COMMENTS OF ASBESTOS DISEASE AWARENESS ORGANIZATION ON DRAFT EPA PART 2 RISK EVALUATION FOR ASBESTOS UNDER THE TOXIC SUBSTANCES CONTROL ACT

EPA-HQ-OPPT-2021-0254
FRL-9347-06-OCSPP

89 Federal Register 26878 (April 16, 2024)

The Asbestos Disease Awareness Organization (ADAO) submits these comments on the U.S. Environmental Protection Agency (EPA) draft Part 2 risk evaluation on asbestos under the Toxic Substances Control Act (TSCA).

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 U.S. ban on asbestos, championing enactment of the Alan Reinstein Ban Asbestos Now Act.

ADAO was founded by Linda Reinstein and Doug Larkin after both of their loved ones were diagnosed with mesothelioma. Slowly but surely, we 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.

For more than 100 years, it has been known that when asbestos fibers become airborne, they can get trapped in the lungs and cause scarring and inflammation. Asbestos exposure causes suffering and fatal illnesses, including mesothelioma, asbestosis, and cancers of the lung, larynx, and ovaries.[1]

In 1976, the National Institute for Occupational Safety and Health (NIOSH) was the first U.S. federal agency to recommend a ban on asbestos in the workplace.

Since TSCA was amended in 2016, ADAO has expressed its views at every stage of the risk evaluation and risk management process, submitting extensive information to EPA, participating in Science Advisory Committee on Chemicals (SACC) public meetings, filing comments and position statements on key milestones and meeting often with EPA leadership and staff.

The Part 2 evaluation is an important step forward in the battle to protect Americans from deadly asbestos, which is responsible for more than 40,000 deaths in the United States every year. The

[1] *2023 Comprehensive Asbestos Report: The Analysis of Imports, Use, Impact on Human Health, and Current Regulations and Policy*. Reinstein https://www.asbestosdiseaseawareness.org/wp-content/uploads/2023/09/2023-Reinstein-Asbestos-Report-New.pd (attached to these comments).

U.S. government has done far too little to understand how and where Americans are being exposed to legacy asbestos that lurks in our homes, schools, workplaces, and in consumer products. Legacy asbestos exposure poses a serious danger to public health and is a major contributor to numerous asbestos-caused diseases, including deadly cancers and mesothelioma.

Millions of workers and consumers are exposed to asbestos from these legacy products, often unknowingly and without any precautions to prevent exposure. A strong, rigorous, and comprehensive analysis of the risks of legacy asbestos has never been conducted and is long overdue. Once we have fully defined these risks, we can turn to the all-important task of saving lives through stronger public education and more protections for at-risk first responders, construction workers repairing, renovating or demolishing older buildings, janitors servicing older HVAC systems, and teachers and students in older schools built with asbestos.

The Part 2 draft determines that exposure to legacy asbestos presents an unreasonable risk of injury to human health, a conclusion that ADAO strongly supports. If EPA's final evaluation affirms this finding, TSCA requires it to conduct a risk management rulemaking to eliminate the unreasonable risk. Currently, an uneven patchwork of federal and state laws and regulations apply to the handling and removal of legacy asbestos. Unfortunately, implementation of these programs is not measuring up to the high level of protection that public health demands. The risk management process will be a unique opportunity for EPA, stakeholders and leading experts to join forces to build public awareness of the threat of legacy asbestos and take steps to manage it more safely.

While EPA has done important work on the Part 1 Chrysotile Asbestos Final Rule, the Part 2 draft risk evaluation has many gaps and weaknesses and does not provide policymakers and public health experts with authoritative and credible guidance on the ongoing health threats from legacy asbestos. Disappointingly, EPA has failed to inform and engage stakeholders and to engage the independent scientific community in peer review. To assure that legacy asbestos remains a high priority, we recommend that EPA:

1. Contract with the National Academy of Sciences to provide a full review of the risks of legacy asbestos and its impact on public health and identify risk management strategies that will more effectively prevent asbestos-related death and disease;
2. In cooperation with other agencies and departments, convene a stakeholder advisory committee to review existing laws and regulations applicable to legacy asbestos, identify gaps in coverage and implementation, and recommend improvements;
3. Conduct an updated study of the presence of asbestos in public, residential and commercial buildings in the US; and
4. Develop educational asbestos prevention materials and conduct extensive community outreach campaigns

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EXECUTIVE SUMMARY

Why the Part 2 Risk Evaluation Is Critical in Protecting Public Health

The EPA Part 1 Chrysotile Asbestos rule bans ongoing importation and use of one asbestos fiber – chrysotile – and six current uses of this fiber and products containing it. While Part 1 is an important step forward in reducing asbestos exposure, Part 2 addresses a more urgent public health threat – legacy asbestos-containing products still in use. From 1900 to the present day, the US has consumed more than 31 million metric tons of asbestos.[2] 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 a pervasive presence in schools, factories, homes, apartments, construction and demolition sites, landfills and commercial and public buildings as well as industrial and commercial equipment. Much of the asbestos is in friable form and can be released into the air when disturbed through normal wear-and-tear, repairs, routine maintenance, renovations, and disasters.

Asbestos causes 40,000 deaths[3] each year and legacy asbestos is a major contributor to this death toll. Understanding legacy asbestos risks is a complex and challenging task. In contrast to Part 1, EPA must address not just chrysotile but all other asbestos fibers – the asbestiform varieties of crocidolite (riebeckite), amosite (cummingtonite-grunerite), anthophyllite, tremolite and actinolite

[2] *2023 Comprehensive Asbestos Report: The Analysis of Imports, Use, Impact on Human Health, and Current Regulations and Policy*. Reinstein https://www.asbestosdiseaseawareness.org/wp-content/uploads/2023/09/2023-Reinstein-Asbestos-Report-New.pdf

[3] https://www.apha.org/policies-and-advocacy/public-health-policy-statements/policy-database/2020/01/10/eliminating-exposure-to-asbestos

— plus richterite-asbestos and winchite-asbestos. The pathways and sources of exposure to legacy asbestos are also considerably more extensive and varied than the six conditions of use (COUs) examined in Part 1. They include, for example, construction and demolition sites where embedded asbestos fibers are released into the air; repair and maintenance work on HVAC systems, floor tile or insulation in homes, apartments and commercial buildings that releases asbestos; long-term disturbance of asbestos-containing materials (ACMs) in buildings that endangers occupants; and exposure to asbestos fibers leaking over time from damaged building components and equipment in factories, schools, and other public buildings.

The goal of the Part 2 evaluation is to examine the magnitude and duration of exposure to legacy asbestos from these and other pathways and then to determine the risks of lung cancer, mesothelioma, and lung disease that they pose to the public. As in Part 1, the bottom line of Part 2 will be a determination of whether exposure to legacy asbestos presents an unreasonable risk of injury to human health. If EPA finalizes its preliminary determination of unreasonable risk, TSCA will require the Agency to conduct a risk management rulemaking to eliminate that risk – just as EPA did in Part 1 for ongoing chrysotile imports and use. This rulemaking will provide a unique opportunity to strengthen the large but incomplete patchwork of federal and state laws and regulations that were put in place to manage legacy asbestos four decades ago. The public health benefits of more comprehensive and effective protections from asbestos exposure would be far-reaching and substantial.

Legacy Asbestos Has Been a Low Priority for EPA and the Part 2 Evaluation Is Long Overdue

There has long been a compelling public health need to update our understanding of the risks of legacy asbestos, but EPA has devoted little attention to this subject since the 1980s. The Agency conducted a study of the prevalence of asbestos in certain classes of buildings across the US in 1984 but it was incomplete at the time and is now out-of-date. Since then, there has been no comprehensive EPA assessment of the presence of asbestos in buildings and other structures, the populations exposed and the health risks from this exposure even though public concerns about the dangers of legacy asbestos persist. The Part 2 draft evaluation represents the first serious examination of legacy asbestos in nearly 40 years.

The comprehensive overhaul of TSCA in 2016 prompted renewed interest in asbestos after a long delay. Following years of work, EPA promulgated a comprehensive asbestos ban rule under TSCA in 1989 but, following an industry challenge, the rule was overturned in 1991 by the Fifth Circuit Court of Appeals.[4] When TSCA was amended in 2016, concern about EPA's failure to ban asbestos under the old law was a strong driver for strengthening EPA's authority. After passage of the amended law, there was broad support for including asbestos in the first round of risk evaluations and risk management rules that EPA was required to conduct. However, EPA initially began these activities without including legacy asbestos, maintaining that it lacked authority under TSCA to conduct risk evaluations on legacy products. ADAO and other groups then challenged EPA's position in the Ninth Circuit Court of Appeals. In November 2019, the Court held that EPA's exclusion of legacy uses and associated disposals from its risk evaluation "contradicts TSCA's plain

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

language" and that its asbestos risk evaluation must consider the risks posed by these sources of exposure.[5]

In its peer review of the draft Part 1 evaluation, the EPA Science Advisory Committee on Chemicals (SACC) expressed deep concerns about the draft's failure to address all asbestos exposure pathways, including legacy asbestos use and disposal. When Part 1 was finalized in late 2020, EPA promised to conduct a Part 2 evaluation addressing legacy asbestos as required by the Ninth Circuit decision as well as other elements missing from Part 1. However, since EPA did not set a timetable for Part 2 or define its scope, ADAO and its partners filed two legal cases in 2021 challenging the limitations of the Part 1 evaluation and seeking a timetable for completing Part 2. EPA and ADAO then negotiated an enforceable consent decree in late 2021 directing the Agency to issue a final Part 2 evaluation by December 1, 2024.

The Draft Part 2 Evaluation Confirms that the Risks of Legacy Asbestos Exposure are Significant, Widespread, and Seriously Threaten Public Health

While the draft Part 2 evaluation has serious flaws and limitations and does not fully characterize the health risk from legacy asbestos exposure, it leaves no doubt that legacy asbestos poses a significant ongoing threat to public health.

The draft demonstrates that legacy asbestos is present in hundreds of thousands of structures, building sites and disposal facilities. It also confirms that the populations at risk are large and diverse and include workers and their families, teachers and students, firefighters and other emergency responders, occupants of abandoned buildings, residents of homes and apartments, do-it-yourself consumers, and people who live or work in the vicinity of construction and demolition sites.

Using available monitoring data from OSHA and NIOSH, the draft evaluation calculates risks of cancer (lung cancer and mesothelioma) and non-cancer health effects (asbestosis, pleural thickening) for a subset of legacy conditions of use (COUs). Several of these estimated risks are above EPA's unreasonable risk benchmarks for cancer and non-cancer endpoints, an alarming finding in light of the unusually large population at risk. Although understated in many ways, these preliminary determinations of unreasonable risk underscore that a large percentage of the deaths and serious diseases caused by asbestos each year are likely attributable to legacy asbestos and these deaths will continue to occur without significant reductions in legacy asbestos exposure.

EPA's Failure to Assure Meaningful Public Engagement and Peer Review for Part 2 is a Step Backward

The far-reaching impacts of legacy asbestos exposure and the complexity and scope of the risk management process call for extensive outreach to key public health stakeholders, the scientific and medical community, labor and the general public. It is surprising and disappointing that EPA has made no effort to conduct this outreach or even to publicize the findings of the Part 2 evaluation. For example, apart from a short Webinar on May 13, EPA has provided no opportunity for

[5] *Safer Chemicals, Healthy Families v. EPA*, 943 F.3d 397 (9th Cir. 2019).

stakeholders to present their views directly to the Agency. Equally important, the draft is poorly organized, bogged down in arcane details and often incomprehensible, even to asbestos experts. Nowhere is there a compelling narrative about the dangers of legacy asbestos and the limitations of current risk management efforts that would engage experts and the public in a robust process on how best to eliminate the urgent risks identified in the evaluation.

Compounding the lack of public engagement is the absence of independent scientific peer review of the draft evaluation. The Agency conducted a limited letter review of a white paper addressing certain health effects,[6] but is not soliciting external expert review of the draft evaluation itself. In letters to EPA, ADAO, other NGOs and the Environmental Protection Network expressed alarm about this omission, but Agency leadership was not responsive to these concerns.

EPA's approach is in sharp contrast to Part 1 and numerous other EPA risk evaluations under TSCA, on which its SACC conducted. a transparent and rigorous peer review. The Part 2 evaluation presents more complex scientific challenges than Part 1. The exposed worker and consumer populations are considerably larger and more diverse, and pathways of exposure and release are more numerous, varied, and complex. As demonstrated in these comments, EPA's methodologies for literature review and exposure analysis have serious flaws that undoubtedly would have concerned knowledgeable experts who critiqued the evaluation. A robust peer review process would have strengthened the credibility and weight of the final evaluation and provided a vehicle to engage asbestos experts, stakeholders and the general public. Without peer review, the evaluation will fail to give policymakers and public health experts the authoritative guidance on the ongoing health threats from legacy asbestos that is necessary to formulate effective strategies to address these risks.

Flaws in Part 2 and Recommendations for Strengthening the Draft Evaluation

While ADAO strongly supports EPA's preliminary determinations of unreasonable risk, the draft Part 2 evaluation contains several gaps, limitations and deficiencies. By excluding important exposure pathways, relying on incomplete data and giving short shrift to populations with aggregate exposures or unique susceptibility to asbestos' health effects, the draft seriously understates legacy asbestos risks.

Key ADAO comments on the draft evaluation include the following:

The Human and Economic Toll of Asbestos

- For over a century, asbestos has been known to cause widespread disease and death. Asbestos is unique among dangerous substances in its widespread use and harmful impacts on the population. The human cost of asbestos exposure has been staggering and the death toll enormous. From 1991 to 2019, more than one million Americans died from preventable asbestos-caused diseases.[7]

[6] https://www.epa.gov/newsreleases/epa-advances-asbestos-part-2-risk-evaluation-seeks-peer-review-white-paper
[7] https://www.asbestosdiseaseawareness.org/newsroom/blogs/adao-asbestos-mortality-report-from-1991-2019/

Gaps in EPA's Analysis of Pathways, Levels and Risks of Legacy Asbestos Exposure

- To put the risks of legacy asbestos in perspective, policymakers and the public need a comprehensive and transparent picture of the prevalence of legacy asbestos in the US—how many buildings have legacy asbestos, what types of buildings are affected, when were they built, how many people inhabit or frequent them and where they are concentrated geographically. The draft evaluation fails to meet this need.
- The Part 2 draft gives short shrift to ACM in buildings as a source of asbestos exposure and lung cancer, mesothelioma and non-cancer disease. Unless it recognizes the risks to occupants of asbestos-containing schools, apartments and public buildings, EPA will fail to protect a large potentially exposed and susceptible population from unreasonable risk, a population which has already been documented to be at risk for asbestos-related death and disease as a result of exposure to ACM in buildings.
- Recent medical screening data confirms that significant amounts of asbestos remain in place at major industrial facilities and demonstrates that damaged and deteriorated asbestos insulation continues to be a systemic threat to worker health.
- Although EPA makes preliminary determinations of unreasonable risk for nearly all the occupational COUs for which it estimates risks, these estimates derive from an incomplete database and disproportionately emphasize unrepresentative studies that understate likely levels of exposure and risk. EPA must (1) draw on a broader universe of studies (going as far back as 1970), (2) give less weight to OSHA and NIOSH data from pre-announced site visits, flawed industry studies and studies in which measured levels reflect the use of PPE, and (3) clarify the impact of different sampling and analytical methods on the representativeness of the data.
- There are over 630,000 miles of asbestos cement pipe buried across the United States. The Part 2 draft, however, classifies asbestos cement pipe as a low priority for evaluation. Thus, the draft does not address risks to workers who renovate, maintain or replace pipes, DIY consumers who engage in these activities directly, or consumers of drinking water containing asbestos leaching from cement pipes.
- EPA undermines its whole chemical risk determination by separating legacy asbestos COUs into those that "contribute" to the unreasonable risk and those that do not. In the latter category are seven COUs that EPA finds do not meet its risk benchmarks and ten COUs for which it lacks "sufficient information" to make quantitative determinations of risk. We strongly urge EPA **not** to identify COUs that do not contribute to the whole chemical unreasonable risk of asbestos. Instead, all COUs for which there is evidence of potential exposure should be included in its unreasonable risk determination. This will avoid narrowing risk management options unnecessarily and enable EPA to consider a broad range of effective risk reduction strategies during its rulemaking process.
- Although firefighters are identified in Part 2 as a highly exposed population, the draft provides limited information on explosions, floods, rail derailments, collapsed buildings and other emergency events that result in uncontrolled asbestos releases and spikes in exposure by emergency responders, bystanders and the general public. An increase in extreme weather events will result in more damage and destruction of ACM-containing

structures, releasing more asbestos into the air and increasing risks to the public and clean-up and emergency response workers.

- EPA'**s** assessment of exposure and risk from legacy asbestos waste is incomplete and inadequate for risk management. EPA's only source of information on the volume of asbestos-containing waste generated, treated and disposed of nationally is its TRI database but TRI reports account for a very small percentage of all such waste. The draft also does not discuss, even in general terms, conditions and management practices at waste treatment and disposal facilities and the potential for offsite releases of asbestos-containing dust to which nearby residents or bystanders are exposed. Thus, the contribution of waste sites to general population risk cannot be determined.

Limited Attention to Vulnerable Populations

- Surprisingly, there is no environmental justice analysis in the draft evaluation even though there are many indicators that disadvantaged frontline communities have disproportionate exposure to legacy asbestos. These communities are often located in close proximity to landfills and other disposal sites that treat or manage asbestos waste as well as to older industrial facilities which contain asbestos insulation, gaskets, flooring, cement pipes and other ACMs. They also have abandoned or substandard housing, schools and other structures containing poorly maintained and unsafe legacy asbestos. And their residents often have high levels of asthma and other health conditions that can be aggravated by asbestos exposure or predispose them to asbestos disease.
- EPAs identification of PESSs and risk analyses for vulnerable populations are inadequate and incomplete. EPA does not quantify risk for smokers separate from the general population although epidemiology studies demonstrating higher risks to smokers provide information about the magnitude of this risk. The draft evaluation also makes no effort to define and quantify the segment of the population with early-life exposure to asbestos or to adjust risk estimates to account for this important risk factor.
- EPA's inclusion of aggregate exposure analysis in Part 2 is a welcome recognition of the multiple sources of exposure to asbestos by a large segment of the population. However, the scope of this analysis is too narrow. EPA must address aggregate exposures across *all* COUs that exceed risk benchmarks so that higher risk populations are clearly identified and all sources of risk to these groups are accounted for.

Ignoring Risks of Asbestos-containing Talc

- EPA's dismissal of the presence of asbestos in talc is irresponsible and ignores extensive evidence of the risks to consumers and workers from asbestos-contaminated talc. The Agency's claim that "exposure is unlikely" because "selective mining practices occur and are successful in generally avoiding deposits that are likely to contain asbestos minerals." is not defensible. There is no reliable means to selectively mine to avoid all asbestos given the mineralogy, inadequate basic analytical techniques used in industry, the limitations of the human eye, and the physical nature of talc ore.

Quantifying Cancer and non-Cancer Risks

- In general, EPA's identification of asbestos's cancer and non-cancer effects is supported by the weight of evidence and aligned with the many previous determinations by leading experts and public health bodies.
- We strongly support EPA's decision to use a single IUR that is applicable to all asbestos fibers without differentiation and assumes that chrysotile and amphiboles are equally potent in causing lung cancer and mesothelioma. This approach has repeatedly been supported by the Agency and its scientific advisors and there is no scientific basis to differentiate between fiber types on the basis of relative carcinogenic potency.
- EPA proposes to adopt a Point of Departure (POD) for non-cancer effects of 2.6×10^{-2} fiber/cc based on dose-response data in the occupational cohort from the O.M. Scott plant in Marysville, Ohio as described in the Libby IRIS assessment. This is the first time EPA has made quantitative non-cancer risk determinations for all asbestos fibers and it adds an important dimension to its risk analysis.

I. Asbestos Is Unique Among Dangerous Substances in its Widespread Use and Harmful Impacts on the Population

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.[8] There is 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."[9] 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, the use of asbestos has been massive in scale. According to the U.S. Geological Survey (USGS):[10]

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

[8] IARC. Monograph 1OOC: Asbestos (Chrysotile, Amosite, Crocidolite, Actinolite· and· Anthophyllite), Lyon: International Agency for Research on Cancer (2012)
[9] 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).
[10] https://pubs.usgs.gov/circ/2006/1298/

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.[11] 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. 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."[12] 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.[13]

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."[14] The danger extends far beyond manufacturing plants— firefighters, construction workers, auto mechanics and repairmen, and schoolteachers are among the workers at highest risk for asbestos exposure and related diseases. Asbestos fibers can also be 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, 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.[15] These findings reinforce the urgent need to eliminate exposure to asbestos and underscore the continuing public health challenge that asbestos presents.

There is overwhelming consensus in the scientific community that there is no safe level of exposure to asbestos. As noted by WHO:[16]

> 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.

[11] http://ghdx.healthdata.org/gbd-results-tool?params=gbd-api-2017 permalink/535c35ab1fc10471f721c9b58eecd3c2
https://www.nera.com/publications/archive/2017/asbestos--economic-assessment-of-bans-and-declining-production-a.html
[13] http://www.euro.who.int/data/assets/pdf_file/0009/341757/Asbestos_EN_WEB_reduced.pdf?ua=1.
[14] https://www.atsjournals.org/doi/full/10.1164/rccm.200310-1436ST
[15] 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.
[16] https://www.who.int/ipcs/assessment/public_health/chrysotile_asbestos_summary.pdf

IARC[17], the Occupational Safety and Health Administration (OSHA)[18], the Department of Health and Human Services,[19] the National Institute for Occupational Safety and Health (NIOSH)[20], 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 Part 1 evaluation,[21] 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)[22] estimated exposures to asbestos below 200-300 fiber/cc years were not associated with increased cancer deaths. By 1980 a publication by McDonald[23] found no increased risk of lung cancer deaths below 20 fiber/cc years, a level ten times lower. By 1998, Iwatsubo et al.[24] 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)[25] 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.[26]

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 during consumption of drinking water contaminated by asbestos cement pipe.

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[17] http://monographs.iarc.fr/ENG/Monographs/vol100C/mono100C.pdf.
[18] https://www.osha.gov/laws-regs/federalregister/1994-08-10
[19] https://ntp.niehs.nih.gov/ntp/roc/content/profiles/asbestos.pdf
[20] https://www.cdc.gov/niosh/docs/2011-159/pdfs/2011-159.pdf
[21] Comments Of Richard A. Lemen, Ph.D., MSPH On EPA's Draft Part 1 Risk Evaluation For Asbestos ("Lemen Statement")
27 May 2020 (Lemen Statement) at 1.
[22] 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.
[23] 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.
[24] 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.
[25] 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.
[26] Lemen Statement.

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 strengthening its asbestos standards in 1999, 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] These risk levels would be deemed unreasonable under EPA TSCA cancer risk benchmarks for exposed workers. This underscores the unique benefits of TSCA in protecting against unsafe 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. Using this authority, the final Part 1 rule prescribes an existing chemical exposure limit (ECEL) for chrysotile asbestos significantly lower than the current OSHA standard.

II. The Draft Evaluation Fails to Provide a Comprehensive Analysis of Pathways, Levels and Risks of Legacy Asbestos Exposure

A. The Draft Lacks Credible Estimates of the Number of Buildings Containing Legacy Asbestos and the Size of the Exposed Population

To put the risks of legacy asbestos in perspective, policymakers and the public need a comprehensive and transparent picture of the prevalence of legacy asbestos in the US – how many buildings have legacy asbestos, what types of buildings are affected, when were they built, how many people inhabit or frequent them and where they are concentrated geographically. The draft evaluation fails to meet this need.

In 1984, EPA conducted a survey of representative regions of the US to determine the presence of friable asbestos-containing materials (ACM) in buildings, the uses of this ACM and the amount of asbestos they contained.[29] 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 survey (representative of a target population of 3.6 million buildings) included significant in person testing and observation of installed asbestos in a randomly selected sample of building types. The report reached several significant conclusions, including:

- 20 percent of buildings had asbestos-containing friable material (499,000-966,000 buildings);
- 16 percent of buildings had asbestos-containing pipe and boiler insulation (239,000-888,000 buildings);
- The average percent asbestos content (weighted by square footage of material) in asbestos-containing sprayed- or troweled-on friable material was 14 percent;
- For asbestos-containing pipe and boiler insulation material, the average percent asbestos content was 70 percent;

[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
[29] USEPA, *Asbestos in Buildings:* A *National Survey of Asbestos-Containing Friable Materials*. Washington, DC: Office of Toxic Substances, EPA 560/5-84-006 (1984).

- An estimated 1,526,000 buildings, or 42 percent of buildings in the survey, had asbestos-containing floor tile; and
- 59 percent of residential apartment buildings (with 10 or more units) that were surveyed contained friable asbestos, the highest percent in any building category.

A 1988 EPA report to Congress, required by the 1986 Asbestos Hazard Emergency Response Act (AHERA), provided additional analysis of the survey data in the 1984 report.[30] It concluded that, for all public and commercial buildings surveyed, 14 percent of all friable asbestos-containing material was damaged (501,000 buildings) and 9 percent was damaged significantly (317,000 buildings). Residential apartment buildings had the highest percentage (23 percent) of damaged asbestos.

The report also concluded that, "more public and commercial buildings contain thermal system insulation asbestos than other kinds of friable asbestos" and it "is generally in worse condition and in higher concentrations than the other asbestos found in public and commercial buildings." It emphasized that this "asbestos represents a potentially serious health hazard to the custodial and maintenance staff, who work with and around this material on a regular basis."

Although schools were omitted from the 1984 survey, the 1988 report found that, based on a separate 1983 EPA national telephone survey of schools, 35 percent (35,000) contained friable asbestos.

According to the 2018 Environmental Protection Agencies (EPA) Office of Inspector General (OIG) Report, "substantial amounts of asbestos, particularly in sprayed form, have been used in school buildings, especially from 1946 through 1972. More than 50 million students from kindergarten through 12th grade attend more than 131,000 public and private school facilities in the United States, and more than 7 million teachers and others work in those schools."[31]

As the only major studies of the presence of ACM in buildings in the last 35 years, the 1984 and 1988 reports provide data of unique value in assessing the risks of legacy asbestos. While out-of-date in some respects,[32] these data are still useful in determining the segment of the building universe with unsafe asbestos, a key data-point in estimating the size of the populations at risk. Nonetheless, the Part 2 draft does not discuss or analyze these reports, nor does it identify new data that would update their findings. Instead, it attempts to use Bureau of Labor Statistics (BLS) and the U.S. Census' Statistics of U.S. Businesses (SUSB) sources to estimate the number of "establishments and worksites" for each ACM occupational exposure scenario. DRE at 249-250. However, these projections are not only highly uncertain but fail to include large portions of the building universe (such as occupied buildings that are not active construction sites but have damaged ACM and schools in which teachers and students are exposed continuously to asbestos).

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[30] ASBESTOS-CONTAINING MATERIALS IN PUBLIC BUILDINGS: A Report To Congress, U.S. Environmental Protection Agency Washington, D.C., February 1988.

[31] *2023 Comprehensive Asbestos Report: The Analysis of Imports, Use, Impact on Human Health, and Current Regulations and Policy*. Reinstein https://www.asbestosdiseaseawareness.org/wp-content/uploads/2023/09/2023-Reinstein-Asbestos-Report-New.pdf

[32] For example, given turnover in the nation's building stock, a smaller percentage of US buildings may contain asbestos today but the condition of the remaining asbestos may be worse.

A critical element in defining the risk posed by legacy asbestos is a detailed breakdown of the key segments of the impacted population – i.e., the number and types of workers, emergency responders, DIY consumers, families with take home exposure, building occupants and members of the general public with asbestos exposure. EPA attempts to quantify the populations at risk but its estimates are buried in an appendix to the draft evaluation, are incomplete and are not translated into understandable top-line numbers. EPA also bases its estimates on unreliable calculations of the number of establishments and worksites where asbestos is present as opposed to sound data on the number of ACM-containing buildings.

Even with these limitations, EPA's estimates of ACM-exposed populations are large: for example, it projects that exposed workers engaged in construction maintenance, renovation and demolition total 4.8 million and that there are 364,300 career firefighters and 676,900 volunteer firefighters with potential asbestos exposure. DRE at 256, 285. Even these estimates, however, are dramatically understated because they do not include occupants of ACM-containing commercial and residential buildings, teachers and students in ACM-contaminated schools, DIY consumers, families with take home exposure and members of the public exposed to indoor or outdoor air with elevated asbestos levels. Quantifying these subpopulations would add several million people to the total number of at-risk Americans.

B. EPA Incorrectly Excludes Teachers, Students and other Building Occupants from the Population at Risk and Fails to Include Them in its Analysis of Risks

The Part 2 draft gives short shrift to ACM in buildings as a source of asbestos exposure and lung cancer, mesothelioma and non-cancer disease due to that exposure. According to Dr. Christine Oliver, "unless this shortcoming is remedied, the EPA will fail to protect a large potentially exposed and susceptible population from unreasonable risk," a population which has already been documented to be at risk for asbestos-related death and disease as a result of exposure to ACM in buildings.[33]

The Part 2 draft makes the faulty assumption that asbestos in building materials does not pose a risk unless "released during construction, modification, or demolition" of ACM "in homes, schools, or commercial buildings" (Executive Summary).[34] According to Dr. Oliver, EPA fails to recognize that "friable ACM that is damaged by age, water, or trauma can in fact release fibers by attrition and in response to perturbation by such events as building vibration and air currents.[35] More aggressive disturbance is not necessary to asbestos fiber release."

EPA conducted a limited systematic review of exposure data from buildings with legacy asbestos but found these data to be inadequate. EPA excluded sampling data obtained in schools under AHERA on the questionable rationale that because they were collected during abatement activities

[33] Comments of L. Christine Oliver, MD, MPH, MS, FACPM (separately submitted to EPA Part 2 docket).
[34] EPA. Draft Risk Evaluation for Asbestos. Part 2. EPA Document # EPA-740-D-24-006. Office of Chemical Safety and Pollution Prevention. April 2024. [+ Pg ##]
[35] Oliver LC. Asbestos in public buildings. In:Rom WN, ed. Environmental Occupational Medicine. New York: Lippincott-Raven. 1998. Pp.387-395.

and considered not representative of "non-occupational exposures."[36,37] Overall, the Agency concluded that "The available information regarding passive or non-source attributed asbestos concentrations in indoor air of residential and public buildings is not sufficient for EPA to conduct a quantitative exposure assessment."DRE at 71. On this basis, the Part 2 draft did not include building occupants (including teachers and students in schools) in its projections of populations at risk and made no determinations of unreasonable risk for these COUs.

As Dr. Oliver explains the ramifications of this omission for the overall completeness of the evaluation:

> The number of building occupants at risk from ACM in buildings because of past and to a lesser extent current exposures likely far exceeds that of the other groups. Determinants of exposure risk are friability, accessibility, and condition of the ACM, as well as patterns of airflow and potential for disturbance.[38] While these exposures may not be as intense overall, proximity to occupational- and DIY-type activities likely result in spikes in intensity. The duration is long for many who work (e.g., government employees, teachers) and live in such buildings; so that cumulative exposures are significant, particularly in light of the fact that, with the possible exception of asbestosis, no threshold exposure has been shown for [asbestos-related disease].

Dr. Oliver elaborates that:

> ACM "in place", in good condition, and undisturbed does not pose a risk to building occupants as long as it stays that way.[39] Over time, however, such ACM ages and may become water-damaged and/or disturbed as a result of repair and maintenance work or, more benignly, building vibration and aggressive air currents. Under these conditions asbestos fibers are released, become airborne, and settle on ceiling tiles, available for inhalation should the ceiling tiles be moved or removed. If the tiles are removed, there is a direct pathway to surfaces and building occupants below. These fibers are then entrained as a result of dusting and cleaning activities and become available once more for inhalation.

An example of asbestos fiber release from such ACM "in-place" in buildings is provided in Sawyer's report of air sampling conducted in the Yale University Art and Architecture Building in 1971.[40] Construction of the building was completed in 1963. A mixture of asbestos (chrysotile) and fiberglass was spray-applied to the ceilings in the building to retard fire and sound. Sawyer notes: "The exposed and friable ceilings soon began gradual deterioration as air currents, ventilation leaks, and vibration resulted in fiber loss." Asbestos air sampling under quiet conditions was

[36] Asbestos-containing materials in schools; final rule and notice. Federal Register. 40 CFR Section 763, 1987.
[37] Asbestos School Hazard Abatement Reauthorization Act of 1990. Pub. L. 101-637,S.1893,1990.
[38] Oliver LC. Asbestos in public buildings. In:Rom WN, ed. Environmental Occupational Medicine. New York: Lippincott-Raven. 1998. Pp.387-395.
[39] Oliver LC. Asbestos in public buildings. In:Rom WN, ed. Environmental Occupational Medicine. New York: Lippincott-Raven. 1998. Pp.387-395.
[40] Sawyer RN. Asbestos exposure in a Yale building. Analysis and resolution. *Environ Res*. 1977;13:146-169.

conducted in 1971; eight samples were collected and analyzed using phase contrast microscopy. Mean concentration for the eight samples was 0.3 f/cm^3, with a range of 0.1-0.5 f/cm^3.

Dr. Oliver emphasizes that EPA's evaluation disregards numerous studies in the literature reporting malignant mesotheliomas (MM) and other asbestos-related diseases in occupants of public and commercial buildings and schools, as well as health care facilities with friable ACM. The summary of this literature in Dr. Oliver's comments on the draft evaluation is compelling and worthy of being quoted in full:

> MM is a sentinel event for asbestos exposure. Case reports and epidemiologic studies have linked the occurrence of MM with the presence of ACM in buildings.[41] [42] [43] [44] Stein et al reported the case of a 54-year-old female office worker diagnosed with malignant pleural mesothelioma.[45] TEM analysis of digested lung tissue revealed findings consistent with amosite asbestos, confirmed by energy dispersive X-ray microanalysis of 40 randomly-selected fibers. The case had always been an office worker and had no known source of asbestos exposure outside of this job. The building in which she worked was constructed 15 years previously, 14 years prior to her mesothelioma diagnosis. The ceiling was spray-on asbestos. Polarized light microscopy (PLM) and dispersion staining of a piece of the ceiling showed it to be 70% asbestos.
>
> Lilienfeld reported three cases of malignant pleural mesothelioma and one case of peritoneal mesothelioma in school teachers whose only known exposure to asbestos was fireproofing/insulation material in the schools in which they taught.[46] Age range was 43 to 64 years; two cases were male, and two female. Duration of work as a school teacher ranged from 15 to 31 years. Analyses of bulk samples of ACM in two cases revealed chrysotile and amosite, and in the third case, chrysotile, amosite and crocidolite. In the case of a 60-year-old teacher in Chicago, asbestos was used in acoustical ceiling tiles, pipe insulation, and other fire protection materials.
>
> Anderson et al carried out a case-control study of MM in Wisconsin males.[47] Data were derived from a population-based statewide cancer registry. Elevated gender- and age-adjusted odds ratios (OR) were observed for schools and for janitors: OR 3.5, 95% confidence interval (CI) 1.5-8.3 and OR 2.83, 95% CI 1.4-6, respectively.

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[41] Stein RC, Kitajewska JY, Kirkham JB, Tait N, Sinha G, Rudd RM. Pleural mesothelioma resulting from exposure to amosite asbestos in a building. Respir Med. 1989;83:237-239.

[42] Lilienfeld DE. Asbestos-associated pleural mesothelioma in school teachers: a discussion of four cases. NY Acad Sci. 1991;643:454-458.

[43] Anderson HA, Hanrahan LP, Schirmer J, Higgins D, Sarrow P. Mesothelioma among employees with likely contact with in-place asbestos-containing building materials. Ann NY Acad Sci. 1991;643:550-572.

[44] Tomasallo CD, Christensen KY, Raymond M, Creswell PD, Anderson HA, Meiman JG. An occupational legacy. Malignant mesothelioma incidence and mortality in Wisconsin. JOEM. 2018;60(12):1143-1149.

[45] Stein RC, Kitajewska JY, Kirkham JB, Tait N, Sinha G, Rudd RM. Pleural mesothelioma resulting from exposure to amosite asbestos in a building. Respir Med. 1989;83:237-239.

[46] Lilienfeld DE. Asbestos-associated pleural mesothelioma in school teachers: a discussion of four cases. NY Acad Sci. 1991;643:454-458.

[47] Anderson HA, Hanrahan LP, Schirmer J, Higgins D, Sarrow P. Mesothelioma among employees with likely contact with in-place asbestos-containing building materials. Ann NY Acad Sci. 1991;643:550-572.

Case identification and exposure histories revealed 12 MMs in school teachers and 29 in building maintenance workers. Nine (75%) of the school teachers had no known exposure to asbestos outside of their work as a teacher. Of the maintenance workers, 10 worked in public schools and seven in other public buildings; 10 (34% of the total group) had no known exposure to asbestos outside of their work in buildings.

Tomasallo et al. carried out a case-control study of mesothelioma incidence and mortality in Wisconsin over the period 1997 to 2013.[48] Occurrence was examined by usual industry and occupation. A twofold increase in risk for mesothelioma mortality was observed in the group of "primary, secondary, and special education teachers": OR 2.10, 95% CI 1.05-4.26. Analysis of the subset of "elementary and middle school teachers", compared to the "professional and related occupations" and all other occupations, revealed significant increase in risk for MM among elementary and middle school teachers: OR 3.50, 95% CI 1.46-8.38 and OR 2.53, 95% CI 1.26-5.09, respectively.

In the fall of 2019 ten public schools in Philadelphia were closed following the diagnosis of MM in a longtime teacher, and the discovery of "shoddy" abatement work by the Philadelphia School District's own teams and outside contractors.[49] Of eight wipe samples of dust collected by teachers in one Philadelphia elementary school and analyzed for asbestos, all contained chrysotile asbestos and two (12.5%) contained amosite asbestos.[50]

In April 2023, *The Observer*, a Sunday newspaper in the U.K. owned by *The Guardian*, published an article describing the results of an analysis of death certificate data by the Office of National Statistics (ONS).[51] Results of the data analysis revealed that 94 educational professionals and 53 healthcare professionals in England had died of MM since 2017.

The paper reported "Of the 94 education professionals, 39 worked in primary and nursery schools, 21 in secondary schools and 21 in further and higher education institutions." Thirteen were special education needs staff, senior education professionals, and Ofsted inspectors. Among the 53 healthcare professionals who died of MM were nurses and midwives, therapists, and doctors, psychologists, and other professions. Attribution was to damaged and deteriorated ACM in schools and other education buildings and in healthcare facilities in England.

[48] Tomasallo CD, Christensen KY, Raymond M, Creswell PD, Anderson HA, Meiman JG. An occupational legacy. Malignant mesothelioma incidence and mortality in Wisconsin. JOEM. 2018;60(12):1143-1149.
[49] Graham KA, Ruderman W. Amid Philly schools' asbestos crisis, city to pay an outside monitor to watchdog jobs. The Philadelphia Inquirer. Updated: February 2020.
[50] Ruderman W, Laker B, Purcell D. New test: 10.7 million fibers on floor at Philadelphia elementary school. The Philadelphia Inquirer. June 6, 2018.
[51] Savage M. Policy Editor. "The tragic cost of under-investment": asbestos blamed for 150 deaths of school and hospital workers in England. The Observer. April 16, 2023.

> Chronic non-cancer outcomes associated with ACM in buildings have been reported in public school custodians.[52,53,54] The principal ARDs observed were pleural plaques and interstitial lung disease (ILD) likely attributable to asbestosis. Oliver et al examined 121 Boston public school custodians (BPSC); of these, 57 (47.5%) reported no exposure to asbestos outside of work as a custodian (NOE). Mean duration of employment for this group was 25.7 years. Twenty-one percent of the NOE group had pleural plaques on chest X-ray vs. 33% for the group overall and 0.7% of a group of 711 laboratory, maintenance, and grounds personnel at a large university in Boston. The number with ILD was 3 (2.5%). Prevalence of pleural plaques increased with increasing duration of work as a custodian. Tasks performed by the BPSC included sweeping and dusting of plaster and/or insulation dust, patching and/or removing torn insulation on pipes and boilers, and boiler maintenance. Air sampling during these activities was not performed on a regular basis.
>
> Custodians in the New York City public schools were surveyed by Selikoff and Levin for radiographic evidence of ARD.[55] Of the 666 surveyed, 246 (37%) had no significant outside asbestos exposure. Over 60% had worked for at least 20 years. Radiographic outcomes for this group were pleural plaques (7%), ILD (17%), and both (3%).
>
> Balmes et al examined 673 school district employees in California.[56] Sixty-three percent (422) reported NOE; the jobs for 102 (24.2%) were classified as custodial or building maintenance. The prevalence of ARD on chest X-ray in the form of pleural plaques and/or ILD was 8.8%. For the group overall, prevalence was increased 30% for those whose duration of employment was greater than 10 years.

It is unclear whether EPA failed to identify these studies or was aware of them but chose not to discuss them in its draft evaluation because they contained no or limited air sampling data. Whatever the case, the studies provide strong qualitative evidence of the causal connection between damaged and deteriorated ACM in schools and other buildings and asbestos-related death and disease on the part of building occupants. Accordingly, they should be given substantial weight in any determination of unreasonable risk for legacy asbestos in buildings.

The draft evaluation also ignored other important information about asbestos exposure by building occupants. As described by Dr. Oliver, EPA either did not consult or chose not to include data from the Health Effects Institute-Asbestos Research (HEI-AR) report entitled *Asbestos in Public and Commercial Buildings: A Literature Review and Search of Current Knowledge* produced in

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[52] Oliver LC. Asbestos in public buildings. In:Rom WN, ed. Environmental Occupational Medicine. New York: Lippincott-Raven. 1998. Pp.387-395.
[53] Levin SM, Selikoff IJ. Radiological abnormalities and asbestos exposure among custodians of the New York City Board of Education. Ann NY Acad Sci. 1991; 643:530-539.
[54] Balmes JR, DaPonte A, Cone JE. Asbestos-related disease in custodial and building maintenance workers from a large municipal school district. Ann NY Acad Sci. 1991;643:540-549.
[55] Levin SM, Selikoff IJ. Radiological abnormalities and asbestos exposure among custodians of the New York City Board of Education. Ann NY Acad Sci. 1991; 643:530-539.
[56] Balmes JR, DaPonte A, Cone JE. Asbestos-related disease in custodial and building maintenance workers from a large municipal school district. Ann NY Acad Sci. 1991;643:540-549.

1991. HEI was charged (by Congress) with undertaking a research program to, among other things, "determine actual airborne asbestos fiber levels prevalent in buildings" and "characterize peak exposure levels and their significance."[57] Results are described both qualitatively and quantitatively.

Similarly, the draft evaluation makes no mention of a compendium of papers from the *Annals of the New York Academy of Sciences* entitled "The Third Wave of Asbestos Disease: Exposure to Asbestos in Place. Public Health Control."[58] To better understand asbestos-related health risks associated with exposure to "in-place" asbestos, these papers present exposure scenarios and qualitative information about dose-response relationships in addition to results of epidemiologic studies.

EPA's systematic review did not search for monitoring data prior to 2000 and was limited to US and Canadian studies. According to Dr. Oliver, much of the relevant asbestos air sampling in public and commercial buildings, including schools, was conducted prior to 2000: in the 1980s and 1990s, and to a lesser extent, the 1970s. There is no reason to believe that older data or asbestos levels measured outside North America would lack relevance to current conditions in US buildings. Because of the limitations of its systematic review, EPA likely overlooked data sources that could have contributed to quantitative exposure assessment of building occupants. As Dr. Oliver recommends, EPA should conduct a second broader systematic review for ACM monitoring data for public, residential, and commercial buildings with expansion of search terms to include hospitals and health care facilities, a larger number of countries, and indoor asbestos sampling data going back to 1970.

C. The Presence of Damaged Asbestos Insulation in Older Industrial Facilities Is a Serious Health Threat to Insulators Who Entered the Workforce After Such Insulation was no Longer Installed

A white paper submitted to EPA by the International Association of Heat and Frost Insulators and Allied Workers attached to these comments) presents documentation of conditions at certain major Chicago-area refineries and power houses. It confirms that significant amounts of asbestos remain in place at major industrial facilities and demonstrates that damaged and deteriorated asbestos continues to be a systemic worker protection concern. In addition, a comprehensive medical screening program showed that a new generation of workers had significant levels of asbestos disease. Specifically, 47% of the Chicago Insulators screened who started work as insulators in the 1980's had asbestos pleural disease as shown by the 2014-2016 screening results. The average age of these Insulators was under 53 and the date of their first employment showed that they did not install asbestos thermal insulation and that their health impairment was solely due to legacy asbestos.

[57] Asbestos in Public and Commercial Buildings: A Literature Review and Synthesis of Current Knowledge. *Health Effect Institute – Asbestos Research (HEI-AR).* 141 Portland Street, Suite 7100, Cambridge, MA. 1991.
[58] Levin SM, Selikoff IJ. Radiological abnormalities and asbestos exposure among custodians of the New York City Board of Education. Ann NY Acad Sci. 1991; 643:530-539.

In short, in industrial facilities as well as residential and public buildings, normal wear-and-tear and poor maintenance practices are responsible for significant legacy asbestos exposure and increases in asbestos disease.

D. EPA's Analysis of Sampling Data for Occupational Exposure is Incomplete and Skewed Toward Unrepresentative Studies that Lead to Underestimates of Risk

Although EPA makes preliminary determinations of unreasonable risk for nearly all the occupational COUs for which it estimates risks, these estimates derive from an incomplete data-base and disproportionately emphasize unrepresentative studies that understate likely levels of exposure and risk.

First, as with studies of building occupant exposure, it appears that EPA's systematic review did not search for occupational monitoring studies conducted before 2000 and only identified studies from the US and Canada.[59] In addition, the bulk of the monitoring data analyzed in the draft are from NIOSH and OSHA data-bases and industry studies, which likely are not representative of actual workplace conditions because employers knew in advance that monitoring would be conducted. Other more credible studies were not included in EPA's analysis. For example, EPA did not access data from state abatement programs, EPA regions enforcing AHERA or NESHAP requirements, or independent research predating 2000.

Finally, many of the studies relied on by EPA likely involved workers wearing personal protective equipment (PPE) even though EPA's policy is to base its determinations of unreasonable risk on exposure measurements that reflect the absence of PPE. No effort was made to screen out studies of workers using PPE or to give such studies less weight than studies in which workers did not use PPE.

In his detailed comments on the draft evaluation submitted to the EPA docket, Dr. Steven Markowitz, a noted occupational exposure expert and member of the SACC review panel **f**or EPA's Part 1 evaluation, explains that:

> EPA relies heavily on OSHA-CEHD data for air monitoring results. OSHA-derived data must be considered with circumspection due to the intervention of OSHA into the workplace (i.e., the Hawthorne effect). The employer and workforce are under best behavior, because OSHA inspections and monitoring are usually planned with adequate preparation time for all concerned. Work practices (e.g., wetting the material) are likely altered for the monitoring session, undermining the value of the monitoring results as a basis to characterize "central tendency" or "high-end" exposures. The goal of this EPA exercise - risk management – is to address likely exposure conditions for workers and ONU's Having an OSHA inspector looking over your shoulder is not a likely event. Notably 200 of the 8-hour TWA results of OSHA were non-detects (p. 285).

[59] ADAO could not find a description of the SR criteria EPA applied to occupational monitoring studies in the draft evaluation or its appendices.

EPA itself acknowledges the Hawthorne effect, commenting that "workers that are aware that they are being monitored may exhibit more hygienic practices if they wish to show that there is lesser exposure." DRE at 120.

Dr. Markowitz also points out that EPA's analysis of monitoring data for the Maintenance, Renovation, and Demolition COUs is inherently implausible, likely because of heavy reliance on unrepresentative data from OSHA inspections:

> On its face, it is hard to believe that the central tendency estimate for "higher-potential workers" for an 8-hour-TWA is 0.0011 f/cc and the 30-minute exposure concentration for the same worker is 0.025 f/cc (Table Apx E-21. p. 286). Noting in the Executive Summary that EPA states that "EPA's high-end estimates cover those situations where existing regulations do not apply," the values in this OES (0.43 f/cc for 8-hour TWA and 0.16 f/cc for 30-minute short-term concentration. These are relatively modest levels of exposure, especially in view of the presumption that protective measures were likely not taken.

As Dr. Markowitz observes, "most of the remaining literature that is used in this OES are industry-based studies, some in the grey literature. Selected studies have important weaknesses, including those by Lange and colleagues, which are not well documented (see below) and, frankly, replete with prejudicial (non-scientific) statements." Dr. Markowitz elaborates on EPA's reliance on questionable industry studies and omission of more credible studies by independent researchers in his review of EPA's exposure assessment for "Handling articles or formulations that contain asbestos."

> In this OES ("handling articles or formulations that contain asbestos"), Section E.13.4.3 indicates that OSHA data and 3 studies were used to obtain data shown in Table Appendix E-4 (p. 258-260). OSHA CEHD data were used; all 8-hour air samples were all non-detects. The comments on the usefulness of OSHA data above apply. The 3 studies addressing the OES "handling articles or formulations that contain asbestos." were Brorby (2013)[60], Garcia (2018)[61] and Lange (2006)[62]. The Brorby study addresses joint compound; the Garcia study: wire gauze pads, and the Lange study ceiling tile, caulking, floor tile, and roofing materials. Additional details on the study results and air sampling findings are provided in an EPA Risk Part 2 Data Tables.
>
> The use of the 3 selected studies to estimate relevant exposures for this OES is not informative. The Brorby study is a modeling study (note the term in its title "Evaluation of a New Model"), even as the "Strengths, Limitations, Assumptions, and

[60] Brorby, G.P., Sheehan, P.J., Berman, D.W., Bogen, K.T. and Holm, S.E. (2013), Exposures from Chrysotile-Containing Joint Compound: Evaluation of New Model Relating Respirable Dust to Fiber Concentrations. Risk Analysis, 33: 161-176. https://doi.org/10.1111/j.1539-6924.2012.01847.x

[61] Garcia E, Newfang D, Coyle JP, Blake CL, Spencer JW, Burrelli LG, Johnson GT, Harbison RD. Evaluation of airborne asbestos exposure from routine handling of asbestos-containing wire gauze pads in the research laboratory. Regul Toxicol Pharmacol. 2018 Jul;96:135-141. doi: 10.1016/j.yrtph.2018.04.020. Epub 2018 Apr 30. PMID: 29723551.

[62] Lange, J., Sites, S., Mastrangelo, G. et al. Exposure to Airborne Asbestos During Abatement of Ceiling Material, Window Caulking, Floor Tile, and Roofing Material. Bull Environ Contam Toxicol **77**, 718–722 (2006). https://doi.org/10.1007/s00128-006-1122-8.

> Uncertainties" section (p. 303) of the EPA document states that using monitoring data is preferable to using modeling. Why weren't original studies used, such as, for example, joint compound. See the following studies: 1) Rohl et al (1972)[63]; Verma et al (1980)[64]; and Gypsum Association (1973).[65]
>
> The Lange et al study is even more problematic. Sampling of various ACM materials (ceiling tile, caulking, floor tile, and roofing materials) were taken during asbestos abatement activities, and OSHA asbestos regulations were followed, as cited by the authors. The worker tasks and activities are never described. The asbestos content of the ACM materials being abated were never measured, or at least reported. The measurement method (i.e., NIOSH 7400) is not cited. The detection limit of the personal and area sampling method was 0.1 f/cc, i.e., the OSHA TWA level. All of these aspects of the study demonstrate scientific deficiencies or do not represent typical conditions for workers managing legacy asbestos materials. Virtually all sample results were below the level of detection. The authors conclude by advising against use of respirators during asbestos abatement and by supporting the re-introduction of chrysotile in the U.S., which "will likely save many lives in the U.S. and other countries" (p. 720).

Dr. Markowitz also raises concerns about the data EPA uses to assess worker exposure during the COU for "use, repair, or removal of appliances and machinery that contain asbestos," where EPA relies on OSHA CED data, 2 NIOSH HHE's, and 7 studies cited in Table Appendix E-4. This OES describes work with gaskets, packing, industrial brakes and clutches, and reinforced packing. According to Dr. Markowitz, one of the cited studies, Madl (2015)[66] is a gasket and packing simulation study. He asks: "Why not rely on, or at least include, a broader set of studies where conditions of asbestos exposure in use of these materials was characterized? Consider Cheng (1991)[67], Millette (1993)[68], Longo (2002)[69], and McKinnery (1992)."[70]

Dr. Markowitz also comments that:

> The number of workers in this OES may be under-estimated. Only three NAICS codes are included: Petroleum refineries; Other Basic Organic Chemical

[63] Rohl AN et al. Exposure to Asbestos in the Use of Consumer Spackling, Patching, and Taping Compounds. Science 189,551-553(1975).doi:10.1126/science.1145211.
[64] Verma DK, Middleton CG. Occupational exposure to asbestos in the drywall taping process. Am Ind Hyg Assoc J. 1980 Apr;41(4):264-9. doi: 10.1080/15298668091424726. PMID: 7395743.
[65] Gypsum Association. Evaluation of Exposure to Asbestos During Mixing and Sanding of Joint Compounds. November 19, 1973, 12 pp.
[66] Madl AK, Devlin KD, Perez AL, et al. Airborne asbestos exposures associated with gasket and packing replacement: a simulation study of flange and valve repair work and an assessment of exposure variables. Regul Toxicol Pharmacol. 2015 Feb;71(1):35-51. doi: 10.1016/j.yrtph.2014.10.017. Epub 2014 Nov 15. Erratum in: Regul Toxicol Pharmacol. 2017 Aug;88:365-366. doi: 10.1016/j.yrtph.2017.05.004. PMID: 25445297.
[67] Cheng RT, McDermott HJ. Exposure to asbestos from asbestos gaskets. Appl Occup Environ Hyg; 6(7): 1991.
[68] Millette, JR. and Mount, MD. A study determining asbestos fiber release during the removal of valve packing. Applied Occupational and Environmental Hygiene, 8(9), 790-793, 1993.
[69] Longo WE, et al. Fiber release during the removal of asbestos-containing gaskets: a work practice simulation. Appl Occup Environ Hyg; 17(1): 55-62, 2002.
[70] McKinnery WN et al. Evaluation of airborne asbestos fiber levels during removal and installation of valve gaskets and packing. Am Ind Hyg Assoc Jl. 53(8):531-532, 1992.

> Manufacturing, and Industrial Machinery and Equipment Wholesalers. What about other manufacturing industries where workers handle gaskets and packing: paper, petrochemical, other inorganic chemical, pharmaceutical, plastics, etc.? If the belief is that asbestos-containing gaskets and packing are no longer in use, then why include the petrochemical industry? The logic of the NAICS composition of this OES is unclear or should be re-visited.

A related concern raised by Dr. Barry Castleman in communications to ADAO is the extremely low and unrepresentative air concentrations EPA cites for asbestos exposure during removal of vinyl floor tiles. DRE at 66. During removal, patches of the felt often do not come up when old flooring is removed and sanding the floor to eliminate patches of cemented asbestos is a common practice of flooring installers. A 1975 internal corporate memo of former asbestos flooring manufacturer Congoleum (attached to these comments) reported sampling for worker exposure from removal of flooring that entailed "much less sanding than normal." Nonetheless, personal sampling for 19-minute periods recorded exposures of 56.1 and 36.7 f/cc. A 14-minute sample taken after sanding stopped was 12.9 f/cc.[71] This report indicates far higher exposure during tile removal than EPA suggests.

ADAO recommends that EPA reanalyze the monitoring data it is using for its estimates of occupational exposure and risk to (1) draw on a broader universe of studies (going as far back as 1970), (2) give less weight to OSHA and NIOSH data from pre-announced site visits, flawed industry studies and studies in which measured levels reflect the use of PPE and (3) clarify the impact of sampling and analytical methods on the representativeness of the data.

E. EPA Should Address Risks from Asbestos Cement Pipe Used for Drinking Water and Wastewater

According to the draft evaluation, asbestos cement pipes manufactured before the 1980s may contain asbestos concentrations ranging from 12 to 15 percent and continue to be used extensively to convey municipal drinking water and wastewater for sewage treatment. DRE at 277. In 2019, EPA's Office of Air Quality Planning and Standards (OAQPS) gave final approval to an alternative work practice standard for asbestos cement pipe replacement (84 Fed. Reg. 26852) and found that:

> There are over 630,000 miles of Asbestos Cement pipe buried across the United States that have reached or will reach the end of their estimated design and useful lives. Like most of our buried infrastructure, the time has come to renovate or replace these systems.[72]

The Part 2 draft, however, classifies asbestos cement pipes as a low priority for evaluation. DRE at 59. Thus, the draft does not address risks to workers who renovate, maintain or replace pipes, DIY consumers who engage in these activities directly, or consumers of drinking water containing asbestos leaching from cement pipes.

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[71] B Castleman, *Asbestos: Medical and Legal Aspects,* 5th Ed., 2005, p, 351.

[72] U.S. EPA. Guidelines for replacing asbestos cement pipe by close tolerance pipe slurrification. (n.d.). Docket No. EPA-HQ-OAR-2017-0427

Replacement or repair of asbestos cement pipes by workers or consumers releases asbestos to the air and results in inhalation exposure. Reports of bursting AC pipe in Houston, Texas documented substantial inhalation of asbestos fibers by poorly protected workers.[73] This exposure scenario will likely become more prevalent because of the growing need to remove or service asbestos cement pipes but is not addressed in Part 2.

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 life-time 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."
>
> 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.[74]

In 1982, EPA set a maximum contaminant level (MCL) for asbestos in drinking water of 7 million fibers per liter (MFL) based on concern about the risk of developing benign polyps from deterioration of asbestos cement in drinking water infrastructure.[75] 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.[76] 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)."[77]

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[73] In 2012, two alert workers and a television reporter in Houston exposed improperly managed ruptures of AC pipe. The KHOU News 11 investigation estimated that 20% of the City of Houston's water pipe infrastructure was AC pipe. KHOU (2012). Ruptures of pipe resulted in significant inhalation exposure to asbestos because of the lack of adequate protective equipment. https://www.youtube.com/watch?v=4Pjt6Sg_5tw (Additional information available upon request.)

[74] ANSI Blog: Keeping Asbestos Out of Drinking Water https://blog.ansi.org/?p=158120

[75] https://www.epa.gov/ground-water-and-drinking-water/national-primary-drinking-water-regulations

[76] https://www.ewg.org/tapwater/contaminant.php?contamcode=1094

[77] 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)at 164. Asbestos | Toxicological Profile | ATSDR (cdc.gov)

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."[78]

The Part 2 draft notes that "[i]nhalation and ingestion are the main exposure pathways of concern" for asbestos. DRE at 143. It elaborates that the 2012 IARC Monograph "acknowledges that several individual studies show a positive association between ingestion of asbestos via drinking water and stomach and colorectal cancer across several different communities." However, EPA then indicates that "there are studies that did not find an association." DRE at 144. Overall, the Agency declines to conduct quantitative dose-response analysis for ingestion routes of exposure "based on the limited information available for these exposures." Id.

Although the evidence of a causal connection between the presence of asbestos in drinking water and gastrointestinal cancers is not conclusive, there is ample reason for concern about this risk in light of the pervasive use of asbestos cement pipe in drinking water systems. While noting limitations in the data, EPA should treat asbestos ingestion from asbestos cement pipe as a contributor to legacy asbestos risks, thereby assuring that it will be addressed in a comprehensive risk management strategy.

F. Even Where Exposure Data Is Limited, EPA Should Identify All COUs with Exposure Potential as Contributors to the Unreasonable Risk of Legacy Asbestos

As Part 1 emphasizes, the Agency is "now making a single unreasonable risk determination for asbestos as a chemical substance." DRE at 189.[79] Asbestos is particularly well-suited for this "whole chemical" risk determination because it presents unique dangers to human health that cannot be effectively addressed on a use-by-use basis:

- Any level of exposure to asbestos can cause cancer and debilitating non-cancer disease
- The toll of death and disease from asbestos has been massive and remains alarmingly high
- Exposure to asbestos exists at all stages of its life cycle and across multiple pathways of exposure
- The level of risk from asbestos is a function of the cumulative impact of multiple exposure pathways and fiber types, not individual COUs in isolation
- A broad cross-section of the US population is exposed to asbestos, including workers, consumers and the general public
- Unreasonable risk can only be prevented by eliminating all sources of exposure

[78] Id at 65.
[79] DRE at 189.

EPA emphasized these factors in deciding to impose a comprehensive ban in its 1989 TSCA rule. As the Agency recognized, risk management measures that focus on individual COUs in isolation from all pathways of exposure will fail to address the full magnitude of the risks it presents.[80]

However, EPA undermines its whole chemical risk determination by separating legacy asbestos COUs into those that "contribute" to the unreasonable risk and those that it says do not. In the latter category are seven COUs that EPA finds do not meet its risk benchmarks and ten COUs for which it lacks "sufficient information" to make quantitative determinations of risk. DRE at 190-191.

COUs in these categories include products for industrial and commercial use, as well as consumer products such as children's toys, hobby materials, ceramics, automotive applications, textiles, ceramics and garden products. While EPA may lack the evidence to show that use of one of these products individually exceeds its numerical cancer risk benchmarks, it is universally recognized that there is no safe level of exposure to asbestos and no asbestos-container products have zero-risk. Moreover, most exposure to asbestos occurs by multiple pathways and a COU that is low risk in isolation may contribute to a significant risk when combined with other sources of exposure. For example, while EPA concludes that asbestos-containing crayons are low risk, DRE at 200-201, these products are broken, mouthed and swallowed when children play with them, and children are not only uniquely susceptible to carcinogens but may have multiple other sources of asbestos exposure.

Thus, we strongly urge EPA **not** to identify COUs that do not contribute to the whole chemical unreasonable risk of asbestos but to include them in its risk determination if there is evidence of potential exposure. This will avoid narrowing risk management options unnecessarily and enable EPA to consider a broad range of effective risk reduction strategies during its rulemaking process.

G. Except for Fires, EPA Ignores Asbestos Exposure from Disasters and Emergency Events

Although firefighters are identified in Part 2 as a highly exposed population, the draft provides limited information on explosions, floods, rail derailments, collapsed buildings and other emergency events that result in uncontrolled asbestos releases and spikes in exposure that present elevated risks to emergency responders, bystanders and the general public. [81] Given the frequency of disasters and their likely increase in the future due to climate change and resulting extreme weather events, this is a troubling omission. An increase in extreme weather events will result in more damage and destruction of ACM-containing structures, releasing more asbestos into the air and increasing risks to the public and clean-up and emergency response workers.

A well-studied disaster resulting in widespread asbestos release was the 2001 attack on the New York World Trade Center (WTC).[82] 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

[80] 54 Fed. Reg. 29460 (July 12, 2989).
[81] Federal Emergency Management Agency. (August 2011). Climate Change: Long Term Trends and their Implications for Emergency Management. https://www.fema.gov/pdf/about/programs/oppa/climate_change_paper.pdf
[82] P. J. Landrigan *et al.*, "Health and environmental consequ nter disaster," *Environmental Health Perspectives,* vol. 112, no. 6, pp. 731-739, May 2004.

settled dust at and around Ground Zero had concentrations ranging between 0.8 and 3.0%. 5000 tons of ACBMs [Asbestos Containing Building Materials] were released during the WTC collapse, and the amount of asbestos fibers in the air was 555 times greater than the permissible level."[83]

While the WTC collapse was an exceptional event, comments submitted to the Part 2 docket by the American Public Health Association (APHA) cite two other examples of significant disaster-related asbestos exposure:

- The wildfires on Maui, Hawaii in August 2023 killed more than 100 people, and contributed to many more injured and displaced. Among the consequences was debris and fire ash containing asbestos.[84,85]
- Demolition of a 55-year-old waterfront hotel in Punta Gorda, Florida was put on hold in March 2024 after the city learned of the asbestos-containing building materials.[86,87] The hotel was severely damaged in 2022 by Hurricane Ian and further damaged by Hurricane Idalia in 2023. For 18 months while the property was closed for business, the public, including trespassers and vandals, were potentially exposed to asbestos.[88]

According to APHA, fires and explosions at industrial facilities also result in large asbestos releases. For example:

- A fire on April 11, 2023, at a recycling facility in Richmond, Indiana sent plumes of smoke through downwind communities. More than 2,000 near-by residents were ordered to evacuate.[89] On April 13, 2023, EPA confirmed the presence of asbestos-containing material in debris from the incident.[90] The agency was involved in hazardous waste clean-up and testing in the community for 11

[83] Y. C. Kim and W. H. Hong, "Optimal management program for asbestos containing materials to be available in the event of a disaster," *Waste Management,* vol. 64, pp. 272-285, Jun 2017.
[84] McAvoy AB. (August 30, 2023) Unclear how many in Lahaina lost lives as Hawaii authorities near the end of their search for dead. Associated Press.
[85] U.S. EPA. Maui Wildfires. Hazardous materials removal (Phase I). (Last updated March 27, 2024) https://www.epa.gov/maui-wildfires/hazardous-materials-removal-phase-1
[86] Orenczuk A. (2024 April 2). "This is a crazy eyesore!"…and asbestos just delayed demolition in Punta Gorda. Fox4 Southwest Florida. https://www.fox4now.com/punta-gorda/this-is-a-crazy-eyesore-and-asbestos-just-delayed-demolition-in-punta-gorda
[87] WINK News. (2024 May). What's next for Punta Gorda hotel with asbestos. [YouTube] https://www.youtube.com/watch?v=ZTEPf6yw94g
[88] Murray D. (2023 July 19). Punta Gorda Waterfront Hotel undergoes partial demolition process. Fox4 Southwest Florida. https://www.fox4now.com/news/local-news/lee-county/punta-gorda-waterfront-hotel-undergoes-partial-demolition-process
[89] Jeong A. (April 12, 2023). Toxic smoke from Indiana industrial fire forces evacuation order for 2,000. Washington Post. https://www.washingtonpost.com/nation/2023/04/11/indiana-fire-evacuations-richmond/
[90] Vespa M, Guevara S, Li DK. (April 13, 2023). Residents near Indiana plastics fire report snowlike debris falling and a taste of chemicals in the air. NBC News. https://www.nbcnews.com/news/us-news/carcinogen-asbestos-in-debris-from-indiana-plastics-fire-rcna79556

months.[91] The disaster clean-up included removal of about 6,000 tons of fire debris including suspected asbestos-containing materials.[92]

- A 2021 warehouse fire in Austin, TX billowed smoke from a structure containing significant amounts of asbestos.[93] More than 100 firefighters responded to the event and were potentially exposed to it, as were many community members, vehicles and structures downwind.

It is not defensible for EPA to disregard releases of asbestos from disaster and emergencies as "unique" events outside the scope of TSCA risk evaluations when they are both foreseeable and expected and result in widespread and harmful exposure.

Dr. Markowitz explains in his comments to the Part 2 docket that, in addition to firefighters and other trained emergency responders, cleanup and remedial work following floods or other disasters typically involves untrained, short-term workers. In NYC, after the World Trade Center collapse and after Hurricane Sandy, there were large numbers of short-term clean-up workers, usually immigrant day laborers. Their work is clean-up and small-scale demolition and doesn't resemble the work of firefighters. The tasks they perform are usually considered unskilled. They rarely have personal protection or are informed about the hazards. They frequently are hired by transient clean-up companies that may not be well-characterized in the NAICS system. This population should at a minimum be described and presumed to have high end exposures for the tasks that they perform in the Part 2 evaluation.

H. EPA's Assessment of Exposure and Risk from Legacy Asbestos Waste is Incomplete and Inadequate for Risk Management

According to the draft evaluation, "EPA expects releases to occur during waste handling, disposal, and treatment." DRE at 307. The amount of waste generated that includes legacy asbestos is enormous. EPA estimates that 188,800,000 tons of Construction and Demolition (C&D) waste were disposed of in 2018 and that as many as 21,399 commercial demolition sites and 25,390 residential demolition sites may generate asbestos waste. DRE at 279. As EPA explains, "[n]on-friable asbestos waste is treated as either construction and demolition or municipal solid waste and can be disposed of in a municipal landfill. Friable asbestos waste is considered a 'non-RCRA' hazardous waste and is not subject to RCRA subtitle C regulation and can be disposed in a municipal landfill but special requirements for containerization, transportation, recordkeeping and disposal are needed." Id. at 306. The Agency also notes that asbestos waste can be incinerated or recycled. EPA's upper-bound estimate of the number of establishments involved in waste handling, disposal, and treatment of asbestos is 4,972. Id. at 307. It estimates that workers potentially exposed to asbestos during waste disposal activities may total 73,000 (id at 309) although this figure does not include the many construction and demolition workers who handle asbestos waste at job sites before disposal occurs.

[91] U.S. EPA. My Way Trading Warehouse Fire. On-Scene Coordinator updates. (Last update March 20, 2024). https://response.epa.gov/site/site_profile.aspx?site_id=16003

[92] City of Richmond. City of Richmond (IN) update and statement marking one year anniversary after My Way Trading Warehouse fire. News release, April 10, 2024. https://www.richmondindiana.gov/news/www-richmondindiana-gov-news

[93] Barer D, Travis A. (May 26, 2021). New details in blaze the potentially exposed over 100 firefighters to asbestos. https://www.kxan.com/investigations/new-details-in-blaze-that-potentially-exposed-over-100-firefighters-to-asbestos/

EPA's only source of information on the volume of asbestos-containing waste generated, treated and disposed of nationally is its TRI database but TRI reports account for a very small percent of all asbestos-containing waste. For example, EPA acknowledges that "TRI data are self-reported, [] have reporting requirements that exclude certain facilities from reporting," only cover friable asbestos and require reporting only where certain volume thresholds are exceeded. Id. at 308-309.The Part 2 draft also lacks any breakdown of the types of disposal sites (municipal, Subtitle C, incineration and recycling) receiving asbestos waste and their locations and thus is unable to address the proximity of these sites to environment justice communities. The draft also does not discuss, even in general terms, conditions and management practices at these facilities and the potential for offsite releases of asbestos-containing dust that result in exposure by nearby residents or bystanders. Thus, the contribution of waste sites to general population exposure to asbestos cannot be determined.

Also absent from the Part 2 draft is any information about asbestos found at Superfund cleanup sites under the Comprehensive Environmental Response, Compensation and Liability Act of 1980 (CERCLA). In September 2023, there were approximately 1,336 Superfund sites on the National Priority List (NPL) and 16 of them contained asbestos hazards.[94] ATSDR also reports that asbestos has been identified in at least 83 of additional waste sites screened for inclusion on the NPL.[95] At several of these sites, releases of asbestos are ongoing and it has been detected in air, groundwater or surface water, creating a potential exposure pathway for nearby communities.

The Part 2 draft analyzes limited monitoring data for workers involved in disposal activities (DRE at 310-312) but acknowledges that this "data does not include process information or worker activities," making it difficult to determine exposure levels for the large number of workers engaged in different facets of waste shipment, treatment and disposal operations.

EPA identifies *Disposal – distribution for disposal* as a COU contributing to unreasonable risk. DRE at 190. However, the disposal activities falling within this determination are not defined, creating uncertainty about the scope of risk management necessary for legacy asbestos disposal, and EPA does not address whether releases from disposal sites contribute to unreasonable risks to the general population.

ADAO is also concerned by EPA's assertions that "the thermal destruction of asbestos results in morphological changes resulting in the formation of non-asbestos fibers (such as forsterite, amorphous silica, and enstatite during the recrystallization process)" and that "very low to non- detectable concentrations of asbestos fibers released to air have been reported during incineration processes." DRE at 46. The reference EPA relies on is a single report which does not account for all the asbestos that goes to incinerators, where combustion temperatures are not uniform and where some asbestos can pass through and be released to the air unaltered.

In its final evaluation, EPA should clarify that it is not suggesting that incineration – which is a broad category that applies to many different processes – is a safe disposal method for asbestos waste in all instances. EPA should also make clear that make clear that asbestos-containing materials are not destroyed during house or other structural fires or other high-temperature conditions lest readers get the mistaken impression that combustion renders asbestos harmless.

[94] Asbestos Superfund Sites | A Clean Up Guide, https://wwwn.cdc.gov/TSP/ToxProfiles/ToxProfiles.aspx?id=30&tid=4n-Up Guide.
[95] Tox Profile at 149.

III. EPA Must Do More to Address the Risks of Legacy Asbestos to Vulnerable Populations

Because legacy asbestos has so many COUs and there are so many exposed population segments, significant variations in exposure and risk exist across the exposure universe. TSCA requires EPA to account for these variations and to give particular attention to subpopulations at greater risk because of higher susceptibility and/or exposure (PESSs). Subpopulations exposed to asbestos from multiple COUs and pathways often are at greater risk from higher exposure and should be treated as PESSs. This is also the case with populations with unique characteristics that increase the likelihood of non-cancer disease and cancer. EPA must not only identify such susceptible subpopulations but specifically address whether they face unreasonable risks and how large they are.

The Part 1 evaluation takes positive initial steps toward protecting vulnerable populations but needs to be significantly strengthened.

A. The Omission of an Environmental Justice Analysis is a Serious Flaw in Part 2

For this Administration, environmental justice concerns converge with TSCA's emphasis on protecting vulnerable populations because disproportionate risks from chemical exposure often impact disadvantaged communities of color.

Surprisingly, however, there is no environmental justice analysis in the draft evaluation even though there are many indicators that disadvantaged frontline communities have disproportionate exposure to legacy asbestos. These communities are often located in close proximity to landfills and other disposal sites that treat or manage asbestos waste as well as older industrial facilities which contain asbestos insulation, gaskets, flooring, cement pipes and other ACMs. They also have abandoned or substandard housing, schools and other structures containing poorly maintained and unsafe legacy asbestos. And their residents often have high levels of asthma and other health conditions that can be aggravated by asbestos exposure or predispose them to asbestos health effects. These are all factors that point to the importance of an environmental justice analysis and EPA should include such an analysis in the final evaluation.

B. EPA Identification of PESSs and Risk Analyses for these Populations Are Inadequate and Incomplete

As required by TSCA, the draft identifies a small number of possible PESS, including occupational exposures, children, individuals who are exposed through DIY activity, and those who smoke. DRE at 178. However, for nearly all of the subpopulations EPA considers, Table 5-26 concludes that only two -- smokers and children – are more susceptible to the health effects of asbestos than the general population. Even for these groups, moreover, EPA makes no attempt to quantify the increase in risk resulting from higher susceptibility.

As an example, EPA asserts that "evidence is not sufficient to quantitatively estimate risk for smokers separate from the general population." But epidemiology studies demonstrating higher risks to smokers provide information about the magnitude of this higher risk. 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 that included smoking ($p_{trend} = <$ 0.0001.[96] 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.[97] Risk was significantly higher in

those with asbestosis (ILO 1980 profusion ≥ 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 could use the RR differences between smokers and non-smokers to calculate the higher incidence of lung cancer in smokers, increase its lung cancer risk estimate for this group accordingly and then determine the percentage of smokers in the asbestos-exposed population. Or it could simply apply a 10X uncertainty factor (UF) to account for the higher risk to smokers per standard EPA IRIS methodology.[98]

Similarly, EPA acknowledges that "[e]pidemiologic evidence has demonstrated that time since first exposure is a key predictor in asbestos-related disease" and therefore "exposures during childhood are associated with greater risk." Yet the draft evaluation makes no effort to define and quantify the segment of the population with early-life exposure to asbestos or to adjust risk estimates to account for this higher susceptibility. Such an analysis would not be difficult: the presence of elevated asbestos levels in many schools indicates that a large portion of the student population has asbestos exposure at a young age and children can be expected to be exposed to asbestos in homes where DIY projects are conducted or contaminated take-home clothing is laundered. Moreover, 2005 EPA guidance recommends increasing quantitative estimates of cancer risk to children by factors of 3X or 10X because risks "generally are higher from early-life exposure than from similar exposure durations later in life."[99] Yet there are no such adjustments in the draft Part 2 evaluation.

EPA also rejects classifying populations with pre-existing disease as a PESS because it "did not identify pre-existing disease factors that are associated with increased susceptibility." Again, the evidence points in a different direction. Nonmalignant asbestos-related disease is important as a marker of increased risk for lung cancer.[100] 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 ≥ 1/0 (odds ratio (OR) 2.03, 95% CI 1.00-4.13).[101] Based on these data, populations with non-cancer asbestos disease should be a PESS for lung cancer because of their higher susceptibility.

Risk for asbestos-related lung cancer and non-cancer lung disease is also increased by a number of other variables, including family, preexisting Chronic Obstructive Pulmonary Disease (COPD) and occupational or environmental exposure to other lung carcinogens (e.g., silica, radon). EPA doesn't consider these

[96] Cullen MR, Barnett MJ, Balmes JR, Cartmel B, Redlich C, Brodkin CA, et al. Predictors of lung cancer among asbestos-exposed men in the β-carotene and retinol efficacy trial. Am J Epidemiol 2005;161(3):260-270.

[97] Markowitz SB, Levin SM, Miiller A, Morabia A. Asbestos, asbestosis, smoking, and lung cancer. New findings from the North American insulator cohort. Am J Respir Crit Care Med 2013;188(1):90-96.

[98] https://www.epa.gov/sites/default/files/2014-12/documents/rfd-final.pdf.

[99] https://www3.epa.gov/airtoxics/childrens_supplement_final.pdf.

[100] Wilkinson P, Hansell DM, Janssens J, Rubens M, Rudd RM, Taylor AN, McDonald C. Is lung cancer associated with asbestos exposure when there are no small opacities on the chest radiograph. Lancet 1995;345:1074-1078;Cullen MR, Barnett MJ, Balmes JR, Cartmel B, Redlich C, Brodkin CA, et al. Predictors of lung cancer among asbestos-exposed men in the β-carotene and retinol efficacy trial. Am J Epidemiol 2005;161(3):260-270.

[101] Hlllerdal G. Pleural plaques and risk for bronchial carcinoma and mesothelioma. Chest 1994;105:144-150.

factors in its PESS analysis although a number of them (together with smoking) are more prevalent in environmental justice communities than the general population.

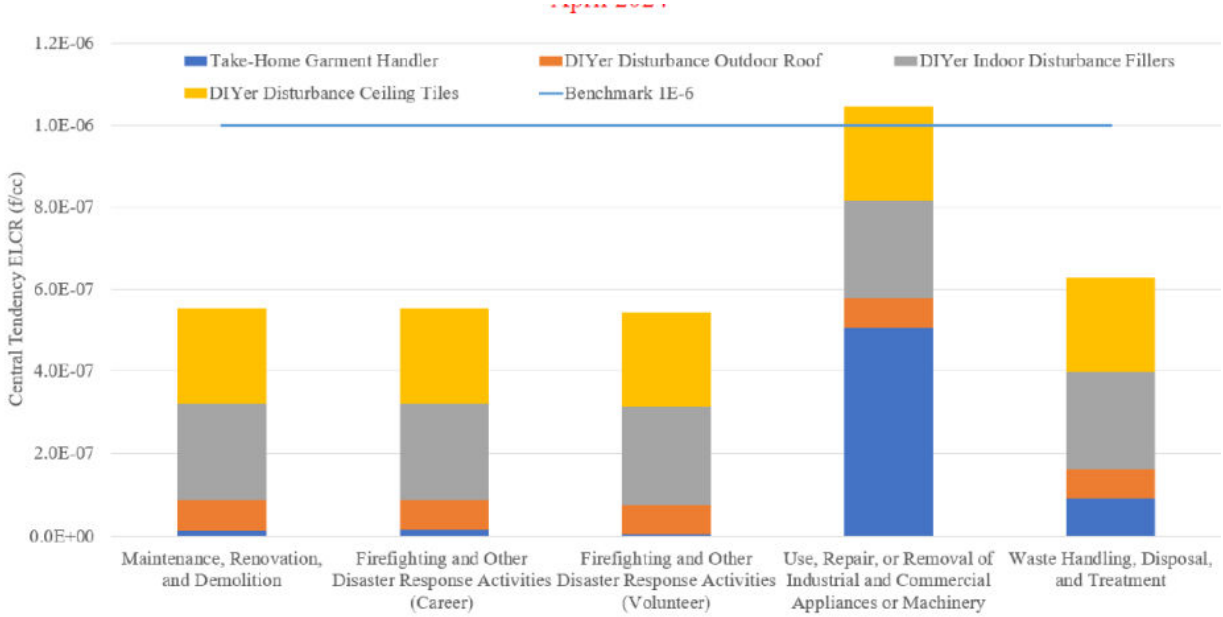
C. EPA's Aggregate Exposure Analysis is a Positive Step but its Scope Is Too Narrow

Aggregate exposure analysis is an important tool to assure that risk evaluations account for and quantify the higher risks to subpopulations with exposure from multiple sources and pathways. TSCA recognizes the benefits of this tool, and it can play a critical role in identifying and addressing subpopulations which are PESSs because of elevated levels of exposure.

EPA's inclusion of aggregate exposure analysis in Part 1 is a welcome recognition of the multiple sources of exposure to asbestos by a large segment of the population. EPA correctly recognizes that:

> A worker may be involved in multiple activities aside from their work requirements that exposes them to asbestos that have varying occupational exposures. DIYers may perform multiple projects that release and exposes them to asbestos fibers. Take-home exposures can occur to workers and DIYers as they handle asbestos-contaminated clothing and do non-occupational renovation activities.

DRE at 396. As EPA depicts the convergence of these sources:



Figure_Apx M-1. Central Tendency Lifetime Cancer Risk Aggregation across Take-Home and DIY Scenarios

However, these are not the only sources that contribute to aggregate exposures. As an example, EPA does not address the aggregate asbestos exposure of persons who occupy residential buildings and schools, breathe asbestos in outdoor air and have elevated exposure to asbestos in communities impacted by waste transport, handling, treatment and disposal.

In addition, EPA acknowledges that cancer risks were only "aggregated across scenarios if the [cancer risk] values for each scenario are *below the non-occupational benchmark* (1×10^{-6} f/cc)" and, as a result, "very few [cancer risk] values can be used in this aggregate analysis." DRE at 396 (emphasis added). Similarly, EPA notes that its aggregate analysis of non-cancer risks was only for DIY consumers and "[t]he majority of the high-end DIY scenarios resulted in MOE values over the benchmark and are not used in the aggregation so very few activities are aggregated." Id. at 399.

EPA's decision to impose these constraints on its analysis of aggregate exposure are not explained and resulted in an incomplete and limited understanding of the combined impact of multiple exposure scenarios on overall risk. It is important for EPA to address aggregate exposures across COUs that *exceed risk benchmarks* so that higher risk populations are clearly identified and all sources of risk to these groups are accounted for. In addition, EPA should provide a *qualitative* assessment of the potential contribution to aggregate risk of COUs for which it lacked sufficient data to generate risk estimates; failing to recognize that these COUs may add to total exposures and thereby elevate risks to certain subpopulations could result in inadequate protection during risk management.

IV. EPA's Dismissal of the Presence of Asbestos in Talc is Irresponsible and Ignores Extensive Evidence of the Risks to Consumers and Workers from Asbestos-contaminated Talc

EPA acknowledges that talc and other mineral formations are known to be contaminated with asbestos (DRE at 304) but claims that "exposure is unlikely" because "selective mining practices occur and are successful in generally avoiding deposits that are likely to contain asbestos minerals." On this basis, EPA says "it will not conduct any further analysis of this COU in this draft risk evaluation." DRE at 306.

This cursory dismissal – without any analysis or explanation -- of an issue that has raised high-profile public health concern in the media and tort litigation and at the Food and Drug Administration (FDA) is reckless and irresponsible. In its review of the Part 1 risk evaluation, the SACC emphasized the importance of contaminated talc as an exposure pathway for asbestos and ADAO and other parties submitted extensive comments describing the links between asbestos in talc and serious health effects. EPA's failure to address asbestos in talc-based products also received negative scrutiny in *Asbestos Disease Awareness Org. v. Wheeler*, 508 F. Supp. 3d 707 (N.D. Cal. 2020), which in turn was a major driver for including talc contamination in EPA's recent asbestos reporting rule under TSCA. In light of this lengthy history, it is flabbergasting and disappointing that EPA would give short shrift to such a central issue

Dr. Jacqueline Moline, currently the Chairperson of the Department of Occupational Medicine, Epidemiology and Prevention at the Donald & Barbara Zucker School of Medicine at Hofstra University/Northwell Health, is a recognized expert on the causal link between asbestos in talc and mesothelioma and ovarian cancer. She submitted a detailed statement on this issue as part of the public comment process for EPA's draft Part 1 evaluation; this statement was apparently ignored by EPA. Dr. Moline is now submitting an additional statement to the docket in response to the draft Part 2 evaluation. Her conclusions are that:

> *The position that that "exposure is unlikely" because "selective mining practices occur and are successful in generally avoiding deposits that are likely to contain asbestos minerals." is not defensible* as there is no reliable means to selectively mine to avoid all asbestos given contexts of the mineralogy, inadequate basic analytical techniques used in industry, the limitations of the human eye, and the physical nature of talc ore. That the USEPA acknowledges that talc and other mineral formations are known to be contaminated with

> asbestos but claims that "exposure is unlikely" because of "selective mining" is incognizant of the geological contexts of mineralization and practices of mining of talc. Furthermore, exposure has been shown to occur following use of talc mined at levels that are associated with subsequent disease. Based on the geological and mineralogical data, along with exposure assessments, I respectfully request that this COU be considered as the hazard it represents. (Emphasis added).

A. There Is Extensive Evidence of the Presence of Asbestos in Talc.

As in her 2020 statement, Dr, Moline emphasizes that talc is commonly associated with many asbestos minerals.

> It should be understood that many asbestos minerals are recognized to occur in talc deposits and derived materials and in associated lithologies (Winter 2001; Chernoskey et al., 1988, Evans and Guggenheim 1988). For example, chrysotile can be stable over a wide range of geologic conditions. Evans (2004) provides a summary of these conditions and comments on the stability of serpentine minerals, synthesizing an extensive literature on the subject. These general conditions are also commensurate with many of the conditions of talc formation (e.g., Chernoskey et al., 1988, Evans and Guggenheim 1988). Tremolite also is well recognized to be associated with talc (e.g., Winter 2001) as is anthophyllite (e.g., Evans and Guggenheim 1988). Furthermore, it is well recognized that metamorphic systems are dynamic geologically, with temperature, pressure, fluid, and structural conditions varying over long periods of geologic time (e.g., Winter 2001). Mineral reactions under these varying conditions may also be incomplete, meaning there may not be enough geologic time, or a sustained amount of a geologic factor or chemical component to drive the reaction to completion, as discussed in Deer, Howie and Zussman (2013). Reactions can also progress for some period and then revert to other minerals such as talc to the asbestiform mineral chrysotile, or talc may react to form tremolite (Winter 2021) or anthophyllite (Evans and Guggenheim, 1988) because of changes in geologic conditions such as temperature, concentrations of CO_2 and other kinetic factors difficult to define.

B. Common Industry Techniques Cannot Reliably Detect Asbestos in Talc

Dr. Moline also cites the December 2021 IWGACP White Paper, a consensus document on asbestos in talc-based consumer products authored by over 30 experts from 8 federal agencies (including 6 from the EPA).[102] Based on this report and other information, Dr. Moline emphasizes that "[a]sbestos cannot be reliably detected in talc materials by techniques commonly used in industry" and that "[a]sbestos and talc particles can be intimately mixed at scales not observable by the unaided eye and hand sorting or visual inspection is wholly unreliable as a detection method." As she summarizes, "'selective mining', even when supported with common industry analytical work, cannot reliably assure asbestos is avoided in the mining process."

[102] FDA / IWGACP (2021) White Paper: IWGACP Scientific Opinions on Testing Methods For Asbestos In Cosmetic Products Containing Talc. Interagency Working Group on Asbestos in Consumer Products.

C. There is Extensive Documentation of the Harmful Effects of Asbestos in Talc

EPA makes no mention of the extensive documentation of the adverse health impacts of asbestos in talc despite the detailed discussion of this evidence in Dr. Moline's 2020 statement and ADAO's 2020 comments. We are summarizing the key points below in the hope that they are not again overlooked by the Agency.

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."[103] 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."[104] It was also found that these talc miners had an excessive death rate from cancers of the lung and pleura.[105]

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.[106] 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.[107] 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.[108] 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 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).109 Tests of other sources of talc have yielded similar results.

Recently, studies by FDA and others have documented the presence of asbestos in a variety of personal care products and cosmetics.[110] 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.[111]

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.[112] 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.[113] 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 and ovarian cancer have found elevated cancer risk, particularly for the most common type of ovarian cancer, serous carcinoma of the ovary.[114]

[103] 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.

[104] 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

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

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

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

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

[109] 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).

[110] 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

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

[112] 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.

[113] 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.

[114] 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.

D. EPA Ignores Evidence of Talc in TSCA-regulated Consumer and Industrial Products

According to Dr. Moline, "EPA's determination that exposure to asbestos from mining talc does not occur fails to take into account peer-reviewed studies that have demonstrated that there is asbestos in consumer products, such as cosmetic talc." In its 2020 comments, ADAO identified several studies finding asbestos in imported talc-based consumer products subject to TSCA:

- In 2000, the Seattle Post Intelligencer confirmed that asbestos had been found in crayons.[115]
- In 2007, the ADAO's product testing confirmed asbestos in five consumer products, including a child's toy.[116]
- 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.[117]
- 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.[118]

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

The Part 2 evaluation acknowledges that "[r]elevant uses of imported talc products that may contain asbestos (*i.e.,* fillers and putties with talc containing asbestos and crayons with talc containing asbestos) were also considered, but there was no reasonably available information identified to provide evidence that import of these products is ongoing." DRE at 17. This claim rings hollow because there is no indication that EPA tested imported talc consumer products or took any other step to obtain "reasonably available information" about them. Moreover, based on the history of finding asbestos in imported talc products, EPA should have treated this COU as "reasonably foreseen" under TSCA and assessed its risks in Part 2.

The Part 2 evaluation does not acknowledge or address the many products derived from domestic or imported talc that are used for industrial purposes yet ADAO discussed these products in detail in its Part 1 comments. According to Geology.com,[119] 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.

- **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. 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 its Part 2 asbestos evaluation.

V. EPA's Analysis of Cancer and Non-Cancer Health Effects Generally Has a Sound Scientific Basis and Does Not Warrant Major Changes

Part 2 provides a limited discussion of the extensive literature on asbestos' cancer and non-cancer effects and draws heavily on the analysis in Part 1. Consistent with the authoritative conclusions of many expert bodies, EPA finds strong evidence that lung cancer, mesothelioma, laryngeal cancer and ovarian cancer are causally related to asbestos exposure. DRE 141-142. EPA concludes that there is suggestive but inconclusive evidence of a causal connection to colorectal cancer. EPA also determines that asbestos exposure has been demonstrated to have adverse non-cancer effects on the respiratory system, including asbestosis, non-malignant respiratory disease (NMRD), pulmonary function impairments, diffuse pleural thickening (DPT), and pleural plaques. Id. at 143-143.

According to EPA, there is evidence of immunological and lymphoreticular effects, but it is inconclusive.

ADAO agrees that, in general, EPA's identification of asbestos's cancer and non-cancer effects is supported by the weight of evidence and aligned with the many previous determinations by leading experts and public health bodies.

A. EPA Properly Selected an IUR that Treated All Fibers as Equivalent in Toxicity

The most challenging task EPA faced was selecting epidemiology studies for dose-response assessment and using them to determine an inhalation unit risk (IUR) for carcinogenicity for the derivation of quantitative risk estimates for legacy asbestos COUs. EPA presented its approach in a White Paper entitled

[115] https://www.cpsc.gov/PageFiles/108033/crayons.pdf
[116] https:/www.asbestosdiseaseawareness.org/archives/364
[117] https://www.ewg.org/release/alert-tests-find-high-levels-asbestos-children-s-makeup-kit
[118] https://uspirg.org/blogs/blog/usp/back-school-asbestos-crayons
[119] https://geology.com/minerals/talc.shtml

Quantitative Human Health Approach to be Applied in the Risk Evaluation for Asbestos Part 2—Supplemental Evaluation including Legacy Uses and Associated Disposals of Asbestos. The epi studies reviewed in the White Paper were similar to those assessed for Part 1 although the focus was on all six fiber types rather than chrysotile alone. The IUR selected in the White Paper was 0.2 per fiber/cc (within the ballpark of the Part 1 IUR).

On October 25, 2023, EPA convened a letter peer review of the White Paper and the seven reviewers responded to EPA's charge questions, which were compiled and released to the public on December 28, 2023.[120] Despite reservations, most of the reviewers did not question EPA's approach to IUR selection, which posited that the six asbestos fibers had equivalent carcinogenic effects. One reviewer, however, objected, maintaining that "it is almost inconceivable that the Agency is suggesting a single IUR for all four fiber types (chrysotile, amosite, tremolite, and crocidolite)" and there "are a myriad of epidemiology studies, toxicology studies, and evaluations of chemical/physical properties . . . that clearly show that these different fiber types have dramatically different cancer potencies (both lung cancer and mesothelioma)."

ADAO scientists strongly disagree with this comment and oppose differentiating between fiber types on the basis of relative carcinogenic potency.

The 1988 IRIS assessment established an IUR of 2.3E-1 per (f/mL) for lung cancer and mesothelioma.[121] 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.[122] 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.[123] OSHA reached the same conclusion in 1986 and 1994.

As explained by leading experts, the IRIS IUR was derived using a dose response model that assumed "equal potency for chrysotile and the amphiboles; equal potency for all fibers longer than 5 mm; and no threshold exposure level for carcinogenicity." [124] The IRIS summary indicated that:[125]

> 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.

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[120] https://www.epa.gov/chemicals-under-tsca/epa-releases-peer-review-comments-white-paper-asbestos-part-2-risk-evaluation.

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

[122] 51 Fed. Reg. 22612 (1986).

[123] 54 Fed. Reg. 29467. (1989)

[124] 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.)

[125] Id at 9.

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."[126] 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."[127] 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.[128]

During the 2008 SAB review, comments by Dr. Mitchell Silverstein and 83 other experts maintained that: [129]

> There 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 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 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." [130]

Consistent with this long history, EPA should categorically reject proposals to set separate IURs for individual asbestos fibers based on claimed differences in toxicity and stick with a single IUR for all six fibers.

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[126] 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).
[127] Id.
[128] Letter from Stephen L. Johnson, EPA Administrator, to Dr. Agnes Kane, Chair of Science Advisory Board Asbestos Committee, December 29, 2008.
[129] 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.
[130] Silverstein et al, at 856.

We do not agree, however, that EPA's proposed IUR of 0.2 per fiber/cc is the best choice of the three options EPA considered in the White Paper. These options were:

- 1988 IRIS Asbestos risk (largely based on EPA (Nicholson) 1986 Asbestos Update) = 0.23, (accounting for all asbestos fiber types); this is based on 14 epidemiologic studies that include occupational exposure to amphiboles, chrysotile, and mixed fibers, depending on the study.
- 2014 Libby-LAA Asbestos Risk = 0.17 (LAA).
- 2020 EPA PART 1 Asbestos Risk Assessment = 0.16 (for chrysotile only).

Several of the letter reviewers preferred the first IUR. As one reviewer explained: "Using 0.23 as the IUR would be the most justifiable method, because it would reflect a risk estimate for a significant number of people (workers and others) who have mixed fiber type legacy asbestos exposures, and is based on actual calculations, not 'rounded.'" ADAO agrees with this logic.

One reason a higher IUR is preferable is that the IUR is only based on dose-response information for lung cancer and mesothelioma and does not factor in laryngeal and ovarian cancer despite clear evidence of causality. Accordingly, the IUR may not be fully protective for all types of cancers related to asbestos exposure. A higher IUR may not fully address this concern but would provide a small increment of added protection.

B. EPA's Quantitative Risk Determinations for Non-Cancer Effects Are an Important Step Forward

In its Part 1 evaluation, EPA did not perform any quantitative risk modeling for non-cancer effects. This was a weakness in Part 1 and ADAO is pleased that EPA is taking a different approach in Part 2. Largely relying on its Libby Amphibole IRIS assessment, EPA proposes to adopts a Point of Departure (POD) for non-cancer effects of 2.6×10^{-2} fiber/cc based on dose-response data in the occupational cohort from the O.M. Scott plant in Marysville, OH described by Lockey et al. (1984) and followed up by Rohs et al. (2008). EPA then applies a combined uncertainty factor of 300 to derive a benchmark Margin of Exposure (MOE) to determine whether measured exposure levels for different COUs present an unreasonable risk of asbestosis and other non-cancer lung diseases. This is the first time EPA has made quantitative non-cancer risk determinations for all asbestos fibers and it adds an important dimension to its risk analysis. While EPA explains the basis for its combined of 300, a greater UF would be defensible to take into account variability in susceptibility to asbestos' non-cancer effects and lack of dose response data for certain endpoints of concern.

CONCLUSION

ADAO is pleased to submit these comments to EPA and looks forward to working with the Agency to address the risks of legacy asbestos under TSCA.

Respectfully submitted,

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