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EPN Comments on the (Chrysotile) Asbestos draft Risk Evaluation
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The Environmental Protection Network (EPN) is an organization comprised of more than 500 U.S. Environmental Protection Agency (EPA) alumni volunteering their time to protect the integrity of EPA, human health, and the environment. We harness the expertise of former EPA career staff and confirmation-level appointees to provide an informed and rigorous defense against current administration efforts to undermine public health and environmental protections.

EPN is submitting these comments to the Science Advisory Committee on Chemicals (SACC) to aid in their review of the asbestos draft risk evaluation. We look forward to the rescheduling of the SACC meeting so that experts on asbestos can offer their feedback on this important risk evaluation.

Risk Characterization, Risk Determination, Risk Management

Mixed results have occurred when EPA has attempted to mitigate risk to asbestos under Toxic Substances Control Act (TSCA). EPA promulgated the Asbestos Ban and Phase Out Rule (ABPR) in 1989. This rule was largely vacated shortly thereafter. The most recent action came in April 2019 when EPA finalized an Asbestos Significant New Use Rule (SNUR) under TSCA Section 5, which prohibits manufacturing (including importing) or processing of discontinued uses of asbestos from restarting without EPA having an opportunity to evaluate each intended use for risks to health and the environment, and to take any necessary regulatory action, which may include a prohibition. Given that this does not represent a permanent ban, the possibility exists that importing, processing or manufacturing as well as discontinued uses could be approved in the future.

We have known for decades that asbestos is a human carcinogen. Many thousands of people have been sickened and have died from exposure to it. As of March 2019, 66 countries have banned the use of asbestos, including all members of the European Union, but not the U.S. An additional 10 nations are placing restrictions on its use. This draft risk evaluation concludes that every condition of use evaluated poses an unreasonable risk to public health, in both the occupational (workers and occupational non-users (ONUs)) and consumer (users and bystanders) setting. *It is time to proceed directly to rulemaking* with a proposal to BAN the importation, manufacture, processing, distribution and use of asbestos for all commercial and consumer uses in the U.S. on an expedited timeline. Over 30 years have passed since the 1989 rule failed, during which alternatives could have been developed. There is no excuse for delaying action any further.

1. **Human Health Hazard Assessment**

   a. Scope, Systematic Review, and Study Selection
   EPA repeatedly presents their rationale for narrowing the focus of the human health assessment to the observation of increased incidences of lung cancer and mesotheliomas in exposed humans. Given this deviation from the norm, the agency modified its evaluation criteria, which still lacks peer review: "the study domains of exposure, outcome, study participation, potential

confounding, and analysis were further tailored to the specific needs of evaluating asbestos studies for their potential to provide information on the exposure-response relationship between asbestos exposure and mortality from lung cancer and from mesothelioma."

While the agency mentioned Systematic Review in the Charge to the Committee, it has asked no questions. Therefore, EPN raises these questions.

i. Was it appropriate to narrow the scope only to certain cancers, i.e., the observations of lung cancer and mesothelioma in epidemiology studies? Should they enhance their discussion of other tumor sites, including if the data were insufficient to support dose-response modeling?
   We raise the latter question in response to the EPA statement on page 131: "Both the U.S. Institute of Medicine and IARC concluded that asbestos causes laryngeal cancer and IARC concluded that asbestos causes ovarian cancer." The evidence for other sites appears to be less definitive.
ii. Did EPA appropriately apply systematic review principles when narrowing the scope of its assessment and screening and selecting the information to use in this abbreviated assessment? In other words, were the tailoring/modification and application of the data evaluation criteria done in a credible manner?

b. Cancer Risk Assessment

In EPA's draft asbestos risk evaluation, risk estimates for workers and consumers are based on the agency's derivation of Inhalation Unit Risk (IUR) values only for lung cancer and mesothelioma, although other tumor types have been observed to occur following asbestos exposure. EPA states in the risk evaluation that over 24,000 asbestos studies were initially identified, but only 26 papers covering seven (7) study cohorts were selected for analysis. EPA provides little information on how they screened this enormous database down to seven studies, but does mention three elimination criteria. First, EPA explains that they eliminated all studies not focused solely on chrysotile exposure because the agency believes that chrysotile is the only asbestos fiber type currently imported into the U.S. as bulk asbestos or available as a component of asbestos-containing products. EPA acknowledges that commercial chrysotile does contain some, likely small, amounts of the other fiber types.

Second, EPA states that they screened out studies lacking sufficiently detailed information on exposure levels and duration.

Third, the agency mentions on page 30 that "several transmission electron microscopy (TEM) papers modeling risk of lung cancer were found, but because there was no TEM-based modeling of mesothelioma mortality, TEM data could not be used to derive a TEM-based IUR." This statement implies that EPA did not analyze or use these studies for any purpose. EPN is concerned that nowhere in the risk evaluation does EPA identify these TEM papers or compare the TEM models of lung cancer to the phase contrast microscopy (PCM) model of lung cancer, which EPA used for the risk evaluation. (EPA derived the lifetime unit risks for lung cancer and mesothelioma separately and then statistically combined them to yield the cancer inhalation unit risks for this evaluation). The decision to ignore these studies seems unjustified because the "Systematic Review Supplemental File on Data Quality Evaluation of Human Health Hazard

Studies: Mesothelioma and Lung Cancer Studies" indicates on pages 10 and 21 that studies using PCM or TEM analyses are appropriate for use. Since TEM detects much smaller fibers than PCM, it would be a good test of the PCM lung cancer model to see how it compared with the TEM model. It would also be interesting to know if the TEM models of lung cancer are based on a linear dose-response or an exponential model. EPA used an exponential model for their PCM lung cancer model based on calculation of Akaike information criterion (AIC) and the use of these criteria as a measure of model fit. Choosing the exponential model is a very consequential declaration since the exponential model results in less conservative IURs than the traditional linear dose-response model. We suggest that EPA describe the mode of action information (or lack thereof) that could further inform model choice based on biological considerations.

EPN questions whether exclusion of TEM studies is an appropriate choice. One could perform a TEM-based assessment for lung cancer and a PCM-based assessment for mesothelioma and still find a way to combine the two into a single value. We note that EPA did use different mathematical models for defining, extending, and extrapolating each tumor type's dose-response, and EPA further presented a rationale for combining the dose-response assessments.

We question whether it was appropriate to exclude from consideration those studies of scenarios in which exposure occurring to both chrysotile and amphibole fiber forms could not be separated out. EPA states on page 30, "As a naturally occurring mineral, chrysotile can co-occur with other minerals, including amphibole forms of asbestos. Trace amounts of these minerals may remain in chrysotile as it is used in commerce. This commercial chrysotile, rather than theoretically "pure" chrysotile, is, therefore, the substance of concern for this assessment." However, on page 134, it states, "In reviewing these available studies, EPA distinguished between studies of exposure settings where only commercial chrysotile was used or where workers exposed only to commercial chrysotile could be identified, and situations where chrysotile was used in combinations with amphibole asbestos forms and the available information did not allow exposures to chrysotile and amphibole forms to be separated. Studies in the latter group were judged to be uninformative with respect to the cancer risks from exposure to commercial chrysotile and were excluded from further consideration (e.g., Slovenia cohort: Dodic et al., (2007; 2003)." The definition of the substance of concern, commercial chrysotile, thus, is unclear on this point.

The agency provides inadequate justification for excluding other tumor types from the assessment. As noted above, authoritative bodies, like the World Health Organization's International Agency for Research on Cancer (IARC), have concluded that there is a causal relationship between asbestos exposure and cancers of the larynx and ovary. As EPA notes on page 131, "No toxicity values or IURs have yet been estimated for either laryngeal or ovarian cancers." We suggest that now is the time to determine if quantitative assessment of these other tumor types is possible and appropriate. We feel EPA has an obligation to screen and summarize the relevant literature, describe the studies in sufficient detail, and determine whether it provides the needed qualitative and quantitative information to include these two tumor types in a combined IUR with lung and mesothelioma.

EPN is also concerned that the draft risk evaluation does not consider the non-cancer risks of asbestosis and pleural thickening in addition to the risks of lung cancer and mesothelioma. EPA determines unreasonable risks for workers when cancer risks exceed 1 death per 10,000, but does not characterize the additional risk of asbestosis, which may be triggered by fewer fibers than are associated with the 1x10-4 cancer risk standard for the occupational setting (page 162: "EPA typically uses a benchmark cancer risk level of 1x10-4" for workers/ONUs for determining the acceptability of the cancer risk in a worker population") and the 1 x 10-6 standard (page 192: "Cancer benchmark is 10-6") for the consumer setting.

EPA notes on page 198 that, based upon a comparison of the 2014 Integrated Risk Information System (IRIS) reference concentration (RfC) for Libby amphibole asbestos for non-cancer health effects with the 1988 IUR for cancer risks, that the 1988 general asbestos cancer toxicity value appeared to be the clear risk driver, as meeting that target risk would result in lower non-cancer risks than those estimated at the RfC. This conclusion is relevant ONLY to the general population to which the 1 x 10-6 standard applies (or, in the case of this draft risk evaluation, consumer uses).

Furthermore, in the current draft risk evaluation, EPA is proposing an IUR that is about 1/3 lower (that is, less conservative) than the 1988 IRIS value. For the 1 x 10-4 occupational standard, EPA concedes (on page 198), "At this (1 x 10-4) risk level, if the non-cancer effects of chrysotile are similar to Libby amphibole asbestos, the non-cancer effects of chrysotile are likely to contribute additional risk to the overall health risk of asbestos beyond the risk of cancer. Thus, the overall health risks of asbestos exposure based on cancer alone are underestimated." We feel that EPA is obligated to determine if underestimation is the case and to what degree. This brief paragraph is not adequate:

> The POD associated with the only non-cancer toxicity value is 0.026 fibers/cc (U.S. EPA, 2014b). Although the non-cancer toxicity of chrysotile may be different from Libby amphibole asbestos, there is uncertainty that the IUR for chrysotile asbestos may not fully encompass the health risks associated with chrysotile exposure. Several of the COU-elated exposures evaluated for human health risks in section 4.2 are at or greater than the POD for non-cancer effects associated with exposure to Libby amphibole asbestos. (page 198)

Adjustments to the determination of reasonable or unreasonable risk may be warranted. A robust, credible assessment of this issue is in order.

2. The Timing of Public Comment and Peer Review

Just when you think it can't get worse, it does. In this, and other draft risk evaluations, the agency persists in misrepresenting their intended practices when stating that "Peer review will be conducted in accordance with EPA's regulatory procedures for chemical risk evaluations, including using the EPA Peer Review Handbook and other methods…." And "EPA believes peer reviewers will be most effective in this role if they receive the benefit of public comments on draft risk evaluations prior to peer review. For this reason, and consistent with standard Agency practice, the public

comment period will precede peer review on this draft risk evaluation."

This time, there was no time to provide comments ahead of the virtual prep meeting as the comments were due by noon of the same day (April 3) that the Federal Register notice, which announced the availability of the asbestos review materials and the schedule of events, was published. There were fewer than three weeks (due date: April 22) granted for public comments to reach the peer review committee before it was to have met on April 27-30, which, once again, was in the middle of the official public comment period. Since no new date has been announced for a rescheduled meeting, it is still possible that it could occur before the end of the full comment period (June 2, 2020). As we have stated repeatedly, this pattern of scheduling is not only inconsistent with the agency's own policies but it is also clearly inadequate for comment submitters and for the peer reviewers alike to study the draft evaluation and supplementary materials and prepare meaningful comments on these substantial and consequential assessments

3. Legacy Uses

In this draft risk evaluation, EPA acknowledges, for the first time, the court decision in Safer Chemicals Healthy Families v. EPA, Nos. 17-72260 et al. (9th Cir. 2019), that obligates the agency to consider legacy uses when conducting assessments in the Existing Chemicals Risk Evaluation program. As noted on pages 18 and 216, "this draft risk evaluation does not reflect consideration of any legacy uses and associated disposal for chrysotile asbestos or other asbestos fiber types as a result of that decision. EPA intends to consider legacy uses and associated disposal in a supplemental scope document and supplemental risk evaluation." Further, on page 232, "EPA will consider legacy uses and associated disposal (which could include the other five asbestiform varieties) in subsequent supplemental documents."

So the question becomes, "How soon?" While we would not suggest that the agency slow walk the chrysotile asbestos assessment so that the supplemental assessment could "catch up," we would recommend that the supplemental assessment proceed with all deliberate speed and that it include assessment of the other five asbestiform varieties in addition to chrysotile. Much of the available literature on asbestos does/cannot distinguish between fiber types, so attempting to parse toxicity profiles among them is not a useful exercise. We are concerned that because legacy asbestos contains multiple fiber types, people in the real world are not exposed to chrysotile alone, and a chrysotile-only risk evaluation and IUR will be inadequate when, as required by TSCA, EPA must examine the risks of legacy asbestos products that are a source of ongoing exposure.

4. Human Exposure and Personal Protective Equipment (PPE)

a. <u>Occupational Exposure</u>
Once again, EPN makes the point that risk determinations should NOT be made based upon the assumption that PPE is being used, for reasons that EPA itself states (on page 59), "The APFs are not assumed to be interchangeable for any COU, any workplace, or any worker. The use of a respirator would not necessarily resolve inhalation exposures since it cannot be assumed that employers implement comprehensive respiratory protection programs for their employees." This statement is supported by the summarization of the 1998 Riala and Riipinen study:

> However, for asbestos, nominal APFs in Table 2-3 may not be achieved for all PPE users (Riala and 2090 Riipinen, 1998), investigated performance of respirators and HEPA units in 21 different exposure abatement scenarios; most involved very high exposures not consistent with COUs identified in this RE. However, for three abatement scenarios, exposure concentrations were below 1 f/cc, which is relevant to the COUs in this draft risk evaluation. In the three scenarios, actual APFs were reported as 50, 5, and 4. The strength of this publication is the reporting of asbestos samples inside the mask, use of worker's own protection equipment, and measurement in different real work conditions. The results demonstrate that while some workers have protection above nominal APF, some workers have protection below nominal APF, *so even with every worker wearing respirator, some of these workers would not be protected"* (emphasis added).

On page 60, EPA notes that, "in 2017 EPA engineers conducted site visits to two chlor-alkali facilities. During these site visits, the observations by EPA engineers' confirmed details of the process descriptions provided by industry and described below." [Question: Did the facilities know EPA was coming ahead of time? It has been revealed in anecdotal and other evidence that if a facility (of any kind) is informed of a pending visit, that they will "clean up" and present a seemingly compliant operation when, in fact, it's not so under ordinary circumstances].

b. <u>Consumer Exposure</u>

Page 109: "The outdoor scenario assumes the do-it-yourself (DIY) brake repair/replacement work is performed in the consumer's residential driveway. It also assumes the additional work associated with this brake work is brake filing and occurs in the residential driveway." We would submit that the latter task would more likely take place inside the garage at a workbench.

Page 110: "EPA assumes, for the outdoor scenario, a consumer does not use compressed air." We disagree. We think the consumer could use compressed air, either from an aerosol can or a portable ("pancake") air compressor, outside or inside the garage.

"EPA assumes the consumer performs arc grinding for the indoor scenario and assumes the consumer performs hand filing for the outdoor scenario." We disagree: one is more likely to do this inside the garage at the workbench, perhaps using an electric grinder for both scenarios.

Exposure "can vary if the consumer has more than one car or works on vintage cars and that same consumer does all of the brake repair/replacement work for all cars they own." We would ask What would be a reasonable upper-bound exposure estimate for this scenario?

Page 113: Bystander—"EPA evaluated consumer bystander exposure for the DIY brake outdoor scenario by applying a reduction factor of 10 to the PBZ value measured outdoors for the consumer user. The reduction factor of 10 was chosen based on a comparison between the PBZ and the < 3 meter from automobile values measured indoors across all activities identified in the study data utilized from Blake (a ratio of 6.5). The ratio of 6.5 was rounded up to 10, to account for an additional reduction in concentration to which a bystander may be exposed in the

outdoor space based on the high air exchange rates and volume in the outdoor."

The Blake study focused on activity conducted in a professional work setting. In this instance, one could understand that any bystander would be some distance away from the action. However, we would suggest that would not be the case in a home setting (garage or driveway). In this instance, the bystander is more likely to be more actively engaged in the exercise and would be closer to the action, either observing to learn, teaching or helping out in some way. Exposure to the bystander should be assumed to be the same as the consumer's.

One could make the same argument for the DIY UTV Exhaust System Gasket Removal/Replacement Scenario—that the bystander is more actively engaged in the activity than would be observed in a professional setting and, therefore, would be assumed to be exposed to the same level of asbestos fibers as the consumer.

5. Aggregate Exposure

EPA identified both occupational and consumer conditions of use (COU) for inclusion in the asbestos risk evaluation. It considered occupational exposure to workers and non-users and consumer exposure to users and bystanders, which include, but are not limited to, male and female workers of reproductive age who are >16 years of age. Bystanders include individuals of all ages.

EPA evaluated exposures (inhalation only) in occupational and consumer settings to estimate risk of health hazard (for cancer only) for the COUs in this draft risk evaluation. Acute and chronic inhalation exposure was considered, although there was no aggregate assessment conducted for individuals who might be exposed in an occupational setting as well as a consumer-use setting. In addition, EPA acknowledged that exposure can potentially occur via all routes. One would expect that dermal exposure is not likely to pose an unreasonable risk under anticipated conditions, given unlikely absorption via that route. However, the same is not true of oral exposure. There are data to show that asbestos has been found in surface and groundwater, (e.g., collected from/near current or former National Priorities List (NPL) hazardous waste sites (ATSDR, 2001) and finished drinking water. A national drinking water standard was promulgated for asbestos in 1992. Furthermore, there are documented instances of airborne asbestos exposure from non-COU sources. Nonetheless, the Office of Pollution Prevention and Toxics (OPPT) continues to decline to conduct aggregate exposure assessments, ignoring exposures not directly related to specific conditions of use, pretending that no other exposures may be occurring in concert with those related to the COUs. Obviously, this leads to an underestimation, perhaps substantially, of the actual risk borne by individuals involved in the COUs.

This asbestos draft risk evaluation did not include the general population in its assessments. We expect to see this group considered in the supplemental evaluation addressing legacy uses.

6. Human Health Risk Determination

In the draft risk evaluation, EPA evaluated the following categories of conditions of use: manufacturing, processing, distribution in commerce, occupational and consumer uses, and disposal.

a. Workers and ONUs

EPA concluded, "For workers in all six COUs identified in this risk evaluation, cancer risks were exceeded for all central tendency and high-end exposures (chlor-alkali industry, stamping of sheet gaskets, use of sheet gaskets in the chemical production industry, oil field brake blocks, aftermarket auto brakes/other vehicle friction products installation and UTV gasket repair). In addition, for ONUs, cancer risks were exceeded for high-end exposure estimates in all of the COUs. For central tendency exposure estimates for ONUs, cancer risks were exceeded for sheet gasket use, oilfield brake block use, and UTV gasket repair."

EPN agrees with the agency's conclusions on the six COUs and encourages EPA to take risk mitigation measures (i.e., a ban) with all deliberate speed.

EPA determined the following COUs did not constitute an unreasonable risk: import of asbestos and asbestos-containing products; distribution of asbestos-containing products; disposal of asbestos-containing sheet gaskets processed and/or used in chemical production and import; use, distribution, and disposal of asbestos-containing brakes for the specialized and large National Aeronautics and Space Administration (NASA) transport plane ("Super Guppy").

EPA's rationale for concluding that the COUs noted above do not present an unreasonable risk is predicated on information received from the regulated community on their workplace practices purporting to protect workers and ONUs. EPN retains a healthy skepticism as to whether these purported practices are, in fact, in place at all times. EPA should have conducted its own investigation of actual handling and use of asbestos before it concluded that these activities do not present an unreasonable risk.

b. Consumers and bystanders

EPA concluded that "for all COUs that were assessed, there were (unreasonable) risks to consumers (DIY) and bystanders for all high-end exposures with the following exceptions: outdoor brake repairs (5 minutes/day in the driveway – benchmark not exceeded for high-end for both DIY and bystanders) and outdoor brake repairs (30 minutes/day in the driveway – benchmark not exceeded for high-end exposures for the bystander only). In addition, risks were noted for central tendency estimates for all COUs (brake and UTV gasket repair/replacement) for both consumers (DIY) and bystanders except for the outdoor exposure scenarios. Outdoor exposure scenarios for brake repair/replacement for 5 minutes in the driveway was the only scenario that did not exceed the benchmark for consumers (DIY) and bystanders. For outdoor exposures of 30 minutes/day once every 3 years, there were no exceedances for either the DIY or bystander for the central tendency exposure scenario."

Some of the outdoor exposure scenarios might result in a shift from a "no unreasonable" finding to an "unreasonable" finding if the recommended modifications to the exposure scenarios were implemented.

7. Environmental Assessment

EPN concurs, in general, with EPA's overall draft assessment of risk posed by chrysotile asbestos to the natural environment. We also offer the following discussion and recommendations on relevant issues and comments.

a. Relative Toxicities/Relative Potencies of Several Types of Asbestos

EPA evaluated risks posed only by chrysotile asbestos and discounted effects from other asbestos fibers. This suggests that EPA believes that the science is sufficient to determine the potency of each fiber type specifically. However, studies in the open literature suggest various orders of toxicity or potency. One study, for example, using embryonic human intestine-derived (I-407) and adult rat liver-derived (ARL-6) epithelial cells, reported that the order of cytotoxicity was chrysotile > amosite > crocidolite [source: https://www.sciencedirect.com/science/article/abs/pii/0013935180901231]. Another asbestos study indicated that relative potency of chrysotile: amosite: crocidolite was 1: 83: 376 [source: https://www.ncbi.nlm.nih.gov/pubmed/30077661]. Yet another study reported that " . . . at exposure levels seen in occupational cohorts it is concluded that the exposure specific risk of mesothelioma from the three principal commercial asbestos types is broadly in the ratio 1: 100: 500 for chrysotile, amosite and crocidolite respectively" [source: https://www.ncbi.nlm.nih.gov/pubmed/11108782].

Given these various approaches to assessing relative toxicities and relative potency, we think that the better approach is to consider health studies on *asbestos fibers in addition to chrysotile, e.g., amosite and crocidolite, because they will provide a broader database for addressing potential effects of asbestos for human health.*

b. Asbestos Bioconcentration

EPA's draft risk evaluation reports that chrysotile asbestos is not likely to bioconcentrate (Belanger, SE; Cherry, DS; Cairns, J; Mcguire, MJ. 1987. Using Asiatic clams as a biomonitor for chrysotile asbestos in public water supplies. Journal of the American Water Works Association, 79 (3): 69-74) because testing indicated bioconcentration factors (BCFs) for asbestos were low in the laboratory (slightly greater than 1). In the field, however, viscera BCFs were as high as 100, and whole clam homogenates ranged from 1,400 to 5,000. These data are consistent with the theory that asbestos fibers are difficult to depurate.

Given those field observations, it is not inconceivable that organisms that ingest clams may well be exposed to relatively large loads of asbestos fibers through their diet in areas where there is considerable asbestos contamination, and could by that route be a possible route for bioconcentrating asbestos.

c. Action Needed to Require Measured Concentrations of Asbestos in Effluent

For certain asbestos manufacturing operations, EPA's draft evaluation reported [lines 1815-1819] that their effluent guidelines establish limits on the allowable levels of total suspended solids (TSS), pH, or chemical oxygen demand (COD). However, the regulations do not establish specific limits for asbestos from those operations where asbestos discharges are

allowed. Thus, without the requirement to measure asbestos concentrations in effluent, estimating asbestos levels in effluent or receiving waters is challenging.

We take this opportunity to strongly recommend that EPA work to amend their effluent guidelines by requiring that (1) concentrations of asbestos in effluent be measured, and (2) specific limits be established for asbestos from those operations where discharges are allowed.