vinyl INSTITUTE

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Re: Vinyl Institute Comments on the Draft Scope of the Risk Evaluation for Vinyl Chloride; EPA-HQ-OPPT-2018-0448

The Vinyl Institute's (VI)[1] Vinyl Chloride Consortium (VC Consortium) appreciates the opportunity to submit these comments on the U.S. Environmental Protection Agency's (EPA or the Agency) draft scope of the risk evaluation for vinyl chloride.[2] Members of the VC Consortium include Formosa Plastics Corporation, Occidental Chemical Corporation, Shintech Inc., Westlake Corporation, and Olin Corporation, and produce virtually all of the vinyl chloride manufactured in the United States. The draft document represents a reasonable starting point for stakeholder input on the Toxic Substances Control Act (TSCA) risk evaluation for vinyl chloride, but numerous changes are necessary to ensure that EPA's risk evaluation is appropriately scoped, compliant with TSCA, and consistent with the Trump Administration's policy goals.

Critically, EPA must acknowledge, in this risk evaluation and across the Agency's TSCA section 6 activities, that unreasonable risk is a term of art under TSCA. While not defined in TSCA or in EPA's regulations, the ordinary meaning of "unreasonable risk" cannot be the same as just any "risk" identified through the traditional tools of risk assessment: it must be "unreasonable."

As we explain in these comments, there are many conditions of use for which qualitative, rather than quantitative, assessment is warranted. Qualitative risk assessment is an important tool to determine that conditions of use (COUs) are not anticipated to present unreasonable risk, at which point a more robust quantitative assessment is unnecessary. The manufacturing, processing, and use of vinyl chloride is already highly regulated, with robust controls used to prevent exposure. In addition, the physical and chemical properties of vinyl chloride mean that many standard routes of exposure are not relevant. In these situations, it is straightforward to determine that there is no potential for risk, let alone an unreasonable risk. For example, the Draft Scope appropriately indicates that EPA will assess the dermal

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[1] The Vinyl Institute, established in 1982, represents the leading producers of vinyl chloride and other monomers, vinyl resins, and ingredient and additive producers for vinyl compounds. The VI serves as the collective voice for the vinyl industry. More information can be found at www.vinylinfo.org.

[2] *Vinyl Chloride; Draft Scope of the Risk Evaluation Under the Toxic Substances Control Act (TSCA); Notice of Availability and Request for Comment*, 90 Fed. Reg. 4,738 (Jan. 16, 2025); *Draft Scope of the Risk Evaluation for Vinyl Chloride*, Docket No. EPA-HQ-OPPT-2018-0448-0617 ("Draft Scope").

exposure route qualitatively. EPA should similarly assess the potential for consumer inhalation exposure from articles and products qualitatively because of the extremely low potential for exposure. Attempting to quantify these scenarios would only provide unscientific, hyper-conservative calculations that would be a waste of EPA's limited resources, and would likely still reach the same conclusion that there is no potential risk due to no (or very little) exposure.

Vinyl chloride is safely manufactured in the United States and the industry adheres to existing, comprehensive regulations governing the safe manufacture and use of vinyl chloride. The industry clearly prioritizes protecting workers, neighbors, and customers. The VC Consortium provides the following comments to assist EPA with defining an appropriate scope for the risk evaluation and to provide key information for EPA's consideration. We have been willing, collaborative good-faith partners to the Agency over the years, and we remain committed to engaging with EPA throughout the TSCA regulatory process for vinyl chloride.[3]

I. Conditions of Use

a. EPA Must Incorporate Practicality into the Identification of COUs

Vinyl chloride is an essential commodity chemical that is a vital part of the value chain for many products. As VI described in its March 18, 2024, comments,[4] vinyl chloride is chemically converted into material endpoints such as PVC resin before ever reaching a consumer. While residual vinyl chloride may still be entrained in the polymer matrix, industry is required to limit the amount of residual vinyl chloride (typically referred to as residual vinyl chloride monomer (RVCM)) under the PVC MACT.[5] Furthermore, industry has implemented programs that evaluate articles for the presence of vinyl chloride, including adherence to voluntary consensus-based standards, such as NSF/ANSI-61 for drinking water pipes. Because PVC products may contain at most a trace amount of vinyl chloride in accordance with Federal regulations and are demonstrated to be safe to manufacture and use, EPA should narrow the draft scope of the vinyl chloride risk evaluation to qualitatively assess products that are produced downstream of PVC resin production and, where feasible, exclude from further evaluation those products that contain no RVCM or virtually undetectable RVCM because such miniscule impurities are not COUs relevant to risk.

[3] VI and its VC Consortium submitted extensive comments during the prioritization phase for VC. Docket No. EPA-HQ-OPPT-2018-0448-0025, EPA-HQ-OPPT-2018-0448-0606. Those comments are referenced and incorporated into these comments.

[4] Docket No. EPA-HQ-OPPT-2018-0448-0025.

[5] Formally known as the National Emission Standards for Hazardous Air Pollutants for Polyvinyl Chloride and Copolymers Production, 40 C.F.R. Part 63 Subpart HHHHHHH.

As noted in the VC Consortium's March 2024 comments, PVC resin following polymerization is typically in the form of solids suspended in liquid or in a slurry form.[6] The resin subsequently is piped to a resin stripper (typically a steam stripper), which removes vinyl chloride and other HAPs to comply with the PVC MACT (and, before that, the VCM NESHAP) stripped resin limits. At downstream customer facilities, PVC resin is compounded with any additives necessary for the end-use application and formed into the needed shape(s) for the application, typically via extrusion. Finished PVC products include both products that are within the scope of TSCA and many articles that are outside the scope of TSCA (*e.g.*, blood bags, medical devices). Given the extremely low potential for vinyl chloride to be present in articles at more than miniscule concentrations, EPA need only perform a qualitative evaluation of these products in order to rule out risk from these articles early in the risk evaluation process.

With respect to downstream exposures, EPA states, without justification, that it plans to assume that vinyl chloride is present at 0.1% (1000 ppm) in any PVC product when vinyl chloride is not specifically listed on a product Safety Data Sheet (SDS).[7] Again, this assumption ignores the existing regulatory framework governed by the Clean Air Act, where residual vinyl chloride is required to be stripped out of all PVC resin types at sufficiently low levels to ensure worker and user safety.[8] A paper presented at the Society of Plastics Engineers VinylTec conference in 2017 illustrates how the average typical amount of residual vinyl chloride is less than 1.0 ppm across all resin types, and in the most commonly used PVC resin type (suspension) it is below 0.3 ppm.[9] The same paper demonstrates that these average typical residual vinyl chloride monomer levels have improved since 2000 across all resin types, with the percent reduction in RVCM over that time ranging from 37% to 76%. EPA's assertion also ignores the fact that most articles do not require an SDS.

Many articles are exempt from the U.S. Occupational Safety and Health Administration's (OSHA) Hazard Communication Standard (HCS), and thus manufacturers, distributors, and employers do not need to provide an SDS.[10] Since PVC resin has already been stripped of vinyl chloride, PVC resins and compounds are typically sold with a Certificate of Analysis (CoA) to demonstrate the low levels of vinyl chloride that are present (assuming vinyl chloride is detected as all). EPA should not take the ultra-conservative and

[6] To this point, we note that *EPA's Draft Chemistry and Fate Assessment Technical Document for Vinyl Chloride* lists the solubility of vinyl chloride as 9,150 mg/L at 20.5 °C, although other sources have reported lower values. *Cf.* Docket No. EPA-HQ-OPPT-2018-0448-0619 at 13 *with* ATSDR, *Toxicological Profile for Vinyl Chloride* (Jan. 2024) at 136, available at: https://www.atsdr.cdc.gov/toxprofiles/tp20.pdf. In any case, it is important that EPA not assume that vinyl chloride is present in various streams (unstripped resin, unstripped wastewater) at the limit of solubility; available data indicates that is not true.

[7] Draft Scope at 48.

[8] *See* 40 C.F.R. Part 63 Subpart HHHHHHH.

[9] Borrelli, F., R., Krock, R., Paradis, et. al., SPE ANTEC 2017 Presentation, May 9, 2017.

[10] *See* 29 C.F.R. § 1900.1200(b)(6)(v).

unsupported position that products are assumed to contain vinyl chloride at 0.1% absent an SDS statement indicating a specific concentration.

EPA must refine and narrow the scope of its planned risk evaluation for vinyl chloride to focus on the manufacture of vinyl chloride and processing of vinyl chloride into other materials and substances. In identifying worker activities associated with COUs within the scope of the risk evaluation, EPA should focus its review: workers "performing other work activities in or near areas where vinyl chloride is used,"[11] will necessarily be a limited population given existing engineering and administrative controls. EPA should limit its evaluation to conditions of use related to production and processing of vinyl chloride and should not include any conditions of use related to transportation, emergency situations, or releases that are already regulated under other statutes and authoritative bodies. For example, the U.S. Department of Transportation (DOT) is responsible for issuing regulations to protect against the risks that are inherent in the transportation of hazardous material in commerce. Furthermore, considering the tragic events that occurred in East Palestine, Ohio in February 2023, the official report on the rail incident by the National Transportation Safety Board (NTSB) strongly indicates that the cars carrying vinyl chloride had functioned properly, and that the vent-and-burn decision made at the scene was not necessary. TSCA is a gap-filling statute, intended to regulate only exposures and uses that are not adequately addressed under other laws.

b. Accidental Releases

EPA states that it generally does not include catastrophic accidents, extreme weather events, and other natural disasters in the scope of risk evaluations; however, the Agency "would consider including such events . . . if the Agency receives information indicating regular and predictable changes in exposures associated with these events."[12] For instance, EPA references a prior preamble discussion where the Agency clarified that it:

> . . . would generally not include within the scope of the risk evaluation exposures associated with future extreme weather events (e.g., hurricanes and wildfires). However, if information reasonably available to the Agency indicated that factors such as rising sea levels or extreme temperatures made worse by climate change were leading to regular and predictable changes in exposures associated with a given condition of use of a chemical substance, EPA would expect to consider those exposures within the scope of the risk evaluation.[13]

[11] Draft Scope at 26.

[12] Draft Scope at 17.

[13] *Procedures for Chemical Risk Evaluation Under the Toxic Substances Control Act (TSCA)*, 88 Fed. Reg. 74,292, 74,299 (Oct. 30, 2023).

As discussed in more detail below, these events are not appropriately part of the COUs for vinyl chloride.

1. Accidental Releases are Rare and Not Part of the Conditions of Use

The inclusion of exposures associated with catastrophic accidents, extreme weather events, and other natural disasters in the conditions of use would require an overly broad interpretation of the term "conditions of use." TSCA defines "conditions of use" to encompass:

> *"the circumstances, as determined by the Administrator, under which a chemical substance is intended, known, or reasonably foreseen to be manufactured, processed, distributed in commerce, used or disposed of."*[14]

The phrase "intended, known, or reasonably foreseen" is a limitation on the circumstances that are part of the COUs for a substance and thus a limitation on the COUs that may be identified and included in the scope of a risk evaluation. It goes without saying that catastrophic accidents, extreme weather events, and other natural disasters and any potential exposures that may arise from them are not "intended" or planned. Instead, the inclusion of these events in the scope of the risk evaluation would hinge on exposures being known or reasonably foreseen or, as EPA put it, "regular and predictable." However, these types of events and their impacts are, by their very nature, anything but "regular and predictable." Indeed, an accident, by its very definition, is an "unforeseen and unplanned event or circumstance" and very clearly falls outside of "intended, known, or reasonably foreseen" limitations of the definition of conditions of use.[15]

Similarly, although a growing body of evidence suggests that extreme weather events and other natural disasters are increasing in frequency, intensity, and extent, there are still many variables (*e.g.*, research gaps, long-term societal actions) that impact the predictability of these events on a large scale.[16] As such, it is unclear how EPA would, for example, quantify, "the *likely* duration, intensity, frequency, and number of exposures" associated with the effects of an extreme weather event or other natural disasters.[17] To do so would toe the line into making conditions of use determinations based on hypothetical scenarios or mere projections. Moreover, there is no evidence that extreme weather events have any linkage to accidental releases of vinyl chloride in particular.

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[14] 15 U.S.C. § 2602(4).

[15] "Accident." Merriam-Webster.com Dictionary, https://www.merriam-webster.com/dictionary/accident.

[16] *See e.g.*, *Fifth National Climate Assessment*, U.S. Global Change Research Program (2023), 1-38, 2-37.

[17] 15 U.S.C. § 2605(b)(4)(F) (emphasis added)

2. Existing Regulatory Programs Address the Potential for Accidents

Accidental releases of vinyl chloride are atypical and not likely to occur with any predictable frequency. The vinyl industry is already subject to several regulations governing emergency response and unplanned releases, which also require preparations to prevent such releases. As such, catastrophic accidents, extreme weather events, and other natural disasters should be excluded from the scope of the risk evaluation in the interest of avoiding duplicative regulation and reducing regulatory burden.

From its inception, TSCA has served to fill gaps in regulation. The 2016 TSCA Amendments "reinforce[d] TSCA's original purpose of filling gaps in Federal law that otherwise did not protect against the unreasonable risks presented by chemicals."[18] To effectuate this purpose, section 9(b)(1) directs EPA to coordinate actions taken under TSCA with the impacts of actions under other federal laws administered in whole or in part by the Agency. If the risk associated with a chemical substance can be addressed, or reduced to a sufficient extent, by actions taken under such other federal laws, EPA must use such other authority, unless the Agency determines that it is in the public interest to use its authority under TSCA. In addition, section 9(d) requires that EPA consult and coordinate with other federal executive departments, agencies, and other appropriate instrumentalities of the federal government to "achiev[e] the maximum enforcement of this chapter while imposing the least burdens of duplicative requirements on those subject to this chapter."

In this case, the risks associated with catastrophic accidents, and the impacts of extreme weather and natural disasters on the vinyl industry, are already mitigated by both EPA-administered statutes and those administered by other agencies. The regulations, administrative, controls, and industry programs that address responses to accidents, should they occur, and the actions necessary to minimize accidents and to prevent recurrence are outlined in the paragraphs below.

Risk Management Program (40 C.F.R. Part 68): Vinyl chloride and PVC producers are subject to EPA's accidental release prevention requirements, commonly called the Risk Management Program (RMP). RMP requires regulated facilities that have more than a threshold quantity of vinyl chloride in a process to develop a risk management plan that: (i) identifies the potential effects of a chemical accident; (ii) identifies steps the facility is taking to prevent an accident; and (iii) spells out emergency response

[18] H. Rep. No. 114-176 (June 23, 2015) at 28.

procedures should an accident occur. This includes the evaluation of risks from natural hazards (*e.g.*, flooding, hurricanes, earthquakes, etc.)[19] that could potentially lead to an accidental release.[20]

Facilities subject to RMP's Program 3 Prevention Program, such as vinyl chloride and PVC resin producers, must meet strict requirements to prevent accidental releases, minimize worker exposure, and protect public health and the environment, if an event does occur:

- **Process Safety and Process Hazard Analysis** – Provide written process safety information identifying and explaining the hazards posed by the production process. In addition, owners or operators of covered processes must also perform a process hazard analysis that identifies, evaluates, and controls the hazards associated with the production process.
- **Operating Procedures** – Develop and implement written procedures that provide clear instructions for safely conducting activities within each covered process, including during normal, temporary, and emergency operations. The procedures must include, for example, safe work practices that control hazards during operations such as lockout/tagout and confined space entry, and address precautions necessary to prevent chemical exposure (*e.g.*, engineering controls, administrative controls, and personal protective equipment).
- **Mechanical Integrity (MI)** – Establish written procedures to maintain the on-going integrity of process equipment using "Recognized and generally accepted good engineering practices" (RAGAGEP) such as API and ASTM standards. One major goal of a MI program is to prevent the loss of primary containment (LOPC) of the process liquids and gases. RMP's MI requirements require that inspections and testing of process equipment be performed for the life of the equipment. Any equipment deficiencies identified must be corrected before further use of the equipment.
- **Incident Investigations** - The owner or operator shall investigate each incident, which resulted in, or could reasonably have resulted in a "catastrophic release."[21] Incident investigations must be initiated not later than 48 hours following the incident. Incident

[19] The March 2024 RMP amendments explicitly address external events such as natural hazards, however, long-standing EPA RMP Emergency Response Program guidance directs owners and operators to consider their susceptibility to hazards both at the facility and in the surrounding environment (*e.g.*, "floods, temperature extremes, tornadoes, earthquakes, and hurricanes"). *See* EPA, General Risk Management Programs Guidance, Chap. 8: Emergency Response (Apr. 2004).

[20] "Accidental release" means an unanticipated emission of a regulated substance or other extremely hazardous substance into the ambient air from a stationary source. 40 C.F.R. § 68.3.

[21] "Catastrophic release" means a major uncontrolled emission, fire, or explosion, involving one or more regulated substances that present imminent and substantial endangerment to public health and the environment. 40 C.F.R. § 68.3.

report findings and recommendations must be promptly addressed and resolutions and corrective actions documented. The report must also be review with affected personnel with tasks relevant to the incident findings.

Finally, emergency response must be coordinated with local emergency planning and response organizations on at least an annual basis.

VCM NESHAP (40 C.F.R. Part 61 Subpart F): This rule affects plants that produce:

(1) Ethylene dichloride by reaction of oxygen and hydrogen chloride with ethylene;

(2) Vinyl chloride by any process; and/or

(3) One or more polymers containing any fraction of polymerized vinyl chloride (*i.e.*, plants that produce PVC resin).

Within the VCM NESHAP, releases of vinyl chloride to the atmosphere from pressure relief devices (PRDs) – *i.e.*, safety equipment used to release an unplanned, non-routine discharge of gas from process equipment to avoid safety hazards or equipment failure – are generally prohibited. An exception exists for "emergency relief discharges," or a discharge which could not have been avoided by taking measures to prevent the discharge.[22]

Hazardous Organic NESHAP (HON) (40 C.F.R. Part 63 Subparts F, G and H): The HON also regulates vinyl chloride released to the atmosphere, including through PRDs. Owners and operators are required to equip PRDs with a device or system that will identify, record, and notify them of a release and release prevention measures (*e.g.*, flow, temperature, liquid level and pressure indicators with deadman switches).

PVC MACT (40 C.F.R. Part 63 Subpart HHHHHHH): Discharges to the atmosphere from PRDs in HAP service are prohibited, so facilities take significant steps to avoid PRD discharges. In addition, such PRDs are required to have a release indicator to signal when an emission release has occurred and to be equipped with an alert system that will "immediately and automatically" notify an operator when the PRD is open, in order to reduce the potential for PRD discharges.

OSHA's Vinyl Chloride Standard (29 C.F.R. § 1910.1017): This regulation applies during (1) the manufacture, reaction, packaging, repackaging, storage, handling or use of vinyl chloride or polyvinyl chloride;[23] and (2) the transportation of vinyl chloride or polyvinyl chloride except to the extent that the Department of Transportation may regulate the hazards covered by the OSHA standard.[24] Under the

[22] 40 C.F.R. § 61.65(a).

[23] Excluding the handling or use of fabricated products made of PVC.

[24] 29 C.F.R. § 1910.1017.

standard, the employer's central obligation is to limit employee exposure to vinyl chloride, including in the event of an emergency. A written operational plan for emergency situations must be developed. If there is an emergency,[25] then employees engaged in "hazardous operation"[26] or correcting situations during a hazardous release must be equipped with respiratory protection and protective garments, which are required to be "clean and dry" for each use.

Hazardous Substance Designations and Release Notifications (40 C.F.R. Part 302): Section 102(a) of EPA's Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) identifies reportable quantities for certain hazardous substances, including vinyl chloride, and sets forth the notification requirements if a release event occurs. For vinyl chloride, any release that exceeds 1 pound in a 24-hour period requires notification to the National Response Center.[27]

Department of Transportation: Railcars carrying vinyl chloride are primarily regulated by the U.S. Department of Transportation (DOT) which sets strict standards for the design, construction, and handling of the railcars, including specific labeling requirements, safety procedures for loading and unloading, and emergency response protocols due to the hazardous nature of vinyl chloride. This includes 49 CFR Part 172 which lists and classifies those materials which the DOT has designated as hazardous materials for purposes of transportation and prescribes the requirements for shipping papers, package marking, labeling, and transport vehicle placarding applicable to the shipment and transportation of those hazardous materials.

II. Exposure

The exposures identified by EPA for potential assessment during the risk evaluation are fairly straightforward. In Section V of these comments, below, we address EPA's conceptual models for addressing these exposures; in this section, we provide additional information concerning exposure and corrections to the Draft Scope.

a. General Population Exposure

General population exposures through air emissions from manufacturing facilities are appropriately regulated under the Clean Air Act (CAA). As VI explained in previous comments, vinyl chloride is

[25] "Emergency" means any occurrence such as, but not limited to, equipment failure, or operation of a relief device which is likely to, or does, result in massive release of vinyl chloride. 29 C.F.R. § 1910.1017(b)(5).

[26] "Hazardous operation" means any operation, procedure, or activity where a release of either vinyl chloride liquid or gas might be expected as a consequence of the operation or because of an accident in the operation, which would result in an employee exposure in excess of the permissible exposure limit. 29 C.F.R. § 1910.1017(b)(7).

[27] 40 C.F.R. §§ 302.5, 302.6(a). Note that while the NRC posts initial incident data to its website, that data has not been validated or investigated; subsequent investigation by the reporting company may find that there was no release.

regulated as a Hazardous Air Pollutant under Section 112.[28] Indeed, EPA recently reviewed emissions from the manufacture of vinyl chloride for residual risk under the CAA and determined that vinyl chloride emissions do not warrant more stringent regulation.

On May 16, 2024, EPA finalized a second residual risk assessment for the Synthetic Organic Chemical Manufacturing Industry (SOCMI) Source Category in support of the Agency's 2024 risk and technology review for 40 CFR Part 63, Subparts F, G, H, and I.[29] As part of this residual risk assessment, EPA calculated and assessed emissions of vinyl chloride. Because these EPA calculations were not available in the docket for the SOCMI rulemaking, the Consortium tasked ALL4 to recreate EPA's calculations concerning cancer, chronic non-cancer, and acute risk from emissions of vinyl chloride from facilities subject to that rule.[30] ALL4's work confirmed that EPA found no evidence of unacceptable risk to populations surrounding SOCMI facilities from vinyl chloride. Accordingly, there is no need for EPA to redundantly assess or regulate general population exposures from air emissions under TSCA.

b. Occupational Exposures

EPA states that it plans to evaluate worker activities where there is a potential for exposure, specifically potential exposure via the inhalation route and the dermal route. The VC Consortium agrees with EPA that the oral route does not present a potential for significant exposure given the physical-chemical properties of vinyl chloride.

EPA reported its expectation that program data from OSHA, Chemical Exposure Health Data (CEHD), and National Institute for Occupational Safety and Health (NIOSH) Health Hazard Evaluations (HHEs) will serve as key data to inform the occupational exposure assessment for vinyl chloride. While EPA did not identify specific air monitoring data within the OSHA and CEHD databases intended for use in the occupational exposure assessment of vinyl chloride as it has in prior draft risk evaluation scopes, 41 NIOSH HHEs were identified as potentially relevant gray literature sources to the occupational exposure assessment for vinyl chloride in the Health Assessment Workspace Collaborative (HAWC) database.[31] Upon review of the available occupational exposure literature identified by EPA, the VC Consortium has

[28] Docket No. EPA-HQ-OPPT-2018-0448-0025 at 8, 10.

[29] *New Source Performance Standards for the Synthetic Organic Chemical Manufacturing Industry and National Emission Standards for Hazardous Air Pollutants for the Synthetic Organic Chemical Manufacturing Industry and Group I & II Polymers and Resins Industry*, 89 Fed. Reg. 42,932 (May 16, 2023). The National Emission Standards for Hazardous Air Pollutants (NESHAP) for SOCMI is otherwise known as the Hazardous Organic NESHAP or "the HON."

[30] For additional information on EPA's CAA risk assessment methodology, please refer to the document titled *Residual Risk Assessment for the Synthetic Organic Chemical Manufacturing Industry (SOCMI) Source Category in Support of the 2024 Risk and Technology Review Final Rule*, Docket No. EPA-HQ-OAR-2022-0730-2807.

[31] TSCA Vinyl Chloride: Environmental Release and Occupational Exposure (2025), available at: https://hawc.epa.gov/assessment/100500391/.

identified potential limitations in the suitability and relevance of exposure data intended for use in the risk evaluation of vinyl chloride.

As explained below, based upon prior review of the occupational exposure literature identified in the HAWC database and the data further reported to contain personal sampling data in Appendix B.2 of the Draft Scope, the VC Consortium recommends EPA carefully consider the context and availability of data within these studies. We advise EPA to consider the appropriateness of the available worker exposure literature in characterizing current worker exposures to vinyl chloride and avoid reliance on studies that (1) incorporate exposures prior to 1975; (2) lack necessary qualitative information describing the purpose of sampling as well as activities that occurred during sampling; (3) represent worst-case conditions or high-end exposure potential; and/or (4) were authored for purposes beyond baseline characterization of occupational exposures to vinyl chloride.

1. Literature References Identified by EPA

In Appendix B.2 of the Draft Scope, EPA summarized information identified from publicly available databases and gray literature references for assessing occupational exposures to vinyl chloride. In total, 225 information sources pertaining to occupational exposure were included in Appendix B.2 and were further stratified by evidence provided. By comparison, through its HAWC database, EPA identified 187 information sources, including 116 peer-reviewed and 71 gray literature references, as potentially relevant to occupational exposure to vinyl chloride; evidently, 38 occupational exposure sources included in Appendix B.2 were not identified in the prior literature search informing the HAWC database or were not screened due to unavailability of the full reference. Of the 225 total sources included in Appendix B.2, 108 were reported to contain personal sampling data.

Of the peer reviewed literature identified in Appendix B.2 to potentially inform the occupational exposure assessment and containing personal sampling data, approximately 29 sources consisted of epidemiological investigations into specific health outcomes associated with exposure to vinyl chloride. Upon review of these 29 sources, approximately six sources reportedly characterized occupational exposure to vinyl chloride through collection of personal samples. Of the remaining epidemiological studies, worker exposure potential was typically determined through organization into predetermined exposure bands based on worker job history, job title, or uniformly assumed historic air concentrations, rather than through collection of personal air samples. While ultimately aligned with the intent of protecting worker health, epidemiological studies generally aim to characterize statistically significant associations between exposure and disease, rather than characterize individual worker exposure experiences during 8-hour work shifts or specific tasks. Thus, we encourage EPA to consider the relevance of epidemiological estimates in representing worker exposures in current occupational environments and the suitability of identifying these estimates as personal sampling data. Furthermore, of the available epidemiological studies on vinyl chloride, all include some exposures occurring prior to the PEL change in 1975; therefore, exposure estimates derived from these studies are anticipated to overestimate exposures in current industrial facilities and are, thus, not appropriate for this risk evaluation.

2. Occupational Exposure Data Identified by EPA

As noted above and explained in the VC Consortium's prior comments during the prioritization process, the OSHA Vinyl Chloride Standard is applicable to (1) the manufacture, reaction, packaging, repackaging, storage, handling or use of vinyl chloride or polyvinyl chloride; and (2) the transportation of vinyl chloride or polyvinyl chloride except to the extent that the Department of Transportation may regulate the hazards covered by the OSHA standard.[32] The OSHA Vinyl Chloride Standard imposes significant and protective requirements including exposure monitoring,[33] designation of regulated areas,[34] medical surveillance with a medical opinion for suitability for continued exposure or removal from exposure on each potentially exposed employee every year of exposure,[35] and personal protective equipment such as respiratory[36] and dermal[37] protection.

Of the 108 sources identified in Appendix B.2 as containing personal sampling data for use in the occupational exposure assessment, 42 consisted of NIOSH HHEs. Contrary to prior draft risk evaluation scopes, such as for 1,2-dichloroethane, in the Draft Scope EPA did not identify specific air monitoring data within the OSHA and CEHD database intended for use in the occupational exposure assessment of vinyl chloride. Notably, OSHA inspection and NIOSH HHE data are typically collected at the request of an employer, employee, or union as a result of workplace complaints or suspected hazard or health concerns. These inspections and evaluations are used to evaluate worst case chemical exposure scenarios and do not intend to completely characterize or represent all employees and daily activities.

Additionally, available records of OSHA and CEHD data lack important sampling details and sufficient qualitative information, such as sampled exposure scenario or task, process descriptions, and sample collection and analysis methodology to reliably allow for interpretation and representation of specific COUs or similar exposure groups (SEGs). While EPA has not indicated whether it intends to perform the occupational exposure assessment for vinyl chloride in the context of SEGs, such analysis performed in prior risk evaluations, such as for 1,1-dichloroethane, was well received by both stakeholders and the Science Advisory Committee on Chemicals (SACC). In the final scope, EPA should commit to taking this approach.

[32] *See* Docket No. EPA-HQ-OPPT-2018-0448-0025 at 4-7.

[33] 29 C.F.R. § 1910.1017(d).

[34] 29 C.F.R. § 1910.1017(e).

[35] 29 C.F.R. § 1910.1017(k).

[36] 29 C.F.R. § 1910.1017(g).

[37] 29 C.F.R. §§ 1910.1017(h)(1)(ii), 1910.132(a), 1910.132(d).

Of the 42 NIOSH HHEs identified by EPA in Appendix B.2, all were published prior to, or in, 1985, and 23 were published prior to, or in, 1975. Data from these HHEs are therefore not likely to represent current industrial practices, and data from studies collected prior to or in 1975 specifically are not likely to reflect changes in exposure control implemented at manufacturing and processing facilities following the promulgation of the new OSHA PEL for vinyl chloride in 1975.

EPA should not rely on OSHA CEHD and NIOSH HHE program data lacking sufficient qualitative information, as the appropriateness of this data for identified COUs or potential analysis by SEG cannot be determined, and use of this data in the risk evaluation would overestimate realistic present-day workplace exposures.

III. Human Health Hazard Information

In the Draft Scope, EPA indicated its intent to focus on four key non-cancer hazards, including liver toxicity, neurotoxicity, immunotoxicity, and developmental toxicity, when considering the hazards for vinyl chloride. This focus is based on EPA's review of prior agency assessments of vinyl chloride hazards. Furthermore, based on carcinogenicity classifications and prior EPA assessments of carcinogenicity for vinyl chloride, the scope also expects to include a detailed evaluation of cancer and genotoxicity hazard data. The VC Consortium separately commissioned Stantec to review the hazard literature for vinyl chloride, with a focus on these endpoints. This review was conducted in a manner consistent with EPA's draft guidance for systematic review under TSCA. The following comments were shaped by that review, including identification of key literature excluded from EPA's Draft Scope (as detailed in Appendix B.5 of the Draft Scope) and a qualitative assessment of the data supporting identification of these hazards.

a. Literature Review and Evaluation

Stantec identified a total of 51 relevant studies of high and medium quality (per EPA's quality rating system) that were not included in EPA's HAWC literature review (Appendix B.5 of the Draft Scope). A list of these studies, including their title, authors, and relevant health endpoints, and quality score is provided in **Attachment 1** to these comments. Of these studies, 47 were epidemiological evaluations, and included 12 high quality and 35 medium quality studies spread across each of the non-cancer and cancer hazards identified by EPA as the focus of the risk evaluation. Of the remaining four toxicological studies, two were deemed to be of high quality, and two were deemed to be of medium quality following application of same quality rating system utilized by EPA.[38] While additional resources were identified for consideration, it is of note that there are limitations to the current body of literature for vinyl chloride. Specifically, none of the medium or high quality studies identified by Stantec (or included in EPA's reference list) included workers that experienced vinyl chloride exposure only after the reduction in the PEL from 500 ppm to 1 ppm in 1975, which likely resulted in dramatically different

[38] EPA, Draft Systematic Review Protocol Supporting TSCA Risk Evaluations for Chemical Substances Version 1.0, (2021).

exposure conditions in the workplace. EPA should take caution in the interpretation of these studies and accord them less weight in a weight of the evidence review, as the studies are not relevant to modern day occupational exposures to vinyl chloride. Furthermore, we urge EPA to consider studies that may not be suitable for use in dose response assessment to assist with drawing conclusions regarding the weight of evidence around a hazard. Even if a study is considered uninformative for dose response assessment, it may contain valuable information suitable for understanding the potential for hazard of vinyl chloride.

As EPA reviews these studies, the VC Consortium notes that EPA's rating system considers how the study was performed, and is not a stand-alone indication of the study's suitability for use in assessing risk for vinyl chloride or its consistency with TSCA science requirements. For example, a study may be rated "high quality" under EPA's rating system while still retaining confounders such as exposure to other chemical substances that necessitate caution and careful review when potentially used during the TSCA vinyl chloride risk evaluation.

1. Liver Toxicity

EPA states that the Agency for Toxic Substances and Disease Registry (ATSDR) concluded that hepatic effects are "a *presumed* health effect for humans" based on evidence of fibrosis, cirrhosis, and steatohepatitis in vinyl chloride workers following chronic-duration inhalation exposures. Stantec's literature search confirmed that the liver is a target for vinyl chloride toxicity on the basis of animal studies. Effects observed were broad ranging and included effects on liver weight, liver enzyme induction, evidence of oxidative stress, and adverse histopathology/changes in morphology of the liver, among others. However, when considering epidemiological literature, evidence for specific liver outcomes were inconsistent. For example, some studies reported increases in cirrhosis of the liver in worker groups with high exposures to vinyl chloride, while other studies reported no increases in liver cirrhosis. One factor we caution EPA to consider during its evaluation of the available hazard data on liver toxicity in humans is to control for alcohol consumption, which is a confounder and major risk factor for the development of liver cirrhosis and other adverse liver outcomes in humans. Notably, of the studies Stantec reviewed, none of those designated as high-quality cohort studies evaluating liver cirrhosis in workers or the general population explicitly controlled for alcohol consumption. Furthermore, a general lack of consistency in categorization or grouping of liver outcomes in human studies makes comparison across studies difficult in determining an overall weight of evidence for liver effects in humans.

2. Neurotoxicity

In the Draft Scope, EPA cites the conclusions from ATSDR that "neurological effects [from exposure to vinyl chloride] are a *presumed* health effect for humans based on limited epidemiological and animal evidence."[39] It was further stated that "there is a moderate level of evidence in animal studies based on clinical signs in multiple acute-duration inhalation studies in rats, mice, and guinea pigs as well as a chronic-duration oral study in rats."[40] Based on Stantec's review, the overall weight of evidence from the animal toxicity literature does not support the occurrence of neurological/behavioral effects following vinyl chloride exposure at moderate exposure levels. Some studies reported motor effects, specifically ataxia and twitching in rats following acute exposures to a very high concentration of 3.8 % vinyl chloride (approximately 38,000 ppm or 98,800 mg/m^3 if assuming 100% purity).[41] Similarly, adverse neurotoxicological effects from chronic exposure studies were also only observed at very high exposure concentrations (*e.g.*, 30,000 ppm); in studies of more moderate exposure (5,000 ppm), exposure to vinyl chloride did not result in non-neoplastic adverse effects in the nervous system.[42] In interpreting the animal studies, we caution EPA about the relevance of adverse effects observed at only very high exposure levels to current occupational exposure scenarios with low exposure potential, if any at all.

With respect to epidemiological evidence of neurological effects, EPA cites to ATSDR's conclusion that there is "a *presumed* health effect for humans." While EPA notes that this statement is based on *limited* epidemiological evidence, Stantec and the VC Consortium caution EPA against drawing such conclusions with as few studies as are available for non-malignant neurological outcomes in humans. Among the five total studies Stantec identified, all substantially differed in terms of type of exposure (occupational, environmental, drinking water) and outcomes (acute symptoms, amyotrophic lateral sclerosis, multiple sclerosis, Parkinson's Disease). The lack of consistent available evidence results in the inability to draw weight of evidence conclusions on these outcomes in humans without further study. Indeed, with current occupational settings representing far lower potential exposures, this limited epidemiological evidence should be given even less weight in a weight of the evidence evaluation.

Based on this collective evidence, while we agree some studies indicate a potential for neurotoxicity from vinyl chloride exposure, the effects observed in animal models have only been observed at very high concentrations and few studies are available in the epidemiological literature to support any consistent reporting of adverse neurotoxicological findings at more moderate exposures. Therefore, as noted above, EPA should not extrapolate high exposure studies to exposure scenarios in the risk

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[39] Draft Scope at 31.

[40] Draft Scope at 31.

[41] D. G. Clark and D. J. Tinston, "Acute inhalation toxicity of some halogenated and non-halogenated hydrocarbons," *Hum Toxicol* 1, no. 3 (Jul 1982), https://doi.org/10.1177/096032718200100306.

[42] P. L. Viola, A. Bigotti, and A. Caputo, "Oncogenic Response of Rat Skin, Lungs, and Bones to Vinyl Chloride," *Cancer Research* 31, no. 5 (1971); V. J. Feron, A. Kruysse, and H. P. Til, "One-year time sequence inhalation toxicity study of vinyl chloride in rats. I. Growth, mortality, haematology, clinical chemistry and organ weights," *Toxicology* 13, no. 1 (May 1979).

evaluation with comparatively low exposure (*i.e.*, today's vinyl chloride manufacturing and processing environment).

3. Immunotoxicity

EPA states that ATSDR concludes that immunological effects are "a *suspected* health effect for humans" based on limited evidence in animal studies. Stantec agrees that the evidence in animal studies is significantly limited as only 12 articles were identified by Stantec's review. ATSDR only included six articles in its review, thereby calling into question the completeness and hence, reliability of its weight of evidence determination. Moreover, ATSDR made the determination of suspected immunotoxicity primarily based on three studies that observed effects such as increases in spleen and thymus weights and lymphocyte proliferation in immunized and non-immunized test animals. However, three other studies reviewed by ATSDR either did not observe the same effects or did not observe dose-dependence, even at higher concentrations. In addition, it is not known if these increases in spleen and thymus weight have any biological significance in humans, as no additional investigations have reported on downstream immunotoxic effects nor were there post-treatment observations to determine if there was a resolution of effects. Of note, in one of the studies that observed increased immunoactivity in immunized test animals, downstream antigen-induced immune responses were reportedly not affected by vinyl chloride exposure.

Regarding humans, EPA notes evidence of increased circulating immune complexes, immunoglobulins, complement factors, and levels of inflammatory cytokines in vinyl chloride workers. Stantec reviewed the studies included in the ATSDR profile as well as those identified in the HAWC literature search. It is of note that while EPA identified 37 inhalation studies assessing immunological effects in the Human Health Hazard heat map located in Appendix B.5 of the Draft Scope, Stantec's review of that article list identified only 16 total articles relevant to immunological effects in humans. Stantec suggests EPA further review of its study categorization methodology to ensure that studies are categorized to the correct endpoint, for accurate weight of evidence assessment.

ATSDR defines a *suspected* health effect on the strength of evidence (low or moderate) in humans and in animals. Based on the evidence reviewed, it is likely that ATSDR's designation is a result of *low* level of evidence in animals and *moderate* level of evidence in humans. However, review of the available epidemiological evidence for immunological outcomes suggests a weaker body of evidence for immunological outcomes in humans than may be suggested by ATSDR's conclusion. For example, very few studies reported on the same outcome, leaving endpoint-specific evidence to the basis of one or two individual studies. Further, while approximately half of the human studies Stantec identified were cohort studies, the fundamental study design in epidemiological research, all of the cohort studies were ranked of low or medium quality, due largely to the lack of adequate vinyl chloride exposure assessment, and only one of the studies was published within the last 25 years. Recent and updated evaluations are particularly important for an assessment of immunotoxicity due to the evolution in classification of autoimmune disorders in recent years. Notably, none of the studies related to vinyl chloride exposure assessed modern autoimmune disorders such as rheumatoid arthritis or psoriasis.

4. Developmental Toxicity

In the Draft Scope, EPA noted the conclusions of ATSDR on the developmental effects of vinyl chloride. EPA stated that "developmental effects are a *suspected* health effect for humans based on evidence from studies in mice and rabbits that were exposed via inhalation during gestation. Human data were limited to a small number of ecological and case-control studies that did not report effects."[43]

While there is agreement with the methodology adopted and studies identified by ATSDR, the conclusions of Stantec's review differ based on the overall weight of evidence from animal studies. Specifically, the reviewed high-quality and medium-quality animal studies showed a lack of significant major developmental toxicity following exposure to vinyl chloride; however, a few minor effects, such as delayed ossification were noted in mice, which were not observed in other model species and were coincident with maternal toxicity. Notably, delayed ossification is an effect that may be secondary to maternal toxicity and/or reduced fetal weight secondary to maternal toxicity. The no observed adverse effect concentration (NOAEC) for developmental toxicity was determined to be 1100 ppm (2860 mg/m^3) in the high-quality study reviewed.[44]

With respect to epidemiological studies, Stantec agrees with EPA that human data for developmental toxicity is limited to a small number of ecological and case-control studies. Overall, the quality of this literature is insufficient to draw conclusions regarding the weight of evidence in humans, based on the nature of the studies themselves and their quality ratings during Stantec's independent review (where no studies evaluating developmental toxicity as an endpoint in humans was considered of high quality).

5. Genotoxicity and Cancer

In the Draft Scope, EPA states that "evidence of the carcinogenicity of vinyl chloride in animals is available from inhalation studies in rats, mice, and hamsters and from oral studies in rats. No studies are currently available for dermally exposed animals."[45] Furthermore, EPA's prior assessments and assessments by other agencies have indicated that vinyl chloride is a human carcinogen, based on a combination of human and animal data. Stantec agrees with this general classification but urges EPA to consider specific points regarding the available literature on animal and human studies.

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[43] Draft Scope at 31 (emphasis added).

[44] S. R. Thornton et al., "Embryo-fetal developmental and reproductive toxicology of vinyl chloride in rats," *Toxicol Sci* 68, no. 1 (Jul 2002), https://doi.org/10.1093/toxsci/68.1.207.

[45] Draft Scope at 32.

With respect to animal studies, Stantec agrees with EPA's conclusion on the lack of currently available dermal studies. Otherwise, the animal studies support evidence of carcinogenicity, specifically liver, lung, and mammary gland carcinomas in different species of animals exposed to vinyl chloride. However, Stantec cautions on the higher sensitivity and predisposition of tested mice to cancer following vinyl chloride exposure compared to other animal species based on the studies reviewed by Stantec. Moreover, the use of animal studies to define the dose response for vinyl chloride is likely to overpredict cancer potency, as demonstrated by Reitz et al.,[46] even when accounting for pharmacokinetic differences between animals and humans through use of physiologically-based pharmacokinetic modeling. This discrepancy may be related to considerations associated with mode of action and/or pharmacodynamic differences between animals and humans related to factors such as DNA repair capacity. Therefore, while the current literature is appropriate for hazard identification for vinyl chloride as it relates to carcinogenicity, it may not be optimal, and may be overly protective, for characterizing dose response in humans. Careful consideration of mode of action and species differences should be incorporated when establishing the dose response and establishing potency factors used in the development of benchmarks for the risk evaluation, including occupational exposure values (OEVs).

With respect to epidemiological studies, EPA states that occupational inhalation exposure to vinyl chloride was associated with liver cancer, including angiosarcoma, hepatocellular carcinoma, and cholangiocellular carcinoma. Stantec found 23 studies that assessed liver cancer outcomes, and Stantec agrees that among these studies, there is some evidence of a positive association between vinyl chloride exposure and liver cancer. Specifically, 13 of 23 studies identified a positive association. However, it is of note that, as previously mentioned, none of the 23 studies, including the seven U.S. cohort studies, were conducted with workers exposed solely after the decrease in PEL from 500 ppm to 1 ppm implemented by OSHA in 1975. These are unlikely to be reflective of current exposure levels, considering modern day vinyl chloride manufacturing and processing.

Regarding other types of cancer, EPA states that recent follow-up studies on occupational cohorts and pooled and meta-analysis studies do not support an association between vinyl chloride exposure and brain cancer, lung cancer, soft tissue cancer, lymphatic/hematopoietic cancer, and malignant melanoma.[47] Stantec agrees that there is insufficient scientific evidence of an association between vinyl chloride exposure and brain cancer, lung cancer, soft tissue cancer, lymphatic/hematopoietic cancer, and malignant melanoma. Stantec identified three additional high quality studies not included in EPA's literature review that confirmed a lack of statistically significant associations between vinyl chloride

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[46] R. H. Reitz et al., "Predicting cancer risk from vinyl chloride exposure with a physiologically based pharmacokinetic model," *Toxicol Appl Pharmacol* 137, no. 2 (Apr 1996), https://doi.org/10.1006/taap.1996.0079.

[47] Draft Scope at 32.

exposure and cancers of the brain and central nervous system,[48] as well as one medium[49] and one high quality study[50] that provided additional evidence against a relationship between vinyl chloride exposure and melanoma. It is also of note that Stantec reviewed nine epidemiological studies that evaluated whether or not there was an association between vinyl chloride and breast cancer, and a majority of the studies (n=6), including five high quality studies, concluded that there was insufficient evidence of an association.[51] However, Stantec agrees with EPA's assessment that cancer studies are limited in their application to women or to individuals outside of the workplace due to insufficient consideration of these populations and environments in the current literature.

IV. Environmental Hazard Information

a. Review of Available Environmental Hazard Information

In the Draft Scope, EPA notes that the Agency reviewed ecotoxicity information available in ECOTOXicology Knowledgebase (ECOTOX), previous assessments, and other literature. Based on the

[48] E. Delzell et al., *A Case-control study of intracranial tumors among Amoco Research Center employees who worked in the 500 building complex*, University of Alabama at Birmingham (2000); U. Fedeli et al., "Mortality from liver angiosarcoma, hepatocellular carcinoma, and cirrhosis among vinyl chloride workers," *Am J Ind Med* 62, no. 1 (Jan 2019), https://doi.org/10.1002/ajim.22922; W. Wu et al., "Cohort and case-control analyses of workers exposed to vinyl chloride: an update," *J Occup Med* 31, no. 6 (Jun 1989), https://doi.org/10.1097/00043764-198906000-00007.

[49] V. B. Smulevich, I. V. Fedotova, and V. S. Filatova, "Increasing evidence of the rise of cancer in workers exposed to vinylchloride," *Br J Ind Med* 45, no. 2 (Feb 1988), https://doi.org/10.1136/oem.45.2.93.

[50] Fedeli et al., "Mortality from liver angiosarcoma, hepatocellular carcinoma, and cirrhosis among vinyl chloride workers."

[51] F. J. Bove et al., "Evaluation of mortality among Marines, Navy personnel, and civilian workers exposed to contaminated drinking water at USMC base Camp Lejeune: a cohort study," *Environ Health* 23, no. 1 (Jul 3 2024), https://doi.org/10.1186/s12940-024-01099-7; T. Carreón et al., "Coronary artery disease and cancer mortality in a cohort of workers exposed to vinyl chloride, carbon disulfide, rotating shift work, and o-toluidine at a chemical manufacturing plant," *Am J Ind Med* 57, no. 4 (Apr 2014), https://doi.org/10.1002/ajim.22299; E. Garcia et al., "Hazardous air pollutants and breast cancer risk in California teachers: a cohort study," *Environ Health* 14 (Jan 30 2015), https://doi.org/10.1186/1476-069x-14-14; J. E. Heck et al., "Exposure to outdoor ambient air toxics and risk of breast cancer: The multiethnic cohort," *Int J Hyg Environ Health* 259 (Jun 2024), https://doi.org/10.1016/j.ijheh.2024.114362; J. M. Madrigal et al., "Carcinogenic industrial air pollution and postmenopausal breast cancer risk in the National Institutes of Health AARP Diet and Health Study," *Environ Int* 191 (Sep 2024), https://doi.org/10.1016/j.envint.2024.108985; Smulevich, Fedotova, and Filatova, "Increasing evidence of the rise of cancer in workers exposed to vinylchloride."; K. A. Mundt et al., "Historical cohort study of 10 109 men in the North American vinyl chloride industry, 1942-72: update of cancer mortality to 31 December 1995," *Occup Environ Med* 57, no. 11 (Nov 2000), https://doi.org/10.1136/oem.57.11.774; K. A. Mundt et al., "Quantitative estimated exposure to vinyl chloride and risk of angiosarcoma of the liver and hepatocellular cancer in the US industry-wide vinyl chloride cohort: mortality update through 2013," *Occup Environ Med* 74, no. 10 (Oct 2017), https://doi.org/10.1136/oemed-2016-104051; DHHS and NIOSH, "Health Hazard Evaluation Report. HETA 81-080-11466. Precision Plastics Company," (Philadelphia, PA: Department of Health and Human Services, National Institute for Occupational Safety and Health, 1982).

reviewed information, EPA concluded that exposure to vinyl chloride might cause acute toxicity to aquatic vertebrates (mortality, growth, and behavior) and invertebrates (mortality, reproduction, behavior), chronic toxicity to aquatic vertebrates (mortality) and chronic toxicity to aquatic invertebrates (reproduction and growth/development), and toxicity to algae (growth inhibition).[52] Data gathered within the ECOTOX database identified environmental hazard information for terrestrial invertebrates (mortality, reproduction, and growth/development).[53] In the case of inhalation, which is a potential exposure route for terrestrial wildlife, the relative contribution to total exposure risk is considered to be negligible in most situations. Stantec generally agrees with these assertions, though emphasizes that based on the available toxicity information, the ecotoxicity of vinyl chloride can be characterized as low. For aquatic species, the available toxicity data could be used to categorize vinyl chloride per EPA's ecotoxicity categories for aquatic species. For example, vinyl chloride would be categorized as practically nontoxic/slightly toxic to fish, at most. Additionally, the majority of the available toxicity values for aquatic species are above the concentrations reported in surface water, indicating a low likelihood of risk. For terrestrial species, although less toxicity data are available, consideration of mammalian data from the CompTox Chemicals Dashboard and the use of the EPA Web Interspecies Correlation Estimation (Web-ICE) to estimate toxicity in avian species could provide valuable insights, since no information for these species is available in ECOTOX. Similar to the aquatic environment, the environmental concentrations of vinyl chloride in air and soil are anticipated to be extremely low, indicating that vinyl chloride is highly unlikely to pose a risk to terrestrial species. Lastly, although vinyl chloride has several structural analogs, conducting a read-across analysis is likely to be unnecessary. The physical-chemical properties of vinyl chloride and low environmental concentrations suggest that these data gaps are not data needs because EPA can qualitatively determine there is no risk based on available information. Additional details for aquatic and terrestrial species as well as analogs are provided below.

1. Aquatic Species

Stantec identified available information on acute toxicity to aquatic vertebrates (mortality, growth, and behavior) and invertebrates (mortality, reproduction, behavior), chronic toxicity to aquatic vertebrates (mortality) and chronic toxicity to aquatic invertebrates (reproduction and growth/development), and toxicity to algae (growth inhibition). Based on available information, vinyl chloride would meet EPA classification tiers as practically nontoxic to moderately toxic, depending on the type of organism.

Stantec independently searched the ECOTOX database for toxicity data for vinyl chloride using its CAS number 75-01-4. The available data (as of February 24, 2025) for aquatic species are provided in **Attachment 2**. For fish, median lethal concentration (LC_{50}) values of 1060 and 1220 mg/L (fingerling) and >14.58 mg/L (larva) were reported. In addition, no-response lethal (NR-LETH) values of 388 and

[52] Draft Scope at 30.

[53] Draft Scope at 30.

1680 mg/L were noted for fish, whereas the no-response zero mortality (NR-ZERO) values ranged from 9.72 to 894 mg/L. For crustaceans, an LC_{50} value of >20 mg/L was noted. In addition, the median effective concentration (EC_{50}) of >20 mg/L and NR-ZERO of 20 mg/L were reported for crustaceans. For mollusks, an LC_{50} of >10 mg/L and a median effective concentration (EC_{50}) of >20 mg/L were noted. For invertebrates, the median inhibitory concentration (IC_{50}) values were the only values available, which ranged from 405 to 806 mg/L. For algae, an EC_{50} of 1.17 mg/L and an IC_{50} of 5.15 mg/L were noted. Additional toxicity values available for aquatic species were the no-observed-effect concentrations (NOECs) of <9.72 mg/L (fish), 0.01 mg/L (crustaceans), and 2.5 mg/L (algae), as well as the lowest-observed-effect concentrations (LOECs) of 0.0001 mg/L (crustaceans, although this LOEC was based on changes in mRNA levels, a nuanced effect not traditionally evaluated in aquatic toxicity studies) and 5 mg/L (algae).

For better context, the toxicity values were compared to the EPA ecotoxicity category thresholds for aquatic species defined by the EPA as follows: very highly toxic (<0.1 mg/L), highly toxic (0.1-1 mg/L), moderately toxic (>1-10 mg/L), slightly toxic (>10-100 mg/L), and practically nontoxic (>100 mg/L).[54] Based on the acute toxicity values, vinyl chloride was practically nontoxic/slightly toxic to fish, slightly toxic to crustaceans and mollusks, practically nontoxic to invertebrates, and moderately toxic to algae (**Table 1**).

Table 1. Ecotoxicity Categories for Aquatic Species - Vinyl Chloride

Organism	Toxicity Value (mg/L) from Studies	Ecotoxicity Category
Fish	LC_{50}: 1060, 1220, >14.58	Practically nontoxic – Slightly toxic
Crustaceans	LC_{50}: >20	Slightly toxic
Mollusks	LC_{50}: >10	Slightly toxic
Invertebrates	IC_{50}: 806, 540, 520, 430, 405	Practically nontoxic
Algae	EC_{50}: 1.17	Moderately toxic

It is noteworthy that according to the ToxGuide™ for Vinyl Chloride by the ATSDR,[55] vinyl chloride was detected in approximately 1% of 2385 surface water samples in 2022 and 289 surface water samples in 2023 at average concentrations of 0.0124 and 0.0068 mg/L, respectively; the maximum concentrations were 0.0613 and 0.0077 mg/L in 2022 and 2023. At these averages, vinyl chloride is unlikely to pose risks to aquatic species.

[54] EPA, "Technical Overview of Ecological Risk Assessment - Analysis Phase: Ecological Effects Characterization," available at: https://www.epa.gov/pesticide-science-and-assessing-pesticide-risks/technical-overview-ecological-risk-assessment-0.

[55] ATSDR, "ToxGuide for Vinyl Chloride," available at: https://www.atsdr.cdc.gov/toxguides/toxguide-20.pdf," (2024).

2. Terrestrial Species

In the Draft Scope, EPA rightly notes that no environmental hazard information for terrestrial organisms were identified in previous assessments. Stantec also agrees that the data currently available within the ECOTOX database is predominantly for terrestrial invertebrates (mortality, reproduction, and growth/development). Lastly, Stantec agrees with the EPA that in the case of inhalation, which is a potential exposure route for terrestrial wildlife, the relative contribution to total exposure risk is considered to be negligible in most situations.

Stantec independently searched the ECOTOX database for toxicity data for vinyl chloride using its CAS number. The available data (as of February 24, 2025) for terrestrial species are provided in **Attachment 3**. For mammals, a no observed effect level (NOEL) of 50 mg/kg was noted for an unspecified vinyl chloride formulation. For insects/spiders, NOELs of 10,000 and 100,000 ppm vinyl chloride were noted. In addition, an NR-LETH of 48,500 ppm, a NOEL of 100,000 ppm, and a lowest observed effect level (LOEL) of 200,000 ppm in insects/spiders were noted for an unspecified vinyl chloride formulation. For worms, a LOEL of 1×10^{-8} mol/L was noted for an unspecified vinyl chloride formulation.

According to the CompTox Chemicals Dashboard, the vinyl chloride median lethal dose (LD_{50}) in rats was 500 mg/kg (**Attachment 4**). Additional toxicity data were also available for mammals in the CompTox Chemicals Dashboard, which can be considered in the absence of mammalian data in ECOTOX. Additionally, the EPA Web Interspecies Correlation Estimation (Web-ICE) could be leveraged to estimate toxicity in avian species. As an example, the rat LD_{50} was input into Web-ICE to assess species sensitivity distributions for terrestrial wildlife (**Attachment 5**). The estimated LD_{50} values in birds ranged from 11 mg/kg (Red billed quelea) to 219 mg/kg (Gray partridge); the median LD_{50} across 17 bird species was 101 mg/kg. The estimated LD_{50} values in other mammals ranged from 45 mg/kg (Ricefield rat) to 731 mg/kg (Deer mouse); the median LD_{50} across nine mammalian species was 446 mg/kg. Based on the median LD_{50} values, vinyl chloride was moderately toxic to birds and mammals. Web-ICE also estimated a hazardous dose for 5% of the species (HD_5) of 24 mg/kg.

It is noteworthy that according to the ToxGuide™ for Vinyl Chloride by ATSDR,[56] vinyl chloride was measured in ambient air at 94 sites across the U.S. in 2022, and the air concentrations ranged from below the level of detection to 1.7 $\mu g/m^3$. This air concentration is several orders of magnitude lower than the inhalation toxicity values reported in mammals, indicating that vinyl chloride is unlikely to pose a risk to mammals via inhalation. ATSDR also noted that the maximum vinyl chloride concentrations in surface water were 0.0613 and 0.0077 mg/L in 2022 and 2023, respectively. These concentrations could be used to estimate the doses that would be expected if terrestrial organisms were to ingest water contaminated with vinyl chloride. For example, the estimated the daily water ingestion rate in bobwhite

[56] ATSDR, "ToxGuide for Vinyl Chloride," available at: https://www.atsdr.cdc.gov/toxguides/toxguide-20.pdf.

quail is 0.10-0.13 g/g.[57] Assuming a body weight of 190 g, a bobwhite quail would ingest 19-25 g (or 19-25 mL, using a water density of 1 g/mL) of water per day. Thus, ingestion of water containing 0.0613 mg/L vinyl chloride would result in daily vinyl chloride doses of approximately 0.006-0.008 mg/kg, which are several orders of magnitude lower than the estimated LD_{50} of 51 mg/kg in bobwhite quail as well as the HD_5 of 24 mg/kg, indicating that vinyl chloride was unlikely to pose a risk to bobwhite quail via ingestion of contaminated water. Similar calculations could be conducted for other terrestrial species.

Additionally, according to the European Chemicals Agency (ECHA) dossier, no significant vinyl chloride concentrations are present in the soil and air compartment, which was supported by exposure assessment with the European Union System for the Evaluation of Substances (EUSES).[58] A regional predicted environmental concentration (PEC) in soil of 7.82×10^{-9} mg/kg (dry weight) and a regional PEC in the atmosphere of 3.53×10^{-6} mg/m^3 were calculated. The maximal local PEC value that was calculated for soil was 0.042 mg/kg (dry weight) and for air 0.019 mg/m^3 (for S-PVC production). The registrant concluded that these concentrations were very low and no significant exposure via the soil and air compartment was assumed; thus, studies on terrestrial organisms were not considered necessary.

b. Analog Identification and Read-Across

Several tools are available to identify analogs for vinyl chloride. These include the Structure Search (BETA) available in CompTox Chemicals Dashboard and Organisation for Economic Co-operation and Development (OECD) quantitative structure-activity relationship (QSAR) Toolbox, which provide structural similarity score. As an example, several analogs were identified for vinyl chloride in CompTox Chemicals Dashboard (see **Table 2** for top analogs based on structural similarity). This indicates that read-across might be possible. However, based on the available information, including toxicity data, environmental concentrations, and physicochemical properties, vinyl chloride risks to aquatic and terrestrial species are highly unlikely. Therefore, review of analog data is unlikely to change the conclusion that vinyl chloride is of low risk to aquatic and terrestrial species.

Table 2. Examples of vinyl chloride analogs identified in CompTox Chemicals Dashboard[59]

DTXSID	Name	CASRN	Similarity
DTXSID30986722	Chloro(~2~H_3_)ethene	6745-35-3	1.00
DTXSID30777596	Lithium 1-chloroethen-1-ide	10363-20-9	0.83
DTXSID201339105	(1E)-1,2-Dichloroethene radical ion (1+)	73245-64-4	0.83
DTXSID8024991	1,2-Dichloroethylene	540-59-0	0.83
DTXSID2024030	(Z)-1,2-Dichloroethylene	156-59-2	0.83
DTXSID7024031	(E)-1,2-Dichloroethylene	156-60-5	0.83

[57] EPA, "Wildlife Exposure Factors Handbook" (1993).

[58] ECHA, "European Chemical Agency: Chloroethylene. EC Number: 200-831-0. From: https://echa.europa.eu/it/registration-dossier/-/registered-dossier/16163/6/4/1 " (2023).

[59] Using CompTox Chemicals Dashboard v2.5.2, available at: https://comptox.epa.gov/dashboard/sss.

DTXSID70824018	chloroethene; zinc	78389-90-9	0.83
DTXSID8021438	1,1-Dichloroethylene	75-35-4	0.71
DTXSID00911107	Chloroethene--1,1-dichloroethene (1/1)	109321-18-8	0.71

Furthermore, in the event EPA deems it necessary to conduct analog identification for vinyl chloride data to fill data gaps on terrestrial toxicity, in spite of the low likelihood of exposure via terrestrial pathways (based on the physical-chemical properties of vinyl chloride and overall low reported presence in relevant environmental media), the VC Consortium urges that EPA consider comments previously submitted by VI on analog identification for vinyl industry-related chemistries.[60] Specifically, we urge EPA to consider its selection of tools and evaluate the literature of all suitable analogs, before selecting data upon which to extrapolate to vinyl chloride.

V. Conceptual Models

a. Conceptual Model for Industrial and Commercial Activities and Uses

EPA indicates that it intends to quantitatively assess concentrations of vinyl chloride vapors in industrial and commercial settings where vinyl chloride is present as a liquefied compressed gas. EPA further indicates that for those settings, EPA intends to quantitively assess inhalation exposures of workers and occupational non-users (ONUs) to those vapors. As noted in Section II.b above, EPA should carefully review published data and past industrial hygiene data and ensure that the conditions used in gathering that data reflect current conditions for those COUs.

EPA indicates that it intends to qualitatively assess the liquid contact pathway and the dermal exposure route. EPA states that "sustained or routine dermal exposure to liquid vinyl chloride is not expected."[61] The VC Consortium agrees that because vinyl chloride is manufactured and processed in an enclosed process and because vinyl chloride rapidly vaporizes at standard temperature and pressure, this is not a relevant pathway for exposure and EPA can readily determine that such pathway does not present an unreasonable risk.

EPA should expand the pathways that it assesses qualitatively rather than quantitatively. As EPA correctly states in the Draft Scope, the concentration of residual vinyl chloride monomer (RVCM) in PVC resin and products is very low due to resin stripping.[62] It is on this basis that EPA concludes that workers handling PVC resin will have low to negligible dermal exposure to vinyl chloride. Similar conclusions can be drawn with respect to inhalation exposure to vinyl chloride: the very low presence of vinyl chloride as an impurity in PVC resin precludes meaningful inhalation exposure from PVC resin. Furthermore, downstream PVC products have even lower or undetectable presence of RVCM. In 1995, the VI

[60] *See, e.g.*, Docket No. EPA-HQ-OPPT-2024-0114-0078 at 18-20.

[61] Draft Scope at 36.

[62] Draft Scope at 36.

commissioned a study on extrusion processing of rigid vinyl materials.[63] The study examined the processes and equipment typically used to blend and extrude PVC rigid compounds used for the fabrication of vinyl pipe. The sampling and testing were conducted in accordance with EPA test reference methods and specifications and was reviewed by the Agency. Volatile organic emissions for processing rigid PVC were approximately 0.054 g/kg, but no hazardous pollutants — including vinyl chloride, benzene, and toluene — were found at the levels of detection. Accordingly, it is appropriate for EPA to qualitatively (rather than quantitatively) assess inhalation exposure for these COUs, and EPA can readily determine that this pathway does not present an unreasonable risk. The VC Consortium intends to submit further information to support this approach.

b. Conceptual Model for Consumer Activities and Uses

EPA indicates that it intends to quantitatively evaluate all consumer articles and products that could lead to inhalation exposure. EPA states that RVCM in consumer products could volatilize and lead to inhalation exposure.[64] The VC Consortium is not aware of any data that shows vinyl chloride will volatilize from any PVC product and become a source for inhalation exposure. Rather, any trace levels of RVCM are expected to remain in the product, with any diffusion extremely slow according to Fickian diffusion principles.

EPA identifies two consumer COUs that may contain vinyl chloride up to 15.4 percent and which EPA intends to assess quantitatively with respect to inhalation exposures: (1) adhesives and sealants; and (2) paints and coatings. The VC Consortium believes these numbers to be in error, given that vinyl chloride is a gas at ambient conditions and that adhesives/sealants and paints/coatings are not produced and distributed in a manner that would provide the necessary pressurization and/or refrigeration to keep 15.4 percent by weight of vinyl chloride in a liquid state in a consumer product. It seems more likely that whatever documentation EPA reviewed conflated vinyl chloride and polyvinyl chloride.

EPA indicates that it intends to qualitatively assess oral exposure from the adhesives and sealants COU and the paints and coatings COU because consumers are unlikely to ingest these products. EPA also indicates that it intends to qualitatively assess oral and dermal exposure via consumer products that contain PVC or related polymers. The VC Consortium agrees that it is appropriate to qualitatively assess these COUs, rather than performing a quantitative analysis.

EPA should expand the pathways that it assesses qualitatively rather than quantitatively. As EPA states in the Draft Scope, and as the VC Consortium notes above, the concentration of RVCM in PVC resin and products is very low due to resin stripping, as well as subsequent drying of the resin.[65] Accordingly, it is

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[63] Ernes, D. A., Griffin, J. P., J. Vinyl Add. Technol. (1996) 2 (3), p. 180.

[64] Draft Scope at 38.

[65] Draft Scope at 36.

appropriate for EPA to qualitatively (rather than quantitatively) assess consumer inhalation exposure, and EPA can readily determine that this pathway does not present an unreasonable risk. The VC Consortium intends to submit further information to support this approach.

c. Conceptual Model for Environmental Releases and General Population Exposure

EPA indicates that it intends to quantitively assess concentrations of vinyl chloride in ambient air. As discussed in Section II.a above, emissions to ambient air are appropriately addressed under the CAA, where EPA recently confirmed that there is no unacceptable risk from vinyl chloride manufacturing units.

EPA indicates that it intends to qualitatively assess several exposures pathways and routes from environmental releases of vinyl chloride. First, EPA states that it has already qualitatively assessed the surface water and sediment exposure pathway and determined that no further evaluation is necessary (*i.e.*, it does not present an unreasonable risk).[66] Second, EPA states that it has already qualitatively assessed the potential release of vinyl chloride into landfill leachate and determined that no further evaluation is necessary (*i.e.*, it does not present an unreasonable risk) because release of vinyl chloride into landfill leachate is unlikely to occur from TSCA COUs.[67] Third, EPA states that it has already qualitatively assessed the release of vinyl chloride from TSCA COUs to groundwater and determined that no further evaluation is necessary (*i.e.*, it does not present an unreasonable risk) "because vinyl chloride released to air is not expected to be transported to groundwater and vinyl chloride released to water or land is expected to largely partition to air."[68] Fourth, EPA states that it has already qualitatively assessed potential exposure through drinking water and determined that no further evaluation is necessary (*i.e.*, it does not present an unreasonable risk) because standard industry practices, widespread building codes, and low detected concentrations of vinyl chloride in drinking water at the distribution entry point establish that there is no significant exposure to vinyl chloride through drinking water.[69] Fifth, EPA states that it has already qualitatively assessed potential exposure through soil and determined that no further evaluation is necessary (*i.e.*, it does not present an unreasonable risk) due to the very low releases to land and limited partitioning from other environmental media.[70] Sixth, EPA states that it has already qualitatively assessed potential exposure through vinyl chloride release to terrestrial environments from biosolids application to soil and determined that no further evaluation is necessary (*i.e.*, it does not present an unreasonable risk).[71] Seventh, EPA states that it has already qualitatively

[66] Draft Scope at 40.

[67] Draft Scope at 40-41.

[68] Draft Scope at 41.

[69] Draft Scope at 41.

[70] Draft Scope at 42.

[71] Draft Scope at 42.

assessed potential exposure of and through terrestrial species and aquatic species (including human consumption of those species) and determined that no further evaluation is necessary (*i.e.*, it does not present an unreasonable risk).[72] Finally, EPA states that it has already qualitatively assessed potential exposure to the general population through oral or dermal exposures. As EPA notes, vinyl chloride concentrations in drinking water are below the MCL, and exposure from consumption of aquatic or terrestrial species is expected to be negligible.[73] Additional analysis of these exposure pathways is not warranted in light of EPA's findings.

The VC Consortium agrees with EPA's reasoning and conceptual model for assessing these exposure pathways and routes qualitatively; EPA has sufficient information on which to make these findings. Many of these pathways can be excluded from the need to conduct quantitative risk analyses due to the physical-chemical properties of vinyl chloride.

VI. EPA's Analysis Plan

In general, EPA's Analysis Plan tracks the delineation of quantitative and qualitative assessments noted in the preceding section of these comments. In that aspect, the VC Consortium largely agrees with EPA's proposed approach. Overall, it is critical that EPA approaches its review with transparency and practicality. For example, the VC Consortium agrees that regulatory limits, reporting thresholds, and disposal requirements are relevant to consider for release estimation and environmental response; however, in the absence of data, EPA should not simply assume that releases are at the maximum allowed under these standards. This approach would be extremely conservative and would not reflect TSCA's scientific standards. For example, companies typically design controls for processes with a compliance margin so that variability and minor excursions do not place them in non-compliance. The VC Consortium is able and willing to assist EPA with any questions the Agency may have in this regard.

As noted in Section II.a above and in Attachment 1, EPA is incorrect to expect that vinyl chloride concentrations in ambient air near industrial sites may be significant.[74] Likely concentrations of vinyl chloride in ambient air are very low, if present at all.

For occupational exposures, EPA states that it plans to review workplace monitoring data collected by government agencies such as OSHA and NIOSH, as well as monitoring data in the published literature. EPA is correct to consider the OSHA PEL for vinyl chloride in the occupational exposure assessment. EPA can justifiably assume that all sites are in compliance with the OSHA PEL; however, it is likely that existing monitoring programs have tailored their sampling methods to demonstrating compliance with the PEL. Accordingly, detection limits would be targeted to the 1 ppm 8-hour TWA. In reviewing data,

[72] Draft Scope at 42-43.

[73] Draft Scope at 43.

[74] Draft Scope at 46.

EPA should not assume that non-detect readings reflect concentrations approaching the detection level or apply EPA's standard approach of treating any non-detect value as a reading at one-half of the detection limit.

For occupational exposures, EPA also states that it intends to assess worker exposure pre-implementation and post-implementation of engineering controls using reasonably available information.[75] EPA provides local exhaust ventilation as an example of such controls. While it is not clear the extent to which EPA intends to utilize this approach, this appears to be out of sync with the requirement that EPA assess the COUs as defined in TSCA, *i.e.*, the intended, known, and reasonably foreseeable circumstances of manufacture, processing, and use. Necessarily, vinyl chloride must be manufactured and processed in enclosed systems; thus, it would be illogical and contrary to the TSCA COU definition to excise intrinsic engineering controls from EPA's assessment of the COUs. Moreover, it would be a waste of EPA's limited resources to attempt to construct and assess such an unrealistic scenario. On PPE, EPA has calculated inhalation exposures both before and after the use of PPE (respirators). As this is a straightforward mathematical calculation, it is appropriate for EPA to continue to do so, but EPA's risk evaluation findings must reflect the actual COUs that include mandatory PPE.

VII. EPA's Plan for Peer Review

EPA states that peer review of the draft risk evaluation will be conducted in accordance with the 2024 Final Risk Evaluation Procedural Rule and EPA's Peer Review Handbook.[76] To ensure development of a high quality risk evaluation for vinyl chloride, the VC Consortium urges EPA to submit the draft risk evaluation to the Science Advisory Committee on Chemicals (SACC) for peer review. Review by the SACC is a crucial check on EPA's scientific assessment of toxicology science, and also serves as an important additional opportunity for stakeholders to weigh in on the science underlying EPA's risk evaluations.

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We thank the EPA for its consideration of these comments. Please do not hesitate to contact me for more information or if any questions arise.

Respectfully submitted,

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[75] Draft Scope at 47.

[76] Draft Scope at 56-57.

VIII. List of Attachments

Attachment 1 – Medium and high quality studies not included in the HAWC database

Attachment 2 – Aquatic species toxicity data for vinyl chloride in the ECOTOX database

Attachment 3 – Terrestrial species toxicity data for vinyl chloride in the ECOTOX database

Attachment 4 – CompTox Chemicals Dashboard mortality response values

Attachment 5 – EPA Web Interspecies Correlation Estimation (Web-ICE)

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Attachment 1 - Medium and High Quality Studies Not Included in the HAWC Literature Search

Title	Authors	Year	Endpoint	Journal	Quality Ranking*
Analysis of a Cluster of Cases of Wegener Granulomatosis	Albert, DA; Albert, AN; Vernace, M, et al.	2005	Immunotoxicity	JCR: Journal of Clinical Rheumatology, 11(4), 188-193.	Medium
Angiosarcoma of the liver: annual occurrence and aetiology in Great Britain	Baxter, P. J.; Anthony, P. P. Macsween, et al.	1980	Cancer	Br J Ind Med 37:213-21	Medium
Cytogenetic monitoring of industrial populations potentially exposed to genotoxic chemicals and of control populations	de Jong, G.; van Sittert, N. J.; Natarajan, A. T.	1988	Cancer Biomarker	Mutat Res 204:451-64	Medium
Ten cases of angiosarcoma of the liver in Shawinigan, Quebec	Delorme, F.; Thériault, G.	1978	Cancer	J Occup Med 20:338-40	Medium
A case-control study of intracranial tumors among Amoco Research Center employees who worked in the 500 building complex	Delzell E., Beall C., Rodu B., et al.	2000	Cancer	BP Amoco Coporation	High
Increased morbidity odds ratio of primary liver cancer and cirrhosis of the liver among vinyl chloride monomer workers	Du, C. L.; Wang, J. D.	1998	Cancer; Liver toxicity	Occupational and environmental medicine, 55(8), 528-532.	Medium
Mortality study of workers in a polyvinyl-chloride production plant	Duck, BW; Carter, JT; Coombes, EJ.	1975	Cancer	The Lancet, 306(7946), 1197-1199.	Medium
Angiosarcoma of the liver in Great Britain in proximity to vinyl chloride sites	Elliott, P.; Kleinschmidt, I.	1997	Cancer	Occupational and environmental medicine, 54(1), 14-18.	Medium
Hepatic disease among workers at a vinyl chloride polymerization plant	Falk H, Creech JL, Heath CW, Johnson MN, Key MM	1974	Cancer; Liver toxicity	Jama, 230(1), 59-63.	Medium
Mortality from liver angiosarcoma, hepatocellular carcinoma, and cirrhosis among vinyl chloride workers	Fedeli, U.; Girardi, P.; Gardiman, G., et al.	2019	Cancer; Liver Toxicity	American journal of industrial medicine, 62(1), 14-20.	High

Title	Authors	Year	Endpoint	Journal	Quality Ranking*
Mutations in apoptotic genes and micronucleus occurrence in vinyl chloride-exposed workers in China	Feng, N; Zheng, G.; Hao, Y., et al.	2017	Cancer Biomarker	Environmental and molecular mutagenesis, 58(1), 39-45	Medium
Mortality experience of workers exposed to vinyl chloride monomer in the manufacture of polyvinyl chloride in Great Britain	Fox, A. J.; Collier, P. F.	1977	Cancer; Liver toxicity	Occupational and Environmental Medicine, 34(1), 1-10.	Medium
Relationship between locations of chromosome breaks induced by vinyl chloride monomer and lymphocytosis	Fucić A, Hitrec V, Garaj-Vrhovac V, et al.	1995	Immunotoxicity	American journal of industrial medicine, 27(4), 565-571.	Medium
A nine-year follow up study of a population occupationally exposed to vinyl chloride monomer	Fucić, A.; Barković, D.; Garaj-Vrhovac, V, et al.	1996	Cancer Biomarker	Mutation Research/Environmental Mutagenesis and Related Subjects, 361(1), 49-53.	Medium
Mortality for Lung Cancer among PVC Baggers Employed in the Vinyl Chloride Industry	Girardi, P.; Barbiero, F.; Baccini, M., et al.	2022	Cancer	International Journal of Environmental Research and Public Health, 19(10), 6246.	Medium
Occupational exposures at a polyvinyl chloride production facility are associated with significant changes to the plasma metabolome	Guardiola, J. J.; Beier, J. I.; Falkner, K. C., et al.	2016	Liver Toxicity Biomarkers	Toxicology and applied pharmacology, 313, 47-56	Medium
Exposure to outdoor ambient air toxics and risk of breast cancer: The multiethnic cohort	Heck, J. E.; He, D.; Wing, S. E., et al.	2024	Cancer	International Journal of Hygiene and Environmental Health, 259, 114362.	High
Persistent liver dysfunction among workers at a vinyl chloride monomer polymerization plant	Ho, S. F.; Phoon, W. H.; Gan, S. L., et al.	1991	Liver Toxicity Biomarkers	Occupational Medicine, 41(1), 10-16.	Medium
Exposure to vinyl chloride monomer: results of a cohort study after a seven year follow up. The French VCM Group.	Laplanche, A., Clavel-Chapelon, F., Contassot, J. C., et al.	1992	Immunotoxicity	Occupational and Environmental Medicine, 49(2), 134-137.	Medium
Vinyl chloride and liver and brain cancer at a polymer production plant in Louisville, Kentucky	Lewis, R.; Rempala, G.; Dell, L. D., et al.	2003	Cancer	Journal of occupational and environmental medicine, 45(5), 533-537.	Medium

Title	Authors	Year	Endpoint	Journal	Quality Ranking*
A case-cohort study of angiosarcoma of the liver and brain cancer at a polymer production plant	Lewis, R.; Rempala, G.	2003	Cancer	Journal of occupational and environmental medicine, 45(5), 538-545.	High
Polymorphisms in glutathione S-transferases in French vinyl chloride workers	Li, Y.; Zhou, M.; Marion, M. J., et al.	2005	Cancer Biomarker	Biomarkers, 10(1), 72-79.	Medium
Polymorphisms in the p53 pathway genes and micronucleus occurrence in Chinese vinyl chloride-exposed workers	Li, Y.; Feng, N. N.; Zhang, G. H., et al.	2013	Cancer Biomarker	International journal of occupational medicine and environmental health, 26, 825-836.	Medium
Gene-environment interactions between DNA repair polymorphisms and exposure to the carcinogen vinyl chloride	Li, Y.; Marion M. J.; Zipprich, J., et al.	2009	Cancer Biomarker	Biomarkers, 14(3), 148-155.	Medium
Spontaneous abortions among women employed in the plastics industry	Lindbohm, M. L.; Hemminki, K.; Kyyrönen, P.	1985	Reproductive/ Developmental	American journal of industrial medicine, 8(6), 579-586.	Medium
Western diet unmasks transient low-level vinyl chloride-induced tumorigenesis; potential role of the (epi-) transcriptome	Liu S.; He L.; Bannister O. B, et al.	2023	Cancer/Genotoxicity	Toxicology and applied pharmacology, 468, 116514.	Medium
Carcinogenic industrial air pollution and postmenopausal breast cancer risk in the National Institutes of Health AARP Diet and Health Study	Madrigal, J. M.; Pruitt, C. N. Fisher, et al.	2024	Cancer	Environment international, 191, 108985.	High
Liver function assessment in workers exposed to vinyl chloride	Maroni, M.; Fanetti, A. C.	2006	Liver Toxicity Biomarkers	International archives of occupational and environmental health, 79, 57-65.	Medium
Unusual splenomegalic liver disease as evidenced by peritoneoscopy and guided liver biopsy among polyvinyl chloride production workers	Marsteller HJ, Lelbach WK, Muller R, et al.	1975	Liver Toxicity Biomarkers	Annals of the New York Academy of Sciences, 246(1), 95-134.	Medium
Proportional mortality among vinyl chloride workers	Monson, R. R.; Peters, J. M.; Johnson, M. N.	1975	Cancer	The Lancet, 304(7877), 397-398.	Medium

Title	Authors	Year	Endpoint	Journal	Quality Ranking*
Exposures to polyvinyl chloride, methyl ketone and other chemicals. The pulmonary and non-pulmonary effect	Oleru, U. G.; Onyekwere, C.	1992	Neurological	International archives of occupational and environmental health, 63, 503-507.	Medium
Lack of mutagenic effect of vinyl chloride monomer in the mammalian spot test	Peter S, Ungvary G.	1980	Genotoxicity	Mutation Research/Genetic Toxicology, 77(2), 193-196.	Medium
Vinyl chloride cytogenetics	Picciano DJ, Flake RE, Gay PC, et al.	1977	Cancer Biomarker	Journal of Occupational Medicine, 527-530.	Medium
Alterations of liver and spleen among workers exposed to vinyl chloride	Popper H, Thomas LB.	1975	Cancer; Liver toxicity	Annals of the New York Academy of Sciences, 246, 172-194.	Medium
Messenger RNA expression and genetic polymorphisms of cell cycle control genes and chromosomal aberrations in Chinese vinyl chloride monomer-exposed workers	Qiu, Y. L.; Sun, P.; Wang, W, et al.	2011	Cancer Biomarker	Journal of occupational and environmental medicine, 53(12), 1442-1446.	Medium
A case of polymyositis with anti-histidyl-t-RNA synthetase (Jo-1) antibody syndrome following extensive vinyl chloride exposure.	Serratrice, J., Granel, B., Pache, X, et al.	2001	Immunotoxicity	Clinical rheumatology, 20, 379-382.	High
Increasing evidence of the rise of cancer in workers exposed to vinyl chloride	Smulevich VB, Fedotova IV, Filatova VS.	1988	Cancer	Occupational and Environmental Medicine, 45(2), 93-97.	Medium
Effects of vinyl chloride on liver function of exposed workers, evaluated by measurements of plasma clearance of the 99mTc-N-2,4-dimethylacetanilido-iminodiacetate complex	Studniarek, M.; Durski, K.; Liniecki, J, et al.	1989	Liver Toxicity Biomarkers	Journal of applied toxicology, 9(4), 213-21	Medium
Early hepatic histologic alterations among chemical (vinyl monomer) workers	Tamburro CH, Makk L, Popper H.	1984	Liver Toxicity Biomarkers	Hepatology, 4(3), 413-418	Medium

Title	Authors	Year	Endpoint	Journal	Quality Ranking*
Mortality surveillance in a large chemical company: the Union Carbide Corporation experience, 1974-1983	Teta, M. J.; Schnatter, A. R.; Ott, M. G, et al.	1990	Cancer	American journal of industrial medicine, 17(4), 435-447.	Medium
The toxicity of vinyl chloride as determined by repeated exposure of laboratory animals	Torkelson TR, Oyen F, Rowe VK	1961	Liver toxicity	American Industrial Hygiene Association Journal, 22(5), 354-361	High
Genetic polymorphisms of XRCC1, HOGG1 and MGMT and micronucleus occurrence in Chinese vinyl chloride-exposed workers	Wang, Q.; Fang, J.; Sun, Y, et al.	2010	Cancer Biomarker	Carcinogenesis, 31(6), 1068-1073	Medium
Role of endoplasmic reticulum stress and oxidative stress in vinyl chloride-induced hepatic steatosis in mice	Wang, Q.; Zhang L.; Chen, S.Q.; Ma W.Y.; Guo Y.L.; Gao Y.; Tian, F.J.; Qiu Y. L.	2019	Liver toxicity	Toxicology and Applied Pharmacology, 381, 114730.	High
Micronucleus occurrence related to base excision repair gene polymorphisms in Chinese workers occupationally exposed to vinyl chloride monomer	Wen-Bin, M. ; Wei, W.; Yu-Lan, Q.; Fang, J.; Zhao-Lin, X.	2009	Cancer Biomarker	Journal of occupational and environmental medicine, 51(5), 578-585	Medium
An increased standardized mortality ratio for liver cancer among polyvinyl chloride workers in Taiwan	Wong RH, Chen PC, Du CL, et al.	2002	Cancer; Liver Toxicity	Occupational and Environmental Medicine, 59(6), 405-409	High
Cohort and case-control analyses of workers exposed to vinyl chloride: an update	Wu W, Steenland K, Brown D, et al.	1989	Cancer; Liver Toxicity	Journal of Occupational and Environmental Medicine, 31(6), 518-523.	High
Exposure to ambient air toxicants and the risk of amyotrophic lateral sclerosis (ALS): A matched case control study	Wu, F.; Malek, A. M.; Buchanich, J. M., et al.	2024	Neurological	Environmental Research, 242, 117719.	High

Title	Authors	Year	Endpoint	Journal	Quality Ranking*
Liver fibrosis associated with potential vinyl chloride and ethylene dichloride exposure from the petrochemical industry	Yuan TH, Chen JL, Shie RH, Yeh YP, Chen YH, Chan CC	2020	Liver Toxicity (Biomarkers)	Science of The Total Environment, 739, 139920.	High
Association of Telomere Length With Chromosomal Damage Among Chinese Workers Exposed to Vinyl Chloride Monomer	Zheng, G. Q. ; Zhang, G. H.; Xu, X. W, et al.	2017	Cancer Biomarker	J Occup Environ Med 59:e252-e256	High
Relative telomere length and gene expression of shelterin complex proteins among vinyl chloride monomer-exposed workers in China	Zheng, G. Q.; Zhang, G. H.; Wu, H. T., et al.	2019	Cancer Biomarker	Environ Mol Mutagen 60:361-367	High
Evaluation in vinyl chloride monomer-exposed workers and the relationship between liver lesions and gene polymorphisms of metabolic enzymes	Zhu, S. M.; Ren, X. F.; Wan, J. X, et al.	2005	Liver toxicity	World J Gastroenterol 11:5821-7	Medium

*For the purposes of the Stantec's assessment, study quality was assessed independent of how it was anticipated the study would be used. Therefore, some studies may have been identified as high or medium quality based on the study characteristics and therefore have some utility in a weight of evidence hazard assessment of vinyl chloride (acknowledging study limitations) but may not be well-suited for other purposes, such as dose response assessment of vinyl chloride (*e.g.*, environmental epidemiology studies that involve multiple potential exposures, or other similarly designed studies).

Attachment 2 - ECOTOX Aquatic Species Data - Vinyl Chloride (75-01-4)

Chemical Analysis	Chemical Purity Mean(%)	Species Scientific Name	Species Common Name	Species Group	Organism Lifestage	Organism Age Mean (Hours)	Exposure Type	Media Type	Number of Doses	Conc 1 Type (Standardized)	Conc 1 Mean or Min-Max (Standardized; AI mg/L)	Effect	Effect Measurement	Endpoint	Observed Duration Mean or Min-Max (Days)	BCF 1 Value (RA)	Reference Number
Unmeasured values (some measured values reported in article)	>98	Chlorella fusca var. vacuolata	Green Algae	Algae		-		Fresh water	1	Active ingredient	0.05	Accumulation	Residue	BCF	1	40	3782
Measured	>99	Oedogonium cardiacum	Green Algae	Algae		-	Static	Fresh water		Active ingredient	0.04174	Accumulation	Residue		3	-	7564
Unmeasured	99.9	Raphidocelis subcapitata	Green Algae	Algae; Standard Test Species	Exponential growth phase (log)	-		Fresh water	7	Active ingredient	1.17	Population	Abundance	EC50	2	-	176965
Unmeasured	99.9	Raphidocelis subcapitata	Green Algae	Algae; Standard Test Species		-	Static	Fresh water	6	Active ingredient	5.15	Population	Abundance	IC50	2	-	176871
Unmeasured	99.9	Raphidocelis subcapitata	Green Algae	Algae; Standard Test Species		-	Static	Fresh water	6	Active ingredient	5	Population	Abundance	LOEC	2	-	176871
Unmeasured	99.9	Raphidocelis subcapitata	Green Algae	Algae; Standard Test Species		-	Static	Fresh water	6	Active ingredient	2.5	Population	Abundance	NOEC	2	-	176871
Unmeasured	99.9	Moina macrocopa	Water Flea	Crustaceans	Juvenile	24 Hours		Fresh water	3	Active ingredient	>20	Intoxication	Immobile	EC50	2	-	176965
Unmeasured	99.9	Moina macrocopa	Water Flea	Crustaceans	Juvenile	24 Hours		Fresh water	3	Active ingredient	>20	Mortality	Mortality	LC50	2	-	176965
Unmeasured	99.9	Moina macrocopa	Water Flea	Crustaceans	Juvenile	24 Hours		Fresh water	3	Active ingredient	20	Mortality	Mortality	NR-ZERO	2	-	176965
Unmeasured	-	Daphnia magna	Water Flea	Crustaceans; Standard Test Species	Neonate	24 Hours	Renewal	Fresh water	-	Formulation	0.0001	Genetics	Vitelline outer layer membrane protein 1 mRNA	LOEC	10	-	174676
Unmeasured	-	Daphnia magna	Water Flea	Crustaceans; Standard Test Species	Neonate	24 Hours	Renewal	Fresh water	-	Formulation	0.0001	Genetics	Juvenile hormone esterase isoform A mRNA	LOEC	10	-	174676
Unmeasured	-	Daphnia magna	Water Flea	Crustaceans; Standard Test Species	Neonate	24 Hours	Renewal	Fresh water	-	Formulation	0.0001	Genetics	Glutathione S-transferase mRNA	LOEC	10	-	174676
Unmeasured	-	Daphnia magna	Water Flea	Crustaceans; Standard Test Species	Neonate	24 Hours	Renewal	Fresh water	-	Formulation	0.01	Genetics	Nitric oxide synthase 2 mRNA	NOEC	10	-	174676
Unmeasured	-	Daphnia magna	Water Flea	Crustaceans; Standard Test Species	Neonate	24 Hours	Renewal	Fresh water	-	Formulation	0.01	Reproduction	Time to first progeny	NOEC	10	-	174676
Unmeasured	-	Daphnia magna	Water Flea	Crustaceans; Standard Test Species	Neonate	24 Hours	Renewal	Fresh water	-	Formulation	0.01	Genetics	Vitellogenin 1 mRNA	NOEC	10	-	174676

Chemical Analysis	Chemical Purity Mean(%)	Species Scientific Name	Species Common Name	Species Group	Organism Lifestage	Organism Age Mean (Hours)	Exposure Type	Media Type	Number of Doses	Conc 1 Type (Standardized)	Conc 1 Mean or Min-Max (Standardized; AI mg/L)	Effect	Effect Measurement	Endpoint	Observed Duration Mean or Min-Max (Days)	BCF 1 Value (RA)	Reference Number
Unmeasured	-	Daphnia magna	Water Flea	Crustaceans; Standard Test Species	Neonate	24 Hours	Renewal	Fresh water	-	Formulation	0.01	Genetics	Vitellogenin 2 mRNA	NOEC	10	-	174676
Unmeasured	-	Daphnia magna	Water Flea	Crustaceans; Standard Test Species	Neonate	24 Hours	Renewal	Fresh water	-	Formulation	0.01	Genetics	Ecdysone receptor B isoform mRNA	NOEC	10	-	174676
Unmeasured	-	Daphnia magna	Water Flea	Crustaceans; Standard Test Species	Neonate	24 Hours	Renewal	Fresh water	-	Formulation	0.01	Reproduction	Progeny counts/numbers	NOEC	10	-	174676
Unmeasured	-	Daphnia magna	Water Flea	Crustaceans; Standard Test Species	Neonate	24 Hours	Renewal	Fresh water	-	Formulation	0.01	Enzyme(s)	Juvenile-hormone esterase	NOEC	10	-	174676
Unmeasured	-	Daphnia magna	Water Flea	Crustaceans; Standard Test Species	Neonate	24 Hours	Renewal	Fresh water	-	Formulation	0.01	Reproduction	Progeny counts/numbers	NOEC	10	-	174676
Unmeasured	-	Daphnia magna	Water Flea	Crustaceans; Standard Test Species	Neonate	24 Hours	Renewal	Fresh water	-	Formulation	0.01	Genetics	Ecdysone receptor A1 mRNA	NOEC	10	-	174676
Unmeasured	-	Daphnia magna	Water Flea	Crustaceans; Standard Test Species	Neonate	24 Hours	Renewal	Fresh water	-	Formulation	0.01	Growth	Growth, general	NOEC	10	-	174676
Unmeasured	-	Daphnia magna	Water Flea	Crustaceans; Standard Test Species	Neonate	24 Hours	Renewal	Fresh water	-	Formulation	0.01	Genetics	Retinoid X Receptor alpha mRNA	NOEC	10	-	174676
Measured	>99	Daphnia magna	Water Flea	Crustaceans; Standard Test Species		-	Static	Fresh water	-	Active ingredient	0.04174	Accumulation	Residue		3	-	7564
Unmeasured values (some measured values reported in article)	>98	Leuciscus idus ssp. melanotus	Carp	Fish		-		Fresh water	1	Active ingredient	0.05	Accumulation	Residue	BCF	3	10	3782
Measured	-	Micropterus salmoides	Largemouth Bass	Fish	Fingerling	-	Static	Fresh water	5	Active ingredient	1060	Mortality	Survival	LC50	4	-	181061
Measured	-	Micropterus salmoides	Largemouth Bass	Fish	Fingerling	-	Static	Fresh water	5	Active ingredient	647-2185	Behavior	Multiple measurement terms entered		0.0417-1	-	181061
Measured	-	Micropterus salmoides	Largemouth Bass	Fish	Fingerling	-	Static	Fresh water	5	Active ingredient	647-2186	Physiology	Respiration		4	-	181061
Measured	>99	Gambusia affinis	Western Mosquitofish	Fish		-	Static	Fresh water		Active ingredient	0.04174	Accumulation	Residue		3	-	7564
Measured	-	Lepomis macrochirus	Bluegill	Fish; Standard Test Species	Fingerling	-	Static	Fresh water	5	Active ingredient	1220	Mortality	Survival	LC50	4	-	181061

Chemical Analysis	Chemical Purity Mean(%)	Species Scientific Name	Species Common Name	Species Group	Organism Lifestage	Organism Age Mean (Hours)	Exposure Type	Media Type	Number of Doses	Conc 1 Type (Standardized)	Conc 1 Mean or Min-Max (Standardized; AI mg/L)	Effect	Effect Measurement	Endpoint	Observed Duration Mean or Min-Max (Days)	BCF 1 Value (RA)	Reference Number
Measured	99.9	Oryzias latipes	Japanese Medaka	Fish; Standard Test Species	Larva	7 Days		Fresh water	3	Active ingredient	>14.58	Mortality	Mortality	LC50	4	-	176965
Unmeasured	99.9	Oryzias latipes	Japanese Medaka	Fish; Standard Test Species	Multiple	-		Fresh water	3	Active ingredient	<9.72	Multiple	Multiple effects reported as one result	NOEC	14	-	176965
Measured	-	Lepomis macrochirus	Bluegill	Fish; Standard Test Species	Fingerling	-	Static	Fresh water	5	Active ingredient	1680	Mortality	Mortality	NR-LETH	0.0417-0.25	-	181061
Measured	-	Esox lucius	Northern Pike	Fish; Standard Test Species		-	Renewal	Fresh water	-	Active ingredient	388	Mortality	Mortality	NR-LETH	10	-	176992
Measured	99.9	Oryzias latipes	Japanese Medaka	Fish; Standard Test Species	Multiple	-		Fresh water	3	Active ingredient	9.72	Mortality	Mortality	NR-ZERO	14	-	176965

Attachment 3 – ECOTOX Terrestrial Data – Vinyl Chloride (75-01-4)

Species Group	Chemical Purity Mean(%)	Organism Lifestage	Organism Age Mean or Min-Max	Exposure Type	Media Type	Number of Doses	Conc 1 Type (Author)	Conc 1 Mean or Min-Max	Conc 1 Units (Author)	Effect	Effect Measurement	Endpoint	Observed Response Mean or Min-Max	Observed Response Units	Response Site	Observed Duration (Days)	Reference Number
Worms; Standard Test Species	99.9		-	Culture medium	Culture	7	Active ingredient	15	mg/L	Mortality	Mortality	LC50	15	mg/L		1	176965
Worms; Standard Test Species	99.9		-	Culture medium	Culture	4	Active ingredient	80	mg/L	Mortality	Mortality	LC50	80	mg/L		3	176965
Worms; Standard Test Species	99.9		-	Culture medium	Culture	7	Active ingredient	15	mg/L	Mortality	Mortality	NR-ZERO	15	mg/L		1	176965
Worms; Standard Test Species	99.9	Adult	-	Culture medium	Culture	7	Active ingredient	60	mg/L	Mortality	Mortality	NR-ZERO	60	mg/L		3	176871
Worms; Standard Test Species	99.9		-	Culture medium	Culture	4	Active ingredient	80	mg/L	Mortality	Mortality	NR-ZERO	80	mg/L		3	176965
Worms; Standard Test Species	99.9	Adult	-	Culture medium	Culture	8	Active ingredient	10-60	mg/L	Genetics	Gene expression		10-60	mg/L		3	176871
Flowers, Trees, Shrubs, Ferns	-	Mature dormant	-	Fumigation			Formulation	1000	ppm	Development	Maturity		1000	ppm	Bud	42	41203
Flowers, Trees, Shrubs, Ferns	-	Mature dormant	-	Fumigation			Formulation	1000	ppm	Physiology	Dormancy break change, plants		1000	ppm	Bud	42	41203
Insects/Spiders	-		0-2 Days	Environmental, unspecified	No substrate	2	Formulation	200000	ppm	Genetics	X-linked recessive lethal	LOEL	200000	ppm		6	66224
Insects/Spiders			0-2 Days	Environmental, unspecified	No substrate	2	Formulation	200000	ppm	Genetics	X-linked recessive lethal	LOEL	200000	ppm		6	66224
Insects/Spiders	99.995		0-2 Days	Environmental, unspecified	Agar	2	Active ingredient	100000	ppm	Genetics	X-linked recessive lethal	NOEL	100000	ppm		0-3	66224
Insects/Spiders	99.995		0-2 Days	Environmental, unspecified	Agar	2	Active ingredient	10000	ppm	Genetics	X-linked recessive lethal	NOEL	10000	ppm		0-3	66224
Insects/Spiders	99.995		0-2 Days	Environmental, unspecified	Agar	2	Active ingredient	10000	ppm	Genetics	X-linked recessive lethal	NOEL	10000	ppm		4-6	66224
Insects/Spiders	-		0-2 Days	Environmental, unspecified	No substrate	3	Formulation	100000	ppm	Genetics	X-linked recessive lethal	NOEL	100000	ppm		6	66224
Insects/Spiders	99.995		0-2 Days	Environmental, unspecified	Agar	2	Active ingredient	10000	ppm	Genetics	X-linked recessive lethal	NOEL	10000	ppm		7-9	66224
Insects/Spiders	99.995		0-2 Days	Environmental, unspecified	Agar	2	Active ingredient	100000	ppm	Genetics	X-linked recessive lethal	NOEL	100000	ppm		4-6	66224
Insects/Spiders	99.995		0-2 Days	Environmental, unspecified	Agar	2	Active ingredient	100000	ppm	Genetics	X-linked recessive lethal	NOEL	100000	ppm		7-9	66224
Insects/Spiders	-	Adult	-	Environmental, unspecified	No substrate	2	Formulation	48500	ppm	Mortality	Mortality	NR-LETH	48500	ppm		2	176688
Insects/Spiders	-		0-2 Days	Environmental, unspecified	No substrate	3	Formulation	10000-100000	ppm	Genetics	X-linked recessive lethal		10000-100000	ppm		6	66224
Insects/Spiders	-	Adult	-	Environmental, unspecified	No substrate	2	Formulation	48500	ppm	Mortality	Mortality		48500	ppm		2	176688

Species Group	Chemical Purity Mean(%)	Organism Lifestage	Organism Age Mean or Min-Max	Exposure Type	Media Type	Number of Doses	Conc 1 Type (Author)	Conc 1 Mean or Min-Max	Conc 1 Units (Author)	Effect	Effect Measurement	Endpoint	Observed Response Mean or Min-Max	Observed Response Units	Response Site	Observed Duration (Days)	Reference Number
Insects/Spiders	-	Adult	-	Environmental, unspecified	No substrate	2	Formulation	48500	ppm	Mortality	Mortality		48500	ppm		2	176688
Insects/Spiders	-	Adult	-	Environmental, unspecified	No substrate	2	Formulation	48500	ppm	Mortality	Mortality		48500	ppm		2	176688
Insects/Spiders	-	Adult	-	Environmental, unspecified	No substrate	2	Formulation	48500	ppm	Mortality	Mortality		48500	ppm		2	176688
Insects/Spiders	-	Adult	-	Environmental, unspecified	No substrate	2	Formulation	48500	ppm	Genetics	Mutation		48500	ppm		2	176688
Insects/Spiders	-	Adult	-	Environmental, unspecified	No substrate	2	Formulation	48500	ppm	Mortality	Mortality		48500	ppm		2	176688
Insects/Spiders	-	Adult	-	Environmental, unspecified	No substrate	2	Formulation	48500	ppm	Mortality	Mortality		48500	ppm		2	176688
Insects/Spiders	-	Adult	42-48 hours	Environmental, unspecified	No substrate	5	Formulation	625-5000	ppm	Behavior	Distance moved, change in direct movement		625-5000	ppm		0.3333	176695
Insects/Spiders	-	Adult	-	Environmental, unspecified	No substrate	2	Formulation	48500	ppm	Genetics	Abnormal chromosomal distribution		48500	ppm		2	176688
Insects/Spiders	-	Adult	-	Environmental, unspecified	No substrate	2	Formulation	48500	ppm	Genetics	Abnormal chromosomal distribution		48500	ppm		2	176688
Insects/Spiders	-	Adult	42-48 hours	Environmental, unspecified	No substrate	4	Formulation	100-10000	ppm	Behavior	Distance moved, change in direct movement		100-10000	ppm		0.0417-0.25	176695
Insects/Spiders	-	Adult	-	Environmental, unspecified	No substrate	2	Formulation	48500	ppm	Genetics	Mutation		48500	ppm		2	176688
Flowers, Trees, Shrubs, Ferns; U.S. Invasive Species	-	Mature dormant	-	Fumigation		-	Formulation	4000	ppm	Physiology	Dormancy break change, plants		4000	ppm	Bud	42	41203
Flowers, Trees, Shrubs, Ferns; U.S. Invasive Species	-	Mature dormant	-	Fumigation		-	Formulation	4000	ppm	Development	Maturity		4000	ppm	Bud	42	41203
Mammals; Standard Test Species	98	Not intact	-	In Vitro	Fresh water	6	Active ingredient	510	mg/L	Biochemistry	Protein content	IC50	510	mg/L		1	112973
Mammals; Standard Test Species	98	Not intact	-	In Vitro	Fresh water	6	Active ingredient	495	mg/L	Cell(s)	Viability	IC50	495	mg/L		1.125	112973
Mammals; Standard Test Species	98	Not intact	-	In Vitro	Fresh water	6	Active ingredient	425	mg/L	Physiology	Standard Metabolic Rate	IC50	425	mg/L		1.125	112973
Mammals; Standard Test Species	98	Not intact	-	In Vitro	Fresh water	6	Active ingredient	480	mg/L	Genetics	RNA synthesis rate	IC50	480	mg/L		1	112973

Species Group	Chemical Purity Mean(%)	Organism Lifestage	Organism Age Mean or Min-Max	Exposure Type	Media Type	Number of Doses	Conc 1 Type (Author)	Conc 1 Mean or Min-Max	Conc 1 Units (Author)	Effect	Effect Measurement	Endpoint	Observed Response Mean or Min-Max	Observed Response Units	Response Site	Observed Duration (Days)	Reference Number
Mammals; Standard Test Species	-		6 weeks	Diet, unspecified	No substrate	3	Formulation	25	mg/kg	Morphology	Organ weight in relationship to body weight	LOEL	25	mg/kg	Liver	3	111454
Mammals; Standard Test Species	-		6 weeks	Diet, unspecified	No substrate	3	Formulation	50	mg/kg	Biochemistry	Bilirubin		50	mg/kg	Serum	3	111454
Mammals; Standard Test Species	-		6 weeks	Diet, unspecified	No substrate	3	Formulation	50	mg/kg	Enzyme(s)	Aspartate aminotransferase		50	mg/kg	Serum	3	111454
Mammals; Standard Test Species	-		6 weeks	Diet, unspecified	No substrate	3	Formulation	25-50	mg/kg	Histology	Multiple measurement terms entered		25-50	mg/kg	Liver	3	111454
Mammals; Standard Test Species	-		6 weeks	Diet, unspecified	No substrate	3	Formulation	25-50	mg/kg	Genetics	Gene expression		25-50	mg/kg	Liver	3	111454
Worms	-	Juvenile	-	Environmental, unspecified	Culture	7	Formulation	0.00000001	mol/L	Development	Slowed, Retarded, Delayed or Non-development		0.00000001	mol/L		4	18349
Worms	-	Juvenile	-	Environmental, unspecified	Culture	7	Formulation	0.00000001-0.001	mol/L	Mortality	Survival		0.00000001-0.001	mol/L		4	18349
Worms	-	Juvenile	-	Environmental, unspecified	Culture	4	Formulation	0.00000001-0.0001	mol/L	Genetics	Mutation		0.00000001-0.0001	mol/L		9	18349
Flowers, Trees, Shrubs, Ferns	-	Mature dormant	-	Fumigation		-	Formulation	10000	ppm	Physiology	Dormancy break change, plants		10000	ppm	Bud	39	41203
Flowers, Trees, Shrubs, Ferns	-	Mature dormant	-	Fumigation		-	Formulation	10000	ppm	Development	Maturity		10000	ppm	Multiple entries	39	41203
Flowers, Trees, Shrubs, Ferns; U.S. Invasive Species	-	Mature dormant	-	Fumigation		-	Formulation	1000	ppm	Development	Maturity		1000	ppm	Multiple entries	10	41203
Flowers, Trees, Shrubs, Ferns; U.S. Invasive Species	-	Mature dormant	-	Fumigation		-	Formulation	0.1	cm3/l	Development	Maturity		0.1	cm3/l	Bud	40	41203
Flowers, Trees, Shrubs, Ferns; U.S. Invasive Species	-	Mature dormant	-	Fumigation		-	Formulation	0.1	cm3/l	Physiology	Dormancy induction, plants		0.1	cm3/l	Bud	40	41203

Attachment 4 - CompTox Chemicals Dashboard Mortality Response Values - Vinyl Chloride (75-01-4)

Type	Value	Units	Exposure Route	Species	Source
LD_{50}	500	mg/kg	Oral	Rat	NLM ChemID Plus
LC_{50}	460,125	mg/m^3	Inhalation	Rat	NLM ChemID Plus

Attachment 5 - EPA Web Interspecies Correlation Estimation (Web-ICE) - Vinyl Chloride (75-01-4)

Surrogate Species	Surrogate Toxicity – LD_{50} (mg/kg)	Estimated Animal Class	Estimated Animal Common Name	Estimated Toxicity – LD_{50} (mg/kg)
Norway Rat	500	Birds	Redwinged blackbird	77.11
			Chukar	121.95
			Red-legged partridge	172.67
			Mallard	211.67
			Canada goose	73.47
			House finch	29.88
			Northern bobwhite	50.72
			Rock dove	41.48
			Japanese quail	195.67
			Chicken	175.51
			House sparrow	174.99
			Gray partridge	219.2
			Ring-necked pheasant	97.95
			Red billed quelea	10.72

Surrogate Species	Surrogate Toxicity – LD_{50} (mg/kg)	Estimated Animal Class	Estimated Animal Common Name	Estimated Toxicity – LD_{50} (mg/kg)
			Common grackle[1]	26.65
			Starling	101.26
			Sharp-tailed grouse	110.17
		Mammals	Dog	502.85
			Guinea pig	420.65
			Meadow vole[1]	82.91
			Mouse	462.1
			Mule deer	446.18
			Rabbit	506.19
			Deer mouse[1]	731.14
			Ricefield rat[1]	45.31
			Roof rat[1]	276.19

[1] Input surrogate toxicity (Norway Rat LD50 = 500 mg/mg) was greater than model maximum for this animal.