April 28, 2023

***Via regulations.gov***

Dr. Michal Freedhoff
Assistant Administrator
U.S. Environmental Protection Agency
Office of Chemical Safety and Pollution Prevention
1200 Pennsylvania Ave., NW
Washington, D.C. 20460-0001

**Re: Comments on EPA's Draft Proposed Approach for Cumulative Risk Assessment of High-Priority Phthalates and a Manufacturer-Requested Phthalate Under the Toxic Substances Control Act, Docket No. EPA-HQ-OPPT-2022-0918**

Dear Assistant Administrator Freedhoff:

Please accept these comments on EPA's *Draft Proposed Approach for Cumulative Risk Assessment of High-Priority Phthalates and a Manufacturer-Requested Phthalate Under the Toxic Substances Control Act* (2023) (the "Phthalates CRA Proposal"). We strongly support EPA's proposal to assess the cumulative risks to human health from phthalates exposure pursuant to the Toxic Substances Control Act's ("TSCA") risk evaluation provisions.[1] TSCA authorizes EPA to conduct risk evaluations on "a category of chemical substances" and mandates that EPA conduct its risk evaluations in a manner consistent with the best available science.[2] Thus, contrary to EPA's assertion in the Phthalates CRA Proposal,[3] TSCA requires EPA to conduct a cumulative risk assessment where, as with phthalates, there is evidence of toxicological similarity across multiple chemicals and co-exposure over a relevant timeframe.[4] Given the robust evidence that multiple phthalates contribute to the same adverse health effects and that the general population and multiple higher-risk subpopulations experience significant co-exposures to multiple phthalates, we urge EPA to expeditiously implement its proposal to complete a phthalates cumulative risk assessment. Failing to do so would defy the best available science and substantially understate risk.

At the same time, EPA's current proposal for a phthalates cumulative risk assessment is too narrow. Among other changes proposed below, to satisfy TSCA and align with the best available science, EPA should expand its proposed cumulative chemical group and its consideration of higher-risk subpopulations, use readily available information to fill identified data gaps, address the effects of non-chemical stressors on human susceptibility to health harm from phthalate exposures, and assess the cumulative risks to wildlife from exposure to phthalates and related chemicals in the environment.

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[1] 15 U.S.C. § 2605(b).
[2] *Id.* § 2625(c)(1), (h).
[3] Phthalates CRA Proposal 17.
[4] *See* Comments of Earthjustice et al. re. Draft Proposed Principles of Cumulative Risk Assessment Under the Toxic Substances Control Act 3–6 (Apr. 28, 2023).

## I. EPA's Proposed Cumulative Chemical Group Correctly Includes Toxicologically Similar Substances but Omits Additional Antiandrogenic Chemicals

We support EPA's proposal to evaluate DEHP, BBP, DBP, DIBP, DCHP, and DINP (collectively, the "Proposed Cumulative Chemical Group") in a cumulative risk assessment. EPA's conclusion that these six phthalates satisfy the criteria for cumulative chemical grouping is consistent with the best available science. The National Research Council of the National Academy of Sciences recommends that the best approach for evaluating risk posed by chemicals of the same class is to conduct a cumulative risk assessment when there is substantial evidence of co-exposures and common adverse health outcomes.[5] In its proposal, EPA provides substantial evidence of simultaneous co-exposures to the chemicals in its Proposed Cumulative Chemical Group in the human population. EPA also correctly determined that the chemicals in its Proposed Cumulative Chemical Group all contribute to antiandrogenic effects, including phthalate syndrome, satisfying the criteria for cumulative chemical grouping based on toxicological similarity.

At the same time, TSCA requires EPA to account for cumulative risk from additional antiandrogenic chemicals. The best available science demonstrates that several additional chemicals, including other ortho-phthalates and non-phthalate chemicals with similar antiandrogenic effects, can act cumulatively in mixtures with the Proposed Cumulative Chemical Group substances.[6] These additional chemicals include the ortho-phthalates dihexyl phthalate and dipentyl phthalate, which are both found in U.S. household dust,[7] along with the registered pesticides linuron, vinclozolin, and procymidone. In several recent dose-response studies, EPA scientists documented the magnitude of the cumulative effects of mixtures containing these chemicals and chemicals in the Proposed Cumulative Chemical Group,[8] and collectively found that these mixtures induced antiandrogenic effects at doses that were orders of magnitude lower than those associated with antiandrogenic effects of individual phthalates. In addition, diisooctyl

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[5] Nat'l Rsch. Council, *Phthalates and Cumulative Risk Assessment: The Tasks Ahead* 3 (2008) ("NRC 2008"), https://www.nap.edu/catalog/12528/phthalates-and-cumulative-risk-assessment-the-tasks-ahead.

[6] Justin M. Conley et al., *Mixed "Antiandrogenic" Chemicals at Low Individual Doses Produce Reproductive Tract Malformations in the Male Rat*, 164 Toxicological Scis. 166 (2018), https://doi.org/10.1093/toxsci/kfy069; Justin M. Conley et al., *A Mixture of 15 Phthalates and Pesticides Below Individual Chemical No Observed Adverse Effects Levels (NOAELs) Produces Reproductive Tract Malformations in the Male Rat*, 156 Env't Int'l, art. no. 106615 (2021), https://doi.org/10.1016/j.envint.2021.106615; Kembra L. Howdeshell et al., *Cumulative Effects of Antiandrogenic Chemical Mixtures and Their Relevance to Human Health Risk Assessment*, 220 Int'l J. Hygiene & Env't Health 179 (2017).

[7] Susanna D. Mitro et al., *Consumer Product Chemicals in Indoor Dust: A Quantitative Metaanalysis of U.S. Studies,* 50(19) Env't Sci. & Tech. 10661 (2016), https://doi.org/10.1021/acs.est.6b02023.

[8] Conley et al. 2018; Conley et al. 2021.

phthalate, a known antiandrogenic ortho-phthalate,[9] remains approved for food-contact use in the United States.[10] This evidence indicates a need to consider the cumulative risks posed by these antiandrogenic chemicals in conjunction with the Proposed Cumulative Chemical Group. If EPA believes that it requires additional information on co-exposures to these chemicals in the human population to consider them for cumulative risk assessment, EPA should use its legal authority under TSCA to obtain this information expeditiously.

That these substances are not currently undergoing TSCA risk evaluation does not justify disregarding their cumulative effects. While EPA is not required to develop an individual risk determination for these additional antiandrogenic chemicals, it cannot rationally ignore evidence that they contribute to cumulative risk. Indeed, EPA already recognizes the imperative to consider chemical exposures from sources that are not subject to EPA's direct regulatory authority under TSCA by proposing to aggregate exposures to the Proposed Cumulative Chemical Group substances from so-called non-TSCA and non-attributable sources. Similarly, EPA has acknowledged that non-chemical stressors should be considered in risk evaluations because they contribute to population susceptibility and cumulative risk.[11] As discussed above, the best available science demonstrates that co-exposure to additional antiandrogenic chemicals increases the risks from exposure to the phthalates in EPA's Proposed Cumulative Chemical Group—evidence that EPA lacks discretion to ignore.[12]

Finally, while we agree with EPA's conclusion that DIDP is not antiandrogenic, co-exposures to DIDP and other toxicologically related phthalates may contribute to cumulative risk. As described in the 2014 report of the U.S. Consumer Product Safety Commission's Chronic Hazard Advisory Panel on Phthalates and Phthalate Alternatives ("CHAP Report"), DIDP is associated with developmental toxicity evidenced by skeletal abnormalities as well as reduced pup survival and weight gain following birth.[13] Additional phthalates also are associated with developmental toxicity—including substances that, like DIDP, are approved for food-contact use and therefore contribute to dietary co-exposures. If EPA elects not to include DIDP in the Proposed Cumulative Chemical Group due to the focus on phthalate syndrome, EPA still must address DIDP's potential to contribute to cumulative risk, either in the phthalates cumulative risk assessment or in its individual risk evaluation for DIDP.

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[9] Anne-Marie Saillenfait et al., *Adverse Effects of Diisooctyl Phthalate on the Male Rat Reproductive Development Following Prenatal Exposure*, 42 Reprod. Toxicology 192 (2013).
[10] 21 C.F.R. § 181.27.
[11] *See* EPA, *Draft Proposed Principles of Cumulative Risk Assessment Under the Toxic Substances Control Act* 8 (2023).
[12] *See* 15 U.S.C. § 2625(h) (requiring EPA to conduct risk evaluations "in a manner consistent with the best available science"); H.R. Rep. No. 94-1679, at 61 (1976) (Conf. Rep.) ("Oftentimes an unreasonable risk will be presented because of the interrelationship or cumulative impact of a number of different substances or mixtures. The conferees intend that the Administrator have authority to protect health and the environment in such situations.").
[13] U.S. Consumer Prod. Safety Comm'n, *Report by the Chronic Hazard Advisory Panel on Phthalates and Phthalate Alternatives* 102–03 (2014).

## II. EPA Must Implement its Proposal to Consider Cumulative Phthalates Exposures from TSCA Conditions of Use, "Non-Attributable" Sources, and "Non-TSCA" Sources

We strongly support EPA's proposal "to combine non-attributable and non-TSCA exposures with exposures from TSCA [conditions of use ("COUs")] when appropriate to determine cumulative exposure."[14] As EPA acknowledges, "certain non-attributable (*e.g.* dust) and non-TSCA (*e.g.* dietary) pathways are anticipated to be major contributors to phthalate exposure that contribute to cumulative risk."[15] Accordingly, a cumulative risk assessment that fails to consider these exposures fully would be fundamentally flawed and produce "an underestimation of risk."[16] Such an assessment would violate TSCA's mandate for EPA to consider all "reasonably available" information relevant to risk evaluations and conduct risk evaluations "in a manner consistent with the best available science."[17] EPA therefore must implement its proposal to aggregate TSCA COU, non-attributable, and non-TSCA exposures for the substances in its Proposed Cumulative Chemical Group and, as discussed below, consider fully all relevant exposure pathways and sources.

Indeed, the best available science demands that EPA consider, and aggregate, non-attributable and non-TSCA exposures with exposures from TSCA COUs in all of its TSCA risk evaluations, unless the data indicate that there are no such exposures for the chemical and populations at issue.[18] The imperative to do so as part of the phthalates cumulative risk assessment is particularly urgent because it is well established that dietary sources—which EPA does not regulate directly under TSCA—are the primary exposure pathway to most phthalates for most people—including for infants and children and with regard to the antiandrogenic phthalates that are the focus of EPA's proposed cumulative risk assessment.[19] Accordingly, ignoring

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[14] Phthalates CRA Proposal 115.
[15] *Id.* at 150.
[16] *Id.* at 114.
[17] 15 U.S.C. § 2625(h), (k).
[18] *See id.*; *see also* Nat'l Rsch. Council, *Science and Decisions: Advancing Risk Assessment* 130, 132 (2009) ("NRC 2009") (emphasizing the "[n]eed for [e]valuation of [b]ackground exposures" in risk assessment because even low-dose exposures to a chemical "may have a relevant biologic effect" when combined with elevated background levels).
[19] *See* CHAP Report 3, 52–53, 59 (concluding that "food, beverages and drugs via direct ingestion … constituted the highest [source of] phthalate exposures to all subpopulations"); Agency for Toxic Substances and Disease Registry, *Toxicological Profile for Di(2-ethylhexyl)phthalate (DEHP)* 2 (2022) (affirming that "[t]he principal route of human exposure to DEHP is oral," approximately half of oral exposure to DEHP among infants and toddlers comes from food, and "ingestion of food (including food from containers that leach DEHP) accounts for 95% of total oral exposure" among children and adults); Expert Decl. of Ami R. Zota, Sc.D., M.S., ¶¶ 3, 16, *In re Env't Def. Fund*, No. 21-1255 (D.C. Cir. filed Dec. 7, 2021) ("Zota Declaration"); Expert Decl. of Russ B. Hauser, M.D., Sc.D., M.P.H., ¶ 17, *In Re Env't Def. Fund*, No. 21-1255 (D.C. Cir. filed Dec. 7, 2021) ("Hauser Declaration").

phthalate exposures from the diet and other so-called "non-TSCA" and "non-attributable" sources would grossly distort EPA's risk assessment.

Further, as part of its aggregation of TSCA COU, non-TSCA, and non-attributable exposures, EPA must consider drinking water exposures to the relevant phthalates. EPA's proposal does not clearly commit the agency to this analysis, stating that "cumulative risk from drinking water attributable to TSCA releases may be included as appropriate," or that drinking water exposures "may . . . be included as a non-attributable source."[20] But considering drinking water exposures is mandatory, as readily available information demonstrates that multiple populations, including consumers and fenceline community residents, are exposed to phthalates in drinking water.[21] Accordingly, EPA must consider all reasonably foreseeable drinking water exposures to the relevant phthalates, whether or not it identifies a TSCA COU as the source of the drinking water contamination.

## III. EPA Must Consider Additional Higher-Risk Subpopulations in its Phthalates Cumulative Risk Assessment

As EPA's proposal acknowledges, TSCA mandates the evaluation and elimination of chemical risks not only to the general population, but also to any relevant "potentially exposed or susceptible subpopulation,"[22] which is "a group of individuals within the general population identified by [EPA] who, due to either greater susceptibility or greater exposure, may be at greater risk than the general population of adverse health effects from exposure to a chemical substance or mixture."[23] EPA correctly identifies pregnant women, women of reproductive age, male infants, male toddlers, and male children as relevant higher-risk subgroups for the phthalates cumulative risk assessment due to their increased susceptibility to phthalate syndrome on the basis of life stage.[24] At the same time, EPA must evaluate the risks to additional subpopulations who experience greater exposure to phthalates than the general population and/or are more susceptible to harm from that exposure.

For example, people of color and economically insecure people experience elevated phthalate exposures compared to the general population.[25] In particular, it is well documented in the literature that Black and Latina women of reproductive age experience both heightened

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[20] Phthalates CRA Proposal 147.
[21] *See*, *e.g.*, 2018 Consumer Confidence Rep. for Public Water Sys. City of Deer Park at 4, https://www.deerparktx.gov/DocumentCenter/View/7677/2018-Water-Report (last visited Apr. 18, 2023) (documenting DEHP in drinking water in Deer Park, Texas); 2021 Consumer Confidence Report for Public Water Sys. City of Deer Park, at 4, https://deerparktx.gov/DocumentCenter/View/10605/2022-CCR-for-Water-Year-2021 (last visited Apr. 18, 2023) (same); Houston Public Works, *Water Quality Report 2021,* at 5 (documenting DEHP in Houston drinking water).
[22] 15 U.S.C. § 2605(b)(4)(A), (b)(4)(F)(i).
[23] *Id.* § 2602(12).
[24] Phthalates CRA Proposal 108.
[25] Zota Declaration ¶¶ 23–24; *see also* Mike Belliveau, Ctr. for Food Safety, *Capped with Toxics* 9 (2021) (Toxic-Free Food Campaign 2021).

exposure to phthalates[26] and greater susceptibility to associated health harms than the general population.[27] In addition, infants and children, regardless of sex, experience disproportionate phthalate exposures compared to other age groups[28] and are more susceptible to associated health harms.[29] Despite this evidence, EPA's proposal states only that additional higher-risk subpopulations "*may* . . . be identified throughout the risk evaluation process and incorporated into a CRA as appropriate," and that the "*individual* phthalates risk evaluations will consider all relevant lifestages, populations, and [potentially exposed or susceptible subpopulations]."[30] That vague promise is inadequate. Given the wealth of evidence documenting inequitable exposure and heightened susceptibility to health harm from phthalates among people of color—including, but not limited to, Black and Latina women; economically insecure people; and infants and children, as well as evidence that "exposure to multiple phthalates will, at a minimum, have additive health effects, if not synergistic health effects, that can magnify the health harms associated with individual phthalates,"[31] EPA must evaluate the risks to these additional higher-risk groups in its cumulative risk assessment.

## IV. EPA Must Aggregate All Relevant Phthalate Co-Exposures Affecting Workers and Fenceline Communities

EPA correctly proposes to aggregate phthalate exposures within occupational and fenceline community populations that are associated with TSCA COUs, non-TSCA uses, and non-attributable sources. This aggregation of multiple exposures to one or more phthalates is essential to account for the real-world exposures within these populations and, as discussed above, to conduct a scientifically valid and lawful cumulative risk assessment.[32] While we generally support EPA's proposed approach in this regard, we note below additional exposure sources omitted from the proposal that EPA must incorporate into its cumulative risk assessment for phthalates.

Regarding workers, EPA acknowledges that, in addition to exposure to one or more phthalates in the workplace, "[w]orkers may have exposures to multiple phthalates through

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[26] *See* Zota Declaration ¶¶ 6, 23–24 and papers cited therein; Kelly K. Ferguson et al., *Urinary Phthalate Metabolites and Biomarkers of Oxidative Stress in Pregnant Women: A Repeated Measures Analysis*, 123 Env't Health Persps. 210 (2015), https://ehp.niehs.nih.gov/doi/pdf/10.1289/ehp.1307996.

[27] Zota Declaration ¶¶ 26–28 and papers cited therein; Michael S. Bloom et al., *Racial Disparity in Maternal Phthalates Exposure; Association with Racial Disparity in Fetal Growth and Birth Outcomes*, 127 Env't Int'l 473 (2019), https://www.sciencedirect.com/science/article/pii/S0160412018329908.

[28] Zota Declaration ¶ 25 and papers cited therein; Hauser Declaration ¶ 26 and papers cited therein.

[29] Zota Declaration ¶ 29 and papers cited therein; Hauser Declaration ¶¶ 24–25, 27, 30 and papers cited therein.

[30] Phthalates CRA Proposal 108 (emphases added).

[31] Hauser Declaration ¶ 35.

[32] 15 U.S.C. § 2625(h), (k); 40 C.F.R. § 702.41(a)(4), (e)(3).

sources occurring outside the workday" from non-attributable and non-TSCA sources.[33] While this is correct, EPA also must consider workers' potential exposure to phthalates outside the workplace from TSCA COUs, including consumer uses. EPA's proposal to estimate cumulative exposures to occupational populations by adding non-attributable exposure, non-TSCA exposure, and cumulative occupational exposure is therefore incomplete.[34] In its draft cumulative risk assessment, EPA must also aggregate relevant TSCA COU exposures that workers may experience outside the workplace.

Regarding fenceline community residents, we likewise support EPA's proposal to consider "exposures [to] multiple phthalates from TSCA, non-attributable, and non-TSCA sources of exposures," as well as "cumulative exposure and risk from single or multiple facility releases."[35] For the final category, EPA's proposal correctly recognizes multiple TSCA COU scenarios that can generate co-exposures to multiple phthalates, *i.e.*, when (1) "a single facility releases more than one phthalate to the ambient air or receiving waterbodies"; (2) "multiple TSCA facilities in close proximity release more than one phthalate to ambient air or receiving waterbodies"; or (3) "a fenceline community is near one or more facilities releasing phthalates [and] is also being exposed through consumer or occupational COUs."[36] In addition, reflecting the reality that fenceline community residents often experience occupational and/or consumer exposures as well, EPA correctly proposes to combine calculated risks to fenceline communities with its estimates of cumulative occupational and consumer exposures. This approach is essential to satisfy TSCA's best-available-science mandate and ensure that EPA's cumulative risk assessment reflects fenceline communities' real-world exposures to phthalates and other chemicals that pose cumulative risks. Accordingly, EPA should eliminate from its proposal language suggesting that this approach is discretionary.[37]

To ensure that its assessment captures all relevant exposures, EPA should conduct a cumulative sentinel exposure assessment to establish the "plausible upper bound of exposure" to the Proposed Cumulative Chemical Group in the human population.[38] TSCA contemplates that EPA will consider sentinel exposures when conducting risk evaluations,[39] and EPA should do so in the phthalates cumulative risk assessment to establish an "upper bound" of cumulative exposures to the Proposed Cumulative Chemical Group. To achieve this, for each chemical within the Proposed Cumulative Chemical Group, EPA should first aggregate high-end exposure

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[33] Phthalates CRA Proposal 141.
[34] *See id.* at 142.
[35] *Id.*
[36] *Id.* at 144.
[37] *See id.* at 148 (stating that "reasonable combinations of exposures" for fenceline community residents who also experience consumer and/or occupational exposure "may be considered, as data allows").
[38] 40 C.F.R. § 702.33; *see* 15 U.S.C. § 2625(c)(1) ("Any action authorized or required to be taken by the Administrator under any provision of this chapter with respect to a chemical substance or mixture may be taken by the Administrator in accordance with that provision with respect to a category of chemical substances or mixtures.").
[39] 15 U.S.C. § 2605(b)(4)(F)(2).

estimates for all reasonably foreseeable exposure scenarios, including, but not limited to, exposures from the workplace, use of consumer products, environmental exposures, fenceline community exposures, and all non-TSCA and non-attributable exposure sources, including dietary exposures and exposures from the use of personal care products. After scaling each phthalate to the potency of the index chemical (assuming EPA characterizes cumulative risk using the relative potency factor approach), EPA should then aggregate each individual chemical's adjusted "upper bound" aggregate exposure estimate to derive a cumulative sentinel risk estimate, which would represent a high-end exposure estimate for the entire Proposed Cumulative Chemical Group. From this value, central-tendency and low-end cumulative exposure estimates can be statistically derived. Low-end cumulative exposure estimates could also be derived using the reverse dosimetry approach for phthalates with available National Health and Nutrition Examination Survey ("NHANES") biomonitoring data to estimate low-end cumulative internal phthalate dose in the general population. EPA can rely on the cumulative sentinel risk estimate to characterize risk for communities that experience high levels of exposures to the Proposed Cumulative Chemical Group from multiple exposure scenarios, especially when a detailed quantitative exposure assessment cannot be completed. In addition, when characterizing risk for the cumulative sentinel exposure scenario, EPA should account for any relevant interacting non-chemical stressors through the use of uncertainty factors, as described below.

Finally, as discussed *infra* Point V, we note that additional data sources beyond those listed in EPA's proposal are available to inform this assessment. And where EPA confronts legitimate data limitations, it must, as the proposal notes, incorporate appropriate modeling and/or "assumptions . . . to determine reasonable combinations of exposure for identified populations."[40] This may include the use of uncertainty factors to account for exposures to relevant phthalates that are not listed on the Toxics Release Inventory ("TRI") or reflected in other databases.[41]

## V. EPA Must Implement—and Expand Upon—its Proposal for Integrating All Reasonably Available Data to Develop Cumulative Exposure Estimates

TSCA requires EPA to consider and integrate all reasonably available exposure information in its risk evaluations.[42] To satisfy this mandate, EPA must implement and expand upon its proposal for collecting and integrating reasonably available exposure information in the phthalates cumulative risk assessment.

EPA correctly proposes to consider multiple sets of relevant EPA programmatic data—including data from the Chemical Data Reporting rule database, the Toxics Release Inventory,

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[40] Phthalates CRA Proposal 148.

[41] In addition, EPA should move expeditiously to finalize the proposed addition of DINP to the TRI and to add BBP, DIBP, and DCHP to the TRI, as all of these chemicals are "known to cause or can reasonably be anticipated to cause in humans" cancer or other chronic health effects that satisfy the statutory criteria for TRI listing. 42 U.S.C. § 11023(d)(2)(B).

[42] 15 U.S.C. § 2625(k); 40 C.F.R. § 702.41(b)(1).

Discharge Monitoring Reports, and the National Emissions Inventory—as well as occupational exposure information from fellow agencies, to calculate cumulative phthalates exposures.[43] Data from all of these sources are reasonably—indeed, readily—available to EPA, and EPA correctly recognizes that considering only a subset of these sources could yield substantial underestimates of both chemical release volumes and the number of chemical release events.[44]

At the same time, as EPA also acknowledges, even considering all of the listed data sources for the six phthalates EPA proposes to consider in its cumulative risk assessment will "leave[] large data gaps in assessing environmental releases and occupational exposures for certain phthalates and certain COUs that will require alternative methods and data sources to fill."[45] Accordingly, EPA must incorporate reasonably available information from additional sources to satisfy TSCA and develop a scientifically sound cumulative risk assessment.

For example, EPA should consider data collected by its Office of Enforcement and Compliance Assurance, as well as regional offices, in the course of compliance investigations and enforcement actions; data collected or generated by EPA's Office of Research and Development, Office of Air and Radiation, Office of Water, and Office of Land & Emergency Management; off-site consequences analyses in Risk Management Plans required under the Clean Air Act; chemical inventories developed pursuant to the Emergency Planning and Community Right-to-Know Act; monitoring and chemical release data from state, tribal, and local authorities; academic data and reports; and data generated and/or collected by local communities. These data are reasonably available and necessary to accurately characterize both average exposures and peak exposures associated with various excess emission events at industrial facilities, such as accidents, spills, malfunctions, start-up and shutdown periods, and extreme weather events. Because such excess emission events are "known" and "reasonably foreseen" consequences of manufacturing, processing, distributing, using, and disposing of phthalates, TSCA requires EPA to consider them in assessing exposure and risk associated with the chemicals' conditions of use.[46]

Finally, when utilizing chemical release information from the TRI and other databases, EPA must consider multiple years of data to generate valid release and exposure estimates given the substantial annual variation in reported release volumes.[47] Specifically, EPA should review

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[43] *See* Phthalates CRA Proposal 136–41, 143–44.

[44] *See* Earthjustice and Louisiana Env't Action Network, *Considerations for Fenceline Community Exposure Assessment* 10–18 (2022) (documenting substantial variation in release volumes and number of release incidents reported to the TRI, NEI, and DMR databases for TSCA high-priority chemicals).

[45] Phthalates CRA Proposal 137; *see also id.* at 138 (acknowledging that "[t]here may be significant challenges with using EPA programmatic data to identify sites with cumulative release and exposure potential"); *id.* at 144 ("In many instances, the [EPA] program data will not cover all potential releases for a given site due to the limited coverage of the selected phthalates in these programs . . . .").

[46] 15 U.S.C. § 2602(4).

[47] *See* Earthjustice & Louisiana Env't Action Network 2022 at 12–15.

at least five years of release data and consider the highest annual release volume for each relevant chemical to estimate a facility's "known" and "reasonably foreseen" phthalates releases.

## VI. EPA Should Implement its Proposed Methodology for Conducting a Cumulative Risk Assessment for the Proposed Cumulative Chemical Group

We generally support EPA's proposed methodologies for completing the cumulative risk assessment for the Proposed Cumulative Chemical Group. EPA appropriately proposes to evaluate the cumulative risk of phthalate syndrome, rely on a dose-addition and relative potency factor model to characterize cumulative risk, and estimate cumulative human phthalate exposures using a scenario-based approach. These methods are largely supported by an abundance of available toxicological and exposure data for the Proposed Cumulative Chemical Group.

At the same time, we wish to emphasize that EPA does not need such an expansive body of toxicological and exposure information to conduct a cumulative risk assessment for other chemicals or chemical categories. While EPA proposes to consider seven key adverse health outcomes that are associated with phthalate syndrome for its phthalates cumulative risk assessment, the best available science indicates that only a single common adverse health endpoint is required to conduct a cumulative risk assessment,[48] and EPA should not limit the grouping of chemicals or chemical categories for subsequent cumulative risk assessments if data for more than one common toxicological endpoint is not available. Similarly, EPA's choice to use the relative potency factor approach for hazard characterization is appropriate to evaluate the cumulative risk of the Proposed Cumulative Chemical Group, but the amount of concordant dose-response data required for this approach could limit the grouping of chemicals or chemical categories in subsequent cumulative risk assessments. In such circumstances, EPA should consider relying on a modified relative potency factor or hazard index approach to evaluate the cumulative risk of chemicals or chemical categories for which concordant dose-response data is not available.

## VII. EPA Should Fully Account for Human Variability and Vulnerability when Evaluating the Potential Cumulative Risks of Developmental Harm from Co-Exposure to Multiple Phthalates

When conducting risk assessments under TSCA, EPA is required to rely on the "best available science"[49] and specifically evaluate risks to "potentially exposed or susceptible subpopulations" such as infants and children.[50] Further, decades of scientific evidence suggests that EPA should improve its methodologies to account for enhanced early-life susceptibility to chemical exposures.[51]

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[48] NRC 2008 at 11–12.
[49] 15 U.S.C. § 2625(h).
[50] *Id*. § 2605(b)(4); *id.* § 2602(12).
[51] *See* Julia R. Varshavsky et al., *Current Practice and Recommendations for Advancing How Human Variability and Susceptibility Are Considered in Chemical Risk Assessment*, 21(Suppl 1) Env't Health Art. No. 133 (2023), https://doi.org/10.1186/s12940-022-00940-1.

While we support EPA's proposal to focus its cumulative risk assessment on phthalate syndrome, EPA must also fully account for the risks of developmental neurotoxicity from co-exposure to multiple toxicologically related phthalates. A growing body of scientific evidence shows that exposure to phthalates *in utero* and during childhood is linked to a higher risk of serious and irreversible harm to brain development. Early-life exposures to DEHP, DBP, DIBP, DINP, and/or BBP have been linked to reduced social responsiveness, social problems, lower vocabulary scores, poorer working memory, lower IQ, and behavioral disorders like attention-deficit hyperactivity disorder (ADHD) in children.[52] Based on the significance of these harms, several leading experts in toxicology, exposure science, and epidemiology concluded in a recent article published in the *American Journal of Public Health* that urgent reforms are needed to "substantially reduce exposures to ortho-phthalates over critical periods of child brain development."[53]

Although the evidence of the neurodevelopmental toxicity potential of phthalates is developing, data gaps for certain phthalates and a lack of concordance in observed outcomes between human and animal studies collectively contribute to uncertainty in the existing data. EPA appropriately acknowledges this uncertainty, concluding that developmental neurotoxicity data is "limited across the high-priority and manufacturer-requested phthalates."[54] However, EPA did not propose methods to account for this uncertainty in the cumulative risk assessment for phthalates, which is critical due to the potential for phthalates to induce neurodevelopmental harm and the enhanced susceptibility to harm from phthalate exposures during early life.

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[52] Stephanie M. Engel et al. *Neurotoxicity of Ortho-phthalates: Recommendations for Critical Policy Reforms to Protect Brain Development in Children*, 111 Am. J. of Pub. Health 687 (2021), https://doi.org/10.2105/AJPH.2020.306014; Elizabeth M. Kamai et al., *Gestational Phthalate Exposure and Preschool Attention Deficit Hyperactivity Disorder in Norway*, 5 Env't Epidemiology, art. no. e161 (2021); Michiel A. van den Dries et al., *Phthalate and Bisphenol Exposure During Pregnancy and Offspring Nonverbal IQ*, 128 Env't Health Persps., art. no. 77009 (2020); Stephanie M. Engel et al., *Prenatal Phthalates, Maternal Thyroid Function, and Risk of Attention-Deficit Hyperactivity Disorder in the Norwegian Mother and Child Cohort*, 126 Env't Health Persps., art. no. 057004 (2018); Drew B. Day et al., *Phthalate Mixtures in Pregnancy, Autistic Traits, and Adverse Childhood Behavioral Outcomes*, 147 Env't Int'l, art. no. 106330 (2021); Sharon Daniel et al., *Prenatal and Early Childhood Exposure to Phthalates and Childhood Behavior at Age 7 Years*, 143 Env't Int'l, art. no. 105894 (2020); Trine Staak Olesen et al., *Prenatal Phthalate Exposure and Language Development in Toddlers from the Odense Child Cohort*, 65 Neurotoxicology & Teratology 34 (2018); Nan Li et al., *Identifying Periods of Susceptibility to the Impact of Phthalates on Children's Cognitive Abilities*, 172 Env't Rsch. 604 (2019); Pam Factor-Litvak et al., *Persistent Associations between Maternal Prenatal Exposure to Phthalates on Child IQ at Age 7 Years*, 9 PloS ONE, art. no. e114003 (2014), https://doi.org/10.1371/journal.pone.0114003.

[53] Engel et al. 2021.

[54] Phthalates CRA Proposal 27.

### A. EPA should expand its default intraspecies variability uncertainty factor to better account for human variability in response to phthalate exposures.

When evaluating the risk posed by chemicals, EPA currently relies on a 10X default adjustment factor to account for intraspecies variability, which EPA often adjusts downward for individual chemicals (as it did in its trichloroethylene risk evaluation) but never upward.[55] This method is based on a scientific recommendation made nearly 70 years ago.[56] Since then, decades of scientific evidence has amassed indicating that this adjustment factor does not capture the full range of human responses to chemical exposures, especially for susceptible subgroups like children, infants, and the developing fetus.[57] Based on observed toxicokinetic differences in chemical metabolism between younger age groups and adults, California EPA's Office of Environmental Health Hazard Assessment ("OEHHA") now relies on a 30X intraspecies adjustment factor.[58] The World Health Organization's International Programme on Chemical Safety ("IPCS") examined human variability in toxicokinetic and toxicodynamic responses to chemical exposures using a probabilistic method, and found that variability at the 99th percentile across the general population was up to 4.2 times higher than what is reflected in EPA's default intraspecies adjustment factor.[59] Dozens of additional studies demonstrate that the variability in human response to chemical exposures far exceeds what EPA currently considers when evaluating human health risk.[60] Given the substantial body of evidence, from both academic and agency studies, recommending improved methodologies to fully account for human variability, we recommend that EPA expand its intraspecies variability factor to at least 42X to fully account for human variability in response to phthalate exposures.

### B. EPA should incorporate an additional uncertainty factor to account for enhanced early-life susceptibility to phthalate exposures.

In addition to increasing the default intraspecies adjustment factor to account for human variability, we urge EPA to incorporate an additional uncertainty factor when evaluating cumulative risk to account for the enhanced susceptibility to phthalate exposures in younger age groups, including children, infants, and the developing fetus. Although EPA is proposing to base the cumulative phthalates risk assessment on developmental toxicity endpoints, expanding the intraspecies uncertainty factor is necessary to fully account for the enhanced susceptibility of

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[55] NRC 2009 at 111.
[56] Varshavsky et al. 2023 at 6; *see also* AJ Lehman et al., *100-fold Margin of Safety*, 18 Ass'n Food & Drug Off. USQ Bull. 33–35 (1954), https://www.scienceopen.com/document?vid=0745a8c4-86f6-43a9-ba83-f78f92d6e8dd.
[57] Varshavsky et al. 2023, at 8 tbl.2.
[58] Cal. Env't Prot. Agency, *Technical Support Document for the Derivation of Noncancer Reference Exposure Levels* (2008), https://oehha.ca.gov/media/downloads/crnr/noncancertsdfinal.pdf.
[59] WHO IPCS, *Guidance Document on Evaluating and Expressing Uncertainty in Hazard Characterization* (2d. ed. 2017), http://www.inchem.org/documents/harmproj/harmproj/harmproj11.pdf.
[60] Varshavsky et al. 2023, at 8 tbl.2.

younger age groups to harm from phthalate exposures. This is particularly necessary because the IPCS-informed (up to 42X) adjustment factor does not account for early-life susceptibility, necessitating additional adjustment to capture the full range of variability in responses to chemical exposures in younger age groups.

Indeed, EPA and Congress have recognized the scientific imperative to apply an adjustment factor to account for early-life susceptibility in an analogous regulatory context. This approach is required by the Food Quality Protection Act ("FQPA") to account for additional susceptibility to harm from dietary pesticide exposures in pregnant women and children. This congressional mandate is supported by scientific evidence "demonstrating that the current default approach [used by EPA] does not protect the human population across life stages of development," especially when considering differences in metabolism in younger age groups compared to adults.[61] Moreover, EPA itself has said that "sound science" requires it to apply the FQPA risk assessment methodologies, including an additional 10X uncertainty factor, to account for the unique risks posed to infants and children from chemical exposure, outside the FQPA context.[62] Consistent with the best available science, EPA should add an additional age-specific uncertainty factor of at least 10X to account for enhanced early-life susceptibility to phthalate exposures when conducting the phthalates cumulative risk assessment under TSCA.

### C. EPA should adopt an additional uncertainty factor to address database uncertainty concerning neurodevelopmental harm from phthalates.

In addition to increasing the default intraspecies adjustment factor to more accurately account for human variability and incorporating an additional 10X uncertainty factor to capture enhanced early-life susceptibility, we urge EPA to incorporate an additional uncertainty factor when evaluating cumulative risk to account for uncertainty concerning the neurodevelopmental toxicity potential of phthalates. Adoption of a database uncertainty factor is merited because of a lack of consistent dose-response relationships between phthalate exposures and neurodevelopmental outcomes, and the potential for neurodevelopmental harm to occur at doses near or below those that are anti-androgenic.

In its proposal, EPA acknowledged the "limited" body of evidence on the neurodevelopmental toxicity of phthalates.[63] In addition, many of the existing associations between prenatal phthalate exposures and neurodevelopmental outcomes are derived from epidemiological studies,[64] increasing the potential for low-dose effects at levels below those observed in animal studies. OEHHA recommends incorporating an additional uncertainty factor of at least 3X to account for certain database deficiencies, particularly for "chemicals with

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[61] *Id.* at 13.
[62] EPA, *Revised Risk Assessment Methods for Workers, Children of Workers in Agricultural Fields, and Pesticides with No Food Uses* 2 (2009); *see also* EPA, Policy Paper on Revised Risk Assessment Method for Workers, Children of Workers in Agricultural Fields, and Pesticides with No Food Uses; Notice of Availability, 74 Fed. Reg. 65,121 (Dec. 9, 2009).
[63] Phthalates CRA Proposal 27.
[64] Engel et al. 2021.

substantial toxicological data gaps, including, but not limited to, developmental toxicity."[65] EPA similarly recommends incorporating an uncertainty factor of up to ten "to account for the potential for deriving an underprotective RfD/RfC as a result of an incomplete characterization of the chemical's toxicity. In addition to identifying toxicity information that is lacking, review of existing data may also suggest that a lower reference value might result if additional data were available."[66] Consistent with recommendations by EPA and OEHHA, here EPA should add an additional uncertainty factor of at least 3X to account for the significant data gaps in the body of evidence on the neurodevelopmental toxicity of phthalates.

## VIII. EPA's Cumulative Risk Assessment Must Address the Effects of Non-Chemical Stressors on Susceptibility to Harm from Substances in the Proposed Cumulative Chemical Group

EPA is specifically directed under TSCA to evaluate risks to "potentially exposed or susceptible subpopulation[s]" "who, due to either greater susceptibility or greater exposure, may be at greater risk than the general population of adverse health effects from exposure to a chemical substance."[67] If EPA finds that a chemical presents unreasonable risk to any potentially exposed or susceptible subpopulation, EPA must regulate the chemical "to the extent necessary so that the chemical substance or mixture no longer presents such risk."[68]

To accurately identify susceptible populations at greater risk from harm, and then identify and eliminate unreasonable risk to such populations, EPA must use the best available science to evaluate the factors that contribute to greater susceptibility and vulnerability.[69] Studies have shown that both intrinsic factors (such as life stage or underlying disease) and extrinsic factors (such as psychosocial stress from economic insecurity, violence, or racial injustice) contribute to susceptibility to harm from chemical exposures.[70] It is well established in the scientific literature that these nonchemical stressors can increase susceptibility to harm from chemical exposures and should be taken into consideration when identifying, and protecting, potentially exposed or

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[65] Cal. Env't Prot. Agency, Off. of Env't Health Hazard Assessment, *Technical Support Document for the Derivation of Noncancer Reference Exposure Levels* at xiii (2008), https://oehha.ca.gov/media/downloads/crnr/noncancertsdfinal.pdf.

[66] EPA Risk Assessment Forum, *A Review of the Reference Dose Concentration Processes* 4–44 (2002), https://www.epa.gov/sites/default/files/2014-12/documents/rfd-final.pdf.

[67] 15 U.S.C. §§ 2605(b)(4)(A), 2602(12).

[68] *Id.* § 2605(a).

[69] *Id*. § 2625(h).

[70] Patricia D. Koman et al., *Population Susceptibility: A Vital Consideration in Chemical Risk Evaluation Under the Lautenberg Toxic Substances Control Act*, 17 PLoS Biology 4 (2019), https://journals.plos.org/plosbiology/article?id=10.1371/journal.pbio.3000372; Cliona M. McHale et al., *Assessing Health Risks from Multiple Environmental Stressors: Moving from G×E to I×E*, 775 Mutational Rsch. 11 (2018), https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5863617/; NRC 2009 at 110, 111, and 213.

susceptible subpopulations.[71] For example, a study conducted by researchers at the Harvard School of Public Health discovered that exposure to high levels of traffic-related air pollution during childhood, an intrinsically susceptible life stage, was associated with an elevated risk of developing asthma only if the exposed children also experienced chronic psychosocial stress in the form of violence.[72]

In general, people of color face disproportionately high levels of non-chemical stressors, like low socioeconomic status and healthcare inequities, which often translate to a greater proportion and severity of negative health outcomes.[73] In the context of phthalate exposures,

> [c]ertain racial and ethnic groups are more exposed and susceptible to harm from phthalate exposures because of the prevalence of associated non-chemical stressors.[74] For example, Black and Latina women of reproductive age experience disproportionately high exposures to certain phthalates,[75] and are more likely to suffer from health harms associated with these exposures[76] likely due to non-chemical stressors commonly

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[71] NRC 2009 at 110, 111; Bruce S. McEwen & Pamela Tucker, *Critical Biological Pathways for Chronic Psychosocial Stress and Research Opportunities to Advance the Consideration of Stress in Chemical Risk Assessment*, 101 Am. J. Pub. Health S131 (2011), https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3222511/; Devon C. Payne-Sturges et al., *Methods for Evaluating the Combined Effects of Chemical and Nonchemical Exposures for Cumulative Environmental Health Risk Assessment*, 15 Intl. J. Envtl. Rsch. & Pub. Health 2797 (2018), https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6313653/.

[72] Jane E. Clougherty, *Synergistic Effects of Traffic-Related Air Pollution and Exposure to Violence on Urban Asthma Etiology*, 115 Envtl. Health Persp. 1140 (2007), https://pubmed.ncbi.nlm.nih.gov/17687439/.

[73] Gilbert C. Gee & Devon Payne-Sturges, *Environmental Health Disparities: A Framework Integrating Psychosocial and Environmental Concepts*, 112 Envtl. Health Perspectives 1645–53 (Dec. 2004), https://pubmed.ncbi.nlm.nih.gov/15579407/.

[74] *See* McHale et al. 2018; Rachel Morello-Frosch et al., *Cumulative Impacts of Inequalities in Environmental Health: Implications for Policy*, 30 Health Affairs 879 (2011), https://doi.org/10.1377/hlthaff.2011.0153.

[75] Tamarra M. James-Todd et al., *Racial/ethnic Disparities in Environmental Endocrine Disrupting Chemicals and Women's Reproductive Health Outcomes: Epidemiological Examples Across the Life Course*, 3 Current Epidemiology Reports 161 (2016), https://doi.org/10.1007/s40471-016-0073-9.

[76] Ami R. Zota et al., *Phthalates Exposure and Uterine Fibroid Burden Among Women Undergoing Surgical Treatment for Fibroids: A Preliminary Study*, 111 Fertility and Sterility 112 (2019), https://doi.org/10.1016/j.fertnstert.2018.09.009; Zota Declaration ¶¶ 6, 23, 24, 27, 28.

experienced by these groups like food insecurity and psychosocial stress from racial injustice.[77]

In a study examining pooled biomonitoring data obtained by NHANES over the course of 12 years in women of reproductive age, cumulative exposures to certain antiandrogenic phthalates, including DEHP, DIBP, DBP, and BBP, were 12% higher in Black women than white women, suggesting a higher potential risk of adverse antiandrogenic health outcomes in Black women from phthalate exposure.[78] Epidemiological studies have confirmed that non-chemical stressors can also enhance the severity of developmental harm from phthalate exposures. For example, in a study examining mother-child pairs, anogenital distance was altered in females born to mothers who were exposed to antiandrogenic phthalate mixtures and also experienced stressful life events during pregnancy.[79]

TSCA requires that EPA account for the effects of non-chemical stressors on human health risk. And yet, in its proposal for assessing the cumulative risk of phthalates within the Proposed Cumulative Chemical Group, EPA failed to acknowledge the need to examine the potential interactions between chemical and non-chemical stressors or any methods that could account for these interactions. For more than a decade, EPA scientists have recognized the need to incorporate and quantify the effects of non-chemical stressors in cumulative risk assessment.[80] EPA can rely on existing methods to examine the contributions of non-chemical stressors to cumulative risk. For example, EPA can use various science-based tools with sociodemographic

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[77] Ami R. Zota & Bhavna Shamasunder, *The Environmental Injustice of Beauty: Framing Chemical Exposures from Beauty Products as a Health Disparities Concern*, 217 Am. J. of Obstetrics & Gynecology 418.E1 (2017), https://doi.org/10.1016/j.ajog.2017.07.020; Ami R. Zota et al., *Recent Fast Food Consumption and Bisphenol A and Phthalates Exposures Among the U.S. Population in NHANES, 2003-2010*, 124 Env't Health Persp. 1521 (2016), https://doi.org/10.1289/ehp.1510803; Zota Declaration¶ 6.

[78] Julia R. Varshavsky et al., *A Novel Method for Calculating Potency-Weighted Cumulative Phthalates Exposure with Implications for Identifying Racial/Ethnic Disparities Among U.S. Reproductive-Aged Women in NHANES 2001–2012*, 50 Envtl. Sci. & Tech. 10616 (2016), https://doi.org/10.1021/acs.est.6b00522.

[79] Tye E. Arbuckle et al., *Do Stressful Life Events During Pregnancy Modify Associations Between Phthalates and Anogenital Distance in Newborns?*, 177 Env't Res., art. no. 108593 (2019), DOI: 10.1016/j.envres.2019.108593; s*ee also* Devon Payne-Sturges et al., *Cumulative Risk Evaluation of Phthalates Under TSCA*, 57 Env't Sci. & Tech. 6403 (2023), https://doi.org/10.1021/acs.est.2c08364.

[80] Cynthia V. Rider et al., *Incorporating Nonchemical Stressors into Cumulative Risk Assessments*, 127 Tox. Scis. 10 (2012), https://doi.org/10.1093/toxsci/kfs088; NRC 2009 at 110, 111, and 213.

indicators, like the CalEnviroScreen,[81] the CDC Social Vulnerability Index ("SVI"),[82] NHANES,[83] and/or the U.S. Census Bureau American Community Survey[84] to identify subpopulations that experience high levels of non-chemical stressors. Data obtained from one or more of these sources can further inform cumulative risk assessment. For example, EPA can use a complex systems approach for cumulative risk assessment to account for community-specific non-chemical stressors identified through SVI.[85] A recent study outlining distinct recommendations for conducting a phthalates cumulative risk assessment further suggests considering non-chemical stressors as "effect modifiers of the dose-response relationship" "[i]f there is evidence of alterations in dose-response relationships (e.g., evidence of effect modification) for individual phthalates by co-exposures to other chemical or nonchemical stressors."[86]

In the case where sufficient data to quantitatively account for non-chemical stressors is not available, EPA can determine whether additional uncertainty factors should be applied during risk characterization. The best available scientific evidence supports the use of additional uncertainty factors to account for multiple non-chemical stressors when assessing risk to potentially exposed or susceptible subpopulations.[87] Several governmental and scientific authorities have supported the use of an additional uncertainty factor to account for potential interactions among chemicals found in mixtures.[88] Additional uncertainty factors should be considered to account for the potential interactions between chemical and non-chemical stressors. This is particularly relevant when assessing risk to residents of fenceline communities or other susceptible subgroups who experience disproportionately high levels of non-chemical stressors compared to the general population. Detailed scientific rationales supporting these recommendations can be found in the publication *Current Practice and Recommendations for*

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[81] *CalEnviroScreen,* Cal. OEHHA, https://oehha.ca.gov/calenviroscreen (last visited Apr. 19, 2023).
[82] Agency for Toxic Substances and Disease Registry, CDC/ATSDR Social Vulnerability Index, https://www.atsdr.cdc.gov/placeandhealth/svi/index.html (last visited Apr. 19, 2023).
[83] *National Health and Nutrition Examination Survey*, CDC https://www.cdc.gov/nchs/nhanes/index.htm (last updated Apr. 25, 2023).
[84] *American Community Survey (ACS)*, U.S. Census Bureau, https://www.census.gov/programs-surveys/acs (last visited Apr. 27, 2023).
[85] Devon C. Payne-Sturges et al., *Defining and Intervening on Cumulative Environmental Neurodevelopmental Risks: Introducing a Complex Systems Approach*, 129 Env't Health Persps. art. no. 35001 (2021), https://doi.org/10.1289/EHP7333.
[86] Payne-Sturges et al. 2023.
[87] Varshavsky et al. 2023.
[88] *See* Swedish Chems. Agency, *An Additional Assessment Factor (MAF) – A Suitable Approach for Improving the Regulatory Risk Assessment of Chemical Mixtures?* (2015), http://www.thomasbackhaus.eu/wp-content/uploads/2015-Backhaus-MAF-Rapport-5-15.pdf; Nat'l Rsch. Council, *Drinking Water and Health, Volume 9: Selected Issues in Risk Assessment* 99, 127–29 (1989), https://nap.nationalacademies.org/catalog/773/drinking-water-and-health-volume-9-selected-issues-in-risk.

*Advancing How Human Variability and Susceptibility Are Considered in Chemical Risk Assessment*.[89] This paper specifically recommends "development of a separate default extrinsic variability factor . . . that would account for exposure to multiple chemical and non-chemical stressors."[90]

## IX. EPA Must Evaluate Ecological Risks from Cumulative Phthalate Exposures

Prior to issuing risk determinations for the high-priority and manufacturer-requested phthalates, EPA must evaluate the risks to wildlife from cumulative phthalate exposures. TSCA compels this; the statute imposes on EPA a coequal obligation to "conduct risk evaluations . . . to determine whether a chemical substance presents an unreasonable risk of injury to health *or the environment.*"[91] And as discussed above, TSCA's risk evaluation mandate requires consideration of cumulative risks.

Existing data and literature demonstrate both the need for ecological cumulative risk assessment of phthalates and the availability of information to support it. Published literature documents significant co-releases of multiple phthalates and related chemicals into the environment.[92] Indeed, much of the cumulative exposure data EPA proposes to rely on for its human health cumulative risk assessment demonstrates that wildlife, too, experience co-exposures to multiple phthalates and other chemicals with related toxicological effects.

Further, EPA has models for ecological cumulative risk assessments conducted by other authoritative bodies for phthalates and other substances. For example, Health Canada published a detailed proposal for an ecological cumulative risk assessment of phthalates in 2015, which outlined a tiered approach similar to EPA's proposed human health cumulative risk assessment and describes methodological options for assessing cumulative ecological risk using concentration addition.[93] Health Canada noted that, while there were no ecological cumulative risk assessments for phthalates in the literature as of 2015, "there are many examples where the cumulative ecological risks posed by other groups of substances have been assessed by the Government of Canada or by other jurisdictions internationally."[94] Further, Health Canada explained that evidence that related chemicals co-occur in environmental media—which EPA summarizes in its proposal—is the most important indicator for ecological cumulative risk assessment and emphasized that such an assessment "is generally considered to provide a much

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[89] Varshavsky et al. 2023.
[90] *Id.* at 13.
[91] 15 U.S.C. § 2605(b)(4)(A) (emphasis added).
[92] *See*, *e.g.*, Tania Montoto-Martinez, et al. *Microplastics, Bisphenols, Phthalates and Pesticides in Odontocete Species in the Macaronesian Region (Eastern North Atlantic)*, 173 Marine Pollution Bull. 113105 (2021), https://doi.org/10.1016/j.marpolbul.2021.113105; Maria C. Vagi et al., *Potential Effects of Persistent Organic Contaminants on Marine Biota: A Review of Recent Research*, 13 Water 2488 (2021), https://doi.org/10.3390/w13182488.
[93] Health Canada, *Proposed Approach for Cumulative Risk Assessment of Certain Phthalates Under the Chemicals Management Plan* (2015).
[94] *Id.* at 41; *see also id.* at 41–42 (citing examples).

more scientifically robust consideration of risk than assessments on a substance-by-substance basis."[95] In addition to drawing from existing published ecological cumulative risk assessments, EPA's Office of Chemical Safety and Pollution Prevention should confer with other EPA offices that are engaged in the development and deployment of methods for assessing cumulative ecological risk from complex chemical mixtures, including the Office of Water, Office of Pesticide Programs, and the Superfund program within the Office of Land and Emergency Management.

In sum, consistent with the best available science and EPA's co-equal obligation to evaluate ecological risk under TSCA, the agency must assess the risks to wildlife from cumulative exposures to phthalates. It has the methods and data needed to do so.

## X. EPA Must Move Expeditiously to Complete its Phthalates Cumulative Risk Assessment and Reach Final Risk Determinations for the High-Priority and Manufacturer-Requested Phthalates

As described here, we urge EPA to implement and—in certain critical respects, expand upon—its proposal for cumulative risk assessment of phthalates undergoing TSCA risk evaluation. EPA must do so expeditiously, mindful of the urgent need for appropriate regulatory safeguards to address the risks these substances pose to people and wildlife and Congress's mandate that EPA complete the pending risk evaluations for the high-priority phthalates no later than June 20, 2023, and for the manufacturer-requested phthalates no later than July 2, 2023.[96] While we are advocating for EPA to expand upon its proposal in key areas, we point to authoritative models, published literature, and readily available data to support these improvements to EPA's proposal so that the agency may make these essential changes efficiently. We welcome questions about our recommendations and how the agency can develop a lawful, scientifically robust, and protective cumulative risk assessment for phthalates without undue delay.

If you have any questions about these comments, please contact Katherine O'Brien, Earthjustice, at kobrien@earthjustice.org.

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[95] *Id.* at 41–42.

[96] *See* 15 U.S.C. § 2605(b)(4)(G) (requiring EPA to complete risk evaluations within three years after initiation and allowing for an extension of up to six months); EPA, *High-Priority Substance Designations Under the Toxic Substances Control Act (TSCA) and Initiation of Risk Evaluation on High-Priority Substances; Notice of Availability*, 84 Fed. Reg. 71,924 (Dec. 30, 2019) (initiating risk evaluations for BBP, DBP, DCHP, DEHP, and DIBP effective December 20, 2019); EPA, *Di-Isodecyl Phthalate (DIDP); Final Scope of the Risk Evaluation to Be Conducted Under the Toxic Substances Control Act (TSCA); Notice of Availability*, 86 Fed. Reg. 48,695 (Aug. 31, 2021) (explaining that EPA initiated DIDP risk evaluation on January 2, 2020); EPA, *Di-isononyl Phthalate (DINP); Final Scope of the Risk Evaluation to Be Conducted Under the Toxic Substances Control Act (TSCA); Notice of Availability*, 86 Fed. Reg. 48,693, 48,694 (Aug. 31, 2021) (same for DINP).

Respectfully submitted,

Alaska Community Action on Toxics
Alianza Nacional de Campesinas, Inc.
Black Women for Wellness
Breast Cancer Prevention Partners
Center for Environmental Health
Center for Food Safety
Center for Science and Democracy, Union of Concerned Scientists
Clean Power Lake County
Defend Our Health
Detroit Hamtramck Coalition for Advancing Healthy Environments
Earthjustice
Environmental Defense Fund
NRDC
Toxic Free NC
Zero Waste Washington