


Exhibit 147

**Supplemental Report of
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**Camp Lejeune Justice Act
United States Federal Court
Eastern District of North Carolina**

Prepared for
United States Department of Justice
950 Pennsylvania Avenue NW
Washington, DC 20530



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February 28, 2025**

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This Supplemental Report is in response to Dr. Bird's Supplemental Report served on February 4, 2025. It is also restricted to the diseases identified in Track 1.

My opinions herein are derived from my education and experience including my knowledge of materials included in my bibliography and other materials with which I am familiar from previous endeavors. These opinions are reached to a reasonable degree of scientific certainty. My opinions in this case were arrived at using the same methodology and scientific rigor that I have applied throughout my academic, professional, and consulting career performing research, authoring peer-reviewed manuscripts for publication, and assessing the scientific merit of ideas and works of other scientists through the peer review process. My approach included reviewing robust and rigorous peer-reviewed literature, regulatory standards, and other relevant literature and resources, assessing the strength of each of the sources; and weighing the limitations of the sources. I reserve the right to develop additional opinions as a need for them may arise and modify these opinions on the basis of emerging information.

1. Dr. Bird Improperly Relied on a Press Release and Disregarded the Underlying Scientific Determination

Dr. Bird has been misled by statements made by EPA's Press Office (EPA Press Office, 2024). A scientist would not rely upon a press release to reach scientific conclusions. Here, there is no evidence that EPA's Press Release was reviewed by or endorsed by scientists knowledgeable in toxicology or medicine. Specifically, the statement in the press release regarding the diseases that trichloroethylene (TCE) is "known to cause" seems to be unique to the Press Release. Regarding TCE, neither the TSCA 2020 Final Risk Evaluation for Trichloroethylene (EPA, 2020) nor the Final Revised Determination of Unreasonable Risk (EPA, 2023) contain the phrase "known to cause." It is important to consider the actual language contained in EPA's scientific documents, including the TSCA final rule for trichloroethylene (89 Fed. Reg. 102568-102635), TSCA Final Rule for PCE (89 Fed. Reg. 103560-103616), EPA's TSCA Risk Evaluation for Trichloroethylene (EPA, 2020), EPA's IRIS assessment for trichloroethylene (EPA, 2011), and EPA's IRIS assessment for tetrachloroethylene (EPA, 2012).

Dr. Bird's blind reliance on the Press Release ignores the appropriate methodology for evaluating causation. One of the issues important in evaluating the applicability of an association between exposure and response is assessing the relationship between (1) the exposure of the subjects, individuals, or animals in which the effect has been reliably associated with exposure and (2) the exposure of the individuals in question. Dr. Bird failed to explain the relevance of the TCE ban with respect to evaluating exposure and the response and its applicability or inapplicability to exposed individuals in this case.

2. Dr. Bird Improperly Assumed that the Exposures Evaluated by TSCA are the Types of Exposures at Camp Lejeune

Dr. Bird indicated that the Toxic Substances Control Act (TSCA) Office's ban of TCE and tetrachloroethylene (PCE) is relevant to supporting his opinions regarding risk. But, first and foremost, it must be clearly understood that the exposures evaluated by TSCA and the populations to which they pertained do not reflect the exposures experienced at Camp Lejeune. Therefore, the risk estimates that supported the ban of TCE do not extend to Camp Lejeune exposures.

2.1 The TSCA Ban of TCE Does Not Apply to Drinking Water

TSCA's evaluation of TCE specifically excluded exposure via drinking water. The TSCA ban is especially clear that the (1) estimates of exposure from the specified conditions of use, (2) resulting risk estimates, and (3) conclusions developed **do not apply to drinking water**. With relation to the analysis of TCE under TSCA, EPA (2020) wrote, “[h]ence, because the drinking water exposure pathway for trichloroethylene is currently addressed in the [Safe Drinking Water Act] regulatory analytical process for public water systems, EPA is not evaluating exposures to the general population from the drinking water exposure pathway in the Risk Evaluation for trichloroethylene under TSCA.” This is further clarified in the Final Rule (89 Fed. Reg. 102568-102635), discussed below.

Second, it is important to consider that TSCA did not reach the decision of an unreasonable risk on the basis of a single exposure scenario. TSCA evaluated 54 conditions of use, which did not include the potential drinking water exposures at Camp Lejeune. The decision was reached on the totality of findings of both non-cancer and cancer risk from the specific TSCA conditions of use and durations of exposure. Dr. Bird (citing 89 Fed. Reg. 102575) wrote, “‘TCE presents an unreasonable risk of injury to human health under the conditions of use based on acute and chronic non-cancer and cancer risks.’ Those are precisely the kinds of exposures experienced by the Marines and civilians who were stationed at Camp Lejeune during the time period applicable.” This is just wrong. EPA's TSCA risk evaluation (EPA, 2020) evaluated 54 conditions of use, none of which are the “kinds of exposures” experienced at Camp Lejeune, namely exposures via drinking water¹ or repeated exposures of anyone except workers and occupational non-users.

Specifically, for cancer risk, TSCA did not evaluate consumers, only workers aged 16 and older and occupational bystanders² exposed for a duration of 40 years (EPA, 2014, 2020). The 40-year exposure duration assumed by TSCA for occupational **cancer** risk is not applicable for exposures identified for Camp Lejeune. For consumers, TSCA only evaluated the **non-cancer** risk of an acute (single day) exposure, explaining, “[c]hronic exposure was not considered relevant to consumers based on expected use patterns.” Therefore, the risks evaluated through TSCA and supporting its TCE ban do not apply to the exposure scenarios evaluated for Camp Lejeune Marines and residents.

It will be made clear in this Supplemental Report that the TSCA ban of TCE, categorically does not apply to drinking water exposures like those that occurred at Camp Lejeune. Inasmuch as Dr. Bird's opinions address a drinking water-based exposure at Camp Lejeune, the TSCA ban of TCE cannot support his conclusions.

2.2. TSCA Makes Clear Why the Wastewater Concentration Does Not Apply to Drinking Water

EPA/OCSPP³ (EPA, 2020) has made it clear, in part through the evaluation of 54 specific conditions of use, that its exposure estimates, risk assessment, and conclusions do not apply to exposure from

¹ The 54 conditions of use examined are identified in Tables 1-3 and 1-4 of the TSCA risk evaluation (EPA, 2020).

² EPA (2020) retains the term “worker,” but uses the term “Occupational Non-Users” to distinguish employees not directly using TCE in the workplace.

³ EPA's Office of Chemical Safety and Pollution Prevention (OSCPP) is the parent organization of the Office of Pollution Prevention and Toxics, which is directly in charge of TSCA. Other EPA Offices at the same level as OCSPP

contaminated drinking water (see Section 13 of my Expert Report). Further, while TSCA did develop an estimate of an allowable concentration of TCE in water, that was wastewater, not drinking water.

Dr. Bird discussed regulation of wastewater concentrations without providing proper context. The Final Rule (89 Fed. Reg. 102568-102635) was clear that this concentration applied to wastewater, and that the relevant population for that condition of use was workers in Publicly Owned Treatment Works (POTW). These workers may be exposed to contaminants volatilizing from a much higher volume of wastewater than the typical U.S. resident is exposed to through much smaller volumes of drinking water. While a typical U.S. citizen uses approximately 82 gallons of drinking water per day,⁴ a POTW facility, on average, processes approximately 2.26 million gallons of wastewater per day.⁵ TSCA's Final Rule (89 Fed. Reg. 102568) followed the Guidance to Protect POTW Workers from Toxic and Reactive Gases and Vapors,⁶ thus distinguishing this potentially exposed population from the general public, noting, in particular, POTW workers' "occupational exposure profiles." TSCA did enact a screening level concentration of 0.00284 mg/L in wastewater, and clarified its application to wastewater only. Dr. Bird did not provide any evidence that Marines or civilians at Camp Lejeune were exposed to TCE under the occupational conditions at any publicly owned wastewater treatment facility.

In fact, EPA specifically distinguished the applicability of this TSCA standard from MCL values, which are developed and implemented pursuant to the Safe Drinking Water Act (SDWA) for application to drinking water. EPA further explains: "This screening level is a level specific to TSCA, to regulate unreasonable risk to workers performing wastewater disposal that are exposed to TCE. This differs from MCLs which regulate public water systems under a different federal statute and do not address exposures to TCE through wastewater" [89 Fed. Reg. 102583]. Understanding the context of TSCA's evaluation is necessary.

2.3 The Findings of Health Impacts at Woburn, Massachusetts Do Not Translate to Exposures at Camp Lejeune

Dr. Bird improperly attempted to place the Camp Lejeune contamination in the context of contamination at Woburn, Massachusetts, by referring to the Press Release (EPA Press Office, 2024). Many of the studies of the risks at Woburn cited by Dr. Bird evaluated childhood leukemia, and it should be noted that TSCA did not evaluate the cancer risk to individuals younger than 16 years of age. Dr. Bird described the contamination at Woburn as "a site where TCE and PCE contamination led to markedly elevated cancer occurrence" without acknowledging the presence of the many other chemicals that also

include the Office of Water (OW) and the Office of Research and Development (ORD). OW uses ORD's risk assessments as the basis for Safe Drinking Water Act regulations and OCSPP uses ORD risk assessments as the basis for TSCA regulations.

⁴ <https://www.epa.gov/watersense/statistics-and-facts>

⁵ <https://css.umich.edu/publications/factsheets/water/us-wastewater-treatment-factsheet#:~:text=Treatment%20of%20Municipal%20Wastewater&text=Almost%2015%2C000%20POTWs%20treat%20and,of%20wastewater%20into%20U.S.%20waterways.>

⁶ Guidance to Protect POTW Workers from Toxic and Reactive Gases and Vapors. US Environmental Protection Agency, Office of Water, Washington, D.C., EPA 812-B-92-001, June 1992.

contaminated Woburn's drinking water. Despite Dr. Bird's references to Woburn, none of the underlying TSCA risk evaluations cite to specific studies of the Woburn cohort.

Multiple chemicals beyond TCE and PCE contaminated the water in Woburn and so Dr Bird's association of only TCE or PCE with the health effects identified at Woburn are suspect. For example, Parker and Rosen (1981)⁷ studied the many chemicals present at in Woburn and described a higher-than-expected incidence of childhood leukemia and a lower than expected incidence of bladder cancer in Woburn.⁸ Parker and Rosen (1981) reported, "[n]o information is available indicating what, if any, contaminants existed prior to May 1979," and identified several volatile organic chemicals in water from wells G and H including chloroform, trichloroethylene, tetrachloroethylene, 1,1,1-trichloroethane, dibromochloromethane, trichlorotrifluoroethane, dichloroethylene, and dichlorotrifluoroethane; and arsenic. Other industrial waste near the cluster of reported leukemia cases contained arsenic, lead, chromium, nickel, zinc, and other metals. The presence of these other chemicals cannot be ignored when discussing the results of studies of Woburn.

Additionally, other studies evaluated exposure to drinking water and recognized the presence of numerous chemicals in Woburn. Cutler et al. (1986)⁹ identified leather tanneries as an important industry in the town, historically, and indicated the finding of pits of buried hides as a source of the foul odor of drinking water. Regarding the tanning industry chemicals that may have accompanied the foul odor from buried hides into drinking water, Rastogi et al. (2007) reviewed occupational cancer effects of occupational exposures in tanneries. Those authors reported, "[t]annery workers have been known from previous studies to have the potential for exposure to numerous known or suspected occupational carcinogens including hexavalent chromium salts, arsenic, organic solvents (benzene, formaldehyde, butyl acetate, ethanol, acetaacetate, toluene and acetone)." In light of the lack of data describing previous drinking water contamination in Woburn, Cutler et al. (1986) wrote, "[t]he lack of environmental data for earlier periods is a major obstacle in establishing a link between specific environmental contaminants and the occurrence of leukemia in Woburn." Thus, it is not possible to attribute health conditions in Woburn to any specific contaminant in the Woburn drinking water.¹⁰ This highlights the significant uncertainty accompanying the extrapolation of effects from Woburn to Camp Lejeune.

⁷ Dr. Bird cited to Parker and Rosen (1981) in his Reports on Kidney Cancer and on Hematopoietic Cancers (December 9, 2024).

⁸ If the findings of Parker and Rosen are to be considered relevant, then the inverse relationship between the increased incidence of renal cancer and the decreased incidence of bladder should be reconciled with the proposed similar mechanism of action (Bird, Supplemental Report). It cannot. Further, EPA (2011,) characterized bladder cancer as a high dose effect observed in some studies, and went so far as to write, "[t]hese studies do not provide evidence against an association between TCE exposure and bladder cancer." EPA (2020) wrote, "[t]here is some evidence of association for bladder or urothelial cancer and high cumulative TCE exposure."

⁹ Dr. Bird cited to Cutler et al. (1986) in his Report on Hematopoietic Cancers (December 9, 2024).

¹⁰ Aspects of these and other epidemiological investigations, including those addressing contamination at Woburn, MA, can be found in the expert reports of Dr. Goodman.

3. Point Estimates Like the Margin of Exposure Values Do Not Quantify Risk

Dr. Bird relied on the Final Rule without consideration of the fact that the margin of exposure (MOE) values do not quantify risk. The MOE-based approach used by TSCA (EPA, 2020) to determine an unreasonable risk must also be evaluated. Generically speaking, an MOE is the ratio of an exposure to the point of departure (POD) value for an effect used in risk assessment. TSCA (EPA, 2020) adopted the uncertainty factor value of 30 from the EPA (2011) TCE risk assessment as the value for the benchmark MOE. TSCA developed MOE values for conditions of use by comparing estimated exposures from conditions of use to the risk assessment POD, and then compared these MOE values to the benchmark MOE value of 30 to determine the acceptability of a chronic TCE exposure.¹¹

Dose response is a central tenet of toxicology - it is generally accepted that as the dose increases, the response increases. This concept is brought into risk assessment through the downward extrapolation of POD values to develop reference dose and reference concentration values that are below those thought to be adverse, thus further reducing the likelihood of adverse outcomes. In the context of the MOE, as the exposure increases, the MOE decreases. For TSCA, when MOE values for autoimmune effects decline below 30, risk is deemed unacceptable.

However, EPA has established that it is inappropriate to use reference concentrations to gauge the risk associated with an exposure that exceeds the reference concentration (Section 10.3 of my Expert Report). The TSCA approach of gauging the acceptability of risk by comparing the MOE of an exposure to a benchmark MOE representing the uncertainty factor used in the reference concentration is a transparent skirting of this prohibition. Here, TSCA has in effect implemented a policy that interprets any exceedance of a reference value as an unacceptable risk. Such a policy runs directly counter to EPA guidance that states “[r]eference values are not predictive risk values; they provide no information about risks at higher exposure levels.” (EPA, 2022).

Further, the hazard quotient is the ratio of an exposure to an acceptable level, which is often a reference concentration. EPA has also cautioned that hazard quotients and hazard index values¹² should not be interpreted as numerical estimates of risk (Sections 10.7.1 to 10.7.3 of my Expert Report). EPA wrote: “The Hazard Index does not define dose-response relationships, and its numerical value should not be construed to be a direct estimate of risk.” (EPA, 1989). Neither do HQ values define dose-response relationships, which is necessary to understand the potential increase of an effect with increasing dose or exposure. These topics are discussed in more detail in sections 8.9.1 and 10.7.3 of my Expert Report.

3.1 The Margin of Exposure for Autoimmune Effects Should Not be the Basis for Unacceptable Risk

TSCA (EPA, 2020) identified the benchmark MOE of 30 to gauge the risk of autoimmune effects (EPA, 2011). In its determination of unreasonable risk, TSCA (EPA, 2023), indicated that chronic non-cancer risk evaluation was based on autoimmune effects. However, TSCA (EPA, 2020) based the determination of the unacceptability of risk on MOE values for autoimmunity that are “based on an early biomarker

¹¹ TSCA indicates that chronic risk is estimated assuming a 40-year duration of exposure.

¹² A hazard index value is the sum of hazard quotient values for all chemicals in a mixture exposure.

that *may not be adverse itself.*" (emphasis added). In other words, the effect protected against by the benchmark MOE may not be adverse.

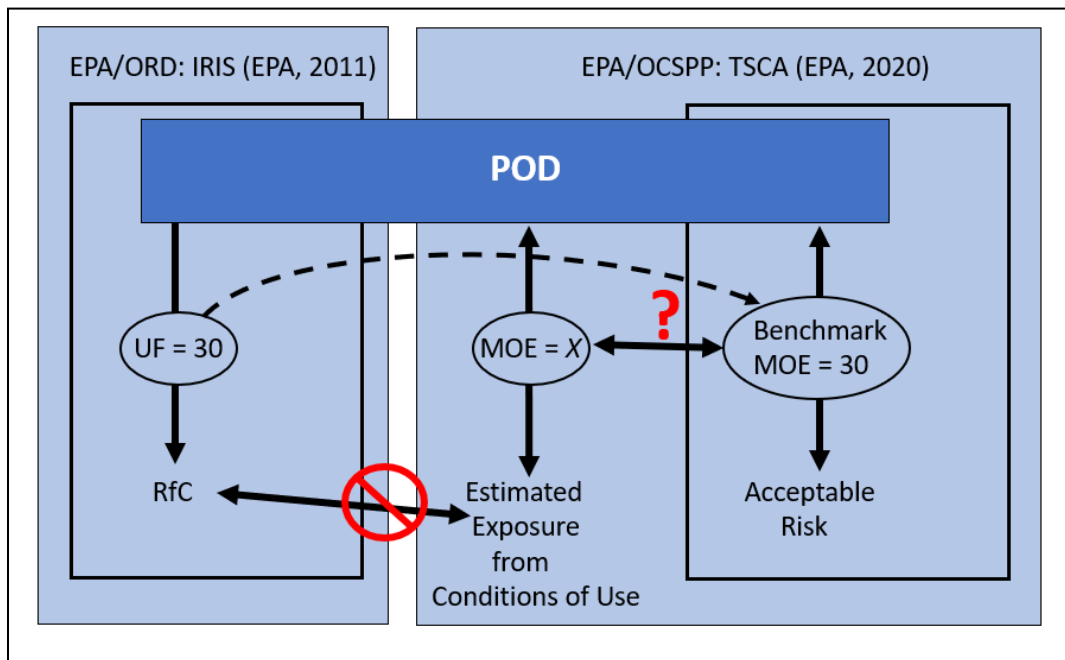
Additionally, in developing an MOE value based on the POD for autoimmune effects, TSCA assumes that the magnitude of the effect will increase with increasing exposure. However, it does not. In discussing the finding of autoantibodies in mice from the study by Keil et al. (2009), TSCA (EPA, 2020) wrote, "[w]hile there was not a consistent dose-response for autoantibodies (responses are similar or even decreased at the higher dose), this inconsistent dose response is in agreement with results from autoimmune-prone MRL +/+ mice in (Griffin et al. 2000)." Without a consistent dose response, estimating the risk from unstudied doses in an unstudied species becomes even more uncertain.

Additionally, TSCA (EPA, 2020) employed the POD value determined by EPA/ORD used to develop the inhalation reference concentration (RfC) in the IRIS assessment for TCE (EPA, 2011). However, EPA/OSCP failed to acknowledge that EPA/ORD clearly indicated that an exceedance of a reference value cannot be used to infer risk (see section 10.3 of my Expert Report).¹³ There are two quantitative values that define a reference value: the POD and the uncertainty factor (UF) values. The RfC is determined by dividing the POD by the UF, shown in the left-most box of Figure 1, below. And comparing an exposure, irrespective of the motive for which it was estimated, to an RfC to quantify the risk of an exceedance exposure is prohibited (shown with the "prohibited" symbol in Figure 1).

However, as shown in the dark blue box in Figure 1, below, TSCA used the POD value from the IRIS risk assessment for TCE as one of the quantitative values when determining the unacceptability of risk. TSCA also used the benchmark margin of exposure of 30, adopted from the uncertainty factor value of 30 from the IRIS assessment for TCE (EPA, 2011), shown by the dashed arrow. As shown in the center of Figure 1, TSCA estimated exposures from conditions of use and determined the MOE for each by comparing the estimated exposure for conditions of use to the POD taken from the IRIS assessment for TCE (). TSCA then compared the MOE for the conditions of use to the benchmark MOE value of 30, shown by the solid arrow and question mark in Figure 1. TSCA reached a decision of "unreasonable risk" if the MOE value for the condition of use was lower than the benchmark MOE of 30. In other words, if the distance between the estimated exposure was closer to the POD (right-most panel) than the distance between the RfC and the POD (left-most panel), TSCA interpreted the exposure from the condition of use to represent an unacceptable risk. Practically speaking, the difference between the TSCA approach just described and the comparison prohibited by EPA/ORD is minimal, if there is any difference at all.

¹³ For a discussion of how a POD does not inform estimation of a response at other doses or exposures, see Section 8.9.1. of my Expert Report.

Figure 1. EPA (2020) failed to acknowledge that using their MOE-based approach is the same as using a reference concentration to quantify risk.



RfC = inhalation Reference Concentration; UF = uncertainty Factor; MOE = margin of exposure

4. EPA's 2011 TCE Inhalation Cancer Slope Factor is Unreliable

Notably, the TSCA Risk Evaluation (EPA, 2020) did not evaluate cancer risk for any population subgroups other than workers and occupational non-users. As discussed, the exposure scenarios for the studied conditions of use do not represent “precisely the kinds of exposures experienced by the Marines and civilians who were stationed at Camp Lejeune,” as improperly indicated by Dr. Bird. This is another reason why the risk estimates that supported the ban of TCE do not extend to Camp Lejeune exposures.

TSCA (EPA, 2020) evaluated TCE cancer risk based on the inhalation cancer slope factor from EPA's IRIS assessment for TCE (EPA, 2011). The inhalation cancer slope factor for TCE is based on findings of renal cell carcinoma from highly exposed workers from Charbotel et al. (2006), and increased four-fold to additionally cover liver/biliary tract cancer and Non-Hodgkin's lymphoma because the data for the latter two cancers were not strong enough to serve as the basis for specific slope factors.¹⁴ Charbotel et al. (2006) questioned the relevance of their findings to lower levels of exposure when they wrote, “[a]lthough this study shows a possible link between high levels of exposure to TCE and increased risk of RCC, further epidemiological studies are necessary to assess the effect of lower levels of exposure.”

¹⁴ As I explained in my report when using these upper bound cancer potency factors to estimate risks, that the uncertainties included in the factors indicate that the “true risk may be as low as zero” (see Section 8.1.2. of my Expert Report).

My Expert Report identified the estimation of TCE kidney effects by both EPA and the National Research Council (NRC) as related to high exposures (Section 8.11.1.). Only for renal cell carcinoma, a kind of kidney cancer, did EPA (2011) assume that a DNA-active mode of action was possible. EPA described kidney effects for TCE as high-dose effects; thus, formation of kidney effects at lower doses is uncertain. The only “major uncertainty” called out by EPA (2011) in the entire TCE risk assessment is identified in the inhalation unit risk estimate for cancer, and that is “in the extrapolation from occupational exposures to lower environmental exposures.” (EPA, 2011). EPA/OCSP (EPA, 2020) also repeated this passage from EPA (2011), without attribution, was the only “major uncertainty” acknowledged in the EPA (2020) Risk Evaluation for Trichloroethylene.¹⁵ EPA (2011) also discusses association of renal cell carcinoma with TCE exposure relating information from the occupational study (Charbotel, 2006), which EPA says showed that only 37 of the 86 renal cell carcinoma cases were from individuals actually exposed to TCE. EPA (2011) also identified uncertainties in the cancer dose-response assessment in Section 6.2.2.4. EPA (2011) did not conclude that TCE was “known to cause” kidney cancer, or any other effect.

Despite these cautions presented in the EPA’s 2011 Toxicological Review for Trichloroethylene (EPA, 2011) and repeated by TSCA (EPA, 2020), EPA (2023) has determined under the conditions of use that were evaluated in the TSCA risk Evaluations (EPA, 2014, 2020), that TCE presents an unreasonable risk of cancer. EPA’s determination should not be extended beyond the conditions of use evaluated by TSCA.

5. EPA Did Not Issue a Complete Ban of PCE

Unlike the ban of TCE “prohibiting the manufacture (including import), processing, and distribution in commerce of TCE for all uses” (89 Fed Reg 102574), “EPA is not finalizing a complete ban on PCE.” (89 Fed. Reg. 103563). Instead, certain conditions of PCE use identified by EPA (e.g., vapor degreasing) will continue¹⁶ through a ten-year phase-out period. Other conditions of use continuing through a phase out period (89 Fed. Reg. 103567) include the use of PCE in some manufacturing processes and in the dry-cleaning industry, where “EPA estimates that 6,000 dry cleaners still use PCE.” (89 Fed. Reg. 103563).¹⁷ Other uses including using PCE as a processing aid in pesticide, fertilizer, and other agricultural chemical manufacturing are not prohibited. This contrasts sharply with the approach taken by EPA toward TCE.

In its TSCA evaluation, EPA looks to the appropriate risk values for the chemical. There are appreciable differences in the level of confidence placed in the identification of risk values for non-cancer and cancer endpoints for TCE and PCE. This imparts a different level of confidence in risk estimates developed, as well. The principal studies used by EPA (2011) to assess the **non-cancer** inhalation risk for TCE was

¹⁵ EPA (2011) also identifies many more areas of uncertainty for both cancer and non-cancer effects.

¹⁶ Under this approach a Workplace Chemical Protection Plan will be established for PCE to ensure worker safety. EPA writes, “[t]he rule bans many uses of PCE and establishes workplace controls, including a Workplace Chemical Protection Program and prescriptive controls, for those uses not prohibited.” <https://www.epa.gov/assessing-and-managing-chemicals-under-tsca/risk-management-perchloroethylene-pce#>

¹⁷ Interestingly, dry cleaners were the subject in the studies that identified and quantified the neurotoxic effect of PCE that served to identify the POD for quantification of risk in EPA’s IRIS program (EPA, 2012) as well as in EPA’s TSCA Risk Evaluation (EPA, 2020b).

conducted in animals exposed by the oral route,¹⁸ while the corresponding **non-cancer** inhalation risk study for PCE was conducted in humans exposed by the inhalation route.¹⁹ EPA (2011) placed “high” confidence in the inhalation RfC for TCE. In contrast, EPA (2012) placed only “medium” confidence in the inhalation RfC for PCE, citing shortcomings in the study design and its resulting data that describe the effect on color vision evaluated.

The study used to estimate the inhalation **cancer** potency of TCE was conducted using humans occupationally exposed by the inhalation route, avoiding the uncertainty in extrapolating risk from the oral route to the inhalation route, and from animals to humans. EPA (2011) characterized TCE as being “carcinogenic to humans.” In contrast, the study used to estimate inhalation cancer potency of PCE was conducted in animals exposed by the oral route, adding two more extrapolation steps than were necessary in developing TCE’s inhalation cancer potency estimate. EPA (2012) characterized PCE as “likely to be carcinogenic in humans by all routes of exposure,” rather than “carcinogenic to humans.” Qualifying the conclusion with “likely” and other issues in the mode of analysis for PCE indicate that cancer risk evaluations are more uncertain for PCE than for TCE. Therefore, EPA’s decision regarding TCE cannot be imparted to PCE.

6. References

The following list of references supplements the list of references produced with or cited within my Report on February 5, 2025. This also includes references listed in this Supplement Report.

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¹⁸ When an acceptable inhalation study is not available, and when an oral study is acceptable, EPA uses physiologically based pharmacokinetic modeling to extrapolate findings from the oral to the inhalation risk. This was done to derive the inhalation RfC for TCE.

¹⁹ It should be understood that the endpoint considered for neurotoxicity for PCE was a decrement in color vision. The only neurologic condition evaluated in Track 1 is Parkinson’s Disease. I am unaware of information describing the similarity of insult, specific tissue region affected or mechanism of action between color vision loss and Parkinson’s disease. For these and other reasons, loss of color vision seems an unacceptable surrogate for Parkinson’s Disease in the context of risk assessment.

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