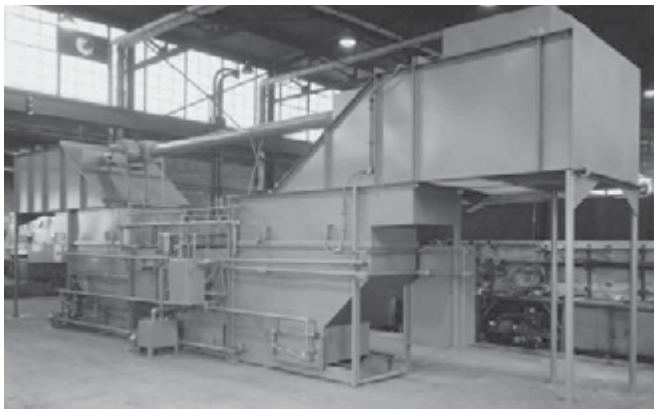
REGULATORY UPDATE

EPA issues draft risk evaluation for trichloroethylene: Implications for possible changes to risk management

Trichloroethylene (TCE) periodically undergoes risk evaluation under the Toxic Substances Control Act (TSCA). In February 2020, EPA released a Draft Risk Evaluation for TCE[1] which could have implications on how TCE exposure and risk are managed in the future. Specifically, there may be less need for emergency regulatory responses and a greater need for a more pragmatic approach to risk management decision-making for TCE.

TCE is often identified in site characterization studies due to its long history of industrial use and its persistence in environmental media. TCE can also confound vapor intrusion and indoor air quality evaluations, in part due to its current use in many commercial and consumer products, and also due to its low toxicity thresholds.

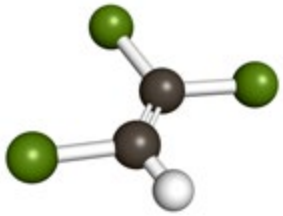


CURRENT RISK MANAGEMENT APPROACH AND CHALLENGES FOR TCE

Risk management policy for TCE has long been based on consideration of carcinogenic health effects. However, following the U.S. Environmental Protection Agency (EPA) updated Toxicological Review for TCE in 2011[2], regulatory agencies expanded their scope to manage risks for TCE exposure in consideration of non-cancer health effects – in particular a specific developmental health effects endpoint identified as congenital heart defects (CHD).

Consideration of developmental health effects introduced a shift in the risk management paradigm for TCE. This is because the threshold concentration for development effects is only slightly higher than the concentration protective for a $1x10^{-6}$ cancer risk (meaning that risk management decisions could be driven by non-cancer risks), and because EPA identified CHD as being a serious health effect associated with a short-term exposure. However, EPA did not satisfactorily define "short-term exposure," leaving individual regulatory agencies to decipher how to use environmental sampling data to evaluate health risk and inform risk management decisions. This led to divergent, and in some cases extreme, state and EPA regulatory responses that included evacuation of occupied buildings when TCE was measured in a single sampling event at a concentration only marginally above the developmental effect threshold concentration. In some cases[3], the difference in TCE indoor air screening levels at which no response is needed (e.g.,

3 µg/m^3) and rapid response is required (e.g., 4 µg/m^3) is as little as 1 µg/m^3, which is within the range of analytical uncertainty for conventional indoor testing methodologies.



In addition to issues associated with interpreting "short-term" exposure, many experts have criticized the body of evidence that EPA relied upon to conclude that TCE is a concern for CHD[4]. In particular, technical reviewers have noted substantial limitations with the single study that EPA used to derive dose-response information for CHD (known as the *Johnson et al. [2003] study*), and that the body of evidence does not support an association between TCE exposure in humans and CHD at environmentally relevant exposures[5]. In response, one state has indicated that "this approach has been highly controversial (the results obtained in the original study indicating increased incidence of fetal cardiac malformations have not been replicated despite several attempts to do so) and has proven to be very problematic as a policy"[6]. Despite criticism, EPA has taken the position that in the absence of convincing information to the contrary, TCE-induced cardiac malformations in rat fetuses is considered valid and relevant to humans[7].

NEW EVIDENCE FURTHER QUESTIONS RELEVANCY OF DEVELOPMENTAL EFFECTS RELATED TO TCE

The TSCA Draft Risk Evaluation considers new evidence from a study published in 2019 by *DeSesso et al.* that employed the same methodology as the *Johnson et al. (2003) study* but failed to find a statistically significant relationship between TCE exposure and CHD[8]. The Draft Risk Evaluation included a weight of evidence evaluation in which EPA concluded that CHD remains a viable endpoint to consider in risk characterization; however, CHD should not be used to inform risk management decisions. Specifically, EPA cited their charge under TSCA to use the best available science, the extent of independent verification, and the weight of scientific evidence, and noted that public health benefits most when EPA relies upon the highest quality information for which the agency has the highest confidence.

IMPLICATIONS FOR FUTURE RISK MANAGEMENT APPROACH FOR TCE

The Draft Risk Evaluation provides an EPA position that risk management decisions for TCE should not be based on developmental effects related to CHD. Significantly, the Draft Risk Evaluation and *DeSesso et al.* study appear to provide "convincing information to the contrary" to support a position that TCE-induced CHD is not valid and relevant to humans, at least to the extent that it is used to guide risk management decisions.

If finalized with the same conclusion, the TSCA Risk Evaluation could have implications for managing TCE exposures and risks in the future, particularly when evaluating the vapor intrusion pathway. Future risk management decisions could include reducing the focus on extreme regulatory responses (i.e., building evacuation) that are based on concerns about developmental effects, using longer-term monitoring data to inform decisions, and a return to more practical risk management decision making for TCE.

If you have questions about TCE regulations, risk assessment, or vapor intrusion challenges, please contact:

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References

[1] See regulations.gov for docket EPA-HQ-OPPT-2019-0500.

[2] Toxicological review of trichloroethylene (CASRN 79-01-6) in support of summary information on the Integrated Risk Information System (IRIS). EPA/635/R-09/011F.

[3] See for example "NJDEP Master Table, Generic Vapor Intrusion Screening Levels" (www.nj.gov/dep/srp/guidance/vaporintrusion/vig_tables.pdf).

[4] See for example: Hardin BD et al. 2005. Trichloroethylene and dichloroethylene: A critical review of teratogenicity. Birth Defects Res A Clin Mol Teratol 73(12):931-955. 10.1002/bdra.20192.

[5] Watson, RE, et al. Trichloroethylene-contaminated drinking water and congenital heart defects: a critical analysis of the literature, Repro. Toxicol. 21: 117-47 (2006).

[6] Indiana Department of Environmental Management. "2016 Screening Levels Table Now Available Clarification Regarding Application of Trichloroethene Indoor Air Screening Levels" March 7, 2016.

[7] Toxicological review of trichloroethylene (CASRN 79-01-6) in support of summary information on the Integrated Risk Information System (IRIS). EPA/635/R-09/011F.

[8] DeSesso JM et al. Trichloroethylene in drinking water throughout gestation did not produce congenital heart defects in Sprague Dawley rats. Birth Defects Res 111(16):1217-1233 (2019)