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Dear Panel Members:

RE: Updated Comments by Paustenbach on EPA Draft Risk Evaluation for Asbestos of March 30, 2020

Thank you for the opportunity to respond to the document entitled "Draft Risk Evaluation for Asbestos" that was released for comment around March 30, 2020. It represents an enormous effort on the part of the Agency, and it is among the most detailed and transparent EPA analyses that I have read. Alas, there are many portions of the document where I believe the Agency will need to make significant changes for it to be considered scientifically rigorous. It is also possible that the Agency will conclude that the document need not be revised, and will not issue a next version, since it does not materially identify a hazard that requires regulatory intervention.

I am submitting these comments about two weeks before the deadline, so the panelists have time to evaluate them carefully.

Background

I am a toxicologist certified by two different boards and a certified industrial hygienist. I have been an adjunct professor at five universities over the years while also actively serving in a professional role in the environmental and occupational health sciences for 45 years. I have written nearly 300 peer-reviewed papers, 55 book chapters, and three books on topics relevant to the work of this committee. I have also edited, perhaps, the most popular text on health risk assessment, which has been used for 30 years by many universities.

I considered it crucial that I submit comments since I have likely studied and conducted more original research in the areas covered in this EPA draft document (e.g., gaskets, packing, and brakes) than most persons in the field. In total, I have published 30 peer-reviewed articles addressing asbestos, of which, the Agency cited eight in its draft document to evaluate asbestos exposures under several scenarios.

My CV is posted on our website.

Relationship to EPA

I have often given lectures in which I note that the contributions of the environmental movement (very much led by the U.S. EPA) brought about a revolutionary improvement in the quality of life for hundreds of millions of Americans. Its influence on global attitudes has been profoundly positive. It was my good fortune to have served as a member of the EPA Board of Scientific Counselors (BOSC) Executive Committee for six years (2008-2014). I had previously served on several Ad Hoc Science Advisory Boards

(SABs) addressing specific chemicals. Today, I am a member of the EPA Science Advisory Board Chemical Assessment Advisory Board (2019 - 2022).

To varying degrees, I have submitted documents to EPA and interacted with the Agency on many levels for nearly 40 years.

Disclosure

This SAB, the Ad-hoc advisory panel, and the EPA ethics lawyers should be aware that I have conducted a considerable amount of research on the topics addressed in this document. I have also consulted with companies who used to have chrysotile asbestos in their brakes and in their gaskets, and those that used asbestos to manufacture other encapsulated products. Generally, I was paid for those efforts. I have also testified in perhaps 400 depositions and 20-30 courtrooms over the years on the hazards of chrysotile in brakes, gaskets, packing, Bakelite, roofing materials, and other encapsulated products.

These organizations, where I served as an expert in these matters, have financially supported some of my published work. Additionally, my previous firm, ChemRisk, performed several million dollars of work in this area without any outside support. The financial ties to any law firm or industry who supported our research or papers have been disclosed in our manuscripts for almost 20 years (unless, for some reason, the editor or publisher did not feel it was appropriate; which rarely was the case).

My colleagues at Paustenbach and Associates and I have invested approximately 200 hours in preparing these comments. We have not received compensation from anyone for this effort, which would typically be billed in the vicinity of approximately $70,000. The opinions expressed within these comments are our own. No lawyers or other experts have seen or commented on this submission.

Executive Summary of My Concerns

Asbestos has been one of the most studied chemicals within all of toxicology, epidemiology, industrial hygiene (exposure science), and occupational medicine. One can spend 30-50 years studying this topic and still not master all issues that relate to its potential to affect human health. There are about 300 papers related to the matters addressed in this *Draft Risk Evaluation for Asbestos* (March 24, 2020), which deserve to be understood by the panel to do a proper peer review.

Note Regarding This SAB Panel

The matters addressed in the 500+ pages of documents associated with this initiative are complicated and controversial. I feel that it was a bit unwise for the Agency to take on this task without considerable input along the way by the most knowledgeable scientists in the field. Perhaps that occurred, but that is not clear to me.

Having served on several EPA SAB panels and having addressed similar panels over the years, I am sympathetic to the challenge by those who have not been immersed in the relevant asbestos literature for one or two decades or more. With the fairly recent restrictions regarding “conflict of interest” (real or perceived), it is almost impossible to convene a panel of experts who know the topics at a depth

necessary to adequately address the more complicated questions posed by the Agency in this document. I would encourage the Agency to consider returning to the prior policy where it asked prospective EPA candidates to disclose their perceived conflicts and then allow the ethics lawyers at EPA decide if those present a problem.

This would allow professionals who are highly accomplished and true experts to be part of future panels. I am not suggesting that the current SAB for this document is inadequate, but I do believe that the Agency would benefit from the wisdom of professionals who serve as experts in litigation or are consulting experts to the private sector.

While developing these comments, I frequently referred to the 200 or more published papers that are relevant to the topics in this document. I hardly think that it is fair for volunteers who are not "deep" into the study of asbestos to expect them to master these topics in a relatively short period of time. But, for many SAB panels, this is precisely what is expected of them.

I applaud this SAB panel as it attempts to master the relevant literature and deal with the many comments which you will undoubtedly receive. I must note, however, that I was disappointed that EPA released this document on March 22 (during the peak of the COVID-19 crisis) and then requested an April 22 submission of comments (still during the crisis). Like 300,000,000 other Americans, all of us have been cloistered. Thus, I was pleased that the Agency extended the deadline and the date of the SAB meeting.

I believe the SAB panels should be aware that, although I am not a lawyer, I am not convinced that the EPA is required to address this topic to fulfill its responsibilities under the Lautenberg Chemical Safety for the 21st Century Act. While not mentioned in the document, it could have a dramatic impact on current and future toxic tort litigation. It may not be a concern of the Agency, but I think it is nevertheless important to recognize.

Overall, I am surprised that in light of the many competing priorities facing EPA, this topic, which may affect 10 - 100 or fewer persons annually in the coming years rather than the 1.5 million persons the Agency identified as at risk, deserved this level of effort. I address these and other matters, as summarized here, and discussed in detail in the comments below.

1) **The foundation of this document is that Americans today, and in the future, are going to be exposed to raw or encapsulated asbestos. This document estimates that it could be as high as nearly 1,500,000 persons per year. In my experience, and based on my knowledge of the topic, that is not even remotely close to being accurate.**

2) **Based on my studies, the number of persons potentially exposed in the coming years is minimal. Indeed, I estimate that perhaps no more than 100 persons in the United States may work with raw or encapsulated asbestos where there could be measurable or significant exposure. In chlor-alkali facilities, many workers are allegedly exposed (according to this document), but the exposures are *de minimus* compared to the OSHA PEL based on the data presented.**

3) **The data presented for the one or two facilities that occasionally punch out "asbestos-containing gaskets" indicate that the exposures of those 2-3 workers, who might work the**

press occasionally, are well controlled. The information presented by the Agency about the number of persons who "might" work with asbestos-containing gaskets in the coming years is not accurate.

4) **Concerning brakes, it is unlikely that there are many persons purchasing or installing brakes with asbestos. Even if one is able to do so, they will almost always be disc brake pads rather than drum brake linings. The Agency's estimate that as many as 750,000 mechanics in the U.S. might be expected to work with such brakes in the coming years is off target. At best, 20-100 "shade tree mechanics" may find drum brakes containing asbestos in the United States and install them, but that is unlikely since drum brakes are only needed for a select few antique cars. Shade-tree mechanics typically work outdoors, so those exposures would be *de minimus* (if they occur at all).**

5) **No dealership or brake shop would knowingly install an imported brake if it contained asbestos. The liability is too significant. As noted, only a handful of car owners may replace their brakes at home, but generally speaking, drum brakes have not been used in cars in the United States for almost 40 years. Furthermore, it is exceedingly rare to find persons in the modern era who would attempt to replace drum brakes themselves. It is a relatively complex process that is best done with equipment typically not found in a non-mechanic's home garage.**

6) **In the document, the Agency claims that it is only concerned with brake changes going forward. As such, the Agency assumes that modern era control technologies for brake changes will be implemented by those in the general public and DIY mechanics. However, all the calculations in the document are based on studies involving techniques used from approximately 1940 to 1980.**

7) **The Agency would be familiar with the exposures in the modern era if it referred to the five or six papers written by NIOSH in the late 1970s and early 1980s. NIOSH specifically measured the exposures associated with using modern techniques. None of those papers were cited in this document.**

8) **Some of the assumptions about exposure frequency for those working with gaskets and brakes are not reasonable based on the existing scientific literature.**

9) **According to the document, only a limited number of asbestos-containing sheet gaskets rolls are imported each year for one specific use in one industry. That is why they can be produced by one firm on a handful of days per year (involving just two employees). In my view, this is not worthy of attention compared to the vast responsibilities of EPA, given that the exposures are insignificant.**

10) **I find it troubling that the Agency has concluded that exposures in certain occupations, which have been known for decades not to pose a significant hazard to workers, have been categorized as presenting an "unreasonable risk to the health of workers." The only way that the Agency could have reached such a conclusion is by changing the cancer potency factor (CPF) for asbestos and, in particular, for chrysotile. The scientific basis for**

this change is flawed and casts serious doubt on the validity of the conclusions of the document.

11) **The Agency appears to have been convinced that it is possible to identify a CPF for chrysotile alone, even though, after nearly 70 years of study, no other body in the world has ever claimed that such a CPF could be offered. The Agency relies on the North and South Carolina textile cohorts for deriving its CPF, which is inappropriate for several reasons. First, some members of this cohort have been known to have been exposed to amphiboles. This was noted in the original manuscripts by Dement et al. (2009), Loomis et al. (2009), and in depositions of Dr. Dement. Beyond that, Roggli et al. (1998) studied the lungs of at least one of these workers and found amphiboles (Roggli et al., 1998; Pavlisko et al., 2020). The amphiboles are at least 100-fold more potent for causing mesothelioma than chrysotile. Thus, the Carolina cohorts are not "chrysotile only" and should not be used to derive a chrysotile CPF. Consultants to EPA (Berman and Crump, 2003), I believe, reached this decision almost 20 years ago when EPA funded their work.**

12) **It is well known in the asbestos literature that if chrysotile could produce mesothelioma (which remains in dispute), it may do so only at doses that are in the vicinity that cause asbestosis (50-400 f/cc-year) (Churg, 1988; Churg et al., 1993; Pierce et al., 2016). Additionally, the fibers are likely to be exceedingly long (like those only found in textile mills) and, therefore, vastly different in length and aspect ratio from the short asbestos fibers that were used as filler in brakes and gaskets.**

13) **In my view, the Agency missed essential epidemiology studies conducted in vehicle mechanics exposed to encapsulated asbestos. At least 15 studies have evaluated these cohorts for the periods when asbestos could have been in brakes in the United States. These are addressed, quantitatively and critically, in several peer-reviewed meta-analyses of lung cancer and/or mesothelioma, which are the most relevant studies for understanding possible cancer risks of chrysotile in brakes and can be extrapolated to gaskets and packing. I would refer the SAB to the comments submitted by Moolgavkar et al., dated May 18, 2020, and by Dr. Garabrant, dated May 20, 2020, for an in-depth discussion of these studies.**

14) **Several papers have addressed the biologic activity or potency of fibers that have been either heated or simply filled with phenolic resins, such as brakes, Bakelite, and gaskets. Bernstein et al. (Bernstein et al., 2003; Bernstein and Hoskins, 2006; Bernstein et al., 2013, 2018, 2020a, 2020b) have found that that asbestos loses its toxicological potency when filled with resins or when the fibers are converted to forsterite. These papers were not mentioned in the EPA document.**

15) **The Agency discusses the possibility that fibers will drift from a point source and possibly cause a serious potential hazard to nearby workers or that they provide an almost infinite source of fibers for resuspension. This is generally not the case, especially for the fibers which have a length of 5-40 µm. Fibers of this length tend to agglomerate, fall due to gravitational forces, or are attracted to one another due to van der Waals forces. The half-life for these so-called OSHA fibers (e.g., longer than 5 µm with an aspect ratio of 3 or**

greater) or equivalent particles to settle out is on the order of approximately 5 minutes, not the hours or days as this document suggests.

16) **The myth that the particles remain in air for days or are easily resuspended, with the potential to cause inhalation exposure for weeks after they are generated lacks scientific merit. The only studies supporting this view contain no quantitative discussion of the air concentrations of OSHA fibers over time (e.g., no settling rates with half-life by fiber size). Rather, quantitative historical reports show that the fibers that remain suspended are generally too small to be of biological importance, i.e., much less than 5 μm. The tendency of these fibers to be resuspended is also quite low as they stick fairly tightly to flat surfaces. The data from the work at the World Trade Center make this abundantly clear (see the report by the oversight panel on exposure in which I participated).**

17) **One cannot rule out that, given the information provided in this document, some brakes purchased online by an individual may contain asbestos. However, as noted by the Motor & Equipment Manufacturers Association (MEMA), these purchases comprised less than one percent of the brakes sold in the United States in 2013, which is likely an overestimate, given that the letter was published over six years ago and that MEMA would like to see these purchases banned. Nonetheless, even if MEMA's estimate are accurate, one must be aware that the brakes sold online are almost certainly disc brakes. Installing or removing a new disc brake should not pose any exposure to the worker. Therefore, even if <1% of the disc brakes are in the marketplace, they are irrelevant from an exposure or health risk standpoint.**

18) **When deriving the cancer potency factor (CPF) for chrysotile, EPA adds both lung cancer epidemiological data to the mesothelioma epidemiological data, which is scientifically inappropriate on several levels. First, during the lung cancer epidemiology for the era in question (the 1940s – 2000s), a substantial portion of the male worker population smoked for short or long periods of time. Studies show that persons frequently do not recall accurately if they smoked for only a few years or find it socially unacceptable to admit to any smoking history. This compromises nearly all studies when attributing lung cancer to an inhaled carcinogen. Without a comprehensive smoking history for each person or a comprehensive knowledge of the exposure to various forms of asbestos (and other airborne carcinogens), these studies cannot be used to identify a "chrysotile only" potency factor for lung cancer. The potency of cigarettes overwhelms the cancer potency of chrysotile. In 1955, Doll did his best to address the potency of asbestos alone but knew that smoking was a confounder that is difficult to control (Doll, 1955).**

19) **Regarding chrysotile only CPF, lung cancer and mesothelioma are generally believed to act through entirely different mechanisms of action. To combine them into one CPF is highly questionable and contrary to the current EPA Cancer Guidelines. Long fiber chrysotile, if these can cause mesothelioma, probably do so via clogging the stoma and subsequent chronic inflammation (Lynn, 1983; Englert et al., 2014). Chrysotile probably increases the lung cancer risk primarily in persons who smoked for some period of time – chrysotile fibers absorb the many carcinogens present in cigarette smoke. Roggli et al and others**

have addressed these issues over the years (Englert et al., 2014). On top of these factors, the Agency has identified the incorrect cohorts for calculating its CPF.

20) **When coupled with the fact that chrysotile is almost certainly a threshold carcinogen, i.e., it acts through two different mechanisms to cause lung cancer and mesothelioma (if it can do so at any reasonable dose), and because it does not appear to cause either disease until asbestosis-producing lifetime doses are reached (probably only with very long fibers), it seems scientifically unfounded to try to derive a single CPF for chrysotile for both diseases. If it were scientifically valid, it would have been done long ago.**

21) **I would direct you to significant EPA contracts and peer review panels, which were oddly not cited in the Agency's document, where it was concluded that chrysotile may not have potency for mesothelioma or lung cancer (at least not until very high cumulative doses and/or long fibers are involved). Be reminded that Dr. Agnes Kane convened an EPA SAB to address this issue and, while the committee failed to reach agreement on a number of topics, it did conclude that the potency of different fiber types should be accounted for in regulatory decision making.**

22) **For its calculation of the CPF, I also believe that the Agency incorrectly assumed that there is no background rate of pleural or peritoneal mesothelioma in persons not exposed to asbestos. There are numerous papers demonstrating that mesothelioma occurs in persons never exposed to asbestos levels above background. The two papers that have recently questioned this view are not convincing especially when compared to the arguments presented by Price and Ware, or Moolgavkar et al., 2009 (and others). This topic deserves significant attention from both SAB panels.**

23) **A considerable number of toxicologists and epidemiologists believe that soon after about 2025 or 2030, a vast majority of mesotheliomas seen in the population were spontaneous in origin. I recognize that the opposing school of thought is that that the latency for causing mesothelioma is longer than what we believed in the past. Some have postulated that the latency maybe 30-35 years or even as high as 45 years. I believe it is highly unlikely that the latency is longer than 40 years, except for perhaps radiation, but that has also been questioned in recent years. Without a lung biopsy, spontaneous mesotheliomas are indistinguishable from mesotheliomas due to asbestos exposure. Given the few workers with appreciable exposure after about 1975-1980, it is much more likely that any mesotheliomas after 40 years of the date of last exposure are spontaneous instead of the suggestion that the latency continues to get longer than once thought.**

24) **It has been evident to most scientists who have studied pleural mesothelioma that its incidence rate increases with age (even in unexposed cohorts), just like every other cancer. The trends are obvious. Critics have claimed that "they were probably exposed to asbestos at some point," but that is usually purely speculation and, for chrysotile, almost no one will claim that asbestos-related disease occurs without very significant exposure of which the person would be aware. This is entirely different than exposures to the amphiboles which can go unrecognized by persons who have been exposed over the years.**

25) **There are not many known causes of mesothelioma but it is well known that the pleural mesothelioma rate is higher in those who have had chest radiation treatments for cancer at mid-life during the 1950-1970/1980s (and who were never exposed to asbestos). Additionally, you often see peritoneal mesothelioma in persons in their 30 - 50s when their exposure history indicates that they were always office workers (not to mention that the latency would not be consistent with the disease if it was solely due to asbestos exposure). Mesothelioma clearly occurs without exposure to asbestos in probably 20 – 25% of cases, if not more. As the typical American enjoys greater longevity, we see an increased incidence of mesothelioma, that is independent of asbestos exposure.**

26) **As mentioned, it is clear from the data in the EPA's document, that few, if any, persons in the United States should ever again be exposed to chrysotile above trivial concentrations. Lifetime doses will almost certainly be 100 to 1,000-fold less than those that should cause an asbestos-related disease. I doubt that as many as 100 persons in the U.S. annually could have measurable exposure to asbestos today from brakes, gaskets, and packing (for various reasons). As mentioned, the available data presented by the Agency does not seem to identify any cohorts which might be routinely exposed above about 1/10 to 1/2 the current OSHA PEL for asbestos (and the number of plausible workers is very low).**

27) **In reviewing this document, one cannot help but think that the EPA is using this analysis as a backdoor method for supporting a ban on asbestos. Personally, I have no problem with a ban as long as critical uses of military or research significance are protected; however, given the flaws in this document, it would appear that a different approach to supporting such a ban is needed.**

28) **It also seems plausible that the EPA is using this document to cajole OSHA into revising its PELs for asbestos. Again, I would support OSHA revisiting its PELs and establishing new and separate PELs for chrysotile, amosite, crocidolite, and tremolite. This is long overdue. However, as noted above, this document is not sufficiently robust on a scientific basis for assisting OSHA in revising the PELs for the various forms of asbestos.**

29) **If there are a handful of applications in essential industries that require the importation of a limited number of asbestos-containing gaskets or packing to produce militarily or economically crucial goods, the Agency could ban the importation of asbestos, except for these special applications. It is not necessary for the Agency to overlook logic, decades of research, scientific literature, and overestimate the size of the plausibly exposed population to discuss exposure scenarios that do not exist (at least not for the almost 1.5 million workers that EPA says could be over-exposed in the coming years).**

30) **This document suggests that hundreds of thousands of persons each year in the future may be exposed to dangerous levels of asbestos. The Agency provides no scientifically credible basis for this claim. In fact, such exposures do not currently exist or are insignificant, i.e., they do not pose a hazard to the handful of potentially exposed workers. Those persons in chlor-alkali facilities, gasket stamping, and those replacing brakes on**

antique cars, based on the data presented in this document, and in my own professional experiences, are not currently over-exposed. You have to discard the OSHA PEL, generate inaccurate exposure scenarios and derive a "new" cancer potency factor for chrysotile alone to conclude that occupational conditions for the six categories of workers evaluated by the Agency (see page 26 of 310) are at "an unreasonable risk to health."

31) **To the best of my knowledge, in the 50-year history of the EPA and OSHA, neither agency has put forth any regulatory document focused on any hazard for which even using CPFs derived from a linearized multistage model (LMS) leads to a prediction of *zero* incremental cancer cases for any of the exposed cohorts (Travis and Hattemer-Frey, 1988; Dudley, 2015). Indeed, it would appear that even if one assumes that 1,000 – 10,000 persons were to be exposed to chrysotile in these applications for the next 40 years, there would not be even a single incremental case of cancer in the exposed population. Based on my review of dozens of regulations, no agency in the federal government has ever been concerned about such trivial risks.**

32) **Thus, I am more than a bit surprised that the alleged hazards described in this document would warrant 310 pages of examination. I was even more surprised that EPA concluded that many exposures in the United States today "present an unreasonable risk to health" (page 26, ln. 1106), even though the worker exposures discussed in this document were usually far below the OSHA PEL.**

33) **The litigation which could be generated by this document could involve claims of billions of dollars related to personal injury associated with alleged asbestos exposures of mechanics, pipefitters, millwrights, oil rig workers, and others who worked between 1945-1985. If EPA wants to encourage future, possibly unnecessary or unwarranted litigation, I can think of no better way to do that than issue this document as currently written.**

I hope that the Agency gives some consideration to my and others' comments before moving forward with finalizing this document. In my view, the analysis is not consistent with the high standards of many EPA initiatives. To believe in the conclusions, one must accept too many unfounded assumptions.

As noted previously, to those of us who have studied this issue, it looks very much like the Agency is trying to use this document to support a ban on the import of asbestos. I would welcome such a ban, but functionally, it is probably not necessary. The litigation environment has prevented firms from using asbestos for nearly 40 years. If the Agency wishes to provide support for such a ban to Congress, there are surely other approaches rather than assemble a cascade of assumptions that lead one to a conclusion that, on the face of it, is not supportable.

The other possible reason for producing this document, I am guessing, is to encourage OSHA to review its PELs for asbestos. That is, reduce them because some consider them to be too high. Or, EPA is encouraging OSHA set separate PELs for each fiber type. Both initiatives would be worthwhile but using this document, which contains many shortcomings, is not the appropriate way to make this happen.

In my comments, I recognize that I frequently repeat the same criticisms for different statements within this document. I did this, page by page, because, in my experience, the comments are often "separated" among various groups in the EPA who assembled the document. When that happens, some members of

those teams do not receive the entire set of comments, but instead, only those that supposedly relate to their contribution. Thus, I chose the "comment by comment" approach.

PAUSTENBACH SPECIFIC COMMENTS

The following comments are intended to be used to improve the quality of the next draft of this document. Depending on the content of the letters to the Agency regarding this document, perhaps the Agency will conclude that it is not appropriate for them to issue a revision in the future. That is likely a decision to be reached by EPA management or OMB but, for the reasons expressed below, I do not believe this document is needed to achieve any requirements of the Lautenberg Act.

Also, I did not assemble these comments using the style, which was identified in the Charge Questions to the panel. I chose not to attempt to direct the SAB panel by suggesting how I would answer those questions. Instead, I commented on the scientific merits of the content of each major category of topics in the document and attempted to introduce additional pieces of scientific knowledge that are available in the literature.

Having written more than 1,000 risk assessments during my career, I applaud the thoroughness and transparency of the EPA analyses. The text was clear, and it focused on the objectives. The team involved in the exposure calculations should be applauded for the transparency and clarity of most of the calculations. Unfortunately, by my way of thinking, more than 90 crucial papers were "missed" in the comprehensive search, and these should be considered in the next draft. These papers, and a better understanding of the subtleties regarding how to read and reconcile this complicated body of literature, is needed to conduct this assessment properly. I hope that my comments allow the SAB and the Agency to do that.

Statement 1: "This draft risk evaluation for asbestos was performed in accordance with the Frank R. Lautenberg Chemical Safety for the 21st Century Act and is being disseminated for public comment and peer review." (Page 17, ln. 665 - 667)

Comment 1: I am supportive of the Agency's desire to adhere to the goals of the Lautenberg initiative. However, in reviewing TSCA, I was unable to find a rationale for justifying the preparation of this document. There are several reasons why its development is confusing to me and likely many other professionals in the environmental health and safety fields. Therefore, some of the claims are bound to be surprising (and misleading) to others outside of our professions.

As I will explain here, your document may be addressing the exposures of quite a small number of persons in the United States today (at least on any routine basis), concerning brakes, gaskets, and packing. And, more importantly, it is logical to conclude that even fewer persons will have exposure to asbestos-containing products in commerce in the coming years due to litigation and the broad awareness of the hazards of over-exposure to asbestos.

This document may apply to less than 50 or 100 persons in the United States today who might have any future opportunity to work with these materials, even occasionally. In the coming years, I doubt that it will apply to more than a handful of persons (excluding those in the Chlor-alkali industry who are apparently not materially exposed due to controls).

In my 40 years of experience, I have not seen EPA, OSHA or NIOSH write such a massive document regarding this few numbers of potentially exposed persons, who's alleged exposures to airborne concentrations of asbestos, based on this document, are much less than the current OSHA Permissible Exposure Limits (PELs) or ACGIH Threshold Limit Values (TLVs) for asbestos.

I cannot speak to exposures in the Chlor-Alkali industry beyond what is presented in the EPA draft, as I have no experience working with that industry. However, simply as a matter of current interest, during the peak of the COVID-19 crisis, it would not seem to be a prudent time to be placing further constraints on this crucial industry that produces the most widely used disinfectant (chlorine), when the data shows that the workers are not over-exposed to asbestos (based upon the OSHA PEL).

First, having studied the topic of asbestos-containing gaskets, packing, and brakes for the past 20 years, EPA should consider just how difficult or even impossible it is to find such materials in the marketplace anywhere in the United States. I was aware of the single sheet gasket company that you identified, which punches out already manufactured sheets from an overseas source. For the benefit of readers, sheet gaskets contain up to 50% of resin-soaked asbestos, and these fibers are embedded in a resin or other polymeric material. They are not released to a measurable degree during routine cutting procedures. It is noteworthy that I have tried valiantly to find such materials so that research could be conducted with them and have generally failed. After many years of searching, I have not found an asbestos-containing brake at any store and, when I purchased them on the internet, they always failed to contain asbestos.

You may not be aware that nearly all major companies in the United States discarded most, if not all asbestos-containing materials or replaced them in the various pieces of equipment during routine scheduled maintenance beginning in or around 1990, if not earlier. That is more than 30 years ago.

Nearly all large firms, between 1974 and 1985, contacted their suppliers and insisted that they halt supplying asbestos-containing products. Documentation for this is readily available. At the same time, these firms put in place policies where all asbestos-containing materials, which were replaced due to wear and tear, were not to contain asbestos materials going forward.

Further, even though it was not required for them to throw away asbestos-containing materials in their warehouses, most large firms, going back 30 to 40 years, discarded these materials rather than use that stock as replacements (even though it was permitted). Often, this was because they were not sure that they could use these parts and still meet the OSHA PEL. By that time in history, billions of dollars in litigation had already begun regarding asbestos-containing material. They had no interest in having any possible future liability from that material.

Another curiosity about this EPA document is that it purports to be a forward-looking assessment. However, the data relied upon were collected 10-25 years ago using materials and equipment from 40-50 years ago! It appears (and is stated) that the 310 pages are predicated on a belief that some handful of people (maybe less than 100 in the nation) are interacting with newly manufactured and imported asbestos-containing materials. If one assumes that those workers in the chlor-alkali business are the only ones who might be exposed to raw chrysotile (but it surely seems well controlled), then I would be surprised if more than 100 persons in our nation (going forward) "might" be exposed in the coming years (or potentially exposed to concentrations greater than the current OSHA PEL).

As noted in the executive summary, there is a heavy emphasis on the Blake et al., paper, which focuses on techniques that were used 60 years ago. Those studies were conducted to understand exposures which occurred from approximately 1940 – 1975/1985; not exposures post-2020 (which are supposed to be the focus of this document). The proper studies to understand exposures going forward, assuming anyone or a handful of persons are using asbestos-containing brakes, would be the exposures which NIOSH quantified in their six different reports (Dement 1972; Johnson et al. 1979; Roberts 1980a, 1980b; Roberts and Zumwalde 1982; Sheehy et al. 1989). Today, I would expect the control technologies that are used to be better than those studied 30-40 years ago by NIOSH. All these factors lead one to question most of the assumptions regarding exposures going forward that are found in this EPA document.

In the late 1960s, the first measurement of a brake job TWA in a vehicle brake repair garage was undertaken by Ford Motor Company (Hickish and Knight 1970). This study was conducted at a Ford dealership in London, during which 11 vehicles were serviced, and the authors' specific aim was to evaluate exposures to airborne fibers associated with blowing out the brake shoes and drums, a practice which the authors noted was "not included in all routine services," and to evaluate compliance with the 1968 British Occupational Hygiene Standard for Chrysotile Asbestos Dust (Hickish and Knight 1970). The authors reported an 8-hour TWA of 0.68 f/cc, which was below the contemporaneous British occupational standard (2 f/cc over a working lifetime of 50 years) and the ACGIH TLV for asbestos of approximately 30 f/cc (i.e., 5 mppcf).

This research was referenced during the 1969 International Conference of Pneumoconiosis in Johannesburg, South Africa, where an international group of scientists convened to discuss the medical and engineering aspects of the asbestos industry, as well as current research findings. It was noted by Dr. W.J. Smither that "[a]sbestos is not indestructible, since most fibres from brake drums are changed into forsterite;" he further noted that "[e]ven in the conditions of brake drum cleaning by compressed air, time-weighted average exposures are within the limits considered negligible by the British Occupational Health Service Standards" (Shapiro, 1970). Moreover, Dr. Irving Selikoff of Mount Sinai Hospital in New York responded that "[b]rake linings do not constitute a hazard" (Shapiro, 1970). This was 50 years ago!

Soon thereafter, similar studies were conducted in the United States, following the promulgation of an occupational standard for asbestos by OSHA. From 1972 through 1989, the National Institute for Occupational Safety and Health (NIOSH) conducted numerous investigations of vehicle brake servicing operations in the United States (Dement 1972; Johnson et al. 1979; Roberts 1980a, 1980b; Roberts and Zumwalde 1982; Sheehy et al. 1989). The consistency among independent 8-hour TWA airborne asbestos concentrations among the NIOSH studies indicates substantial similarities in brake repair operations and the asbestos content in brake linings. In addition, independent studies of airborne asbestos levels in brake servicing garages were published in the 1980s and 1990s by researchers in the United States (Moore 1988), Germany (Rodelsperger et al. 1986), Sweden (Plato et al. 1995), and Australia (Yeung et al. 1999). The exposures of hundreds of mechanics were evaluated in these studies, and nearly all of them supported a view that asbestos exposure to mechanics during brake job practices were not generally believed to be of concern.

You have identified one firm which punches out gaskets that contain chrysotile, which I was aware of. However, I am not aware of any data which indicates that anyone else is interacting with asbestos-containing materials in 2020. I discuss this numerous times in my comments.

Another observation is that to the best of my knowledge, in the 50-year history of EPA and OSHA, neither agency has put forth any regulatory document focused on any hazard where even using cancer potency factors derived from the linearized multistage model (LMS), the prediction is that there will not be a single case of cancer for any of the exposed cohort (Travis and Hattemer-Frey, 1988; Dudley, 2015). Thus, I am more than a bit surprised that the alleged hazards described in this document would warrant 310 pages of examination. I was even more surprised that EPA concluded that many exposures in the United Stated today "… present an unreasonable risk to health." (page 26, ln. 1106) even though this document indicated that worker exposures which EPA discussed were usually far below the OSHA PEL.

Additionally, it appears to me that this document is outside of the purview of EPA, as it deals with occupational exposures, which should be handled by OSHA. I am well aware that EPA has some responsibility for ensuring that new and existing materials that are in the marketplace do not or will not pose a significant health risk to the public or workers (*vis a vis*) a premanufacture notification (PMN) process. But, what is addressed here does not fall in that bucket because the number of workers that might plausibly be over-exposed is virtually zero (unless the assumption is that the appropriate OSHA PEL should be 0.01 f/cc (or lower)) on an 8 hour TWA basis).

Brakes

To conduct the five to nine different studies that my former colleagues and I performed between 2002 and 2017, we had to spend dozens of hours searching for the very products that this document is raising concerns about. During that time frame, we failed to identify any asbestos-containing brakes in any retail establishment (including more than 20 NAPA dealerships, small garages, and backwoods warehouses). Thus, in my experience, any belief by EPA that persons are going to be exposed in the coming years (except perhaps for the one location that they identified) is difficult to accept.

The only location of which I am aware where some asbestos-containing brakes were retained is an auto restoration facility in California that specialized in restoring very large and heavy automobiles from the 1950s and 1960s. The reason they used the old asbestos brakes is that they feared that the replacement fillers in the brakes (used to replace asbestos) might not have been adequate to stop a vehicle of that weight. Therefore, for the few dozen cars that they sent each year to Scandinavian countries, they often wanted to be sure that each would stop as appropriate. Even 15 years ago, only a couple dozen people continued to work on these vehicles each year. I believe nearly all such work ceased in about 2015. It makes no difference; in more recent years, they were installing new brakes for which there is no exposure to asbestos.

I am not aware of the site which you described on page 87. It is noted that "… asbestos containing automobile components are used in a single-vehicle which is manufactured domestically but only exported and sold outside of the United States." (Page 87, ln 3120 – 3121). The actual site should be disclosed to determine if such a facility still actually produces such vehicles with asbestos-containing material. Assuming such a place exists, it is reasonable to assume that work with asbestos-containing material would only involve the installation of brakes. Thus, there would be no exposure to asbestos during this process.

The Agency agrees that there would be no exposure associated with the installation of new brakes, as stated in this document (pg. 96, ln. 3519 – 3521). Keep in mind, there is no need to grind a new brake prior to it being installed on a modern car.

In short, going forward, I cannot imagine a scenario where any full-time mechanic would be installing brakes that contain chrysotile asbestos. I could not find one, especially a new one, even when I tried with considerable effort for research purposes. One cannot rule out that some brakes from outside the country might slip into the United States, but the number must be vanishingly small. The <1% number cited by the Agency in this document seems unsupportable, and I suggest that that it deserves further study.

Gaskets

Concerning gaskets, for the past 20 years, I have studied the release of asbestos from this product for the years that they contained asbestos. My experience in recent years, and probably like other researchers, in being able to find an asbestos-containing gasket is similar to my inability to find an asbestos-containing brake. After contacting 30 – 50 major firms, I was unable to find a sheet gasket that contained asbestos, after approximately the year 2000.

Most firms indicated that the asbestos-containing gaskets were destroyed in the 1980s. To conduct simulation studies, I was able to locate small pieces of sheet gaskets in a pipe fabrication shop outside Bangor, Maine, in 2005. In short, these materials have not been generally available for use in the United States for at least 30 years.

Virtually all of these published studies involved the removal of gaskets from valves and fittings that were located in scrap metal storage areas near chemical plants. These old pieces of pipe were from the 1950-1970 era. Even then, a large percentage of the gaskets we found in these “boneyards” did not contain asbestos.

I am not surprised that EPA was able to locate only a single manufacturer who still punches out asbestos-containing gaskets for the refinery and/or chlor-alkali industries (which are imported in rolls from overseas). The Agency accurately characterizes that single site as having four employees (two potentially exposed) who only have a few dozen hours of exposure each year. What is noteworthy is that these workers have been studied and all are exposed to less than the PEL.

Therefore, going forward, it would appear that it is implausible that persons will be exposed to asbestos-containing gaskets, except when removing gaskets from equipment that is 30 or more years old. Even then, every pipefitter that might replace gaskets on this older equipment would appropriately assume that asbestos might be in those gaskets and will be governed by current practices, as well as OSHA regulations, that mandate controls to limit exposure, even if asbestos-containing material is not present. Fortunately, as the EPA document indicates, those exposures are generally less than the OSHA PEL, and are usually immeasurable small when the work is conducted outdoors.

From what I can determine, EPA has identified one company that purchases sheet gaskets in the United States, with use limited to one or two industries. In consideration of the significant cost of asbestos litigation activity in the U.S., the Agency might consider that this one remaining user might have excellent reasons for continuing to produce asbestos gaskets in such a litigious environment.

Packing

Consistent with my experience with brakes and gaskets, I have, for over the past 10-20 years, been unable to identify any asbestos-containing packing in the marketplace or historical warehouses. This is consistent with my prior statements that these materials were disposed of many years ago. I know of no firm that imports asbestos-containing packing materials because of the potential exposure to litigation and because there are many acceptable substitute materials for this application.

Even if there was asbestos in new packing materials, as EPA has learned from its review of the literature, there is virtually no exposure to those who install packing and only very modest exposure when removing old dry packing from 40+year-old equipment. In my reading of the Lautenberg regulation, it only applies to materials for which there is expected use (and some exposure) going forward. It does not appear to require evaluations of exposures from 30 to 80 years ago since there is virtually no chance of exposure going forward.

Today, the number of persons who perform repairs on equipment with packing, which was installed before 1975, must be nearly zero. Packing is usually replaced every 2-5 years (depending on the application). Therefore, surely the last plausible exposures must have been around 1985 or 1990 (over 30 years ago). Additionally, asbestos was used infrequently in packing after about 1975.

I saw no information in the Agency's document, which would contradict my experience and these opinions. Again, in consideration of the significant cost of asbestos litigation activity in the United States, there can be no reasonable expectation by the Agency, or any other party, that such future "new" use of asbestos-containing material would occur in this country.

Statement 2: "The preliminary conclusions, findings, and determinations in this draft risk evaluation are for the purposes of identifying whether asbestos presents unreasonable risk or no unreasonable risk under the conditions of use, in accordance with TSCA section 6, and are not intended to represent any findings under TSCA section 7." (page 17, ln. 674 - 677).

Comment 2: This statement is consistent with my understanding of TSCA. It seems that EPA only has some authority to regulate or ban materials that are currently in commerce and for which there is ongoing exposure. Even though this document states on numerous occasions that exposures through new products are occurring and will continue to happen. This is simply not true, except at that single gasket stamping company (employing four persons) and the chlor-alkali industry. As described by EPA, the exposures at these facilities are well controlled, and the employees appear to be using PPE when needed.

Perhaps there are some installations of asbestos-containing brakes from overseas by that single firm which EPA identified that makes cars for use and sale abroad (I expect that this might involve race cars for which asbestos might be a desirable component in the brakes). As I will discuss, if this exposure is associated with the production of a race car, there will be no exposure because it involves the installation only, and arcing is not needed on a new vehicle if it has a drum brake.

As described in the document, there are some albeit well-controlled exposures in the Chlor-alkali industry. However, based on the data presented for that industry, no exposures are approaching the current OSHA PEL. Because of the current COVID-19 crisis, this would not seem to be a prudent time to

be placing even greater constraints on an industry that produces the most widely used disinfectant, chlorine.

In consideration of the current considerable cost of asbestos litigation activity in the United States, it is reasonable to assume that the few remaining companies noted as using asbestos-containing material are only doing so out of critical need. They almost certainly are continuing their attempts to find a replacement for asbestos (not out of fear of overexposure to asbestos but rather the fear of litigation).

As noted above, the current draft conclusions that any/all of these uses present an unreasonable risk to health is unjustified based on the very data provided in this EPA document. Trial lawyers continue to put forward an argument that exposures in some occupations between 1950 - 1980 posed some degree of incremental risk to some persons (reasonable persons can choose to disagree). However, these claims are entirely unrelated to the American workforce going forward since the opportunities for exposure to asbestos have been virtually eliminated.

Concerning the discussion about brake blocks in the oil fields, I found no information in the document that brake blocks for these rigs have contained asbestos over the past 35 years. I checked a few suppliers. Even if some do continue to contain chrysotile which is not discussed in their MSDS, it seems that this analysis is not relevant to the Lautenberg initiative. Also, if it were, given the handful of persons who could be potentially exposed and the low level of possible exposure, these data indicate that any regulatory action is unwarranted (especially if there is some concern about the potential failure of replacement materials that might not contain asbestos (e.g. might cause an injury or fatality).

I do have a concern that this document is intended to support, under the Lautenberg Initiative, an attempt by EPA to ban the import of all asbestos-containing products. I have supported such a ban for many years, except for the use of asbestos in those few applications where there is no plausible replacement. Fortunately, all of these few remaining applications of which I am aware, involve products made only with encapsulated chrysotile asbestos.

It is nearly impossible, in the modern era, to have worker exposures exceed $1/10^{th}$ or ½ of the numerical OSHA PEL when working with these encapsulated products. Also, the duration of exposure is usually minutes per year, rather than hours or days; therefore, the 8-hour time-weighted average of exposures to brakes or gaskets (going forward) is vanishingly small.

It is my understanding that there may be specific needs by the military (like balloon gaskets used on high-pressure lines in aircraft like the Apollo) that are critical use applications, and apparently require encapsulated chrysotile products. Those items are never manipulated, and even if they were, they would likely be unable to release airborne asbestos in concentrations anywhere close to the OSHA PEL (and quite often, they are immeasurably small).

Statement 3: "To meet these TSCA § 26 science standards, EPA used the TSCA systematic review process described in the Application of Systematic Review in TSCA Risk Evaluations document (U.S. EPA, 2018a)." (page 17, ln. 681 -683).

Comment 3: I applaud the Agency for choosing to implement the systematic review process; however, I have found that perhaps as many as 90 published papers that are relevant to this risk evaluation were

not cited. With that in mind, it is clear to me that there were significant shortcomings in the application of this systematic review.

After reading these comments, the SAB and EPA staff can decide if this observation is correct, but in any event, a proper systematic review should have identified all relevant published papers, and it did not.

Statement 4: "Under TSCA, EPA has promulgated several regulations for asbestos, including the Asbestos Ban and Phase Out rule of 1989, which was then largely vacated in 1991, and under the Asbestos Hazard Emergency Response Act (AHERA), which requires inspection of schools for asbestos." (Page 17, ln. 694 - 697)

Comment 4: As mentioned previously, in light of EPA's disappointment in having their asbestos ban vacated in 1991, it should come as no surprise that readers may believe that the sole purpose of writing this risk assessment is EPA's renewed attempt to ban all uses of asbestos. As I previously stated, I support a general ban, albeit with exceptions for the rare circumstances noted above.

If EPA still desires a ban, there surely must be methods other than this document to achieve this outcome. Indeed, the current asbestos litigation in the U.S. has essentially banned asbestos in new products and has limited continuing uses to only those critical use applications identified above.

Statement 5: "Based on 2019 data, the total amount of raw asbestos imported into the U.S. was 750 metric tons. The asbestos-containing products that EPA has identified as being imported and used are sheet gaskets, brake blocks, aftermarket automotive brakes/linings, other vehicle friction products, and other gaskets." (Page 17, ln. 706 – 709; page 18, ln. 710 - 711).

Comment 5: Based upon what I could find in this document, there is a modest amount of documentation that supports EPA's belief that 750 metric tons of raw asbestos are imported. But it very well may be true. This represented more asbestos than I have believed was required by this industry.

Based on the Agency's document, virtually all of the raw asbestos imported is used to create diaphragms for the chlor-alkali industry. The description of the use of raw asbestos (pages 61 – 65) is informative in this regard. If accurate, based on the data presented on pages 66 – 68, almost none of the approximately 400 persons identified in Table 2-7 are exposed above the OSHA PEL (Table 2-4, 2-5 and 2-6). And, if these data were segregated by decade, for example, if full shift breathing zone samples were to be examined after 1990, it is likely no one would have had exposures over the current OSHA PEL for the past 30 years. For that reason, I am surprised the Agency concluded that these workers would perform activities in the coming years that place them at "unreasonable risk to health" (page 26).

Indeed, as Table 2-5 shows, the 650 samples collected between 1996 and 2017, even at the 95th percentile of exposures, the concentrations were at 0.050 fibers/cc. Based upon my 40 years of reviewing industrial hygiene data, it is reasonable to assume that a significant fraction of those 650 samples were at non-detected concentrations and that the 95th percentile value is driven by substituting ½ the Limit of Detection (LOD) for those Non-Detects.

In those situations where a significant fraction of the data are "ND" (i.e., considered censored data), it is not appropriate to simply substitute ½ the LOD or the LOD divided by the square root of 2 when

presenting these kinds of summary statistics. Many times over the years, I have seen data sets where 90% of the data were below the limit of detection (basically no detectable exposure for any worker), but when the ½ LOD value was inserted, and the risk goals were 1 in 1,000,000, it was concluded that the risks were unacceptable (when the opposite was true). Such erroneous conclusions can result if the responsible person does not look carefully at the raw data.

For this reason, special statistical analyses are used for highly censored data. EPA has issued guidance in this regard, and it would be useful for them to apply that approach to the data from the chlor-alkali industry. EPA recommends, and I also support, many of the approaches recommended by Dr. Helsel over the past 30 years.

Interestingly, it is not clear why Table 2-4 has 11 f/cc as the maximum result when, as noted in the footnote, it should read 0.019 fibers/cc. This incongruency is perhaps just a typographical error that needs to be corrected.

The data presented in this document suggests that the exposures to raw asbestos in that industry are remarkedly well-controlled and are certainly are not of regulatory interest. Again, all the information presented by the Agency for this cohort indicates that these data are inconsistent with the current draft claim that (A) Processing and Industrial use of Asbestos Diaphragms in Chlor-alkali Industry, and (B) Processing and Industrial Use of Asbestos-Containing Sheet Gaskets in Chemical Production "present an unreasonable risk to health" (as stated in this document). Based on the available data, they do not, unless one embraces this newly proposed chrysotile cancer potency factor (CPF).

I was surprised that the Agency did not follow historical protocol and its guidelines for setting a CPF. Usually, EPA would run this proposal through the IRIS program; if that occurred, it is not stated in this document. If I am correct, one might be suspicious of an attempt by EPA to embed a very controversial regulatory decision (a new CPF) within an alleged TSCA related exposure assessment or risk assessment. I think it would be wise to obtain a view from the IRIS group and OMB on the CPF, so they could conduct an economic impact assessment (if this has not already been performed).

Approach

Statement 6: "EPA used reasonably available information (defined in 40 CFR 702.33 as "information that EPA possesses, or can reasonably obtain and synthesize for use in risk evaluations, considering the deadlines for completing the evaluation"), in a fit-for-purpose approach, to develop a risk evaluation that relies on the best available science and is based on the weight of the scientific evidence." (page 18, ln. 716 - 719)

Comment 6: It is unclear to me what this sentence is intended to tell the reader. However, if one were to look at the historical documents of the past 20 – 30 years, I think it would be apparent to the Agency that there is virtually no opportunity for exposure to chrysotile asbestos in the coming years in a consumer product.

On page 50, it is noted that "For the data sources that passed full-text screening, EPA evaluated their quality and each 1713 data source received an overall confidence of high, medium, low or unacceptable." Further, in section 2.3.1.6 Oil Field Brake Blocks, the single study considered by EPA was rated "low" in the systematic review. While it was not rated "unacceptable," the limitations of

this study that might have been only 2 or 3 samples suggest the minimal nature of this data. It is difficult to see how EPA could rely on such flimsy data as “fit-for-purpose” or “best available science” or rely on such lightweight “weight of the scientific evidence.”

If the agency questions my professional belief that there are no exposures to be anticipated in the coming years for more than a handful of people in the United States, I believe that this information can be submitted by the private sector during a reasonable comment period by informed parties (which I believe will be available after the public meeting of SAB).

Informed parties have evaluated this question for the past 30-50 years. The past 40 years of asbestos-related litigation has brought millions of corporate documents into the public sector, to which the Agency may or may not have access. If requested, I am confident that it will be forthcoming.

As stated above, it is my view that there are at least 90 published papers or reports that are relevant to this risk evaluation that were not cited. With that in mind, I fear that there are significant shortcomings in the application of this draft systematic review. After reading my comments, the SAB and EPA staff can decide if this observation is correct. In any event, this document should include all relevant published papers.

The following is a partial list of those that I suggest adding to the review:

Epidemiologic Studies or Manuscripts Involving Vehicle Mechanics and Asbestos-Related Disease

1. Mcdonald and Mcdonald, 1980;
2. Agudo et al., 2000;
3. Coggon et al., 1995;
4. Hessel et al., 2004;
5. McElvenny et al., 2005;
6. Milham and Ossiander, 2001;
7. NIOSH, 2002;
8. Olsen and Jensen, 1987;
9. Petersen and Milham, 1980;
10. Rake et al., 2009;
11. Rolland et al., 2005;
12. Rolland et ak., 2010;
13. Spirtas et al., 1985;
14. Teschke et al., 1997;
15. Teta et al., 1983;
16. Benhamou et al., 1988;
17. Consonni et al., 2010;
18. Corbin et al., 2011;
19. Gustavsson et al., 1990;
20. Hrubek et al., 1992;
21. Lerchen et al., 1987;
22. MacArthur et al., 2009;
23. Morabia et al., 1992;
24. Vineis et al., 1988;
25. Williams et al., 1977;

26. Boillat and Lob, 1973;
27. Coggon et al., 1995b;
28. Elliehausen et al., 1985;
29. Marcus et al., 1987;
30. Albin et al., 1990;
31. Berman and Crump, 2003;
32. Hodgson et al., 2005;
33. Lacquet and van der Linden, 1980;
34. McDonald, 1984;
35. Yarborough, 2007, 2006;
36. Dement, 1972;
37. Johnson et al., 1979;
38. Roberts 1980a, 1980b;
39. Roberts and Zumwalde, 1982;
40. Moore, 1988;
41. Rodelsperger et al., 1986;
42. Plato et al., 1995;
43. Aguilar-Madrid et al., 2010;
44. Dubrow and Wegman, 1984;
45. Enterline and McKiever, 1963;
46. Finkelstein, 1995;
47. Gustavsson et al., 1990;
48. Hansen, 1989;
49. Hansen, 2003;
50. Hodgson et al., 1997;
51. Jarvholm and Brisman, 1988
52. Kelsh et al., 2007;
53. Laden et al., 2004;
54. Leigh, 1996;
55. Menck and Henderson, 1976;
56. Merlo et al., 2010;
57. Milham and Ossiander, 2010;
58. Milne et al., 1983;
59. Peto et al., 2009;
60. Roelofs et al., 2013
61. Rushton et al., 1983;
62. Schwartz, 1987;
63. Spirtas et al., 1994;
64. Swanson and Burns, 1993;
65. Tomasallo et al., 2018;
66. Van den Borre and Deboosere, 2015;
67. Wong, 2001
68. Berry and Newhouse, 1983;
69. Browne and Smither, 1983;
70. Clin et al., 2011;
71. Finkelstein, 1989;

72. Finkelstein and Meisenkothen, 2010;
73. Kogan et al., 1993;
74. Newhouse et al., 1982;
75. Newhouse and Sullivan, 1989
76. Pang et al., 1997;
77. Skidmore and Dufficy, 1983;
78. Szeszenia-Dabrowska et al., 2015

Relevant Papers Involving Health Effects Regarding the Removal/Installation of Brakes or Gaskets

1. Balzer, 1968;
2. Balzer and Cooper, 1968;
3. Cheng and McDermott, 1991;
4. Federal Register, 1994, 1972;
5. Lindell, 1973;
6. Marr, 1964;
7. Selikoff, 1970;
8. Selikoff et al., 1979;
9. Liukonen et al., 1978;
10. Jones, 1981;
11. Anderson et al., 1982;
12. CONSAD Research Corporation and Clayton Environmental Consultants, Inc., 1984;
13. Boelter and Spencer, 2003;
14. Longo et al., 2002;
15. Madl et al., 2007;
16. McKinnery, Jr. and Moore, 1992;
17. Millette and Mount, 1993;
18. NIOSH, 1994;
19. Spencer, 1998a,
20. Spencer, 1998b
21. Williams et al., 2007

Statement 7: "EPA reviewed the information and evaluated the quality of the methods and reporting of results of the individual studies using the evaluation strategies described in Application of Systematic Review in TSCA Risk Evaluations (U.S. EPA, 2018a)." (page 18, ln. 722 - 724)

Comment 7: I am not sure of the processes used by EPA to evaluate the quality of the methods; however, I will say that this document is among the most thoroughly transparent analyses that I have read in my 40-year career as a risk assessor. I applaud the exposure assessors for "grinding through" all of these exposure scenarios — congratulations to the group who did this.

Given the key papers not considered by EPA, it is clear to me that there were significant shortcomings in the application of this systematic review. After reading my comments, the SAB and EPA staff can decide if this observation is correct.

Statement 8: "EPA evaluated exposures (inhalation only) to asbestos in occupational and consumer settings to estimate risk of health hazard (cancer only) for the COUs in this draft risk evaluation." (page 19, ln. 755 - 756)

Comment 8: It appears that EPA did a reasonably good job in the vast majority of its evaluations regarding exposures to asbestos. Unfortunately, many good papers were missed. So, the exposure assessors could not do a great job in the assessment. Also, no weight was given to the fact that the exposures were to occur in the coming years. I may not agree with all of the Agency's assumptions, which has many effects on the conclusions, but I appreciated the care in its execution. The problem with the document is not the exposure assessment; it is the assumptions about exposure duration and frequency, the appropriateness of their new cancer potency factor, and the number of exposed persons that I find problematic (and fundamentally unfounded scientifically).

Statement 9: "EPA used the Framework for Human Health Risk Assessment to Inform Decision Making (U.S. EPA, 2014a) to evaluate, extract, and integrate asbestos' dose-response information. EPA evaluated the large database of health effects associated with asbestos exposure cited in numerous U.S. and international data sources." (page 19, ln. 767 - 770)

Comment 9: The process through which the Agency applied this guidance document to derive a cancer potency factor (CPF) is unclear to me, and it is certainly inadequate. As noted previously, I think it would have been appropriate to have run this proposal for a new (and novel) CPF for chrysotile only through IRIS.

I was surprised that there was not an exhaustive discussion of the appropriateness or inappropriateness of relying on historical epidemiology studies of chrysotile workers (textile workers in particular). However, almost certainly, it was not appropriate to rely exclusively on the North Carolina and South Carolina cohorts. The supporting document to the proposal, entitled "DRAFT evaluation for Asbestos: Systematic Review Supplemental file: Data Quality Evaluation of Human Health Hazard Studies: Mesothelioma and Lung Cancer Studies" (March 2020) is good for what it attempted to do. Like the exposure assessment, the discipline and patience of the staff involved in writing this supplement are extraordinary.

It is essential that EPA consider the meta-analyses of epidemiological studies of the auto mechanics (i.e. Wong, 2001; Goodman et al., 2004; Pierce et al., 2008; Garabrant et al., 2016). Other relevant cohorts are described by Berman and Crump, 2008. There are some little known facts about the Balangero cohort in Italy that deserve attention (Pierce et al., 2016; Piolatto et al., 1990; Pira et al., 2009). I am hopeful that those who know them will submit comments.

I am not sure that the Chongqing (China) cohort is worthy of much attention (Deng et al., 2012; Courtice et al., 2016; Pierce et al., 2016). Some have claimed that this study has too many shortcomings to be reliable. I will hope someone else offers comments on this topic.

The North Carolina Cohort

As readers will find, the EPA proposed cancer potency factor for chrysotile was based almost entirely on the studies of North and South Carolina textile mills (Dement et al., 2009; Loomis et al., 2009; Elliot et

al., 2012). In my view, when assessing the carcinogenic potency and toxicity of chrysotile, one needs to appreciate the importance of fiber length (among other things).

Fiber dimension has long been recognized as a likely contributor to defining the potency of asbestos, regardless of fiber type. Since the late 1940s, a substantial amount of research has been conducted in which fiber length has been either directly or indirectly considered. Specifically, it has been known since the early 1970s that long, thin fibers present a greater risk of disease than shorter fibers (Stanton, 1973; Stanton et al., 1977; Lippmann, 1988; Eastern Research Group, Inc., 2002, 2003a; ATSDR 2002).

Biological mechanisms in the lung allow for faster and more efficient clearance of short fibers compared to long fibers. Retention of the long asbestos fibers in the lung is believed to lead to the development of disease (ATSDR 2002; ERG 2003a; Berman and Crump, 2008a, 2008b).

In 2002, The Agency for Toxic Substances and Disease Registry sponsored the convening of an expert panel to address the influence of fiber length on asbestos-related health effects (ATSDR 2002). These panelists were asked to comment on the physiological fate, as well as non-carcinogenic and carcinogenic health effects associated specifically with asbestos and synthetic vitreous fibers less than 5 µm in length. Overall, the ATSDR-sponsored "panelists agreed that there is a strong weight of evidence that asbestos and short vitreous fibers shorter than 5 µm are unlikely to cause cancer in humans" based on "findings from epidemiologic studies, laboratory animal studies, and in vitro genotoxicity studies, combined with the lung's ability to clear short fibers" (Eastern Research Group, Inc., 2003b). As noted previously, the U.S. EPA contracted the preparation of a technical support document to establish a protocol to assess asbestos-related (Berman et al. 2003). Based on modeling results presented in the technical support document, Berman and Crump concluded that the best estimate of risk for mesothelioma for fibers between 5 and 10 µm in length was one three-hundredth of the risk assigned to fibers longer than 10 µm. The authors explained that "results from [their] review of the supporting literature suggest that the optimum cutoff for increased potency occurs at a length that is closer to 20 µm [rather] than 10 µm." Further, Berman and Crump (Berman et al. 2003) reported that the best estimate of the potency of fibers shorter than 5 µm for mesothelioma is zero. A second expert panel convened by the U.S. EPA agreed that the risk associated with fibers less than 5 µm in length is "very low and could be zero" (Eastern Research Group, Inc., 2003c).

Importance of Cumulative Dose

As noted by Pierce et al. (2008, 2016) and many others, the lifetime cumulative dose is critical to understand the increased lifetime cancer risk. I am not confident that enough care was invested in understanding the uncertainties in the exposure estimates presented by Dement et al., 2009. Tables two and three from that article contain a plethora of information and a considerable amount of uncertainty. Some critical information is missing (at least from what I could determine). For example, a key element of Table 3 is the number of None Detected samples that were observed for each exposure zone. As noted previously, this can be important; although for this cohort, it appears that there was an ample number of samples with detectable concentrations.

The biggest shortcoming in the Systematic Review of the epidemiologic studies is the lack of acknowledgment of the importance of the mechanic studies (see articles listed above). To understand the relevant asbestos exposures or risks associated with working with gaskets, brakes, and packing, I would argue that the textile workers are an inappropriate cohort. This is because they were exposed to

asbestos with a different fiber length and product composition than workers exposed to asbestos in gaskets, brakes, and packing. In these encapsulated products, the asbestos fibers are shorter and have been soaked in a hard-phenolic resin. I recommend to the Agency that they include the relevant articles I mentioned above, and the ones cited in the Garabrant et al. (2016) paper to understand the epidemiology of these relevant cohorts.

It is unclear to me why the Agency did not include many of the relevant toxicological and epidemiological studies regarding asbestos fibers in gaskets, brakes, and packing, especially given the vast body of work that has been performed on these fibers. In any event, as a result of this significant deficiency, there is virtually no support for the conclusions of this draft risk characterization that relies almost exclusively on the new CPF. This CPF was improperly based on a study of a textile mill cohorts (which involves exposure to inordinately long asbestos fibers that were not soaked in resins). Additionally, it is worth repeating that at least some of these workers were also exposed to amphiboles during their careers.

Although I will discuss this in more detail in later comments, I hope that it is apparent to the Agency that textile fibers are often greater than 20, 30, and 40 microns in length (well described in the Dement et al., 2009 article). The toxicology studies, as well as some of the lung burden data, clearly show that these very long fibers are the only ones that might have the potential to cause mesothelioma and are probably the primary driver for lung cancer (Berman and Crump, 2003 and others). I believe the article by Hodgson and Darnton, (2000) supports the view that the risk of exposure to raw long fiber chrysotile is much different than exposures to short-fiber chrysotile, especially compared to those soaked in phenolic resin.

Some have proposed fairly elegant and novel hypotheses about why these long fibers in the textile industry “might” be able to cause mesothelioma due to clogging up the exit portals of the lung and causing black spots in that location, because the fibers are too long to get through the portal (Donaldson et al., 2010; Barlow et al., 2017).

It is understandable that unless the Agency scientists have spent a lifetime studying asbestos, that the myriad of subtleties and complexities associated with understanding chrysotile potency would not be fully understood. For example, the fibers of interest for block brakes, automobile brakes, elevator brakes, conveyor brakes, and virtually all other asbestos-containing brakes before 1985 are almost all shorter than five microns in length and almost certainly lack biological activity. The lack of biologic activity of phenolic filled fibers was hypothesized by Langer, 2003 and his hypothesis was later proven to be accurate in a series of studies published by Bernstein and Hoskins, (2006) and Bernstein et al. (2013, 2018). Neither the Langer paper nor the three Bernstein studies are cited in this document. As an aside, the Bernstein papers were published in the top-rated toxicology research journal in our profession.

As noted, in my view, there are four significant meta-analyses of cancer risks to vehicle mechanics who were involved with brake repair that were not cited (Wong, 2001; Goodman et al., 2004; Hessel et al., 2004; Garabrant et al., 2016). These meta-analyses evaluated the epidemiology studies which evaluated vehicle mechanics during the era when asbestos was present in some brakes. They also clearly showed that asbestos exposure from brakes before 1985 (when 70% of these products contained asbestos) had no increased risk of mesothelioma or lung cancer in those cohorts. To get a good understanding of these critical papers, I suggest a careful review of Garabrant et al. (2016), which is a meta-analysis of all key papers.

See the following papers:

1. Mcdonald and Mcdonald, 1980;
2. Spirtas et al., 1985;
3. Teta et al., 1983;
4. Goodman et al., 2004;
5. Rake et al., 2009;
6. Aguilar-Madrid et al., 2010;
7. Merlo et al., 2010;
8. Rolland et al., 2010;
9. Roelofs et al., 2013;
10. McElvenny et al., 2005;
11. Health & Safety Executive, 2013;
12. Milham,2011;
13. NIOSH, 2011
14. Hessel et al., 2004;
15. Teschke et al., 1997;
16. Agudo et al., 2000;
17. Hansen et al., 2003;
18. Woitowitz and Rödelsperger, 1994

What do the meta-analyses of mechanics tell us?

I believe that the Agency will find that the authors of the four meta-analyses found no increased risk of lung cancer or mesothelioma when these studies were carefully evaluated. When you couple that observation with the historical data that I have presented in several publications (which suggests that these workers were likely exposed on average to 0.04 f/cc; a mean value to which there is broad agreement including within the EPA), one would infer that this represents a no-observed effect level for chrysotile fibers. Note that EPA indicated that a good best estimate of the 8-hour TWA exposure of mechanics during the years that brakes contained asbestos is 0.04 f/cc (Weil, 1985), which is similar to the concentration identified in Paustenbach et al. (2003).

On a lifetime basis, this should represent a practical threshold for chrysotile asbestos from an encapsulated material (e.g., 40 years x 0.04 f/cc = 1.6 f/cc-years). Given the robust nature of these data , one would not need to turn to the linearized multistage (LMS) model and apply it to entirely different exposure scenarios (i.e., the textile cohorts in North and South Carolina), to derive a new CPF for chrysotile. When many epidemiological studies yield no increased risk and you understand the magnitude of exposure, it would seem that these data would be considered reliable.

The LMS is primarily intended for genotoxic carcinogens and not those that act through a mechanism such as repeated cytotoxicity which is almost certainly the mechanism for chrysotile to produce mesothelioma (producing one type of inflammation in a portion of the pleura) and, probably, for lung cancer (producing a different type of inflammation in a different area of the lung) (Lynn, 1983). Given chrysotile's lack of genotoxicity and robust epidemiological data, the LMS model is almost certainly not appropriate. Many toxicologists have argued that safe levels of exposure can be derived by the classic safety factor approach applied to a genuine or apparent human NOEL (f/cc-year).

It may be noted by trial lawyers, who will likely be submitting comments that all the meta-analyses were conducted by experts who have testified on behalf of defendants. That is true, but I would hope that the Agency will weigh the quality of the work and the fact that they survived peer review in journals with solid impact factors. Rather than rely on innuendo that these papers are biased or flawed, due to the source of funding, I would expect the Agency to weigh them exclusively on their scientific merit.

As noted, it is not surprising that these epidemiological studies showed no increased risk of cancer or mesothelioma because, as stated above, these fibers have been soaked in phenolic resin and/or many have had their chemical composition altered by the intense heat associated with braking. EPA is correct on noting that "dose is everything." As emphasized throughout these comments, chrysotile potency for mesothelioma and/or lung cancer is dictated by dose, fiber type, and length (also, aspect ratio) and the doses of chrysotile to be considered carcinogenic need to approach 50-400 f/cc-years (Pierce et al., 2016)

As discussed at the Monticello meeting two years ago, where 60 of the top asbestos scientists met for three days, it was generally concluded that chrysotile would not increase the risk of lung cancer until doses are sufficient to produce asbestosis. These papers are presented in a special edition of Toxicology and Applied Pharmacology (The Monticello Conference on Elongated Mineral Particles, December 2018). None of those papers are cited in this document and I would urge EPA to review them.

My views about chrysotile and mesothelioma

Concerning mesothelioma, I have said for years, as have many epidemiologists, pathologists, toxicologists, occupational hygienists, and physicians, that it remains unclear that pure chrysotile can increase the risk of mesothelioma.

This is described in an elegant manner by Hodgson and Darton (2005), which is surprisingly not cited in this document. In their seminal paper, they say that they cannot rule out that chrysotile has no potency for mesothelioma (e.g., may not have the capacity to produce mesothelioma by itself). Since then, I believe that the bulk of the scientific community has concluded that we cannot rule out the possibility that chrysotile may not be able to cause mesothelioma when long fiber chrysotile are involved, such as that reported by Dement et al. (2009). The basis for the belief by some that long fiber chrysotile might pose a mesothelioma hazard is described by those who have studied the stoma which they believe might explain why persons exposed in textile mills might at greater risk of mesothelioma. This is due to the inhalation of high doses of chrysotile fibers longer than 30 µm (e.g., black spots) (Wang, 1975; Boutin et al., 1996; Mitchev et al., 2002; Craighead, 2008; Donaldson et al., 2010; Roggli and Sharma, 2014).

I think that the Agency would benefit from a careful review of Pierce et al. (2008; 2016), as well as several other relevant papers that I have brought to their attention. Numerous papers suggest that only at asbestosis producing lifetime doses might there be a possible increased risk for lung cancer and mesothelioma for chrysotile. These doses are more than 100-fold greater than the historical exposures of auto mechanics who worked with asbestos-containing brakes.

Exposure of Auto Mechanics

Many papers have attempted to characterize the exposures and associated risk of mechanics through 1985. Due to the relatively strict OSHA regulation passed in 1994 on asbestos, that is still in effect today, and due to the truly voluminous and costly litigation that has surrounded asbestos for more than 35 years, there is virtually no chance that exposures of significance have occurred since then in the United States. This is supported by the very data presented in this EPA document.

Therefore, it is entirely unclear to me why the Agency, through its responsibility for administering the Toxic Substances Control Act, which is supposed to anticipate and prevent future exposures, would assemble such a complex document as this draft (given the lack of exposure of Americans for the past 30+ years). Such exposures will never occur again in the United States, except for what I estimate to be approximately 100 persons identified in this document, who may be involved in critical use applications that will continue to diminish over time. As noted in this EPA document, these types of exposures have been studied, and such exposures appear to be already well-controlled, as identified in this document.

New Cancer Potency Factor

It is recognized that, in this document, when EPA applies their new cancer potency factor (CPF), they calculate a risk that is greater than 1 in a million, and sometimes more than one in ten thousand, for those who handle gaskets and install brakes.

However, because the basis for the CPF is seriously flawed (as it is based upon fibers from amosite contaminated textile mills, and not encapsulated fibers from brakes, clutches, or gaskets), I would suggest that the risk estimates for pipefitters and millwrights (gaskets), vehicle mechanics (brakes) and millwrights (packing) are not accurate. Beyond missing the importance of phenolic resin filled fibers with respect to biological potency, the Agency has adopted a novel approach where they combine the mesothelioma risk and the lung cancer risk to derive their new proposed CPF. In my view, this raises serious questions regarding the accuracy of the risk estimates for the six different exposure groups which they studied.

All the information presented here suggests to me that this document will require a great deal of work before it would yield credible estimates of risk for the workers who have been identified by EPA. I believe that this document may need to be "tabled" because, in addition to many shortcomings, the number of potentially exposed persons in the coming years is well below that of regulatory significance.

Even after one considers what I believe are shortcomings in the analyses, it is surprising that EPA management and the Office of Management and Budget (OMB) would conclude that perhaps millions of dollars of effort were appropriately invested in this endeavor. I hold this view partly because the document assumes that exposures will exist going forward in time (and there is no credible evidence to support this belief), and even if they did, if one assumes ten thousand persons are exposed, using a proper CPF, the Agency would conclude that in the United States no one working in these identified scenarios would develop a single (one) case of cancer due to these exposures.

To the best of my knowledge, and based on my review of historical regulations, there has never been a regulatory or societal concern about exposure scenarios that do not produce a theoretical risk of producing a single incidence of cancer in the exposed population (Travis and Hattemer-Frey, 1988).

Statement 10: "The IUR for asbestos developed in 1988 was based on 14 epidemiologic studies that included occupational exposure to chrysotile, amosite, or mixed-mineral exposures [chrysotile, amosite, crocidolite]." (page 19, ln. 778 - 780)

Comment 10: Yes, the IUR developed in 1988 was based on a blend of exposures to various forms of asbestos. As noted by the Agency (and OSHA), using such an approach is not appropriate to understand the cancer risks posed by chrysotile alone. As identified above, the studies used by the Agency in this assessment to estimate the potency of chrysotile alone are not the appropriate ones.

As previously mentioned, it is my view that the Agency has conceptually approached this evaluation in an improper scientific manner by attempting to combine lung cancer epidemiological studies with mesothelioma epidemiological studies to derive a cancer potency factor (CPF). I am not aware of the Agency proposing such an approach in the past and do not believe that it is appropriate here.

The following are some of the reasons why I think the Agency needs to rethink its approach to estimating the cancer potency for chrysotile:

1) The studies of lung cancer and exposure to chrysotile are almost certainly confounded by the inadequate smoking history of the persons in the cohorts. It is well known, for example, that persons often fail to report when they smoked for one to five, or two to ten years in their lifetime (sometimes because it is socially unattractive, sometimes before it heavily influences their insurance rates and sometimes because they forget). This is particularly important when studying lung cancer because smoking is known to be a vastly more potent lung carcinogen than chrysotile. In short, if you do not have a good smoking history, the results of an epidemiological study involving lung cancer are unacceptably confounded. There are some techniques for attempting to correct for smoking when the smoking history is unknown; however, those can be flawed if there is a significant difference between typical persons in the control group vs. those in the exposed cohort. As discussed in Goodman et al., 1999, there can be considerable heterogeneity in smoking habits between occupational groups and the controls.

2) It is generally believed that chrysotile cannot cause lung cancer unless the lifetime cumulative doses approach those that cause asbestosis. I do not understand why the Agency would consider it appropriate to combine chrysotile potency, which is dependent upon fiber length and heavy doses (which causes chronic inflammation) with an entirely separate disease (mesothelioma). These distinct diseases almost certainly act through different mechanism(s) of action. This is not scientifically appropriate, and it is contrary to the EPA's Cancer Risk Assessment Guidelines (EPA, 2005).

3) It is entirely possible, based upon several extensive evaluations of the epidemiology data, that pure chrysotile does not cause mesothelioma at any plausible dose. If it does, it is most likely to occur only in the cohorts of textile workers, which were studied in North and South Carolina (Dement et al., 2009; Loomis et al., 2009; Elliot et al., 2012). This is because they

were primarily exposed to very long chrysotile fibers, and many of the doses were at high concentrations and were the cause of asbestosis in many workers. Additionally, the use of crocidolite is well documented in the South Carolina textile plant. Between 1950 and 1975, approximately 2000 pounds of crocidolite yarn was used in this plant to make tape or braided packing, compared to the six to eight million pounds of chrysotile that were used annually (Dement, 1980; Dement, 1983; Hein, 2007).

4) As Dr. David Garabrant noted in his submitted comments to the Agency, "These cohorts are not ideal for claiming that they were only exposed to chrysotile. There is evidence that some or many of these workers were exposed to amosite at different times in their careers. In a paper by Dement et al. (2009), they acknowledged that in Plant #3, "amosite was carded, twisted and woven between approximately 1963 and 1976." In 1989, Sebastien et al. conducted a fiber analysis of the lung tissue of workers from the South Carolina Textile plant cohort. Seventy-two lung specimens were obtained for analysis. The authors found "non-trivial concentrations (>0.1 f/µg)" of amosite and crocidolite in 32% of the samples. Commercial amphiboles were found only in samples of employees who were hired before 1940; there was no crocidolite detected in those who worked at the textile plant after 1940 (Sebastien et al., 1989). In 1997, Green et al. confirmed these results when 28% of the textile workers' lung samples that were analyzed were found to contain amosite and/or crocidolite (Green, 1997). In a subset of lung tissues derived from the above cohorts used by Sebastien and Green, Case analyzed 64 samples and reported results similar to both previous studies, that 32% of the fibers analyzed from the textile cohort samples were amosite and crocidolite. Similar to Sebastien, these workers were found to have stopped working in the textile plant between 1938 and 1947 (Case, 2000)."

Therefore, using the epidemiology studies for North and South Carolina textile workers is inappropriate for assessing the possible cancer hazard of persons exposed to brake wear debris, clutch debris, or encapsulated airborne particles from gaskets (encapsulated) or packing (which has been soaked in an oily lubricant). The vast majority of fibers (by weight) from these exposures involves fibers that are less than ten microns in length and are filled with some type of polymeric material. These exposures are in no way analogous or even vaguely similar to exposure to raw, long chrysotile fibers found in the textile mills.

Numerous animal studies have clearly shown that chrysotile fibers shorter than five microns lack biologic activity, and those between five and ten microns, may also not be potent enough for causing lung cancer or mesothelioma (Berman and Crump, 2003; Hodgson et al., 2005a; Berman and Crump, 2008a). Most exposures associated with the historical work with asbestos from brakes and gaskets involves fibers less than five microns in length. Asbestos exposure becomes a health concern only when relatively high concentrations of asbestos fibers (compared to background) are inhaled. It has been stated by Churg et al. that at the very least, doses of chrysotile required to produce asbestosis (25 f/cc years to 100 f/cc years) are necessary to cause lung cancer and, perhaps, mesothelioma (Churg, 1988; Churg et al., 1993).

Moreover, Pierce et al. summarized the NOAELs reported in the literature for predominately chrysotile-exposed cohorts and found that the preponderance of the cumulative chrysotile NOAELs for

mesothelioma fell in the range of approximately 15 to 500 f/cc-years (Pierce et al. 2008). Recent analyses have suggested that chrysotile may not even be a risk factor for mesothelioma at any reasonable airborne fiber concentration (Hodgson et al. 2005; Yarborough 2006; Eastern Research Group, Inc., 2003).

Another factor that appears to have a significant impact on the likelihood of developing an asbestos-related disease, especially for chrysotile, is the fiber length. Most experts, virtually all scientific bodies, and OSHA have concluded that only fibers which are greater than 5 µm in length are biologically significant. For chrysotile, recent research indicates that fibers may need to approach or exceed 20 µm in length for them to have carcinogenic potential (Eastern Research Group, Inc., 2003).

In 2002, The Agency for Toxic Substances and Disease Registry sponsored the convening of an expert panel to address the influence of fiber length on asbestos-related health effects (ATSDR, 2002). These panelists were asked to comment on the physiological fate, as well as non-carcinogenic and carcinogenic health effects associated specifically with asbestos and synthetic vitreous fibers less than 5 µm in length.

Overall, the ATSDR-sponsored panelists agreed that "there is a strong weight of evidence that asbestos and short vitreous fibers shorter than 5 µm are unlikely to cause cancer in humans" based on "findings from epidemiologic studies, laboratory animal studies, and in vitro genotoxicity studies, combined with the lung's ability to clear short fibers" (Eastern Research Group, Inc., 2003b). As noted previously, the U.S. EPA contracted the preparation of a technical support document to establish a protocol to assess asbestos-related disease (Berman et al. 2003). Based on modeling results presented in the technical support document, Berman and Crump concluded that the best estimate of risk for mesothelioma for fibers between 5 and 10 µm in length was one three-hundredth of the risk assigned to fibers longer than 10 µm. The authors explained that "results from [their] review of the supporting literature suggest that the optimum cutoff for increased potency occurs at a length that is closer to 20 µm [rather] than 10 µm" (Berman et al. 2003). Further, Berman and Crump (Berman et al. 2003) reported that the best estimate of the potency of fibers shorter than 5 µm for mesothelioma is zero. A second expert panel convened by the U.S. EPA agreed that the risk associated with fibers less than 5 µm in length is "very low and could be zero" (Eastern Research Group, Inc., 2003a).

In contrast, a significant fraction of fibers found in those textile mills exceeded 20 microns in length, which mechanistically might or might not possibly be responsible for some increased risk for lung cancer or mesothelioma. Berman and Crump (2008), working under an EPA funded study, suggested that for chrysotile, potency may only be present for fibers in the vicinity of 25-40 microns.

Statement 11: "As stated in Section 3.2, epidemiological studies on mesothelioma and lung cancer in cohorts of workers using chrysotile in commerce were identified that could inform the estimation of an exposure-response function allowing for the derivation of a chrysotile asbestos IUR." (page 19, ln. 786 - 788)

Comment 11: As stated in previous comments, there are virtually no products in commerce today that contain chrysotile. Secondly, the only epidemiology studies that are relevant for the products discussed in this assessment are the studies (listed above) that evaluated vehicle mechanics. These were weighed for quality by Garabrant et al. (2016) in their meta-analyses. They showed that there was no increased risk to these cohorts. The few studies which suggested an increased risk had significant shortcomings, which are described in the Garabrant et al. (2016) paper.

On a mechanistic level, the lack of potency of brake wear debris and clutch debris have been described in numerous articles. These include Langer, (2003), Paustenbach et al. (2004), Korchevskiy et al. (2019), Boyles et al. (2019), Poland and Duffin, (2019), and Bernstein et al. (2020a, 2020b).

Statement 12: "For workers, cancer risks in excess of the benchmark of 1 death per 10,000 (or 1 x 10-4) were indicated for all conditions of use under high-end and central tendency exposure scenarios when PPE was not used. With the hypothetical use of PPE at APF of 10 (except for chlor-alkali processing and use and sheet gasket use), most risks were reduced for central tendency estimates but still persisted for sheet gasket stamping, auto brake replacement, other vehicle friction products and utility vehicle (UTV use and disposal) gasket replacement for high-end exposure estimates (both 8-hour and short-term durations)." (page 20, ln. 840 - 846)

Comment 12: I have several levels of concern about this statement.

First, the historical objective (benchmark) for occupational exposures to carcinogens has been 1 in 1000 workers (OSHA, 1999) going back to the 1970s. It was not until the NIOSH Chemical Carcinogen Policy published in 2017 (Whittaker et al., 2017) was released that a benchmark of 1 in 10,000 was proposed (e.g., 1 in 10,000 risk to workers). Thus far, to the best of my knowledge, this is a proposed policy and does not currently have regulatory standing.

Second, it is not traditional nor appropriate to base policy or regulation only on the high-end exposure scenario.

Third, as I have discussed above, the basis for the risk estimates is a flawed cancer potency factor derived for the unique exposure scenarios which existed in the textile mills between 1935 and approximately 1975.

Fourth, in my view, given that this EPA document focuses on brake wear debris (in no small measure), the cancer potency factor should be based on the epidemiology studies for brake mechanics who worked in the 1940 – 1980 era. An assessment of these data indicates that the brake wear debris has no potency for lung cancer or mesothelioma (Wong, 2001; Goodman et al., 2004; Garabrant et al., 2016; Garabrant and Pastula, 2018).

Fifth, the finding that epidemiological studies of those exposed to brake wear debris and gasket debris from automobiles and trucks were not at increased risk of lung cancer or mesothelioma should not be surprising given what has been learned about the dose to workers and the results of various animal studies (Bernstein et al., 2003; Bernstein and Hoskins, 2006; Bernstein et al., 2013, 2018, 2020a, 2020b).

Statement 13: "For ONUs, cancer risks in excess of the benchmark of 1 death per 10,000 (or 1 x 10-4) were indicated for both central tendency and high-end exposures for sheet gasket use (in chemical production) and UTV gasket replacement." (page 21, ln. 841 - 853)

Comment 13: For the last 30 years, EPA has had a benchmark of 1 in 10,000 to 1 in 1,000,000, and this depends on the size of the exposed population, and a cost-benefit analysis regarding reasonable versus tolerable risk (Whipple, 1988; Travis and Hattemer-Frey, 1988). As illustrated in the Travis et al. (1987)

paper, which I still believe is accurate and also reflects today's regulatory environment, none of the federal agencies have shown concern about cancer risks where it is predicted that not a single case of cancer would be prevented if the regulatory action took place. To the best of my knowledge, it is usually expected that hundreds or thousands of lives will be saved through a regulatory action, although there have been exceptions.

As stated previously in these comments, it appears to me that between 10 – 100 persons in the United States are likely to be exposed going forward to asbestos for the five conditions that are described in this document (except for chlor-alkali). Also, from what I can determine, virtually all of the groups that are apparently of concern to EPA currently are exposed at the upper end to airborne concentrations less than 0.05 fibers/cc on an 8-hour time-weighted basis. These concentrations are far less than the current OSHA PEL. If EPA wants to take issue with the OSHA PEL, they can take that up in the customary fashion regarding rulemaking, but it does not seem appropriate to question it in this document.

As noted, the 1 in 10,000 criteria does not have regulatory standing as far as I can determine. Indeed, from 1970 to 2017, the objective of most regulations involving workers was to control the risk to one in a thousand (assuming more than 1000 persons are exposed to this level of risk). It was only in July 2017 that NIOSH issued a document titled "Current Intelligence Bulletin 68 (NIOSH Chemical Carcinogen Policy) where they suggested going to a theoretical risk of 1 in 10,000." To the best of my knowledge, that was a policy, not a regulation or even an agreed objective for EPA and NIOSH. Further, for asbestos, for OSHA, the 1 in 10,000 risk level would have assumed mixed fiber exposure. OSHA has never attempted to set a chrysotile only PEL or cancer potency factor for pure chrysotile. In part, OSHA has not attempted to set a separate PEL for chrysotile because they know if the risks are acceptable for exposure to mixed fibers then exposure to pure chrysotile would be much less.

Perhaps most important, if controlling risk to the one in 10,000 level is the objective of EPA, I believe that is already achieved for the gasket stamping plant, for the brake mechanics, for those involve with gaskets, for those in the chlor-alkali business, and those who might interact with the brakes in the oil fields. This belief is based on the data in the EPA document.

The Agency does not provide sufficient scientific basis for the cancer potency factor that they have applied because the cohort upon which they rely (the Carolina textile workers) is not a chrysotile only cohort. As I have stated previously, some of the best researchers which EPA has relied upon for years to understand the cancer potency of chrysotile have said that there is a high probability that chrysotile alone does not cause mesothelioma (except perhaps when fibers longer than 20 – 40 microns are present in the lungs at concentrations sufficient to cause asbestosis) (Hodgson and Darnton, 2000; Berman and Crump, 2003; Pierce et al., 2016, 2008).

Uncertainties

Statement 14: "Uncertainties have been identified and discussed after each section in this risk evaluation. In addition, Section 4.3 summarizes the major assumptions and key uncertainties by major topic: uses of asbestos, occupational exposure, consumer exposure, environmental risk, IUR derivation, cancer risk value, and human health risk estimates." (page 21, ln. 869 - 872)

Comment 14: It is refreshing to see the Agency be thorough concerning wanting to make the uncertainties in the assessment transparent. For the topics discussed, in my view, the agency has done a reasonable job of identifying many of them.

However, I would suggest that the most significant areas of uncertainty are not discussed in the document. Among the many that deserve attention are the following:

1. The significance of fiber length to the toxicity and potency of chrysotile in the Carolina textile factories vs the products addressed in this assessment (brakes, gaskets, and packing).

2. The fact that one or two or more of the cases of mesothelioma in the Carolinas involved persons who are known to be exposed to the amphiboles during their work careers. This compromises its use as the basis for estimating a cancer potency factor (CPF) for pure chrysotile (beyond the fact that the fibers are 3 - 10-fold longer in the textile mill vs the vast majority in brakes and gaskets).

3. This document should present risks using the CPF that EPA and OSHA have used up until this time to illustrate the significance of their proposed CPF in determining whether excessive risk exists in any population.

4. Based upon my previous comments, I think the Agency should be transparent concerning the number of workers that are plausibly anticipated to be exposed in the coming years due to the import of new asbestos material. As I have said, based on my experience, I would be surprised if more than 20 – 100 people are going to be exposed each year to newly imported asbestos (other than in the chlor-alkali industry), and it appears that even the chlor-alkali workers are exposed to what is generally considered to be acceptable concentrations (according to OSHA).

5. As noted, the Agency is reminded that even if the <1% of brakes which might contain asbestos are coming from Canada, Russia, China or India, those should virtually all be brake pads (rather than drum brakes). Thus, since there is no exposure during installation or removal, they should not be of concern to EPA going forward under TSCA.

Statement 15: "A specific IUR was developed in this risk evaluation for combined mesothelioma and lung cancer following exposure to chrysotile asbestos. There is evidence that other cancer endpoints may also be associated with exposure to the commercial forms of asbestos." (page 22, ln. 925 - 927)

Comment 15: As discussed in the Berman and Crump, 2008 paper (and others identified in previous comments), chrysotile has different potency factors for lung cancer and mesothelioma, as derived from published studies of occupationally exposed cohorts. Besides, as noted previously, there is a real question as to whether chrysotile alone can cause mesothelioma (certainly at concentrations below that which causes asbestosis). It is recognized that other cancers could be associated with exposure to amphiboles, but this is generally not true for populations exposed to pure chrysotile.

Studies of the Calidria mines would be informative for understanding pure chrysotile as it is extremely pure. The last that I looked, the epidemiology of the miners was uneventful (Ilgren, 2010).

Beyond this, I believe that the majority of credible epidemiologist and toxicologists believe that when trying to assess the lung cancer hazard for persons exposed to asbestos, or any other potential lung carcinogen, that if you do not have a good smoking record, it is impossible for persons exposed from 1950 through 2000 to have any sense that the agent of interest caused lung cancer in the cohort. This is because the potency of tobacco smoking for lung cancer is so much greater than that of chrysotile fibers.

It has been said that even one year of smoking at any time will negate the ability to say that a particular chemical or fiber is the cause of lung cancer in a given person. Of course, there are exceptions to that thought, since some agents themselves are such potent lung carcinogens that it is impossible not to identify them as the causative agent.

But what is important here is that chrysotile does not have that potency for causing lung cancer. The Agency recognized the issue of confounding between smoking, chrysotile and lung cancer because they stated that on page 23 (paragraph 1) "Most of the studies of asbestos-exposed workers did not have information to control for cigarette smoking, which is an important risk factor for lung cancer in the general population. However, the bias related to this failure to control for smoking is believed to be small."

Statement 16: "Most of the studies of asbestos-exposed workers did not have information to control for cigarette smoking, which is a significant risk factor for lung cancer in the general population. However, the bias related to this failure to control for smoking is believed to be small." (page 23, ln. 945 - 947)

Comment 16: I do not agree with this statement; see my previous comments regarding the inability to "correct" for smoking history. Smoking is the number one cause of lung cancer in the United States. How can "failure to not control for smoking" be small? For chrysotile, it is believed that it requires concentrations at sufficient to cause asbestosis to increase the risk of lung cancer (Berman and Crump, 2008b).

Statement 17: "The occupational exposure assessment made standard assumptions of 240 days per year, 8 hours per day over 40 years starting at age 16 years." (page 23, ln. 955 - 957)

Comment 17: This seems extreme to me but is consistent with a 40-year working lifetime. However, this typical assumption of a 40-year working lifetime was developed to conduct evaluations retrospectively. So, when it is used for a prospective risk assessment, one should have a reasonable expectation that the exposures will occur for that period.

For this EPA document, for the five exposure scenarios identified, I saw no evidence that even a handful of workers who might be involved in those occupations would likely to be exposed for 40 years going forward. I suggest choosing a more appropriate assumption of duration for a prospective risk assessment.

However, as I have said repeatedly in my comments, I do not see much exposure to asbestos in the coming years. We need to remember that OSHA is responsible for regulating "day to day" exposures in the workplace, not EPA.

Statement 18: "Workers exposed to asbestos in workplace air, especially if they work directly with asbestos, are most susceptible to the health effects associated with asbestos." (page 23, ln. 972 - 973)

Comment 18: The word "most susceptible" used in this document is novel to me. In this document, it states:

> "TSCA § 3(12) states that "the term 'potentially exposed or susceptible subpopulation' means a group of individuals within the general population identified by the Administrator who, due to either greater susceptibility or greater exposure, may be at greater risk than the general population of adverse health effects from exposure to a chemical substance or mixture, such as infants, children, pregnant women, workers, or the elderly."

Historically, it has been my experience, as a toxicologist and risk assessor, that the word susceptibly was and should be limited to those who are biologically more susceptible. For example, exposure of children to lead, exposure of workers to airborne methylene chloride who have a deficiency of specific p450 enzymes, and asthmatics exposed to sensitizers are whom we categorize as most susceptible. This has been the traditional application of the term. I think the statement in TSCA regarding "greater exposure than the general population" is meant to address workers versus the public and those workers who are believed to be heavily exposed.

Based on the data presented in this document, and my own professional experience, I would not consider any of these workers whom the Agency is trying to protect for the next 40 years to be heavily exposed (and, therefore, not "most susceptible" using this definition). Indeed, if they are currently exposed, if they are exposed at all, it is to extremely low airborne concentrations of chrysotile asbestos.

In summary, relatively high exposure and susceptibility should not be confused as similar terms as they are, of course, entirely different concepts.

Statement 19: "There is also some evidence of genetic predisposition for mesothelioma related to having a germline mutation in BAP1 (Testa et al., 2011)." (page 23, ln. 982 - 983)

Comment 19: I agree that there is a strong possibility that there is a gene mutation, probably not BAP1, which will eventually be found that will allow us to identify those who have a predisposition to asbestos toxicity or, more specifically, to mesothelioma. At this time, it is speculation that we have any understanding of what might make any specific person more genetically susceptible to mesothelioma than someone else.

Statement 20: "The EPA has defined aggregate exposure as "the combined exposures to an individual from a single chemical substance across multiple routes and across multiple pathways (40 CFR § 702.33)."" (page 24, ln. 993 - 995)

Comment 20: This statement is correct, but as the Agency has said in this document, it is irrelevant to this analysis.

Statement 21: "In this risk evaluation, the EPA considered sentinel exposure the highest exposure given the details of the COU and the potential exposure scenarios. EPA considered sentinel exposures by considering risks to populations who may have upper bound (e.g., high-end, high intensities of use) exposures." (page 24, ln. 1008 - 1011)

Comment 21: In my experience, the Agency has rarely identified sentinel exposures. However, if they plan to do so on a routine basis going forward, these are not the cohorts that deserve such a label given their low level of exposures to asbestos.

Statement 22: "In each risk evaluation under TSCA section 6(b), EPA determines whether a chemical substance presents an unreasonable risk of injury to health or the environment, under the conditions of use." (page 24, ln. 1014 - 1015)

Comment 22: I agree with this statement. However, as noted previously, there are so many flaws in the derivation of the cancer potency factor (CPF) for chrysotile (presented in this document) that it is not appropriate for anyone to categorize any of these exposures as "presenting an unreasonable injury to health."

Attempts to use this document to identify a new CPF, which would suggest that the current OSHA PEL is unreasonably high, seems inappropriate. This topic is discussed later in these comments. The Agency has commissioned over the past 30 years many panels and numerous contractors to attempt to refine the CPF for chrysotile (including the Berman and Crump papers), and they have failed to reach agreement on a better CPF than the one currently used by EPA (prior to this draft dacoument). It has been evident for more than 40 years, as evidenced by the different TLVS for the fiber types in 1979 (ACGIH, 1980), that the amphiboles had very different toxicity from chrysotile fibers (Hodgson and Darnton, 2000; Hodgson et al., 2005; Bernstein et al., 2020a, 2020b).

For the sake of transparency, as noted before, I believe that the calculations for risk in this document should be based upon the current CPF for asbestos with modification by a factor of 100 to reflect the near-consensus view that chrysotile is at least 100 fold less potent than amosite for mesothelioma (Hodgson and Darnton, 2000; Hodgson et al., 2005b). For the vast majority of cohorts that have been studied, the predominant factor when evaluating the risks was the percent of amosite in the product. This understanding was not presented in this document.

Statement 23: "EPA also takes into consideration the Agency's confidence in the data used in the risk estimate. This includes an evaluation of the strengths, limitations, and uncertainties associated with the information used to inform the risk estimate and the risk characterization. The rationale for the risk determination is discussed in Section 5.2." (page 24, ln. 1022 - 1025)

Comment 23: I agree that the Agency should give serious consideration to the "… the strengths, limitations, and uncertainties associated with the information used to inform the risk estimate and the risk characterization". However, for the reasons that I have stated previously, the enormous degree of uncertainty regarding lung cancer and mesothelioma cancer potency factors used for chrysotile is not adequately disclosed for the reasons already mentioned.

Risk to Workers

Statement 24: "The conditions of use of asbestos that present an unreasonable risk to workers include processing and industrial use of asbestos-containing diaphragms, processing and industrial use of asbestos-containing sheet gaskets and industrial use of asbestos-containing brake blocks, aftermarket automotive asbestos-containing brakes/linings, other vehicle friction products, and other asbestos-containing gaskets. A full description of EPA's determination for each condition of use is in Section 5.2." (page 25, ln. 1076 - 1080)

Comment 24: I have reviewed the data represented in this EPA document. It appears that the workers in these facilities are nearly all exposed to time weighed average concentrations that are well below the current OSHA PEL of 0.1 fibers/cc. As shown in Table 2-5, there were 650 samples collected between 1996 and 2017. The table indicates that the 50th percentile concentration was 0.006 fibers/cc, and the upper 95th percentile concentration was 0.05 fibers/cc (eight-hour time-weighted averages full-shift samples).

These data do not support a finding of "unreasonable risk." Indeed, based on the current "law of the land," they are considered acceptable or "safe." Even if EPA insists that the OSHA PEL for asbestos is "way out of date" (which it is), then if one were to carefully assess the toxicology and epidemiology of chrysotile, especially chrysotile soaked in resin, one would conclude that the exposures in all of the facilities discussed in the EPA document would be considered "safe," acceptable or "tolerable"…..but certainly not presenting "an unreasonable risk."

Chlor-Alkali

In my 45 years of practice as an industrial hygienist, and having reviewed more than a million samples results, I have never seen data which illustrates such a high level of control for any airborne toxicant that I have studied.

In my professional experience, this is an incredibly narrow range of sample results for short-term, allegedly high exposure, sampling. In other words, statistically, in general, one would expect a very low level of uncertainty concerning these results.

These data clearly show that compared to the guideline, which applies to the United States for the protection of workers, the regulatory and risk assessment community would not conclude that these:

> "… conditions of use of asbestos that present an unreasonable risk to workers include processing and industrial use of asbestos-containing diaphragms, processing and industrial use of asbestos-containing sheet gaskets and industrial use of asbestos-containing brake blocks, aftermarket automotive asbestos-containing brakes/linings, other vehicle friction products, and other asbestos-containing gaskets" (page 25)

The available information does not support a finding of "unreasonable risk."

Processing and Industrial Use of Asbestos-Containing Sheet Gaskets

I have reviewed the description of the gasket making facility in the text of the document. It begins on page 74 and ends approximately on page 77.

The EPA document indicates that the number of workers at the Branham's facilities (two people in total) assumes that both facilities have only one employee who processes asbestos-containing gaskets. Two other workers do not handle asbestos-containing gaskets, and two others work in the office. The data presented indicate that the short-term personal breathing zone sample (as presented in Table 2-9), which are presumed to be the highest concentrations during the work shift, average 0.0249 fibers/cc (this is not shown in table 2-9).

In my professional experience, this is an incredibly narrow range of sample results for short-term, allegedly high, exposure results. In other words, statistically, one would say that there is a very low level of uncertainty regarding the magnitude of exposure. I expect that many of these samples were at or near the limit of detection, and therefore might be "substitute values" for the LOD. That is, given the low concentrations, I believe that many of these had no detectable fibers, and ½ the LOD was substituted for these values.

In short, in the field of risk assessment, using the currently available risk criteria (e.g., the OSHA PEL), one would have to conclude that these exposures do not pose "unreasonable risk to workers." This is especially true since we are talking about protecting only two workers in the United States, who were exposed only to a limited number of days per year. Further, given the high lack of certainty that chrysotile at any concentration can cause mesothelioma, makes the EPA's concern about the healthfulness of these two workers is unwarranted.

Be reminded that the fibers that go into polymeric sheet gasket materials, even when produced overseas, are made from Grade 7R chrysotile asbestos (Pigg, 1994), which is generally less than five microns in length. As an aside, one needs to consider that when chrysotile fibers are soaked in polymeric materials, that they have entirely different chemical properties than when they are not saturated. And those characteristics probably influence chronic toxicity. The recent data by Bernstein et al., (2020a, 2020b) (which is not cited in this document) indicates that fibers in polymeric materials have lost their biological activities (if they had it in the first place due to particle length and aspect ratio).
The available scientific data does not support a finding of "unreasonable risk."

Oil Field Brake Blocks

I have reviewed this section of the document, which starts on page 82 and ends on page 86.
The Agency did a good job in describing the use of this product and its life-expectancy (page 83), it is accurately noted that on page 82 that "The main brake can have several different designs, such as a friction band brake, a disc brake, or a modified clutch." The agency should be aware that these are three different braking mechanisms that each yield different kinds of brake wear debris.

The agency also correctly notes that (page 83, paragraph 3):

> "The brake blocks are enclosed in the drawworks, so it is not necessary to clean off brake dust under normal operations. The drawworks is washed down prior to removal and installation of

> brake blocks—a task that could lead to water releases of asbestos dust. Brake block servicing typically takes place outdoors or in a large service bay inside a shop (Popik, 2018)."

This means that there is no potential exposure to airborne brake debris from this braking system, as debris from this brake drops to the ground, so there is no operator nearby who is likely to be exposed to airborne brake dust and, as noted by the Agency, these systems are washed by water before servicing. It is important to note that because there is no exposure to asbestos associated with this particular braking system, the Agency does not present airborne concentrations to which workers were exposed. I fail to understand why the document does not state that "based on what we know, there are no data indicating measurable exposure…. therefore, it is not worthy of additional discussion."

It is noteworthy that the EPA has assumed that because they found a safety data sheet supplied by a supplier that some brake blocks in the past contained asbestos and that "some" continue to contain asbestos even though there is no information to support that assumption. Indeed, I believe that it is inaccurate.

On page 85, the agency states that:

> "It is reasonable to assume that wear of the brake blocks over time will release some asbestos fibers to the workplace air. However, the magnitude of these releases and resulting worker exposure levels is not known. In an effort to provide a risk estimate for this COU, the exposure scenario described in the previous section will be used. Table 2-13 presents the exposure data used for the risk estimates for brake block usage."

To estimate exposure to airborne asbestos from these brakes, even though there is no evidence that these brakes contain asbestos today, and the agency has indicated that there is no opportunity for exposure to airborne asbestos from these brakes, they built Table 2-13 which has a title "Summary of Asbestos Exposures During Use in Brake Blocks for EPA's Risk Evaluation." Regrettably, the Agency provides no robust data to support that table. The Agency assumes that the exposures are similar to those observed in the punching of sheet gaskets, which encompasses two employees exposed to near non-detectable concentrations.

The Agency has taken the central tendency values from Table 2-11 for the two employees involved with punching sheet gaskets and has assumed that these are the same concentrations that might be involved with persons in the oil industry, for which they have already shown that there is no exposure. Unfortunately, the Agency gives the impression that about 530,000 employees could be potentially exposed (even though, if exposed, the concentrations are surely immeasurably low).

Instead, the Agency presents Table 2-12 (Summary of Total Establishments in Relevant Industries and Potentially Exposed Workers and ONUs for Oilfield Brake Blocks). In this summary, the Agency gives the impression that 17,831 firms who have a total employment of about 530,000 people could be exposed to asbestos from these braking systems. I see no justification for why such an impression would want to be given to anyone since no one is exposed to asbestos in this industry from these brakes (the debris falls into the soil, no worker is expected to be standing beside this braking mechanism, and it is washed down before servicing).

Further, I believe it is likely that most or all firm firms phased out of using asbestos-containing blocks or band brakes years ago. I would suggest that the agency would need to confirm that in 2020, that any such applications of chrysotile in oilfield brake blocks continues to exist.

In section 2.3.1.6 Oil Field Brake Blocks, the single study considered by EPA was rated "low" in the systematic review. While it was not rated "unacceptable," the limitations of this study that might have been only 2 or 3 samples suggest the minimal nature of this data. It is difficult to see how EPA could rely on such flimsy data as "fit-for-purpose" or "best available science" or rely on such lightweight "weight of the scientific evidence."

I found no information in the document that brake blocks in this industry have contained asbestos over the past 35 years. Even if true, it seems that this analysis is not warranted under the Lautenberg initiative. If it were, given the handful of persons who could be potentially exposed (which I believe to be zero) and the magnitude of possible exposures, these data indicate that any regulatory action is unwarranted, especially in consideration of such a critical use scenario where the failure of such materials can result in multiple injuries to workers.

Please note that in 2018, the EPA decided that a ban on imported asbestos was not necessary at that time. This deserves to be discussed somewhere in this document.

As I am sure the Agency is aware, it is nearly impossible, in the modern era, due to litigation, to have worker exposures exceed 1/10th or ½ of the numerical PEL when working with these products. Also, the duration of exposure is usually minutes, rather than hours or days; therefore, the 8-hour time-weighted average of these exposures is diminishingly small.

It is my understanding that there may be specific needs by the military (like balloon gaskets used on high-pressure lines in aircraft like the Apollo) that are critical uses that require encapsulated chrysolite products. I am not aware of where you can find such items. Even if found, those items are never manipulated, and even if they were, they would likely be unable to release airborne asbestos in measurable concentrations.

Just as "Popik, 2018" references personal email communications in support of EPA positions, I would cite a recent post by a Certified Industrial Hygienist and Certified Safety Professional on the American Industrial Hygiene Association online forum, Catalyst, on 4/24/2019, regarding brakes on drilling rig draw works:

> "The brakes on drilling rig draw works are critical control devices as they are relied upon to suspend extremely heavy loads. If there is a failure of brakes on draw works, the outcome can be one or more fatalities, one would rate the risk of such an event in the extreme "high" end of a risk matrix."

In addition to pointing out the criticality of such continued use of asbestos-containing material, if any, such implies the role of these brakes to be "holding" brakes, not dynamic brakes. Crane brakes are an excellent example of "holding" brakes that have been studied. Based upon the one available study (Spencer et al., 1999) on the topic in the industrial hygiene peer-reviewed literature, "There were no asbestos fibers detected by the TEM method from air samples collected during the operation of the cranes."

Automotive Brakes and Clutches

I have reviewed this section of the document, which starts on page 87 and ends on page 97.
In my view, it would be wise for the Agency in the introductory paragraph to note that asbestos-containing parts were phase-out of cars by all of the various automobile manufactures beginning around 1972. Virtually all cars in the United States did not have asbestos in their brakes, gaskets or clutches by 1984 or 1985. I believe that ample evidence can be provided from documents in the litigation to support this view.

Some door handles and knobs may have contained encapsulated asbestos through 1988. But, various studies have shown that it is virtually impossible to remove the asbestos after it gets in a product like Bakelite (Mowat et al., 2005).

It is noted on page 87/310 that:

> "Asbestos was previously a component of many automobile parts, including brakes, clutches, gaskets, seam sealants, and exhaust systems (Blake et al., 2008; Rohl et al., 1976); and older model vehicles still in operation may have various asbestos-containing parts. Additionally, aftermarket automotive parts made from asbestos can be purchased from online retailers, and it is possible that such products exist in older stockpiles."

I strongly recommend that the Agency not give the impression that these parts are readily available or that many cars in the nation today contain asbestos-containing brakes. The current text is misleading if not wholly incorrect. It is not fair to suggest that older model vehicles still in operation may have asbestos-containing parts (perhaps this could potentially be true for an antique car that has not been driven in several decades but this is not of regulatory consequence).

My criticisms are based on the following:

1. I believe that the last asbestos-containing brakes manufactured in the United States were from 1986 to 1988. That is anecdotal because probably 70% or more of the cars made in the United States did not contain asbestos-containing brakes by 1978 through 1980.

2. There are a few product lines that still had asbestos-containing brakes after 1980, but they were few in number. If asbestos was in the brakes after 1980, this was because the companies had not yet been able to demonstrate their ability to meet safety standards to ensure that the cars would stop as needed and perform as required across weather conditions from -40 F below zero to 180 F. Needless to say, that is no small challenge for cars and light trucks, which ranged in weight during that era over a wide span.

3. EPA need not worry about persons, today, replacing asbestos-containing brakes in cars. Brakes are typically replaced every three to four years, on average; therefore, logically few cars which were repaired after 1988 should contain asbestos-containing brakes, as that was more than 30 years ago. Since about 1980 or so, most of the brakes are disc brakes, so there is no grinding/arcing that is needed and there is no accumulation of brake dust.

4. Beginning in the year 2002, I began searching for asbestos-containing brakes at auto stores across the nation, and I found none.

5. Today, in my view, I know of no seller or storage facility, where I or anyone should be able to find or purchase asbestos-containing brakes. Even antique car restoration facilities, to the best of my knowledge, long ago exhausted their supplies of old asbestos-containing drum brakes.

6. All this discussion is moot, because as the published data indicates, the exposures of mechanics to asbestos, even in the 1975 – 1985 era, were minimal on a TWA basis. Beyond that, we now know that even the relatively few airborne fibers released during a brake change appear to lack much biologic activity (Langer, 2003; Bernstein et al., 2015, 2018).

7. Beyond the reasons mentioned above, the Agency should have no concerns about exposures to brake dust (that which is released during grinding) or brake wear debris (due to historical braking) in the coming years. Note that the above-mentioned scientific facts explain why virtually all credible epidemiology studies of vehicle mechanics for the years 1955 – 1985 do not show an increased risk of lung cancer or mesothelioma (Garabrant et al., 2016).

To reinforce my views that the EPA need not worry about future exposure to asbestos-containing brakes is the fact that the only location that I have been able to find such items is in an abandoned junkyard in the California desert. And those brakes were rusted so tight to the brake mechanism that I needed a cutting torch to remove them.

Further, I found it noteworthy that the Agency did not offer any information or data which supported their view that these brakes will be in commerce in the future. On a practical level, the reason that most companies that had asbestos-containing materials threw them away in the 1970s, as it became clear that the legal liability for retaining these materials was breathtaking, and these pieces were of no material worth as such. It is difficult for me to believe that chrysotile asbestos brakes are currently entering the United States to a significant degree (this is discussed elsewhere) and if they are, they will be disc brakes.

Asbestos Brakes in Cars Produced in the US and Sold Overseas

The agency stated (top of page 87/310) that:

> "Currently, information indicates asbestos containing automobile components are used in a single vehicle which is manufactured domestically, but only exported and sold outside of the United States. However, the potential remains for some older vehicles to have asbestos-containing parts and for foreign-made aftermarket parts that contain asbestos to be imported and installed by consumers in cars when replacing brakes or clutches. EPA is aware of one car manufacturer that imports asbestos-containing automotive friction products for new vehicles, but those vehicles are then exported and not sold in the United States."

I am a bit surprised that this is true but, if it is, I expect that very few of these cars are manufactured (perhaps less than 50 a year) in the United States. As I recall, a handful of sports cars like the McLaren

were converted to right side drives in Los Angeles and sent to the EU annually, perhaps this is what the Agency is referring to here.

If accurate, there can be no more than a handful of persons engaged in this activity. But more importantly, there is no exposure to asbestos during the installation of brakes, even if there were asbestos-containing brakes used during the installation process. Specifically, there is no brake wear debris or brake dust because grinding is not needed on a modern car. Note, none of the historical evaluations are relevant because, in this document, the Agency has decided that state of art techniques will be used in this assessment. Additionally, all the new cars would have disc brakes.

All of the facts mentioned above make me wonder why EPA would feature this exposure scenario in this document and states (page 26/310) that:

Occupational Conditions of Use that Present an Unreasonable Risk to Health
• Processing and Industrial use of Asbestos Diaphragms in Chlor-alkali Industry • Processing and Industrial Use of Asbestos-Containing Sheet Gaskets in Chemical Production • Industrial Use and Disposal of Asbestos-Containing Brake Blocks in Oil Industry • Commercial Use and Disposal of Aftermarket Automotive Asbestos-Containing Brakes/Linings • Commercial Use and Disposal of Other Vehicle Friction Products • Commercial Use and Disposal of Other Asbestos-Containing Gaskets

Statement 25: "For asbestos, the employee permissible exposure limit (PEL) is 0.1 fibers per cubic centimeter (f/cc) as an 8-hour, time-weighted average (TWA) and/or the excursion limit of 1.0 f/cc averaged over a sampling period of 30 minutes." (page 59, ln. 2065 - 2067)

Comment 25: Views about the Current OSHA PEL

This sentence raises a concern that this document (Draft Risk Evaluation for Asbestos) is being used to justify proposing another ban on the import of asbestos-containing materials or as a "backdoor" method for pressuring OSHA to lower the asbestos PEL. Either action would be acceptable to me as a health professional (excluding the apparent need to use asbestos in the Chlor-alkali industry).

If EPA has concluded that it is appropriate to regulate chrysotile differently than amosite or crocidolite, then it would be a considerable improvement over the current state of affairs. For too many years, we have carelessly used the word asbestos to apply to all five major fiber types, as well as some cleavage fragments, even though there are nearly six decades of research showing that each is chemically distinct and poses a unique biological hazard. The reason for the dramatic difference in half-life and potency was recently demonstrated in a paper by Korchevskiy et al., 2019. Additionally, an excellent article by Garabrant and Pastula (2018) highlights the apparent potency differences among all these fiber types, plus the elongated mineral particles, as reflected in many epidemiology studies.

It is noteworthy that the only reason that EPA concluded that changing brakes or replacing gaskets going forward could pose an "unreasonable risk of harm" is that they have derived a new cancer potency factor (CPF) intended explicitly for chrysotile.

There are several significant problems with the derivation of their CPF, which I will discuss in detail later. First, it is the application of this rather stringent newly derived CPF that drives the risk calculations for all of the exposure scenarios.

Secondly, the foundation of the CPF is the epidemiology studies of the Carolina textile mills. It was assumed that they were only exposed to pure chrysotile, but that was not the case. As acknowledged in a recent deposition of Dr. Dement (one of the authors of those studies), he admitted that it was likely that some persons who developed mesothelioma were also exposed to the amphiboles. Indeed, that was confirmed by Dr. Roggli, who examined their lung tissues (Roggli et al., 1998; Pavlisko et al., 2020).

Thirdly, it is not scientifically reasonable to attempt to combine two separate diseases with two distinct mechanisms of action, with a high probability that the lung cancer data are flawed, to derive a combination CPF for chrysotile.

Fourth, beyond these three points, there have been 40 years of debate about whether chrysotile can even cause mesothelioma, and it is reasonably well known that it does not produce mesothelioma, except at concentrations that could cause asbestosis (Weill, 2018). Almost certainly, the dose-response is not linear for lung cancer and chrysotile exposure, and it has a threshold related to the dose, which causes asbestosis.

If the Agency wishes to lower the PEL for asbestos and to regulate according to each form of asbestos, I applaud that effort. However, this is entirely inappropriate from a regulatory standpoint, since OSHA is responsible for setting PELs. Therefore, it seems appropriate for EPA to suggest to OSHA to begin a rulemaking process for the three commercial forms of amphiboles, rather than attempt to "backdoor" a regulatory process via this proposal.

Risk to Consumers

Statement 26: "For consumers, EPA determined that the conditions of use that present an unreasonable risk are use of aftermarket automotive asbestos-containing brakes/linings and other asbestos-containing gaskets. A full description of EPA's determination for each condition of use is in Section 5.2." (page 26, ln. 1089 - 1092)

Comment 26: For the reasons I discussed the near impossibility previously to exposure to asbestos-containing brakes in the coming years, I believe that this section of the document is irrelevant. Of the disc brakes that are imported, even if <1% did contain asbestos, there is still no exposure from installing them.

Risk to Bystanders (from consumer uses)

Statement 27: "EPA determined that the conditions of use that present an unreasonable risk to bystanders are use of aftermarket automotive asbestos-containing brakes/linings and other asbestos-containing gaskets. A full description of EPA's determination for each condition of use is in Section 5.2." (page 26, ln. 1094 - 1097)

Comment 27: For the reasons, I discussed the near impossibility previously to exposure to asbestos-containing gaskets in the coming years, other than that single factory with four employees who's exposure seems to be well-controlled, I believe that this section of the document irrelevant.

Statement 28: (page 26, ln. 1106 - 1107)

Occupational Conditions of Use that Present an Unreasonable Risk to Health
• Processing and Industrial use of Asbestos Diaphragms in Chlor-alkali Industry • Processing and Industrial Use of Asbestos-Containing Sheet Gaskets in Chemical Production • Industrial Use and Disposal of Asbestos-Containing Brake Blocks in Oil Industry • Commercial Use and Disposal of Aftermarket Automotive Asbestos-Containing Brakes/Linings • Commercial Use and Disposal of Other Vehicle Friction Products • Commercial Use and Disposal of Other Asbestos-Containing Gaskets

Consumer Uses and Disposal that Present an Unreasonable Risk to Health
• Aftermarket Automotive Asbestos-Containing Brakes/Linings • Other Asbestos-Containing Gaskets

Comment 28: For comments that I have offered thus far, and based on the comments to follow, I see no adequate scientific basis for concluding that any of these occupational exposures (even if they could plausibly occur) would pose "an unreasonable risk to health."

Statement 29: "EPA published the scope of the risk evaluation for asbestos (U.S. EPA, 2017c) in June 2017, and the problem formulation in June 2018 (U.S. EPA, 2018d), which represented the analytical phase of risk evaluation in which "the purpose for the assessment is articulated, the problem is defined, and a plan for analyzing and characterizing risk is determined" as described in Section 2.2 of the Framework for Human Health Risk Assessment to Inform Decision Making." (page 28, ln. 1120 - 1124)

Comment 29: From what I can determine, the Agency generally followed this framework document, except that the systematic review does not seem thorough, and there was an insufficient examination of the quality of the critical assumptions regarding both the exposure and cancer potency.

Statement 30: "As explained in the Risk Evaluation Rule (82 Fed. Reg. 33726 (July 20, 2017)), the purpose of peer review is for the independent review of the science underlying the risk assessment." (page 28, ln. 1143 - 1145)

Comment 30: Given the EPA's strict policies regarding perceived conflicts of interest, it is difficult to convene a panel of experts who know the thousands of pages of information on a topic as deep as asbestos. Therefore, even though the current panels contain many credible and accomplished professionals, only one or two persons on the panel or ad hoc group have been seriously studying

asbestos toxicology and epidemiology for the past ten or 20 years. This makes it extremely difficult to obtain "an independent review of the science underlying the risk assessment." All one can hope is that many persons invest dozens of hours of writing comments on the document and that the panelists take the time to read them.

Statement 31: "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." (page 28, ln. 1147 - 1148)

Comment 31: I wholeheartedly agree with this and hope that the panel members have the time to carefully read the comments and the supporting published or unpublished literature on asbestos toxicology and epidemiology. The time between the deadline for submitting comments and the time the panel is convened is quite short for the panelists to digest the comments and find the relevant papers.

Statement 32: "The COUs in this draft risk evaluation for asbestos are limited to only a few categories of ongoing uses, and chrysotile is the only type of asbestos fiber identified for these COUs[3]." (page 29, ln. 1178 - 1179)

Comment 32: As stated previously, I believe that the Agency has addressed any significant plausible future exposures, and, as noted earlier, other than for the Chlor-alkali industry, there is likely to be no measurable exposure going forward, except for more than a handful of people in the country.

Statement 33: "The COUs included in this draft risk evaluation that EPA considers to be known, intended, or reasonably foreseen are the manufacture/import, use, distribution and disposal of asbestos diaphragms, sheet gaskets, other gaskets, oilfield brake blocks, aftermarket automotive brakes/linings, and other vehicle friction products and the processing of asbestos diaphragms and sheet gaskets." (page 29, ln. 1183 - 1187)

Comment 33: For the reasons mentioned, I do not believe that EPA has a basis for assuming that there will be "known, intended, or reasonably foreseen" exposures in the future, which are worthy of regulatory consideration. It is a stretch to get to 100 exposed persons (certainly exposed to no more than 1/100th the current OSHA PEL) rather than the 1,000,000 or more people which the Agency thinks could be exposed.

Thus, since so few persons may be exposed, the Agency cannot meet the requirement that at least a few cancer deaths would be prevented by advancing this document. Usually, dozens of lives are to be saved by this type of regulation.

Statement 34: "After EPA confirmed that chrysotile asbestos is the only type of asbestos still being imported into the U.S. either in raw form or in products, EPA developed a chrysotile IUR4 to be used in the RE. The IUR for asbestos developed in 1988 was based on 14 epidemiologic studies that included occupational exposure to chrysotile, amosite, or mixed-mineral exposures (chrysotile, amosite, crocidolite)." (page 30, ln. 1208 -- 1212)

Comment 34: As noted previously, the decision to derive and propose a chrysotile only cancer potency factor (CPF) for mesothelioma and lung cancer is admirable. However, I do not think it is scientifically reasonable to create one CPF for both endpoints (especially not for chrysotile).

Statement 35: "In developing a PCM-based IUR in this risk evaluation, several 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." (page 30, ln. 1229 - 1231)

Comment 35: This is a reasonable assumption at this time.

Statement 36: "Over 24,000 studies were initially identified for consideration during the Systematic Review process to determine whether the IUR needed to be updated." (page 30, 1240 - 1242)

Comment 36: I am surprised that there are that many papers concerning asbestos that could be considered relevant.

Statement 37: "Different modeling choices and combinations of cancer-specific unit risks yielded candidate IUR values ranging from 0.08 to 0.33 per f/cc, indicating low model-based uncertainty. The IUR chosen is 0.16 per f/cc and it was applied to the COUs to calculate lifetime risks for workers and consumers." (page 31, ln. 1246 -1249)

Comment 37: Based on over 40 years of experience in deriving cancer potency factors (CPFs) for carcinogenic chemicals, it seems implausible to me that the agency would believe that the plausible range of CPFs would be that narrow. Anyone with considerable experience in this area would know that the span of CPF for a chemical like this begins at zero and increases with the type of model that is applied with the data. Most of these models will all yield reasonable or plausible CPFs, especially for a generally non-genotoxic carcinogen like chrysotile. Usually, the range of plausible CPFs will often span two orders of magnitude.

Statement 38: "Therefore, this use will only be evaluated in occupational settings for one use that EPA identified for cars that are manufactured with asbestos-containing brakes in the U.S. but are exported and not sold in the U.S. However, removing and installing asbestos brakes in older vehicles by both professional mechanics and DIY consumers will be evaluated (see Table 1-4. below)." (page 37, ln. 1433 - 1436)

Comment 38: As noted previously, it is unlikely that more than 100 people per year work with asbestos-containing brakes, even up to four or five times per year, across the United States. If it occurs at all, this does not seem to be worthy of regulatory attention. Those persons will be handling disc brakes where there is no potential for exposure.

Statement 39: "Although EPA conducted a comprehensive search and screening process as described above, EPA made the decision to leverage the literature published in previous assessments[6] when identifying relevant key and supporting data[7] and information for developing the asbestos risk

evaluation. This is discussed in the Strategy for Conducting Literature Searches for Asbestos: Supplemental Document to the TSCA Scope Document (EPA-HQ-OPPT-2016-0736)." (page 44, ln. 1521 - 1523)

Comment 39: It is unclear to me what leveraging the literature means. This needs to be explained in a more precise manner.

Statement 40: "EPA did not have a previous, recent risk assessment of asbestos on which to build; therefore, initially the Systematic Review included a very large number of papers for all areas. Initially, studies were limited to those published after 1987, containing at least one of the six fiber types identified under TSCA." (page 45, ln. 1571 - 1573)

Comment 40: For this type of analysis, it seems that the agency should have primarily relied upon one of the more important projects which they funded almost 20 years ago (Berman and Crump, 2003, 2008b). Also, for assessing the risk of exposure to phenolic encapsulated materials, like brakes, packing, and gaskets, the Agency should have relied upon the meta-analyses studies of auto mechanics by Garabrant et al., 2016; Goodman et al., 2004; Wong, 2001; Pierce et al., 2008. They are infinitely more relevant to this document's scientific underpinnings than the textile workers' exposure cohorts in the Carolinas (Loomis et al., 2009; Dement et al., 2011; Elliot et al., 2012).

2.2.2.1 **Processing and Industrial Use of Asbestos Diaphragms in Chlor-alkali Industry (page 53)**

Statement 41: "EPA attempted to identify other companies that fabricate asbestos-containing sheet gaskets in the United States but could not locate any. Therefore, it is not known how many sites fabricate imported sheet gaskets containing asbestos in the United States." (page 54, ln. 1878 - 1880)

Comment 41: It is my best judgment that it is unlikely that other significant potential users have been missed by the Agency.

Statement 42: Table 2-2. Crosswalk of Conditions of Use and Occupational and Consumer Scenarios Assessed in the Risk Evaluation (pages 55 – 56)

Table 2-2. Crosswalk of Conditions of Use and Occupational and Consumer Scenarios Assessed in the Risk Evaluation

COU	Scenario	Form of asbestos
Diaphragms for Chlor-Alkali Industry (Processing and Use)	Occupational	Imported raw asbestos (used to fabricate diaphragms)
Brake Block Use (Use)	Occupational	Imported article
Sheet Gaskets Stamping (Processing)	Occupational	Imported sheets
Sheet Gaskets In chemical production (Use)	Occupational	Gaskets imported or purchased in US
Brakes Installation in exported cars (Use)	Occupational	Imported brakes
Brakes Repair/replacement (Use and Disposal)	Occupational (repair shops)	Imported brakes
Brakes Repair/replacement (Use and Disposal)	Consumer (DIY)	Imported (Internet purchase)
UTV Gaskets Manufacture UTV in US (Use and Disposal)	Occupational	Imported gaskets
UTV Gaskets Repair/replacement (Use and Disposal)	Occupational (repair shops)	Imported gaskets
UTV Gaskets Repair/replacement (Use and Disposal)	Consumer (DIY)	Imported gaskets

Comment 42: It is my best judgment that it is unlikely that the Agency has missed other significant potential users.

Statement 43: "Several of the asbestos-containing products identified as COUs of asbestos are not friable as intact products; however, non-friable asbestos can be made friable due to physical and chemical wear and normal use of asbestos-containing products. Exposures to asbestos can potentially occur via all routes; however, EPA anticipates that the most likely exposure route is inhalation for workers and ONUs." (page 56, ln. 1950 - 1953)

Comment 43: For asbestos-containing brakes and gaskets, which are going to be challenging to find in nearly any facility, you will find that they are not friable. For the very old gaskets in historical pipes, which are unlikely to still be in any active industrial factory, they are generally removable as a single unit or in large chips. Therefore, exposures to measurable respirable airborne fibers are highly unlikely.

Statement 44: "Where available, EPA used inhalation monitoring data from industry, trade associations, or the public literature." (page 56, ln. 1958 - 1959)

Comment 44: The agency appears to have done a good job assembling the available exposure data, except for several unpublished gasket studies before 1986.

Statement 45: "EPA typically assumes that ONU inhalation exposure is comparable to area monitoring results that may be available or assumes that ONU exposure is likely lower than workers." (page 56, ln. 1964 - 1966)

Comment 45: I recognize that it has recently become more common for some professionals within the agencies or in the IH profession to assume that area monitoring yields lower airborne concentrations that personal monitoring. However, it should be recognized that for most of the past 50 years, that has not been the case because area monitors were usually placed in locations where the highest exposures were expected and where workers rarely spent time.

Over the last ten years, many industrial hygienists have been unaware that area samples were normally collected in that matter. Instead, they have assumed that the person's breathing zone brought them closer to the source of the exposure (which is often true). However, historically, area samples were placed near the point source to see if any worker could be overexposed. It was a method for identifying the maximum possible concentration and, then, if it were substantial, you would begin to monitor workers.

Statement 46: "A central tendency estimate was assumed to be representative of occupational exposures in the center of the distribution for a given COU. EPA's preference was to use the 50th percentile of the distribution of inhalation exposure data as the central tendency. In cases where other approaches were used, the text describes the rationale for doing so." (page 57, ln. 1998 - 2001)

Comment 46: This approach is appropriate.

2.3.1.3 Chlor-Alkali Industry

2.3.1.4 Sheet Gaskets

Statement 47: "This section describes how asbestos-containing rubberized sheeting is processed into gaskets." (page 70, ln. 2496)

Comment 47: This description appears to be thorough.

Statement 48: "One known company in the United States (Branham Corporation) processes (or fabricates) gaskets from asbestos-containing rubberized sheeting." (page 71, ln. 2514 - 2515)

Comment 48: I am a bit surprised that anyone is stamping these gaskets out in the United States. I had assumed that these gaskets, which are used for a unique situation, were shipped into the United States in sealed bags. It is acknowledged on page 74 that they have only one worker who processes asbestos-containing gaskets.

He or she may be the sole person in the United States who punches out the asbestos-containing gaskets from sheets. Fortunately, the available industrial hygiene data indicates that they are not overexposed to asbestos.

Statement 49: "Industry representatives have informed EPA that the stamping process creates no visible dust, due in part to the fact that the asbestos fibers are non-friable and encapsulated in rubberized sheet material (ACC, 2018). This statement is consistent with EPA's observations during the site visit, in which no significant dust accumulations were observed on or near Branham's stamping machines. However, EPA's observations are based on a single, announced site visit. More importantly, sampling data reviewed for this operation do indicate the presence of airborne asbestos. This suggests that the stamping releases some asbestos into the workplace air." (page 73, ln. 2573 - 2579)

Comment 49: In an absolute sense, one can state that some asbestos fibers are released into the air during the punching process. However, given the strict OSHA requirements regarding asbestos during the past 30 – 40 years, it seems implausible that any company would process asbestos-containing sheet gaskets unless exposures were very well controlled (which they appear to be from the Agency's data).

Because the demand is so low and because no facility wants them on their site (except chlor-alkali facilities or a refinery), it is not surprising that only one employee in the United States is involved in this activity. The installation of pre-punched asbestos-containing gaskets poses no exposure.

Statement 50: "EPA received one month of worker activity data for Branham's Mississippi facility, and these data indicated that, in May 2018, the worker spent no more than 70 minutes per day processing asbestos-containing gaskets (Branham, 2018)." (page 74, ln. 2605 - 2607)

Comment 50: I am not surprised that exposures are limited to 70 minutes per day, and I would be surprised if this activity occurred more than a few times per year.

Comment 51: "The worker exposure levels from the Kentucky facility will be used in this draft risk evaluation because Branham officials informed EPA that they do not anticipate considerable increases or decreases in production demand for asbestos-containing sheet gaskets." (page 74, ln. 2609 - 2611)

Comment 51: This seems appropriate.

Comment 52: "A 2013 industrial hygiene evaluation performed by consultants from Environmental Health Management (EHM) concluded that measured asbestos exposures at Branham's Kentucky facility were not high enough to require respiratory protection (EHM, 2013); however, the worker uses the N95 masks to comply with Branham procedures." (page 74, ln. 2616 - 2620)

Comment 52: This is not surprising, given the data about this facility.

Statement 53: "EPA received slightly varying estimates of the number of workers at Branham's facilities and the specific locations where they work (ACC, 2018; Branham, 2018). Based on these estimates, EPA assumes that both facilities have one worker who processes asbestos-containing gaskets, two workers who process other non-asbestos containing gaskets in the same open floor area (and are considered to be ONUs), and at least two workers in the office space. Therefore, EPA assumes that asbestos-containing gasket stamping at this company (i.e., at both facilities combined) includes two directly exposed workers and four ONUs." (page 74, ln. 2631 - 2637)

Comment 53: As stated previously, investing a lot of time and effort into understanding the exposures of these workers, who are under the OSHA PEL for asbestos, appears to be a poor use of resources that could be applied to higher priority projects in the EPA.

Statement 54: "These estimates are based on the one company known to stamp asbestos-containing sheet gaskets. It is unknown if other U.S. companies perform this same stamping activity. EPA attempted to identify other companies that cut/stamp asbestos-containing sheet gaskets in the United States but could not locate any.: (page 74, ln. 2639 - 2642)

Comment 54: I would be surprised if there were another facility that stamps asbestos-containing gaskets. My searches have not identified another such facility in the United States.

Statement 55: "EPA first considered the 2011 to 2016 nationwide exposure data provided by OSHA and the history of NIOSH HHEs, but neither resource included exposure data relevant to stamping of asbestos-containing sheet gaskets." (page 75, ln. 2650 - 2651)

Comment 55: This is not surprising to me.

Statement 56: "Branham hired EHM consultants to conduct this study, which involved a day of PBZ monitoring at the Kentucky facility in December 2012. The EHM consultants measured PBZ concentrations for one worker - the worker who operated the stamping machine to process asbestos-containing gaskets - and issued a final report of results in 2013 (EHM, 2013). The EHM consultants measured worker inhalation exposures associated with a typical day of processing asbestos-containing gaskets and reported that samples were collected "during work periods when the maximum fiber concentrations were expected to occur"" (EHM, 2013). (page 75, ln. 2666 - 2671)

Comment 56: I agree that this is a reasonable approach.

Statement 57: "Ten short-term samples, all approximately 30 minutes in duration, were collected from one worker throughout an 8-hour shift. Samples were analyzed by PCM following NIOSH Method 7400." (page 75, ln. 2677 - 2678)

Comment 57: Although limited, this number is likely adequate for a stationary point source.

Statement 58: "The short-term exposures ranged from 0.008 fibers/cc to 0.059 fibers/cc. Table 2-9. lists the individual measurement results and corresponding sample durations. Based on the short-term results, the EHM consultants calculated an 8-hour TWA exposure of 0.014 fibers/cc, which assumed no exposure during periods without sampling." (page 75, ln. 2680 - 2683)

Comment 58: This seems to represent extraordinarily good controls for asbestos exposures in this facility.

Statement 59: Table 2-10 Summary of Asbestos Exposures During Sheet Gasket Stamping Used in EPA's Risk Evaluation (page 77)

Table 2-10 Summary of Asbestos Exposures During Sheet Gasket Stamping Used in EPA's Risk Evaluation

Occupational Exposure Scenario	Full-Shift Exposures (fibers/cc)					
	Workers			ONUs		
	Central Tendency	High-end	Confidence Rating	Central Tendency	High-end	Confidence Rating
Sheet gasket stamping: 8-hr TWA exposure	0.014	0.059	Medium	0.0024	0.010	Medium
Sheet gasket stamping: Short-term exposures (approximate 30-minute duration)	0.024	0.059	Medium	0.0042	0.010	Medium

Comment 59: Table 2-10 seems adequate for determining compliance with the OSHA PEL for asbestos.

Statement 60: "The "bystander" locations in this study were between 5 and 10 feet from the gasket removal activity, and asbestos concentrations were between 2.5 and 9 times lower than those measured for the worker. Based on these observations, EPA assumes that ONU exposures for this COU are a factor of 5.75 (i.e., the midpoint between 2.5 and 9) lower than the directly exposed workers. This concentration reduction factor falls within the range of those reported for other asbestos COUs." (page 77, ln. 2728 - 2732)

Comment 60: The Agency's general approach to estimating the exposures of bystanders is reasonably accurate. I would refer them to a paper by Donovan et al., 2011, which specifically evaluates bystander exposures when working with asbestos-containing materials. That paper and the two letters to the editor regarding that paper (Egilman and Schilling, 2012; Donovan et al., 2012) are the best available information for estimating these bystander exposures.

Statement 61: "Installed gaskets typically remain in operation anywhere from a few weeks to three years; the time-frame before being replaced is largely dependent upon the temperature and pressure

conditions (ACC, 2018), whether due to detected leaks or as part of a routine maintenance campaign." (page 78, ln. 2782 - 2784)

Comment 61: This statement is accurate, based upon my experiences as a toxicologist and industrial hygienist.

Statement 62: "Though Chemours has an active program to replace asbestos-containing gaskets with asbestos-free alternatives and this program has resulted in considerable decreases in asbestos-containing gasket use (EPA-HQ-OPPT-2016-0736-0067), gaskets formulated from non-friable chrysotile asbestos-containing sheeting continue to be the only product proven capable of withstanding certain extreme operating conditions and therefore provide a greater degree of process safety and integrity than unproven alternatives according to industry (ACC, 2017b)." (page 78, ln. 2797 - 2802)

Comment 62: The observation that few applications require asbestos-containing materials does not surprise me. However, sometimes there is simply not an appropriate substitute for a polymeric material to contain anything other than an asbestos filler.

Comment 63: "When removing old gaskets for replacement, trained maintenance workers wear respiratory protection—either an airline respirator or cartridge respirator with P-100 HEPA filters, although the APF for this respiratory protection was not specified (ACC, 2017a)." (page 79, ln. 2810 - 2813)

Comment 63: For approximately the last 30 years (since 1990), pipefitters, boilermakers, steamfitters, insulators, and laborers have been taught to assume that when working with any very old gasket or old insulation, that they should assume that it contains asbestos and to take appropriate precautions.

Statement 64: "One publication was a 1996 study of maintenance workers who removed braided gaskets and sheet gaskets at a chemical plant in the Netherlands (Spence and Rocchi, 1996)." (page 80, ln. 2849 - 2850)

Comment 64: If the Agency is interested, there are many more workplace studies of gasket removal studies that are available (conducted by different firms) and have been highlighted in a previous comment.

Statement 65: "The other publication was a 2006 study that used a simulated work environment to characterize worker and ONU exposure associated with gasket removal onboard a naval ship or at an onshore site (Mangold et al., 2006)." (page 80, ln. 2859 - 2861)

Comment 65: The Mangold paper is a solid article (I was a co-author), but there are others that the Agency should be aware of, and I would be happy to share that information if requested.

Statement 66: "Other peer-reviewed publications were identified and evaluated but not considered in this assessment because they pertained to heavy-duty equipment (Boelter et al., 2011), a maritime

setting with confined spaces (Madl et al., 2014), and braided packing (Boelter et al., 2002)." (page 80, ln. 2871 - 2873)

Comment 66: It is appropriate not to use those papers to understand automobile or truck brake exposures to asbestos, but the Agency should be reminded that 99.9% of asbestos is degraded during the braking process to forsterite or related compounds (Jacko et al., 1973; Rowson, 1978; Williams and Muhlbaier, 1982; Boelter et al., 2007; Madl et al., 2009).

Statement 67: "The samples evidently were collected to assess compliance with OSHA occupational exposure limits, suggesting that they were analyzed using PCM. Asbestos levels in these samples ranged from 0.0026 to 0.094 fibers/cc, with an average of 0.026 fibers/cc (ACC, 2017a). The documentation provided for these sampling events does not indicate the sampling duration or the amount of time that workers performed gasket removal activity, nor were the raw data provided." (page 80, ln. 2881 - 2886)

Comment 67: Since these samples were collected to assess OSHA compliance, it is acceptable to assume that they were collected for either 30 minutes or 8 hours. In either case, it appears that the exposures are entirely satisfactory when compared to the OSHA PEL for asbestos.

Throughout these comments, I do not want to imply that the current OSHA PEL is considered acceptable to all professionals within the occupational health community. The problem with the OSHA PEL is that it applies to chrysotile, amosite, and crocidolite as if they were equally toxic or potent for producing cancer. In the 1978 ACGIH TLVs, the differences in potencies were recognized when they established two different TLVs.

It is also true that because there are not separate limits for each form of asbestos, the 0.1 f/cc PEL for all asbestos is generally not considered adequate for crocidolite and, probably, not for amosite. It is entirely possible to derive reasonable PELS simply based on the NOEL for chrysotile and using the Hodgson & Darnton ratio of potencies (Hodgson and Darnton, 2000). For example, some have suggested that the NOEL for chrysotile is between 100 and 400 f/cc-year (Pierce et al., 2016). Assuming that is accurate, for purposes of illustration, then the NOEL or practical threshold for amosite might be 200/100 or 2.0 f/cc-year (or a PEL of 0.05 f/cc). If one uses 50 f/cc-year as the low end for developing asbestosis for chrysotile, then it is 50/100 then divided by 40 years (a PEL of 0.01 f/cc for amosite). This is ten-fold less than the current PEL of 0.1 f/cc.

It is regrettable that after physicians, toxicologists and epidemiologists realized that these three forms of asbestos had entirely different toxicological properties and carcinogenic potencies, they continue to be regulated equivalently with only one occupational exposure limit (OEL).

For example, as noted above, it is clear to me that the OSHA PEL of 0.1 fibers/cc poses an unacceptable risk to workers if crocidolite is the airborne toxicant. If it is amosite, depending on the risk criterion which is found acceptable (either 1 in 1,000 or 1 in 10,000 for workers), it is probably not sufficiently strict. However, for chrysotile, a PEL of 0.1 fibers/cc seems perfectly acceptable. A lifetime cumulative dose of 0.1 fibers/cc yields a total lifetime dose of 4 fibers/cc-years; this is not unlike the lifetime cumulative dose for vehicle mechanics who were involved with brake changes from 1945 to 1985 (approximately when brakes no longer contained asbestos) and there has been no increased risk of asbestos related disease in that population.

Since nearly all of the epidemiology studies of merit show no increased risk of mesothelioma in the auto mechanics during that period, it is reasonable to infer that four fibers/cc-years for chrysotile did not pose a significant health risk for mechanics who worked from 1940-1975. The obvious caveat to using the experience of mechanics to estimate the risk to those exposed to pure chrysotile is that the fibers associated with braking appear to have lost their biologic activity due to conversion to forsterite or being filled with phenolic resins (Bernstein et al., 2018, 2003).

Statement 68: "The high-end 8-hour TWA exposure value for workers (0.094 fibers/cc) is based on the highest exposure measurement reported for gasket removal activity at titanium dioxide manufacturing facilities (ACC, 2017a). Again, the sample duration for this measurement was not provided, and so this concentration represents a high-end by extrapolating the value to represent an entire shift." (page 81. ln. 2900 - 2903)

Comment 68: This seems like an unusually strict approach, if not unreasonable, for estimating the eight-hour time-weighted average exposure for a person removing gaskets. By definition, this is an episodic event, which usually does not last longer than a few minutes. And in my experience in chemical plants, it would be highly unusual for pipefitters to replace more than a handful of gaskets each week unless they were assigned exclusively to the preventative maintenance program for pumps or valves at a chemical plant. Such jobs are scarce since they are too monotonous.

Going forward, it would be surprising if 20 – 200 old asbestos containing gaskets are replaced annually going forward, which is an exposure scenario outside the purview of EPA and is not of regulatory significance to any agency.

Statement 69: Table 2-11. Summary of Asbestos Exposures During Sheet Gasket Use Used in EPA's Risk Evaluation (page 81)

Table 2-11. Summary of Asbestos Exposures During Sheet Gasket Use Used in EPA's Risk Evaluation

Occupational Exposure Scenario	8-hr TWA Exposure Levels (fibers/cc)					
	Workers			ONUs		
	Central Tendency	High-end	Confidence Rating	Central Tendency	High-end	Confidence Rating
Sheet gasket use: 8-hr TWA exposure	0.026	0.094	Medium	0.005	0.016	Medium

Comment 69: As noted several times in my comments, there are virtually no persons in the United States who should be interacting with asbestos-containing gaskets, especially going forward. The only source of which we are aware is discussed in this EPA document. They get sold into a small market and since there is no exposure when they are installed, I do not understand the focus of the Agency on that exposure route.

The high-end values in Table 2-11 are not consistent with the published literature or the experiences of most of the industrial hygienists in the field for an eight-hour TWA (even 20 – 30 years ago). On a TWA basis, the major review articles (for when this work was performed in the 1950-1980s) should be the focus of this document. As an exposure assessor, you would probably divide by 20-fold or more due to

the infrequency of the worker's contact with these materials. In short, I am hard-pressed to think of anyone exposed in the coming years to asbestos associated with gaskets (except in some refineries and chlor-alkali facilities).

Since the Agency is looking at this from a going-forward basis, it is more reasonable to assume that no one will be changing asbestos-containing gaskets in the coming years. As the gaskets do not contain asbestos (with few, if any, exceptions) or for gaskets that have not been changed in 20 years (a nearly impossible situation), the exposures will be extremely short and rare.

ONU Exposures

Statement 70: "As noted previously, one study (Mangold et al., 2006) measured "bystander" exposure during asbestos-containing gasket removal. The bystander locations were between 5 and 10 feet from the gasket removal activity, and concentrations were between 2.5 and 9 times lower than those measured for the worker." (page 81, ln. 2916 - 2918)

Comment 70: I have no issue with the Agency citing the data from the Mangold et al. (2006) paper, but I recommend that they rely primarily on the paper by Donovan et al. (2011) for bystanders. However, for the reasons expressed in Comment 69, there can be no bystander exposure since there is no likely one in the modern era changing an asbestos-containing brake (that might release dust).

I hope the panel and ad hoc panel have come to recognize that 85% of this document is describing scenarios that will likely rarely occur in the coming years and, if they do, the number of persons potentially exposed is vanishingly small. In short, the foundation of the building that is attempting to be built by the Agency is highly suspect.

2.3.1.6 Oil Field Brake Blocks

Statement 71: "The rotary drilling rig of an oil well uses a drawworks hoisting machine to raise and lower the traveling blocks during drilling. The drawworks is a permanently installed component of a mobile drilling rig package, which can be either "trailerized" or self-propelled. Therefore, there is no on-site assembly of the drawworks. Except for initial fabrication and assembly prior to installation on a new rig, the drawworks is not set or installed in an enclosed building (Popik, 2018)." (page 82, ln. 2953 - 2957)

Comment 71: I have no reason to question this statement.

Statement 72: "EPA obtained a safety data sheet (SDS) from Stewart & Stevenson Power Products, LLC for "chrysotile woven oilfield brake blocks, chrysotile woven plugs, and chrysotile molded oilfield brake blocks."" (page 83, ln. 3000 = 3001)

Comment 72: The date on the MSDS received from Stewart & Stevenson Power Products, LLC, is not revealed in this document. It should be noted that the information pertaining to asbestos-containing brake blocks at https://stewartandstevenson.com/assets/files/pdf/rig/BrakeBlocks2011.pdf was copyrighted in 2010, and https://stewartandstevenson.com/assets/files/pdf/rig/silverline-sp-brake-

blocks.pdf is copyrighted 2012. Therefore, this information is not current but is instead approximately 8 to 10 years old.

As noted previously, I would be surprised if there were many brake blocks containing asbestos still used in these mobile drilling rigs. This can be easily determined by interviewing an industrial hygienist or mechanics in that industry. However, even if some or all of these rigs still use asbestos-containing brake blocks, the exposure to workers would be to the brake wear debris. This, at most, would have the same characteristics as automobile brake wear debris from the 1960s.

Researchers, including the U.S. Public Health Service, and the U.S. EPA observed as early as the 1960s that, while forsterite had a chemical composition similar to chrysotile, the material was amorphous and nonfibrous (Lynch and Ayer, 1968; Anderson et al., 1973; Jacko et al., 1973; Rowson, 1978). More importantly, these researchers consistently reported that only a very small fraction (typically much less than 1%) of the wear debris in drum brakes consisted of asbestos fibers (Jacko et al., 1973; Rowson, 1978; Williams and Muhlbaier, 1982; Paustenbach et al., 2004; Boelter et al., 2007; Madl et al., 2009).

Not only is the chrysotile almost entirely converted to forsterite during braking, but recent work also confirms prior suspicions that the chrysotile fibers found in the air from brake wear debris lack the biologic activity of asbestos. Dr. Langer of NYU has conducted a chemical analysis of brake wear debris. He noted that "Using heating studies and milling as an approximation of thermal and mechanical shear stress that chrysotile is subjected to on a brake lining, biological blunting is shown to begin much earlier than the olivine transformation process. Minimal degradation of the chrysotile surface structure imparts a disproportionately great effect on its biological activity" (Langer, 2003).

Dr. Langer's hypotheses were confirmed in two major studies in 2014 and 2015 by Dr. Bernstein and his colleagues (Bernstein et al., 2014, 2015). They reported that "These results provide the support that brake-dust derived from chrysotile containing brake drums would not initiate a pathological response in the lung or the pleural cavity following short term inhalation" (Bernstein et al., 2015). Thus, the available information is that even the few fibers that were sometimes found in the breathing zone in various studies of mechanics in the 1960s and 1970s had little or no carcinogenic potency.

As important, based on the use of these portable rigs, these exposures are going to be outdoors, which means that there is nearly infinite dilution, such that a bystander would have no measurable exposure to the brake wear debris. Furthermore, the holding brake portion of these brakes would not be expected to release debris at even the low rate of automobile brakes and they are washed down prior to being replaced.

In short, this exposure scenario probably should not be in the document. It would appear that there is no possible measurable exposure to chrysotile associated with this exposure scenario.

Statement 73: "At least one U.S. company imports and distributes non-metallic, asbestos-woven brake blocks used in the drawworks of drilling rigs. Although the company no longer fabricates brake blocks using asbestos, the company confirmed that it imports asbestos-containing brake blocks on behalf of some clients for use in the oilfield industry." (page 83, ln. 3007 - 3010)

Comment 73: There appears to be a high level of uncertainty in this statement. Even if they are sold into the United States, for the reasons mentioned in comment 72, I am hard-pressed to find a plausible exposure scenario.

2.3.1.6.3 Number of Sites and Potentially Exposed Workers – Oil Field Brake Blocks

Statement 74: "EPA identified one U.S. facility that imports asbestos-containing brake blocks (Popik, 2018). It is unknown how many other facilities import asbestos-containing brake blocks. It is also unknown how many customers receive brake blocks from the sole facility identified by EPA. Unlike some of the other COUs, the lack of any information on oilfield brake block COU necessitated the use of other established methods to estimate the number of potentially exposed workers." (page 84, ln. 3024 - 3028)

Comment 74: Rather than for the Agency to "guess" the number of brake blocks still in use that are asbestos-containing and the frequency of interaction with those brakes, they should conduct interviews of the appropriate persons.

It feels like pure speculation that this exposure scenario exists today and, if it does, I am unable to see how anyone is exposed to airborne chrysotile due to the work being outdoors, the fact that the dust will fall to the ground since there is no brake drum, the brakes are washed prior to replacement, and because no one would be expected to stand near the brake block (and the asbestos released should be forsterite).

Statement 75: "EPA did not identify any studies that contain exposure data related to asbestos-containing brake blocks but did identify one published study that contains limited air sampling data for asbestos-containing brake bands (Steinsvag et al., 2007). In the absence of any other exposure data, the limited data provided in this study were used to estimate exposures to workers from brake block installation, servicing, and removal." (page 85, ln. 3047 - 3049)

Comment 75: I have read the Steinsvag et al., 2006 paper. The authors state that "these samples were analyzed by stationary samples of asbestos fibers on the drilling floor at one installation in 1988." They indicated that asbestos-containing brake bands caused the exposure at that facility.

The appropriate method for interpreting these data (I assume that they could represent the mean of four or five samples; the number of samples is unstated) is rather simple. As stated previously, when industrial hygienists want to understand the potential for a point source to release vapors, dusts or fibers, it has not been uncommon over the past 50 years to place a sampling device near that source. The objective is to identify whether or not there is any possible hazard from these substances to persons who might pass by or work within four to ten feet of the point source.

These appear to be "screening level" samples. In this particular case, it makes sense that an industrial hygienist would collect three to five samples to determine if the source was worthy of further study. Given that there is no need for a person to be near these brakes, during the daily course of their job, these should be considered as sample data that represent peak exposure levels for the time that a person would stand near an operating brake. I imagine that this is no more than an hour a month, if at all.

Therefore, I do take issue with the Agency's assumption that this is representative of an eight-hour TWA exposure of someone in this industry. Indeed, I think that this overestimates the current data by at least a factor of 100 to 1000, and one can conclude from these data that this is a negligible source of exposure to asbestos. Therefore, in my view, the alleged possible health hazards can be disregarded.

In consideration of the measured 0.02 - 0.03 fibers/cc, assuming one hour of exposure on any particular day, the eight-hour TWA would be approximately 0.003 fibers/cc. Furthermore, assuming one day a month of such exposure, a 45-year career cumulative exposure would be 0.006 fiber/cc-year.

If this concentration is considered unacceptable, then an industrial hygienist should do a three-day sampling of a facility that uses brake blocks. This would determine the representative concentration of asbestos in these facilities today, if any, assuming that they still use asbestos. This would validate or refute the assumption that this poses an unacceptable risk to a worker's health today.

As mentioned previously, it is my impression that these concerns are under the purview of OSHA. Note that these samples were collected approximately 32 years ago (when asbestos was a component in brake blocks).

Statement 76: "The information available to EPA confirms that some brake blocks used in domestic oilfields contain asbestos, as demonstrated by an SDS provided by a supplier. It is reasonable to assume that wear of the brake blocks over time will release some asbestos fibers to the workplace air. However, the magnitude of these releases and resulting worker exposure levels is not known. In an effort to provide a risk estimate for this COU, the exposure scenario described in the previous section will be used. Table 2-13 presents the exposure data used for the risk estimates for brake block usage." (page 85 – 86, ln. 3081 - 3086)

Comment 76: As discussed in a previous comment, using the data from the Steinsvag et al., 2007 paper is not appropriate, since this was an area sample collected adjacent to the brake. As it is unclear that anyone would have a job description that would require spending much time in the vicinity of that braking mechanism, I recommend that this exposure scenario be dropped (due to the lack of information, the outdoor environment, and lack of worker proximity to the source). Thus, table 2-13 should be significantly altered or dropped.

Indeed, the magnitude of exposure from brakes on cranes has been studied, and resulting worker exposure levels are known; based upon the Spencer Balzer paper in the industrial hygiene peer-reviewed literature (Spencer et al., 1999). The authors noted that "There were no asbestos fibers detected by the TEM method from air samples collected during the operation of the cranes" (Spencer et al., 1999). Interestingly, some of these cranes had the operator sitting within 2 feet of the exposed brake for these cranes (the brake is in the cabin). I assume there is no measurable exposure because of the conversion to forsterite or because it is open to the ground and the particles simply "drop out" of the cab.

2.3.1.7 Aftermarket Automotive Brakes/Linings and Clutches

Statement 77: "The use of asbestos in automotive parts has decreased dramatically in the last 30-40 years. Several decades ago, virtually all vehicles had at least some asbestos-containing components.

Currently, information indicates asbestos containing automobile components are used in a single vehicle, which is manufactured domestically but only exported and sold outside of the United States. However, the potential remains for some older vehicles to have asbestos-containing parts and for foreign-made aftermarket parts that contain asbestos to be imported and installed by consumers in cars when replacing brakes or clutches." (page 87, ln. 3118 - 3124)

Comment 77: This was discussed in a previous comment. The Agency notes that "the potential remains for some older vehicles to have asbestos-containing parts."

It is my impression that EPA would have no jurisdiction over the rare exposures that might occur today by replacing an asbestos-containing automobile brake, since this is regulated by OSHA. This is undoubtedly not regulated under the TSCA umbrella. More importantly, as noted previously, it is unfathomable that there would be any exposure to asbestos, as asbestos-containing brakes have not been sold inside the United States since the early 1980s (except for the alleged sales on the internet which I cannot substantiate).

Ten to fifteen years ago, when I attempted to study the issue of "current use," I did visit antique auto restoration shops. I can state that sometimes an old car, found in a hay barn, did find its way to a vintage auto-restoration shop. Those cars had to be about 40-50 years old, and "some" still contained asbestos-containing brakes. However, by then, as noted by EPA in this document, nearly every shop used wet removal techniques, and exposures, even 15 years ago, were negligible and infrequent (even rare).

Concerning the Agency's belief that "Currently, information indicates asbestos containing automobile components are used in a single-vehicle which is manufactured domestically, but only exported and sold outside of the United States," I do not believe that this is an exposure scenario worthy of discussion.

The number of plausibly exposed persons is probably less than 30-100 persons in the United States (if any). Given the approximate one to four billion dollars that auto manufacturers have spent in litigation costs involving asbestos-containing material sold before the 1980s, it is implausible that a car manufacturer today uses asbestos-containing components. Since EPA has said that they are assuming, in this document, that modern-era brake removal practices are and will be used going forward, then there would be no measurable exposure in this hypothetical exposure scenario.

One cannot rule out that given the information provided by EPA in this document, that brakes purchased online by an individual might contain asbestos. However, as noted by the Motor & Equipment Manufacturers Association (MEMA), this comprised less than one percent of the brakes sold in the United States in 2013. I believe this is an overestimate because that trade organization has wanted them banned. More importantly, they are not transparent about the fact that over 99% of those imports would be disc brakes, where no exposure during installation or repair occurs.

Most importantly, from an exposure standpoint, even if this is true, installing a new disc (or drum) brake should not pose an exposure risk (except in the rare cases when grinding must occur, and the machine does not have proper ventilation). Grinding of a drum brake usually only occurs in an antique car restoration shop. Additionally, as virtually all modern cars have disc brakes, that even if the brakes did contain asbestos, there would be no opportunity for exposure, as the brake wear debris falls to the ground during braking. Even if <1% of the brake market did contain asbestos, it is irrelevant concerning the potential adverse effects on an individual or on public health.

As noted, EPA said that for the sake of this document, they were assuming modern era practices would be used (so there is almost certainly no exposure, as grinding would not be utilized).

Concerning the Agency's belief that some "… foreign-made aftermarket parts that contain asbestos to be imported and installed by consumers in cars when replacing brakes or clutches", I cannot rule out that brakes from foreign countries may be manufactured containing asbestos and are potentially being sold in the U.S. market. However, as noted previously, I have gone to dozens of part stores in an attempt to find such brakes and have failed to find any, and I have attempted to purchase them on the internet (and have failed).

If EPA is aware of a current source, then they should identify it in this document. In January 2011, the supreme court of India banned the use of asbestos in their country. China and Russia had proposed a similar ban during that time frame. As previously noted, it is implausible that any U.S.-based corporation would sell these brakes in the United States due to the liability of potential litigation.

Statement 78: "EPA is aware of one car manufacturer that imports asbestos-containing automotive friction products for new vehicles, but those vehicles are then exported and not sold in the United States." (page 87, ln. 3126 - 3127)

Comment 78: This was discussed in a previous comment.

To the best of my knowledge, if this is true, it would be only for a handful of vehicles, which are produced in the U.S. but sold overseas. Even if true, the exposure scenario is irrelevant because there is no exposure to asbestos associated with installing new brakes on a new car! The Agency should be more transparent in discussing which automobile shop(s) they are discussing in this section.

2.3.1.7.1 Process Description – Aftermarket Automotive Brakes/Linings and Clutches

Statement 79: "Based on the long history of the use of asbestos in automobile parts, and because aftermarket automotive parts may still be available for purchase, the Agency believes this COU is still ongoing." (page 87, ln. 3141 - 3142)

Comments 79: I do not believe that this statement is accurate, and I hope that my previous comments about its lack of validity are convincing.

Automobile Brakes

Statement 80: "New automobiles manufactured in the United States had brake assemblies with asbestos-containing components. (page 87, ln. 3157 – 3158)

Comment 80: As noted in my paper (Paustenbach et al., 2004), to the best of my knowledge, the last new car to have an asbestos-containing brake was made around 1984 (there are claims that

some models had them until 1986 or 1987), and the vast majority of automobiles had already converted to non-asbestos containing brakes by 1979.

Statement 81: "Since the mid-1990s, material and design improvements have led to most cars being manufactured with disc brakes, effectively phasing out drum brakes in passenger automobiles (Richter et al., 2009)." (page 88, ln. 3169 - 3171)

Comment 81: I am a co-author of the Richter et al. (2009) article that the Agency has cited, and the Agency has not correctly represented its contents.

On page 459, the full quote is, "The introduction of disc brakes in the 1960s in the United States (1950s in Europe) further reduced the need for machining activities, because the old disc brake pads were simply removed and replaced with new ones. By the mid-1970s, most automobiles in the United States had a combination of front disc brakes and rear drum brakes. By the 1990s, most automobiles sold in the United States had disc brakes on all four wheels."

This needs to be corrected.

Statement 82: "Use of asbestos-containing braking systems began to decline in the 1970s due to many factors, including toxicity concerns, rising insurance costs, regulatory scrutiny, challenges associated with disposing of asbestos-containing waste, and availability of asbestos-free substitutes (Paustenbach et al., 2004)." (page 88, ln. 3180 - 3182)

Comment 82: This has been appropriately quoted from my 2004 paper. I would add that litigation associated with asbestos that has occurred since 1975 ensured that no asbestos-containing auto parts would be present in cars in the post-1980 or 1985 era.

Statement 83: "However, the Agency knows of at least one company that imports asbestos-containing friction products for use in cars assembled in the U.S., but those vehicles are exported for sale and are not sold domestically." (page 88)

Comment 83: This was addressed in a previous comment.

Statement 84: "Today, individual consumers can find aftermarket automotive products marketed as containing asbestos through online retailers." (page 89, ln. 3204 - 3205)

Comment 84: I am shocked that there continue to be sales of asbestos-containing brakes from China and Canada, as noted in the letter "RE: Request to Designate Asbestos as a High-Priority Substance Under TSCA § 6 Due to Use in Brake Friction Materials" (by the Motor & Equipment Manufacturer Association), but I will assume that this letter is accurate. However, the Agency should be more transparent in noting that MEMA states that <1% of all of the brakes sold in the US might contain asbestos in 2013. I believe all would be disc brakes so there should be no exposure, as the wear debris would fall to the road during braking.

Statement 85: "Despite this trend, asbestos in automotive parts is not banned at the federal level, and foreign suppliers face no restrictions (other than those currently in place in the states of California and Washington) when selling asbestos-containing brake products to business establishments and individuals in the United States. The Motor and Equipment Manufacturers Association informed EPA that approximately $2.2 million of asbestos-containing brake materials were imported into the United States in 2014 (MEMA, 2016)." (page 89, ln. 3217 - 3222)

Comment 85: This was addressed in a previous comment.

Statement 86: "Consistent with the history for brakes, friction materials in clutches moved from asbestos-containing to asbestos-free designs over recent decades. By the 1980s, automobile manufacturers began using various asbestos-free substitutes in clutch assemblies (Jiang et al., 2008); and by 2000, most automobiles in the United States were no longer made with asbestos-containing clutches (Cohen and Van Orden, 2008). However, aftermarket clutch parts may contain asbestos. As evidence of this, Jiang et al. (2008) reported purchasing 27 boxes of asbestos-containing clutch discs that had been stockpiled at a parts warehouse (Jiang et al., 2008), suggesting that stockpiles of previously manufactured asbestos-containing clutch assemblies could be available." (page 90, ln. 3244 - 3251)

Comment 86: I am a co-author of the Jiang et al. (2008) article. It is incorrect to suggest that it is plausible in 2020 to obtain clutches or brakes that contain asbestos from a warehouse. As noted in our article, we found clutches manufactured before 1980 in a remote warehouse that stored old parts for the restoration of antique vehicles. This was a rare find, even approximately 15 years, ago when we were able to locate those components.

We believe the bulk of exposures when handling boxes of old brakes or clutches was due to dust on the outside of the box. This dust contained asbestos which apparently fell on the boxes while they were being stored near the production area (for short or long periods of time). We saw little dust inside the boxes.

Statement 87: "While brake linings and pads at installation may contain between 40 and 50 percent chrysotile asbestos (i.e., fibers longer than 5 micrometers) (OSHA, 2006), brake dust is largely made up of particles and fibrous structures less than 5 micrometers in length, which would no longer be measured as asbestos by PCM." (page 91, ln. 3297 - 3300)

Comment 87: This comment is irrelevant concerning the future possible exposures to asbestos for brake mechanics. No brakes in the marketplace today, other than perhaps those few imported from Canada or China (identified above), contain asbestos. I am currently investigating whether these foreign brakes that contain asbestos are still entering the United States. I checked ten years ago, and I was unable to find them.

As noted, several times, that as they are disc brakes, there would be no exposure to asbestos, as the wear debris would fall to the roadway.

Statement 88: "Other researchers have reported lower values, indicating that brake dust typically contains less than 1 percent asbestos (Paustenbach et al., 2003). This wearing and degradation of asbestos in brake parts must be considered when assessing worker exposures." (page 91, ln. 3302 - 3305)

Comment 88: I agree with this statement.

Statement 89: "(Cohen and Van Orden, 2008) evaluated clutch assemblies from a vehicle salvage yard and found that clutch plates, on average, contained 43 percent asbestos, while the dust and debris in clutch housings, on average, contained 0.1 percent asbestos (Cohen and Van Orden, 2008)." (page 91, ln. 3318 - 3320)

Comment 89: It is noteworthy that when the authors studied the replacement of these clutches, even during the era when they contained asbestos, there were very low concentrations of asbestos measured in the air near the workers. Since the Agency is concerned only about future exposures, I would expect that virtually no workers will be handling asbestos-containing clutches in the coming years in the United States. No US manufacturer has used asbestos in a clutch for over 40 years.

I suggest dropping this discussion of clutches because it is not applicable in the modern era or going forward.

Statement 90: "First, clutches generally do not need to be repaired as frequently. By estimates made in 2008, clutches typically last three times longer than brake linings (Cohen and Van Orden, 2008). Second, a common clutch repair method is to remove and replace the entire clutch assembly, rather than replacing the clutch disc component (Cohen and Van Orden, 2008). These two factors likely result in clutch repair asbestos exposures being lower than comparable brake repair asbestos exposures." (page 92, ln. 3322 - 3327)

Comment 90: It seems that the Agency is aware that in the coming years, there is virtually no likelihood that persons will be exposed to asbestos from clutch repairs for the reasons stated above and in previous comments.

Statement 91: "In the late 1980s, NIOSH conducted a series of industrial hygiene surveys on brake repair facilities, and the Agency estimated that 155,000 brake mechanics and garage workers in the United States were potentially exposed to asbestos (OSHA, 2006). In 1994, OSHA estimated as part of its updated asbestos rulemaking that 676,000 workers performed automotive repair activities, and these workers were found in 329,000 establishments (i.e., approximately two workers per establishment) (Federal Register, 1994)." (page 92, ln. 3331 - 3336)

Comment 91: I agree that over 40 years ago, there were a large number of mechanics potentially exposed to asbestos while performing brake work (albeit at very low exposures). Since mechanics have not had access to asbestos-containing brakes since then, I would not expect them to be exposed to asbestos in the future. Professional mechanics would not be permitted to use aftermarket brakes, which contained asbestos, since they would not meet the specifications of the brake manufacturer.

For a reasonably accurate description of the number of mechanics who might be exposed to asbestos due to brakes, I would refer you to the paper by Finley et al., (2012, 2013). I am a co-author of those papers, and we discuss why Dr. Lemen's analysis was critically flawed.

Today, because virtually all cars have disc brakes, that even if they did contain asbestos, there would be no opportunity for exposure, as the brake wear debris falls to the ground during braking.

Statement 92: "Accordingly, EPA estimates that this COU has 749,900 ONUs." (page 92, ln. 3346 - 3347)

Comment 92: Among all the many claims that I find unjustified in this document, this one is among the most unfounded.

I fail to find a justification for using this number for the reasons stated in previous comments. Indeed, I would expect that no persons going forward (rather than 749,900 persons who the Agency predicts will be exposed) would be exposed to asbestos while performing automotive brake or clutch work now, in the past 20 years, or in the coming years. The rationale for my view is simple; the brakes and clutches have not contained asbestos for more than 40 years.

Additionally, if there are imported brakes with asbestos, they would virtually all be disc brakes, which would not be associated with exposure to brake wear debris (the brake wear debris drops to the road) or arcing debris (because they don't require arcing) during the installation process.

Beyond the lack of persons working with asbestos-containing materials and the fact that even if they did, the exposures are expected to be minimal in the post-1980 era; Blake et al. (2003) conducted a simulation study to evaluate airborne asbestos fiber concentrations during brake replacement, filing, sanding, and arc grinding of new brake shoes, as well as during cleanup of the work area. This dataset applied to the era approximately from 1940 – 1975.

Personal 8-hour TWA concentrations during brake removal and replacement simulations ranged from 0.0031-0.0009 f/ml (PCM equivalent). Additional results revealed that the highest exposures occurred during arc grinding, with estimated personal 8-hour TWA concentrations ranging from 0.0347-0.0935 f/ml, while all other tasks were less than 0.02 f/ml. The authors noted that all samples and estimated 8-hour TWAs were below historical and current occupational exposure limits (Blake et al., 2003).

The Agency is using this data to describe exposures after 2020. The Agency should not be using information to describe practices that ended 25-35 years ago when performing exposure assessments going forward.

In addition to the above, research by Bernstein et al. (2020a, 2020b, 2015, 2014) indicated that even when there is exposure to brake wear debris from brakes that contained asbestos, the fibers have lost their biologic activity since they are filled with phenolic resin.

These resins were baked into place during the brake manufacturing process. Consistent with Langer's hypothesis (Langer, 2003), even the debris from an arcing machine, where the chrysotile had not been converted to forsterite, failed to produce an asbestos-like toxicological response in animal models (Bernstein et al., 2018).

One is hard-pressed to suggest that any discussion of the future hazards to mechanics who install brakes remain in this EPA document.

Statement 93: "For additional insights into OSHA sampling results, EPA considered the findings published by Cowan et al. (2015). These authors summarized OSHA workplace compliance measurements from 1984 to 2011, which included 394 PBZ samples obtained from workers at automotive repair, services, and parking facilities (Cowan et al., 2015)." (page 92, ln. 3358 - 3361)

Comment 93: EPA correctly noted that in the Cowan et al. (2015) article, who was a former colleague, as shown in Table 2-14, none of the air samples collected after 1990 contained asbestos. Of the total 152 personal samples collected between 1984 and 2011 at automobile dealers and service stations, 144 had no detectable concentrations of asbestos (Cowan et al., 2015). For the eight samples where asbestos was measured, the average concentration was about ½ the current OSHA PEL, and they were collected between 1984 and 1999. Of the 42 area samples collected at those facilities, all had no detectable concentrations of asbestos in the air. This information is from Table 2 of the Cowan et al. (2015) article. It appears that the Agency failed to recognize the importance of these data.

The results presented in this table show that after 1990, for the 119 samples detected, all had no detectable asbestos concentrations. It seems disingenuous to list at the bottom of that table, a range of values from 0.0031 – 35.6 fibers/cc, implying that it represents the typical range of exposures for the last 35 years. Indeed, all of those samples shown in that range were detected before 1990.

This confirms the validity of my previous comments regarding a lack of exposure to asbestos over the last 35 years. It also supports my view that if foreign-made brakes containing asbestos are entering the market, it is not reflected in the data. This includes the data sampled by OSHA and those reported by Paustenbach et al. (2003); Jiang et al. (2008); Richter et al. (2009).

It would be useful for EPA to review the Cowan et al. (2015) paper again, as, on page 627, the authors noted that:

> "There are limitations to using OSHA compliance sampling data to evaluate the true exposure potential of workers to asbestos or other chronic disease agents over time. First, the OSHA compliance measurements do not represent random sampling of workplace conditions, nor do they represent an industrial hygiene risk management strategy, limiting the generalizability of these data (Coble et al., 2001; Gomez, 1993; Henneberger et al., 2004). OSHA compliance samples are collected for different purposes than those samples collected for the purposes of exposure and risk management. Hewett (2001) pointed out that these two different goals (compliance vs. risk management) are often misinterpreted or misused. The OSHA compliance sample is simply a snapshot in time, providing a yes or no answer to the question of whether a specific worker exposure scenario is in compliance with the regulatory standards over an 8-h period of time (Leidel, 1977; Tuggle, 1981). The proper interpretation of these single-shift sampling data points is very important in the context of evaluating true exposures to workers."

Table 2-14. PBZ Asbestos Concentrations Measured by OSHA for Workers at Automotive Repair, Services, and Parking Facilities

Time Frame	Number of Samples	Number of Samples Non-Detect for Asbestos	Number of Samples with Detected Asbestos	Range of Detected Asbestos Concentrations (fibers/cc)
1984-1989	274	241	33	0.0031 – 35.6
1990-1999	101	101	0	N/A
2000-2009	17	17	0	N/A
2010-2011	2	2	0	N/A
Total	394	361	33	0.0031 – 35.6

Notes: Data from (Cowan et al., 2015).
Data are personal breathing zone (PBZ) concentrations of unknown duration.

Statement 94: "Specifically, EPA considered five NIOSH in-depth survey reports published in 1987 and 1988 (Cooper et al., 1988, 1987; Godbey et al., 1987; Sheehy et al., 1987a; Sheehy et al., 1987b) and a 1989 NIOSH publication that reviewed these findings (OSHA, 2006). (page 93, ln. 3374 - 3376)

Comment 94: It is noted in the Agency's document that they assume that proper dust control measures have generally been used since 1988 and, as such, they assume that there will be virtually no exposures in the coming years.

Given that working premise, I do not understand why this document estimates exposures to be unacceptable high going forward for the approximately 750,000 mechanics in the United States. The data and their assumptions about no exposure make this entire analysis of brakes and automobiles irrelevant.

Statement 95: "EPA also considered the published literature on asbestos exposures associated with automobile brake repair." (page 93, ln. 3386 - 3387)

Comment 95: This entire section on brakes and clutches on pages 93 – 97 of this document seem irrelevant, in light of the previous comment.

Statement 96: "PBZ samples were collected during seven test runs, and measured asbestos concentrations ranged from 0.0146 fibers/cc to 0.4368 fibers/cc, with the highest level observed during arc grinding operations." (page 93, ln. 3394 - 3395)

Comment 96: This discussion about arc grinding seems irrelevant, especially when discussing future asbestos exposures. This is because it is assumed that proper engineering controls will be used, and Bernstein et al. (2014, 2015) work shows that even in these air samples, the generated dust is not biologically active.

Beyond that, drum brakes have not been used in cars in the United States since about 1980. There is minimal exposure, if any, to brake dust or brake wear debris with a disc brake.

Statement 97: "EPA selected a central tendency 8-hour TWA exposure value for workers (0.006 fibers/cc) by assuming the median short-term exposure level could persist for an entire workday. This is a reasonable assumption for full-time brake repair mechanics, who may conduct 40 brake repair jobs per week, and a protective assumption for automotive mechanics who do not repair brakes throughout their shifts." (page 95, ln. 3451 - 3456)

Comment 97: For exposures that occurred among some mechanics from 1940 to 1980, the Agency's selection of a central tendency of 0.006 fibers/cc for an eight-hour TWA concentration is reasonable.

However, for the reasons stated above, one would not expect even a handful of people in the future to be exposed to asbestos while working with automobiles.

Therefore, the section involving brakes and clutches needs to be rewritten, or the exposure scenario dropped. The Agency needs to make it clear that they are not claiming that exposures going forward poses an unreasonable risk, given the low plausible airborne asbestos concentrations (if any) and the few people likely exposed while performing automobile repairs.

Statement 98: "PCM-based personal exposure measurement in an automotive repair facility may overstate asbestos exposures, which some studies have demonstrated through TEM analyses of filter samples (Blake et al., 2003; Weir et al., 2001)." (page 95, ln. 3482 - 3484)

Comment 98: The Agency is correct in noting that PCM data almost always yields higher results than the TEM data. This is due to the number of fiberglass fibers and clothing fibers in the air of many workplaces that are detected and classified as asbestos-like fibers in the PCM method.

In the take-home studies that my colleagues and I have performed, often 50% of the PCM fibers were due to clothing.

Statement 99: "Automotive repair facilities involve many machining operations that can release non-asbestos airborne fibers, such as cellulose fibers from brushes and metal and plastic fragments from body repair (Blake et al., 2008)." (page 95, ln. 3486 - 3488)

Comment 99: This is an accurate statement.

Statement 100: "While EPA has verified that U.S. automotive manufacturers are not installing asbestos brakes on new cars for domestic distribution, EPA has identified a company that is importing asbestos-containing brakes and installing them in their cars in the United States. These cars are exported and not sold domestically." (page 96, ln. 3501 - 3504)

Comment 100: This has been addressed several times in previous comments.

2.3.1.8.1 Installing New Brakes on New Cars for Export Only

Statement 101: "EPA did not identify any studies that contain exposure data related to installation of asbestos-containing brakes from an Original Equipment Manufacturer (OEM). As a result, the exposure assessment approach used for the aftermarket automotive brakes/linings and clutches described in Section 2.3.1.7 was also used for this COU and is reported here in Table 2-16." (page 96, ln. 3512 - 3515)

Comment 101: The Agency's assumptions about how automotive repair work currently and in the future involves exposure to asbestos has not been relevant since the mid-1980s (if not years before); as such, I do not understand their rationale for stating that nearly 1,000,000 automobile mechanics will likely be exposed to asbestos in the coming years.

2.3.1.8.2 Use of Brakes/Frictional Products for a Single, Larg[e] Transport Vehicle (NASA Super-Guppy).

Statement 102: "This section evaluates asbestos exposures associated with brake block replacement for the Super Guppy Turbine (SGT) aircraft, which is operated by the National Aeronautics and Space Administration (NASA)." (page 97, 3541 - 3543)

Comment 102: Due to the few numbers of persons potentially exposed to asbestos who work on this aircraft, and the certainty that they are utilizing appropriate engineering controls for asbestos dust, this exposure scenario should not be of regulatory concern.

According to the Agency's document, only one or two persons are involved with replacing the brake pads of this aircraft. This document states that all work is done under wet methods or a high-efficient particulate vacuum. Additionally, the data presented by the EPA on page 100 indicates that there is no measurable exposure. Specifically, "Three of the five sampling results that NASA provided were labeled as "8-hour TWA" observations, and EPA considered these to be representative of full-shift exposures. The three results for this exposure duration were: <0.003 fibers/cc, <0.006 fibers/cc, and <0.0089 fibers/cc (NASA, 2020a). To calculate the central tendency for full shift exposure, EPA replaced the three observations with one-half of the detection limit and calculated the arithmetic mean of those three values. By this approach, EPA calculated a central tendency concentration of <0.003 fibers/cc."

Gaskets

2.3.1.9 Other Gaskets-Utility Vehicles (UTVs)

Statement 103: "EPA has identified the use of asbestos-containing gaskets in the exhaust system of a specific type of utility vehicle available for purchase in the United States. This COU is identified as "other gaskets" in Table 1-4. of Section 1.4.2. It is known that these UTVs are manufactured in the United States, so EPA expects that there is potential for exposures to workers who install the gaskets during assembly and workers who may repair these vehicles." (page 100, ln. 3662 - 3666)

Comment 103: It is my understanding that this industry voluntarily discontinued the use of asbestos-containing materials in these vehicles manufactured in the United States. Even if they did not, there is

no exposure when installing asbestos-containing gaskets, and the removal of these gaskets, as discussed in the document, under any plausible scenario, produces low airborne concentrations of chrysotile.

For this exposure scenario to be relevant to TSCA, it would have to be reasonable to expect that these gaskets would be available in the future and used in these vehicles. It has been established in this document that the only gaskets manufactured in the United States that contain asbestos are of limited use in the Chlor-alkali facilities (and perhaps in some high acid streams in refineries).

As previously noted, it has been challenging for those of us conducting retrospective exposure analyses to find new asbestos-containing gaskets over the past 30 years in the U.S.. Thus, this exposure scenario seems unnecessary and unfounded since the EPA is focused on possible future exposures.

As many as 50 years ago within the occupational health community, exposures to asbestos from asbestos-containing gaskets had not been considered to pose a significant health risk (even by Dr. Selikoff, see his paper in 1970 – Selikoff, 1970). I can find no reason for this exposure scenario to be presented with the "context" regarding the current environment and that in the U.S. in the coming years.

Statement 104: "EPA's estimate of occupational inhalation exposures is based on a 2006 study (Paustenbach et al., 2006), in which workers at a muffler shop removed exhaust systems from 16 vehicles. The vehicle model years ranged from 1946 to 1970; and 12 of the 16 vehicles were found to have asbestos in some combination of the mufflers, manifold gaskets, and exhaust pipe gaskets. The measured asbestos content in these components ranged from 9.5 to 80.1 percent, with only chrysotile asbestos fibers detected." (page 104, ln. 3752 - 3756)

Comment 104: This is an accurate statement. However, it is noteworthy that in my many years of research on this matter, our group did not find automobiles manufactured after 1975, which had asbestos-containing gaskets in the exhaust system. I considered myself fortunate to be able to find abandoned autos that had them so we could conduct our work (which was only possible in California where the cars don't rust heavily, and they tend to be collected).

2.3.1.9.5 Data Assumptions, Uncertainties and Level of Confidence

Statement 105: "Further, this assessment assumes that data from one publication (Paustenbach et al., 2006) are representative of exposures for this condition of use. However, the job activities and exposure scenarios considered in the publication differ from the UTV-related exposures in at least two ways." (page 105, ln. 3807 - 3810)

Comment 105: In my opinion, the measured exposures mentioned here are representative of gasket practices in the pre-1980s era. They are not relevant going forward.

Statement 106: "It is unclear if the asbestos content in the automobile exhaust systems from pre-1970 automobiles are representative of the asbestos content in today's UTV exhaust systems." (page 105, ln. 3816 - 3817)

Comment 106: I see no reason to expect asbestos exposures post-2020 since gaskets have not been made of asbestos for nearly 40 years (except for the uses mentioned in this document). The exception is the handful of oversea utility vehicle manufacturers that may have used asbestos-containing gaskets.

Perhaps as importantly, the Agency has suggested that the average concentration for estimating asbestos exposure during gaskets replacements is 0.005 fibers/cc, which is a small fraction of the OSHA PEL. Additionally, 16 of the 21 area samples were non-detects for asbestos via PCM analysis in the cited Paustenbach et al. (2006) article.

Statement 107: "Moreover, five of the personal breathing zone samples collected from mechanics had filters overloaded with particulate, and these samples were not analyzed. The authors noted that the overloaded filters may have resulted from particulate matter released while mechanics used torches to cut and weld exhaust pipes, but EPA cannot rule out the possibility that these overloaded filters might have contained elevated levels of asbestos." (page 105, ln. 3834 - 3838)

Comment 107: The Agency needs to reflect on the reasonableness of their concerns regarding the possibility that some of the filters from the Paustenbach et al. (2006) study were overloaded with asbestos.

To be honest, the concern is almost laughable; as among the 23 valid samples, 17 had no detectable concentrations of asbestos by PCM, and six had maximum concentrations up to 0.0505 fibers/cc (Paustenbach et al., 2006). All the data indicate virtually no airborne fibers, so why would the Agency suggest that we overloaded the others with asbestos?

The TEM analyses identified asbestos fibers in seven of the sampling filters. I would suggest that the Agency embrace this sentence: "Overall, based on the PCM analysis of the 23 valid samples, the study authors reported an average worker asbestos concentration of 0.024 fibers/cc and a maximum concentration of 0.066 fibers/cc (Paustenbach et al., 2006)."

I collected the air samples for many of the activities in that article. Working under a 20+-year-old car introduces you intimately to the amount of road dust that resides in a car's undercarriage. We collected 23 samples, 17 had no detectable concentrations of asbestos (by PCM).

There is a near-zero probability that the six samples that had detectable asbestos concentrations were overloaded with chrysotile asbestos fibers from an encapsulated gasket. I recollect that those six samples were visually dark due to what appeared to be dirt from the undercarriage.

2.3.1 Consumer Exposures

Statement 108: "This section summarizes the data used for estimating consumer inhalation exposures to asbestos for two potential do-it-yourself (DIY) scenarios: (1) brake repair/replacement and (2) gasket repair/replacement in Utility Vehicles (UTVs). Specifically, the brake repair/replacement scenario involves repair or installation of imported aftermarket brake pads (disc brakes) or brake shoes (drum brakes) containing asbestos." (page 107, ln. 3877 - 3881)

Comment 108: As noted previously, the Agency states that they performed this analysis under the assumption that proper engineering controls would be used during brake replacements. This would eliminate exposures to not only the worker but also for any bystander. Thus, it is not clear why this analysis was presented in this document.

On a more practical note, as previously stated in my comments, few functioning cars still use drum brakes. By and large, these were eliminated 40 years ago for most cars in the United States. Disc brake pads present no opportunity for exposure during installation and virtually no chance of exposure when replaced.

Furthermore, I expect that few persons, in the modern era, would attempt to replace a drum brake shoe assembly without significant experience, and of course, they would be repairing antique automobiles. Beyond that, since less than 1% of new brakes "might" contain asbestos, according to the Agency's estimates, and given the relatively few drum brakes that are replaced yearly in the United States, this makes it improbable that there would be any asbestos exposure. Therefore, I would recommend that this exposure scenario is without foundation and can be removed from this document.

As an aside, the Agency's proposed approach for estimating bystander exposure is a reasonable one. However, a more elegant approach has been presented by Donovan et al. (2011). Also, the underlying calculations are discussed in quite a bit of detail in subsequent letters to the editor from Egilman and Schilling (2012) and Donovan et al. (2012). Over the past ten tears, numerous authors have adopted that approach to estimate bystander exposures to asbestos fibers.

Statement 109: "EPA has found no reasonably available information to suggest that asbestos-containing brakes are manufactured in the United States, and based on stakeholder outreach, the Agency does not believe that any domestic car manufacturer installs asbestos-containing brakes in new cars sold domestically.[10] However, consumers can purchase asbestos-containing brakes as an aftermarket replacement part for cars as well as asbestos containing gaskets for UTV exhaust systems." (page 108, ln. 3893 - 3897)

Comment 109: As noted in many previous comments, in the post-2020 era, it seems to be highly unlikely that any non-mechanic would attempt to replace a drum brake on a 40-year-old car. This exposure scenario was more reasonable from the 1950s through the 1970s. As cars manufactured over the past 40 years rarely have drum brakes, this exposure scenario is not worthy of the Agency's attention.

The complexity of the calculations that are presented are needless given the minimal number of potentially exposed persons in the United States, who are not mechanics, that would work with brakes on a car and be assured that it could be driven safely.

2.3.2.1 Consumer Inhalation Exposures of Do-It-Yourself (DIY) Mechanics During Brake Repair: Approach and Methodology

Statement 110: "This consumer assessment addresses potential scenarios in which a DIY consumer installs, repairs or replaces existing automobile brakes with imported aftermarket brake pads or shoes containing asbestos; including brake linings and clutches." (page 108, ln. 3911 - 3913)

Comment 110: As discussed in previous comments, if this exposure scenario is plausible, it likely only affects a small handful of persons across the country. As they do not have the equipment or mechanical skills, and only approximately <1% of imported brakes from China or Canada allegedly contain asbestos.

Beyond this, the EPA stated that they assumed modern brake replacement techniques were to be used, and these would eliminate exposure.

Statement 111: "While peer-reviewed literature indicates much of the asbestos brake pad or shoe use has been phased out and the majority of existing cars on the road do not have asbestos brakes (Finley et al., 2007), asbestos-containing brakes and shoes can still be purchased in the United States." (page 108, ln. 3913 - 3916)

Comment 111: I have covered this in detail in previous comments. It is challenging to find brakes that contain asbestos being sold over the internet.

However, even after they are sold, and after the brakes have been driven 25,000 – 70,000 miles, the asbestos exposures would be likely immeasurable small due to the degradation of the chrysotile to forsterite (Jacko et al., 1973; Rowson, 1978; Williams and Muhlbaier, 1982; Boelter et al., 2007; Madl et al., 2009), dilution by wind currents, the wear debris dropping to the roadway, and possible use of wet methods. Importantly, these fibers have lost their biologic activity for causing an asbestos-producing disease (Bernstein et al., 2018, 2003).

Note, as before, one has to assume that the brakes imported today will all be disc brakes.

Statement 112: "Consumer exposure during DIY brake repair is expected to differ from occupational brake repair in four ways (Versar, 1987): (1) consumers generally do not have a fully equipped professional garage to perform auto repairs (in some cases, the repairs would occur in an enclosed garage); (2) consumers would not wear respirators, mitigate dust emissions, or have available the professional equipment found in commercial repair shops; (3) consumers have limited experience, and thus the time required to make repairs would be longer; and (4) consumers are unlikely to perform more than one brake job per year and it was assumed that only one consumer would perform the task of replacing asbestos brakes or shoes." (page 109, ln. 3935 - 3942)

Comment 112: Although the Agency has already stated that they assume that brakes will be changed under conditions that do not produce exposure to asbestos, this document continues to try to build the narrative that this work practice will result in asbestos exposures. This is even though brakes in the United States have not contained asbestos for more than 30 years.

I find it difficult to understand why a 30-year-old study, which was intended to replicate workplace practices performed 40 – 50 years ago, would be relied upon to predict exposure in the coming years.

As noted previously by the Agency, these activities were not to be a part of their analysis because of the assumption that there would be no asbestos exposure when proper work practices were used.

Statement 113: "Duration of Activity: Available literature indicates a typical "brake job" for a professional brake mechanic for a single vehicle takes between one and two hours (Paustenbach et al., 2003). No data were found in existing literature on the length of time needed for a DIY consumer to perform a brake job. EPA assumes a consumer DIY brake repair/replacement event could take twice as long as a professional mechanic, or about three hours (double the mean of time found in the literature for professional mechanics)." (page 109, ln. 3972 - 3978)

Comment 113: The quote from my 2003 paper is accurate. In my opinion, to conduct a quantitative evaluation of a homeowner (DIYer), replacing a drum brake should not be worthy of regulatory review.

I fear that this document has probably cost taxpayers the better part of one or two million dollars to design, identify a contractor, to competitively bid it, to issue the award, then have it executed by the contractor, reviewed by the EPA project officer and their staff, then prepare written comments, which then needed to be considered by the contractor and turned in to a final product.

Then there was a pre-meeting with stakeholders before it was printed in the Federal Register, then it was issued for comment in the Federal Register, which resulted in persons having to read several hundred pages of comments and respond to them. But even before the agency issues its response, approximately 20 outside consulting scientists (e.g., ad hoc SAB members) were paid to read the document and all of its comments and then convene for a three-day meeting.

I have long admired much of EPA's activities, but as a taxpayer and scientist I have to believe that in the era of the COVID virus, global warming, and the need better to control the distribution of plastic debris across the planet, the EPA must "have bigger fish to fry."

Statement 114: "Cleaning methods: EPA assumes, for the indoor scenario, a consumer may use compressed air to clean brake assemblies since it was historically utilized, is still readily available to consumers (canned air or air compressor systems), and nothing prohibits consumers from using compressed air. EPA assumes, for the outdoor scenario, a consumer does not use compressed air." (page 110, ln. 3980 - 3984)

Comment 114: Based on my experiences over the past 40 years, 90% of brake jobs conducted by consumers were done outdoors (especially in the era which involved the removal of brake drums containing a nuisance quantity of dust particles). No mechanic would want to blow the dust out in a garage.

Further, it is disappointing that the Agency has made almost implausible assumptions throughout this risk assessment. I dare say that I have never met anyone who even alleged to have blown out brake drums using a portable air compressor in their garage with the doors closed.

Statement 115: "Possible additional work during repair/installation of brakes: EPA assumes a consumer may perform additional work on brakes, like arc grinding, hand filing, or hand sanding of brake pads as part of the brake repair/replacement work." (page 110, ln. 3986 - 3988)

Comment 115: I disagree with the Agency that today, a DIYer would sand the brake pads in an attempt to remove glazing. Glazing occurred more often in the 1950s – 1970s than it has since 1985 due to the better design of automobiles, the lack of brake drums on modern cars, and the adoption of disc brakes.

I remember my father, who was a service manager of over 20 mechanics throughout his lifetime, complaining that he had just put a glaze on the brakes because he went down a steep hill with the brake firmly placed against the floor and could hear the screech of the resulting glaze. Even as an experienced mechanic, in 1965, he had no interest in jacking up the car in the front yard and attempting to remove the brake mechanism, hand sand the face of the brake, and reinstall it with the hope that it would quit squealing.

In the year 2001, when I began seriously studying asbestos, I attempted to find a grinder for brake shoes, which was used in the prior 20 years. It was not easy to find that device. They had either long been replaced with more modern arcing devices or were thrown away because they were no longer necessary. Modern arcing machines are manufactured, but they are rare since it is rare that one works on a car with drum brakes.

I recognize that some arcing devices are still sold today, but there is a minimal chance that they are purchased by a DIYer weekend mechanic and kept in their garage. Indeed, I have never seen an arcing device in the past 40 years that was in anyone's home garage.

Statement 116: "Frequency of brake repair jobs: EPA assumes the average consumer performs a single brake repair/replacement job about once every three years." (page 110, ln. 3995 - 3996)

Comment 116: This is a reasonable assumption if they are talking about the average consumer hiring a professional to change the brakes.

Statement 117: "For two baseline tests, no additional manipulation of the brake shoes (such as filing, sanding, or arc grinding) was conducted. The remaining four tests involved additional manipulation of the brake shoes as follows:

1) arc grinding of the new shoes to precisely match each shoes' radius to that of its companion brake drum (n = 2), and

2) sanding to bevel the edges and remove the outermost wear surfaces on each shoe (n=1), and 3) filing to bevel the square edges of the shoe friction material prior to installation (n=1)." (page 111, ln. 4049 - 4057)

Comment 117: As noted previously, the Agency is investing a significant number of hours in performing calculations about exposure scenarios that are not remotely plausible to exist in the United States going forward.

2.3.2.1.2 Exposure Data for Use in Risk Evaluation – Do-It-Yourself (DIY) Mechanics During Brake Repair

Statement 118: "Consumer inhalation exposure to asbestos for the DIY brake repair/replacement scenario was assessed for both the consumer user (individual doing the brake repair/replacement work) and a bystander (individual observing the brake work or present within the garage during the brake work). Consumer inhalation exposure was evaluated for two conditions for the consumer user and bystander." (page 112, ln. 4076 - 4079)

Comment 118: I do not take issue with most of the exposure calculations in this document, other than the fact that they are generally implausible in the post-2020 era. Creating Tables like 2-26 seems unnecessary given all of the other problems that have been identified in the document, and the airborne concentrations will be well below the OSHA PEL for asbestos.

Statement 119: "For purposes of utilizing the information provided in Table 2-26 within this evaluation, EPA applied the personal breathing zone (PBZ) values to the DIY consumer user for the indoor and outdoor scenarios under the assumption that hands on work would result in exposure within the PBZ of the individual." (page 112, ln. 4093 - 4095)

Table 2-26. Exposure concentrations from Blake (2003) and Sheehy (1989) studies to the DIY user during various activities

Study	Activity	Duration	Concentration (fibers/cc)		Location	Confidence Rating
		(hours)	PBZ	<3 m from auto		
(Blake et al., 2003)	Brake shoe removal/ replacement	1.5	0.0217	0.00027	Indoors	Medium
		1.4	0.0672	0.0258	Indoors	Medium
	Filing brakes	1.7	0.0376	0.0282	Indoors	Medium
	Hand sanding Brakes	1.6	0.0776	0.0133	Indoors	Medium
	Arc-grinding Brakes	1.7	0.4368	0.0296	Indoors	Medium
		1.6	0.2005	0.0276	Indoors	Medium
	Cleaning facility	0.5	0.0146	0.0069	Indoors	Medium
(Sheehy et al., 1989)	Brake shoe removal/ replacement	Unknown [a]	0.007	Not monitored[b]	Outdoors	Medium

[a] No monitoring duration was provided within the study.
[b] This study did not include outdoor area monitoring which could be applied to the bystander

Comment 119: I believe that most would agree that this is an overly conservative assumption.

Statement 120: "EPA applied the area monitoring data obtained less than 3 meters from the automobile for the DIY bystander for the indoor scenario under the assumption that the bystander could be an observer closely watching the work being performed, an individual learning how to do brake

repair/replacement work, or even a child within the garage while the brake work is being performed." (page 113, ln. 4099 - 4102)

Comment 120: In my opinion, this exposure scenario is nearly implausible.

Statement 121: 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[11]. (page 113, ln. 4109 - 4112)

Comment 121: This is a reasonable assumption, but I would prefer the numbers suggested by Donovan et al. (2011).

Do it Yourself (DIY) Consumer User

Indoor Scenario

Statement 122: "While this activity may not be common practice for all brake repair/replacement activities, affordable grinding machines are readily available to those DIY consumers interested in purchasing and utilizing such equipment. Additionally, such equipment is also available for rental from various stores." (page 113, ln. 4123 - 4125)

Comment 122: This was discussed in a previous comment.

Statement 123: "For this risk evaluation, EPA used the average of the two-brake shoe removal/replacement values within the (Blake et al., 2003) study as the central tendency value for the indoor scenario." (page 113, ln. 4130 - 4131)

Comment 123: This assumption is acceptable.

Statement 124: "While the use of compressed air is not a recommended practice, no reasonably available information was found that surveyed actual cleaning methods used or preferred by DIY consumers for this scenario. EPA therefore utilized these values to evaluate consumer inhalation exposure with the understanding that they may represent a more conservative exposure concentration value." (page 114, ln. 4135 - 4139)

Comment 124: I do not understand the logic behind this statement. The Agency has clearly stated in their document that their analysis is based on the assumption that proper dust control measures will be used by persons involved with the brake change. Beyond that, this exposure scenario is only remotely plausible in the post-2020 era, for probably no more than 100 or 200 persons in the US (if any).

Outdoor Scenario

Statement 125: "EPA utilized the personal breathing zone concentration from the (Blake et al., 2003) study obtained during filing of brakes for the high-end exposure concentration for the consumer user under the outdoor scenario. Although this value was obtained in an indoor environment it is a potential additional work activity that could also be performed outside. Additionally, even though it is outdoors, it is expected that filing work would entail the consumer user's personal breathing zone to be very close to the brakes being filed and therefore high air exchange rates and outdoor volumes would not be expected to have a considerable impact on the exposure during such work." (page 114, ln. 4142 - 4148)

Comment 125: Prior comments addressed this matter.

Statement 126: "The (Sheehy et al., 1989) study is the only study identified through the systematic review process which included PBZ monitoring data for a DIY consumer user during outdoor brake repair/replacement work." (page 114, ln. 4152 - 4154)

Comment 126: I have no issue with this statement.

Bystander

Indoor Scenario

Statement 127: "EPA utilized the (Blake et al., 2003) area sampling data obtained within three meters from the automobile on which the work is being performed to represent exposure concentrations for the bystander under the indoor scenario." (page 114, ln. 4163 - 4165)

Comment 127: This has been addressed in a previous comment. Using the Blake et al. (2003) data for future exposure scenarios is not appropriate.

Outdoor Scenario

Statement 128: "There were no area monitoring data for the outdoor work in (Sheehy et al., 1989) which could be representative of potential bystander exposure. As a surrogate, EPA used the analysis of reduction factors (RFs) based on available data for the gasket ONU exposure scenario." (page 114, ln. 4174 - 4176)

Comment 128: As noted in a previous comment, my preference would be using the Donovan et al. (2011) article for assessing bystander exposures.

3.3.2.1.3. Exposure Estimates for DIY Brake Repair/Replacement Scenario

Statement 129: "EPA assessed chronic exposures for the DIY brake repair/replacement scenarios based on the exposure concentrations, assumptions, and exposure conditions described above. Because

reasonably available information was not found to characterize exposure frequencies and lifetime durations, EPA made the following assumptions:

• Exposure frequency of 3 hours on 1 day every 3 years or 0.04 days per year. This considers car maintenance recommendations that brakes be replaced every 35,000 miles, and the average annual miles driven per driver in the United States is 13,476 miles/year (U.S. DOT, 2018).

• Exposure duration of 62 years. This assumes exposure for a DIY consumer user starts at 16 years old and continues through the average adult lifetime (78 years). EPA also used a range of exposures (for both age at first exposure and duration of exposure); these are further described in Section 4.2.3 of the Risk Characterization." (page 115, ln. 4189 - 4201)

Comment 129: The amount of work invested in this document and the care by which it was prepared reminds me of what a professor told me when he read my 20-page report about a lab experiment that I conducted in grad school. That is, elegant and precise writing will not cover up the faulty thinking that underpins the analysis.

Statement 130: Table 2-28 (page 115)

Table 2-28. DIY Brake/Repair Replacement - Exposure Levels for EPA's Risk Evaluation

Condition of Use	Category	Exposure Concentrations (fibers/cc)		Confidence Rating
		Central Tendency	High-End	
Aftermarket automotive parts – brakes (Indoor)	DIY User	0.0445	0.4368	Medium
	Bystander	0.0130	0.0296	Medium
Aftermarket automotive parts – brakes (outdoors)	DIY User	0.007	0.0376	Medium
	Bystander	0.0007	0.0038	Medium-Low

Comment 130: I believe that the calculated results are unreasonably high, for the reasons mentioned in previous comments. The problem is that the Agency has selected a scenario that involves purchasing the <1% of imported brakes from China or Canada that allegedly contain asbestos, and then uses many unreasonable exposure assumptions to describe an activity that has not been performed by many persons in the last 35 years.

Statement 131: "The volume of a former automobile repair facility is considerably larger than a typical residential garage and will have different air exchange rates. While this could raise some uncertainties related to the applicability of the measured data to a DIY consumer user environment, the locations of the measurements utilized for this evaluation minimize that uncertainty. The PBZ values are very near the work area and should not be affected by the facility volume or air exchange rates." (page 116, ln. 4221 - 4225)

Comment 131: I agree with this statement.

Statement 132: "While industry practices have drifted away from the use of compressed air to clean brake drums/pads, no reasonably available information was found in the literature indicating consumers have discontinued such work practices. To consider potential consumer exposure to asbestos resulting from brake repair/replacement activities, EPA uses data which included use of compressed air. However, EPA recognizes this may be a more conservative estimate because use of compressed air typically could cause considerable dust/fibers to become airborne if it is the only method used." (page 116, ln. 4249 - 4254)

Comment 132: I have addressed my concerns about this statement in previous comments.

AGENCY ANALYSIS CONCERNING GASKETS

2.3.2.2 Consumer Exposures Approach and Methodology – DIY Gaskets in UTVs

Statement 133: "There was no reasonably available information found in the published literature related to DIY consumer exhaust system gasket repair/replacement activities on UTVs." (page 117, ln. 4293 - 4294)

Comment 133: Prior comments address my concern about this statement.

Statement 134: "Thirty studies relating to gasket repair/replacement were identified and reviewed as part of the systematic review process for exposure. These studies were compared against a series of criteria to evaluate how representative the studies are for DIY consumer exhaust system gasket repair/replacement activity." (page 117, ln. 4300 - 4303)

Comment 134: Perhaps after the public meeting, some of the commenters can submit several unpublished studies on this topic. A large fraction of them offer credible data.

Statement 135: "When compared to these criteria, three of the thirty studies were fully evaluated; a 2006 study by Blake (Blake et al., 2006), a 2005 study by Liukonen ((Liukonen and Weir, 2005), and a 2006 study by Paustenbach (Paustenbach et al., 2006), as shown in Table 2-29." (page 117 – 118, ln. 4309 - 4314)

Table 2-29. Summary of Studies Satisfying Factors Applied to Identified Literature

Reference	Occupational	Consumer	Data Quality Rating (Score)
(Blake et al., 2006)	Yes	No	Medium (2.1)
(Liukonen and Weir, 2005)	Yes	No	Medium (2.0)
(Paustenbach et al., 2006)	Yes	No	Medium (1.7)

Comment 135: I appreciate that you relied on one of my papers, and I consider the others by Liukonen and Weir (2005) and Blake et al. (2006) to be worthy of citation and serious consideration. However, all of us were addressing exposures that existed 40+ years ago.

Statement 136: "For non-detectable samples reported within a study at their respective sensitivity limits, statistics were calculated based on the full sensitivity value for that sample. For non-detectable samples reported within a study below their respective sensitivity limits, statistics were calculated based on one-half the sensitivity limit for that sample. For non-detectable samples reports at levels greater than their respective sensitivity limits, statistics were calculated based on one-half the reported non-detectable value. Table 2-30 summarizes the data based on the methodologies described here." (page 118, ln. 4332 - 4337)

Table 2-30. Summary Results of Asbestos Exposures in Gasket Repair Studies

Study Engine Work Sample Type	Air Sample Data			Air Sample Concentrations (Fibers/cc)			Confidence Rating
	Sample Size	Non-Detectable Samples	Mean Sample Duration (Minutes)	Minimum	Maximum	Mean	
(Blake et al., 2006)	**28**	**14**	**140**	**0.002**	**0.027**	**0.007**	Medium
Engine Dissembly	**15**	**4**	**128**	**0.003**	**0.027**	**0.009**	Medium
Area	9	2	135	0.003	0.008	0.005	Medium
Personal	6	2	117	0.007	0.027	0.015	Medium
Engine Reassembly	**13**	**10**	**153**	**0.002**	**0.008**	**0.003**	Medium
Area	9	9	154	0.002	0.008	0.003	Medium
Personal	4	1	153	0.003	0.008	0.005	Medium
(Liukonen and Weir, 2005)							
Engine Dissembly	**29**	**26**	**53**	**0.004**	**0.060**	**0.018**	Medium
Area	10	10	58	0.004	0.059	0.016	Medium
Observer	3	3	43	0.004	0.057	0.026	Medium
Outdoor	2	2	112	0.006	0.006	0.006	Medium
Personal	14	11	44	0.011	0.060	0.019	Medium
(Paustenbach et al., 2006)							
Engine Dissembly	**94**	**61**	**39**	**0.002**	**0.066**	**0.014**	Medium
Area	22	15	46	0.002	0.015	0.005	Medium
Bystander	44	29	40	0.004	0.030	0.012	Medium
Personal	28	17	32	0.006	0.066	0.024	Medium

Comment 136: The approach described here is not unreasonable, but there are more elegant techniques that can be used when there is a high percentage of censored data (Ganser and Hewett, 2010; Helsel, 1990; Hewett and Ganser, 2007). Additionally, I would refer the Agency to many papers by Dr. Hessel on this subject.

Statement 137: "After review and consideration of all the information within each of the three studies, EPA used the (Paustenbach et al., 2006) study to evaluate DIY consumer exposure to asbestos resulting

from removal/replacement of exhaust system gaskets for this risk assessment. This study was used because it was specific to exhaust system work involving asbestos-containing gaskets. It also includes information applicable to a DIY consumer user (the individual[s] doing the gasket work) and the bystander (the individual[s] observing the gasket work).” (page 119, ln. 4343 - 4348)

Comment 137: I appreciate that you considered our 2006 article credible.

2.3.2.2.2 Exposure Estimates for DIY UTV Exhaust System Gasket 4393 Removal/Replacement Scenario

Statement 138: “There was no reasonably available information found within the literature providing specific information about the frequency of gasket change-out and it is expected that frequency can vary depending on the location of the gasket and the number of gaskets needing change-out at any one time. The exhaust system gasket on the engine manifold may be exposed to more extreme temperature fluctuations than one on the muffler and therefore experience more wear and tear requiring replacement more frequently. EPA assumes, for this evaluation, one or more gaskets will be replaced once every three years.” (page 120, ln. 4407 - 4412)

Comment 138: This assumption is acceptable for assessing exposures in the past, but of course, gasket replacement varied with geographic location. However, this discussion in not reasonable when you are looking forward to the post-2020 era.

Statement 139: “Exposure durations were assumed to be 62 years. This assumes exposure for the DIY consumer user starts at 16 years old and continues through the average adult lifetime of 78 years. Table 2-31 provides a summary of the data utilized for this evaluation.” (page 120, 4414 - 4416)

Table 2-31. Estimated Exposure Concentrations for UTV Gasket Repair/Replacement Scenario – DIY Mechanic and Bystander

Condition of Use	Type	Exposure Concentrations Fibers/cc		Confidence Rating
		Central Tendency	High-end	
UTV gasket Repair/replacement (Paustenbach et al., 2006)	DIY Consumer	0.024	0.066	Medium
	Bystander	0.012	0.030	Medium

Comment 139: I pray that I am not performing gasket work on my vehicles when I am 78 years of age.

Statement 140: “There is some uncertainty associated with the assumption that UTV exhaust system gasket repair/replacement activities would take a consumer a full three hours to complete. An internet search revealed some videos suggesting gasket replacement would take a DIY consumer 30 minutes to complete. This value mirrors the sampling time-frames within the (Paustenbach et al., 2006) study. However, the time needed for a DIY consumer to complete a full UTV exhaust system gasket repair/replacement activity can vary depending on several factors including location of gaskets, number

of gaskets, size of gasket, and adherence of the gasket and residual material once the system is opened up and the gasket is removed." (page 121, 4450 - 4457)

Comment 140: I think that this is an agonizing level of detail that is not warranted for this type of analysis, especially as these gaskets are, almost certainly, not an asbestos-containing material in the post-2020 era.

Statement 141: "There is some uncertainty associated with the assumption that an individual would be associated with using an UTV for the entire average adult lifetime of 78 years beginning at 16 years of age. It is possible certain individuals may be involved with UTV work prior to 16 years of age. While older individuals may not be associated with their personal UTV and related gasket work up to age 78, they may provide assistance on gasket work or perhaps change from a consumer "user" to a consumer "bystander"." (page 122, ln. 4468 - 4472)

Comment 141: I think that this is an agonizing level of detail that is not warranted in this type of analysis because I believe the exposure scenario is unlikely.

2.3.2.3 Summary of Inhalation Data Supporting the Consumer Exposure Assessment

Statement 142: "Table 2-32 contains a summary of the consumer inhalation exposure data used to calculate the risk estimates in Section 4.2.3." (page 122, ln. 4491 - 4492)

Table 2-32. Summary of Consumer Inhalation Exposures

Condition of Use	Duration	Type	Exposure Concentrations, fibers/cc		Confidence Rating
			Central Tendency	High-end	
Brakes Repair/Replacement (Indoors)	3 hours once every 3 years	DIY Consumer	0.0445	0.4368	Medium
		Bystander	0.0130	0.0296	Medium
Brakes Repair/Replacement (Outdoors)	3 hours once every 3 years	DIY Consumer	0.007	0.0376	Medium
		Bystander	0.0007	0.0038	Medium-Low
UTV gasket Repair/replacement	3 hours once every 3 years	DIY Consumer	0.024	0.066	Medium
		Bystander	0.012	0.030	Medium

Comment 142: I believe that this is an agonizing level of detail that is not warranted given the low probability that this exposure scenario is present today or in the future.

2.3.3 Potentially Exposed or Susceptible Subpopulations

Statement 143: "TSCA requires that a risk evaluation "determine whether a chemical substance presents an unreasonable risk of injury to health or the environment, without consideration of cost or other non-risk [f]actors, including an unreasonable risk to a potentially exposed or susceptible subpopulation identified as relevant to the risk evaluation by the Administrator, under the conditions of use." TSCA § 3(12) states that "the term 'potentially exposed or susceptible subpopulation' means a group of individuals within the general population identified by the Administrator who, due to either greater susceptibility or greater exposure, may be at greater risk than the general population of adverse health effects from exposure to a chemical substance or mixture, such as infants, children, pregnant women, workers, or the elderly."" (page 123, ln. 4498 - 4505)

Comment 143: I believe that the Agency is incorrect on their use of the term "susceptible subpopulations." It was originally intended to address persons in the general public who may or may not be more susceptible than the typical working person. That is, "such as infants, children, pregnant women, workers, or the elderly."

I do not believe that this type of analysis warrants a susceptible subpopulation discussion. If for no other reason than that, the degree of exposure is remarkedly small, and there is a low probability that infants, children, pregnant women, and the elderly probably will be involved with replacing brakes or gaskets on 40-year-old vehicles in the coming years. As noted, if any part might contain asbestos, it will be the disc brake (for which there will be no exposure).

Statement 144: "Of the human receptors identified in the previous sections, EPA identifies the following as potentially exposed or susceptible subpopulations due to their greater exposure to asbestos and considered them in the risk evaluation:

Consumers and bystanders associated with consumer (DIY) use. Asbestos has been identified as being used in products (aftermarket automotive brakes and linings and other gaskets in UTVs) available to consumers; however, only some individuals within the general population may use these products (i.e., DIYers or DIY mechanics). Therefore, those who do use these products are a potentially exposed or susceptible subpopulation due to greater exposure." (page 123, ln. 4515 – 4521; 4533 - 4537)

Comment 144: The sensitive subpopulations discussion was never intended to be applied to a situation where only a handful of adults across the nation might be exposed to the scenarios presented in this 310-page document.

The worries about sensitive subpopulations have historically focused on exposure scenarios where millions of citizens are exposed to appreciable levels of a toxicant (e.g., during days when there is an inversion, and high levels of particulate matter put children, joggers, asthmatics, those with COPD, and the elderly at increased risk).

Statement 145: "CPS is a monthly survey of households conducted by the Bureau of Census for the Bureau of Labor Statistics and provides a comprehensive body of data on the labor force characteristics. Statistics for the following subpopulations of workers and ONUs are provided: adolescents, adult men and women. As shown in Table 2-33, men make up the majority of the workforce in the asbestos COUs.

In other sectors, women (including those of reproductive age and elderly women) make up a larger portion of wholesale and retail trade." (page 124, ln. 4545 - 4548)

Table 2-33. Percentage of Employed Persons by Age, Sex, and Industry Sector (2017 and 2018 worker demographics from BLS)

Age Group	Sex	Mining, quarrying, and oil and gas extraction	Manufacturing	Wholesale and retail trade
		Oilfield Brake Block	Chlor-Alkali; Gasket stamping; Gasket use in chemical plants	Auto brake; UTV
Adolescent (16-19 years)	Male	0.4%	0.8%	3.0%
	Female	0.0%	0.4%	3.2%
Adults (20-54 years)	Male	68.2%	52.9%	42.8%
	Female	9.2%	22.2%	35.4%
Elderly (55+)	Male	19.4%	17.5%	12.3%
	Female	3.3%	7.3%	9.6%

Comment 145: As noted in a previous comment, I do not believe that there is any exposure to asbestos from brakes for the workers in oil fields that will be measurable for the conditions that are described in this EPA document. Additionally, there is something wrong with this table, as each subcategory should equal 100%.

3.2 Human Health Hazards

Statement 146: "Many authorities have established that there are causal associations between asbestos exposures and lung cancer and mesotheliomas (NTP, 2016; IARC, 2012; ATSDR, 2001a; U.S. EPA, 1988b; IARC, 1987; U.S. EPA, 1986; IARC, 1977). Although asbestos is also associated with other types of cancers, there are no Inhalation Unit Risk (IUR) values available for these other cancers." (page 128, ln. 4698 - 4700)

Comment 146: It is essential to recognize that like everything else in medicine and toxicology, that the adverse effects are dependent upon dose. This is particularly true with short fiber chrysotile. None of these diseases are caused by low-level exposure to chrysotile, especially to fibers less than 10 microns in length, and these comprise the majority in fibers encapsulated in brakes, gaskets and packing.

Statement 147: "Thus, this draft risk evaluation uses the EPA-derived chrysotile IUR described in Section 3.2.4 to calculate risk estimates." (page 128, ln. 4706 - 4707)

Comment 147: The Agency's derived IUR, in my view, is scientifically unfounded. It is based upon many faulty assumptions that have been generally known to be untrue for many, many years. I will address a handful of them below.

1) The Agency assumes that mesothelioma does not exist in the background population. This is a scientifically unfounded assumption. Mesothelioma is like any other cancer in that the incident rate increases with age. I have found no one who can refute the papers by Price and Ware (2009) on this topic or those by Moolgavkar et al. (2009).

2) The assumption that mesothelioma does not appear in the background is flawed on many levels. I would refer the Agency to the famous papers by Tomasetti and Vogelstein (2015), Tomasetti et al. (2017), and Moolgavkar et al. (2010).

3) When the Agency corrects for the actual background incidence of both pleural and peritoneal mesothelioma, this will affect their derived cancer potency factor. It is noteworthy that peritoneal mesothelioma has never been associated with chrysotile only cohorts, and few persons, if any, over the last 40 years have had exposures to amphiboles that would have increased their risk or caused peritoneal mesothelioma. It is unclear to me where the Agency was adding plural and/or peritoneal mesothelioma in their calculated cancer potency factor.

4) Case reports have suggested an association between radiation therapy and the subsequent development of malignant mesothelioma (Antman et al. 1983; Cavazza et al. 1996; Hofmann et al. 1994). Specifically, Antman et al. (1983) published a case series that described four patients who developed mesothelioma after radiation therapy, none of which reported any known exposure to asbestos; their mesotheliomas were diagnosed between 10 and 31 years following their radiation treatments (Antman et al. 1983). Further, the authors noted that five additional cases of mesothelioma were reported in the literature before the publication of their study and stated that the “histology of the radiation-associated mesotheliomas could not be differentiated from those associated with asbestos contact” (Antman et al. 1983, p. 698). Cavazza et al. (1996) discussed an additional eight patients who developed malignant mesothelioma in sites of prior radiation therapy for a different primary tumor. Of these patients, four had no known history of prior asbestos exposure, while the exposure history of the four others was unknown. In a more recent study, Teta et al. (2007) examined the occurrence of mesothelioma in patients who previously suffered from Hodgkin (HL) and non-Hodgkin (NHL) lymphoma. The results indicated a statistically significant increased risk of mesothelioma for HL males who underwent radiation treatment (SIR: 6.59; 95% CI: 1.79, 16.87). There was also an increased risk of mesothelioma for both NHL men and women who underwent radiation treatment (SIR: 2.24; 95% CI: 1.07, 4.12) (Teta et al. 2007). Lastly, Goodman et al. (2009) performed a literature review and concluded that the case-report and epidemiology literature indicated that “ionizing radiation can play a causal role in mesothelioma” (p. 1245).

5) The Agency seems to have embraced a belief that the Carolina Textile Mill cohort studies cited throughout this document only had exposures to pure chrysotile fibers. That is not correct. Dement acknowledges in his paper that there were potential exposures to amphiboles in the North Carolina facility (Dement et al., 2009; Loomis et al., 2009). In a deposition taken by Dr. Dement, he again reiterated that there was possible exposure to

amphiboles there, and he acknowledges that Dr. Roggli had found amphibole fibers in one or two workers in that facility (Roggli et al., 1998; Pavlisko et al., 2020). Thus, that cohort cannot be used to support this analysis. This entire EPA document is inappropriate and scientifically flawed because of this.

6) This entire document is meant to evaluate the health hazards posed by fibers from encapsulated products. None of the fibers at the textile mills were encapsulated. Encapsulation is necessary to highlight because the fibers are filled with polymeric or phenolic chemicals. As shown by Bernstein et al. (2018, 2003), these fibers do not appear to have any biologic activity, which is remotely similar to raw asbestos.

7) In my view, using the textile worker data to predict the hazard of short fiber chrysotile is without foundation. It is well known from the Berman and Crump studies that chrysotile probably fails to have the capacity to cause mesothelioma except at asbestosis producing air concentrations and only when the fibers are in the vicinity of 20 – 40 microns in length (Berman and Crump, 2003, 2008b). A large proportion of the fibers at these textile mills were in the 40-micron range. Comparatively, brake wear debris from chrysotile fibers are 99% shorter than 5 microns (Paustenbach et al., 2004). The most appropriate cohort for this EPA document and for the exposure scenarios for which it focuses is the 12 epidemiology studies that address exposures of mechanics to brake dust and brake wear debris (See previous comments). Surprisingly, none of them, nor the four meta-analyses of these studies were cited in this document.

8) I reviewed the "Systematic Review Supplemental File," which supports the primary EPA document [EPA document # EPA-740-R1-8012]. None of the 12 relevant studies of mechanics are cited or discussed. None of the four meta-analyses are cited or discussed. None of the toxicology papers which discuss why there is a threshold dose or a threshold for asbestosis, lung cancer, and mesothelioma concerning short-fiber chrysotile are discussed. Additionally, none of the EPA sponsored reviews on this topic are mentioned in this supplemental document.

9) It is difficult how a thorough, systematic review would miss those papers when a large fraction of them are mentioned in my 2004 article, which is frequently cited by the Agency in this document. I would direct you to figure nine in that 2004 paper. That figure was built nearly 20 years ago, and since then, twice as many articles on the subject have been published.

Statement 148: "In the PF document, it was stated that the asbestos RE would focus on epidemiological inhalation data on lung cancer and mesothelioma for all TSCA Title II fiber types, just as stated in the 1988 EPA IRIS Assessment on Asbestos (U.S. EPA, 1988b)." (page 129, ln. 4718 - 4720)

Comment 148: It is not clear to me why the agency used this approach. This rationale needs to be explained in more detail.

Statement 149: "EPA identified key and supporting studies from previous peer reviewed assessments and new studies published since 1988 and evaluated them against the data quality criteria developed for asbestos. The evaluation criteria were tailored to meet the specific needs of asbestos studies and to determine the studies' potential to provide information on the exposure-response relationship between asbestos exposure and mortality from lung cancer and from mesothelioma." (page 130, ln. 4786 - 4789)

Comment 149: Although on the surface, it appears like the Agency did an exemplary job examining the literature, for some reason, many of the relevant chrysotile asbestos toxicology (related to encapsulated asbestos) and epidemiology papers were omitted for consideration in this document. I think this was because the Agency had a belief that they had to only look at chrysotile "only" exposed cohorts, which they believed (incorrectly) were found in the various studies of the Carolina textile mill workers.

This needs to be rectified in the next iteration of this document.

3.2.3.1 Mode of Action (MOA) considerations for asbestos

Statement 150: "As stated in IRIS Assessment on Libby Amphibole Asbestos (2014c) for asbestos in general, International Agency for Research on Cancer (IARC) has proposed a mechanism for the carcinogenicity of asbestos fibers [see Figure 4-2 in (IARC, 2012)]. Asbestos fibers lead to oxidant production through interactions with macrophages and through hydroxyl radical generation from surface iron. Inhaled fibers that are phagocytosed by macrophages may be cleared or lead to frustrated phagocytosis, which results in macrophage activation, release of oxidants, and increased inflammatory response, in part due to inflammasome activation." (page 131, 4830 - 4836)

Comment 150: This is a legitimate proposed mechanism, but for completeness, other mechanisms that have been discussed in the literature should be included. This is particularly important for chrysotile since it generally lacks genotoxicity (Barlow et al., 2013, 2017). As a result, one sees a heavily non-linear response in the low dose region in most predominately chrysotile exposed cohorts. Much more could be said about this topic, but it goes beyond the scope of these comments.

I will note that a chapter in Lynn (1983) text does address some of the likely mechanisms of action for the carcinogenicity of chrysotile.

Statement 151: "Mineral fibers may also lead to direct genotoxicity by interfering with the mitotic spindle and leading to chromosomal aberrations." (page 132, ln. 4839 - 4840)

Comment 151: This is one of several possible mechanisms. For completeness, the Agency should discuss the numerous other alternative hypotheses (Lynn, 1983; Englert et al., 2014).

The paragraphs below are from Englert et al. (2014):

"The studies reported by Wagner et al.[5] and by Davis et al.[43] both showed a close association between the severity of interstitial fibrosis (i.e. asbestosis) and the development of pulmonary neoplasms. This finding suggests that pulmonary parenchymal tumors in asbestos-exposed animals derive from a metaplastic and hyperplastic epithelial response in areas of interstitial fibrosis that in some instances progressed to neoplasia. Davis and Cowie[239] have addressed this question in greater detail. These

authors note that when adenomas or very early carcinomas are found, they are frequently in the center of areas of advanced asbestosis with exuberant epithelial metaplasia/hyperplasia. In studies comparing the pathological effects of various mineral fibers, there has also been a close association between the severity of pulmonary fibrosis and tumor development.[101, 240, 241]

In an analysis of data from several different studies,[43, 68] a strong correlation was observed between the percentage of lung occupied by fibrosis and the occurrence of pulmonary tumors ($p < 0.001$).[239] Tumors which developed in association with low-recorded levels of fibrosis (involving less than 4% of the lung area), were either advanced tumors occupying a single lung lobe or early tumors originating from the center of areas of interstitial fibrosis (Figure 10-10). While these studies support a role for fibrosis in the development of asbestos-associated tumors, they do not definitively answer the question as to whether fibrosis is an absolute prerequisite for the development of pulmonary tumors in experimental animals, which would require examination of a relatively large population of rats during the period of early tumor development.[239] Furthermore, the results may not be relevant to the great majority of lung cancers occurring in asbestos workers, in which cigarette smoke is an important cofactor.[242, 243]

Role of Fiber Dimensions

Inhalation studies have indicated that in an analogous fashion to fibrogenic potential, long fibers have the greatest carcinogenic potential in experimental animal models.[5] Davis et al.[68], using an amosite preparation with extremely few fibers greater than 5 µm in length, reported no tumors in rats following long-term inhalation, whereas a clear excess of lung carcinomas and pleural mesotheliomas developed in rats breathing an amosite cloud containing considerable numbers of fibers 5 µm or greater in length. Similar but less clear-cut results were obtained in a study of long and short preparations of chrysotile asbestos.[68] In this latter study, some longer fibers were still present in the ”short fiber” chrysotile preparation, although the ‘long fiber’ preparation (on an equal-mass basis) had five times as many fibers 5 µm or greater in length and 80 times as many fibers 30 µm or greater in length. Both long and short chrysotile preparations produced mesotheliomas in more than 90% of rats following intraperitoneal injection of 25 mg. However, at a dose level of 2.5 mg, the short-fiber preparation produced only one-third as many mesotheliomas as the long fiber preparation, which still produced mesotheliomas in more than 90% of the animals injected. At a dose of 0.25 mg, the long-fiber preparation still produced tumors in 66% of rats.[68] The dose of short-fiber chrysotile that resulted in no mesothelial tumors in 24 rats (injected intraperitoneally) was calculated to contain 57 million fibers greater than 8 µm in length.[68]

Further complicating the carcinogenic potential of chrysotile are the relatively short half-lifes of the longer, disease causing fibers. Bernstein et al. conducted a 5-day inhalation study and found the half-life of chrysotile fibers >20 µm to be 16 days and the half-life of fibers 5-20 µm to be 29.4 days.[252] This was in contrast to amosite which had a half-life greater than 1000 days for all lengths of fibers in rat lungs.[253] Studies using mineral fibers other than asbestos have also shown a strong association between fiber length and carcinogenicity.[68, 241, 254-256] Given all of these findings as well as other epidemiologic studies, the Agency for Toxic Substances and Disease Registry published an expert report in 2003 concluding that asbestos fibers less than 5 □m were “unlikely to cause cancers in humans.”[257]

The Effect of Smoke Exposure on Asbestos Injury

In the previous discussion, mechanisms by which asbestos fibers might interact directly with DNA and chromosomes, and thus as an initiator of carcinogenesis, were emphasized. However, there is considerable epidemiologic data indicating that, with respect to carcinoma of the lung, asbestos interacts in a multiplicative fashion with cigarette smoke to enhance greatly the rate of neoplastic transformation. In this sense, asbestos behaves as a classic promoter of carcinogenesis. Numerous

studies have explored various mechanisms by which asbestos could interact with cigarette smoke components in the process of carcinogenesis.[318]

One mechanism of interaction might be the adsorption of polycyclic aromatic hydrocarbons or other carcinogenic compounds within cigarette smoke onto the surface of the asbestos fiber, which then could act as a carrier particle, providing prolonged and intimate contact of the adsorbed carcinogens with respiratory epithelial cells."

Statement 152: "However, EPA's research to identify COUs indicated that only chrysotile asbestos is currently being imported in the raw form or imported in products." (page 132, ln. 4864 - 4865)

Comment 152: To my knowledge, the only raw asbestos imported into the United States today is that which is used by the Chlor-Alkali industry (as noted by the Agency).

Statement 153: "The epidemiologic studies available for risk assessment all include populations exposed to commercial chrysotile asbestos, which may contain small, but variable amounts of amphibole asbestos." (page 132, ln. 4869 - 4870)

Comment 153: As the Agency is aware, due to the high potency of the amphiboles, even their presence in low concentrations in raw fibers generally dictates the mesothelioma hazard. Trace contamination of chrysotile by tremolite typically does not pose a significant risk; however, depending upon the percent of contamination, it can substantially influence the risk of developing an asbestos-related disease.

3.2.4.2 Rationale for Asbestos-Specific Data Evaluation Criteria

Statement 154: "In the PF step of the asbestos assessment, it was accepted that exposure to asbestos was a known cause of lung cancer and mesothelioma, and that the purpose of the systematic review would be the identification of studies which Inhalation could inform the estimation of an exposure-response function allowing for the derivation of an asbestos 4880 inhalation unit risk for lung cancer and mesothelioma combined." (page 132 – 133, ln. 4877 - 4881)

Comment 154: As noted in previous comments, it is not appropriate to attempt to calculate a single cancer potency factor for both mesothelioma and lung cancer for chrysotile. As stated before, no lung cancer study can be considered credible unless you have a detailed understanding of the smoking history of each person in the exposed cohort. This is particularly true for predominately male cohorts who lived in the United States between 1940 and 2000.

Interaction between cumulative asbestos exposure and smoking and their combined effect on lung cancer risk has been studied since the 1960s. Many of these studies have evaluated the interaction effect between cigarette smoking and high-level cumulative asbestos exposures, such as those experienced by miners, millers, textile workers, and insulators (Saracci, 1977; Selikoff and Hammond, 1968; Hammond et al., 1979; Selikoff et al., 1980; Berry et al., 1985; Erren et al., 1999; Liddell and Armstrong, 2002; H. Wang et al., 2012; Yano et al., 2010; X. R. Wang et al., 2012; X. Wang et al., 2012; Wang et al., 2013).

Markowitz et al. (2013) conducted a follow-up study on a subset of the cohort of insulation workers previously studied by Selikoff et al. (1979) and Hammond et al. (1979). The authors compared lung cancer mortality rates among those that underwent clinical examination between 1981 and 1983 (n = 2,377) to lung cancer mortality rates from "blue collar" workers studied in a separate (but contemporaneous) cohort with no reported asbestos exposure. According to Markowitz et al. (2013), asbestos exposure (in the absence of asbestosis) resulted in a rate ratio of 3.6 (95% CI: 1.7-7.6); smoking in the absence of asbestos exposure resulted in a lung cancer rate ratio of 10.3 (95% CI: 8.8-12.2). However, most importantly, in the absence of asbestosis, asbestos exposure in combination with smoking resulted in a lung cancer rate ratio of 14.4 (95% CI: 10.7-19.4). The authors concluded that this supports that in the absence of asbestosis, the joint effect of asbestos exposure and cigarette smoking is additive, which is contrary to earlier reports of a multiplicative interaction.

Villeneuve et al. (2012) conducted a population-based case-control study of men in Canada between 1994 and 1997, in which the authors assessed the combined effect of occupational asbestos exposure and cigarette smoking on the risk of lung cancer. The authors stratified occupational asbestos exposure according to exposure magnitude: low exposure (which included welders and boiler operators, mechanics, non-electrician repairmen, pipefitters, and construction workers), medium exposure (which included boiler and pipe insulators, firefighters, and plasters), and high exposure (which included miners and insulation blowers/sprayers). The authors also stratified smoking history by cumulative intensity: <10 pack-years, 10 to <40 pack-years, and ≥40 pack-years. The authors reported that, for each of the three smoking history categories, there was approximately a two-fold increase lung cancer risk among the medium or high asbestos exposure groups compared to those with no exposure history, which the authors concluded was "consistent with a multiplicative relationship" (Villeneuve et al., 2012). However, the risk of lung cancer among those with low asbestos exposure was no different for those with no asbestos exposure for each of the cumulative smoking categories. This supports that at low levels of asbestos exposure, no synergy exists between asbestos and cigarette smoking.

Additionally, Gustavsson (2002) performed a case-control investigation of lung cancer risk associated with occupational asbestos exposure among male residents of Stockholm, Sweden, aged 40 to 75 years. To assess the combined effect of smoking and asbestos exposure on lung cancer risk, the authors grouped cases by both cumulative asbestos exposure and level of tobacco use. Cumulative asbestos exposures ranged from 0 to 20.4 f/cc-years; however, only 18 (6%) of the participants estimated to have been exposed to asbestos had exposures that exceeded 4.5 f/cc-years. The authors characterized the interaction between asbestos exposure and smoking as "submutiplicative but slightly more than additive"; however, for those with cumulative exposures ≤ 2.49 f/cc-years, which is similar to the upper-bound cumulative exposure for career automobile mechanics [as reported by Finley et al. (2007)], the joint effect of asbestos and cigarette smoke exposures on lung cancer risk was not statistically different from those with the same smoking history and no history of exposure to asbestos (Gustavsson, 2002). This supports that at low levels of asbestos exposure, no synergy exists between asbestos and cigarette smoking.

In addition, Hrubek et al. (1992) conducted a mortality study to assess the relative risk for various causes of death among 300,000 veterans who served in the U.S. Armed Forces between 1917 and 1940, including those who served as auto mechanics. The authors stratified smoking history into three categories: nonsmokers, current smokers, and combined smoking status. The authors reported that they did not observe an increased risk of lung cancer in auto mechanics and repairmen who smoked relative to smokers in the referent population (RR = 1.2; 95% CI: 0.93-1.49) (Hrubek et al., 1992). These data

support that low-level chrysotile exposure associated with work as a vehicle mechanic did not increase the risk of developing lung cancer in excess of smoking alone (i.e., no synergy exists).
In summary, though a synergistic relationship for lung cancer risk between asbestos exposure and smoking has been demonstrated in some studies of cohorts exposed to high cumulative asbestos exposure levels, there is no evidence that this relationship exists at low cumulative asbestos exposure levels, such as those experienced by a career automobile mechanics.

Statement 155: "In terms of evaluating the quality of outcome information, lung cancer is relatively straightforward to evaluate as an outcome. Specific International Classification of Disease (ICD) codes for lung cancer have existed for the entire time period of the studies evaluated here making it possible to identify cases from mortality databases." (page 133, ln. 4909 - 4912)

Comment 155: I disagree with the Agency's statement regarding "lung cancer is relatively straightforward to evaluate as an outcome." The only straightforward aspect is that there is a disease classification that has been around for 40 – 60 years. Concerning the claim that lung cancer is due to chrysotile is, considered by most independent physicians, a challenging claim to make. One has to consider the person's smoking history (pack-years), the age they began smoking, and the age in which they stopped smoking, as well as numerous other factors. This has to be coupled with a sound knowledge of the typical fiber length of chrysotile to which the worker was exposed, as well as their cumulative dose (fibers/cc/year). And of course, it is essential to have a thorough knowledge of the presence of any amphiboles present in the chrysotile.

Statement 156: "EPA identified studies of five independent occupational cohorts exposed only to commercial chrysotile that provided adequate data for assessment of lung cancer risks: asbestos textile manufacturing workers in North Carolina and South Carolina, USA (Loomis et al., 2009; Hein et al., 2007) and Chongqing, China (Deng et al., 2012) and chrysotile miners in Québec, Canada (Liddell et al., 1997), and Qinghai, China (2014; Wang et al., 2013b). A pooled analysis of the two U.S. studies (NC and SC) asbestos textile cohorts (Elliott et al., 2012) also provides informative data. In addition, Berman and Crump (2008) provide informative risk estimates for the Québec miner cohort based on modeling dose-response data that were not available in the original study." (page 135, ln. 4981 - 4988)

Table 3-2. Study Cohort, Individual studies and Study Quality of Commercial Chrysotile Asbestos Reviewed for Assessment of Lung Cancer and Mesothelioma Risks

Study Cohort	Author, Year	HERO ID	Study Quality**
South Carolina, US	(Berman and Crump, 2008)	626405	Lung Cancer 1.6 High Mesothelioma 1.7 Medium
	(Brown et al., 1994)	3081832	
	(Cole et al., 2013)	3078261	
	(Dement et al., 1983b)	67	
	(Dement et al., 1994)	3081766	
	(Dement and Brown, 1994)	3081783	
	(Edwards et al., 2014)	3078061	
	(Elliott et al., 2012)	1247861	
	(Hein et al., 2007)	709498	
	(Loomis et al., 2012)	1257856	
	(SRC, 2019c)	5080236	
	(Stayner et al., 1997)	3081241	
	(Stayner et al., 2008)	2604140	
Qinghai, China - miners	(Wang et al., 2012)	2572504	Lung Cancer 1.6 High
	(Wang et al., 2013b)	2548289	
	(Wang et al., 2014)	2538846	
Balangero, Italy*	(Piolatto et al., 1990)	3082492	
	(Pira et al., 2009)	2592425	
	(Pira et al., 2017)	5060134	
	(Rubino et al., 1979)	178	

Comment 156: As discussed in a previous comment, it is not appropriate to combine the lung cancer and mesothelioma risks (as presented in this table), when attempting to describe the possible cancer risk due to exposure to chrysotile.

Statement 157: "A re-parametrization with α = exp (Beta0) is called the linear relative rate model. For epidemiologic studies where, individual data analysis was conducted, other models have been used for modeling lung cancer. These include both linear relative rate model (e.g., (Hein et al., 2007)), the Cox proportional hazard model (e.g., (U.S. EPA, 2014c; Wang et al., 2014) and other log-linear relative rate models (e.g., (Elliott et al., 2012; Loomis et al., 2009). Results from all these model types were considered to be informative in estimating the lung cancer potency factor (KL) and were carried forward for further consideration." (page 137, ln. 5039 - 5044)

Comment 157: No amount of modeling of epidemiology data can make up for the serious differences in what is being inhaled by these workers in these different cohorts compared to auto mechanics or millwrights 50 years ago. One has to consider the differences in fiber length, aspect ratio, purity of the chrysotile, and other factors before grouping them and considering them equivalent.

3.2.4.5 Study Descriptions and Model Fitting Results

Statement 158: "The asbestos exposure data and exposure assessment methods in studies of the Charleston, South Carolina textile plant (Elliott et al., 2012; Hein et al., 2007) are exceptionally detailed compared to most asbestos studies. The methods used were innovative at the time, a large number of exposure measurements cover the relevant study period, and detailed process and work history information were available and utilized in estimating exposures. The exposure data used in studies of North Carolina plants (Loomis et al., 2019; Elliott et al., 2012) are also high quality. The methods were similar to those developed for the studies of the South Carolina plant. However, relative to the South Carolina study, the number of exposure measurements is smaller, and the historical process and work-history data are less detailed. Nevertheless, the exposure data are of higher quality than those utilized in other studies of occupational cohorts exposed to chrysotile. For both U.S. textile cohorts, the exposure assessment methods and results have been published in full detail." (page 140 – 141, ln. 5192 - 5202)

Comment 158: I would agree that the exposure assessment is reasonably thorough in the North and South Carolina textile mill studies. I believe that the dataset can be studied in more detail and that it would reveal other important insights. I would not quibble with them over most of the exposure assumptions in this statement as the shortcoming of studying these cohorts and attempting to describe the risks of low-level to short fiber chrysotile has been discussed in previous comments.

North Carolina asbestos textile plants [carried forward for unit risk derivation]

Statement 159: "Loomis et al. (2019; 2009) reported on mortality in a cohort of workers in four North Carolina asbestos textile mills that had not been studied previously. Three of the plants were operationally similar to the South Carolina plant, but did not have equivalent exposure controls. They produced yarns and woven goods from raw chrysotile fibers, mostly imported from Canada. A fourth, smaller plant produced several asbestos products using only purchased yarns. The latter plant lacked adequate exposure data and was included in comparisons of cohort mortality to the general population, but not in exposure-response analyses for lung cancer or mesothelioma." (page 143, ln. 5300 - 5306)

Comment 159: Any comments that I might have would go beyond the scope of this document. All I say is that the Carolina cohorts are entirely inappropriate for predicting the risks of exposure to short fiber chrysotile, which have absorbed phenolic resins (which is what is seen in gaskets, brakes, and other products). See comments (submitted on 5-20-2020) by Dr. David Garabrant to the Agency on this document for a more in-depth discussion of these cohorts.

Statement 160: Table 3-8 (page 149, ln. 5556 - 5558)

Table 3-8. Comparison of Lifetime Units Risks of Lung Cancer by Industry

Industry	Lifetime unit risks of lung cancer	
	MLE	95% UB
Textiles	7.60E-3 – 1.66E-1	1.17E-2 – 2.50E-1
Mining	2.05E-3 – 1.53E-1	2.90E-3 – 4.57E-1

Textiles cohorts (Loomis et al., 2009; Hein et al., 2007); Mining cohorts (Quebec, Canada; Qinghai, China). The cohort from Chongqing, China was not included here, but those values are intermediate and would not change the ranges provided here.

Comment 160: Any comments that I might have would go beyond the scope of this document. See comment 159.

3..4.6.1 Combining Lung Cancer Unit Risk and Mesothelioma Unit Risk

Statement 161: "Once the cancer-specific lifetime unit risks are obtained, the two are then combined. It is important to note that this estimate of overall potency describes the risk of mortality from cancer at either of the considered sites and is not just the risk of an individual developing both cancers concurrently. Because each of the unit risks is itself an upper bound estimate, summing such upper bound estimates across mesothelioma and lung cancer mortality is likely to overpredict the upper bound on combined risk. Therefore, following the recommendations of the Guidelines for Carcinogen Risk Assessment (U.S. EPA, 2005), a statistically appropriate upper bound on combined risk was derived as described below." (page 151, ln. 5598 - 5604)

Comment 161: As noted in previous comments, I would urge the Agency to be cautious about applying any variation of the linearized multistage model to short fiber chrysotile fibers. I believe that if you use the most recent EPA guidelines for carcinogens, there is strong evidence that short fiber chrysotile is a threshold carcinogen. It seems to me, and to many other who have published on this topic that due to the lack of genotoxicity (Barlow et al., 2013) and the minimal probability for it to produce lung cancer (Berman and Crump, 2003; Pierce et al., 2008; Garabrant et al., 2016; Pierce et al., 2016), most low dose extrapolation models that insist on linearity at very low doses are not appropriate. I was delighted to see that Dr. Crump is on the ad hoc panel, and he can speak on this matter with authority.

3.2.4.7.1 Selecting the Preferred Model Forms for Lung Cancer

Statement 162: "Between the linear relative rate and exponential model forms for lung cancer mortality in both SC and NC cohorts, the exponential models clearly fit better (Elliott et al., 2012). Table 2 of that publication shows that the standard model fit metric, called the Akaike Information Criterion (AIC; smaller values indicate better fit), for the SC exponential model was 2656.96 and for the SC linear model was 3039.5." (page 153, ln. 5641 - 5644)

Comment 162: Any additional comments that I might have would go beyond the scope of this document.

Statement 163: "Limiting the results in Table 3-6 to lung cancer results based on the better fitting exponential models yielded four combinations that were essentially equivalent in terms of statistical fit and study quality (Table 3-7)." (page 154, ln. 5661 - 5663)

Comment 163: From the mentioned text, it is not clear why these models are "essentially equivalent" This needs to be better explained to be more apparent to the reader.

Statement 164: "TSCA § 3(12) states that "the term 'potentially exposed or susceptible subpopulation' means a group of individuals within the general population identified by the Administrator who, due to either greater susceptibility or greater exposure, may be at greater risk than the general population of adverse health effects from exposure to a chemical substance or "mixture, such as infants, children, pregnant women, workers, or the elderly."" (page 155 – 156, ln. 5717 - 5721)

Comment 164: I have discussed the problems with the Agency's use of sensitive subpopulation in previous comments. To be specific, I am not aware of any biological reasons why an individual would be more sensitive than others to chrysotile at the extremely low doses, which have been discussed in this document (which are related to exposure to gaskets and brakes that occurred nearly 40 years ago).

Statement 165: "Factors affecting susceptibility examined in the available studies on asbestos include life stage, gender, genetic polymorphisms and lifestyle factors. There is some evidence of genetic predisposition for mesothelioma related to having a germline mutation in BAP1 (Testa et al., 2011). Cigarette smoking in an important risk factor for lung cancer in the general population. In addition, lifestage is important relative to when the first exposure occurs. The long-term retention of asbestos fibers in the lung and the long latency period for the onset of asbestos-related respiratory diseases suggest that individuals exposed earlier in life may be at greater risk to the eventual development of respiratory problems than those exposed later in life (ATSDR, 2001a)." (page 156, ln. 5728 - 5735)

Comment 165: In an absolute sense, this might be true. I believe there is growing evidence that BAP1 may not be as important as once thought.

However, practically speaking, the Agency's concern for the exposure to young persons is unjustified because the occupational exposure for the exposure scenarios described here will not exist in the coming years (you have to go back 40 years for all of these exposures scenarios to be relevant).

Additionally, the four sentences shown above describe many complex biological phenomena in such a simplistic manner, that I would urge the Agency to remove the paragraph from the document or expand each of these topics in individual sections that are fully explained.

Statement 166: Table 4-3 (page 161 – 162, ln. 5906 - 5907)

Table 4-3. Reported Respirator Use by COU for Asbestos Occupational Exposures

Condition of Use	Monitoring Data?	Respirator Use Text	APF for Risk Calculation
Chlor-alkali	Yes, provided by industry (EPA-HQ-OPPT-2016-0736-0052, Enclosure C)	Workers engaged in the most hazardous activities (e.g., those with the highest likelihood of encountering airborne asbestos fibers) use respiratory protection. Examples include workers who: handle bags of asbestos; clean up spilled material; operate glove boxes; and perform hydroblasting of spent diaphragms. The types of respirator used range from half-face air-purifying respirators to supplied air respirator hoods, depending on the nature of the work.	Half-face air-purifying APF of 10 Supplied air respirator hoods APF of 25 *for specific tasks*[3] **APF to use for the risk calculation: 10 to 25**
Sheet gasket stamping	Yes, provided by industry	Workers wear N95 filtering facepiece masks. A site-specific industrial hygiene evaluation determined that asbestos exposures were not high enough to require employee respirator use. (Note: the EPA risk estimates indicate that these workers should be wearing appropriate respirators, which is not an N95 mask. See footnote 1).	Half mask with N95[1] **Hypothetical APF to use for the risk calculation: 10 to 25**
Sheet gasket use (Chemical Production)	Yes, provided by industry	When replacing or servicing asbestos-containing sheet gaskets, workers in the titanium dioxide industry wear respirators, either airline respirators or cartridge respirators with P-100 HEPA filters.	Cartridge respirators with P-100 HEPA filters APF 10 Airline respirators: APF 10

Condition of Use	Monitoring Data?	Respirator Use Text	APF for Risk Calculation
			APF to use for the risk calculation: 10
Oilfield brake blocks	Yes, from the literature	No information is reasonably available on respirator use for this COU.	**Hypothetical APF to use for the risk calculation: 10 to 25**
Aftermarket automotive brakes and clutches	Yes, provided in literature	An unknown amount of respirator use occurs among these workers. OSHA's asbestos standard requires establishments to use control methods to ensure that exposures are below permissible exposure limits. OSHA has also reported: "Respiratory protection is not required during brake and clutch jobs where the control methods described below are used" (OSHA, 2006). Nonetheless, some respirator use among workers in this industry is expected.	**Hypothetical APF to use for the risk calculation: 10 to 25**
Other gasket vehicle friction product (UTV)	No[2]	No information is reasonably available on respirator use for this COU, but worker activities are expected to be similar to those for aftermarket automotive brakes and clutches.	**Hypothetical APF to use for the risk calculation: 10 to 25**

[1] OSHA Asbestos Standard 1910.1001 states that negative pressure and filtering masks should not be used for asbestos exposure. The N95 is a negative pressure mask.
[2] EPA is using worker exposure data from the sheet gasket replacement in the chemical manufacturing industry as a surrogate for the exposures that may occur when workers service UTV friction products.
Source: (OSHA, 2006). Asbestos-Automotive Brake and Clutch Repair Work: Safety and Health Information Bulletin. SHIB 07-26-06. Available online at: https://www.osha.gov/dts/shib/shib072606.html.
[3] See Table 2-7.

Comment 166: I have discussed why this table is not relevant in previous comments. I could say more, but I would have to dwell on other aspects of my previous comments.

4.2.2.2 Risk Estimation for Cancer Effects Following Chronic Inhalation Exposures for Sheet Gasket Stamping

Statement 167: "Table 4-11 presents the ELCRs for workers stamping gaskets from sheets, using exposure data from two sampling durations (8-hour full shift; 30 minute short-term). The central tendency and high-end exposure values are presented along with the ELCR for each exposure distribution in Table 4-11 and Table 4-12. The exposure levels (personal samples) for full shift workers are from Table 2-10 The high-end 8-hour TWA exposure value for workers (0.059 fibers/cc) is an estimate, and this full-shift exposure level was not actually observed. This estimate assumes the highest measured short-term exposure of the gasket stamping worker could persist for an entire day." (page 167, ln. 6069 - 6075)

Table 4-11. Excess Lifetime Cancer Risk for Sheet Gasket Stamping Full Shift Workers and ONUs (from Table 2-10, Personal Samples) before consideration of PPE and any relevant APF

Occupational Exposure Scenario	Exposure Levels (fibers/cc)				ELCR (40 yr exposure starting at age 16 years)			
	Asbestos Worker		ONU		Asbestos Worker		ONU	
	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end
Sheet gasket stamping: 8-hr TWA exposure	0.014	0.059	0.0024	0.010	**3.3 E-4**	**1.4 E-3**	5.6 E-5	**2.3 E-4**

Asbestos Workers: ELCR $_{(\text{Central Tendency})}$ = 0.014 f/cc • 0.2192 • 1.5 • 0.0707 per f/cc
Asbestos Workers: ELCR $_{(\text{High-end})}$ = 0.059 f/cc • 0.2192 • 1.5 • 0.0707 per f/cc
ONU: ELCR $_{(\text{Central Tendency})}$ = 0.0024 f/cc • 0.2192 • 1.5 • 0.0707 per f/cc
ONU: ELCR $_{(\text{High-end})}$ = 0.01 f/cc • 0.2192 • 1.5 • 0.0707 per f/cc

Comment 167: This seems like a great deal of precision and number crunching for the exposure of one or two employees who work a few hours per week on this production line in the coming years. This scenario does not seem important in that the airborne concentrations are so low (as identified in this document), the chrysotile fibers are short and encapsulated, and the exposures are only a handful of hours per year. It genuinely should not be of interest to the Agency.

Statement 168: "Table 4-12 presents the inhalation cancer risk estimates for workers stamping sheet gaskets and for ONUs exposed to asbestos, using an averaging of short-term exposures (assuming 30 minutes) and full shift exposures (7.5 hours per day of the full shift TWA exposure) based on monitoring data. The central tendency short-term exposure value for workers (0.024 fibers/cc) is the arithmetic mean of ten short-term measurements reported in a study of one worker at a company that stamps sheet gaskets containing asbestos." (page 168, ln. 6090 - 6095)

Table 4-12. Excess Lifetime Cancer Risk for Sheet Gasket Stamping Short-term Exposures within an 8-hour Full Shift (from Table 2-10, Personal Samples) before consideration of PPE and any relevant APF

Occupational Exposure Scenario	Exposure Levels (Fibers/cc)				ELCR (40 yr exposure starting at age 16 years)			
	Asbestos Worker		ONU		Asbestos Worker		ONU	
	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end
Sheet gasket stamping: **Short-term exposures** (~30- minute; and ~30-minute short term samples within a full shift)*.	30 min value: 0.024 8-hr TWA: 0.015*	0.059 0.059*	0.0042 0.0025*	0.010 0.010*	--- **3.5 E-4**	--- **1.4 E-3**	--- 5.6 E-5	--- **2.3 E-4**

*Short-term exposures are assumed to be 30 minutes in duration. For the purposes of risk estimation, short term exposures are averaged with full shift exposure by assuming 30 minutes per day of short-term exposure with an additional 7.5 hours per day of the full shift TWA exposure.
ELCR = {[(0.5 hour)*EPC$_{(30\ \text{minute})}$ + (7.5 hours)* EPC$_{(\text{Full Shift})}$] / 8 hours} • 0.2192 • 1.5 • 0.0707.
Asbestos Worker: ELCR $_{(\text{Central Tendency})}$ = {[(0.5 hour)*0.024 + (7.5 hours)* 0.014] / 8 hours} • 0.2192 • 1.5 • 0.0707.
Asbestos Worker: ELCR $_{(\text{High-end})}$ = {[(0.5 hour)*0.059 + (7.5 hours)* 0.059] / 8 hours} • 0.2192 • 1.5 • 0.0707.

Comment 168: I do not see how an occupation with high-end exposures to asbestos that are approximate ½ the OSHA PEL can be considered an unacceptable health risk (given the minimal duration of exposure and the few occasions of possible exposure per year). The only way this happens is if one applies a much more conservative cancer potency factor (CPF) to the exposures. And, as I have said, I can see no reason for promulgating this CPF based on the LMS model to epidemiological data on the wrong cohort for encapsulated short-fiber chrysotile!

One cannot help but think the Agency is trying to place pressure on NIOSH and OSHA to update the asbestos PEL (and probably encourage them to set 3-5 different factors for the various forms of asbestos). I would not be against such a move by OSHA, but using this document to force that issue, with all the associated costs and inaccuracies, does not seem to be the correct approach to achieve this goal.

As noted, several times in this document, in my 40 years as a toxicologist and industrial hygienist, I have never seen an occupational assessment where a risk of 3.5 ^ 10-4 cancer risk for two to four employees is considered an unacceptable cancer risk.

With risks this low, you would consider even 1000 persons a year exposed routinely to this substance as being an acceptable occupational risk (and this is without consideration with the personal protective equipment, which both of these employees use on a routine basis, and the dust control measures used at this facility).

Statement 169: "For full shift worker scenarios, the benchmark cancer risk estimate of 1x10-4 was exceeded for workers with high-end exposures when a hypothetical APF of 10 was applied; all other worker scenarios were below the benchmark (central tendency for hypothetical APFs of 10 and 25 and high-end exposures with an APF of 25. Since the assumption is that ONUs do not wear respirators, application of APFs do not apply and so their risk estimates do not change (i.e., the benchmark cancer risk estimate of 1x10-4 was exceeded for ONUs for high-end exposures)." (page 169, ln. 6124 - 6129)

Table 4-15. Excess Lifetime Cancer Risk for Sheet Gasket Stamping Short-term Exposures within an 8-hour Full Shift (from Table 4-12) after consideration of PPE using an APF=10 for both full-shift and short-term exposures (excluding ONUs)

Occupational Exposure Scenario	ELCR (40 yr exposure starting at age 16 years)	
	Asbestos Worker	
	Central Tendency	High-end
Sheet gasket stamping: Short-term exposures	3.5 E-5	1.4 E-4

Comment 169: Based upon Table 4-15, it seems that the Agency would conclude that the few employees working in sheet gasket stamping are not at any significant risk of harm. In light of the background risk of cancer being approximately 40%, historically, governments around the world would consider these two employees to be at *de minimis* increased risk of cancer (this is especially true since the cancer potency factor used is almost certainly for short-fiber chrysotile).

It is unclear why they failed to reach that conclusion.

4.2.2.4 Risk Estimation for Cancer Effects Following Chronic Inhalation Exposures for Oilfield Brake Blocks

Statement 170: "Qualitatively, the information available to EPA confirms that some brake blocks used in domestic oilfields contain asbestos, as demonstrated by a safety data sheet provided by a supplier. It is reasonable to assume that wear of the brake blocks over time will release some asbestos fibers to the air. However, the magnitude of these releases and resulting worker exposure levels are not known. Only 1 study on brake blocks was located and used to estimate exposures." (page 171, ln. 6196 - 6200)

Comment 170: Any comments that I might have, beyond what I have already said about this exposure scenario, would go beyond the scope of this document.

4.2.2.5 Risk Estimation for Cancer Effects Following Chronic Inhalation Exposures for Aftermarket Auto Brakes and Clutches

Statement 171: "Exposure data from aftermarket auto brakes and clutches were presented for two sampling durations (8-hour TWA and short-term) in Table 2-15. The exposure levels are based on an 8-hour TWA from Table 2-15., which are based on 7 studies found in the literature. ELCRs for short-term data from Table 2-15. are also presented." (page 173, ln. 6230 - 6233)

Table 4-23. Excess Lifetime Cancer Risk for Repairing or Replacing Aftermarket Auto Brakes and Clutches in an Occupational Setting, 8-hour TWA Exposure (from Table 2-15.) before consideration of PPE and any relevant APF

Occupational Exposure Scenario	Exposure Levels (Fibers/cc)				ELCR (40 yr exposure starting at age 16 years)			
	Asbestos Worker		ONU		Asbestos Worker		ONU	
	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end
Repairing or replacing brakes with asbestos-containing aftermarket automotive parts: 8-hour TWA exposure	0.006	0.094	0.0007	0.011	**1.4 E-4**	**2.2 E-3**	1.6 E-5	**2.6 E-4**

Asbestos Workers: ELCR $_{(\text{Central Tendency})}$ = 0.006 f/cc • 0.2192 • 1.5 • 0.0707 per f/cc
Asbestos Workers: ELCR $_{(\text{High-end})}$ = 0.094 f/cc • 0.2192 • 1.5 • 0.0707 per f/cc
ONU: ELCR $_{(\text{Central Tendency})}$ = 0.0007 f/cc • 0.2192 • 1.5 • 0.0707 per f/cc
ONU: ELCR $_{(\text{High-end})}$ = 0.011 f/cc • 0.2192 • 1.5 • 0.0707 per f/cc

Comment 171: My thoughts on this topic were addressed in previous comments. In reviewing the data presented in this table, I am ever more convinced that this exposure scenario is irrelevant under TSCA and is not of regulatory significance. I would recommend dropping it from this document.

Statement 172: (page 174, ln. 6263 - 6276)

Table 4-24. Excess Lifetime Cancer Risk for Repairing or Replacing Aftermarket Auto Brakes and Clutches in an Occupational Setting, Short-term Exposures Within an 8-hour Full Shift (from Table 2-15.) before consideration of PPE and any relevant APF

Occupational Exposure Scenario	Exposure Levels (Fibers/cc)				ELCR (40 yr exposure starting at age 16 years)			
	Asbestos Worker		ONU		Asbestos Worker		ONU	
	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end
Repairing or replacing brakes with asbestos-containing aftermarket automotive parts: short-term exposure (~30- minute; and ~30-minute short term samples within a full shift)*.	30 min value: 0.006 8-hr TWA: 0.006*	0.836 0.140*	0.0007 0.0007*	0.100 0.011*	--- **1.4 E-4**	--- **3.3 E-3**	--- 1.6 E-5	--- **2.6 E-4**

*Short-term exposures are assumed to be 30 minutes in duration. For the purposes of risk estimation, short term exposures are averaged with full shift exposure by assuming 30 minutes per day of short-term exposure with an additional 7.5 hours per day of the full shift TWA exposure.

ELCR = {[(0.5 hour)*$EPC_{(30\ minute)}$ + (7.5 hours)* $EPC_{(Full\ Shift)}$] / 8 hours}. • 0.2192 • 1.5 • 0.0707.

Asbestos Worker: ELCR $_{(Central\ Tendency)}$ = {[(0.5 hour)*$EPC_{(30\ minute)}$ + (7.5 hours)* $EPC_{(Full\ Shift)}$] / 8 hours} • 0.2192 • 1.5 • 0.0707.

Asbestos Worker: ELCR $_{(High\text{-}end)}$ = {[(0.5 hour)*$EPC_{(30\ minute)}$ + (7.5 hours)* $EPC_{(Full\ Shift)}$] / 8 hours} • 0.2192 • 1.5 • 0.0707.

Asbestos Worker: ELCR (Central Tendency) = {[(0.5 hour)*0.006 + (7.5 hours)* 0.006] / 8 hours} • 0.2192 • 1.5 • 0.0707.

Asbestos Worker: ELCR (High-end) = {[(0.5 hour)*0.836 + (7.5 hours)* 0.094] / 8 hours} • 0.2192 • 1.5 • 0.0707.

ONU: ELCR (Central Tendency) = {[(0.5 hour)*0.0007 + (7.5 hours)* 0.0007] / 8 hours} • 0.2192 • 1.5 • 0.0707.

ONU: ELCR (High-end) = {[(0.5 hour)*0.1 + (7.5 hours)* 0.011] / 8 hours} • 0.2192 • 1.5 • 0.0707.

Comment 172: My previous comments have addressed my concerns about the data presented in this table.

4.2.2.5 Risk Estimation for Cancer Effects Following Chronic Exposures for Other Vehicle Friction Products

Statement 173: "As discussed in Section 2.3.1.8, EPA is using the exposure estimates for aftermarket auto brakes and clutches for the other vehicle friction products COU. Therefore, the risk estimates will mimic those for the aftermarket auto brakes scenarios. Exposure data from aftermarket auto brakes and clutches were presented for two sampling durations (8-hour TWA and short-term) in Table 2-15. The exposure levels are based on an 8-hour TWA from Table 2-15., which are based on 7 studies found in the literature. ELCRs for short-term data from Table 2-15. are also presented." (page 176, ln. 6315 - 6320)

Comment 173: For the reasons mentioned in previous comments, I do not believe that the risks predicted here have been accurately characterized.

Statement 174: Table 4-29 (page 176, ln. 6327 - 6338)

Table 4-29. Excess Lifetime Cancer Risk for Installing Brakes and Clutches in Exported Cars in an Occupational Setting, 8-hour TWA Exposure (from Table 2-15.) before consideration of PPE and any relevant APF

Occupational Exposure Scenario	Exposure Levels (Fibers/cc)				ELCR (40 yr exposure starting at age 16 years)			
	Asbestos Worker		ONU		Asbestos Worker		ONU	
	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end
Installing brakes with asbestos-containing automotive parts: 8-hour TWA exposure	0.006	0.094	0.0007	0.011	**1.4 E-4**	**2.2 E-3**	1.6 E-5	**2.6 E-4**

Asbestos Workers: ELCR $_{(\text{Central Tendency})}$ = 0.006 f/cc • 0.2192 • 1.5 • 0.0707 per f/cc
Asbestos Workers: ELCR $_{(\text{High-end})}$ = 0.094 f/cc • 0.2192 • 1.5 • 0.0707 per f/cc
ONU: ELCR $_{(\text{Central Tendency})}$ = 0.0007 f/cc • 0.2192 • 1.5 • 0.0707 per f/cc
ONU: ELCR $_{(\text{High-end})}$ = 0.011 f/cc • 0.2192 • 1.5 • 0.0707 per f/cc

Table 4-23. presents the inhalation cancer risk estimates for workers repairing and replacing auto brakes and clutches and for ONUs exposed to asbestos. For workers, the benchmark cancer risk estimate of $1x10^{-4}$ was exceeded for central tendency and high-end. For ONUs, the cancer benchmark was exceeded for the high-end only. Estimates exceeding the benchmark are shaded in pink and bolded.

Comment 174: For the reasons mentioned in previous comments, I do not believe that the risks predicted here have been accurately characterized.

Statement 175: Table 4-30 (page 177, ln. 6343 - 6362)

Table 4-30. Excess Lifetime Cancer Risk for Installing Brakes and Clutches in Exported Cars in an Occupational Setting, Short-term Exposures Within an 8-hour Full Shift (from Table 2-15.) before consideration of PPE and any relevant APF

Occupational Exposure Scenario	Exposure Levels (Fibers/cc)				ELCR (40 yr exposure starting at age 16 years)			
	Asbestos Worker		ONU		Asbestos Worker		ONU	
	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end
Repairing or replacing brakes with asbestos-containing aftermarket automotive parts: short-term exposure (~30- minute; and ~30-minute short term samples within a full shift)*.	30 min value: 0.006 8-hr TWA: 0.006*	0.836 0.140*	0.0007 0.0007*	0.100 0.011*	--- **1.4 E-4**	--- **3.3 E-3**	--- 1.6 E-5	--- **2.6 E-4**

*Short-term exposures are assumed to be 30 minutes in duration. For the purposes of risk estimation, short term exposures are averaged with full shift exposure by assuming 30 minutes per day of short-term exposure with an additional 7.5 hours per day of the full shift TWA exposure. ELCR = {[(0.5 hour)*EPC$_{(30\ \text{minute})}$ + (7.5 hours)* EPC$_{(\text{Full Shift})}$] / 8 hours}. • 0.2192 • 1.5 • 0.0673.
Asbestos Worker: ELCR $_{(\text{Central Tendency})}$ = {[(0.5 hour)*EPC$_{(30\ \text{minute})}$ + (7.5 hours)* EPC$_{(\text{Full Shift})}$] / 8 hours} • 0.2192 • 1.5 • 0.0707.
Asbestos Worker: ELCR $_{(\text{High-end})}$ = {[(0.5 hour)*EPC$_{(30\ \text{minute})}$ + (7.5 hours)* EPC$_{(\text{Full Shift})}$] / 8 hours} • 0.2192 • 1.5 • 0.0707.
Asbestos Worker: ELCR $_{(\text{Central Tendency})}$ = {[(0.5 hour)*0.006 + (7.5 hours)*0.006] / 8 hours} • 0.2192 • 1.5 • 0.0707.
Asbestos Worker: ELCR $_{(\text{High-end})}$ = {[(0.5 hour)*0.836 + (7.5 hours)*0.094 / 8 hours} • 0.2192 • 1.5 • 0.0707

Comment 175: For the reasons mentioned in previous comments, I do not believe that the risks predicted here have been accurately estimated or characterized.

4.2.2.7 Risk Estimation for Cancer Effects Following Inhalation Exposures for Gasket Installation/Servicing in UTVs

Statement 176: "Multiple publications (see Section 2.3.2.2) report on occupational exposures associated with installing and servicing gaskets in automobiles. The exposure data used for this COU are presented in Table 2-23. Data on the exposure at the central and high-end estimates are presented along with the ELCR for each exposure distribution in Table 4-35." (page 180, ln. 6428 – 6431)

Table 4-35. Excess Lifetime Cancer Risk for UTV Gasket Installation/Servicing in an Occupational Setting, 8-hour TWA Exposure (from Table 2-23.) before consideration of PPE and any relevant APF

Occupational Exposure Scenario	Exposure Levels (Fibers/cc)				ELCR (40 yr exposure starting at age 16 years)			
	Asbestos Worker		ONU		Asbestos Worker		ONU	
	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end
UTV (based on gasket repair/replacement in vehicles: 8-hr TWA exposure)	0.024	0.066	0.005	0.015	**5.6 E-4**	**1.5 E-3**	**1.2 E-4**	**3.5 E-4**

Asbestos Workers: ELCR $_{(Central\ Tendency)}$ = 0.024 f/cc • 0.2192 • 1.5 • 0.0707 per f/cc
Asbestos Workers: ELCR $_{(High\text{-}end)}$ = 0.066 f/cc • 0.2192 • 1.5 • 0.0707 per f/cc
ONU: ELCR $_{(Central\ Tendency)}$ = 0.005 f/cc • 0.2192 • 1.5 • 0.0707 per f/cc
ONU: ELCR $_{(High\text{-}end)}$ = 0.015 f/cc • 0.2192 • 1.5 • 0.0707 per f/cc

Table 4-35. presents the inhalation cancer risk estimates for workers installing and/or servicing gaskets in utility vehicles and for ONUs exposed to asbestos. For both workers and ONUs, the benchmark cancer risk estimate of $1x10^{-4}$ was exceeded for both central tendency and high-end exposures. Estimates exceeding the benchmark are shaded in pink and bolded.

Comment 176: For the reasons mentioned in previous comments, I do not believe that the risks predicted here have been accurately characterized. Further, given all the factors discussed previously, the plausible number of exposed persons in the coming years is nil or significantly small (and the duration of exposure and airborne concentrations will undoubtedly be extremely low).

4.2.2.8 Summary of Risk Estimates for Cancer Effects for Occupational Inhalation Exposure Scenarios for All COUs

Statement 177: "Table 4-38 summarizes the risk estimates for inhalation exposures for all occupational exposure scenarios for asbestos evaluated in this RE. 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. Risk estimates that exceed the benchmark (i.e., cancer risks greater than the cancer risk benchmark) are shaded and in bold." (page 181, ln. 6470 – 6474)

Table 4-38. Summary of Risk Estimates for Inhalation Exposures to Workers and ONUs by COU

Asbestos Sheets – Gasket Stamping Section 4.2.2.2	Worker	Central Tendency (8-hr)	**3.3 E-4**	3.3 E-5	1.3 E-5
		High-end (8-hr)	**1.4 E-3**	**1.4 E-4**	5.6 E-5
		Central Tendency short term	**3.5 E-4**	3.5 E-5[e]	1.4 E-5[f]
		High-end short term	**1.4 E-3**	**1.4 E-4**[e]	5.6 E-5[f]
	ONU	Central Tendency (8-hr)	5.6 E-5	N/A	N/A
		High-end (8-hr)	**2.3 E-4**	N/A	N/A
		Central Tendency short term	5.6 E-5	N/A	N/A
		High-end short term	**2.3 E-4**	N/A	N/A
Asbestos Sheet Gaskets – use (based on repair/ replacement data from TiO_2 industry) Section 4.2.2.3	Worker	Central Tendency (8-hr)	**6.0 E-4**	6.0 E-5	2.4 E-5
		High-end (8-hr)	**2.2 E-3**	**2.2 E-4**	8.8 E-5
	ONU	Central Tendency (8-hr)	**1.2 E-4**	N/A	N/A
		High-end (8-hr)	**3.7 E-4**	N/A	N/A
Oil Field Brake Blocks Section 4.2.2.4	Worker	Central Tendency (8-hr)	**7.0 E-4**	7.0 E-5	2.8 E-5
	ONU	Central Tendency (8-hr)	**4.6 E-4**	N/A	N/A
Aftermarket Auto Brakes Section 4.2.2.5	Worker	Central Tendency (8-hr)	**1.4 E-4**	1.4 E-5	5.6 E-6
		High-end (8-hr)	**2.2 E-3**	**2.2 E-4**	8.8 E-5
		Central Tendency short-term	**1.4 E-4**	1.4 E-5[e]	5.6 E-6[f]
		High-end short-term	**3.3 E-3**	**3.3 E-4**[e]	**1.3 E-4**[f]
	ONU	Central Tendency (8-hr)	1.6 E-5	N/A	N/A
		High-end (8-hr)	**2.6 E-4**	N/A	N/A
		Central Tendency short-term	1.6 E-5	N/A	N/A
		High-end short-term	**2.6 E-4**	N/A	N/A

Comment 177: For the reasons mentioned in previous comments, I do not believe that the risks predicted here have been accurately estimated (because I think the cancer potency factor is not appropriate) or adequately characterized.

4.2.3.1 Risk Estimation for Cancer Effects Following Episodic Inhalation Exposures for DIY Brake Repair/Replacement

Statement 178: "EPA assessed chronic chrysotile exposures for the DIY (consumer) and bystander brake repair/ replacement scenario based on repeated exposures resulting from recurring episodic exposures from active use of chrysotile asbestos related to DIY brake-related activities. These activities include concomitant exposure to chrysotile asbestos fibers which are reasonably anticipated to remain within indoor and outdoor use facilities. It is well-understood that asbestos fibers in air will settle out in dust and become re-entrained in air during any changes in air currents or activity within the indoor and outdoor use facilities. On the other hand, in occupational settings, regular air sampling would capture

both new and old fibers and have industrial hygiene practices in place to reduce exposures." (page 183, ln. 6503 - 6510)

Comment 178: The impression that the Agency is giving readers is entirely without scientific merit for the fibers of biological and/or regulatory interest. It is folklore that fibers in the vicinity of 5 – 40 microns in length (those of biological importance) can be "reasonably anticipated to remain within indoor and outdoor use facilities." This is an effort to continue repeating statements first developed by persons who never studied the subject because it sounded reasonable to them or because some persons reported finding some concentration of short fibers in air long after their generation. Indeed, the research showing that this is not accurate dates back to the early 1960s (Corn and Stein, 1965; Corn and Stein, 1966; Hinds, 1999).

The Agency is incorrect in its statement that "It is well-understood that asbestos fibers in the air will settle out in the dust and become re-entrained in the air during any changes in air currents or activity within the indoor and outdoor use facilities." Not surprisingly, the statement is offered without a reference.

It seems that this is nothing more than the propagation of a myth that was put forward by experts for the plaintiffs and some government scientists over the last 40 years. This is compounded by several erroneous statements by NIOSH and several of their guidance documents, alleging that this must be the case. Without a quantitative discussion of the length and diameter of the fibers of interest, as well as a quantitative discussion of the half-life for deposition, and some accounting for the factors which influence deposition and resuspension, any general discussion of fiber transport is uninformative (and often nonsense).

Before explaining why statements about fibers being suspended for long periods of time are generally incorrect or uninformative, I am reminded of a lecture by the former head of OSHA, Dr. Morton Corn, who was also chairman of the industrial hygiene department at Johns Hopkins and the University of Pittsburgh, and who received his Ph.D. from Harvard under the direction of Dr. Silverman, on this very topic (Corn and Stein, 1965). Dr. Corn was happy to share with his students and me the following statement – "if these fibers were so easily resuspended as everyone would like you to believe, then all of us would take our dusty and dirty cars and drive them 70 miles per hour on the interstate and expect them to be sparkling clean when we got home. This is not true, and the topic of fiber resuspension is much more complicated than meets the eye."

In 2015, my colleagues and I evaluated the removal rates of chrysotile fibers (PCME size range) from air and compared this rate to modeled estimates of the settling rate of asbestos from air based on simple theoretical calculations relying only on factors such as gravitational settling and/or dilution ventilation (Sahmel et al. 2015). This study measured airborne chrysotile fiber concentrations within the breathing zone (six replicate events), over time and under known ventilation conditions. A fiber concentration decay curve was then developed from the PCME sampling results to estimate an overall concentration decay half-life and the time required to reduce the airborne concentration of PCME chrysotile fibers by 99%. Based on the results of the study, the chrysotile PCME concentration decay half-life for the six replicate events ranged from 1.1 to 11.4 minutes, with an estimated mean half-life of 4.8 minutes (95% CI: 3.4-8.6 minutes), while the average time for 99% removal of PCME chrysotile fibers from air was 32 minutes (95% CI: 22-57 minutes). These findings are consistent with current and previous research showing that a reasonable estimate of time necessary for the removal of 99% of relevant fibers from air ranged from approximately 20 to 80 minutes.

The studies described herein indicate that fibers in the size range of interest to human health (i.e., >5 µm in length) settle and are otherwise removed from the air more rapidly than would be estimated based on gravitational settling or ventilation alone. Specifically, experimental studies show that airborne fiber concentrations decrease rapidly after disturbance, with significant reductions in the first 30 minutes and nearly all fibers removed after one hour. Hinds (1999) noted, "[a]erosol particles attach firmly to any surface they contact" and stated that when particles contact one another, they will readily adhere to form agglomerates or larger masses in the air (Hinds 1999).

Current particulate dispersion models tend to rely mainly on two factors in evaluating the rate at which fibers settle out of the atmosphere: gravitational settling rate and the ventilation/air exchange rate (Timbrell 1965; Drivas et al. 1996; Keil 2000b; Keil et al. 2009). In both gravitational settling and ventilation models, particular limitations (depending on the model) can oftentimes lead to an underestimate of the settling rate for asbestos fibers.

These limitations can include the lack of appropriate ventilation rates (gravitational settling models), as well as not factoring the behavior of small particles that can readily become attached to surfaces (Reist 1984; Hinds 1999). Research in particle dynamics has shown through laboratory and field studies that asbestos fibers and other particulates remain airborne for shorter periods than would be expected based on either gravitational settling models or ventilation model estimates alone (Corn and Stein 1966; Moorcroft and Duggan 1984; Sahmel et al. 2015).

Certain factors other than ventilation and gravitational settling velocity can substantially affect the removal efficiency of particulates from the air including van der Waals forces, electrostatic interactions, impaction onto surfaces, agglomeration, diffusion, Brownian motion, and cohesion due to water molecules (Drinker and Hatch 1954; Zimon and Corn 1969; Reist 1984; Hinds 1999; Sahmel et al. 2015). Reist (1984) confirmed that in turbulent airflows, such as what might be expected in a typical industrial workplace, particles are likely to be removed from the air by mechanisms other than settling (Reist 1984). Also, models that have predicted a very long settling time were usually for fibers that were 1 or 2 µm in length (or shorter) which are generally considered biologically irrelevant by OSHA and the toxicology community (Berman 2010; ERG 2003b, 2003a; Platek et al., 1985: Stanton 1973; Stanton et al. 1977).

Many practitioners have utilized modeling as a tool to estimate airborne asbestos at various distances as long ago as the early 1970s (Bragg et al. 1974; Sawyer and Spooner 1978; USEPA 1978; NRC 1981). The National Research Council's Committee on Indoor Pollutants indicated that the diameter of a fiber was the primary predictor of the settling velocity out of still air from a height of three meters, such that a fiber five µm long and one µm in diameter remained in the air for approximately four hours. This statement is not informative because it does not address the percent of the fibers that were originally released that remain suspended at 15, 30, 60, 90 or 240 minutes after release. It is the basis for dozens of citations in the literature even though it is nearly meaningless from a health risk assessment standpoint.

In contrast, a fiber five µm long and just 0.1 µm in diameter stayed in the air for much longer, almost 20 hours (NRC 1981). Similarly, the U.S. EPA (1978) stated that settling velocity was strongly dependent on fiber diameter and less dependent on fiber length (USEPA, 1978). Based on theoretical calculations of settling velocity for fibers with the same aspect ratio and varying fiber lengths, the U.S. EPA predicted that fibers with an aspect ratio of 5:1 and lengths of 5, 2, and 1 µm took 4, 20, and 80 hours,

respectively, to settle out of still air from a height of three meters. Again, without characterizing the deposition half-life, this statement is meaningless because 90% of the fibers between 5-20 um are known to be deposited in about 90-100 minutes after release.

In various publications, NIOSH and EPA have sometimes carelessly said that asbestos fibers can remain suspended for hours or days (which is not valid for those fibers of biological significance). As stated, without a quantitative characterization of the deposition half-life by fiber length, non-quantitative discussions of fiber deposition or transport are meaningless. A good example of a statement that is not informative, but certainly eye-catching, is presented in EPA's 1986 Guidance for Preventing Asbestos Disease Among Auto Mechanics which states, "[t]he asbestos fibers released from brake and clutch work can be scattered throughout a garage, where they present a hazard for months or years" (USEPA, 1986). The pitfall of that broad claim is that they do not specify the length or diameter of the fibers, and they do not mention the concentration of airborne particles (which influences coagulation or agglomeration), nor the turbulence. Interestingly, at higher airborne concentrations, the time for the vast majority of the fibers/particles to be removed (deposited) is even shorter or when there is much humidity in the air.

The statements presented here about fiber deposition and resuspension can be found in numerous published papers and texts. I would encourage the Agency to study the texts on particle physics by Drinker and Hatch (1954), Reist (1984), Hinds (1999) and Vincent (1995). The fundamental concepts that are provided in their work have also been presented in courtrooms in several jurisdictions across the nation by Dr. Corn, , myself, and others who have studied this topic. Recently a judge embraced those views and struck the plaintiff witnesses who embraced the often vague statements offered in various EPA, OSHA and NIOSH documents regarding fiber transport or suspension.

Excess lifetime cancer risk for people engaging in DIY brake repair (consumers) and replacement

4.2.3.2 Risk Estimation for Cancer Effects following Episodic Inhalation Exposures for UTV Gasket Repair/replacement

Statement 179: "EPA assessed chrysotile exposures for the DIY (consumer) and bystander UTV gasket repair/replacement scenario based on aggregated exposures resulting from recurring episodic exposures from active use of chrysotile asbestos related to DIY brake-related activities. These activities include concomitant exposure to chrysotile asbestos fibers which are reasonably anticipated to remain within indoor use facilities. It is well-understood that asbestos fibers in air will settle out in dust and become re-entrained in air during any changes in air currents or activity indoors. On the other hand, in occupational settings, regular air sampling would capture both new and old fibers and have industrial hygiene practices in place to reduce exposures." (page 189, ln. 6704 - 6711)

Comment 179: This level of detailed analysis of a trivial risk remains a mystery to me. If the Agency feels compelled to study this, that is their right to spend the hours and funds to do so. However, they are assessing phantom exposures using an unsubstantiated cancer potency factor for an extremely small exposure cohort.

I cannot imagine finding an asbestos-containing gasket for a car in the coming years, and even if it could be found, there is no exposure associated with installing a pre-cut gasket.

4.3 Assumptions and Key Sources of Uncertainty

Key Assumptions and Uncertainties in the Uses of Asbestos in the U.S.

Statement 180: "EPA researched sources of information to identify the intended, known, or reasonably foreseen asbestos uses in the U.S. Beginning with the February, 2017 request for information (cite public meeting on Feb 14th) on uses of asbestos and followed by both the Scope document (June (2017c)) and Problem Formulation (June (2018d)), EPA has refined its understanding of the current conditions of use of asbestos in the U.S. This has resulted in identifying chrysotile asbestos as the only fiber type manufactured, imported, processed, or distributed in commerce at this time and under six COU categories." (page 193, ln. 6818 - 6823)

Comment 180: I have asked for many years for both the EPA and OSHA to notify key experts when rulemaking is occurring on topics for which perhaps no more than 10 or 20 persons can offer solid advice. I have been working in this space for approximately 20 - 40 years. Given that the agency cites some of the more visible scientists in the document, it only makes sense that they would notify us about this and similar documents by EPA.

Statement 181: "EPA also received data from one company that fabricates sheet gaskets and one company that uses sheet gaskets. These data were used, even though there are limitations, such as the representativeness of practices in their respective industries." (page 195, ln. 6904 - 6906)

Comment 181: I am not sure of the relevance of these statements, especially given the few numbers of persons exposed, the low levels of airborne asbestos concentrations measured in these facilities, and the lack of potential future exposures to asbestos.

Statement 182: "All the raw chrysotile asbestos imported into the U.S. is used by the chlor-alkali industry for use in asbestos diaphragms. The number of chlor-alkali plants in the U.S. is known and therefore the number of workers potentially exposed is fairly certain. In addition, estimates of workers employed in this industry were provided by the chlor-alkali facilities." (page 195, ln. 6908 - 6911)

Comment 182: This topic was addressed in previous comments.

4.3.4 Key Assumptions and Uncertainties in the Consumer Exposure Assessment

Statement 183: "In Blake et al. (2003), worker exposures were measured at a former automobile repair facility which had an industrial sized and filtered exhaust fan unit to ventilate the building during testing while all doors were closed. A residential garage is not expected to have a filtered exhaust fan installed and operating during DIY consumer brake repair/replacement activities." (page 195, ln. 6932 - 6935)

Comment 183: I appreciate that the EPA is attempting to be thorough by including an uncertainty analysis. However, I think that all this dedicated effort to characterize insignificant exposures and truly negligible risks is not an optimal use of taxpayer money.

Statement 184: "There is also some uncertainty associated with the assumption that a bystander would remain within three meters from the automobile on which the brake repair/replacement work is being conducted for the entire three-hour period EPA assumes it takes the consumer user to complete the work. However, considering a residential garage with the door closed is relatively close quarters for car repair work, it is likely anyone observing (or learning) the brake repair/replacement work would not be able to stay much further away from the car than three meters. Remaining within the garage for the entire three hours also has some uncertainty, although it is expected anyone observing (or learning) the brake repair/replacement work would remain for the entire duration of the work or would not be able to observe (or learn) the task." (page 196, ln. 6949 - 6957)

Comment 184: I agree that it is highly unlikely that anyone would spend much time closer than three meters to a brake repair job. What I find difficult to understand is the purpose of this analysis, as the anticipated exposures in the years ahead are either unlikely or limited to a handful of people in the country.
As noted previously, the only explanation for detailing exposure scenarios that almost certainly will not be experienced by more than a few dozen persons in the coming years feels like a "backdoor" attempt to justify banning asbestos imports into the United States or to strong-arm OSHA into lowering its PEL. Professionally, I am not opposed to either action, but I believe that due process should be followed, and there must be a solid scientific underpinning for the claims made to justify the ban.

Statement 185: "While industry practices have drifted away from the use of compressed air to clean brake drums/pads, no information was found in the literature indicating consumers have discontinued such work practices. To consider potential consumer exposure to asbestos resulting from brake repair/replacement activities, EPA uses data which included use of compressed air. However, EPA recognizes this may be a more conservative estimate because use of compressed air typically could cause considerable dust/fibers to become airborne if it is the only method used." (page 196, 6959 - 6964)

Comment 185: Although I want to be respectful, it is disingenuous for the Agency to claim that they could not find a published paper describing the use of compressed air generators in the garages of homeowners. One only needs to rely on their personal experiences and logic to recognize that it is nearly impossible to imagine a scenario, where in 2020, a homeowner, who was not a mechanic, changing a drum brake on an antique vehicle in their garage, then using compressed air to blow out the brake drum. This would distribute a significant amount of dust into their face and their garage environment. Logically, it is highly unlikely that someone would use compressed air to willingly distribute millions of particles of brake wear debris into their garage and antique automobiles. That is why this work was nearly always performed outdoors.

Statement 186: "The endpoint for both mesothelioma and lung cancer was mortality, not incidence. Incidence data are not available for any of the cohorts. Nevertheless, mortality rates approximate incidence rates for cancers such as lung cancer and mesothelioma because the survival time between cancer incidence and cancer mortality is short. Therefore, while the absolute rates of lung cancer mortality at follow-up may underestimate the rates of lung cancer incidence, the uncertainty for lung cancer is low." (page 197, ln. 7022 - 7026)

Comment 186: As noted in previous comments, there is no scientific basis for attempting to calculate a single cancer potency factor (CPF) for chrysotile for lung cancer and mesothelioma. As stated previously, the Agency has selected the wrong cohorts to calculate the CPF. Instead, they should have focused on understand the three or four meta-analyses of brake mechanics, which showed no increased risk of mesothelioma or lung cancer following exposure to chrysotile. Combining the two endpoints, as discussed previously, due to their different mechanisms of actions, is not consistent with EPA's guidelines. Also, one mechanism for producing cancer due to chrysotile is almost certainly one that has a practical or genuine threshold (lung cancer). Likewise, the mechanism for producing mesothelioma, for chrysotile, may well have a threshold. One thing is certain, the shape of the dose-response curves in the observable range are much different and to blend them into a single dose-response curve poses numerous conceptual and mathematical challenges which are not easily overcome.

4.3.7 Confidence in the Human Health Risk Estimations

Workers/Occupational Non-Users

Statement 187: "Depending on the variations in the exposure profile of the workers/occupational non-users, risks could be under- or over-estimated for all COUs. The estimates for extra cancer risk were based on the EPA-derived IUR for chrysotile asbestos. The occupational exposure assessment made standard assumptions of 240 days per year, 8 hours per day over 40 years starting at age 16 years. This assumes the workers and occupational non-users are regularly exposed until age 56." (page 199, ln. 7106 - 7110)

Comment 187: As much as I applaud the Agency for trying to introduce mathematical confidence intervals to this risk estimation process, it is a classic misuse of risk assessment methods to attempt to characterize the confidence in the estimates given that the exposures of relevance in this document are not likely to occur in the upcoming years. Additionally, the entire foundation for this document's cancer potency factors is inappropriate for short-fiber chrysotile, as the incorrect cohort was selected (for numerous reasons presented here and as discussed in Garabrant's comments, as well as those by Moolgavkar et al).

Assumptions About Bystanders

Statement 188: "The EPA Exposure Factors Handbook (2011) provides the risk assessment community with data-derived values to represent human activities in a variety of settings. For the purposes of this draft risk evaluation, understanding the amount of time consumers spend in a garage is important to develop an exposure scenario for DIYers/mechanics who change their own brakes or gaskets and bystanders to those activities. Table 16-16 in the Handbook, entitled Time Spent (minutes/day) in Various Rooms at Home and in All Rooms Combined, Doers Only, has a section on time spent in a garage." (page 202, ln. 7187 - 7192)

Comment 188: This topic was addressed in previous comments.

Potential Number of Impacted Individuals

Statement 189: "Table 4-54 provides an estimate of the number of impacted individuals for both occupational and consumer exposure scenarios. Some of the estimates have a higher level of confidence than others. For example, EPA is fairly certain about the number of chlor-alkali workers given the information submitted by industry. For some of the other COUs, while there may be some knowledge about the potential number of workers/consumers in a particular COU, there is a lack of information/details on the market share of asbestos-containing products available to both workers and consumers. This makes it difficult to assess level of both certainty and confidence estimating the potential number of impacted individuals using asbestos for the COUs (except for chlor-alkali) in this draft risk evaluation. For ONUs and bystanders, there is a similar lack of understanding of the potential number of potentially impacted individuals." (page 203, ln. 7235 - 7244)

Table 4-54. Summary of Estimated Number of Exposed Workers and DIY Consumers[a].

Condition of Use	Industrial and Commercial		DIY	
	Workers	ONU	Consumer	Bystanders
Asbestos diaphragms – chlor-alkali	75-148	<2900-3000	-	-
Sheet gaskets – stamping	≥2	≥4	-	-
Sheet gaskets – use	25/facility (no. of facilities Unknown)	13/facility (no. of facilities Unknown	-	-
Oilfield brake blocks	<61,695 (total; number exposed to asbestos unknown) [(c)]	<66,108 (total; number in vicinity of asbestos Unknown[(c)]	-	-
Aftermarket automotive brakes/linings, clutches	749,900	749,000	31,857,106	Unknown
Other Vehicle Friction Products (brakes installed in exported cars)	Unknown	Unknown	-	-
Other gaskets – UTVs	~1500 (total; number exposed to asbestos unknown[(d)]	Unknown	Unknown	Unknown

[a] See Text for details.

Comment 189: With all due respect to the Agency, if they are attempting to characterize the number of persons who will be exposed in the coming years to these exposure scenarios, the information in this table is not credible for the following reasons (as noted in previous comments):

1) The description of persons exposed to raw asbestos in the Chlor-alkali industry was surely much less than the values offered here. Beyond that, what is more important, is that hundreds of samples indicated that those workers were usually exposed to non-detectable airborne concentrations of asbestos or were exposed to minimal amounts.

2) I agree with the available information that approximately two people are exposed (nationally) in stamping sheet gaskets that contain asbestos.

3) There is virtually no exposure in installing a pre-punched gasket, since there is no cutting or hole punching involved.

4) I have already noted that it appears most likely that no one is routinely or even sporadically exposed to airborne brake wear debris in the outdoor environment, related to brakes on portable drilling rigs. The number offered in this table is either meant to mislead or deceive, or the Agency was ill-informed.

5) Concerning installing imported aftermarket brakes, after 20 years of searching, I have been unable to identify such a brake on the internet, that is not to say that they do not exist or that some are not sold in the United States. However, they are not as easy to find as the document would lead one to believe. Even in the industry trade association, which has a vested interest in preventing them from being imported into the United States, they could not identify even 1% of the brakes imported into the U.S. as being asbestos containing. Although not stated, you have to assume that 99% of the <1% are disc brakes, for which there is no exposure.

6) Therefore, it is seriously misleading to suggest that 750,000 brake mechanics a year are going to be exposed to asbestos-containing brake linings in the coming years, when few, if any, cars made in the United States have uses drum brakes in the past 40 years.

7) Concerning friction products in other motor vehicles, all I can say is that for the past ten years, few if any of these vehicles entered the United States with asbestos-containing brakes. The threat of litigation and the severe consequence should make such sales unreasonable.

8) Concerning gaskets, the only possible exposure to degraded asbestos-containing gaskets is when a pipefitter might replace a gasket on a very old piece of equipment that was to be brought back into service. I have had a difficult time finding pipes in a scrap yard that contained asbestos gaskets. Most of this scrap metal was sent to China in the 1995 – 2010 era during their massive building boom wherein they consumed virtually all of the world's supply of scrap metals (including old piping, tanks, boilers, old cars, and old steel from abandoned buildings). As an aside, for five years, I watched the San Francisco/Oakland Bay Bridge disassembled piece by piece and dropped into boats, which went directly to China. Thus, I am confident that it would be near impossible to find many asbestos-containing gaskets in equipment that needed to be replaced by a non-asbestos containing gasket.

4.4 Risk Conclusions

Human Health Risk Conclusions to Workers

Statement 190: "With the assumed use of respirators as PPE at APF of 10, most risks would be reduced but still persisted for sheet gasket stamping, sheet gasket use, auto brake replacement, and UTV gasket replacement. When respirators with an APF of 25 was assumed, risk was still indicated for the auto brakes high-end short-term exposure scenario only. It is important to note that based on published evidence for asbestos (see Section 2.3.1.2), nominal APF may not be achieved for all respirator users. ONUs were not assumed to be using PPE to reduce exposures to asbestos." (page 208, ln. 7418 - 7423)

Table 4-55. Summary of Risk Estimates for Inhalation Exposures to Workers and ONUs by COU (Cancer benchmark is 10^{-4})

Life Cycle Stage/Category	Subcategory	Occupational Exposure Scenario	Popula-tion	Exposure Duration and Level	Cancer Risk Estimates (before applying PPE)	Cancer Risk Estimates (with APF=10[c])	Cancer Risk Estimates (with APF=25[c])
	Asbestos Sheets – Gasket Stamping	Section 2.3.1.4	Worker	Central Tendency (8-hr)	**3.3 E-4**	3.3 E-5	1.3 E-5
				High-end (8-hr)	**1.4 E-3**	**1.4 E-4**	5.0 E-5
				Central (w/ short-term)	**3.5 E-4**	3.5 E-5[e]	1.4 E-5[f]
	Aftermarket Auto Brakes	Section 2.3.1.7	Worker	Central Tendency (8-hr)	**1.4 E-4**	1.4 E-5	5.6 E-6
				High-end (8-hr)	**2.2 E-3**	**2.2 E-4**	8.8 E-5
				Central (w/ short-term)	**1.4 E-4**	1.4 E-5[e]	5.6 E-6[f]
				High (w/ short-term)	**3.3 E-3**	**3.3 E-4[e]**	**1.3 E-4[f]**
			ONU	Central (8-hr)	1.6 E-5	N/A	N/A
				High (8-hr)	**2.6 E-4**	N/A	N/A
				Central (w/ short-term)	1.6 E-5	N/A	N/A
				High (w/ short-term)	**2.6 E-4**	N/A	N/A

	Other Vehicle Friction Products	2.3.1.8	Worker	Central Tendency (8-hr)	**1.4 E-4**	1.4 E-5	5.6 E-6
				High-end (8-hr)	**2.2 E-3**	**2.2 E-4**	8.8 E-5
				Central (w/ short-term)	**1.4 E-4**	1.4 E-5[e]	5.6 E-6[f]
				High (w/ short-term)	**3.3 E-3**	**3.3 E-4**[e]	**1.3 E-4**[f]
			ONU	Central (8-hr)	1.6 E-5	N/A	N/A
				High (8-hr)	**2.6 E-4**	N/A	N/A
				Central (w/ short-term)	1.6 E-5	N/A	N/A
				High (w/ short-term)	**2.6 E-4**	N/A	N/A

Comment 190: Although the calculations that were applied to this scenario were correctly executed; however, because the cancer potency factor was incorrectly derived, the exposures are not going to occur in the future with asbestos-containing gaskets and brakes (surely for no more than a few dozen persons) and because the hours of exposure are over-estimated in these calculations, the suggestion that this exposure scenario poses an unacceptable risk (especially going forward) is without scientific foundation.

4.5.3 Human Health Risk Conclusions to Consumers

Statement 191: "Table 4-56 provides a summary of risk estimates for consumers and bystanders. Cancer risks were exceeded for all consumer and bystander UTV gasket replacement exposure scenarios. For consumer and bystander brake replacement scenarios conducted indoors, cancer risk estimates were exceeded for both central tendency and high-end exposures. For outdoor scenarios, cancer risks were exceeded for high-end exposures for 5 minutes/day scenario for DIYers. In addition, cancer risks were exceeded for both DIYers and bystanders for the 30 minutes/day scenario." (page 7437 – 7442)

Table 4-56. Summary of Risk Estimates for Inhalation Exposures to Consumers and Bystanders by COU (Cancer benchmark is 10^{-6})

Life Cycle Stage/Category	Subcategory	Consumer Exposure Scenario	Population	Exposure Duration and Level	Cancer Risk Estimates
Imported asbestos products	Brakes Repair/replacement Indoor, compressed air, once every 3 years for 62 years starting at 16 years, exposures at 30% of active used between uses, 1 hour/d in garage	Section 4.2.3.1	DIY	Central Tendency	**4.3 E-5**
				High-end	**4.2 E-4**
			Bystander	Central Tendency	**2.6 E-5**
				High-end	**6.0 E-5**
	Brakes Repair/ replacement Indoor, compressed air, once every 3 years for 62 years starting at 16 years, exposures at 30% of active used between uses, 8 hours/d in garage	Section 4.2.3.1	DIY	Central Tendency	**3.4 E-4**
				High-end	**3.4 E-3**
			Bystander	Central Tendency	**2.6 E-5**
				High-end	**6.0 E-5**
	Brakes Repair/ replacement Outdoor, once every 3 years for 62 years starting at 16 years, exposures at 2% of active used between uses, 5 min/d in driveway	Section 4.2.3.1	DIY	Central Tendency	9.9 E-8
				High-end	5.3 E-7
			Bystander	Central Tendency	2.1 E-8
				High-end	1.1 E-7
	Brakes Repair/ replacement Outdoor, once every 3 years for 62 years starting at 16 years, exposures at 2% of active used between uses, 30 min/d in driveway	Section 4.2.3.1.	DIY	Central Tendency	2.9 E-7
				High-end	**1.5 E-6**
			Bystander	Central Tendency	5.9 E-8
				High-end	3.2 E-7
	Brakes Repair/replacement Indoor, compressed air, once at 16 years, staying in residence for 10 years, 1 hour/d in garage	Section 4.2.3.1	DIY Bystander	Central Tendency	**5.6 E-6**
				High End	**5.5 E-5**
			Bystander	Central Tendency	**3.2 E-6**
				High-end	**7.3 E-6**
Imported Asbestos Products	Gaskets Repair/ replacement in UTVs Indoor, 1 hour/d, once every 3 years for 62/20 years starting at 16 years exposures at 30% of active used between uses, 1 hour/d in garage	Section 4.3.2.2	DIY	Central Tendency	**2.3 E-5**
				High-end	**6.4 E-5**
			Bystander	Central Tendency	**2.4 E-5**
				High-end	**6.1 E-5**
	Gaskets Repair/ replacement in UTVs	Section 4.3.2.2	DIY	Central Tendency	**1.8 E-4**
				High-end	**5.1 E-4**

Life Cycle Stage/Category	Subcategory	Consumer Exposure Scenario	Population	Exposure Duration and Level	Cancer Risk Estimates
	Indoor, 1 hour/d, once every 3 years for 62 years starting at 16 years exposures at 30% of active used between uses, 8 hour/d in garage		Bystander	Central Tendency	2.4 E-5
				High-end	6.1 E-5
	Gasket Repair Repair/replacement Indoor, once at 16 years, staying in residence for 10 years, 1 hour/d in garage	Section 4.2.3.2	DIY	Central Tendency	3.0 E-6
				High end	8.3 E-6
			Bystander	Central Tendency	3.08 E-6
				High-end	7.16 E-6

Comment 191: Please refer to comment 190. If the person conducting the work is not at risk, it is implausible that there would be bystanders at risk. Please note that the number of bystanders times the number of risk estimates should equal at least one person getting cancer. Otherwise, the increased risk is insignificant.

Risk Determination

Unreasonable Risk

Statement 192: "EPA uses the term "indicates unreasonable risk" to show EPA concern that the chemical substance may have the potential to present unreasonable risk, recognizing that other factors may be considered in making a determination of presents/does not present unreasonable risk. EPA only assessed cancer endpoints in the asbestos risk evaluation. For cancer endpoints, EPA uses the term "greater than risk benchmark" as one indication for the potential of a chemical substance to present unreasonable risk; this occurs, for example, if the lifetime cancer risk value is 5x10-2, which is greater than the benchmarks of 1x10-4 to 1x10-6. Conversely, EPA uses the term "does not indicate unreasonable risk" when EPA does not have a concern for the potential of the chemical substance to present unreasonable risk. More details are described below." (page 213, ln. 7480 - 7488)

Comment 192: My only suggestion is that it is the responsibility of the Agency to consider the number of persons who are likely to be exposed when determining whether an exposure scenario is deemed unacceptable. In this particular scenario, I am hard-pressed to think that in the coming years, more than a handful of people could be exposed to this exposure scenario. I have discussed many times in my comments why these exposures are certainly not going to occur, except in rare circumstances.

5.1.2 Risks to Human Health

Statement 193: "EPA estimates cancer risks by estimating the incremental increase in probability of an individual in an exposed population developing cancer over a lifetime (excess lifetime cancer risk (ELCR)) following exposure to the chemical under specified use scenarios. However, for asbestos, EPA used a less than lifetime exposure calculation because the time of first exposure impacts the cancer outcome (see Section 4.2.1). Standard cancer benchmarks used by EPA and other regulatory agencies are an

increased cancer risk above benchmarks ranging from 1 in 1,000,000 to 1 in 10,000 (i.e., 1x10-6 to 1x10-4 or also denoted as 1 E-6 to 1 E-4) depending on the subpopulation exposed. Generally, EPA considers benchmarks ranging from 1x10-6 to 1x10-4 as appropriate for the general population, consumer users, and nonoccupational PESS.28 7515

For the purposes of this risk determination, EPA uses 1x10-6 as the benchmark for consumers (e.g., do-it-yourself mechanics) and bystanders. In addition, consistent with the 2017 NIOSH guidance,29 EPA uses 1x10-4 as the benchmark for individuals in industrial and commercial work environments subject to Occupational Safety and Health Act (OSHA) requirements. It is important to note that 1x10-4 is not a bright line, and EPA has discretion to make risk determinations based on other benchmarks and considerations as appropriate. It is also important to note that exposure-related considerations (e.g., duration, magnitude, population exposed) can affect EPA's estimates of the ELCR." (page 214, ln. 7507 - 7523)

Comment 193: It is a policy decision whether the Agency sets a risk criteria of 10^-6 for consumers and 10^-4 for workers. The critical point made in these comments is that I have not quibbled with those risk criteria. It is just that the calculations used to determine those criteria are not scientifically justifiable.

Further, as noted, I am not aware of a single circumstance in U.S. regulatory history where actions were taken when not even one theoretical life would be lost in the exposed cohort. These cohorts that are not overexposed using the OSHA PEL acceptability criteria and future exposures will undoubtedly be less than those that occurred over 30 – 50 years ago.

5.2 Risk Determination for Chrysotile Asbestos

Statement 194: "EPA's determination of unreasonable risk for the conditions of use of chrysotile asbestos is based on health risks to workers, occupational non-users (exposed to asbestos indirectly by being in the same work area), consumers, and bystanders (exposed indirectly by being in the same vicinity where consumer uses are carried out).

As described in sections 4, significant risk were identified for lung cancer and mesothelioma. Section 26 of TSCA requires that EPA make decisions consistent with the "best available science."" (page 215, ln. 7563 - 7569)

Comment 194: As noted in previous comments, the conclusions mentioned above are unjustified on many levels.

Statement 195: "During risk evaluation, the only fiber type of asbestos that EPA identified as manufactured (including imported), processed, or distributed under the conditions of use is chrysotile, the serpentine variety. Chrysotile is the prevailing form of asbestos currently mined worldwide. Therefore, it is reasonable to assume that commercially available products fabricated overseas are made with chrysotile. Any asbestos being imported into the U.S. in articles for the conditions of use EPA has identified in this document is believed to be chrysotile." (page 216, ln. 7577 - 7581)

Comment 195: I agree with this statement.

Statement 196: Table 5-5 (page 225)

Table 5-5. Risk Determination for Chrysotile Asbestos: Commercial Use and Disposal of Aftermarket Automotive Asbestos-Containing Brakes/Linings and Other Vehicle Friction Products
(Commercial Mechanic Brake Repair/Replacement is Representative for both COUs; refer to section 4.2.2.5 and 4.2.2.6 for the risk characterization)

Criteria for Risk Determination	Exposed Population	
	Workers	Occupational Non-Users
Life cycle Stage	Commercial Use	Commercial Use
TSCA Section 6(b)(4)(A) Unreasonable Risk Determination	Presents an unreasonable risk of injury to health (workers and occupational non-users)	
Unreasonable Risk Driver	Cancer resulting from chronic inhalation exposure	Cancer resulting from chronic inhalation exposure
Benchmark (Cancer)	10^{-4} excess cancer risks	10^{-4} excess cancer risks
Risk Estimates without PPE	**8-hour TWA** 1.4 E-4 Central Tendency 2.2 E-3 High-end **Short Term** 1.4 E-4 Central Tendency 3.3 E-3 High-end	**8-hour TWA** 1.6 E-5 Central Tendency 2.6 E-4 High-end **Short Term** 1.6 E-5 Central Tendency 2.6 E-4 High-end
Risk Estimates with PPE	**APF = 1** Workers are not assumed to wear respirators; Respirators only required by OSHA if PEL exceeded. **8-hour TWA** 1.4 E-4 Central Tendency 2.2 E-3 High-end **Short Term** 1.4 E-4 Central Tendency 3.3 E-3 High-end	Not Assessed; ONUs are not assumed to wear respirators
Risk Considerations	EPA calculated risk estimates based on data provided in the published literature and OSHA monitoring data (Table 2-14). Although OSHA standards require certain work practices and engineering controls to minimize dust, respiratory PPE is not required unless the permissible exposure limit (PEL) is exceeded. With the expected absence of PPE, the cancer benchmark is exceeded	EPA calculated risk estimates data provided in the published literature. ONU inhalation exposures are expected to be lower than inhalation exposures for workers. EPA estimated a reduction factor of 8.4 (Section 2.3.1.7) for ONUs. Because asbestos fibers released during the worker activities described in Section 2.3.1.7.2 can settle and again become airborne

Comment 196: As noted in previous comments, the risk estimates presented here are flawed on many different levels. However, I applaud the staff for the care and patience that they have expended into this tedious analysis. I am sorry that I cannot support most of the Agency's conclusions in this document.

Concluding Comments

For the past 15 years, many trial lawyers, as well as some EPA and OSHA scientists have claimed that much of the published research, including mine, cited in this EPA document was generated only for the purposes of litigation to inform the courts. Some of the research has also been dismissed with the assertion that no one after 1985 would be routinely exposed to asbestos-containing products.

Interestingly, now the Agency is claiming, erroneously, that over 1,000,000 persons in the future are going to be exposed to asbestos and that the same work that has been routinely rejected is now relevant to understanding public health risks. It is satisfying to know that the totality of scientific literature on this topic is being considered for the benefit of all.

I hope the SAB panels and staff find the comments useful.

Respectfully,

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President, Paustenbach and Associates

And in conjunction with

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Toxicologist, Paustenbach and Associates

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