# UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

## Comments of Safer Chemicals Healthy Families, Center for Environmental Health, Earthjustice, Environmental Health Strategy Center, Environmental Working Group, Natural Resources Defense Council, and Toxic Free NC on EPA's Draft Risk Evaluation For 1,4-Dioxane under Section 6(b) of TSCA

Submitted via Regulations.gov (August 30, 2019)

Docket ID EPA-HQ-OPPT-2019-0238

## EXECUTIVE SUMMARY

Safer Chemicals Healthy Families (SCHF), Center for Environmental Health, Earthjustice, Environmental Health Strategy Center, Environmental Working Group, Natural Resources Defense Council, and Toxic Free NC submit these comments on EPA's draft risk evaluation for 1,4-dioxane under section 6(b) of the Toxic Substances Control Act (TSCA).[1] Our organizations are national and grassroots 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.

**The Public Comment and Peer Review Process for the Initial TSCA Risk Evaluations Is Inadequate**

Before turning to the 1,4-dioxane evaluation, we reiterate our serious concern about the inadequate public comment and peer review process for all 10 evaluations being conducting to fulfill EPA's mandate under section 6(b)(2)(A) of TSCA. Because of the essential role of risk evaluations in determining the safety of existing chemicals of concern and eliminating unreasonable risks, these initial evaluations provide a critical test of EPA's progress in achieving the public health goals of Congress. The serious gaps and deficiencies in the first three draft evaluations underscore the importance of full public comment and peer review so that EPA receives the feedback necessary to improve the final versions. Regrettably, however, EPA's rushed public comment and peer review process is putting enormous pressure on stakeholders and the Science Advisory Committee on Chemicals (SACC) itself. This is greatly weakening their ability to conduct the thorough review and analysis of the draft evaluations essential for providing thoughtful and probing scientific feedback to the Agency.

As some of our groups emphasized in their July 11, 2019 letter to Assistant Administrator Dunn, EPA is following the highly irregular procedure of convening the SACC to review draft evaluations *well before* the close of the public comment period. This is preventing the SACC members from considering informed stakeholder input based on a thorough analysis of these drafts. Moreover, the SACC reviews have been scheduled only a few weeks after the release of the draft evaluations, limiting the ability of SACC members to carefully examine their contents and providing the public with inadequate time to develop presentations

[1] 84 Federal Register 31315 (July 1, 2019).

to the SACC. Despite our concerns, EPA is following the same truncated process for its latest draft evaluation on 1-bromopropane, released on August 12,[2] and apparently for the remaining six draft evaluations still in progress.

By prioritizing speed over public participation and over ensuring SACC access to thoughtful independent scientific input, the Agency is undermining the credibility and quality of its evaluations, which are already under a significant cloud, and fostering the perception that it is trying to wall off SACC from critical voices that it thinks could adversely influence its conclusions and recommendations. *We continue to request that EPA: (a) schedule future SACC meetings on the remaining draft evaluations after the close of the relevant comment periods so these comments can be provided to and considered by SACC in a timely manner, and (b) avoid overlapping comment periods on the evaluations.*

**EPA's Draft Evaluation Fails to Address and Account for All Pathways of Exposure that Put the Public at Risk**

1,4-Dioxane is a multi-site, multi-species carcinogen that has prompted public health concern at the state and national level for many years. Human exposure to 1,4-dioxane is widespread and derives from multiple sources:

- 1,4-dioxane is produced as a byproduct during manufacture of ethoxylated and other chemicals and has been documented as an impurity in extensively used personal care and cleaning products. Use of these products may expose millions of consumers to 1,4-dioxane directly and results in releases of the chemical to surface water and wastewater when the products are washed "down the drain" after use.
- Public drinking water supplies in many parts of the country have been found to contain 1,4-dioxane and millions of people have consumed this contaminated drinking water. Drinking water contamination is the result of a combination of factors, including ongoing "down the drain" discharges of consumer products to wastewater systems that cannot effectively remove 1,4-dioxane; historical disposal of 1,4-dioxane-containing industrial wastes that have migrated to groundwater and surface water and in turn to drinking water sources; and ongoing releases of 1,4-dioxane from manufacturing, processing and use activities. Private drinking water supplies may also contain 1,4-dioxane in significant concentrations.
- The general population, and vulnerable subpopulations such as children, are subject to significant, ongoing, and cumulative exposures of 1,4-dioxane through product use and ingestion of drinking water.
- A large number of workers (perhaps millions according to EPA) are exposed to 1,4-dioxane during manufacture and at numerous downstream facilities where it is intentionally used in a wide variety of industrial applications.
- 1,4-dioxane is also released to air and disposed of as waste by manufacturing and use facilities.[3]

[2] 84 Federal Register 39830. The SAAC meeting to review this evaluation will be on September 10-12 while comments are due on October 11. Submissions to the SACC must be made by August 30, the same day comments are due on the 1,4-dioxane and HBCD draft evaluations.

[3] According to EPA's Toxics Release Inventory for 2014, 96,437 pounds of 1,4-dioxane were released to the air, 24,262 to surface water, and 422,943 pounds were transferred from the facility for off-site disposal (US EPA TRI

As part of its Safer Consumer Products program, California's Department of Toxic Substances Control (DTSC) has described some of the multiple pathways of human exposure to 1,4-dioxane as follows:[4]

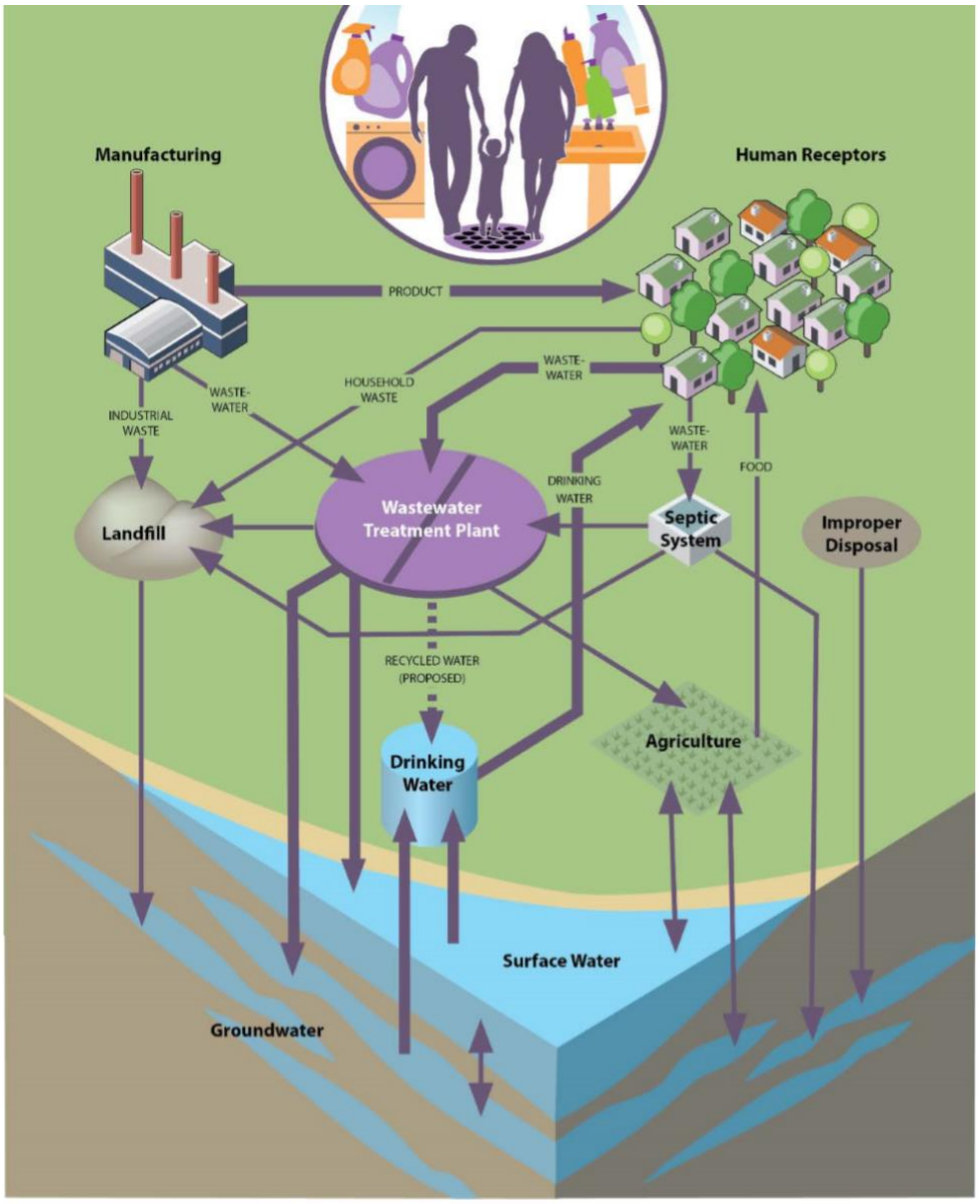


**Figure 4.** Exposure pathways of 1,4-dioxane, focusing on exposures most relevant for consumer products. Bold arrows emphasize the potential impacts of consumer products on the drinking water exposure pathway. The dotted line relates to the future direct use of treated wastewater as drinking water.

Explorer, Release Chemical Report database, available at https://iaspub.epa.gov/triexplorer/tri_release.chemical). These are also pathways of human exposure that EPA fails to account for in its risk evaluation.

[4] California Department of Toxic Substances Control (DTSC). 2019. Work Plan Implementation: 1,4-Dioxane in Personal Care and Cleaning Products. P 6. Available at https://dtsc.ca.gov/wp-content/uploads/sites/31/2019/05/Background-Document_14-dioxane.pdf ("CA DTSC Background Document")

As this figure shows, any credible determination of 1,4-dioxane's human health risk must integrate and combine these multiple exposure pathways and account for their aggregate contribution to risk. Thus, as discussed below, the California Department of Toxic Substances Control (DTSC) has initiated a process to limit the presence of 1,4-dioxane in cleaning and personal care products due to product-related and drinking water ingestion exposure scenarios. Similarly, the New York State Legislature recently passed a bill imposing limits on 1,4-dioxane in these products.[5]

EPA's draft evaluation, however, only addresses one dimension of 1,4-dioxane exposure – risks to workers engaged in the chemical's manufacture, processing and use. The evaluation expressly excludes use of personal care and cleaning products containing 1,4-dioxane as an impurity and consumption of contaminated drinking water – sources of exposure that put many millions of Americans at risk.

EPA explicitly recognizes that it "did not evaluate hazards or exposures to the general population in this risk evaluation, and there is no risk determination for the general population" (Draft Evaluation at 156), but fails to acknowledge that these gaps in coverage leave the most significant public health risks from 1,4-dioxane exposure unaddressed. Without assessing these risks, the evaluation will fail to inform the public and regulators about 1,4-dioxane's impacts on public health and lay a foundation for meaningful action to reduce exposure and risk.

EPA has not provided a credible legal or scientific rationale for excluding consumer product and drinking water pathways of exposure from the draft evaluation.

EPA justifies its exclusion of consumer products on the ground that the 1,4-dioxane present in these products is produced as a byproduct from ethoxylation of other chemicals and therefore will be within the scope of the risk evaluation it may conduct for these chemicals. However, TSCA does not allow EPA to omit some pathways of exposure from an evaluation because they might be addressed in a future evaluation for a different set of chemicals. Rather, both intended and byproduct forms of manufacture are "conditions of use" under TSCA and the risks of exposure are identical regardless of the method of production. Moreover, EPA has articulated no plan or timetable to conduct a risk evaluation on ethoxylated chemicals and, given its constrained resources and the limited number of substances it is required to evaluate under the law, such an evaluation is unlikely in the foreseeable future. Revealingly, ethoxylated chemicals are not among the 20 chemicals that EPA proposed for the next set of risk evaluations under TSCA,[6] an indication that they are a low priority for future action.[7]

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[5] See https://www.nysenate.gov/legislation/bills/2019/s4389

[6] 84 Fed. Reg. 44300 (August 23, 2019).

[7] EPA may never evaluate all of the ethoxylated chemicals that result in 1,4-dioxane production as a byproduct. There are many classes of ethoxylated chemicals that that may form 1,4-dioxane as a byproduct during manufacture. Moreover, the sulfation process (and manufacture of alcohol ethoxysulfates) is also significantly responsible for the formation of 1,4-dioxane, not just ethoxylation (manufacture of alcohol ethoxylates). The American Cleaning Institute (ACI) underscored this point at the Aug. 21 DTSC workshop in Sacramento and it was a prominent theme in remarks from industry.
https://dtsc.ca.gov/wp-content/uploads/sites/31/2019/08/Kathleen-S_ACI-presentation-Aug-21-2019.pdf

Finally, conducting separate risk evaluations on intended and byproduct uses of 1,4-dioxane will mean that neither evaluation will provide a complete or accurate picture of the chemical's health risks. This is because both sets of uses contribute to total human exposure and cannot be meaningfully evaluated in isolation from each other. For example, workers are exposed to 1,4-dioxane through both plant-specific and general population pathways. Thus, as SACC members noted in their review of the draft evaluation, consumer product exposures should be addressed in combination with all other sources of exposure to 1,4-dioxane.

The exclusion of drinking water exposure from the 1,4-dioxane risk evaluation is an outgrowth of EPA's broader policy decision to ignore all environmental release scenarios in TSCA risk evaluations on the premise that they are being effectively addressed under other environmental laws. Thus, EPA asserts that, because "the drinking water exposure pathway for 1,4-dioxane is being addressed under the regular analytical processes to identify and evaluate drinking water contaminants" under the Safe Drinking Water Act (SDWA), "there is no need to include this pathway in the risk evaluation for 1,4-dioxane under TSCA" (Problem Formulation at 43). However, EPA has not set a National Primary Drinking Water Regulation under SDWA for 1,4-dioxane. Nor is there any realistic prospect that such a regulation will be developed any time soon. Thus, if drinking water sources of exposure are not included in the ongoing TSCA risk evaluation, the odds that they will be addressed under SDWA are remote. Moreover, the purpose of TSCA is require a holistic examination of total risk from all sources. SDWA, by contrast, would only address one source of exposure. EPA's decision to ignore environmental releases that contribute to total human risk – the unique purview of TSCA – thus turns the law on its head.

The exclusion of drinking water exposures also violates Section 6(b)(4)(A) of TSCA, which requires that EPA risk evaluations address potentially exposed or susceptible subpopulations under the conditions of use. As discussed below, California and others have identified vulnerable populations who ingest contaminated drinking water, and workers are exposed to 1,4-dioxane through both workplace and general population pathways. By excluding consideration of drinking water exposures, EPA failed to address risks to vulnerable populations as directed by section 6(b)(4)(A).

*Thus, EPA should revise the draft evaluation so it addresses the contribution of consumer products and drinking water contamination to total exposure and risk from 1,4-dioxane.*

**EPA'S Evaluation of Risks to Workers Is Flawed and Does Not Support Its Conclusion That Nearly All Occupational Exposure Scenarios Do Not Present Unreasonable Risks**

EPA's risk determinations for workers are based on limited and poorly described workplace monitoring data that it then extrapolates to a wide variety of manufacturing and processing conditions. The absence of adequate information on worker exposure could have been avoided if EPA had used its TSCA information collection authority to obtain all existing worker monitoring data and require industry to conduct additional monitoring where necessary to reliably assess occupational risks.

EPA also failed to recognize that workers have multiple routes of exposure, including both in their places of employment and in their homes, where they may use consumer products and ingest drinking water containing 1,4-dioxane. The most protective approach would be to conduct an "aggregate exposure assessment" that accounts for the total exposure and risk resulting from these combined sources. EPA

further understates worker exposures by calculating risk levels for dermal and inhalation exposure separately and not combining them to account for concurrent exposure by both routes.

For several workplace exposure scenarios, EPA calculates cancer risks above its "unreasonable risk" benchmark of 1 x $10^{-4}$ but then determines that use of Personal Protective Equipment (PPE) lowers these risks below the benchmark and makes them "reasonable" under TSCA. The assumption that employers are now effectively protecting workers through PPE is arbitrary and without basis. The OSHA permissible exposure limit (PEL) for 1,4-dioxane is several decades old and is set at a level (100 ppm or 360 mg/m3 as an 8-hour TWA) that fails to protect workers against significant cancer risk. It is doubtful that employers are implementing more stringent exposure limits (through PPE or other controls) in the absence of any legal obligation to do so. Moreover, it is well-known and documented that respirators and other protective gear are used only intermittently by workers even where they are legally required, that Safety Data Sheets (SDS) and directions for safe use are often misunderstood or ignored, and that employers often fail to provide adequate training and equipment to their workers. Like OSHA, EPA should therefore make determinations of unreasonable risk to workers without taking into account the effects of PPE. The questionable assumption that voluntary use of PPE will protect workers should not be an excuse to ignore risks that would otherwise be unreasonable and would warrant regulatory action to protect workers.

**EPA's Risk Determinations for 1,4-Dioxane Are Weakened by Numerous Data Gaps That Should Have Been Identified and Addressed Before Initiating the Evaluation**

These data gaps involve essential information on 1,4-dioxane's health and environmental effects and release into the environment. The absence of this information creates major uncertainties in EPA's evaluation and likely understate human health and ecological risks. EPA should have applied an additional Uncertainty Factor (UF) of 10 to account for absence of critical health effects information. If this UF is incorporated in the Agency's Margin of Exposure (MOE) analysis, risks that EPA now deems reasonable will become unreasonable.

**Because There Is No Scientific Support for a Threshold MOA, EPA's Cancer Risk Estimates and Unreasonable Risk Determinations Should Be Based Only on a Linear Low Dose Extrapolation**

EPA and other authoritative bodies have classified 1,4-dioxane as "likely to be carcinogenic to humans." However, departing from EPA's 2013 IRIS assessment, the draft evaluation presents cancer risk estimates using linear and non-linear low dose extrapolation methods and leaves the door open to basing the final evaluation on the non-linear approach. Although industry has pushed hard for this approach, it is contrary to the Agency's cancer risk assessment guidelines, the conclusions of the 2013 IRIS assessment, and the detailed analysis of 1,4-dioxane's mode of action (MOA) in the draft TSCA evaluation itself. The final evaluation should only present linear estimates of cancer risk.

## I. THE DRAFT EVALUATION UNJUSTIFIABLY DISREGARDS HEALTH RISKS FROM THE PRESENCE OF 1,4 DIOXANE IN CONSUMER PRODUCTS

EPA's evaluation fails to address the contribution to human exposure of 1,4-dioxane's presence in consumer products and thus ignores its risks to consumers. This is a fundamental gap in the evaluation.

It lacks any justification under TSCA and will greatly weaken the value of the evaluation in protecting public health.

### A. 1,4-Dioxane's Presence as an Impurity in Numerous Personal Care and Cleaning Products Is a Significant Pathway of Human Exposure and Contributor to Risk

1,4-Dioxane is generated during the production of alcohol ethoxysulfates and alcohol ethoxylates that are extensively used as surfactants in shampoo, body wash, dish detergent, laundry detergent and many other products. In its 2015 initial assessment of 1,4-dioxane, EPA states: "Workers and consumers may be exposed to 1,4-dioxane present as a contaminant in products such as personal care products, paints, adhesives, varnishes, cleaners and detergents."[8] EPA further comments that "[w]hile personal care products are regulated by the FDA, uses in paints, adhesives, varnishes, cleaners and detergents fall under TSCA authority."

There are considerable data on the types of consumer products containing 1,4-dioxane and the levels present. The reported findings include the following:

- *Bath products, hair treatment, and lotions marketed toward children*: 1,4-dioxane detected in 47/82 products tested. Average = 1.54 ug/g (ppm); range = 0.23-15.3 ppm, with 2 above 10 ppm.[9]
- *Dish detergent* (average 4.6 ppm, max 7.7 ppm), *body wash* (average 4.43 ppm, max 35 ppm), *laundry detergent* (average 4.61 ppm, max 14 ppm), *shampoo* (average 1.66 ppm, max 5.5 ppm), *bubble bath* (average 2.43 ppm, max 11 ppm), *body wash & shampoo combined* (average 2.04 ppm, max 7.6 ppm), *hand soap* (average 0.93 ppm, max 1.9 ppm), *lotion* (average 0.16 ppm, max 0.92 ppm), and *baby wipes* (average 0.01 ppm, max 0.01 ppm)[10]
- *Kleenex Hair and Body Wash and Kleenex Foam Hair and Body Wash* (both 0-0.1%)[11]

[8] US EPA. 2015. TSCA Work Plan Chemical Problem Formulation and Initial Assessment: 1,4-Dioxane. P 8. Available at https://www.epa.gov/sites/production/files/2017-06/documents/14_dioxane_problem_formulation_and_intial_assessment.pdf

[9] These data are from a 2018 FDA study accepted for publication in July 2019. See: US FDA. 1,4-Dioxane in Cosmetics: A Manufacturing Byproduct, under the heading "How much 1,4-dioxane is present in cosmetics?" available at https://www.fda.gov/cosmetics/potential-contaminants-cosmetics/14-dioxane-cosmetics-manufacturing-byproduct#How (accessed August 29, 2019). The study's abstract is available from here: https://www.sciencedirect.com/science/article/pii/S0021967319307848.

[10] These are the concentrations in personal care or cleaning products from studies in the last 10 years, as summarized by California's DTSC in Table 1 of its August 2019 Alternatives Analysis Threshold Discussion Proposal, available at: https://dtsc.ca.gov/wp-content/uploads/sites/31/2019/08/14-Dioxane-Draft-AAT-for-August-2019-Workshop.pdf. An image of Table 1 is also copied into this document below.

[11] The safety data sheets for these products are available at: http://www.na.kccustomerportal.com/Documents/Upload/Application/2811/Learning%20Center/Article/KLEENEX%20Hair%20and%20Body%20Wash%2091557_GHS_EN.pdf and http://www.na.kccustomerportal.com/Documents/Upload/Application/2811/Learning%20Center/Article/KLEENEX%20%20Foam%20Hair%20and%20Body%20Wash%2091553_11553_GHS_EN.pdf. They were originally referenced in EPA's February 2017 "Preliminary Information on Manufacturing, Processing, Distribution, Use, and Disposal" for 1,4-Dioxane, available at https://www.epa.gov/sites/production/files/2017-02/documents/14-dioxane.pdf.

- *Art supplies* (1000-5000 ppm), *baby bibs* (under 100 ppm), *clothing* (under 100 ppm), *blankets/throws* (under 100 ppm), *footwear* (under 100 ppm), and *toys/games* (under 100 ppm to under 5,000 ppm).[12]
- *Bath/pool water toy* (either 100-500 ppm or 500-1,000 ppm, or there are two toys)[13]
- *Possibly in 8,000+ shampoos, soaps, lotions, sunscreens, toothpastes and cosmetics, 200+ of which are marketed to children and infants* (levels not specified). The Environmental Working Group (EWG) analyzed its Skin Deep® cosmetics database for ingredients produced through ethoxylation, such as polyethylene, polyethylene glycol (PEG) and ceteareth (since 1,4-dioxane can be a contaminant in ethoxylated chemicals) and found these ingredients in over 8,000 personal care products, including shampoos, soaps, lotions, sunscreens, toothpastes and cosmetics.[14]
- *Sprayway Leather Conditioner & Cleaner*, SW-991, Aerosol-06/19/2015: contains 0.01-0.1% by weight[15]
- *Anti-freeze (for consumers)* – in 1988, contained "100 to 3,400 ppb," according to the ATSDR Toxicological Profile for 1,4-dioxane.[16] In a 2002 report, the European Chemicals Bureau listed the range in anti-freeze and deicing products as 0.1-22 ppm (in turn, citing to an Australian (NICNAS) study from 1998).[17]

These products are used by a large portion of the US population. For example, EWG conducted an online survey in 2004 "of the cosmetics and personal care products used by 2,300 people" and found that 1 in 5 adults is potentially exposed every day to 1,4-dioxane.[18] According to EWG, more than 8,000 personal

[12] These numbers represent concentrations of 1,4-dioxane in children's products reported by manufacturers under the Washington State Children's Safe Products Act from 2013 to 2019, accessed via the Washington State Department of Ecology's "Children's Safe Product Act Reported Data" portal - https://apps.ecology.wa.gov/cspareporting/Reports/ReportViewer.aspx?ReportName=ChemicalReportByCASNumber.

[13] This data shows concentrations of 1,4-dioxane in children's product(s) reported by manufacturers under Vermont law and regulations, as shown in the database accessed from this webpage: https://www.healthvermont.gov/environment/children/chemical-disclosure-program-childrens-products-manufacturers

[14] Faber, Scott, "Trump's EPA Ignores Hidden Carcinogen Lurking in Cosmetics," June 26, 2017, available at https://www.ewg.org/planet-trump/2017/06/trump-s-epa-ignores-hidden-carcinogen-lurking-cosmetics; Faber, Scott, and Jared Hayes, "Hundreds of Kids' Cosmetics Products May Contain Hidden Carcinogen," July 11, 2017, available at https://www.ewg.org/enviroblog/2017/07/hundreds-kids-cosmetics-products-may-contain-hidden-carcinogen; EWG, "EWG Surveys Personal Care Product Companies About 1,4-Dioxane," April 17, 2017, available at https://www.ewg.org/release/ewg-surveys-personal-care-product-companies-about-14-dioxane.

[15] See safety data sheets from the Sprayway product webpage at http://www.spraywayinc.com/content/leather-conditioner-cleaner and in the Consumer Product Information Database here https://www.whatsinproducts.com/brands/show_msds/1/18766.

[16] US ATSDR. 2012. Toxicological Profile for 1,4-Dioxane. P 176. Available at https://www.atsdr.cdc.gov/toxprofiles/tp187.pdf (citing to a 1989 study).

[17] European Chemicals Bureau. 2002. European Union Risk Assessment Report for 1,4-dioxane. P 49. Available at https://echa.europa.eu/documents/10162/a4e83a6a-c421-4243-a8df-3e84893082aa.

[18] EWG, "EWG Research Shows 22 Percent of All Cosmetics May Be Contaminated With Cancer-Causing Impurity," February 8, 2007, available at https://www.ewg.org/news/news-releases/2007/02/08/ewg-research-shows-22-percent-all-cosmetics-may-be-contaminated-cancer

care products may contain 1,4-dioxane as a contaminant, with more than 200 of these products marketed for children and babies.[19]

Adolescents - with their unique sensitivity to the effects of carcinogens - use more cosmetics and body care products daily than an average adult woman. Participants in EWG's study of teen girls' chemical body burden used an average of 17 personal care products each day. These products fall under multiple product categories known to contain ethoxylated chemistry.[20]

California's Department of Toxic Substances Control (DTSC) has identified 1,4-dioxane as a Chemical of Concern under its Safer Consumer Products regulations and is in the process of designating Priority Products.[21] Based on a survey of publicly reported data, DTSC provided a snapshot of the potentially affected product universe as follows:[22]

[19] Faber, Scott, and Jared Hayes, "Hundreds of Kids' Cosmetics Products May Contain Hidden Carcinogen," July 11, 2017, available at https://www.ewg.org/enviroblog/2017/07/hundreds-kids-cosmetics-products-may-contain-hidden-carcinogen.

[20] EWG, "Teen Girls' Body Burden of Hormone-Altering Cosmetics Chemicals: Detailed Findings," September 24, 2008, available at https://www.ewg.org/research/teen-girls-body-burden-hormone-altering-cosmetics-chemicals/detailed-findings

[21] DTSC is considering listing (1) beauty, personal care, and hygiene products and (2) cleaning products containing 1,4-dioxane as "Priority Products" and then proposing an Alternatives Analysis Threshold (AAT) of 1 ppm, which is the level at which manufacturers would be required to develop an alternatives analysis, stop selling the product, or reformulate. DTSC is proposing to restrict 1,4-dioxane in these products because when they are used and washed down the drain, they end up in wastewater and "contribute to continuous, low-level, widespread contamination of municipal wastewater with 1,4-dioxane." According to DTSC, this can be expected to lead to groundwater and drinking water contamination. DTSC's proposed AAT was subject to discussion at its August 21 public workshop and public comments are being accepted until August 30. CA DTSC. Safer Products: 1,4-Dioxane in Personal Care and Cleaning Products. Available at https://dtsc.ca.gov/scp/1-4-dioxane/ (last accessed August 29, 2019).

[22] California DTSC. 2019. 1,4-Dioxane in Personal Care and Cleaning Products: Seeking Input on Alternatives Analysis Threshold. P 3. Available at https://dtsc.ca.gov/wp-content/uploads/sites/31/2019/08/14-Dioxane-Draft-AAT-for-August-2019-Workshop.pdf

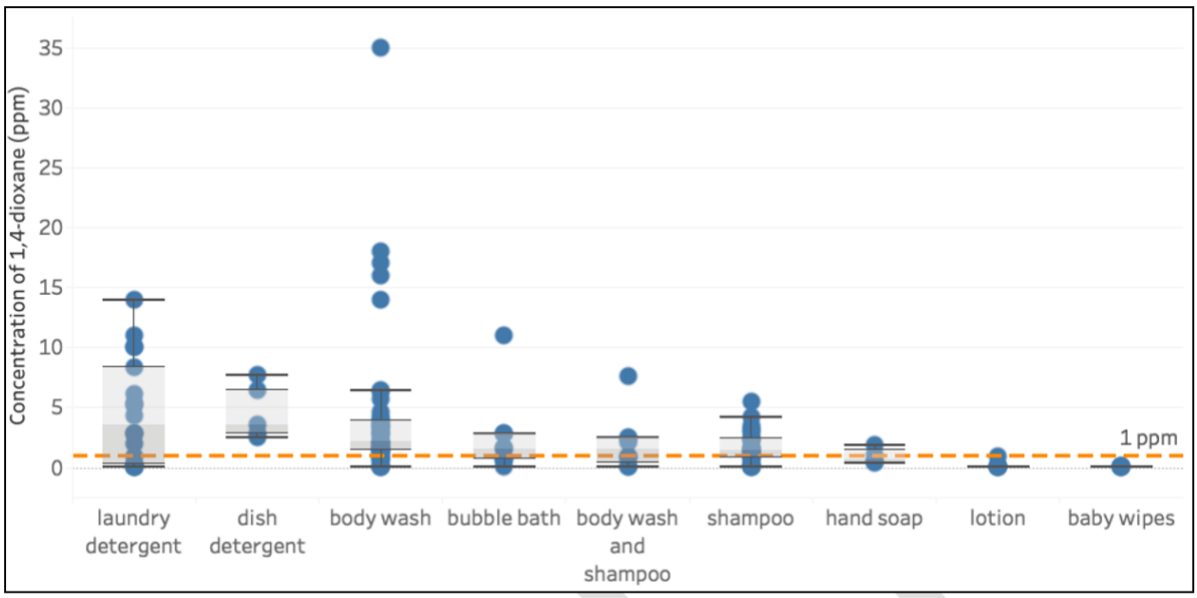


*Figure 1. 1,4-Dioxane in products from studies conducted within the last 10 years. Yellow dotted line represents the proposed AAT value of 1 ppm. Detailed data available in Table 1.*

It further summarized the results of available product testing as follows:

**Table 1.** Summary of 1,4-dioxane concentrations found in personal care and cleaning products in the last 10 years.[11,12]

| | | | 1,4-dioxane in product, $C_p$ (µg/g)* | |
|---|---|---|---|---|
| **Product Type** | **n** | **< 1 ppm** | **Max** | **Mean** |
| Dish detergent | 5 | 0% | 7.7 | 4.60 |
| Body wash | 42 | 19% | 35 | 4.43 |
| Laundry detergent | 18 | 33% | 14 | 4.61 |
| Shampoo | 23 | 39% | 5.5 | 1.66 |
| Bubble bath | 9 | 44% | 11 | 2.43 |
| Body wash and shampoo | 8 | 50% | 7.6 | 2.04 |
| Hand soap | 4 | 75% | 1.9 | 0.93 |
| Lotion | 6 | 100% | 0.92 | 0.16 |
| Baby wipes | 5 | 100% | 0.01 | 0.01 |

* 1 µg/g = 1 ppm

For laundry detergent, shampoo, and body wash, DTSC provided these estimates of the frequency of product use (and of exposure to 1,4-dioxane):

**Table 2**. Volume and frequency estimates for product use and activity.

| Product type (Activity) | Product use per activity $m_{pa}$ (g) | Water use per activity $V_a$ (L) | Number of times performed per person per year |
|---|---|---|---|
| Laundry detergent (load of residential laundry) | 98.5[14] | 189[15] | 100[16] |
| Shampoo (shower) | 13.1[18] | 65[19] | 312[17] |
| Body wash (shower) | 15.5[18] | 65[19] | 312[17] |

In short, 1,4-dioxane is present in numerous consumer products at significant levels and these products are likely used extensively by a large segment of the population, including children, pregnant women and other vulnerable and susceptible subpopulations.

### B. There is No Legal or Scientific Justification for Excluding Consumer Product Exposure from EPA's Risk Evaluation

The presence of 1,4-dioxane in consumer products is integral to understanding overall pathways and levels of consumer exposure and hence human health risk in three critical respects. First, consumers are directly exposed to 1,4-dioxane when using these products and are at risk of adverse health effects from that exposure. Second, when personal care and cleaning products are discharged "down the drain" following consumer use, 1,4-dioxane is not removed by most standard wastewater treatment systems and therefore passes through to surface water or groundwater, ultimately resulting in its presence in drinking water for human consumption. Third, workers exposed to 1,4-dioxane during its manufacture and processing may also use consumer products containing the chemical and ingest contaminated drinking water, adding to the levels to which they are exposed.

These sources of exposure are interrelated and cumulative and must be considered in combination to determine the total risk that 1,4-dioxane presents to the general population and vulnerable subpopulations.[23] However, EPA is not undertaking this comprehensive assessment because it has determined that "production of 1,4-dioxane as a by-product from ethoxylation of other chemicals and presence as a contaminant in industrial, commercial and consumer products will be excluded from the scope of the risk evaluation."[24] EPA has justified this exclusion on the ground that "[t]hese 1,4-dioxane activities will be considered in the scope of the risk evaluation for ethoxylated chemicals."

[23] General population risk is also influenced by air emissions and waste disposal. EPA ignores these pathways of exposure in its draft evaluation but they should be included in any comprehensive assessment of risk to the general population.

[24] US EPA. 2017. Scope of the Risk Evaluation for 1,4-Dioxane. P 8. Available at https://www.epa.gov/sites/production/files/2017-06/documents/dioxane_scope_06-22-2017.pdf ("EPA 2017 Scoping Document")

This rationale is simply not credible legally or scientifically. Whether produced intentionally or as a byproduct during ethoxylation, 1,4-dioxane is "manufactured" as defined in TSCA and its presence in consumer products (whether intended or not) is a "condition of use" under section 3(4) because it is a "circumstance. . . under which [1,4-dioxane] is known or reasonably foreseen to be manufactured. . . ." Section 6(b)(4) of TSCA requires risk evaluations to address "the conditions of use" of a chemical substance and EPA has no discretion to address some conditions of use but not others.[25] Even if EPA had such discretion, it could not defensibly exclude conditions of use simply because they involve "unintentional" manufacture of a substance since the purpose of producing a chemical is unrelated to its potential for exposure and risk.[26]

Moreover, the reality is that EPA has no plan or timetable to conduct a risk evaluation on ethoxylated chemicals and, given its constrained resources and the limited number of substances it is required to evaluate under the law, there is no realistic possibility of such an evaluation in the foreseeable future.[27] Congress required that the 1,4-dioxane risk evaluation be completed by December 22, 2020, or six months later if an extension is needed. Any risk evaluation for ethoxylated chemicals – assuming it is conducted at all – would be initiated long after this deadline passes and thus would not excuse EPA's failure to conduct a complete evaluation addressing all of 1,4-dioxane's conditions of use within the timeframe set by Congress.

Finally, even if EPA were able to evaluate ethoxylated chemicals any time soon, this bifurcated approach would not protect public health. Conducting separate risk evaluations on intended and byproduct uses of 1,4-dioxane will mean that neither evaluation will provide a complete or accurate picture of the chemical's health risks. This is because both sets of uses contribute to total human exposure and cannot be meaningfully evaluated in isolation from each other. Thus, as SACC members noted in their review of the draft evaluation, consumer product exposures are best addressed in combination with all other sources of exposure to 1,4-dioxane. EPA's failure to do so as part of the ongoing risk evaluation will result in a serious understatement of human health risk and fail to protect "potentially exposed or susceptible subpopulations," in violation of section 6(b)(4) of TSCA.

For these reasons, EPA should remove the exclusion of consumer products from its risk evaluation and rework the draft evaluation so these products are assessed in combination with other contributors to 1,4-dioxane exposure.

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[25] In a challenge to EPA's framework rule for conducting risk evaluations under TSCA, a number of the signatory groups have taken the position that the law requires risk evaluations to address all conditions of use and that EPA cannot pick and choose among the uses that it assesses. *Safer Chemicals Health Families et al v. Environmental Protection Agency et al*. (Ninth Cir. No. 17-72260). This case has been briefed and argued but not yet decided.

[26] EPA's failure to consider the risks posed by 1,4-dioxane's manufacture as a byproduct stands in stark contrast to its refusal to consider HBCD's degradation products in its draft risk evaluation for that chemical. https://www.epa.gov/assessing-and-managing-chemicals-under-tsca/draft-risk-evaluation-cyclic-aliphatic-bromide-cluster In the HBCD risk evaluation, EPA is evaluating the chemical that causes 1,5,9-cyclododecatriene to be present as a contaminant in sediment, yet it still refuses to consider the risks associated with this degradation product. EPA makes no attempt to explain why byproducts are better addressed when evaluating the parent chemical, but degradation products are not.

[27] EPA notes in its Scoping Document that chemicals that are produced by ethoxylation and could contain 1,4-dioxane as an impurity include alkyl ether sulphates (AES, anionic surfactants) and other ethoxylated substances, such as alkyl, alkylphenol and fatty amine ethoxylates; polyethylene glycols and their esters; and sorbitan ester ethoxylates. (EPA 2017 Scoping Document at 21)

## II. EPA'S FAILURE TO ADDRESS 1,4-DIOXANE CONTAMINATION IN DRINKING WATER FURTHER UNDERSTATES EXPOSURE AND RISK

The draft evaluation only examines worker exposure to 1,4-dioxane during manufacture and processing. It does not address consumption of contaminated drinking water or the combined impact on the general population of concurrent consumption of drinking water and consumer products containing 1,4-dioxane.

The number of people who ingest, touch or breathe 1,4-dioxane in consumer products and contaminated drinking water is much greater than the number of impacted workers (and workers, too, are exposed to 1,4-dioxane outside their places of employment). Thus, the exclusion of these exposed populations results in an incomplete picture of how 1,4-dioxane impacts public health. Without considering the entire exposed population, EPA's determination that 1,4-dioxane does not present an unreasonable risk of injury is flawed and incomplete.

### A. Contamination of Drinking Water with 1,4-Dioxane is Widespread and Significant

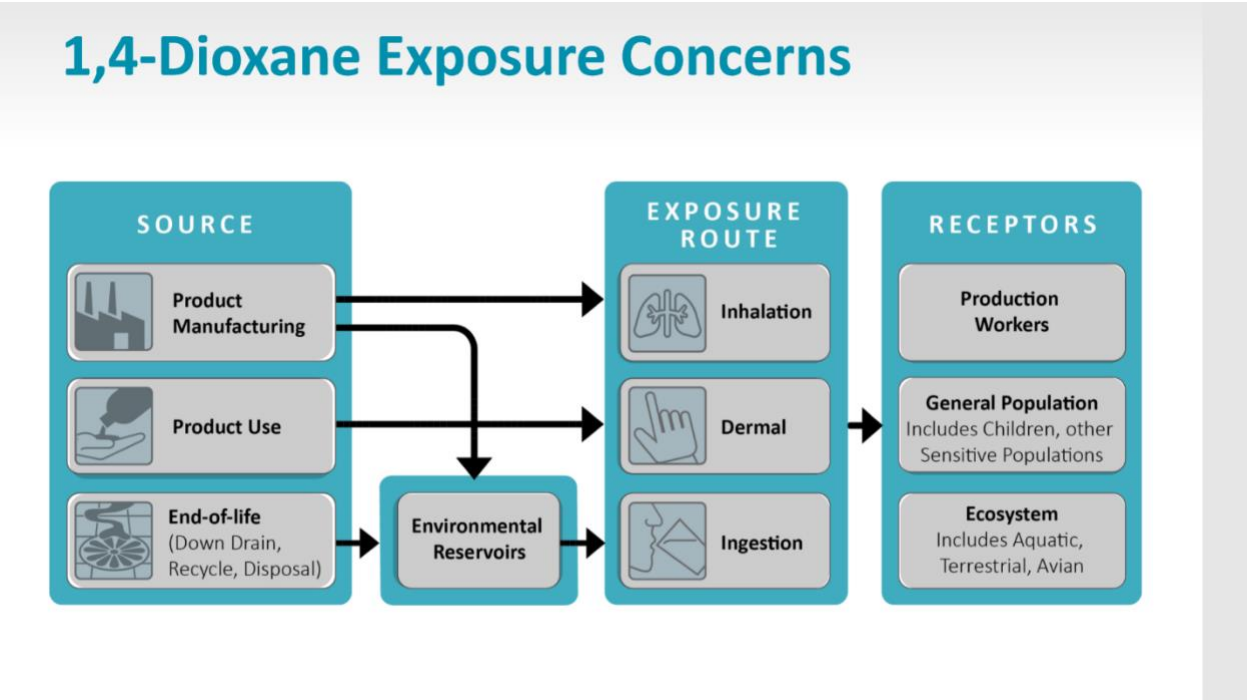
Contamination of drinking water with 1,4-dioxane is a long-standing concern in many regions of the US and has drawn considerable attention from local communities and state and federal regulators. According EWG, "1,4-Dioxane in drinking water sources can come from wastewater discharges, toxic waste and Superfund sites, as well as industrial facilities where plastics and solvents have been manufactured or used … Tracing this contamination to a specific source can be difficult because 1,4-dioxane can be carried, through ground or surface water, away from the original discharge site."[28] "Groundwater plumes that contain the chemical Trichloroethane (TCA) are very likely to also contain 1,4-dioxane. … The elevated levels found in many laundry detergents make laundromats a potential point-source of contamination for 1,4-dioxane."[29] "Down the drain" discharges of personal care and cleaning products contaminated with 1,4-dioxane also contribute to groundwater and drinking water contamination because it is not removed by most standard wastewater treatment systems and therefore is released to water bodies in their effluent.[30]

DTSC has depicted the sources of drinking water contamination and its role in overall exposure to 1,4-dioxane as follows:

[28] Benesh, Melanie, et al. "Hidden Carcinogen Taints Tap Water, Consumer Products Nationwide." September 6, 2017, available at https://www.ewg.org/research/hidden-carcinogen-taints-tap-water-consumer-products-nationwide ("2017 EWG Report").

[29] Citizens Campaign for the Environment, "Protect Drinking Water from 1,4-Dixoane," available at https://www.citizenscampaign.org/14dioxane (last accessed August 29, 2019).

[30] CA DTSC Background Document




EWG estimated that as of 2017, for the 2010-2015 dataset, 1,4-dioxane "was detected in samples of drinking water supplies for nearly 90 million Americans in 45 states."[31] It further concluded that, within the same timeframe, over 7 million people in 27 states were exposed to drinking water with levels above 0.35 ppb, a concentration that EPA has determined is "the amount of 1,4-dioxane expected to cause no more than one additional case of cancer in 1 million people who drink and bathe with the water over a lifetime."[32] These numbers probably underestimate the extent of contamination because medium and small water systems may not test regularly for 1,4-dioxane and private wells are not required to test at all.[33]

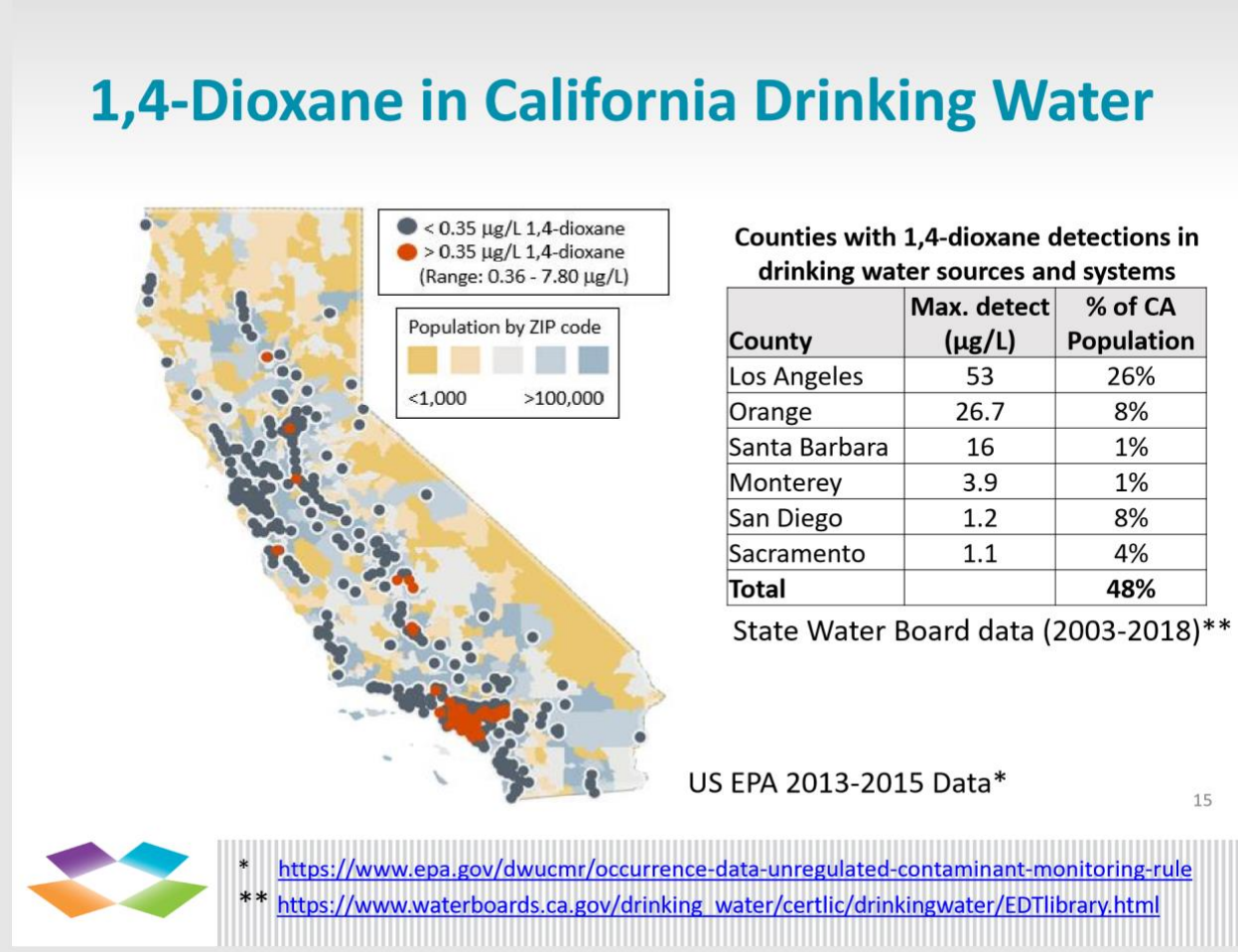
According to EWG, as of 2017 and for the 2010-2015 dataset, the states with the largest number of people exposed to at least 0.35 ppb of 1,4-dioxane in drinking water were "California, with 2.5 million people exposed; North Carolina, with 1.2 million; and New York, with 700,000."[34] Utilities with the highest average contamination levels (between 4x and 17x 0.35 ppb) were as follows:[35]

- 7 utilities in California (in and around LA): 1.4- 4.9 ppb
- 1 utility in MN (north of Minneapolis in New Brighton): 2.99 ppb
- 3 utilities in NJ (Camden and elsewhere): 2.03-2.7 ppb

[31] 2017 EWG Report
[32] As described by EWG in its 2017 report (2017 EWG Report)
[33] 2017 EWG Report
[34] Id.
[35] 2017 EWG Report, Table 1: https://cdn3.ewg.org/sites/default/files/u352/EWG_1%2C4-D_Table1_C03.pdf?_ga=2.92416349.1101494957.1566307622-1695567012.1541443516

- 5 utilities in NY (Long Island): 1.26-2.93 ppb. Note that 2013 testing of water from a Hicksville well found 33 ppb and the well was shut off "as soon as it was discovered to be contaminated."[36]
- 8 utilities in NC (Cape Fear River basin in and around Fayetteville): 2.02-5.83 ppb
- 1 utility in PA (near Pittsburgh in Beaver Falls): 2.66 ppb

California DTSC has depicted the presence of 1,4-dioxane drinking water contamination across the state as follows:[37]




According to DTSC, "combined exposure from product use and drinking water . . . is of particular concern for children and those with liver disease, … who may be more sensitive to exposure to 1,4-

[36] According to EWG's Tap Water database page for Hicksville WD, https://www.ewg.org/tapwater/system-contaminant.php?pws=NY2902829&contamcode=2049, and Mosco, Steve, "Glass Half Empty," *Hicksville News*, March 16, 2017, https://hicksvillenews.com/2017/03/16/glass-half-empty/.

[37] CA DTSC presentation slides entitled "1,4-Dioxane in Personal Care and Cleaning Products Public Meeting" dated June 28 2019, available at https://dtsc.ca.gov/wp-content/uploads/sites/31/2019/07/DTSC-Presentation.pdf.

dioxane than the general population."[38] In addition, "[e]nvironmental justice communities, which are already subject to socioeconomic and health stressors and other types of pollution … may be particularly impacted by the additional exposure to 1,4-dioxane from consumer products." DTSC's mapping of the overlap between these communities and drinking water contamination also shows that 1,4-dioxane "in [drinking water] in some of these communities exceeds levels of concern established by the U.S. EPA and the State Water Board."

Thus, in California and other states, drinking water contamination represents a widespread source of exposure and risk impacting millions of people and vulnerable subpopulations.

### B. EPA's Rationale for Disregarding Drinking Water Exposure Reflects a Misunderstanding of the Safe Drinking Water Act and an Indefensible Interpretation of TSCA

The exclusion of drinking water exposure from the 1,4-dioxane risk evaluation is an outgrowth of EPA's broader policy decision to ignore environmental release scenarios in TSCA risk evaluations. As stated in its Problem Formulation for 1,4-dioxane, EPA will not use these evaluations to address "pathways under other environmental statutes, administered by EPA, which adequately assess and effectively manage exposures and for which long-standing regulatory and analytical processes already exist."[39] Consistent with this rationale, EPA's position is that because "the drinking water exposure pathway for 1,4-dioxane is being addressed under the regular analytical processes to identify and evaluate drinking water contaminants" under the Safe Drinking Water Act (SDWA), there is no need to include this pathway in the risk evaluation for 1,4-dioxane under TSCA. As a result, "EPA did not evaluate hazards or exposures to the general population in this risk evaluation, and there is no risk determination for the general population" (Draft Evaluation at 156).

One might infer from these statements that EPA is actively engaged in using its SDWA authorities to limit the presence of 1,4-dioxane in drinking water to protect human health. However, the reality is far different. 1,4-dioxane was one of numerous contaminants for which drinking water utilities were required to conduct monitoring under EPA's third Unregulated Contaminant Monitoring Rule (UMCR 3). Otherwise, however, EPA has not addressed 1,4-dioxane under SDWA. There is no National Primary Drinking Water Regulation under SDWA for 1,4-dioxane. Nor is there any realistic prospect that such a regulation will be developed given the lengthy process SDWA requires to select candidates for regulation, the protracted rulemaking process required for selected contaminants, the large number of candidates for regulation and the nearly complete absence of new drinking water regulations over the last few decades. Thus, it is fanciful to assume that anything resembling a TSCA risk evaluation will be conducted for 1,4-dioxane under SDWA. If drinking water sources of exposure are not included in the ongoing TSCA risk evaluation, they will likely never be addressed.

Just as EPA lacks authority to exclude consumer products containing 1,4 dioxane from its risk evaluation, so it lacks authority to ignore discharges of 1,4-dioxane to surface water and ultimately drinking water during the use of these products: these discharges are likewise "conditions of use" of 1,4-dioxane that

[38] CA DTSC Background Document, at P 4.
[39] US EPA. 2018. "Problem Formulation of the Risk Evaluation for 1,4-Dioxane." P 42. Available at https://www.epa.gov/sites/production/files/2018-06/documents/14-dioxane_problem_formulation_5-31-18.pdf

EPA must evaluate under section 6(b)(4) of TSCA. Moreover, in evaluating these uses, EPA must consider not only risks to the general population but risks to "potentially exposed or susceptible subpopulations." As discussed above, California and others have identified vulnerable populations (including children, people with liver disease and environmental justice communities) who ingest contaminated drinking water, and workers also have elevated risks because they are exposed through both workplace and general population pathways.

Even if SDWA did provide an effective vehicle to regulate 1,4-dioxane in drinking water, it would not result in a comprehensive evaluation of risk from all pathways of exposure, including drinking water contamination, use of consumer products, air emissions, waste disposal, and inhalation or dermal contact in the workplace. The purpose of TSCA is to fill this gap by requiring a holistic examination of total risk from all sources. Thus, risk evaluations under section 6(b)(4)(A) must determine "whether a chemical substance presents an unreasonable risk of injury to health or the environment" – a requirement that entails examining all sources of exposure relevant to the chemical substance as a whole. 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 environmental releases that result in pathways of human exposure, whether they might theoretically be controlled under other environmental laws or not.

If Congress had intended a blanket exemption for environmental releases 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 derive from environmental exposure pathways and could be addressed under other environmental laws.

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

In amending TSCA in 2016, Congress sought to promote "effective implementation" of the 1976 law's objectives. See S. Rep. No. 114-67, 114th Cong., 1st Sess. (2015) at 2. 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."[40] Excluding all pathways of chemical exposure through air, water and soil from risk evaluations would be directly contrary to these Congressional expectations.

EPA's position that other environmental laws should displace TSCA risk evaluations for *all* chemicals arbitrarily assumes that these laws provide equivalent protection of public health and the environment and that there is no added benefit in addressing environmental pathways of exposure under TSCA. But in reality, these other laws vary greatly in the degree of protection they afford against chemical risks and the extent of their application to unsafe chemicals. These limitations are precisely why Congress gave EPA comprehensive authority over chemical risks under TSCA in 1976 and strengthened that authority in 2016. SDWA is a prime example. It does not require EPA to evaluate the risks of specific contaminants unless it selects them for drinking water standards – a path that EPA has followed for only a handful of chemicals since SDWA was amended in 1997 and is extremely unlikely for 1,4-dioxane. And even in the remote event 1,4-dioxane were selected for regulation, the TSCA risk-based framework would be more protective than SDWA's, which requires cost-benefit balancing in setting limits for drinking water contaminants, the very approach rejected in the 2016 TSCA amendments.[41]

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

In 2016, Congress underscored the chemical-specific focus of this analysis by revising section 9(b)(2) so that, in deciding whether to regulate under TSCA or another law, EPA must "consider . . . all relevant aspects of the risk" in question and make a "comparison of the estimated costs and efficiencies" of addressing the risk under TSCA and other laws. Commenting on this language, the law's Senate Democratic sponsors explained that it allowed EPA to regulate under other laws in lieu of TSCA only where the "Administrator has already determined that a risk to health or the environment associated with a chemical substance or mixture could be eliminated or reduced to a sufficient extent by additional actions taken under other EPA authorities."[42]

This approach presupposes that EPA has already used the TSCA risk evaluation process to identify the risks of a chemical and the exposure pathways contributing to those risks and thus has an informed basis to determine whether they "could be eliminated or reduced to a sufficient extent" under another law. However, if EPA has not examined the specific pathways of environmental exposure and their

[40] Congressional Record – Senate 3517 (June 7, 2016).
[41] 42 U.S.C. §300g-1
[42] Congressional Record – Senate 3517 (June 7, 2016).

contribution to total risk under TSCA, then it cannot conduct the analysis that section 9(b) requires because it will be unable to evaluate the relative strengths of using TSCA or another law to eliminate the risk. By presuming that other laws are *always* superior to TSCA in identifying and reducing the risks of chemicals in environmental media, EPA's blanket exclusion of environmental releases thus turns section 9(b) on its head.

In sum, EPA's exclusion of drinking water exposure from its risk evaluation is unjustified under TSCA and will result in an understatement of 1,4-dioxane's risks. EPA should remove this exclusion and revise the draft evaluation so it addresses the contribution of drinking water contamination to total risk in combination with other sources of exposure. The proper approach is for EPA to aggregate exposures from these multiple pathways in order to account for the overall exposure to 1,4-dioxane by the general population and vulnerable subpopulations.

## III. EPA'S EVALUATION OF RISKS TO WORKERS IS FLAWED AND DOES NOT SUPPORT EPA'S CONCLUSION THAT NEARLY ALL OCCUPATIONAL EXPOSURE SCENARIOS DO NOT PRESENT UNREASONABLE RISKS

Having excluded consumer and general population exposure, EPA's evaluation of 1,4-dioxane's risks to human health is focused entirely on exposure by workers during the course of their employment. Despite this narrow scope, EPA identifies a wide range of industrial activities where worker exposure to 1,4-dioxane may occur and a large occupational population (over 4 million workers) that may be exposed. Using cancer risk as the driver, EPA determines risk levels to workers and occupational non-users (ONUs) for each discrete activity and use sector. It concludes that a few categories of workers (engaged in the manufacture, use as an intermediate and disposal of 1,4-dioxane) face an unreasonable risk of cancer but the bulk of the exposed worker population does not.

EPA's conclusion that 1,4-dioxane does not present an unreasonable risk of injury to most workers is flawed and unjustified in several respects: (1) the worker exposure information EPA relies on is limited and unreliable; (2) EPA does not aggregate multiple exposure pathways in determining overall worker risks; (3) the Agency's risk determination is based on the fallacious and unsupportable assumption that workers are using personal protective equipment (PPE) on a continuous basis; and (4) EPA's risk determinations for workers emphasize central tendency exposure levels and give less weight to high-end exposures.

### A. The Lack of Sufficient Monitoring Data and Limited Information about Several 1,4-Dioxane Uses Create Large Uncertainties in EPA's Determinations of Worker Risk

EPA's risk determinations for workers are based on limited and poorly described workplace monitoring data by the inhalation route that it then extrapolates to a wide variety of manufacturing and processing conditions despite its uncertain relevance. The principal source of data for EPA's analysis is monitoring conducted by BASF, a 1,4-dioxane manufacturer. However, EPA itself recognized that "the BASF data had limitations including lack of descriptions of worker tasks, exposure sources, and possible engineering controls" (Draft evaluation at 54). EPA also relied on workplace exposure estimates provided in the EU Risk Assessment for 1,4-dioxane to assess exposures in industrial applications but similarly acknowledged that "[t]he data sets used are limited and mostly lacked specific descriptions of

worker tasks, exposure sources, and possible engineering controls to provide context" and were "only presented in ranges with key statistics (i.e. median or average and 90th percentile), so EPA was unable to directly calculate final values from the raw data and relied on estimates provided in the 2002 EU Risk Assessment" (Draft evaluation at 60). [43]

In all, EPA identified 13 separate industrial applications from diverse sectors where worker exposure to 1,4-dioxane is expected. The Agency acknowledged that it "did not find specific details for most of these processes" but claimed that "typical operations are expected to be similar across these uses" (p. 58). This assumption is arbitrary and simplistic given the obvious differences between, for example, wood pulping and pharmaceutical manufacturing and the large number of proposed workers EPA estimated for some operations (4,094,000 plus 178,000 bystanders alone for functional fluids used in open systems).

Moreover, EPA has no worker monitoring data for dermal exposure and relies entirely on modeling to estimate dermal exposure levels.

The absence of detailed information on the conditions of worker exposure and 1,4-dioxane concentrations during worker operations could have been avoided if EPA had used its TSCA section 4 and 8 information collection authority to obtain all existing worker monitoring data and related information from industry and to require industry to conduct additional monitoring where necessary to reliably assess occupational risks. Combined with the serious data gaps on toxicity, the high level of uncertainty in EPA's assessment of occupational exposure calls into question the validity of EPA's determinations of cancer and non-cancer risk to exposed workers.

### B. EPA Improperly Concluded that Aggregate Exposure Assessment Was Not Warranted for Workers

EPA understated risks to workers by ignoring the contribution of consumer products and drinking water to overall worker exposure. In reality, workers have multiple routes of exposure, including both in their places of employment and in their homes, where they may use consumer products and ingest drinking water containing 1,4-dioxane. The most protective approach would be to conduct an "aggregate exposure assessment" that accounts for the total exposure and risk resulting from these combined sources. Section 6(b)(4)(F) of TSCA directs EPA to consider using such aggregate exposure methodologies. However, EPA's draft concludes – wrongly – that, because "of the limited nature of all

[43] Given its acknowledgement of the limitations of the monitoring data it relied on, it is surprising that EPA declined to use NIOSH and OSHA monitoring studies that likely provided useful information, considering that they received some of the highest scores in EPA's systematic review rating process. These studies were among 44 engineering release and occupational exposure studies that EPA rated "acceptable" under its systematic review criteria but eliminated due to its "hierarchy of preferences." Moreover, EPA rejected as "unacceptable" a highly relevant OSHA monitoring study because the data were in a text file instead of an Excel file (see pg. 105 of the 1,4-dioxane Supplemental File: Data Quality Evaluation of Environmental Releases and Occupational Exposure Data). Instead, EPA included studies from NICNAS (the Australian government's regulatory body for industrial chemicals) and lower scoring studies from BASF despite both a commitment to interagency collaboration (as per EPA's risk evaluation process rule and stating in the draft risk evaluation that the Agency prefers monitoring studies over modeling studies (HBCD Draft Risk Evaluation for Cyclic Aliphatic Bromide Cluster pgs 175-176).

routes of exposure to individuals (i.e., occupational) resulting from the conditions of use of 1,4-dioxane, a consideration of aggregate exposures of 1,4-dioxane was deemed not to be applicable for this risk evaluation" (p. 152). EPA should recalculate its determinations of risk to workers to account for aggregate 1,4-dioxane exposure from the workplace, consumer products and drinking water.

EPA further understates worker exposures and risk by calculating risk levels for dermal and inhalation exposure separately and not combining them to account for concurrent exposure by both routes. This is unjustified since workers are likely exposed to 1,4-dioxane both by breathing it and by dermal contact during the workplace uses addressed in EPA's risk evaluation.

### C. EPA Lacks any Legal or Technical Basis to Conclude that Cancer Risks that Exceed Its Benchmark Are Not Unreasonable Because Workers Will Use PPE

To determine whether cancer risks to workers are unreasonable, EPA uses a risk benchmark of $1 \times 10^{-4}$: risks exceeding this threshold are considered unreasonable, whereas lower risks are not. (Given that EPA has previously used a risk range of $1 \times 10^{-4}$ to $1 \times 10^{-6}$ as a trigger for regulatory action, the selection of the upper end of this range to define unreasonable risk is arbitrary and unprotective.[44]) For several workplace exposure scenarios, EPA calculates cancer risks above the benchmark in the absence of personal protective equipment (PPE), but then determines that use of PPE would lower the risk below the benchmark for most exposed workers. On this basis, it concludes that risks to these workers are not unreasonable.

EPA's position that the use of PPE is effectively protecting workers from unsafe exposure is not grounded in reality. OSHA has a permissible exposure limit (PEL) for 1,4-dioxane, but it is several decades old and is set at a level (100 ppm or 360 mg/m3 as an 8 hour TWA[45]) that does not reflect currently available carcinogenicity data and fails to protect workers against significant cancer risk.[46] It is doubtful that employers are implementing more stringent exposure limits in the absence of any legal obligation to do so. Thus, even if some PPE are in use during certain workplace tasks, they are likely insufficient to reduce exposure to levels that provide effective protection against cancer risks. As for the claimed obligation of employers 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

[44] 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., 1x10-6 to 1x10-4) depending on the subpopulation exposed. Generally, EPA considers 1 x 10-6 to 1x 10-4 as the appropriate benchmark for the general population, consumer users, and non-occupational potentially exposed or susceptible subpopulations (PESS)" (p. 155). However, EPA then asserts that $1 \times 10^{-4}$ should be the unreasonable risk threshold for occupational exposure based on OSHA precedent. 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.

[45] 29 CFR § 1910.1000. The OSHA standard was adopted as part of the TLV list established shortly after OSHA's creation and does not include any PPE requirements.

[46] By contrast, California OSHA has set a limit of 0.28 ppm and the NIOSH Recommended Exposure Limit (REL) is 1 ppm as a 30-minute ceiling. The 8-hour TWA exposure levels that EPA presents in its risk evaluation based on limited monitoring and modeling exceed the California and NIOSH limits for most industrial uses. Moreover, if representative of industry practice, the sparse monitoring data cited in the EPA evaluation indicates that workplace protection programs for 1,4-dioxane are at best sporadic and limited in scope.

protection measures they deem appropriate.[47] It is implausible that employers (including many small businesses) have the resources or expertise to analyze the carcinogenicity data base for 1,4-dioxane and set and enforce stringent exposure limits that OSHA itself has not adopted. As EPA's risk evaluation itself recognizes, "[t]he use of a respirator would not necessarily resolve inhalation exposures since it cannot be assumed that employers have or will implement comprehensive respiratory protection programs for their employees" (p. 53).[48] Similarly, while EPA assumes that workers would always be provided and consistently use impervious gloves, it acknowledges that "[d]ata about the frequency of effective glove use – that is, the proper use of effective gloves – is very limited in industrial settings" (p. 293). Occupational bystanders are even less likely than directly exposed workers to uniformly wear effective gloves.

EPA's reliance on PPE to eliminate otherwise unreasonable risks is contrary to real-world experience in the workplace that EPA itself has previously cited to demonstrate that the use of PPE cannot effectively mitigate risks to workers and that stronger protections such as a ban on certain chemical uses are necessary to satisfy TSCA.[49] It is well-known and documented that respirators and other protective gear are used intermittently by workers even where they are legally required, that Safety Data Sheets (SDS) and directions for safe use are often misunderstood or ignored, and that employers often fail to provide adequate training and equipment to their workers. This is why OSHA and other worker protection authorities first examine whether serious risks can be addressed with improved engineering controls and work practices and turn to PPE only as a last resort.[50] Like OSHA, EPA should make determinations of risk to workers without taking into account the effects of PPE; whether engineering controls, PPE or additional measures should be required is a risk management decision that should follow a determination of unreasonable risk to workers.[51] The possible use of PPE should not be an excuse to conclude that unsafe workplace exposure levels do not present an unreasonable risk.

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[47] 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).

[48] According to the risk evaluation, "[t]he complexity and burden of wearing respirators increases with increasing APF [Assigned Protection Factor]" (p. 53). For a number of use categories, EPA determined that respirators with APFs of 50 or above were needed on a continuous basis to reduce cancer risks to levels that EPA deems not unreasonable.

[49] Methylene Chloride and N-Methylpyrrolidone; Regulation of Certain Uses Under TSCA Section 6(a), 82 Fed. Reg. 7464, 7481 (Jan. 19, 2017).

[50] If a chemical presents a significant risk, OSHA and NIOSH manage that risk using the "hierarchy of controls," under which hazard elimination, substitution, engineering and administrative controls are all prioritized over the use of PPE. OSH, Ctrs. for Disease Control & Prevention, updated Jan. 13, 2015, https://www.cdc.gov/niosh/topics/hierarchy/. The hierarchy of controls has been endorsed by OSHA, NIOSH, the American Society of Safety Engineers, the American Industrial Hygiene Association, the American Conference of Governmental Industrial Hygienists, the American Public Health Association, the American Federation of Labor and Congress of Industrial Organizations, and many others. As explained by NIOSH, "[t]he hierarchy of controls normally leads to the implementation of inherently safer systems" because chemical regulation and substitution are "more effective and protective" than PPE. Id.

[51] Indeed, EPA's own draft evaluation recognizes that "[t]he most effective controls are elimination, substitution, or engineering controls. Respirators, and any other personal protective equipment, are 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" (p 52).

Not only has EPA ignored the considerable body of evidence calling into question the effectiveness of PPE but it assumes that PPE are reliably and consistently used by 1,4-dioxane manufacturers and users without any confirmatory evidence. For example, it could have surveyed companies about their workplace practices and visited plants to observe how 1,4-dioxane is used and handled and ascertain whether and when PPE is in place. Yet it took none of these steps and thus has no real-world basis to rely on PPE for the absence of unreasonable risk. Unfortunately, this lack of diligence extended to other aspects of EPA's worker exposure assessment: it could have inspected plants in different use sectors to observe the 1,4-dioxane processing conditions and required industry to submit or conduct additional workplace monitoring but failed to do so, instead relying on speculation and modeling that reduce the transparency, precision and reliability of its determinations of risks to workers.

### D. Central Tendency Exposure Levels Should Not Drive Risk Determinations to the Exclusion of High-End Exposures

EPA's assessment of workplace risks differentiates between "central tendency" and "high-end" exposure levels. According to the draft evaluation, a "central tendency is assumed to be representative of exposures in the center of the distribution (50th percentile) for a given condition of use." p. 151. By definition, the "central tendency" would thus exclude the large portion of the worker population with higher levels of exposure. EPA apparently believes that it can base its determinations of unreasonable risk on central tendency exposure levels, discounting the risks to highly exposed workers where the central tendency risks are not unreasonable. However, TSCA explicitly rules out this approach.

Section 3(12) of the law defines a "potentially exposed or susceptible subpopulation" as a "group of individuals . . who, due to . . . greater exposure, may be at greater risk . . . of adverse health effects from exposure to a chemical substance." The law specifically identifies workers as such a subpopulation. Under section 6(b)(4)(A) of TSCA, EPA risk evaluations must determine whether the substance "presents an unreasonable risk of injury" to a "potentially exposed or susceptible subpopulation." Thus, EPA must address whether risks to highly exposed workers are unreasonable, and, if so, must make a determination to that effect. It cannot conclude that 1,4-dioxane does not present an unreasonable risk based solely on central tendency exposure levels.[52]

## IV. NUMEROUS GAPS IN HEALTH AND ENVIRONMENTAL EFFECTS DATA WEAKEN THE STRENGTH OF THE DRAFT EVALUATION AND COULD HAVE BEEN ADDRESSED BY EPA

As with the PV29 evaluation, EPA's risk determinations for 1,4-dioxane are weakened by numerous data gaps that should have been identified and addressed before conducting the evaluation. These data gaps create major uncertainties in EPA's assessment of hazard and exposure for human health and ecological endpoints.

As EPA acknowledges, it has only limited and inadequate data on 1,4 dioxane's reproductive and developmental toxicity and no data at all on its developmental neurotoxicity. No studies have been

[52] For the most part, EPA has considered high-end exposure levels in its risk determinations, but this is not uniformly the case.

conducted on endocrine effects and immunotoxicity. Nor have dermal toxicity studies been conducted for any endpoint, despite the prominence of this route of exposure in EPA's assessment of risk to workers. The result is that EPA seeks to quantify dermal toxicity values by the highly uncertain technique of extrapolating inhalation toxicity studies to dermal exposure. The data on dermal absorption are also limited and, as EPA itself acknowledges, insufficient to conclude that absorption by this route is insubstantial. Given the extensive assessments of 1,4-dioxane by EPA's IRIS program, ATSDR, the EU, and OPPT itself in 2015, EPA was undoubtedly aware of these data gaps several years ago and could have used its TSCA testing authority to require the missing studies had it sought to do so.

EPA guidance calls for an Uncertainty Factor (UF) of up to 10 "to account for the potential for deriving an underprotective RfD/RfC as a result of an incomplete characterization of the chemical's toxicity."[53] Where a prenatal toxicity study and a two-generation reproduction study are missing, the full factor of 10 is typically applied.[54] Since this is the case for 1,4-dioxane, the Benchmark MOE for acute/short-term inhalation risks should be increased from 300 to 3,000, and the chronic inhalation and dermal Benchmark MOEs from 30 to 300. For a number of worker groups, this would mean that the MOE would no longer be adequate, in some cases even where PPE is used, and the EPA risk determinations would shift from reasonable to unreasonable.

Although EPA concludes that 1,4-dioxane does not present an unreasonable risk of injury to the environment, there is a dearth of data in the draft evaluation to support this conclusion. EPA claims that "recent monitoring data on ambient surface water levels indicate relatively low levels" (p. 213) but cites no data to support this claim. Moreover, the evaluation only provides one environmental fate and transport study and heavily relies on modeling to estimate other fate and transport parameters. Most significantly, EPA relies on acute toxicity data for only a few organisms and cites chronic toxicity data only for fish. These gaps – which EPA easily could have required industry to fill – preclude scientifically supportable determinations about 1,4-dioxane's risks to the environment.

## V. BECAUSE THERE IS NO SCIENTIFIC SUPPORT FOR A THRESHOLD MOA, EPA'S CANCER RISK ESTIMATES SHOULD BE BASED ON A LINEAR LOW DOSE EXTRAPOLATION

1,4-dioxane has demonstrated carcinogenic effects in multiple animal species and strains, in males and females, and by inhalation and drinking water ingestion. In rodent studies, it has caused liver, nasal cavity, kidney, peritoneal, mammary gland, Zymbal gland, and subcutis tumors. Few chemicals have induced so many tumors across such a broad spectrum of test systems. In its 2013 IRIS assessment, EPA concluded that "1,4-dioxane is 'likely to be carcinogenic to humans' based on evidence of multiple tissue carcinogenicity in several 2-year bioassays conducted in three strains of rats, two strains of mice, and in

[53] "A Review of the Reference Dose and Reference Concentration Processes," December 2002, Prepared for the Risk Assessment Forum U.S. Environmental Protection Agency, available at https://www.epa.gov/sites/production/files/2014-12/documents/rfd-final.pdf

[54] Dourson, ML; Felter, SP; Robinson, D. (1996) Evolution of science-based uncertainty factors in noncancer risk assessment. Regul Toxicol Pharmacol 24:108–120

guinea pigs."[55] After reviewing these studies, the draft risk evaluation incorporates this conclusion. (p. 107)

However, the draft evaluation then presents cancer risk estimates using linear and non-linear low dose extrapolation methods and leaves the door open to basing its final evaluation on the non-linear approach, which would greatly understate 1,4-dioxane's cancer risks. There is no scientific or policy justification for this approach: it is contrary to the Agency's cancer risk assessment guidelines, the conclusions of its 2013 IRIS assessment, the findings of state regulators, and the detailed analysis of 1,4-dioxane's mode of action (MOA) in the draft TSCA evaluation itself.

EPA's 2005 Guidelines for Carcinogen Risk Assessment emphasize the high level of evidence necessary to depart from the presumption of linearity for carcinogens:[56]

> 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. (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's 2013 peer-reviewed IRIS assessment for 1,4-dioxane concluded that there was inadequate evidence for a non-linear MOA:

> Dose-response and temporal data support the occurrence of cell proliferation prior to the development of liver tumors (JBRC, 1998; Kociba et al., 1974) in the rat model. However, the dose-response relationship for induction of hepatic cell proliferation has not been characterized, and it is unknown if it would reflect the dose-response relationship for liver tumors in the 2-year rat and mouse studies*. Conflicting data from rat and mouse bioassays (JBRC, 1998; Kociba et al., 1974) suggest that cytotoxicity may not be a required precursor event for 1,4-dioxane-induced cell proliferation. Data regarding a plausible dose response and temporal progression (see Table*

[55] US EPA. 2013. Toxicological Review of 1,4-Dioxane (with inhalation update) In Support of Summary Information on the Integrated Risk Information System (IRIS). Available at https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0326tr.pdf

[56] https://www.epa.gov/sites/production/files/2013-09/documents/cancer_guidelines_final_3-25-05.pdf

*4-21) from cytotoxicity and cell proliferation to eventual liver tumor formation are not available. Also, Kociba et al. (1974) reported renal degeneration, necrosis, and regenerative proliferation in exposed rats, but no increase in the incidence of kidney tumors, which does not support a cytotoxicity/cell proliferation MOA.*

For nasal tumors, there is a hypothesized MOA that includes metabolic induction, cytotoxicity, and regenerative cell proliferation (Kasai et al., 2009). The induction of CYP450 has some support from data illustrating that following acute oral administration of 1,4-dioxane by gavage or drinking water, CYP2E1 was inducible in nasal mucosa (Nannelli et al., 2005). CYP2E1 mRNA was increased approximately two- to threefold in nasal mucosa (and in the kidney, see Section 3.3) in the Nannelli et al. (2005) study. While cell proliferation was observed following 1,4-dioxane exposure in both a 2-year inhalation study in male rats (1,250 ppm) (Kasai et al., 2009) and a 2-year drinking water study in male (274 mg/kg-day) and female rats (429 mg/kg-day), no evidence of cytotoxicity in the nasal cavity was observed (Kasai et al., 2009); therefore, cytotoxicity, as a key event, is not supported. Nasal lesions, including inflammation, hyperplasia, and metaplasia, were frequently seen in inhalation studies conducted by the NTP with no evidence of nasal carcinogenicity (Haseman and Hailey, 1997; Ward et al., 1993). Following a 13-week inhalation study in rats, a concentration-dependent increase of 1,4-dioxane in the blood was observed (Kasai et al., 2008). Studies have shown that water-soluble, gaseous irritants cause nasal injuries such as squamous cell carcinomas (Morgan et al., 1986). Similarly, 1,4-dioxane, which has been reported as a miscible compound (Hawley and Lewis, 2001), also caused nasal injuries that were concentration-dependent, including nasal tumors (Kasai et al., 2009). Additionally, it has been suggested that in vivo genotoxicity may contribute to the carcinogenic MOA for 1,4-dioxane (Kasai et al., 2009) (see Section 4.7.3.6 for further discussion). *Collectively, these data are insufficient to support the hypothesized MOAs.*

There are no data available regarding any hypothesized MOA by which 1,4-dioxane produces kidney, lung, peritoneal (mesotheliomas), mammary gland, Zymbal gland, and subcutis tumors (p.91-92) (emphasis added).

Although some new information has become available since 2013, the conclusions of the draft risk evaluation mirror the IRIS analysis:

*The relationship between cell proliferation, hyperplasia, and 1,4-dioxane mediated tumor formation has not been established.* Though several publications (Dourson et al., 2017; Dourson et al., 2014; McConnell, 2013) do provide evidence of cytoplasmic vacuolar degeneration and hepatocellular necrosis in rat and non-neoplastic lesions, the animal data does not support a dose response relationship between cell proliferation, hyperplasia, and liver tumors in rat and mouse studies. Kociba et al. (1974) reported hepatic degeneration and regenerative hyperplasia at or below dose levels that produced liver tumors, but incidence for these effects was not reported. *Therefore, a dose-response relationship could not be evaluated, and the events cell proliferation and hyperplasia are not supported by available data. Finally, the doses in hepatotoxicity studies where cytotoxicity and cell proliferation were observed were greater than cancer bioassay dose levels. Integrating data across studies, dose-response relationships*

*between cytotoxicity and tumor formation are not well established in the rat and mouse data and are inconsistent among bioassays and across exposure duration.*

*EPA determined that evidence is not sufficient to support a MOA of cytotoxicity followed by sustained cell proliferation as a required precursor to tumor formation related to the metabolic saturation and accumulation of the parent compound, 1,4-dioxane (Dourson et al., 2017; Kociba et al., 1975).* In addition, while genotoxicity is evident from high doses with in vitro and in vivo studies the occurrence at high doses and potential confounding with cytotoxicity does not support a mutagenic mode of action hypothesis at low doses in vivo. Other than liver tumors, no plausible MOA has been hypothesized for the other tumor types associated with exposure to 1,4dioxane. As a result, the proposed dose response approach for liver and other tumors is to show best fit of threshold and linear models applied to tumor data and linear default extrapolation in the absence of known MOA (emphasis added) (p. 101)

Thus, the draft evaluation concluded that:

> For cancer risk estimates, in the absence of a known MOA for liver tumors or other tumor types, a linear low-dose extrapolation approach was used to estimate the dose-response at doses below the observable range. *There was a high degree of uncertainty in any of the MOA hypotheses considered in this evaluation (e.g., mutagenic mode of action or threshold response to cytotoxicity and regenerative hyperplasia for liver tumors). Linear extrapolation is the default approach when there is uncertainty about the MOA* (p. 150) (emphasis added).

Two states have similarly considered recent claims of a threshold MOA for 1,4-dioxane carcinogenicity and rejected them. [57] For example, New Jersey carefully reviewed the 2014 paper by Dourson et al. reporting the results of a review of liver slides from the 1978 NCI cancer study.[58] The paper argued that 1,4-dioxane causes liver tumors in rats and mice through a pathway involving cytotoxicity (as indicated by hypertrophy and necrosis) followed by regenerative hyperplasia, and that a threshold approach is therefore appropriate for risk assessment. However, New Jersey scientists disagreed, explaining that:

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

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[57] https://www.michigan.gov/documents/deq/deq-aqd-toxics-14-DioxaneTSG_Report_2015_487415_7.pdf; https://www.state.nj.us/dep/dsr/supportdocs/11-chemicals-response.pdf.

[58] Dourson, M; Reichard, J; Nance, P; Burleigh-Flayer, H; Parker, A; Vincent, M; Mcconnell, EE. (2014). Mode of action analysis for liver tumors from oral 1,4-dioxane exposures and evidence-based dose response assessment. Regul Toxicol Pharmacol 68: 387-401.

Given the broad consensus (including by EPA scientists) that a threshold MOA for 1,4-dioxane is not supportable, EPA should remove its non-linear dose-response extrapolation from its final risk evaluation.

## CONCLUSION

The draft risk evaluation for 1,4-dioxane is incomplete and flawed and seriously understates risks to human health. EPA should make significant revisions to the draft evaluation to address its many deficiencies and reissue it for additional public comment and peer review.

We appreciate this opportunity to present comments on the draft 1,4-dioxane risk evaluation.

If you have any questions about these comments, please contact SCHF counsel, Bob Sussman, at bobsussman1@comcast.net or 202-716-0118.

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