ENVIRONMENTAL PROTECTION AGENCY

Notice of Proposed Rulemaking: Phasedown of Hydrofluorocarbons: Establishing the Allowance Allocation and Trading Program Under the American Innovation and Manufacturing Act; Docket ID No EPA–HQ–OAR–2021–0044, 86 Fed. Reg. 27150 (May 19, 2021)

Comments of the

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HSIA Comments on Proposed HFC Allocation Rule

These comments address regulations proposed by EPA to implement the American Innovation and Manufacturing Act, as enacted on December 27, 2020. 86 Fed. Reg. 27150 (May 19, 2021). The primary purpose of the proposed regulations is to establish a program to allocate rights to produce and consume hydrofluorocarbons (HFCs) in furtherance of the HFC phase down schedule in the AIM Act. The Halogenated Solvents Industry Alliance, Inc. (HSIA) represents manufacturers and users of chlorinated solvents essential to the manufacture of HFCs and of HFC alternatives in the family of hydro-olefins (HFOs).

HSIA's interest in the proposed regulations arises out of EPA's concern about, and solicitation of comments on, how the phasedown of HFC production could affect production of HFCs and HFC substitutes – and associated air pollution emissions – at individual facilities, particularly in communities that are disproportionately burdened by air pollution. These environmental justice (EJ) concerns are said to be due to the release of toxic chemicals that are feedstocks, catalysts, or byproducts in the production of HFCs or HFC substitutes. 86 Fed. Reg. at 27159.

The Agency's Regulatory Impact Analysis (RIA), where these concerns are addressed more specifically, "focuses mainly on characterizing baseline emissions of air toxics that are also associated with chemical feedstock use for HFC production." It also contains an analysis of community characteristics within one and three miles of the nine HFC/HFO production facilities identified, described as a uniquely granular assessment of the characteristics of these facilities and the communities where they are located. The analysis indicates that the total baseline cancer risk and total respiratory risk from air toxics (not all of which stem from HFC production) varies, but is generally higher within one to three miles of an HFC production facility. In this regard, EPA states: "It is not clear the extent to which these baseline risks are directly related to HFC production, but some HFC production feedstocks, catalysts, and byproducts are toxic, particularly with respect to potential carcinogenicity (e.g., carbon tetrachloride, tetrachloroethylene, trichloroethylene, etc.). Additionally, some HFC alternatives, e.g., HFOs, use the same chemicals as feedstocks in their production or released as byproducts, potentially raising concerns about local exposure to them." *Id.*

EPA requests whether other regulatory authorities would be more appropriate to address potential impacts outside of the AIM program. *Id*. The comments below support continued utilization of the Clean Air Act's existing, comprehensive regulatory programs implemented through rule and/or federal and state air permitting programs, *e.g.*, the Protection of Stratospheric Ozone and the National Emission Standards for Organic Hazardous Air Pollutants, to evaluate any suspect or potential HFC/HFO emissions impact instead of developing a separate and duplicative evaluation through the AIM Act regulatory process.

Finally, EPA solicits comment on key assumptions underlying the EJ analysis. *Id.* at 27160. Foremost among these "key assumptions" is that the three principal feedstock "chemicals (carbon tetrachloride, perchloroethylene, and trichloroethylene) are known to present an unreasonable risk of injury to the health of workers or occupational non-users in processing as a

reactant or intermediate in industrial gas manufacturing," referencing Risk Evaluations for each of these substances completed by EPA in the past year under the Toxic Substances Control Act (TSCA). RIA at p. 114. These Risk Evaluations, the first issued after the passage of the Lautenberg Act, are untested, have not been subject to rigorous review, are not consistent with the statutory requirements of TSCA, do not include an EJ analysis, and overestimate both the hazard and exposure of chlorinated HFC/HFO feedstocks.[1]

The "unreasonable risk" to which workers are said to be exposed is purely a matter of linear extrapolation of dose-response data to assumed (and greatly exaggerated) worker exposure scenarios. The Risk Evaluations provide no credible evidence of increased cancer incidence or other adverse health effects in worker populations exposed to the solvents.

Moreover, the Risk Evaluations themselves are completely silent as to any risk posed by these chemicals to fenceline communities. Nor is there even any attempt to quantify concentrations of the solvents at the fencelines of the nine HFC/HFO production facilities in question. As will be shown, it is entirely inappropriate scientifically to extrapolate from a conclusion that a worker is at risk from an exposure (even if it were correct) to the conclusion that an inhabitant of a fenceline community where concentrations of that chemical are close to background levels is similarly at risk.[2]

I. Congress Has Recognized that Feedstock Emissions Are Too Insignificant to Be a Concern and Provided Other Authority to Protect Fenceline Communities

In the 1990 Clean Air Act (CAA) Amendments (42 U.S.C. § 7671d(a)(2)) and the more recent AIM Act, Congress expressly excluded feedstock uses from production restrictions on the basis that the chemical is "used and entirely consumed (except for trace quantities)," in essence a judgment that such uses are *de minimis*. Thus, production or import of carbon tetrachloride, which depletes stratospheric ozone, was prohibited as of 2010 under CAA Title VI but allows for limited exceptions such as *feedstock or process agent uses*. (A similar definition in the AIM Act excludes HFCs used as feedstock or process agents from the phase down.) It is illogical that uses so trivial they have been excluded from regulation to address the overriding global environmental concerns of stratospheric ozone depletion and climate change could now be drivers of hypothesized community risk for EJ purposes.

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[1] Because of the unique judicial review provisions of TSCA, as amended in 2016 by the Frank R. Lautenberg Chemical Safety for the 21st Century Act, 15 U.S.C. § 2605(i)(2) ("Lautenberg Act"), HSIA is unable to show that they are unsupported by substantial evidence or otherwise unlawful under TSCA until they are used as the basis for a final rule under TSCA § 6(a), 15 U.S.C. § 2605(a).

[2] On June 30, 2021, the EPA Office of Pollution Prevention & Toxics announced that it will be carrying out an analysis of the fenceline risk posed by the chlorinated feedstocks as part of its TSCA risk evaluations. https://www.epa.gov/newsreleases/epa-announces-path-forward-tsca-chemical-risk-evaluations. It is not clear whether and to what extent there has been any coordination with the Air Office, but this "more comprehensive exposure assessment of fenceline communities" will obviously be directly relevant to the EJ analysis in the instant rulemaking. The notice states: "EPA intends to withdraw the previously issued orders for those conditions of use for which no unreasonable risk was found for all the first 10 risk evaluations. The agency then intends to issue revised unreasonable risk determinations for these chemicals as a "whole substance" and seek public comment on this approach." Thus, it would seem premature to rely on these Risk Evaluations, which are not complete, for an EJ analysis at this time.

Elsewhere in the 1990 CAA Amendments, Congress also created a comprehensive program to regulate sources of "hazardous air pollutants" such as the chlorinated solvents. CAA § 112(d) (42 U.S.C. § 7412(f)(2)) provides that EPA shall "promulgate standards . . . to provide an ample margin of safety to protect public health in accordance with this section." Further, CAA § 112(f)(2) (42 U.S.C. § 7412(f)(2)) provides "if standards promulgated pursuant to subsection (d) and applicable to a category or subcategory of sources emitting a pollutant (or pollutants) classified as a known, probable or possible human carcinogen do not reduce lifetime excess cancer risks to the individual most exposed to emissions from a source in the category or subcategory to less than one in one million, the Administrator shall promulgate standards under this subsection for such source category." Pursuant to these authorities EPA has already adopted standards applicable to fluorinated gas manufacturing that specifically regulate the risk now identified in the proposed rule. Clearly § 112 is the authority that Congress provided to address any fenceline risk from manufacturing fluorinated gasses. It is remarkable that these comprehensive regulations are not mentioned in the RIA.

The notice states (86 Fed. Reg. at 27159) that "EPA solicits comment on whether other regulatory authorities would be more appropriate to address any inadvertent or unexpected distributional effects that are identified, for example, if a producer obtained allowances in sufficient quantities to grow HFC production, which could potentially increase air emissions at that location. In such instances, where other authorities may be a more appropriate avenue, EPA expects that effects would be addressed through that avenue outside of AIM Act regulatory processes under timelines appropriate to those other programs."

Given the existence of mature regulatory programs under CAA § 112(d) and (f) directed specifically at the risks in question, it appears that these programs are the "more appropriate avenue" sought by EPA. The principal such program is the National Emission Standards for Organic Hazardous Air Pollutants from the Synthetic Organic Chemical Manufacturing Industry ("HON Rule"), 40 CFR part 63, subparts F, G, H, and I, which covers facilities manufacturing fluorochemicals using chlorinated solvents as feedstocks.[3] In 2006, EPA's Risk and Technology Review determined that no changes to the existing CAA § 112(d) rule were required under CAA § 112(f) because the current level of control both (i) reduced hazardous air pollutant (HAP) emissions to levels that present an acceptable level of risk and (ii) protects public health with an ample margin of safety. The finding regarding an ''ample margin of safety'' was based on a consideration of the additional costs of further control and the relatively small reductions in health risks that would be achieved by an alternative. The level of risk from the HON Rule was found acceptable for the following reasons: The maximum individual lifetime cancer risk was estimated to be 100-in-1 million, and this level of risk occurred at only two facilities. There were no people with estimated cancer risks greater than 100-in-1 million (the presumptively acceptable level of maximum individual lifetime cancer risk under applicable law) resulting from exposure to HON HAP emissions. For the exposed population, total annual cancer incidence was estimated at 0.14 cases per year. The Hazard Index (HI) values (representing long-term noncancer public health risks) barely exceeded 1. *See* 71 Fed. Reg. 76603, 76605 (Dec. 21, 2006).

[3] The National Emission Standards for Hazardous Air Pollutants: Miscellaneous Organic Chemical Manufacturing Rules ("MON") also applies to some HFC/HFO feedstock facilities. EPA completed the Risk and Technology Review (RTR) for the MON in August 2020 [85 Fed. Reg. 49084 (August 12, 2020)].

Further, in the Risk and Technology Review for the HON Rule, EPA expressly:

> "reaffirmed its commitment to ensuring environmental justice for all people, regardless of race, color, national origin, or income level. To ensure environmental justice, we assert that we shall integrate environmental justice considerations into all of our programs and policies, and, to this end, have identified eight national environmental justice priorities. One of the priorities is to reduce exposure to air toxics. At proposal, EPA requested comment on the implications of environmental justice concerns relative to the two options proposed since some HON facilities are located near minority and low-income populations. We received one comment regarding environmental justice concerns that is addressed in the response to comments document." 71 Fed. Reg. at 76614.

Finally, the RIA does not address the role of state and local regulators, and the fact that all of the facilities surveyed have been permitted for the releases reported to TRI, and presumably are in compliance with these permits.

II. The TSCA Risk Evaluations Are Deficient and Should Not Be Used as a Basis for EJ Regulation

TSCA § 26(h) and (i) require EPA to use scientific information, technical procedures, measures, methods, protocols, methodologies, and models consistent with the best available science and to base its decisions on the weight of the scientific evidence. The Risk Evaluations referred to in the AIM proposal, which are the inaugural TSCA Risk Evaluations issued after the passage of the Lautenberg Act, are still undergoing review by EPA. Reliance upon the Risk Evaluations by other regulatory programs outside of TSCA areas is both premature and inappropriate because of the overestimates of hazards and worker exposures, described further below.

TSCA § 6(b)(4)(F), as revised by the Lautenberg Act, requires that EPA's risk evaluations must, among other things:

- "integrate and assess available information on hazards and exposures for the conditions of use of the chemical substance, including information that is relevant to specific risks of injury to health or the environment and information on potentially exposed or susceptible subpopulations identified as relevant by the Administrator;"

- "take into account, where relevant, the likely duration, intensity, frequency, and number of exposures under the conditions of use of the chemical substance;" and

• "describe the weight of the scientific evidence for the identified hazard and exposure."

New TSCA § 26(h) requires that, for each risk evaluation (as "a decision based on science") that "the Administrator shall use scientific information, technical procedures, measures, methods, protocols, methodologies, or models, employed in a manner consistent with the best available science, and shall consider as applicable—

(1) the extent to which the scientific information, technical procedures, measures, methods, protocols, methodologies, or models employed to generate the information are reasonable for and consistent with the intended use of the information;

(2) the extent to which the information is relevant for the Administrator's use in making a decision about a chemical substance or mixture;

(3) the degree of clarity and completeness with which the data, assumptions, methods, quality assurance, and analyses employed to generate the information are documented;

(4) the extent to which the variability and uncertainty in the information, or in the procedures, measures, methods, protocols, methodologies, or models, are evaluated and characterized; and

(5) the extent of independent verification or peer review of the information or of the procedures, measures, methods, protocols, methodologies, or models."

TSCA § 26(i), as added by the Lautenberg Act, provides simply that "The Administrator shall make decisions under sections 4, 5, and 6 based on the weight of the scientific evidence."

A. Existing EPA Cancer Risk Methodology is Not Best Available Science

As noted above, the RIA relies solely on findings of unreasonable risk to workers in HFO/HFC facilities issued by a different EPA program office, and these findings are not supported by substantial evidence, including best available science, as required by TSCA §§ 6, 26. In seeking to stretch these findings to cover fenceline communities, the RIA goes further and suggests that general population exposures to the three chlorinated solvents of approximately 2 ug/m^3 may represent a one in 1 million risk of cancer. There is absolutely no support in the record for such a conclusion. We submit that the foundation of the methodology that arrives at such absurd outcomes must be rigorously supported by EPA before that methodology is applied for regulatory purposes. In this regard, we attach as Attachment 1 the complete volume of a recent journal which calls into question this methodology: *Assessing the Scientific Basis of the Linear No Threshold (LNT) Model with Threshold Models for Cancer Risk Assessment of*

Radiation and Chemicals (If LNT were a biologically valid dose-response model, the appearance and evolution of life on Earth would not have been possible), edited by Edward Calabrese and Robert Golden, Chemico-Biological Interactions, 301: 1-146 (2019). We also provide in Attachment 2 a discussion of how EPA's current cancer risk methodology fails to comply with the statutory directives.

B. Deficient Hazard Assessments

Recognizing that a hazard assessment is different for each separate substance, taking into account differences in the toxicological data, this section focuses on CTC to simplify the presentation in these comments. Relevant information showing similar deficiencies in EPA's treatment of TCE and PCE is provided in Attachments 3 and 4, respectively.[4]

The CTC Risk Evaluation used a linear non-threshold model coupled with an assumption that the principal study relied upon did not produce a no-observed-adverse-effect level (NOAEL), both in disregard of advice provided by outside peer reviewers. As a result, as described in more detail below, the estimates are overly conservative by at least a thousand-fold. The Risk Evaluation relied on Nagano *et al*., (2007) to derive both the cancer inhalation unit risk (IUR) and the dermal slope factor. The IUR estimates based on Nagano *et al*. (2007) were calculated by the EPA IRIS Program in 2010. The IUR selected for carbon tetrachloride via the inhalation pathway was 6×10^{-6} $(\mu g/m^3)^{-1}$, which was associated with pheochromocytomas in the male mouse. The data set on pheochromocytomas in the male mouse was also judged by the EPA IRIS Program to yield the highest estimate of risk.[5] As in the case of the dermal exposure assessment, this approach patently departs from EPA's recognition, in calling for the use of "best available science," that "scientific knowledge about risk is rapidly changing and … risk information may need to be updated over time."[6]

In the IRIS CTC assessment, EPA concluded that there is insufficient information on the mode of action (MOA) of CTC for mouse liver tumors at low doses and the mouse pheochromocytomas to support a non-linear dose-response approach for assessing cancer risk. A majority (four out of six) of the external peer reviewers, however, recommended that potential CTC cancer risk should be based on a non-linear threshold method. To quote directly from the IRIS response to reviewer comments: "Two reviewers considered it appropriate to present a linear low-dose extrapolation approach as an alternative approach, but that based on available evidence, the nonlinear method seems more appropriate." A fifth reviewer stated that use of a linear dose response model is "difficult to defend and is not a preferable approach" [and a] "sixth reviewer did not agree that a linear assessment is justified for carbon tetrachloride." Even one of the two reviewers who believed that a low-dose linear approach was the "most clear, prudent and scientifically defensible approach" noted that use of a nonlinear approach is "reasonable to

[4] The LNT papers from Chemico-Biological Interactions 301: 1-146 (2019) were omitted from Attachment 4 as they are presented separately as Attachment 1 in the previous paragraph. Also omitted from Attachment 4 is the Excel spreadsheet containing the critical data from the New York State Part 232 Dry Cleaning Compliance Inspection Reports (this is referenced in Attachment 4 as Appendix 9).

[5] Risk Evaluation at 167.

[6] EPA Guidelines at 23.

consider," although noting that such an approach might use an additional, possibly 10-fold, uncertainty factor to assure protection of both cancer and non-cancer endpoints.[7]

The final Risk Evaluation included a nonlinear dose-response assessment, but departed from the advice of the TSCA Science Advisory Committee on Chemicals (SACC), which was quite clear that a threshold MOA should be used for CTC:

> "The Committee concluded that the weight of a considerable body of scientific evidence indicates that the relationship between carbon tetrachloride dose/exposure and its genotoxic response is nonlinear with a steep dose-response. Less is known about mechanisms underlying adrenal gland tumors in rodents or apparent glioblostomas [sic] in workers. Most of the Committee members recommended that the EPA consider adoption and implementation of a threshold MOA when estimating cancer risks."[8]
>
> Indeed, the Committee highlighted the following recommendation:
>
> "Recommendation 55: Consider adoption of a threshold-type MOA in estimating the carcinogenic risks of carbon tetrachloride.
>
> "Mechanisms underlying the carcinogenicity of carbon tetrachloride in the rodent liver have been studied extensively. Using a WOE approach, it is likely that the relationship between carbon tetrachloride dose per exposure and its genotoxic response is nonlinear with a steep dose response. This conclusion is primarily based upon the MOA identified from numerous genotoxicity investigations, as well as several important factors that support/indicate a nonlinear dose-response. These include recognition that:
>
> 1. The primary site of carbon tetrachloride bioactivation and adverse effects is the smooth endoplasmic reticulum, a site removed from the nucleus and DNA;
>
> 2. The moieties which are formed are highly reactive and unlikely to travel far in the aqueous cytoplasm from their site of formation;
>
> 3. The observed genotoxic effects appear to result from indirect mechanisms related to oxidative and lipid peroxidation-mediated DNA damage, or damage occurring due to necrosis and apoptosis;
>
> 4. Carbon tetrachloride metabolite-induced lipid peroxidation is an exponential chain reaction, such that a single initiation event can lead to formation of many reactive species. Thus, the extent of damage can have a distinct nonlinear component;

[7] https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0020tr.pdf at A-25.

[8] https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0499-0046 at 50.

5. High levels of hepatoprotective agents and antioxidants are present in hepatocytes;

6. A close relationship is manifest between cytotoxicity and genotoxicity;

7. Oxidative and lipoperoxidation-related DNA damage occurs spontaneously in untreated cells, and has been shown to be efficiently repaired; and

8. Apoptosis and recognition and destruction of transformed cells by the immune system are additional protective mechanisms that argue against use of a linear dose-response model."[9]

The Committee concluded:

"[A]lthough the Evaluation claims to have 'Evaluated the weight of the scientific evidence based on the available human health hazard data for carbon tetrachloride,' the Committee noted that convincing support for this claim is lacking. In particular, the Evaluation refers repeatedly to a concern that low-level exposures to carbon tetrachloride may somehow act through genotoxic mechanisms (evidence for this notwithstanding); indeed, this concern is its underlying justification for using the "default" approach of applying a linearized model to the tumor mouse bioassay data in order to predict low-dose cancer-risk. But the weight of evidence clearly indicates that any genotoxicity caused by carbon tetrachloride can occur only at exceedingly high levels of exposure, and is caused not by carbon tetrachloride directly, but only indirectly after high levels of lipid peroxide by-products (such as reactive aldehydes) have accumulated intracellularly. . . . No support is provided for EPA's designation of an 'alternate MOA' that combines cytotoxic mechanisms at relatively high CCl4 doses with 'alternate, non-cytotoxic mechanisms' at lower doses."[10]

Although the Risk Evaluation includes cancer risk estimates derived using a non-linear approach, the calculations are based on a point of departure (POD) of 5 ppm. EPA interpreted the increase in liver tumors in the female mice at this concentration as a treatment-related lowest observed-adverse-effect level (LOAEL). As noted by the SACC, however, the scientific justification for using a nonlinear approach here is that the MOA for CTC-induced liver tumors involves cytotoxicity and proliferation from the highly reactive radical metabolites of CTC. Thus, liver toxicity is a precursor key event to CTC-induced liver tumors. In the Nagano study there was no indication of liver toxicity in the livers of female mice exposed to 5 ppm. Accordingly, EPA's use of 5 ppm as a LOAEL for its derivation of cancer risk is incompatible with the underlying assumption regarding the MOA. Given the preponderance of science evidence for the cytotoxic-proliferative MOA for CTC carcinogenicity, the weight of the

[9] *Id.* at 51-52.

[10] *Id.* at 39 (references omitted and emphasis added).

evidence suggests that the increase in female mouse liver tumors at 5 ppm occurred by chance and that this exposure concentration is instead a NOAEL.

Indeed, the SACC stated:

> "No support is provided for the EPA's designation of an "alternate MOA" that combines cytotoxic mechanisms at relatively high carbon tetrachloride doses with "alternate, non-cytotoxic mechanisms" at lower doses. What is meant by an "alternate non-cytotoxic mechanism" (Evaluation Page 124, line 4005)? This appears to be speculation that something must be occurring to produce an increased incidence in liver adenomas in the female mice dosed at five ppm. Consideration should be given to the possibility that this was a chance occurrence in a single study. The historical incidence of this benign tumor in control Crj:BDF1 mice is as high as 10%. Had three of 50 control females exhibited liver adenoma in this particular experiment, the difference between them and the five ppm dose group would not have been statistically significant. There was no increase in liver carcinoma incidence in the females dosed at five ppm and no significant increase over controls in combined benign and malignant liver tumors. It should also be noted there was no increase in hepatocellular adenoma or carcinoma in the male mice dosed at five ppm. Male mice metabolically activate more carbon tetrachloride and experience a higher incidence of liver cancer then do females."[11]

The peer review excerpts quoted above make clear that the Committee disagreed with EPA and supported a non-linear assessment based on a 5 ppm NOAEL. Further, the Committee made clear its view that EPA was not using a weight-of-the-evidence approach. This is highly significant given the admonition in TSCA § 26(i) that "[t]he Administrator shall make decisions under sections 4, 5, and 6 based on the weight of the scientific evidence." It is unusual for peer reviewers to place so much emphasis on a recommendation, and even more unusual for EPA to disregard such a recommendation when it echoes earlier advice received from different external peer reviewers on the same subject.

Significantly, there is a recent and readily available Substance Evaluation Conclusion for CTC prepared by France as a part of the substance evaluation process under the REACH Regulation (EC) No 1907/2006 (Attachment 5). Unlike the EPA Evaluation, but consistent with the outside peer reviewers here, this weight-of-the-evidence review combines a nonlinear, threshold mode of action with a nongenotoxic mode of action:

> "Taking into account the results of genotoxicity data, CCl4 [CTC] is not considered as a direct genotoxic agent but acts as a carcinogen by a threshold mode of action. Cytotoxicity and regeneration seem therefore to be a main factor in the apparition [sic] of (pre-)neoplastic lesions. In conclusion, CCl4 is considered to act as a carcinogen by a threshold mode of action."

Based on this conclusion, the French evaluation derives a NOAEL of 5 ppm (32 mg/m³)

[11] *Id.* at 50-51.

for hepatoadenomas and carcinomas in both species after chronic exposure to CTC via the inhalation route. This is in line with the workplace limit enforced by OSHA (10 ppm) and that recommended by the American Conference of Governmental Hygienists (5 ppm), and some thousand times higher than the level deemed acceptable by EPA. To meet the TSCA requirements, EPA should recognize the 5 ppm NOAEL and use it, along with a nonlinear MOA, as the basis for a revised cancer risk assessment.

C. Deficient Dermal Exposure Assessments

The Risk Evaluation concludes that the chlorinated solvents present unreasonable risks to workers under almost all conditions of use, including fluorinated gas manufacturing, with or without Personal Protective Equipment (PPE). For dermal exposure, although unsupported by actual data, EPA finds unreasonable cancer risks to workers even with the most protective glove use (Protection Factor of 20). In the absence of dermal exposure data, EPA relied on models to estimate the amount the solvent that is retained by workers from dermal contact. These "worst-case scenarios" assume unrealistic dermal exposure durations and fail to recognize basic industrial hygiene (IH) practices, as well as engineering controls required by the NESHAP. Thus, they are clearly inapplicable to facilities that use the solvents as feedstocks.

The use of chlorinated solvents in the production of other chemicals occurs in closed system process units where potential dermal contact is limited to short-term tasks in the operation of unit activities. The typical tasks that could potentially involve contact with the solvents are handling of transfer lines for vessel charging/uncharging and collecting samples from process points for laboratory analysis. In general, these tasks would involve limited direct contact with liquid, and the duration of any
potential contact with the liquid is very short (*i.e.*, minutes). The discussion that follows is directed at the Risk Evaluation for Carbon Tetrachloride (CTC). Similar considerations apply to EPA's assessments for the other two solvents.

EPA overestimated dermal exposure for workers using Kasting and Miller with the following assumptions that do not reflect conditions in the manufacturing and processing workplace: (1) one dermal contact with undiluted CTC which coats fully one or both hands per work shift; (2) workers do not wash their hands at any point during the 8-hour work shift if gloves are not worn; and (3) a worker wears the same pair of gloves for the entire 8-hour work shift without stopping to wash their hands and/or change their gloves. Incredibly, EPA provides no documentation or justification for these assumptions other than the intent to establish a theoretical "worst-case scenario." As a result of these conservative assumptions, EPA has substantially overestimated worker exposure to solvent from dermal contact in facilities that use them as a reactant or intermediate.

According to EPA, risk evaluations under TSCA § 6(b) are not screening level risk assessments, but are intended to "use scientific information, technical procedures, measures, protocols, methodologies and models consistent with the best available science." Therefore, EPA should consider in its dermal exposure models assumptions that are relevant and appropriate to actual workplace practices for the uses being evaluated. Unfortunately, these Risk Evaluations failed to acknowledge basic IH practices. For facilities with closed systems, any potential dermal

exposures are for short durations and, combined with the industry standards for good IH practices at these facilities which require removal and disposal of potentially contaminated gloves and hand washing after each task completion, do not justify an 8-hour period for absorption of the solvent through skin. Moreover, the solvents will evaporate from the skin and gloves between exposure periods. A more realistic approach to estimating the dermal dose of these compounds in workers in closed system facilities can be obtained using the IH Skin Perm model.[12] This tool is commonly used by practitioners of IH and exposure assessment to produce reliable estimates of dermal exposure, and it takes into account losses to evaporation and estimates the mass that is absorbed. In addition, IH SkinPerm can be used to evaluate the impacts of differing patterns of exposure on fractional and total dose of absorption, *i.e.*, it allows for the incorporation of realistic exposure patterns.

Using the IH Skin Perm model and a more realistic, albeit still conservative, period for exposure and absorption after tasks, allowing for handwashing, and assuming skin exposure had occurred for up to 1 hour before removal, we can estimate the dermal absorbed dose for use of the chlorinated solvents as a reactant or intermediate in the production of other chemicals. Such an estimate for absorption of CTC provides 2.78 mg/day from exposure to two full ungloved hands. In comparison, the Risk Evaluation estimated the amount of CTC absorbed to be 90 mg/day for two full hands (high-end estimate). Thus, the impact of using a more realistic approach to estimating the high-end dermal CTC dose over one hour results in an approximately 32-fold reduction in the dermal dose.

This overestimate of dermal dose is expected also to hold true in the Risk Evaluations for gloved hands, the only difference being that there is reduced dermal uptake from glove use, and this is accounted for by a workplace protection factor. It is also important to note that these models assume that a worker is exposed to neat or undiluted chemical. Such exposure is highly unlikely in facilities that use chlorinated solvents as reactants or intermediates in closed systems. As a result of using unrealistic worst-case assumptions in its dermal exposure assessments, EPA substantially overestimated worker exposure to the solvents from dermal contact by at least several orders of magnitude. *Thus, if the revised scenarios were applied in the risk characterizations, there would be no unreasonable risk to workers from dermal exposure!*

Recognition of standard work practices and reliance on reasonable and realistic exposure data are critical to meet the statutory requirements of TSCA. EPA's reliance on hypothetical assumptions for modeling of the amount of solvent that is absorbed by workers from dermal contact cannot be justified. Assumptions used for estimating worker exposures should be as relevant as possible for the use being evaluated. EPA's use of unrealistic dermal exposure assumptions has led to erroneous conclusions regarding the health risks to workers using chlorinated solvents as feedstock in closed systems. Because the Risk Evaluations are intended to determine whether such use presents an unreasonable risk of injury to workers under TSCA § 6(b), which requires rulemaking to mitigate risks found to be unreasonable, it is imperative that it be revised to reflect the "best available science."

[12] IH SkinPerm is a peer-reviewed exposure assessment tool published by the American Industrial Hygiene Association (AIHA) Exposure Assessment Strategies Committee.

III. More Information Is Needed on Background Concentrations and Sources

The RIA states, in most relevant part:

"Four of the chemicals in Table 6-8 were part of the first 10 evaluated by EPA and have a completed risk assessment. Three of those chemicals (carbon tetrachloride, perchloroethylene, and trichloroethylene) are known to present an unreasonable risk of injury to the health of workers or occupational non-users in processing as a reactant or intermediate in industrial gas manufacturing. The fourth chemical (methylene chloride) was found to not pose an unreasonable risk to workers for this condition of use, but it is subject to an Occupational Safety and Health Administration (OSHA) eight-hour time-weighted average permissible exposure limit (PEL). This is an upper limit of the airborne concentration to which an employee may be exposed.

"In addition, National Air Toxic Assessment (NATA) data from 2014 (the most recent year available) for census tracts within and outside of a 1 and 3 mile distance are used to approximate the cumulative baseline cancer and respiratory risk due to air toxics exposure for communities near an HFC production facility.

"The EJ analysis demonstrates that:

o The characteristics of the community near HFC production facilities are heterogeneous;

o Total baseline cancer risk and total respiratory risk from air toxics (not all of which stem from HFC production) varies, but is generally higher, and in some cases much higher within 1–3 miles of an HFC production facility;

o Higher percentages of low income and Black or African American individuals live near HFC production facilities compared to the overall or rural average at the national level;

o It is not clear the extent to which these baseline risks are directly related to HFC production, but some of HFC feedstocks and byproducts are toxic; and

o Multiple HFC alternatives are available, some of which have toxic profiles for the chemicals used as feedstocks in their production.

"Given limited information regarding which substitutes will be produced where, it is unclear to what extent this proposed rule will impact baseline risks

> from hazardous air toxics for communities living near HFC and HFC substitute production facilities. EPA is seeking information to help better characterize these changes and their implications for nearby communities for analysis of the final rule."[13]

It appears that, assertions regarding "very high baseline risks from air toxics" near these facilities notwithstanding, "EPA has not undertaken an analysis of how the emissions of various HFC feedstocks, catalysts, and byproducts affect nearby communities (e.g., through the use of a fate and transport model or the modeling of main exposure pathways). Nor does it have information at this time on how workers may be exposed to these chemicals or the characteristics of workers at these facilities." RIA p. 115. Going forward, EPA should provide the part of the EJ analysis that used the 2014 NATA data to show that "total baseline cancer risk and total respiratory risk from air toxics (not all of which stem from HFC production) varies, but is generally higher, and in some cases much higher within 1–3 miles of an HFC production facility." This information seems to be a primary driver for the EJ concern. What chemicals were included and what was their contribution to both total baseline cancer risk and total respiratory risk?

Because the RIA does not quantify any contribution from HFC/HFO production, it is not possible to understand the impact of use of chlorinated solvent feedstocks in fluorinated gas production without substantial supplementation of the record. Clearly, a better understanding of background concentrations and site releases is needed as EPA considers the questions it has raised in the EJ context.

[13] RIA, pp. 114 *et seq.*