UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

Comments of Safer Chemicals Healthy Families on EPA's Draft Risk Evaluation for Methylene Chloride under Section 6(b) of TSCA

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Safer Chemicals Healthy Families (SCHF) submits these comments on EPA's draft risk evaluation for methylene chloride (MC) under section 6(b) of the Toxic Substances Control Act (TSCA).[1] These comments provide our views to the Scientific Advisory Committee on Chemicals (SACC) in advance of its December 3-4 meeting to review the draft evaluation.

Safer Chemicals Healthy Families is a non-profit organization whose work is focused on issues of chemical safety. We use a variety of channels to improve public understanding of the health and environmental risks of unsafe chemicals, reduce their presence in consumer products and on retail shelves, and strengthen federal laws and regulations that protect the public. SCHF has played a prominent role in persuading leading retailers to stop sales of paint remover products containing MC and N-Methylpyrrolidone (NMP). Working with the families of young men who died from MC exposure, we were also instrumental in calling on EPA to finalize its proposed ban on MC-based paint removers for commercial and consumer use.

As we show below, the draft risk evaluation confirms earlier EPA findings that MC poses serious risks to consumers and workers of death, incapacitating neurotoxic effects, cancer and liver toxicity. Although the findings of the draft evaluation are alarming, we believe that EPA has in fact understated MC's risks in several respects:

- EPA does not combine inhalation and dermal exposures, even though they occur concurrently, and thus fails to account for their combined contribution to total risk.
- EPA excludes all MC environmental releases in determining human exposure, including air emissions, which are unusually large and account for elevated ambient air concentrations in many urban areas.
- EPA makes no effort to examine aggregate risk from multiple pathways, such as concurrent workplace, consumer product, and environmental exposures, which are common occurrences for many individuals and communities.
- EPA does not fully account for the increased susceptibility to MC's acute effects by pregnant women, the elderly, fetuses, children, people engaged in vigorous physical activity, users of alcohol and Individuals suffering from lung and heart disease.
- EPA has not explained and justified why the Inhalation Unit Risk (IUR) for carcinogenicity in its draft evaluation is lower than in previous MC evaluations and clarified how the IUR accounts for the higher prevalence of the GST-T1 null (-/-) genotype in large US subpopulations.

[1] 84 Federal Register 31315 (July 1, 2019).

- The draft evaluation does not use all necessary uncertainty factors (UFs) in calculating benchmark margins of exposure (MOE) for acute and chronic non-cancer effects, making no allowance for known vulnerable populations and uncertainties in the MC database.
- As in several other evaluations, EPA has assumed that workers will be protected from unsafe exposures by Personal Protective Equipment (PPE) – an assumption that is contrary to known workplace realities and established worker protection policy and practice.

We urge SACC to focus on these issues in reviewing EPA's draft evaluation and to urge EPA to modify the evaluation if it agrees with our concerns.

I. The Draft Evaluation Demonstrates Serious Acute and Chronic Risks to Workers and Consumers

EPA's concerns about the acute and chronic hazards of MC are long-standing. The Agency began evaluating the chemical in 1991. In 2011, EPA's Integrated Risk Information System (IRIS) issued a final MC toxicity assessment[2] and in 2014, the Agency finalized its TSCA risk assessment for MC-based paint removers.[3] Because of the serious risks demonstrated in that assessment, EPA proposed in early 2017 to ban these products under TSCA for both consumer and commercial use.[4] While EPA dragged its feet on finalizing this proposal, four more deaths occurred from inhalation of MC fumes during paint remover use. Following a public outcry and pleas by the families of the decedents, EPA belatedly finalized its consumer use ban in March of this year but postponed action to protect workers using MC paint strippers.[5]

Drawing heavily on its earlier scientific analysis, the draft risk evaluation broadens the findings of the 2014 risk assessment to include the many other consumer and commercial uses of MC. The population exposed to MC from these uses is extremely large, totaling more than 8 million workers and several million consumers and exposed bystanders.[6]

As EPA has earlier found, the draft risk evaluation concludes that "[r]isks from acute exposures include central nervous system risks such as central nervous system depression and a decrease in peripheral vision, each of which can lead to workplace accidents and which are precursors to more severe central nervous system effects such as incapacitation, loss of consciousness, and death" (p. 30). These effects occur because MC fumes act as a CNS depressant and also metabolize in the body into carbon monoxide, cutting off the supply of oxygen and killing users in as few as ten minutes. According to a recent analysis (attached) by

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[2] Toxicological Review of Dichloromethane (Methylene Chloride) (CASRN 75-09-2) in Support of Summary Information on the Integrated Risk Information System (IRIS) [EPA IRIS Assessment]. (EPA/635/R-10/003F). Washington, D.C. http://www.epa.gov/iris/toxreviews/0070tr.pdf U.

[3] TSCA Work Plan Chemical Risk Assessment, Methylene Chloride: Paint Stripping Use. (740-R1-4003). Environmental Protection Agency, Office of Chemical Safety and Pollution Prevention. https://www.epa.gov/sites/production/files/2015-09/documents/dcm_opptworkplanra_final.pdf {Workplan Risk Assessment}

[4] 82 Fed. Reg. 7464 (January 19, 2017).

[5] 84 Fed. Reg. 11420 (March 23, 2019).

[6] The risk evaluation provides estimates of exposed workers for each of the commercial/industrial conditions of use it examines and these estimates total over 8 million for all exposed workers. EPA's 2017 paint remover proposal estimated that 1.3 consumers were exposed to these products (82 Fed. Reg. 7475-6). An even larger number of consumers are probably exposed to the 15 types of consumer products encompassed by the draft evaluation although EPA does not estimate the size of this population.

scientists at the University of California San Francisco, 83 deaths have been linked to acute exposure to MC since 1980. This likely understates the actual number; EPA indicated in its 2017 proposed rule that numerous additional deaths were probably either unreported or erroneously attributed to other causes.[7]

The draft risk evaluation also concludes that MC has serious chronic health effects. It determines that "[t]here is sufficient evidence of methylene chloride carcinogenicity from animal studies" and that the chemical has produced tumors at multiple sites, in males and females, in rats and mice, by oral and inhalation exposure, and in multiple studies." (p. 264). It further finds that "inhalation and oral studies identified liver effects as sensitive non-cancer effect linked with exposure to methylene chloride in animals." (p. 260)

The draft evaluation addresses 15 consumer products that contain MC. It concludes that these products present acute risks similar in nature and magnitude to the paint remover risks on which EPA based its consumer use ban. *Specifically, for all but one of the 15 products, projected acute exposures in one or more of EPA's use scenarios were above or alarmingly close to MC levels causing neurotoxic effects in human studies.* (p.36) As a result, for inhalation or dermal exposure or both, margins of exposure (MOE) were well below the "benchmark MOE" that EPA used to define unreasonable risk. For several of the products, the MOEs were unprotective not only for product users but for consumer bystanders as well.

The risks to workers identified in the draft evaluation were equally alarming. *The evaluation found inhalation and/or dermal MOEs below the benchmark MOE for all 31 of the industrial and commercial conditions of use it analyzed.* (pp. 34-36) These conditions include the commercial use of MC-containing paint removers (pp. 685-725). The risk evaluation incorporates verbatim large portions of EPA's 2014 risk assessment and thus reaffirms the rationale for the proposed ban on commercial use of these products that EPA failed to finalize earlier this year.

II. The Draft Risk Evaluation Significantly Understates Risks to Consumers and Workers

Although the draft evaluation demonstrates that MC presents serious and unreasonable risks to health, we believe it overlooks several factors that, if considered, would result in even larger estimates of risk and a higher probability of acute and chronic health effects under expected levels of exposure.

A. EPA Fails to Combine Concurrent Inhalation and Dermal Exposures

The draft risk evaluation concludes that MOEs for acute dermal and inhalation exposure are below benchmark MOEs for several worker and consumer use scenarios. (Table 4-104) EPA also calculates cancer risks for both inhalation and dermal exposure. The inhalation cancer risks are in many cases higher than 1×10^{-4}; the dermal risks are above 1×10^{-6} and in some cases 1×10^{-5} in no glove scenarios. (Table 4-71) However, while recognizing that "[i]nhalation and dermal exposures are assumed to occur simultaneously for workers and consumers," EPA did not aggregate exposures across these routes and calculate a total risk accounting for the contribution of both. (p. 304) EPA acknowledges that this approach "may lead to an underestimate of exposure." However, it explains that it "chose not to employ simple additivity of exposure [routes] . . . because of the uncertainties present in the current exposure estimation procedures." Id.

[7] 82 Fed. Reg. 7482.

It is not clear what "uncertainties" EPA is referring to. In fact, EPA used inhalation toxicity data to extrapolate from the acute to the dermal route, using toxicokinetic information to estimate dermal doses at which the effects seen in inhalation studies would occur, as explained below:

> "EPA did not identify toxicity studies by the dermal route that were adequate for dose-response assessment. Dermal candidate values, therefore, were derived by route-to-route extrapolation from the inhalation PODs as mentioned above. The inhalation PODs were extrapolated using a POD based on either human data i.e., acute exposures or the BMDLHEC a value from animals adjusted to account for animal to human extrapolation using the PBPK model the preferred approach because this incorporates methylene chloride specific toxicokinetic data. Therefore, the equations for extrapolating from inhalation PODs to the dermal route account for human inhalation and body weight, shown below, assume average exposure factors from the Exposure Factors Handbook . . ." (p. 282)

Since EPA had sufficient confidence in route-to-route extrapolation to base estimates of dermal risk on the results of inhalation studies, it is hard to understand why this same approach could not be used to determine overall exposure by the two routes combined. Thus, EPA's MOEs and cancer risk estimates should have assumed exposure levels reflecting the contribution of each route, as opposed to calculating risks for each route individually. This would result in substantially lower MOEs and higher estimated cancer risks for use scenarios where both dermal and inhalation exposures are concurrent. **SACC should urge EPA to use combined dermal and inhalation exposures to determine MC's risks in its final evaluation.**

B. EPA's Exclusion of All Environmental Releases Fails to Consider Important Human Exposure Pathways

As in the case of previous evaluations, EPA has excluded environmental releases from its risk determinations, ignoring significant contributors to overall risk.

According to the EPA problem formulation,[8] "[i]Inhalation serves as the expected primary route of exposure for the general population due to both [MC's] volatility and propensity to be released to air from ongoing commercial and industrial activities." (p.39) Because MC is frequently applied as a solvent in open processes, significant loss of vapors to the atmosphere is expected. The most recent round of reporting for the Toxics Release Inventory ("TRI") showed MC air emissions of 2.9 million pounds in 2018.[9] Because of the reporting thresholds for TRI reporting, this figure does not reflect the emissions of the many small commercial operations that use MC as a solvent; it thus underestimates total releases to air. According to the 2000 ATSDR toxicological profile:

> "It has been estimated that 85% of the total amount of methylene chloride produced in the United States is lost to the environment (EPA 1985e), about 86% of which is released to the atmosphere (EPA 1982a). Thus, about 73% (370 million pounds) of the U.S. production volume for 1988 (500 million pounds), of methylene chloride was lost to the atmosphere in 1988."[10]

[8] https://www.epa.gov/sites/production/files/2018-06/documents/mecl_problem_formulation_05-31-18.pdf
[9] EPA, 2018 TRI Factsheet: Chemical – Dichloromethane, https://enviro.epa.gov/triexplorer/chemical.html?pYear=2018&pLoc=000075092&pParent=TRI&pDataSet=TRIQ1
[10] https://www.atsdr.cdc.gov/toxprofiles/tp14.pdf, at 178.

Because of these releases, the Problem Formulation finds that "levels of methylene chloride in the ambient air are widespread and shown to be increasing." (p. 39) Not unexpectedly, ambient levels tend to be highest in urban areas with a concentration of MC user facilities. According to the problem formulation, the "2011 National Air Toxics Assessment (NATA) modeled concentrations for various air toxics nationwide at a census tract level. This screening level tool modeled a maximum total methylene chloride concentration of 5,000 parts per trillion (18 µg/m3) and maximum human inhalation exposure concentrations of 3,900 parts per trillion (14 µg/m3)." (p. 36) ATSDR reports that:

> "Methylene chloride was among the chemicals monitored in a statewide survey of hazardous air pollutants by the Arizona Hazardous Air Pollutants Monitoring Program. The average amount of methylene chloride detected in air ranged from 0.61 ppm on a hillside in Yavapai County to 1.62 ppm in Phoenix (Zielinska et al. 1998). Concentrations of methylene chloride in urban areas and in the vicinity of hazardous waste sites are generally one to two orders of magnitude higher."[11]

ATSDR emphasizes that "groups within the general population that could have potentially high exposures . . . include individuals living in proximity to sites where methylene chloride was produced or sites where methylene chloride was disposed, and individuals living near one of the 1,569 NPL hazardous waste sites where methylene chloride has been detected in some environmental media (HazDat 1996)."[12]

As in prior risk evaluations, EPA justifies excluding the contribution of air emissions to total MC exposure on the ground that those exposures are "adequately assess[ed] and effectively manage[d]" under "other environmental statutes …. i.e., the Clean Air Act ("CAA")." (p. 428) While MC is a CAA Hazardous Air Pollutant ("HAP"), this does not justify ignoring air emissions in TSCA risk evaluations. Title III of the CAA initially mandates technology-based -- not risk based – emission limits. Once these limits are in place, the law gives EPA at least eight more years to evaluate residual risks and set risk-based emission standards under CAA section 112(f).[13] However, these standards would only consider emission-related risks, and thus would not take into account aggregate health risks from all sources of exposure. Moreover, EPA's emission standards would apply only to "major" sources, which are defined as facilities that emit more than 10 tons per year of any single HAP or 25 tons per year of all HAPs.[14] This definition would likely not cover the thousands of smaller establishments that in the aggregate account for substantial MC air emissions.

in its review of the 1,4-dioxane draft, the SACC questioned EPA's similar rationale for failing to consider drinking water pathways of exposure and consumer uses:[15]

> "Some Committee members stated that omission of consumers and the general United States (U.S.) population is inappropriate, unless risk assessments *have been* completed at this point in time. Exposure scenarios that include consumers are important given the known presence of 1,4-Dioxane in plastics, other commercially available products, surface water, drinking water, groundwater, and in sediments. The Committee also had concerns that the omission of these multiple routes of exposure puts workers who inhale or ingest 1,4-Dioxane outside the workplace at even greater risk."

[11] Id at 183.
[12] Id at 189.
[13] 42 U.S.C. § 7412(f)(2).
[14] 42 U.S.C. § 7412(a)(1).
[15] 1,4-Dioxane and HBCD SACC Report, at 20.

The SACC added that:[16]

> "The Committee discussed that if each program office of the EPA says others are assessing the risks and thus not including them in their assessment, the U.S. public will be left with no overall assessment of risks. If risks have been assessed by other program offices of EPA then the Agency should present them as part of the underlying data to support this TSCA Evaluation—if not, the Agency must gather the data for an assessment or include an assessment based on the assumption of near-worst-case exposures."

The SACC underscored that "[g]eneral human population and biota exposure must be assessed for inhalation, ingestion, and dermal routes [and that] [d]ifferent sub-populations may have different extents of exposure, but each route must be assessed."[17] EPA's narrower approach, it said, "strayed from basic risk assessment principles by omitting well known exposure routes such as water consumption by all occupationally and non-occupationally-exposed humans as well as similar exposures to other biological receptors."[18]

The MC risk evaluation ignores – in the SACC's words – basic "risk assessment principles" by excluding "well-known exposure routes" and failing to provide an "overall assessment of risks." Consumers who use MC-based products are also exposed to MC air emissions, particularly if they live near emitting facilities, and may also be exposed to MC through drinking water or proximity to waste sites. Similarly, workers exposed to MC at their places of employment may also inhale MC from ambient air or breathe vapors from MC-containing products used in their homes. **SACC should urge EPA to consider all known routes of exposure and, when people are exposed by multiple routes, to determine their collective contribution to overall risk.**

III. The Draft Evaluation Does Not Fully Address Risks to Vulnerable Populations

EPA also understates MC's risks because it fails to fully account for vulnerable populations and does not use all necessary uncertainty factors (UFs) in calculating benchmark MOEs. In one case, EPA has scaled back risk estimates in its 2014 risk assessment but has not justified using less protective approaches.

A. Numerous Groups Are at Increased Risk of MC's Acute Effects

Of particular concern is the greater vulnerability of certain population groups to the risks of CNS depression, coma and death from acute exposure to MC. As EPA's evaluation indicates, these groups include pregnant women, the elderly, fetuses, children, people engaged in vigorous physical activity, users of alcohol and Individuals suffering from lung and heart disease (pp. 274-275).

For example, the draft indicates that "EPA considers that increased COHb levels resulting from inhalation exposure to methylene chloride may also result in adverse effects in individuals with cardiac disease, a sensitive subpopulation." (p.259). EPA indicates that a smaller increase in COHb (between 2.5 and 4.5%) may precipitate angina than the increase (5.1%) at which neurotoxic symptoms were observed in Putz et al. (1979), the key human study EPA used as its POD for acute effects. (p. 275) Thus, unsafe MC concentrations would be lower for heart patients than for healthy individuals in the general population.

EPA also explained that:

[16] Id.
[17] Id.
[18] Id.

> "Fetuses, infants and toddlers are also potentially susceptible to methylene chloride exposure. Hemoglobin in the fetus has a higher affinity for CO than does adult hemoglobin. Thus, the neurotoxic and cardiovascular effects may be exacerbated in fetuses and in infants with higher residual levels of fetal hemoglobin when exposed to high concentrations of methylene chloride. (OEHHA, 2008b). Alexeeff and Kilgore (1983) identified an age-related difference in nervous system responses among mice as well." (p.387)

These groups, too, may be harmed by smaller CO increases, at lower exposure levels, than healthy adults.

EPA attempted to account for the enhanced susceptibility of these groups by applying a default intraspecies uncertainty/variability factor (UF) of 10. (p. 274). However, this UF is customarily used by EPA to account for normal expected variations in sensitivity within the healthy population. Here, by contrast, EPA has identified specific subgroups with biological characteristics that make it likely that they will experience adverse acute effects at lower concentrations than healthy adults. **To provide protection to these groups, a UF beyond the normal 10 X factor should be applied, as EPA has done for other susceptible groups such as infants and children.**[19]

B. EPA Has Not Justified Using a Lower IUR Than It Used in its 2011 IRIS and 2014 TSCA Assessments

The Inhalation Unit Risk (IUR) for cancer in the draft risk evaluation is 1-38 x 10^{-6} per mg/m^3. (p. 304) In the 2014 EPA assessment, the IUR was 1 x 10^{-5} per mg/m^3,[20] based on dose-response modeling in the 2011 IRIS assessment, which uses the same IUR.[21] The differences in the IURs means that the cancer risk estimates in the 2014 assessment are 6-7 times greater than in the draft evaluation. The draft evaluation does not explain how and why the two IURs are different beyond briefly acknowledging that EPA's IUR modeling has changed from 2014. It is troubling that EPA has departed from two final, peer reviewed assessments without transparently justifying this significant change in approach and specifically asking the SACC to review its scientific validity.

One possible explanation of the different IURs may involve polymorphisms, genetic factors that may increase susceptibility to cancer for some portion of the population. The IRIS assessment explains that a –

> "major pathway for dichloromethane metabolism involves the conjugation of dichloromethane to GSH, catalyzed by GST. This results in the formation of a GSH conjugate that is eventually metabolized to CO2 (Figure 3-1). The conjugation of dichloromethane to GSH results in formation of two reactive intermediates that have been proposed to be involved in dichloromethane toxicity, S-(chloromethyl)glutathione and formaldehyde."(p.14)

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[19] EPA, Consideration of the FQPA Safety Factor and Other Uncertainty Factors In Cumulative Risk Assessment of Chemicals Sharing a Common Mechanism of Toxicity, February 28, 2002, available at https://www.epa.gov/sites/production/files/2015-07/documents/apps-10x-sf-for-cra.pdf; EPA 2005 Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens (Supplemental Guidance, SG). https://www.epa.gov/risk/supplemental-guidance-assessing-susceptibility-early-life-exposure-carcinogens; Barton HA, Cogliano VJ, Flowers L, Valcovic L, Setzer RW, Woodruff TJ. Assessing susceptibility from early-life exposure to carcinogens. Environ Health Perspect. 2005;113(9):1125-33. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1280390/

[20] Workplan Risk Assessment, at 68.

[21] EPA IRIS Assessment, at 271.

The assessment states that "[r]esults from studies of GST-T1 genotypes in human blood samples indicate that average prevalences of the GST-T1 null (-/-) genotype are higher in Asian ethnic groups (47–64%) than in other groups, including Caucasians (19–20%), African-Americans (22%), and mixed groups (19%)." (p. 15) Noting that "the known polymorphisms for GST-T1 expression were integrated into the human model," it then explains that the "distributions of human inhalation unit risk values (from which the recommended [i.e., mean] values were taken) show that the 99th percentiles are approximately seven- and sixfold higher than means for liver and lung cancer, respectively." (p.253)

If EPA's risk evaluation failed to use the 99th percentile IURs to account for the high prevalence of GST-T1 expression in some population groups but instead used the means, this would explain why the IUR in the evaluation is 6-7 times lower than in the 2014 and IRIS assessments. It is very hard to know for sure, however, based on the lack of clarity in the draft evaluation. EPA's only comment on this issue is that:

> "Sampling of the full distribution of GSTT genotypes in the human population (GSTT1+/+, GSTT1+/- and GSTT1 -/-) was done to derive the I An acute, 3-hour exposure to 100 ppm dichloromethane demonstrated evidence of immunosuppression in CD-1 mice (Aranyi et al., 1986). . . Use of the upper-bound estimate for the full population distribution of the GSTT1 genotypes is considered sufficiently protective of sensitive sub-populations." (p. 659)

The SACC should ask EPA to clarify the exact reason for the lower IUR in the risk evaluation and then recommend whether EPA should use this IUR or the higher IURs in the 2011 IRIS and 2014 risk assessments.

IV. EPA Should Add an Uncertainty Factor of 3 for Database Uncertainty

Critical gaps exist in understanding of MC's human health effects. According to the 2011 IRIS assessment, "[t]he inhalation database lacks adequate developmental neurotoxicity and immunotoxicity studies at chronic low exposures." (p. 264) IRIS explained the need for additional data for the first endpoint as follows:

> "One data uncertainty identified is the potential for neurodevelopmental effects. Animal bioassays have not identified gross or microscopic effects on neural tissues from long-term exposures or single (Schwetz et al., 1975) or multigenerational (Nitschke et al., 1988b) developmental toxicity studies, albeit with the limitations regarding dosing protocol. However, behavioral changes were observed in pups born to rats exposed to high levels (4,500 ppm) of dichloromethane (Bornschein et al., 1980; Hardin and Manson, 1980); 4,500 ppm was the only dose used in this study. Thus, uncertainty exists as to the development of neurological effects from lower gestational exposures in animals or in humans." (p. 264)

In the case of immunotoxicity, the assessment said:

> "An acute, 3-hour exposure to 100 ppm dichloromethane demonstrated evidence of immunosuppression in CD-1 mice (Aranyi et al., 1986). This study used a functional immune assay that is relevant to humans (i.e., increased risk of Streptococcal pneumonia-related mortality and decreased clearance of Klebsiella bacteria). Chronic and/or repeated exposure

studies evaluating functional immunity are not available and represent a data gap." (pp. 263-64)

EPA guidance calls for an UF of 3 where the absence of complete data creates uncertainty in determining a chemical's health effects.[22] The IRIS assessment applied this UF for MC:

> "In consideration of the entire database for dichloromethane, a database UF of 3 was selected. This UF accounts for limitations in the two-generation reproductive toxicity study (i.e., discontinuous exposure throughout the lifecycle) and limitations in the design of the available developmental studies (including a lack of neurodevelopmental endpoints). There is an additional potential concern for immunological effects as suggested by a single acute inhalation study, specifically immunosuppressive effects that may be relevant for infectious diseases spread through inhalation." (p. 196)

While recognizing the same data-gaps as IRIS,[23] the draft risk evaluation does not apply a UF for data uncertainty. **SACC should urge EPA to add this UF in determining the benchmark MOE for MC's non-cancer effects.**

V. EPA's Unreasonable Risk Determinations for Workers Should Not Assume They Will be Protected by PPE

As in previous risk evaluations, EPA proposes to determine that MC's risks to workers are not unreasonable where the assumed use of Personal Protective Equipment (PPE) would reduce exposures to "acceptable" levels even though the risks would be unreasonable in the absence of PPE. However, as EPA's draft evaluations recognize and SAAC has itself underscored, this approach departs from established workplace protection policy and is contrary to the realities of worker exposure to unsafe chemicals.

To reduce worker risks below levels of concern, the EPA draft evaluations assume that "workers and occupational non-users wear respirators for the entire duration of the work activity throughout their career" and "are properly trained and fitted on respirator use." According to EPA, "similar assumptions apply to the use of gloves and their expected elimination of any dermal exposure."[24] However, the 1-BP draft risk evaluation[25] acknowledges that "[f]ew literature sources indicate the use of respirators in 1-BP conditions of use" (p. 57) and notes that "none of the workers surveyed at a Chinese facility wore PPE" (p.59) and that "small commercial facilities performing dry cleaning and spot cleaning are unlikely to have a respiratory protection program" (p. 24). The 1.4-dioxane evaluation[26] likewise recognizes that "[t]he use of a respirator would not necessarily resolve inhalation exposures since it cannot be

[22] USEPA, Reference Dose/Reference Concentration (RfD/RfC) Technical Panel, A Review of the Reference Dose And Reference Concentration Processes, December 2002, available at https://www.epa.gov/osa/review-reference-dose-and-reference-concentration-processes

[23] For example, EPA states that it "did not carry immune system effects forward for dose-response because epidemiological, animal and mechanistic data are limited and inconclusive." (p. 380)

[24] Draft Risk Evaluation for HBCD, June 2019, available at https://www.epa.gov/sites/production/files/2019-07/documents/hbcd_draft_risk_evaluation_062719_hero_link_0.pdf, at 381.

[25] https://www.epa.gov/sites/production/files/2019-08/documents/01._1-bp_draft_risk_evaluation_hero_links_external.pdf.

[26] https://www.epa.gov/sites/production/files/2019-06/documents/1_14-dioxane_draft_risk_evaluation_06-27-2019.pdf

assumed that employers have or will implement comprehensive respiratory protection programs for their employees" (p. 53). Similarly, EPA emphasizes that "[d]ata about the frequency of effective glove use – that is, the proper use of effective gloves – is very limited in industrial settings" (p. 293). And it adds that gloves provide effective protection only "if proven impervious to the hazardous chemical, and if worn on clean hands and replaced when contaminated or compromised." (p. 180).

Indeed, EPA has previously rejected reliance on respirators to reduce exposure to MC. In its January 2017 proposed ban on MC paint removers, EPA acknowledged that "not all workers may be able to wear respirators … Individuals with impaired lung function due to asthma, emphysema, or chronic obstructive pulmonary disease, for example, may be physically unable to wear a respirator."[27] EPA further observed that "individuals with facial hair, like beards or sideburns that interfere with a proper face-to-respirator seal, cannot wear tight fitting respirators," and "respirators may also present communication problems, vision problems, worker fatigue, and reduced work efficiency."[28] These considerations apply with special force to the many small facilities where MC is used and processed and where basic worker protection programs are often lacking.

The SACC has emphasized similar considerations in questioning EPA's reliance on PPE in several draft evaluations. Thus, in its review of the PV29 draft, it noted that "the analysis in the Evaluation does not discuss or account for the fact that downstream commercial users may be oblivious to chemical risks and lack even rudimentary industrial hygiene measures."[29] Similarly, in reviewing the 1,4-dioxane evaluation, the SACC concluded that the "consensus of the Committee believes that PPE may not be consistently and properly worn, as EPA assumed"[30] and noted that "[g]love use should not always be assumed to be protective" and, if worn improperly, gloves "could actually lead to higher exposures."[31] The SACC emphasized that, "[b]ecause respirators are inherently uncomfortable and potentially unreliable for long-term use, the use of respirators for more than relatively short terms is not considered appropriate in typical industrial hygiene practice." As it concluded, "8-hour use of PPE should not be used in the risk characterization of inhaled 1,4-Dioxane. Risk estimates should be presented without the use of PPE as reasonable worst case."[32]

In the case of HBCD, the SACC noted that "it was unreasonable to assume workers would wear PPE for entire 8-hour shifts due to underlying medical conditions, facial hair, discomfort, and other issues" and added that:[33]

> "[M]any members of the Committee believed EPA should place more emphasis on the limited likelihood that respiratory protection will be adopted without specific occupational exposure guidelines for HBCD . . . Dust exposures in the construction trades (especially residential construction) continue to represent an occupational health concern because of the many small-to-medium size operators and the use of temporary (and, not infrequently, undocumented) workers. Workers in these small-to-medium enterprises may not be likely to adopt personal

[27] 82 Fed. Reg. 7479.
[28] *Id.*
[29] SACC Report on PV29 at 37.
[30] SACC Report on 1,4-dioxane and HBCD, at 86.
[31] Id. at 55.
[32] Id. at 53.
[33] Id at 118.

protective equipment (PPE) controls, so EPA's characterization of reasonable risk relying on use of PPE is not sufficiently supported by the practical realities of many workplaces."

Because of the limitations of PPE, OSHA and NIOSH manage chemical risks using the "hierarchy of controls," under which hazard elimination, substitution, engineering and administrative controls are all prioritized over the use of PPE.[34] As explained by NIOSH, "[t]he hierarchy of controls normally leads to the implementation of inherently safer systems" because chemical regulation and substitution are "more effective and protective" than PPE. EPA's own risk evaluation for 1,4-dioxane likewise recognizes that "[t]he most effective controls are elimination, substitution, or engineering controls [and that] "[r]espirators, and any other personal protective equipment. . . , should only be considered when process design and engineering controls cannot reduce workplace exposure to an acceptable level" (p 52). Thus, the SACC review of the HBCD evaluation stressed that "[m]any Committee members were concerned with the reliance on PPE or engineering controls to reduce risk, as that is contrary to the hierarchy of controls." [35]

The SACC should tell EPA that its reliance on PPE to determine that unsafe MC exposures do not present unreasonable risks is not grounded in workplace realities and sound worker protection policy.

Conclusion

We appreciate this opportunity to comment on the draft MC risk evaluation and look forward to sharing our views with the SACC.

Please contact SCHF counsel Bob Sussman with any questions at bobsussman1@comcast.net

[34] OSH, Ctrs. for Disease Control & Prevention, updated Jan. 13, 2015, https://www.cdc.gov/niosh/topics/hierarchy/.

[35] SACC Report on 1,4-dioxane and HBCD, at 73.