

Exhibit 124

SUPPLEMENTAL REPORT TO 12/08/2024 (SUBMITTED ON 12/09/2024) GENERAL CAUSATION REPORT: TETRACHLOROETHYLENE AND PD

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1. IMPACT OF RECENT EPA PRESS RELEASE AND RULINGS ON THE 12/08/2024 GENERAL CAUSATION REPORT

1.1 12/09/2024 EPA press release.

On 12/09/2024, the United States Environmental Protection Agency (USEPA) issued a press release entitled “Biden-Harris Administration Announces Latest Actions under Nation’s Chemical Safety Law to Protect People from Cancer-Causing Chemicals Trichloroethylene and Perchloroethylene”¹. The intent of this press release was to convey information on forthcoming final rulings on trichloroethylene (TCE) and tetrachloroethylene (also known as perchloroethylene, PCE), with a subtitle of *“Final EPA rules ban all uses of TCE, all consumer uses and many commercial uses of PCE, require worker protections for all remaining uses under the Toxic Substances Control Act”*.

The press release is EPA’s own assessment of the impact on public health of the rulings:

[“TCE is an extremely toxic chemical known to cause liver cancer, kidney cancer, and non-Hodgkin’s lymphoma. TCE also causes damage to the central nervous system, liver, kidneys, immune system, reproductive organs, and fetal heart defects. These risks are present even at very small concentrations. Under today’s rule, all uses of TCE will be banned over time (with the vast majority of identified risks eliminated within one year), and safer alternatives are readily available for the majority of uses.”]

[“PCE is known to cause liver, kidney, brain and testicular cancer, as well as damage to the kidney, liver and immune system, neurotoxicity, and reproductive toxicity. Today’s final rule will better protect people from these risks by banning manufacture, processing and distribution in commerce of PCE for all consumer uses and many commercial uses, while allowing some workplace uses to continue only where robust workplace controls can be implemented.”]

In this press release, the EPA conveyed to all audiences that a primary factor for the forthcoming rulings banning and or significantly limiting use was neurotoxicity concerns for both PCE and TCE. Thus, EPA’s own assessment of impact of these new rulings clearly recognizes prior and potential future harm to human health. Specifically neurological health that has occurred or could occur due to both PCE or TCE exposure.

1.2 12/17/2024 final EPA ruling on TCE.

On 12/17/2024 EPA issued a final ruling on TCE, entitled “Trichloroethylene (TCE); Regulation Under the Toxic Substances Control Act (TSCA)”². The EPA’s own rationale for this ruling further supports the General Causation Report:

1.2.1 The term “*neurotoxicity*” is stated 5 times in the ruling under the following sections:

- I. Executive Summary. D. *Why is the agency taking this action* (p. 102572)? In this section, EPA notes “*neurotoxicity*” is a significant adverse outcome associated with repeated TCE exposures. EPA further states in reference to “*neurotoxicity*” and other non-cancer adverse outcomes: “*This final rule will eliminate the unreasonable risk to human health from TCE...*”.
- I. Executive Summary. E. *What are the estimated incremental impacts of this action* (p. 102574)? EPA notes in this section that “*The actions in this final rule are expected to achieve health benefits to the American public...*”. “*Neurotoxicity*” is specifically noted as one unreasonable risk that will be mitigated by banning virtually all manufacture and uses.
- II. Background. C. *Summary of EPA’s Risk Evaluation Activities on TCE*. 3. Description of Unreasonable Risk (p. 102576). “*Neurotoxicity*” is specifically noted as one of the unreasonable non-cancer risks from TCE exposure.
- V. TSCA Section 6(c)(2) Considerations. A. *Health Effects and the Magnitude of Human Exposure* (p. 102611). EPA states that “*neurotoxicity*” was identified in a 2020 Risk Evaluation as an identified non-cancer adverse outcome from chronic exposures.
- V. TSCA Section 6(c)(2) Considerations. D. *Reasonable Ascertainable Economic Costs of the Final Rule*. 2. Costs and Benefits of the Regulatory Action and of the One or More Primary Alternative Regulatory Actions Considered by the Administrator (p. 102615). In this section EPA noted, specifically non-cancer endpoints are critical considerations in weighing the balance of costs and benefits of this ruling. Here, a specific non-monetized benefit is the mitigating “*neurotoxicity*” risk from TCE exposure.

1.2.2 The term “*Parkinson’s disease*” (PD) is specifically mentioned in the ruling:

- 1. Executive Summary. E. *What are the estimated incremental impacts of this action* (p. 102574)? The EPA specifically notes epidemiological links to PD in this section.

1.2.3 Expert scientific opinion on EPA’s ruling on the 12/08/2024 General Causation Report. The most relevant US Government Authority on cost benefit analysis of TCE use is the EPA. In this ruling, the EPA has determined the costs (adverse outcomes) to human health outweigh the benefits of TCE use. The rationale for the ruling bolsters the General Causation Report in the following ways:

- The clear recognition that neurotoxicity is a significant health concern with TCE exposure, PD being one adverse neurotoxic outcome.
- That banning virtually all TCE use will mitigate unreasonable future neurotoxicity health concerns from TCE exposures, PD being one adverse neurotoxic outcome.

These determinations further support the overall scientific conclusion from the General Causation Report that: Tetrachloroethylene (PCE) is at least as likely as not a cause of PD. The TCE ruling specifically bolsters the following scientific evidence in the General Causation Report that led to the overall conclusion: *Structural similarity and structural activity relationships to trichloroethylene (TCE), which is a known PD risk factor based upon collective epidemiological and neurotoxicological data. There is also a lack of scientific support for the alternate hypothesis that 1 additional chlorine atom and 1 less hydrogen atom (PCE vs TCE) would be protective in preventing PD risk.* EPA's ruling rational is inclusive of overall neurotoxicity and, specifically PD risk. Given the extensive scientific documentation in the General Causation Report that PCE neurotoxicity would be expected to be similar to TCE, with respect to PD, EPA's ruling for TCE further supports plausibility of PCE induced PD relevant risk.

1.3 12/18/2024 final EPA ruling on PCE.

On 12/18/2024 EPA issued a final ruling on PCE, entitled "*Perchloroethylene (PCE); Regulation Under the Toxic Substances Control Act (TSCA)*"³. EPA's own rationale for this ruling further supports the General Causation Report:

1.3.1 The term "neurotoxic- (neurotoxic, neurotoxicant, neurotoxicity)" is states 15 times in the ruling under the following sections:

- I. Executive Summary. D. *Why is the Agency taking this action (p. 103562)?* "Neurotoxicity" is stated 2 times. Specifically, EPA states: "*The most sensitive health effect driving the unreasonable risk of PCE and selected as the basis for this rule is neurotoxicity from chronic exposure.*" "*For PCE, impaired visual and cognitive function and diminished color discrimination following chronic exposures represent the most sensitive endpoint indicating neurotoxicity, based on epidemiological data reported in two studies that identified lowest observed adverse effect levels for color confusion and impaired pattern recognition and reaction time in pattern memory.*". These statements indicated that EPA considers the nervous system as the most sensitive biological target in repose to PCE. Moreover, PCE neurotoxicity appears to involve multiple, wide ranging nervous system targets, adversely affecting both cognitive function and vision. As stated in the General Causation Report, epidemiological data also strongly suggest a plausible causative link between PCE and PD, alongside mechanistic data showing similarities to PD relevant TCE neurotoxicity. Thus, extensive, broad neurotoxicity factors in the EPA ruling plus the PD specific analysis in the General Causation Report bolster scientific conclusions.
- I. Executive Summary. E. *What are the estimated incremental impacts of this action (p. 103564)?* "Neurotoxic- (neurotoxic, neurotoxicity)" is stated 2 times. Specifically, EPA states that the nonmonetized benefits include risk reduction of "neurotoxicity", with even broader neurotoxicity cited for the ruling than in the Executive Summary: "*Neurotoxic effects associated with PCE exposure in human studies include visual deficits, impaired cognition, and neurodevelopmental outcomes from prenatal and early childhood exposure to PCE such as increased affinity of engaging in drug, alcohol, and tobacco use as a teen or adult*". These statements further underscore the broad neurotoxicity concerns of PCE. As stated in the General Causation Report, epidemiological data

also strongly suggest a plausible causative link between PCE and PD, alongside mechanistic data showing similarities to PD relevant TCE neurotoxicity. Thus, extensive, broad neurotoxicity factors in the EPA ruling plus the PD specific analysis in the General Causation Report bolster scientific conclusions.

- II. Background. A. *Overview of Perchloroethylene (PCE)* (p. 103564). The very first sentence of the opening background statement on PCE states firstly, definitively, and prominently that PCE is a neurotoxicant. Thus, extensive, broad neurotoxicity factors in the EPA ruling plus the PD specific analysis in the General Causation Report bolster scientific conclusions.
- II. Background. C. *Summary of EPA's Risk Evaluation Activities on PCE*. 3. Description of Unreasonable Risk (p. 103565). "*Neurotoxic- (neurotoxic, neurotoxicity)*" is stated 2 times. Again, EPA states "*neurotoxicity*" as the most unreasonable non-cancer risk: "*EPA identified neurotoxicity as the most robust and sensitive endpoint for non- cancer adverse effects from acute inhalation and dermal exposures and as the most robust and sensitive endpoint for non-cancer adverse effects from chronic inhalation and dermal exposures for all conditions of use.*". Neurotoxicity is clearly a driving factor in the PCE ruling. Thus, extensive, broad neurotoxicity factors in the EPA ruling plus the PD specific analysis in the General Causation Report bolster scientific conclusions.
- IV. Provisions of Final Rule. B. *Workplace Chemical Protection Program (WCPP)*. 3. Existing Chemical Exposure Limit (ECEL) (p. 103583). EPA is finalizing with slight modification that owners or operators must ensure the airborne concentration of PCE within the personal breathing zone of potentially exposed persons remains at or below 0.14 ppm as an 8-hour TWA ECEL, with an action level finalized as 0.10 ppm as an 8-hour TWA. EPA states specifically that "*neurotoxicity*" is a reason for this ruling.
- IV. Provisions of Final Rule. C. *Prescriptive Controls*. 2. Workplace Requirements for Energized Electrical Cleaner. c. Labeling Requirements for Energized Electrical Cleaner (p. 103592). EPA states the following labelling requirement: "*This product contains perchloroethylene (PCE) (CASRN 127-18-4), a chemical determined by the Environmental Protection Agency to present unreasonable risk of injury to health under the Toxic Substances Control Act (TSCA), based on neurotoxicity and other adverse health effects.*". In this statement, EPA recognizes under the extremely limited conditions that PCE will now be used, those with exposure potential should be explicitly warned of "*neurotoxicity*". Thus, extensive, broad neurotoxicity prevention in the EPA ruling plus the PD specific analysis in the General Causation Report bolster scientific conclusions.
- V. TSCA Section 6(c)(2) Considerations. A. *Health Effects of PCE and the Magnitude of Human Exposure to PCE* (p. 103596). "*Neurotoxicity*" is broadly stated 4 times. EPA states explicitly under TSCA, the ruling resulting in extreme limitations on PCE use stemmed from neurotoxicity as a primary concern. Neurotoxicity is clearly a driving factor in the PCE ruling. Thus, extensive, broad neurotoxicity factors in the EPA ruling plus the PD specific analysis in the General Causation Report bolster scientific conclusions.

- V. TSCA Section 6(c)(2) Considerations. *D. Reasonably Ascertainable Economic Consequences of the Final Rule.* 2. Costs and Benefits of the Regulatory Action and of the One or More Primary Alternative Regulatory Actions Considered by the Administrator (p. 103598). EPA states that the ruling is expected to achieve health benefits for the American public. EPA specifically states that mitigating “neurotoxicity” concerns is a primary driver of the ruling. Neurotoxicity is clearly a driving factor in the PCE ruling. Thus, extensive, broad neurotoxicity factors in the EPA ruling plus the PD specific analysis in the General Causation Report bolster scientific conclusions.
- PART 751—REGULATION OF CERTAIN CHEMICAL SUBSTANCES AND MIXTURES UNDER SECTION 6 OF THE TOXIC SUBSTANCES CONTROL ACT. § 751.609 Workplace requirements for laboratory use. Here again, EPA restates the following labelling requirement: *“This product contains perchloroethylene (PCE) (CASRN 127–18–4), a chemical determined by the Environmental Protection Agency to present unreasonable risk of injury to health under the Toxic Substances Control Act (TSCA), based on neurotoxicity and other adverse health effects.”*. In this statement, EPA recognizes under the extremely limited conditions that PCE will now be used, those with exposure potential should be explicitly warned of neurotoxicity. Thus, extensive, broad neurotoxicity prevention in the EPA ruling plus the PD specific analysis in the General Causation Report bolster scientific conclusions.

1.3.2 Expert scientific opinion on EPA’s ruling on the 12/08/2024 General Causation Report. The most relevant US Government Authority on cost benefit analysis is the EPA. In this ruling, the EPA has determined the costs (adverse outcomes) to human health outweigh the benefits of PCE, resulting in major restrictions on use. The rationale for the ruling bolsters the General Causation Report in the following ways:

- The clear recognition that neurotoxicity is a significant health concern with PCE exposure.
- That banning manufacture, processing and distribution in commerce of PCE for all consumer uses and many commercial uses, while allowing some workplace uses to continue only where robust workplace controls can be implemented will mitigate unreasonable future neurotoxicity health concerns from PCE exposures.

These determinations further support the overall scientific conclusion from the General Causation Report that: Tetrachloroethylene (PCE) is at least as likely as not a cause of PD. The PCE ruling specifically and repeatedly states general neurotoxicity as a critical rationale for the ruling. While neurotoxicity is stated in this ruling even more frequently than in the TCE ruling, PCE neurotoxicity is referred to much more broadly. Here, there is much less detailed knowledge for specific neurological diseases PCE may cause (vs. TCE), again PCE having far less citable studies in this area of research. What is clear is that the EPA has ruled that PCE enters and adversely affects the brain. Thus, the PCE ruling also bolsters the following scientific evidence in the General Causation Report that led to the overall conclusion: *Structural similarity and structural activity relationships to trichloroethylene (TCE), which is a known PD risk factor based upon collective epidemiological and neurotoxicological data. There is also a lack of scientific support for the alternate hypothesis that 1 additional chlorine atom and 1 less hydrogen atom (PCE*

vs TCE) would be protective in preventing PD risk. Specifically, rather than any portion of the PCE ruling suggesting that PCE is less neurotoxic than TCE, the ruling states “neurotoxicity” as even a more prominent factor in the ruling than TCE. Given the extensive scientific documentation in the General Causation Report that PCE neurotoxicity would be expected to be similar to TCE, with respect to PD, EPA’s ruling for PCE further supports plausibility of PCE induced PD relevant risk.

2. SUMMARY OF SCIENTIFIC CONCLUSIONS FROM 12/08/2024 GENERAL CAUSATION REPORT FOR REFERENCE AND WITH ANNOTATIONS*,**

**See section 6 of General Causation Report.*

***Annotations to original conclusions are in bolded italics.*

2.1 Tetrachloroethylene (PCE) is at least as likely as not a cause of PD (PD) based upon the following scientific evidence:

BASED ON THE FOREGOING ANALYSIS, AND BASED UPON MY EDUCATION, TRAINING AND EXPERIENCE, IT IS MY OPINION TO A REASONABLE DEGREE OF SCIENTIFIC CERTAINTY THAT TETRACHLOROETHYLENE (PCE) IS AT LEAST AS LIKELY A CAUSE OF PD (PD). MY ANALYSIS INCLUDES THE FOLLOWING:

2.1.1 Structural similarity and structural activity relationships to trichloroethylene (TCE), which is a known PD risk factor based upon collective epidemiological and neurotoxicological data. There is also a lack of scientific support for the alternate hypothesis that 1 additional chlorine atom and 1 less hydrogen atom (PCE vs TCE) would be protective in preventing PD risk*.

***EPA ruling for TCE specifically notes neurotoxicity, inclusive of PD risk, which is also specifically noted. Given the structural similarities (to PCE) and noted neurotoxic effects, the TCE ruling further bolsters support for chloroethylene exposure, inclusive of PCE as a PD risk factor.*

***EPA ruling for PCE specifically notes neurotoxicity, even more prominently and frequently as a basis for the decision on TCE. While there are some indications in the PCE ruling on broad adverse outcomes, it is clear there has been less research focus on specific neurological diseases that PCE may cause (vs TCE). Here again, Given the structural similarities (to TCE) and noted neurotoxic effects, the PCE ruling further bolsters support for chloroethylene exposure, inclusive of PCE as a PD risk factor.*

2.1.2 Epidemiology of chlorinated ethylene solvents (inclusive of PCE) and PD*.

**EPA ruling for TCE specifically notes epidemiological links to PD. Given the structural and noted neurotoxic effects (also see 2.1.1 above and section 6 of General Causation Report), the*

TCE ruling further bolsters support for chloroethylene exposure, inclusive of PCE as a PD risk factor.

2.1.3 Direct experimental evidence that PCE toxicity is mediated by critical PD pathogenic pathways.

2.1.4 Hill considerations met directly for PCE, or by scientifically relevant analogy for TCE.

I AM BEING COMPENSATED \$500 AN HOUR FOR MY TIME DEVOTED TO INVESTIGATING THE RELEVANT ISSUES AND DRAFTING THIS REPORT.

A handwritten signature in blue ink, appearing to read "Jason Cannon", is positioned above a horizontal line.

01/02/2025

Jason Cannon, Ph.D.

3. REFERENCES

1. USEPA. Biden-Harris Administration Announces Latest Actions under Nation's Chemical Safety Law to Protect People from Cancer-Causing Chemicals Trichloroethylene and Perchloroethylene. <https://www.epa.gov/newsreleases/biden-harris-administration-announces-latest-actions-under-nations-chemical-safety-law>; United States Environmental Protection Agency; 2024.
2. USEPA. Trichloroethylene (TCE); Regulation Under the Toxic Substances Control Act (TSCA). Federal Register: RIN 2070-AK83; ENVIRONMENTAL PROTECTION AGENCY; 2024. p. 102568-635.
3. USEPA. Perchloroethylene (PCE); Regulation Under the Toxic Substances Control Act (TSCA). Federal Register: RIN 2070-AK84; 2024. p. 103560-616.