

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
Comments of Safer Chemicals Healthy Families et al on EPA's Draft Risk
Evaluation for 1-Bromopropane (1-BP) under Section 6(b) of TSCA

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Safer Chemicals Healthy Families (SCHF), Natural Resources Defense Council, Earthjustice and Environmental Health Strategy Center submit these comments on EPA's draft risk evaluation for 1-bromopropane (1-BP) under section 6(b) of the Toxic Substances Control Act (TSCA).¹ Our organizations are national, state and local groups committed to assuring the safety of chemicals used in our homes, workplaces and the many products to which our families and children are exposed each day. We took a leadership role during the TSCA legislative process, advocating the most protective and effective legislation possible to reduce the risks of toxic chemicals in use today. We strongly support a proactive approach to implementing the new law that uses the improved tools that Congress gave EPA to deliver significant health and environmental benefits to the American public.

1-BP is a solvent with widespread consumer and industrial uses and significant potential for exposure. There are long-standing concerns about 1-BP's harmful effects on human health, as EPA concluded in its 1-BP risk assessment in 2016. The draft risk evaluation confirms and restates these concerns, finding that 1-BP causes cancer, reproductive harm, damage to developing fetuses, and kidney, liver and neurological effects. Accordingly, it concludes that 1-BP presents an unreasonable risk of injury for several use and exposure scenarios under TSCA.

The EPA Scientific Advisory Committee on Chemicals (SACC) reviewed the draft evaluation on September 10-12, 2019. According to SACC members and comments by stakeholders, while EPA's draft correctly identifies 1-BP's harmful human health effects, it understates the risks that these effects pose to workers, consumers and vulnerable subpopulations. We urge EPA to respond to these concerns in its final evaluation and make upward adjustments in its estimates of risk. Even without these adjustments, it is clear that the combination of serious health effects and high exposure should compel EPA to ban all consumer and most industrial uses of 1-BP under section 6(a) of TSCA.

Our comments make the following key points:

- The draft risk evaluation confirms that 1-BP has several serious health effects, including a high likelihood that pregnant women and fetuses will suffer severe harm as a result of short-term exposure. While EPA continues work on the evaluation, the Agency must take immediate action to warn the public of these risks and urge manufacturers and users to greatly reduce or eliminate acute exposure to 1-BP. The dangers of acute exposure to 1-

¹ 84 Federal Register 39830 (August 12, 2019). https://www.epa.gov/sites/production/files/2019-08/documents/01_1-bp_draft_risk_evaluation_hero_links_external.pdf.

BP are too serious to delay action until completion of the risk evaluation and follow-up rulemaking, which could take several years.

- EPA has excluded air emissions from the scope of the draft 1-BP evaluation with no legal or scientific justification. This exclusion disregards an important contributor to human exposure and risk. There is considerable evidence that 1-BP air emissions are significant and widespread and represent a major pathway of exposure to the general population and vulnerable subpopulations. These emissions also add to other sources of exposure by workers and consumers who are already at high risk of adverse health effects. Failure to account for air emissions in EPA's risk evaluation is both a violation of TSCA and a major gap in public health protection.
- The draft evaluation concludes that inhalation margins of exposure (MOEs) are below the benchmark MOE by up to three orders of magnitude for virtually all workers and many occupational bystanders. It also concludes that inhalation and dermal cancer risks are above 1 in 10,000 (1×10^4) for nearly all manufacturing and use activities. However, EPA then adjusts its MOEs and cancer risk estimates on the unsupported and unrealistic assumption that workers will continuously use respirators and gloves that protect them from exposure. There is no legal requirement for respirator and glove use during exposure to 1-BP and EPA itself acknowledges that there is considerable uncertainty whether workers will continuously wear personal protective equipment (PPE). Thus, EPA's determinations of unreasonable risk for 1-BP should be based on workplace exposure levels in the absence of PPE – an approach that requires EPA to conclude that nearly all workers face unreasonable risks.
- Although finding that 1-BP poses serious risks of several adverse health effects, EPA underestimates the magnitude of these risks in key respects. For example, EPA overlooked significant contributors to consumer exposure, such as concurrent use of multiple products and repeated use scenarios resulting in chronic exposure. Similarly, EPA failed to combine its cancer risk estimates for inhalation and dermal contact, even though these two types of exposure occur concurrently for workers and consumers. Correcting these and other mistakes would significantly increase non-cancer and cancer risks relative to the EPA benchmarks.
- The draft risk evaluation emphasizes the strong weight of the evidence for 1-BP's neurotoxicity in animal and human studies. However, it uses only rat studies on 1-BP to calculate an MOE and does not rely on human studies that showed neurotoxic effects at levels 10X below concentrations producing these effects in rats. Human evidence is generally preferable to animal data and, in this instance, multiple human studies are available that show consistent evidence of neurotoxicity in exposed workers at low concentrations. The most health protective approach is to use these studies to determine the MOE for this endpoint.

- As demonstrated in the draft risk evaluation, 1-BP has been shown to be a multi-site carcinogen in rats and mice. EPA used a linear low-dose extrapolation method to estimate cancer risks at occupational exposure levels. At the SACC meeting, industry commenters took issue with this approach, arguing that there is evidence for a threshold Mode of Action (MOA). EPA properly rejected this position in the draft evaluation, concluding that the weight of the evidence favors a mutagenic MOA and the evidence for a threshold MOA is weak and inconclusive.
- The draft evaluation concludes “that 1-BP does not present unreasonable risk to the environment under the identified conditions of use.” The data supporting this determination are weak and limited and derive almost entirely from ECHA dossiers summarizing industry studies that EPA could not obtain and that neither it nor the public has reviewed. To rely on these unseen studies for determinations of unreasonable risk would violate rudimentary data quality and reliability standards and TSCA’s mandate to base decisions on the “best available science.” EPA should give no weight to the ECHA studies and determine that it lacks evidence to conclude that 1-BP does not present an unreasonable risk to the environment. Using its authority under section 4, the Agency should require industry to conduct aquatic toxicity studies that provide a sound scientific basis for evaluating environmental risk.
- EPA continues to apply a flawed systematic review method despite serious concerns raised by commenters and the SACC. It is critical that EPA address the SACC recommendations in its peer review report on Pigment Violet 29 in all of the ongoing risk evaluations and proceed with the delayed National Academy of Sciences (NAS) review to which EPA committed several months ago.

I. Acute Exposure to 1-BP Presents an Imminent and Substantial Risk of Reproductive and Developmental Harm Requiring Immediate Protection of Workers and Consumers

While the draft risk evaluation confirms that 1-BP has several serious health effects, one finding stands out as raising immediate concern: according to the evaluation, there is a high likelihood that pregnant women and fetuses will suffer severe harm as a result of short-term exposure to 1-BP. While EPA continues work on the evaluation, the Agency must take immediate action to warn the public of these risks and urge manufacturers and users to greatly reduce or eliminate acute exposure to 1-BP. The dangers of acute exposure to 1-BP are too serious to delay action until completion of the risk evaluation and follow-up rulemaking, which could take several years.

A. The Draft Evaluation Concludes that There is Strong Evidence of a Causal Connection between Reproductive and Developmental Risks and Acute Exposure

The draft risk evaluation states that “[r]eproductive and developmental toxicity were identified as critical targets for 1-BP exposure based on a constellation of effects reported across studies, including a two-generation reproduction study (WIL Research, 2001), which showed adverse effects on male and female reproductive parameters, and the developing conceptus” (p. 160). According to the Agency, “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).” EPA concluded that there was “high confidence” in these studies because they “were of longer duration with effects observed more consistently than other high-quality studies that were evaluated.”

The Agency emphasized that “[e]vidence supporting fetal development as a sensitive target of 1-BP exposure is provided by a number of laboratory animal studies.” It elaborated that:

Overall, the general consistency of findings indicative of impaired development across species, as reported in multiple studies from independent laboratories, is taken as evidence of a causative association between 1-BP exposure and developmental toxicity.
(Emphasis added) (p.160)

EPA then assessed reproductive and development risks for both acute and chronic exposure. Explaining its decision to evaluate risks of acute effects, the Agency said that “multiple publications suggest that some developmental effects (e.g., decreased live litter size and increased post-implantation loss) may result from a *single exposure during a critical window of development.*” (Emphasis added) (p.185). The Agency indicated that developmental effects were “considered the most sensitive [endpoints] identified for an acute exposure duration and are considered to be biologically relevant to the potentially exposed or susceptible subpopulation (i.e., adults of reproductive age and their offspring).”

To examine these acute risks, EPA selected Points of Departure (PODs) for acute developmental effects² from animal studies and, after making adjustments to reflect differences in metabolism and uptake between rodents and humans, converted them to a Human Equivalent Concentration (HEC) for inhalation and a Human Equivalent Dose (HED) for dermal exposure. Expressed as an 8-hour HEC, EPA determined that the POD for the decreased live litter size was a Benchmark Concentration lower confidence limit (BMCL) of 31 ppm and the POD for the increased post-implantation loss was a BMCL of 24 ppm. (p.166). The HEDs calculated by EPA were 19 mg/kg/day (decreased litter size) and 11/mg/day (post implantation loss). EPA used the 8-hour HECs and HEDs to assess risks for acute occupational exposure.

EPA also calculated 24-hour HECs and HEDs to assess the risks of consumer exposure. The 24-hour HECs were 6 ppm (decreased live litter size) and 10 ppm (post-implantation loss). The 24-hour HEDs were 19 mg/kg/day and 11/mg/day, the same as the 8-hour values.³

² “EPA defines a POD as the dose-response point that marks the beginning of a low-dose extrapolation. This point can be the lower bound on the dose for an estimated incidence, or a change in response level from a dose-response model (i.e., BMD), a NOAEL or a LOAEL for an observed incidence or change in the level of response.” (p. 164)

³ EPA apparently failed to convert the worker HEDs to a 24-hour HED for consumers. Had it done so, the HEDs would have been significantly lower.

B. The Evaluation Demonstrates That Acute MOEs for Exposed Workers and Consumers Are Well Below Benchmarks

Having determined PODs/HECs for acute developmental effects, EPA then examined how they compared to human exposure levels resulting from consumer product use and 1-BP’s presence in the workplace.

Consumer Product Exposure. According to the draft evaluation, 1-BP is used in the following consumer products:

Table 2-42. Consumer Uses and Routes of Exposure Assessed

Consumer Uses	Routes of Exposure
<ol style="list-style-type: none"> 1. Adhesive Accelerant (Liquid Pump Spray) 2. General Purpose Spray Cleaner (Liquid Spray/Aerosol) 3. Spot Cleaner and Stain Remover (Liquid Spray/Aerosol) 4. Mold Cleaning and Release Product (Liquid Spray/Aerosol) 5. General Cleaners and Degreasers (Liquid Spray/Aerosol) 6. Electronics Degreasers (Liquid Spray/Aerosol) 	Inhalation and Dermal
<ol style="list-style-type: none"> 7. Coin and Scissors Cleaner (Liquid Bath) 8. Automobile AC Flush (Liquid) 	Inhalation and Dermal
<ol style="list-style-type: none"> 9. Insulation (Off-gassing) 	Inhalation

Using established modeling techniques, EPA determined low, medium and high intensity use scenarios for short-term exposures to each product type and calculated corresponding 24-hour Time Weighted Average (TWA) exposure levels. These scenarios were developed for both direct users and bystanders and for inhalation (all uses) and dermal (3 uses) pathways of exposure. EPA then compared its product-by-product exposure estimates to the HECs/HEDs and derived margins of exposure (MOEs) – a ratio of the HEC/HED to human exposure levels. To determine how risky the consumer uses are, EPA compared the actual MOEs to a “benchmark MOE” of 100.⁴ This benchmark accounts for “uncertainty/ adjustment factors” in predicting effects in humans on the basis of animal studies and is used by EPA as a ‘yardstick’ for determining the unreasonableness of risks.

For numerous products and use scenarios, EPA’s draft risk evaluation found that actual consumer exposures were above or alarmingly close to the HEC/HED. As a result, in many cases, the MOEs were below 1 and, in nearly all instances, were far lower than the benchmark EPA used to determine unreasonable risk.

Figures 1a and 1b compare the MOE’s for high, moderate, and low intensity use of 1-BP for both users and bystanders to the benchmark MOE of 100 (represented by the red line). These charts

⁴ The benchmark MOE of 100 reflects a 10X UF for inter- and intra-species variability and a 10X UF to account for variation in sensitivity within human populations. (p. 191-192). Arguably, EPA should have applied another 10X UF for extrapolation from a Lowest Observed Adverse Effect Level (LOAEL) to a No Observed Adverse Effect Level (NOAEL).

Figure 1a. Risks of concern for developmental effects (post-implantation loss) by consumer product users for all uses and scenarios

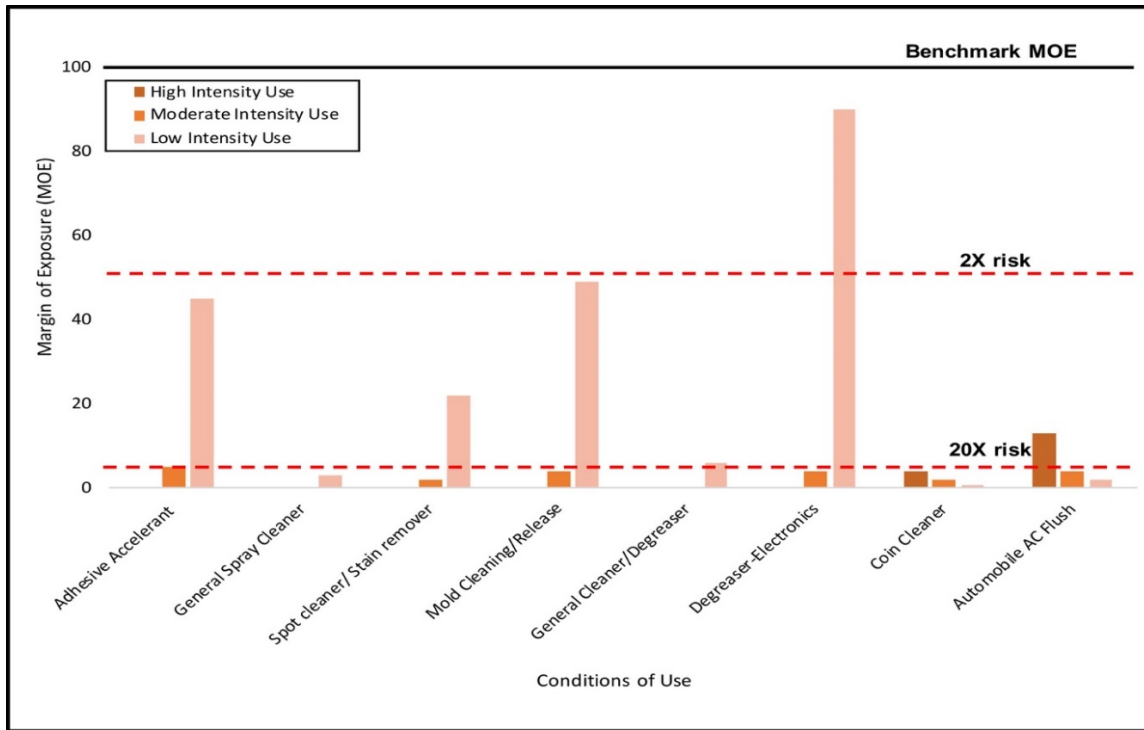
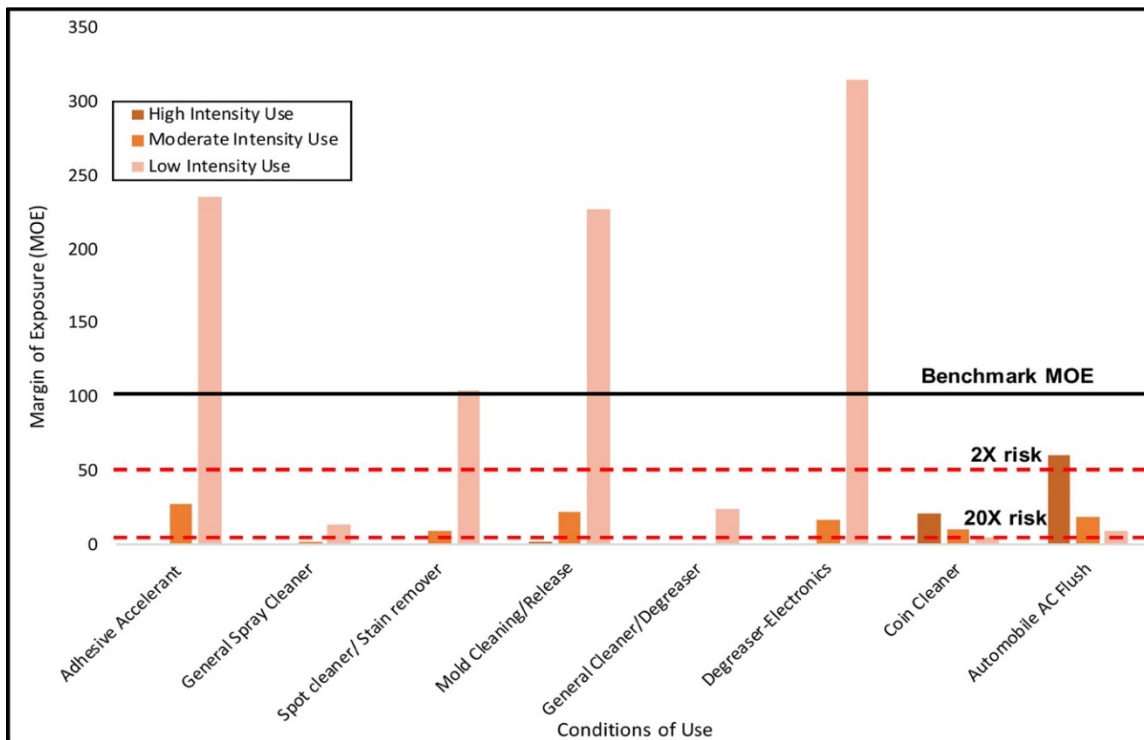


Figure 1b. Risks of concern for developmental effects (post-implantation loss) by consumer product bystanders for all uses and scenarios

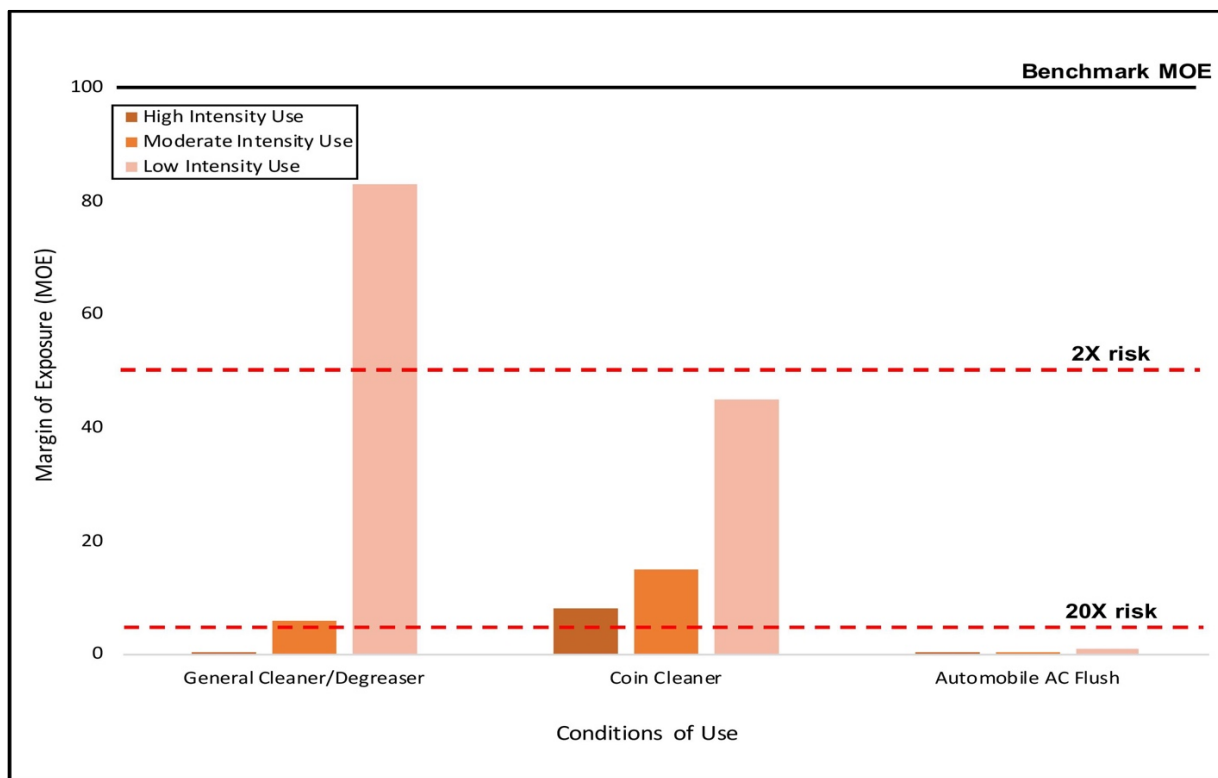


represent a subset of the data on 1-BP's developmental effects, specifically post-implantation loss for the F0 generation (the originally affected generation, not subsequent birth cohorts).

As shown in Figures 1a and 1b, MOEs were below the benchmark for all high- and medium-intensity use scenarios and nearly all low-intensity use scenarios. In almost all cases, MOEs were not protective for both direct consumer users and bystanders and for both of the adverse developmental effects (reduced litter size and post-implantation loss) linked to acute 1-BP exposure.

As shown in Figure 2 below, EPA also calculated dermal exposure MOEs for three types of consumer products (general cleaner/degreaser, coin cleaner, and automobile AC Flush) for which it determined that exposure by this route was likely.⁵

Figure 2. Non-cancer risk estimates for 24-hr dermal exposure following adult consumer uses of 1-BP



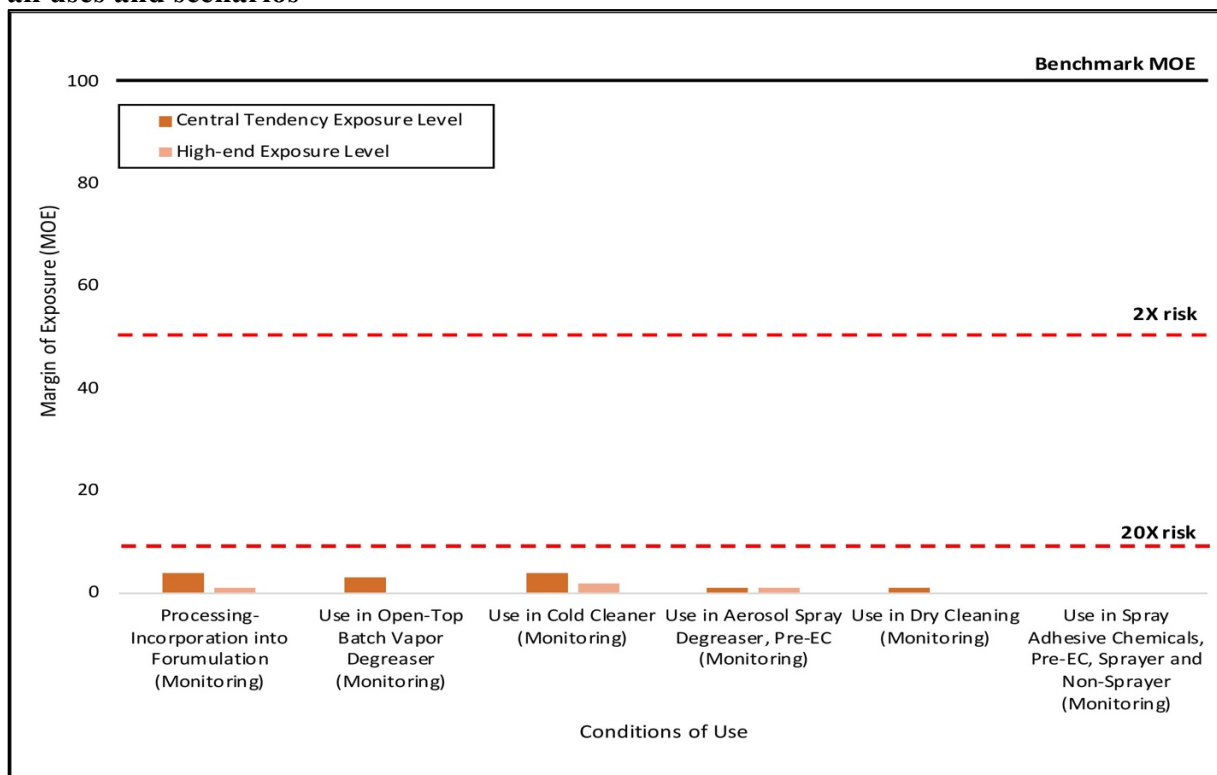
Again, most MOEs were well below the benchmark and some were below the HED, meaning that human exposure levels were higher than the doses predicted to cause adverse effects. As all of these products also result in inhalation of 1-BP, the risks of dermal exposure would be additive, making the overall MOE even smaller (and the use/exposure riskier).

⁵ It is not explained why EPA felt that the other consumer products lacked the potential for dermal exposure. By their very nature, these products would seem likely to result in inhalation and dermal contact during use.

Worker Exposure. The draft evaluation also estimated developmental and reproductive risks to workers for industrial and commercial applications of 1-BP, including several using open processes with the potential for significant worker exposure.

For each of these 1-BP applications, the draft risk evaluation used monitoring data (where available) and modeling to estimate acute exposure levels for exposed workers. Using the same procedure it applied to consumer products, it then compared these levels to 8-hour HECs representing the exposure levels at which acute developmental and reproductive effects would be expected to occur in workers inhaling 1-BP. Using this comparison, EPA calculated MOEs for each exposure scenario and acute developmental effect and determined whether these MOEs were above or below the benchmark MOE of 100. As shown in Figure 3, **MOEs were again below the benchmark by up to three orders of magnitude for all workers and many occupational non-users (ONUs) and in a few instances were 1 or even below (meaning that actual exposures were equal to or higher than the HEC).**

Figure 3. Risks of concern for developmental effects (post-implantation loss) workers for all uses and scenarios



C. EPA Must Take Immediate Action to Protect Exposed Workers and Consumers

In the face of these unequivocal findings,⁶ EPA must take three immediate actions to protect pregnant women and fetuses from acute exposure to 1-BP while it finalizes its risk evaluation and completes rulemaking under TSCA section 6(a):

- EPA should list 1-BP under section 5(b)(4) of TSCA as a chemical that “present[s] or may present an unreasonable risk to human health and the environment.” This listing will increase the transparency of EPA’s decision making, provide additional disclosure of exports of products containing 1-BP, and enhance awareness of the harmful effects of acute exposure.
- EPA should issue and broadly disseminate a health advisory that warns the public of 1-BP’s risks to fertility and fetal development following acute exposure and urges women of child-bearing age to avoid exposure to these products if they are present in their homes.
- At the same time, the Agency should send letters to all 1-BP manufacturers, industrial users and manufacturers of BP-containing consumer products that:
 - (1) urge that retailers and distributors stop sales of consumer products containing 1-BP;
 - (2) call upon manufacturers, processors and commercial users to take immediate steps to reduce workplace concentrations of 1-BP below the NIOSH Recommended Exposure Limit (REL) of 0.3 ppm, placing principal reliance on engineering controls, and implement comprehensive safety and health programs that include worker education and training, hazard communication, and exposure monitoring;
 - (3) advise manufacturers and distributors of 1-BP and all products containing the chemical to immediately revise product labels and Safety Data Sheets (SDSs) to prominently warn workers of 1-BP’s acute reproductive and developmental hazards and recommend immediate reductions in exposure below the NIOSH REL, backed up by worker training, education and monitoring; and
 - (4) urge firms using 1-BP to investigate and adopt safer substitutes.

While EPA could initially seek voluntary industry commitments to implement these measures, it should not hesitate to make them mandatory using its TSCA section 7 “imminent hazard” authority if firms fail to act quickly and effectively to protect workers and consumers. Since it gives rise to acute exposure, the threat 1-BP poses to pregnant women and their offspring is imminent. The effects of concern are severe, and their occurrence is likely based on EPA’s own risk evaluation. Thus, acute exposure to 1-BP plainly satisfies the TSCA section 7(b) definition of an “imminently hazardous chemical substance.”

⁶ The signatories to these comments and several other groups wrote to EPA Administrator Wheeler on October 9 to request immediate action by the Agency. The letter and backup paper accompanying it are attached to this petition.

We believe that distributors and retailers should immediately remove all consumer products containing 1-BP from commerce because no other step will meaningfully protect product users and bystanders. As the draft risk evaluation emphasizes, consumers are extremely unlikely to use protective equipment and, even if they did, the acute risks would be unacceptable under EPA's criteria. Product labels (which now are inadequate) would not likely change consumer behavior, as EPA has noted in previous section 6 proposals for other solvents, and bystanders would not see label warnings in any event. Since there are known safer substitutes for 1-BP in these applications, no possible justification exists for putting consumers at risk.

There is an equally compelling need for industry to immediately implement substantial reductions in workplace exposure. Deep reductions in exposure through engineering controls or safer alternatives are required now to reduce the risk and even these reductions may be inadequate to provide full protection against acute adverse reproductive and developmental effects.

We look forward to early action by EPA to address the imminent risks of acute 1-BP exposure.

II. EPA Significantly Understates Exposure and Risk by Excluding Air Emissions and other Environmental Releases from Its Risk Evaluation

EPA has excluded air emissions from the scope of the draft 1-BP risk evaluation on the ground that “the Clean Air Act adequately assesses and effectively manages risks to the general population . . . via the ambient air pathway.” (p. 38). This exclusion lacks a sound legal and scientific basis and disregards an important contributor to human exposure and risk. As discussed below, there is considerable evidence that 1-BP air emissions are significant and widespread and represent a major pathway of exposure by the general population and vulnerable subpopulations. These emissions also add to other sources of exposure by workers and consumers who are already at high risk of adverse health effects. Failure to account for air emissions in EPA's risk evaluation is thus a violation of TSCA and a major gap in public health protection, as SACC members commented during the September 10-12 meeting to review the draft evaluation.

A. 1-BP Air Emissions Are Significant and Result in Widespread Exposure

EPA's 2018 Problem Formulation for the 1-BP evaluation calls attention to its high volatility and emphasizes “air as a primary medium of environmental release.”⁷ Noting 1-BP's “relatively long hydroxy radical oxidation half-life (t_{1/2} 14 days),” the Problem Formulation underscores the contribution of facility emissions to “near facility human receptors and the general population.”⁸

Recent data from the Toxic Release Inventory (TRI) confirms the large volume of 1-BP air emissions. The chemical was added to the TRI in 2015 and the initial round of reporting occurred in 2017. Forty three (43) facilities reported fugitive emissions totaling 394,469 pounds

⁷ Problem Formulation of the Risk Evaluation for 1-Bromopropane, May 2018 at 39
https://www.epa.gov/sites/production/files/2018-06/documents/1bp_problem_formulation_05-31-18.pdf

⁸ Problem Formulation at 39.

and 26 reported stack emissions totaling 232,191 pounds.⁹ The Problem Formulation notes several reasons why the TRI reports likely understate air emissions. One is that many emitting facilities “could be below the threshold for reporting.”¹⁰ This would include small facilities using 1-BP as a vapor degreaser, as a component of adhesives and sealants, and in dry cleaning and other cleaning operations. If accounted for, these facilities would greatly increase the number of 1-BP emitting sources and the overall volume of emissions. For example, the draft risk evaluation estimates between 500 and 2500 establishments that use 1-BP for vapor degreasing and between 1000 and 5000 that use the chemical as an aerosol spray degreaser. (pp. 70, 83).

EPA’s earlier draft risk assessment on 1-BP likewise omitted air emissions. The 2016 review of the Chemical Safety Advisory Committee (CSAC) faulted the assessment because it:¹¹

fails to account for other potential sources/pathways of exposures to the general population (e.g., emissions from dry cleaning facilities exposing other building occupants or populations in close proximity) and assumes that exposures occur only in the workplace or in homes where there is direct use of spray adhesives, degreasers, and/or cleaners. 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.

The CSAC emphasized that “[i]t is, thus, highly likely that exposures to 1-bromopropane occur in populations living or working in close proximity to facilities using 1-BP” and that “exposures occurring in close proximity to facilities using 1-BP could result in a disproportionate health risk in low-income communities and communities of color . . .”¹²

As EPA notes in its draft evaluation,¹³ further evidence of the contribution of air emissions to general population exposure is provided by studies documenting urinary bromide concentrations in large numbers of workers, pregnant women, children and adults. In its 2016 review, CSAC summarized this evidence as follows:¹⁴

Supporting information for inclusion of chronic exposures to the general public includes biomonitoring studies evaluating the presence of a metabolite of 1-BP. The Committee noted that at least two recent studies have documented the presence of N-acetyl-S-(n-propyl)-l- cysteine, a urinary biomarker of exposure for 1-bromopropane in pregnant

⁹ Id. at 32-33.

¹⁰ Id.

¹¹ Minutes of the May 24-25, 2016 Chemical Safety Advisory Committee Meeting https://www.khlaw.com/Files/29464_Chemical_Safety_Advisory_Committee_Minutes.pdf, at 21.

¹² Id..

¹³ Risk Evaluation, at 148-149.

¹⁴ CSAC minutes , at 22-23.

mothers ((Boyle, Viet et al., 2016), data from the National Children’s Study) and in children 6-11 years from the general U.S. population (Jain, 2015). The authors reported detection frequencies of 99% and 60.8% in pregnant mothers and children, respectively. Additionally, 2011-2012 data from the National Health and Nutrition Examination Survey (NHANES) released by the Centers for Disease Control and Prevention (CDC, 2015) also provide supporting evidence that there is widespread exposure in the general U.S. population.

Although EPA erroneously decided not to rely on these data in its draft evaluation,¹⁵ it recognized that N-acetyl-S-(n-propyl)-l-cysteine “is considered to be a valid biomarker for 1-BP exposure,”¹⁶ that “urinary bromide concentration appears to be a useful index of 1-BP exposure” and that occupational exposure studies have “consistently identified significant correlations” between 1-BP concentration in the ambient air and levels of 1-BP or its metabolites in urine.”¹⁷ The CSAC reviewers of the 2016 draft risk assessment emphasized that, while not conclusive, these studies point toward the “possibility of low level but very widespread non-occupational exposure to 1-BP,” and recommended that these bio-monitoring studies be used as “[s]upporting information for inclusion [in the risk assessment] of chronic exposures to the general public.”¹⁸ EPA must consider this bio-monitoring data to establish background levels of 1-BP and to evaluate risks to potentially exposed and susceptible subpopulations, including workers, pregnant women, and children that are already have 1-BP in their blood from air emissions.

B. Air Emissions Likely Present a Significant Cancer Risk to the General Population

Available data also provides a basis to estimate 1-BP air concentrations in communities near emitting facilities and to determine risks of cancers and other adverse effects. The NTP (2013) monograph on 1-BP noted that "EPA has estimated 1-bromopropane concentrations in ambient air at a distance of 100 meters from average-adhesive use model facilities via air dispersion modeling to be 0.138 mg/m³ [0.0274 ppm] and 1.38 mg/m³ [0.274 ppm] for high- adhesive use facilities (Wolf et al., 2003; also cited as Morris and Wolf, 2003 in NTP's 13th Report on Carcinogens)." ¹⁹

More recently, the Halogenated Solvents Industry Alliance (HSIA), one of the parties petitioning to list 1-BP as a Hazardous Air Pollutant (HAP) under the Clean Air Act (CAA), submitted an exposure and risk assessment to EPA in support of its petition. HSIA estimated 1-BP emissions

¹⁵ EPA based this decision on concern that N-acetyl-S-(n-propyl)-l-cysteine may be a urinary marker for not just 1-BP but other brominated compounds but it could have used the three years following the CSAC report to require further research to confirm its specificity for measuring 1-BP.

¹⁶ Risk Evaluation at 148.

¹⁷ Id at 146.

¹⁸ CSAC Minutes, at 14, 22.

¹⁹ NTP. (2013). National Toxicology Program Report on Carcinogens Monograph on 1-Bromopropane. NIH Publication No. 13-5982. September 2013.

for five representative facilities: a narrow tube manufacturing/ degreasing operation, two dry cleaners, and two furniture manufacturing/spray adhesive facilities. It then used the latest version of the EPA's Human Exposure Model (HEM) to model facility emissions and estimate downwind concentrations at actual residences near the facilities. Applying a cancer unit risk estimate to the modeled ambient concentrations, the assessment concluded that "9,000 people [are] estimated to have cancer risk greater than 1-in-1 million."²⁰ If a similar method were used to estimate cancer risk for the thousands of other 1-BP user facilities, the at risk population would be far higher.

As the HSIA assessment shows and the CSAC review concludes, 1-BP air emissions likely present a significant risk of cancer and other adverse health effects to the general population. This risk is additive to the unacceptable cancer and other risks that users of consumer products and workers already face based on the draft evaluation. Moreover, given 1-BP's neurotoxic effects and developmental and reproductive toxicity, people living near emitting facilities -- and children and pregnant women in particular -- represent "potentially exposed or susceptible subpopulations" for which special protection is required in TSCA risk evaluations. Failure to account for health risks from air emissions would thus result in a flawed and incomplete evaluation that ignores the "best available science" and understates 1-BP's risks to public health.

C. The Pending Petition to List 1-BP as a HAP Does Not Justify Excluding Air Emissions from the Draft Evaluation

After initially deciding in the Problem Formulation to address air emissions, EPA has now reversed course in the draft evaluation, arguing that air emissions should be excluded on the ground that:²¹

. . . in December 2016, EPA, through its Office of Air and Radiation (OAR), issued a draft notice of the agency's rationale for granting petitions to add 1-BP to the list of HAPs contained in section 112(b)(1) of the CAA. Under Section 112 of the CAA, EPA is required to regulate and control emissions of listed hazardous air pollutants. After further consideration of the likely future applicability of EPA regulations to 1-BP EPA has determined that the Clean Air Act adequately assesses and effectively manages risks to the general population and the environment for terrestrial receptors via the ambient air pathway. As a result, EPA has modified the conceptual models since the problem formulation. Based upon consultation within the Agency, OCSPP understands from OAR that the Agency is finishing review of comments on this draft notice and intends to finalize an action before the end of 2019.

EPA wrongly claims that the potential for future CAA regulation eliminates the need to evaluate air-related risks under TSCA.

²⁰ 82 Federal Register 2354, 2361 (January 9, 2017) (preliminary EPA decision granting petition).

²¹ Risk Evaluation at 38.

First of all, 1-BP has not yet been listed as a HAP. The petition for HAP listing has been pending since 2010. While the Obama EPA made a preliminary decision to grant the petition in December 2016,²² the Trump EPA has taken no action since then and, given its overall track record, there is no guarantee that it will finalize the listing in 2019. To exclude an important risk pathway from evaluation under TSCA based on a future regulatory action that has been pending for nearly a decade and may not happen is reckless and unjustified.

Even if 1-BP were listed as a HAP, the premise that regulation under the CAA adequately protects the general population and eliminates the need to evaluate the contribution of air emissions to overall risk under TSCA is without basis and ignores the purpose of TSCA risk evaluations. Under section 6(b)(4)(A), these evaluations must determine “whether a chemical substance presents an unreasonable risk of injury to health or the environment” – a requirement that entails examining all sources of exposure to the substance. Similarly, section 6(b)(4)(A) provides that a risk evaluation must determine the substance’s risks under “the conditions of use.” This broad term spans the entire life cycle of a chemical and is defined under section 3(4) to mean “the circumstances . . . under which a chemical substance is intended, known, or reasonably foreseen to be manufactured, processed, distributed in commerce, used, or disposed of.” These “circumstances” clearly include air emissions that result in pathways of human exposure, whether they might be addressed under the CAA or not.

If Congress had intended a blanket exemption of air emissions from risk evaluations under section 6(b), it surely would have said so explicitly, given the far-reaching impact of such an exemption. But not only is there no such exemption in the law but its legislative history and structure demonstrate that Congress intended TSCA to provide a comprehensive framework for identifying and managing chemical risks, including those that could be addressed under other environmental laws like the CAA.

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

²² See note 20, *supra*.

In amending TSCA in 2016, Congress sought to promote “effective implementation” of the 1976 law’s objectives. See S. Rep. No. 114-67, 114th Cong., 1st Sess. (2015) at 2. At the time it strengthened TSCA, Congress affirmed that the intent of the original law—to give EPA “authority to look at the hazards [of chemicals] in total,” S. Rep. No. 94-698, at 2—remained “intact.” S. Rep. No. 114-67, at 7. Indeed, in a statement accompanying the law’s passage, its Senate Democratic sponsors underscored that, with the expanded authorities conferred by Congress, TSCA should not be “construed as a ‘gap filler’ statutory authority of last resort” but “as the primary statute for the regulation of toxic substances.”²³

EPA’s position that other environmental laws should displace TSCA risk evaluations arbitrarily assumes that these laws provide equivalent protection of public health and the environment and that there is no added benefit in evaluating the risks presented by environmental pathways of exposure under TSCA. But in reality, these other laws vary greatly in the degree of protection they afford against chemical risks and the extent of their application to unsafe chemicals. These limitations are precisely why Congress gave EPA broad authority over chemical risks under TSCA in 1976 and strengthened that authority in 2016 by requiring EPA to conduct comprehensive risk evaluations on chemicals of concern.

In this case, a HAP listing will not assure that EPA evaluates the health risks of 1-BP emissions or takes effective action to protect the exposed population from unreasonable health risks. Title III of the CAA initially mandates technology-based -- not risk based – emission limits. Once these limits are in place, the law gives EPA at least eight more years to evaluate residual risks and set risk-based emission standards under CAA section 112(f).²⁴ In contrast to TSCA risk evaluations, these standards would only consider emission-related risks, and thus would not take into account aggregate health risks from all sources of exposure.

Moreover, EPA’s emission standards would apply only to “major” sources, which are defined as facilities that emit over 10 tons per year of any single HAP or 25 tons per year of all HAPs.²⁵ This definition would likely not cover the thousands of smaller establishments that in the aggregate account for substantial 1-BP air emissions. It is possible that these facilities could be regulated under the CAA as “area sources” but whether and when their emissions would be restricted and how much protection EPA must afford are highly uncertain.²⁶

In sum, even if 1-BP is eventually listed as a HAP, its regulation under the CAA would be inadequate to compensate for EPA’s failure to evaluate the contribution of air emissions to overall exposure and risk under TSCA. TSCA does not permit EPA to evaluate whether 1-BP

²³ Congressional Record – Senate 3517 (June 7, 2016).

²⁴ 42 U.S.C. § 7412(f)(2).

²⁵ 42 U.S.C. § 7412(a)(1).

²⁶ EPA has no obligation under 112(f) to set risk-based standards for emissions of 1-BP from area sources. Instead, EPA’s only mandatory obligations to address risk from emissions from area sources are contained in 112(k), which requires EPA to reduce cancer- and non-cancer risk from exposure to all hazardous air pollutants in the aggregate, but would permit EPA to allow unreasonable risk from 1-BP to persist, so long as it sufficiently reduces risk from other pollutants.

“presents”—i.e. at this moment—an unreasonable risk of injury on the basis of uncertain and speculative future events that are, at best, years off. EPA should therefore include these emissions in its ongoing risk evaluation for 1-BP and determine their contribution to cancer and non-cancer health risks, both in themselves and in combination with other exposure and risk pathways for workers, consumers and communities.

III. EPA Should Determine that All Worker Exposures Exceeding Its Risk Benchmarks Present Unreasonable Risks under TSCA, Without Taking into Account Personal Protective Equipment

The draft evaluation concludes that inhalation MOEs are below the benchmark MOE by up to three orders of magnitude for virtually all workers and many occupational bystanders. It also concludes that inhalation and dermal cancer risks are above 10^{-4} for nearly all manufacturing and use activities.²⁷ However, EPA then adjusts its MOEs and cancer risk estimates on the unsupported and wholly unrealistic assumption that workers will continuously use respirators and gloves that protect them from exposure. As shown below, there is no legal requirement for respirator and glove use during exposure to 1-BP and EPA itself acknowledges that there is considerable uncertainty whether workers will continuously wear personal protective equipment (PPE). Thus, consistent with OSHA policy, EPA’s determinations of unreasonable risk for 1-BP should be based on workplace exposure levels in the absence of PPE – an approach that requires EPA to conclude that nearly all workers face unreasonable risks.

A. The Draft Evaluation Shows that 1-BP Presents Significant Risks of Multiple Adverse Health Effects to Nearly All Exposed Workers

The draft risk evaluation identifies numerous industrial and commercial applications of 1-BP. Several of these applications involve open processes with the potential for significant worker exposure, such as:

- Formulation of mixtures containing 1-BP

²⁷ EPA’s decision to select 10^{-4} as its benchmark for unreasonable cancer risks to exposed workers is unjustified. As explained in the draft evaluation, “Standard cancer benchmarks used by EPA and other regulatory agencies are an increased cancer risk above benchmarks ranging from 1 in 1,000,000 to 1 in 10,000 (i.e., 1×10^{-6} to 1×10^{-4}) depending on the subpopulation exposed. Generally, EPA considers 1×10^{-6} to 1×10^{-4} as the appropriate benchmark for the general population, consumer users, and non-occupational potentially exposed or susceptible subpopulations (PESS)” (p. 257). EPA cites NIOSH and OSHA practice as precedent for using 1×10^{-4} as the unreasonable risk threshold for cancer in the workplace. However, EPA does not explain why this precedent should control decision-making under TSCA, a different law, or why workers should receive less protection than other exposed subpopulations. Moreover, because workers are exposed to 1-BP not just in the workplace but through air emissions and other pathways, they should receive the level of protection as the general population for the combination of pathways contributing to the overall cancer risk they face.

- Industrial and commercial use as solvent for cleaning and degreasing, including vapor degreaser (batch vapor degreaser – open top and closed loop, inline vapor degreaser), cold cleaner aerosol spray degreaser/cleaner.
- Industrial and commercial use as adhesives and sealants.
- Industrial and commercial use as cleaning and furniture care products, including dry cleaning, spot cleaner and other liquid, spray and aerosol cleaners.
- Other industrial and commercial uses: arts, crafts, hobby materials (adhesive accelerant); automotive care products (engine degreaser, brake cleaner, refrigerant flush); anti-adhesive agents (mold cleaning and release product); building/construction materials not covered elsewhere (insulation); electronic and electronic products and metal products; functional fluids (closed/open-systems) – refrigerant/cutting oils; asphalt extraction; laboratory chemicals; and temperature indicator – coatings.

These uncontrolled applications occur at numerous small sites and involve a large worker population. For example, EPA estimates that between 22 and 99 sites formulate 1-BP into mixtures and employ up to 1,046 workers; that between 500 and 2,500 establishments use 1-BP as a vapor degreaser and employ up to 24,000 exposed workers; that 1,000 to 5,000 businesses use 1-BP-based aerosol solvents, with up to 12,300 exposed workers; and that 100 to 280 facilities use 1-BP spray adhesive products in foam cushion manufacturing, with up to 4,200 exposed workers. EPA found that at least half of these exposed workers are women. (p,23).

For each of these 1-BP applications, the draft risk evaluation uses monitoring data (where available) and modeling to estimate acute and chronic exposure levels (high-end and central tendency) for exposed workers and occupational non-users (ONUs). It then compares these levels to HECs (for inhalation) and HEDs (for dermal contact), representing the exposure levels at which chronic adverse effects would be expected to occur in humans based on the results of animal studies. Using this comparison, for each exposure scenario and end-point, EPA calculates MOEs and compares them to a “benchmark MOE” of 100. As EPA uses this methodology, it presumes that an unreasonable risk exists where the actual MOE is **below** the benchmark MOE.

The draft evaluation provides these MOE comparisons for a range of chronic adverse health effects causally related to 1-BP exposure, including liver, kidney, reproductive, developmental and neurotoxic effects. For carcinogenicity, the evaluation estimates excess cancers by multiplying the occupational scenario-specific estimates for both workers and ONUs by EPA’s inhalation unit risk (IUR). Excess cancer risks are expressed as the number of cancer cases per

million and then compared to “benchmark” levels of 10^{-4} , 10^{-5} , and 10^{-6} incremental individual lifetime risk.

The evaluation concludes that, in the absence of PPE, inhalation MOEs are below the benchmark MOE by up to three orders of magnitude for virtually all workers and many ONUs (except in 1-BP manufacturing and processing) and in some instances are 1 or even below (meaning that actual exposures are equal to or higher than the HEC). It also concludes that inhalation and dermal cancer risks (without PPE) are each above 10^{-4} for nearly all manufacturing and use activities and for nearly all workers and most ONUs.

However, EPA also calculates alternate MOEs and cancer risks assuming that workers would wear and be protected by respirators (APF=50). As EPA explained, the MOE “estimates for these respirator scenarios assume 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. 194). EPA made similar calculations of dermal risk assuming the use of protective gloves. Even under these unrealistic assumptions about the exposure reduction provided by respirators and gloves, the MOEs and cancer risks were still unacceptable for some use scenarios, However, in other cases, the assumed use of PPE resulted in MOEs above the benchmark MOE and in cancer risks lower than the 10^{-4} benchmark, as shown in the table below:

Table 4-47. Inhalation Cancer Risk Estimates for Occupational Use of 1-BP (Benchmark = 1×10^{-4})

Condition of Use	Category		IUR (ppm ⁻¹)	Cancer Risk		Respirator APF	Cancer Risk with Respirator		Exposure Data Type
				Central tendency	High-end		Central tendency	High-end	
Manufacturing (US)	-	Worker	0.004	1.43E-04	5.54E-04	10	1.E-05	5.54E-05	Monitoring Data
Import, Repackaging, Processing -- Incorporation into Article	-	Worker	0.004	1.82E-05	1.23E-04	10	2.E-06	1.23E-05	Model (Deterministic)
Processing - Incorporation into Formulation	-	Worker	0.004	6.03E-03	3.88E-02	50	1.E-04	7.75E-04	Monitoring Data
	-	ONU	0.004	5.17E-04	3.26E-03	N/A	N/A	N/A	
Vapor Degreasing, Open-Top	-	Worker	0.004	1.07E-02	1.01E-01	50	2.E-04	2.03E-03	Monitoring Data
	-	ONU	0.004	3.18E-05	4.40E-03	N/A	N/A	N/A	
	Pre-EC	Worker	0.004	2.82E-03	3.68E-02	50	6.E-05	7.35E-04	Model (Probabilistic)
	Post-EC	Worker	0.004	2.82E-04	3.68E-03	50	6.E-06	7.35E-05	
	Pre-EC	ONU	0.004	1.49E-03	2.09E-02	N/A	N/A	N/A	
	Post-EC	ONU	0.004	1.49E-04	2.09E-03				
Vapor Degreasing, closed-loop	-	Worker	0.004	5.63E-05	7.35E-04	10	6.E-06	7.35E-05	Model (Probabilistic)
	-	ONU	0.004	2.97E-05	4.19E-04	N/A	N/A	N/A	
Cold Cleaning	-	Worker	0.004	6.84E-03	1.52E-02	50	1.E-04	3.04E-04	Monitoring Data
	-	ONU	0.004	4.13E-03	5.33E-03	N/A	N/A	N/A	
	-	Worker	0.004	8.23E-04	1.84E-02	50	2.E-05	3.67E-04	Model (Probabilistic)
	-	ONU	0.004	4.33E-04	1.05E-02	N/A	N/A	N/A	
Aerosol Degreasing	Pre-EC	Worker	0.004	2.55E-02	6.47E-02	50	5.E-04	1.29E-03	Monitoring Data
	Post-EC	Worker	0.004	8.74E-03	1.13E-02	50	2.E-04	2.26E-04	
	-	Worker	0.004	9.52E-03	3.62E-02	50	2.E-04	7.24E-04	Model (Probabilistic)
	-	ONU	0.004	1.60E-04	1.44E-03	N/A	N/A	N/A	

In the final section of the draft evaluation, EPA concludes that, for operations where worker exposures are within non-cancer and cancer benchmarks “when expected use of PPE is considered,” 1-BP will be deemed **not** to present an unreasonable risk under TSCA. (pp. 260-277). This would mean that EPA would take no action to require protections for workers in rulemaking for 1-BP under section 6(a) of TSCA.

B. There Is No Legal or Technical Basis to Assume that PPE Will Adequately Protect Workers from Unsafe 1-BP Exposure

We strongly disagree that worker exposure scenarios that are unsafe in the absence of respirators and gloves can be determined not to present unreasonable risks on the assumption that PPE will adequately protect workers. Any respirator use (let alone continuous respirator use for all workplace operations as assumed by EPA) is highly uncertain during nearly all of the 1-BP conditions of use addressed in the risk evaluation. The risk evaluation acknowledges that “[f]ew literature sources indicate the use of respirators in 1-BP conditions of use” (p. 57) and notes that “none of the workers surveyed at a Chinese facility wore PPE” (p.59) and that “small commercial facilities performing dry cleaning and spot cleaning are unlikely to have a respiratory protection program” (p. 24).

There is no OSHA Permissible Exposure Limit (PEL) for 1-BP and OSHA regulations do not prescribe the use of respirators and protective gloves for workers exposed to 1-BP. While employers may have a general obligation to consider all relevant data and control exposure accordingly, OSHA regulations give employers wide latitude to interpret evidence of workplace risks and to select worker protection measures they deem appropriate.²⁸

In the case of 1-BP, a review of product labels and SDSs indicates that they do not prominently highlight its adverse health effects or recommend aggressive respirator and glove use. Moreover, the small facilities that use 1-BP typically lack the expertise and resources for advanced worker protection programs and, even where PPE are recommended in SDSs, are unlikely to make respirators and protective gloves consistently available and assure compliance. While EPA may “expect” that PPE is used at these facilities, the risk evaluation provides no documentation that this is in fact the case.

The draft evaluation notes that, consistent with OSHA policy, “[t]he most effective controls are elimination, substitution, or engineering controls [such as] process enclosure, local exhaust ventilation (LEV), and general dilution ventilation.”²⁹ (p.57). EPA further acknowledges that

²⁸ OSHA’s PPE standard requires employers to assess the hazards workers face but to provide PPE only when the employer deems such measures “necessary.” 29 C.F.R. § 1910.132(a).

²⁹ According to the evaluation, “EPA does not have information on the effectiveness and prevalence of engineering controls” (p. 57). However, monitoring data cited by EPA in the draft evaluation shows that workplace levels for

“[p]ersonal protective equipment, such as respirators and gloves, is the last means of worker protection in the hierarchy of controls and should only be considered when process design and engineering controls cannot reduce workplace exposure to an acceptable level.” (Id.)

Based on the recognized limitations of respirators, in its proposed TSCA rules banning use of Trichloroethylene (TCE) in aerosol and vapor degreasing operations, EPA rejected respirator use as a worker protection strategy and concluded that eliminating worker exposure was the only effective mechanism for managing TCE’s health risks. As EPA explained the inadequacy of respirators:³⁰

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

These considerations apply equally to 1-BP, a volatile solvent with a very similar use profile to TCE. As EPA concluded for TCE, the only effective way to protect workers from acute developmental effects is to “ensure that employees are no longer at risk from exposure.”³¹ Thus, consistent with OSHA policy, EPA’s determinations of unreasonable risk under TSCA should be based on workplace exposure levels in the absence of PPE. If – as EPA has shown -- these exposure levels present an unreasonable risk, EPA should proceed to rulemaking under section 6(a), as required by TSCA, and compel industry to implement engineering controls and other measures necessary to eliminate the unreasonable risk.

IV. The Draft Evaluation Understates Risks to Consumers and Workers

While EPA’s draft demonstrates that 1-BP poses serious risks of several adverse health effects, it underestimates the magnitude of these risks to workers and consumers in key respects. As shown

most conditions of use are three or more orders of magnitude above the NIOSH recommended exposure limit (REL) of 0.3 parts per million (ppm). This suggests that effective engineering controls are generally lacking.

³⁰ 82 Federal Register 7432, 7445 (January 19, 2007).

³¹ Id at 7444.

below, EPA should more accurately account for likely levels and conditions of exposure that are greater than assumed in the draft evaluation and increase its risk estimates accordingly.

A. EPA's Assessment of Consumer Exposure Was Incomplete and Overlooked Significant Contributors to Exposure and Risk

EPA's risk estimates for consumer products fail to recognize that consumers may use multiple products containing 1-BP simultaneously. They also assume that consumer product use is a one-time event, not an ongoing source of exposure. Thus, the evaluation does not address chronic exposure scenarios, even though it acknowledges that this "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." (p. 132). The CSAC review of the 2016 EPA risk assessment highlighted these limitations:³²

The Committee suggests that the Agency consider multiple uses on any day of the various aerosol products and use on multiple days per week. A do-it-yourselfer may have multiple items that need cleaning/degreasing/gluing in a single project and thus may use the product multiple times on a given day or multiple days in a week.

Unfortunately, EPA did not adopt these suggestions. Not only did this result in an underestimate of potential acute exposure but EPA also failed to address whether consumers are at risk of longer-term health effects, including impacts of 1-BP on reproduction and fetal development observed in repeated dose studies, kidney and liver toxicity, neurotoxicity and cancer.

Resulting in a further underestimate of risks to consumers, EPA acknowledges that "[t]his evaluation assumes a background concentration of zero for the chemical of concern during evaluation of consumer exposure." (p.132). However, as discussed in Part II above, widespread and substantial air emissions of 1-BP likely result in general population exposure, including by consumers who also use products containing 1-BP. In addition, exposed workers may use these products while at home and some residences and apartments may be co-located with dry-cleaning facilities or commercial businesses that release 1-BP vapors. The CSAC review faulted EPA's earlier risk assessment for not considering these scenarios:³³

[S]everal Committee members commented that there should have been more consideration of exposures from co-residence near dry cleaning facilities, community-level exposures in areas nearby industrial or dry cleaning operations, and the general population (a concern raised by the NHANES data and apparent appearance of 1-BP in some consumer product databases).

It is disappointing that EPA did not follow these recommendations. If background levels of 1-BP and other contributors to exposure were considered in conjunction with use of consumer products, overall consumer exposure levels would be higher and of longer duration than assumed in the draft evaluation. This would result in larger projected risks, including from both acute and

³² CSAC Minutes, at 14.

³³ Id., at 18.

longer-term exposures, particularly for vulnerable subpopulations with multiple exposure pathways.

For example, EPA's evaluation of risks to consumers only examines end-points -- acute reproductive and developmental effects -- that are relevant to women of child-bearing age and fetuses. However, expanding the evaluation to include multiple-exposure scenarios and general population exposure from air emissions would require EPA to include other end-points that can harm infants and children, men of reproductive age and other groups that are now excluded from EPA's assessment of risks to consumers.

A final concern is that, while EPA determined that MOEs for both inhalation and dermal acute exposure to consumer products were extremely low, it did not aggregate exposure from these two routes. This would provide a more accurate picture of risk since users of the products experience dermal and inhalation exposure simultaneously. The combined risk would be significantly larger than the risk for each pathway alone and the MOEs would show an even greater likelihood of adverse developmental and reproductive effects.

B. The Draft Evaluation Understates Occupational Exposures and Risks

As with consumers, EPA evaluated risks of chronic health effects to workers by separately estimating exposure by the inhalation and dermal routes. Again, however, EPA failed to aggregate exposures for both routes and thus did not calculate an overall level of risk.

For example, EPA describes the inhalation cancer risk for workers as follows:

The benchmark cancer risk estimate of 1×10^{-4} was exceeded for all of the uses in workers and occupational non-users for both central tendency and high-end exposure estimates for both monitoring and modeling data with or without the use an APF in most cases with few exceptions. (p. 229)

.For dermal exposure, EPA found that:

The benchmark cancer risk estimate (1×10^{-4}) was exceeded for all conditions of use (Bins 1-5) when no gloves were used (p. 237)

Obviously, since workers have concurrent inhalation and dermal exposure, these two risk estimates should be combined. This would significantly increase the overall cancer risk relative to the EPA benchmark, raising the level of concern for carcinogenicity in the workplace and requiring deeper reductions in exposure to provide adequate worker protections.

In addition, as with consumer product use, EPA's estimates of occupational risk assume that background concentrations of 1-BP are zero. However, workers are part of the general population and thus are exposed to ambient air levels of 1-BP outside of the workplace. If their places of employment are in highly industrialized areas, they may also be exposed to 1-BP

emissions from neighboring facilities. EPA should factor in these sources of elevated exposure when it estimates occupational risks because they add to the exposures from direct occupational exposure.

EPA also underestimates risks to children who spend time in dry cleaning shops operated by their parents and are exposed to 1-BP as a result. EPA assumes that exposure by these children is only for four hours a day when in fact it could be several hours longer during weekends and school vacations or for children not yet of school age. (pp. 92-93). EPA also assumes that exposure by children is not chronic and therefore does not estimate risks for 1-BP health effects, including cancer and neurotoxicity, linked to repeated dose exposure. However, it is plausible that children of dry cleaner workers could spend an extended period in dry cleaning shops and could be further exposed to 1-BP if they live in apartments directly above these shops. Thus, chronic exposure by this subpopulation is a realistic scenario for which EPA should estimate exposure levels and health risks. Finally, EPA risk policy dictates applying an uncertainty factor of up to 10X to account for the greater susceptibility to toxicants of infants and children.³⁴ Applying this uncertainty factor would increase both cancer and non-cancer risks to this subpopulation by an order of magnitude.

V. The Draft Evaluation Fails to Account for Evidence of Neurotoxicity in Humans at Lower Doses than in Animal Studies

The draft risk evaluation emphasizes the strong weight of the evidence for 1-BP's neurotoxicity:

Neurotoxicity has been identified as a critical effect for 1-BP based on over 15 years of behavioral ... neurochemical, and neurophysiological studies in rodents as well as cross-sectional studies and case reports in humans (Appendices I.1, I.3, and I.4). Overall, there is considerable support for the finding of peripheral neurotoxicity, and consistency in reports of impaired peripheral nerve function (sensory and motor) and adverse neuromuscular impacts. The effects are progressive in terms of exposure duration and concentration, and range from subtle changes in nervous system function and neurochemistry progressing to physiological manifestations of neuron damage to structural evidence of neuronal pathology. (p. 161)

³⁴ The Food Quality Protection Act (FQPA) requires EPA to apply a 10X default safety factor for the protection of infants and children in the absence of reliable data that support use of a different safety factor. EPA, Consideration of the FQPA Safety Factor and Other Uncertainty Factors In Cumulative Risk Assessment of Chemicals Sharing a Common Mechanism of Toxicity, February 28, 2002, available at <https://www.epa.gov/sites/production/files/2015-07/documents/apps-10x-sf-for-cra.pdf>. EPA's 2005 Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens provides the policy and scientific rationale for extending this approach to all chemical assessments across the Agency. EPA 2005 Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens (Supplemental Guidance, SG). <https://www.epa.gov/risk/supplemental-guidance-assessing-susceptibility-early-life-exposure-carcinogens>; Barton HA, Cogliano VJ, Flowers L, Valcovic L, Setzer RW, Woodruff TJ. Assessing susceptibility from early-life exposure to carcinogens. *Environ Health Perspect.* 2005;113(9):1125-33. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1280390/>

In the evaluation, EPA used rat studies on 1-BP to establish a POD for assessing neurotoxicity risks and calculated an HEC of 25 ppm to determine an MOE. (p. 173). The Agency did not rely on human data although worker studies showed neurotoxic effects at levels 10X below the HEC, as described in the CSAC review of the 2016 risk assessment:³⁵

Of note, the lowest HEC (25 ppm) is derived from a neurotoxicity study in rats. The epidemiological studies reporting neurotoxicity in workers using or manufacturing 1-BP were not readily useable for dose response assessment. However, they do provide some information on exposures of workers with neurological deficits. For example, Ichihara et al. (2004) measured 8-hr TWA 1-BP exposures of individual workers in a 1-BP production factory and found a range of exposures from 0.34 to 49 ppm, with a GM of 2.92 ppm. Fifteen of 27 workers in the 1-BP factory exhibited neurological deficits relative to control workers.

EPA's rationale for relying exclusively on the animal data for its neurotoxicity MOE is that "the reports of effects in factory workers with lower exposures are limited by questions about exposure characterization, measurement techniques, and sensitivity." (p. 247). At the same time, EPA emphasizes that its findings of "[n]eurotoxicity produced by 1-BP are based on rodent and human literature, with considerable similarities in both qualitative and quantitative outcomes." (Id).

Human evidence is generally preferable to animal data and, in this instance, multiple human studies are available that show consistent evidence of neurotoxicity in exposed workers at low concentrations. The most health protective approach is to use these studies to determine the POD/HEC for this endpoint. Otherwise, EPA's evaluation will understate the well-documented and serious neurotoxic risks of 1-BP to people.

VI. EPA Correctly Used a Linear Extrapolation to Estimate Cancer Risks from Exposure to 1-BP

As demonstrated in the draft risk evaluation, 1-BP has been shown to be a multi-site carcinogen in rats and mice. Applying the criteria in EPA's 2005 Guidelines for Carcinogen Risk Assessment,³⁶ the evaluation concludes that:

1-BP may be considered "*Likely to be Carcinogenic in Humans*" based on the positive findings for carcinogenicity in more than one test species together with positive findings for the direct reactivity of 1-BP with DNA and evidence that both 1-BP and its metabolites are positive in mutagenicity studies and other types of studies that assess genetic toxicity. (Emphasis in original)

³⁵ CSAC Minutes, at 66.

³⁶ EPA 2005. Guidelines for Carcinogen Risk Assessment. Pg. 84-85. Available from: https://www.epa.gov/sites/production/files/2013-09/documents/cancer_guidelines_final_3-25-05.pdf

The evaluation uses a linear low-dose extrapolation method to estimate cancer risks at occupational exposure levels. At the SACC meeting, industry commenters took issue with this approach, arguing that there is evidence for a Mode of Action (MOA) involving a threshold and that a non-linear approach based on this MOA is a more defensible method of risk estimation.

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

Elucidation of a mode of action for a particular cancer response in animals or humans is a data-rich determination. *Significant information should be developed to ensure that a scientifically justifiable mode of action underlies the process leading to cancer at a given site. In the absence of sufficiently, scientifically justifiable mode of action information, EPA generally takes public health protective, default positions* regarding the interpretation of toxicologic and epidemiologic data animal tumor findings are judged to be relevant to humans, and cancer risks are assumed to conform with low dose linearity (emphasis added) (1-10 through 1-11).

The Guidelines add that:

When the weight of evidence evaluation of all available data are insufficient to establish the mode of action for a tumor site and when scientifically plausible based on the available data, linear extrapolation is used as a default approach, because linear extrapolation generally is considered to be a health-protective approach. *Nonlinear approaches generally should not be used in cases where the mode of action has not been ascertained.* (emphasis added) (3-21)

A nonlinear approach should be selected when there are sufficient data to ascertain the mode of action and conclude that it is not linear at low doses and the agent does not demonstrate mutagenic or other activity consistent with linearity at low doses. (3-22).

EPA has correctly applied these principles in the draft evaluation. The Agency found that “the overall weight of the scientific evidence supports a mutagenic MOA for 1-BP induced carcinogenicity” (p.159). While acknowledging that “the results from Ames and other genotoxicity tests for 1-BP have been mixed,” it emphasized that the negative “studies do not provide clear evidence against a mutagenic mode of action of 1-BP carcinogenicity based on several conceptual, and methodological uncertainties” (p. 163) It concluded that, “[g]iven the lack of specific mechanistic or dose-response information, linear extrapolation from the point of departure is recommended.” (p. 159)

In reviewing the 2016 1-BP assessment, CSAC concurred with these conclusions. It stated that “[m]ost Committee members agreed the existing evidence supports a conclusion of mutagenicity/genotoxicity as the primary mode of action (MOA) for 1-BP and some of its metabolites” (p. 49-50) and “concluded that the available evidence supports using a low-dose linear model to assess dose-response.”³⁷

³⁷ CSAC Minutes, at 50.

EPA's final evaluation should retain this approach.

VII. EPA Lacks Support for Its Finding of No Unreasonable Risk for Environmental Effects

The draft evaluation concludes “that 1-BP does not present unreasonable risk to the environment under the identified conditions of use.” (p. 23) The data supporting this determination are weak and inconclusive and mostly derive of studies unavailable to EPA and the public and of unknown quality and reliability.

EPA's Charge Question 5.1 to the SACC acknowledges that “[o]nly a few environmental test data endpoints (including ECHA) are available in the public domain for 1-BP. Most are from the ECHA website.” The ECHA dossiers are data summaries prepared by industry, not actual study reports. While ECHA posts these summaries on its Website, it does not evaluate either the summaries or underlying studies for quality and reliability. Thus, neither ECHA nor any other government agency has vouched for either the accuracy of the summaries or the validity of the study findings they describe and the methods with which the studies were conducted.

As it admitted in the Charge Question, EPA itself failed to obtain and review the studies reported in the ECHA summaries:

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.

Having failed to obtain the studies and thus to review them, the Agency nonetheless “decided to use the experimental data . . . [g]iven that the ECHA environmental test data results are in the public domain.” EPA thus chose to rely on the ECHA summaries even though the “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*.” (p. 141) In fact, EPA admitted that “only a single acute fish toxicity study identified during the literature search process ((Geiger et al., 1988))” was evaluated for data quality using the systematic review protocol. Yet EPA justified using the ECHA summaries on the basis of a “qualitative” evaluation of the reported findings that showed that the “hazard conclusions of these summaries are consistent with the results of the fish study that was reviewed for data quality.” *Id.* To use a single acute study as “qualitative” confirmation of ECHA-reported studies that EPA has never obtained or reviewed is, to say the least, a misapplication of the concept of data quality.

No data were available to characterize the hazards of chronic 1-BP exposure to aquatic species. EPA compensated for this data gap by estimating hazards from chronic exposure using an acute-to-chronic ratio (ACR). (p.139-140) However, in calculating this ratio, EPA relied on the ECHA summaries of acute studies. Without independent verification of the ECHA summaries and the studies they describe, there is no assurance that the chronic toxicity value EPA derived was correct.

Review of full studies – as opposed to ECHA summaries – is a rudimentary safeguard to assure that the summaries accurately reflect the reported findings and do not overlook other noteworthy findings and that the test protocol was sound and reliably executed. Moreover, only if the actual study results are available is it possible for peer reviewers and public commenters to evaluate EPA’s interpretation of the study results and use of the data in making the “weight of evidence” determination of environmental risk that TSCA requires.

Since it has long been aware of the limited ecotoxicity data on 1-BP, EPA could easily have required 1-BP’s manufacturers to conduct aquatic toxicity testing early in the TSCA risk evaluation process. In this event, publicly available test results would now be available for use in the risk evaluation and the data could be reviewed not only by EPA but by the SACC and public commenters. Having failed to follow this path, EPA now lacks a valid scientific basis to determine that 1-BP does not present an unreasonable risk to the environment. This determination should thus be removed from the final risk evaluation, and EPA should use its TSCA authority to secure necessary studies of 1-BP’s ecological risks.

VIII. Continued Application of EPA’s TSCA Systematic Review Methodology to Draft Risk Evaluations Is Highly Problematic in Light of SACC Concerns Which EPA Has Failed to Address

The 1-BP draft risk evaluation continues to rely on the TSCA EPA’s systematic review method³⁸ despite the serious scientific flaws previously identified by numerous commenters and summarized in a recent peer-reviewed commentary published in the *American Journal of Public Health*.³⁹

In addition to the many deficiencies of the original TSCA systematic review method, recent risk evaluations (including for 1-BP) incorporate a new approach of relying on “key and supporting/influential information.” This approach was not previously published or peer reviewed, has not been subject to public comment, and raises concerns about bias in the weighting of studies in EPA’s risk determinations. EPA has not defined key terms like “key,” “supporting” and “influential,” encouraging subjective judgments which are not grounded in objective criteria.

Recent evaluations have also been based on a “hierarchy of preferences,” another new concept that was not part of the original TSCA systematic review document and has likewise not been subject to peer review or public comment. The 1-BP evaluation briefly explains this approach in a footnote on page 45 as follows:

³⁸ EPA (2018) Application of Systematic Review in TSCA Risk Evaluations.

³⁹ Singla V, Sutton P, Woodruff TW. (2019) The Environmental Protection Agency Toxic Substances Control Act Systematic Review Method May Curtail Science Used to Inform Policies, With Profound Implications for Public Health. *Am J Public Health*. doi: 10.2105/AJPH.2019.305068

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

EPA does not explain why some types of studies should receive preference over others in determining the weight of evidence for a particular endpoint and on what basis these studies should be assigned to a "higher level." Again, EPA has failed to provide objective criteria for determining which evidence to rely on and which to exclude, undermining transparency and consistency in the systematic review process and encouraging subjective judgments.

In its peer review of the Draft Risk Evaluation of C.I. Pigment Violet 29 (PV29),⁴⁰ the EPA SACC highlighted several areas of concern with the TSCA systematic review method and made numerous recommendations for how EPA should address these questions about the method's scientific basis. EPA has not addressed these recommendations and must do so expeditiously as it continues to issue and finalize risk evaluations. Below, we summarize the areas of most concern raised by the SACC that remain unaddressed:

- *"The Agency rationale for developing the TSCA SR should include a comparison to other SR approaches and describe the rationale for major differences."*⁴¹
- *"The Committee discussed the need to publish peer reviewed pre-established protocols for each of the Agency's reviews prior to performing the actual risk assessment. The protocol for PV29 was created concurrently with the review, which is contrary to best practices for systematic reviews."*⁴²
- *"The Committee noted that the TSCA SR weighted scoring system may be inappropriate if there is disagreement in the weighting of different metrics. For example, a certain study characteristic that may be a "fatal flaw" would be weighted equally to other more minor elements. The "Agency should provide justification for using a weighted scoring system and the rationale for the specific metrics used for differential weighting in its evaluation of studies."*⁴³
- *"Regarding data integration, the Committee discussed the benefits of including a more thorough and inclusive data integration discussion in the TSCA SR for PV29... there is a*

⁴⁰ US EPA (2019) Peer Review of the Draft Risk Evaluation for Pigment Violet 29 (PV29). Available: <https://www.regulations.gov/docket?D=EPA-HQ-OPPT-2018-0604> D=EPA-HQ-OPPT-2018-0604

⁴¹ Id. at 26.

⁴² Id. at 27.

⁴³ Id. at 26-7.

need in the Evaluation for a thorough description and outline for how all evidence and data are integrated into a final weight of evidence conclusion”⁴⁴

Given the SACC’s critical role as a scientific peer reviewer of the TSCA program, it is critical that EPA address the SACC’s comments through changes to its systematic review method prior to finalizing the 1-BP and other ongoing TSCA risk evaluations. The SACC also strongly recommended that EPA move forward with National Academy of Sciences (NAS) of its TSCA systematic review method – a commitment on which EPA is dragging its feet.

Conclusion

1-BP is an unsafe chemical with significant worker, consumer and general population exposure. EPA’s risk evaluation confirms previous determinations that 1-BP causes cancer, reproductive harm, damage to developing fetuses, and kidney, liver and neurological effects. EPA further concludes that, for most exposure scenarios, the risks posed by these effects are unreasonable under TSCA. We agree that 1-BP presents unreasonable risks, and in fact believe that EPA’s findings of serious reproductive and developmental effects from acute exposure are so troubling that they require immediate action to protect women of child-bearing age and fetuses from imminent harm. Moreover, alarming as they are, EPA’s determinations of risk underestimate the magnitude of exposure by consumers, workers and communities: properly estimated, 1-BP’s risks are in fact significantly greater than the draft evaluation reflects. EPA’s evaluation must therefore be revised to show larger risks than presented in the draft. We believe that, with these adjustments, the risk evaluation will make a compelling case that all consumer and most commercial uses of 1-BP should be banned under section 6(a) of TSCA.

We appreciate the opportunity to submit comments on the draft 1-BP evaluation.

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Respectfully submitted.

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⁴⁴ Id. at 27.