Asbestos Disease Awareness Organization™
Voice of the Victims

Thursday, May 28, 2020

The Honorable Andrew Wheeler
Administrator
U.S. Environmental Protection Agency 1200 Pennsylvania Avenue NW
Washington DC, 20460

Re: Draft TSCA Risk Evaluation for Asbestos: EPA-HQ-OPPT-2019-0501-0001; 85 FR 18954 (April 3, 2020)

Dear Administrator Wheeler:

Please find the attached comments of the Asbestos Disease Awareness Association (ADAO) on EPA's draft risk evaluation for asbestos under the Toxic Substances Control Act (TSCA).

Asbestos is likely the most hazardous substance in widespread use since the industrial revolution and is responsible for millions of deaths worldwide. Asbestos is universally recognized to have no safe level of exposure. Although it has been banned in over 60 countries, most uses of asbestos are lawful in the US. ADAO has been a strong and outspoken advocate for a comprehensive ban on asbestos in this country.

As ADAO shows in the attached comments, we are deeply concerned by the many omissions and flaws in the draft risk evaluation. Although EPA has determined that asbestos presents unreasonable risks under the limited conditions of use it addresses, these risks are greatly understated because EPA –

- delays consideration of legacy asbestos exposure to an uncertain future risk evaluation
- focuses on only one asbestos fiber type
- does not consider cancers and non-cancer lung effects linked to asbestos
- ignores environmental exposure to asbestos
- excludes asbestos-contaminated talc products and exposures
- lacks basic information about asbestos importation and use that it should have obtained using TSCA information collection authorities
- unjustifiably relies on respirators to protect workers from asbestos exposure, and
- does not account for increased risks to subpopulations with greater susceptibility to asbestos or multiple pathways of exposure

EPA also departs from the well-established scientific framework for estimating asbestos risks and calculates an IUR considerably lower than the long-standing IUR adopted by IRIS in the 1980s.

These flaws have resulted in a risk evaluation that fails to present a full and accurate picture of the threat that asbestos poses to public health and will undermine asbestos risk management policies now in place. We believe a stronger and more comprehensive risk evaluation based on the best available science is essential both to provide a compelling basis for action under TSCA to eliminate exposure to asbestos and to assure that the public is fully informed about the serious, ongoing danger of asbestos to public health.

We appreciate the Agency's consideration of our comments.

Sincerely,
Linda Reinstein, Asbestos Disease Awareness Organization, President and Cofounder
Robert Sussman, A Asbestos Disease Awareness Organization, Counsel
cc: Assistant Administrator Alex Dunn

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

Comments of Asbestos Disease Awareness Organization on Draft EPA Risk Evaluation for Asbestos Under the Toxic Substances Control Act

EPA-HQ-OPPT-2019-0501-0001; 85 Fed. Reg. 18954 (April 3, 2020)

Submitted By:

Linda Reinstein, Asbestos Disease Awareness Organization, President and Cofounder

Robert Sussman, Asbestos Disease Awareness Organization Counsel

May 27, 2020

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Comments of Asbestos Disease Awareness Organization on Draft EPA Risk Evaluation for Asbestos Under the Toxic Substances Control Act

EPA-HQ-OPPT-2019-0501-0001; 85 Fed. Reg. 18954 (April 3, 2020)

SUMMARY

The Asbestos Disease Awareness Organization (ADAO) submits these comments on the Environmental Protection Agency (EPA) draft risk evaluation on asbestos under the Toxic Substances Control Act (TSCA).

About ADAO

Launched in 2004, ADAO is now the largest independent non-profit organization in the U.S. dedicated to eliminating asbestos-caused diseases. ADAO is far more than an asbestos victims' organization; our cutting-edge research, ongoing product testing, and educational efforts have enabled us to be a leading stakeholder in prevention policy. We have been a strong and outspoken advocate for a comprehensive US ban on asbestos, championing enactment of the Alan Reinstein Ban Asbestos Now Act of 2019 (ARBAN), which has bi-partisan support in both houses of Congress.

ADAO was founded by Linda Reinstein and Doug Larkin after both of their loved ones were diagnosed with mesothelioma. Slowly but surely, it grew into a network of around 50,000 individuals as more and more victims, families, scientists, nonprofits, and trade unions joined us in pursuit of our shared goal of eliminating asbestos-related diseases. ADAO's Science and Prevention Advisory Boards are comprised of world class experts in asbestos-related disease and exposure. Many Board members contributed their insights to the development of these comments, and several are commenting separately and/or making oral statements to the SAAC during its upcoming June 8 public meeting.

Since TSCA was amended in 2016, ADAO has expressed its views at every stage of the risk evaluation process, submitting extensive information to EPA, filing comments and position statements on key milestones and meeting often with EPA leadership and staff.

Dangers of Asbestos

Asbestos is likely the most hazardous substance in widespread use since the industrial revolution and is responsible for millions of deaths worldwide. Asbestos is universally

recognized to have no safe level of exposure.[1] Although it has been banned in over 60 countries, most uses of asbestos are lawful in the US.

The death toll from asbestos exposure in the US remains alarmingly high. A recent study by Dr. Jukka Takala DSc, MSc, BSC, President of the International Commission of Occupational Health (ICOH), and colleagues reported that asbestos-related diseases are causing an average of 39,275 deaths in the United States annually -- more than double the previous estimates of 15,000 per year.[2] These findings reinforce the urgent need to eliminate exposure to asbestos and underscore the continuing public health challenge that asbestos presents.

Asbestos: A Poster Child for TSCA Reform

EPA's draft evaluation is its first comprehensive assessment of asbestos risks since the 1980s and responds to the new mandates in the 2016 TSCA amendments, which were enacted in part because of frustration with EPA's inability to regulate asbestos. In 1989, the Agency issued a rule under section 6(a) of TSCA prohibiting most asbestos uses[3] but, following an industry challenge, the rule was overturned in 1991 by the Fifth Circuit Court of Appeals based on limitations in TSCA unrelated to the risks of asbestos.[4] During the TSCA reform process in 2016, there was bipartisan agreement that asbestos was a poster child for TSCA's failure to protect public health, and that any new law needed to ensure that EPA could finally do its job and ban asbestos. Many in Congress and the public hoped EPA would use its expanded authority to conduct risk evaluations and regulate unsafe chemicals to reinstate the 1989 asbestos ban.

In December 2016, shortly after the passage of the new law, EPA selected ten chemicals for initial risk evaluations, including asbestos.[5] ADAO and many others hoped that the risk evaluation would initiate a process leading to a comprehensive asbestos ban. However, this hope faded as EPA narrowed the scope of the risk evaluation, used a questionable Significant New Use Rule (SNUR) to track but not ban the reintroduction of discontinued asbestos products, and refused to use its broad TSCA information collection authorities to require reporting by industry of essential use and exposure information.

Failing to Fulfill the Promise of Amended TSCA

The draft risk evaluation confirms our concerns about EPA's failure to act forcefully to address asbestos. It contains numerous exclusions and limitations that result in an incomplete picture of

[1] Part I of these comments provides an overview of the scientific consensus on the risks of asbestos and its enormous economic and human toll in the US.

[2] S. Furuya, O. Chimed-Ochir, K. Takahashi, A. David, and J. Takala, "Global Asbestos Disaster," *International Journal of Environmental Research and Public Health,* vol. 15, no. 5, p. 15, 2018.

[3] Asbestos: Manufacture, Importation, Processing, and Distribution in Commerce Prohibitions (54 Federal Register 29460, July 12, 1989) (FRL–3476–2)

[4] *Corrosion Proof Fittings v. EPA*, 947 F.2d 1201 (5th Cir. 1991).

[5] 81 Federal Register 91927 (December 19, 2016).

the current impacts of asbestos on public health and underestimate exposure and risk. The evaluation also departs from the established scientific framework used by EPA and other agencies over the last three decades, using novel and questionable methodologies that result in risk estimates considerably lower than in previous assessments.

Importance of SACC Review

For these reasons, careful and probing review of the draft evaluation by the independent EPA Science Advisory Committee on Chemicals (SACC) is essential. Unfortunately, the SACC review has been handicapped by EPA's insistence on an unrealistic and compressed schedule at the very time that the scientific and medical community is overwhelmed by the coronavirus pandemic.[6] We hope that SACC members and consultants will be able to give the draft evaluation the full attention it deserves but are deeply concerned that EPA's flawed process will prevent a robust peer review.

Significant Ongoing Exposure to Asbestos

Although some have characterized the remaining active uses of asbestos as negligible, the EPA evaluation demonstrates that ongoing exposure from these uses is in fact significant. Current asbestos users include 15 plants in the large chlorine manufacturing industry. According to the United States Geological Survey (USGS), this industry imported 750 metric tons of raw asbestos in 2018 from Brazil and Russia.[7] Asbestos brake linings and gaskets remain in use in US vehicle manufacturing and in the large aftermarket for auto replacement parts. Both the chemical and oil industries may be large users of asbestos-containing products although EPA lacks information on the full extent of these uses.

Even with the limited information available to EPA, the Agency estimates that close to a million workers are exposed to asbestos from current commercial uses.[8] Given the absence of worker population estimates for a number of uses, the actual number may be higher – even without counting the millions of workers who are exposed to "legacy" asbestos in homes, businesses and schools across the US. EPA also estimates that 31,857,106 consumer do-it-yourselfers (DIYs) may have exposure to asbestos when replacing brake pads in their own or others'

[6] On March 30, ADAO wrote to Administrator Wheeler to request a delay of the SACC review – a request also made by some SACC members. EPA initially denied this request and then postponed the SACC meeting until early June because a quorum of members could not attend the originally scheduled late April meeting. Even with this postponement, there is reason for concern that the SACC will be unable to provide the detailed and thoughtful feedback essential for meaningful peer review. For a chemical as dangerous and well-studied as asbestos, truncating the SACC process in this manner is unacceptable.

[7] EPA Office of Chemical Safety and Pollution Prevention, *Draft Risk Evaluation for Asbestos,* March 2020 (Risk Evaluation), at 33.

[8] Risk Evaluation at 205, Table 4-54. This includes 167,000 oil industry workers and Occupational Non-users (ONUs) who may be exposed to asbestos at up to 21,670 sites (pp. 84-85) and 749,900 workers in automotive repair and maintenance shops who may be exposed to asbestos brake linings, clutches and gaskets (p.92).

vehicles.[9] The public health benefits of protecting these large worker and consumer populations from exposure to asbestos are undeniable.

Findings of the Draft Risk Evaluation

The draft risk evaluation finds that nearly all ongoing commercial and consumer uses of asbestos reviewed by the Agency *present an unreasonable cancer risk under TSCA to workers, occupational non-users, consumers, and bystanders***.** This includes asbestos-containing diaphragms, asbestos-containing sheet gaskets, asbestos-containing brake blocks, aftermarket asbestos-containing brakes/linings, other vehicle friction products, and other asbestos-containing gaskets. Importantly, the unreasonable risk findings apply to the chlor-alkali industry, the one remaining importer of raw bulk asbestos in the United States, which has argued for decades that its use of asbestos is safe.

Flaws and Limitations in the Draft Evaluation

While ADAO strongly supports these conclusions, we believe that, overall, the draft evaluation contains several gaps, limitations and deficiencies. By excluding numerous exposure pathways and asbestos-related diseases and disregarding relevant studies, the draft seriously understates asbestos-related risks. It is important for SACC to highlight these flaws in its recommendations to EPA. Unless significantly revised, the evaluation will be a step backward in scientific understanding and weaken current policies to protect Americans from cancer and other serious diseases linked to asbestos exposure. A stronger risk evaluation that uses more comprehensive exposure information and better science and removes the many exclusions and limitations in the draft would both reinforce EPA's determinations of unreasonable risk and support more protective and stringent restrictions as asbestos moves into the TSCA risk management process.

Our concerns about the draft evaluation – presented in detail in the remainder of these comments – are as follows:

- The draft evaluation does not address the risks of legacy asbestos products despite a US court of appeals decision requiring EPA to evaluate these risks. Legacy asbestos is pervasive in US buildings and is a significant contributor to ongoing asbestos-related death and disease. A comprehensive assessment of the risks of asbestos to the US population is impossible without accounting for exposure to legacy asbestos.

- The risk evaluation only addresses the chrysotile form of asbestos and disregards other recognized fiber types. Legacy asbestos products contain multiple fibers and some current products (like asbestos-contaminated talc) include a mix of fibers. Thus, real-world exposure is to multiple fiber types. As a result, prior assessments have drawn on available data for all commercially used fibers to estimate risks. There is no credible basis for differentiating among asbestos fibers based on lung cancer and mesothelioma

[9] Id. at 204-205.

risk. Thus, a risk evaluation focused only on chrysotile exposure is scientifically unjustified. EPA should expand the evaluation to include the six fiber types included in the TSCA definition of asbestos plus the extremely hazardous "Libby amphibole."

- EPA's Inhalation Unit Risk (IUR) – the driver of EPA's risk determinations – would lower estimated asbestos risks considerably as compared to the broadly accepted Integrated Risk Information System (IRIS) IUR and uses a flawed approach similar to the "binning" framework that the EPA Science Advisory Board (SAB) rejected in 2008. Like IRIS, EPA should base the IUR on a broad mix of studies for different fibers and industries, treat all fibers as equally potent and use the protective linear model for low dose extrapolation.

- The draft risk evaluation is based solely on the carcinogenicity endpoints of lung cancer and mesothelioma. It does not address other types of tumors (like ovarian and laryngeal cancers) and serious non-cancer lung diseases (like asbestosis) known to be caused by asbestos. EPA itself acknowledges that these omissions result in a substantial underestimation of risk. All cancer and non-cancer endpoints for asbestos should be included in the final evaluation.

- Departing from TSCA's comprehensive framework for chemical risk management and disregarding previous SACC recommendations, the draft evaluation excludes all environmental pathways of exposure to asbestos. Asbestos present in ambient air, drinking water and waste is a well-documented source of exposure that overlaps with and magnifies workplace and consumer product sources. Experts consider environmental exposure a significant contributor to overall risk. EPA must consider these exposure pathways in its final risk evaluation.

- EPA has ignored the documented presence of asbestos contamination in talc-based crayons and other consumer products to which infants and children are exposed as well as in workplaces where industrial talc is used. It is known that exposure to asbestos-contaminated talc can cause mesothelioma and ovarian cancer. There is no legal justification for ignoring these TSCA-regulated sources of exposure merely because asbestos is present in talc as a contaminant and not an intended constituent. TSCA has consistently been interpreted to apply to impurities and, even if not intended, the "known" or "reasonably foreseen" presence of asbestos in talc is a TSCA "condition of use" that the risk evaluation must address.

- The draft evaluation relies on limited submissions by industry and publicly available information to identify ongoing conditions of use and determine the magnitude and extent of current asbestos exposure. In following this approach, the Agency rejected petitions in 2018 from ADAO, other organizations and 18 Attorneys General (AGs) to require reporting under TSCA section 8(a) by importers, processors and users of raw asbestos and asbestos-containing products. Although EPA insisted at the time that it did not need any additional information, the draft evaluation demonstrates otherwise. Throughout the evaluation, EPA admits that it lacks reliable information on the

quantities of asbestos involved in most ongoing uses, the companies and number of facilities using asbestos-containing products, the nature of the use operation and the total number of workers and consumers exposed. It also lacks adequate workplace monitoring data for all conditions of use and has erroneously excluded at least two documented uses (asbestos yarn and cement) from the draft evaluation. These gaps could have been avoided if EPA used its TSCA information collection authorities, as the petitioners requested. EPA should now use these authorities to obtain the data and other information necessary for an informed risk evaluation.

- With no supporting evidence except broad and unverified industry assurances, EPA concludes that asbestos importation, distribution in commerce and certain disposal activities do not present an unreasonable risk of injury. In fact, as the industry itself recognizes, spills, accidents or damaged bags and containers of asbestos can result in exposure during loading, unloading, transportation and waste shipment and handling. These exposures are likely to present significant risks to workers and bystanders given the dangers of exposure to even small amounts of asbestos. EPA's final evaluation should reverse the conclusions of the draft and determine that all importation, distribution in commerce and disposal of asbestos presents an unreasonable risk of injury.

- EPA should not base its risk determinations for workers on the assumed use of respirators to reduce exposure. As SACC has previously advised, relying on personal protective equipment (PPE) to reduce risk to workers is contrary to the established industrial hygiene policy of using product substitution, work practices and engineering controls as the primary tools for worker protection, with PPE as a last resort. SACC has also recognized that use of PPE in the real world is highly variable and uneven. Asbestos is no exception: EPA has provided no evidence to demonstrate consistent, reliable and protective use of respirators across the conditions of use addressed in its evaluation.

- EPA's risk evaluation fails to address risks to potentially exposed or susceptible subpopulations (PESSs) which require special protection under TSCA. These subpopulations include individuals exposed to asbestos across multiple routes and pathways and persons at increased risk such as cigarette smokers and individuals with underlying lung disease.

I. Asbestos Impact on Public Health

For over a century, asbestos has been known to cause widespread disease and death. In a monograph on asbestos published in 2012, the International Agency for Research on Cancer (IARC) found the following cancers in humans to be causally related to asbestos exposure: lung cancer, malignant mesothelioma, ovarian cancer, and cancer of the larynx.[10] There is

[10] IARC. Monograph 1OOC: Asbestos (Chrysotile, Amosite, Crocidolite, Actinolite· and· Anthophyllite), Lyon: International Agency for Research on Cancer (2012)

considerable evidence in the scientific literature of causal associations with gastrointestinal cancers and kidney cancer. Non-malignant diseases are also caused by asbestos. These include asbestosis and asbestos-related pleural thickening. "There is general agreement among scientists and health agencies . . . [e]xposure to any asbestos type (i.e., serpentine [chrysotile] or amphibole) can increase the likelihood of lung cancer, mesothelioma, and nonmalignant lung and pleural disorders."[11] Accordingly, all fiber types in commercial use have been regulated with equal stringency by OSHA, EPA and other government agencies.

For the last 120 years, use of asbestos has been massive in scale. According to the U.S. Geological Survey (USGS):[12]

- From 1900 to today, the U.S. has consumed more than 31 million metric tons of asbestos;
- From 1991 to 2002, the U.S. has mined 111,420 metric tons of asbestos until the last domestic mine closed in 2002;
- From 1991 to 2018 the EPA has allowed 280,325 metric tons of asbestos to be imported.

The human cost of asbestos exposure has been staggering and the death toll enormous. From 1991 to 2017, more than one million Americans died from preventable asbestos-caused diseases.[13] These deaths represent only a snapshot in time; the total number of deaths during the 100+ years of asbestos use is much larger. The economic cost of inaction has been and remains immense: "The economic burden of lung cancer and mesothelioma associated with occupational and para-occupational asbestos exposure is substantial." According to the World Health Organization (WHO) report *Asbestos Economic Assessment of Bans and Declining Production and Consumption,* "[t]he substantial costs associated with the continued use of asbestos potentially outweigh any other economic benefit."[14] The annual global health care costs associated with the health effects of asbestos are estimated to be US $ 2.4–3.9 billion, excluding the additional costs of pain, suffering and welfare losses.[15]

The American Thoracic Society has stated that "[a]sbestos has been the largest single cause of occupational cancer in the United States and a significant cause of disease and disability from nonmalignant disease."[16] The danger extends far beyond manufacturing plants— firefighters, construction workers, auto mechanics and repairmen and school teachers are among the workers at highest risk for asbestos exposure and related diseases. Asbestos fibers can also be

[11] U.S. Public· Health Service, U.S. Department of Health & Human Services. Toxicological Profile for asbestos. Atlanta: Agency for Toxic Substances and Disease Registry; (2001) (ToxProfile). .
[12] https://pubs.usgs.gov/circ/2006/1298/
[13] http://ghdx.healthdata.org/gbd-results-tool?params=gbd-api-2017 permalink/535c35ab1fc10471f721c9b58eecd3c2
[14] https://www.nera.com/publications/archive/2017/asbestos--economic-assessment-of-bans-and-declining-production-a.html
[15] http://www.euro.who.int/data/assets/pdf_file/0009/341757/Asbestos_EN_WEB_reduced.pdf?ua=1.
[16] https://www.atsjournals.org/doi/full/10.1164/rccm.200310-1436ST

carried home on the workers' clothing, skin, and hair, thus exposing their family members to non-occupational asbestos exposure.

Despite the elimination of many asbestos products due to corporate liability, asbestos deaths – calculated to be nearly 40,000 per year as noted above – remain high, demonstrating that millions of Americans have been significantly exposed to asbestos in the past and many others are exposed now.

There is overwhelming consensus in the scientific community that there is no safe level of exposure to asbestos. As noted by WHO:[17]

> Bearing in mind that there is no evidence for a threshold for the carcinogenic effect of asbestos, including chrysotile, and that increased cancer risks have been observed in populations exposed to very low levels, the most efficient way to eliminate asbestos-related diseases is to stop using all types of asbestos.

IARC[18], the Occupational Safety and Health Administration (OSHA)[19], the Department of Health and Human Services,[20] the National Institute for Occupational Safety and Health (NIOSH)[21], the World Health Organization (WHO) and a number of other regulatory and public health bodies recognized asbestos as a human carcinogen decades ago.

In his comments on the draft evaluation,[22] Dr. Richard Lemen, formerly Acting Director of NIOSH and Assistant Surgeon General of the United States and co-chair of ADAO's SAB, has explained the sequence of studies demonstrating the absence of a safe level of asbestos exposure:

> Epidemiological studies that have actually studied the effect of low levels of asbestos exposure have concluded that there are no "*safe"* doses of exposure. Such studies have derived these conclusions by actually following those with *"low"* dose exposures. The danger in relying on only select epidemiology studies is revealed by the growth in our historical understanding of asbestos hazards. In the early 1970s, studies by McDonald (1973)[23] estimated exposures to asbestos below 200-300 fiber/cc years were not

[17] https://www.who.int/ipcs/assessment/public_health/chrysotile_asbestos_summary.pdf
[18] http://monographs.iarc.fr/ENG/Monographs/vol100C/mono100C.pdf.
[19] https://www.osha.gov/laws-regs/federalregister/1994-08-10
[20] https://ntp.niehs.nih.gov/ntp/roc/content/profiles/asbestos.pdf.
[21] https://www.cdc.gov/niosh/docs/2011-159/pdfs/2011-159.pdf
[22] COMMENTS OF RICHARD A. LEMEN, Ph.D., MSPH ON EPA's DRAFT RISK EVALUATION FOR ASBESTOS 27 May 2020 (Lemen Statement) at 1.
[23] McDonald, J.C., 1973. Cancer in chrysotile mines and mills. In: Biological Effects of Asbestos, International Agency for Research on Cancer. Eds. P. Bogovski, J.C. Gilson, V. Timbrell, J.C. Wagner. IARC Scientific Publications No. 8: 189-194.

associated with increased cancer deaths. By 1980 a publication by McDonald[24] found no increased risk of lung cancer deaths below 20 fiber/cc years, a level ten times lower. By 1998, Iwatsubo et al.[25] found exposures of 0.5-0.99 fiber/cc years produced four-fold increased risk of cancer. And by the early 2000s, Rodelsperger et al. (2001)[26] found roughly eight-fold increased risk at exposures above 0.15 fiber/cc years. 200 … 20 … 0.5 … 0.1 … The historical lessons repeatedly show we are incapable of identifying a threshold level of exposure below which individuals are not at risk of asbestos disease.

Asbestos fibers can become respirable when asbestos-containing materials and products are disturbed or become friable. The primary route of asbestos entry into the body is inhalation; however, fibers are also ingested and are found in drinking water.

OSHA has three standards to protect workers from the hazards of asbestos in the workplace. These standards apply to the general Industry, shipyards, and construction. However, OSHA standards are by law limited by considerations of economic and technical feasibility.[27] Thus, in adopting its asbestos standards, OSHA conceded that they would not eliminate significant cancer risks to workers. Rather, the Agency estimated 3-4 workers per 1,000 would develop lung cancer even if every employer fully complied with asbestos exposure limits.[28] This underscores the unique benefits of TSCA in protecting against workplace exposures. Under TSCA section 6(a), EPA has authority to fill gaps in worker protection by imposing additional requirements (including a ban on importation and use) where necessary to eliminate unreasonable risks to workers.

II. The Draft Evaluation Does Not Address the Risks of Legacy Asbestos Products Despite a Court Decision Requiring EPA to Evaluate These Risks

A. Legacy Asbestos Is Pervasive in Buildings Across the US and is a Major Contributor to Asbestos Disease and Death

For most of the twentieth century, numerous asbestos-containing products—including attic and wall insulation, pipes and boilers, floor tiles, gaskets, roofing, shingles and siding—were widely used in constructing homes, schools, apartments, public buildings, offices, stores, and factories. This asbestos remains in place in millions of structures across the country. Much of the asbestos is in friable form and can be released into the air when disturbed during routine

[24] McDonald, J.C., Liddell, F.D.K., Gibbs, G.W., Eyssen, G.E., McDonald, A.D., 1980. Dust exposures and mortality in chrysotile mining, 1910-75. Br J Indust Med; 37: 11-24.
[25] Iwatsubo, Y., Pairon, J.C., Boutin, C., Ménard, O., Massin, N., Caillaud, D., Orlowski, E., Galateau-Salle, F., Bignon, J., Brochard, P., 1998. Pleural mesothelioma: Dose-response relation at low levels of asbestos exposure in a French population-based case-control study. Am J Epid; 148(2): 133-142.
[26] Rodelsperger, K., Jockel, K.-H., Pohlabeln, H., Romer, Woitowitz, H.-J., 2001. Asbestos and man-made vitreous fibers as risk factors for diffuse malignant mesothelioma: results from a German hospital-based case-control study, Am. J. Ind. Med., 39, 262-275.
[27] *American Textile Mfgs. Institute, Inc. v. Donovan (ATM)*, 452 U.S. 490, 508-11 (1981).
[28] https://www.osha.gov/laws-regs/standardinterpretations/1999-07-23

building maintenance and upkeep. A large population of workers and consumers is exposed to legacy asbestos on an ongoing basis.

The incidence of asbestos-related disease is elevated in populations with exposure to legacy asbestos. A 2013 study by NIOSH researchers examined cancer incidence and mortality among firefighters in San Francisco, Chicago, and Philadelphia and found that "the population of firefighters in the study had a rate of mesothelioma two times greater than the rate in the U.S. population as a whole" and that "it was likely that the[se] findings were associated with exposure to asbestos, a known cause of mesothelioma."[29] Studies have also found that school teachers, particularly in elementary and middle schools, are at higher risk of mesothelioma than the general population, due to the widespread presence of asbestos in schools built in the 1960s and 1970s.[30]

There is also widespread exposure to asbestos-containing debris that enters waste streams during renovation and demolition of buildings where legacy asbestos is present. Asbestos waste continues to be generated and managed in the U.S. in significant quantities. The movement of asbestos waste in commerce and poor waste management at landfills and construction sites pose a significant danger to workers and the public.

Emergency response crews and volunteers (as well as building occupants) are at high risk of legacy asbestos exposure in the wake of fires and other disasters. Where the duration of exposure is prolonged and more exposure events occur, the risk of asbestos-related disease is increased.[31] A well-studied disaster resulting in widespread asbestos release was the 2001 attack on the New York World Trade Center (WTC).[32] When the twin towers collapsed, "thousands of tons of particulate matter consisting of cement dust, glass fibers, lead, asbestos, polycyclic aromatic hydrocarbons (PAHs)" and other pollutants were expelled into the environment. The pollutants spread over Manhattan and Brooklyn for miles beyond the WTC site. Although the elevated airborne levels of asbestos declined eventually, the settled dust at and around Ground Zero had concentrations ranging between 0.8 and 3.0%.

Waste management is crucial after any disaster, due to the large amounts of debris waste that typically contains toxic substances, including asbestos. "Poor waste management not only causes environmental pollution in water, soil and air, but also causes harm to human health, particularly that of workers. 5000 tons of ACBMs [Asbestos Containing Building Materials] were

[29] R. D. Daniels *et al.*, "Mortality and cancer incidence in a pooled cohort of US firefighters from San Francisco, Chicago and Philadelphia (1950-2009)," *Occupational and Environmental Medicine,* vol. 71, no. 6, pp. 388-397, Jun 2014.
[30] https://www.inquirer.com/education/a/mesothelioma-philadelphia-school-district-lea-dirusso-cancer-20191121.html
[31] C. Bianchi and T. Bianchi, "Malignant mesothelioma: Global incidence with asbestos," (in English), *Industrial Health,* Review vol. 45, no. 3, pp. 379-387, Jun 2007.
[32] P. J. Landrigan *et al.*, "Health and environmental consequ nter disaster," *Environmental Health Perspectives,* vol. 112, no. 6, pp. 731-739, May 2004.

released during the collapse of the World Trade Center in 2001, and the amount of asbestos fibers discharged was 555 times greater than the permissible level."[33]

In 1984, EPA conducted a survey to determine the extent of the use of friable asbestos-containing materials in US buildings and the amount of asbestos in them.[34] The survey focused on federally owned buildings; apartment buildings; and commercial buildings. Single-family homes, small rental properties, schools, factories and non-federal public buildings were not addressed. The report reached several significant conclusions, including that 20 percent of buildings had asbestos-containing friable material, 16 percent of buildings had asbestos-containing pipe and boiler insulation, the average asbestos content in friable material was 14 percent. and 14 percent of asbestos-containing material was significantly damaged.

No comprehensive assessment of legacy asbestos exposure has been conducted in the last 35 year despite the likelihood that EPA's 1984 findings are out-of-date and no longer represent the extent of damaged asbestos in buildings and the level of risk of disease and death which this asbestos now presents. The TSCA risk evaluation is a critical tool to update our understanding of the current prevalence and condition of legacy asbestos in US buildings, the number of people exposed and the magnitude of the ongoing risk.

While EPA may believe that legacy asbestos is adequately regulated under existing laws and regulations, this is a misconception, According to the comments of Brent Kynoch, chair of ADAO's Prevention Committee and a recognized expert in asbestos abatement:[35]

> While EPA might assume that employers have knowledge of the presence of ACM in buildings, this is generally not true. There is no EPA requirement to do a complete building survey (inspection) for the presence of ACM except that which is required for schools (K-12, 40 CFR Part 763, Subpart E). This means that there are a vast number of buildings where there never has been a complete survey, nor have workers been trained even at the basic awareness level as is required by EPA (schools) and OSHA in their regulations. To this day there are many workers on a daily basis performing necessary tasks with no knowledge of the presence of ACM in their work, nor have they been training in required worker protection and work practices. These "unknowing, unprotected" exposures are not considered by EPA in the Risk Evaluation, which obviously leads to an under assessment of the exposures and risk associated with existing asbestos.

[33] Y. C. Kim and W. H. Hong, "Optimal management program for asbestos containing terials to be available in the event of a disaster," *Waste Management,* vol. 64, pp. 272-285, Jun 2017.
[34] USEPA, *Asbestos in Buildings:* A *National Survey of Asbestos-Containing Friable Materials*. Washington, DC: Office of Toxic Substances, EPA 560/5-84-006 (1984).
[35] Comments of J. Brent Kynoch, Managing Director Environmental Information Association, in response to the Draft Risk Evaluation for Asbestos at 2.

A comprehensive examination of potential risks under TSCA will lay the groundwork for new, more effective protections against legacy asbestos exposure.

B. EPA's Repeated Assertion that TSCA Does Not Apply to Legacy Asbestos has been Rejected by a US Court of Appeals

Since initiating its risk evaluation in December 2016, the Agency has claimed that the risks of legacy asbestos are beyond its authority under TSCA. This interpretation was rejected by the U.S. Court of Appeals for the Ninth Circuit in its Noveeaders mber 14, 2019 decision in *Safer Chemicals, Healthy Families v USEPA*, No. 17-72260 (9th Cir. Nov. 14, 2019). The Ninth Circuit held "that EPA's exclusion of legacy uses and associated disposals contradicts TSCA's plain language" (id. at 46) and that "TSCA's 'conditions of use' definition plainly addresses conditions of use of chemical substances that will be used or disposed of in the future, regardless of whether the substances are still manufactured for the particular use" (id at 53). The Court was well aware that its conclusion applied to asbestos, noting that "[f]or example, although asbestos is now infrequently used in making new insulation, it remains in place in previously installed insulation" (id. at 46) and that "future disposal of asbestos insulation . . . unambiguously falls within TSCA's definition of 'conditions of use'" (id. at 54)

However, EPA indicates that it "continues to review the recent court decision" and that "this draft risk evaluation does not reflect consideration of any legacy uses and associated disposal for chrysotile asbestos or other asbestos fiber types as a result of that decision. EPA intends to consider legacy uses and associated disposal in a supplemental scope document and supplemental risk evaluation."[36] We believe the exclusion of legacy exposure from the current evaluation is a fundamental flaw and needs to be corrected.

TSCA requires risk evaluations to look holistically at all sources of exposure that contribute to risk.[37] The SACC has been critical of prior risk evaluations that omit sources of exposure and fail to aggregate the contribution of different exposure pathways to overall risk. Thus, in its report on the draft evaluation for 1,4-dioxane, 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."[38] EPA's narrower approach, it said, "strayed from basic risk assessment principles by omitting well known exposure routes such as water consumption by all

[36] Risk Evaluation at 18.

[37] Risk evaluations under section 6(b)(4)(A) must determine "whether a chemical substance presents an unreasonable risk of injury to health or the environment." This requirement cannot be met without examining all sources of exposure that contribute to health and environmental risk. Similarly, section 6(b)(4)(A) provides that a risk evaluation must determine the substance's risks under "the conditions of use." This broad term spans the entire life cycle of a chemical and is defined under section 3(4) to mean "the circumstances . . . under which a chemical substance is intended, known or reasonably foreseen to be manufactured, processed, distributed in commerce, used or disposed of."

[38] 1,4-Dioxane and HBCD SACC Report, at 18.

occupationally and non-occupationally-exposed humans as well as similar exposures to other biological receptors."[39]

Workers and consumers exposed to the asbestos-containing products addressed in the draft evaluation are likely also exposed to legacy asbestos in their places of employment, homes and other buildings they frequent. Unless these sources of exposure are assessed in combination, risks of asbestos-related disease will be underestimated. Moreover, since the population exposed to legacy asbestos is much larger than the worker and consumer population exposed to current-asbestos containing products, the omission of legacy uses will result in a severely incomplete and misleading picture of the magnitude and level of asbestos exposure and the overall US incidence of asbestos-related mortality and disease.

We believe that EPA should expand the current evaluation to address all legacy asbestos exposure rather than artificially bifurcate current and legacy asbestos uses and assess the latter in a future evaluation several years in the future. At an absolute minimum, EPA should account for legacy exposures in making risk determinations for the ongoing asbestos uses addressed in the current evaluation. In its 2017 framework rule for TSCA risk evaluations, EPA claimed (erroneously as it turned out) that TSCA did not require it to make risk findings for legacy exposures but also said that "[i]n a particular risk evaluation, EPA may consider background exposures from legacy use, associated disposal, and legacy disposal as part of an assessment of aggregate exposure or as a tool to evaluate the risk of exposures resulting from non-legacy uses."[40] Asbestos is clearly a compelling case for following this approach.

III. The Risk Evaluation Should Not be Limited to Chrysotile but Should Encompass All Recognized Asbestos Fibers

A. EPA and Other Agencies Have Addressed All Asbestos Fiber Types Without Differentiation and Used Data on All Fibers to Estimate Risks and Set Exposure Limits

The definition of asbestos in Title II of TSCA (section 202) encompasses six types of fibers:

> The term ''asbestos'' means asbestiform varieties of— (A) chrysotile (serpentine), (B) crocidolite (riebeckite), (C) amosite (cummingtonite-grunerite), (D) anthophyllite, (E) tremolite, or (F) actinolite.

This definition, which is codified in Title II of TSCA, is incorporated verbatim in EPA's Asbestos-Containing Materials in Schools Rule, Asbestos Ban and Phaseout Rule, Asbestos National Emission Standards for Hazardous Air Pollutants (NESHAP) and recent significant new use rule under TSCA requiring notification of resumption of discontinued asbestos uses. OSHA workplace standards have likewise applied to the six fiber types without differentiation and

[39] Id.
[40] 82 Fed. Reg. 33726, 33730 (July 20, 2017).

have been based on risk assessments that derive from available studies for all the recognized fibers.

The 1988 peer reviewed IRIS assessment for asbestos, which was the basis for the EPA TSCA regulations in 1989 banning most asbestos uses,[41] established a single Inhalation unit risk (IUR) value for the six fiber types using studies of exposure to multiple fiber types.[42] The IRIS summary indicated that:[43]

> There is some evidence which suggests that the different types of asbestos fibers vary in carcinogenic potency relative to one another and site specificity. It appears, for example, that the risk of mesothelioma is greater with exposure to crocidolite than with amosite or chrysotile exposure alone. This evidence is limited by the lack of information on fiber exposure by mineral type. Other data indicates that differences in fiber size distribution and other process differences may contribute at least as much to the observed variation in risk as does the fiber type itself.

In 2008, the EPA Superfund program proposed a departure from the IRIS IUR which used an "interim approach to account for the potential differences of cancer potency between different mineral types and particle size distributions at different human exposure conditions."[44] The proposal would establish a "'multi-bin' mathematical approach to estimate cancer risk according to mineral groups (amphibole or chrysotile) and particle size (length and width) based on transmission electron microscopy." The EPA Science Advisory Board (SAB) was asked to review the Superfund proposal and during its public meeting on July 21-22, 2008, numerous asbestos scientists expressed strong opposition to using "bins" to differentiate between the risks of fiber types.

In its November 14, 2008 letter to EPA Administrator Johnson and accompanying report, the SAB Asbestos Committee advised that its members "generally agreed that the scientific basis as laid out in the technical document in support of the proposed method is weak and inadequate" and that "the document was woefully inadequate with respect to the representation of available information on epidemiology, toxicology, mechanism of action and susceptibility."[45] In response to these concerns, EPA Administrator Stephen Johnson accepted the Committee's conclusion ''that the quality of the available exposure data was generally insufficient to support the effort EPA proposed'' and announced that the proposed risk assessment would not be pursued further.[46]

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[41] These regulations were almost entirely overturned in the 1991 decision in *Corrosion Proof Fittings v. EPA* by the U.S. Court of Appeals for the Fifth Circuit.
[42] https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0371_summary.pdf#nameddest=rfc.
[43] Id at 9.
[44] SAB Consultation on EPA's *Proposed Approach for Estimation of Bin-Specific Cancer Potency Factors for Inhalation Exposure to Asbestos,* November 14, 2008 (SAB Consultation).
[45] Id.
[46] Letter from Stephen L. Johnson, EPA Administrator, to Dr. Agnes Kane, Chair of Science Advisory Board Asbestos Committee, December 29, 2008.

The new draft TSCA risk evaluation seeks to revive the discredited approach of developing fiber-specific potency values – in this case focused on chrysotile because, according to EPA, the products covered by the evaluation all contain the chrysotile form of asbestos. Disturbingly, the draft does not acknowledge the 2008 SAB deliberations and explain why EPA has now concluded that fiber-by-fiber risk determinations are warranted despite their rejection twelve years ago.

B. A Chrysotile-Only Approach Does Not Account for Real-World Exposure to Multiple Fibers

There are additional factors that weigh against limiting the draft evaluation to chrysotile and not considering exposure to amphiboles and other fibers in estimating asbestos risks to consumers and workers.

Of most importance, legacy asbestos products contain a mix of fibers, not just chrysotile, and there is widespread ongoing exposure to multiple fibers due the presence of these products in millions of buildings. For example, vermiculite contaminated with amphibole has been used as insulation in some 10-30 million homes and can be released into indoor and outdoor air when there is disruption caused by extreme weather or home remodeling or demolition, exposing residents and construction workers. Other types of building materials now installed in homes and other structures were made with amphibole, including shingles, roofing materials, insulation around pipes and boilers. Amphibole fibers can be released when these building components are disrupted, such as during repairs, maintenance and demolition work. Asbestos fibers are also known to be released during fires in buildings and these fibers (which include amphiboles) pose a well-documented risk to firefighters and other emergency responders , as noted above.

Workers and consumers exposed to current chrysotile-containing products may also have been exposed to other fibers earlier in their work careers when construction materials containing these fibers were still in active use. They may also be exposed currently to these fibers as a result of the presence of legacy construction materials in homes and other structures where they now reside and work. Determining asbestos risks to these workers and consumers based solely on their exposure to chrysotile in current products would fails to account for all pathways of exposure and understate risks.

Even if (contrary to our recommendations), EPA defers consideration of legacy asbestos exposure to a future risk evaluation, it will at that time need to account for the risks of multiple fiber types and will be unable to base risk determinations on studies pertaining to chrysotile alone. For this reason, as well as the lack of scientific basis for fiber-specific determinations of carcinogenic potency, EPA should rework the current draft evaluation so it uses a single set of risk values for all asbestos fibers.

In addition, chrysotile, tremolite and anthophyllite fibers are currently found as contaminants in talc-based consumer products and in industrial talc used in tire manufacturing and other

industries. As discussed below, asbestos contained in talc is a significant pathway of exposure for many Americans and should be included in the EPA risk evaluation. Workers and consumers now exposed to current chrysotile-containing products are potentially exposed to talc and thus to other asbestos fibers which contribute to overall risk.

There is also air contamination from naturally occurring rock containing amphibole, which has been found in ambient air in numerous locations where this rock is mined and processed for use in producing cement and other building materials. Studies demonstrate that exposure to these fibers can contribute to mesothelioma risk.[47] This would be an additional source of exposure to asbestos fibers other than chrysotile.

Thus, the EPA evaluation should examine the risks of all fiber types customarily defined as asbestos. Basing risk estimates on chrysotile-related studies alone both lacks a sound scientific basis and is not reflective of asbestos exposure in the real world.

C. EPA Should Include Libby Amphibole in the Fiber Types Addressed in the Risk Evaluation

The non-asbestiform varieties of winchite and richterite, which are often referred to as "Libby

Amphibole," do not technically fall within the TSCA Title II definition of asbestos but are generally recognized as having the properties of asbestos, including the capacity to cause serious health effects. Libby Amphibole was found in vermiculite ore mined near Libby, MT and extensively distributed throughout the United States during the 20th century. As ADAO has repeatedly requested, Libby Amphibole should be addressed in EPA's risk evaluation because of its large contribution to asbestos-related mortality and disease and ongoing potential for widespread exposure.

According to an article published in 2017, Dr. Brad Black, CEO and Medical Director for the Center for Asbestos Related Disease (CARD) clinic in Libby stated, "I think the mortality rates are really high here, with just the non-malignant effects from the material…The [burden of] progressive fibrotic disease has been very significant. It has not just involved the former vermiculite workers, who obviously had very high exposures, but also those who were exposed environmentally. We've lost a number of people to lung disease, including people who just lived and worked in Libby, but not at the mine."[48]

Since its opening in 2000, The CARD clinic has screened approximately 7500 people, and of those, Dr. Black estimates that 3400-3500 have some level of asbestos-related disease.[49] According to Dr. Black, 700 – 800 people are screened every year. In Libby and the surrounding Lincoln county, the rate of lung disease is 50-60% higher than the national average. This rate is

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[47] Pan et al., *Residential proximity to naturally occurring asbestos and mesothelioma in California.* Am. J. Respir. Crit. Care Med. 172:1019- 1025 (2005).
[48] A. van Dorn, "Libby: the long legacy of a public health disaster," (in eng), *Lancet Respir Med,* vol. 5, no. 3, pp. 174-175, 03 2017.
[49] Id.

likely an underestimation because death certificates do not always appropriately link the cause of death to asbestos[. Furthermore, Dr. Black has found that low-level exposure, even lower than the allowed exposure rate in the workplace, causes harmful pleural plaquing. [50]

For decades, vermiculite mined in Libby was used throughout the U.S. to produce Zonolite attic insulation, which is estimated to be in as many as 35 million US homes, buildings, and offices.[51] During its investigations at the Libby mine, EPA obtained over 80,000 vermiculite concentrate shipping invoices from W.R. Grace for the period that the company owned the mine (1964–1990). An analysis of EPA's summary of these invoices indicated that a total of approximately 6,109,000 tons of vermiculite concentrate were shipped to 245 sites across the country.[52] W.R. Grace processed an estimated 200,000 tons of vermiculite from the Libby mine each year until the mine finally ceased operations in 1990. Mining and processing of vermiculite containing this form of asbestos in Libby, Montana resulted in EPA declaring a public health emergency in this small town in 2008.

In sum, Libby Amphibole is a significant contributor to legacy asbestos exposure through its widespread use in attic insulation across the US, its continued presence at the Libby mining site and the likelihood of further exposure from contamination of the many inactive where it was used to produce Zoolite attic insulation. No assessment of legacy asbestos exposure and risk would be complete it if did not include this type of asbestos.

IV. EPA's Inhalation Unit Risk (IUR) Understates Asbestos Risks

The basis for EPA's proposed determination of unreasonable risk is its Inhalation Unit Risk (IUR) for chrysotile asbestos, which it uses to estimate risk levels for each of the conditions of use addressed in its draft evaluation. These risk levels are then compared to EPA "benchmarks" for evaluating the "unreasonableness" of cancer risks. For workers, a cancer risk of 1×10^{-4} or greater is considered unreasonable; for the general population and consumers, the threshold is 1×10^{-6}.

The 1988 IRIS assessment established an IUR of 2.3E-1 per (f/mL) for lung cancer and mesothelioma.[53] EPA and other agencies have relied on this IUR (or modifications) for exposure limits for asbestos over the last 32 years. The IRIS IUR is applicable to all asbestos fibers without differentiation and assumes that chrysotile and amphiboles are equally potent in causing both lung cancer and mesothelioma.[54] In 1989, when adopting a ban on most asbestos products, EPA again concluded that its risk assessment should assume that all asbestos fibers have equal potency.[55] OSHA reached the same conclusion in 1986 and 1994.

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[50] Id.

[51] https://www.usgs.gov/news/usgs-scientists-develop-new-tool-determine-if-vermiculite-insulation-contains-asbestos

[52] https://www.atsdr.cdc.gov/asbestos/sites/national_map/Summary_Report_102908.pdf

[53] https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0371_summary.pdf#nameddest=rfc

[54] 51 Fed. Reg. 22612 (1986).

[55] 54 Fed. Reg. 29467. (1989)

By contrast, the IUR in the EPA risk evaluation is 0.16 (per f/cc) (p. 155). If finalized, the new IUR would be 50 percent below the IRIS value. This would have significant implications for exposure limits now in place or adopted in the future since the new IUR would indicate that the risk of lung cancer and mesothelioma for asbestos is smaller than IRIS concluded.

The IRIS IUR was based on an analysis of 15 epidemiological studies examining a mix of asbestos fibers and industries. As explained by leading experts, the IUR was derived using a dose response model that:[56]

> assumed the following: Equal potency for chrysotile and the amphiboles; equal potency for all fibers longer than 5 mm; no threshold exposure level for carcinogenicity; a multiplicative interaction between asbestos exposure and cigarette smoking for lung cancer; relative risks for lung cancer that vary linearly with cumulative exposure lagged by 10 years; and death rates for mesothelioma that vary as a linear function of concentration and a cubic function of time since first exposure.

In contrast, the new IUR is specific to chrysotile and derives from two studies (in North Carolina and South Carolina) of textile workers. EPA picks the North Carolina study as the driver for mesothelioma and the South Carolina study as the driver for lung cancer and then uses an "exponential" model to extrapolate to lower levels of exposure on the ground that it represents the "best fit" to the data. On this basis, EPA rejects a linear method of extrapolation – the approach used in the IRIS assessment.

We believe the science does not support lowering the IUR as proposed by EPA and that the draft evaluation uses a flawed approach similar to the "binning" framework that the EPA SAB reviewed and rejected in 2008. Dr. Lemen has emphasized that "In light of the long history of scientific and regulatory agencies around the world evaluating the risks for disease among workers and others exposed to asbestos and concluding there is no evidence of a threshold or *safe* level of exposure to asbestos, any new evaluation should proceed cautiously and with respect for well-established science and public health policies."[57] EPA has not met this standard:

There are five fundamental problems with the new EPA IUR as described below.

A. The IUR Should not be Limited to Chrysotile and Studies of Textile Workers But Should Reflect All Asbestos Fibers and Use Epidemiological Data For All Industries

[56] Michael A. Silverstein, Laura S. Welch, and Richard Lemen, *Developments in Asbestos Cancer Risk Assessment*, Am. J. Ind. Med. 52:850–858, 2009 (Silverstein et al.)

[57] Lemen Comments at 3.

During development of the now-abandoned binning approach in the early 2000s, there was strong opposition to developing separate potency factors for chrysotile and other fibers, as proposed by Berman and Crump in papers commissioned by the Superfund program. For example, a 2003 expert review of an early version of the binning approach expressed the following concerns: [58]

> The 2003 report repeated earlier cautions that grossly imperfect exposure characterization in the epidemiology studies creates substantial uncertainties in the estimation of potency factors, including both random and systematic biases. Among the specific data flaws mentioned were unrepresentative sampling strategies, use of surrogate measures in the absence of actual asbestos measures, lack of data from earlier time periods, and reliance on area samples rather than personal breathing zone measures. Concerns were raised by members of the 2003 expert panel convened by the EPA that the epidemiologic exposure data underlying the risk assessment models was inadequate, particularly for estimating fiber size specific risk estimates. It was also noted that the results for lung cancer were unstable and highly dependent on which studies were included. Sensitivity calculations by one reviewer, Dr. Leslie Stayner, found that when the Quebec miners and millers were excluded chrysotile had twice the potency of amphiboles, but when the South Carolina textile workers were excluded the amphiboles had ten times the potency of chrysotile.

During the 2008 SAB review, comments by Dr. Mitchell Silverstein and 83 other experts maintained that: [59]

> The proposed OSWER method will produce unreliable estimates of risk and should not be used for public health purposes. It relies on exposure assessments that are irreparably flawed, a problem that cannot be overcome by statistical modeling."[60] The comments emphasized that "[t]here is no compelling scientific basis for estimating different potency factors for lung cancer by fiber type and OSWER should take bins that assume this off the table. Stayner, Dankovic and Lemen have reasoned convincingly that 'there is absolutely no epidemiologic or toxicologic evidence to support the argument that chrysotile asbestos is any less potent than other forms of asbestos for inducing lung

[58] Silverstein et al at 852.

[59] Stayner L et al. Occupational exposure to chrysotile asbestos and cancer risk: A review of the amphibole hypothesis. Am J Public Health. 1996;86:179-186.

[60] Comments to EPA Science Advisory Board Asbestos Committee on Office of Solid Waste and Emergency Response (OSWER) Proposed Approach for Estimation of Bin-Specific Cancer Potency Factors for Inhalation Exposure to Asbestos, submitted July 7, 2008 by Michael Silverstein, MD, MPH on behalf of 83 co-signers

cancer' and that 'chrysotile appears to be just as potent a lung carcinogen as the other forms of asbestos.'

Looking back on the 2008 SAB review, Silverstein et al offered this overview: "The repeated efforts by the EPA to characterize the relative cancer potencies for different asbestos fiber types and sizes have not been able to overcome the limitations of the exposure data in the epidemiological studies, and the resulting problems with the 2008 model led EPA to conclude that it could not be used to make public policy decisions."[61] They endorsed the continuing viability of the IRIS approach from the 1980s because it "made appropriately conservative assumptions in estimating the risk for asbestos across all fiber types, for example, in assuming equal potency and not attempting to determine exact risks for subgroups of fiber types." Overall, Silverstein et al underscored that the "epidemiologic evidence on asbestos exposure and health outcomes is limited in important ways that render a *fiber specific* asbestos risk assessment troublesome" (emphasis in original). Dr. Lemen indicates that EPA's draft evaluation suffers from the same flaw: "The idea of separating out studies that "isolate" the effects of chrysotile and basing risk estimates on these studies alone is inherently flawed because of the uncertainty in the exposure levels in these studies and the difficulty of using limited data (i.e. the two textile studies) to attribute specific potency factors to individual fibers."[62]

B. By Developing a Chrysotile-Specific IUR, the Draft Evaluation Inappropriately Fails to Consider and Integrate a Large Body of Epidemiological Studies

EPA's draft does exactly what Silverstein et al describe as "troublesome" – it conducts a *fiber specific* asbestos risk assessment.

Because EPA concluded (erroneously in our view) that "commercial chrysotile is . . . the substance of concern for this quantitative assessment," it "sought to derive an IUR specific to chrysotile asbestos."[63] This led to the further decision that "studies of populations exposed only to chrysotile provide the most informative data for the purpose of developing the TSCA risk estimates." [64] As a result, while EPA's literature search identified more than 24,000 studies on asbestos, its risk evaluation was based on only 26 papers covering seven occupational cohorts.[65]

In this winnowing process, "EPA distinguished between studies of exposure settings where only commercial chrysotile was used or where workers exposed only to commercial chrysotile could be identified, and situations where chrysotile was used in combinations with amphibole asbestos forms and the available information does not allow exposures to chrysotile and amphibole forms to be separated. Studies in the latter group were judged to be uninformative

[61] Silverstein et al, at 856.
[62] Lemen Comments at 5.
[63] Risk Evaluation at 132.
[64] Id.
[65] Id at 134.

with respect to the cancer risks from exposure to commercial chrysotile and were excluded from further consideration."[66]

In the end, "EPA identified studies of five independent occupational cohorts exposed only to commercial chrysotile that it believed provided adequate data for assessment of lung cancer risks."[67] See Table 3-2. From these studies, EPA chose the North Carolina study to quantify mesothelioma risk and South Carolina study to quantify lung cancer risk and derived a single composite IUR for the two types of malignancy combined.[68] This reliance on two studies – out of the large number of epidemiological studies on asbestos – greatly magnifies uncertainties since different studies have shown different levels of risk and all studies (including the North and South Carolina studies) have limitations in tracking exposure levels, deaths and other inputs that weaken their reliability standing alone.

Given that there is no valid basis to conclude that chrysotile in less potent than other fibers, a robust "weight of evidence" analysis of the epidemiological literature should include studies on *all fiber types*. As described in EPA's risk evaluation rule, an assessment of the weight of evidence requires "a systematic review method, applied in a manner suited to the nature of the evidence or decision, that uses a pre-established protocol to comprehensively, objectively, transparently, and consistently, identify and evaluate each stream of evidence, including strengths, limitations, and relevance of each study and to integrate evidence as necessary and appropriate based upon strengths, limitations, and relevance."[69] However, EPA's draft evaluation falls short of this standard, because it excludes most of the epidemiological literature on asbestos and thus fails to systematically "integrate evidence . . .based on [i]ts strengths, limitations and relevance."

By contrast, the IRIS IUR was "based on an analysis of the unit exposure risk for lung cancer and mesothelioma in 11 studies [later increased to 15]." In this analysis,[70] "all studies that provide exposure-response information [were] utilized . . . along with estimates of the uncertainty of such data. An appropriate weighted average of the relationships found in different studies, taking into account observable differences in exposure circumstances, yields an overall exposure-response relationship." EPA also considered adopting the approach used in the draft risk evaluation – "select[ing] the study or studies with the best exposure data, assuming an adequate measure of effect" – but rejected it. The Agency said that this approach might have "particular merit in evaluating the risk from an agent whose exposure can be well characterized, such as that from a single chemical species" but that "this is not the case with asbestos." As it indicated, in the asbestos epidemiological studies, "[c]urrent estimates of what such concentrations might have been can be inaccurate, since individual exposures were highly

[66] Id.
[67] Id. 135.
[68] Id. 155.
[69] 40 CFR § 702.33.
[70] EPA Office of Research and Development, Office of Health and Environmental Assessment, *Airborne Asbestos Health Assessment Update,* EPA/S00/8-84/003F June 1986 (OHEA Assessment)

variable. Further, while disease response now can be established through epidemiological studies, these, too, can be misleading because of methodological limitations."

We recommend that the draft risk evaluation be revised so that the IUR is based on all suitable studies for all asbestos fiber types.

C. *The use of an "exponential" rather than a "linear model" to determine cancer potency departs from past asbestos risk assessments and EPA policy and is not protective*

EPA's 2005 Guidelines for Carcinogen Risk Assessment[71] emphasize the high level of evidence necessary to depart from the presumption of linearity for carcinogens:

> Elucidation of a mode of action for a particular cancer response in animals or humans is a data-rich determination. *Significant information should be developed to ensure that a scientifically justifiable mode of action underlies the process leading to cancer at a given site. In the absence of sufficiently, scientifically justifiable mode of action information, EPA generally takes public health protective, default positions* regarding the interpretation of toxicologic and epidemiologic data animal tumor findings are judged to be relevant to humans, and cancer risks are assumed to conform with low dose linearity (emphasis added) (1-10 through 1-11).

The Guidelines add that:

> When the weight of evidence evaluation of all available data are insufficient to establish the mode of action for a tumor site and when scientifically plausible based on the available data, linear extrapolation is used as a default approach, because linear extrapolation generally is considered to be a health-protective approach. Nonlinear *approaches generally should not be used in cases where the mode of action has not been ascertained.* (emphasis added) (3-21). A nonlinear approach should be selected when there are sufficient data to ascertain the mode of action and conclude that it is not linear at low doses and the agent does not demonstrate mutagenic or other activity consistent with linearity at low doses. (3-22).

It is broadly recognized that there is no threshold for asbestos-induced lung cancer and mesothelioma and that any level of exposure to asbestos can cause these diseases. As EPA stated in the 1986 OEHA assessment that forms the basis for the IRIS IUR:[72]

> In the discussion of the time relationship of lung cancer risk and asbestos exposure, the data can be interpreted in terms of a multistage model of cancer in which asbestos appears to act at a single late stage. The continued high risk following cessation of exposure results from the continued presence of asbestos in the lungs. This model is compatible with a linear dose-response relationship.

[71] EPA Cancer Guidelines, at 84-85.
[72] OEHA Assessment, at 25.

Further:[73]

> The accumulated data suggest that the excess risk of death from lung cancer from asbestos exposure is proportional to the cumulative exposure (the duration times the intensity) and the underlying risk in the absence of exposure. . . . As with lung cancer, the risk [of mesothelioma] appears to be proportional to the cumulative exposure to asbestos in a given period.

However, the draft risk evaluation bases its IUR on an "exponential" rather than a "linear" model for dose response. As it explains, "[b]etween the linear relative rate and exponential model forms for lung cancer mortality in both SC and NC cohorts, the exponential models clearly fit better."[74]

Significantly, EPA's choice of a non-linear model is based solely on statistical "curve fitting." The risk evaluation cites no mechanistic or other biological basis to conclude that the exponential model better approximates asbestos' mode of action – a prerequisite for overcoming the presumption of linearity under EPA's cancer risk assessment guidelines. In fact, others who have examined the SC and NC data have concluded that "the best model for lung cancer was linear on a multiplicative scale with the best data fit obtained when the threshold was set at zero."[75] Equally important, EPA only performs its "curve fitting" analysis on two epidemiology studies. However, as described above, the preferable approach would have been to base the IUR on all studies providing dose-response information for asbestos fibers – a much larger body of data. Without analyzing all these studies, EPA makes a large leap of faith in concluding that the exponential model is broadly applicable to asbestos carcinogenicity.

Given the large uncertainties in individual studies due to imprecision in exposure measurement and disease tracking, the "good fit" of the exponential model for the SC and NC cohorts might be unique to these studies and inapplicable to others. As emphasized in Silverstein et al, "[t]rying to turn fundamentally unreliable data into valid and reliable output is statistical alchemy, no matter how sophisticated and complex the mathematical models."[76] Dr. Lemen similarly states: "The exponential response curve used in the Draft Risk Evaluation is suspect and may understate risk because the imprecision in the exposure levels does not provide a strong basis for this type of "curve fitting" modeling. This exponential model should not replace the more conservative linear approach that EPA and others have traditionally used for asbestos." [77]

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[73] Id at 1.
[74] Risk evaluation at 153.
[75] Markowitz, *Asbestos-Related Lung Cancer and Malignant Mesothelioma of the Pleura: Selected Current Issues, Semin. Respir. Care Med. 36:334-346 (2015)*
[76] Silverstein et al at 855-866.
[77] Lemen Comments at 6.

The EPA risk evaluation compares IURs based on the exponential and linear models for the textile worker studies. The high end of the range for the linear model runs is an IUR 0.33 per f/cc (SC cohort) while the lower end of the range for the exponential runs is an IUR of 0.08 per f/cc (NC cohort).[78] This is a four-fold difference, illustrating the implications of model choice for the protectiveness of the IUR and the risk levels EPA uses for its determinations of unreasonable risk.

EPA should revise its IUR derivation to use the traditional, protective linear low-dose model consistent with its cancer risk assessment guidelines.

D. EPA's Use of Mortality Rather than Incidence Data for the IUR Is Underprotective

The IUR in the draft evaluation is based on cancer mortality rather than cancer incidence because of the absence of lung cancer or mesothelioma incidence data in the two asbestos textile cohorts on which EPA relies. The draft evaluation acknowledges that this results in an underestimation of risk:[79]

> Thus, because the cancer slope factor (KM) is based on the absolute risk, any missed incident cases of mesothelioma will necessarily underestimate the total mesothelioma risk associated with chrysotile asbestos and in the absolute risk model even one incident case close to the follow-up date and missed in follow-up will increase the risk estimate.

However, EPA argues that the absence of incidence data in the two studies only has a limited impact on the risk estimates:[80]

> According to the National Cancer Institute's Surveillance Epidemiology and End Results (SEER) data on cancer incidence, mortality, and survival (Howlader et al., 2013), the median length of survival for lung cancer is less than 1 year, with 2-year survival for males about 25% and 5-year survival for males about 17%. For lung cancer, any bias would be expected to be low because the cancer slope factor (KL) is estimated based upon the relative risk. For mesothelioma, the median length of survival with mesothelioma is less than 1 year, with 2-year survival for males about 20%, and 5-year survival for males about 6%.

Thus, EPA concludes that any bias introduced by omission of incidence data for lung cancer will be low based on an assumed low relative risk due to the low number of survivors.

However, the SEER data cited by EPA are for the period 1975-2010.[81] As described in the comments of Dr. Christine Oliver, a member of ADAO's SAB, "[t]hey They are likely outdated

[78] Risk evaluation, Table 3-11.
[79] Risk evaluation at 155.
[80] Id at 154-154.
[81] Howlader N, Noone AM, Krapcho M, Garshell J, Newman N, Altekruso SF, et al, eds. SEER Cancer Statistics Review, 1975-2010. Bethesda, MD: National Cancer Institute. 2013.

because of the feasibility and effectiveness of low dose chest CT (LDCT) lung cancer screening in reducing mortality from lung cancer, and improved treatment of malignant mesothelioma."[82] Dr. Oliver explains that:

> The NIH National Lung Screening Trial (NLST) was stopped in 2010 when it became clear that LDCT screening significantly reduced mortality from lung cancer.[18] Eligibility criteria for screening were established by a number of organizations and government bodies and programs became operational across the country soon thereafter.
>
> In February, 2020 the Centers for Disease Control and Prevention examined and reported the prevalence of LDCT screening for lung cancer in 2017 in 10 states among survey participants who met U.S. Preventive Services Task Force (USPSTF) eligibility criteria and those who did not.[19] Weighted percentage for the total who underwent LDCT screening was 12.5 (10.4-14.9) for those who met the USPSTF criteria, and 7.9 (6.8-9.1) for those who did not. For those who are screened, longer survival has been shown.

Based on recent trends toward longer survival for lung cancer and mesothelioma, we believe EPA is minimizing the impact on the IUR of failing to account for disease incidence. *We recommend that EPA adjust the IUR upward by an amount that reflects the projected number of missed cases of cancer and mesothelioma corresponding to the latest data on survival rates.*

E. The Draft Evaluation Improperly Minimizes the Significance of Differences in PCM and TEM Measurement Techniques In Interpreting the Studies It Uses for the IUR

Phase contrast microscopy (PCM), historically the most common method for counting asbestos fibers, measures only those fibers >5 mm in length and does not have the resolution needed to identify fibers <0.25 mm in diameter.[83] Transmission electron microscopy (TEM) is a more sensitive method, now generally preferred, and is more effective in identifying smaller and thinner fibers believed to be more carcinogenic. According to Silverstein et al, "[u]ndercounting of thin chrysotile fibers could inadvertently lead to the incorrect attribution of observed risks to the thicker measurable fiber types" and this could bias dose-response analysis.[84] For this reason, "efforts have been made to convert total fiber counts to counts of specific fiber types, lengths, and diameters based on data in the late 1970s and early 1980s in which samples were analyzed with TEM."[85] However, this conversion has proven difficult and the 2008 SAB review rejecting the binning approach indicated that a "primary concern is the lack of available data to

[82] COMMENTS OF L. CHRISTINE OLIVER, MD, MPH, MSc, FACPM ON EPA DRAFT RISK EVALUATION FOR ASBESTOS (Oliver Comments), at 4.
[83] Dement J, Kuempel E, Zumwalde R, Smith R, Stayner L, Loomis D. 2008. Development of a fibre size-specific job-exposure matrix forairborne asbestos fibres. Occup Environ Med 65:605–612.
[84] Silverstein et al, at 853.
[85] Id at 854.

estimate the TEM specific levels of exposure for the epidemiological studies utilized in this analysis.''[86]

Recalculating PCM-based exposure levels as TEM-based levels is difficult because ''PCM and TEM results do not correlate well, and no generally applicable conversion factor exists between the two measurement techniques."[87] As explained in the 1986 OEHA assessment:[88]

> Modern counting techniques may be utilized to evaluate work practices and ventilation conditions believed to be typical of earlier activities. However, it is always difficult to duplicate materials and conditions of earlier decades so that such retrospective estimates are necessarily uncertain. Alternatively, fiber counting techniques using the particle counting instrumentation of earlier years can be used now to evaluate a variety of asbestos containing aerosols. The comparative readings would then serve as a calibration of the historic instrument in terms of fiber concentrations. Unfortunately, the calibration depends on the type and size distribution of the asbestos used in the process under evaluation and on the quantity of other dust present in the aerosol. *Thus, no universal conversion has been found between earlier dust measurements and current fiber counts*

In the draft risk evaluation, EPA acknowledged that "the IUR for 'current use' asbestos (i.e., chrysotile) is based solely on studies of PCM measurement as TEM-based risk data are limited in the literature and the available TEM results for chrysotile lack modeling results for mesothelioma."[89] However, it asserted that "[i]n TEM studies of NC and SC . . . models that fit PCM vs TEM were generally equivalent (about 2 AIC units), indicating that fit of PCM is similar to the fit of TEM (for these two cohorts). According to EPA. this "provid[es] confidence in those PCM measurements for SC and NC" and enables the Agency to conclude that "exposure uncertainty is considered low in the cohorts used for IUR derivation."

However, Dr. Oliver has looked closely at the PCM and TCM data for the two cohorts and concluded that the fits were not in fact equivalent for the two measurement methods:[90]

> Thus, these findings indicate a similar fit to the risk models of TEM and PCM fiber counts, but the number of fibers and the range of fiber sizes counted by TEM is much greater than that of PCM. The greatest strength of association with lung cancer was with fibers <0.25 μ in diameter, a width that is undetectable by PCM. Loomis et al. conclude as follows: "… the association of lung cancer with fibres <0.25 μ in diameter in the combined cohort supports the use of TEM and other improved methods to assess

[86] SAB Consultation
[87] Snare J. 2005. OSHA Standard Interpretation letter from Assistant Secretary Jonathan Snare to U.S. Senator Conrad Burns. 6/30/2005.Silverstein et al at 854.
[88] OEHA Assessment at 43.
[89] Risk Evaluation at 197.
[90] Oliver Comments at 6.

> asbestos fibre exposure for research and regulatory purposes; these fibres are too small to be counted with the PCM method that has been the standard since the 1960s."

Dr. Oliver concluded that the "adoption by the EPA of a new statistical model to calculate an IUR for asbestos should be left until a time when the exposure analyses using TEM have reached a point of practical scientific and regulatory application." We agree with this conclusion and believe that the uncertainties cited by Dr. Oliver greatly weaken confidence in use of the PCM data from the North Carolina study to derive an IUR and further illustrate why deriving an IUR from a single study is a flawed approach.

V. The Exclusion of Other Cancer Types and Non-Cancer Disease Is a Further Reason Why the Draft Evaluation Understates Risk

A. EPA Does Not Address Several Other Cancers Linked to Asbestos Exposure

The draft risk evaluation is based solely on the carcinogenicity endpoints of lung cancer and mesothelioma. It does not address other types of tumors or serious non-cancer lung diseases known to be caused by asbestos. EPA suggests that the omission of these endpoints has little impact on its risk estimates, but this is incorrect.

IARC has found that a causal association between exposure to asbestos and cancer of the larynx is clearly established based on:[91]

> fairly consistent findings of both the occupational cohort studies as well as the case-control studies, plus the evidence for positive exposure-response relationships between cumulative asbestos exposure and laryngeal cancer, cancer of the larynx reported in several of the well-conducted cohort studies. This conclusion was further supported by the meta-analyses of 29 cohort studies encompassing 35 populations and of 15 case-control studies of asbestos exposure and laryngeal cancer, cancer of the larynx undertaken by the [Institute of Medicine] (2006).

IARC also concluded that a causal association between exposure to asbestos and cancer of the ovary was clearly established, based on "strongly positive cohort mortality studies of women with heavy occupational exposure to asbestos."[92]

Numerous studies also show a positive association between exposure to asbestos and cancer of the pharynx, based on the positive findings of a series of well-conducted cohort studies of

[91] IARC. Monograph 1OOC:- Asbest s (Chrysotile, Amosite, Crocidolit Actinolite· and· Anthophyllite), Lyon: International Agency for Research on Cancer (2012) (citing IOM, Asbestos: Selected Cancers. Institute of Medicine of the National Academy of Science [http://!>..<!oks.nap.edu/catalog/11665.html] (2006)) (IARC Monograph)

[92] Id.

populations occupationally exposed to asbestos.[93] IARC likewise found a positive association between exposure to asbestos and cancer of the colorectum, based on the "fairly consistent findings of the occupational cohort studies, plus the evidence for positive exposure-response relationships between cumulative asbestos exposure and cancer of the colorectum consistently reported in the more detailed cohort studies."[94] These findings, combined with evidence of asbestos-related kidney tumors,[95] point to a pattern of gastrointestinal malignancies caused by ingestion of asbestos fibers[96] – a route of exposure addressed by IRIS but excluded from the draft risk evaluation.

EPA admits that failure to include cancers of the larynx and ovary "is a downward bias leading to lower IUR (inhalation unit risk) estimates in an overall cancer health assessment."[97] However, it appears to defend this omission on the basis that there is a "lack of sufficient numbers of workers to estimate risks of ovarian and laryngeal cancer."[98] The basis for this statement is unclear since EPA provides no discussion and analysis of the data base for these cancers. EPA does indicate that "lacked quantitative estimates of the risks of cancers of the larynx and the ovary from chrysotile asbestos"[99] but this begs the question whether quantitative risks for these tumors could be determined using studies for all six fiber types – arguably a more valid basis for IUR derivation as discussed above.

EPA also minimizes its omission of cancers of the larynx and ovary by pointing out that the "selected IUR was chosen to compensate for this risk."[100] According to the draft, the four calculated IURs for the NC study ranged between 0.08 and 0.16 per f/cc and EPA picked the higher end of the range because it "was most likely to cover the total risk of incident cancers."[101] Why a slightly higher IUR would be sufficient to account for the risk of other cancers is not obvious since EPA did not review the database for these cancers and has no basis for estimating the magnitude of the risk they represent. Moreover, the range of IURs from which EPA selected its definitive IUR was itself not conservative because it was based on the exponential rather than the more defensible linear model as discussed above.

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[93] Selikoff et al., Asbestos-associated deaths among insulation workers in the United States and Canada, 1967-1987. Ann NY Acad Sci, 643: 1 Third Wave 1-14 (1991); Sluis-Cremer et al., *The mortality of amphibole miners in South Africa, 1946-80,* Br. J. Indust. Med. 49: 566-575 (1992); Reid et al. *Aerodigestive and gastrointestinal tract cancers and exposure to crocidolite (blue asbestos): incidence and mortality amongformer crocidolite workers,* Int. J. Cancer 111: 757-761 (2004); Pira et al., *Cancer mortality in a cohort of asbestos textile workers,* Br. J. Cancer, 92: 580-586 (2005).

[94] IARC Monograph

[95] Smith et al., *Asbestos and kidney cancer: the evidence supports a causal association,* Am. J. Ind. Med. 16(2):159-66 (1989).

[96] Frumkin et al., *Asbestos exposure and gastrointestinal malignancy review and meta-analysis,*- J. Indust. Med.14: 79-95 (1988); Gamble, *Risk of gastrointestinal cancers from inhalation and ingestion of asbestos* Regul. Toxicol. Pharmacol., 52: Supp1S124-S153 (2008).

[97] Draft risk evaluation, at 197.

[98] Id.

[99] Id at 155.

[100] Id. at 197.

[101] Id. at 155.

B. EPA Fails to Address Serious Non-Cancer Diseases (Asbestosis and Pleural Plaque) Related to Asbestos Exposure

Dr. Oliver emphasizes the important role of nonmalignant asbestos-related disease in morbidity and mortality in the United States. As she explains:[102]

> Of greatest import is asbestosis, non-malignant disease of the lung parenchyma. Contrary to common belief, asbestosis is not a disease of the past. As recently as 2014, the death rate in the U.S. was approximately 6/million persons/year. Using asbestos exposure data from the South Carolina textile plant relied upon by the EPA in its IUR calculation, Stayner et el and Hein et al. modeled the relationship between occupational exposure to asbestos and asbestosis. The data show a linear relationship between cumulative exposure to chrysotile and asbestosis in white men 60 to 74 years of age born in 1920 or later. . . . Both the disease itself and invasive procedures may be associated with considerable morbidity and/or mortality.

Pleural plaques are generally asymptomatic but may be associated with impairment in pulmonary function. Death from respiratory failure attributable to asbestos-related diffuse pleural thickening has been reported.[12]

Workers with asbestos-related interstitial lung disease experience diminished pulmonary function, including measures of forced vital capacity (FVC) and of the gas exchange capability of the lung (i.e., diffusing capacity).[103] The pleural disease caused by exposure to the Libby Amphibole Asbestos can result in significant decreases in pulmonary function and the ability to carry out activities of daily living.[104] Individuals with this disease suffer from severe chest pain requiring narcotic medication.

As described in Dr. Oliver's comments, "[n]onmalignant asbestos-related disease is important as a marker of increased risk for lung cancer" as well a causing significant loss of pulmonary function, reduced mobility and suffering in its own right.[105] In a hospital-based case-control study, Wilkinson et al observed a twofold increase in lung cancer risk in 93 patients with definite or probable occupational asbestos exposure and ILO radiographic profusion of small opacities of $\geq$ 1/0 (odds ratio (OR) 2.03, 95% CI 1.00-4.13).[13] In a cohort study of 1,532 smokers with occupational asbestos exposure and lung cancer, Cullen et al observed a significant increase in lung cancer risk with increasing ILO profusion $\geq$ 1/0, taking into account co-variates

[102] Oliver Comments at 3.
[103] Lilis R, Miller A, Godblod J, Chan E, Selikoff IJ. 1991. Pulmonary function and pleural fibrosis: Quantative relationships with an integrative index of pleural abnormalities. Am J Industr Med 20:145–161. Miller A, Lilis R, Godbold J, Wu X. 1996. Relation of spirometric function to radiographic interstitial fibrosis in two large workforces exposed to asbestos: An evaluation of the ILO profusion score. OccupEnviron Med 53:808–812. Miller A, Warshaw R, Nezamis J. Diffusing capacity and forced vital capacity in 5,003 asbestos-exposed workers: relationships to interstitial fibrosis (ILO profusion score) and pleural thickening. Am J Ind Med. 2013 Dec;56(12):1383-93.
[104] Black B, Szeinuk J, et al. Rapid progression of pleural disease due to exposure to Libby amphibole: "Not your grandfather's asbestos related disease." Am J Ind Med, 57 (11) (2014), pp. 1197-1206
[105] Oliver Comments at 3.

that included smoking (p_{trend} = < 0.0001.[14] Markowitz et al observed a significant increase lung cancer risk among both smokers and non-smokers in their study of 2,377 North American insulation workers.[15] Risk was significantly higher in those with asbestosis (ILO 1980 profusion $\geq$ 1), and higher in those who smoked. Among non-smokers with asbestosis, the authors observed rate ratio (RR) 7.40, 95% CI 4.0-13.7; and among smokers with asbestosis, RR 36.8, 95% CI 30.1-45.

EPA acknowledges that the IUR "does not include any risks that may be associated with non-cancer health effects" and that "[p]leural and pulmonary effects from asbestos exposure (e.g., asbestosis and pleural thickening) are well documented."[106] EPA also recognizes that the risk associated with these diseases is likely additive to the cancer risk, at least in occupational settings:

> [I]n occupational settings, with workers and ONUs exposed in a workplace, EPA considered risks of cancer per 10,000 people. At this risk level, if the non-cancer effects of chrysotile are similar to Libby amphibole asbestos, the non-cancer effects of chrysotile are likely to contribute additional risk to the overall health risk of asbestos beyond the risk of cancer. Thus, the overall health risks of asbestos based on cancer alone are underestimated.

EPA notes that its 2014 IRIS assessment of Libby amphibole asbestos[107] derived a Reference Concentration (RfC) for non-cancer health effects but there "is no [RfC] for these non-cancer health effects specifically for chrysotile."[108] Dr. Oliver takes issue with this reasoning:[109]

> An RfC has been calculated for nonmalignant disease occurring in association with exposure to Libby amphiboles. Alleged differences in potency between chrysotile asbestos and the amphiboles apply to malignant mesothelioma only and have not been shown for other cancers and nonmalignant disease, i.e. lung cancer, laryngeal cancer, ovarian cancer, and nonmalignant respiratory disease, including asbestosis. If an RfC was calculated for Libby amphiboles for nonmalignant disease, why can the same not be done for chrysotile asbestos? [§4.3.5, lines 1038-1041].

Indeed, EPA itself points out that "[s]everal of the [condition of use]-related exposures evaluated for human health risks in [its risk evaluation] are at or greater than the POD for non-cancer effects associated with exposure to Libby amphibole asbestos."[110] *This underscores the importance of developing a RfC for chrysotile and other fiber types and using it in the risk evaluation to determine the likelihood of non-cancer health effects (additive to the projected*

[106] Risk evaluation at 198.
[107] EPA, *Toxicological Review of Libby Amphibole Asbestos* In Support of Summary Information on the Integrated Risk Information System (IRIS), December 2014
[108] Risk evaluation at 198.
[109] Oliver Comments at 2.
[110] Risk Evaluation at 198.

malignancies) at projected exposure levels for workers and consumers. Such an RfC should be included in the final evaluation.

VI. Departing from TSCA and SAAC Recommendations, EPA Has Improperly Excluded All Environmental Pathways of Exposure to Asbestos, Further Underestimating Risk

The draft evaluation recognizes that "general population exposures to chrysotile asbestos may occur from industrial and/or commercial uses; industrial releases to air, water or land; and other conditions of use."[111] However, EPA has excluded these exposure pathways from the evaluation on the ground that they "are covered under the jurisdiction of other environmental statutes, administered by EPA, which adequately assess and effectively manage those exposures, i.e., CAA, SDWA, CWA, and RCRA." As a result, EPA "did not evaluate hazards or exposures to the general population in this risk evaluation, and there is no risk determination for the general population." As EPA acknowledged:[112]

> EPA did not evaluate the following: emission pathways to ambient air from commercial and industrial stationary sources or associated inhalation exposure of the general population or terrestrial species; the drinking water exposure pathway for asbestos; the human health exposure pathway for asbestos in ambient water; emissions to ambient air from municipal and industrial waste incineration and energy recovery units; on-site releases to land that go to underground injection; or on-site releases to land that go to asbestos National Emission Standards for Hazardous Air Pollutants (NESHAP) (40 CFR part 61, subpart M) compliant landfills or exposures of the general population (including susceptible populations) or terrestrial species from such releases.

EPA has followed the same approach in all other risk evaluations under amended TSCA.

As discussed below, excluding all environmental exposure pathways from the asbestos risk evaluation will defeat the central TSCA goal of providing a comprehensive picture of its risks to humans and the environment. This exclusion also ignores repeated SAAC concerns that the protectiveness of EPA risk evaluations will be compromised if they fail to address environmental pathways of exposure. In the case of asbestos, there is ample evidence that these pathways are significant.

A. TSCA Requires Risk Evaluations to Address All Pathways of Exposure

Risk evaluations under section 6(b)(4)(A) must determine "whether a chemical substance presents an unreasonable risk of injury to health or the environment." This requirement cannot be met without examining all sources of exposure that contribute to health and environmental risk. Similarly, section 6(b)(4)(A) provides that a risk evaluation must determine the substance's

[111] Id. at 25.
[112] Id. at 216.

risks under "the conditions of use." This broad term spans the entire life cycle of a chemical and is defined under section 3(4) to mean "the circumstances . . . under which a chemical substance is intended, known or reasonably foreseen to be manufactured, processed, distributed in commerce, used or disposed of." These "circumstances" clearly include environmental releases that result in pathways of human exposure, whether or not they might be controlled under other environmental laws.

If Congress had intended a blanket exemption for environmental releases from risk evaluations under section 6(b), it surely would have said so explicitly. But not only is there no such exemption in the law, but its legislative history and structure demonstrate that Congress intended TSCA to provide a comprehensive framework for identifying and managing chemical risks, including those that derive from environmental exposure pathways that are subject to other environmental laws.

When it enacted TSCA in 1976, Congress recognized that then-existing environmental laws were "clearly inadequate" to address the "serious risks of harm" to public health from toxic chemicals. H.R. Rep. No. 94-1341, at 7 (1976); see S. Rep. No. 94-698, at 3 ("[W]e have become literally surrounded by a manmade chemical environment. … [T]oo frequently, we have discovered that certain of these chemicals present lethal health and environmental dangers."). While other federal environmental laws focused on specific media, such as air or water, none gave EPA authority to "look comprehensively" at the hazards of a chemical "in total." S. Rep. No. 94-698, at 2. Congress designed TSCA to fill these "regulatory gaps," S. Rep. No. 94-698, at 1, through a comprehensive approach to chemical risk management that considered "the full extent of human or environmental exposure," H.R. Rep. No. 94-1341, at 6.

B. SACC has faulted EPA risk evaluations for excluding environmental pathways of exposure

The SACC has repeatedly raised concerns about EPA's failure to consider environmental pathways of human exposure. Thus, in its review of the 1,4-dioxane draft risk evaluation, the SACC said:[113]

> 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.

The SACC added that:[114]

[113] 1,4-Dioxane and HBCD SACC Report, at 18.
[114] Id.

> 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."[115] 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."[116]

The SACC review of the 1-BP draft risk evaluation similarly took EPA to task for failing to consider air emissions and other environmental releases: [117]

> The lack of consideration for general population exposures excludes a vast extent of the US population (workers, consumers, school children, and other populations) who are exposed to 1-BP, perhaps on a daily basis. The lack of consideration of the general population exposure is concerning given the strong evidence of widespread exposure to a chemical that may be 1-BP based (from biomonitoring data).

The SACC report for the methylene chloride evaluation raised similar concerns:[118]

> "Several Committee members expressed concern that large quantities of methylene chloride are volatilized to ambient air from diverse and disperse uses and that there is no COU that provides a basis for setting any limit on these emissions. While EPA asserts that the Clean Air Act (CAA) can be used to control these emissions, Committee members thought the CAA would address only a fraction of total emissions, i.e. only from Major Sources as defined by the 1990 CAA Amendments."

The Report added that:[119]

> Concern was expressed that many of the methylene chloride releases to the environment are unaccounted for, and the Committee recommended EPA consider using a mass-balance approach to match amount manufactured/imported with amounts

[115] Id.
[116] Id.
[117] SACC 1-BP Report at 17.
[118] SACC Methylene Chloride Report at 75.
[119] Id at 15.

used in products, recycled or disposed, and released to the environment. . . . Discharges to air, ground water, soils and sediments are not considered.

The SACC expressed concern that "readers of this Evaluation receive a partial picture of risks, finding for example, that recycling and proper disposal present the only environmental hazards under TSCA" and that "this incomplete picture of risks may be used to promote improper releases and disposal of methylene chloride."[120]

Despite the strength of SACC's concerns, EPA has refused to reconsider its exclusion of environmental pathways of exposure from its risk evaluations. SACC needs to reiterate its concerns about the exclusion of these pathways in connection with the asbestos risk evaluation.

C. Releases of Asbestos to Air, Drinking Water and Soil Add to Exposure and Risk

Air Emissions. According to ATSDR's 2001 Toxicological Profile for asbestos,[121] "[i]n urban areas, most ambient air concentrations range from 0.1 to 10 ng/m3 (3x10-6–3x10-4 PCM f/mL), but may range up to 100 ng/m3 (3x10-3 PCM f/mL) as a result of local sources. . . . Near industrial operations involving asbestos, levels may be as high as 50–5,000 ng/m3 (10.0015–15 PCM f/mL) (IARC 1977)." Dr. Arthur Frank reports that asbestos air concentrations "in excess of 0.000I f/cc (urban) are not true ambient background exposures and probably represent environmental exposures from a point source" and "can cause mesothelioma in humans**."[122]**

In certain parts of the country with high asbestos levels in rock formations, air emissions may result from rock mining. A 2005 study in California found that "residential proximity to [naturally occurring asbestos] shows an independent and dose–response association with mesothelioma risk. The findings are biologically plausible in view of the known strong association of occupational asbestos exposure and mesothelioma, and the observation of an association of NOA and mesothelioma in other areas of the world."[123]

Another air emission source is asbestos releases during building repair, modeling and demolition. These releases will result in the greatest exposure to construction workers, but nearby residents, pedestrians and bystanders may be exposed as well. ATSDR also notes that "[a]sbestos fibers may be released to indoor air due to the possible disturbance of asbestos-containing building materials such as insulation, fireproofing material, dry wall, and ceiling and floor tile (EPA 1991b; HEI 1991; Spengler et al. 1989) . . . In a survey performed by EPA (1988c),

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[120] Id.

[121] *Toxicological profile for asbestos* (update). (CIS/03/00067). Atlanta, Georgia: U.S. Department Of Health And Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, 2001, http://www.atsdr.cdc.gov/toxprofiles/index.asp. (Toxprofile), at 158-159.

[122] In his comments on the draft evaluation, Dr. Arthur Frank, co-chair of ADAO's SAB, has submitted a lengthy affidavit reviewing the literature on asbestos (Frank Affidavit). The quote in the text can be found at p. 110.

[123] Pan et al., *Residential proximity to naturally occurring asbestos and mesothelioma risk in California.* Am. J. Respir. Crit. Care Med. 172:IOI 9-1025 (2005).

levels of asbestos in 94 public buildings that contained asbestos ranged from not detected (ND) to 0.2 TEM f/mL (ND–3x10-3 PCM f/mL), with an arithmetic mean concentration of 0.006 TEM f/mL (10-4 PCM f/mL) (Spengler et al. 1989)."[124] Similarly, ATSDR indicates that, "[i]n studies from a Health Effects Institute-Asbestos Research Study, mean concentrations of fibers >5 µm ranged from 0 to 2.5x10-4 f/mL in public and commercial buildings and from 1.0x10-5 to 1.11x10-3 f/mL in schools and universities (Lee et al. 1992)."[125]

Indoor air exposure will be greatest where asbestos building components are damaged and poorly managed. The exposed population in public buildings includes workers, visitors, teachers and students. Exposure may be particularly high in asbestos-containing schools in which students and teachers are present for extended periods (30-40 hours per week). These pathways of exposure illustrate how the exclusion legacy asbestos from the draft evaluation results in an underestimation of risk.

The EPA evaluation also does not assess air emissions from facilities which use or process asbestos and asbestos-containing products for the ongoing conditions of use that are the focus of the evaluation. As described by EPA, use and disposal of asbestos-containing brake blocks in the oil industry and commercial and consumer use of aftermarket automotive asbestos-containing brakes/linings has the potential for off-site air emissions that could impact nearby residents and bystanders. However, there is no indication that EPA attempted to obtain emissions data from facilities engaged in these activities. While asbestos air emissions reported for the TRI Inventory are relatively low, TRI requirements apply only to large facilities and do not capture environmental releases from the smaller operations accounting for several of the conditions of use addressed in the evaluation.

Drinking Water. According to the American National Standards Institute (ANSI), asbestos enters our water supplies from "the deterioration of asbestos-cement pipes, which make up between 12-15 percent of drinking water systems in the United States and can be found all over Europe, Japan, and Australia. Over time, damage to these pipes erodes the cement, allowing asbestos fibers to seep into the water. Many of these municipal water distribution systems were built in the early-to-mid 1900s, with an average recommended lifetime of 70 years. Since these pipelines are used long past their peaks and subject to harsh water and soil conditions, they are more prone to breakage, adding to the level of contamination."[126]

Another source of asbestos in drinking water is leaching of natural occurring asbestos from soil and rock erosion and "loose fibers spreading into the environment from nearby construction sites or landfills. Disposing of older asbestos products in the environment can create toxic runoff that eventually flows into watersheds." [127]

[124] ToxProfile at 161-2.
[125] Id. at 163.
[126] ANSI Blog: Keeping Asbestos Out of Drinking Water https://blog.ansi.org/?p=158120
[127] Id.

In 1982, EPA set a maximum contaminant level (MCL) for asbestos in drinking water of 7 million fibers per liter (MFL).[128] According to the Environmental Working Group (EWG), monitoring required by EPA has detected asbestos in the drinking water of 34 water suppliers in 12 states serving a combined population of 241,000 people.[129] Exceedances of the MCL have been detected in some of these drinking water systems. ATSDR reports that asbestos "concentrations in most areas are <1 MFL (EPA 1979b), but values of 1–100 MFL and occasionally higher have been detected in areas contaminated by erosion from natural asbestos deposits (EPA 1976; Kanarek et al. 1980) or from mining operations (Sigurdson et al. 1981) . . . The amount of asbestos contributed from asbestos cement pipe is negligible in some locations (Hallenbeck et al. 1978) but may result in concentrations of 1–300 MFL at other locations (Craun et al. 1977; Howe et al. 1989; Kanarek et al. 1981)."[130]

There is evidence that ingestion of drinking water containing asbestos is a cause of gastrointestinal malignancies. According to ATSDR, a "number of epidemiological studies have been conducted to determine if human cancer incidence is higher than expected in geographical areas where asbestos levels in drinking water are elevated (usually in the range of 1–300 MFL) . . . Most of these studies have detected increases, some of which were statistically significant, in cancer death or incidence rates at one or more tissue sites (mostly gastrointestinal) in populations exposed to elevated levels of asbestos in their drinking water."[131]

Unlike IRIS, EPA chose to only evaluate risks from inhalation of asbestos and ignore ingestion. This is obviously a serious omission given the ample documentation of the presence of asbestos in drinking water and evidence that exposure by this source is linked to gastrointestinal cancers. EPA should expand its risk evaluation to assess the ingestion route of exposure.

Contaminated Waste. Asbestos-contaminated waste is prevalent both at inactive waste sites and active landfills and industrial facilities. ATSDR reports that asbestos has been identified in at least 83 of the 1,585 hazardous waste sites that have been proposed for inclusion on the EPA Superfund National Priorities List (NPL).[132] At several of these sites, asbestos has been detected in air, groundwater or surface water, creating a potential exposure pathway for nearby communities.

Under the TRI program, facilities are required to report asbestos releases to the environment if they are in a covered industrial code and manufacture (including import) or process more than 25,000 pounds of friable asbestos, or if they otherwise use more than 10,000 pounds of friable

[128] https://www.epa.gov/ground-water-and-drinking-water/national-primary-drinking-water-regulations
[129] https://www.ewg.org/tapwater/contaminant.php?contamcode=1094
[130] ToxProfile at 164.
[131] Id at 65.
[132] Id at 149.

asbestos. The 2018 EPA Problem Formulation for the asbestos risk evaluation summarizes TRI reports for asbestos in 2015 as follows:[133]

> In 2015, 36 facilities reported a total of approximately 25 million pounds of friable asbestos waste managed. Of this total, zero pounds were recovered for energy, approximately 188,000 pounds were treated, and nearly 25 million pounds were disposed of or otherwise released into the environment.

Because of the limitations on the application of TRI reporting requirements, it is likely that the amount of asbestos-containing waste managed or disposed of was significantly higher.

Notably, friable asbestos wastes managed on or off-site nearly tripled from 2009 to 2015. As EPA commented in the Problem Formulation:[134]

> From TRI data available using TRI Explorer, Table 2-6 shows that there has been a relatively large increase in total on-site and off-site disposal or other releases of friable asbestos since 2009 [Citation omitted] From 2009 to 2015, total on-site and off-site disposal or other releases of friable asbestos have risen from 8.8 million pounds to nearly 25.6 million pounds, respectively. As previously noted, the vast majority of the total on-site and off-site disposal or other releases of friable asbestos are released to land . . . The industry accounting for the highest release quantities of friable asbestos is the hazardous waste treatment and disposal sector, followed by the petroleum and other chemical and electric sectors.

The movement of asbestos waste in commerce and large volumes of waste managed at landfills and manufacturing sites are obviously a significant source of exposure to workers and nearby communities but are not meaningfully addressed in the draft evaluation.[135]

Asbestos present in ambient air, drinking water and waste overlaps with other sources of exposure. Thus, consumers who are exposed to asbestos in brake linings may also exposed to asbestos in drinking water or at nearby waste sites as well as to legacy asbestos in their homes, workplaces, schools and other public buildings. The same is true of workers exposed to asbestos from the conditions of use addressed in the draft evaluation. They may be exposed to

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[133] EPA Office of Pollution Prevention and Toxics, *Problem Formulation of the Risk Evaluation for Asbestos* (*pp*roblem formulation*),* May 2018, https://www.epa.gov/sites/production/files/2018-06/documents/asbestos_problem_formulation_05-31-18.pdf, at 27.

[134] Id. at 28-29. Although TRI releases subsequently declined modestly, they remained high. For example, total TRI asbestos releases for 2017 were 20,556,023 pounds.

[135] While EPA finds no unreasonable risk to health or the environment for occupational populations for the disposal of asbestos sheet gaskets scraps during gasket stamping and the disposal of spent asbestos processed in chemical manufacturing plants, this finding is based on the assumed absence of exposure as represented by industry – an assumption that seems questionable given the large volume of asbestos-containing waste managed at chlor-alkali facilities. Draft risk evaluation at 218. Moreover, EPA did not address the risks of asbestos waste management at other industrial facilities and landfills, several of which reported substantial asbestos releases under TRI.

asbestos not only at their workplaces but by consuming contaminated drinking water and inhaling asbestos fibers from outdoor and indoor air in their communities and homes.

Dr. Frank has observed that "[t]here are many case reports of mesothelioma in individuals with brief or low dose 'environmental' or home exposure."[136] EPA has previously noted that, because of the nature of asbestos and its interaction with the human body, each exposure increases the likelihood of developing an asbestos-related disease.[137] According to one expert, "there is a real burden of environmental asbestos exposure in industrialized countries that could account for approximately 20% of all mesotheliomas."[138] Thus, environmental exposure to asbestos is a significant contributor to risk and its omission from the EPA evaluation is a fundamental flaw.

VII. The Draft Risk Evaluation Ignores Consumer and Worker Exposure to Asbestos-Contaminated Talc

A major gap in the draft evaluation is its failure to address the risks of talc contaminated with asbestos. The presence of asbestos in talc is well-documented and there is no justification under TSCA to exclude it from the asbestos risk evaluation.

The dangers of asbestos-contaminated talc are described in the Statement of Dr. Jacqueline Moline, a member of ADAO's SAB and the Chairperson of the Department of Occupational Medicine, Epidemiology and Prevention at the Donald & Barbara Zucker School of Medicine at Hofstra University/Northwell Health. The key points in Dr. Moline's Statement are summarized below.

A. *Talc Deposits Are Often Co-Located with Asbestos Deposits*

Talc is a mineral mined from underground deposits. It is comprised of silicon, magnesium, and oxygen. Talc's most useful characteristic is its ability to absorb moisture, including both water-based and oil-based human moisture and perspiration. Crushing raw talc transforms it to a powder. Talc is mined domestically and imported from several countries. Talc deposits have been identified in mineral formations that include, or are located near, asbestos deposits. Because of this co-location, asbestos-containing talc has been identified and documented by geologists for years. These asbestos containing deposits can contain tremolite or anthophyllite, both forms of amphibole asbestos as well as chrysotile. According to the International Agency for Research on Cancer (IARC), "...as late as 1973, some talc products sold in the USA contained detectable levels of chrysotile asbestos, tremolite or anthophyllite (Rohl et al, 1976) and it is

[136] Frank Affidavit at 117.
[137] Environmental Protection Agency. A Guide for Ship Scrappers: Tips for Regulatory Compliance. Environmental Protection Agency; Report No.: 315-B-.00-001 (2000).
[138] Goldberg et al., Possible effect of environmental exposure to asbestos on geographical variation in mesothelioma rates. Occup. Environ. Med. 67:417-421 (2010).

possible that they remained on the market in some places in the world for some time after that (Jehan, 1984)."

B. Talc Has Been Linked to Asbestos-Related Diseases

Health officials long ago noted that New York talc miners were dying from lung scarring, including asbestos bodies in the scarred lung tissues and pathology "similar to [findings] reported in asbestosis."[139] New York state labor protection officials noted that other writers had attributed talc lung scarring to the fibrous varieties of talc, and observed that, for New York talc miners, "In general, the clinical, [chest X-ray], and pathological findings were similar to those observed in asbestosis."[140] It was also found that these talc miners had an excessive death rate from cancers of the lung and pleura.[141]

Starting in 2002, there have been published reports of cases of mesothelioma, considered a signal tumor for asbestos exposure, among New York talc miners. An epidemiology report sponsored by R. T. Vanderbilt Company found 2 cases among the 782 white men who had been employed for at least one day at the New York talc mines between 1948-1989.[142] Meanwhile, independent pathologists reported finding at least 8 confirmed cases of mesothelioma among New York state talc miners and millers as of 1986, and subsequently added 5 additional cases.[143] Commercial amphibole (amosite and crocidolite) asbestos fibers were virtually absent in the lung tissues of all 10 cases subjected to pathological examination, indicating that other occupational asbestos exposures (e.g., in construction) were not responsible for these mesotheliomas of these workers.

A recent effort by Finkelstein to update Honda et al. (2002) provides further evidence that asbestos-containing talc causes mesothelioma.[144] Finkelstein's update of Honda, using assumptions that would lead to an underestimate of the risk of mesothelioma (underestimating number of mesotheliomas from the cohort and overestimating number of person years at risk ("PYR")), "found [t]here were at least five new cases of mesothelioma in the cohort and mesothelioma incidence rates were at least five (1.6-11.7) times the rate in the general population." Based on this finding, Finkelstein concluded that "it is prudent, on the

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[139] FW Porro et al., Pneumoconiosis in the Talc Industry. *Am. J. Roent. Radium Therapy* 47: 507-524, 1942. Quote from FW Porro et al., Pathology of Talc Pneumoconiosis with Report of an Autopsy. *North. N. Y. Med. J.* 3: 23-25, 1946.

[140] M Kleinfeld et al., Talc Pneumoconiosis. *Arch. Ind. Health* 12: 66-72, 1955; M Kleinfeld et al., Talc Pneumoconiosis/A Report of Six Patients with Postmortem Findings. *Arch. Env. Health* 7: 101-115, 1963

[141] M Kleinfeld et al, Mortality among Talc Miners and Millers in New York State. *Indust. Hyg. Review* 9: 3-12, 1967.

[142] Y Honda et al., Mortality among Workers at a Talc Mining and Milling Facility. *Ann. Occup. Hyg.* 46: 575-585, 2002).

[143] MJ Hull et al., Mesothelioma among Workers in Asbestiform Fiber-bearing Talc Mines in New York State. *Ibid.* Suppl. 1, 132-136, 2002

[144] Finkelstein, Malignant Mesothelioma Incidence Among Talc Miners and Millers in New York State, Am. J. Ind. Med. 55(10):863-8 (Oct. 2012).

balance of probabilities, to conclude that dusts from New York State talc ores are capable of causing mesothelioma in exposed individuals."

There is substantial evidence that talcs from other areas also contain substantial amounts of asbestos (or asbestiform fibers) that can cause mesothelioma. For example, talc from Death Valley, California often contains amphibole asbestos that can cause mesothelioma. Van Gosen identified amphibole asbestos in numerous talcs from the Death Valley mines. Recently, Compton examined white talc ore from the Grantham Mine (source of ore for Sierra Talc and later owned by Johns-Manville Corp.). Using polarized light microscopy (PLM), Compton found "[t]he mineral sample was found to contain 5-15% (by volume) tremolite/actinolite as determined by PLM. The sample contains asbestiform fibers consistent with fibrous tremolite (see Figures 2 and 3) and fibrous talc'" (italics in original).[145] Tests of other sources of talc have yielded similar results.

Recently, studies by the Food and Drug Administration (FDA) and others have documented the presence of asbestos in a variety of personal care products and cosmetics.[146] These include baby powder and a wide range of talc-based makeup products sold by Claire's and Justice. There has been considerable public concern about these findings and one major manufacturer, Johnson & Johnson, recently withdrew talc-based baby powder from the US market.[147]

A recent article by Dr. Moline and her colleagues reported on 33 individuals with mesothelioma with no other identifiable source of exposure apart from their consistent use of cosmetic talc.[148] This case series included six individuals for whom tissue digestion analysis was performed, confirming the presence of asbestos and talc fibers in their tissue. In 2020, Emory et al. published a larger case series of 75 additional patients with cosmetic talcum powder exposure and mesothelioma.[149] There are now over 110 cases of mesothelioma reported in the peer-reviewed medical literature identifying mesothelioma among users of cosmetic talc. In addition, recent studies that have looked at the relationship between perineal talc exposure

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[145] Compton, Report of Results: MVAJ 1054 Analysis of Grantham Mine Talc for asbestos, Prepared for: Maune Raichle Hartley French & Mudd, LLC, 70 Washington St., Suite 425 Oakland, CA 94607 (July 8, 2015).

[146] October 11, 2019 - AMA Analytical Services, Inc. Summary of Asbestos and Talc Analysis - Johnson & Johnson - Baby Powder Lot #22318RB, From: www.fda.gov/media/131989/download
October 11, 2019 - AMA Analytical Services, Inc. (Supporting Data) INV-106924_ LabReview-2.1: AMA Laboratory Report 308006 (56 pages); https://www.fda.gov/cosmetics/cosmetics-recalls-alerts/fda-advises-consumers-stop-using-certain-cosmetic-products

[147] https://www.nytimes.com/2020/05/19/business/johnson-baby-powder-sales-stopped.html

[148] Moline J, Bevilacqua K, Alexandri M, Gordon RE. Mesothelioma Associated with the Use of Cosmetic Talc. J Occup Environ Med. 2019 Oct 10. doi: 10.1097/JOM.0000000000001723. [Epub ahead of print] PubMed PMID: 31609780.

[149] Emory TS, Maddox JC, Kradin RL. Authors' response to "malignant mesothelioma following exposure to cosmetic talc: Association, not causation." Am J of Ind Med (2020)DOI:10.1002/ajim.23106;
Andrion, Alberto, et al. Malignant Peritoneal Mesothelioma in a 17-Year-Old Boy with Evidence of Previous Exposure to Chrysotile and Tremolite Asbestos, Human Pathology, Volume 25, No. 6 (June 1994).
Musti, et al., Exposure to Asbestos and Mesothelioma Risk of Onset of Primary Ovarian, Description of Two Cases, 2009.

and ovarian cancer have found elevated cancer risk, particularly for the most common type of ovarian cancer, serous carcinoma of the ovary.[150]

C. Talc Has Significant Consumer and Industrial Uses Subject to TSCA

Although talc-based baby powder and cosmetics are regulated by the FDA, there are several talc-based consumer products subject to TSCA. There is considerable data documenting the presence of amphiboles and other asbestos fibers in a number of these products:

- In 2000, the Seattle Post Intelligencer confirmed that asbestos had been found in crayons.[151]
- In 2007, the ADAO's product testing confirmed asbestos in five consumer products, including a child's toy.[152]
- In 2015, the Environmental Working Group's (EWG) product testing confirmed four brands of crayons contained asbestos, all of them manufactured in China: Amscan Crayons, Disney Mickey Mouse Clubhouse 10 Jumbo Crayons, Nickelodeon Teenage Mutant Ninja Turtle Crayons, and Saban's Power Rangers Super Megaforce 10 Jumbo Crayons.[153]
- In 2018, U.S. Public Interest Research Group tested six kinds of crayons from various brands. Green Playskool crayons were found to contain tremolite asbestos fibers.[154]

The presence of asbestos in these products is of particular concern because of their use by children.

ADAO has submitted this evidence of asbestos contamination in children's products to EPA and urged that it to take action under TSCA.[155] EPA has not responded.

Talc also has extensive industrial uses which are subject to TSCA. According to Geology.com,[156] these uses include:

- **Plastics** -- In 2011, about 26% of the talc consumed in the United States was used in the manufacturing of plastics. It is mainly used as a filler.

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[150] Kadry MT, Farhat N, Karyakina NA, Shilnikova N, Ramoju S, Gravel CA, Krishnan K, Mattison D, Wen SW, Krewski D. Critical Review of the Association between Perineal Use of Talc Powder and Risk of Ovarian Cancer. Reproductive toxicology (Elmsford, NY). 2019 Dec. 90:88-101. Berge W, Mundt K, Luu H, Boffetta P. Genital use of talc and risk of ovarian cancer: a meta-analysis. European Journal of Cancer Prevention. 2018 May 1;27(3):248-57.

[151] https://www.cpsc.gov/PageFiles/108033/crayons.pdf

[152] https:/www.asbestosdiseaseawareness.org/archives/364

[153] https://www.ewg.org/release/alert-tests-find-high-levels-asbestos-children-s-makeup-kit

[154] https://uspirg.org/blogs/blog/usp/back-school-asbestos-crayons

[155] E.g. Asbestos Disease Awareness Organization's Docket Submission In Response to EPA's Problem Formulation Document for Asbestos Released on June 1, 2018, August 13, 2018.

[156] https://geology.com/minerals/talc.shtml

- **Ceramics –** In the United States in 2011, about 17% of the talc consumed was used in the manufacturing of ceramics products such as bathroom fixtures, ceramic tile, pottery, and dinnerware.
- **Paint --** Most paints are suspensions of mineral particles in a liquid. The liquid portion of the paint facilitates application, but after the liquid evaporates, the mineral particles remain on the wall. Talc is used as an extender and filler in paints.
- **Paper** -- Most papers are made from a pulp of organic fibers. This pulp is made from wood, rags, and other organic materials. Finely ground mineral matter is added to the pulp to serve as a filler. Talc as a mineral filler can improve the opacity, brightness, and whiteness of the paper. Talc also can also improve the paper's ability to absorb ink. In 2011, the paper industry consumed about 16% of the talc used in the United States.
- **Roofing Materials** -- Talc is added to the asphaltic materials used to make roofing materials to improve their weather resistance. It is also dusted onto the surface of roll roofing and shingles to prevent sticking. In 2011, about 6% of the talc consumed in the United States was used to manufacture roofing materials.
- **Other Uses --** Ground talc is used as a lubricant in applications where high temperatures are involved. It has also been used in the rubber industry to prevent rubber products from sticking. Talc powder is used as a carrier for insecticides and fungicides. It can easily be blown through a nozzle and readily sticks to the leaves and stems of plants. Its softness reduces wear on application equipment.

These industrial uses likely expose thousands of workers to talc powder by inhalation and dermal contact. The extent to which this talc contains asbestos is not known, but typically industrial-grade talc undergoes less extensive processing than talc used in personal care products and is more likely to contain impurities. Given the link between talc-based baby powder and mesothelioma and ovarian cancer in women, industrial talc exposure may well be a cause of asbestos-related death and disease. This risk should be addressed by EPA in the asbestos evaluation.

D. TSCA Applies to Asbestos Contaminants in TSCA-Regulated Consumer and Industrial Applications of Talc

At the April 7 SAAC preparatory meeting, EPA was questioned about the omission of asbestos-contaminated talc from the draft evaluation. In response, EPA staff asserted that contaminants in consumer and industrial products are outside the scope of TSCA risk evaluations because their presence is "inadvertent." This is an incorrect interpretation of the law.

It has always been EPA's policy to treat contaminants found in substances or mixtures as manufactured for commercial purposes under TSCA, regardless of whether the contaminant is "intended" to be present. Thus, EPA's premanufacture notice (PMN) regulations under section 5 require manufacturers of "new chemicals" to notify EPA of "impurities" found in these substances[157] and EPA has used its authorities under section 5 of TSCA to restrict these

[157] 40 CFR § 720.45(b).

impurities where they may present unreasonable risks to health or the environment. The PMN regulations define "impurity" as a "chemical substance which is unintentionally present with another chemical substance."[158] They also state that the term "manufacture or import for commercial purposes" applies to: [159]

> substances that are produced coincidentally during the manufacture, processing, use, or disposal of another substance or mixture, including byproducts that are separated from that other substance or mixture and impurities that remain in that substance or mixture. Byproducts and impurities without separate commercial value are nonetheless produced for the purpose of obtaining a commercial advantage, since they are part of the manufacture of a chemical substance for commercial purposes.

Thus, asbestos that is mined "coincidentally" during the mining of talc and lacks "separate commercial value" is nonetheless "manufactured for commercial purposes" under TSCA and is subject to TSCA authorities.

These authorities include the risk evaluation requirements in section 6(b) of TSCA. Under section 6(b)(4), these evaluations must determine whether a substance "presents an unreasonable risk of injury to health or the environment . . . under its conditions of use." Section 3(4) of TSCA defines "conditions of use" as the "circumstances . . . under which a chemical substance is intended, known or reasonably foreseen to be manufactured, processed, distributed in commerce, used, or disposed of." Clearly, impurities that are not "intended" to be manufactured but are "known or reasonably foreseen" to be produced during the manufacture of another substance fall within this definition. Thus, its inadvertent presence in commercially mined talc is a "condition of use" of asbestos which must be addressed in EPA's asbestos risk evaluation.

VIII. As EPA Acknowledges, it Lacks Basic Information About Ongoing Asbestos Uses and Exposure and Its Risk Determinations are Unreliable and Incomplete as a Result

Despite petitions from ADAO and several states, EPA has refused to use its TSCA information collection authorities to obtain reports from industry on ongoing imports, uses and exposures for asbestos and asbestos-containing products. Instead, EPA has elied on voluntary submissions to assess exposure and risk scenarios for workers and consumers. As a result, EPA does not have reliable and complete information on the quantities of asbestos and asbestos-containing products being imported, the number of companies and sites using these imports, the nature of these use activities, and the number of workers and consumers who are exposed. Nor does EPA have sufficient monitoring data to make reliable estimates of exposure. These are fundamental

[158] 40 CFR § 720.3(m).
[159] 40 CFR § 720.3(r).

flaws in the draft evaluation which call into question the completeness and adequacy of its determinations of unreasonable risk and the conditions of use requiring evaluation under TSCA.

A. EPA Rejected Petitions Requesting that It Use Its TSCA Reporting Authorities to Obtain Use and Exposure Information on Asbestos

On September 25, 2018, ADAO and five other organizations petitioned EPA to promulgate reporting requirements for asbestos under the information collection authorities in section 8(a) of TSCA. The petition, based on section 21 of TSCA, was prompted by EPA's admission in its problem formulation that it lacked fundamental information necessary for an informed understanding of asbestos use and exposure. The petitioners asked EPA to require mandatory reporting under TSCA section 8(a) so that it could fully and reliably assess asbestos risks in its upcoming TSCA evaluation.

As the petition showed, the problem formulation lacked basic information about the quantities of asbestos contained in imported products, their import volumes, the sites where they are used and the number of exposed individuals. For example, the problem formulation identified one company that imported asbestos-containing brake blocks for oil field use, but acknowledged that "[i] is unclear how widespread the continued use of asbestos brake blocks is for use in oilfield equipment."[160] Similarly, the problem formulation identified a chemical manufacturer, Chemours, which uses imported sheet gaskets containing 80 percent asbestos but did not address how many other manufacturers use these gaskets, the aggregate amount of asbestos they contain, and the conditions of use that may result in release of and exposure to asbestos fibers.[161] The problem formulation also cited USGS experts who, based on import records, believe that "asbestos-containing products that continue to be imported include . . . asbestos brake linings (automotive brakes/linings, other vehicle friction products)" but acknowledged that "the import volume of products containing asbestos is not known."[162] EPA further recognized that consumer exposure could occur from "changing asbestos-containing brakes or brake linings or cutting" but conceded that "[c]onsumer exposures will be difficult to evaluate since the quantities of these products that still might be imported into the United States is not known."[163]

The petition emphasized that the reporting requirements under TSCA that applied to other substances did not cover asbestos because, as EPA advised a major asbestos importer (Occidental Chemical) on July 28, 2017, asbestos is exempt from reporting under the TSCA Chemical Reporting Rule (CDR) because it is a "naturally occurring substance." The petition asked EPA to close this reporting loophole by amending and expanding the CDR rule to assure that it captured basic information about the importation and use of asbestos and asbestos-containing products that EPA would need for its TSCA risk evaluation.

[160] Problem Formulation, at 25.
[161] Id.
[162] Id at 22.
[163] Id at 39.

On January 31, 2019, a similar petition to require asbestos reporting under section 8(a) of TSCA was filed by 18 Attorneys General (AGs) representing 17 states and the District of Columbia. The AG petition likewise cited the need for additional information to inform the asbestos risk evaluation.

EPA denied the ADAO petition on December 21, 2018, asserting that it had "conducted extensive research and outreach" and already obtained all the information about asbestos use and exposure it needed for an informed risk evaluation:[164]

> EPA does not believe that the requested amendments would result in the reporting of any information that is not already known to EPA. As noted in more detail in Unit IV, EPA conducted extensive research and outreach to develop its understanding of import information on asbestos-containing products in support of the ongoing asbestos risk evaluation. After more than a year of research and stakeholder outreach, EPA believes that the Agency is aware of all ongoing uses of asbestos and already has the information that EPA would receive if EPA were to amend the CDR requirements.

The Agency denied the state AG petition on similar grounds on April 30, 2019.[165]

It turns out, however, that the draft risk evaluation suffers from the exact same information gaps identified in the ADAO petition and that the draft in fact expressly acknowledges these gaps, just as the earlier problem formulation did.

B. EPA Recognizes that It Lacks Critical Information on Commercial and industrial Uses of Asbestos

Throughout the draft risk evaluation, EPA admits that it does not know the quantities of asbestos involved in these uses, the companies and number of facilities using the asbestos-containing products, the nature of the use operation and the number of workers and consumers exposed.

Thus, the Executive Summary of the draft evaluation notes that:[166]

> Only two workers were identified for stamping sheet gaskets, and two TiO2 manufacturing facilities were identified in the U.S. who use asbestos-containing gaskets. However, EPA is not certain if asbestos-containing sheet gaskets are used in other industries and to what extent. For the other COUs, no estimates of the number of potentially exposed workers were submitted to EPA by industry or its representatives.

[164] 84 Fed. Reg. 3396 (February 12, 2019). ADAO and its co-petitioners subsequently filed suit to challenge the petition denial in the United States District Court for the Northern District of California.
[165] 84 Fed. Reg. 20062 (May 8, 2019). The AGs have also challenged the petition denial and their case and the ADAO case have been consolidated.
[166] Risk Evaluation at 22-23.

Similarly, EPA qualifies its risk determinations by acknowledging that, outside the chlor-alkali industry,[167]

> while there may be some knowledge about the potential number of workers/consumers in a particular COU, there is a lack of information/details on the market share of asbestos-containing products available to both workers and consumers. This makes it difficult to assess level of both certainty and confidence estimating the potential number of impacted individuals using asbestos for the COUs (except for chlor-alkali) in this draft risk evaluation. For ONUs and bystanders, there is a similar lack of understanding of the potential number of potentially impacted individuals.

Overall, because only EPA received only a "handful" of voluntary submissions from industry, "there are many uncertainties with respect to the extent of use, the number of workers and consumers involved and the exposures that might occur from each activity."[168]

These information gaps extend to the number of facilities using asbestos-containing products and the processing conditions at these facilities. Thus, aside from a single company, "it is unknown if other U.S. companies import asbestos-containing sheet material to stamp gaskets"[169] and whether such companies "perform this same stamping activity."[170] Because EPA could not "identify other companies that cut/stamp asbestos-containing sheet gaskets in the United States, . . . it is not known how many sites cut or stamp imported asbestos-containing sheet gaskets."[171] As a result, while EPA had limited monitoring data from the single processor of asbestos-containing sheet material, it "is uncertain if these monitoring data are representative of the entire U.S. population of workers that are potentially exposed during asbestos-containing sheet gasket processing."[172]

Downstream use of sheet gaskets represents another area of large uncertainty. EPA noted that "[a]sbestos-containing gaskets are used primarily in industrial applications with extreme operating conditions, such as . . . in many chemical manufacturing and processing operations." However, despite efforts "to identify all industrial uses of asbestos-containing gaskets, . . . the primary use known to the Agency is among titanium dioxide manufacturing facilities."[173] Even for this sector, however, "no estimates of the number of potentially exposed workers were submitted to EPA by industry or its representatives" and while EPA received information from one manufacturer, [o]ther titanium dioxide manufacturing plants that operate under similar conditions in the United States are thought to use asbestos-containing gaskets to prevent chlorine leaks, but EPA does not have information to confirm this."[174]

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[167] Id at 203.
[168] Id at 193.
[169] Id at 71.
[170] Id at 74.
[171] Id. at 74-75.
[172] Id. at 77.
[173] Id at 78.
[174] Id at 79.

The potentially widespread use of asbestos in oil field brake blocks likewise could not be meaningfully assessed because of limited information. While EPA identified one company that "imports asbestos-containing brake blocks on behalf of some clients for use in the oilfield industry, [i]t is unclear if any other companies fabricate or import asbestos-containing brake blocks, or how widespread the continued use of asbestos brake blocks is in oilfield equipment."[175] Even for the one importer, it is "unknown how many customers receive brake blocks from the sole facility identified by EPA."[176] Thus, "EPA was not able to identify the volume of imported asbestos-containing brake blocks, the number of brake blocks used nationwide, nor the number of workers exposed as a result of installation, removal, and disposal activities."[177]

Finally, while EPA attempts to assess consumer exposures during "do it yourself" brake and gasket repair and replacement, "[t]he number of consumers impacted by these COUs is unknown because the number of products containing asbestos for these COUs is unknown."[178]

This information is basic for a meaningful exposure and risk evaluation and its absence creates large uncertainties in characterizing asbestos's conditions of use – uncertainties for which EPA compensated by resorting to guesswork and speculation. Had EPA granted the section 21 petitions of ADAO and the State AGs, it would have been able to use TSCA reporting authorities to require all importers and processors of raw asbestos and asbestos-containing products to submit reports on their activities. These reports would have assured that EPA was in possession of complete and accurate information about the quantities of asbestos entering the US, the number of importers and secondary users, the sites where asbestos-containing products were in use and the number of exposed employees.

EPA's failure to require TSCA reporting on asbestos to inform its ongoing risk evaluation is a marked departure from previous EPA initiatives to address the risks of asbestos. To support its comprehensive rulemaking to ban most uses of asbestos in the 1989, EPA used its TSCA section 8(a) reporting authority in 1982 to collect information on industrial and commercial uses of asbestos.[179] Congress then enacted, and President Reagan signed, the Asbestos Information Act of 1988 imposing a one-time requirement for current and former manufacturers and processors to report asbestos-containing products to EPA.[180] EPA collected extensive information under the law, which it released to the public on February 13, 1990.[181] EPA's failure

[175] Id. at 83.
[176] Id at 84.
[177] Id at 86.
[178] Id. at 107.
[179] 47 Federal Register 33207 (July 30, 1982) (40 CFR 763.60).
[180] Pub. L. 100-577. To implement the law, EPA published a notice on April 18, 1989 (54 FR 15622) establishing a process and schedule for reporting information required by the Act. In a subsequent notice, the Agency informed submitters that it would not accept CBI claims. 54 Fed. Reg. 38736 (Sept. 20. 1989).
[181] 55 Fed. Reg. 5144.

to now utilize these mandatory information collection tools has severely handicapped the current risk evaluation.

C. The Limited Workplace Monitoring Data in the Draft Evaluation Reflects EPA's Inadequate and Ineffective Information Collection Efforts under TSCA

Sparseness of Monitoring Data for Most Conditions of Use. The draft risk evaluation lacks meaningful exposure monitoring data for nearly all the conditions of use it addresses. The sparseness of the monitoring on which EPA draws is surprising and troubling in light of extensive OSHA regulation of workplace exposure to asbestos and longstanding concern about the risks of industrial use of asbestos. EPA itself acknowledges that, "[w]here there are few data points available, it is unlikely the results will be representative of worker exposure across the industry."[182]

For example, EPA emphasizes that "[a]n important consideration for worker exposure is the extent to which sheet gasket stamping releases asbestos-containing fibers, dusts and particles" but that it could not confirm these releases because it only visited a single site and had very limited monitoring data.[183] As it acknowledged, "the exposure data [it relied on] are based on observations from a single reference that presents worker exposure monitoring data for a single company, and documentation for this study is incomplete."[184] Similarly, "EPA did not identify any studies that contain exposure data related to asbestos-containing brake blocks" used in the oil industry. While it "is reasonable to assume that wear of the brake blocks over time will release some asbestos fibers to the workplace air," the "magnitude of these releases and resulting worker exposure levels is not known."[185]

EPA also reports that a US auto manufacturer uses asbestos-containing brakes to produce vehicles for export but it "did not identify any studies that contain exposure data related to installation of asbestos-containing brakes" in these vehicles.[186] Along the same lines, the Agency "identified the use of asbestos-containing gaskets in the exhaust system of a specific type of utility vehicle available for purchase in the United States. . . . It is known that these UTVs are manufactured in the United States, so EPA expects that there is potential for exposures to workers who install the gaskets during assembly and workers who may repair these vehicles."[187] Nonetheless, despite estimating a large number of potentially exposed workers, "[n]o information from OSHA, NIOSH, or the scientific literature was available on occupational exposures to asbestos associated with installing and servicing gaskets in UTVs."[188]

[182] Risk Evaluation at 195.
[183] Id. at 73.
[184] Id. at 81.
[185] Id at 85-86.
[186] Id. at 96.
[187] Id. at 100.
[188] Id at 103.

Finally, despite estimating an extremely large population of DIY consumer users of asbestos brake lining and gaskets, EPA acknowledges having "very little information specific to consumer exposures,"[189] requiring it to extrapolate from occupational data of uncertain relevance and use "best professional judgment."

Monitoring at Chlor-alkali Plants. The one condition of use for which extensive workplace monitoring data are available is the production and use of asbestos diaphragms for chlor-alkali production. However, EPA obtained these data from voluntary submissions by individual producers and their trade association, ACC.[190] It is uncertain whether the companies provided all the monitoring data in their possession or selected certain data for submission to EPA. In addition, data was not provided for 5 of the 15 plants in the industry.[191] For this reason and because the information EPA received covered varying time periods, it "may not be representative for the exposures in all companies." As EPA explained, "operations [across plants] are different, where some of them hydroblast and reuse their chrysotile asbestos-containing diaphragms and others replace them," with different exposures during these two activities.[192]

EPA also cautioned that "it is uncertain if certain high-exposure activities are captured in this dataset, such as exposures when cleaning spilled asbestos within a container from damaged bags."[193] EPA had little ability to verify the completeness of the chlor-alkali data because it made site visits to only 2 of the 15 plants using asbestos diaphragm cells, these visits were announced in advance, and EPA was not accompanied by knowledgeable experts from OSHA and NIOSH.[194]

EPA could have investigated whether the chlor-alkali industry monitoring data addressed all high-exposure activities by using a checklist developed by the industry itself. The Chlorine Institute's Pamphlet 137 elaborates on the many parts of the process life cycle that give rise to asbestos exposures of workers and environmental contamination.[195] These exposures include:

- Losses from torn sacks in shipment, unloading, and storage of asbestos sacks
- Waste from vacuuming areas where torn sacks are discovered and patched
- Waste in water used to wash dust off sacks, with the danger of drying of contaminated surfaces and airborne release

[189] Id at 108.
[190] The ACC data is of no value because it does not identify the plants where measurements were taken, as EPA recognizes.
[191] Id at 66. As noted in the Comments of Barry Castleman ScD on the draft evaluation at 6, "[o]ne company (Axiall) provided only 18 measurements of daily average exposure, for only one year, at only one plant. Axiall operated at least 3 diaphragm cell plants (*North American Chlor-Alkali Industry Plants and Production Data Report for 2015,* Chlorine Institute, 2016, Table 1)."
[192] Risk Evaluation at 195.
[193] Id. at 70.
[194] Id. at 60.
[195] Chlorine Institute, *Guidelines: Asbestos Handling for the Chlor-Alkali Industry* (Oct. 2008) (Chlorine Industry Guidelines).

- Dust from contaminated protective clothing shaken or air-blown, and from transport and laundering
- Dust from bag opening, delumping, and emptying (even if done with minimal disturbance under a vented hood or in an enclosure)
- Dust from handling and storage of partially filled sacks and disposal of empty sacks
- Transfer of asbestos from sacks for weighing and dumping into a tank for mixing with water, even if done with functioning local exhaust ventilation or in an enclosure
- Spilling, splashing, and drying of asbestos slurry transferred to operating cells
- Storage, handling, and installation of completed diaphragms in cells
- Shipping, receiving, storage, and assembly of pre-deposited diaphragms
- Spills of all kinds even if recognized and clean-up efforts are immediately made
- Incomplete protection of respirators even if properly fitted, maintained, and stored
- Release from hydro-blasting to remove asbestos diaphragms and drying of material on surfaces and clothing afterwards.
- Waste disposal of over 300 mt/yr of asbestos, even if the waste has OSHA warning labels and the dangers are posted at waste sites.

EPA could have closely examined the industry monitoring data to confirm whether it captured these high-exposure activities, but apparently failed to do so.

EPA's reliance on monitoring data voluntarily submitted by industry greatly weakened its ability to assess worker and consumer exposure. As EPA admits, industry cooperation was extremely limited for most use conditions, some companies that worked with EPA did not submit any data, and even the data EPA received was not necessarily complete or representative of asbestos use conditions. The Agency's exposure assessment would have been substantially more robust and reliable if EPA had used its mandatory information collection authority under TSCA to assure that it received all the industry asbestos monitoring data available.[196] Multiple tools were available for this purpose. EPA could have added asbestos to its section 8(d) rule,[197] which requires submission of all health and safety studies, including monitoring studies. Alternatively, after using section 8(a) reporting to identify all importers and processors of asbestos and asbestos-containing products and all asbestos use sites, it could have used its subpoena authority under TSCA section 13 to require these entities to submit the monitoring data in their possession. And if significant data gaps still remained, EPA could use its section 4 testing authority to require monitoring by companies that lacked data on levels of worker exposure. Unfortunately, none of these steps were taken, greatly limiting EPA's understanding of asbestos exposure levels.

[196] OSHA rules require employers to retain all workplace monitoring data for 30 years. EPA could simply have required the submission of all asbestos monitoring data subject to this requirement. It could also have worked closely with OSHA to make sure it had access to the results of all monitoring conducted by OSHA. While the draft risk evaluation refers to NIOSH and OSHA-generated monitoring data, it does not explain the efforts EPA made to systematically obtain this information.

[197] 40 CFR Part 716.

D. *EPA Has Failed to Justify Excluding Asbestos Fabric and Cement from the Draft Evaluation*

EPA's June 2018 asbestos problem formulation identifies imports of asbestos cement products and woven products as current conditions of use that would be addressed in EPA's risk evaluation.[198] The problem formulation cites USGS experts to confirm imports of these products and USGS annual reports have consistently included them in its descriptions of asbestos import activity. [199] However, EPA does not include cement and woven products in the draft risk evaluation because it "has not found any evidence to suggest that woven products (other than those that are already covered under a distinct COU such as brake blocks used in draw works) or cement products imported into the United States contain asbestos."[200] EPA even maintains that it "is highly certain that manufacturing (including import), processing, or distribution of asbestos is not intended, known or reasonably foreseen beyond the 6 product categories identified herein."[201]

These are surprising claims given that International Trade Commission import summaries have consistently shown substantial incoming shipments of asbestos fabric and cement. According to Dr. Castleman, "'[r]ecorded US imports of asbestos yarn and thread from Mexico were 51 metric tons in 2018 and 85 m.t. in 2019" and totaled 518 metric tons in 2011-2015.[202] US imports of asbestos pipes have been valued at $115 million by the Observatory of Economic Complexity (OEC).[203] As the basis for ignoring this evidence, EPA says that it "contacted potential foreign exporters of asbestos woven products and asbestos cement products, and these foreign companies informed EPA that they do not have customers in the United States."[204] However, EPA only talked to two Mexican companies, not all listed foreign exporters of these product classes. The contacts were by phone and the companies (who were not subject to TSCA) did not provide written statements to EPA.[205] Dr. Castleman underscored the inadequacy of this evidence:[206]

> Asking a Mexican corporation that EPA has no jurisdiction over, about the wrong trade category of imported asbestos textile products, provides no useful information, let alone reassurance. Similarly, US imports of (asbestos) cement, recorded as coming from China, Taiwan, and Honduras in 2017-2018, could hardly be appraised by asking a Mexican manufacturer and somebody at the American Water Works Association (EPA Draft, p. 37). Ports of origin for cement (HTS code 6811.40.0000) imports in 2019 were

[198] Asbestos Problem Formulation at 25.
[199] https://prd-wret.s3-us-west-2.amazonaws.com/assets/palladium/production/s3fs-public/atoms/files/mcs-2019-asbes.pdf
[200] Risk Evaluation at 37.
[201] Id. at 193.
[202] Castleman Comments at 4-5.
[203] https://oec.world/en/profile/hs92/6811/
[204] Risk Evaluation at 37.
[205] While referenced in the risk evaluation, the records of these telephone conversations could not be directly accessed in EPA's HERO database.
[206] Castleman Comments at 5.

Canada, China, Germany, and Vietnam; these include products made in other countries and trans-shipped through the listed countries-of-origin. EPA failed to identify, let alone contact the US importers of record of these products (Appendix C, p. 251).

As these comments suggest, telephone calls with two foreign suppliers are far from definitive evidence of the absence of imports. Instead, using its authorities under TSCA section 8(a), EPA should have required reports from importers identifying all asbestos-containing products entering the US, the quantities of these imports and the American end-users.

EPA suggests that the inclusion of asbestos woven and cement products in its Significant New Use Rule (SNUR) (40 CFR §721.11095) is an adequate substitute for addressing them in the draft risk evaluation.[207] This is incorrect. The SNUR makes no finding of unreasonable risk for these or other asbestos-containing products nor is it a prohibition on their use; instead, it merely requires EPA to be notified before their reintroduction into US commerce. By contrast, the risk evaluation would determine unreasonable risk for all asbestos conditions of use and lay the foundation for a ban of products subject to that determination. Accordingly, we request that EPA treat asbestos woven and cement products as TSCA conditions of use and add them to the draft evaluation.

IX. EPA Concludes Without Any Basis that Importation, Distribution in Commerce and Disposal of Asbestos and Asbestos-containing Products do not Present an Unreasonable Risk of Injury

EPA finds no unreasonable risk to health or the environment for "import and distribution in commerce of asbestos for all the conditions of use" and "for the disposal of asbestos sheet gaskets scraps during gasket stamping and the disposal of spent asbestos gaskets used in chemical manufacturing plants."[208]

These sweeping conclusions – which conflict with the findings of draft risk evaluations for other, less dangerous chemicals – receive minimal discussion. The only justification EPA provides is that it "assumed the absence of exposure to asbestos" because "[r]aw asbestos and asbestos-containing products are imported into the U.S. in a manner where exposure to asbestos is not expected to occur."[209] According to EPA, this is because "raw asbestos is imported in bags wrapped in plastic where they are contained in securely locked shipping containers [that] remain locked until they reach the chlor-alkali plants" and because asbestos "articles (or asbestos-containing products) are assumed to be imported and distributed in commerce in a non-friable state, enclosed in sealed boxes."[210]

[207] Risk Evaluation at 216.
[208] Id at 218.
[209] Id at 217-218.
[210] Id at 218.

To the extent they have any support at all, EPA's assumption of no exposure during raw asbestos imports is based on self-serving statements by the chlor-alkali industry which EPA has made no effort to independently confirm.

While fluctuating from year to year, asbestos imports by the chlor-alkali industry have been substantial. In 2018, USGS found that imports of raw asbestos totaled 750 metric tons,[211] twice the amount originally estimated. Raw asbestos comes into the US at multiple ports of entry, including Houston, Texas, New Orleans, Louisiana, Norfolk, Virginia, Port Everglades, Florida, Savannah, Georgia, and Newark, New Jersey. Exposure to asbestos can occur at several stages: the unloading of ships, storage of asbestos in customs territory, loading of asbestos on trucks or trains, transport from ports of entry to chlor-alkali manufacturing sites, and unloading of asbestos containers at these sites. During any of these activities, accidents or improper handling could rupture shipping containers and bags, releasing raw asbestos powder in quantities that put workers or bystanders at risk.

The draft evaluation acknowledges that damaged shipping containers are known to arrive in the US and "[p]ort and warehouse workers manage and remediate any damaged containers."[212] Damaged containers are also received by plants: "After arriving at the plant, the shipping container with raw asbestos is inspected, and any damaged containers are shipped back to the sender." In addition, "workers' first task after opening the containers is to inspect bags for leaks. If bags are broken or loose asbestos is evident, the area is controlled to prevent accidental exposure, the bags are repaired, and the location is barricaded and treated as an area requiring cleanup."[213]

Similarly, the Chlorine Institute's Pamphlet 137 identifies several stages of the asbestos life cycle that give rise to exposure by workers and environmental contamination.[214] These exposure scenarios include losses from torn sacks in shipment, unloading, and storage of asbestos sacks and waste from vacuuming areas where torn sacks are discovered and patched.

EPA has even less information about the importation of asbestos-containing articles and products than it has about the importation of raw asbestos for use in chlor-alkali production. As discussed above, EPA does not know how many import shipments are received each year, the amounts of asbestos they contain, where they enter the United States, the types of shipping containers used, the number of US sites receiving shipments and whether shipment occurs by rail, truck or barge. To reach the blanket conclusion that no asbestos exposure occurs during these activities is pure speculation. In fact, given the opportunities for exposure to raw asbestos described above, the risks of accidental releases and spills would seem even greater during importation and distribution of asbestos-containing products.

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[211] https://prd-wret.s3-us-west-2.amazonaws.com/assets/palladium/production/s3fs-public/atoms/files/mcs-2019-asbes.pdf
[212] Draft Evaluation at 61.
[213] Id.
[214] Chlorine Industry Guidelines.

In short, damaged shipping containers and bags and spills, leaks and accidents during importation and distribution in commerce are "known" or "reasonably foreseen" occurrences during the life-cycle of imported raw asbestos and asbestos-containing products and thus fall within the definition of "conditions of use" in section 3(4) of TSCA. To assume that that these occurrences never happen and there is no unreasonable during asbestos importation and distribution in commerce is arbitrary and unjustified and ignores plausible exposure scenarios that are uniquely dangerous given the absence of any safe level of exposure to asbestos.[215]

X. EPA Should Not Base Risk Determinations on the Assumed Use of Respirators Since Engineering Controls Should be the Primary Form of Protection and Evidence of Actual Use of Respirators Is Minimal

As in past evaluations, EPA bases determinations of unreasonable risk on the assumed use of respirators for some asbestos conditions of use. The assumption of respirator use does not change EPA's conclusion that these conditions of use present unreasonable risks but it does reduce risk estimates significantly and could be used to justify exposure restrictions that are insufficiently protective. SACC has repeatedly questioned the appropriateness of EPA's reliance on respirators and other Personal Protective Equipment (PPE) and this caution is particularly relevant to asbestos, for which evidence of real-world respirator use is extremely limited.

A. The SACC Has Repeatedly Raised Serious Concerns About EPA's Undue Reliance on PPE to Determine the Absence of Unreasonable Risk

In each of its reviews of draft evaluations, the SACC has raised concerns about EPA's undue reliance on respirators and other PPE in its risk determinations. In its report on the PV29 draft, the SACC 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." 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."[216] 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."[217]

[215] EPA "also assumes the absence of asbestos exposure during the occupational disposal of asbestos sheet gaskets scraps during gasket stamping and the disposal of spent asbestos gaskets used in chemical manufacturing plants. This assumption is based on the work practices followed and discussed in section 2.3.1 that prevent the release of asbestos fibers." Draft Evaluation at 218. This makes no sense since EPA acknowledges that it only knows of one manufacturer in the chemical industry that uses sheet gaskets and has no basis to extrapolate the work practices of that company to the rest of the industry.

[216] These "heightened exposures" could occur as a result of "contamination of the interior of the glove" (if workers were not properly trained in glove use and replacement) or by "acting as a reservoir" for contaminants (if the gloves were not impermeable). Such occlusion (greater penetration of the skin where contaminants build up inside the glove because it is permeable) would result in *greater* dermal exposure than in the "no glove" scenario.

[217] Id. at 53.

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:[218]

> "[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 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."

The SACC report on 1-BP indicated that "[p]ersons familiar with PPE use realize that nominal protection factors may not be achieved in actual practice"[219] and concluded that EPA "[a]ssumptions about PPE use are likely unrealistic for many of the scenarios and so the determination of whether a condition of use results in an acceptable or unacceptable risk should be based on no PPE use, with the possible exception of in a manufacturing facility."[220]

The SACC report on the methylene chloride risk evaluation reinforced these points, stating that "[m]ost Committee members agreed that EPA's assumptions of PPE use likely do not reflect actual conditions in most workplaces."[221] The SACC added that:[222]

> "The Agency's reliance on appropriate use of personal protective equipment (PPE), including both respirators and gloves, is not supported by current research literature or industrial hygiene practice. The mere presence of a regulation requiring respirators does not mean that they are used or used effectively. Inadequacies in respirator programs are documented. Respirators require multiple respiratory protection (RP) compliance factors in order to perform as certified. Brent et al. (2005) used data from the NIOSH and Bureau of Labor Statistics (BLS) joint survey on Respirator Usage in Private Sector Firms, (BLS, 2001) to examine the adequacy of respirator protection programs in private industries. They found "large percentages of establishments requiring respirator use [under OSHA or the Mine Safety and Health Administration (MSHA) regulations] had indicators of potentially inadequate respirator programs." Later, Janssen et al. (2014) reported that 'APFs do not apply to RPD used in the absence of a fully compliant RP program; less than the expected level of protection is anticipated in these situations.' Moving beyond program elements, the frequency of proper use of gloves and respirators is largely unknown."

[218] Id at 118.
[219] SACC Report on 1-BP, at 30-31.
[220] Id at 66.
[221] SACC Report on methylene chloride, at 17.
[222] Id at 36.

B. Evidence of Consistent and Reliable Respirator Use by Asbestos-Exposed Workers Is Lacking

EPA recognizes that, under the well-established workplace hierarchy of controls, use of respirators to reduce exposure is a last resort, justified only when other more effective protections are infeasible: [223]

> OSHA requires employers utilize the hierarchy of controls to address hazardous exposures in the workplace. The hierarchy of controls prioritizes the most effective measures to address exposure; the first of which is to eliminate or substitute the harmful chemical (e.g., use a different process, substitute with a less hazardous material), thereby preventing or reducing exposure potential. Following elimination and substitution, the hierarchy prioritizes engineering controls to isolate employees from the hazard, followed by administrative controls, or changes in work practices to reduce exposure potential (e.g., source enclosure, local exhaust ventilation systems, temperature). Administrative controls are policies and procedures instituted and overseen by the employer to protect worker exposures. As the last means of control, the use of personal protective equipment (PPE) (e.g., respirators, gloves) is required when the other control measures cannot reduce workplace exposure to an acceptable level.

Under OSHA standards for asbestos and other substances, respirator use is allowed only "when the occupational exposure limit is exceeded after feasible engineering, work practice, and administrative controls have been put in place."[224]

OSHA Respirator requirements for asbestos are extremely specific:[225]

> OSHA's 29 CFR § 1910.1001(g)(2)(ii), however, is specific to asbestos and states that employers must - when the employee chooses to use a powered air-purifying respirator (PAPR), and it provides adequate protection to the employee - provide an employee with a tight-fitting PAPR instead of a negative pressure respirator selected according to § 1910.1001(g)(3). In addition, OSHA 1910.1001(g)(3) states that employers must not select or use filtering facepiece respirators for protection against asbestos fibers. Therefore, filtering facepieces (N95), quarter masks, helmets, hoods, and loose fitting facepieces should not be used. OSHA's 29 CFR § 1910.1001(g)(3)(ii) also indicates that high-efficiency particulate air (HEPA) filters for PAPR and non-powered air-purifying respirators should be provided.

Moreover, even for allowable respirators, "nominal APFs [Assigned Protection Factors] . . . may not be achieved for all PPE users" exposed to asbestos**.** EPA references research (Riala and Riipinen, 1998) assessing the performance of respirators and HEPA units in 21 different

[223] Risk Evaluation at 57-58
[224] Id., at 58.
[225] Id.

exposure abatement scenarios, based on "asbestos samples from inside the mask, use of the worker's own protection equipment, and measurement in different real work conditions." According to EPA, the "results demonstrate that while some workers have protection above nominal APF, some workers have protection below nominal APF, so even with every worker wearing respirator, some of these workers would not be protected."[226]

EPA assumed respirator use for three conditions of use – chlor-alkali production, sheet gasket stamping and sheet gasket use in the chemical industry – based on reports by industry. For the remaining conditions of use, EPA lacked any information on respirator usage although it claimed without documentation that "some respirator use among workers in [the aftermarket for brakes and clutches] industry is expected."[227] EPA also lacked information on the type of respirator used except for chlor-alkali production and sheet gasket use. Thus, for all but these conditions of use, it based risk determinations on "hypothetical" APFs of 10-25. Even where APFs were reported by industry, EPA cautioned that "there is some uncertainty [in relying on these APFs because the] . . . nominal APF may not be achieved for all respirator users."[228]

Moreover, as reported by industry, the extensiveness of respirator use was uneven and limited. For chlor-alkali production, ACC submissions indicated that "workers wear respiratory PPE during three tasks (Asbestos Unloading/Transport, Glovebox Weighing and Asbestos Handling, and Hydroblasting), but do not wear respiratory PPE during five of the tasks (Asbestos Slurry, Depositing, Cell Assembly, Cell Disassembly, and Filter Press)." These submissions also revealed that, where used, "respiratory PPE is not worn throughout an entire 8-hour shift."[229] Moreover, EPA noted that workplace asbestos levels were no lower and in some cases higher for tasks requiring PPE than for tasks lacking PPE. Finally, since "respiratory PPE is not worn for all worker tasks where occupational exposure monitoring data indicates the presence of airborne asbestos fibers, the potential [exists] for released asbestos fibers to settle and to again become airborne," putting workers at risk of additional exposure.[230]

It is noteworthy that while EPA made two announced visits to chlor-alkali plants, it did not verify first-hand the wearing of respirators during the work tasks for which ACC reported respirator use, nor did it make any effort to systematically determine whether respirator practices were consistent and equally protective across the 15 plants in the industry.

Outside of the chlor-alkali industry, reports of respirator use provided raised even more questions. For example, the one company processing sheet gaskets indicated that an N95 respirator was worn by a worker cutting gaskets. However, EPA noted that the OSHA asbestos standard did not allow use of such respirators.[231] In addition, neither this company nor the one identified gasket user in the chemical industry provided any details about their programs for

[226] Id. at 59-60.
[227] Id 161-162.
[228] Id. at 219.
[229] Id. at 220.
[230] Id.at 221.
[231] Id. at 222.

respiratory protection. The OSHA respiratory protection standard (29 CFR 1910.134) contains numerous elements, *e.g.,* for program administration; worksite-specific procedures; respirator selection; employee training; fit testing; medical evaluation; and respirator cleaning, maintenance, and repair. EPA has emphasized that the "use of a respirator would not necessarily resolve inhalation exposures since it cannot be assumed that employers implement comprehensive respiratory protection programs for their employees."[232]

For both of these sectors, as discussed above, EPA acknowledged that numerous other companies might process and use asbestos sheet gaskets, but it had no knowledge of how many such companies exist or who they are. Obviously, then, EPA lacks information about respirator use beyond the two companies it contacted and has no basis to assume that workers uniformly wear respirators across these conditions of use.

For all these reasons, EPA risk determinations for exposed workers should not be based on assumed respirator use but should estimate risks without factoring in reductions in exposure attributable to respirators.

XI. EPA's Risk Evaluation Fails to Account for Risks to Highly Exposed or Susceptible Subpopulations Entitled to Protection Under TSCA

Under TSCA, EPA risk evaluations must identify potentially exposed or susceptible subpopulations (PESS) and determine whether the specific risks to these subpopulations are unreasonable. EPA did not comply with this requirement.

Subpopulations with higher than average exposure to asbestos include individuals who are exposed by multiple routes and/or across multiple pathways and conditions of use. However, EPA declined to define and address these subpopulations:[233]

> Aggregate exposures for asbestos were not assessed by routes of exposure, since only inhalation exposure was evaluated in the RE. EPA chose not to employ simple additivity of exposure pathways at this time within a condition of use because of the uncertainties present in the current exposure estimation procedures. This lack of aggregation may lead to an underestimate of exposure but based on physical chemical properties the majority of the exposure pathway is believed to be from inhalation exposures. Pathways of exposure were not combined in this RE. Although it is possible that workers exposed to asbestos might also be exposed as consumers (e.g., by changing brakes at home), the number of workers/uses is potentially small. The individual risk estimates already indicate risk; aggregating the pathways would increase the risk.

Because of this approach, EPA underestimated exposure and risk in several ways. First, it is known that consumers ingest asbestos-contaminated drinking water. EPA admitted that failure

[232] Id. at 59.
[233] Id. at 207.

to aggregate exposures for drinking water and inhalation "may lead to an underestimate of exposure" but rationalized this failure on the ground that "the majority of the exposure pathway is believed to be from inhalation exposures." This may be true, but EPA is obligated to consider risks to PESS, and persons exposed by both routes clearly comprise a PESS.

Second, there are likely workers who are exposed to asbestos during their employment, from indoor and outdoor air or waste disposal, and from consumer activities such as DIY brake pad replacement. EPA acknowledges that these overlapping exposures may occur but dismisses them because the "number of workers/uses is small." Even if this were correct, it doesn't negate EPA's obligation under TSCA to determine the risks to a subpopulation with elevated exposure. EPA also says that "aggregating the pathways would increase the risk" but accounting for this increased risk is very reason why Congress directed EPA to address PESSs.

Finally, workers and consumers with asbestos exposure from the conditions of use addressed in the risk evaluation are also likely to be exposed to legacy asbestos. EPA recognizes "the potential for exposure to legacy asbestos for any populations or subpopulation, due to activities such as home or building renovations, as well as occupational or consumer exposures identified in this RE."[234] It explains, however, that, "[l]egacy asbestos exposure is not considered in the RE at this time which could underestimate exposures and thus, risks." Again, the prospect of understating exposures and risks is the very reason why EPA should not delay consideration of legacy exposure to a future risk evaluation. The risks of legacy asbestos and currently active conditions of use are intertwined and must be addressed in combination.

EPA also recognizes that there are subpopulations with greater susceptibility to asbestos. Consistent with several studies, it notes that a "source of variability in susceptibility between people is smoking history or the degree of exposure to other risk factors with which asbestos interacts" such as preexisting lung conditions.[235] It also indicates that "the long-term retention of asbestos fibers in the lung and the long latency period for the onset of asbestos-related respiratory diseases suggest that individuals exposed earlier in life may be at greater risk to the eventual development of respiratory problems than those exposed later in life." In addition, "[t[here is also some evidence of genetic predisposition for mesothelioma related to having a germline mutation in BAP1."

While EPA may have identified susceptible subpopulations, however, TSCA requires it to do more – it must estimate the size of these subpopulations and estimate the level of incremental risk they face beyond risk levels for the general population.

CONCLUSION

[234] Id.
[235] Id. at 206.

ADAO is deeply concerned by the many omissions and flaws in the draft risk evaluation. Although EPA has determined that asbestos presents unreasonable risks under the limited conditions of use it addresses, these risks are greatly understated because EPA –

- delays consideration of legacy asbestos exposure to an uncertain future risk evaluation
- focuses on only one asbestos fiber type
- does not consider cancers and non-cancer lung effects linked to asbestos
- ignores environmental exposure to asbestos
- excludes asbestos-contaminated talc products and exposures
- lacks basic information about asbestos importation and use that it should have obtained using TSCA information collection authorities
- unjustifiably relies on respirators to protect workers from asbestos exposure, and
- does not account for increased risks to subpopulations with greater susceptibility to asbestos or multiple pathways of exposure

EPA also departs from the well-established scientific and regulatory framework for estimating asbestos risks and calculates an IUR considerably lower than the long-standing IUR adopted by IRIS in the 1980s.

These flaws have resulted in a risk evaluation that fails to present a full and accurate picture of the threat that asbestos poses to public health and will undermine asbestos risk management policies now in place.

ADAO believes a comprehensive asbestos ban is long overdue and urges Congress to take immediate actions to ban asbestos that EPA cannot or will not take under TSCA.

Respectfully submitted,

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