March 27, 2025

Comments from University of California, San Francisco Program on Reproductive Health and the Environment to the TSCA Science Advisory Committee on Chemicals (SACC)

We appreciate the opportunity to provide information for SACC members to consider in their peer review of EPA's 2024 draft risk evaluation for 1,3-Butadiene.

TSCA requires EPA to determine whether a chemical substance presents an unreasonable risk of injury to health or the environment.[1] This requires EPA to look at the chemical as a whole, not individual uses, and is also consistent with TSCA's requirement to use the "best available science" which requires consideration of aggregate exposures.[2]

We submitted comments to EPA on the draft risk evaluation and supplement and are providing excerpts here that are most relevant to the charge questions. Our full comments are available here.[3]

Table of Contents

[1] 15 USC §2605(b).
[2] 15 USC §2625(h).
[3] https://prhe.ucsf.edu/sites/g/files/tkssra341/f/resources/2025.03.05_1-3%20Butadiene%20Draft%20Risk%20Evaluation_UCSF%20PRHE%20comments.pdf

Charge question 2: General Population Exposure Assessment and Analysis

a)ii)3) Please comment on EPA's conclusion that refined modeling of ambient air concentrations was necessary to inform cancer risk evaluation.

EPA's risk characterization for 1,3-butadiene in the draft risk evaluation clearly supports its determination of unreasonable cancer risks for communities living near facilities manufacturing and processing 1,3-butadiene, based on the results of the Integrated Indoor-Outdoor Air calculator (IIOAC).[4] The refined modeling in the draft risk evaluation using the Human Exposure Model (HEM) provides additional support for the unreasonable risk determination. However, in reality, EPA is underestimating risks because it inappropriately disregards some risks of concern to fenceline communities .[5]

Separate from the draft risk evaluation, EPA also released a more recent supplement that uses emissions data from the National Emissions Inventory (NEI) to estimate cancer risk, but EPA's supplement is too limited to inform any conclusions regarding general population risk from 1,3-butadiene emissions. Any analysis EPA conducts of cancer risk using NEI emissions should incorporate data for all facilities, model risks at the fenceline, and model combined risks of 1,3-butadiene in all locations where two or more facilities are within 50 km of each other. To ensure fenceline communities are adequately protected, where there are important differences between TRI and NEI-based risk estimates, EPA should seek to determine the reason for these differences and, in the absence of compelling evidence to the contrary, should always use the higher risk estimate in drawing any conclusions and in determination of unreasonable risk. Our full comments on the supplement begin on pg. 7.

EPA underestimates risks to fenceline communities because it did not consider real-world exposures, increased susceptibility, and cumulative exposures.

EPA's analysis underestimates risks to communities because it failed to consider aggregate exposures, reasonably available chemical release data, increased susceptibility, and cumulative exposures. The best available scientific protocols and methodologies for conducting risk assessments require consideration of all exposure pathways, accounting for aggregate and cumulative exposures, as well as increased susceptibility to harm.[6] Residents of fenceline communities must be considered a "potentially exposed or susceptible subpopulation" because they face greater chemical exposures due to their proximity to polluting facilities and

[4] U.S. EPA (2024). Draft Risk Evaluation for 1,3-Butadiene, p. 119.
[5] U.S. EPA (2024). Draft Risk Evaluation for 1,3-Butadiene, p. 121.
[6] Rayasam, S. D. G., Koman, P. D., Axelrad, D. A., Woodruff, T. J., & Chartres, N. (2022). Toxic Substances Control Act (TSCA) Implementation: How the Amended Law Has Failed to Protect Vulnerable Populations from Toxic Chemicals in the United States. *Environmental science & technology*, *56*(17), 11969–11982. https://doi.org/10.1021/acs.est.2c02079.

contaminated sites, and they often experience greater harm from those exposures due to their cumulative exposures to multiple chemicals as well as other non-chemical stressors such as poverty and racial discrimination.

a. EPA did not adequately evaluate real world exposures to 1,3-butadiene.

EPA acknowledges that communities are exposed to 1,3-butadiene from all sources - those related to TSCA conditions of use, and those not related to TSCA conditions of use (COUs). However, it declines to consider aggregate (total) exposure to 1,3-butadiene in its risk evaluation "because TSCA only provides authority to regulate exposures resulting from TSCA COUs and does not provide authority to regulate beyond TSCA COUs."[7] Whether or not the exposure comes from a TSCA condition of use, it will contribute to overall exposure and to risk. EPA must consider all sources of 1,3-butadiene exposures in its general population risk evaluation.

EPA did not consider all relevant and available chemical release data in its fenceline exposure assessment. We support EPA's use of Toxics Release Inventory data from multiple reporting years, and we also support EPA's stated intent to incorporate data from the National Emissions Inventory in the final risk evaluation ("EPA intends to incorporate exposures and risks analyses based on the 2017 and 2020 NEI reported releases for the finalized draft risk evaluation").[8] However, chemical incidents and releases also result in exposures to fenceline communities and as these events are "known" and "reasonably foreseen" consequences of chemical manufacturing, transportation, use, and disposal, they must be considered under TSCA.[9] For example, the Chemical Safety and Hazard Investigation Board released a report detailing a 2019 explosion and fire at a facility in Texas with large releases of 1,3-butadiene.[10] Additionally, facility start up, shut down and malfunction conditions also result in releases of 1,3-butadiene and exposures to fenceline communities which must be included in EPA's assessment.[11] In January of 2024, a winter storm in Texas resulted in "upset" events, with facilities reporting multiple chemical releases, including thousands of pounds of 1,3-butadiene, to the Texas Commission on Environmental Quality.[12]

b. EPA did not account for increased susceptibility of fenceline communities.

People living in fenceline communities are more likely to experience adverse health effects from chemical exposures than the general population due to a variety of factors that make them more

---

[7] U.S. EPA (2024). Draft General Population Exposure for 1,3-Butadiene, p. 26.
[8] U.S. EPA (2024). Draft General Population Exposure for 1,3-Butadiene, p. 8.
[9] 15 U.S.C. § 2602(4).
[10] Chemical Safety and Hazard Investigation Board (2022). Investigation report: TPC Group Chemical Plant Butadiene Unit. https://www.csb.gov/tpc-port-neches-explosions-and-fire/.
[11] Memorandum from Janet McCabe, Deputy Adm'r, EPA, to Reg'l Adm'rs, EPA 2 (Sept. 30, 2021), https://www.epa.gov/system/files/documents/2021-09/oar-21-000-6324.pdf (withdrawing Oct. 9, 2020, memorandum addressing startup, shutdown, and malfunctions in state implementation plans).
[12] Environment Texas (2024). Texas emissions events during January 2024 winter storm. https://environmentamerica.org/texas/center/resources/texas-emissions-events-during-january-2024-winter-storm/; The Texas Tribune (2024). Texas companies reported releasing 1 million pounds of excess pollution during recent cold snap. https://www.texastribune.org/2024/01/26/texas-pollution-emissions-cold-weather-upsets/.

susceptible to harm.[13] These factors can include biological traits like age, genetic makeup, and pre-existing health conditions, which are collectively considered *intrinsic* factors.[14]

Susceptibility to harm from chemical exposures can also be increased by external stressors, which include psychosocial stress from experiencing income inequality, violence, racism, healthcare inequity, food insecurity, or extreme weather.[15] In general, people of color in the United States experience disproportionately high levels of these external stressors, collectively known as *extrinsic* susceptibility factors, and as a result, people of color are more susceptible to negative health outcomes from chemical exposures.[16]

While any individual internal or external factor can enhance susceptibility, people living in fenceline communities often experience multiple intrinsic and extrinsic factors simultaneously, which increases the potential for even greater susceptibility to adverse effects from chemical exposures.[17] EPA does not consider increased susceptibility when assessing risks to fenceline communities. EPA thus fails to use risk assessment methodologies that are "consistent with the

---

[13] McHale, C. M., Osborne, G., Morello-Frosch, R., Salmon, A. G., Sandy, M. S., Solomon, G., Zhang, L., Smith, M. T., & Zeise, L. (2018). Assessing health risks from multiple environmental stressors: Moving from G×E to I×E. Mutation research. Reviews in mutation research, 775, 11–20. https://doi.org/10.1016/j.mrrev.2017.11.003.

[14] National Academies of Sciences, Engineering, and Medicine. 2023. Building Confidence in New Evidence Streams for Human Health Risk Assessment: Lessons Learned from Laboratory Mammalian Toxicity Tests. Washington, DC: The National Academies Press. https://doi.org/10.17226/26906.

[15] Morello-Frosch, R., Zuk, M., Jerrett, M., Shamasunder, B., & Kyle, A. D. (2011). Understanding the cumulative impacts of inequalities in environmental health: implications for policy. Health affairs (Project Hope), 30(5), 879–887. https://doi.org/10.1377/hlthaff.2011.0153; McHale, C. M., Osborne, G., Morello-Frosch, R., Salmon, A. G., Sandy, M. S., Solomon, G., Zhang, L., Smith, M. T., & Zeise, L. (2018). Assessing health risks from multiple environmental stressors: Moving from G×E to I×E. Mutation research. Reviews in mutation research, 775, 11–20. https://doi.org/10.1016/j.mrrev.2017.11.003; Payne-Sturges, D. C., Scammell, M. K., Levy, J. I., Cory-Slechta, D. A., Symanski, E., Carr Shmool, J. L., Laumbach, R., Linder, S., & Clougherty, J. E. (2018). Methods for Evaluating the Combined Effects of Chemical and Nonchemical Exposures for Cumulative Environmental Health Risk Assessment. International journal of environmental research and public health, 15(12), 2797. https://doi.org/10.3390/ijerph15122797; Gee, G. C., & Payne-Sturges, D. C. (2004). Environmental health disparities: a framework integrating psychosocial and environmental concepts. Environmental health perspectives, 112(17), 1645–1653. https://doi.org/10.1289/ehp.7074l; Solomon, G. M., Morello-Frosch, R., Zeise, L., & Faust, J. B. (2016). Cumulative Environmental Impacts: Science and Policy to Protect Communities. Annual review of public health, 37, 83–96. https://doi.org/10.1146/annurev-publhealth-032315-021807; Koman, P. D., Singla, V., Lam, J., & Woodruff, T. J. (2019). Population susceptibility: A vital consideration in chemical risk evaluation under the Lautenberg Toxic Substances Control Act. PLoS biology, 17(8), e3000372. https://doi.org/10.1371/journal.pbio.3000372; National Academies of Sciences, Engineering, and Medicine. 2023. Building Confidence in New Evidence Streams for Human Health Risk Assessment: Lessons Learned from Laboratory Mammalian Toxicity Tests. Washington, DC: The National Academies Press. https://doi.org/10.17226/26906.

[16] Gee, G. C., & Payne-Sturges, D. C. (2004). Environmental health disparities: a framework integrating psychosocial and environmental concepts. Environmental health perspectives, 112(17), 1645–1653. https://doi.org/10.1289/ehp.7074; Payne-Sturges, D. C., Scammell, M. K., Levy, J. I., Cory-Slechta, D. A., Symanski, E., Carr Shmool, J. L., Laumbach, R., Linder, S., & Clougherty, J. E. (2018). Methods for Evaluating the Combined Effects of Chemical and Nonchemical Exposures for Cumulative Environmental Health Risk Assessment. International journal of environmental research and public health, 15(12), 2797. https://doi.org/10.3390/ijerph15122797.

[17] Environmental Justice Health Alliance for Chemical Policy Reform et al. (2018). Life at the Fenceline: Understanding Cumulative Health Hazards in Environmental Justice Communities. https://ej4all.org/assets/media/documents/Life%20at%20the%20Fenceline%20-%20English%20-%20Public.pdf.

best available science,"[18] and understates the risks posed to fenceline communities. It is well established in the scientific literature that both intrinsic and extrinsic factors can increase susceptibility and thus must be taken into consideration when evaluating risks to "potentially exposed or susceptible subpopulations,"[19] including fenceline communities.

Further, the National Academy of Sciences has warned that failing to account for both intrinsic and extrinsic susceptibility factors could lead to a vast underestimation of risks from chemical exposures in the human population.[20] The SACC raised similar concerns in its evaluation of EPA's proposed Fenceline Assessment Approach, and stressed the importance of considering the impact of non-chemical stressors in chemical risk evaluation.[21] The SACC further recommended that EPA could apply safety factors to account for factors like co-occurrence of multiple chemical and non-chemical stressors.[22]

To comply with TSCA and adhere to recommendations provided by EPA's own scientific peer reviewers, EPA must consider not only fenceline communities' increased exposures but also their heightened susceptibility to 1,3-butadiene as a result of intrinsic and extrinsic susceptibility factors. EPA should apply additional adjustment factors to account for fenceline communities' increased susceptibility. To account for increased susceptibility to harm in younger age groups, California EPA's Office of Environmental Health Hazard Assessment (OEHHA) now relies on a 30X intra-species adjustment factor that is three times higher than the one currently used by EPA.[12] We recommend that EPA apply an expanded intra-species adjustment factor of 42X, consistent with the 42-fold human variability in toxicokinetic and toxicodynamic responses to chemical exposures observed by the WHO using a probabilistic method.[23] Application of this expanded adjustment factor will more adequately capture human variability in the response to

---

[18] 15 U.S.C. § 2625(h).

[19] National Research Council (2009). Science and Decisions: Advancing Risk Assessment. pp 110-111.Washington, DC: The National Academies Press. https://doi.org/10.17226/12209; Morello-Frosch, R., Zuk, M., Jerrett, M, Shamasunder, B., & Kyle, A. D. (2011). Understanding the cumulative impacts of inequalities in environmental health: implications for policy. Health affairs (Project Hope), 30(5), 879–887. https://doi.org/10.1377/hlthaff.2011.0153; McHale, C. M., Osborne, G., Morello-Frosch, R., Salmon, A. G., Sandy, M. S., Solomon, G., Zhang, L., Smith, M. T., & Zeise, L. (2018). Assessing health risks from multiple environmental stressors: Moving from G×E to I×E. Mutation research. Reviews in mutation research, 775, 11–20. https://doi.org/10.1016/j.mrrev.2017.11.003; Payne-Sturges, D. C., Scammell, M. K., Levy, J. I., Cory-Slechta, D. A., Symanski, E., Carr Shmool, J. L., Laumbach, R., Linder, S., & Clougherty, J. E. (2018). Methods for Evaluating the Combined Effects of Chemical and Nonchemical Exposures for Cumulative Environmental Health Risk Assessment. International journal of environmental research and public health, 15(12), 2797. https://doi.org/10.3390/ijerph15122797.

[20] National Research Council (2009). Science and Decisions: Advancing Risk Assessment. pp 9-10. Washington, DC: The National Academies Press. https://doi.org/10.17226/12209.

[21] U.S. EPA (2022). Final Report on Draft TSCA Screening Level Approach for Assessing Ambient Air and Water Exposures to Fenceline Communities Version 1.0 pp 49.Available: https://www.epa.gov/system/files/documents/2022-01/draft-fenceline-report_sacc.pdf.

[22] U.S. EPA (2022). Final Report on Draft TSCA Screening Level Approach for Assessing Ambient Air and Water Exposures to Fenceline Communities Version 1.0 pp 65.Available: https://www.epa.gov/system/files/documents/2022-01/draft-fenceline-report_sacc.pdf.

[23] WHO IPCS (2017). Guidance Document on Evaluating and Expressing Uncertainty in Hazard Characterization. Available: http://www.inchem.org/documents/harmproj/harmproj/harmproj11.pdf.

1,3-butadiene exposures, including in highly exposed or susceptible subpopulations, and is consistent with recommendations made by scientific experts.[24]

c. EPA did not consider cumulative risk of exposures to multiple chemicals sharing common adverse outcomes with 1,3-butadiene.

EPA fails to consider communities' cumulative exposures to other chemicals, in addition to 1,3-butadiene, from a variety of sources and pathways (see more in Comment 8, cumulative risk assessment, below). In doing so, EPA is ignoring the real-world exposures and risks faced by many fenceline communities**.** EPA's failure to consider cumulative exposures is particularly problematic for chemicals that contribute to common adverse health outcomes, which could increase the likelihood of harm to communities exposed to 1,3-butadiene.[25] For EPA to assess fenceline communities' risks without considering cumulative exposures is not "consistent with the best available science,"[26] in violation of TSCA. The National Research Council has not only recommended the consideration of cumulative exposures in risk evaluations, but has also warned that "risk assessment might become irrelevant in many decision contexts" without it.[27] TSCA requires EPA to use scientifically supported approaches and methodologies to "integrate and assess available information on hazards and exposures," including those that contribute to cumulative risks in fenceline communities.[28] This information includes a recent study that outlined methods for identifying cumulative exposures to chemicals that contribute to similar adverse health effects in highly exposed and susceptible groups.[29] Consistent with

---

[24] Varshavsky, J. R., Rayasam, S. D. G., Sass, J. B., Axelrad, D. A., Cranor, C. F., Hattis, D., Hauser, R., Koman, P. D., Marquez, E. C., Morello-Frosch, R., Oksas, C., Patton, S., Robinson, J. F., Sathyanarayana, S., Shepard, P. M., & Woodruff, T. J. (2023). Current practice and recommendations for advancing how human variability and susceptibility are considered in chemical risk assessment. Environmental health : a global access science source, 21(Suppl 1), 133. https://doi.org/10.1186/s12940-022-00940-1.

[25] National Research Council (2008). Phthalates and Cumulative Risk Assessment: The Tasks Ahead. pp 4-11. Washington, DC: The National Academies Press. https://doi.org/10.17226/12528; Solomon, G. M., Morello-Frosch, R., Zeise, L., & Faust, J. B. (2016). Cumulative Environmental Impacts: Science and Policy to Protect Communities. Annual review of public health, 37, 83–96. https://doi.org/10.1146/annurev-publhealth-032315-021807; Rayasam, S. D. G., Koman, P. D., Axelrad, D. A., Woodruff, T. J., & Chartres, N. (2022). Toxic Substances Control Act (TSCA) Implementation: How the Amended Law Has Failed to Protect Vulnerable Populations from Toxic Chemicals in the United States. *Environmental science & technology*, *56*(17), 11969–11982. https://doi.org/10.1021/acs.est.2c02079; Vandenberg, L. N., Rayasam, S. D. G., Axelrad, D. A., Bennett, D. H., Brown, P., Carignan, C. C., Chartres, N., Diamond, M. L., Joglekar, R., Shamasunder, B., Shrader-Frechette, K., Subra, W. A., Zarker, K., & Woodruff, T. J. (2023). Addressing systemic problems with exposure assessments to protect the public's health. Environmental health : a global access science source, 21(Suppl 1), 121. https://doi.org/10.1186/s12940-022-00917-0; Pullen Fedinick, K., Yiliqi, I., Lam, Y., Lennett, D., Singla, V., Rotkin-Ellman, M., & Sass, J. (2021). A Cumulative Framework for Identifying Overburdened Populations under the Toxic Substances Control Act: Formaldehyde Case Study. International journal of environmental research and public health, 18(11), 6002. https://doi.org/10.3390/ijerph18116002.

[26] 15 U.S.C. § 2625(h).

[27] National Research Council (2009). Science and Decisions: Advancing Risk Assessment. pp 9-10.Washington, DC: The National Academies Press. https://doi.org/10.17226/12209; National Research Council (2008). Phthalates and Cumulative Risk Assessment: The Tasks Ahead. Pp. 4-11. Washington, DC: The National Academies Press. https://doi.org/10.17226/12528.

[28] 15 U.S.C. § 2605(b)(4)(F)(i).

[29] Pullen Fedinick, K., Yiliqi, I., Lam, Y., Lennett, D., Singla, V., Rotkin-Ellman, M., & Sass, J. (2021). A Cumulative Framework for Identifying Overburdened Populations under the Toxic Substances Control Act:

recommendations made by scientific experts,[30] EPA could apply additional adjustment factors to account for any cumulative risks to fenceline communities that are exposed to 1,3-butadiene and to other chemicals with common adverse outcomes.

Comments on the supplement to the 1,3-butadiene risk evaluation

In the 1,3-Butadiene Draft Risk Evaluation, EPA appropriately determined that air emissions of 1,3-butadiene pose an unreasonable risk of cancer to the general public, with fenceline communities exposed to risks greater than 1-in-a million. EPA's previous analysis was based on emissions data from the Toxics Release Inventory (TRI). EPA's new supplement uses emissions data from the National Emissions Inventory (NEI) to estimate cancer risk, but only for 9 facilities with previous risk estimates greater than 1-in-100,000. The supplement does not present risk estimates for approximately 700 other facilities with data on 1,3-butadiene emissions in NEI, including the facilities with the highest emissions.

EPA reports that the cancer risks for the 9 facilities modeled are lower when using NEI data as compared with risk estimates derived with TRI data, but it provides no explanation for this result. EPA compared the TRI and NEI-based estimates at census block centroids, when a more appropriate comparison would be at the fenceline for each facility, approximated as 100 meters from the release point. In the case of at least one out of the 9 facilities, the estimate of 2017 1,3-butadiene emissions in the NEI is approximately 300-fold lower than the TRI value; EPA does not discuss why it has 2 emissions estimates for the same facility in the same year that are so drastically different. In the absence of a concrete explanation based on strong and verifiable evidence, EPA should base any conclusions on the higher emissions estimate to ensure protection of fenceline communities. In addition, the EPA supplement estimates risk based only on emissions from single facilities in isolation rather than aggregate ambient concentrations resulting from combined emissions of multiple facilities. Two out of the 9 facilities modeled by EPA in the supplement are both located in Orange, TX, but EPA's analysis does not recognize that the air inhaled by community residents will have 1,3-butadiene from both facilities, as well as emissions of the chemical from numerous additional facilities in Orange and other nearby cities. As a result, EPA's supplement fails to apply the best available science and underestimates real-world cancer risks to the most-burdened fenceline communities.

EPA's supplement is too limited to inform any conclusions regarding general population risk from 1,3-butadiene emissions. Any analysis EPA conducts of cancer risk using NEI emissions should incorporate data for all facilities, model risks at the fenceline, and model combined risks of 1,3-butadiene in all locations where two or more facilities are within 50 km of each other. To ensure fenceline communities are adequately protected, where there are important differences between TRI and NEI-based risk estimates, EPA should seek to determine the reason for these

---

Formaldehyde Case Study. International journal of environmental research and public health, 18(11), 6002. https://doi.org/10.3390/ijerph18116002.

[30] Varshavsky, J. R., Rayasam, S. D. G., Sass, J. B., Axelrad, D. A., Cranor, C. F., Hattis, D., Hauser, R., Koman, P. D., Marquez, E. C., Morello-Frosch, R., Oksas, C., Patton, S., Robinson, J. F., Sathyanarayana, S., Shepard, P. M., & Woodruff, T. J. (2023). Current practice and recommendations for advancing how human variability and susceptibility are considered in chemical risk assessment. Environmental health : a global access science source, 21(Suppl 1), 133. pp.3. https://doi.org/10.1186/s12940-022-00940-1.

differences and, in the absence of compelling evidence to the contrary, should always use the higher risk estimate in drawing any conclusions and in determination of unreasonable risk.

Our detailed comments on the supplement to the 1,3-Butadiene Draft Risk Evaluation address the following issues:

1. *EPA's Draft Risk Evaluation appropriately determined that air emissions of 1,3-butadiene pose an unreasonable risk of cancer to the general public. EPA's limited and flawed supplement analyzing a small subset of emitting facilities should have no impact on this conclusion and is not an adequate basis for decision-making.*

2. *EPA's supplement significantly underestimates risk by disregarding the aggregate exposures of the general public to 1,3-butadiene in communities located near multiple emitting facilities.*

3. *EPA's supplement underestimates risk by disregarding many of the facilities with the largest emissions reported in the NEI.*

Detailed Comments:

1. *EPA's Draft Risk Evaluation appropriately determined that air emissions of 1,3-butadiene pose an unreasonable risk of cancer to the general public. EPA's limited and flawed supplement analyzing a small subset of emitting facilities should have no impact on this conclusion and is not an adequate basis for decision-making.*

In the draft risk evaluation, EPA concluded that the general population is at unreasonable risk from air exposures to 1,3-butadiene based on exposed populations with cancer risks greater than the 1-in-a-million benchmark. EPA's draft risk evaluation shows that 47 facilities pose cancer risks greater than 1-in-a-million to nearby residents.[31] These risk estimates were derived using data from the Toxics Release Inventory (TRI).

In the draft risk evaluation, EPA stated its intent to conduct further modeling using data from the National Emissions Inventory (NEI):

> EPA intends to incorporate exposures and risks analyses based on the 2017 and 2020 NEI reported releases for the finalized draft risk evaluation.[32]

We support EPA's intent to incorporate analysis using NEI data alongside the TRI estimates in the final risk evaluation, however EPA's supplementary analysis using only a small amount of NEI data does not satisfy TSCA's requirement for using the best available science. EPA has released for comment a very limited analysis using NEI data for only 9 facilities – or roughly 1%

[31] U.S. EPA (2024). Draft Human Exposure Model (HEM) TRI 2016 to 2021 Exposure and Risk Analysis for 1,3-Butadiene, "Population Risk" tab.
[32] U.S. EPA (2024). Draft General Population Exposure for 1,3-Butadiene, p. 8.

of the more than 700 facilities with 2020 data in the NEI. No firm conclusions can be drawn from such a limited analysis, and the purpose of this analysis in unclear.

EPA's supplement compares the original TRI-based risk estimates for each of the 9 facilities alone to NEI-based estimates and concludes that use of the NEI data results in lower cancer risks at each site. However, the scope of the supplementary analysis is much narrower than the previous analysis conducted using TRI data. EPA described its analysis of risks from 1,3-butadiene air emissions using TRI data in the 1,3-Butadiene Draft Risk Evaluation as follows:

> For general population exposures, including exposures to fenceline communities, EPA modeled air concentrations from facilities, focusing on the distances of 100 m, 100 to 1,000 m, and 1,000 m from release points, and aggregated exposures from multiple facilities from all releasing facilities within a 50,000-meter radius to the general population within a given census block based on 2020 census data.[33]

The supplement did not conduct the same modeling. In contrast to the draft risk evaluation, EPA's conclusion in the supplementary analysis is based on inappropriate comparisons of the estimated "census block with the highest risk" from TRI and NEI for each facility,[34] instead of comparing risks at 100 meters. Census block centroids can be located far from the fenceline, and thus the cancer risk estimates being compared may substantially understate the risk to community residents exposed to the highest concentrations of 1,3-butadiene. EPA's analysis using NEI data should instead be assessing 1,3-butadiene concentrations and risks within 100 meters of the release point at each facility.

In addition, the supplemental analysis using NEI data considers risks only from individual facilities, and not aggregate exposures to community residents from all facilities within 50 km (see further comment below).

Further, it is not clear that EPA has modeled each site with all relevant NEI data. For example, EPA provides the identifier for the Shell Norco plant as EISD 8239511.[35] However, the Excel file presenting the NEI data reports emissions from this plant with both EISD 8239511 and an additional identifier of EISD 8018911.[36] It appears that EPA's modeling excluded the emissions from Shell Norco reported in the NEI for EISD 8018911, and therefore has underestimated the population risk from Shell Norco emissions.

EPA makes no attempt to determine the reasons for any discrepancies between the TRI-based and NEI-based risk estimates. For the Total Energies facility in Port Arthur, TX, EPA reports a 2000-fold lower risk when using the NEI: 7.4E-05 risk based on TRI and 3.7E-08 risk based on NEI.[37] EPA should not report such large differences in risk without some effort to determine the underlying reasons. In this case, it appears that the TRI and NEI have vastly different emissions estimates for this facility; for 2017, TRI emissions (TRI ID 77640FNLNDHIGHW) are reported

---

[33] U.S. EPA (2024). Draft Risk Evaluation for 1,3-Butadiene, p. 112.
[34] U.S. EPA (2025). 1,3-Butadiene TRI and NEI Risk Estimate Comparison Analysis, Table 1.
[35] U.S. EPA (2025). 1,3-Butadiene TRI and NEI Risk Estimate Comparison Analysis, Table 1.
[36] U.S. EPA (2024). Draft Air Releases (NEI 2017) for 1,3-Butadiene, "2017 & 2020 Summary_Point" tab.
[37] U.S. EPA (2025). 1,3-Butadiene TRI and NEI Risk Estimate Comparison Analysis, Table 1.

as 143,508 pounds[38] (equal to 65,231 kg) and NEI emissions (EISD 4863111) are 223 kg,[39] or 300-fold lower. The NEI value seems implausible and could be an error; until a reason for the discrepancy is determined, the NEI-based risk estimate for this facility should be regarded as highly unreliable and should be disregarded.

EPA should proceed with a thorough analysis making use of NEI data, as it previously indicated. This analysis may be useful in identifying additional locations at high risk that were not identified by the TRI analysis. To ensure protection of fenceline communities, in any instance where there are significant differences between TRI-based and NEI-based estimates, EPA should use the higher risk for risk characterization and unreasonable risk determination unless it has detailed and thorough documentation of facility-specific evidence to substantiate that erroneous data were used to develop that estimate.

2. *EPA's supplement significantly underestimates risk by disregarding the aggregate exposures of the general public to 1,3-butadiene in communities located near multiple emitting facilities.*

EPA's supplement reports cancer risk estimates for each of 9 facilities considered in isolation, without assessing the combined community exposures to 1,3-butadiene emitted from multiple neighboring sources. Two out of the 9 facilities that EPA modeled with NEI data are located in Orange, TX: Lion Elastomers (EISD 5780411, zip code 77630) and Arlanxeo (EISD 3961411, zip code 77630). Residents living near one of these plants are also very likely to be exposed to emissions from the other plant. EPA's approach treats each facility as if they are hundreds of miles apart rather than in close proximity to one another, and thus significantly underestimates the risk.

For 1,3-butadiene, this issue is much more extensive than just 2 neighboring plants. In addition to Lion Elastomers and Arlanxeo, at least three other large 1,3-butadiene emitters are found in Orange (EISDs 4190211, 5780411, 10678011, zip codes 77630 and 77631) and at least 13 more emitting facilities are located in the neighboring Golden Triangle cities of Beaumont, Port Neches, and Port Arthur, TX (zip codes 77640, 77643, 77651, 77701, 77704, 77705) – a total of at least 18 facilities in the 2020 NEI.[40] Other communities outside of the Golden Triangle also are exposed to 1,3-butadiene emitted by multiple facilities.

Any determination of risk to fenceline communities or the general population that does not consider the combined air concentrations of 1,3-butadiene from multiple emitters will understate risk. The risk estimates presented in EPA's supplement are not consistent with the best available science because they disregard how the close proximity of multiple facilities results in greater concentrations of 1,3-butadiene in neighboring comunities.

[38] U.S. EPA (2024). Draft Air Releases (TRI) for 1,3-Butadiene, "2016-2021 TRI" tab.
[39] U.S. EPA (2024). Draft Air Releases (NEI 2017) for 1,3-Butadiene, "2017 & 2020 Summary_Point" tab.
[40] U.S. EPA (2024). Draft Air Releases (NEI 2017) for 1,3-Butadiene, "2017 & 2020 Summary_Point" tab.

3. *EPA's supplement underestimates risk by disregarding many of the facilities with the largest emissions reported in the NEI.*

EPA's supplement presents risk estimates for only 9 facilities – only a small portion of the 47 facilities found to exceed 1-in-a-million risk using TRI data, and a smaller portion of the more than 700 facilities with 2020 data in the NEI. The 9 facilities addressed in the supplement are also not the sites with the greatest emissions in the NEI, and therefore are unlikely to present the greatest risks when modeling with NEI data.

Table 1 presents NEI emissions estimates for all facilities with emissions greater than 5,000 kg in either 2017 or 2020. The facility with the greatest emissions is the Channelview complex in Channelview, TX, with 77,032 kg emitted in 2020 – approximately 2.5 times the emissions of the highest-emitting plant that was modeled (the Arlanxeo facility in Orange, TX – 31,345 kg in 2020). The Chocolate Bayou Plant in Alvin, TX, with 47,617 kg emitted in 2017 also has greater emissions than any one of the 9 facilities modeled by EPA, and 3 additional facilities have emissions greater than 20,000 kg in 2017 and/or 2020.

EPA should use NEI data for all facilities and not a small, selective subset. If EPA's intent is to provide an illustrative analysis of what NEI-based results might look like using only a small number of facilities, it should focus that analysis on the highest-emitting facilities while also examining locations where multiple 1,3-butadiene emitters are located in close proximity to one another.

Table 1. Emissions of 1,3-butadiene reported in EPA's National Emissions Inventory (NEI) for 2017-2020: Facilities with emissions of 5,000 kg/yr or more.

Facility Name	City	State	Zip	EIS Facility Identifier	Year	NEI Emissions (kg/yr)	Included in EPA supplement?
CHANNELVIEW COMPLEX	CHANNELVIEW	TX	77530	4925111	2020	77,032	NO
CHANNELVIEW COMPLEX	CHANNELVIEW	TX	77530	4925111	2017	48,514	NO
CHOCOLATE BAYOU PLANT	ALVIN	TX	77512	5632411	2017	47,617	NO
ORANGE PLANT (Arlanxeo)	**ORANGE**	**TX**	**77630**	**3961411**	**2020**	**31,345**	**Yes**
DEER PARK PLANT	DEER PARK	TX	77536	4168511	2017	29,450	NO
SABIC Innovative Plastics US LLC	Ottawa	IL	61350	7339111	2020	28,810	NO
CHOCOLATE BAYOU PLANT	ALVIN	TX	77512	5632411	2020	27,326	NO
SABIC Innovative Plastics US LLC	Ottawa	IL	61350	7339111	2017	24,949	NO

Facility Name	City	State	Zip	EIS Facility Identifier	Year	NEI Emissions (kg/yr)	Included in EPA supplement?
ORANGE PLANT (Arlanxeo)	**ORANGE**	**TX**	**77630**	**3961411**	**2017**	**23,681**	**Yes**
BAYTOWN OLEFINS PLANT	BAYTOWN	TX	77520	4056511	2017	22,630	NO
BAYTOWN OLEFINS PLANT	BAYTOWN	TX	77520	4056511	2020	20,134	NO
PORT NECHES OPERATIONS C4 PLANT (TPC Group)	**PORT NECHES**	**TX**	**77651**	**13407911**	**2017**	**19,169**	**Yes**
DEER PARK CHEMICALS	DEER PARK	TX	77536	4168511	2020	15,219	NO
HOUSTON CHEMICAL PLANT	HOUSTON	TX	77017	4941211	2020	14,652	NO
BEAUMONT CHEMICAL PLANT (Goodyear)	**BEAUMONT**	**TX**	**77704**	**5653011**	**2017**	**13,560**	**Yes**
FORMOSA POINT COMFORT PLANT	POINT COMFORT	TX	77978	5633411	2017	13,381	NO
Shell Chemical LP - Norco Chemical Plant - East Site	**Norco**	**LA**	**70079**	**8239511**	**2017**	**12,268**	**Yes**
BORGER PLANT	BORGER	TX	79007	6157311	2020	11,977	NO
BASF TOTAL NAFTA REGION OLEFINS COMPLEX	PORT ARTHUR	TX	77640	6445411	2017	11,256	NO
VICTORIA SITE	VICTORIA	TX	77901	5679711	2020	10,908	NO
BEAUMONT CHEMICAL PLANT	BEAUMONT	TX	77704	4930211	2017	10,735	NO
ORANGE SITE	ORANGE	TX	77631	10678011	2020	10,702	NO
BEAUMONT CHEMICAL PLANT (Goodyear)	**BEAUMONT**	**TX**	**77705**	**5653011**	**2020**	**9,959**	**Yes**
PHILTEX RYTON PLANT	BORGER	TX	79007	6157311	2017	9,282	NO
VICTORIA SITE	VICTORIA	TX	77901	5679711	2017	9,275	NO
PORT NECHES OPERATIONS C4	**PORT NECHES**	**TX**	**77651**	**13407911**	**2020**	**9,142**	**Yes**

Facility Name	City	State	Zip	EIS Facility Identifier	Year	NEI Emissions (kg/yr)	Included in EPA supplement?
PLANT (TPC Group)							
GALENA PARK TERMINAL	GALENA PARK	TX	77547	6533811	2017	8,955	NO
TEXAS OPERATIONS	LONGVIEW	TX	75607	4941511	2017	7,545	NO
BASF TOTAL NAFTA REGION OLEFINS COMPLEX	PORT ARTHUR	TX	77640	6445411	2020	7,436	NO
Union Carbide Corp - St Charles Plant	Hahnville	LA	70057	7202911	2020	7,369	NO
BAYPORT FACILITY (Dixie Chemical)	**LA PORTE**	**TX**	**77571**	**4862611**	**2020**	**7,353**	**Yes**
Firestone Polymers LLC - Lake Charles Facility	**Sulphur**	**LA**	**70665**	**8465911**	**2017**	**7,307**	**Yes**
INVISTA SARL SABINE RIVER SITE	ORANGE	TX	77631	10678011	2017	7,303	NO
PORT NECHES SYNTHETIC RUBBER PLANT	PORT NECHES	TX	77651	5651611	2020	6,411	NO
EQUISTAR CHEMICALS, LP	CLINTON	IA	52732	5509711	2020	6,159	NO
HOUSTON PLANT	HOUSTON	TX	77017	4168611	2020	6,106	NO
Sasol Chemicals (USA) LLC - Lake Charles Chemical Complex	**Westlake**	**LA**	**70669**	**8468011**	**2020**	**6,030**	**Yes**
HOUSTON PLANT	HOUSTON	TX	77017	4168611	2017	6,029	NO
GALENA PARK TERMINAL	GALENA PARK	TX	77547	6533811	2020	5,948	NO
Westlake Chemical OpCo LP	Calvert City	KY	42029	18100711	2020	5,836	NO
CEDAR BAYOU PLANT	HOUSTON	TX	77029	4056411	2020	5,591	NO
HOUSTON CHEMICAL PLANT	HOUSTON	TX	77017	4941211	2017	5,265	NO

Facility Name	City	State	Zip	EIS Facility Identifier	Year	NEI Emissions (kg/yr)	Included in EPA supplement?
NOVA Chemicals Olefins LLC - Geismar Ethylene Plant	Geismar	LA	70734	7445911	2020	5,083	NO

Source: U.S. EPA (2024). Draft Air Releases (NEI 2017) for 1,3-Butadiene, "2017 & 2020 Summary_Point" tab. Facility names shown in parentheses obtained from U.S. EPA (2025). 1,3-Butadiene TRI and NEI Risk Estimate Comparison Analysis, Table 1.

Facilities shown in **bold** were modeled in EPA's 1,3-butadiene supplementary analysis.

Charge Question 4: Occupational Exposure Assessment

EPA's determination of unreasonable risk in occupational settings inappropriately discounts and disregards exposure levels of 50% of workers, including high-end exposures, without justification and violates TSCA's requirement to assess risks to groups with greater exposures.

EPA's risk characterization for 1,3-butadiene clearly supports its determination of unreasonable risk to workers, but EPA has underestimated risks to workers because there are significant flaws in the exposure assumptions it used. The use of high-end exposure estimates to inform risk characterization is consistent with the best available science, EPA's practice in previous TSCA risk evaluations, and with the statutory requirements of TSCA. However, in the 1,3-butadiene Draft Risk Evaluation, EPA failed to use high-end exposure estimates to inform unreasonable risk determinations for workers for chronic non-cancer and cancer risks, effectively ignoring the higher-than-average risks that occur among 50% of workers.

EPA's use of central tendency estimates only for chronic exposure assumes that the exposure levels for all workers will generally fall near the "average exposures" over time. EPA inaccurately rationalizes the use of central tendency over high-end estimates:

> Central tendency is used for EPA's preliminary risk determination for chronic non-cancer and lifetime cancer estimates since longer-term average exposure (e.g., 250 days per working years or 78 years for cancer estimates) *would bias toward central tendency* (i.e., the more common risk estimates) vs. higher-end values (i.e., less common risk estimates or 95th percentile or value at which 95% of all measurements fall below it).[41]

This statement is simply the definition of the central tendency, which is representative of only typical or more common levels in the population, and not a rationale for disregarding chronic exposures and risks to workers with higher than typical chronic exposure levels. In choosing to rely on only the central tendency, EPA does not consider whether there is unreasonable risk to the 50% of the population with exposures greater than the central tendency. Further, it fails to meet

[41] U.S. EPA (2024). Draft Risk Evaluation for 1,3-Butadiene, p. 112 (Emphasis added).

its obligation under TSCA to identify any unreasonable risks to PESS, which include groups who "due to…greater exposure, may be at greater risk than the general population."[42] EPA's current approach fails to capture the risk for individuals with higher-than-average chronic exposures, such as those in the 99th percentile, who may be at much higher risk.

EPA assumes that central tendencies are more appropriate for chronic non-cancer and cancer risks "for longer-term average exposure."[43] This approach is misleading and assumes that **all** long-term exposure will align with the average of short-term measurements. EPA's approach incorrectly assumes that there is no variability across workplaces in long-term concentrations of 1,3-butadiene and that each day's concentration of 1,3-butadiene is independent of the levels in the same facility in previous days. In fact, different workplaces have different equipment and different procedures that are highly likely to result in consistent and highly-correlated day-to-day concentrations (a facility with high levels last month and last year is likely to have high levels today and tomorrow), and thus differences in chronic exposure concentrations. Long-term exposure estimates should consider not just average exposures, but also those workers who are exposed at higher levels over sustained periods of time. Relying on central tendency alone will underestimate the real-world exposure and potential harm to these workers.

Moreover, EPA inappropriately uses single-day averages from monitoring data as a basis for the risk estimates without additional adjustments to account for the limitations of the data. The use of single-day averages will very likely miss days with high peak concentrations, underestimating the risk to more highly exposed workers. Furthermore, the available monitoring data is not fully representative of the full range of facilities that produce or process 1,3-butadiene. It is reasonably foreseeable that there are facilities where exposures could be much higher than indicated by the available measurements.

EPA's failure to use high-end exposure estimates results in an understatement of unreasonable risks to workers. For a number of COUs EPA considered high-end exposure estimates only for intermediate (30-day) non-cancer risks, while considering only central tendency exposure estimates in the determination of chronic (longer than 30 days) non-cancer and cancer unreasonable risks. This results in underestimating chronic unreasonable risks.

For example, EPA found that the COU Manufacturing – Infrastructure/ Distribution Operations contributed to unreasonable risk from intermediate non-cancer *only*. However, EPA should have also found that this COU contributed to unreasonable risk for both chronic non-cancer and cancer impacts. EPA's calculated high-end risk estimates (which were disregarded in the unreasonable risk determination) for both chronic non-cancer and cancer were at levels that it considers unreasonable for central tendency estimates (non-cancer MOE = 11 and cancer risk = 3.4E-04).

It is unclear why EPA is not considering the risk estimates it has already calculated based on high-end exposures in its determinations of unreasonable risk.

[42] 15 U.S.C. §2602(12).
[43] U.S. EPA (2024). Draft Risk Evaluation for 1,3-Butadiene, p. 112.

According to Table 5-4, all of the following COUs should be considered as contributing to unreasonable risks for chronic non-cancer or cancer impacts, based on risk estimates using high-end exposures:

- Manufacturing – Infrastructure/ Distribution Operations – for chronic non-cancer and cancer risks
- Manufacturing – Instrument and Electrical – for 8-hour and 12-hour TWA cancer risks
- Manufacturing – Laboratory Technician – for chronic non-cancer and cancer risks
- Manufacturing – Machinery and Specialists – for chronic non-cancer and cancer risks
- Manufacturing – Maintenance – for chronic non-cancer and cancer risks
- Manufacturing – Maintenance – Turnaround – for chronic non-cancer and cancer risks
- Manufacturing – Operations Onsite – for chronic non-cancer and cancer risks
- Manufacturing – Safety Health and Engineering – for chronic non-cancer and cancer risks
- Processing – Processing as a Reactant – Intermediate – Infrastructure/ Distribution Operations – for chronic non-cancer and cancer risks
- Processing – Processing as a Reactant – Intermediate – Instrumental and Electrical – for cancer risks
- Processing – Processing as a Reactant – Monomer used in polymerization process – Worker – for chronic non-cancer and cancer risks
- Processing – Processing as a Reactant – Monomer used in polymerization process – ONU (12-hr TWA) – for cancer risks
- Processing – Incorporation into Formulation, Mixture, or Reaction Product – Infrastructure/ 2210 Distribution Operations – for chronic non-cancer and cancer risks
- Processing – Incorporation into Formulation, Mixture, or Reaction Product – Instrument and Electrical (8-hr and 12-hr TWA) – for cancer risks
- Processing – Incorporation into Formulation, Mixture, or Reaction Product – Laboratory Technician – for chronic non-cancer and cancer risks
- Processing – Incorporation into Formulation, Mixture, or Reaction Product – Machinery and Specialists – for chronic non-cancer and cancer risks
- Processing – Incorporation into Formulation, Mixture, or Reaction Product – Maintenance – for chronic non-cancer and cancer risks
- Processing – Incorporation into Formulation, Mixture, or Reaction Product – Maintenance – Turnaround – for chronic non-cancer and cancer risks
- Processing – Incorporation into Formulation, Mixture, or Reaction Product – Operations Onsite – for chronic non-cancer and cancer risks
- Processing – Incorporation into Formulation, Mixture, or Reaction Product – Safety Health and Engineering – for chronic non-cancer and cancer risks
- Processing – Incorporation into Article – Other: Polymer in: Rubber and plastic product manufacturing (Worker) – for chronic non-cancer and cancer risks

Furthermore, Table 5-4 presents the risk estimates in a way that is unclear and difficult for readers to interpret accurately. There is a noticeable omission of data for the COU "Processing--Processing as a Reactant – Intermediate." Table 5-4 should be revised in the final risk evaluation to improve clarity, include the missing data, and enhance overall transparency.

Overall, the use of central tendency estimates in the unreasonable risk determination does not ensure that workers with greater than the median exposure level (half of workers) are adequately protected. By prioritizing use of high-end chronic exposure and risk estimates for determining unreasonable risk and addressing exposures at the 95th or 99th percentiles, EPA would better reflect the risks to workers who are at greater risk, fulfilling its mandate to protect worker health.

Charge question 5: Human Health Hazard

b)iii) Please comment on EPA's preliminary conclusion that ovarian atrophy is not appropriate for extrapolating to human risk due to differences in species-specific metabolites and substantial uncertainty in quantifying the relevant metabolite concentrations in humans.

EPA underestimates non-cancer risks because it inappropriately excludes the most sensitive non-cancer endpoint, ovarian atrophy, without appropriate scientific justification.

EPA inappropriately relies on a hypothesized mechanism of action (MOA) proposed in Kirman et al. 2012 to dismiss ovarian atrophy in its assessment for non-cancer hazard identification.[44] EPA adopts the proposed Kirman MOA with slight modification and uses it to suggest "there may be greatly reduced sensitivity in humans" to ovarian toxicity from 1,3-butadiene.[45] This is not supported by the scientific evidence.

First, the MOA hypothesized by Kirman and by EPA does not have supporting evidence in the critical key event, follicle depletion (which EPA calls Key Event 3). EPA acknowledges that "the mechanism for how 1,3-butadiene metabolites lead to follicle depletion is unclear."[46] Thus EPA is making a scientific decision based on an unsubstantiated hypothesis.

It is for exactly this reason, that in 2013, one year after the Kirman study was published, the California Office of Environmental Health Hazard Assessment (OEHHA) found "There is currently no accepted mode of action for the acute or chronic effects of butadiene exposure noted in this document," including ovarian atrophy.[47] Further, OEHHA found that humans are very likely *more* sensitive to ovotoxic effects, noting "Humans differ substantially from mice in lifespan and in the time available for chronic exposure to effect ovotoxicity which is far longer in humans, and the generally greater robustness of the mouse reproductive system relative to the human."[48]

---

[44] U.S. EPA (2024). Draft Human Health Hazard Assessment for 1,3-Butadiene, p. 22; C.R. Kirman, R.L. Grant, Quantitative human health risk assessment for 1,3-butadiene based upon ovarian effects in rodents, Regulatory Toxicology and Pharmacology, Volume 62, Issue 2, 2012, Pages 371-384, ISSN 0273-2300, https://doi.org/10.1016/j.yrtph.2011.11.001.
[45] U.S. EPA (2024) Draft Human Health Hazard Assessment for 1,3-Butadiene, p. 27.
[46] U.S. EPA (2024) Draft Human Health Hazard Assessment for 1,3-Butadiene, p. 24.
[47] California Office of Environmental Health Hazard Assessment (2013) 1,3-Butadiene reference exposure levels. p. 30. https://oehha.ca.gov/media/downloads/crnr/072613bentcrel.pdf.
[48] California Office of Environmental Health Hazard Assessment (2013) 1,3-Butadiene reference exposure levels. p. 35. https://oehha.ca.gov/media/downloads/crnr/072613bentcrel.pdf.

Further, the conflict of interest statement for Kirman states that the American Chemistry Council provided funding for the work, yet EPA does not consider this financial conflict of interest in assessing the quality of the Kirman study and evaluating its conclusions in comparison to the conclusions from OEHHA's assessment.[49] The NASEM and SACC have both recommended that EPA account for the bias that can result from financial conflicts of interest when assessing the quality of studies.[50]

EPA is not using the best available science when it discounts the ovarian atrophy endpoint and is instead using a hypothetical scenario with insufficient data to bolster a weak MOA analysis that other independent, authoritative sources rejected. Ovarian atrophy is the most sensitive non-cancer health hazard and there is sufficient relevant, high-quality data for EPA to use this endpoint in its non-cancer dose-response assessment.

f)i) Please comment on EPA's evaluation and incorporation of new epidemiological cohort data in the derivation of updated cancer hazard values, including study selection for dose-response analysis.

EPA underestimates cancer risks because of reliance on inaccurate exposure estimates in the calculation of inhalation unit risk (IUR).

As 1,3-butadiene has a mutagenic mode of action, EPA appropriately used a linear cancer assessment approach. However, the inhalation unit risk (IUR) EPA derived is about 10-fold lower (less potent) than the value calculated by EPA in its 2002 Integrated Risk Information System (IRIS) assessment.[51] EPA notes this is primarily due to using revised, higher exposure estimates from Macaluso et al. 2004: "when comparable exposure-response models are used, differences in key parameter estimates are due primarily to changes in exposure estimates for the SBR [styrene-butadiene rubber] cohort."[52]

<u>EPA inappropriately used overestimated modeled exposure estimates rather than the actual measured exposures for the primary occupational health study.</u> EPA states that the exposure estimates in Macaluso, et al.2004 "were revised upward by as much as an order of magnitude," compared to the EPA IRIS assessment.[53] This is accurate. The EPA IRIS assessment incorporated measured exposure data from NIOSH. EPA also states that "Macaluso et al. (2004)

[49] UCSF Program on Reproductive Health and the Environment. We Need the Best Science Free of Conflicts of Interest so Environmental Health Decision-Making Can Protect Public Health. https://prhe.ucsf.edu/sites/g/files/tkssra341/f/wysiwyg/UCSF%20PRHE%20EPA%20COI%20v1.pdf.
[50] National Research Council. Review of EPA's Integrated Risk Information System (IRIS) Process, p. 79. Washington, DC: National Academies Press; 2014.
[51] US EPA (2002). Integrated Risk Information System: 1,3-Butadiene. https://iris.epa.gov/static/pdfs/0139_summary.pdf.
[52] U.S. EPA (2024). Draft Human Health Hazard Assessment for 1,3-Butadiene, p. 67; Macaluso M, Larson R, Lynch J, Lipton S, Delzell E. Historical estimation of exposure to 1,3-butadiene, styrene, and dimethyldithiocarbamate among synthetic rubber workers. J Occup Environ Hyg. 2004 Jun;1(6):371-90. doi: 10.1080/15459620490452004. PMID: 15238328.
[53] U.S. EPA (2024). Draft Human Health Hazard Assessment for 1,3-Butadiene. p. 66.

revised the exposure estimates for 1,3-butadiene that incorporated additional information, including historical industrial hygiene surveys by NIOSH."[54] It is not accurate that Macaluso et al. 2004 incorporated historical industrial hygiene surveys by NIOSH. Macaluso only compared their modeled estimates of 1,3-butadiene levels to actual measured 1,3-butadiene levels collected by NIOSH. In almost all cases, Macaluso's modeled estimates are higher, sometimes quite significantly, than NIOSH measurements (see Table 1).

Table 1. *Data from Table VIII of Macaluso et al. 2004. Job Group-Specific 1,3-Butadiene (BD) Time-Weighted Average (TWA) Exposure Measurements from NIOSH Surveys and Estimates from Macaluso, 2004.*

	1,3-butadiene TWA (ppm)			
	NIOSH measurements		**Macaluso, 2004 modeled estimates**	
Job group	**Mean (SD)**	**Range**	**Mean**	**90% uncertainty interval**
Tank farm operator	2 (4)	0–24	13	2–113
Reactor operator	1.8 (4)	0–25	4	0–28
Recovery operator	No data			
Finishing operator	0.35 (1)	0–7	0	—
Maintenance, skilled	1.8 (7)	0–43	3.8	0–22
Maintenance, unskilled	No data			
Laboratory technician	3 (7)	0–38	5	0–58
All workers	1.1 (4)	0–43	2	2–2

There is little rationale presented in Macaluso et al. 2004 as to why modeled exposure estimate would be more reliable than the measurements taken by NIOSH.

There are clear financial conflicts of interest in both Macaluso et al. 2004 and Sathiakumar et al. 2021, the key studies EPA relied on for derivation of the new IUR: funding from the trade group that promotes chemical manufacturer's interests, the American Chemistry Council (formerly the Chemical Manufacturers' Association). The acknowledgement from Macaluso et al. 2004 says:

> This study was funded by the International Institute of Synthetic Rubber Producers and the Olefins Panel of the Chemical Manufacturers' Association.[55]

The funding statement from Sathiakumar et al. 2021 says:

> International Institute of Synthetic Rubber Producers, American Chemistry Council (Olefins Panel) and Styrene Information and Research Center. The sponsors were given an opportunity to provide comments on a draft of this paper. However, the contract between the University of Alabama at Birmingham and the sponsors stipulated that the

[54] U.S. EPA (2024). Draft Human Health Hazard Assessment for 1,3-Butadiene. p. 65.
[55] Macaluso M, Larson R, Lynch J, Lipton S, Delzell E. Historical estimation of exposure to 1,3-butadiene, styrene, and dimethyldithiocarbamate among synthetic rubber workers. J Occup Environ Hyg. 2004 Jun;1(6):371-90. doi: 10.1080/15459620490452004. PMID: 15238328.

> academic investigators should independently carry out the design, conduct and reporting of the study. Accordingly, the authors made all decisions about the contents of this paper.[56]

EPA should calculate the IUR for 1,3-butadiene with the original exposure information used in its 2002 IRIS assessment which are based on measured data rather than biased modeled estimates, and incorporate updated information for the cohort to include women as well as men and a longer timeline. While EPA states that "differences in key parameter estimates are due primarily to changes in exposure estimates"[57] it is important for it to demonstrate this transparently with the updated data set.

[56] Sathiakumar N, Bolaji BE, Brill I, Chen L, Tipre M, Leader M, Arora T, Delzell E. 1,3-Butadiene, styrene and lymphohaematopoietic cancers among North American synthetic rubber polymer workers: exposure-response analyses. Occup Environ Med. 2021 Dec;78(12):859-868. doi: 10.1136/oemed-2020-107197. Epub 2021 Jun 9. PMID: 34108254; PMCID: PMC8606437.

[57] U.S. EPA (2024). Draft Human Health Hazard Assessment for 1,3-Butadiene, p. 67.