BCF)%20and.pdf.

4. EPA states: "[T]here were no available sediment, soil, nor avian toxicity studies found in the scientific literature for 1-BP. The toxicity of 1-BP is expected to be low based on the lack of on-topic environmental hazard data for 1-BP to sediment and terrestrial organisms in the published literature and the physical/chemical/fate properties (relatively high volatility (Henry's Law constant of 7.3X10-3 atm-m3/mole), high water solubility (2.4 g/L), and low log Koc (1.6) suggesting that 1-BP will only be present at low concentrations in these environmental compartments." (Problem Formulation for 1-BP at p. 41)

Astoundingly, EPA here relies on the lack of available data to conclude toxicity must be low.

5. EPA states: "For most high-priority chemical substances level(s) can be characterized through a combination of available monitoring data and modeling approaches." (Problem Formulation for 1-BP at p. 57)

EPA simply asserts this as fact, even as it seeks (as noted on this same page) more of the very same data. And for 1-BP, EPA has acknowledged there are no monitoring data available (Problem Formulation for 1-BP at p. 34).

6. EPA states: "Additionally, for conditions of use where no measured data on releases are available, EPA may use a variety of methods including the application of *default assumptions*. *** EPA will also review data sources containing estimated data and *identify data gaps*." (Problem Formulation for 1-BP at p. 58)

While defaults have their place, there is no excuse for EPA failing to even mention its authority to require the development and submission of the information it needs. And to date, EPA has done little to nothing to identify data gaps, and instead actively seeks to avoid doing so.

7. EPA states: "If measured values resulting from sufficiently high-quality studies are not available (to be determined through the systematic review process), chemical properties will be estimated using EPI Suite, SPARC, and other chemical parameter estimation models. Estimated fate properties will be reviewed for applicability and quality." (Problem Formulation for 1-BP at p. 60)

Again EPA skips right over any mention of mandating data development or submission.

8. EPA states: "EPA will review *reasonably available* data that may be used in developing, adapting or applying exposure models." (Problem Formulation for 1-BP at p. 61, emphasis added)

In its final risk evaluation rule, EPA defines "reasonably available" as 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 CFR § 702.33, emphasis added. Yet, in the problem formulation, EPA makes no mention of efforts to use its authorities to generate or obtain needed information.

9. EPA states: "For some OSHA data, NAICS codes included with the data will be matched with potentially applicable conditions of use, and *data gaps will be identified where no data are found for particular conditions of use. EPA will attempt to address data gaps identified as described in steps 2 and 3 below*." (Problem Formulation for 1-BP at p. 64, emphasis added) Step 2 entails the use of data on surrogate chemicals. Step 3 entails the use of models. No step is indicated that would entail requiring submission or development of the needed data.

10. EPA states: "Review reasonably available exposure data for surrogate chemicals that have *uses and chemical and physical properties similar to 1-BP*. *** For several uses including use of adhesives, and cleaning products, EPA believes that trichloroethylene and other similar solvents may share the same or similar conditions of use and may be considered as surrogates for 1-BP." (Problem Formulation for 1-BP at p. 64, emphasis added)

EPA makes no mention of the need for surrogate chemicals to have similar environmental and biological fate as well as chemical and physical properties. Nor does it appear to be planning to compare the chemicals on the basis of any available toxicity information. While EDF does not oppose including surrogate data when relevant, it should not be the option of first resort and be used to excuse EPA from actively pursuing such data through its information authorities.

11. EPA states: "*If sufficient dermal toxicity studies are not identified* in the literature search to assess risks from dermal exposures, then a *route-to-route extrapolation* from the inhalation and oral toxicity studies would be needed to assess systemic risks from dermal exposures." (Problem Formulation for 1-BP at p. 70, emphasis added)

Again, EPA makes no mention of filling the data gap.

EPA should use its information authorities to fill the above data gaps in order to develop a risk evaluation consistent with the best available science and reasonably available information.

E. 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 it lacks "sediment and terrestrial toxicity test data" (p. 246; *see*

also Problem Formulation for 1-BP at p. 41). "The toxicity of 1-BP is expected to be low based on the lack of on-topic environmental hazard data for 1-BP to sediment and terrestrial organisms in the published literature." Problem Formulation for 1-BP at p. 41. 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.

9. EPA needs to implement the requirements of TSCA § 14 when reviewing materials for this risk evaluation.

EPA has an affirmative obligation to review at least 25% of non-chemical identity confidentiality claims under TSCA, 15 U.S.C. § 2613(g), and EPA has stated that it is implementing that obligation by "review[ing] every fourth submission received that contains non-chemical identity [confidential business information (CBI)] claims."[187] 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 *** for which notification is required under section 5." 15 U.S.C. § 2613(b)(2)(A). TSCA also requires disclosure of "any information reported to, or *otherwise obtained by*, [EPA] *from* a health and safety study which relates to [such] a chemical substance. . . ." *Id.* § 2613(b)(2)(B) (emphases added). Thus, any health and safety studies and related information on these chemicals 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"

[187] 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).

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.

Moreover, in the problem formulation, EPA states: "Based on market information from other sources, EPA expects degreasing and spray adhesive to be the primary uses of 1-BP; however, the *exact use volumes associated with these categories are claimed CBI* in the 2016 CDR (U.S. EPA, 2016a)." (Problem Formulation for 1-BP at p. 27, emphasis added)

EPA's failure to conduct the timely reviews TSCA mandates of CBI claims made in submissions under the CDR – which were collected three years ago – is resulting in the public being precluded from understanding the extent of consumer uses of this chemical. EPA should complete its review of those CBI claims in a timely manner.

10. EPA needs to ensure that environmental justice is appropriately considered, analyzed, and addressed in the risk evaluation.

Environmental justice is "the fair treatment and meaningful involvement of all people regardless of race, color, national origin, or income with respect to the development, implementation and enforcement of environmental laws, regulations and policies."[188] 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."[189] EPA has committed to integrate environmental justice into "everything" the agency does in order to "reduce[ ] disparities in the nation's most overburdened communities."[190]

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

[188] EJ 2020 GLOSSARY, https://www.epa.gov/environmentaljustice/ej-2020-glossary.
[189] *Id.*
[190] 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. In addition, EPA does not appear to have undertaken any outreach oriented towards ensuring the meaningful involvement of environmental justice communities in the risk evaluation process. EPA must address environmental justice in the risk evaluation, both by incorporating an analysis into the evaluation and ensuring meaningful involvement by environmental justice communities in the development of the risk evaluation.

A. The risk evaluation is subject to Executive Order 12898.

Executive Order 12898 directed federal agencies to identify and address "disproportionately high and adverse human health or environmental effects of its programs, policies, and activities on minority populations and low-income populations." Exec. Order No. 12898, 59 Fed. Reg. 7629 (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 evaluations undoubtedly fall within this capacious definition, qualifying as "activities" of EPA, carried out as part of its "programs" and pursuant to its "policies." As agency actions that may affect the level of protection provided to human health or the environment, the risk evaluations under TSCA must address environmental justice communities.[191] EPA's own guidance on considering environmental justice defines "agency action" to include risk assessments.[192] 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.[193] 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

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[191] *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.
[192] *Id.* at 1.
[193] U.S. EPA, Risk Evaluation Rule Response to Comments at 1, https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0654-0109.

of pollution on "minority populations and low-income populations."[194] The failure to do so in the draft risk evaluation, in particular by failing to consider minority, low-income, and indigenous communities when identifying potentially exposed or susceptible populations, does not "carry out the spirit," or the letter, of the Executive Order. EPA must prepare an actual environmental justice analysis to comply with the Executive Order.

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.[195]

Those gaps in coverage were often a result of controlling pollution solely "through technology-based regulation or an individual chemical-by-chemical approach."[196] 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.

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[194] Exec. Order No. 12898; *see also* U.S. Office of Inspector General, *EPA Needs to Consistently Implement the Intent of the Executive Order on Environmental Justice* at 9-10 (Mar. 2004), https://www.epa.gov/sites/production/files/2015-10/documents/20040301-2004-p-00007.pdf (explaining that the intent of the Executive Order, in part, was to place EPA's focus on minority and low-income communities).

[195] National Environmental Justice Advisory Council, Cumulative Risks/Impacts Work Group, *Ensuring Risk Reduction in Communities with Multiple Stressors: Environmental Justice and cumulative Risks/Impacts* at 7 (Dec. 2004), https://www.epa.gov/sites/production/files/2015-04/documents/ensuringriskreducationnejac.pdf (emphasis added).

[196] *Id.* at 11.

In addition to the general gaps in coverage, environmental justice communities are often disproportionately exposed to sources of chemical contamination. For instance, a report by the General Accounting Office revealed that:

- three-quarters of hazardous waste landfill sites in eight southeastern states were located in communities whose residents were primarily poor and African-American or Latino, and
- race and ethnicity were the most significant factors in deciding where to place landfills, waste and environmentally hazardous facilities.[197]

EPA's exclusion from the draft risk evaluation of exposure pathways resulting from environmental releases—particularly the exclusion of the ambient air pathway despite extensive air 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."[198]

Moreover, EPA's exclusions of exposure pathways linked to disposal sites 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.[199]

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[197] 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.
[198] EJ 2020 GLOSSARY, https://www.epa.gov/environmentaljustice/ej-2020-glossary.
[199] National Environmental Justice Advisory Council, Cumulative Risks/Impacts Work Group, *Ensuring Risk Reduction in Communities with Multiple Stressors: Environmental Justice and cumulative Risks/Impacts* at 24 (Dec. 2004), https://www.epa.gov/sites/production/files/2015-04/documents/ensuringriskreducationnejac.pdf (emphasis added).

Failing to consider exposures linked to disposal systematically underestimates the background level of exposures faced by many environmental justice communities. In order to determine whether those communities will face an unreasonable risk of injury from the chemicals undergoing risk evaluation, EPA must consider exposures from disposal.

11. Assessment factors do not lead to conservative calculations; in fact, assessment factors account for real-world sources of variability as well as database limitations.

In the problem formulation, EPA states that it used "conservative assumptions" when assessing aquatic environmental exposures. *See, e.g.*, Problem Formulation for 1-BP at p. 35. These types of 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 are used to calculate "lower bound effect levels (referred to as the concentration of concern; COC) that would likely encompass more sensitive species not specifically represented by the available experimental data. Also, assessment factors are included in the COC calculation to account for differences in inter- and intra-species variability, as well as laboratory-to-field variability." *Id*. at 42. 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.[200]

In evaluating risks, EPA should recognize that AFs ensure greater accuracy and do not provide a safety factor rendering the evaluation "conservative."

12. 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 states 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 analyzing the human and animal data that meet the systematic review data quality criteria described in the *Application of Systematic Review in TSCA Risk Evaluations* (U.S. EPA, 2018) document. *** 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-BP at p. 69. EPA has not explained, either here or in its OCSPP 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 B at 55-57, and EPA should implement those requirements consistent with that approach.

13. 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;

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[200] 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).

and (3) data integration of the scientific data used in risk evaluations developed under TSCA." Problem Formulation for 1-BP at p. 15.

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).[201]
>
> 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.[202]

EPA's IRIS program reflects this NAS recommendation by developing problem formulation and assessment protocols for each of its assessments.[203] OCSPP needs to develop full protocols for each of its risk evaluations, and should consult with the IRIS program on how best to do so in consideration of requirements under TSCA.

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[201] 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).
[202] *Id.* at 6 (emphases added).
[203] 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.

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 asserts that data integration will use systematic review methods, but EPA never explains how data integration will occur. For example, EPA states:

> 6) Evaluate the weight of the evidence of environmental release data.
> EPA will rely on the weight of the scientific evidence when evaluating and integrating environmental release data. 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.

Problem Formulation for 1-BP at p. 59; *see also* Problem Formulation for 1-BP at pp. 60, 62, 66, 68, 70.

In fact, EPA has not described its approach to data (evidence) integration in its problem formulation, 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.[204]

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EDF appreciates the opportunity to provide comments and EPA's consideration of them.

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[204] 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.