

Using the Best Available Science to Assess Hazards and Risks of Industrial Chemicals Will Ensure Better Public Health Decisions

RECOMMENDATION

EPA must use the most scientifically up-to-date approaches to evaluate the hazards and risks of industrial chemicals and environmental pollutants that inform decision-making to protect public health.

ISSUE SUMMARY

Science has advanced on how industrial chemicals and environmental pollutants can adversely influence people's health, but methods for evaluating the evidence to use in policy decision-making has not kept pace with this science. There is well-supported evidence showing there are health risks from common every-day exposures to harmful chemicals due to factors that amplify the risks such as background exposures to multiple pollutants, pre-existing disease, genetic and social vulnerabilities, ongoing disease processes and susceptible life stages such as fetal and child development. Well-documented examples include particulate matter,¹ other forms of air pollution,² and lead.³ Further, these factors can influence risks such that risks can occur at any level.

Despite some methodological improvements, EPA's overall framework for using scientific evidence in hazard and risk assessment has stayed largely the same since the 1970s even though multiple authoritative review bodies and scientists have called for improved approaches.^{4,5,6,7,8,9} Of particular concern is that EPA continues to use an approach that assumes no risks below a level assumed to be "safe" for the general population, including sensitive groups, such as infants, children, pregnant women and marginalized communities. The lack of progress in updating approaches for identifying and evaluating environmental health risks is especially problematic considering the continued increases in chemical manufacture and use,¹⁰ and increasing trends in chronic disease,¹¹ particularly among the most vulnerable. EPA urgently needs to update its framework for incorporating current scientific knowledge into evidence-based science policies and practices to better reflect how industrial pollutants affect health with the goal of reducing harmful exposures and improving health and health equity.

PROPOSED ACTIONS

- 1. EPA should assume that all health effects, both cancer and noncancer, have some probability of occurring at any level of exposure and should quantify risks accordingly, unless proven otherwise, as recommended by authoritative scientific bodies.**
- 2. EPA should correct its definition of potentially exposed and susceptible populations, similar to the definition in the 2017 TSCA proposed risk evaluation framework rule.**
- 3. EPA should implement an improved default human variability adjustment factor of at least 30 fold for human risk assessment for all health endpoints to capture the wide range of factors contributing to differences in human response to chemical exposures including early life vulnerabilities, pre-existing health disparities, and common disease processes.**
- 4. U.S. regulatory authorities should have a consistent approach for characterizing exposures to environmental pollutants for rulemaking.**
- 5. EPA should consider classes of chemicals to accelerate risk management and avoid regrettable substitutions.** Specifically, EPA should consider, at a minimum, the 6 phthalates banned by CPSC under review as a group under TSCA Sec 26(c).

SUPPORTING EVIDENCE

EPA should assume that all health effects, both cancer and noncancer, have some probability of occurring at any level of exposure and should quantify risks accordingly, unless proven otherwise, as recommended by authoritative scientific bodies.

Human health risk assessment, and subsequent policy and regulatory decisions, can be substantially improved by using

quantitative methods to estimate health risks for all identified health effects. Currently, noncancer risk estimates are based on a bright line that does not specify a particular risk level (e.g., Reference Dose, RfD or concentration RfC) and assumes a threshold, below which there is no observed effect — but this does not mean that there is no/zero effect in the population.¹² Cancer risks on the other hand are expressed as probabilities (e.g., 1 in a million risk) based on the assumption that there is no exposure level of a chemical without some cancer risk.¹³ Treating noncancer risk estimates similarly to how cancer risk estimates are treated would better reflect current scientific understanding of health risks, provide more useful and actionable information to the public and decision-makers about environmental health risks, and allow policymakers to better estimate the health benefits of environmental regulations.

The most current scientific understanding shows that due to ongoing background exposures from multiple chemicals, common pre-existing diseases (e.g., diabetes), and factors that contribute to variability in response to chemical exposures (e.g., genetics and life stage vulnerabilities), risks will extend to all foreseeable population exposures.^{14,15,16,17} This was affirmed by the NAS in 2009,¹⁸ that recommended transitioning away from an assumption of “safety” and toward dose-response methods that quantify risk at doses within the experimental range as well as below it. Yet EPA continues to use scientifically unsupported “bright-line” methods which incorrectly imply a zero-risk level.

Methods are available and have been demonstrated as a way to implement an approach for estimating risk of all health effects that also includes factors to account for life stage vulnerability, coexposures to other pollutants, genetics, pre-existing conditions, and social factors including poverty and racism/discrimination.

We specifically recommend that EPA:

- Use established methods (e.g., probabilistic assessment) to quantify the level of risk for all identified health effects in parallel with RfD/point of departure calculation for every newly proposed noncancer benchmark (e.g., RfD) in an EPA IRIS assessment.^{19,20,21}
 - Estimating the exposure level to the population for different risk levels of all identified health effects (e.g., -1 in 100,000, 1 in 10,000, 1 in 1,000), in all EPA regulatory programs, including TSCA and SDWA, will give decision-makers better information about how exposures in the population translate into population risks for different health endpoints.
- Use established methods (e.g., probabilistic assessment) to quantify health risks from exposures and produce risk estimates under TSCA as part of risk evaluations. EPA should also use these risk calculations to quantify benefits under TSCA and better identify policy options to reduce exposures.²²

- Develop training materials that can explain to a variety of stakeholder audiences why these methods are useful, and how they can be implemented in a risk assessment and risk management framework.

EPA should correct its definition of potentially exposed and susceptible populations, similar to the definition in the 2017 TSCA proposed risk evaluation framework rule.

Current scientific understanding indicates that intrinsic factors (such as pre-existing diseases) and extrinsic factors (such as stress due to food insecurity and/or poverty) can increase susceptibility to environmental chemical exposure risks. Under the current law, EPA must consider impacts of chemicals on potentially susceptible subpopulations; however its current definition does not capture the reality of susceptibility.

Naming the factors that should be considered for susceptible populations is an important step to ensure consideration of these factors in hazard and risk assessment. In 2017 EPA proposed an expanded definition of susceptible populations as part of its TSCA risk evaluation framework rule, and EPA should incorporate a more robust definition into existing and proposed policies and guidelines.

An expanded version of EPA's 2017 proposed definition is below:

Potentially susceptible subpopulation means a group of individuals within the general population who, due to greater susceptibility may be at greater risk than the general population of adverse health effects from exposure to a chemical substance or mixture, including but not limited to infants, children, pregnant women, workers, or the elderly. Susceptibility can be due to both intrinsic (e.g., life stage, reproductive status, age, gender, genetic traits) and acquired (e.g., pre-existing disease, geography, socioeconomic, racism/discrimination, cultural, workplace) factors when identifying this population.

EPA should implement an improved default human variability adjustment factor of at least 30 fold for human risk assessment for all health endpoints to capture the wide range of factors contributing to differences in human response to chemical exposures including early life vulnerabilities, pre-existing health disparities, and common disease processes.

Development of risk-based estimates of harm from environmental chemical exposures are typically based on animal and human studies that do not necessarily reflect the full range of human variability/susceptibility that may occur across the population (**see Proposed Actions 1, 2, and 3**) Many authoritative scientific bodies currently use or have recommended adjustment factors which better account for human variability than EPA's current default adjustment factor of 10.

We recommend the following which could be easily adopted while enhancing the current data that informs these factors:

- A default adjustment factor for human variability in response to chemical exposures of at least 30, unless there are chemical-specific data to the contrary, should be applied to all health endpoints.^{23,24,25} This would align with using the same methodological approach for all health endpoint risks.
 - EPA's current assessment method for cancer does not adjust for individual variability in cancer susceptibility.
- The International Programme on Chemical Safety (IPCS) report identified up to a 14-fold range of human variability in response to chemical exposures when human toxicokinetic and toxicodynamic (TK and TD) data was combined probabilistically.²⁶

U.S. regulatory authorities should have a consistent approach for characterizing exposures to environmental pollutants for rulemaking.

Federal agencies do not take the same approach in considering what extent of the population is exposed to industrial chemicals and/or pollution for the purposes of rulemakings. For example, EPA's Pesticide Office often calculates exposures for at least the 99th percentile of the population, while other offices only account for exposures of the 95th percentile of the population.

Considering just the 95th percentile of the population potentially leaves a large portion of the population — 16 million people²⁷ — at higher exposure levels and thus unprotected. In order to adequately protect the population, policy and regulatory exposure rules should all consider the same percentile of the population and should encompass at least the 99th percentile, similar to the Pesticide office. Those that are left unprotected by the exposure estimates (the top .1 percentile) should be robustly characterized with regard to susceptibility factors such as their geography and demographics in order to ensure transparency around who is and is not potentially protected.

EPA should consider classes of chemicals to accelerate risk management and avoid regrettable substitutions. Specifically, EPA should consider, at a minimum, the 6 phthalates banned by CPSC under review as a group under TSCA Sec 26(c).

Chemicals are usually assessed for their risk and addressed through public policy via a chemical-by-chemical approach. While this can be useful, it is also time- and resource-intensive. Chemicals that are more studied and identified as hazardous may be replaced with less well-studied chemicals, under the assumption that little data indicates no risk.²⁸ This can result in substitution of hazardous chemicals with chemicals that have similar structure and function (e.g., bisphenols), may be relatively untested, and can be as or more harmful than the original chemical; otherwise known as a regrettable

substitution.²⁹ Further, assessing chemicals one at a time can underestimate hazard and risks as scientific evidence shows that multiple chemical exposures acting on the same health endpoint can result in increased risk compared to individual chemical exposures. For example, assessing phthalates individually will result in underestimation of risk because multiple phthalates can act together to affect the same health endpoint (male reproductive development), and thus there is increased risk from cumulative exposures. Additionally, assessing cumulative exposures better reflects exposures experienced by the public, providing a more accurate estimate of risk.^{30,31,32}

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