



**Draft Risk Evaluation for  
Asbestos**

*March 2020*

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550 **ACKNOWLEDGEMENTS**

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551 This report was developed by the United States Environmental Protection Agency (U.S. EPA), Office of  
552 Chemical Safety and Pollution Prevention (OCSP), Office of Pollution Prevention and Toxics (OPPT).

553

554 **Acknowledgements**

555 The OPPT Assessment Team gratefully acknowledges participation and/or input from Intra-agency  
556 reviewers that included multiple offices within EPA, Inter-agency reviewers that included multiple  
557 Federal agencies, and assistance from EPA contractors GDIT (Contract No. CIO-SP3,  
558 HHSN316201200013W), ERG (Contract No. EP-W-12-006), Versar (Contract No. EP-W-17-006), ICF  
559 (Contract No. EPC14001) and SRC (Contract No. EP-W-12-003). EPA also acknowledges the  
560 contributions of technical experts from EPA's Office of Research and Development and epidemiologists  
561 subcontracted to SRC who contributed to the development of the IUR.

562

563 **Docket**

564 Supporting information can be found in public docket: [EPA-HQ-OPPT-2016-0736](https://www.epa.gov/ocsp/epa-hq-oppt-2016-0736).

565

566 **Disclaimer**

567 Reference herein to any specific commercial products, process or service by trade name, trademark,  
568 manufacturer or otherwise does not constitute or imply its endorsement, recommendation or favoring by  
569 the United States Government.

570

571

572 **ABBREVIATIONS**


---

573	ABPO	1989 Asbestos Ban and Phase Out Rule
574	ACC	American Chemistry Council
575	ADC	Average Daily Concentration
576	AHERA	Asbestos Hazard Emergency Response Act
577	ASHAA	Asbestos School Hazard Abatement Act
578	ASHARA	Asbestos School Hazard Abatement Reauthorization Act
579	ATSDR	Agency for Toxic Substances and Disease Registry
580	CAA	Clean Air Act
581	CASRN	Chemical Abstracts Service Registry Number
582	CBI	Confidential Business Information
583	CDR	Chemical Data Reporting
584	CERCLA	Comprehensive Environmental Response, Compensation and Liability Act
585	COU	Condition of Use
586	CPSC	Consumer Product Safety Commission
587	CWA	Clean Water Act
588	DIY	Do-It-Yourself
589	EG	Effluent Guideline
590	ELCR	Excess Lifetime Cancer Risk
591	EMP	Elongated Mineral Particle
592	EPA	Environmental Protection Agency
593	EPCRA	Emergency Planning and Community Right-to-Know Act
594	EU	European Union
595	FDA	Food and Drug Administration
596	f/cc	Fibers per cubic centimeter
597	FHSA	Federal Hazardous Substance Act
598	g	Gram(s)
599	HAP	Hazardous Air Pollutant
600	HEPA	High-Efficiency Particulate Air
601	HTS	Harmonized Tariff Schedule
602	IARC	International Agency for Research on Cancer
603	IRIS	Integrated Risk Information System
604	IUR	Inhalation Unit Risk
605	K <sub>l</sub>	Lung cancer potency factor
606	K <sub>m</sub>	Mesothelioma potency factor
607	LADC	Lifetime Average Daily Concentration
608	lb	Pound
609	LTL	Less Than Lifetime
610	LOEC	Lowest Observable Effect Concentration
611	MAP	Model Accreditation Plan
612	MCLG	Maximum Contaminant Level Goal
613	µm	Micrometers
614	MFL	Million Fibers per Liter
615	mppcf	million particles per cubic foot of air
616	mg	Milligram(s)
617	MPa	Megapascal
618	MSHA	Mine Safety and Health Administration
619	mV	Millivolt
620	NAICS	North American Industry Classification System

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621	ND	Non-detects (value is < analytical detection limit)
622	NEI	National Emissions Inventory
623	NESHAP	National Emission Standard for Hazardous Air Pollutants
624	NIH	National Institutes of Health
625	NIOSH	National Institute for Occupational Safety and Health
626	NPL	National Priorities List
627	NTP	National Toxicology Program
628	OCSPP	Office of Chemical Safety and Pollution Prevention
629	OEM	Original Equipment Manufacturer
630	ONU	Occupational Non-User
631	OPPT	Office of Pollution Prevention and Toxics
632	OSHA	Occupational Safety and Health Administration
633	PCM	Phase Contrast Microscopy
634	PECO	Population, Exposure, Comparator and Outcome
635	PEL	Permissible Exposure Limit
636	PESO	Pathways/Processes, Exposure, Setting and Outcomes
637	PF	Problem Formulation
638	POD	Point of Departure
639	POTW	Publicly Owned Treatment Works
640	PPE	Personal Protective Equipment
641	ppm	Part(s) per Million
642	RCRA	Resource Conservation and Recovery Act
643	RA	Risk Assessment
644	RESO	Receptors, Exposure, Setting/Scenario and Outcomes
645	RfC	Reference Concentration
646	RIA	Regulatory Impact Analysis
647	RR	Relative Risk
648	SDS	Safety Data Sheet
649	SDWA	Safe Drinking Water Act
650	SNUN	Significant New Use Notice
651	SNUR	Significant New Use Rule
652	TSFE	Time Since First Exposure
653	TCCR	Transparent, Clear, Consistent, and Reasonable
654	TEM	Transmission Electron Microscopy
655	TRI	Toxics Release Inventory
656	TSCA	Toxic Substances Control Act
657	TURA	Toxics Use Reduction Act
658	TWA	Time Weighted Average
659	U.S.	United States
660	USGS	United States Geological Survey
661	UTV	Utility vehicle
662	WHO	World Health Organization



663 **EXECUTIVE SUMMARY**

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664

665 This draft risk evaluation for asbestos was performed in accordance with the Frank R. Lautenberg  
666 Chemical Safety for the 21st Century Act and is being disseminated for public comment and peer  
667 review. The Frank R. Lautenberg Chemical Safety for the 21st Century Act amended the Toxic  
668 Substances Control Act (TSCA), the Nation's primary chemicals management law, in June 2016. As per  
669 EPA's final rule, *Procedures for Chemical Risk Evaluation Under the Amended Toxic Substances  
670 Control Act (82 FR 33726)*, EPA is taking comment on this draft and will also obtain peer review on this  
671 draft risk evaluation for asbestos. All conclusions, findings, and determinations in this document are  
672 preliminary and subject to comment. The final risk evaluation may change in response to public  
673 comments received on the draft risk evaluation and/or in response to peer review, which itself may be  
674 informed by public comments. The preliminary conclusions, findings, and determinations in this draft  
675 risk evaluation are for the purposes of identifying whether asbestos presents unreasonable risk or no  
676 unreasonable risk under the conditions of use, in accordance with TSCA section 6, and are not intended  
677 to represent any findings under TSCA section 7.

678

679 TSCA § 26(h) and (i) require EPA to use scientific information, technical procedures, measures,  
680 methods, protocols, methodologies and models consistent with the best available science and to base its  
681 decisions on the weight of the scientific evidence. To meet these TSCA § 26 science standards, EPA  
682 used the TSCA systematic review process described in the *Application of Systematic Review in TSCA  
683 Risk Evaluations* document ([U.S. EPA, 2018a](#)). The data collection, evaluation, and integration stages of  
684 the systematic review process are used to develop the exposure, fate and hazard assessments for risk  
685 evaluations.

686

687 Asbestos is subject to federal and state regulations and reporting requirements. Asbestos is reportable to  
688 the Toxics Release Inventory (TRI) under Section 313 of the Emergency Planning and Community  
689 Right-to-Know Act (EPCRA) but is only reportable in the friable form at concentration levels of 0.1%  
690 or greater. It is designated a Hazardous Air Pollutant (HAP) under the Clean Air Act (CAA), and is a  
691 hazardous substance under the Comprehensive Environmental Response, Compensation and Liability  
692 Act (CERCLA). Asbestos is subject to National Primary Drinking Water Regulations (NPDWR) under  
693 the Safe Drinking Water Act (SDWA) and designated as a toxic pollutant under the Clean Water Act  
694 (CWA) and as such is subject to effluent limitations. Under TSCA, EPA has promulgated several  
695 regulations for asbestos, including the Asbestos Ban and Phase Out rule of 1989, which was then largely  
696 vacated in 1991, and under the Asbestos Hazard Emergency Response Act (AHERA), which requires  
697 inspection of schools for asbestos. On April 25, 2019, EPA finalized an Asbestos Significant New Use  
698 Rule (SNUR) under TSCA Section 5 that prohibits manufacture (including import) or processing of  
699 discontinued uses of asbestos from restarting without EPA having an opportunity to evaluate each  
700 intended use for risks to health and the environment and to take any necessary regulatory action, which  
701 may include a prohibition.

702

703 Asbestos has not been mined or otherwise produced in the U.S. since 2002. Although there are several  
704 known types of asbestos, the only form of asbestos known to be imported, processed, or distributed for  
705 use in the United States at the posting of this draft risk evaluation is chrysotile. Raw chrysotile asbestos  
706 currently imported into the U.S. is used exclusively by the chlor-alkali industry. Based on 2019 data, the  
707 total amount of raw asbestos imported into the U.S. was 750 metric tons. EPA has also identified the  
708 importation of asbestos-containing products; however, the import volumes of those products are not  
709 fully known. The asbestos-containing products that EPA has identified as being imported and used are

710 sheet gaskets, brake blocks, aftermarket automotive brakes/linings, other vehicle friction products, and  
711 other gaskets. In this draft risk evaluation, EPA evaluated the following categories of conditions of use  
712 (COU) for chrysotile asbestos: manufacturing; processing; distribution in commerce; occupational and  
713 consumer uses; and disposal.

714

715 Approach

716 EPA used reasonably available information (defined in 40 CFR 702.33 as “*information that EPA*  
717 *possesses, or can reasonably obtain and synthesize for use in risk evaluations, considering the deadlines*  
718 *for completing the evaluation*”), in a fit-for-purpose approach, to develop a risk evaluation that relies on  
719 the best available science and is based on the weight of the scientific evidence. EPA used previous  
720 analyses as a starting point for identifying key and supporting studies to inform the exposure, fate, and  
721 hazard assessments. EPA also evaluated other studies published since the publication of previous  
722 analyses. EPA reviewed the information and evaluated the quality of the methods and reporting of  
723 results of the individual studies using the evaluation strategies described in *Application of Systematic*  
724 *Review in TSCA Risk Evaluations* ([U.S. EPA, 2018a](#)).

725

726 During development of this risk evaluation, the only fiber type of asbestos that EPA identified as  
727 imported, processed, or distributed under the COUs in the United States is chrysotile, the serpentine  
728 variety. Chrysotile is the prevailing form of asbestos currently mined worldwide, and so it is assumed  
729 that a majority of commercially available products fabricated overseas are made with chrysotile. Any  
730 asbestos being imported into the U.S. in articles is believed to be chrysotile. The other five forms of  
731 asbestos are now subject to a SNUR as described previously<sup>1</sup>.

732

733 EPA evaluated the following categories of COU of chrysotile asbestos in this draft risk evaluation:  
734 manufacturing; processing; distribution in commerce; occupational and consumer uses; and disposal for  
735 the following COUs: use of diaphragms in the chlor-alkali industry, sheet gaskets in chemical  
736 production facilities, oilfield brake blocks, aftermarket automotive brakes/linings, other vehicle friction  
737 products, and other gaskets. EPA continues to review the recent court decision in *Safer Chemicals*  
738 *Healthy Families v. EPA*, Nos. 17-72260 et al. (9th Cir. 2019), and this draft risk evaluation does not  
739 reflect consideration of any legacy uses and associated disposal for chrysotile asbestos or other asbestos  
740 fiber types as a result of that decision. EPA intends to consider legacy uses and associated disposal in a  
741 supplemental scope document and supplemental risk evaluation.

742

743 In the [problem formulation](#) ([U.S. EPA, 2018d](#)) (PF), EPA identified the conditions of use and presented  
744 three conceptual models and an analysis plan for this draft risk evaluation. These have been carried into  
745 the draft risk evaluation where EPA has quantitatively evaluated the risk to human health using  
746 monitoring data submitted by industry and found in the scientific literature through systematic review  
747 for the COUs (identified in Section 1.4.3 of this draft risk evaluation). During the PF phase of the Risk  
748 Evaluation, EPA was still in the process of identifying potential asbestos water releases for the TSCA

---

<sup>1</sup> This requires notification to, and review by, the Agency should any person wish to pursue manufacturing, importing, or processing crocidolite (riebeckite), amosite (cummingtonite-grunerite), anthophyllite, tremolite or actinolite (either in raw form or as part of articles) for any use (40 CFR 721.11095). Therefore, under the final asbestos SNUR, EPA will be made aware of manufacturing, importing, or processing for any intended use of crocidolite (riebeckite), amosite (cummingtonite-grunerite), anthophyllite, tremolite or actinolite (either in raw form or as part of articles). If EPA finds upon review of the Significant New Use Notice (SNUN) that the significant new use presents or may present an unreasonable risk (or if there is insufficient information to permit a reasoned evaluation of the health and environmental effects of the significant new use), then EPA would take action under TSCA section 5(e) or (f) to the extent necessary to protect against unreasonable risk.

749 COUs to determine the need to evaluate risk to aquatic and sediment-dwelling organisms. After the PF  
750 was released, EPA continued to search EPA databases as well as the literature and attempted to contact  
751 industries to shed light on potential releases to water. The reasonably available information indicated  
752 that there were minimal or no surface water releases of asbestos associated with the COUs in this draft  
753 risk evaluation.

754  
755 EPA evaluated exposures (inhalation only) to asbestos in occupational and consumer settings to  
756 estimate risk of health hazard (cancer only) for the COUs in this draft risk evaluation. In occupational  
757 settings, EPA evaluated inhalation exposures to workers and occupational non-users, or ONUs. EPA  
758 used inhalation monitoring data submitted by industry and literature sources, where reasonably  
759 available and that met TSCA systematic review data evaluation criteria, to estimate potential inhalation  
760 exposures. In consumer settings, EPA evaluated inhalation exposures to both consumers (Do-it-  
761 Yourselfers or DIY mechanics) and bystanders and used estimated inhalation exposures, from literature  
762 sources where reasonably available and that met data evaluation criteria, to estimate potential  
763 exposures using a range of user durations. These analyses are described in Section 2.3 of this draft risk  
764 evaluation.

765  
766 EPA evaluated reasonably available information for human health hazards and identified hazard  
767 endpoints for cancer. EPA used the Framework for Human Health Risk Assessment to Inform Decision  
768 Making ([U.S. EPA, 2014a](#)) to evaluate, extract, and integrate asbestos' dose-response information. EPA  
769 evaluated the large database of health effects associated with asbestos exposure cited in numerous U.S.  
770 and international data sources. Many authorities have established that there are causal associations  
771 between asbestos exposures and cancer ([NTP, 2016](#); [IARC, 2012](#); [ATSDR, 2001a](#); [U.S. EPA, 1988b](#);  
772 [IARC, 1987](#); [U.S. EPA, 1986](#); [IARC, 1977](#)).

773  
774 Given the well-established carcinogenicity of asbestos for cancer, EPA, in its PF document, decided to  
775 limit the scope of its systematic review to cancer and to inhalation exposures with the goal of updating,  
776 or reaffirming, the existing 1988 EPA inhalation unit risk (IUR) for general asbestos ([U.S. EPA,](#)  
777 [1988b](#)). Therefore, the literature was reviewed to determine whether a new IUR needed to be  
778 developed. The IUR for asbestos developed in 1988 was based on 14 epidemiologic studies that  
779 included occupational exposure to chrysotile, amosite, or mixed-mineral exposures [chrysotile, amosite,  
780 crocidolite]. However, EPA's research to identify COUs indicated that only chrysotile asbestos is  
781 currently being imported in the raw form or imported in products. In addition, most studies of  
782 populations exposed only to chrysotile provide the most informative data for the purpose of developing  
783 the TSCA risk estimates for the COUs for asbestos in this document. EPA will consider legacy uses  
784 and associated disposal in subsequent supplemental documents.

785  
786 As stated in Section 3.2, epidemiological studies on mesothelioma and lung cancer in cohorts of workers  
787 using chrysotile in commerce were identified that could inform the estimation of an exposure-response  
788 function allowing for the derivation of a chrysotile asbestos IUR. EPA could not find any recent risk  
789 values in the literature for chrysotile asbestos since the IRIS IUR value was the result of contemporary  
790 data from the 1980s.

791  
792 EPA derived the chrysotile IUR based on review of the epidemiology literature describing occupational  
793 cohorts exposed to commercial chrysotile that provided adequate data for the assessment of lung cancer  
794 and mesothelioma risks. EPA developed data evaluation criteria specifically to assess the quality of  
795 epidemiology studies of asbestos and lung cancer and mesothelioma. The study domains of exposure,  
796 outcome, study participation, potential confounding, and analysis were further tailored to the specific  
797 needs of evaluating asbestos studies for their potential to provide information on the exposure-response

798 relationship between asbestos exposure and mortality from lung cancer and from mesothelioma. In terms  
799 of evaluating exposure information, asbestos is unique among these first 10 TSCA chemicals  
800 undergoing risk evaluation as it is a fiber and has a long history of different exposure assessment  
801 methodologies. For mesothelioma, this assessment is also unique with respect to the impact of the  
802 timing of exposure relative to the cancer outcome as the time since first exposure plays a dominant role  
803 in modeling risk. The most relevant exposures for understanding mesothelioma risk were those that  
804 occurred decades prior to the onset of mesothelioma and subsequent mesothelioma mortality.

805  
806 Cancer potency values were either extracted from published epidemiology studies or derived from the  
807 data within those studies. Once the cancer potency values were obtained, they were adjusted for  
808 differences in air volumes between workers and other populations so that those values can be applied  
809 to the U.S. population as a whole in standard EPA life-table analyses. The life-table methodology  
810 allows the estimation of an exposure concentration associated with a specific extra risk of cancer  
811 mortality caused by chrysotile asbestos. According to standard practice, the lifetime unit risks for lung  
812 cancer and mesothelioma were estimated separately and then statistically combined to yield the cancer  
813 inhalation unit risk. Less-than-lifetime or partial lifetime unit risks were also derived for a range  
814 of exposure scenarios based on different ages of first exposure and different durations of exposure  
815 (e.g., 20 years old and 40 years of exposure).

816  
817 Risk Characterization

818 Environmental Risk: Based on the reasonably available information in the published literature,  
819 provided by industries using asbestos, and reported in EPA databases, there is minimal or no releases  
820 of asbestos to surface water associated with the COUs that EPA is evaluating in this risk evaluation.  
821 Thus, EPA believes there is low or no potential for environmental risk to aquatic or sediment-dwelling  
822 receptors from the COUs included in this risk evaluation because water releases associated with the  
823 COUs are not expected and were not identified. Terrestrial pathways, including biosolids, were  
824 excluded from risk evaluation at the PF stage.

825  
826 Human Health Risks: EPA identified cancer risks from inhalation exposure to chrysotile asbestos.  
827 For workers and ONUs, EPA estimated cancer risk from inhalation exposures to asbestos using IUR  
828 values and exposures for each COU. EPA estimated risks using several occupational exposure  
829 scenarios related to the central and high-end estimates of exposure without the use of personal  
830 protective equipment (PPE), and with potential PPE for workers using asbestos. Industry submissions  
831 indicated that some workers used respirators for certain tasks, but not others, while other workers used  
832 ineffective respirators (sheet gasket stampers using N95 respirators is not protective based on OSHA  
833 regulations). Although hypothetical respirator usage with an applied protection factor (APF) of 10 and  
834 25 was calculated for all COUs, actual respirator use was limited to an APF of 10 (the use of sheet  
835 gaskets) and APFs of 10 and 25, in some cases, for chlor-alkali diaphragm use. No other APFs were  
836 indicated for any other COU. For asbestos, nominal APFs (e.g., 25) may not be achieved for all PPE  
837 users. More information on respiratory protection, including EPA's approach regarding the  
838 occupational exposure scenarios for asbestos, is in Section 2.3.1.2.

839  
840 For workers, cancer risks in excess of the benchmark of 1 death per 10,000 (or  $1 \times 10^{-4}$ ) were indicated  
841 for all conditions of use under high-end and central tendency exposure scenarios when PPE was not  
842 used. With the hypothetical use of PPE at APF of 10 (except for chlor-alkali processing and use and  
843 sheet gasket use), most risks were reduced for central tendency estimates but still persisted for sheet  
844 gasket stamping, auto brake replacement, other vehicle friction products and utility vehicle (UTV use  
845 and disposal) gasket replacement for high-end exposure estimates (both 8-hour and short-term  
846 durations). Although not expected to be worn given the reasonably available information, when PPE

847 with an APF of 25 was applied, risk was still indicated only for the high-end, short term exposure  
848 scenario for the auto brakes and other vehicle friction products. EPA's estimates for worker risks for  
849 each occupational scenario are presented by each COU in Section 4.2.2 and summarized in Table 4-38.  
850

851 For ONUs, cancer risks in excess of the benchmark of 1 death per 10,000 (or  $1 \times 10^{-4}$ ) were indicated for  
852 both central tendency and high-end exposures for sheet gasket use (in chemical production) and UTV  
853 gasket replacement. In addition, cancer risks for ONUs were indicated for high-end exposures only for  
854 chlor-alkali, sheet gasket stamping, and auto brakes. ONUs were not assumed to be using PPE to reduce  
855 exposures to asbestos used in their vicinity. EPA's estimates for ONU risks for each occupational  
856 exposure scenario are presented by each COU in Section 4.2.2 and summarized in Table 4-38.  
857

858 For consumers (Do-it-Yourselfers, or DIY) and bystanders of consumer use, EPA estimated cancer  
859 risks resulting from inhalation exposures with a range of user durations, described in detail in Section  
860 4.2.3. EPA assumed that consumers or bystanders would not use PPE.  
861

862 For consumers and bystanders, cancer risks in excess of the benchmark of 1 death per 1,000,000 (or  $1 \times 10^{-6}$ )  
863 were indicated for most COUs for consumer exposure scenarios. Risks were indicated for all  
864 high-end exposures for both consumers and bystanders for brake and UTV gasket indoor scenarios;  
865 and the high-end consumer outdoor scenarios (for 30-minute exposures). EPA's estimates for  
866 consumer and bystander risks for each consumer use exposure scenario are presented in Section 4.2.3  
867 and summarized in Table 4-48.  
868

869 *Uncertainties.* Uncertainties have been identified and discussed after each section in this risk  
870 evaluation. In addition, Section 4.3 summarizes the major assumptions and key uncertainties by major  
871 topic: uses of asbestos, occupational exposure, consumer exposure, environmental risk, IUR  
872 derivation, cancer risk value and human health risk estimates.  
873

874 Beginning with the February, 2017 request for information on uses of asbestos ([see 2017 Public](#)  
875 [Meeting](#)) and followed by both the Scope document (June (2017c)) and Problem Formulation (June  
876 (2018d)), EPA has refined its understanding of the current conditions of use of asbestos in the U.S.  
877 Chrysotile asbestos is the only fiber type imported, processed, or distributed in commerce for use in  
878 2019. All the raw asbestos imported into the U.S. is used by the chlor-alkali industry for use in asbestos  
879 diaphragms. The remaining COUs are for articles that contain chrysotile asbestos and EPA received  
880 voluntary acknowledgement from a handful of industries that fall under these COU categories.  
881 Therefore, EPA evaluated manufacturing, processing, distribution in commerce, occupational and  
882 consumer uses, and disposal of chrysotile asbestos in this draft risk evaluation.  
883

884 By finalizing the asbestos SNUR on April 25, 2019 to include manufacturing (including import) or  
885 processing discontinued uses not already banned under TSCA, EPA is highly certain that manufacturing  
886 (including import), processing, or distribution of asbestos is not intended, known or reasonably foreseen  
887 beyond the six product categories in this risk evaluation. EPA will consider legacy uses and associated  
888 disposal in subsequent supplemental documents.  
889

890 For occupational exposures, the number of chlor-alkali plants in the U.S. is known and therefore the  
891 number of workers potentially exposed is fairly certain. The number of workers potentially exposed for  
892 other COUs is less certain. Only two workers were identified for stamping sheet gaskets, and two TiO<sub>2</sub>  
893 manufacturing facilities were identified in the U.S. who use asbestos-containing gaskets. However, EPA  
894 is not certain if asbestos-containing sheet gaskets are used in other industries and to what extent. For the  
895 other COUs, no estimates of the number of potentially exposed workers were submitted to EPA by

896 industry or its representatives, so estimates were used and were based on market estimates for that work  
897 category; but with no information on the market share for asbestos containing products. Therefore,  
898 numbers of workers potentially exposed were estimated and, based on the COU, these estimations have  
899 a range of uncertainty from low (chlor-alkali) to high (sheet gasket use, oilfield brake blocks,  
900 aftermarket automotive brakes/linings, other vehicle friction products and other gaskets).

901  
902 Exposures for ONUs can vary substantially. Most data sources do not sufficiently describe the proximity  
903 of these employees to the exposure source. As such, exposure levels for the ONU category will vary  
904 depending on the work activity. It is unknown whether these uncertainties overestimate or underestimate  
905 exposures.

906  
907 A review of reasonably available literature for consumer exposure estimates related to brake  
908 repair/replacement activities by a DIY consumer was limited and no information for consumer exposure  
909 estimates related to UTV exhaust system gasket repair/replacement activities was found. This absence of  
910 scenario-specific exposure information required EPA to use surrogate monitoring data from  
911 occupational studies to evaluate consumer risk resulting from exposure to asbestos during these two  
912 activities. The surrogate occupational studies tended to be based on older studies that may or may not  
913 reflect current DIY consumer activities, including best practices for removing asbestos containing  
914 materials. In addition, EPA is uncertain about the number of asbestos containing brakes that are being  
915 purchased online and installed in cars (classic cars or new cars) or gaskets that are being replaced in  
916 UTVs.

917  
918 After the PF was released, EPA continued to search EPA databases and all publicly available literature  
919 and contact industries to shed light on potential releases to water from the COUs in this risk evaluation  
920 for the purpose of evaluating risk to aquatic or sediment-dwelling organisms. EPA found minimal or no  
921 releases of asbestos to surface water associated with the COUs in this risk evaluation. In addition, there  
922 are no reported releases of asbestos to water from TRI. EPA views the uncertainty that this introduces as  
923 low.

924  
925 A specific IUR was developed in this risk evaluation for combined mesothelioma and lung cancer  
926 following exposure to chrysotile asbestos. There is evidence that other cancer endpoints may also be  
927 associated with exposure to the commercial forms of asbestos. IARC concluded that there was sufficient  
928 evidence in humans that commercial asbestos (chrysotile, crocidolite, amosite, tremolite, actinolite, and  
929 anthophyllite) was causally associated with lung cancer and mesothelioma, as well as cancer of the  
930 larynx and the ovary. The lack of sufficient numbers of workers to estimate risks of ovarian and  
931 laryngeal cancer is a downward bias leading to lower IUR estimates in an overall cancer health  
932 assessment; however, the selected IUR was chosen to compensate for this bias (See Section 3.2.4).

933  
934 The endpoint for both mesothelioma and lung cancer was mortality, not incidence. Incidence data are  
935 not available for any of the cohorts. Nevertheless, mortality rates approximate incidence rates for  
936 cancers such as lung cancer and mesothelioma because the survival time between cancer incidence and  
937 cancer mortality is short. Therefore, while the absolute rates of lung cancer mortality may underestimate  
938 the rates of lung cancer incidence, the uncertainty for lung cancer is low. For mesothelioma, the median  
939 length of survival with mesothelioma is less than 1 year for males, with less than 20% surviving after 2-  
940 years and less than 6% surviving after 5-years. Because the mesothelioma model is absolute risk, this  
941 leads to an under-ascertainment on mesothelioma risk, however, the selected IUR was chosen to  
942 compensate this bias (See Section 3.2.4)

943

944 Epidemiologic studies are observational and as such are potentially subject to confounding and selection  
945 biases. Most of the studies of asbestos exposed workers did not have information to control for cigarette  
946 smoking, which is an important risk factor for lung cancer in the general population. However, the bias  
947 related to this failure to control for smoking is believed to be small. It is unlikely that smoking rates  
948 among workers in the chosen epidemiology studies differed substantially enough with respect to their  
949 cumulative chrysotile exposures to induce important confounding in risk estimates for lung cancer (see  
950 Section 4.3.7). Mesothelioma is not related to smoking and thus smoking could not be a confounder for  
951 mesothelioma.

952

953 Depending on the variations in the exposure profile of the workers/occupational non-users and  
954 consumers/bystanders, risks could be under- or over-estimated for all COUs. The estimates for extra  
955 cancer risk were based on the EPA-derived IUR for chrysotile asbestos. The occupational exposure  
956 assessment made standard assumptions of 240 days per year, 8 hours per day over 40 years starting at  
957 age 16 years. This assumes the workers and ONUs are regularly exposed until age 56. If a worker  
958 changes jobs during their career and are no longer exposed to asbestos, this may overestimate exposures.  
959 However, if the worker stays employed after age 56, it would underestimate exposures.

960

961 Potentially Exposed Susceptible Subpopulations (PESS): TSCA § 6(b)(4) requires that EPA conduct a  
962 risk evaluation to “*determine whether a chemical substance presents an unreasonable risk of injury to*  
963 *health or the environment, without consideration of cost or other non-risk factors, including an*  
964 *unreasonable risk to a potentially exposed or susceptible subpopulation identified as relevant to the risk*  
965 *evaluation by the Administrator, under the conditions of use.*” TSCA § 3(12) states that “*the term*  
966 *‘potentially exposed or susceptible subpopulation’ means a group of individuals within the general*  
967 *population identified by the Administrator who, due to either greater susceptibility or greater exposure,*  
968 *may be at greater risk than the general population of adverse health effects from exposure to a chemical*  
969 *substance or mixture, such as infants, children, pregnant women, workers, or the elderly.*”

970

971 EPA identified certain human subpopulations who may be more susceptible to exposure to asbestos than  
972 others. Workers exposed to asbestos in workplace air, especially if they work directly with asbestos, are  
973 most susceptible to the health effects associated with asbestos. Although it is clear that the health risks  
974 from asbestos exposure increase with heavier exposure and longer exposure time, investigators have  
975 found asbestos-related diseases in individuals with only brief exposures. Generally, those who develop  
976 asbestos-related diseases could show no signs of illness for decades after exposure.

977

978 A source of variability in susceptibility between people is smoking history or the degree of exposure to  
979 other risk factors with which asbestos interacts. In addition, the long-term retention of asbestos fibers in  
980 the lung and the long latency period for the onset of asbestos-related respiratory diseases suggest that  
981 individuals exposed earlier in life may be at greater risk to the eventual development of respiratory  
982 problems than those exposed later in life ([ATSDR, 2001a](#)). There is also some evidence of genetic  
983 predisposition for mesothelioma related to having a germline mutation in BAP1 ([Testa et al., 2011](#)).

984

985 Cancer risks were indicated for all the worker COUs and most of the consumer/bystander COUs. In  
986 addition, several subpopulations (e.g., smokers, genetically predisposed individuals, workers who  
987 change their own asbestos-containing brakes) may be more susceptible than others to health effects  
988 resulting from exposure to asbestos. These conditions are discussed in more detail for potentially  
989 exposed or susceptible subpopulations and aggregate exposures in Section 4.4 and Section 4.5.

990

991 Aggregate and Sentinel Exposures: Section 6(b)(4)(F)(ii) of TSCA requires the EPA, as a part of the risk  
992 evaluation, to describe whether aggregate or sentinel exposures under the conditions of use were  
993 considered and the basis for their consideration. The EPA has defined aggregate exposure as “*the*  
994 *combined exposures to an individual from a single chemical substance across multiple routes and*  
995 *across multiple pathways* (40 CFR § 702.33).” Exposures to asbestos were evaluated by the inhalation  
996 route only. Inhalation and oral exposures could occur simultaneously for workers and consumers. EPA  
997 chose not to employ simple additivity of exposure pathways at this time within a COU since the most  
998 critical exposure pathway is inhalation and the target being assessed is combined lung cancer and  
999 mesothelioma.

1000  
1001 Aggregate exposures for asbestos were not assessed by all routes of exposure, since only inhalation  
1002 exposure was evaluated in the RE. Pathways of exposure were also not combined in this RE. EPA  
1003 recognizes that it is possible that workers exposed to asbestos might also be exposed as consumers (by  
1004 changing asbestos-containing brakes at home).

1005  
1006 The EPA defines sentinel exposure as “*the exposure to a single chemical substance that represents the*  
1007 *plausible upper bound of exposure relative to all other exposures within a broad category of similar or*  
1008 *related exposures* (40 CFR § 702.33).” In this risk evaluation, the EPA considered sentinel exposure the  
1009 highest exposure given the details of the COU and the potential exposure scenarios. EPA considered  
1010 sentinel exposures by considering risks to populations who may have upper bound (e.g., high-end, high  
1011 intensities of use) exposures

#### 1012 Risk Determination

1013 In each risk evaluation under TSCA section 6(b), EPA determines whether a chemical substance  
1014 presents an unreasonable risk of injury to health or the environment, under the conditions of use. The  
1015 determination does not consider costs or other non-risk factors. In making this determination, EPA  
1016 considers relevant risk-related factors, including, but not limited to: the effects of the chemical substance  
1017 on health and human exposure to such substance under the conditions of use (including cancer and non-  
1018 cancer risks); the effects of the chemical substance on the environment and environmental exposure  
1019 under the COU; the population exposed (including any potentially exposed or susceptible  
1020 subpopulations); the severity of hazard (including the nature of the hazard, the irreversibility of the  
1021 hazard); and uncertainties. EPA also takes into consideration the Agency’s confidence in the data used  
1022 in the risk estimate. This includes an evaluation of the strengths, limitations, and uncertainties associated  
1023 with the information used to inform the risk estimate and the risk characterization. The rationale for the  
1024 risk determination is discussed in Section 5.2.

1025  
1026  
1027 Environmental Risk: As described in the problem formulation ([U.S. EPA, 2018d](#)), other Agency  
1028 regulations adequately assess and effectively manage exposures from asbestos releases to terrestrial  
1029 pathways, including biosolids, for terrestrial organisms. After the PF was released, EPA continued to  
1030 search EPA databases as well as the literature and contacted industries to shed light on potential releases  
1031 of asbestos to water from the TSCA COUs. Based on the reasonably available information in the  
1032 published literature, provided by industries using asbestos, and reported in EPA databases, there is  
1033 minimal or no releases of asbestos to surface water associated with the COUs in this risk evaluation.  
1034 Therefore, EPA concludes there is no unreasonable risk to aquatic or sediment-dwelling environmental  
1035 organisms. Details are provided in Section 4.1.

1036  
1037 Risk of Injury to Health: EPA’s determination of unreasonable risk for specific COUs of asbestos listed  
1038 below are based on health risks to workers, occupational non-users, consumers, or bystanders from  
1039 consumer use. The health effect driver for EPA’s determination of unreasonable risk is cancer from



1040 inhalation exposure. As described below, risks to the general population were not relevant for these  
1041 conditions of use.

1042  
1043 There are physical-chemical characteristics that are unique to asbestos, such as insolubility in water,  
1044 suspension and duration in air, transportability, the friable nature of asbestos-containing products, which  
1045 attribute to the potential for asbestos fibers to be released, settled, and to again become airborne under  
1046 the conditions of use (re-entrainment). Also unique to asbestos is the impact of the timing of exposure  
1047 relative to the cancer outcome; the most relevant exposures for understanding cancer risk were those that  
1048 occurred decades prior to the onset of cancer and subsequent cancer mortality. In addition to the cancer  
1049 benchmark, the physical-chemical properties and exposure considerations are important factors in  
1050 considering risk of injury to health. To account for the exposures for ONUs and, in certain cases  
1051 bystanders, EPA derived a distribution of exposure values for calculating the risk for cancer by using  
1052 area monitoring data (i.e., fixed location air monitoring results) where available for certain conditions of  
1053 use and when appropriate applied exposure reduction factors, using data from published literature (see  
1054 Sections 2.3.1 and 2.3.2 for details on ONU and bystander methods, respectively). The risk  
1055 determination for each COU in this risk evaluation considers both central tendency and high-end risk  
1056 estimates for workers, ONUs, consumers and bystanders. Where relevant EPA considered PPE for  
1057 workers. For many of the COUs both the central tendency and high-end risk estimates exceed the risk  
1058 benchmark for each of the exposed populations evaluated. However, the risk benchmarks do not serve as  
1059 a bright line for making risk determinations and other relevant risk-related factors were considered. In  
1060 particular there are severe and irreversible health effects associated with asbestos inhalation exposures  
1061 and fibers can become airborne again and available for exposure, which resulted in EPA focusing on the  
1062 high-end risk estimates rather than central tendency risk estimates to be most protective of workers,  
1063 ONUs, consumers, and bystanders. Additionally, EPA's confidence in the data used in the risk estimate  
1064 is considered.

1065  
1066 Risk to the General Population: General population exposures to chrysotile asbestos may occur from  
1067 industrial and/or commercial uses; industrial releases to air, water or land; and other conditions of use.  
1068 As part of the PF for asbestos, EPA found those exposure pathways are covered under the jurisdiction of  
1069 other environmental statutes, administered by EPA, which adequately assess and effectively manage  
1070 those exposures, i.e., CAA, SDWA, CWA, and RCRA. EPA believes that the TSCA risk evaluation  
1071 should focus on those exposure pathways associated with TSCA uses that are not subject to the  
1072 regulatory regimes discussed above because these pathways are likely to represent the greatest areas of  
1073 concern to EPA for unmanaged risks. Therefore, EPA did not evaluate hazards or exposures to the  
1074 general population in this risk evaluation, and there is no risk determination for the general population.

1075  
1076 Risk to Workers: The conditions of use of asbestos that present an unreasonable risk to workers include  
1077 processing and industrial use of asbestos-containing diaphragms, processing and industrial use of  
1078 asbestos-containing sheet gaskets and industrial use of asbestos-containing brake blocks, aftermarket  
1079 automotive asbestos-containing brakes/linings, other vehicle friction products, and other asbestos-  
1080 containing gaskets. A full description of EPA's determination for each condition of use is in Section 5.2.

1081  
1082 Risk to Occupational Non-Users (ONUs): EPA determined that the conditions of use that present  
1083 unreasonable risks for ONUs include processing and industrial use of asbestos-containing diaphragms,  
1084 processing and industrial use of asbestos-containing sheet gaskets and industrial use of asbestos-  
1085 containing brake blocks, aftermarket automotive asbestos-containing brakes/linings, other vehicle  
1086 friction products, and other asbestos-containing gaskets. A full description of EPA's determination for  
1087 each condition of use is in Section 5.2.

1088

1089 Risk to Consumers: For consumers, EPA determined that the conditions of use that present an  
1090 unreasonable risk are use of aftermarket automotive asbestos-containing brakes/linings and other  
1091 asbestos-containing gaskets. A full description of EPA’s determination for each condition of use is in  
1092 Section 5.2.

1093

1094 Risk to Bystanders (from consumer uses): EPA determined that the conditions of use that present an  
1095 unreasonable risk to bystanders are use of aftermarket automotive asbestos-containing brakes/linings  
1096 and other asbestos-containing gaskets. A full description of EPA’s determination for each condition of  
1097 use is in Section 5.2.

1098

1099 Summary of risk determinations:

1100 EPA has preliminarily determined that there are no conditions of use presenting an unreasonable risk to  
1101 environmental receptors (see details in Section 5.1).

1102 EPA has preliminarily determined that the following conditions of use of asbestos present an  
1103 unreasonable risk of injury to health to workers (including, in some cases, occupational non-users) or to  
1104 consumers (including, in some cases, bystanders). The details of these determinations are presented in  
1105 Section 5.2.<sup>2</sup>

1106

**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

1107

**Consumer Uses and Disposal that Present an Unreasonable Risk to Health**

- Aftermarket Automotive Asbestos-Containing Brakes/Linings
- Other Asbestos-Containing Gaskets

1108

1109 EPA has determined that the following conditions of use of asbestos do not present an unreasonable risk  
1110 of injury to health. The details of these determinations are presented in section 5.2.

1111

1112

1113

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<sup>2</sup> Although EPA has identified both industrial and commercial uses here for purposes of distinguishing scenarios in this analysis, the Agency interprets the authority over “any manner or method of commercial use” under TSCA section 6(a)(5) to reach both.

**Conditions of Use that Do Not Present an Unreasonable Risk to Health**

- Import of asbestos and asbestos-containing products
- Distribution of asbestos-containing products
- Disposal of asbestos-containing sheet gaskets processed and/or used in chemical production
- Import, use, distribution and disposal of asbestos-containing brakes for the specialized and large National Aeronautics and Space Administration (NASA) transport plane (“Super Guppy”)

1114

# 1 INTRODUCTION

---

This document presents the draft risk evaluation for asbestos under the Frank R. Lautenberg Chemical Safety for the 21st Century Act which amended the Toxic Substances Control Act, the Nation's primary chemicals management law, in June 2016.

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](#). EPA has received information and comments specific to individual chemicals and of a more general nature relating to various aspects of the risk evaluation process, technical issues, and the regulatory and statutory requirements. EPA has considered comments and information received at each step in the process and factored in the information and comments as the Agency deemed appropriate and relevant including comments on the published problem formulation for asbestos. Thus, in addition to any new comments on the draft risk evaluation, the public should re-submit or clearly identify at this point any previously filed comments, modified as appropriate, that are relevant to this risk evaluation and that the submitter feels have not been addressed. EPA does not intend to further respond to comments submitted prior to the publication of this draft risk evaluation unless they are clearly identified in comments on this draft risk evaluation.

As per EPA's final rule, [Procedures for Chemical Risk Evaluation Under the Amended Toxic Substances Control Act](#) (82 Fed. Reg. 33726 (July 20, 2017)), this draft risk evaluation will be subject to both public comment and peer review. EPA is providing 60 days for public comment on this draft risk evaluation, including the submission of any additional information that might be relevant to the science underlying the risk evaluation and the outcome of the systematic review associated with asbestos. This satisfies TSCA (15 U.S.C. 2605(b)(4)(H)), which requires EPA to provide public notice and an opportunity for comment on a draft risk evaluation prior to publishing a final risk evaluation.

Peer review will be conducted in accordance with EPA's regulatory procedures for chemical risk evaluations, including using the [EPA Peer Review Handbook](#) and other methods consistent with Section 26 of TSCA (*See* 40 CFR 702.45). 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. Peer review will therefore address aspects of the underlying science as outlined in the charge to the peer review panel such as hazard assessment, assessment of dose-response, exposure assessment, and risk characterization. EPA believes peer reviewers will be most effective in this role if they receive the benefit of public comments on draft risk evaluations prior to peer review. For this reason, and consistent with standard Agency practice, the public comment period will precede peer review on this draft risk evaluation. The final risk evaluation may change in response to public comments received on the draft risk evaluation and/or in response to peer review, which itself may be informed by public comments. EPA will respond to public and peer review comments received on the draft risk evaluation and will explain changes made to the draft risk evaluation for asbestos in response to those comments in the final risk evaluation.

The PF identified the conditions of use (COUs) and presented three conceptual models and an analysis plan. Based on EPA's analysis of the COU, physical-chemical and fate properties, environmental releases, and exposure pathways, the PF preliminarily concluded that further analysis was necessary for

1159 exposure pathways to workers, consumers, and surface water, based on a qualitative assessment of the  
1160 physical-chemical properties and fate of asbestos in the environment. However, during development of  
1161 the PF, EPA was still in the process of identifying potential asbestos water releases for the COUs. After  
1162 the PF was released, EPA continued to search EPA databases as well as the literature and either engaged  
1163 in a dialogue with industries or reached out for a dialogue to shed light on potential releases to water.  
1164 The reasonably available information indicated that there were surface water releases of asbestos;  
1165 however, not all releases are subject to reporting (e.g., effluent guidelines) or are applicable (e.g.,  
1166 friability). Based on the reasonably available information in the published literature, provided by  
1167 industries using asbestos, and reported in EPA databases, there is minimal or no releases of asbestos to  
1168 surface water associated with the COUs in this risk evaluation. Therefore, EPA concludes there is no  
1169 unreasonable risk to aquatic or sediment-dwelling environmental organisms (See Section 4.1).

1170  
1171 Asbestos has been regulated by various Offices of EPA for years. The risk evaluation (RE) for asbestos  
1172 has posed some unique challenges to OPPT. Unlike the other nine chemicals that are part of the “First  
1173 10” risk evaluations under the Lautenberg Act of 2016, asbestos is a naturally occurring fiber, which  
1174 poses its own set of issues, including defining: (1) the COU (by asbestos fiber type); (2) the appropriate  
1175 inhalation unit risk (IUR) value to use for the hazard/dose-response process; and (3) the appropriate  
1176 exposure assessment measures.

1177  
1178 The COUs in this draft risk evaluation for asbestos are limited to only a few categories of ongoing uses,  
1179 and chrysotile is the only type of asbestos fiber identified for these COUs<sup>3</sup>. Ongoing uses of asbestos in  
1180 the U.S. were difficult to identify despite using an extensive list of resources. To determine the COU of  
1181 asbestos and inversely, activities that do not qualify as COUs, EPA conducted extensive research and  
1182 outreach. EPA identified activities that include import of raw asbestos, used solely in the chlor-alkali  
1183 industry, and import and use of asbestos-containing products. The COUs included in this draft risk  
1184 evaluation that EPA considers to be known, intended, or reasonably foreseen are the manufacture/  
1185 import, use, distribution and disposal of asbestos diaphragms, sheet gaskets, other gaskets, oilfield brake  
1186 blocks, aftermarket automotive brakes/linings, and other vehicle friction products and the processing of  
1187 asbestos diaphragms and sheet gaskets. Some of these COUs are very specialized. Since the PF, three  
1188 uses were removed from the scope of the RE based on further investigation (see Section 1.4.3); these  
1189 uses include woven products, cement products, and packings (from “gaskets and packings”). EPA  
1190 determined that there is no evidence to support that asbestos-containing woven products, cement  
1191 products, or packings are COUs of asbestos. These three uses were added to the Significant New Use  
1192 Rule (SNUR) for asbestos (40 CFR 721.11095). The Asbestos SNUR is an Agency action  
1193 complementary to this risk evaluation and taken under TSCA section 5 to prohibit any manufacturing  
1194 (including import) or processing for discontinued uses of asbestos from restarting without EPA having  
1195 an opportunity to evaluate them to determine risks to health or the environment and take any necessary  
1196 regulatory action, which may include a prohibition. The final asbestos SNUR ensures that any  
1197 manufacturing (including import) and processing for all discontinued uses and types of asbestos that are  
1198 not already banned are restricted from re-entering the U.S. marketplace without notification to EPA and  
1199 review and any necessary regulatory action by the Agency. Thus, should any person wish to

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<sup>3</sup> Please note that EPA continues to review the recent court decision in *Safer Chemicals Healthy Families v. EPA*, Nos. 17-72260 et al. (9th Cir. 2019). This draft risk evaluation does not reflect consideration of legacy uses and associated disposal as a result of that decision. EPA is still seeking public comment on and peer review of this version, however. EPA intends to consider legacy uses and associated disposal in a supplemental scope document and supplemental risk evaluation.

1200 manufacture, import, or process asbestos for an activity that is not a COU identified in this document or  
1201 subject to an existing ban, then EPA would review the risk of the activity associated with such a use in  
1202 accordance with TSCA section 5.  
1203

1204 During the investigation of the COUs, EPA also determined that asbestos is no longer mined in the U.S.,  
1205 and that only chrysotile asbestos is being imported. The other five forms of asbestos identified for this  
1206 risk evaluation, including crocidolite (riebeckite), amosite (cummingtonite-grunerite), anthophyllite,  
1207 tremolite or actinolite, are no longer manufactured, imported, or processed in the United States and are  
1208 also now subject to the SNUR. After EPA confirmed that chrysotile asbestos is the only type of asbestos  
1209 still being imported into the U.S. either in raw form or in products, EPA developed a chrysotile IUR<sup>4</sup> to  
1210 be used in the RE. The IUR for asbestos developed in 1988 was based on 14 epidemiologic studies that  
1211 included occupational exposure to chrysotile, amosite, or mixed-mineral exposures (chrysotile, amosite,  
1212 crocidolite). As a naturally occurring mineral, chrysotile can co-occur with other minerals, including  
1213 amphibole forms of asbestos. Trace amounts of these minerals may remain in chrysotile as it is used in  
1214 commerce. This commercial chrysotile, rather than theoretically “pure” chrysotile, is therefore the  
1215 substance of concern for this assessment. The epidemiologic studies available for risk evaluation all  
1216 include populations exposed to commercial chrysotile asbestos, which may contain small, but variable  
1217 amounts of amphibole asbestos. Because the only form of asbestos imported, processed, or distributed  
1218 for use in the United States today is chrysotile, studies of populations exposed only to chrysotile provide  
1219 the most informative data for the purpose of updating the TSCA risk estimates for the COUs for  
1220 asbestos in this document. EPA will consider legacy uses and associated disposal in subsequent  
1221 supplemental documents.  
1222

1223 Related to the focus on chrysotile asbestos is the method of identifying asbestos in studies used to  
1224 develop the IUR. The IUR is based on fiber counts made by phase contrast microscopy (PCM) and  
1225 should not be applied directly to measurements made by other analytical techniques. PCM  
1226 measurements made in occupational environments were used in the studies used to support the  
1227 derivation of the chrysotile IUR. PCM detects only fibers longer than 5 µm and >0.4 µm in diameter,  
1228 while transmission electron microscopy (TEM), often found in environmental monitoring  
1229 measurements, can detect much smaller fibers. In developing a PCM-based IUR in this risk evaluation,  
1230 several TEM papers modeling risk of lung cancer were found, but because there was no TEM-based  
1231 modeling of mesothelioma mortality, TEM data could not be used to derive a TEM-based IUR.  
1232

1233 EPA stated in the PF that the asbestos RE would focus on epidemiological data on lung cancer and  
1234 mesothelioma. The 1988 IUR identified asbestos as a carcinogen causing both lung cancer and  
1235 mesothelioma from inhalation exposures and derived a unit risk to address both cancers (for all TSCA  
1236 Title II fiber types – see Section 1.1). EPA is not aware of any other chrysotile-specific IUR for the  
1237 asbestos types included in this RE or any other risk-based values having been estimated for other types  
1238 of cancer for asbestos by either EPA or other government agencies. For the derivation of a chrysotile  
1239 asbestos IUR, epidemiological studies on mesothelioma and lung cancer in cohorts of workers using  
1240 chrysotile in commerce were identified to inform the estimation of an exposure-response function. Over  
1241 24,000 studies were initially identified for consideration during the Systematic Review process to  
1242 determine whether the IUR needed to be updated. This large number of studies posed its own unique  
1243 challenges, including development of data quality review standards specific to asbestos.

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<sup>4</sup> Inhalation Unit risk (IUR) is typically defined as a plausible upper bound on the estimate of cancer risk per µg/m<sup>3</sup> air breathed for 70 years. For asbestos, the IUR is expressed as cancer risk per fibers/cc (in units of the fibers as measured by PCM).

1244  
1245 EPA derived an IUR for chrysotile asbestos using five epidemiological study cohorts analyzing lung  
1246 cancer and mesothelioma. EPA derived cancer-specific unit risks using lifetables. Different modeling  
1247 choices and combinations of cancer-specific unit risks yielded candidate IUR values ranging from 0.08  
1248 to 0.33 per f/cc, indicating low model-based uncertainty. The IUR chosen is 0.16 per f/cc and it was  
1249 applied to the COUs to calculate lifetime risks for workers and consumers.

1250  
1251 EPA estimated risks for workers, occupational non-users (ONUs), consumers (do-it-yourself [DIY]  
1252 mechanics) and bystanders for the COUs identified. Inhalation exposure scenarios were used to estimate  
1253 risks for cancer based on the EPA-derived IUR for chrysotile asbestos. This assessment is unique with  
1254 respect to the timing of exposure relative to the cancer outcome as the time since first exposure plays a  
1255 dominant role in modeling risk. Occupational exposures assumed 240 days/year for 8-hour workdays for  
1256 40 years starting at 16 years old; with other starting ages and exposure durations also presented.  
1257 Occupational exposures for chlor-alkali and sheet gasket workers and ONUs were based on monitoring  
1258 data supplied by companies performing the work. Consumer exposures were based on study data  
1259 provided in the literature for gasket replacement and brakes. Consumer exposures assumed that DIY  
1260 mechanics for both COUs changed brakes or gaskets once every three years (the task taking three hours)  
1261 over a lifetime and that exposures lingered between the episodic exposures.

1262  
1263 In this draft risk evaluation, Section 1 presents the basic physical-chemical characteristics of asbestos, as  
1264 well as a background on regulatory history, COUs, and conceptual models, with particular emphasis on  
1265 any changes since the publication of the PF. This section also includes a discussion of the systematic  
1266 review process utilized in this draft risk evaluation. Section 2 provides a discussion and analysis of the  
1267 exposures, both health and environmental, that can be expected based on the COUs for asbestos. Section  
1268 3 discusses environmental and health hazards of asbestos. Section 4 presents the risk characterization,  
1269 where EPA integrates and assesses reasonably available information on health and environmental  
1270 hazards and exposures, as required by TSCA (15 U.S.C. 2605(b)(4)(F)). This section also includes a  
1271 discussion of any uncertainties and how they impact the draft risk evaluation. Section 5 presents EPA's  
1272 proposed determination of whether the chemical presents an unreasonable risk under the COU, as  
1273 required under TSCA (15 U.S.C. 2605(b)(4)).

## 1274 1275 **1.1 Physical and Chemical Properties and Environmental Fate**

1276 Asbestos is a “generic commercial designation for a group of naturally occurring mineral silicate fibers  
1277 of the serpentine and amphibole series” ([IARC, 2012](#)). The Chemical Abstract Service (CAS) definition  
1278 of asbestos is “a grayish, non-combustible fibrous material. It consists primarily of impure magnesium  
1279 silicate minerals.” The general CAS Registry Number (CASRN) of asbestos is 1332-21-4; this is the  
1280 only asbestos CASRN on the TSCA Inventory. However, other CASRNs are available for specific fiber  
1281 types.

1282  
1283 TSCA Title II (added to TSCA in 1986), Section 202 defines asbestos as the “asbestiform varieties of  
1284 six fiber types – chrysotile (serpentine), crocidolite (riebeckite), amosite (cummingtonite-grunerite),  
1285 anthophyllite, tremolite or actinolite.” The latter five fiber types are amphibole varieties. In the *Problem*  
1286 *Formulation of the Risk Evaluation for Asbestos* (EPA-HQ-OPPT-2016-0736-0131) ([U.S. EPA, 2018d](#)),  
1287 physical and chemical properties of all six fiber types were presented. As discussed in more detail in  
1288 Section 1.4, this risk evaluation has focused on chrysotile given EPA's knowledge of the COUs of  
1289 asbestos, and EPA will consider legacy uses and associated disposal in subsequent supplemental  
1290 documents. Table 1-1. lists the physical/chemical properties for the six fiber types of asbestos. As with  
1291 all silicate minerals, the basic building blocks of asbestos fibers are silicate tetrahedra  $[\text{SiO}_4]^{4-}$  where

1292 four oxygen atoms are covalently bound to the central silicon. These tetrahedra occur as sheets [Si<sub>4</sub>O<sub>10</sub>]  
 1293 in chrysotile. In the case of chrysotile, an octahedral brucite layer having the formula [Mg<sub>6</sub>O<sub>4</sub>(OH)<sub>8</sub>] is  
 1294 intercalated between each silicate tetrahedral sheet.  
 1295  
 1296

**Table 1-1. Physical and Chemical Properties of Asbestos Fiber Types<sup>a</sup>**

	Chrysotile	Amosite	Crocidolite	Asbestiform Tremolite	Asbestiform Anthophyllite	Asbestiform Actinolite
Essential composition	Mg silicate with some water	Fe, Mg silicate	Na, Fe silicate with some water	Ca, Mg silicate with some water	Mg silicate with iron	Ca, Mg, Fe silicate with some water
Color	White, gray, green, yellowish	Ash gray, greenish or brown	Lavender, blue, greenish	Gray-white, greenish, yellowish, bluish	Grayish white, also brown-gray or green	Greenish
Luster	Silky	Vitreous to pearly	Silky to dull	Silky	Vitreous to pearly	Silky
Surface area <sup>b</sup> (m <sup>2</sup> /g)	13.5-22.4	2.25-7.10	4.62-14.80	No data	No data	No data
Hardness (Mohs)	2.5-4.0	5.5-6.0	4.0	5.5	5.5-6.0	6.0
Specific gravity	2.4-2.6	3.1-3.25	3.2-3.3	2.9-3.2	2.85-3.1	3.0-3.2
Optical properties	Biaxial positive parallel extinction	Biaxial positive parallel extinction	Biaxial extinction inclined	Biaxial negative extinction inclined	Biaxial positive extinction parallel	Biaxial negative extinction inclined
Refractive index	1.50-1.55	1.64	1.7 pleochroic	1.61	1.61	1.63 weakly pleochroic
Flexibility	High	Good	Good	Poor, generally brittle	Poor	Poor
Texture	Silky, soft to harsh	Coarse but somewhat pliable	Soft to harsh	Generally harsh, sometimes soft	Harsh	Harsh
Spinnability	Very good	Fair	Fair	Generally poor, some are spinnable	Poor	Poor
Tensile strength (MPa)	550-690 (80,000-100,000 lb/in <sup>2</sup> )	110-620 (16,000-90,000 lb/in <sup>2</sup> )	690-2100 (100,000-300,000 lb/in <sup>2</sup> )	7-60 (1,000-8,000 lb/in <sup>2</sup> )	≤30 (≤ 4,000 lb/in <sup>2</sup> )	≤7 (≤ 1,000 lb/in <sup>2</sup> )
Fiber size, median true diameter (μm) <sup>c</sup>	0.06 <sup>e</sup>	0.26	0.09	No data	No data	No data



	Chrysotile	Amosite	Crocidolite	Asbestiform Tremolite	Asbestiform Anthophyllite	Asbestiform Actinolite
Fiber size, median true length (µm) <sup>d</sup>	0.55 <sup>e</sup>	2.53	1.16	No data	No data	No data
Resistance to: Acids	Weak, undergoes fairly rapid attack	Fair, slowly attacked	Fair	Fair	Fair	Fair
Bases	Very good	Good	Good	Good	Very good	Fair
Zeta potential (mV) <sup>d</sup>	+13.6 to +54	-20 to -40	-32	No data	No data	No data
Decomposition temperature (°C)	600-850	600-900	400-900	950-1,040	No data	No data

<sup>a</sup> [Badollet \(1951\)](#)  
<sup>b</sup> [Addison et al. \(1966\)](#)  
<sup>c</sup> [Hwang \(1983\)](#)  
<sup>d</sup> [Virta \(2011\)](#)  
<sup>e</sup> The reported values for diameter and length are median values. As reported in Virta (2011), “Industrial chrysotile fibers are aggregates...that usually exhibit diameters from 0.1 to 100 µm; their lengths range from a fraction of a millimeter to several centimeters, although most chrysotile fibers used are < 1 cm.”

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## 1.2 Uses and Production Volume

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The only form of asbestos manufactured (including imported), processed, or distributed for use in the United States today is chrysotile. The United States Geological Survey (USGS) estimated that 750 metric tons of raw chrysotile asbestos were imported into the U.S. in 2018 ([USGS, 2019](#)). This raw asbestos is used exclusively by the chlor-alkali industry and imported amounts tend to range between 300 and 800 metric tons during a given year ([USGS, 2019](#)).

In addition to the use of raw imported chrysotile asbestos by the chlor-alkali industry, EPA is also aware of imported asbestos-containing products; however, the import volumes of those products are not fully known. 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. More information about the uses of asbestos and EPA’s methodology for identifying COUs is provided in Section 1.4.1 of this document. EPA will consider legacy uses and associated disposal in subsequent supplemental documents.

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## 1.3 Regulatory and Assessment History

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EPA conducted a search of existing domestic and international laws, regulations and assessments pertaining to asbestos. EPA compiled this summary from data available from federal, state, international and other government sources, as cited in 7Appendix A. EPA evaluated and considered the impact of at least some of these existing laws and regulations to determine what, if any further analysis might be necessary as part of the risk evaluation. Consideration of the nexus between these regulations and the TSCA COU evaluated in this risk evaluation were developed and described in the PF document.

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***Federal Laws and Regulations***

Asbestos is subject to federal statutes or regulations, other than TSCA, that are implemented by other offices within EPA and/or other federal agencies/departments. A summary of federal laws, regulations and implementing authorities is provided in Appendix A.1.

***State Laws and Regulations***

Asbestos is subject to statutes or regulations implemented by state agencies or departments. A summary of state laws, regulations and implementing authorities is provided in Appendix A.2.

***Laws and Regulations in Other Countries and International Treaties or Agreements***

Asbestos is subject to statutes or regulations in countries other than the United States and/or international treaties and/or agreements. A summary of these laws, regulations, treaties and/or agreements is provided in Appendix A.3.

Table 1-2. Assessment History of Asbestos provides assessments related to asbestos conducted by other EPA Programs and other organizations. Depending on the source, these assessments may include information on COU, hazards, exposures and potentially exposed or susceptible subpopulations.

**Table 1-2. Assessment History of Asbestos**

<b>Authoring Organization</b>	<b>Assessment</b>
<b>EPA assessments</b>	
EPA, Integrated Risk Information System (IRIS)	<a href="#">IRIS Assessment on Asbestos (1988b)</a>
EPA, Integrated Risk Information System (IRIS)	<a href="#">IRIS Assessment on Libby Amphibole Asbestos (2014c)</a>
EPA, Region 8	<a href="#">Site-Wide Baseline Ecological Risk Assessment, Libby Asbestos Superfund Site, Libby Montana (U.S. EPA, 2014b)</a>
EPA, Drinking Water Criteria Document	<a href="#">U.S. EPA Drinking Water Criteria Document for Asbestos (1985)</a>
EPA, Ambient Water Quality Criteria for Asbestos	<a href="#">Asbestos: Ambient Water Quality Criteria (1980)</a>
EPA, Final Rule (40 CFR Part 763)	<a href="#">Asbestos; Manufacture, Importation, Processing and Distribution in Commerce Prohibitions (1989)</a>
EPA, Asbestos Modeling Study	Final Report; Asbestos Modeling Study ( <a href="#">U.S. EPA, 1988a</a> )
EPA, Asbestos Exposure Assessment	<a href="#">Revised Report to support ABPO rule (1988)</a>
EPA, Nonoccupational Exposure Report	Revised Draft Report, Nonoccupational Asbestos Exposure ( <a href="#">Versar, 1987</a> )
EPA, Airborne Asbestos Health Assessment Update	<a href="#">Support document for NESHAP review (1986)</a>
<b>Other U.S.-based organizations</b>	

Authoring Organization	Assessment
National Institute for Occupational Safety and Health (NIOSH)	<a href="#">Asbestos Fibers and Other Elongate Mineral Particles: State of the Science and Roadmap for Research (2011)</a>
Agency for Toxic Substances and Disease Registry (ATSDR)	<a href="#">Toxicological Profile for Asbestos (2001a)</a>
National Toxicology Program (NTP)	<a href="#">Report on Carcinogens, Fourteenth Edition (2016)</a>
CA Office of Environmental Health Hazard Assessment (OEHHA), Pesticide and Environmental Toxicology Section	<a href="#">Public Health Goal for Asbestos in Drinking Water (2003)</a>
<b>International</b>	
International Agency for Research on Cancer (IARC)	<a href="#">IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Arsenic, Metals, Fibres, and Dusts. Asbestos (Chrysotile, Amosite, Crocidolite, Tremolite, Actinolite, and Anthophyllite) (2012)</a>
World Health Organization (WHO)	<a href="#">World Health Organization (WHO) Chrysotile Asbestos (2014)</a>

## 1.4 Scope of the Evaluation

### 1.4.1 Refinement of Asbestos Fiber Type Considered in this Risk Evaluation

During risk evaluation, EPA determined that the only form of asbestos manufactured (including imported), processed, or distributed for use in the United States today is chrysotile. The other five forms of asbestos are no longer manufactured, imported, or processed in the United States and are now subject to a significant new use rule (SNUR) that requires notification of and review by the Agency should any person wish to pursue manufacturing, importing, or processing crocidolite (riebeckite), amosite (cummingtonite-grunerite), anthophyllite, tremolite or actinolite (either in raw form or as part of articles) for any use (40 CFR 721.11095). Therefore, under the final asbestos SNUR, EPA will be made aware of manufacturing, importing, or processing for any intended use of the other forms of asbestos. If EPA finds upon review of the Significant New Use Notice (SNUN) that the significant new use presents or may present an unreasonable risk (or if there is insufficient information to permit a reasoned evaluation of the health and environmental effects of the significant new use), then EPA would take action under TSCA section 5(e) or (f) to the extent necessary to protect against unreasonable risk.

Data from USGS indicates that the asbestos being imported for chlor-alkali plants is all chrysotile. Virta (2006) notes that when South Africa closed its amosite and crocidolite mines (in 1992 and 1997 respectively), worldwide production of amosite and crocidolite ceased. Virta (2006) concluded that almost all of the world’s production of asbestos is chrysotile and that “[s]mall amounts, probably less than a few thousand tons, of actinolite, anthophyllite, and tremolite asbestos are produced for local use in countries such as India, Pakistan, and Turkey.”

1364 Chrysotile is the prevailing form of asbestos currently mined worldwide, therefore, commercially  
1365 available products fabricated overseas are made with chrysotile. Any asbestos being imported into the  
1366 U.S. in articles for the COUs EPA has identified is believed to be chrysotile. Based on EPA's  
1367 determination that chrysotile is the only form of asbestos imported into the U.S. as both raw form and as  
1368 contained in articles, EPA is performing a quantitative evaluation for chrysotile asbestos only in this risk  
1369 evaluation. EPA will consider legacy uses and associated disposal in subsequent supplemental  
1370 documents.  
1371

#### 1372 **1.4.2 Refinement of Evaluation of Releases to Surface Water**

1373 EPA did not evaluate the risk to aquatic species from exposure to surface water in its PF. During the PF  
1374 phase of the Risk Evaluation, EPA was still in the process of identifying potential asbestos water  
1375 releases for the TSCA COUs. After the PF was released, EPA continued to search EPA databases as  
1376 well as the literature and attempted to contact industries to shed light on potential releases to water. The  
1377 available information indicated that there were surface water releases of asbestos; however, not all  
1378 releases are subject to reporting (e.g., effluent guidelines) or are applicable (e.g., friability). Based on the  
1379 reasonably available information in the published literature, provided by industries using asbestos, and  
1380 reported in EPA databases, there is minimal or no releases of asbestos to surface water associated with  
1381 the COUs that EPA is evaluating in this risk evaluation (see Appendix D).  
1382

#### 1383 **1.4.3 Conditions of Use Included in the Risk Evaluation**

1384 TSCA § 3(4) defines the COU as “the circumstances, as determined by the Administrator, under which  
1385 a chemical substance is intended, known, or reasonably foreseen to be manufactured, processed,  
1386 distributed in commerce, used, or disposed of.” Throughout the scoping (2017c), PF (2018d), and risk  
1387 evaluation stages, EPA identified and verified the uses of asbestos.  
1388

1389 To determine the COU of asbestos and inversely, activities that do not qualify as COU, EPA conducted  
1390 extensive research and outreach. This included EPA's review of published literature and online  
1391 databases including the most recent data available from EPA's Chemical Data Reporting program  
1392 (CDR), Safety Data Sheets (SDSs), the U.S. Geological Survey's Mineral Commodities Summary and  
1393 Minerals Yearbook, the U.S. International Trade Commission's DataWeb and government and  
1394 commercial trade databases. EPA also reviewed company websites of potential manufacturers,  
1395 importers, distributors, retailers, or other users of asbestos. EPA also received comments on the *Scope of*  
1396 *the Risk Evaluation for Asbestos* (EPA-HQ-OPPT-2016-0736-0086, 2017c) that were used to inform  
1397 the COU. In addition, prior to the June 2017 publication of the scope document, EPA convened  
1398 meetings with companies, industry groups, chemical users, and other stakeholders to aid in identifying  
1399 COU and verifying COU identified by EPA.  
1400

1401 EPA has removed from the risk evaluation any activities that EPA has concluded do not constitute COU  
1402 – for example, because EPA has insufficient information to find certain activities are circumstances  
1403 under which the chemical is actually “intended, known, or reasonably foreseen to be manufactured,  
1404 processed, distributed in commerce, used or disposed of.”  
1405

1406 Since the PF document was published in June 2018 (U.S. EPA, 2018d), EPA has further refined the  
1407 COU of asbestos for risk evaluation. The activities that EPA has determined are not COU in this  
1408 document are packings, woven products, and cement products. Asbestos “packings” are listed under a  
1409 broader category of “gaskets, packings, and seals” and more detailed data revealed that only imported  
1410 gaskets, not packings, contain asbestos. EPA concluded that “woven and knitted fabrics,” which are

1411 reported in USGS’s 2016 Minerals Yearbook under Harmonized Tariff Schedule (HTS) code  
1412 6812.99.0004 are misreported (see Appendix C for further explanation). Upon further review, EPA  
1413 determined that woven products are not a COU but are precursors to asbestos-containing products or  
1414 physical attributes of the asbestos. EPA contacted potential foreign exporters of asbestos woven  
1415 products and asbestos cement products, and these foreign companies informed EPA that they do not  
1416 have customers in the United States ([U.S. EPA, 2018b, c](#)). The Agency has not found any evidence to  
1417 suggest that woven products (other than those that are already covered under a distinct COU such as  
1418 brake blocks used in draw works) or cement products imported into the United States contain asbestos.  
1419 Furthermore, EPA discussed the use of asbestos in cement pipe with a trade organization, who indicated  
1420 that domestic production, importation, or distribution for such a use is neither known to be currently  
1421 ongoing nor foreseeable ([AWWA, 2019](#)). Based on outreach activity and lack of evidence, EPA does  
1422 not believe asbestos packings, asbestos woven products (that are not already covered under a separate  
1423 and ongoing COU), or asbestos cement products are COU of asbestos in the United States, and  
1424 therefore, packings, woven products, and cement products are no longer under consideration for this risk  
1425 evaluation and are now subject to the asbestos SNUR under TSCA section 5. Table 1-3. represents the  
1426 activities that have been removed from the scope of the risk evaluation since the PF document was  
1427 published in June 2018. EPA will consider legacy uses and associated disposal in subsequent  
1428 supplemental documents.

1429 **Table 1-3. Categories Determined Not to be Conditions of Use After Problem Formulation**

Product Category	Example
Asbestos Cement Products	Cement pipe
Asbestos Woven Products	Imported Textiles
Asbestos Packings	Dynamic or mechanical seals

1431  
1432 EPA has verified that U.S. automotive manufacturers are not installing asbestos brakes on new cars for  
1433 domestic distribution or use. Therefore, this use will only be evaluated in occupational settings for one  
1434 use that EPA identified for cars that are manufactured with asbestos-containing brakes in the U.S. but  
1435 are exported and not sold in the U.S. However, removing and installing asbestos brakes in older vehicles  
1436 by both professional mechanics and DIY consumers will be evaluated (see Table 1-4. below). The only  
1437 use that was identified for the “other gaskets” category was for a specific utility vehicle (UTV) that has  
1438 an asbestos-containing gasket in its exhaust system.

1439  
1440 Based on the above discussion, the COUs that are included in this risk evaluation are described in Table  
1441 1-4.

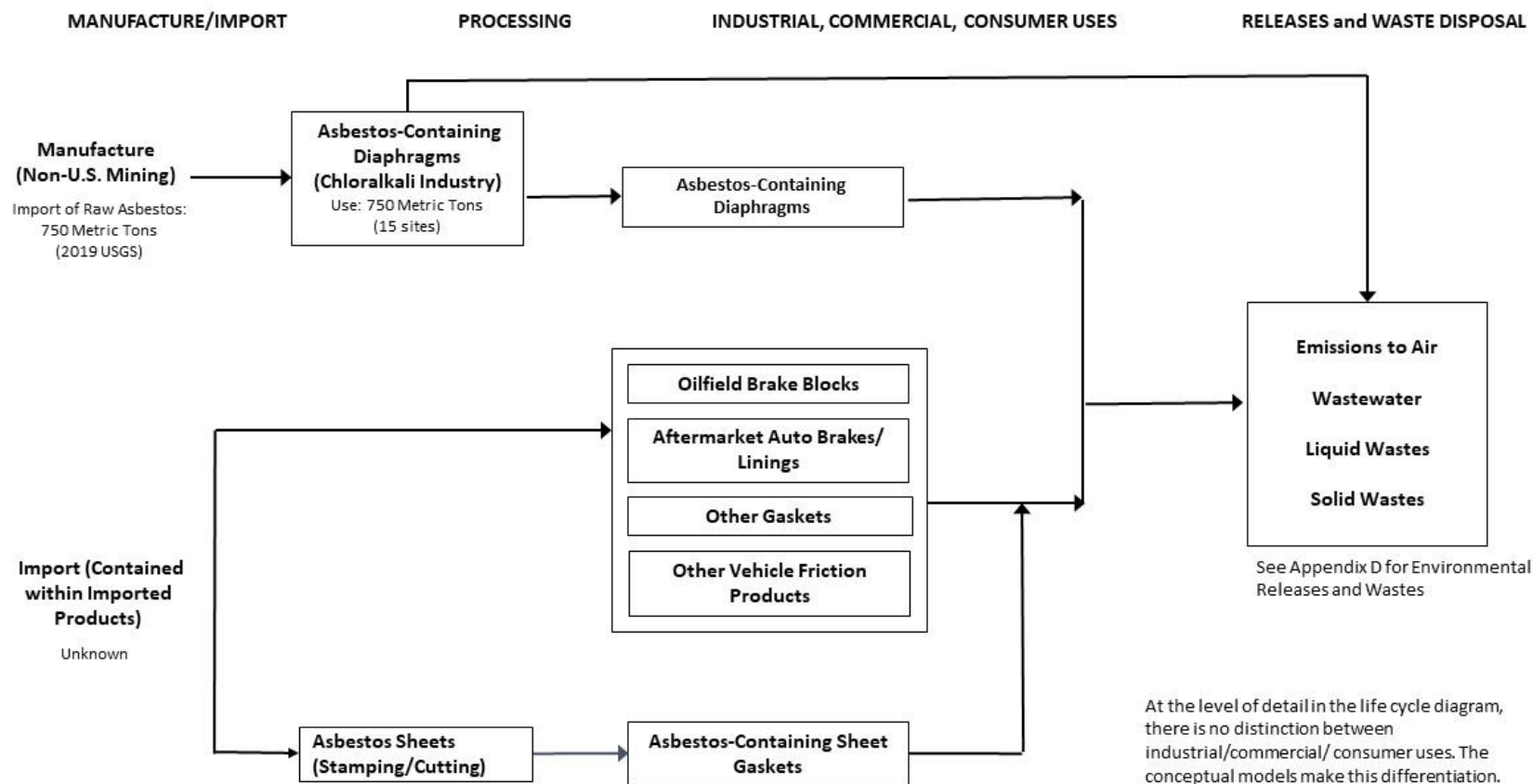
1442  
1443 The life cycle diagram is presented in Figure 1-1.  
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1446

**Table 1-4. Categories of Conditions of Use Included in this Risk Evaluation**

<b>Product Category</b>	<b>Example</b>
Asbestos Diaphragms	Chlor-alkali Industry
Sheet Gaskets	Chemical Production
Oilfield Brake Blocks	Oil Industry
Aftermarket Automotive Brakes/Linings	Foreign aftermarket brakes sold online
Other Vehicle Friction Products	Brakes installed in exported cars
Other Gaskets	Utility Vehicles

1447  
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1449

1450 **Figure 1-1. Asbestos Life Cycle Diagram**

1451 The life cycle diagram depicts the COUs that have been assessed in this risk evaluation. It has been updated to reflect the removal from the PF  
 1452 of woven products, cement products, and packing (see Section 1.4.3) as well as using the 2018 import volume of raw asbestos.

1453

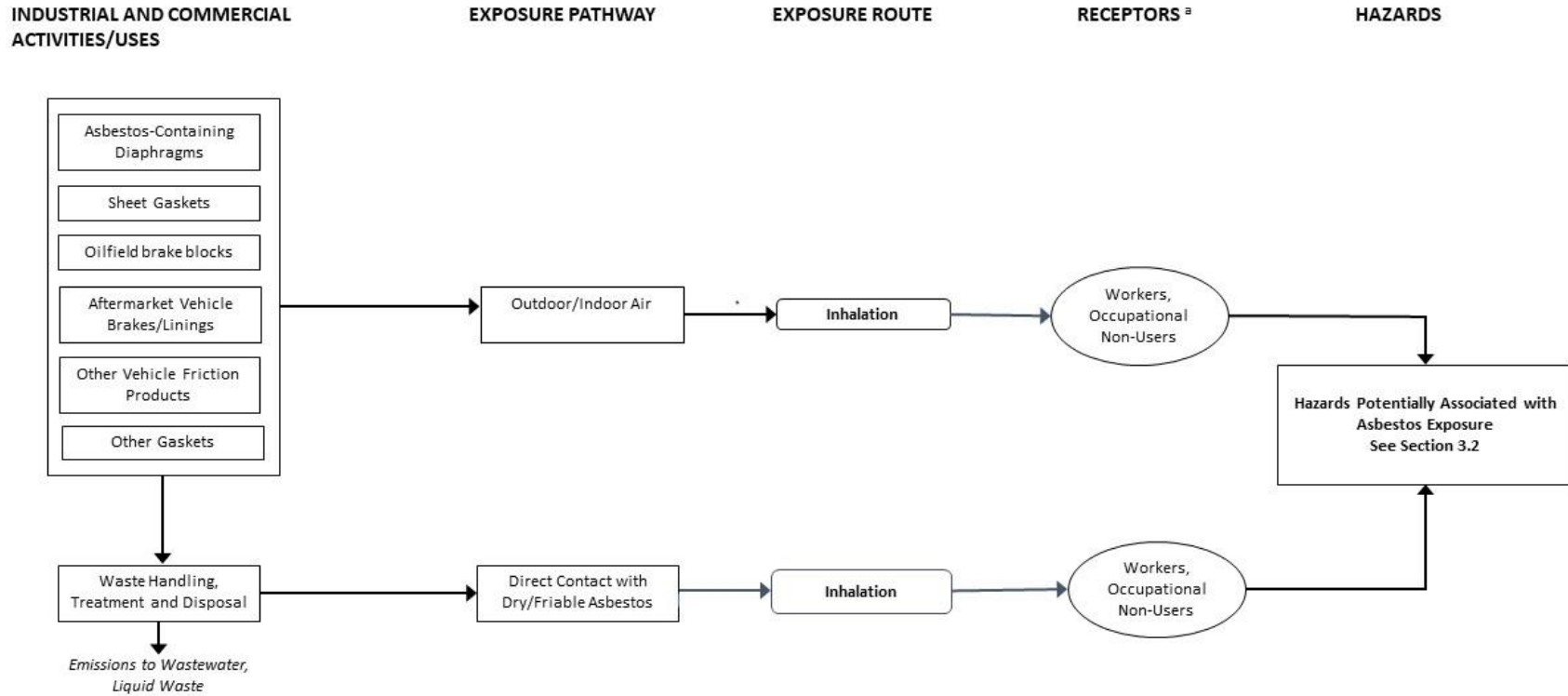
#### 1.4.4 Conceptual Models

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The conceptual models have been modified to reflect the refined COUs of asbestos described in Section 1.4.1. Figure 1-2. and Figure 1-3 present the conceptual models for industrial and commercial uses and consumer uses, respectively. The asbestos conceptual model for environmental releases and wastes from the refined COUs was removed and is discussed in Releases and Exposure to the Environment Supplementary Information Appendix D since it is not being considered in the RE. This was discussed in the Introduction and further discussed in Section 1.4.3.



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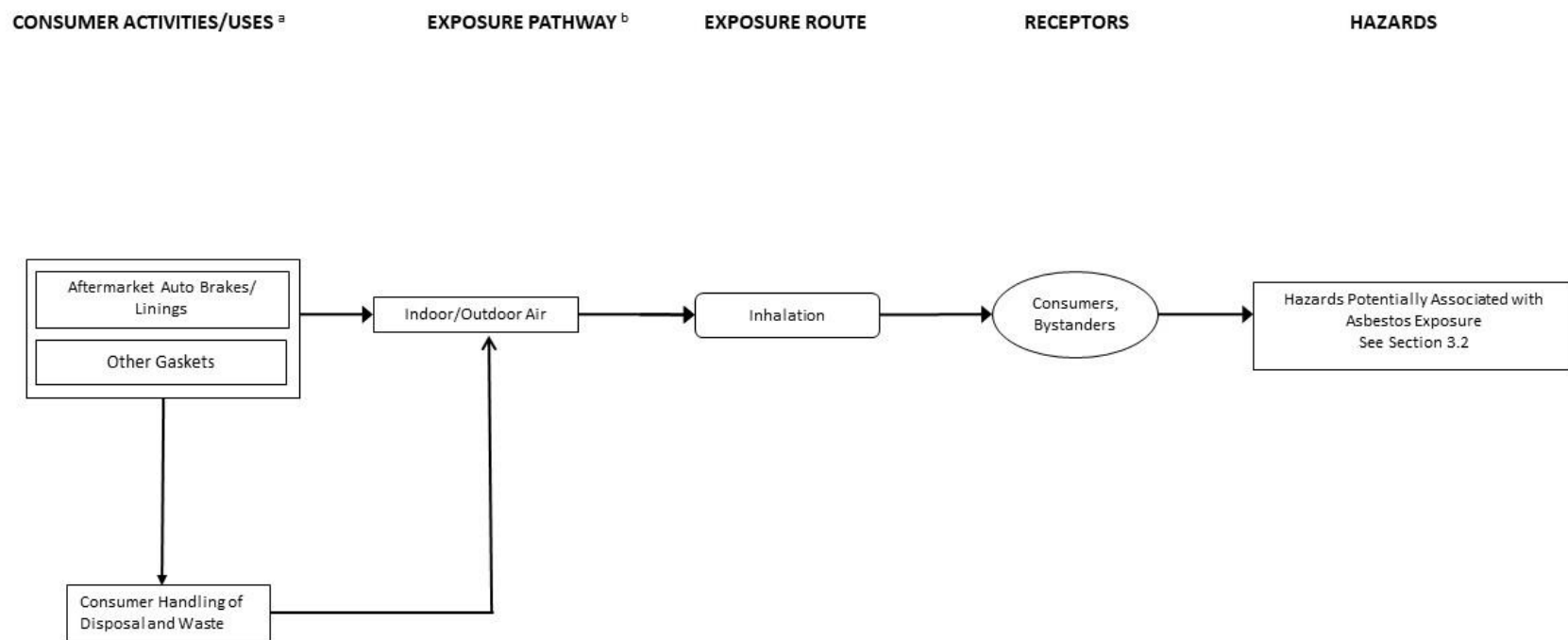


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**Figure 1-2. Asbestos Conceptual Model for Industrial and Commercial Activities and Uses: Potential Exposures and Hazards**

The conceptual model presents the exposure pathways, exposure routes and hazards to human receptors from industrial and commercial activities and uses of asbestos.

<sup>a</sup> Receptors include PESS.



1469

1470 **Figure 1-3. Asbestos Conceptual Model for Consumer Activities and Uses: Potential Exposures and Hazards**

1471 <sup>a</sup>Woven products were removed from this model after the PF was published. Upon further review, EPA determined that woven products are  
1472 not a COU but are precursors to asbestos-containing products or physical attributes of the asbestos. Utility vehicle gaskets were added during  
1473 RE.

1474 <sup>b</sup>Products may be used during indoor and outdoor activities.

1475 <sup>c</sup>Receptors include PESS.

1476

## 1.5 Systematic Review

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TSCA requires EPA to use scientific information, technical procedures, measures, methods, protocols, methodologies and models consistent with the best available science and base decisions under Section 6 on the weight of scientific evidence. Within the TSCA risk evaluation context, the weight of the scientific evidence is defined as “a systematic review method, applied in a manner suited to the nature of the evidence or decision, that uses a pre-established protocol to comprehensively, objectively, transparently, and consistently identify and evaluate each stream of evidence, including strengths, limitations, and relevance of each study and to integrate evidence as necessary and appropriate based upon strengths, limitations, and relevance” (40 C.F.R. 702.33).

To meet the TSCA 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](#)). The process complements the risk evaluation process in that the data collection, data evaluation and data integration stages of the systematic review process are used to develop the exposure and hazard assessments based on reasonably available information. EPA defines “reasonably available information” to mean information that EPA possesses, or can reasonably obtain and synthesize for use in risk evaluations, considering the deadlines for completing the evaluation (40 CFR 702.33).

EPA is implementing systematic review methods and approaches within the regulatory context of the amended TSCA. Although EPA will make an effort to adopt as many best practices as practicable from the systematic review community, EPA expects modifications to the process to ensure that the identification, screening, evaluation and integration of data and information can support timely regulatory decision making under the aggressive timelines of the statute.

### 1.5.1 Data and Information Collection

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EPA planned and conducted a comprehensive literature search based on key words related to the different discipline-specific evidence supporting this risk evaluation (e.g., environmental fate and transport; engineering releases and occupational exposure; exposure to general population, consumers and environmental exposure, and environmental and human health hazard). EPA then developed and applied inclusion and exclusion criteria during the title and abstract screening to identify information potentially relevant for the risk evaluation process. The literature and screening strategy as specifically applied to asbestos is described in the *Strategy for Conducting Literature Searches for Asbestos: Supplemental Document to the TSCA Scope Document* ([EPA-HQ-OPPT-2016-0736](#)), and the results of the title and abstract screening process were published in the *Asbestos (CASRN 1332-21-4) Bibliography: Supplemental File for the TSCA Scope Document*, [EPA-HQ-OPPT-2016-0736](#)) ([U.S. EPA, 2017b](#)).

For studies determined to be on-topic (or relevant) after title and abstract screening, EPA conducted a full text screening to further exclude references that were not relevant to the risk evaluation. Screening decisions were made based on eligibility criteria documented in the form of the populations, exposures,

1516 comparators, and outcomes (PECO) framework or a modified framework.<sup>5</sup> Data sources that met the  
1517 criteria were carried forward to the data evaluation stage. The inclusion and exclusion criteria for full  
1518 text screening for asbestos are available in Appendix D of the *Problem Formulation of the Risk*  
1519 *Evaluation for Asbestos* ([U.S. EPA, 2018d](#)).

1520  
1521 Although EPA conducted a comprehensive search and screening process as described above, EPA made  
1522 the decision to leverage the literature published in previous assessments<sup>6</sup> when identifying relevant key  
1523 and supporting data<sup>7</sup> and information for developing the asbestos risk evaluation. This is discussed in the  
1524 [Strategy for Conducting Literature Searches for Asbestos: Supplemental Document to the TSCA Scope](#)  
1525 [Document](#) (EPA-HQ-OPPT-2016-0736). In general, many of the key and supporting data sources were  
1526 identified in the comprehensive *Asbestos Bibliography: Supplemental File for the TSCA Scope*  
1527 *Document* ([U.S. EPA, 2017a, b](#)). However, there were instances during the releases and occupational  
1528 exposure data search for which EPA missed relevant references that were not captured in the initial  
1529 categorization of the on-topic references. EPA found additional relevant data and information using  
1530 backward reference searching, which is a technique that will be included in future search strategies. This  
1531 issue is discussed in Section 4 of the [Application of Systematic Review for TSCA Risk Evaluations](#) ([U.S.](#)  
1532 [EPA, 2018a](#)). Other relevant key and supporting references were identified through targeted  
1533 supplemental searches to support the analytical approaches and methods in the asbestos risk evaluation  
1534 (e.g., to locate specific information for exposure modeling) or to identify new data and information  
1535 published after the date limits of the initial search.

1536  
1537 EPA used previous chemical assessments to quickly identify relevant key and supporting information as  
1538 a pragmatic approach to expedite the quality evaluation of the data sources, but many of those data  
1539 sources were already captured in the comprehensive literature search as explained above. EPA also  
1540 considered newer information on asbestos not taken into account by previous EPA chemical assessments  
1541 as described in the [Strategy for Conducting Literature Searches for Asbestos: Supplemental Document](#)  
1542 [to the TSCA Scope Document](#) (EPA-HQ-OPPT-2016-0736). EPA then evaluated the relevance and  
1543 quality of the key and supporting data sources, as well as newer information, instead of reviewing all the  
1544 underlying published information on asbestos. A comprehensive evaluation of all of the data and  
1545 information ever published for a substance such as asbestos would be extremely labor intensive and  
1546 could not be achieved considering the deadlines specified in TSCA Section 6(b)(4)(G) for conducting  
1547 risk evaluations.

1548  
1549 This pragmatic approach allowed EPA to maximize the scientific and analytical efforts of other  
1550 regulatory and non-regulatory agencies by accepting, for the most part, the relevant scientific knowledge  
1551 gathered and analyzed by others except for influential information sources that may have an impact on  
1552 the weight of the scientific evidence and ultimately the risk findings. The influential information (i.e.,  
1553 key/supporting) came from a smaller pool of sources subject to the rigor of the TSCA systematic review

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<sup>5</sup> A PESO statement was used during the full text screening of environmental fate and transport data sources. PESO stands for Pathways and Processes, Exposure, Setting or Scenario, and Outcomes. A RESO statement was used during the full text screening of the engineering and occupational exposure literature. RESO stands for Receptors, Exposure, Setting or Scenario, and Outcomes.

<sup>6</sup> Examples of existing assessments are EPA's chemical assessments (e.g., previous work plan risk assessments, problem formulation documents), ATSDR's Toxicological Profiles, EPA's IRIS assessments and ECHA's dossiers. This is described in more detail in the [Strategy for Conducting Literature Searches for Asbestos: Supplemental File for the TSCA Scope Document](#) (EPA-HQ-OPPT-2016-0736).

<sup>7</sup> Key and supporting data and information are those that support key analyses, arguments, and/or conclusions in the risk evaluation.

1554 process to ensure that the risk evaluation used the best available science and the weight of the scientific  
1555 evidence.

1556  
1557 Figure 1-4 to Figure 1-8 depict the literature flow diagrams illustrating the results of this process for  
1558 each scientific discipline-specific evidence supporting the draft risk evaluation. Each diagram provides  
1559 the total number of references at the start of each systematic review stage (i.e., data search, data  
1560 screening, data evaluation, data extraction/data integration) and those excluded based on criteria guiding  
1561 the screening and data quality evaluation decisions.

1562  
1563 EPA bypassed the data screening step for data sources that were highly relevant to the draft risk  
1564 evaluation and moved these sources directly to the data quality evaluation step, as described above.  
1565 These data sources are depicted as “key/supporting data sources” in the literature flow diagrams. Note  
1566 that the number of “key/supporting data sources” were excluded from the total count during the data  
1567 screening stage and added, for the most part, to the data evaluation stages depending on the discipline-  
1568 specific evidence. The exception was the releases and occupational exposure data sources that were  
1569 subject to a combined data extraction and evaluation step as shown in Figure 1-5.

1570  
1571 EPA did not have a previous, recent risk assessment of asbestos on which to build; therefore, initially  
1572 the Systematic Review included a very large number of papers for all areas. Initially, studies were  
1573 limited to those published after 1987, containing at least one of the six fiber types identified under  
1574 TSCA. In addition, only observational human studies were searched for the health hazard assessment.  
1575 The risk evaluation was further refined to identify studies pertaining to only mesothelioma and lung  
1576 cancer as health outcomes, as well as studies containing information specific to chrysotile asbestos only.

1577  
1578 As the process for the risk evaluation proceeded, more data became available and the systematic review  
1579 was refined. This included exposure and engineering citations, e.g., correspondences with industry,  
1580 considered to be on-topic and used to inform the likelihood of exposure. The nature of these documents  
1581 is such that the current framework as outlined in the *Application of Systematic Review in TSCA Risk*  
1582 *Evaluations* (U.S. EPA, 2018a) is not well suited for the review of these types of references. And as  
1583 such, these references, were handled on a case by case basis and are cited in the references section of  
1584 this document.

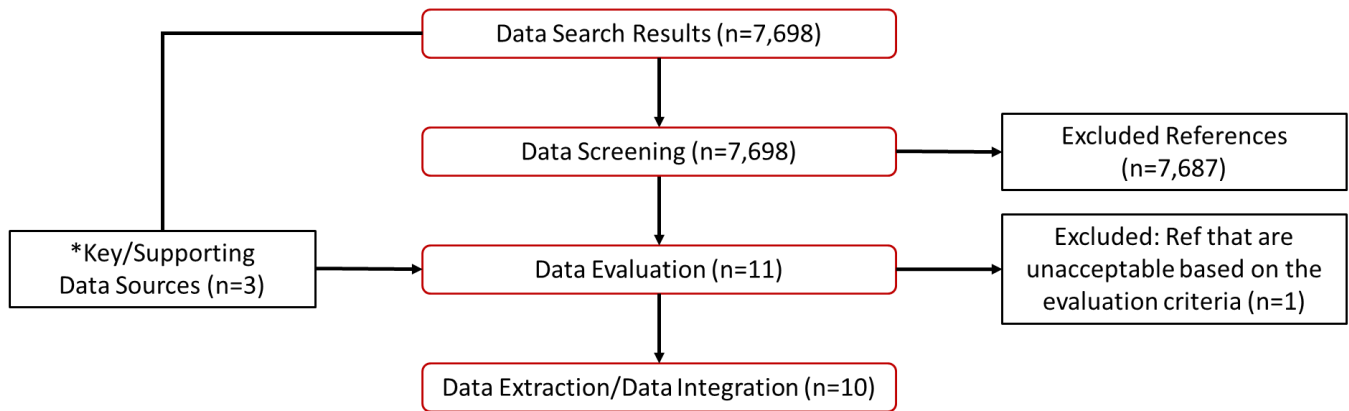
1585  
1586 Information for fate assessment for the first 10 chemical risk evaluations considered the physical  
1587 chemical properties of the chemical and environmental endpoints. For the first 10 chemicals, EPA  
1588 assessed chemical fate as defined by traditional fate endpoints, for example, solubility, partitioning  
1589 coefficients, biodegradation and bioaccumulation – properties that do not apply to asbestos minerals. As  
1590 such, there were few discipline-specific papers identified in the fate systematic review of asbestos  
1591 literature (Figure 1-4).

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**Figure 1-4. Key/Supporting Data Sources for Environmental Fate**

**Note 1:** Literature search results for the environmental fate of asbestos yielded 7,698 studies. Of these studies 7,687 were determined to be off-topic or they did not meet screening criteria (such as non-primary source data or lacking quantitative fate data). The remaining studies entered full text screening for the determination of relevance to the risk evaluation. There were three key and/or supporting data sources identified, the primary literature cited in these sources were passed directly to data evaluation. One primary study was deemed unacceptable based on the evaluation criteria for fate and transport studies and the remaining 10 primary studies were carried forward to data extraction/data integration according to Appendix F in [Application of Systematic Review for TSCA Risk Evaluations \(U.S. EPA, 2018a\)](#). The data evaluation and data extraction files are provided in Appendix F in this draft RE.

**Note 2:** Data sources identified relevant to physical-chemical properties were not included in this literature flow diagram. The data quality evaluation of physical-chemical properties studies can be found in the supplemental document, [Data Quality Evaluation of Physical-Chemical Properties Studies \(U.S. EPA, 2019j\)](#) and the extracted data are presented in Table 1-1.

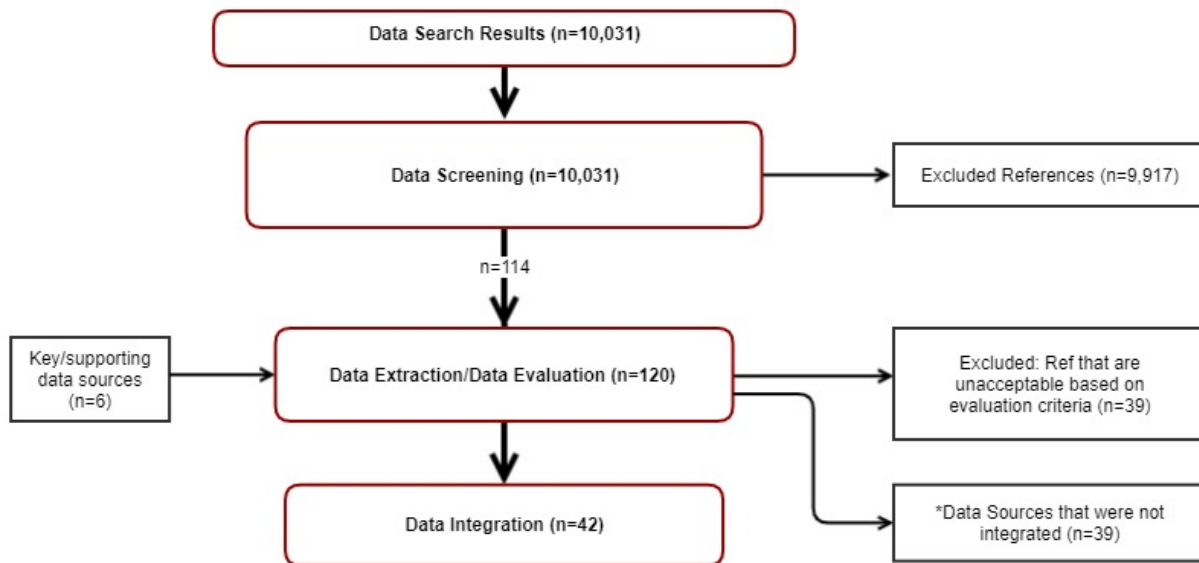


\*These are key and supporting studies from existing assessments (e.g., EPA IRIS assessments, ATSDR assessments, ECHA dossiers) that were considered highly relevant for the TSCA risk evaluation. These studies bypassed the data screening step and primary references cited therein were passed directly to the data evaluation step.

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Figure 1-5. Key/Supporting Data Sources for Engineering Releases and Occupational Exposure

**Note:** Literature search results for environmental release and occupational exposure yielded 10,031 data sources. Of these data sources, 114 were determined to be relevant for the risk evaluation through the data screening process. These relevant data sources were entered into the data extraction/evaluation phase. After data extraction/evaluation, EPA identified several data gaps and performed a supplemental, targeted search to fill these gaps (e.g., to locate information needed for exposure modeling). The supplemental search yielded six relevant data sources that bypassed the data screening step and were evaluated and extracted in accordance with Appendix D in *Application of Systematic Review for TSCA Risk Evaluations* (U.S. EPA, 2018a). Of the 120 sources from which data were extracted and evaluated, 39 sources only contained data that were rated as unacceptable based on serious flaws detected during the evaluation. Of the 81 sources forwarded for data integration, data from 42 sources were integrated, and 39 sources contained data that were not integrated (e.g., lower quality data that were not needed due to the existence of higher quality data, data for release media that were removed from scope after data collection). The data evaluation and data extraction files are provided as separate files (See Appendix B in this draft RE).



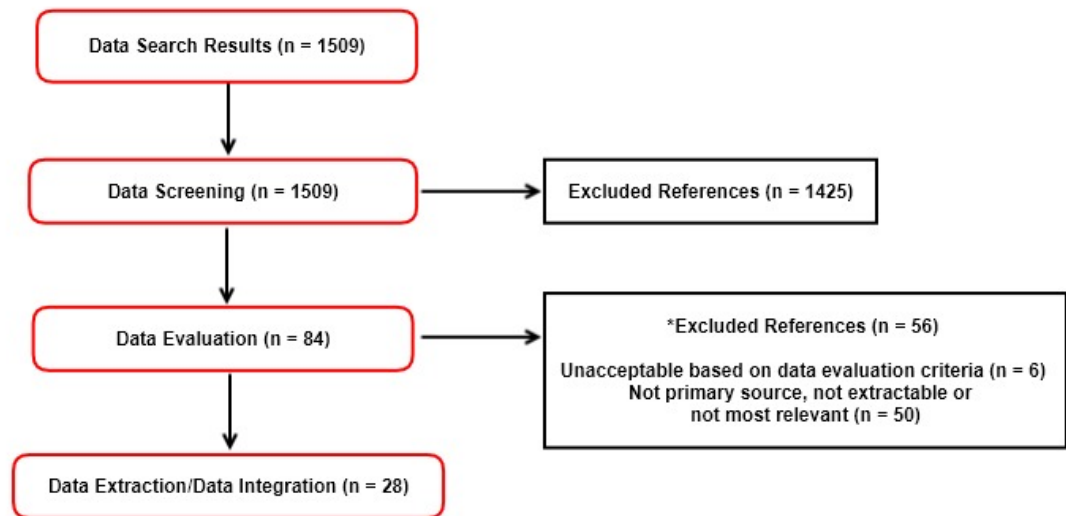
\*The quality of data in these sources (n=39) were acceptable for risk assessment purposes, but they were ultimately excluded from further consideration based on EPA's integration approach for environmental release and occupational exposure data/information. EPA's approach uses a hierarchy of preferences that guide decisions about what types of data/information are included for further analysis, synthesis and integration into the environmental release and occupational exposure assessments. EPA prefers using data with the highest rated quality among those in the higher level of the hierarchy of preferences (i.e., data > modeling > occupational exposure limits or release limits). If warranted, EPA may use data/information of lower rated quality as supportive evidence in the environmental release and occupational exposure assessments.

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**Figure 1-6. Key/Supporting Data Sources for Consumer and Environmental Exposure**

**Note:** Literature search results for consumer and environmental exposure yielded 1,509 data sources. Of these data sources, 84 made it through data screening and into data evaluation. These data sources were then evaluated based on a set of metrics to determine overall relevancy and quality of each data source. The data evaluation stage excluded an additional 56 data sources based on unacceptability under data evaluation criteria (6), not considered a primary source of data, no extractable data, or overall low relevancy to the COUs evaluated (50). The remaining 28 data sources that made it to data evaluation had data extracted for use within the risk evaluation. The data evaluation and data extraction files are provided as separate files (See Appendix B in this draft RE).



\*The quality of data in these sources were acceptable for risk assessment purposes and considered for integration. The sources; however, were not extracted for a variety of reasons, such as they contained only secondary source data, duplicate data, or non-extractable data (i.e., charts or figures). Additionally, some data sources were not as relevant to the PECO as other data sources which were extracted.

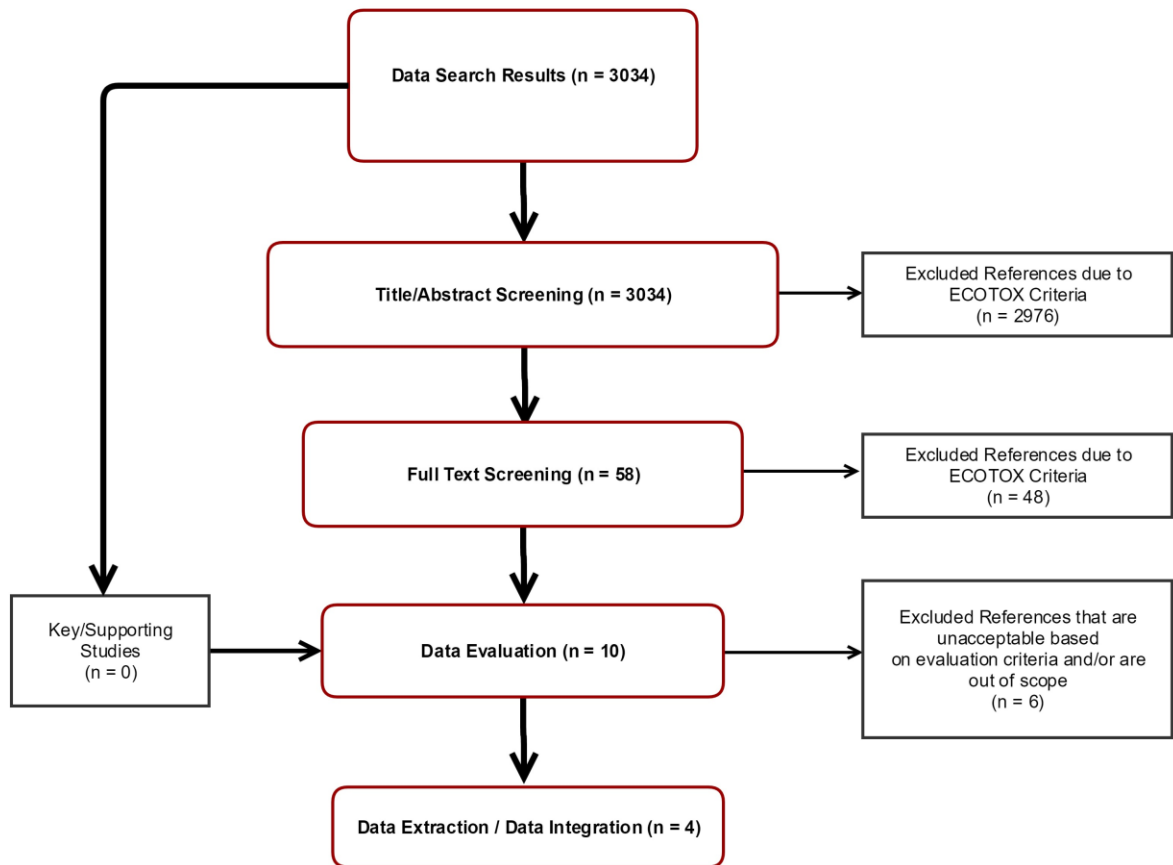
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**Figure 1-7. Key /Supporting Data Sources for Environmental Hazard**

**Note:** The environmental hazard data sources were identified through literature searches and screening strategies using the ECOTOX Standing Operating Procedures. Additional details can be found in the *Strategy for Conducting Literature Searches for Asbestos: Supplemental Document to the TSCA Scope Document*, ([EPA-HQ-OPPT-2016-0736](#)). During PF, EPA made refinements to the conceptual models resulting in the elimination of the terrestrial exposure pathways. Thus, environmental hazard data sources on terrestrial organisms were determined to be out of scope and excluded from data quality evaluation. The data evaluation file is provided as a separate file (See Appendix B in this draft RE).

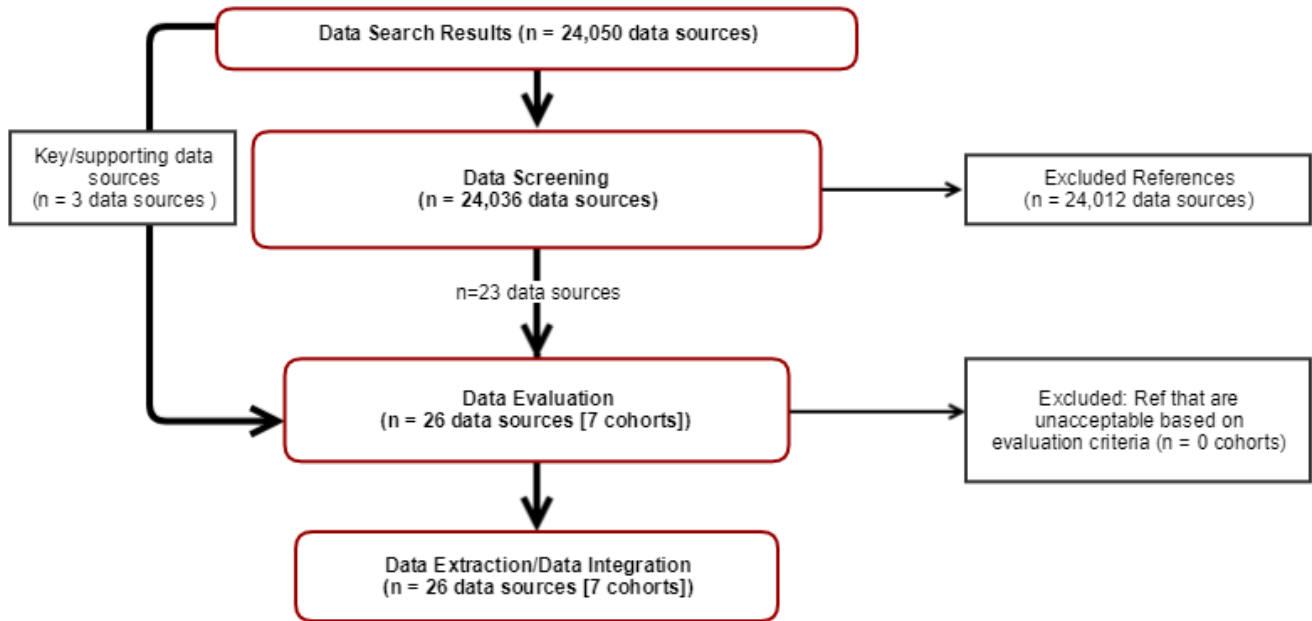


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**Figure 1-8. Key/Supporting Data Sources for Human Health Hazard**

**Note:** Studies were restricted to only mesothelioma and lung cancer as health outcomes, and further restricted to studies containing information specific to chrysotile asbestos only. The data evaluation and data extraction files are provided as separate files (See Appendix B in this draft RE).



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### 1.5.2 Data Evaluation

During the data evaluation stage, EPA assessed the quality of the data sources using the evaluation strategies and criteria described in the *Application of Systematic Review in TSCA Risk Evaluations* (U.S. EPA, 2018a). For the data sources that passed full-text screening, EPA evaluated their quality and each data source received an overall confidence of high, medium, low or unacceptable.

1715

For evaluation of human health hazard studies, the quality criteria presented for epidemiologic studies in the *Application of Systematic Review in TSCA Risk Evaluations* (U.S. EPA, 2018a) 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 (Section 3.2.3.1). The results of the data quality evaluations are summarized in the Supplemental File. Supplemental files (see Appendix B) also provide details of the data evaluations including individual metric scores and the overall study score for each data source.

1723

### 1.5.3 Data Integration

Data integration includes analysis, synthesis and integration of information for the risk evaluation. During data integration, EPA considers quality, consistency, relevancy, coherence and biological plausibility to make final conclusions regarding the weight of the scientific evidence. As stated in the *Application of Systematic Review in TSCA Risk Evaluations* (U.S. EPA, 2018a), data integration

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1729 involves transparently discussing the significant issues, strengths, and limitations as well as the  
1730 uncertainties of the reasonably available information and the major points of interpretation ([U.S. EPA,](#)  
1731 [2018e](#)) EPA defines “reasonably available information” to mean information that EPA possesses, or can  
1732 reasonably obtain and synthesize for use in risk evaluations, considering the deadlines for completing  
1733 the evaluation (*Procedures for Chemical Risk Evaluation Under the Amended Toxic Substances Control*  
1734 *Act* (82 FR 33726)).

1735  
1736 EPA used previous assessments (see Table 1-2. Assessment History of Asbestos ) to identify key and  
1737 supporting information and then analyzed and synthesized available lines of evidence regarding  
1738 asbestos’ chemical properties, environmental fate and transport properties, and its potential for exposure  
1739 and hazard. EPA’s analysis also considered recent data sources that were not considered in the previous  
1740 assessments (as explained in Section 1.5.1 of this document), as well as reasonably available  
1741 information on potentially exposed or susceptible subpopulations.

## 1742 **2 EXPOSURES**

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1743 For TSCA exposure assessments, EPA evaluated exposures and releases to the environment resulting  
1744 from the conditions of use applicable to asbestos. Post-release pathways and routes were described to  
1745 characterize the relationship or connection between the conditions of use for asbestos (Section 1.4.1) and  
1746 the exposure to human receptors, including potentially exposed or susceptible subpopulations (PESS)  
1747 and ecological receptors. EPA considered, where relevant, the duration, intensity (concentration),  
1748 frequency and number of exposures in characterizing exposures to asbestos.

### 1750 **2.1 Fate and Transport**

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1751 Asbestos is a persistent mineral fiber that can be found in soils, sediments, lofted in air and windblown  
1752 dust, surface water, ground water and biota ([ATSDR, 2001b](#)). Asbestos fibers are largely chemically and  
1753 biologically inert in the environment. They may undergo minor physical changes, such as changes in  
1754 fiber length or leaching of surface minerals, but do not react or dissolve in most environmental  
1755 conditions ([Favero-Longo et al., 2005](#); [Gronow, 1987](#); [Schreier et al., 1987](#); [Choi and Smith, 1972](#)).

1756  
1757 The reasonably available data/information on the environmental fate of asbestos is found in Appendix F.  
1758 Those data are summarized below.

1759  
1760 Chrysotile asbestos forms stable suspensions in water; surface minerals may leach into solution, but the  
1761 underlying silicate structure remains unchanged at neutral pH ([Gronow, 1987](#); [Bales and Morgan, 1985](#);  
1762 [Choi and Smith, 1972](#)). Small asbestos fibers (<1 µm) remain suspended in air and water for significant  
1763 periods of time and may be transported over long distances ([Jaenicke, 1980](#)). Asbestos fibers will  
1764 eventually settle to sediments and soil, and movement therein may occur via erosion, runoff or  
1765 mechanical resuspension (wind-blown dust, vehicle traffic, etc.) ([ATSDR, 2001b](#)).

1766  
1767 Limited information is available on the bioconcentration or bioaccumulation of asbestos. Aqueous  
1768 exposure to chrysotile asbestos (10<sup>4</sup>-10<sup>8</sup> fibers/liter) results in embedding of fibers in the tissues of  
1769 aquatic organisms ([Belanger et al., 1990](#); [Belanger et al., 1986c](#); [Belanger et al., 1986a, b](#)). In controlled  
1770 laboratory experiments, asbestos had a negligible bioconcentration factor (BCF slightly greater than 1)  
1771 ([Belanger et al., 1987](#)). Asbestos is not expected to bioaccumulate in food webs ([ATSDR, 2001b](#)).

1773 Asbestos may be released to the environment through industrial or commercial activities, such as  
1774 processing raw asbestos, fabricating/processing asbestos containing products, or the lofting of friable  
1775 asbestos during use, disturbance and disposal of asbestos containing products.

## 1776 **2.2 Releases to Water**

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### 1777 **2.2.1 Water Release Assessment Approach and Methodology**

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1778 The environmental exposure characterization focuses on aquatic releases of asbestos from facilities that  
1779 manufacture, process, or use asbestos under industrial and/or commercial COUs included in this  
1780 document. To characterize environmental exposure, EPA assessed point estimate exposures derived  
1781 from measured concentrations of asbestos in surface water in the United States. Measured surface water  
1782 concentrations were obtained from EPA's Water Quality Exchange (WQX) using the Water Quality  
1783 Portal (WQP) tool, which is the nation's largest source of water quality monitoring data and includes  
1784 results from EPA's STORage and RETrieval (STORET) Data Warehouse, the United States Geological  
1785 Service (USGS) National Water Information System (NWIS), and other federal, state, and tribal sources.  
1786 A literature search was also conducted to identify other peer-reviewed or authoritative gray sources of  
1787 measured surface water concentrations in the United States, but no data were found.  
1788

1789 As discussed in the PF document, because the drinking water exposure pathway for asbestos is currently  
1790 addressed in the Safe Drinking Water Act (SDWA) regulatory analytical process for public water  
1791 systems, this pathway (drinking water for human health) will not be evaluated in this draft RE. The  
1792 Office of Water does not have an ambient water quality criterion for asbestos for aquatic life. Thus,  
1793 potential releases from industrial and commercial activities associated with the TSCA COUs included  
1794 this document to surface water were considered in this draft RE. However, identifying or estimating  
1795 asbestos concentrations in water to evaluate risk to environmental receptors has been challenging.  
1796 During the PF phase of the RE, EPA was still in the process of identifying potential asbestos water  
1797 releases for the TSCA COUs. After the PF was released, EPA continued to search other sources of data  
1798 including TRI data, EPA environmental and compliance monitoring databases, including permits,  
1799 industry responses to EPA questions, and other EPA databases. Details of these investigations are  
1800 included in Appendix D and summarized below.  
1801

1802 TRI reports (Table APX D-2) show that there were zero pounds of friable asbestos reported as released  
1803 to water via surface water discharges in 2018. In addition, TRI reports zero pounds of friable asbestos  
1804 transferred off-site to publicly owned treatment works (POTWs) or to non-POTW facilities for the  
1805 purpose of wastewater treatment. The vast majority of friable asbestos waste management was disposal  
1806 to hazardous waste landfills and to non-hazardous waste landfills.  
1807

1808 EPA issues Effluent Limitations Guidelines and Pretreatment Standards, which are national regulatory  
1809 standards for industrial wastewater discharges to surface waters and POTWs (municipal sewage  
1810 treatment plants). EPA issues these guidelines for categories of existing sources and new sources under  
1811 Title III of the [Clean Water Act \(CWA\)](#). The standards are technology-based (i.e., they are based on the  
1812 performance of treatment and control technologies); they are not based on risk or impacts upon  
1813 receiving waters (see [Industrial Effluent Guidelines](#) for more information). For most operations covered  
1814 by effluent guidelines and standards for the asbestos manufacturing point source category (40 CFR 427),  
1815 the discharge of all pollutants is prohibited. For certain asbestos manufacturing operations, the effluent  
1816 guidelines establish limits on the allowable levels of total suspended solids (TSS), pH, or chemical  
1817 oxygen demand (COD). The regulations do not establish specific limits for asbestos from those  
1818 operations where discharges are allowed. Thus, without the requirement to measure asbestos  
1819 concentrations in effluent, estimating asbestos levels in effluent or receiving waters is challenging.

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EPA investigated industry sector, facility, operational, and permit information regulated by NPDES (National Pollutant Discharge Elimination System) under the CWA to identify any permit limits, monitoring and reporting requirements, and any discharge provisions related to asbestos. The CWA prohibits point source pollutant discharges into waters of the United States unless specifically authorized under the Act, for example through a permit under section 402 (by EPA or an authorized state) that establishes conditions for discharge. Available data were accessed through EPA’s Envirofacts and Enforcement and Compliance History Online (ECHO) systems to identify any evidence of asbestos discharge pertaining to the COUs being evaluated herein. EPA found that no asbestos discharges pertaining to the COUs were reported, and no specific asbestos violations were reported. None of the industrial permits pertaining to the COUs (i.e., chlor-alkali and sheet gasket facilities) had requirements to monitor asbestos. No violation of TSS standards or pH standards were reported.

EPA reports asbestos levels in drinking water from compliance monitoring data from 1998 through 2011 in two separate [six year review cycles](#) (see Table 2-1). However; these data cannot be traced to a specific COU in this draft risk evaluation. In addition, the data are from public water supplies and most likely represent samples from finished drinking water (i.e., tap water) or some other representation that may not reflect the environment in which ecological organisms exist. For these two reasons, these data may not be relevant in assessing the environmental release pathway.

**Table 2-1. EPA OW Six Year Review Cycle Data for Asbestos in Drinking Water, 1998-2011**

Review Cycle	Number of Systems Sampled	Number of Systems with Detections ≥ Minimum Reporting Level (MRL of 0.2 MFL)	Number of Systems with Detections > the MCL of 7 MFL
1998-2005	8,278	268 (3.2%)	14 (<0.2%)
2006-2011	5,785	214 (3.7%)	8 (<0.1%)

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**2.2.2 Water Releases Reported by Conditions of Use**

**2.2.2.1 Processing and Industrial Use of Asbestos Diaphragms in Chlor-alkali Industry**

As noted in the PF, EPA staff visited two separate chlor-alkali facilities in March of 2017 to better understand how asbestos is used, managed and disposed of. The American Chemistry Council (ACC) provided a process description of on-site wastewater treatment methods employed by chlor-alkali facilities to manage and treat wastewater based on their NPDES permits. Some companies in the chlor-alkali industry are known to collect all used diaphragms, hydroblast the asbestos off the screen on which the diaphragm is formed, and filter press the asbestos-containing wastewater. This water in these cases is collected to a sump, agitated, and transferred to a filter press. The filter press contains multiple filter plates with polypropylene filter elements (8 to 100 µm pore size). After solids separation, the filters are removed to large sacks for disposal to a landfill that accepts asbestos-containing waste per federal and state asbestos disposal regulations. The effluent is filtered again and discharged to the facility’s wastewater collection and treatment system ([See Attachment B in ACC Submission](#)). Asbestos releases from chlor-alkali facility treatment systems to surface water and POTWs are not known. While the treatment technologies employed would be expected to capture asbestos solids, the precise treatment efficiency is not known. Chlor-alkali facilities are not required to monitor effluents for asbestos releases, and EPA’s broader research into this COU did not find asbestos water release data.

1860 Another data source considered for asbestos water releases from chlor-alkali facilities was the TRI.  
1861 According to the TRI reporting requirements, industrial facilities are required to disclose asbestos waste  
1862 management practices and releases only for the portion of asbestos that is friable. TRI reporting is not  
1863 required for other forms of asbestos (e.g., non-friable asbestos, asbestos in aqueous solutions) ([U.S.  
1864 EPA, 2017e](#)). Consistent with this qualification in the TRI reporting requirements, no chlor-alkali  
1865 facilities reported asbestos surface water discharges to TRI in reporting year 2018. All chlor-alkali  
1866 facilities reported zero surface water discharges and zero off-site transfers for wastewater treatment.

#### 1867 **2.2.2.2 Processing Asbestos-Containing Sheet Gaskets**

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1868 Based on reasonably available process information provided during an EPA site visit, sheet gasket  
1869 stamping occurs in a warehouse setting with stamping machines (Branham email(s) and observations  
1870 during August 2, 2018 plant visit to Gulfport, MS) ([Branham, 2018](#)). The warehouse has no industrial  
1871 wastewater or water systems, except for potable uses. Housekeeping practices used in relevant work  
1872 areas at the facility EPA visited included a weekly “wipe-down” of equipment (e.g., machine presses,  
1873 dies) and workstations (e.g., table tops) with damp rags, which were disposed of with asbestos-  
1874 containing gasket scraps. This waste was double bagged, sealed, labeled as asbestos, placed in special  
1875 container, and disposed in a landfill permitted to accept asbestos wastes. This company has two sites and  
1876 does not report to TRI for friable asbestos and does not have NPDES permits.  
1877

1878 EPA attempted to identify other companies that fabricate asbestos-containing sheet gaskets in the United  
1879 States but could not locate any. Therefore, it is not known how many sites fabricate imported sheet  
1880 gaskets containing asbestos in the United States. If other companies stamp gaskets in the same way that  
1881 EPA observed at one facility, it could then be assumed that there will not be water releases. However, it  
1882 is not possible to rule out incidental releases of asbestos fibers in wastewater at other fabrication  
1883 facilities if different methods are used, but any amounts of release cannot be quantified.

#### 1884 **2.2.2.3 Industrial Use of Sheet Gaskets at Chemical Production Plants**

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1885 Based on reasonably available process information for the titanium dioxide (TiO<sub>2</sub>) production facility–  
1886 the example used in this draft RE for chemical production plants--described by ACC ([ACC, 2017b](#)) and  
1887 EPA knowledge of the titanium manufacturing process, the purpose of the gasket is to seal equipment  
1888 components. The information indicates that after maintenance workers remove a gasket from a flange,  
1889 he or she will double-bag and seal the gasket and label the bag “asbestos,” and place it in special  
1890 containers for disposal in a landfill permitted to accept asbestos wastes. It appears that there are no water  
1891 releases during use of asbestos gaskets or disposal, and water is not used as an exposure control method;  
1892 therefore, releases to water are not anticipated. However, it is not possible to rule out incidental releases  
1893 of asbestos fibers in wastewater at other facilities if different methods are used, but any amounts of  
1894 release cannot be quantified.  
1895

#### 1896 **2.2.2.4 Industrial Use and Disposal of Asbestos-Containing Brake Blocks in Oil 1897 Industry**

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1898 EPA attempted to evaluate potential water releases of asbestos from use in oil field brake blocks. EPA  
1899 found no reasonably available data or publications documenting asbestos releases from the use of oil  
1900 field brake blocks to water. The only relevant information obtained was an industry contact’s remark  
1901 that workers wash down drawworks before removing used brake blocks and installing new ones ([Popik,  
1902 2018](#)) – a comment that suggests some asbestos fibers may be released into water during this practice.  
1903 The TRI reporting requirements do not apply to the three NAICS codes believed to best represent the  
1904 industries that use oil field brake blocks. No other reasonably available data, such as relevant sampling  
1905 data, publications, or other quantitative insights were found to inform the release assessment. The

1906 reasonably available information currently available for this COU is insufficient for deriving water  
 1907 release estimates.

1908  
 1909 Regarding solid waste, used brake blocks are replaced when worn down to 0.375-inch thickness at any  
 1910 point. Because the remaining portions of the used blocks still contain asbestos, they will be handed as  
 1911 solid waste and are likely handled similarly to used asbestos-containing sheet gaskets: bagged and sent  
 1912 to landfills permitted to accept asbestos waste. The SDS obtained for asbestos-containing brake blocks  
 1913 includes waste disposal. It suggests associated waste should be sent to landfills ([Stewart & Stevenson,  
 1914 2000](#)). While these brake blocks are generally considered non-friable when intact, it is unclear if the  
 1915 asbestos in the used brake blocks is friable or remains non-friable.

1916

1917 **2.2.2.5 Commercial Use, Consumer Use, and Disposal of Aftermarket Automotive**  
 1918 **Asbestos-Containing Brakes/Linings, Other Vehicle Friction Products, and Other**  
 1919 **Asbestos-Containing Gaskets**

1920 EPA determined that water releases for aftermarket asbestos-containing automotive parts (brakes,  
 1921 clutches, gaskets, utility vehicle (UTV) gaskets) do not involve the use of water during the removal and  
 1922 clean up. EPA has not identified peer-reviewed publications that measure water releases of asbestos  
 1923 associated with processing, using, or disposing of aftermarket automotive products.

1924 **2.2.3 Summary of Water Releases and Exposures**

1925 During the PF phase of the RE, EPA was still in the process of identifying potential asbestos water  
 1926 releases for the TSCA COUs in this document. After the PF was released, EPA continued to search EPA  
 1927 databases as well as the literature and attempted to contact industries to shed light on potential releases  
 1928 to water. Very little information was located that indicated that there were surface water releases of  
 1929 asbestos; however, not all releases are subject to reporting (e.g., effluent guidelines) or are applicable  
 1930 (e.g., friability). Based on the reasonably available information in the published literature, provided by  
 1931 industries using asbestos, and reported in EPA databases, there is minimal or no releases of asbestos to  
 1932 surface water associated with the COUs that EPA is evaluating in this risk evaluation.

1933

1934 **2.3 Human Exposures**

1935 EPA evaluated both occupational and consumer scenarios for each COU. The following table provides a  
 1936 description of the COUs and the scenario (occupational or consumer) evaluated in this RE.

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1938 **Table 2-2. Crosswalk of Conditions of Use and Occupational and Consumer Scenarios Assessed in**  
 1939 **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

COU	Scenario	Form of asbestos
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

1940

1941

### 2.3.1 Occupational Exposures

1942

For the purposes of this assessment, EPA considered occupational exposure of the total workforce of exposed users and non-users, which include, but are not limited to, male and female workers of reproductive age who are >16 years of age. This section summarizes the key occupational acute and chronic inhalation exposure concentrations for asbestos.

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EPA only evaluated inhalation exposures to workers and occupational non-users (ONUs) in association with asbestos manufacturing, import, processing, distribution and use in industrial applications and products in the Risk Evaluation. The physical condition of asbestos is an important factor when considering the potential human pathways of exposure. 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. ONUs do not directly handle asbestos or asbestos-containing products but are present during their work time in an area where asbestos or an asbestos-containing product is or may be present.

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Where available, EPA used inhalation monitoring data from industry, trade associations, or the public literature. For each COU, EPA separately evaluates exposures for workers and ONUs. A primary difference between workers and ONUs is that workers may handle chemical substances and have direct contact with chemicals, while ONUs are working in the general vicinity but do not handle the chemical substance. Examples of ONUs include supervisors/managers, and maintenance and janitorial workers who might access the work area but do not perform tasks directly with asbestos or asbestos containing products. For inhalation exposure, in cases where no ONU sampling data are available, 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.



1969 ***Components of the Occupational Exposure Assessment***

1970 The occupational exposure assessment of each COU comprises the following components:

- 1971 • **Process Description:** A description of the COU, including the role of asbestos in the use;  
1972 process vessels, equipment, and tools used during the COU; and descriptions of the worker  
1973 activities, including an assessment for potential points of worker exposure.
- 1974 • **Worker Activities:** Activities in which workers may be potentially exposed to asbestos.
- 1975 • **Number of Sites and Potentially Exposed Workers:** Estimated number of sites that use  
1976 asbestos for the given COU; estimated number of workers, including ONUs, who could  
1977 potentially be exposed to asbestos for the given COU.
- 1978 • **Occupational Inhalation Exposure Results:** EPA used exposure monitoring data provided by  
1979 industry, when it was available, to assess occupational inhalation exposures. EPA also  
1980 considered worker exposure monitoring data published in the peer-reviewed literature. In all  
1981 cases, EPA synthesized the reasonably available information and considered limitations  
1982 associated with each data set. Later in this section, EPA reports central tendency and high-end  
1983 estimates for exposure distribution derived for workers and for ONUs for each COU and  
1984 acknowledges the limitations associated with these exposure estimates.
- 1985 • **Inhalation Exposure Results for Use in the Risk Evaluation:** Central tendency and high-end  
1986 estimates of inhalation exposure to workers and ONUs.

1987 **2.3.1.1 Occupational Exposures Approach and Methodology**

1988 EPA reviewed reasonably available information from OSHA, NIOSH, the peer-reviewed literature,  
1989 industries using asbestos or asbestos-containing products, and trade associations that represent this  
1990 industry (e.g., ACC) to identify relevant occupational inhalation exposure data. Quantitative data  
1991 obtained during Systematic Review were used to build appropriate exposure scenarios when monitoring  
1992 data were not reasonably available to develop exposure estimates. For uses with limited available  
1993 exposure data the assessment used similar occupational data and best professional judgment to estimate  
1994 exposures. In these cases, EPA used assumptions to evaluate risk.

1995 ***General Inhalation Exposures Approach and Methodology***

1997 EPA provided occupational exposure results for each COU that were representative of *central tendency*  
1998 estimates and *high-end* estimates when possible. A central tendency estimate was assumed to be  
1999 representative of occupational exposures in the center of the distribution for a given COU. EPA's  
2000 preference was to use the 50<sup>th</sup> percentile of the distribution of inhalation exposure data as the central  
2001 tendency. In cases where other approaches were used, the text describes the rationale for doing so. EPA  
2002 provided high-end estimates at the 95<sup>th</sup> percentile. If the 95<sup>th</sup> percentile was not available, or if the full  
2003 distribution was not known and the preferred statistics were not available, EPA used a reported  
2004 maximum value or other bounding estimate to represent the high-end estimate.

2005 **2.3.1.2 Consideration of Engineering Controls and Personal Protective Equipment**

2006 OSHA requires employers utilize the hierarchy of controls to address hazardous exposures in the  
2007 workplace. The hierarchy of controls prioritizes the most effective measures to address exposure; the  
2008 first of which is to eliminate or substitute the harmful chemical (e.g., use a different process, substitute  
2009 with a less hazardous material), thereby preventing or reducing exposure potential. Following  
2010 elimination and substitution, the hierarchy prioritizes engineering controls to isolate employees from the  
2011 hazard, followed by administrative controls, or changes in work practices to reduce exposure potential  
2012 (e.g., source enclosure, local exhaust ventilation systems, temperature). Administrative controls are  
2013 policies and procedures instituted and overseen by the employer to protect worker exposures. As the last

means of control, the use of personal protective equipment (PPE) (e.g., respirators, gloves) is required, when the other control measures cannot reduce workplace exposure to an acceptable level.

**Respiratory Protection and OSHA Standards**

OSHA has standards that are applicable to occupational exposure to asbestos including the Respiratory Protection Standard (29 CFR § 1910.134); and the Asbestos Standard (29 CFR § 1910.1001). Both standards have multiple provisions that are highlighted below.

OSHA’s 29 CFR § 1910.134 requires employers to perform a hazard assessment to determine what hazardous exposures exist, if any, and how to mitigate such exposures. The occupational hazard assessment is the basis for the implementation of control measures. Certain industries address workplace hazards by implementing engineering and administrative control measures. When these measures do not fully mitigate the hazard, respiratory protection may be used. Respirator selection provisions are provided in § 1910.134(d) and require that appropriate respirators be selected based on the respiratory hazard(s) to which the worker will be exposed and workplace and user factors that affect respirator performance and reliability. Assigned protection factors (APFs) are provided in Table 1 under § 1910.134(d)(3)(i)(A) (see below in Table 2-3.). APFs refer to the level of respiratory protection that a respirator or class of respirators is expected to provide to employees when the employer implements a continuing, effective respiratory protection program.

**Table 2-3. Assigned Protection Factors for Respirators in OSHA Standard 29 CFR 1910.134<sup>eg</sup>**

Type of Respirator <sup>a, b</sup>	Quarter Mask	Half Mask	Full Facepiece	Helmet/Hood	Loose-fitting Facepiece
1. Air-Purifying Respirator	5	10 <sup>c</sup>	50		
2. Powered Air-Purifying Respirator (PAPR)		50	1,000	25/1,000 <sup>d</sup>	25
3. Supplied-Air Respirator (SAR) or Airline Respirator					
• Demand mode		10 <sup>f</sup>	50		
• Continuous flow mode		50 <sup>f</sup>	1,000	25/1,000 <sup>d</sup>	25
• Pressure-demand or other positive-pressure mode		50 <sup>f</sup>	1,000		
4. Self-Contained Breathing Apparatus (SCBA)					
• Demand mode		10 <sup>f</sup>	50	50	
• Pressure-demand or other positive-pressure mode (e.g., open/closed circuit)			10,000	10,000	

<sup>a</sup> Employers may select respirators assigned for use in higher workplace concentrations of a hazardous substance for use at lower concentrations of that substance, or when required respirator use is independent of concentration.

<sup>b</sup> The assigned protection factors are only effective when the employer implements a continuing, effective respirator program as required by 29 CFR § 1910.134, including training, fit testing, maintenance, and use requirements.

<sup>c</sup> This APF category includes filtering facepieces and half masks with elastomeric facepieces.

<sup>d</sup> The employer must have evidence provided by the respirator manufacturer that testing of these respirators demonstrates performance at a level of protection of 1,000 or greater to receive an APF of 1,000. This level of performance can best be demonstrated by performing a workplace protection factor (WPF) or simulated workplace protection factor (SWPF) study or equivalent testing. Absent such testing, all other PAPRs and SARs with helmets/hoods are to be treated as loose-fitting facepiece respirators and receive an APF of 25.

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<sup>e</sup> These APFs do not apply to respirators used solely for escape. For escape respirators used in association with specific substances covered by 29 CFR § 1910 subpart Z, employers must refer to the appropriate substance-specific standards in that subpart. Escape respirators for other IDLH atmospheres are specified by 29 CFR § 1910.134(d)(2)(ii).

<sup>f</sup> These respirators are not common.

<sup>g</sup> Respirators with bolded APFs satisfy the OSHA requirements for asbestos and an appropriate respirator should be selected based on the air concentration. Filtering facepiece respirators do not satisfy OSHA requirements for protection against asbestos fiber.

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

APFs are intended to guide the selection of an appropriate class of respirators to protect workers after a substance is determined to be hazardous, after an occupational exposure limit is established, and only when the occupational exposure limit is exceeded after feasible engineering, work practice, and administrative controls have been put in place. 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.

Using the OSHA PEL for asbestos of 0.1 f/cc, a half-mask negative pressure HEPA filtered facepiece (when fitted properly) can provide protection in atmospheres with up to 1.0 f/cc [0.1 f/cc multiplied by the APF of 10].

*Only the respirator types and corresponding APFs bolded in Table 2-3. meet the OSHA requirements for asbestos.* The specific respiratory protection required in any situation is selected based on air monitoring data. OSHA specifies that the Maximum Use Concentration (MUC) be calculated to assess respirator selection. The MUC is the maximum amount of asbestos that a respirator can handle from which an employee can be expected to be protected when wearing a respirator. The APF of the respirator or class of respirators is the amount of protection that it provides the worker compared to not wearing a respirator. The permissible exposure limit for asbestos (0.1 f/cc) sets the threshold for respirator requirements. The MUC can be determined mathematically by multiplying the APF specified for a respirator by the OSHA PEL, short-term exposure limit, or ceiling limit.

The APFs are not assumed to be interchangeable for any COU, any workplace, or any worker. The use of a respirator would not necessarily resolve inhalation exposures since it cannot be assumed that employers implement comprehensive respiratory protection programs for their employees. Table 2-3. can be used as a guide to show the protectiveness of each category of respirator. Based on the APFs specifically identified for asbestos and presented in Table 2-3, inhalation exposures may be reduced by a factor of 10 to 10,000 assuming employers institute a comprehensive respiratory protection program.

However, for asbestos, nominal APFs in Table 2-3 may not be achieved for all PPE users ([Riala and Riipinen, 1998](#)), investigated performance of respirators and HEPA units in 21 different exposure abatement scenarios; most involved very high exposures not consistent with COUs identified in this RE. However, for three abatement scenarios, exposure concentrations were below 1 f/cc, which is relevant to the COUs in this draft risk evaluation. In the three scenarios, actual APFs were reported as 50, 5, and 4.

The strength of this publication is the reporting of asbestos samples inside the mask, use of worker’s own protection equipment, and measurement in different real work conditions. The results demonstrate that while some workers have protection above nominal APF, some workers have protection below nominal APF, so even with every worker wearing respirator, some of these workers would not be protected.

### 2.3.1.3 Chlor-Alkali Industry

This section reviews the presence of chrysotile asbestos in semi-permeable diaphragms used in the chlor-alkali industry and evaluates the potential for worker exposure to asbestos.

#### 2.3.1.3.1 Process Description – Asbestos Diaphragms

Asbestos (raw chrysotile) is used in the chlor-alkali industry for the fabrication of semi-permeable diaphragms, which are used in the production of chlorine and sodium hydroxide (caustic soda). The incorporation of asbestos is vital because it is chemically inert and able to effectively separate the anode and cathode chemicals in electrolytic cells (USGS, 2017). Figure 2-1. below shows a typical diaphragm after it has been formed.



**Figure 2-1. Closeup of a Chrysotile Diaphragm Outside of the Electrolytic Cell  
Photograph courtesy of the American Chemistry Council**

Chlor-alkali industry representatives have stated that three companies own a total of 15 chlor-alkali facilities in the United States that use asbestos-containing semi-permeable diaphragms onsite. Some of these facilities fabricate diaphragms onsite from asbestos, and other facilities receive fabricated diaphragms from other chlor-alkali facilities and send them back when the diaphragms reach the end of service life. EPA does not expect exposures to occur when handling fabricated diaphragms. Based on information provided by ACC, the management of asbestos in the chlor-alkali industry is performed in a closely controlled process from its entry into a port in the United States through all subsequent uses. ACC reports that engineering controls, PPE, employee training, medical surveillance, and personal monitoring are all used to monitor and mitigate worker exposures (ACC submission, see Enclosure C).

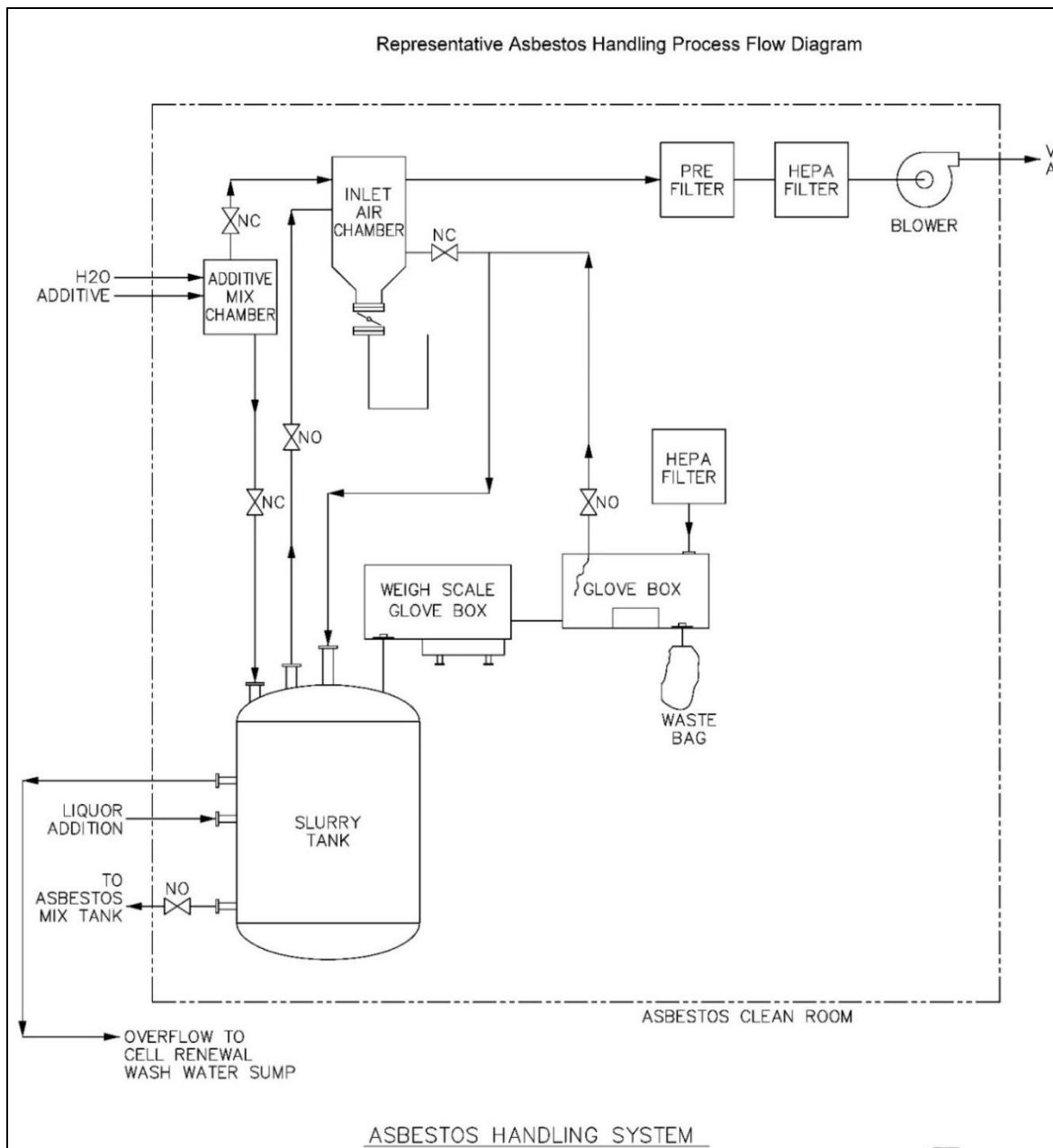
The remainder of this section is based on a description of the chlor-alkali diaphragm manufacturing process and associated asbestos controls. ACC provided this information to EPA, and it is included in the docket (ACC Submission). Unless otherwise specified, all process details presented in the following paragraphs are based on this docket submission. In addition, in 2017 EPA engineers conducted site visits to two chlor-alkali facilities. During these site visits, the observations by EPA engineers’ confirmed details of the process descriptions provided by industry and described below. Other citations are

2133 included in the following paragraphs only for specific details not covered in the main docket reference  
2134 ([EPA-HQ-OPPT-2016-0763-0052](#)).

2135  
2136 After arriving at the plant, the shipping container with raw asbestos is inspected, and any damaged  
2137 containers are shipped back to the sender. Port and warehouse workers manage and remediate any  
2138 damaged containers in conformance with OSHA's asbestos standard for general industry, which  
2139 includes requirements for PPE and respiratory protection (as described above in Section 2.3.1.2).  
2140 Asbestos within the containers is sealed in bags, and workers' first task after opening the containers is to  
2141 inspect bags for leaks. If bags are broken or loose asbestos is evident, the area is controlled to prevent  
2142 accidental exposure, the bags are repaired, and the location is barricaded and treated as an area requiring  
2143 cleanup; workers involved in this activity wear PPE and use respiratory protection, per requirements in  
2144 OSHA's asbestos standard. Plastic-wrapped pallets are labeled per OSHA's hazard communication and  
2145 asbestos standards. Any loose asbestos from punctured bags inside the container is collected using  
2146 HEPA-filtered vacuum cleaners or wetted with water and cleaned up before unloading can proceed.  
2147 Damaged bags are repaired or placed in appropriately labeled, heavy-duty plastic bags. Workers not  
2148 involved in cleanup are prohibited from entering the area until cleanup is complete. When moving the  
2149 asbestos bags into storage locations, care is taken to ensure that bags are not punctured, and personnel  
2150 moving the bags wear specific PPE, including respirators. Storage areas are isolated, enclosed, labeled,  
2151 secured and routinely inspected. Any area or surface with evidence of asbestos is cleaned by a HEPA-  
2152 filtered vacuum or wetted and cleaned up by trained employees wearing PPE.

2153  
2154 To create asbestos-containing diaphragm cells, sealed bags of asbestos are opened, and the asbestos is  
2155 transferred to a mixing tank. At some plants, this process is fully automated and enclosed, in which the  
2156 sealed bags of asbestos are placed on a belt conveyor. The conveyor transfers the sealed bag to an  
2157 enclosure above a mixing vessel. Mechanical knives cut open the bag, and the asbestos and bag  
2158 remnants fall via a chute into the mixing vessel. In other cases, opening of the sealed bags takes place in  
2159 glove boxes. Empty bags are placed into closed and labeled waste containers, either through a port in the  
2160 glove box or during the automated process. The glove boxes are sealed containers with gloves built into  
2161 the side walls, which allow workers to manipulate objects inside while preventing any exposure from  
2162 occurring. Glove boxes also allow workers to open sealed bags and transfer asbestos to a mixing tank  
2163 via a closed system maintained under vacuum.

2164  
2165 Once in the mixing vessel, the raw asbestos used to create a diaphragm is blended with a liquid solution  
2166 of weak caustic soda and salt, thus forming a chrysotile asbestos slurry. Modifiers (e.g., Halar®,  
2167 Teflon®) are added to the slurry. Figure 2-2. shows a process flow diagram of an example glove-box-  
2168 based asbestos handling system and slurry mix tank.



**Figure 2-2. Process Flow Diagram of an Asbestos Handling System and Slurry Mix Tank Image Courtesy of the American Chemistry Council Source: EPA-HQ-OPPT-2016-0736-0106**

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The chrysotile asbestos slurry is deposited onto a metallic screen or perforated plate to form the diaphragm, using a vacuum to evenly apply the slurry across the screen or plate. The diaphragm is drained to remove unbound (free) water and then placed in an oven to dry and harden the asbestos. The modifiers sinter and fuse to the asbestos, the asbestos fuses to the screen or plate, and the asbestos becomes non-friable. After cooling, the diaphragm is installed in the electrolytic cell.

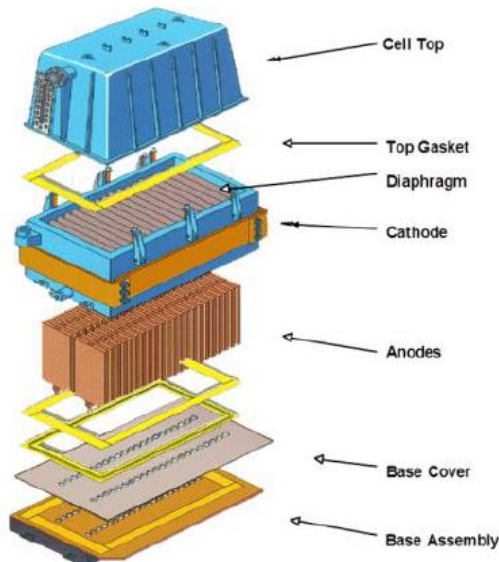
The amount of asbestos used for each diaphragm ranges from 50 to 250 pounds (depending on cell size) and a typical chlor-alkali facility will use about 5 to 25 tons of raw asbestos per year. Industry representatives stated during meetings with EPA that a standard-sized manufacturing cell has a surface

2183 area of 70 m<sup>2</sup> and each cell typically has 20 chrysotile asbestos diaphragms within it, although cell sizes  
2184 vary ([EPA Preliminary Information](#)).

2185  
2186 The chlor-alkali chemical production process involves the separation of the sodium and chloride atoms  
2187 of salt in saltwater (brine) via electricity to produce sodium hydroxide (caustic soda), hydrogen, and  
2188 chlorine. This reaction occurs in an electrolytic cell. The cell contains two compartments separated by a  
2189 semi-permeable diaphragm, which is made mostly of chrysotile asbestos. The diaphragm prevents the  
2190 reaction of the caustic soda with the chlorine and allows for the separation of both materials for further  
2191 processing.

2192  
2193 The cell will typically operate for one to three years before it must be replaced due to a loss of  
2194 conductivity. Many factors can determine the life of a cell, including the brine quality and the cell size.  
2195 During the March 2017 site visit, EPA learned that at least one facility bags and discards the whole  
2196 diaphragm apparatus. However, other chlor-alkali facilities reuse parts of the electrolytic cell, including  
2197 the screen or plate on which the chrysotile diaphragm was formed. The spent asbestos diaphragm is not  
2198 reusable and must be hydroblasted off the screen in a cleaning bay (remaining in a wet state) in order for  
2199 the screen to be reused. The excess water used during this process is filtered prior to discharge to the  
2200 facility's wastewater collection and treatment system. The filtered waste is placed into containers,  
2201 sealed, and sent to a landfill that accepts asbestos-containing waste per federal and state asbestos  
2202 disposal regulations ([EPA Preliminary Information](#)). Figure 2-3. illustrates components and construction  
2203 of an electrolytic cell.

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**Figure 2-3. Electrolytic Cell Construction**  
Image courtesy of the American Chemistry Council  
Source: ([See Enclosure B](#))

### 2.3.1.3.2 Worker Activities – Asbestos Diaphragms

Workers may be potentially exposed to asbestos during various activities associated with constructing, using, and deconstructing asbestos diaphragms, including:

- Inspecting or handling broken bags
- Remediating loose asbestos inside the shipping container
- Opening the bag and handling raw asbestos
- Preparing the diaphragm using asbestos slurry
- Installing the diaphragm in an electrolytic cell (assembly)
- Maintaining the electrolytic cells
- Removing, dismantling, and hydroblasting diaphragms

Based on information provided by industry, when receiving and unloading bags at the facility, workers may be protected through the use of PPE, including respiratory protection (e.g., half-mask respirator with HEPA filters), work gloves, and disposable particulate suits ([See Enclosure C](#)).

As noted previously, some facilities have fully automated and enclosed systems for transferring sealed bags of asbestos to mixing vessels. However, some chlor-alkali facilities transfer materials to a glovebox for weighing operations, during which workers typically wear PAPRs, gloves, and disposable particulate suits ([See Enclosure C](#)). The specific practices for loading dry asbestos from 40-kg bags into the glovebox have not been provided to EPA and likely vary depending on the facility and the glovebox configuration. While some gloveboxes are designed to form a seal with drum-sized product containers, others may require open handling to load the material from the bulk bag into the glovebox.

Slurry preparation involves enclosed processes and wet methods, which minimize airborne exposure potential. Because this is a wet process, workers typically wear gloves and boots with disposable particulate suits, but do not wear respirators even though the short-term (15-minute sampling time) ambient air concentrations were reported to be 0.02 fibers/cc at 50<sup>th</sup> percentile and as high as 0.04 fibers/cc ([See Enclosure C](#)).

For preparing diaphragms, wet asbestos slurry is deposited onto diaphragm screens. One facility stated that the wetted diaphragms are vacuum-dried before being placed in ovens to set ([Axiall-Westlake, 2017](#)). While forming the diaphragms, workers typically wear gloves and boots with disposable particulate suits but do not wear respirators even though the short-term (15-minute sampling time) ambient air concentrations were reported to be 0.0125 fibers/cc at 50<sup>th</sup> percentile and as high as 0.1 fibers/cc which is the OSHA PEL ([See Enclosure C](#)).

For cell assembly, the asbestos contained in the diaphragm is reported to be non-friable (See Enclosure C), thereby eliminating exposure potential. Workers typically wear impermeable gloves and boots but do not wear respirators ([See Enclosure C](#)). Following cell assembly, the diaphragm is inspected and then joined with other parts to complete the electrolytic cell. The short-term (15-minute sampling time) ambient air concentrations for this process were reported to be as high as 0.154 f/cc ([See Enclosure C](#)). Once the diaphragm is in the cell for use in the electrolytic chlor-alkali production process, asbestos exposure from the diaphragms is not expected to occur because the cells are sealed throughout production.

Chlor-alkali facilities use different practices for handling used diaphragms. Some facilities recondition their own diaphragms; some facilities send their used diaphragms to other facilities for reconditioning; and other facilities dispose of used diaphragms and do not recondition them. At the facilities that do perform reconditioning, worker cell repair activities involve disassembling cells and then hydroblasting



2263 diaphragms to remove the asbestos coating. For disassembly, workers typically wear impermeable  
2264 gloves, boots, goggles, and disposable particulate suits but do not wear respirators even though the short  
2265 term (15-minute sampling time) ambient air concentrations were reported to be 0.016 fibers/cc at 50<sup>th</sup>  
2266 percentile and as high as 0.45 fibers/cc ([See Enclosure C](#)). For hydroblasting, workers wear a supplied  
2267 air respirator hood, a waterproof suit, impermeable gloves, and boots ([See Enclosure C](#)). This activity  
2268 occurs in blasting rooms, and workers (while wearing PPE) may be present in these rooms during  
2269 hydroblasting activity ([Axiall-Westlake, 2017](#)).

2270  
2271 For one site EPA visited, the hydroblasting itself was not enclosed but was conducted in a dedicated  
2272 area. The asbestos handling area (slurry mixing, oven, diaphragm disassembly, and hydroblasting area)  
2273 was walled off on three sides with a series of giant pull down doors. The fourth side wall did not extend  
2274 to the ceiling. The layout of such areas may be different at other sites.

2275  
2276 Wastewater from hydroblasting is filter pressed to remove asbestos before discharge from the facility.  
2277 Workers who perform this task typically wear impermeable gloves, boots, and disposable particulate  
2278 suits but do not wear respirators even though the short term (15-minute sampling time) ambient air  
2279 concentrations were reported to be 0.0275 fibers/cc at 50<sup>th</sup> percentile and as high as 0.2 fibers/cc ([See](#)  
2280 [Enclosure C](#)). Filters with filter cakes are then removed from the plate press and bagged for disposal.  
2281 Additionally, two specific practices are expected to minimize workers' asbestos exposures while  
2282 completing this disposal activity: (1) all workers who handle wastes wear PPE, including respirators  
2283 (PAPR) and (2) workers wet solid waste before double-bagging the waste, sealing it, and placing it in  
2284 roll-off containers for eventual transfer to an asbestos landfill ([EPA-HQ-OPPT-2016-0763-0478](#)).

### 2285 **2.3.1.3.3 Number of Sites and Potentially Exposed Workers – Asbestos** 2286 **Diaphragms**

2287 During a meeting with EPA in January 2017, industry representatives stated that in the United States,  
2288 three companies own a total of 15 chlor-alkali plants that continue to fabricate and use asbestos  
2289 (chrysotile)-containing semipermeable diaphragms onsite ([EPA-HQ-OPPT-2016-0736-0069](#)). These  
2290 three companies are Olin Corporation, Occidental Chemical Corporation, and Westlake Corporation. A  
2291 fourth company, Axiall Corporation, previously operated chlor-alkali facilities in the United States, but  
2292 Westlake Corporation acquired this company in 2016. Throughout this section, the companies are  
2293 referred to as Olin, Occidental, and Axiall-Westlake, with the latter referring to chlor-alkali facilities  
2294 currently owned by Westlake, which includes some facilities that were previously owned by Axiall.

2295  
2296 To confirm this facility count, EPA reviewed two other data sources. First, EPA reviewed Chemical  
2297 Data Reporting (CDR) data. Only Olin and Axiall-Westlake reported importing asbestos in 2015. Each  
2298 company reported using asbestos at fewer than 10 sites. Second, EPA reviewed the 2017 TRI data and  
2299 identified a total of 11 facilities reporting information on friable asbestos: three Olin facilities; one  
2300 Axiall-Westlake facility; and seven Occidental facilities. However, it is possible that some of the  
2301 existing chlor-alkali facilities did not have asbestos usage characteristics that would have triggered TRI  
2302 reporting. These two data sources are consistent with the finding that 15 chlor-alkali facilities fabricate  
2303 or use asbestos-containing diaphragms onsite.

2304  
2305 In 2016 CDR, Olin reported a total of at least 25 and fewer than 50 workers who are likely exposed to  
2306 asbestos across all of the company's chlor-alkali facilities, and Axiall-Westlake reported a total of at  
2307 least 50 and fewer than 100 workers who are likely exposed to asbestos across all of the company's  
2308 chlor-alkali facilities. This results in an estimate of at least 75 (25 plus 50) and fewer than 148 (49 plus  
2309 99) workers likely exposed, although this estimate does not include Occidental facilities. As noted  
2310 previously, Occidental facilities did not report to CDR.

2311  
2312 ACC has indicated that approximately 100 workers nationwide in the chlor-alkali industry perform daily  
2313 tasks working with and handling dry asbestos. ACC’s estimate is within the range derived from 2016  
2314 CDR and includes Occidental facilities.

2315  
2316 Regarding potential ONU exposure, EPA considered the fact that area restrictions and other safety  
2317 precautions adopted by the chlor-alkali industry help ensure that no ONU (other than directly exposed  
2318 workers) are near the asbestos diaphragm fabrication processes and use ([EPA-HQ-OPPT-2016-0763-  
2319 0052](#)). However, EPA’s observations during site visits suggest that asbestos exposure might occur to  
2320 workers outside these processes. Additionally, some ONUs (e.g., janitorial staff) may work near the  
2321 asbestos diaphragm fabrication processes. For purposes of this assessment, EPA assumes an equal  
2322 number of ONUs (100) may be exposed to asbestos released from diaphragm fabrication processes and  
2323 use.

#### 2324 **2.3.1.3.4 Occupational Inhalation Exposures – Asbestos Diaphragms**

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2325 To identify relevant occupational inhalation exposure data, EPA reviewed reasonably available  
2326 information from OSHA, NIOSH, the peer-reviewed literature, the chlor-alkali industry, and trade  
2327 associations that represent this industry (e.g., ACC).

#### 2328 **Analysis of Exposed Workers**

2329  
2330 EPA first considered the 2011 to 2016 nationwide exposure data provided by OSHA and the history of  
2331 NIOSH Health Hazard evaluations (HHEs). The OSHA data did not include any observations from the  
2332 chlor-alkali NAICS codes (i.e., 325181 for 2011 and 325180 for 2012 to 2016). Of the NIOSH HHEs  
2333 reviewed, only two were conducted at chlor-alkali facilities, but these evaluations focused on chlorine  
2334 and mercury exposures, not asbestos exposure. One NIOSH HHE considered a facility that received  
2335 disassembled diaphragms for servicing ([Abundo et al., 1994](#)). NIOSH found that the anodes contained  
2336 80 to 90 percent chrysotile asbestos, but the settled dusts from the electrode-servicing facility did not  
2337 have detectable asbestos. The quantitation limit for the dust sampling was not specified. Finally, the  
2338 peer-reviewed literature did not include recent quantitative reports of worker asbestos exposures in the  
2339 chlor-alkali industry.

2340  
2341  
2342 To assess occupational inhalation exposures, EPA used exposure monitoring data provided by industry.  
2343 Data were provided by the three companies that currently use asbestos in the United States chlor-alkali  
2344 industry. Occidental provided exposure monitoring data for six facilities for 1996 to 2016 ([Occidental  
2345 Data, see Volume 2](#)); Axiall-Westlake provided data for 2016 from a single facility ([Axiall, Attachments  
2346 1 and 2](#)); and Olin provided data for 2012 to 2019 from three chlor-alkali facilities and a fourth facility  
2347 that reprocesses anodes ([Olin Corp, 2017](#)). ACC also provided data for 1996 to 2016 ([ACC Data](#)). The  
2348 data that ACC provided were collected at the same chlor-alkali facilities referenced above, and some of  
2349 the data provided by ACC may include duplicates with the data provided by the individual companies.  
2350 This section focuses on PBZ data for asbestos workers.

2351  
2352 The following tables summarize occupational exposure results of different exposure durations for the  
2353 fabrication, use, and disposal of asbestos diaphragms in the chlor-alkali industry. The exposure durations  
2354 considered are full-shift samples, 30-minute average samples, and additional samples of other durations.  
2355 The tables summarize 1,378 sampling results based on the combined PBZ samples from Axiall-  
2356 Westlake, Occidental, Olin, and ACC. Axiall-Westlake, Occidental, and Olin provided a numerical  
2357 sample duration for each sample. For these two data sets (i.e., the combined set from three companies  
2358 and the ACC data), EPA designated samples with durations between 420 and 680 minutes as “full-shift,

samples,” as these durations characterize workers with either 8-hour or 10-hour shifts. The data provided by ACC did not include numerical sample durations. Rather, the ACC data had sample duration descriptions of either “short-term sample” or “full-shift sample,” which EPA assumes refers to 30-minute and 8-hour average observations, respectively. EPA assumes ACC’s sample data were PBZ samples, though this was not clear from the documentation provided.

For samples with results less than the limit of detection (LOD) or limit of quantitation (LOQ), surrogate values were used based on statistical analysis guidelines for occupational exposure data that were developed for EPA (U.S. EPA, 1994). These guidelines call for replacing non-detects with the LOD or LOQ divided by two or divided by the square root of two, depending on the skewness of the data distributions. However, at least half of the samples for every sample averaging time considered were measured concentrations above the detection limit. As a result, the 50<sup>th</sup> and 95<sup>th</sup> percentile concentrations were sensitive only to the magnitude of the measured concentrations and not the strategy used for non-detect replacement.

Table 2-4 and Table 2-5 provide both full-shift and short-term sample summaries. Table 2-6 summarizes PBZ data for all other sampling durations, and Table 2-7 summarizes all short-term samples by exposure group, with additional breakdown by task.

**Table 2-4. 30-min Short-Term PBZ Sample Summary\***

Sample Type	Date Range of Samples	Number of Samples	Maximum Result (f/cc)	50th Percentile (f/cc)	95th Percentile (f/cc)
PBZ	2004 to 2017	384	11**	0.032	0.35

\*Data from Olin, Occidental and ACC

\*\*Note: The maximum concentration in this table (11 fibers/cc) was originally reported as being an “atypical result.” The employer in question required respirator use until re-sampling was performed. The follow-up sample found an exposure concentration (0.019 fibers/cc) more than 500 times lower.

**Table 2-5. Full-Shift\* PBZ Sample Summary**

Sample Type	Date Range of Samples	Number of Samples	Maximum Result (f/cc)	50th Percentile (f/cc)	95th Percentile (f/cc)
PBZ	1996 to 2017	650	0.41	0.0060	0.050

\* Includes both 8-hr and 10-hr TWA sample results.

**Table 2-6. Summary of PBZ Sampling Data for All Other Durations**

Sample Type	Date Range of Samples	Number of Samples	Maximum Result (f/cc)	50 <sup>th</sup> Percentile (f/cc)	95 <sup>th</sup> Percentile (f/cc)
PBZ	2004 to 2019	344	0.91	0.029	0.260

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**Table 2-7 Summary of ACC Short-Term PBZ Sampling Data by Exposure Group (samples from 2001 to 2016)**

Exposure Group / Task Name(s)	Number of Samples	Maximum Result (f/cc)	50th Percentile (f/cc)	95th Percentile (f/cc)
Asbestos Unloading/Transport	8	0.12	0.01	0.09865
Glovebox Weighing and Asbestos Handling	150	1.7	0.0295	0.44
Asbestos Slurry *	5	0.04	0.02	0.036
Depositing *	27	0.1	0.0125	0.0601
Cell Assembly *	31	0.077	0.012	0.0645
Cell Disassembly *	49	0.45	0.016	0.0732
Filter Press *	36	0.2	0.0275	0.1315
Hydroblasting	20	0.51	0.14	0.453

\* Task-specific PPE does not include respirators ([See Enclosure C](#))

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**Analysis of ONUs**

At chlor-alkali facilities, ONU exposures to asbestos are expected to be limited because most asbestos handling areas are likely designated regulated areas pursuant to the OSHA asbestos standard, with access restricted to employees with adequate personal protective equipment. However, EPA considered the possibility of ONU exposure when employees not engaged in asbestos-related activities work near or pass through the regulated areas and may be exposed to asbestos fibers released into the workplace. These employees may include maintenance and janitorial staffs.

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EPA considered area monitoring data (i.e., fixed location air monitoring results) as an indicator of this exposure potential. Across the four sampling data sets provided by industry, only the data provided by Olin included area sampling results ([Olin Corp, 2017](#)). The area monitoring data from Olin’s Alabama, Arkansas, and Louisiana facilities include 15 full-shift asbestos samples collected at fixed locations. The asbestos concentration levels are reported as either 0.004 fibers/cc [N=11] or 0.008 fibers/cc [N=4]. EPA has reason to believe these are all non-detect observations. The notes fields in the sample results identified as 0.008 fibers/cc state “detection limit was 0.008 fibers/cc.” The data that Olin provided for its fourth (Texas) facility do not clearly distinguish whether measurements are area samples or personal breathing zone samples.

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As true exposure values below any limit of detection (LOD) are distributed from zero to the limit of detection, the value of the detection level represents the high end of the distribution of the observations below LOD. To estimate the central tendency, EPA used the mean of the values which was 0.005 fibers/cc and divided by 2 for a central tendency exposure estimate of 0.0025 fibers/cc. The high-end exposure estimate of <0.008 fibers/cc is the higher of the two reported LODs. These values will be used to represent ONU full-shift TWA exposure distribution values in this draft risk evaluation.

**2.3.1.3.5 Exposure Results for Use in Risk Evaluation**

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Table 2-8 presents asbestos exposure data that EPA used in the risk evaluation for workers and ONUs in the chlor-alkali industry. EPA’s basis for selecting the data points appears after the table.

**Table 2-8 Summary of Asbestos Exposures During Processing and Use in the Chlor-Alkali Industry Used in EPA’s Risk Evaluation**

Occupational Exposure Scenario	Exposure Levels (fibers/cc)					
	Workers			ONUs		
	Central Tendency	High-end (95 <sup>th</sup> percentile)	Confidence Rating	Central Tendency	High-end	Confidence Rating
Producing, handling, and disposing of asbestos diaphragms: full-shift TWA exposure	0.0060	0.050	High	0.0025	0.008	Medium
Producing, handling, and disposing of asbestos diaphragms: short-term TWA exposure (30 mins)	0.032	0.35	High	--	--	--

“—” indicates no data reported

The data in Table 2-8 provide a summary of exposure values among workers and ONUs who produce, handle, and dispose of asbestos diaphragms at chlor-alkali facilities. These data represent a complex mix of worker activities with varying asbestos exposure levels. It should be noted that not all activities include use of respirators (Table 2-7). The data points in Table 2-8 were compiled as follows (details presented in *Supplemental File: Occupational Exposure Calculations (Chlor-Alkali)* ([U.S. EPA, 2019b](#)):

- Table 2-8 lists the full-shift TWA exposure levels that EPA used in this risk evaluation. The central tendency value for workers (0.0050 fibers/cc) is the median value of the exposure samples provided by Olin, Occidental and ACC, while the high-end value (0.036 fibers/cc) is the calculated 95<sup>th</sup> percentile (see Table 2-5).
- For ONU exposure estimates area samples were used. Two chlor-alkali facilities provided a total of 15 area samples which were all below the limit of detection (LOD). There were two different detection limits in the two submissions. As true exposure values below any limit of detection are distributed from zero to the limit of detection, the value of the detection level represents the high end of the distribution of the observations below LOD. Central tendency exposure concentrations were calculated by using one-half the detection limit for individual samples; and the high-end concentration is assumed to be the highest detection limit provided.
- The central tendency short-term TWA exposure value for workers was based on short-term (30-minute) sampling data provided by industry. The value in Table 2-5 (0.032 fibers/cc) is the median value of all 30-minute personal samples submitted. The high-end short-term TWA

2453 exposure value for workers (0.35 fibers/cc) is the calculated 95<sup>th</sup> percentile value for the  
2454 compiled industry short-term exposure data. These values are based on all employee tasks  
2455 combined. Refer to Table 2-7 for specific employee tasks (e.g., asbestos handling, filter press  
2456 operation) with higher short-term exposure levels.  
2457

#### 2458 **2.3.1.3.6 Data Assumptions, Uncertainties and Level of Confidence**

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2459 The exposure data shown in Table 2-8 are based monitoring results from the chlor-alkali industry.  
2460 Worker exposure sampling data are available from all three companies (i.e., Occidental, Olin, Axiall-  
2461 Westlake) that currently operate the entire inventory of chlor-alkali facilities nationwide and the overall  
2462 confidence ratings from systematic review for these data were all rated high. Tables 4 through 7  
2463 summarize more than 1,000 individual exposure sampling results, which represent extensive coverage of  
2464 the estimated 100 directly exposed workers. Each company submission of monitoring data includes a  
2465 variety of worker activities. Therefore, this collection of monitoring data likely captures the variability  
2466 in exposures across the different chlor-alkali sites and likely captures the variability in exposures during  
2467 normal operations within a single site.  
2468

2469 EPA notes several limitations with these data:  
2470

- 2471 • the data provided by Axiall-Westlake, Occidental, and Olin represent worker exposures for the  
2472 individual companies. However, the data provided by ACC may include duplicates with the data  
2473 provided by the three companies. The extent of duplicate entries is not known and cannot be  
2474 assessed from the information provided; and  
2475
- 2476 • the monitoring data capture all of the chlor-alkali facilities that use asbestos. However, it is  
2477 uncertain if certain high-exposure activities are captured in this dataset, such as exposures when  
2478 cleaning spilled asbestos within a container from damaged bags.  
2479

2480 EPA used the data for the risk evaluation because of the large number of samples, both full shift and  
2481 short term, and the range of worker activities that will likely capture the variability in exposures.  
2482

2483 EPA considered the quality and uncertainties of the data to determine a level of confidence for the  
2484 assessed inhalation exposures for this COU. The primary strength of this assessment is the use of  
2485 monitoring data from all the sites, which is the highest approach of the inhalation exposure assessment  
2486 approach hierarchy. Based on these strengths and limitations of the data, the overall confidence for  
2487 EPA's assessment of occupational inhalation exposures for this scenario is high.  
2488

2489 Based on these strengths and limitations of the data, the overall confidence for the worker 8-hr TWA  
2490 and short-term data is high.  
2491

2492 For the ONU data – which were all non-detectable area samples – there is medium confidence for this  
2493 set of data.  
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#### 2495 **2.3.1.4 Sheet Gaskets**

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2496 This section describes how asbestos-containing rubberized sheeting is processed into gaskets.

### 2.3.1.4.1 Process Description – Sheet Gasket Stamping

Gaskets are commonly used in industry to form leakproof seals between fixed components (e.g., pipes). Figure 2-4. shows an asbestos-containing gasket and depicts a typical gasket installation for pipe fittings. While many asbestos-free gaskets are commercially available and widely used, asbestos-containing gaskets continue to be the material of choice for industrial applications where gasket material is exposed to extreme conditions such as titanium dioxide manufacturing (e.g., high temperature, high pressure, presence of chlorine). Based on correspondence from ACC, gaskets made from non-asbestos materials reportedly do not provide an adequate seal under these extreme conditions (ACC, 2018).



**Figure 2-4. Typical Gasket Assembly**

**From left to right: photograph of a gasket; illustration of a flange before gasket installation; and illustration of a pipe and flange connection after gasket installation. Photograph taken by EPA; Illustrations from Wikipedia.**

One known company in the United States (Branham Corporation) processes (or fabricates) gaskets from asbestos-containing rubberized sheeting. This stamping activity occurs at two Branham facilities: one in Gulfport, Mississippi and the other in Calvert City, Kentucky. Branham imports the sheeting from a Chinese supplier, and the sheets contain 80 percent (minimum) chrysotile asbestos encapsulated in 20 percent styrene-butadiene rubber (EPA-HQ-OPPT-2016-0736-0067). Branham supplies its finished non-friable asbestos-containing gaskets to several customers, primarily chemical manufacturing facilities in the United States and abroad (see Section 2.3.1.5). It is unknown if other U.S. companies import asbestos-containing sheet material to stamp gaskets.

EPA communicated with industry to understand how Branham typically processes gaskets from asbestos-containing sheeting. This communication includes an October 2017 meeting between EPA and industry representatives, written communications submitted by industry representatives and ACC, and an August 2018 EPA site visit to the Branham gasket stamping facility in Gulfport Mississippi. An overview of the manufacturing process follows.

Rolls of imported asbestos-containing rubberized sheeting are transported inside bolt-locked, sealed containers from the port of entry to the Branham facilities. Branham then stores these rolls in the original inner plastic film wrapping until use. Incoming sheets are typically 1/16-inch thick and weigh

2532 0.6167 pounds per square foot ([ACC, 2018](#)). Branham employees stamp and cut gaskets to customer  
2533 size specifications in a production area. Various other operations occur simultaneously at the Branham  
2534 facilities to include stamping of non-asbestos gaskets using similar stamping machines. These other  
2535 operations occur approximately 20 feet away from the stamping machines used to make asbestos-  
2536 containing gaskets ([EHM, 2013](#)). As noted later in this section, EPA considers the workers supporting  
2537 other nearby operations to be ONUs for this risk evaluation.  
2538

2539 At the Branham facility visited by EPA, workers used three stamping machines to cut the imported  
2540 asbestos-containing sheets into desired sizes. The facility reportedly does not saw gasket material  
2541 ([Branham, 2018](#)), and EPA did not see evidence of this practice during its site visit. The stamping  
2542 machines can be adjusted to make products of varying diameters, from 4 inches to 4 feet. Figure 2-5.  
2543 shows a worker wearing a face mask while operating one of the stamping machines, which uses round  
2544 headed dies attached to a blade. Blades are not removed from the dies, and the dies are seldom “re-  
2545 ruled” (where the rule blade would be pressed back into the wooden die frame).  
2546  
2547  
2548



2549 **Figure 2-5. Asbestos-Containing Stamping Operation**

2550 **Photographs courtesy of Branham Corporation and used with Branham’s permission**  
2551  
2552

2553  
2554 Figure 2-6. shows a photograph of the rule blade, which is approximately 0.010 inches thick.  
2555  
2556



2557 **Figure 2-6. Rule Blade for Stamping Machine**

2558 **Photographs courtesy of Branham Corporation and used with Branham’s permission**  
2559



After stamping the sheet, workers place the finished gasket in individual 6-mm thick resealable bags. These are double-bagged with a warning label and ultimately placed in a container for shipping to customers. Figure 2-7. shows the warning label that Branham applies to asbestos-containing gasket products.



**Figure 2-7. Asbestos Warning Label on Finished Gasket Product Photograph taken by EPA and used with Branham’s permission**

An important consideration for worker exposure is the extent to which sheet gasket stamping releases asbestos-containing fibers, dusts and particles. 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.

The principal cleanup activity during the stamping operation is collection of unused asbestos-containing scrap sheeting, also referred to by the facility as “lattice drops.” Workers manually collect this material and place it in 6-mm thick polyethylene bags, which are then sealed in rigid containers and shipped to the following landfills permitted to receive asbestos-containing waste ([ACC, 2018](#)):

- Asbestos-containing waste from Branham’s Kentucky facility are transported by Branham to the Waste Path Sanitary Landfill at 1637 Shar-Cal Road, Calvert City, Kentucky.
- Asbestos-containing waste from Branham’s Mississippi facility are transported by Team Waste to the MacLand Disposal Center at 11300 Highway 63, Moss Point, Mississippi.

No surface wipe sampling data are available to characterize the extent of settled dust and asbestos fibers present during this operation. The Branham facilities informed EPA that they do not use water, including to wash away scrap or other debris or perform wet mopping, and EPA confirmed this during the site visit. Once per week, however, workers use a damp cloth to wipe down the stamping machine area. Spent cloths from this wiping are bagged and placed in the same rigid containers with the unused scrap material for eventual disposal.

2599 **2.3.1.4.2 Worker Activities – Cutting of Asbestos-containing Sheet Gaskets**

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2600 Worker activities most relevant to potential asbestos exposure include receiving asbestos-containing  
2601 rubber sheeting, processing gaskets by stamping, packaging finished gaskets for shipment, and  
2602 collecting asbestos containing scrap waste.

2603  
2604 The amount of time that workers conduct cutting asbestos-containing sheets varies with production  
2605 demand and other factors. EPA received one month of worker activity data for Branham’s Mississippi  
2606 facility, and these data indicated that, in May 2018, the worker spent no more than 70 minutes per day  
2607 processing asbestos-containing gaskets ([Branham, 2018](#)). Branham informed EPA that the worker at the  
2608 Kentucky facility perform asbestos-containing gasket stamping activity two to three days per week  
2609 ([Branham, 2018](#)). The worker exposure levels from the Kentucky facility will be used in this draft risk  
2610 evaluation because Branham officials informed EPA that they do not anticipate considerable increases  
2611 or decreases in production demand for asbestos-containing sheet gaskets.

2612  
2613 Information on worker PPE use was based on photographs provided by Branham, information in facility  
2614 documents, and observations that EPA made during its site visit. When handling and stamping asbestos-  
2615 containing sheeting and when collecting scraps for disposal, the worker wears safety glasses, gloves, and  
2616 N95 disposable facepiece masks, consistent with Branham procedures ([ACC, 2017a](#)). A 2013 industrial  
2617 hygiene evaluation performed by consultants from Environmental Health Management (EHM)  
2618 concluded that measured asbestos exposures at Branham’s Kentucky facility were not high enough to  
2619 require respiratory protection ([EHM, 2013](#)); however, the worker uses the N95 masks to comply with  
2620 Branham procedures.

2622 **2.3.1.4.3 Number of Sites and Potentially Exposed Workers – Sheet Gasket**  
2623 **Stamping**

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2624 Branham operates two facilities that process asbestos-containing gaskets, with one worker at each  
2625 facility who stamps the asbestos-containing sheet gaskets. During its site visit to one facility, EPA  
2626 observed that stamping of asbestos-containing sheeting occurs in a 5,500 square foot open floor area.  
2627 Other employees work in this open space, typically at least 20 feet away from where asbestos-containing  
2628 gaskets are processed. EPA considers these other employees to be ONUs. The facility also included a  
2629 fully-enclosed air-conditioned office space, where other employees worked.

2630  
2631 EPA received slightly varying estimates of the number of workers at Branham’s facilities and the  
2632 specific locations where they work ([ACC, 2018](#); [Branham, 2018](#)). Based on these estimates, EPA  
2633 assumes that both facilities have one worker who processes asbestos-containing gaskets, two workers  
2634 who process other non-asbestos containing gaskets in the same open floor area (and are considered to be  
2635 ONUs), and at least two workers in the office space. Therefore, EPA assumes that asbestos-containing  
2636 gasket stamping at this company (i.e., at both facilities combined) includes two directly exposed workers  
2637 and four ONUs.

2638  
2639 These estimates are based on the one company known to stamp asbestos-containing sheet gaskets. It is  
2640 unknown if other U.S. companies perform this same stamping activity. EPA attempted to identify other  
2641 companies that cut/stamp asbestos-containing sheet gaskets in the United States but could not locate  
2642 any. Therefore, it is not known how many sites cut or stamp imported asbestos-containing sheet gaskets.

2644 **2.3.1.4.4 Occupational Inhalation Exposure Results – Sheet Gasket Stamping**

2645 To identify relevant occupational inhalation exposure data, EPA reviewed reasonably available  
2646 information from OSHA, NIOSH, the published literature, and industry. All research steps are  
2647 documented below, with more detailed discussion on the most relevant data source, which EPA  
2648 determined was the monitoring results conducted at a Branham facility.

2649  
2650 EPA first considered the 2011 to 2016 nationwide exposure data provided by OSHA and the history of  
2651 NIOSH HHEs, but neither resource included exposure data relevant to stamping of asbestos-containing  
2652 sheet gaskets. For instance, the OSHA data did not include any asbestos results for the gasket  
2653 manufacturing NAICS code 339991.

2654  
2655 EPA also considered the published literature on asbestos exposures associated with sheet gasket  
2656 stamping. This search identified two studies that presented original worker exposure monitoring data.  
2657 One was a 1998 study of sheet gasket production in Bulgaria ([Stroková et al., 1998](#)). However, the study  
2658 lacked specific details on worker activities and the sampling and analysis method used, and the overall  
2659 representativeness of 20-year old processing activities in Bulgaria to today’s operations is unclear. The  
2660 other was a 2000 publication as part of litigation support that examined exposures in a simulated work  
2661 environment ([Fowler, 2000](#)), but this more recent study involved cutting gasket material with a  
2662 conventional woodworking bandsaw - a practice that likely generates elevated asbestos exposures and is  
2663 not representative of Branham’s stamping operations.

2664  
2665 EPA determined that a worker exposure monitoring study conducted at one of the Branham facilities  
2666 provides the most relevant data for this COU. Branham hired EHM consultants to conduct this study,  
2667 which involved a day of PBZ monitoring at the Kentucky facility in December 2012. The EHM  
2668 consultants measured PBZ concentrations for one worker - the worker who operated the stamping  
2669 machine to process asbestos-containing gaskets - and issued a final report of results in 2013 ([EHM,  
2670 2013](#)). The EHM consultants measured worker inhalation exposures associated with a typical day of  
2671 processing asbestos-containing gaskets and reported that samples were collected “during work periods  
2672 when the maximum fiber concentrations were expected to occur” ([EHM, 2013](#)). The EHM consultants  
2673 did not measure or characterize ONU exposures, although EPA believes that two ONUs are present at  
2674 each Branham facility where asbestos-containing sheet gaskets are processed.

2675  
2676 The EHM consultants measured worker inhalation exposure during asbestos-containing gasket stamping  
2677 operations. Ten short-term samples, all approximately 30 minutes in duration, were collected from one  
2678 worker throughout an 8-hour shift. Samples were analyzed by PCM following NIOSH Method 7400.

2679  
2680 The short-term exposures ranged from 0.008 fibers/cc to 0.059 fibers/cc. Table 2-9. lists the individual  
2681 measurement results and corresponding sample durations. Based on the short-term results, the EHM  
2682 consultants calculated an 8-hour TWA exposure of 0.014 fibers/cc, which assumed no exposure during  
2683 periods without sampling. (Note: The periods without sampling appear to be the worker’s break and  
2684 lunch, when exposure would be expected to be zero.)

2685  
2686 The EHM consultants’ study report includes a data summary table, which indicates that the primary  
2687 worker activity covered during the sampling was “cutting gaskets” (i.e., operation of the stamping  
2688 machines); however, the EHM consultants also acknowledged that the worker who was monitored  
2689 collected scrap material while PBZ sampling occurred ([EHM, 2013](#)). EPA infers from the document that  
2690 the sampling represents conditions during a typical workday and covers multiple worker activities.

2691  
2692**Table 2-9. Short-Term PBZ Asbestos Sampling Results (EHM, 2013)**

Duration (minutes)	Result (fibers/cc)
30	0.059
27	0.031
36	0.020
32	0.026
29	0.028
35	0.010
40	0.018
29	0.008
30	0.008
25	0.033

2693

**2.3.1.4.5 Exposure Data for Use in Risk Evaluation – Sheet Gasket Stamping**2694  
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Table 2-10 presents the worker and ONU exposure concentrations that EPA used in this risk evaluation. The following assumptions were made in compiling these data:

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- The central tendency 8-hour TWA exposure value reported for workers (0.014 fibers/cc) was taken from the single calculated value from the personal exposure monitoring study of a Branham worker (EHM, 2013). The calculated value was derived from the ten sampling points shown in Table 2-9., assuming no exposure occurred when sampling was not conducted.
- 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.
- The central tendency short-term exposure value for workers (0.024 fibers/cc) is the arithmetic mean of the ten short-term measurements reported in the EHM study report on the Branham worker (EHM, 2013).
- The high-end short-term exposure value for workers (0.059 fibers/cc) is the highest measured short-term exposure of the Branham worker. This exposure value occurred during a 30-minute sample (EHM, 2013).

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Table 2-10 presents the asbestos exposure data that EPA used in this draft risk evaluation for evaluating risks to workers and ONUs for the COU of processing asbestos-containing sheet gaskets. Given the small number of sampling data points available to EPA, only central tendency and high-end estimates are presented and other statistics for the distribution are not calculated.

2720  
2721

**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

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ONU Exposures

EPA did not identify any ONU exposure measurements for this COU. However, the literature includes “bystander” exposure studies that EPA could use to estimate ONU exposures. Specifically, one publication ([Mangold et al., 2006](#)) measured “bystander” exposure during asbestos-containing gasket removal. 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.

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**2.3.1.4.6 Data Assumptions, Uncertainties and Confidence Level**

The exposure data shown in Table 2-10 are based on 10 PBZ samples collected from one worker performing sheet gasket stamping on a single day at a single facility. EPA used the data from this study for the risk evaluation because it was the only study available that provided direct observations for asbestos-containing sheet gasket stamping operations in the United States. EPA considered the quality and uncertainties of the data to determine a level of confidence for the assessed inhalation exposures for this COU. The primary strength of this assessment is the use of monitoring data, which is the highest approach of the inhalation exposure assessment approach hierarchy. The overall confidence rating from systematic review for these data was high. These monitoring data were provided to EPA by a single company that processes asbestos-containing sheet gaskets with data representing one of its two facilities. However, it is not known how many companies and facilities in total process asbestos-containing sheet gaskets in the United States. Therefore, EPA is uncertain if these monitoring data are representative of the entire U.S. population of workers that are potentially exposed during asbestos-containing sheet gasket processing. The monitoring data were sampled throughout the day of the worker performing the sheet gasket stamping; therefore, these data likely capture the variability in exposures across the various sheet gasket stamping activities. However, it is uncertain if the single sampling day is representative of that facility’s sheet gasket stamping days throughout the year.

Based on these strengths and limitations of the data, the overall confidence for EPA’s assessment of occupational inhalation exposures for this scenario is medium.

2754 **2.3.1.5 Use of Gaskets in Chemical Production**

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2755 **2.3.1.5.1 Process Description – Sheet Gasket Use**

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2756 Asbestos-containing gaskets are used primarily in industrial applications with extreme operating  
2757 conditions, such as high temperatures, high pressures, and the presence of chlorine or other corrosive  
2758 substances. Such extreme production conditions are found in many chemical manufacturing and  
2759 processing operations. These include: the manufacture of titanium dioxide and chlorinated  
2760 hydrocarbons; polymerization reactions involving chlorinated monomers; and steam cracking at  
2761 petrochemical facilities. EPA has attempted to identify all industrial uses of asbestos-containing gaskets,  
2762 but the primary use known to the Agency is among titanium dioxide manufacturing facilities.  
2763

2764 EPA communicated with the titanium dioxide industry to understand typical industrial uses of asbestos-  
2765 containing gaskets. This communication includes an October 2017 meeting between EPA and industry  
2766 representatives and written communications submitted by industry representatives and ACC. An  
2767 overview of asbestos-containing gasket use in the titanium dioxide manufacturing industry follows.  
2768

2769 Branham supplies asbestos-containing gaskets to at least four titanium dioxide manufacturing facilities  
2770 worldwide. Two are Chemours facilities located in DeLisle, Mississippi and New Johnsonville,  
2771 Tennessee; and the other two are located outside the United States ([Mingis, 2018](#)). The manufacture of  
2772 titanium dioxide occurs at process temperatures greater than 1,850 degrees Fahrenheit and pressures of  
2773 approximately 50 pounds per square inch, and it involves multiple chemicals, including chlorine,  
2774 toluene, and titanium tetrachloride ([ACC, 2017b](#)). Equipment, process vessels, and piping require  
2775 durable gasket material to contain these chemicals during operation. The Chemours facilities use the  
2776 Branham products - sheet gaskets composed of 80 percent (minimum) chrysotile asbestos, fully  
2777 encapsulated in styrene-butadiene rubber - to create tight chemical containment seals for these process  
2778 components ([ACC, 2017b](#)). One of these facilities reports replacing approximately 4,000 asbestos-  
2779 containing gaskets of various sizes per year, but any given year's usage depends on many factors (e.g.,  
2780 the number of major turnarounds) ([ACC, 2017b](#)).  
2781

2782 Installed gaskets typically remain in operation anywhere from a few weeks to three years; the time-  
2783 frame before being replaced is largely dependent upon the temperature and pressure conditions ([ACC,  
2784 2018](#)), whether due to detected leaks or as part of a routine maintenance campaign. Used asbestos-  
2785 containing gaskets are handled as regulated non-hazardous material. Specifically, they are immediately  
2786 bagged after removal from process equipment and then placed in containers designated for asbestos-  
2787 containing waste. Containerized waste (volume not known) from both Chemours domestic titanium  
2788 dioxide manufacturing facilities is eventually sent to the following landfills, which are permitted to  
2789 receive asbestos-containing waste ([ACC, 2017b](#)):  
2790

- 2791 • Asbestos-containing waste from Chemours' Tennessee facility is transported to the West  
2792 Camden Sanitary Landfill at 2410 Highway 70 West, Camden, Tennessee.
- 2793
- 2794 • Asbestos-containing waste from Chemours' Mississippi facility is transported to the Waste  
2795 Management Pecan Grove Landfill at 9685 Firetower Road, Pass Christian, Mississippi.  
2796

2797 Though Chemours has an active program to replace asbestos-containing gaskets with asbestos-free  
2798 alternatives and this program has resulted in considerable decreases in asbestos-containing gasket use  
2799 ([EPA-HQ-OPPT-2016-0736-0067](#)), gaskets formulated from non-friable chrysotile asbestos-containing  
2800 sheeting continue to be the only product proven capable of withstanding certain extreme operating  
2801 conditions and therefore provide a greater degree of process safety and integrity than unproven

2802 alternatives according to industry ([ACC, 2017b](#)). A single titanium dioxide manufacturer can have  
2803 approximately 4,000 gaskets of various sizes distributed throughout the plant which are periodically  
2804 replaced during facility shutdowns.

2805 **2.3.1.5.2 Worker Activities – Sheet Gasket Use**

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2806 Worker activities most relevant to asbestos exposure include receiving new gaskets, removing old  
2807 gaskets, bagging old gaskets for disposal, and inserting replacement gaskets into flanges and other  
2808 process equipment. Asbestos-containing gaskets are received and stored in individual resealable 6-mm  
2809 thick plastic bags. Trained maintenance workers wear leather gloves when handling the gaskets for  
2810 insertion into a flange. When removing old gaskets for replacement, trained maintenance workers wear  
2811 respiratory protection—either an airline respirator or cartridge respirator with P-100 HEPA filters,  
2812 although the APF for this respiratory protection was not specified ([ACC, 2017a](#)). Respiratory protection  
2813 is used during this task to protect workers in cases where the originally non-friable asbestos in the  
2814 gaskets has become friable over the service life ([ACC, 2017a](#)).

2815 **2.3.1.5.3 Number of Sites and Potentially Exposed Workers – Sheet Gasket**  
2816 **Use**

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2817 As noted previously, EPA is aware of two Chemours titanium dioxide manufacturing facilities that use  
2818 asbestos-containing gaskets in the United States. However, no estimates of the number of potentially  
2819 exposed workers were submitted to EPA by industry or its representatives. As gaskets are replaced  
2820 during plant shutdowns, this potential number would be low as some workers would be off site during  
2821 the shutdown.

2822  
2823 To estimate the number of potentially exposed workers and ONUs at these two facilities, EPA  
2824 considered 2016 data from the Bureau of Labor Statistics for the NAICS code 325180 (Other Basic  
2825 Inorganic Chemical Manufacturing). These data suggest an industry-wide aggregate average of 25  
2826 directly exposed workers per facility and 13 ONUs per facility. EPA therefore estimates that the two  
2827 Chemours facilities combined have approximately 50 directly exposed workers and 26 ONUs.

2828  
2829 These estimates are based on the one company known to use asbestos-containing gaskets at its titanium  
2830 dioxide manufacturing facilities. Other titanium dioxide manufacturing plants that operate under similar  
2831 conditions in the United States are thought to use asbestos-containing gaskets to prevent chlorine leaks,  
2832 but EPA does not have information to confirm this ([Mingis, 2018](#)).

2833 **2.3.1.5.4 Occupational Inhalation Exposures – Sheet Gasket Use**

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2834 To identify relevant occupational inhalation exposure data, EPA reviewed reasonably available  
2835 information from OSHA, NIOSH, the published literature, and industry. All research steps are  
2836 documented below, with more detailed discussion on the most relevant data source, which EPA  
2837 determined was the monitoring results submitted by ACC for a Chemours titanium dioxide  
2838 manufacturing facility.

2839  
2840 EPA first considered the 2011 to 2016 nationwide exposure data provided by OSHA and the history of  
2841 NIOSH HHEs, but neither resource included asbestos exposure data for the titanium dioxide  
2842 manufacturing industry.

2843  
2844 EPA also considered the published literature on worker asbestos exposure attributed to gasket removal.  
2845 This search did not identify publications that specifically addressed asbestos-containing gasket use in the  
2846 titanium dioxide manufacturing industry. However, two peer-reviewed publications measured worker  
2847 exposures of gasket removal in settings like those expected for this industry:

- 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](#)). The study considered two types of sheet gasket removal activity: gaskets that could be easily removed with a putty knife without breaking, and gaskets that required more intensive means (and longer durations) for removal. Among the data for sheet gasket removal, the highest worker exposure concentration—with asbestos presence confirmed by TEM analysis—was 0.02 fibers/cc for a 141-minute sample. A slightly higher result was reported in a different sample, but TEM analysis of that sample found no detectable asbestos. The overall representativeness of a study more than 20 years old to today’s operations is unclear.
- 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](#)). The simulations considered various gasket removal scenarios (e.g., manual removal from flanges, removal requiring use of a knife, removal requiring use of power wire brushes). The 8-hour TWA PBZ exposures that were not conducted on marine vessels and therefore considered most relevant to the sheet gasket removal ranged from 0.005 to 0.023 fibers/cc. The representativeness of these simulations to an industrial setting is unclear. However, the study provides useful insights on the relative amounts of asbestos exposure between workers and ONUs. The simulated gasket removal scenarios with detected fibers suggested that exposure levels decreased by a factor of 2.5 to 9 between the gasket removal site and the “area/bystander” locations, approximately 5 to 10 feet away.

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](#)).

EPA determined that worker exposure data submitted by ACC for one of the Chemours titanium dioxide manufacturing facilities provide the most relevant data for this COU. ACC stated that only trained Chemours mechanics remove asbestos-containing gaskets, and they use respiratory protection when doing so (either an atmosphere-supplying respirator or an air-purifying respirator) ([ACC, 2017a](#)). According to the information provided to EPA, 34 worker exposure samples have been collected since 2009 during removal of asbestos-containing gaskets, but the number of workers that were evaluated is not known (based on discussions with Chemours during a visit to EPA in October 2017). 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.

### **2.3.1.5.5 Exposure Results for Use in Risk Evaluation – Sheet Gasket Use**

Table 2-11. presents the worker exposure concentrations that EPA is using in this risk evaluation for use of asbestos-containing gaskets at titanium dioxide manufacturing facilities. The following assumptions were made in compiling these data:



- The central tendency 8-hour TWA exposure value for workers (0.026 fibers/cc) is based on the average asbestos exposure measurement reported for gasket removal at titanium dioxide manufacturing facilities ([ACC, 2017a](#)). Though the supporting documentation does not specify sample duration, EPA assumes, based on discussions with Chemours, the average reported concentration can occur throughout an entire 8-hour shift (e.g., for workers removing gaskets throughout a day during a maintenance campaign).
- 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.
- Because the documentation for the 34 worker exposure samples does not include sample duration, EPA cannot assume the central tendency and high-end values apply to short-term exposures. More specifically, if the original data were for full-shift exposures, then assuming those data points apply to short-term durations would understate these exposures. Therefore, EPA has determined that no reasonably available data are available for evaluating worker short-term exposures for this COU.

**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

ONU Exposures

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. 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 factor is based on a study that evaluated exposures in an enclosed setting and therefore may overstate ONU exposures for gasket removal activity in outdoor environments. ONUs may include other maintenance workers, operators, and supervisors.

**2.3.1.5.6 Data Assumptions, Uncertainties and Level of Confidence**

The exposure data shown in Table 2-11. are based on observations from a single reference that presents worker exposure monitoring data for a single company, and documentation for this study is incomplete. EPA estimates that using the 34 direct observations for gasket removal workers likely offers the most representative account of actual exposures, rather than relying on data from the published literature taken from other settings. Moreover, the central tendency concentration shown in Table 2-11. is higher than results from the relevant literature that EPA reviewed, suggesting that the data source considered ([ACC, 2017a](#)) does not understate exposures.

2934 EPA considered the quality and uncertainties of the data to determine a level of confidence for the  
2935 assessed inhalation exposures for this COU. The primary strength of this assessment is the use of  
2936 monitoring data, which is the highest approach of the inhalation exposure assessment approach  
2937 hierarchy. The overall confidence rating from systematic review for these data was rated medium. These  
2938 monitoring data were provided to EPA by industry and represent actual measurements made during  
2939 asbestos-containing sheet gasket removal at a titanium dioxide manufacturing facility in the United  
2940 States. However, the total number of facilities using asbestos-containing sheet gaskets in the United  
2941 States is not known, and EPA could not determine if the industry-provided monitoring data are  
2942 representative of all U.S. facilities that use asbestos-containing sheet gaskets. The monitoring data were  
2943 collected from 2009 through 2017; therefore, the data likely capture temporal variability in the facility's  
2944 operations.

2946 Based on these strengths and limitations of the data, the overall confidence for EPA's assessment of  
2947 occupational inhalation exposures for this scenario is medium.  
2948

### 2949 **2.3.1.6 Oil Field Brake Blocks**

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2950 This section reviews the presence of chrysotile asbestos in oil field brake blocks and evaluates the  
2951 potential for worker exposure to asbestos during use.

#### 2952 **2.3.1.6.1 Process Description – Oil Field Brake Blocks**

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2953 The rotary drilling rig of an oil well uses a drawworks hoisting machine to raise and lower the traveling  
2954 blocks during drilling. The drawworks is a permanently installed component of a mobile drilling rig  
2955 package, which can be either "trailerized" or self-propelled. Therefore, there is no on-site assembly of  
2956 the drawworks. Except for initial fabrication and assembly prior to installation on a new rig, the  
2957 drawworks is not set or installed in an enclosed building ([Popik, 2018](#)).

2958  
2959 The drawworks consists of a large-diameter steel spool, a motor, a main brake, a reduction gear, and an  
2960 auxiliary brake. The drawworks reels the drilling line over the traveling block in a controlled fashion.  
2961 This causes the traveling block and its hoisted load to be lowered into or raised out of the wellbore  
2962 ([Schlumberger, 2018](#)). The drawworks components are fully enclosed in a metal housing. The brake  
2963 blocks, which ride between an inner brake flange and an outer metal brake band, are not exposed during  
2964 operation of the drawworks ([Popik, 2018](#)).

2965  
2966 The brake of the drawworks hoisting machine is an essential component that is engaged when no motion  
2967 of the traveling block is desired. The main brake can have several different designs, such as a friction  
2968 band brake, a disc brake, or a modified clutch. The brake blocks are a component of the braking system  
2969 ([Schlumberger, 2018](#)). According to product specification sheets, asbestos-containing brake blocks are  
2970 most often used on large drilling drawworks and contain a wire backing for added strength. They are  
2971 more resistant than full-metallic blocks, with good flexibility and a favorable coefficient of friction. The  
2972 asbestos allows for heat dissipation and the woven structure provides firmness and controlled density of  
2973 the brake block. Workers in the oilfield industry operate a drilling rig's brakes in an outdoor  
2974 environment and must periodically replace spent brake blocks ([Popik, 2018](#)).

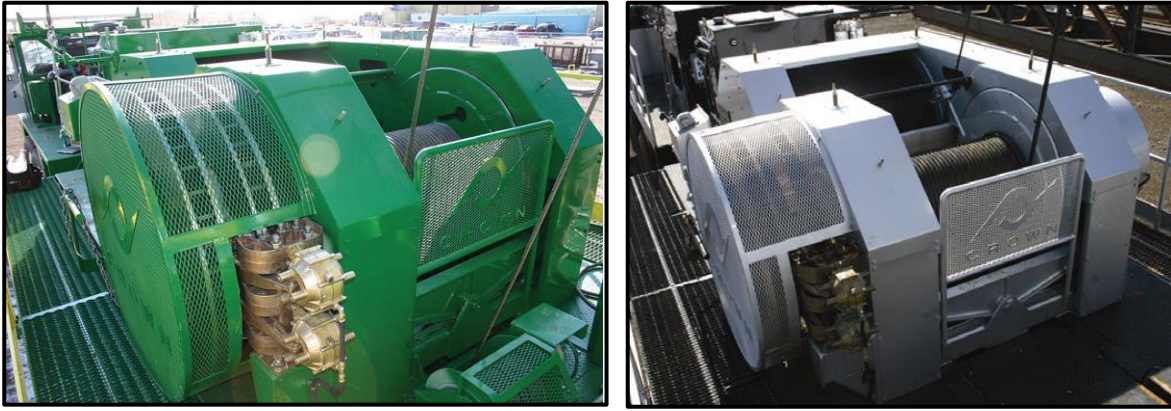


Figure 2-8. Photographs of Typical Oil Field Drawworks

Photograph courtesy of Stewart & Stevenson and used with Stewart & Stevenson’s permission

Drawworks can have either one or two drums, with each drum usually containing two bands, and each band usually containing 10 brake blocks, resulting in a total of 20 to 40 brake blocks per drawworks. The configuration can vary depending on the size of the drawworks. An industry contact specified brake block dimensions of 8 to 12 inches wide by 12 inches long by 0.75 to 1.125 inches thick and weighing six to seven pounds per block. The percent asbestos composition of the brake blocks is unknown (Popik, 2018).

Brake blocks do not require maintenance other than replacement when worn down to a 0.375-inch thickness at any point in the block. The brake blocks typically last between 2 and 3 years under daily operation of the drawworks. Due to the heterogeneous pressure distribution inherent in the mechanics of the brake band design, the brake blocks wear differently depending on their position within the band. However, efforts are made to equalize the tapering pressure distribution by grading the brake block material in order to achieve a more uniform friction at all points along the brake band. (Popik, 2018).

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).

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.” The SDS recommends avoiding drilling, sanding, grinding, or sawing without adequate dust suppression procedures to minimize air releases and inhalation of asbestos fibers from the brake blocks. The SDS recommends protective gloves, dust goggles, and protective clothing. The SDS also specifies that used brake block waste should be sent to landfills (Stewart & Stevenson, 2000).

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. It is unclear if any other companies fabricate or import asbestos-containing brake blocks, or how widespread the continued use of asbestos brake blocks is in oilfield equipment. However, EPA understands from communications with industry that the use of asbestos brake blocks has decreased significantly over time and continues to decline (Popik, 2018).

**2.3.1.6.2 Worker Activities – Oil Field Brake Blocks**

Worker activities include receipt of asbestos-containing brake blocks, removing old brake blocks, bagging old brake blocks for disposal, and installing new brake blocks into drawworks machinery. The activities that may result in asbestos exposure include installing and servicing brake blocks (which may also expose workers in the vicinity). Additionally, workers at the drawworks may be exposed to asbestos fibers that are released as the brake blocks wear down over time. EPA has not identified PPE and industrial hygiene practices specific to workers removing and installing asbestos-containing brake blocks.

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

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.

To estimate the number of potentially exposed workers, EPA used 2016 Occupational Employment Statistics data from the Bureau of Labor Statistics (BLS) and 2015 data from the U.S. Census’ Statistics of U.S. Businesses. EPA used BLS and Census data for three NAICS codes: 211111, Crude Petroleum and Natural Gas Extraction; 213111, Drilling Oil and Gas Wells; and 213112, Support Activities for Oil and Gas Operations. Table 2-13 summarizes the total establishments, potentially exposed workers, and ONUs in these industries. EPA does not have an estimate of the number of establishments in these industries that use asbestos-containing brake blocks. Therefore, EPA presents these results as bounding estimates of the number of establishments and potentially exposed workers and ONUs.

For each of the three NAICS codes evaluated, Table 2-12. presents EPA’s estimates of industry-wide aggregate averages of directly exposed workers per site and ONUs per site. EPA estimates an upper bound of 21,670 sites, 61,695 directly exposed workers, and 66,108 ONUs.

**Table 2-12. Summary of Total Establishments in Relevant Industries and Potentially Exposed Workers and ONUs for Oilfield Brake Blocks**

NAICS Codes	NAICS Description	Total (Entire Industry Sector)				Workers with Relevant Occupations			
		Total Firms	Total Establishments	Total Employees	Average Employees per Establishment	Workers in Relevant Occupations	Occupational Non-Users	Workers per Site	ONUs per Site
211111	Crude Petroleum and Natural Gas Extraction	6,270	7,477	124,847	17	15,380	32,704	2	4
213111	Drilling Oil and Gas Wells	1,973	2,313	89,471	39	10,256	7,397	4	3
213112	Support Activities for Oil and	9,591	11,880	314,589	26	36,059	26,007	3	2

NAICS Codes	NAICS Description	Total (Entire Industry Sector)				Workers with Relevant Occupations			
		Total Firms	Total Establishments	Total Employees	Average Employees per Establishment	Workers in Relevant Occupations	Occupational Non-Users	Workers per Site	ONUs per Site
	Gas Operations								
<b>All NAICS</b>		17,834	21,670	528,907	27	61,695	66,108	3	3

**2.3.1.6.4 Occupational Inhalation Exposures – Oil Field Brake Blocks**

3046  
 3047 EPA did not identify any studies that contain exposure data related to asbestos-containing brake blocks  
 3048 but did identify one published study that contains limited air sampling data for asbestos-containing brake  
 3049 bands ([Steinsvag et al., 2007](#)). In the absence of any other exposure data, the limited data provided in  
 3050 this study were used to estimate exposures to workers from brake block installation, servicing, and  
 3051 removal. The study references stationary samples of asbestos fibers taken in 1988 from the drilling floor  
 3052 at an unnamed facility in Norway’s offshore petroleum industry. Use of asbestos was generally banned  
 3053 in Norway in late 1984, but asbestos brake bands were used in the drilling drawworks on some  
 3054 installations until 1991. The study notes: “...the design of the drilling area might have led to migration  
 3055 of fibers from the brake bands into the drilling cabin or down one floor to the shale shaker area”  
 3056 ([Steinsvag et al., 2007](#)).

3057  
 3058 Stationary samples were taken at two locations: “above brake drum” and “other samples, brake dust.”  
 3059 Reported arithmetic mean concentrations of asbestos fibers for both locations were 0.03 and 0.02  
 3060 fibers/cc, respectively. However, because the publication does not indicate what activities workers  
 3061 performed during sample collection, no inferences can be made regarding whether the results pertain to  
 3062 brake installation, removal, servicing, or repair. The study involved an unknown number of  
 3063 measurements made over an unknown duration of time. While the study does not identify the sample  
 3064 collection methods or the fiber counting algorithms, some text suggests that the presence of asbestos in  
 3065 the samples was confirmed by electron microscope. The study reports the following additional details  
 3066 about the asbestos content of the brake lining: “The composition of the brake lining was: 41% asbestos,  
 3067 28% rayon and cotton, 21% binding agent, 9% brass chip” ([Steinsvag et al., 2007](#)).

3068  
 3069 The sample measurements were made over an unknown duration of time, and EPA is assuming  
 3070 measurements are representative of an 8-hr TWA. EPA assumes the measurements taken above the  
 3071 brake drum are most relevant to worker exposures, as workers are likely to work nearest the brakes, such  
 3072 as operating a brake handle to control the speed of the drawworks or replacing the brake blocks. EPA  
 3073 assumes the other brake dust samples are relevant to ONU exposures as their exact sampling location is  
 3074 not specified but the arithmetic mean concentration is lower than that of the samples taken above the  
 3075 brake drum. Since these two results are both arithmetic means, EPA assumed the values were 0.03 and  
 3076 0.02 fibers/cc for 8-hour TWA, for workers and ONUs, respectively. This study was rated “low” in  
 3077 systematic review ([Steinsvag et al., 2007](#)).

3078

**2.3.1.6.5 Exposure Results for Use in Risk Evaluation – Oil Field Brake Blocks**

3079  
 3080  
 3081 The information available to EPA confirms that some brake blocks used in domestic oilfields contain  
 3082 asbestos, as demonstrated by an SDS provided by a supplier. It is reasonable to assume that wear of the  
 3083 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.

**Table 2-13. Summary of Asbestos Exposures During Use in Brake Blocks for EPA’s Risk Evaluation**

Occupational Exposure Scenario	8-hr TWA Exposure Levels (fibers/cc)			
	Workers		ONUs	
	Central Tendency	Confidence Rating	Central Tendency	Confidence Rating
Brake Blocks: 8-hr TWA exposure	0.03	Low	0.02	Low

ONU Exposures

EPA has not identified specific data on potential ONU inhalation exposures from brake block use. It is assumed that ONUs do not directly handle brake blocks and drawworks machineries, and it is also assumed that drawworks are always used and serviced outdoors close to oil wells. Given the limited information identified above, the lower of the two reported values in the Norway study will be used to represent ONU exposures for this COU.

**2.3.1.6.6 Data Assumptions, Uncertainties and Level of Confidence**

The extent of brake block usage and associated worker exposures are highly uncertain. EPA was not able to identify the volume of imported asbestos-containing brake blocks, the number of brake blocks used nationwide, nor the number of workers exposed as a result of installation, removal, and disposal activities. Further, the study reviewed in this section examined asbestos exposures in 1988 in Norway’s offshore petroleum industry and is of unknown relevance to today’s use of oil field brake blocks in the United States. No other data for brake blocks could be located.

EPA considered the quality and uncertainties of the data to determine a level of confidence for the assessed inhalation exposures for this condition of use. The primary strength of this assessment is the use of monitoring data, which is the highest approach of the inhalation exposure assessment approach hierarchy. However, the monitoring data are limited a single offshore oil platform in Norway in 1988. It is unknown if these data capture current-day U.S. oil field or offshore platform operations. It is also unknown if the monitoring data capture the variabilities in the day-to-day operations of the single offshore platform sampled in the study.

Based on these strengths and limitations of the data, the overall confidence for EPA’s assessment of occupational inhalation exposures for this scenario is low.

### 2.3.1.7 Aftermarket Automotive Brakes/Linings and Clutches

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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.

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. This COU is categorized as “other vehicle friction products” in Table 1-4. of Section 1.4.2 of this risk evaluation. This section reviews the presence of chrysotile asbestos in aftermarket automotive parts and evaluates the potential for worker exposure to asbestos. The section focuses on asbestos in light-duty passenger vehicles, including cars, trucks, and vans.

Note that for occupational exposure for this COU, the use of compressed air as a work practice will not be considered because, in addition to the EPA current best practice guidance ([EPA-747-F-04-004](#)), there is a provision in the OSHA Asbestos Standard: 29 CFR § 1910.1001(f)(1)(ix): Compressed air shall not be used to remove asbestos or materials containing asbestos unless the compressed air is used in conjunction with a ventilation system which effectively captures the dust cloud created by the compressed air.

#### 2.3.1.7.1 Process Description – Aftermarket Automotive Brakes/Linings and Clutches

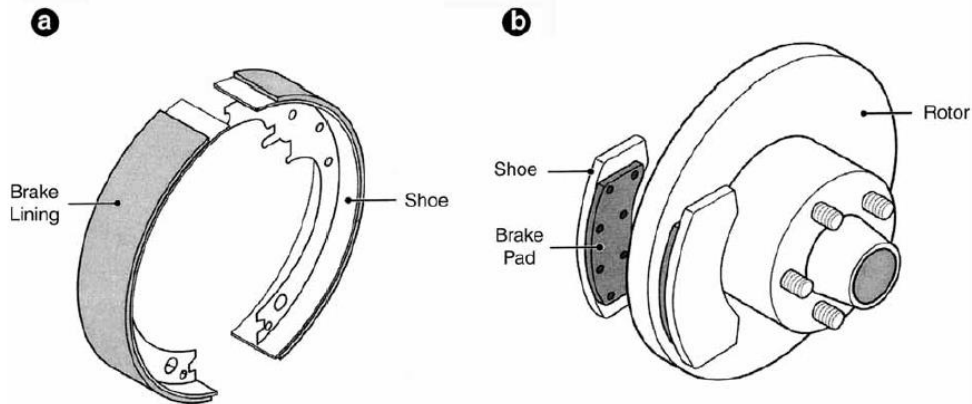
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Based on the long history of the use of asbestos in automobile parts, and because aftermarket automotive parts may still be available for purchase, the Agency believes this COU is still ongoing. Over the past few decades, automobile weights, driving speeds, safety standards, and applicable environmental regulations have changed considerably. These and other factors have led to changes in materials of choice for automobile parts. 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. This section focuses on asbestos in brakes/linings and clutches because repairs for these parts - and hence potential occupational exposure to asbestos - are more likely than repairs for other vehicle components that were known to previously contain asbestos (e.g., seam sealants). For the purpose of this risk evaluation, EPA generally refers to brakes in the following sections, but this term also includes brake linings, brake pads, and clutches.

##### Automobile Brakes

Chrysotile asbestos fibers offer many properties (e.g., heat resistance, flexibility, good tensile strength) that are desired for brake linings and brake pads ([Paustenbach et al., 2004](#)). New automobiles manufactured in the United States had brake assemblies with asbestos-containing components. For instance, NIOSH reported in the late 1980s that friction materials in drum brakes typically contained 40 to 50 percent asbestos by weight ([OSHA, 2006](#)). Other researchers reported that some brake components during these years contained as much as 73 percent asbestos, by weight ([Blake et al., 2003](#)).

3163 The two primary types of automobile brakes are drum brakes and disc brakes, and chrysotile asbestos  
 3164 has been found both in linings for drum brake assemblies and pads in disc brake assemblies (see Figure  
 3165 2-9.). Drum brakes were more prevalent than disc brakes in older vehicles. When the vehicle operator  
 3166 engages drum brakes, the brake shoes (which contain friction materials) contact the rotating brake drum,  
 3167 and this contact slows the vehicle. Disc brakes are much more common today than drum brakes, and  
 3168 they function by applying brake pads (which contain friction materials) to the surface of the revolving  
 3169 brake disc, and this contact slows the vehicle. Since the mid-1990s, material and design improvements  
 3170 have led to most cars being manufactured with disc brakes, effectively phasing out drum brakes in  
 3171 passenger automobiles (Richter et al., 2009).  
 3172  
 3173



3174  
 3175 **Figure 2-9. Illustrations of brake assembly components: (a) a brake lining designed to be used**  
 3176 **with an internal drum brake and (b) a brake pad designed for use with a disc brake.**  
 3177 **Source:** (Paustenbach et al., 2004).  
 3178  
 3179

3180 Use of asbestos-containing braking systems began to decline in the 1970s due to many factors, including  
 3181 toxicity concerns, rising insurance costs, regulatory scrutiny, challenges associated with disposing of  
 3182 asbestos-containing waste, and availability of asbestos-free substitutes (Paustenbach et al., 2004). In  
 3183 1989, EPA issued a final rule that banned the manufacturing and importing of many asbestos-containing  
 3184 products, including automobile brake pads and linings (Federal Register, 1989). While the court  
 3185 overturned most of this ban in 1991, many manufacturers had already begun to phase out asbestos-  
 3186 containing materials and develop alternatives, including the non-asbestos organic fibers that are almost  
 3187 universally used in automobile brake assemblies today (Paustenbach et al., 2004). By 2000, domestic  
 3188 manufacturers had eliminated asbestos from virtually all brake assemblies in automobiles (Paustenbach  
 3189 et al., 2004). EPA is not aware of any automobile manufacturers that currently use asbestos products in  
 3190 brake assemblies for U.S. vehicles. In fact, the Agency received verification from five major vehicle  
 3191 manufacturers that asbestos-containing automotive parts are no longer used and import data has been  
 3192 misreported under the wrong Harmonized Tariff Schedule (HTS) code. However, the Agency knows of  
 3193 at least one company that imports asbestos-containing friction products for use in cars assembled in the  
 3194 U.S., but those vehicles are exported for sale and are not sold domestically. The COU identified for this  
 3195 scenario is specified as “other vehicle friction products” in Table 1-34, and the exposure values are  
 3196 based on aftermarket auto brakes (see Section 2.3.1.8).  
 3197

3198 The history of asbestos in aftermarket brake products has followed a similar pattern. For decades,  
 3199 asbestos was found in various aftermarket brake replacement parts (e.g., pads, linings, and shoes); but  
 3200 the same factors listed in the previous paragraph led to a significant decline in the use of asbestos in  
 3201 aftermarket vehicle friction products. Nonetheless, the literature indicates that asbestos-containing



3202 replacement brake materials continued to be available from parts suppliers into the 2000s; researchers  
3203 were able to purchase these materials in 2008 from a vintage auto parts facility ([Madl et al., 2008](#)).  
3204 Today, individual consumers can find aftermarket automotive products marketed as containing asbestos  
3205 through online retailers.

3206  
3207 In more recent years, state laws and regulations have limited sales of asbestos-containing aftermarket  
3208 brake parts, even among existing stockpiles. In 2010, for instance, the state of Washington passed its  
3209 “Better Brakes Law,” which prohibits manufacturers, retailers, wholesalers, and distributors from selling  
3210 brake friction material that contains more than 0.1 percent asbestiform fibers ([Washington State, 2010](#)).  
3211 In the same year, the state of California passed legislation with similar requirements. The not-to-exceed  
3212 limit of 0.1 percent asbestiform fibers in aftermarket brake parts now essentially extends nationwide,  
3213 due to a memorandum of understanding between EPA and multiple industry stakeholders (e.g., Motor  
3214 and Equipment Manufacturers Association, Automotive Aftermarket Suppliers Association, Brake  
3215 Manufacturers Council) ([U.S. EPA, 2015](#)).

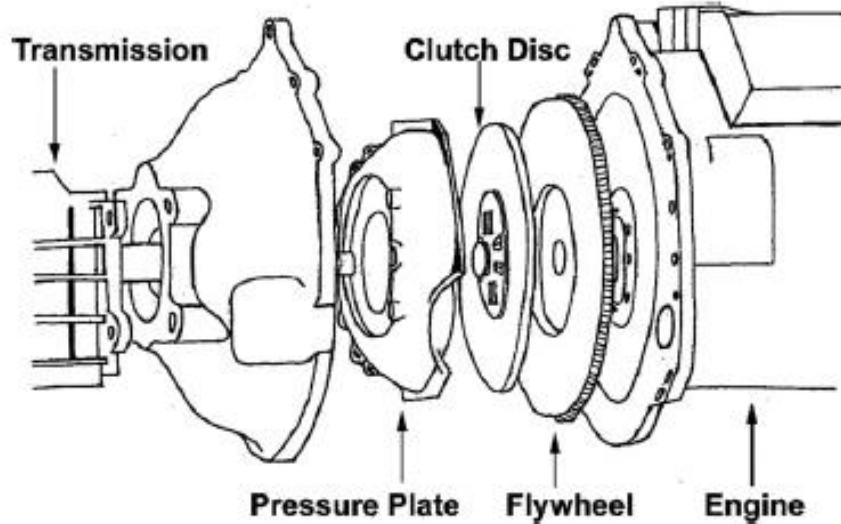
3216  
3217 Despite this trend, asbestos in automotive parts is not banned at the federal level, and foreign suppliers  
3218 face no restrictions (other than those currently in place in the states of California and Washington) when  
3219 selling asbestos-containing brake products to business establishments and individuals in the United  
3220 States. The Motor and Equipment Manufacturers Association informed EPA that approximately \$2.2  
3221 million of asbestos-containing brake materials were imported into the United States in 2014 ([MEMA,  
3222 2016](#)). In 2018, the U.S. Geological Survey indicated that “an unknown quantity of asbestos was  
3223 imported within manufactured products,” such as brake linings ([USGS, 2019](#)).

3224  
3225 Based on this context, asbestos is currently found in automobile brakes in the United States due to two  
3226 reasons: (1) vehicles on the road may have asbestos-containing brakes, whether from original  
3227 manufacturers (primarily for older and vintage vehicles) or aftermarket parts; and (2) vehicles may have  
3228 new asbestos-containing brakes installed by establishments or individuals that use certain imported  
3229 products.

### 3230 Automobile Clutches

3231 In a manual transmission automobile, which currently accounts for less than 5 percent of automobiles  
3232 sold in the United States, the clutch transfers power generated by the engine to the drive train. The  
3233 schematic in Figure 2-10. shows a typical clutch assembly. Because it lies at the interface between two  
3234 rotating metallic surfaces, the clutch disc typically contains friction materials. Decades ago, the friction  
3235 material of choice was chrysotile asbestos, which previously accounted for between 30 and 60 percent of  
3236 the friction material in clutch discs ([Jiang et al., 2008](#)).  
3237

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**Figure 2-10. Schematic of a clutch assembly. The clutch disc is made of friction material, which may contain asbestos.**

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3242

**Source:** ([Jiang et al., 2008](#)).

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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.

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Asbestos-containing aftermarket clutches may be found as imports from foreign suppliers, although the extent to which this occurs is not known. No barriers currently exist to these imports, as asbestos in automotive clutches is not banned at the federal level and the brake laws passed in 2010 in the state of California and the state of Washington do not apply to clutches.

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### **2.3.1.7.2 Worker Activities – Aftermarket Automotive Brakes/Linings and Clutches**

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This section describes worker activities for repair and replacement of both brakes and clutches, including the types of dust control measures that are typically used. For both types of parts, asbestos exposure may occur during removal and disposal of used parts, while cleaning the assemblies, and during handling and installation of new parts.

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#### Automobile Brake Repair and Replacement

For both drum brakes and disc brakes, maintenance, repair, inspection, and replacement jobs typically involve several basic steps. Workers first need access to the brake assembly, which is typically accomplished by elevating the vehicle and removing the wheel. They then remove dust and debris from the brake apparatus using methods described below. Replacement or repair of parts follows, during which workers use various mechanical means to remove old parts and install new ones.

Two critical issues for exposure assessment are the work practices used to remove dust and debris from the brake assembly and the asbestos content of this material:

1. Work practices for automobile brake repair have changed considerably over the years. In the 1970s, use of compressed air to clean brake surfaces was commonplace (Rohl et al., 1976). While effective at quickly preparing surfaces for repair, this practice caused brake dust and other material to become airborne, leading to potential asbestos exposures among workers and ONUs. The practice also caused asbestos-containing dust to settle at locations throughout the workplace, which became a source of future exposure.

Concerns about asbestos exposure during brake repair led NIOSH to perform a series of industrial hygiene evaluations in the late 1980s to investigate the effectiveness of different dust control strategies. Based on the results of these studies and other factors, OSHA amended its asbestos standard in 1994 to require workers performing brake repair and replacement tasks to control dusts (Federal Register, 1994). OSHA's standard established acceptable work practices for brake and clutch repair, with the extent of controls depending on the number of jobs performed per week. Examples of acceptable work practices for brake dust removal include: use of a negative pressure enclosure equipped with a HEPA-filtered vacuum, use of low-pressure wet cleaning methods, and use of wet wipe methods (Federal Register, 1994). This regulation is an important consideration for interpreting worker exposure studies because observed exposure levels prior to promulgation of OSHA's amended asbestos standard may not be representative of exposures at establishments that currently comply with OSHA requirements.

2. The second important consideration for exposure assessment is the asbestos content in brake dust. Due to the high friction environment in vehicle braking, asbestos fibers in the brake material degrade both chemically and physically. 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. In 1989, NIOSH reviewed brake dust sampling data and concluded "the vast majority of samples" reviewed contained less than 5 percent asbestos (OSHA, 2006). 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.

The amount of time that workers repair and replace automobile brakes depends on many factors. The literature suggests that a typical "brake job" for a single vehicle takes between 1 and 2 hours (Paustenbach et al., 2003). While most automotive mechanics perform various repair tasks, some specialized mechanics work exclusively on brakes. The literature also suggests that the number of brake repair jobs performed by automotive service technicians and mechanics range from 2 to 40 per week (Madl et al., 2008).

#### Automobile Clutch Repair and Replacement

Repairing and replacing asbestos-containing clutch assemblies could also result in asbestos exposure. Workers typically elevate vehicles to access the clutch assembly, remove dust and debris, and perform repair and replacement tasks accordingly. Like asbestos in brakes, asbestos in clutch discs degrades with use. (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).

3321  
3322 However, clutch repair and replacement differ from brake work in two important ways. First, clutches  
3323 generally do not need to be repaired as frequently. By estimates made in 2008, clutches typically last  
3324 three times longer than brake linings ([Cohen and Van Orden, 2008](#)). Second, a common clutch repair  
3325 method is to remove and replace the entire clutch assembly, rather than replacing the clutch disc  
3326 component ([Cohen and Van Orden, 2008](#)). These two factors likely result in clutch repair asbestos  
3327 exposures being lower than comparable brake repair asbestos exposures.

3328 **2.3.1.7.3 Number of Sites and Potentially Exposed Workers – Aftermarket**  
3329 **Automotive Brakes/Linings and Clutches**

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3330 EPA considered several data sources when estimating the number of workers directly exposed to  
3331 asbestos when working with aftermarket automotive products. In the late 1980s, NIOSH conducted a  
3332 series of industrial hygiene surveys on brake repair facilities, and the Agency estimated that 155,000  
3333 brake mechanics and garage workers in the United States were potentially exposed to asbestos ([OSHA,](#)  
3334 [2006](#)). In 1994, OSHA estimated as part of its updated asbestos rulemaking that 676,000 workers  
3335 performed automotive repair activities, and these workers were found in 329,000 establishments (i.e.,  
3336 approximately two workers per establishment) (Federal Register, 1994). EPA considers the best current  
3337 estimate of this worker population to be from the Bureau of Labor Statistics, which estimates that  
3338 749,900 workers in the United States were employed as automotive service technicians and mechanics  
3339 in 2016 ([U.S. BLS, 2019](#)). This includes workers at automotive repair and maintenance shops,  
3340 automobile dealers, gasoline stations, and automotive parts and accessories stores.

3341  
3342 ONU exposures associated with automotive repair work are expected to occur because automotive repair  
3343 and maintenance tasks often take place in large open bays with multiple concurrent activities. EPA did  
3344 not locate published estimates for the number of ONUs for this COU. However, consistent with the  
3345 industry profile statistics from OSHA’s 1994 rulemaking, EPA assumes that automotive repair  
3346 establishments, on average, have two workers who perform automotive repair activities. Accordingly,  
3347 EPA estimates that this COU has 749,900 ONUs.

3348 **2.3.1.7.4 Occupational Inhalation Exposures – Aftermarket Automotive**  
3349 **Brakes/Linings and Clutches**

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3350 To identify relevant occupational inhalation exposure data, EPA reviewed reasonably available  
3351 information from OSHA, NIOSH, and other literature. All research steps are documented below, with  
3352 more detailed discussion on the most relevant data sources, which EPA determined to be the post-1980  
3353 studies conducted by NIOSH and the post-1980 publications in the peer-reviewed literature.

3354  
3355 Automobile Brake Repair and Replacement

3356 EPA first considered worker exposure data from OSHA compliance inspections. EPA reviewed data that  
3357 OSHA provided for 2011 to 2016 inspections, but these data did not include any PBZ asbestos  
3358 measurements for the automotive repair and maintenance industry. For additional insights into OSHA  
3359 sampling results, EPA considered the findings published by Cowan *et al.* ([2015](#)). These authors  
3360 summarized OSHA workplace compliance measurements from 1984 to 2011, which included 394 PBZ  
3361 samples obtained from workers at automotive repair, services, and parking facilities ([Cowan et al.,](#)  
3362 [2015](#)). Because the samples were taken for compliance purposes, all measurements were presumably  
3363 made using OSHA-approved methods (i.e., PCM analyses of filters). Table 2-14. summarizes these data,  
3364 which suggest that asbestos exposures for this COU decreased from the mid-1980s to 2011.

**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.

EPA then considered relevant NIOSH publications, focusing on those published since 1980, because earlier publications evaluated work practices (e.g., compressed air blowdown of brake dust) that are no longer permitted. 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). The NIOSH studies investigated PBZ asbestos exposures among workers who employed various dust removal methods while servicing brakes. These methods included use of vacuum enclosures, HEPA-filtered vacuums, wet brushing, and aerosol sprays. In three of the NIOSH studies, the average (arithmetic mean) asbestos concentration over the 2-hour duration of brake repair jobs was below the detection limit (0.004 fibers/cc). The other two studies reported average (arithmetic mean) asbestos concentrations over the brake job duration of 0.006 fibers/cc and 0.007 fibers/cc. NIOSH’s summary of the five studies concluded that “exposures can be minimal” provided workers use proper dust control methods (OSHA, 2006).

EPA also considered the published literature on asbestos exposures associated with automobile brake repair. This review focused on post-1980 publications that reported original asbestos PBZ measurements for business establishments in the United States. Three publications met these criteria (all were given a high rating in the data evaluation; see supplemental file (U.S. EPA, 2019f)):

- The first study was published in 2003, but it evaluated asbestos exposure for brake repair jobs conducted on vehicles with model years 1965-1968. The study considered work practices commonly used during the 1960s, such as compressed air blowdowns and arc grinding and sanding of surfaces (Blake et al., 2003). 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. This range of measurements was for sample durations ranging from 30 minutes to 107 minutes. These observations were considered in the occupational exposure evaluation even though they likely represent an upper-bound estimate of today’s exposures.
- The second study, conducted in 2008, measured worker asbestos exposure during the unpacking and repacking of boxes of asbestos-containing brake pads and brake shoes (Madl et al., 2008). The asbestos-containing brake materials were originally manufactured for 1970-era automobiles, and the authors obtained the materials from vintage parts suppliers and repair facilities. The study evaluated how exposure varied with several parameters, including type of brake material (e.g., drum, shoe) and worker activity (e.g., packing, unpacking, cleaning). The range of personal

breathing zone concentrations observed across 70 short-term samples was 0.032 fibers/cc to 0.836 fibers/cc, with the highest exposure associated with unpacking and packing 16 boxes of asbestos-containing brake pads over approximately 30 minutes. EPA used bystander measurements from this study to assess ONU exposures for this COU.

- The third study examined asbestos exposures during brake repair operations, considering various worker activities ([Weir et al., 2001](#)). EPA did not use this study’s measurements in the occupational exposure evaluation because the publication lacked details necessary for a thorough review. For instance, this study (in contrast to all others considered) did not report on the complete data set, the time-weighted average exposure values did not include an exposure duration, and the TEM metrics were qualitative and vague. For these and other reasons, the study was considered for contextual information, but not quantitatively in the exposure assessment.

Automobile Clutch Repair and Replacement

EPA considered the same automotive brake repair and replacement information sources when assessing asbestos exposure during automobile clutch repair and replacement but did not identify relevant data from OSHA monitoring data or NIOSH publications. EPA identified three peer-reviewed publications ([Blake et al., 2008](#); [Cohen and Van Orden, 2008](#); [Jiang et al., 2008](#)) that measured worker asbestos exposure during automotive clutch repair. Though the clutch repair data are limited in comparison to brake repair exposure data, the three studies suggest that worker asbestos exposure while repairing or replacing asbestos-containing clutches are lower than corresponding exposures for brake repair and replacement activity. As noted earlier, EPA used the available brake repair data as its basis for deriving exposure estimates for the entire COU of working with aftermarket automotive parts.

**2.3.1.7.5 Exposure Data for Use in Risk Evaluation – Aftermarket Auto Brakes/Linings and Clutches**

Table 2-15. presents the asbestos exposure data that EPA used in the risk evaluation for working with asbestos-containing aftermarket automotive parts. EPA’s basis for selecting the data points appears after the table.

**Table 2-15. Summary of Asbestos Exposures During Replacement of Aftermarket Automotive Parts Used in EPA’s Risk Evaluation**

Occupational Exposure Scenario	Exposure Levels (fibers/cc)					
	Workers			ONUs		
	Central Tendency	High-end	Confidence Rating	Central Tendency	High-end	Confidence Rating
Repairing or replacing brakes with asbestos-containing aftermarket automotive parts: 8-hour TWA exposure	0.006	0.094	Medium	0.0007	0.011	Medium
Repairing or replacing brakes with asbestos-containing aftermarket automotive parts: short-term exposure	0.006	0.836	Medium	0.0007	0.100	Medium

3443 Worker Exposures

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- The central tendency short-term TWA exposure value for workers is based on the seven studies found to include relevant measurements ([Madl et al., 2008](#); [Blake et al., 2003](#); [Cooper et al., 1988, 1987](#); [Godbey et al., 1987](#); [Sheehy et al., 1987a](#); [Sheehy et al., 1987b](#)). For each study, EPA identified the central tendency short-term exposure, which was either reported by the authors or inferred from the range of data points, and the value in Table 2-15. (0.006 fibers/cc) is the median of those central tendencies. Most of the studies selected for review do not present 8-hour TWA exposure values. They instead typically report “brake job TWA exposures”—or exposures that occur over the duration of a single brake repair activity. 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.
  - The high-end short-term TWA exposure value for workers (0.836 fibers/cc) is the highest short-term personal breathing zone observation among the seven studies that met the review criteria ([Madl et al., 2008](#)). The high-end 8-hour exposure value for workers (0.094 fibers/cc) is based on a study ([Blake et al., 2003](#)) that used arc grinding during brake repair with no exposure controls, which is a representation of a high-end exposure scenario of today’s work practices.

3464 ONU Exposures

3465 EPA has not identified data on potential ONU inhalation exposures from after-market auto brake

3466 scenarios. ONUs do not directly handle brakes and the ONU exposure estimates in Table 2-15. were

3467 generated by assuming that asbestos concentrations decreased by a factor of 8.4 between the worker

3468 location and the ONU location. EPA derived this reduction factor from a publication ([Madl et al., 2008](#))

3469 that had concurrent worker and bystander exposure measurements where the bystander was

3470 approximately 5 feet from the worker. The value of 8.4 is the average concentration reduction across

3471 four concurrent sampling events.

3472

3473 **2.3.1.7.6 Data Assumptions, Uncertainties and Level of Confidence**

3474 The universe of automotive repair establishments in the United States is expected to have large

3475 variability in the determinants of exposure to asbestos during brake repair. These exposure determinants

3476 include, but are not limited to, vehicle age, type of brake assembly (disc vs. drum), asbestos content of

3477 used and replacement parts, dust control measures used, number of vehicles serviced per day, and

3478 duration of individual repair jobs. It is uncertain if the studies EPA cited for exposure data fully capture

3479 the distribution of determinants of exposure of current automotive brake jobs, and some of the studies

3480 reviewed for this draft risk evaluation are based on practices that are not widely used today.

3481

3482 PCM-based personal exposure measurement in an automotive repair facility may overstate asbestos

3483 exposures, which some studies have demonstrated through TEM analyses of filter samples ([Blake et al.,](#)

3484 [2003](#); [Weir et al., 2001](#)). PCM measurements are based entirely on dimensional criteria and do not

3485 confirm the presence of asbestos, as can be done through supplemental analyses by TEM or another

3486 confirmatory method. Automotive repair facilities involve many machining operations that can release

3487 non-asbestos airborne fibers, such as cellulose fibers from brushes and metal and plastic fragments from

3488 body repair ([Blake et al., 2008](#)).

3489

3490 EPA considered the quality and uncertainties of the data to determine a level of confidence for the  
 3491 assessed inhalation exposures for this condition of use. The primary strength of this assessment is the  
 3492 use of monitoring data, which is the highest approach of the inhalation exposure assessment approach  
 3493 hierarchy. The overall confidence ratings from systematic review for these data were high. The  
 3494 monitoring data were all collected from U.S.-based vehicular maintenance and repair shops. While these  
 3495 studies were conducted after the implementation of the OSHA rule, many of the studies were conducted  
 3496 in the late 1980s and may not be representative of current operations.

3497  
 3498 Based on these strengths and limitations of the data, the overall confidence for EPA’s assessment of  
 3499 occupational inhalation exposures for this scenario is medium.

**2.3.1.8 Other Vehicle Friction Products**

3500 While EPA has verified that U.S. automotive manufacturers are not installing asbestos brakes on new  
 3501 cars for domestic distribution, EPA has identified a company that is importing asbestos-containing  
 3502 brakes and installing them in their cars in the United States. These cars are exported and not sold  
 3503 domestically.  
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3505  
 3506 In addition, there is a limited use of asbestos-containing brakes for a special, large transport plane (the  
 3507 “Super-Guppy”) by the National Aeronautics and Space Administration (NASA) that EPA has recently  
 3508 learned about. In this public draft risk evaluation, EPA is providing preliminary information for public  
 3509 input and the information is provided in a brief format.

**2.3.1.8.1 Installing New Brakes on New Cars for Export Only**

3510  
 3511 EPA did not identify any studies that contain exposure data related to installation of asbestos-containing  
 3512 brakes from an Original Equipment Manufacturer (OEM). As a result, the exposure assessment  
 3513 approach used for the aftermarket automotive brakes/linings and clutches described in Section 2.3.1.7  
 3514 was also used for this COU and is reported here in Table 2-16.  
 3515

3516  
 3517 Most, if not all, of the literature that EPA reviewed pertained to servicing vehicles that were already  
 3518 equipped with asbestos-containing brakes and clutches; requiring the removal of asbestos-containing  
 3519 parts and installing non-asbestos-containing replacement parts. When removing an asbestos-containing  
 3520 part, one of the main sources of exposure is the dust and debris that must be removed from the brake  
 3521 housing, which is not the case for installing OEM asbestos-containing components on new vehicles.  
 3522 Therefore, the aftermarket auto brakes/linings and clutches exposure value used to assess this COU may  
 3523 be an overestimate. The actual exposure for OEM installation is likely to be lower.  
 3524

**Table 2-16. Other Vehicle Friction Products Exposure Levels (from Aftermarket Automotive Parts exposure levels)**

Occupational Exposure Scenario	Exposure Levels (fibers/cc)					
	Workers			ONUs		
	Central Tendency	High-end	Confidence Rating	Central Tendency	High-end	Confidence Rating
Installing brakes with asbestos-containing automotive parts: 8-hour TWA exposure	0.006	0.094	Low	0.0007	0.011	Low



Installing brakes with asbestos-containing automotive parts: short-term exposure	0.006	0.836	Low	0.0007	0.100	Low
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Data Assumptions, Uncertainties and Level of Confidence

The assumptions and uncertainties described above under Section 2.3.1.7 apply here. In addition, the procedure for installing asbestos containing brakes/friction products into a new vehicle does not involve removing of old asbestos-containing brakes/friction products. Thus, the actual exposure is likely to be much lower than estimated here.

Based on these strengths and limitations of the underlying data described above and in Section 2.3.1.7, the overall confidence for EPA’s assessment of occupational inhalation exposures for this scenario is low.

**2.3.1.8.2 Use of Brakes/Frictional Products for a Single, Large Transport Vehicle (NASA Super-Guppy)**

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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). The SGT aircraft (Figure 2-11) is a specialty cargo plane that transports oversized equipment, and it is considered a mission-critical vehicle (NASA, 2020b). The aircraft brake blocks contain chrysotile asbestos, and this section evaluates potential worker exposures associated with servicing the brakes. All observations in this section are based on information provided by NASA.



**Figure 2-11. NASA Super Guppy Turbine Aircraft  
Photograph courtesy of NASA**

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*Aircraft and Brake Description*

Only one SGT aircraft is in operation today, and NASA acquired it in 1997. The SGT aircraft averages approximately 100 flights per year (NASA, 2020a). When not in use, it is hangered at the NASA

3555 Aircraft Operating Division's (AOD) El Paso Forward Operating Location in El Paso, Texas. This is  
3556 also where the aircraft is serviced ([NASA, 2020b](#)).

3557  
3558 The SGT aircraft has eight landing gear systems, and each system has 32 brake blocks. The individual  
3559 blocks (Figure 2-12) contain 43 percent chrysotile asbestos; and they are 4 inches long, 4 inches wide,  
3560 and 1 inch thick ([NASA, 2020b](#)). Each brake block weighs approximately 12.5 ounces.  
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3562  
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3564 **Figure 2-12. Brakes for NASA Super Guppy Turbine Aircraft**  
3565 **Photograph courtesy of NASA**  
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3567 *Worker Activities*

3568 Replacing asbestos-containing brake blocks is the principal worker activity potentially associated with  
3569 asbestos exposure, and this task is performed by four certified technicians. According to NASA, the  
3570 brake blocks are not replaced due to excessive wear; rather, they are typically replaced because they  
3571 have become separated from the brake system or because they have become covered with hydraulic  
3572 fluid or other substances ([NASA, 2020a](#)). This is an important observation, because in EPA's judgment,  
3573 worn brake blocks would be more likely to contain dusts to which workers would be exposed.  
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3575 In materials provided to EPA, NASA described the process by which workers replace brake blocks. This  
3576 process begins by removing the brakes from the landing gear. To do so, the SGT aircraft is raised at the  
3577 axle pads, and the landing gear is opened to allow workers access to the individual brake systems. The  
3578 workers remove the brakes from the aircraft and clean the brakes at an outdoor wash facility.  
3579

3580 The certified technicians then take the breaks into a ventilated walk-in booth (Figure 2-13), which is  
3581 where brake block replacement occurs. According to a NASA job hazard analysis, workers use wet  
3582 methods to control release of asbestos dust during this task ([NASA, 2020a](#)). The workers use spray  
3583 bottles containing a soap-water mixture to keep exposed surfaces damp when replacing brake blocks.  
3584 Waste dusts generated during this activity are collected using a high-efficiency particulate air vacuum;  
3585 and all asbestos-containing wastes, including vacuumed waste, are double-bagged ([NASA Occupational](#)  
3586 [Health, 2020](#)) and disposed of according to waste management regulations for asbestos ([NASA, 2020b](#)).  
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**Figure 2-13. Ventilated Walk-in Booth Where Brakes Pads Are Replaced**  
Photograph courtesy of NASA

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3593 The four certified technicians for SGT aircraft brake replacement receive annual training on asbestos.  
3594 The training course addresses asbestos health hazards, work practices to reduce generation of airborne  
3595 asbestos dust, and information on how PPE can reduce exposures ([NASA Occupational Health, 2020](#)).  
3596 The training also indicates that brake replacement workers who follow proper methods for controlling  
3597 asbestos dust releases are not required to use respiratory protection ([NASA Occupational Health, 2020](#)).  
3598 Respirator usage is also not required because measured exposures were below applicable occupational  
3599 exposure limits ([NASA, 2020a](#)). Despite respiratory protection not being required, NASA informed  
3600 EPA that some certified technicians choose to use half mask air-purifying respirator with P-100  
3601 particulate filters when replacing brake blocks ([NASA, 2020a](#)).  
3602

3603 Brake pad replacement for the one SGT aircraft occurs infrequently, approximately four times per year  
3604 ([NASA, 2020a](#)). According to NASA, the four certified technicians who service the aircraft spend  
3605 approximately 12 hours per year replacing brake pads.  
3606

#### 3607 *Number of Sites and Potentially Exposed Workers*

3608 Brake pad replacement for the SGT aircraft occurs at only one site nationwide: a NASA facility located  
3609 in El Paso, Texas ([NASA, 2020b](#)).  
3610

3611 Over the course of a year, only four certified technicians at this location perform brake pad replacement;  
3612 and one or two of these technicians will perform individual brake pad replacements ([NASA, 2020b](#)).  
3613 Because the brake replacement work occurs in a ventilated walk-in booth, asbestos fibers likely are not  
3614 released into the general workspace where ONUs may be exposed.  
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3616 Therefore, for this condition of use, EPA assumes four workers may be exposed, and no ONUs are  
3617 exposed.  
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#### 3619 *Worker Inhalation Exposures*

3620 EPA's estimate of occupational inhalation exposures for this condition of use are based on five worker  
3621 exposure samples that NASA collected in 2014 ([NASA, 2020a](#)). The sampling was conducted according  
3622 to NIOSH Method 7400, and asbestos was not found above the detection limit in any of the samples.  
3623 EPA estimated worker exposure levels for the risk evaluation as follows:

- 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 the detection limit and calculated the arithmetic mean of those three value. By this approach, EPA calculated a central tendency concentration of <0.003 fibers/cc. For the high-end full shift exposure estimate, EPA used the highest detection limit across the three samples.
- Two of the five sampling results that NASA provided were labeled as being evaluated for “30-minute excursion limits”; and EPA considered these to be representative of short-term exposures. The two results, based on sampling durations of 30 and 35 minutes, were: <0.044 fibers/cc and <0.045 fibers/cc. Following the same approach that was used for full shift exposures, EPA estimated a central tendency short-term exposure of <0.022 fibers/cc and a high-end short-term exposure of <0.045 fibers/cc.

Based on these assumptions, EPA will use these exposure values in this risk evaluation:

- Full Shift: Central Tendency – <0.003 f/cc
- Full Shift: High-End – <0.0089 f/cc
- Short-Term: Central Tendency – <0.022 f/cc
- Short-Term: High-End – <0.045 f/cc

EPA assigned a confidence rating of “high” for these exposure data. This rating was based on the fact that monitoring data are available from the one site where this condition of use occurs. Further, replacement of SGT aircraft brake blocks occurs approximately 12 hours per year, and the five available sampling events spanned more than 4 hours. Therefore, the available data, which were collected using an appropriate NIOSH method, represent almost one-third of the worker activity over an entire calendar year. The spatial and temporal coverage of these data are greater than those for any other condition of use in this risk evaluation.

#### *ONU Inhalation Exposures*

As noted previously, EPA assumes no ONU exposures occur, because the worker activity with the highest likelihood of releasing asbestos occurs in a walk-in ventilated booth, where ONUs are not present.

### **2.3.1.9 Other Gaskets-Utility Vehicles (UTVs)**

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#### **2.3.1.9.1 Process Description – UTV Gasket installation/Serviceing**

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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.

To derive occupational exposure values for this risk evaluation, EPA is drawing on a review of several studies in the literature which characterize exposure scenarios from asbestos-containing gasket replacement in different types of vehicles.

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**2.3.1.9.2 Worker Activities – UTV Gasket Installation/Service**

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The UTV manufacturers receive the pre-cut gaskets which are then installed during manufacture of the UTV. The gaskets may be removed during servicing of the exhaust system.

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Thirty studies relating to gasket repair/replacement were identified and reviewed as part of the systematic review process for the consumer exposure scenario (see Section 2.3.2.2); resulting in identifying three studies as being relevant to gasket installation and replacement in vehicles (see Table 2-29).

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**2.3.1.9.3 Number of Sites and Potentially Exposed Workers – UTV Gasket Installation/Service**

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EPA estimated the number of UTV service technicians and mechanics potentially exposed to asbestos by assuming that asbestos-containing gaskets are most likely to be replaced at UTV dealerships that sell these vehicles.<sup>8</sup> However, no NAICS codes are specific to UTV dealers. These establishments are classified under the 4-digit NAICS 4412, “Other Motor Vehicle Dealers.” Table 2-17. lists the specific industries included in that 4-digit NAICS. The industry most relevant to UTV dealers is the 7-digit NAICS code 4412281, “Motorcycle, ATV, and personal watercraft dealers.” The 2012 Economic Census reports 6,999 establishments in this industry.

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**Table 2-17. Number of Other Motor Vehicle Dealers**

2012 NAICS code	2012 NAICS Code Description	Number of Establishments
<b>4412</b>	<b>Other motor vehicle dealers</b>	<b>14,249</b>
44121	Recreational vehicle dealers	2,605
441222	Boat dealers	4,645
<b>441228</b>	<b>Motorcycle, ATV, and all other motor vehicle dealers</b>	<b>6,999</b>
4412281	Motorcycle, ATV, and personal watercraft dealers	5,098
4412282	All other motor vehicle dealers	1,901
Source: (U.S. Census Bureau, 2016a).		

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The Economic Census also reports the product and service line statistics for retail establishments down to the 6-digit NAICS code level. Product and service code 20593 represents “All-terrain vehicles (ATVs) and personal watercraft.” Out of the 6,999 establishments in the 6-digit NAICS code 441228, Table 2-18. shows that 2,989 of them deal in ATVs and personal watercraft. For purposes of this assessment, EPA assumes that approximately half of them (1,500 establishments, see Table 2-18.) sell and repair UTVs and ATVs, and that the other half specialize in personal watercraft.

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<sup>8</sup> While UTV owners may have their vehicles serviced at repair and maintenance shops that are not part of dealerships, the total number of sites and workers exposed may not necessarily change from the estimates in this analysis. More vehicles being repaired in other types of repair shops would mean fewer vehicles being repaired (and fewer workers exposed) in dealerships. This analysis simplifies the estimates by assuming that engine repairs all occur at dealerships.

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**Table 2-18. Number of ATV and Watercraft Dealers in NAICS 44128**

2012 NAICS Code	2012 NAICS Code Description	Products and Services Code	Products and Services Code Description	Number of Establishments
441228	Motorcycle, ATV, and all other motor vehicle dealers	20593	All-terrain vehicles (ATVs) & personal watercraft	2,989
Source: ( <a href="#">U.S. Census Bureau, 2016b</a> ).				

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**Table 2-19. Estimated Number of UTV Dealers**

Description	Number of Establishments
Estimated number of dealerships repairing and maintaining UTVs/ATVs	1,500

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The next step in estimating potentially exposed workers is to determine the number of workers engaged in UTV repairs. This number had to be estimated because the Bureau of Labor Statistics does not provide employment data by occupation for NAICS 4412281 and because Standard Occupational Classification (SOC) codes are not specific to workers engaged in UTV repairs. Reasonably available information to estimate potentially exposed workers is SOCs at the 4-digit NAICS level (NAICS 4412), which includes dealers in recreational vehicles, boats, motorcycles and ATVs. Table 2-20. presents SOCs that reflect the types of workers that may repair engines and identifies 41,930 workers in relevant occupations in NAICS 4412.<sup>9</sup>

**Table 2-20. Selected Mechanics and Repair Technicians in NAICS 4412 (Other Motor Vehicle Dealers)**

Occupation (SOC code)	Employment
First-Line Supervisors of Mechanics, Installers, and Repairers (491011)	4,140
Aircraft Mechanics and Service Technicians (493011)	120
Automotive Service Technicians and Mechanics (493023)	3,360
Motorboat Mechanics and Service Technicians (493051)	9,800
Motorcycle Mechanics (493052)	13,250

<sup>9</sup> This count excludes occupations in NAICS 4412 that are less likely to engage in engine repair involving gaskets similar to those found in UTVs. Thus, Table 4 does not include occupations such as Electrical and Electronic Equipment Mechanics, Installers, and Repairers (SOC 492000), Automotive Body and Related Repairers (SOC 493021), Mobile Heavy Equipment Mechanics, Except Engines (SOC 493042), Tire Repairers and Changers (SOC 493093) and Outdoor Power Equipment and Other Small Engine Mechanics (SOC 493053). The latter covers workers who repair items such as lawn mowers, chain saws, golf carts, and mobility scooters, which do not generally have engines similar to UTVs.

Occupation (SOC code)	Employment
Recreational Vehicle Service Technicians (493092)	11,260
<b>Total</b>	<b>41,930</b>
Source: ( <a href="#">U.S. BLS, 2019</a> ).	

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Based on the estimates for NAICS 4412 in Table 2-17. and Table 2-20., Table 2-21. calculates that across all entities in NAICS 4412, approximately 3 employees per dealership engage in occupations potentially relevant to UTV repairs.

**Table 2-21. Number of Employees per Establishment in NAICS 4412 in Relevant Occupations**

Number of other motor vehicle dealers (NAICS 4412) (see Table 2-17.)	14,429 establishments
Number of mechanics and repair technicians in NAICS 4412 that may repair engines in recreational vehicles, boats, motorcycles, ATVs, etc. (see Table 2-20.)	41,930 employees
<b>Estimated average number of employees per establishment that may repair motor vehicle engines (calculated as 41,930 divided by 14,429)</b>	<b>~3 employees per establishment</b>

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Assuming that the average number of mechanic and service technicians across NAICS 4412 is applicable to NAICS 4412281, Table 2-22. combines the estimate of 1,500 dealerships repairing and maintaining UTVs/ATVs from Table 2-19. Estimated Number of UTV Dealers with the estimated average of 3 employees per establishment from Table 2-21. to generate an estimate of 4,500 total employees that may repair UTV engines.

**Table 2-22. Estimated Number of Sites and Employees for UTV Engine Repair**

Description	Number of establishments
Estimated number of dealerships repairing and maintaining UTVs/ATVs (see Table 2-19. Estimated Number of UTV Dealers)	1,500
Estimated average number of employees per establishment that may repair motor vehicle engines (see Table 2-21.)	3
Estimated total number of employees that may repair UTV	4,500

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#### **2.3.1.9.4 Occupational Inhalation Exposures for Use in Risk Evaluation - UTV Gasket Installation/Serviceing**

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No information from OSHA, NIOSH, or the scientific literature was available on occupational exposures to asbestos associated with installing and servicing gaskets in UTVs. EPA therefore considered studies of similar worker exposure scenarios to use as a surrogate. Multiple publications (see Section 2.3.2.2) report on occupational exposures associated with installing and servicing gaskets in automobiles. However, EPA located only one study ([Paustenbach et al., 2006](#)) that examined exposures associated with replacing vehicle exhaust systems, which is the UTV component where asbestos-containing gaskets are found. Therefore, EPA based its occupational inhalation exposure assessment for UTV gasket installation and servicing on this study.

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Worker Exposures

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.

The study considered multiple types of exhaust system projects, including removal of different combinations of mufflers, exhaust pipes, and exhaust manifolds and conversion from single to dual exhaust systems. The time needed to remove an exhaust system and install a new one lasted up to 4 hours, but workers reportedly spent less than one minute handling or coming into contact with gaskets. All jobs were performed indoors at the muffler shop, with service bay doors closed, and no other vehicle repair work occurring at the same time.

Personal breathing zone measurements were taken using sampling materials consistent with NIOSH Method 7400. Overall, 23 valid personal breathing zone samples were collected from mechanics and tested with PCM. Some additional samples were taken, but they were overloaded with particulate material and could not be analyzed. Among the 23 valid samples, 17 were non-detect for asbestos by PCM analysis; and 6 samples contained asbestos at concentrations up to 0.0505 fibers/cc. The TEM analyses identified asbestos fibers in 7 of the sampling filters.

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. (Note: 1) The authors reported an average “PCM-adjusted” concentration that is 18 percent lower than the un-adjusted result. The adjustment accounts for the amount of fibers confirmed by TEM as being asbestos. 2) This appears to be a detection level 0.132 f/cc divided by two, contrary to more standard division by square root of two (approximately 1.4), thus underestimating the maximum concentration. The average and maximum concentrations pertain to the times when sampling occurred, and sampling durations ranged from 9 to 65 minutes. The study authors calculated an 8-hour TWA exposure concentration of 0.01 fibers/cc, based on a worker performing four exhaust system removal tasks in one shift.

EPA used the personal breathing zone (PBZ) values for the worker as follows: the last row in Table 2-30 shows the maximum concentration calculated from the information within the study ([Paustenbach et al., 2006](#)) as the high-end estimated concentration for the worker and the mean concentration calculated from the information within the study as the central tendency concentration (see Table 2-23 below).

**Table 2-23. UTV Gasket Installation/Service Exposure Levels for EPA’s Risk Evaluation**

Occupational Exposure Scenario	8-hr TWA Exposure Levels (fibers/cc)					
	Asbestos Worker			ONU		
	Central Tendency	High-end	Confidence Rating	Central Tendency	High-end	Confidence Rating
UTV	0.024	0.066	Medium	0.005	0.015	Medium

ONU Exposures



3792  
3793 The same publication ([Paustenbach et al., 2006](#)) includes area sampling results that EPA found  
3794 appropriate for ONU exposures (rather than what the paper defines as a bystander). These samples were  
3795 collected at breathing zone height at locations near the ends of the muffler shop bays where the exhaust  
3796 system work was performed. The area sample durations ranged from 25 to 80 minutes, and these  
3797 samples were collected during exhaust system work. Overall, 21 area samples from these locations were  
3798 analyzed by PCM; and 16 of these samples were non-detects for asbestos. Among the PCM data from  
3799 this subset of area samples, the authors report that the average asbestos concentration was 0.005  
3800 fibers/cc and the maximum asbestos concentration was 0.015 fibers/cc. The study authors did not report  
3801 8-hour TWA concentrations for the area sample locations. EPA used these average and maximum  
3802 asbestos concentrations to characterize ONU exposures.  
3803

#### 3804 **2.3.1.9.5 Data Assumptions, Uncertainties and Level of Confidence**

3805 A principal assumption made in this assessment is that worker asbestos exposures for removing  
3806 automobile exhaust systems are representative of worker asbestos exposures associated with installing  
3807 and servicing gaskets found in UTV exhaust systems. Further, this assessment assumes that data from  
3808 one publication ([Paustenbach et al., 2006](#)) are representative of exposures for this condition of use.  
3809 However, the job activities and exposure scenarios considered in the publication differ from the UTV-  
3810 related exposures in at least two ways.

3811  
3812 First, the publication used in this analysis ([Paustenbach et al., 2006](#)) considered older automobiles. This  
3813 focus was intentional, because newer vehicles generally do not have asbestos-containing exhaust  
3814 systems. However, all vehicles considered in the study were more than 35 years old at the time the  
3815 research was published. According to the study, the highest concentrations of asbestos in the removed  
3816 gasket was 35.5 to 48.9 percent. It is unclear if the asbestos content in the automobile exhaust systems  
3817 from pre-1970 automobiles are representative of the asbestos content in today's UTV exhaust systems.  
3818

3819 Second, because the study considered vintage automobiles that presumably contained older parts, it is  
3820 likely that the asbestos-containing gaskets in the exhaust systems had worn down with use and time.  
3821 These older gaskets presumably would be more prone to release fibrous asbestos into the air, as  
3822 compared to newer gaskets (which typically are pre-formed with the asbestos encapsulated in a binding  
3823 agent or some other matrix) ([Paustenbach et al., 2006](#)). Therefore, the asbestos concentrations measured  
3824 during the study may overstate the concentrations that might occur during UTV exhaust system  
3825 servicing.  
3826

3827 Additionally, EPA identified two sources of uncertainty pertaining to the data analysis. One pertains to  
3828 the uncertainties associated with non-detect observations. For the average worker exposure  
3829 concentration, 74 percent of the samples were non-detects; and the study authors replaced these  
3830 observations with one-half the detection limit when calculating average concentrations (instead of more  
3831 standard division by square root of 2, approximately 1.4). Similarly, for the area sampling results used  
3832 for ONU exposures, 76 percent of the samples were non-detects.  
3833

3834 Moreover, five of the personal breathing zone samples collected from mechanics had filters overloaded  
3835 with particulate, and these samples were not analyzed. The authors noted that the overloaded filters may  
3836 have resulted from particulate matter released while mechanics used torches to cut and weld exhaust  
3837 pipes; but EPA cannot rule out the possibility that these overloaded filters might have contained elevated  
3838 levels of asbestos.  
3839

3840 Based on these strengths and limitations of the data, the overall confidence for EPA’s assessment of  
 3841 occupational inhalation exposures for this scenario is medium.  
 3842

3843 **2.3.1.10 Summary of Inhalation Occupational Exposure Assessment**

3844 Table 2-24. summarizes the inhalation exposure estimates for all occupational exposure scenarios.  
 3845 Where statistics can be calculated, the central tendency estimate represents the 50th percentile exposure  
 3846 level of the available data set, and the high-end estimate represents the 95th percentile exposure level.  
 3847 The central tendency and high exposures for ONU are derived separately from workers, often by using a  
 3848 reduction factor. See the footnotes for an explanation of the concentrations used for each COU.  
 3849  
 3850

**Table 2-24. Summary of Occupational Inhalation Exposures**

Condition of Use	Duration	Type	TWA Exposures, fibers/cc (see footnotes)		
			Central Tendency	High-end	Confidence Rating
Diaphragms for Chlor-Alkali Industry (Processing and Use)	Full Shift	Worker	0.0060 (a)	0.036 (a)	High
		ONU	0.0025 (b)	<0.008 (b)	High
	Short-term	Worker	0.032 (a)	0.35 (a)	Medium
		ONU	No data	No data	-
Sheet gaskets – stamping (Processing)	Full Shift	Worker	0.014 (c)	0.059 (c)	Medium
		ONU	0.0024 (d)	0.010 (d)	Medium
	Short-term	Worker	0.024 (c)	0.059 (c)	Medium
		ONU	0.0042 (d)	0.010 (d)	Medium
Sheet gaskets – use	Full Shift	Worker	0.026 (e)	0.094 (e)	Medium
		ONU	0.005 (d)	0.016 (d)	Medium
	Short-term	Worker	No data	No data	-
		ONU	No data	No data	-
Oilfield brake blocks - Use	Full Shift	Worker	0.03 (f)	No data	Low
		ONU	0.02 (f)	No data	Low
	Short-term	Worker	No data	No data	-
		ONU	No data	No data	-
Aftermarket automotive brakes/linings, clutches (Use and Disposal)	Full Shift	Worker	0.006 (g)	0.094 (g)	Medium
		ONU	0.0007 (h)	0.011 (h)	Medium
	Short-term	Worker	0.006 (g)	0.836 (g)	Medium
		ONU	0.0007 (h)	0.100 (h)	Medium
Other Vehicle Friction Products	Full Shift	Worker	0.006 (g)	0.094 (g)	Medium
		ONU	0.0007 (h)	0.011 (h)	Medium

Condition of Use	Duration	Type	TWA Exposures, fibers/cc (see footnotes)		
			Central Tendency	High-end	Confidence Rating
(brakes installed in exported cars) (Use)	Short-term	Worker	0.006 (g)	0.836 (g)	Medium
		ONU	0.0007 (h)	0.100 (h)	Medium
Other gaskets – UTVs (Use and Disposal)	Full Shift	Worker	0.024 (i)	0.066 (i)	Low
		ONU	0.005 (i)	0.015 (i)	Low
	Short-term	Worker	No data	No data	-
		ONU	No data	No data	-

- 3851 (a) Chronic exposure concentrations for the chlor-alkali industry are based on worker exposure monitoring data. Central  
3852 tendency concentrations are 50<sup>th</sup> percentile values and high-end concentrations are 95<sup>th</sup> percentile values.
- 3853 (b) Short-term exposure concentrations for the chlor-alkali industry are based on area monitoring data. Central tendency  
3854 concentrations are 50<sup>th</sup> percentile values and high-end concentrations are 95<sup>th</sup> percentile values.
- 3855 (c) Concentrations for sheet gasket stampers are based on worker exposure monitoring data (10 samples). For chronic  
3856 exposures, central tendency is the single full-shift TWA data point available; and high-end assumes the highest  
3857 observed short-term exposure persists over an entire shift. For short-term exposures, central tendency is the median  
3858 concentration observed, and high-end is the highest concentration observed.
- 3859 (d) Concentrations for ONUs at sheet gasket stamping facilities and sheet gasket use facilities were estimated by EPA  
3860 using a concentration-decay factor for bystander exposures derived from the literature.
- 3861 (e) Concentrations for sheet gasket use are based on descriptive statistics provided to EPA of 34 worker exposure  
3862 monitoring samples. The central tendency concentration is the arithmetic mean and the high-end concentration is the  
3863 highest measured value.
- 3864 (f) Concentrations for oil field brake blocks are based on two data points—arithmetic mean exposure for different  
3865 worker activities—reported in the scientific literature.
- 3866 (g) Concentrations for aftermarket automotive parts are based on worker exposure monitoring data documented in seven  
3867 studies. For chronic exposures, the central tendency concentration is the median of the arithmetic mean exposure  
3868 values reported across the seven studies; and the high-end concentration is the highest TWA exposure concentration  
3869 reported. For short-term exposures, the same data set was used but data were summarized for individual  
3870 observations, not the full-shift TWA values.
- 3871 (h) Concentrations for ONUs at auto repair facilities were estimated by EPA using a concentration-decay factor for  
3872 bystander exposures derived from the literature, based on studies of this industry.
- 3873 (i) Asbestos air measurements from Paustenbach et al., (2006): Removal and replacement of exhaust system gaskets  
3874 from vehicles manufactured before 1974 with original and old exhaust systems.  
3875

3876 **2.3.2 Consumer Exposures**

3877 This section summarizes the data used for estimating consumer inhalation exposures to asbestos for two  
3878 potential do-it-yourself (DIY) scenarios: (1) brake repair/replacement and (2) gasket repair/replacement  
3879 in Utility Vehicles (UTVs). Specifically, the brake repair/replacement scenario involves repair or  
3880 installation of imported aftermarket brake pads (disc brakes) or brake shoes (drum brakes) containing  
3881 asbestos. The gasket repair/replacement in the UTV scenario involves removal or installation of  
3882 aftermarket gaskets for UTV exhaust systems containing asbestos. Inhalation exposures are evaluated  
3883 for both the individual doing the repair/replacement work and a potential bystander observing the work  
3884 within the immediate area. For each scenario, it is assumed that consumers and bystanders will not be  
3885 wearing any personal protective equipment. The number of consumers impacted by these COUs is  
3886 unknown because the number of products containing asbestos for these COUs is unknown.  
3887

3888 Dermal exposures are not assessed for consumers in this draft risk evaluation. The basis for excluding  
3889 this route is the expected state of asbestos being only solid/fiber phase. While asbestos may deposit on  
3890 open/unprotected skin, it will not absorb into the body through the protective outer skin layers.  
3891 Therefore, a dermal dose resulting from dermal exposure is not expected.

3892  
3893 EPA has found no reasonably available information to suggest that asbestos-containing brakes are  
3894 manufactured in the United States, and based on stakeholder outreach, the Agency does not believe that  
3895 any domestic car manufacturer installs asbestos-containing brakes in new cars sold domestically.<sup>10</sup>  
3896 However, consumers can purchase asbestos-containing brakes as an aftermarket replacement part for  
3897 cars as well as asbestos containing gaskets for UTV exhaust systems.

3898  
3899 The DIY consumer brake assessment and UTV gasket replacement assessment rely on qualitative and  
3900 quantitative data obtained during the data extraction and integration phase of Systematic Review to build  
3901 appropriate exposure scenarios and develop quantitative exposure estimates using personal inhalation  
3902 monitoring data in both the personal breathing zone and the immediate area of the work. The literature  
3903 search resulted in very little information specific to consumer exposures, thus the consumer assessment  
3904 relies heavily on the review of occupational data, and best professional judgment. Many of the studies in  
3905 existing literature are older (dating back to late 1970s). When possible, EPA used the most recent studies  
3906 available and also considered data quality and adequacy of the data. Targeted literature searches were  
3907 conducted as appropriate to augment the initial data obtained and to identify supplemental information  
3908 such as activity patterns and exposure factors specific to consumers.

#### 3909 **2.3.2.1 Consumer Inhalation Exposures of Do-It-Yourself (DIY) Mechanics During** 3910 **Brake Repair: Approach and Methodology**

3911 This consumer assessment addresses potential scenarios in which a DIY consumer installs, repairs or  
3912 replaces existing automobile brakes with imported aftermarket brake pads or shoes containing asbestos;  
3913 including brake linings and clutches. While peer-reviewed literature indicates much of the asbestos  
3914 brake pad or shoe use has been phased out and the majority of existing cars on the road do not have  
3915 asbestos brakes ([Finley et al., 2007](#)), asbestos-containing brakes and shoes can still be purchased in the  
3916 United States. This scenario evaluates potential consumer inhalation exposure to asbestos during  
3917 removal of the old brakes or shoes containing asbestos, cleaning of the brake housing, shoes, and wheel  
3918 assembly, as well as installation and grinding of the newly installed brakes or shoes containing asbestos.

3919  
3920 Brake repair and replacement typically involve several basic steps. For both drum brakes and disc  
3921 brakes, the first step is to access the brake assembly by elevating the vehicle and removing the wheel.  
3922 The next step is to remove the old brake pads or shoes followed by cleaning the brake apparatus using  
3923 various cleaning equipment such as dry or wet brush, wet rag, brake cleaning fluid, or compressed air.  
3924 Although EPA does not recommend the work practice of blowing brakes with compressed air ([U.S.](#)  
3925 [EPA, 2007](#)), there is insufficient information indicating such practice has been fully discontinued by the  
3926 consumer. After the brake apparatus is cleaned, new pads or shoes are installed. In some situations,  
3927 installation of new pads may require additional work such as brake shoe arc grinding. This additional  
3928 work may be more likely when consumers are working on vintage vehicles and brake shoes do not fit  
3929 exactly inside the brake drum.

---

<sup>10</sup> EPA is aware of one car manufacturer who imports asbestos-containing automotive friction products for new vehicles, but those vehicles are then exported and not sold in the United States.

3931 Systematic review of the reasonably available literature on brake repair and replacement resulted in  
3932 insufficient inhalation personal/area monitoring studies specifically for DIY consumer brake repair.  
3933 Therefore, the DIY brake repair/replacement exposure assessment uses surrogate monitoring data from  
3934 occupational brake repair studies. EPA recognizes that brake repair/replacement by a professional  
3935 mechanic may involve the use of different equipment and procedures. Consumer exposure during DIY  
3936 brake repair is expected to differ from occupational brake repair in four ways ([Versar, 1987](#)): (1)  
3937 consumers generally do not have a fully equipped professional garage to perform auto repairs (in some  
3938 cases, the repairs would occur in an enclosed garage); (2) consumers would not wear respirators,  
3939 mitigate dust emissions, or have available the professional equipment found in commercial repair shops;  
3940 (3) consumers have limited experience, and thus the time required to make repairs would be longer; and  
3941 (4) consumers are unlikely to perform more than one brake job per year and it was assumed that only  
3942 one consumer would perform the task of replacing asbestos brakes or shoes. Considering the expected  
3943 differences between brake repair/replacement work conducted by a professional mechanic and a DIY  
3944 consumer, EPA identified several factors to consider during the systematic review process for using  
3945 professional mechanic information as a surrogate for the DIY consumer. The goal was to examine the  
3946 activity patterns monitored in the various occupational studies and only select those studies which are  
3947 expected to represent a DIY consumer scenario.

3948  
3949 Specifically, EPA only considered activity patterns within the various occupational studies  
3950 representative of expected DIY consumer activity patterns and work practices. EPA also considered only  
3951 those studies with information related to typical passenger vehicles (automobiles, light duty trucks,  
3952 mini-vans, or similar vehicle types); it is not expected that a typical DIY consumer would perform brake  
3953 repair/replacement work on heavy duty trucks, tractor trailers, airplanes, or buses. Furthermore,  
3954 consideration was given to reasonably available literature which had monitoring data in the personal  
3955 breathing zone of the potential DIY consumer and area monitoring within a garage. Lastly, EPA  
3956 considered those studies where the work was performed without localized or area engineering controls  
3957 as it is unlikely a DIY consumer will have such controls (e.g., capture hoods, roof vents, industrial  
3958 exhaust fans baghouses, etc.) within their residential garage.

3959  
3960 The following assumptions are used to assess consumer inhalation exposure to asbestos during DIY  
3961 brake repairs:

- 3962  
3963 • Location: EPA presents an indoor and an outdoor scenario for brake repair and  
3964 replacement work. The indoor scenario assumes the DIY brake repair/replacement is  
3965 performed in the consumer's residential garage with the garage door closed. It also  
3966 assumes the additional work associated with this brake work is arc grinding and occurs  
3967 within the garage with the garage door closed. The outdoor scenario assumes the DIY  
3968 brake repair/replacement work is performed in the consumer's residential driveway. It  
3969 also assumes the additional work associated with this brake work is brake filing and  
3970 occurs in the residential driveway.
  - 3971  
3972 • Duration of Activity: Available literature indicates a typical "brake job" for a  
3973 professional brake mechanic for a single vehicle takes between one and two hours  
3974 ([Paustenbach et al., 2003](#)). No data were found in existing literature on the length of  
3975 time needed for a DIY consumer to perform a brake job. EPA assumes a consumer  
3976 DIY brake repair/replacement event could take twice as long as a professional  
3977 mechanic, or about three hours (double the mean of time found in the literature for  
3978 professional mechanics).
- 3979

- 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.
- 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. EPA assumes the consumer performs arc grinding for the indoor scenario and assumes the consumer performs hand filing for the outdoor scenario. Concentrations resulting from brake work including this additional work is utilized as the high-end estimate for consumer exposure. The central tendency is based on changing out brakes only with no additional work.
- Frequency of brake repair jobs: EPA assumes the average consumer performs a single brake repair/replacement job about once every three years. Brakes in cars and small trucks are estimated to require replacement approximately every 35,000 to 60,000 miles (Advance Auto Parts, [website](#) accessed on November 12, 2018). The three-year timeline is derived by assuming the need to replace brakes every 35,000 miles, and an average number of annual miles driven per driver in the United States of 13,476 miles/year (U.S. DOT, 2018). This can vary if the consumer has more than one car or works on vintage cars and that same consumer does all of the brake repair/replacement work for all cars they own.
- Brake type: EPA assumes exposure to asbestos is similar during the replacement of disc brake pads and drum brake shoes.

**2.3.2.1.1 Consumer Exposure Results – Do-It-Yourself (DIY) Mechanics During Brake Repair**

Utilizing the factors and the assumptions discussed above, EPA identified five relevant studies which could be applied to the expected DIY consumer brake repair/replacement scenario. These references as well as the data quality scores are provided in the following table:

**Table 2-25. Summary of Studies Satisfying Conditions/Factors for Use in Consumer DIY Brake Exposure Scenario**

Reference	Occupational Exposures?	Consumer/DIY Exposures?	Data Quality Rating (Score)
( <a href="#">Sheehy et al., 1989</a> )	Yes	Yes	Medium (1.7)
( <a href="#">Blake et al., 2003</a> )	Yes	No	Medium (1.8)
( <a href="#">Paustenbach et al., 2003</a> )	Yes	No	High (1.0)
( <a href="#">Yeung et al., 1999</a> )	Yes	No	Medium (2.0)
( <a href="#">Kakooei et al., 2011</a> )	Yes	No	Medium (2.0)

Monitoring data from two of the five studies (([Sheehy et al., 1989](#)) and ([Blake et al., 2003](#))) were used to evaluate consumer inhalation exposure to asbestos resulting from brake repair/replacement work. These studies were U.S. studies which used standard sampling and analysis methods (including both PCM and TEM analyses) for asbestos. ([Sheehy et al., 1989](#)) provided DIY consumer exposure data for work conducted outdoors (although limited to two samples). Although professional mechanics were conducting the brake repair/replacement work in the ([Blake et al., 2003](#)) study, the work practices utilized by the professional mechanics were comparable to a DIY consumer in that neither engineering controls nor personal protective equipment were used. The third U.S. study ([Paustenbach et al., 2003](#)) was a supplemental study used to inform the length of time it takes a DIY consumer to complete brake repair/replacement work. The final two studies were non-U.S. studies. ([Yeung et al., 1999](#)) was a secondary study and did not provide supplemental/raw data. Additionally, all breathing zone and area samples from this study were below the PCM detection limits. ([Kakooei et al., 2011](#)) had a limited description of the exposure scenario and therefore may not be representative of the expected DIY consumer activity. Neither of these non-US studies will be further described in this risk evaluation.

A brief summary of the two monitoring studies used for this evaluation is provided below.

([Sheehy et al., 1989](#)) measured air concentrations during servicing of rear brakes on a full-size van. The work was performed outdoors, on a drive-way, by a DIY consumer. The DIY consumer wet the drum brake with a spray can solvent to dissolve accumulated grease and dirt. The mechanic then used a garden hose to flush the surfaces with water. The duration of the monitoring activity was not provided.

([Blake et al., 2003](#)) measured air concentrations in the personal breathing zone of professional mechanics performing brake repair/replacement work. ([Blake et al., 2003](#)) evaluated asbestos exposure for brake repair jobs conducted on passenger vehicles from model years 1965-1968. The study sought to use tools and practices common to the mid-1960s for cleaning, repairing, and replacing the brakes. In six separate tests, brake shoe change-outs were conducted on all four wheels of a car which had already been fitted with new asbestos containing brake shoes and then driven for 1,400 miles prior to the monitoring. The monitoring began with driving the test car into the service bay and ended upon return from a test drive after the brake-change out. The total brake change-out monitoring period was 85 to 103 minutes in duration. In general, all tests involved removing the wheel and tire assemblies, followed by the brake drum. The drum was then placed on the concrete floor creating a shock which broke loose the brake dust. Each brake assembly was then blown out using compressed shop air. 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).

These activities encompassed approximately 12.5 minutes, 4.1 minutes, and 9.7 minutes of the monitoring period, respectively. An additional test was conducted during cleaning only (sweeping) for a total of 30 minutes by the mechanic after four brake change test runs. The tests were conducted in a former automobile repair facility (7 bays, volume of 2,000 m<sup>3</sup>) with the overhead garage doors closed. An exhaust fan equipped with a filter was installed 16 meters away from the brake changing area and operated during all brake changes to ventilate the building. However, smoke testing showed no air movements toward the exhaust fans suction beyond 8 meters from the fan. PCM and TEM analyses

were conducted on all samples except for the seventh test; which was cleaning the work area after all brake changes were complete and for which only PCM analysis was conducted.

(Blake et al., 2003) included area sampling collected from seven locations within the building during each test run, including four samples within 3 meters of the vehicle, one sample within 3 meters of the arc grinding station, and two samples >3 meters from the automobile. Background samples were not collected.

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

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.

- 1) All brake work conducted indoors
- 2) All brake work conducted outdoors

The monitoring data extracted from the (Blake et al., 2003) and (Sheehy et al., 1989) studies are presented in Table 2-26. A discussion of this information follows Table 2-26.

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

Study	Activity	Duration (hours)	Concentration (fibers/cc)		Location	Confidence Rating
			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 <sup>a</sup>	0.007	Not monitored <sup>b</sup>	Outdoors	Medium

<sup>a</sup> No monitoring duration was provided within the study.

<sup>b</sup> This study did not include outdoor area monitoring which could be applied to the bystander

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. EPA assumes exposure to asbestos resulting from brake repair/replacement work occurs for the entire three-hour period it takes the DIY consumer to conduct the work.



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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. EPA assumes the bystander remains within 3 meters of the automobile on which the work is being done for the entire three-hour period it takes for the DIY consumer to conduct the work.

EPA evaluated consumer bystander exposure for the DIY brake outdoor scenario by applying a reduction factor of 10 to the PBZ value measured outdoors for the consumer user. The reduction factor of 10 was chosen based on a comparison between the PBZ and the < 3meter from automobile values measured indoors across all activities identified in the study data utilized from Blake (a ratio of 6.5). The ratio of 6.5 was rounded up to 10, to account for an additional reduction in concentration to which a bystander may be exposed in the outdoor space based on the high air exchange rates and volume in the outdoor<sup>11</sup>.

Table 2-27 provides a summary of the data utilized for this evaluation.

**Table 2-27. Estimated Exposure Concentration for DIY Consumer User and Bystander**

Condition of Use	Estimated Consumer Exposure Concentration (f/cc)				Confidence Rating
	DIY User		Bystander		
	Central Tendency	High-end	Central Tendency	High-end	
Aftermarket Automotive Parts-Brakes (Indoor)	0.0445	0.4368	0.0130 <sup>a</sup>	0.0296 <sup>a</sup>	Medium
Aftermarket Automotive Parts-Brakes (Outdoor)	0.007	0.0376	0.0007 <sup>b</sup>	0.0038 <sup>b</sup>	Medium (DIY) Medium-Low (Bystander)

4117  
4118  
4119  
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<sup>a</sup> Based on area samples, see text.

<sup>b</sup> Reduction factor of 10 used, see text.

DIY Consumer User

4121

Indoor Scenario

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The highest concentration values reported in (Blake et al., 2003) occurred during arc grinding of the brake shoes. 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. Because such equipment is readily available to the consumer, EPA utilized the average of the two arc-grinding values from (Blake et al., 2003) as the high-end concentration for the indoor environment under this exposure scenario.

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. These values were

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<sup>11</sup> Although exposures would be very low and are not quantified here, an assumption is made in Section 4.2.3 to allow for cancer risk estimation for bystanders from outdoor brake replacement.

4132 measured during brake repair/replacement activities only (no additional work like grinding/filing) and  
4133 do include the use of compressed air. However, compressed air was only used to blow out residual dust  
4134 from brake drums after the majority of residual dust is broken out by placing the brakes on the floor with  
4135 a shock to knock off loose material. While the use of compressed air is not a recommended practice, no  
4136 reasonably available information was found that surveyed actual cleaning methods used or preferred by  
4137 DIY consumers for this scenario. EPA therefore utilized these values to evaluate consumer inhalation  
4138 exposure with the understanding that they may represent a more conservative exposure concentration  
4139 value.

#### 4140 Outdoor Scenario

4141 EPA utilized the personal breathing zone concentration from the ([Blake et al., 2003](#)) study obtained  
4142 during filing of brakes for the high-end exposure concentration for the consumer user under the outdoor  
4143 scenario. Although this value was obtained in an indoor environment it is a potential additional work  
4144 activity that could also be performed outside. Additionally, even though it is outdoors, it is expected that  
4145 filing work would entail the consumer user's personal breathing zone to be very close to the brakes  
4146 being filed and therefore high air exchange rates and outdoor volumes would not be expected to have a  
4147 considerable impact on the exposure during such work.

4148  
4149  
4150 EPA used the average monitored outdoor concentration measured in the personal breathing zone from  
4151 the ([Sheehy et al., 1989](#)) study to represent the central tendency value for the consumer user under the  
4152 outdoor scenario. The ([Sheehy et al., 1989](#)) study is the only study identified through the systematic  
4153 review process which included PBZ monitoring data for a DIY consumer user during outdoor brake  
4154 repair/replacement work. The duration of the monitoring in ([Sheehy et al., 1989](#)) was not specified for  
4155 the outdoor work, EPA assumes monitoring occurred for the entire expected duration for the DIY  
4156 consumer user to complete the work. As the study describes, the DIY consumer user utilized various  
4157 wetting techniques on the brakes to clean grease, dirt, and flush the surface of the drums. Considering  
4158 these methods were utilized, EPA assumes compressed air was not used for the outdoor scenario.

#### 4159 Bystander

##### 4160 Indoor Scenario

4161  
4162 EPA utilized the ([Blake et al., 2003](#)) area sampling data obtained within three meters from the  
4163 automobile on which the work is being performed to represent exposure concentrations for the bystander  
4164 under the indoor scenario. These values are expected to be representative of bystander exposure under  
4165 the assumptions described above in that individuals who may remain within the garage during brake  
4166 repair/replacement work would be in close quarters within a typical consumer garage for the entire  
4167 three-hour period. The high-end value utilized was the highest area concentration monitored within three  
4168 meters from the automobile. This value occurred during arc-grinding of the brake shoe. The central  
4169 tendency value utilized was the average of the two area sampling concentrations monitored within three  
4170 meters from the automobile during brake shoe removal/replacement activities.

##### 4171 Outdoor Scenario:

4172  
4173 There were no area monitoring data for the outdoor work in ([Sheehy et al., 1989](#)) which could be  
4174 representative of potential bystander exposure. As a surrogate, EPA used the analysis of reduction  
4175 factors (RFs) based on available data for the gasket ONU exposure scenario. Those data showed people  
4176 5-10 feet away from the user had measured values from 2.5 to 9-fold lower than the exposure levels  
4177 measured for the user. For that COU, EPA used the mean of 5.75 as the RF; which was in the range of  
4178 RFs from other COUs. Because there were no such measured data available to estimate an RF for  
4179 outdoor DIY brake work, EPA selected an RF of 10 that was greater than the range of RFs for other  
4180

COUs, but still allowed evaluation of potential bystander exposure in an outdoor scenario even though such exposure is expected to be low due to high air exchange rates and the volume of the outdoor space. EPA therefore applied a reduction factor of 10 to the data utilized for consumer users to represent the concentration to which the bystander is exposed under the outdoor scenario. This reduction factor was applied to both the central tendency and high-end estimates to represent potential exposure of the bystander.

### 2.3.2.1.3 Exposure Estimates for DIY Brake Repair/Replacement Scenario

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.

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

Condition of Use	Category	Exposure Concentrations		Confidence Rating
		(fibers/cc)		
		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

### 2.3.2.1.4 Data Assumptions, Uncertainties and Level of Confidence

Due to lack of reasonably available information on DIY consumer exposures, the consumer assessment relies on reasonably available occupational data obtained under certain conditions expected to be more representative of a DIY consumer user scenario (no engineering controls, no PPE, residential garage). However, the studies utilized have uncertainties associated with the location where the work was done. 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. While this presents some uncertainty, the study ([Blake et al., 2003](#)) performed smoke testing and found that air movement was limited to within eight meters of the installed and operating exhaust fan. Based on this testing, it is reasonable to assume that the existence of the exhaust fan would have limited effect on the measured concentrations within the PBZ of the DIY consumer and limited effect on the measured concentrations

4218 at the area monitors which were within three meters of the automobile being worked on because both  
4219 locations (automobile and area monitoring stations) were more than eight meters from the exhaust fan.  
4220

4221 The volume of a former automobile repair facility is considerably larger than a typical residential garage  
4222 and will have different air exchange rates. While this could raise some uncertainties related to the  
4223 applicability of the measured data to a DIY consumer user environment, the locations of the  
4224 measurements utilized for this evaluation minimize that uncertainty. The PBZ values are very near the  
4225 work area and should not be affected by the facility volume or air exchange rates. The area samples  
4226 utilized for bystander estimates were obtained within three meters from the automobile on which the  
4227 work was being done, so while affected more by volume and air exchange rates, the effects should be  
4228 limited as air movement appeared to be minimal based on the smoke testing conducted in the ([Blake et  
4229 al., 2003](#)) study.

4230  
4231 There is some uncertainty associated with the assumed length of time the brake repair/replacement work  
4232 takes. EPA assumes it takes a DIY consumer user about three hours to complete brake  
4233 repair/replacement work. This is two times as long as a professional mechanic. While it is expected to  
4234 take a DIY consumer longer, it is also expected DIY consumer users who do their own brake  
4235 repair/replacement work would, over time, develop some expertise in completing the work as they  
4236 continue to do it every three years.

4237  
4238 There is also some uncertainty associated with the assumption that a bystander would remain within  
4239 three meters from the automobile on which the brake repair/replacement work is being conducted for the  
4240 entire three-hour period EPA assumes it takes the consumer user to complete the work. However,  
4241 considering a residential garage with the door closed is relatively close quarters for car repair work, it is  
4242 likely anyone observing (or learning) the brake repair/replacement work would not be able to stay much  
4243 farther away from the car than three meters. Remaining within the garage for the entire three hours also  
4244 has some uncertainty, although it is expected anyone observing (or learning) the brake  
4245 repair/replacement work would remain for the entire duration of the work or would not be able to  
4246 observe (or learn) the task.

4247  
4248 The assumptions and uncertainties associated with a consumer's use of compressed air to clean brake  
4249 drums/pads are discussed above. While industry practices have drifted away from the use of compressed  
4250 air to clean brake drums/pads, no reasonably available information was found in the literature indicating  
4251 consumers have discontinued such work practices. To consider potential consumer exposure to asbestos  
4252 resulting from brake repair/replacement activities, EPA uses data which included use of compressed air.  
4253 However, EPA recognizes this may be a more conservative estimate because use of compressed air  
4254 typically could cause considerable dust/fibers to become airborne if it is the only method used. The  
4255 ([Blake et al., 2003](#)) study notes that compressed air was used to clean residual dust from brake drums,  
4256 but it was only used after "shocking" dust free by placing the brake drums on the ground to knock dust  
4257 free. As a result, the bulk of the dust would be on the ground and a limited portion would be removed  
4258 through the use of compressed air.

4259  
4260 EPA has an overall medium confidence rating for the *literature, studies, and data* utilized for the  
4261 Consumer DIY Brake Repair/Replacement COU. This is based on the existence of monitoring data in  
4262 both the personal breathing zone and area sampling associated directly with brake repair/replacement  
4263 activities. The studies utilized are also representative of expected consumer working conditions for a  
4264 DIY consumer. Both factors would indicate a high confidence in the studies and data used. However,  
4265 since the data utilized is based on a professional mechanic performing the brake repair/replacement  
4266 work rather than a DIY consumer, the overall confidence is medium.

4267  
 4268 EPA has an overall medium confidence rating for the *exposure results associated with the consumer*  
 4269 *user* under the Consumer DIY Brake Repair/Replacement COU for both indoor and outdoor work. This  
 4270 is based on the use of direct monitored personal breathing zone data for the individual doing the work in  
 4271 an indoor and outdoor location.

4272  
 4273 EPA has an overall medium confidence rating for the *exposure results associated with the bystander*  
 4274 *indoor location* under the Consumer DIY Brake Repair/Replacement Scenario. This is based on the  
 4275 existence of area monitoring data obtained in the immediate vicinity of the brake repair/replacement  
 4276 work in an indoor location which is representative of where a bystander may reside during brake  
 4277 repair/replacement work within a residential garage.

4278  
 4279 EPA has an overall medium-low confidence rating for *exposure results associated the bystander*  
 4280 *outdoor location* under the Consumer DIY Brake Repair/Replacement Scenario. This is based on the  
 4281 absence of area monitoring data in an outdoor work location resulting in the need to apply an adjustment  
 4282 factor to estimate bystander exposure concentrations.

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4284 **2.3.2.2 Consumer Exposures Approach and Methodology – DIY Gaskets in UTVs**

4285 This exposure assessment looks at a potential consumer exposure scenario where a DIY consumer  
 4286 removes, cleans, handles, and replaces gaskets associated with exhaust systems on UTVs which may  
 4287 contain asbestos. This scenario falls under the “other gaskets” COU in Table 1-4 of this draft risk  
 4288 evaluation. Asbestos exposure is estimated for the DIY consumer user (the individual performing the  
 4289 gasket repair work) as well as a bystander who may observe the gasket work. This scenario also assumes  
 4290 all the work is conducted indoors (within a garage) and both the consumer and bystander remain in the  
 4291 garage for the entirety of the work.

4292

4293 There was no reasonably available information found in the published literature related to DIY  
 4294 consumer exhaust system gasket repair/replacement activities on UTVs. As a result, EPA expanded the  
 4295 search to include information on occupational gasket repair/replacement for automobiles and identified  
 4296 several studies with relevant information. The gasket repair/replacement scenario relies on monitored  
 4297 values obtained in an occupational setting and considers only those environments and working  
 4298 conditions that may be representative of a DIY consumer user scenario.

4299

4300 Thirty studies relating to gasket repair/replacement were identified and reviewed as part of the  
 4301 systematic review process for exposure. These studies were compared against a series of criteria to  
 4302 evaluate how representative the studies are for DIY consumer exhaust system gasket repair/replacement  
 4303 activity. The first two criteria involved identifying whether the studies were automotive in nature and  
 4304 whether there was enough information about automotive gaskets within the study. EPA also focused on  
 4305 primary sources of data and not secondary or supplemental sources. The final criterion was to review the  
 4306 studies to ensure they were consistent with an expected DIY consumer scenario of removal, cleaning,  
 4307 and replacing gaskets. For example, studies involving machining or processing of gaskets were not  
 4308 considered as it is unlikely a DIY consumer gasket repair/replacement activity would involve machining  
 4309 and gasket processing. When compared to these criteria, three of the thirty studies were fully evaluated;  
 4310 a 2006 study by Blake ([Blake et al., 2006](#)), a 2005 study by Liukonen (([Liukonen and Weir, 2005](#)), and a  
 4311 2006 study by Paustenbach ([Paustenbach et al., 2006](#)), as shown in Table 2-29.

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

Reference	Occupational	Consumer	Data Quality Rating (Score)
-----------	--------------	----------	-----------------------------

( <a href="#">Blake et al., 2006</a> )	Yes	No	Medium (2.1)
( <a href="#">Liukonen and Weir, 2005</a> )	Yes	No	Medium (2.0)
( <a href="#">Paustenbach et al., 2006</a> )	Yes	No	Medium (1.7)

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The ([Blake et al., 2006](#)) study measured worker asbestos exposure during automotive gasket removal/replacement in vintage car engines. The ([Liukonen and Weir, 2005](#)) study measured worker asbestos exposure during automotive gasket removal/replacement on medium duty diesel engines. The ([Paustenbach et al., 2006](#)) study measured worker asbestos exposure during gasket removal/replacement on automobile exhaust systems of vintage cars (ca. 1945-1975). All three studies were conducted in the United States and used air sampling methods in compliance with NIOSH methods 7400 and 7402 for PCM and TEM, respectively. All three studies demonstrate that the highest exposure to asbestos occurs during removal of old gaskets and cleaning of the area where the gasket was removed. All three studies received a medium-quality rating through EPA’s systematic review data evaluation process.

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Relevant data from each of the three studies identified in Table 2-29 were extracted. Extracted data included vehicle or engine type, sampling duration, sample size, exposure concentrations, and units of measurement. The extracted data were transcribed into Microsoft Excel for further analysis to calculate minimum, maximum, and mean concentrations by study, activity type, and sample type. All the extracted data and calculated values are included in *Supplemental File: Consumer Exposure Calculations* ([U.S. EPA, 2019a](#)). All analysis and calculations for the three studies were performed based on the raw data rather than summary data provided by each study due to differences in the summary methodologies across the studies. 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.

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**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	
( <a href="#">Blake et al., 2006</a> )	28	14	140	0.002	0.027	0.007	Medium
Engine Disassembly	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
( <a href="#">Liukonen and Weir, 2005</a> )							

**PEER REVIEW DRAFT. DO NOT CITE OR QUOTE**

<b>Engine Disassembly</b>	<b>29</b>	<b>26</b>	<b>53</b>	<b>0.004</b>	<b>0.060</b>	<b>0.018</b>	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
<a href="#">(Paustenbach et al., 2006)</a>							
<b>Engine Disassembly</b>	<b>94</b>	<b>61</b>	<b>39</b>	<b>0.002</b>	<b>0.066</b>	<b>0.014</b>	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

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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).

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The [\(Paustenbach et al., 2006\)](#) study was conducted in two phases in Santa Rosa, CA during 2004 at an operational muffler shop that has been open since 1974 and specializes in exhaust repair work. The repair facility was about 101 feet by 48 feet with five service bay doors. The vehicles studied were located near the center of the garage. During the study, the bay doors were closed, and no heating, air condition, or ventilation systems were used.

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The [\(Paustenbach et al., 2006\)](#) study looked at 16 vehicles manufactured before 1974 with original or old exhaust systems likely to have asbestos containing gaskets at either the flanges of the muffler system or the manifold of the engine where the exhaust system connects. The study looked at four different types of muffler work: 1) removal of exhaust system up to the flange; 2) removal of exhaust system including manifold gaskets; 3) conversion from single to dual exhaust system; and 4) removal of muffler system up to the manifold with installation of an asbestos donut gasket. Two mechanics performed the exhaust repair work and neither mechanic wore respiratory protection. The mechanics removed the gaskets with either their fingers or by prying with a screwdriver, and any residual gasket material was scraped off with the screwdriver or pulled off by hand.

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All airborne samples were collected using MCE filters consistent with NIOSH method 7400. Personal breathing zone air samples were collected from the right and left lapel of the mechanic, and area air samples were collected at four locations about four feet from the vehicle. Background and ambient air samples were also collected both indoors and outdoors. A total of 134 air samples were collected, but some samples could not be analyzed due to overloaded filters. Other samples were excluded because they were taken during work on vehicles with non-asbestos gaskets. Ultimately, 82 air samples (23 personal, 38 area, and 21 background) were analyzed by PCM, and 88 air samples (25 personal, 41 area, and 22 background) were analyzed by TEM. Samples below the analytical sensitivity limit were included in the statistical analysis by substituting a value of one-half the sensitivity limit.

**2.3.2.2.1 Consumer Inhalation Exposures – DIY Gaskets in UTVs**

Consumer inhalation exposure to asbestos for the DIY exhaust system gasket removal/replacement scenario was assessed for both the DIY consumer user (individual doing the exhaust system gasket removal/replacement work) and a bystander (individual observing the exhaust system gasket removal/replacement work within the garage).

DIY Consumer User

EPA used the PBZ values from ([Paustenbach et al., 2006](#)) identified in Table 2-30 for the DIY consumer user. The maximum concentration was used as the high-end estimated concentration for the consumer user and the mean concentration was used as the central tendency concentration.

EPA used the bystander values from ([Paustenbach et al., 2006](#)) identified in Table 2-30 for the DIY consumer bystander. The bystander values from ([Paustenbach et al., 2006](#)) represent monitoring within four feet of the automobile on which the exhaust system work was being performed. The maximum concentration from Table 2-30 was utilized as the high-end estimated concentration for the consumer bystander and the mean concentration was utilized as the central tendency concentration.

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

EPA assessed exposures for the DIY UTV exhaust system gasket removal/replacement scenario based on the exposure concentrations, assumptions, and exposure conditions described above. There was no reasonably available information found within the literature providing specific information about the length of time it would take for a DIY consumer to complete an exhaust system gasket removal/replacement activity on a UTV. The studies from which data was extracted have sample periods ranging from 32 minutes to 154 minutes to complete various gasket work for a professional mechanic (assuming the sampling time within these studies was equal to the time it took to complete the gasket work). Therefore, EPA assumes, for this evaluation, the exhaust system work would take the DIY consumer three hours to complete which is approximately two times the average sample periods across the studies extracted.

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.

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.

**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 ( <a href="#">Paustenbach et al., 2006</a> )	DIY Consumer	0.024	0.066	Medium
	Bystander	0.012	0.030	Medium

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### 2.3.2.2.3 Data Assumptions, Uncertainties and Level of Confidence

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There were no reasonably available information identified through systematic review providing consumer specific monitoring for UTV exhaust system gasket repair/replacement activities. Therefore, this evaluation utilized published monitoring data obtained in an occupational setting of professional mechanics, as a surrogate for estimating consumer exposures associated with UTV gasket removal/replacement activities. There is some uncertainty associated with the use of data from an occupational setting for a consumer environment due to differences in building volumes, air exchange rates, available engineering controls, and the potential use of PPE. As part of the literature review, EPA considered these differences and utilized reasonably available information representative of the expected consumer environment. The ([Paustenbach et al., 2006](#)) study was conducted in an occupational setting, but no engineering controls were utilized. Additionally, no additional heating, ventilation, and air condition systems were utilized during the study. The monitored values used were the PBZ data which are not expected to be impacted by differences in the ventilation rates, work area volume, or air exchange rates. Similarly, the area monitoring data utilized for bystander exposure were obtained four feet from the automobile on which the work was being performed where differences in the ventilation rates, work area volume, or air exchange rates should have minimal effect on the concentrations to which the bystander is exposed.

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There is some uncertainty associated with the use of an automobile exhaust system gasket repair/replacement activity as a surrogate for UTV exhaust system gasket repair/replacement activity due to expected differences in the gasket size, shape, and location. UTV engines and exhaust systems are expected to be smaller than a full automobile engine and exhaust system, therefore the use of an automobile exhaust system gasket repair may slightly overestimate exposure to the consumer. At the same time, the smaller engine and exhaust system of a UTV could make it more difficult to access the gaskets and clean the surfaces where the gaskets adhere therefore increasing the time needed to clean and time of exposure resulting from cleaning the surfaces which could underestimate consumer exposure.

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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.

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There is some uncertainty associated with the assumption that UTV exhaust system gasket repair/replacement activities would be necessary and performed by a consumer once every three years. A general internet search (“google”) did not identify how often certain gaskets associated with the exhaust systems of UTVs would last or need to be replaced. Some information was found on ATV Maintenance including repacking the exhaust silencer of ATVs annually on machines that are frequently used or every few years on machines used seasonally. Other information found online suggested

4465 whenever you do exhaust system maintenance, you should also replace gaskets to ensure an ongoing  
 4466 effective seal for safety and efficiency.

4467  
 4468 There is some uncertainty associated with the assumption that an individual would be associated with  
 4469 using an UTV for the entire average adult lifetime of 78 years beginning at 16 years of age. It is possible  
 4470 certain individuals may be involved with UTV work prior to 16 years of age. While older individuals  
 4471 may not be associated with their personal UTV and related gasket work up to age 78, they may provide  
 4472 assistance on gasket work or perhaps change from a consumer “user” to a consumer “bystander”.  
 4473

4474 The EPA has an overall medium confidence rating for the *literature, studies, and data* utilized for the  
 4475 Consumer DIY UTV Exhaust System Gasket Repair/Replacement COU. This is based on the existence  
 4476 of monitoring data in both the personal breathing zone and area sampling associated directly with gasket  
 4477 repair/replacement activities. The studies utilized are also representative of expected consumer working  
 4478 conditions for a DIY consumer. Both factors would indicate a high confidence in the studies and data  
 4479 used. However, since the data utilized is based on a professional mechanic performing the brake  
 4480 repair/replacement work rather than a DIY consumer, the overall confidence is medium.  
 4481

4482 The EPA has an overall medium confidence rating *for the exposure results* associated with the consumer  
 4483 user and bystander under the Consumer DIY Exhaust System Gasket Repair/Replacement COU. This is  
 4484 based on the use of direct monitored personal breathing zone data for the individual doing the work and  
 4485 the existence of area monitoring data obtained in the immediate vicinity of the gasket repair/replacement  
 4486 work in an indoor location which is representative of where a bystander may reside during gasket  
 4487 repair/replacement work within a residential garage.  
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**2.3.2.3 Summary of Inhalation Data Supporting the Consumer Exposure Assessment**

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 4491 Table 2-32 contains a summary of the consumer inhalation exposure data used to calculate the risk  
 4492 estimates in Section 4.2.3.  
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**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

### 2.3.3 Potentially Exposed or Susceptible Subpopulations

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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 factors, 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.”

During problem formulation ([U.S. EPA, 2018d](#)), EPA identified potentially exposed and susceptible subpopulations for further analysis during the development and refinement of the life cycle, conceptual models, exposure scenarios, and analysis plan. In this section, EPA addresses the potentially exposed or susceptible subpopulations identified as relevant based on *greater exposure*. EPA addresses the subpopulations identified as relevant based on *greater susceptibility* in Section 3.2.5

In developing the draft risk evaluation, the EPA analyzed the reasonably available information to ascertain whether some human receptor groups may have greater exposure than the general population to the hazard posed by asbestos. Exposures of asbestos would be expected to be higher amongst groups living near facilities covered under the COUs in this draft risk evaluation, groups with asbestos-containing products in their homes, workers who use asbestos as part of their work, and groups who have higher age and route specific intake rates compared to the general population.

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:

- Workers and occupational non-users for the COUs in this draft risk evaluation (chlor-alkali, sheet gaskets, oilfield brake blocks, aftermarket automotive brakes and linings, other frictional products and other gaskets [UTVs]). EPA reviewed monitoring data found in published literature and submitted by industry including both personal exposure monitoring data (direct exposure) and area monitoring data (indirect exposures). Exposure estimates were developed for users (males and female workers of reproductive age) exposed to asbestos as well as non-users or workers exposed to asbestos indirectly by being in the same work area of the building. Also, adolescents and female workers of reproductive age (>16 to less than 50 years old) were also considered as a potentially exposed or susceptible subpopulations
- 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.
- Other groups of individuals within the general population who may experience greater exposures due to their proximity to conditions of use identified in Section 1.4.3 that result in releases to the environment and subsequent exposures (e.g., individuals who live or work near manufacturing, processing, use or disposal sites).

4542 For occupational exposures, EPA assessed exposures to workers and ONUs for the asbestos COUs.  
 4543 Table 2-33 presents the percentage of employed workers and ONUs who may be susceptible  
 4544 subpopulations within select industry sectors relevant to the asbestos COUs. The percentages were  
 4545 calculated using Current Population Survey (CPS) data for 2017. CPS is a monthly survey of households  
 4546 conducted by the Bureau of Census for the Bureau of Labor Statistics and provides a comprehensive  
 4547 body of data on the labor force characteristics. Statistics for the following subpopulations of workers and  
 4548 ONUs are provided: adolescents, adult men and women. As shown in Table 2-33, men make up the  
 4549 majority of the workforce in the asbestos COUs. In other sectors, women (including those of  
 4550 reproductive age and elderly women) make up a larger portion of wholesale and retail trade.

4551

4552

4553 **Table 2-33. Percentage of Employed Persons by Age, Sex, and Industry Sector (2017 and 2018**  
 4554 **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%

4555

4556 Manufacturing – The Manufacturing sector comprises establishments engaged in the mechanical,  
 4557 physical, or chemical transformation of materials, substances, or components into new products.  
 4558 Establishments in the sector are often described as plants, factories, or mills. For asbestos, this sector  
 4559 covers the COUs that occur in an industrial setting, including processing and using chlor-alkali  
 4560 diaphragms, gasket stamping, and gasket use in chemical plants.

4561

4562 Wholesale and retail trade – The wholesale trade sector comprises establishments engaged in  
 4563 wholesaling merchandise, generally without transformation, and rendering services incidental to the sale  
 4564 of merchandise. Wholesalers normally operate from a warehouse or office. This sector likely covers  
 4565 facilities that are engaged in the handling of imported asbestos-containing articles (i.e., aftermarket  
 4566 automotive parts, other vehicle friction products and other gaskets.

4567

4568 Adolescents, or persons between 16 and 19 years in age, are generally a small part of the total  
 4569 workforce. Table 2-34 presents further breakdown on the percentage of employed adolescents by  
 4570 industry subsectors. As shown in the table, they comprise less than 2 percent of the workforce, with the  
 4571 exception of wholesale and retail trade subsector where asbestos may be used in UTV gaskets and auto  
 4572 brakes.

**Table 2-34. Percentage of Employed Adolescents by Industry Sector (2017 and 2018 worker demographics from BLS)**

Sector	COU	Adolescents (16-19 years)
Mining, quarrying, and oil and gas extraction	Oilfield Brake Block	0.89%
Manufacturing	Chlor-Alkali; Gasket cut; Gasket use in chemical plants	1.50%
Wholesale and retail trade	Auto brake; UTV	6.13%

For consumer exposures, EPA assessed exposures to users and bystanders. EPA assumes, for this evaluation, consumer users are male or female adults (greater than 16 years of age). Bystanders could be any age group ranging from infants to adults.

### 3 HAZARDS (Effects)

#### 3.1 Environmental Hazards

##### 3.1.1 Approach and Methodology

EPA conducted comprehensive searches for data on the environmental hazards of asbestos, as described in *Strategy for Conducting Literature Searches for Asbestos: Supplemental File for the TSCA Scope Document* ([EPA-HQ-OPPT-2016-0736-0083](#)).

Only the on-topic references listed in the Ecological Hazard Literature Search Results were considered as potentially relevant data/information sources for this risk evaluation. Inclusion criteria were used to screen the results of the ECOTOX literature search (as explained in the *Strategy for Conducting Literature Searches for Asbestos: Supplemental File for the TSCA Scope Document*). Since the terrestrial pathways, including biosolids, were eliminated in the PF, EPA only reviewed the aquatic information sources following problem formulation using the data quality review evaluation metrics and the rating criteria described in the [Application of Systematic Review in TSCA Risk Evaluations](#) (U.S. EPA, 2018a). Data from the evaluated literature are summarized below and in Table 3-1, in a supplemental file (U.S. EPA, 2019d) and in Appendix E (data extraction table). Following the data quality evaluation, EPA determined that of the six on-topic aquatic toxicity studies, four of these studies were acceptable for use in risk assessment while the two on-topic aquatic plants studies were rated as unacceptable based on the evaluation strategies described in (U.S. EPA, 2018a). The studies rated as unacceptable were not used in this risk evaluation. EPA also identified the following documents sources of environmental hazard data for asbestos: 45 FR 79318, 1980; [ATSDR \(2001a\)](#); [U.S. EPA \(2014c\)](#); [U.S. EPA \(2014b\)](#); [WHO \(2014\)](#); [IARC \(2012\)](#) and Site-Wide Baseline Ecological Risk Assessment, Libby Asbestos Superfund Site, Libby Montana (U.S. EPA, 2014b).

##### 3.1.2 Hazard Identification – Toxicity to Aquatic Organisms

Reasonably available information indicated that the hazards from chronic exposure to fish and aquatic invertebrates following exposure to asbestos at concentrations ranging from  $10^4$ -  $10^8$  fibers/L (which is equivalent to 0.01 – 100 Million Fibers/Liter (MFL)) resulted in significant effects to development and

reproduction. Sublethal effects were observed following acute and chronic exposure to asbestos at concentrations lower than 0.01 MFL; for example, reduction in siphoning abilities in clams. As summarized below and in Appendix Table\_APX E-1: On-topic Aquatic Toxicity Studies Evaluated for Chrysotile Asbestos, four citations were determined to be acceptable in quality and relevance for this risk evaluation. All four citations received a rating of high quality following the data quality evaluation process.

Belanger (1986c) exposed larval coho salmon (*Oncorhynchus kisutch*) and juvenile green sunfish (*Lepomis cyanellus*) to chrysotile asbestos at concentrations that were environmentally relevant during the time of the study and reported behavioral and pathological stress caused by chrysotile asbestos. No treatment related increases in mortality were detected. Coho were exposed for 40 days at 3.0 MFL and 86 days at 1.5 MFL, while sunfish were exposed for 52 days at 3 MFL and 67 days at 1.5 MFL. According to the study, coho larvae exposed to 1.5 MFL were significantly more susceptible to an anesthetic stress test, becoming ataxic and losing equilibrium faster than control fish. Juvenile green sunfish developed behavioral stress effects in the presence of 1.5 and 3.0 MFL. Specifically, the coho and green sunfish exposed to 3.0 MFL had sublethal effects, which include the following: epidermal hypertrophy superimposed on hyperplasia, necrotic epidermis, lateral line degradation, and lesions near the branchial region. Lateral line abnormalities were associated with a loss of the ability to maintain normal orientation in the water column.

In addition, Belanger (1986b) and Belanger (1986a) investigated the effects of chrysotile asbestos exposure on larval, juvenile, and adult Asiatic clams (*Corbicula sp.*). Exposure to 0.01 MFL caused a significant reduction in release of larva by brooding adults as well as increased mortality in larvae. Reduced siphoning activity and fiber accumulation in clams were observed in the absence of food after 96-hr of exposure to 0.0001 and 0.1 MFL chrysotile asbestos, respectively (Belanger et al., 1986b). Sublethal and reproductive effects observed following 30 days of exposure to 0.0001 to < 100 MFL chrysotile include the following: 1) fiber accumulation in gill and visceral tissues, 2) decreased siphoning activity and shell growth of adult clams, 3) decreased siphoning activity, shell growth, and weight gain of juveniles, 4) reduction of larva releases, and 5) larva mortality.

Lastly, Belanger (1990) studied the effects of chrysotile asbestos at concentrations of 0, 0.0001, 0.01, 1, 100 or 10,000 MFL on all life stages of Japanese Medaka (*Oryzias latipes*), including egg development, hatchability, and survival; reduction in growth of larval to juvenile fish; reproduction performance; and larval mortality. Eggs were exposed to chrysotile until hatching for 13-21 days, larvae-juvenile fish were exposed to chrysotile for 13 weeks, and juvenile-adult fish were exposed to chrysotile for 5 months. Asbestos did not substantially impair egg development, hatchability or survival. At concentrations of 1 MFL or higher, hatching of eggs was delayed, larval Medaka experienced growth reduction, and fish developed thickened epidermal tissue. Juvenile fish exposed to 10,000 MFL suffered 98% mortality by 42 days and 100% mortality by 56 days.

For additional perspective on understanding the environmental hazard of asbestos materials, EPA considered other related documents on asbestos. For example, EPA Region 8 reviewed the same data by Belanger *et al.* discussed above for the Libby Superfund Site ecological risk assessment (U.S. EPA, 2014b) and considered the data adequate for asbestos in general, but not relevant for the Libby site specifically.

### **3.1.3 Weight of Scientific Evidence**

During the data integration stage of systematic review EPA analyzed, synthesized, and integrated the reasonably available information into Table 3.1. This involved weighing scientific evidence for quality

and relevance, using a weight of scientific evidence (WoE) approach, as defined in 40 CFR 702.33, and noted in TSCA 26(i) ([U.S. EPA, 2018a](#)).

During data evaluation, EPA reviewed on-topic environmental hazard studies for data quality and assigned studies an overall quality level of high, medium, or low based on the TSCA criteria described in the *Application of Systematic Review in TSCA Risk Evaluations* ([U.S. EPA, 2018a](#)). While integrating environmental hazard data for asbestos, EPA gave more weight to relevant information that were assigned an overall quality level of high or medium.

The ten on-topic ecotoxicity studies for asbestos included data from aquatic organisms (i.e., vertebrates, invertebrates, and plants) and terrestrial species (i.e., fungi and plants). Following the data quality evaluation, EPA determined that four on-topic aquatic vertebrate and invertebrate studies were acceptable while the two on-topic aquatic plants studies were unacceptable based on the evaluation strategies described in the *Application of Systematic Review in TSCA Risk Evaluations* ([U.S. EPA, 2018a](#)). Since the terrestrial pathways were eliminated in the PF, EPA excluded three studies on terrestrial species as terrestrial exposures were not expected under the COUs for asbestos. One amphibian study was excluded from further review and considered out of scope because it was not conducted on chrysotile asbestos. Ultimately the four aquatic toxicity studies were rated high in quality and used to characterize the adverse effects of chrysotile asbestos to aquatic vertebrate and invertebrate organisms from chronic exposure, as summarized in Table 3-1. Any information that EPA assigned an overall quality of unacceptable was not used. The gray literature EPA identified for asbestos had minimal or no information about environmental hazards and were consequently not used. EPA determined that data and information were relevant based on whether they had biological, physical/chemical, and environmental relevance ([U.S. EPA, 1998](#)):

- Biological relevance: correspondence among the taxa, life stages, and processes measured or observed and the assessment endpoint.
- Physical/chemical relevance: correspondence between the chemical or physical agent tested and the chemical or physical agent constituting the stressor of concern.
- Environmental relevance: correspondence between test conditions and conditions in the environment.

**Table 3-1. Environmental Hazard Characterization of Asbestos**

Duration	Test Organism	Endpoint	Hazard Value <sup>c</sup>	Unit	Effect Endpoint(s)	References <sup>c</sup>
<b>Aquatic Organisms</b>						
Acute	Aquatic invertebrates	96-hr LOEC	0.0001-100	MFL <sup>d</sup>	Reduction in siphoning activity; Fiber accumulation	<a href="#">Belanger et al. (1986b)</a> (High)
Chronic	Fish	13-86 day NOEC <sup>a</sup>	0.01-1.5	MFL	Behavioral stress (e.g., aberrant swimming and loss of equilibrium); Egg development, hatchability, and survival; Growth; Mortality	<a href="#">Belanger et al. (1990)</a> (High); <a href="#">Belanger et al. (1986c)</a> (High);
		13-86 day LOEC <sup>b</sup>	1-3			
	Aquatic invertebrates	30-day LOEC	0.0001-100	MFL	Reduction in siphoning activity; Number of larvae released; Alterations of gill tissues; Fiber accumulation in tissues; Growth; Mortality	<a href="#">Belanger et al. (1986b)</a> (High); <a href="#">Belanger et al. (1986a)</a> (High)

<sup>a</sup>NOEC, No Observable Effect Concentration.

<sup>b</sup>LOEC, Lowest Observable Effect Concentration.

<sup>c</sup>Values in the tables were reported by the study authors and combined in ranges (min to max) from different effect endpoints. The values of the NOEC and LOEC can overlap because they may be based on different effect endpoints. For example, fish NOEC = 1.5 MFL was based on behavioral stress (e.g., aberrant swimming and loss of equilibrium) and fish LOEC = 1 MFL was based on significant reduction in growth of larval individuals. See Table\_APX E-1 for more details.

<sup>d</sup>MFL, Million Fibers/Liter.

<sup>e</sup>Data quality evaluation scores for each citation are in the parenthesis.

#### 3.1.4 Summary of Environmental Hazard

A review of the high-quality aquatic vertebrate and invertebrate studies indicated that chronic exposure to waterborne chrysotile asbestos at a concentration range of  $10^4$ - $10^8$  fibers/L, which is equivalent to 0.01 to 100 MFL, may result in reproductive, growth and/or sublethal effects to fish and clams. In addition, acute exposure of waterborne chrysotile asbestos at a concentration range of  $10^2$ - $10^8$  fibers/L to clams demonstrated reduced siphoning activity.

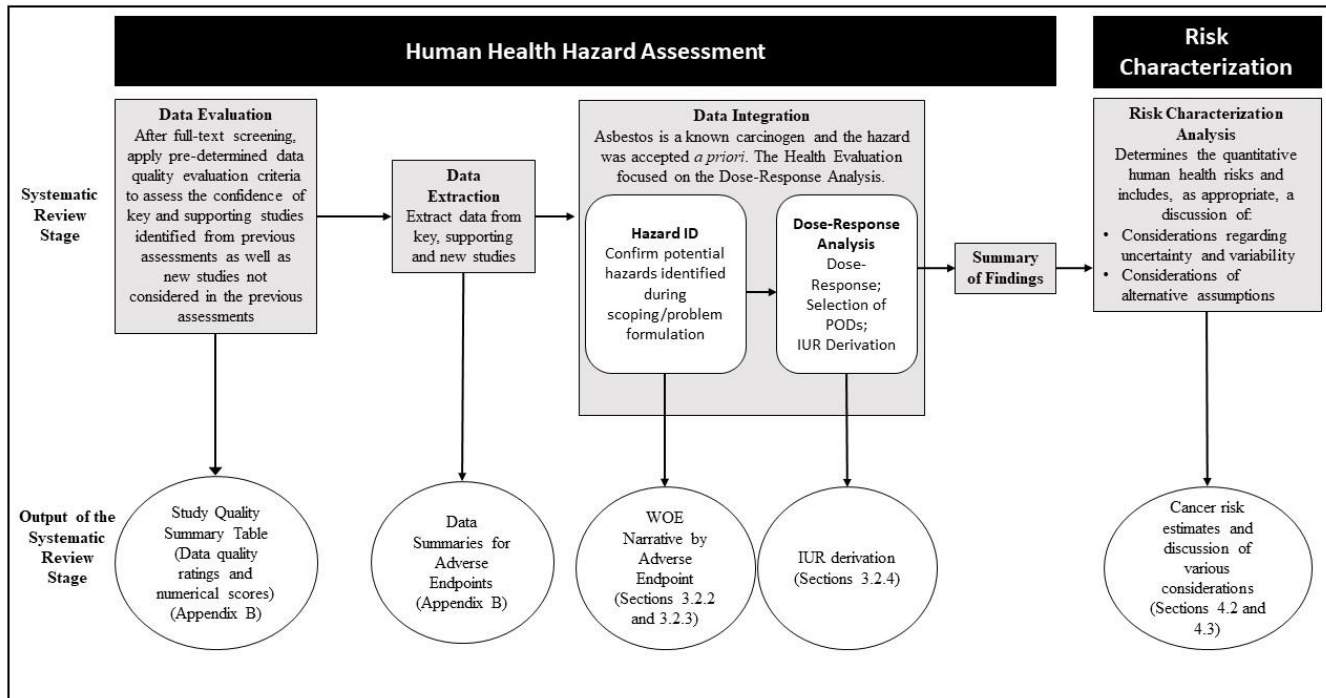
### 3.2 Human Health Hazards

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. Given the well-established carcinogenicity of asbestos for lung cancer and mesothelioma, EPA, in its PF document, decided to limit the scope of its systematic review to these two specific cancers and to inhalation exposures with the goal of updating, or reaffirming, the existing EPA IUR for general asbestos (U.S. EPA, 1988b). As explained in Section 1.4.1, EPA has determined that the asbestos fiber associated with the COUs in this draft risk evaluation is chrysotile. Thus, this draft risk evaluation uses the EPA-derived chrysotile IUR described in Section 3.2.4 to calculate risk estimates.

#### 3.2.1 Approach and Methodology

EPA used the approach described in Figure 3-1 to evaluate, extract and integrate asbestos human health hazard and dose-response information. This approach is based on the *Application of Systematic Review in TSCA Risk Evaluations* (U.S. EPA, 2018a) and the *Framework for Human Health Risk Assessment to Inform Decision Making* (U.S. EPA, 2014a).





4714  
4715 **Figure 3-1. EPA Approach to Hazard Identification, Data Integration, and Dose-Response**  
4716 **Analysis for Asbestos**  
4717

4718 In the PF document, it was stated that the asbestos RE would focus on epidemiological inhalation data  
4719 on lung cancer and mesothelioma for all TSCA Title II fiber types, just as stated in the 1988 EPA IRIS  
4720 Assessment on Asbestos ([U.S. EPA, 1988b](#)). This was based on the large database on the health effects  
4721 associated with asbestos exposure which has been cited in numerous U.S. and international data sources.  
4722 These data sources included, but were not limited to, EPA IRIS Assessment [IRIS Assessment on](#)  
4723 [Asbestos \(1988b\)](#), [IRIS Assessment on Libby Amphibole Asbestos \(2014c\)](#), National Toxicology  
4724 Program (NTP) [Report on Carcinogens, Fourteenth Edition \(2016\)](#), NIOSH [Asbestos Fibers and Other](#)  
4725 [Elongate Mineral Particles: State of the Science and Roadmap for Research \(2011\)](#), ATSDR  
4726 [Toxicological Profile for Asbestos \(2001a\)](#), IARC [Monographs on the Evaluation of Carcinogenic Risks](#)  
4727 [to Humans. Arsenic, Metals, Fibres, and Dusts. Asbestos \(Chrysotile, Amosite, Crocidolite, Tremolite,](#)  
4728 [Actinolite, and Anthophyllite\) \(2012\)](#), and [World Health Organization \(WHO\) Chrysotile Asbestos](#)  
4729 [\(2014\)](#).

4730  
4731 EPA conducted comprehensive searches for reasonably available information on health hazards of  
4732 asbestos, as described in *Strategy for Conducting Literature Searches for Asbestos: Supplemental File*  
4733 *for the TSCA Scope Document* (EPA-HQ-OPPT-2016-0736). The relevant studies were evaluated using  
4734 the data quality criteria in the *Application of Systemic Review in TSCA Risk Evaluations* document ([U.S.](#)  
4735 [EPA, 2018a](#)). The process and results of this systematic review are available in a supplemental  
4736 document (see *Systematic Review Supplemental File: Data Quality Evaluation and Data Extraction of*  
4737 *Human Health Hazard Studies*).  
4738

4739 This EPA human health hazard assessment consists of hazard identification and dose-response  
4740 assessment as described in EPA's *Framework for Human Health Risk Assessment to Inform Decision*  
4741 *Making* ([U.S. EPA, 2014a](#)). Hazards were identified from consensus documents. EPA integrated  
4742 epidemiological studies of asbestos with other readily available information to select the data to use for

dose-response assessment. Dose-response modeling was performed for the hazard endpoints with adequate study quality and acceptable data sets.

After publication of the PF document, EPA determined that only chrysotile asbestos is still imported into the U.S. either in raw form or in products; the other five forms of asbestos have neither known, intended, nor reasonably foreseen manufacture, import, processing, or distribution. EPA will consider legacy uses and associated disposal in subsequent supplemental documents. Therefore, for this document, in order to inform the estimation of an exposure-response function allowing for the derivation of a chrysotile asbestos IUR, EPA identified epidemiological studies on mesothelioma and lung cancer in cohorts of workers using chrysotile in commerce. To identify studies with the potential to be used to derive an inhalation unit risk (IUR), EPA also screened and evaluated new studies that were published since the EPA IRIS assessment conducted in 1988.

The new literature was screened against inclusion criteria in the PECO statement, and the literature was further screened to identify only hazard studies with inhalation exposure to chrysotile asbestos. Cohort data deemed as “key” was entered directly into the data evaluation step based on its relevance to the risk evaluation. The relevant (e.g., useful for dose-response for the derivation of the IUR) study cohorts were further evaluated using the data quality criteria for human studies. Only epidemiological hazard studies by inhalation and only chrysotile asbestos exposures were included.

EPA developed unique data quality criteria for epidemiological studies on asbestos exposure and mesothelioma and lung cancer (see Systematic Review Supplemental File: Data Quality Evaluation and Data Extraction of Human Health Hazard Studies). EPA considered studies of low, medium, or high confidence for dose-response analysis for the derivation of the IUR. Information that was rated unacceptable was not included in the risk evaluation ([U.S. EPA, 2018a](#)). The Systematic Review Supplemental File: Data Quality Evaluation and Data Extraction of Human Health Hazard Studies presents the data quality information on human health hazard endpoints (cancer) for all acceptable studies (with low, medium, or high scores). See section 3.2.4.

Following the data quality evaluation, EPA extracted a summary of data from each relevant cohort. In the last step, the strengths and limitations of the data among the cohorts of acceptable quality were evaluated for each cancer endpoint and a weight-of-the-scientific evidence narrative was developed. Data for either mesothelioma or lung cancer was modeled to determine the dose-response relationship. Finally, the results were summarized, and the uncertainties were presented. The process is described in Section 3.2.4.

Section 3.2.4.3 describes the epidemiological studies chosen for the derivation of the IUR for chrysotile asbestos.

### **3.2.2 Hazard Identification**

Asbestos has an existing EPA IRIS Assessment, an ATSDR Toxicological Profile, and many other U.S. and international assessments (see Section 1.3); hence, many of the hazards of asbestos have been previously compiled and reviewed. Most of the information in these assessments is based on inhalation exposures to human populations. Only inhalation exposures in humans are evaluated in the risk evaluation of asbestos. 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.

4791

4792 During scoping and PF, EPA reviewed the existing EPA IRIS health assessments to ascertain the  
4793 established health hazards and any known toxicity values. EPA had previously, in the IRIS assessment  
4794 on asbestos ([U.S. EPA, 1988b](#)), identified asbestos as a carcinogen causing both lung cancer and  
4795 mesothelioma from inhalation exposures and derived an IUR to address both cancers. No toxicity values  
4796 or IURs have yet been estimated for other cancers that have been identified by the International Agency  
4797 for Research on Cancer (IARC) and other government agencies. Given the well-established  
4798 carcinogenicity of asbestos for lung cancer and mesothelioma, EPA, in its PF document, had decided to  
4799 limit the scope of its systematic review to these two specific cancers and to inhalation exposures with  
4800 the goal of updating, or reaffirming, the existing unit risk. As explained in Section 1.4, the only COUs of  
4801 asbestos or asbestos containing products assessed in this risk evaluation are for chrysotile asbestos.  
4802 Thus, an IUR value for chrysotile asbestos only was developed. EPA will consider legacy uses and  
4803 associated disposal in subsequent supplemental documents.

4804

### **3.2.3 Cancer Hazards**

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4805 Many authorities have established that there are causal associations between asbestos exposures and  
4806 lung cancer and mesotheliomas in humans based on epidemiologic studies ([NTP, 2016](#); [IARC, 2012](#);  
4807 [ATSDR, 2001a](#); [U.S. EPA, 1988b](#); [IARC, 1987](#); [U.S. EPA, 1986](#); [IARC, 1977](#)). EPA also noted in the  
4808 scope that there is a causal association between exposure to asbestos and cancer of the larynx and cancer  
4809 of the ovary ([IARC, 2012](#)), and that there is also suggestive evidence of a positive association between  
4810 asbestos and cancer of the pharynx ([IARC, 2012](#); [NRC, 2006](#)), stomach ([IARC, 2012](#); [ATSDR, 2001a](#))  
4811 and colorectum ([NTP, 2016](#); [IARC, 2012](#); [NRC, 2006](#); [ATSDR, 2001a](#); [NRC, 1983](#); [U.S. EPA, 1980](#)).  
4812 In addition, the scope document reported increases in lung cancer mortality in both workers and  
4813 residents exposed to various asbestos fiber types, including chrysotile, as well as fiber mixtures ([IARC,](#)  
4814 [2012](#)). Mesotheliomas, tumors arising from the thin membranes that line the chest (thoracic) and  
4815 abdominal cavities and surround internal organs, are relatively rare in the general population, but are  
4816 often observed in populations of asbestos workers. All types of asbestos fibers have been reported to  
4817 cause mesothelioma – including chrysotile asbestos ([IARC, 2012](#); [U.S. EPA, 1988b, 1986](#)).

4818 During PF, EPA reviewed the existing EPA IRIS health assessments ([U.S. EPA, 2014c, 1988b](#)) to  
4819 ascertain the established health hazards and any known toxicity values. EPA had previously ([U.S. EPA,](#)  
4820 [1988b, 1986](#)) identified asbestos as a carcinogen causing both lung cancer and mesothelioma and  
4821 derived a unit risk based on epidemiologic studies to address both cancers. The U.S. Institute of  
4822 Medicine ([IOM, 2006](#)) and the International Agency for Research on Cancer ([IARC, 2012](#)) have  
4823 evaluated the evidence for causation of cancers of the pharynx, larynx, esophagus, stomach, colon, and  
4824 rectum, and IARC has evaluated the evidence for cancer of the ovary. Both the U.S. Institute of  
4825 Medicine and IARC concluded that asbestos causes laryngeal cancer and IARC concluded that asbestos  
4826 causes ovarian cancer. No toxicity values or IURs have yet been estimated for either laryngeal or  
4827 ovarian cancers.

4828

#### **3.2.3.1 Mode of Action (MOA) considerations for asbestos**

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4829

4830 As stated in [IRIS Assessment on Libby Amphibole Asbestos \(2014c\)](#) for asbestos in general,  
4831 International Agency for Research on Cancer (IARC) has proposed a mechanism for the carcinogenicity  
4832 of asbestos fibers [see Figure 4-2 in ([IARC, 2012](#))]. Asbestos fibers lead to oxidant production through  
4833 interactions with macrophages and through hydroxyl radical generation from surface iron. Inhaled fibers  
4834 that are phagocytosed by macrophages may be cleared or lead to frustrated phagocytosis, which results  
4835 in macrophage activation, release of oxidants, and increased inflammatory response, in part due to  
4836 inflammasome activation. Free radicals may also be released by interaction with the iron on the surface  
4837 of fibers. Increased oxidant production may result in epithelial cell injury, including DNA damage.

4838 Frustrated phagocytosis may also lead to impaired clearance of fibers, with fibers being available for  
4839 translocation to other sites (e.g., pleura). Mineral fibers may also lead to direct genotoxicity by  
4840 interfering with the mitotic spindle and leading to chromosomal aberrations. Asbestos exposure also  
4841 leads to the activation of intracellular signaling pathways, which in turn may result in increased cellular  
4842 proliferation, decreased DNA damage repair, and activation of oncogenes. Research on various types of  
4843 mineral fibers supports a complex mechanism involving multiple biologic responses following exposure  
4844 to asbestos (i.e., genotoxicity, chronic inflammation/cytotoxicity leading to oxidant release, and cellular  
4845 proliferation) in the carcinogenic response to mineral fibers [see Figure 4-2, (IARC, 2012)].  
4846

### 4847 **3.2.4 Derivation of a Chrysotile Asbestos Inhalation Unit Risk**

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#### 4848 **3.2.4.1 Derivation of a Chrysotile Asbestos Inhalation Unit Risk**

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4851 As stated in Section 3.2.3, epidemiological studies on mesothelioma and lung cancer in cohorts of  
4852 workers using chrysotile in commerce were identified that could inform the estimation of an exposure-  
4853 response function allowing for the derivation of a chrysotile asbestos IUR. In addition, EPA could not  
4854 find any recent risk numbers in the literature for the types of asbestos regulated under TSCA since the  
4855 IRIS IUR<sup>12</sup> value, which had been developed in the 1980s. Thus, rather than update or reaffirm the  
4856 existing IUR for general asbestos, EPA developed a chrysotile-specific IUR in this risk evaluation.  
4857

4858 EPA did not have a previous, recent risk assessment of asbestos on which to build; therefore, the  
4859 literature was reviewed to determine whether a new IUR needed to be developed. As the RE process  
4860 progressed, several decisions were made that refined and narrowed the scope of the RE. It was  
4861 determined during PF that the RE would focus on epidemiologic data on mesothelioma and lung cancer  
4862 by the inhalation route. The existing EPA IUR for asbestos was developed in 1988 was based on 14  
4863 epidemiologic studies that included occupational exposure to chrysotile, amosite, or mixed-mineral  
4864 exposures (chrysotile, amosite, crocidolite). However, EPA's research to identify COUs indicated that  
4865 only chrysotile asbestos is currently being imported in the raw form or imported in products. The other  
4866 five forms of asbestos identified for this risk evaluation are no longer manufactured, imported,  
4867 processed, or distributed in the United States. This commercial chrysotile is therefore the substance of  
4868 concern for this quantitative assessment and thus EPA sought to derive an IUR specific to chrysotile  
4869 asbestos. The epidemiologic studies available for risk assessment all include populations exposed to  
4870 commercial chrysotile asbestos, which may contain small, but variable amounts of amphibole asbestos.  
4871 Because chrysotile is the only form of asbestos in the United States with COUs in this document, studies  
4872 of populations exposed only to chrysotile provide the most informative data for the purpose of  
4873 developing the TSCA risk estimates for the COUs for chrysotile asbestos. EPA will consider legacy uses  
4874 and associated disposal in subsequent supplemental documents.

#### 4875 **3.2.4.2 Rationale for Asbestos-Specific Data Evaluation Criteria**

---

4876 For the first 10 TSCA REs, a general set of study evaluation criteria was developed. These data  
4877 evaluation criteria were not tailored to any specific exposure or outcome. In the PF step of the asbestos  
4878 assessment, it was accepted that exposure to asbestos was a known cause of lung cancer and  
4879 mesothelioma, and that the purpose of the systematic review would be the identification of studies which

---

<sup>12</sup> Inhalation Unit risk (IUR) is typically defined as a plausible upper bound on the estimate of cancer risk per  $\mu\text{g}/\text{m}^3$  air breathed for 70 years. For asbestos, IUR is expressed as cancer risk per fibers/cc (in units of the fibers as measured by PCM).

4880 could inform the estimation of an exposure-response function allowing for the derivation of an asbestos  
4881 inhalation unit risk for lung cancer and mesothelioma combined. The study domains of *exposure*,  
4882 *outcome*, *study participation*, *potential confounding*, and *analysis* were further tailored to the specific  
4883 needs of evaluating asbestos studies for their potential to provide information on the exposure-response  
4884 relationship between asbestos exposure and mortality from lung cancer and from mesothelioma ([U.S.  
4885 EPA, 2019h](#)).

4886  
4887 In terms of evaluating *exposure* information, asbestos is unique among these first 10 TSCA chemicals as  
4888 it is a fiber and has a long history of different exposure assessment methodologies. For mesothelioma,  
4889 this assessment is also unique with respect to the impact of the timing of exposure relative to the cancer  
4890 outcome as the time since first exposure plays a dominant role in modeling risk. The most relevant  
4891 exposures for understanding mesothelioma risk were those that occurred decades prior to the onset of  
4892 cancer and subsequent cancer mortality. Asbestos measurement methodologies have changed over those  
4893 decades, from early measurement of total dust particles measured in units of million particles per cubic  
4894 foot of air (mppcf) by samplers called midjet impingers to fibers per milliliter (f/ml), or the equivalent  
4895 fibers per cubic centimeter (f/cc), where fiber samples were collected on membrane filters and the fiber  
4896 count per volume of air was measured by analyzing the filters using phase contrast microscopy (PCM).  
4897 In several studies encompassing several decades of asbestos exposures, matched samples from midjet  
4898 impingers and membrane filters were compared to derive job- (or location-) specific factors allowing for  
4899 the conversion of earlier midjet impinger measurements to estimate PCM measurement of asbestos air  
4900 concentrations. While some studies were able to provide these factors for specific locations and jobs,  
4901 other studies were only able to derive one factor for all jobs and locations. The use of such data has  
4902 allowed asbestos researchers to investigate the risk of asbestos and successfully model lung cancer and  
4903 mesothelioma mortality over several decades of evaluation ([U.S. EPA, 2014c](#), [1988b](#), [1986](#)). Thus, the  
4904 general exposure evaluation criteria were adjusted to be specific to exposure assessment methodologies  
4905 such as midjet impingers and PCM with attention to the use of job-exposure-matrices (JEMs) to  
4906 reconstruct workers' exposure histories and the reporting of key metrics needed to derive exposure-  
4907 response functions for lung cancer and mesothelioma.

4908  
4909 In terms of evaluating the quality of *outcome* information, lung cancer is relatively straightforward to  
4910 evaluate as an outcome. Specific International Classification of Disease (ICD) codes for lung cancer  
4911 have existed for the entire time period of the studies evaluated here making it possible to identify cases  
4912 from mortality databases. On the other hand, there was no diagnostic code for mesothelioma in the  
4913 International Classification of Diseases prior to the introduction of the 10<sup>th</sup> revision (ICD-10) which was  
4914 not implemented in United States until 1999. Before ICD-10, individual researchers employed different  
4915 strategies (e.g., had to go beyond ICD codes and generally searched original death certificates for  
4916 mention of mesothelioma, considered certain ICD rubrics). Thus, the general outcome evaluation criteria  
4917 were adjusted to be specific to mesothelioma and outcome ascertainment strategies.

4918  
4919 Mesothelioma is a very rare cancer. As noted by U.S. EPA ([2014c](#)), the “Centers for Disease Control  
4920 and Prevention estimated the death rate from mesothelioma, using 1999 to 2005 data, as approximately  
4921 23.2 per million per year in males and 5.1 per million per year in females ([CDC, 2009](#)).” While  
4922 extremely rare, the overwhelmingly dominant cause of mesothelioma is asbestos exposure ([Tossavainen,  
4923 1997](#)) making the observance of mesothelioma in a population a very specific indicator for asbestos  
4924 exposure. It is critical to understand that the prevailing risk model for mesothelioma models is an  
4925 absolute risk model of mesothelioma mortality which assumes there is no risk at zero exposure ([U.S.  
4926 EPA, 1986](#); [Peto et al., 1982](#); [Peto, 1978](#)). This use of an absolute risk model differs from is in stark  
4927 contrast to the standard use of a relative risk model for lung and other cancers. For the relative risk  
4928 model, the risk of lung cancer in an asbestos exposed population multiplies a background risk in an

4929 unexposed population. Thus, an important consideration of study quality is the evaluation of that  
4930 comparison population. However, for mesothelioma, no comparison population is needed to estimate the  
4931 absolute risk among people exposed to asbestos, and therefore the criteria in the *study participation*  
4932 domain (that include comparison population) were adjusted for mesothelioma.

4933  
4934 In terms of evaluating *potential confounding*, the generic potential confounding section was adapted to  
4935 recognize that there are both direct and indirect methods for controlling for some confounders. –  
4936 specifically, that methodologies that involve internal comparisons within a working population may  
4937 indirectly control for smoking and other factors assuming when these factors do not vary with asbestos  
4938 exposure concentrations in the workplace. In contrast, mesothelioma is much simpler to evaluate for  
4939 potential confounding as diagnostic X-ray contrast medium “Thorotrast” and external beam  
4940 radiotherapy are the only other known risk factors for mesothelioma, and this rare exposure these are  
4941 unlikely to be a confounder. because these are rare procedures are not routinely done on healthy  
4942 workers. screening programs typically x-ray all workers – regardless of their cumulative asbestos  
4943 exposure.

4944  
4945 In terms of *analysis*, the evaluation criteria were needed to be adapted for both mesothelioma and lung  
4946 cancer. For mesothelioma, the Peto model (Peto et al., 1982; Peto, 1978) was traditionally used for  
4947 summary data published in the literature (U.S. EPA, 1986) rather than raw individual-level data, so  
4948 studies were considered acceptable that only reported sufficient information to fit modeling using the  
4949 Peto model by the authors or the presentation of sufficient information to fit the Peto model *post hoc*  
4950 was considered acceptable. For lung cancer, a wider selection of statistical models was acceptable, with  
4951 the preference generally given to modeling that used individual data in the analysis. Grouped data  
4952 modeling will also be reported but would be carried forward to the summary only if no individual data  
4953 modeling were available.

### 4955 **3.2.4.3 Additional considerations for final selection of studies for exposure-response**

4956 As shown in Figure 1-8, EPA’s literature search identified more than 24,000 studies, but for the final  
4957 data evaluation 26 papers covering seven cohorts were identified, and these cohorts are listed in Table  
4958 3-2.

4959  
4960 In reviewing these available studies, EPA distinguished between studies of exposure settings where only  
4961 commercial chrysotile was used or where workers exposed only to commercial chrysotile could be  
4962 identified, and situations where chrysotile was used in combinations with amphibole asbestos forms and  
4963 the available information does not allow exposures to chrysotile and amphibole forms to be separated.  
4964 Studies in the latter group were judged to be uninformative with respect to the cancer risks from  
4965 exposure to commercial chrysotile and were excluded from further consideration (e.g., Slovenia cohort:  
4966 Dodic et al., (2007; 2003).

4967  
4968 All the studies determined to be informative for lung cancer and mesothelioma analysis were based on  
4969 observation of historical occupational cohorts. Some cohorts have been the subject of multiple  
4970 publications; in these cases, only data from the publication with the longest follow-up for each cohort or  
4971 the most relevant exposure-response data were used unless otherwise specified.

4972  
4973 Studies were deemed informative for lung cancer risk assessment if either the relative risk of lung cancer  
4974 per unit of cumulative chrysotile exposure in fibers per cc-year (f/cc-yrs) from fitting log-linear or  
4975 additive relative risk models or the data needed to fit such models as described below were available.  
4976 The group of Balangero, Italy cohort studies including Pira et al.,(2009) was excluded for lack of results

4977 from models using a continuous measure of exposure. Studies that presented lung cancer risks only in  
 4978 relation to impinger total dust exposure were excluded from consideration unless they provided at least a  
 4979 data-based, study-specific factor for converting concentrations from mppcf to f/cc.  
 4980

4981 EPA identified studies of five independent occupational cohorts exposed only to commercial chrysotile  
 4982 that provided adequate data for assessment of lung cancer risks: asbestos textile manufacturing workers  
 4983 in North Carolina and South Carolina, USA ([Loomis et al., 2009](#); [Hein et al., 2007](#)) and Chongqing,  
 4984 China ([Deng et al., 2012](#)) and chrysotile miners in Québec, Canada ([Liddell et al., 1997](#)), and Qinghai,  
 4985 China ([2014](#); [Wang et al., 2013b](#)). A pooled analysis of the two U.S. studies (NC and SC) asbestos  
 4986 textile cohorts ([Elliott et al., 2012](#)) also provides informative data. In addition, Berman and Crump  
 4987 ([2008](#)) provide informative risk estimates for the Québec miner cohort based on modeling dose-response  
 4988 data that were not available in the original study.  
 4989

4990 Studies were considered informative for mesothelioma risk assessment if risk estimates from fitting the  
 4991 EPA mesothelioma model to individual-level data or data needed to fit the model as described below  
 4992 were available. None of the original publications reported risk estimates from fitting the Peto model.  
 4993 However, Berman & Crump ([2008](#)) provide risk estimates for the Québec miners from analyses of  
 4994 original, individual-level data ([Liddell et al., 1997](#)) and for South Carolina from analysis of grouped data  
 4995 ([Hein et al., 2007](#)). Comparable risk estimates were generated for North Carolina textile workers  
 4996 ([Loomis et al., 2009](#)) using tabulated mesothelioma data ([Loomis et al., 2019](#)). Data needed to fit Peto  
 4997 mesothelioma model have not been published for any other cohort exposed to chrysotile only.  
 4998

4999 **Table 3-2. Study Cohort, Individual studies and Study Quality of Commercial Chrysotile Asbestos**  
 5000 **Reviewed for Assessment of Lung Cancer and Mesothelioma Risks**

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

Study Cohort	Author, Year	HERO ID	Study Quality**
North Carolina, US	( <a href="#">Berman and Crump, 2008</a> )	626405	Lung Cancer 1.7 Medium  Mesothelioma 1.5 High
	( <a href="#">Dement et al., 2008</a> )	626406	
	( <a href="#">Elliott et al., 2012</a> )	1247861	
	( <a href="#">Loomis et al., 2009</a> )	3079232	
	( <a href="#">Loomis et al., 2010</a> )	2225695	
	( <a href="#">Loomis et al., 2012</a> )	1257856	
	( <a href="#">Loomis et al., 2019</a> )	5160027	
	( <a href="#">SRC, 2019a</a> )	5080241	
Salonit Anhovo, Slovenia*	( <a href="#">Dodic Fikfak, 2003</a> )	3080279	
	( <a href="#">Dodic Fikfak et al., 2007</a> )	3079664	
Quebec, Canada	( <a href="#">Berman and Crump, 2008</a> )	626405	Lung Cancer Low (professional judgement)  Mesothelioma Medium (professional judgement)
	( <a href="#">Gibbs and Lachance, 1972</a> )	3580825	
	( <a href="#">Liddell et al., 1997</a> )	3081408	
	( <a href="#">Liddell et al., 1998</a> )	3081200	
	( <a href="#">Liddell and Armstrong, 2002</a> )	3080504	
	( <a href="#">Mcdonald et al., 1993a</a> )	3081910	
	( <a href="#">Mcdonald et al., 1993b</a> )	3081911	
	( <a href="#">SRC, 2019b</a> )	5080232	
	( <a href="#">Vacek, 1998</a> )	3081118	
Chongqing, China – asbestos products factory including textiles	( <a href="#">Courtice et al., 2016</a> )	3520560	Lung Cancer 1.4 High
	( <a href="#">Deng et al., 2012</a> )	2573093	
	( <a href="#">Wang et al., 2014</a> )	2538846	
	( <a href="#">Yano et al., 2001</a> )	3080569	

5001 \* Cohorts from Italy and Slovenia are not considered further (see text above the table)

5002 \*\* Detailed information on Study quality is in Systematic Review Supplemental File: Data Quality  
5003 Evaluation and Data Extraction of Human Health Hazard Studies  
5004

5005 **3.2.4.4 Statistical Methodology**

5006 The first step towards deriving a cancer unit risk for risk estimation is to identify potency factors for  
5007 lung cancer and mesothelioma. Cancer potency values are either extracted from published epidemiology  
5008 studies or derived from the data within those studies. Once the cancer potency values have been  
5009 obtained, they are adjusted for differences in air volumes between workers and other populations. Those  
5010 adjusted values can be applied to the U.S. population as a whole in the standard EPA life-table analyses.  
5011 These life-table analyses allow for the estimation of an exposure concentration associated with a specific  
5012 extra risk of cancer mortality caused by asbestos. The unit risks for lung cancer and mesothelioma are  
5013 estimated separately and then combined to yield the cancer inhalation unit risk.

5014 **3.2.4.4.1 Cancer Risk Models**

5015 A cancer risk model predicts the probability of cancer in an individual with a specified history of  
5016 exposure to a cancer-causing agent. In the case of inhalation exposure to asbestos, the cancer effects of



chief concern are lung cancer and mesothelioma, and exposure history is the product of the level and timing of the asbestos exposure. The most common model forms are described below.

Lung Cancer

For lung cancer, the risk for grouped data from epidemiologic studies from exposure to asbestos is usually quantified using a linear relative risk model of the following form (Berman and Crump, 2008; U.S. EPA, 1988b, 1986):

$$RR = \alpha (1 + CE \cdot K_L)$$

where:

- RR = Relative risk of lung cancer
- CE = Cumulative exposure to asbestos (f/cc-yrs), equals the product of exposure concentration (f/cc) and the duration of exposure (years). In many publications, exposure estimates are “lagged” to exclude recent exposures, since lung cancer effects usually take at least 10 years to become apparent. In this case, cumulative exposure is indicated as CE<sub>10</sub> to represent the 10-year lag period.
- K<sub>L</sub> = Lung cancer potency factor (f/cc-yrs)<sup>-1</sup>.
- α = The ratio of baseline (unexposed) risk in the study population compared to the reference population. If the reference population is well-matched to the study population, α is usually assumed to be constant=1 and is not treated as a fitting parameter. If the general population is used as the reference population, then α may be different from 1 and is treated as a fitting parameter.

A re-parametrization with α = exp (β<sub>0</sub>) 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 (K<sub>L</sub>) and were carried forward for further consideration.

Mesothelioma

For mesothelioma, the risk model is usually an absolute risk model that gives the risk of death from mesothelioma in an individual following exposure to asbestos that is a function of the concentration and length of time since first exposure. The model form (originally proposed by (Peto et al., 1982; Peto, 1978) and subsequently used by others, including U.S. EPA (1986) and Berman and Crump (2008)) is:

$$I_m = C \cdot K_M \cdot Q$$

where:

- I<sub>m</sub> = Rate of mesothelioma (cases per person year)
- C = Concentration of asbestos (f/cc)
- K<sub>M</sub> = Mesothelioma potency factor (f/cc-yrs<sup>3</sup>)<sup>-1</sup>
- Q = A cubic function of the time since first exposure (TSFE) and the duration (d) of exposure, as follows:

- for TSFE < 10                                    Q = 0
- for 10 ≤ TSFE < d + 10                    Q = (TSFE – 10)<sup>3</sup>
- for TSFE ≥ d + 10                          Q = (TSFE – 10)<sup>3</sup> – (TSFE – 10 – d)<sup>3</sup>

3.2.4.4.2 Derivation of Potency Factors

Values for the cancer potency factors ( $K_L$  and  $K_M$  in the equations above) are derived by fitting a risk model to available exposure-response data from epidemiological studies of workers exposed to asbestos. Fitting is performed using the method of Maximum Likelihood Estimation (MLE), assuming that the observed number of cases in a group is a random variable described by the Poisson distribution.

In general, the preferred model for fitting utilizes individual-level observations. This allows for the exposure metric to be treated as a continuous variable, and also allows for the inclusion of categorical covariates of potential interest such as gender, calendar interval, race, and birth cohort. When the individual data are not available, then the data for individuals may be grouped according to a key exposure metric (CE10 for lung cancer, TSFE for mesothelioma), and the mid-point of the range for each model parameter is usually used in the fitting. In cases where the upper bound of the highest exposure category was not reported in the publication, the value for the upper bound was assumed to be the maximum exposure reported in the publication.

In cases where study authors reported a potency factor derived using an appropriate model, that value was retained for consideration. In cases where the authors did not report a potency factor derived by an appropriate method, EPA estimated the potency factor by fitting a model to grouped data, if they were reported. EPA fitting was performed using SAS. Appendix G provides the SAS codes that were employed. As a quality check, calculations were also performed using Microsoft Excel. Both methods yielded the same results to 3 or more significant figures.

When the potency factors were estimated by the study authors, EPA relied upon the confidence bounds reported by the authors. These were generally Wald-type bounds. Because, the inhalation unit risk (see below) is derived from the one-sided 95th% upper bound (which is equivalent to the upper bound of the two-sided 90th% upper bound), if the authors reported a two-side 95% confidence interval (i.e., from the 2.5<sup>th</sup> to the 97.5<sup>th</sup> bounds), EPA estimated the two-sided 90% confidence interval by back calculating the 5<sup>th</sup> and 95<sup>th</sup> confidence bounds, assuming a normal distribution.

When EPA performed the fitting, 90% two-sided confidence bounds around the potency factors were derived using the profile likelihood method. In this method, the  $100(1-\alpha)$  confidence interval is computed by finding the two values of the potency factor that yield a log-likelihood result that is equal to the maximum log-likelihood minus  $0.5 \cdot \chi^2(1-\alpha, 1)$ , i.e., central chi-square distribution with one degree of freedom and confidence level  $1-\alpha$ . For a 90% confidence interval, this is equal to the maximum log-likelihood minus 1.353.

3.3.4.4.3 Extrapolation from Workers to the general population to derive inhalation unit risk

Because EPA defines the cancer inhalation unit risk for asbestos as an estimate of the increased cancer risk from inhalation exposure to a concentration of 1 f/cc for a lifetime<sup>13</sup>, and the cancer potency factors are derived by fitting risk models to exposure-response data based on workers, it is necessary to adjust the worker-based potency factors to derive values that are applicable to an individual with a different

<sup>13</sup> Note that the lifetime inhalation unit risk is then applied to specific environmental exposure scenarios applicable to current asbestos uses; for specific worker exposure scenarios, the extrapolation factor described may not be applied.

5109 exposure pattern (e.g., a resident with continuous exposure). The extrapolation is based on the  
5110 assumption that the ratio of the risk of cancer in one population compared to another (both exposed to  
5111 the same level of asbestos in air) is related to the ratio of the amount of asbestos-contaminated air that is  
5112 inhaled per unit time (e.g., per year).

5113  
5114 For workers, EPA assumes a breathing rate of 10 m<sup>3</sup> of air per 8-hour work day ([U.S. EPA, 2009](#)). If  
5115 workplace exposure is assumed to occur 240 workdays/year, the volume of air inhaled in a year is  
5116 calculated as follows:

$$\text{Volume Inhaled (worker)} = 10 \text{ m}^3/\text{workday} \cdot 240 \text{ workdays/yr} = 2,400 \text{ m}^3/\text{yr}$$

5117  
5118  
5119  
5120 For a resident, EPA usually assumes a breathing rate of 20 m<sup>3</sup>/day ([U.S. EPA, 2009](#)). If exposure is  
5121 assumed to be continuous (24 hours per day, 365 days per year), the volume inhaled in a year is  
5122 calculated as follows:

$$\text{Volume Inhaled (resident)} = 20 \text{ m}^3/\text{day} \cdot 365 \text{ days/yr} = 7,300 \text{ m}^3/\text{yr}$$

5123  
5124  
5125  
5126 In this case, the extrapolation factor from worker to resident is:

$$\text{Extrapolation factor} = 7,300 / 2,400 = 3.042$$

5127  
5128  
5129  
5130 In the tables below (Section 3.2.4.5), the potencies are shown as calculated from epidemiological  
5131 studies, and the worker to other populations extrapolation factor is applied in the life-table analyses so  
5132 that the unit risks and IUR incorporate that extrapolation factor.

#### 5133 5134 3.2.4.4.4 Life-Table Analysis and Derivation of Inhalation Unit Risk

5135 Potency factors are not analogous to lifetime unit risks or cancer slope factors, and do not directly  
5136 predict the excess risk of lung cancer or mesothelioma in an exposed individual. Rather, the potency  
5137 factors are used in lifetable analyses for lung cancer and mesothelioma to predict the risk of dying as a  
5138 result of the exposure in a specified year of life. However, it is important to recognize that cancer risk in  
5139 a particular year of life is conditional on the assumption that the individual is alive at the start of the  
5140 year. Consequently, the risk of dying of an asbestos-related cancer within a specified year of life is  
5141 calculated as the product of two terms: the probability of being alive at the start of the year and the  
5142 probability of dying of the asbestos exposure within the specified year. The lifetime risk is then the sum  
5143 of all the yearly risks. This procedure is performed to calculate the lifetime risk both for an unexposed  
5144 individual (R<sub>0</sub>) and for an individual with exposure to asbestos (R<sub>x</sub>).

5145  
5146 “Extra risk” for cancer is a calculation of risk which adjusts for background incidence rates of the same  
5147 type of cancer, by estimating risk at a specified exposure level only among the fraction of the population  
5148 not expected to develop the cancer due to background causes, and is calculated as follows ([U.S. EPA,  
5149 2012](#)):

$$\text{Extra Risk} = (R_x - R_0) / (1 - R_0)$$

5150  
5151  
5152  
5153 For mesothelioma, because background risk (R<sub>0</sub>) is assumed to be zero, extra risk is the same as absolute  
5154 risk (R<sub>x</sub>).

5156 The unit risk is risk of incident cancer<sup>14</sup> per unit asbestos concentration (fiber/cc) in inhaled air. The unit  
5157 risk is calculated by using life table analysis to find the exposure concentration (EC) that yields a 1%  
5158 (0.01) extra risk of cancer. The 1% value is referred to as the Benchmark Response (BMR). This value  
5159 is used because it represents a cancer response level that is near the low end of the observable range  
5160 ([U.S. EPA, 2012](#)). Given the EC at 1% extra risk (EC<sub>01</sub>), the unit risk is the slope of a linear exposure-  
5161 response line from the origin through the EC<sub>01</sub>:

$$\text{Unit risk} = 0.01 / \text{EC}_{01}$$

5162  
5163  
5164  
5165 A unit risk value may be calculated based on both the best estimate and the 95% upper confidence  
5166 bound (UB) on the potency factor. The value based on the upper 95% confidence bound is normally  
5167 used for decision-making, since it corresponds to a lower 5% confidence bound (LB) on the exposure  
5168 level yielding 1% extra risk (LEC<sub>01</sub>). Inhalation unit risk is derived by statistically combining risks of  
5169 lung cancer and mesothelioma. This procedure is described below in the section on combining unit risks.  
5170

5171 Life table calculations require as input the all-cause and cause-specific mortality rates for the general  
5172 population in each year of life. The all-cause mortality data were obtained from the National Vital  
5173 Statistics Report Vol 66 No 3 Table 1 ([2017](#)), which provides data from the U.S. population in 2013.  
5174 Lung-cancer mortality rates were obtained by downloading 2016 mortality data for malignant neoplasms  
5175 of trachea, bronchus and lung (ICD-10 C33-C34) from CDC Wonder ([http://wonder.cdc.gov/ucd-  
5176 icd10.html](http://wonder.cdc.gov/ucd-icd10.html)). Because cause-specific mortality rates were given for 5-year intervals, the cause-specific  
5177 rate for each 5-year interval was applied to each age within the interval. For mesothelioma, the mortality  
5178 rate in the absence of asbestos exposure was assumed to be zero.  
5179

5180 The detailed equations for calculating lifetime excess cancer risk for a specified exposure concentration  
5181 in the presence of competing risks are based on the approach used by NRC ([1988](#)) for evaluating lung  
5182 cancer risks from radon. The equations are detailed in Appendix H. The SAS code for lung cancer life  
5183 table analysis was provided to EPA by NIOSH<sup>15</sup> and was adapted for use by a) entering the mortality  
5184 data noted above, b) adding an equation to compute extra risk, and c) adding a macro to solve for the  
5185 EC. The SAS code for mesothelioma was created by inserting user-defined equations for the  
5186 mesothelioma risk model into the NIOSH code. The SAS codes for performing the mesothelioma and  
5187 lung cancer life table calculations are provided in Appendix I. As a quality check, life table calculations  
5188 were also performed using Microsoft Excel. Both methods yielded the same results to 3 or more  
5189 significant figures.  
5190

### 5191 **3.2.4.5 Study Descriptions and Model Fitting Results**

5192 The asbestos exposure data and exposure assessment methods in studies of the Charleston, South  
5193 Carolina textile plant ([Elliott et al., 2012](#); [Hein et al., 2007](#)) are exceptionally detailed compared to most  
5194 asbestos studies. The methods used were innovative at the time, a large number of exposure  
5195 measurements cover the relevant study period, and detailed process and work history information were  
5196 available and utilized in estimating exposures. The exposure data used in studies of North Carolina  
5197 plants ([Loomis et al., 2019](#); [Elliott et al., 2012](#)) are also high quality. The methods were similar to those  
5198 developed for the studies of the South Carolina plant. However, relative to the South Carolina study, the

---

<sup>14</sup> IUR is for incident cancer, but the data available from epidemiology studies are only in terms of mortality (see Section 3.2.4.8)

<sup>15</sup> Beta Version. SAS 30NOV18, provided by Randall Smith, National Institute for Occupational Safety & Health.

5199 number of exposure measurements is smaller, and the historical process and work-history data are less  
5200 detailed. Nevertheless, the exposure data are of higher quality than those utilized in other studies of  
5201 occupational cohorts exposed to chrysotile. For both U.S. textile cohorts, the exposure assessment  
5202 methods and results have been published in full detail.

5203  
5204 Studies of the asbestos products factory in Chongqing, China ([Courtice et al., 2016](#); [Wang et al., 2013b](#);  
5205 [Deng et al., 2012](#); [Yano et al., 2001](#)) provide informative data on a cohort that has not been included in  
5206 previous risk assessments. The methods used to estimate worker exposures for exposure-response  
5207 analyses appear to have emulated those used in the U.S. textile-industry studies. Nevertheless,  
5208 confidence in the exposure data is lower because exposure measurements were made only in later years  
5209 in the study period, the number of measurements is small, and the methodology is not reported in detail.

5210  
5211 Information about the assessment of exposures for the Québec asbestos mining and milling cohorts is  
5212 presented in several papers ([Liddell and Armstrong, 2002](#); [1998](#); [Vacek, 1998](#); [Liddell et al., 1997](#);  
5213 [1993a](#); [1980a](#); [Mcdonald et al., 1980b](#)), but the reports are lacking important details and are sometimes  
5214 in conflict. Nevertheless, it is evident that exposure measurements do not cover the entire study period.  
5215 The number of measurements is not consistently reported but appears to be smaller than for either of the  
5216 U.S. textile cohorts, while the number of distinct jobs was larger. Moreover, all the reported  
5217 measurements were of total dust, rather than fibers. Some reports have suggested or used a conversion  
5218 factor, but the use of single factor for all operations is likely to introduce substantial exposure  
5219 misclassification since the relationship between total dust and fiber counts has been shown to vary  
5220 considerably by process.

5221  
5222 Fewer details are available about the assessment of exposures for studies of chrysotile miners in China  
5223 ([2014](#); [2013b](#); [Wang et al., 2012](#)). Although workshop- and job title-specific fiber concentrations were  
5224 estimated in the study in China, these estimates were based on a small number of paired samples and  
5225 important details of the exposure assessment are not available. The quality of the exposure data is  
5226 therefore difficult to judge.

5227  
5228 Cohorts are listed in order of the quality of exposure assessment with the highest quality cohorts first.  
5229 The cohorts from SC and NC were judged to have the highest quality exposure assessment and only  
5230 those results were carried forward for consideration on the cancer-specific unit risks and the overall  
5231 IUR. For the rest of the cohorts, results of modeling are reported, but not carried forward.

5232  
5233 ***South Carolina asbestos textile plant [carried forward for unit risk derivation]***

5234  
5235 Mortality in a cohort of workers at an asbestos textile plant in Charleston, South Carolina, USA has been  
5236 reported in several papers ([Elliott et al., 2012](#); [2008](#); [Hein et al., 2007](#); [Stayner et al., 1997](#); [Brown et al.,](#)  
5237 [1994](#); [1994](#); [Dement et al., 1983a](#)). Workers employed for at least one month between 1940 and 1965  
5238 were included; the cohort originally included only white men but was later expanded to include non-  
5239 whites and women.

5240  
5241 The Charleston plant produced asbestos textiles from raw chrysotile fibers imported from Canada  
5242 (Québec and British Columbia) and Rhodesia (now Zimbabwe). Purchased crocidolite yarns were also  
5243 woven in a small separate operation for about 25 years, but crocidolite was never carded or spun on site  
5244 ([Dement et al., 1994](#)). The total amount of crocidolite handled was 0.03% of the amount of asbestos  
5245 processed annually ([Dement et al., 1994](#)).

5247 Methods and results of exposure assessment for this cohort were published in detail by Dement et al.,  
 5248 (1983b) and summarized in subsequent publications (e.g., (Hein et al., 2007)). Engineering controls for  
 5249 dust levels were introduced in the plant beginning in the 1930s and the facility was believed to represent  
 5250 the best practice in the industry at the time (Dement et al., 1983b). Estimates of individual exposure  
 5251 were based on 5952 industrial hygiene air samples between 1930 and 1975. All samples before 1965  
 5252 were obtained by midget impinger; both impinger and membrane filter samplers were used from 1965  
 5253 until 1971, and afterward only membrane filter samplers were used. Phase-contrast microscopy (PCM)  
 5254 was used in conjunction with membrane filter sampling to estimate concentrations of fibers  $\geq 5\mu\text{m}$  in  
 5255 length. Further details of historical fiber counting rules are not reported, but fibers  $<0.25\ \mu\text{m}$  in diameter  
 5256 cannot be visualized by PCM and are normally not counted. Paired and concurrent samples by both  
 5257 methods were used to estimate job and operation-specific conversion factors from mppcf to f/cc. One  
 5258 hundred and twenty paired samples were collected in 1965 and 986 concurrent samples were collected  
 5259 during 1968-1971. Statistical analysis of the data indicated no significant trends in fiber/dust ratios over  
 5260 time and no significant differences among operations, except for preparation. Consequently, conversion  
 5261 factors of 8 PCM f/cc per mppcf for preparation and 3 PCM f/cc per mppcf for all other operations were  
 5262 adopted for further analysis. Fiber concentrations were estimated for 9 departments and 4 job categories  
 5263 by linear regression, accounting for time-related changes in process and dust control. Individual  
 5264 cumulative exposures were estimated by linking this job-exposure-matrix to detailed occupational  
 5265 histories for each worker.

5266  
 5267 The most up to date data for lung cancer and mesothelioma in the cohort were reported by Hein et al.  
 5268 (2007) based on follow-up of 3072 workers through 2001; 198 deaths from lung cancer and 3 deaths  
 5269 from mesothelioma were observed. Quantitative exposure-response relationships for lung cancer were  
 5270 estimated by Poisson regression modeling using a linear relative rate form. Cumulative chrysotile  
 5271 exposure in f/cc-yrs was lagged by 10 years and entered as a continuous variable with sex, race and age  
 5272 as covariates. Elliott et al. (2012) performed a similar analysis, except some members of the cohort were  
 5273 excluded to improve comparability with a cohort of textile workers from North Carolina (see below).  
 5274

5275 Hein et al. (2007) did not report exposure-response analysis or detailed data for mesothelioma in the  
 5276 Charleston cohort. All death certificates for deaths before ICD-10 in 1999 were investigated (Hein,  
 5277 personal communication) for mention of mesothelioma (3 deaths), no mesothelioma deaths after 1999  
 5278 were observed. Berman & Crump (2008) estimated  $K_M$  for the cohort from analyses of the original data  
 5279 obtained from the study investigators (see Table 3-3).  
 5280  
 5281  
 5282

**Table 3-3. Model Fitting Results for the South Carolina Cohort**

Endpoint	Source	Table in original publication	Potency Factor		Exposure Concentration associated with BMR (1% Extra Risk) (f/cc)		Lifetime Unit Risk (per f/cc)	
			MLE	95% UB	EC <sub>01</sub> MLE	LEC <sub>01</sub> 5% LB	MLE	95% UB
Lung Cancer	Hein et al. (2007) linear	Table 5	1.98E-02	2.80E-02	7.15E-2	5.06E-2	1.40E-01	1.98E-01
	EPA modeling of Hein et al. (2007) grouped data linear	Table 3	1.73E-02	2.22E-02	8.19E-2	6.38E-2	1.22E-1	1.57E-1

	Elliott et al. (2012) linear	Table 2	2.35E-02	3.54E-02	6.03E-2	4.00E-2	1.66E-1	2.50E-1
	Elliott et al. (2012) exponential	Table 2	5.13E-03	6.36E-03	2.44E-1	1.97E-1	4.09E-2	5.07E-2
<b>Mesothelioma</b>	Berman and Crump (2008) based on Hein et al. (2007)	Table 4	1.5E-09	3.3E-09	4.0E-1	1.8E-1	2.5E-2	5.5E-2

- 1) Details for the modeling for lung cancer are provided in Appendix G, Section 1. Details for the modeling of mesothelioma is provided in Berman and Crump (2008)
- 2) In EPA modeling of Hein et al. (2007) grouped data, alpha=1 and upper bound on the highest exposure interval was assumed 699.8 f/cc (the maximum exposure reported in the publication).
- 3) In calculations involving Elliott et al. (2012), the 95% upper bound on potency factor was calculated from the reported 97.5% upper bound as described above.
- 4) Berman and Crump (2008) reported mesothelioma potency number ( $K_M$ ) with 2 significant digits.

Selection of the results from the South Carolina cohort

As discussed above, for lung cancer, the modeling of individual data is preferred so results from Hein et al. (2007) as well as two results of Elliott et al. (2012) were carried forward for further consideration. For mesothelioma, only the results of modeling of the South Carolina cohort data by Berman and Crump (2008) are available, and those are will be carried forward for the unit risk derivation.

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

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. One of the three larger plants also carded, twisted and wove amosite fibers in a separate facility for 13 years (Loomis et al., 2009). Quantitative data on the amounts of amosite used are not available. However, the operation was isolated from general production and no amosite fibers were found in TEM analysis of archived samples from that plant or any other (Elliott et al., 2012).

Workers employed at least 1 day between 1950 and 1973 were enumerated from company records: 5770 workers (3975 men and 1795 women) and files of state and national health agencies were included and followed for vital status through 2003. Causes of death were coded to the ICD revision in force at the time of death. All conditions mentioned on the death certificate, including intermediate causes and other significant conditions were coded. Death certificate data were examined for any mention of mesothelioma and for ICD codes often applied to mesothelioma before a specific code for mesothelioma was introduced in 1999. Only one worker in the cohort, who did not develop lung cancer or mesothelioma, had a history of employment in the operation where amosite had been used.

Exposure assessment methods and results are described by Dement et al. (2009). The approach was similar to that used in South Carolina (Dement et al., 1983b) with updated statistical methods. Asbestos fiber concentrations were estimated from 3420 air samples taken from 1935 to 1986. Sampling until 1964 was by impinger; membrane filter sampling was introduced in 1964 and both methods were used until 1971, with only membrane filter sampling thereafter. Fibers longer than 5 μm captured on membrane filters were counted by PCM to estimate concentrations; further details of historical fiber

counting rules are not available. Paired and concurrent samples by both methods were used to estimate plant-, operation- and period-specific factors for converting dust to PCM-equivalent fiber concentrations. Fiber/dust ratios did not change significantly over time, so plant- and operation-specific conversion factors (range 1.6 (95% CI 0.4-2-8) fibers/mppcf to 8.0 (95% CI 7.4-8.7) fibers/mppcf) were used for further analysis. Fiber concentration data were analyzed using multivariable mixed models to estimate average concentrations by plant, department, job and time period. The operation and job categories of the job-exposure matrix were similar to those developed for South Carolina (2009; Dement et al., 1983a). These estimates were linked to individual work history records to estimate average and cumulative exposure to asbestos fibers for each worker. Detailed job titles within departments were missing for 27% of workers, mostly short-term; in these cases, exposure was estimated using the plant, period and department average (Loomis et al., 2009). For years prior to 1935, when no exposure measurements and few work history records were available, exposures were assumed to have been equal to those in 1935, before dust controls were implemented.

In total, 277 deaths from lung cancer occurred during follow-up. Exposure-response analyses for lung cancer included 3803 workers in production jobs in 3 of the 4 study plants and 181 lung cancer deaths. Data were analyzed using conventional log-linear Poisson regression models adjusted for age, sex, race, decade of follow-up and birth cohort. Results were reported as relative rates per 100 f/cc-yrs with exposure lags of 0 to 30 years (Loomis et al., 2009).

Elliott et al. (2012) also evaluated exposure-response relationships for lung cancer in the North Carolina cohort using Poisson regression with both log-linear and additive relative rate model forms. Models were adjusted for age, sex, race, calendar period and birth cohort. Results were reported per 100 f/cc-yrs of cumulative fiber exposure with lags of 0, 10 or 20 years.

During the follow-up of the North Carolina cohort, four deaths were coded to mesothelioma according to the ICD-10, and, prior to the implementation of ICD-10, four deaths coded as cancer of the pleura and one death coded as cancer of the peritoneum were observed (2019; Loomis et al., 2009). Because Loomis et al. (2019) reported only pleural cancers before ICD-10, EPA modeled the exposure-response for mesothelioma using data from 1999 onward when ICD-10 was in use (see Table 3-4).

**Table 3-4. Model Fitting Results for the North Carolina Cohort**

Endpoint	Source	Table in original publication	Potency Factor		Exposure Concentration associated with BMR (1% Extra Risk) (f/cc)		Lifetime Unit Risk (per f/cc)	
			MLE	95% UB	EC <sub>01</sub> MLE	LEC <sub>01</sub> 5% LB	MLE	95% UB
Lung Cancer	Elliott et al. (2012) linear	Table 2	1.20E-3	2.71E-3	1.180	5.23E-1	8.47E-3	1.91E-2
	Elliott et al. (2012) exponential	Table 2	9.53E-4	1.40E-3	1.32	8.95E-1	7.60E-3	1.12E-2
	Loomis et al. (2009) exponential	Table 6	1.01E-3	1.47E-3	1.24	8.53E-1	8.06E-3	1.17E-2
	EPA modeling of Loomis et al. (2009) grouped data linear	Table 5	8.08E-4	1.31E-3	1.75	1.08	5.71E-3	9.25E-3



<b>Mesothelioma</b>	EPA modeling of Loomis et al. (2019)	Table S1b	2.44E-9	5.04E-9	2.45E-1	1.19E-1	4.08E-2	8.42E-2
---------------------	--------------------------------------	-----------	---------	---------	---------	---------	---------	---------

- 1) Details for the modeling are provided in Appendix G, Section 2.
- 2) In EPA modeling of the Loomis et al. (2009) lung cancer grouped data, alpha=1 and the upper bound on the highest exposure interval was assumed 2,194 f/cc (the maximum exposure reported in the publication).
- 3) In calculations involving Loomis et al. (2009) and Elliott et al. (2012) lung cancer modeling, the 95% upper bound on potency factor was calculated from the reported 97.5% upper bound as described above.
- 4) In EPA modeling of the Loomis et al. (2019) mesothelioma data, the two top TSFE groups were combined by adding cases and person-years; TSFE, concentration and duration were calculated by averaging person-year-weighted results for both groups.

Selection of the results from the North Carolina Cohort

As discussed above, for lung cancer, the modeling of individual data is preferred so results from Loomis et al. (2009) as well as two results of Elliott et al. (2012) are carried forward for further consideration. The mesothelioma results from the Loomis et al. (2019) sub-cohort of workers that were evaluated with ICD-10 are carried forward for unit risk derivation.

*Chongqing, China, asbestos products factory*

An initial report on mortality among workers at a plant in Chongqing, China, that produced a variety of asbestos products was published by Yano et al. (2001). A fixed cohort of 515 men employed at least one year and active as of 1 January 1972 was established and followed for mortality using plant records. Women were not included in the original cohort as none were hired before 1970. Further analyses based on extended follow-up were reported in subsequent papers (Courtice et al., 2016; Wang et al., 2013b; Deng et al., 2012). The 2008 follow-up of the cohort added 279 women employed between 1970 and 1972 (Wang et al., 2013b).

The Chongqing plant opened in 1939 and expanded in the 1950s; a range of asbestos products, including textiles, friction materials, rubber-impregnated goods and cement were produced (Yano et al., 2001). The plant is reported to have used chrysotile asbestos from two mines in Sichuan Province; amphibole contamination in bulk samples from these mines assessed by transmission electron microscopy (TEM) was found to be below the limit of detection (LOD <0.001%, (Courtice et al., 2016; Yano et al., 2001). An independent study of commercial chrysotile extracted from six mines in China reported tremolite content of 0.002 to 0.312% by weight (Tossavainen et al., 2001), but it is not clear whether these mines supplied chrysotile to the Chongqing factory.

Deng et al. (2012) reported on the methods of exposure assessment. Fiber concentrations for four operations (raw materials processing, textile carding and spinning, textile weaving and maintenance, and rubber and cement production) were estimated from 556 area measurements taken every 4 years from 1970 to 2006. Only total dust was measured before 1999, while paired measurements of dust and fibers were taken subsequently. A total of 223 measurements of fiber concentration by PCM were available. Paired dust and fiber samples from 1999-2006 were used to estimate dust to PCM fiber-equivalent concentrations for the 1970-1994 using an approach similar to that of Dement et al. (2009) and the estimated and measured concentrations were combined for analysis; however, no details were reported on what operations and jobs these estimates represent. Individual cumulative fiber exposures were estimated from the concentration data and the duration of employment in each area of the plant. Work histories were reported to have been stable with few job changes (Deng et al., 2012).

5406 Exposure-response data for lung cancer in the Chongqing cohort have been reported in several papers.  
 5407 Deng et al. (2012) analyzed data for 586 men and women followed to 2006 and reported quantitative risk  
 5408 estimates for cumulative chrysotile exposure obtained by fitting log-linear and additive relative rate  
 5409 models with adjustment for age, smoking and calendar period. Wang et al. (2014) published additional  
 5410 analyses of the same study population but truncated the follow-up period from 1981 to 2006 to make it  
 5411 more comparable with a study of Chinese asbestos miners (described below). The vital status of this  
 5412 cohort was updated to 2008 and an analysis including follow-up from 1972 to 2008 was published by  
 5413 Courtice et al. (2016). The latter papers provide quantitative risk estimates from internal analyses with  
 5414 log-linear relative rate models. Papers on the Chongqing cohort provide informative exposure-response  
 5415 information in units of f/cc-years from Cox or Poisson regression analyses. However, there is potential  
 5416 for misclassification of exposures due to the relatively small number of exposure measurements, the lack  
 5417 of fiber measurements before 1999 and use of area rather than personal sampling (Deng et al., 2012).  
 5418 Fitting results from Deng et al. (2012) are provided in Table 3-5.  
 5419  
 5420

**Table 3-5. Model Fitting Results for the Chongqing China Cohort**

Endpoint	Source	Table in original publication	Potency Factor		Exposure Concentration associated with BMR (1% Extra Risk) (f/cc)		Lifetime Unit Risk (per f/cc)	
			MLE	95% UB	EC <sub>01</sub> MLE	LEC <sub>01</sub> 5% LB	MLE	95% UB
Lung Cancer	Deng et al. (2012) exponential	Table 3	2.08E-3	3.02E-3	6.03E-1	4.15E-1	1.66E-2	2.41E-2
	Deng et al. (2012) Linear	Table 3	4.21E-3	4.56E-3	3.36E-1	3.11E-1	2.97E-2	3.22E-2

5421 Details for the modeling are provided in Deng et al. (2012)  
 5422

5423 Data for mesothelioma were reported for follow-up through 2008 of the expanded cohort including  
 5424 women (Wang et al., 2013b). Three deaths coded as mesothelioma according to the ICD-10 (2 among  
 5425 men and 1 among women) were recognized and only SMRs were reported separately for men and  
 5426 women (Wang et al., 2013b). Data on the exposure levels of the mesothelioma cases are not available,  
 5427 however, so model fitting was not possible. No other analyses of mesothelioma have been reported for  
 5428 the Chongqing cohort.  
 5429

### *Québec, Canada asbestos mines and mills [not carried forward]*

5430  
 5431 Data from studies of miners, millers and asbestos products factory workers at several facilities in  
 5432 Québec, Canada are reported in multiple publications (Liddell and Armstrong, 2002; 1998; Vacek, 1998;  
 5433 Liddell et al., 1997; 1993a; 1980a; McDonald et al., 1980b). The earliest publication, McDonald et al.  
 5434 (1980b), included 11,379 miners and millers from Québec, Canada who were born between 1891 and  
 5435 1920 and had worked for at least a month in the mines and mills and were followed to 1975. Additional  
 5436 findings based on follow-up of the cohort to 1988 were reported by McDonald et al. (1993a), and further  
 5437 extended to 1992 by Liddell et al. (1997). Trace amounts of tremolite have been reported in samples  
 5438 from the Canadian mines (IARC, 2012), with the amounts varying between mines (Liddell et al., 1997).  
 5439  
 5440

5441 The most detailed description of exposure assessment methods used in the Québec studies is given by  
 5442 Gibbs and Lachance (1972). Additional details and updates are given in later publications (e.g., Liddell

5443 [et al., 1997](#); [McDonald et al., 1980b](#)). Total dust concentrations (in mppcf) were estimated using midget  
5444 impinger measurements taken from 1948 to 1966 ([Gibbs and Lachance, 1972](#)). Several different figures  
5445 are reported for the total number of dust measurements used to estimate exposures: Gibbs and Lachance  
5446 ([Gibbs and Lachance, 1972](#)) reported 3096; McDonald et al. ([1980b](#)) reported “well over 4000,” and  
5447 McDonald et al. ([1980a](#)) reported 10,205. Annual dust concentrations for 5783 unique jobs were  
5448 assigned according a 13-point scale with categories of 0.5, 2, 7, 12, 17, 22, 27, 32, 37, 42, 47, 70 and  
5449 140 mppcf. The authors describe the categories as “approximating to the mean”, but the methods of  
5450 analyzing the exposure measurements and developing the categories are not reported. Different  
5451 approaches were used to estimate exposures in earlier and later years when dust data were judged to be  
5452 inadequate; exposures in years before 1948 were reportedly estimated by expert assessment based on  
5453 interviews with workers and company personnel, while those after 1966 were estimated by extrapolation  
5454 from the previously measured levels ([Liddell et al., 1997](#)). Cumulative dust exposure (in mppcf-years)  
5455 for each worker was estimated from the assigned dust concentrations and individual work histories;  
5456 estimated exposures in years before 1938 were multiplied by 1.65 to account for longer work weeks at  
5457 that time ([Liddell et al., 1997](#)). Fibers reportedly accounted for 8-15% of total dust ([Gibbs and](#)  
5458 [Lachance, 1972](#)). Most exposure-response analyses for the cohort were reported relative to cumulative  
5459 dust exposure in mppcf. However, in a case-control study of lung cancer, McDonald et al. ([1980a](#))  
5460 adopted an overall conversion factor of 3.14 f/cc per mppcf, citing 11,819 fiber measurements (methods  
5461 of measurement and analysis not described), “unfortunately with little overlap” with the dust data. In  
5462 another publication, McDonald et al. ([1980b](#)) suggested fiber concentrations per cc would be between 1  
5463 and 7 per mppcf. Liddell et al. ([1984](#)) subsequently reported conversion factors ranging from 3.44 to  
5464 3.67 f/cc per mppcf. Gibbs (1994) reported a 95% confidence interval of  $0.58(D)^{0.68}$  to  $55.7(D)^{0.68}$ , where  
5465 D is the dust concentration measured by impinger, for the ratio of fibers to dust (units not specified).  
5466 Gibbs and Lachance ([1972](#)), reported that the correlation between midget impinger and membrane filter  
5467 counts (0.32) was poor and suggested that “no single conversion factor was justified”. Berman ([2010](#))  
5468 performed an analysis of dust samples from the Québec mines and found that one third of the PCM  
5469 structures samples in the dust were not asbestos, and that about one third of structures counted by PCM  
5470 were also counted by TEM. These findings along with the uncertainties concerning what is an  
5471 appropriate conversion factor raise significant concerns about the accuracy of the f/cc estimates of  
5472 exposure from the Québec studies.

5473  
5474 Most analyses of the Québec cohort compared workers’ mortality to the general population using SMRs  
5475 (e.g., [Liddell et al., 1997](#); [1993a](#); [McDonald et al., 1980b](#)). Liddell et al. ([1998](#)) conducted a nested case-  
5476 control study of lung cancer in a subset of workers at the mines and mills that were included in the  
5477 previous cohort studies and workers from an asbestos products factory. Subsequent publications by  
5478 Vacek et al. ([1998](#)), and Liddell and Armstrong ([2002](#)) presented more detailed analyses on a subset of  
5479 the cohort to examine the role of intensity and timing of exposure, and of potential effect modification  
5480 by cigarette smoking. All exposure-response analyses of lung cancer in the Québec studies utilized total  
5481 dust exposure expressed in mppcf. Estimates of  $K_L$  or analogous additive relative risk measures have not  
5482 been reported for these studies.

5483  
5484 Liddell et al. ([1997](#)) reported 38 cases of mesothelioma in the last follow-up through 1992. The same  
5485 publication also reported that mesothelioma as a cause of death was almost unknown in Quebec until  
5486 1960, which was more than 40 years after start of the cohort’s exposure. Because of that, the method of  
5487 ascertainment for mesothelioma for the cohort was considered to be insufficient because it did not  
5488 include likely mesothelioma deaths and mesothelioma results are not reported in a way to allow for  
5489 derivations of  $K_M$  for the cohort once mesothelioma reporting in Quebec became reliable.

5491 Berman and Crump (2008) estimated  $K_L$  for the Québec cohort from analyses of original data obtained  
 5492 from the study investigators. A single conversion factor for all operations of 3.14 fibers/cc per mppcf  
 5493 was assumed in this analysis. Results are presented in Table 3-6.  
 5494  
 5495

**Table 3-6. Model Fitting Results for the Quebec, Canada Cohort**

Endpoint	Source	Table in original publication	Potency Factor		Exposure Concentration associated with BMR (1% Extra Risk) (f/cc)		Lifetime Unit Risk (per f/cc)	
			MLE	95% UB	EC <sub>01</sub> MLE	LEC <sub>01</sub> 5% LB	MLE	95% UB
Lung Cancer	Berman and Crump (2008) modeling of grouped data linear	Table B1	2.9E-4	4.10E-4	4.88	3.45	2.05E-3	2.90E-3

1. Details for the modeling are provided in Berman and Crump (2008).
2. In Berman and Crump (2008) modeling of the grouped data,  $\alpha=1.15$  was fitted.

***Qinghai, China asbestos mine [not carried forward]***

5501 Wang et al. (2014; 2013a; 2012) reported findings from exposure-response analyses of a cohort of 1539  
 5502 workers at a chrysotile mine in Qinghai Province, China who were on the registry January 1, 1981 and  
 5503 had been employed for at least one year. The cohort was followed for vital status from 1981 to 2006.  
 5504

5505 The mine opened in 1958 (no closing date reported) and produced commercial chrysotile with no  
 5506 detectable tremolite content (LOD 0.1%, (Wang et al., 2012)). Total dust concentrations in the mine  
 5507 were measured periodically between 1984 and 1995 by area sampling in fixed locations (Wang et al.,  
 5508 2012). Sampling was performed according to Chinese national standards. The number of measurements  
 5509 during this period is not reported. An additional 28 measurements were taken in 2006 in 8 different  
 5510 workshops. Dust concentrations in  $\text{mg}/\text{m}^3$  were converted to f/cc using a linear regression model based  
 5511 on 35 paired measurements taken in 1991. Fiber concentrations were estimated by workshop and job  
 5512 title for the period 1984-2006, apparently using a single conversion factor. The estimation methods are  
 5513 not described in detail in English-language publications, but further details may be available in Chinese-  
 5514 language publications referenced by Wang et al. (2013a; 2012), but not reviewed here. As recognized by  
 5515 the authors (Wang et al., 2013a), there is potential for exposure measurement error due to the conversion  
 5516 from mppcf to f/cc-yrs which was based on 35 paired samples that were collected in only one year, for  
 5517 an unspecified number of operations.  
 5518

5519 Wang et al. (2013a) report estimates of SMRs and standardized rate ratios (SRRs) for lung cancer by  
 5520 categorical levels of f/cc-yrs, stratified by smoking status. EPA used these combined data for smokers  
 5521 and non-smokers to estimate a value and confidence interval for  $K_L$  based on the linear relative risk  
 5522 model.  
 5523

5524 Wang et al. (2014) presented rate ratios for categorical and continuous exposure variables using log-  
 5525 linear Cox proportional hazards models adjusted for age and smoking. The findings from the Cox model  
 5526 are useful for risk assessment in that asbestos exposure is modeled as a continuous variable using  
 5527 individual level data, which generally provides a more statistically powerful examination of exposure-  
 5528 response relationships than a grouped analysis. Furthermore, the Cox PH analyses by Wang et al. (2014)  
 5529 adjusted for smoking, whereas the earlier SMR and SRR analyses (Wang et al., 2013a) did not. Fitting  
 5530 results are shown in Table 3-7.

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No data on mesothelioma have been reported for the Qinghai mining cohort.

**Table 3-7. Model Fitting Results for the Qinghai, China Cohort**

Endpoint	Source	Table in original Publication	Potency Factor		Exposure Concentration associated with BMR (1% Extra Risk) (f/cc)		Lifetime Unit Risk (per f/cc)	
			MLE	95% UB	EC <sub>01</sub> MLE	LEC <sub>01</sub> 5% LB	MLE	95% UB
Lung Cancer	EPA modeling of Wang et al. (2013a) grouped data linear	Tables 5 and 6	2.16E-2	6.47E-2	6.56E-2	2.19E-2	1.53E-1	4.57E-1
	Wang et al. (2014) exponential	Table 3	1.82E-3	2.63E-3	6.89E-1	4.77E-1	1.45E-2	2.10E-2

- 1) Details for the modeling are provided in Appendix I, Section 3.
- 2) In EPA modeling of the Wang et al. (2013a) grouped data, alpha was fitted (1.21) and the upper bound on the highest exposure interval was assumed 1097 f/cc (the maximum exposure reported in Wang et al. (2014) for this cohort). The data in Tables 5 and 6 were combined in modeling.
- 3) In calculations involving Wang et al. (2014) results of lung cancer modeling, the reported hazard ratio at exposure level of 100 f/cc-yrs was 1 and it was used to calculate the potency factor as follows: potency factor =  $\ln(1.2) / 100$ .

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Cancer risk ranges by Industry

Historically, it has been proposed in the asbestos literature, that cancer risks may differ by industry (e.g., U.S. EPA (1986), Berman and Crump (2008) and references therein). While lifetime unit risks of mesothelioma are derived only from the two cohorts (the NC and SC textiles cohorts), the lifetime unit risks of lung cancer are available from both those two-cohorts and from two other cohorts (Quebec, Canada; Qinghai, China) and that allows comparison of lung cancer risks by industry (textile vs. mining); one remaining cohort included multiple industries and was not included in the comparison (Chongqing, China). Because there are only two cohorts in each industry category, only a rough comparison is possible by looking at range of risks for each industry. Results are in Table 3-8 below. It is clear that the range of risks in each cell is very wide; however, this limited data indicates that among these cohorts exposed only to chrysotile asbestos, the lifetime unit risks of lung cancer are not different between textile and mining industries.

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**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.

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3.2.4.6 Summary of Results of North and South Carolina Cohorts

As discussed above, the cohorts from NC and SC, and the models based on individual-level data are listed in the Table 3-9 below.

Table 3-9. Cohorts and Preferred Statistical Models for SC and NC Cohorts

Cohort	Endpoint	Source	Potency Factor		Exposure Concentration associated with BMR (1% Extra Risk) (f/cc)		Lifetime Unit Risk (per f/cc)	
			MLE	95% UB	EC <sub>01</sub> MLE	LEC <sub>01</sub> 5% LB	MLE	95% UB
South Carolina	Lung Cancer	Hein et al. (2007) linear	1.98E-2	2.80E-2	7.15E-2	5.06E-2	1.40E-1	1.98E-1
		Elliott et al. (2012) linear	2.35E-2	3.54E-2	6.03E-2	4.00E-2	1.66E-1	2.50E-1
		Elliott et al. (2012) exponential	5.13E-3	6.36E-3	2.44E-1	1.97E-1	4.09E-2	5.07E-2
	Mesothelioma	Berman and Crump (2008) based on Hein et al. (2007)	1.5E-9	3.3E-9	4.0E-1	1.8E-1	2.5E-2	5.5E-2
North Carolina	Lung Cancer	Elliott et al. (2012) linear	1.20E-3	2.71E-3	1.18	5.23E-1	8.47E-3	1.91E-2
		Elliott et al. (2012) exponential	9.53E-4	1.40E-3	1.32	8.95E-1	7.60E-3	1.12E-2
		Loomis et al. (2009) exponential	1.01E-3	1.47E-3	1.24	8.53E-1	8.06E-3	1.17E-2
	Mesothelioma	EPA modeling of Loomis et al. (2019)	2.44E-9	5.04E-9	2.45E-1	1.19E-1	4.08E-2	8.42E-2

Addressing underascertainment of mesothelioma

Unlike for lung cancer, where the relative risk model is used, the model used for mesothelioma is an absolute risk model. For mesothelioma, the undercounting of cases (underascertainment) is a particular concern given the limitations of the ICD classification systems used prior to 1999. In practical terms, this means that some true occurrences of mortality due to mesothelioma are missed on death certificates and in almost all administrative databases such as the National Death Index. Even after the introduction of a special ICD code for mesothelioma with the introduction of ICD-10 in 1999, detection rates were still imperfect (Camidge et al., 2006; Pinheiro et al., 2004), and the reported numbers of cases typically reflect an undercount of the true number (note that the North Carolina cohort was updated in 2003, soon after the introduction of ICD-10). The undercounts are explained by the diagnostic difficulty of mesothelioma, both because of its rarity, variety of clinical presentations, and complexity of cytological confirmation. For example, primary diagnosis of pleural mesothelioma is by chest exam and pleural effusion, but the latter is absent in 10-30% of pleural mesothelioma cases (e.g., Ismail-Khan et al., 2006).

5583 There is no single or set of morphological criteria that are entirely specific for mesothelioma ([Whitaker,](#)  
 5584 [2000](#)). Peritoneal mesothelioma diagnosis is challenging to differentiate between mesothelioma and  
 5585 ovarian or peritoneal serous carcinoma, with these tumors have a common histogenesis, may be difficult  
 5586 to differentiate morphologically and co-express many of the diagnostic markers ([Davidson, 2011](#)). To  
 5587 account for various sources of underascertainment of mesothelioma deaths, U.S. EPA ([2014c](#)),  
 5588 following Kopylev et al. ([2011](#)), developed a multiplier of risk for mesothelioma deaths before and after  
 5589 introduction of ICD-10. Although this procedure was developed based on the Libby Worker cohort, the  
 5590 problematic diagnostic issues described above are agnostic to the fiber type exposure. The developed  
 5591 multiplier ([U.S. EPA, 2014c](#)) is 1.39 with confidence interval (0.80, 2.17). Table 3-10 shows the  
 5592 mesothelioma unit risks adjusted for underascertainment.  
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**Table 3-10. Addressing Underascertainment of Mesothelioma**

Cohort	Source	Mesothelioma Unit risk (per f/cc)	Mesothelioma UB unit risk (per f/cc)	Adjusted Mesothelioma Unit Risk (per f/cc)	Adjusted Mesothelioma UB risk (per f/cc)
South Carolina	Berman and Crump ( <a href="#">2008</a> ) based on Hein et al. ( <a href="#">2007</a> )	2.5E-2	5.5E-2	3.48E-2	7.65E-2
North Carolina	EPA modeling of Loomis et al. ( <a href="#">2019</a> )	4.08E-2	8.42E-2	5.67E-2	1.17E-1

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5596 **3.2.4.6.1 Combining Lung Cancer Unit Risk and Mesothelioma Unit**  
 5597 **Risk**

5598 Once the cancer-specific lifetime unit risks are obtained, the two are then combined. It is important to  
 5599 note that this estimate of overall potency describes the risk of mortality from cancer at either of the  
 5600 considered sites and is not just the risk of an individual developing both cancers concurrently. Because  
 5601 each of the unit risks is itself an upper bound estimate, summing such upper bound estimates across  
 5602 mesothelioma and lung cancer mortality is likely to overpredict the upper bound on combined risk.  
 5603 Therefore, following the recommendations of the *Guidelines for Carcinogen Risk Assessment* ([U.S.](#)  
 5604 [EPA, 2005](#)), a statistically appropriate upper bound on combined risk was derived as described below.  
 5605

5606 Because the estimated risks for mesothelioma and lung cancer mortality were derived using maximum  
 5607 likelihood estimation, it follows from statistical theory that each of these estimates of risk is  
 5608 approximately normally distributed. For independent normal random variables, a standard deviation for  
 5609 a sum is easily derived from individual standard deviations, which are estimated from confidence  
 5610 intervals: standard deviation = (upper bound – central estimate) ÷  $Z_{0.95}$ , where  $Z_{0.95}$  is a standard normal  
 5611 quantile equal to 1.645. For normal random variables, the standard deviation of a sum is the square root  
 5612 of the sum of the squares of individual standard deviations. It is important to mention here that  
 5613 assumption of independence above is a theoretical assumption, but U.S. EPA ([2014c](#)) conducted an  
 5614 empirical evaluation and found that the assumption of independence in this case does not introduce  
 5615 substantial error.  
 5616

5617 In order to combine the unit risks, first obtain an estimate of the standard deviation (SD) of the sum of  
 5618 the individual unit risks as:

$$SD = \sqrt{[(UB\ LC - CE\ LC) \div 1.645]^2 + [(UB\ M - CE\ M) \div 1.645]^2}$$

5621

5622 Where,  
 5623 UB – upper bound unit risk; CE – central estimate of unit risk; LC – lung cancer  
 5624 M – mesothelioma

5625  
 5626 Then, the combined central estimate of risk (CCE) of mortality from either mesothelioma or lung cancer  
 5627 is  $CCE = (CE_{LC} + CE_M)$  per fiber/cc, and the combined IUR is  $CCE + SD \times 1.645$  per fiber/cc.  
 5628

5629 **3.2.4.7 Inhalation Unit Risk Derivation**

5630 To illustrate the range of estimates in the estimates of the IUR, central risks and upper bounds for the  
 5631 combined IUR for South and North Carolina cohorts are presented in Table 3-10.  
 5632

5633 **Table 3-11. Range of Estimates of Estimated Central Unit Risks and IURs for North and South**  
 5634 **Carolina Cohorts**

Lung Cancer Source	Central Unit Risk Lung Cancer	Upper Bound Unit Risk Lung Cancer	Mesothelioma Source	Central Unit Risk Meso	Upper Bound Unit Risk Meso	Combined Central Unit Risk (Lung Cancer + Meso)	Lifetime Cancer IUR (per f/cc)
South Carolina Cohort							
Hein et al. (2007) Linear	1.40E-1	1.98E-1	Berman and Crump (2008) based on Hein et al. (2007)	3.48E-2	7.65E-2	0.175	0.25
Elliott et al. (2012) Linear	1.66E-1	2.50E-1	Berman and Crump (2008) based on Hein et al. (2007)	3.48E-2	7.65E-2	0.201	0.29
Elliott et al. (2012) Exponential	4.09E-2	5.07E-2	Berman and Crump (2008) based on Hein et al. (2007)	3.48E-2	7.65E-2	0.076	0.12
North Carolina Cohort							
Elliott et al. (2012) Linear	8.47E-3	1.91E-2	EPA modeling of Loomis et al. (2019)	5.67E-2	1.17E-1	0.065	0.13
Elliott et al. (2012) Exponential	7.60E-3	1.12E-2	EPA modeling of Loomis et al. (2019)	5.67E-2	1.17E-1	0.064	0.12
Loomis et al. (2009) Exponential	8.06E-3	1.17E-2	EPA modeling of Loomis et al. (2019)	5.67E-2	1.17E-1	0.065	0.13
Combinations of South and North Carolina Cohorts lung and mesothelioma unit risks							
SC Hein et al. (2007) Linear	1.40E-1	1.98E-1	NC EPA modeling of Loomis et al. (2019)	5.67E-2	1.17E-1	0.197	0.28



Lung Cancer Source	Central Unit Risk Lung Cancer	Upper Bound Unit Risk Lung Cancer	Mesothelioma Source	Central Unit Risk Meso	Upper Bound Unit Risk Meso	Combined Central Unit Risk (Lung Cancer + Meso)	Lifetime Cancer IUR (per f/cc)
SC Elliott et al. (2012) Linear	1.66E-1	2.50E-1	NC EPA modeling of Loomis et al. (2019)	5.67E-2	1.17E-1	0.223	0.33
SC Elliott et al. (2012) Exponential	4.09E-2	5.07E-2	NC EPA modeling of Loomis et al. (2019)	5.67E-2	1.17E-1	0.098	0.16
NC Elliott et al. (2012) Linear	8.47E-3	1.91E-2	SC Berman and Crump (2008) based on Hein et al. (2007)	3.48E-2	7.65E-2	0.043	0.09
NC Elliott et al. (2012) Exponential	7.60E-3	1.12E-2	SC Berman and Crump (2008) based on Hein et al. (2007)	3.48E-2	7.65E-2	0.042	0.08
NC Loomis et al. (2009) Exponential	8.06E-3	1.17E-2	SC Berman and Crump (2008) based on Hein et al. (2007)	3.48E-2	7.65E-2	0.043	0.08

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The values of the estimated IURs range from 0.08 per f/cc to 0.33 per f/cc. There is about a four-fold difference between lowest and highest IUR estimates – a very low range of model uncertainty in risk assessment.

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### 3.2.4.7.1 Selecting the Preferred Model Forms for Lung Cancer

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. For the NC exponential model, the AIC was 2020.53 compared to 2327.1 for the linear model (Elliott et al., 2012). When AIC-based comparisons are made, differences in AIC within 2 AIC units are generally considered to be indistinguishable with respect to model fit; models with AIC 10 units higher than the best model “have either essentially no support, and might be omitted from further consideration, or at least those models fail to explain some substantial explainable variation in the data” (Burnham and Anderson, 2002). For lung cancer in both South Carolina and North Carolina, the fit of the exponential models is hundreds of AIC units lower than the linear relative rate models. Such differences in AIC clearly differentiate the quality of the model fit, and although the linear model (which is the traditional EPA model and is used for lung cancer modeling in asbestos assessment (U.S. EPA, 1988b) is shown in the Table 3-11 for comparison, only the exponential models-based risks for lung cancer are used in the final IUR derivation. For the results from North Carolina, there were two candidate exponential models (Elliott et al., 2012; Loomis et al., 2009). Both used Poisson regression and controlled for the same set

of covariates, but the Loomis et al. (2009) publication reported on 181 lung cancer deaths while Elliot et al.(2012) reported on 159 lung cancer deaths. Only the North Carolina lung cancer results from Loomis et al. (2009) were further advanced in the IUR derivation.

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).

**Table 3-12. Estimated Central Unit Risks and IURs for North and South Carolina Cohorts and Preferred Models for Lung Cancer and Mesothelioma**

Lung Cancer Source	Central Unit Risk Lung Cancer	Upper Bound Unit Risk Lung Cancer	Mesothelioma Source	Central Unit Risk Meso	Upper Bound Unit Risk Meso	Combined Central Unit Risk (Lung Cancer + Meso)	Lifetime Cancer IUR (per f/cc)
SC Elliott et al. (2012) Exponential	4.09E-2	5.07E-2	SC Berman and Crump (2008) based on Hein et al. (2007)	3.48E-2	7.65E-2	0.076	0.12
NC Loomis et al. (2009) Exponential	8.06E-3	1.17E-2	NC EPA modeling of Loomis et al. (2019)	5.67E-2	1.17E-1	0.065	0.13
SC Elliott et al. (2012) Exponential	4.09E-2	5.07E-2	NC EPA modeling of Loomis et al. (2019)	5.67E-2	1.17E-1	0.098	0.16
NC Loomis et al. (2009) Exponential	8.06E-3	1.17E-2	SC Berman and Crump (2008) based on Hein et al. (2007)	3.48E-2	7.65E-2	0.043	0.08

None of these combinations of IUR estimates account for two important biases – each of which underestimates the true risk of incident cancer associated with exposure to chrysotile asbestos.

### 3.2.4.8 Biases in the Cancer Risk Values

#### Bias in use of mortality data

The endpoint studied for both mesothelioma and lung cancer was mortality, not cancer incidence. Cancer incidence data are not available for any of the chrysotile asbestos cohorts. According to the National Cancer Institute’s Surveillance Epidemiology and End Results (SEER) data on cancer incidence, mortality, and survival (Howlader et al., 2013), the median length of survival for lung cancer is less than 1 year, with 2-year survival for males about 25% and 5-year survival for males about 17%. For lung cancer, any bias would be expected to be low because the cancer slope factor (K<sub>L</sub>) is estimated based upon the relative risk. For mesothelioma, the median length of survival with mesothelioma is less

than 1 year, with 2-year survival for males about 20%, and 5-year survival for males about 6%. Thus, because the cancer slope factor ( $K_M$ ) is based on the absolute risk, any missed incident cases of mesothelioma will necessarily underestimate the total mesothelioma risk associated with chrysotile asbestos and in the absolute risk model even one incident case close to the follow-up date and missed in follow-up will increase the risk estimate.

Bias in assessing of mortality corresponding to other cancer endpoints

There is evidence that other cancer endpoints may also be associated with exposure to the commercial forms of asbestos. IARC concluded that there was sufficient evidence in humans that commercial asbestos (chrysotile, crocidolite, amosite, tremolite, actinolite, and anthophyllite) was causally associated with lung cancer and mesothelioma, as well as cancer of the larynx and the ovary (Straif et al., 2009). EPA lacked quantitative estimates of the risks of cancers of the larynx and the ovary from chrysotile asbestos. While the additional risks from ovarian and laryngeal cancer are likely to be smaller than the risks of lung cancer and mesothelioma, failing to account for those risks in the IUR necessarily underestimates the total cancer risk associated with chrysotile asbestos.

**3.2.4.9 Selection of the final IUR for Chrysotile Asbestos**

Due to the downward biases described above, the largest IUR (0.16 per f/cc) was selected from the four combinations that were essentially equivalent in terms of statistical fit and study quality in Table 3-8. This largest estimate was most likely to cover the total risk of incident cancers.

**Table 3-13. Estimates of Selected Central Risk and IUR for Chrysotile Asbestos**

Lung Cancer Source	Central Unit Risk Lung Cancer	Upper Bound Unit Risk Lung Cancer	Mesothelioma Source	Central Unit Risk Meso	Upper Bound Unit Risk Meso	Combined Central Unit Risk (Lung Cancer + Meso)	Lifetime IUR (per f/cc)
SC Elliott et al. (2012) Exponential	4.09E-2	5.07E-2	NC EPA modeling of Loomis et al. (2019)	5.67E-2	1.17E-1	0.098	0.16

The definition of the IUR is for a lifetime of exposure. For the estimation of lifetime risks for each condition of use, the partial lifetime (or less than lifetime) IUR has been calculated using the lifetable approach and values for different combination of age of first exposure and duration of exposures are presented in Appendix J.

Uncertainties in the cancer risk values are presented in Section 4.3.7.

**3.2.5 Potentially Exposed or Susceptible Subpopulations**

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 factors, 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

5720 exposure, may be at greater risk than the general population of adverse health effects from exposure to a  
5721 chemical substance or mixture, such as infants, children, pregnant women, workers, or the elderly.”

5722 During problem formulation ([U.S. EPA, 2018d](#)), EPA identified potentially exposed and susceptible  
5723 subpopulations for further analysis during the development and refinement of the life cycle, conceptual  
5724 models, exposure scenarios, and analysis plan. In this section, EPA addresses the potentially exposed or  
5725 susceptible subpopulations identified as relevant based on *greater susceptibility*. EPA addresses the  
5726 subpopulations identified as relevant based on *greater exposure* in Section 2.3.3.

5727  
5728 Factors affecting susceptibility examined in the available studies on asbestos include lifestage, gender,  
5729 genetic polymorphisms and lifestyle factors. There is some evidence of genetic predisposition for  
5730 mesothelioma related to having a germline mutation in BAP1 ([Testa et al., 2011](#)). Cigarette smoking in  
5731 an important risk factor for lung cancer in the general population. In addition, lifestage is important  
5732 relative to when the first exposure occurs. The long-term retention of asbestos fibers in the lung and the  
5733 long latency period for the onset of asbestos-related respiratory diseases suggest that individuals  
5734 exposed earlier in life may be at greater risk to the eventual development of respiratory problems than  
5735 those exposed later in life ([ATSDR, 2001a](#)). Appendix J of this RE illustrates this point in the IUR  
5736 values for less than lifetime COUs. For example, the IUR for a one-year old child first exposed to  
5737 chrysotile asbestos for 40 years is 1.31 E-1 while the IUR for a 20-year old first exposed to asbestos for  
5738 40 years is 5.4 E-2.  
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## 5740 **4 RISK CHARACTERIZATION**

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### 5741 **4.1 Environmental Risk**

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5742 EPA made refinements to the conceptual models during the PF that resulted in the elimination of the  
5743 terrestrial exposure, including biosolids, pathways. Thus, environmental hazard data sources on  
5744 terrestrial organisms were determined to be out of scope and excluded from data quality evaluation and  
5745 further consideration in the risk evaluation process.

5746  
5747 In the PF, EPA identified the need to better determine whether there were releases to surface water and  
5748 sediments from the COUs in this risk evaluation and whether risk estimates for aquatic (including  
5749 sediment-dwelling) organisms should be included in the risk evaluation. Thus, reasonably available  
5750 environmental hazard data/information on aquatic toxicity was carried through the systematic review  
5751 process (data evaluation, data extraction and data integration).

5752  
5753 EPA reviewed reasonably available information on environmental hazards posed by chrysotile asbestos.  
5754 A total of four on-topic and in scope environmental hazard studies were identified for chrysotile  
5755 asbestos and were determined to have acceptable data quality with overall high data quality (7Appendix  
5756 E). In addition, the *Systematic Review Supplemental File: Asbestos Data Quality Evaluation of*  
5757 *Environmental Hazard Studies* presents details of the data evaluations for each study, including scores  
5758 for each metric and the overall study score. These laboratory studies indicated reproductive,  
5759 development, and sublethal effects at a concentration range of 10<sup>4</sup>-10<sup>8</sup> fibers/L, which is equivalent to  
5760 0.01 to 100 MFL, to aquatic environmental receptors following chronic exposure to chrysotile asbestos.

5761  
5762 On the exposure side of the equation, Table 2-1 presents asbestos monitoring results from the last two  
5763 six-year Office of Water sampling programs (encompassing 1998 through 2011). Results of the next six-  
5764 year review cycle is anticipated to be completed in 2023. The data show a low number of samples

(approximately 3.5% of over 14,000 samples over a 12-year period) above the reported minimum reporting limit (MRL) of 0.2 MFL. This exposure value is within the range of hazard values reported to have effects on aquatic organisms (0.01 to 100 MFL). EPA believes there is low or no potential for environmental risk to aquatic or sediment-dwelling receptors from the COUs included in this risk evaluation because water releases associated with the COUs are not expected and were not identified.

Also, after the PF was released, EPA was still in the process of identifying potential asbestos water releases for the TSCA COUs. EPA continued to search EPA databases as well as the literature and engaged in a dialogue with industries and reached out for a dialogue to shed light on potential releases to water. The available information indicated that there were surface water releases of asbestos; however, not all releases are subject to reporting (*e.g.*, effluent guidelines) or are applicable (*e.g.*, friability). Based on the reasonably available information in the published literature, provided by industries using asbestos, and reported in EPA databases, there is little to no evidence of releases of asbestos to surface water associated with the COUs that EPA is evaluating in this risk evaluation. Therefore, EPA concludes there is low or no risk to aquatic or sediment-dwelling organisms. In addition, terrestrial pathways, including biosolids, were excluded from analysis at the PF stage.

## 4.2 Human Health Risk

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### 4.2.1 Risk Estimation Approach

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EPA usually estimates extra cancer risks for repeated exposures to a chemical using an equation where Risk = Human Exposure (*e.g.*, LADC) x IUR. Then estimates of extra cancer risks would be interpreted as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to the potential carcinogen (*i.e.*, incremental or extra individual lifetime cancer risk).

However, as discussed in Section 3.2, this assessment is unique with respect to the impact of the timing of exposure relative to the cancer outcome as the time since first exposure plays a dominant role in modeling risk. The most relevant exposures for understanding mesothelioma risk were those that occurred decades prior to the onset of cancer and subsequent cancer mortality. For this reason, EPA has used a less than lifetime exposure calculation.

The general equation for estimating cancer risks for less than lifetime exposure from inhalation of asbestos, from the Office of Land and Emergency Management Framework for Investigating Asbestos-contaminated Superfund Sites ([U.S. EPA, 2008](#)), is:

$$ELCR = EPC \cdot TWF \cdot IUR_{LTL}$$

where:

ELCR = Excess Lifetime Cancer Risk, the risk of developing cancer as a consequence of the site-related exposure

EPC = Exposure Point Concentration, the concentration of asbestos fibers in air (f/cc) for the specific activity being assessed

IUR<sub>LTL</sub> = Less than lifetime Inhalation Unit Risk per f/cc

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[For example: the notation for the less than lifetime IUR could start at age 16 with 40 years duration  $IUR_{(16,40)}$ . Values for different combination of starting age and duration can be found in Table\_Apx K-1 in Appendix K.

TWF = Time Weighting Factor, this factor accounts for less-than-continuous exposure during a one-year exposure<sup>16</sup>, and is given by:

$$TWF = \left[ \frac{\text{Exposure time (hours per day)}}{24 \text{ hours}} \right] \cdot \left[ \frac{\text{Exposure frequency (days per year)}}{365 \text{ days}} \right]$$

The general equation above can be extended for more complex exposure scenarios by computing the time-weighted-average exposure of multiple exposures (*e.g.*, for 30-minute task samples within a full 8-hour shift). Similarly, when multiple exposures may each have different risks, those may be added together (*e.g.*, for episodic exposures during and between DIY brake work).

There are three points to emphasize in the application of the general equation:

1. The EPC must be expressed in the same units as the IUR for chrysotile asbestos. The units of concentration employed in this risk evaluation are f/cc as measured by phase contrast microscopy<sup>17</sup>.
2. The concentration-response functions on which the chrysotile asbestos IUR is based varies as a function of time since first exposure. Consequently, estimates of cancer risk depend not only on exposure concentration, frequency and duration, but also on age at first exposure. Therefore, it is essential to use an IUR value that matches the exposure period of interest (specifically the age of first exposure and the duration of exposure).
3. When exposures of full-shift occupational workers are to be evaluated, the TWF should be adjusted to account for differences in inhalation volumes between workers and non-workers. As noted in Appendix G, EPA assumes workers breath 10 m<sup>3</sup> air during an 8-hour shift and non-workers breath 20 m<sup>3</sup> in 24 hours. The hourly ratio of those breathing volumes is the volumetric adjustment factor for workers ( $V_{(worker)}$ ) [(10/8) / (20/24) = 1.5]. Thus, for workers, the formula,  $ELCR = EPC \cdot TWF \cdot IUR_{LTL}$ , is extended as  $ELCR = EPC \cdot TWF \cdot V \cdot IUR_{LTL}$ .

$$TWF_{(worker)} = (8 \text{ hours} / 24 \text{ hours}) \cdot (240 \text{ days} / 365 \text{ days}) = 0.2192, \text{ and}$$

$$V_{(worker)} = 1.5$$

If the worker began work at age 16 years and worked for 40 years, the appropriate unit risk factor for cancer risk of chrysotile asbestos (taken from Table\_Apx K-1 (Less Than Lifetime (or Partial lifetime) IUR) in Appendix K) would be:

$$IUR_{(16,40)} = 0.0707 \text{ per f/cc}$$

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<sup>16</sup> See U.S. EPA (1994) and Part F update to RAGS inhalation guidance (U.S. EPA, 2009).

<sup>17</sup> PCM-equivalent (PCMe) concentrations measured using TEM could also be used.

Based on these two factors, the excess lifetime cancer risk would be computed as:

$$\text{ELCR} = \text{EPC in f/cc} \cdot 0.2192 \cdot 1.5 \cdot (0.0707 \text{ per f/cc})$$

**BOX 4-1**

**IUR values for other combinations of age at first exposure and duration of exposure can be found in Table\_Apx K-1: Less Than Lifetime (or Partial lifetime) IUR and in Appendix L: Sensitivity Analysis of Exposures for DIY/Bystander Scenarios**

For example:

- First exposure at age 16 with 62 years exposure:  $\text{IUR}_{(16,62)} = 0.0768$  per f/cc
- First exposure at age 16 with 40 years exposure:  $\text{IUR}_{(16,40)} = 0.0707$  per f/cc
- First exposure at age 16 with 20 years exposure:  $\text{IUR}_{(16,20)} = 0.0499$  per f/cc
- First exposure at age 0 with 78 years exposure:  $\text{IUR}_{(0,78)} = 0.16$  per f/cc

The use scenarios and populations of interest for cancer risk estimation for partial lifetime chronic exposures are presented in **Table 4-1**.

EPA provided occupational exposure results representative of *central tendency* conditions and *high-end* conditions. A central tendency was assumed to be representative of occupational exposures in the center of the distribution for a given condition of use. EPA used the 50<sup>th</sup> percentile (median), mean (arithmetic or geometric), mode, or midpoint values of a distribution as representative of the central tendency scenario. EPA's preference was to provide the 50<sup>th</sup> percentile of the distribution. However, if the full distribution was not known, EPA assumed that the mean, mode, or midpoint of the distribution represented the central tendency depending on the statistics available for the distribution. EPA provided high-end results at the 95<sup>th</sup> percentile. If the 95<sup>th</sup> percentile was not available, or if the full distribution was not known and the preferred statistics were not available, EPA estimated a maximum or bounding estimate in lieu of the high-end. Refer to Table 2-24. and Table 2-25 for occupational and consumer exposures.

EPA received occupational monitoring data for some of the uses (chlor-alkali and sheet gaskets) and those data were used to estimate risks. For the other COUs, EPA used monitoring information from the reasonably available information. Risks for both workers and ONUs were estimated when data were reasonably available. Cancer risk was calculated for the central and high-end exposure estimates. Excess cancer risks were expressed as number of cancer cases per 10,000 (or  $1 \times 10^{-4}$ ).

It was assumed that the exposure frequency (i.e., the amount of days per year for workers or occupational non-users exposed to asbestos) was 240 days per year and the occupational exposure started at age 16 years with a duration of 40 years. EPA typically uses a benchmark cancer risk level of  $1 \times 10^{-4}$  for workers/ONUs and  $1 \times 10^{-6}$  for consumers/bystanders for determining the acceptability of the cancer risk in a population. For consumers (DIY and bystanders; see Section 4.2.3.1), the exposure frequency assumed was 62 years, assuming exposure starting at 16 years old and continuing through their lifetime (78 years). Exposure frequency was also based on data from the EPA Exposure Factors Handbook ([U.S. EPA, 2011](#)) for exposure to chrysotile asbestos resulting from the COUs. As noted in Box 4-1, other age/duration assumptions may be made.

Table 4-1. Use Scenarios and Populations of Interest for Cancer Endpoints for Assessing Occupational Risks Following Inhalation Exposures to Chrysotile Asbestos

Populations and Toxicological Approach	Occupational Use Scenarios of Asbestos
<b>Population of Interest and Exposure Scenario:</b> <i>Users</i>	Adult and youth workers (>16 years old) exposed to chrysotile asbestos 8 hours/day for 240 days/year for working 40 years
<b>Population of Interest and Exposure Scenario:</b> <i>Occupational Non-Users (ONUs)</i>	Adults and youths of both sexes (>16 years old) indirectly exposed to chrysotile asbestos while being in the same building during product use.
<b>Health Effects of Concern, Concentration and Time Duration</b>	<p><b>Cancer Health Effects:</b> Lung Cancer/Mesothelioma</p> <p><b>Chrysotile Asbestos Cancer IUR (see Section 3.2.4)</b></p> <ul style="list-style-type: none"> <li>• Lifetime Inhalation Unit Risk per f/cc (from Table 3-13)                             <ul style="list-style-type: none"> <li>○ Mesothelioma or Lung Cancer,</li> <li>○ 0.16 per f/cc</li> </ul> </li> <li>• <u>Less than Lifetime</u> Inhalation Unit Risk per f/cc (IUR<sub>LT</sub>)                             <ul style="list-style-type: none"> <li>○ Uses values from life tables for different combination of starting age of exposure and duration (see Table APX-K-1)</li> </ul> </li> </ul> <p>Uses a Time Weighting Factor, this factor accounts for less-than-continuous exposure during a one-year exposure</p>
<b>Notes:</b> Adult workers (>16 years old) include both healthy female and male workers.	

Table 4-2. Use Scenarios and Populations of Interest for Cancer Endpoints for Assessing Consumer Risks Following Inhalation Exposures to Chrysotile Asbestos

Populations and Toxicological Approach	Use Scenarios of Asbestos
<b>Population of Interest and Exposure Scenario:</b> <i>Users (or Do-It-Yourselfers; DIY)</i>	<b>Consumer Users:</b> Adults and youths of both sexes (>16 years old) exposed to chrysotile asbestos
<b>Population of Interest and Exposure Scenario:</b> <i>Bystanders</i>	Individuals of any age indirectly exposed to chrysotile asbestos while being in the same work area of the garage as the consumer
<b>Health Effects of Concern, Concentration and Time Duration</b>	<p><b>Cancer Health Effects:</b>                      Lung Cancer/Mesothelioma</p> <p><b>Chrysotile Asbestos Cancer IUR (see Section 3.2.4)</b></p> <ul style="list-style-type: none"> <li>• Lifetime Inhalation Unit Risk per f/cc (from Table 3-13)                             <ul style="list-style-type: none"> <li>○ Mesothelioma or Lung Cancer,</li> <li>○ 0.16 per f/cc</li> </ul> </li> <li>• <u>Less than Lifetime</u> Inhalation Unit Risk per f/cc (IUR<sub>LT</sub>)                             <ul style="list-style-type: none"> <li>○ Uses values from life tables for different combination of starting age of exposure and duration (see Table APX-J-1)</li> </ul> </li> </ul> <p>Uses a Time Weighting Factor, this factor accounts for less-than-continuous exposure during a one-year exposure</p>



Populations and Toxicological Approach	Use Scenarios of Asbestos
Re-entrainment <sup>18</sup> of asbestos can occur indoors in a garage. Both users and bystanders can be exposed.	

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**Reported Respirator Use by COU**

EPA evaluated inhalation exposure for workers and consumers using personal monitoring data either from industry or journal articles. Respirators may be used when effective engineering controls are not feasible as per OSHA’s 29 CFR § 1910.134(a). The knowledge of the range of respirator APFs is intended to assist employers in selecting the appropriate type of respirator that could provide a level of protection needed for a specific exposure scenario. EPA received information from industry on certain COUs that specified the types of respirators currently being used. This information is summarized in Table 4-3. The APF EPA suggests be applied for this risk calculation is provided in bold (based on the discussion in Section 2.3.1.2). When no respirator usage was provided or it was deemed inadequate for the COU, EPA provided a hypothetical APF. 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.

**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 <sup>3</sup>  <b>APF to use for the risk calculation: 10 to 25</b>
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 <sup>1</sup>  <b>Hypothetical APF to use for the risk calculation: 10 to 25</b>
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

<sup>18</sup> Settled Asbestos Dust Sampling and Analysis 1st Edition Steve M. Hays, James R. Millette CRC Press 1994

Condition of Use	Monitoring Data?	Respirator Use Text	APF for Risk Calculation
			<b>APF to use for the risk calculation: 10</b>
Oilfield brake blocks	Yes, from the literature	No information is reasonably available on respirator use for this COU.	<b>Hypothetical APF to use for the risk calculation: 10 to 25</b>
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.	<b>Hypothetical APF to use for the risk calculation: 10 to 25</b>
Other gasket vehicle friction product (UTV)	No <sup>2</sup>	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.	<b>Hypothetical APF to use for the risk calculation: 10 to 25</b>

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<sup>1</sup> 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.  
<sup>2</sup> 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>.  
<sup>3</sup> See Table 2-7.

As determined in the problem formulation and again in Section 3.2.2, exposures to asbestos were evaluated for the inhalation route only. Inhalation and dermal exposures are assumed to occur simultaneously for workers and consumers. EPA chose not to employ simple additivity of exposure pathways at this time within a condition of use because of the uncertainties present in the current exposure estimation procedures and this may lead to an underestimate of exposure.

**4.2.2 Risk Estimation for Workers: Cancer Effects Following Less than Lifetime Inhalation Exposures by Conditions of Use**

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  $1 \times 10^{-4}$  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.

5930 For all COUs that were assessed, there were risks to workers without respirators as personal protective  
 5931 equipment (PPE) for both central and high-end exposure estimates; including those scenarios for which  
 5932 short-term exposure concentrations were available to include in the analysis. When PPE were applied  
 5933 (some known, some hypothetical), risks were not exceeded for some COUs (chlor-alkali and oilfield  
 5934 brake blocks) but they were exceeded for others (sheet gasket stamping – central and high-end, short-  
 5935 term exposure estimates; sheet gasket use – high-end exposure estimate; aftermarket auto brakes and  
 5936 other vehicle friction products – high-end and high-end short-term exposure estimates; and other gaskets  
 5937 [UTV] – high-end exposure estimates). Industry submissions indicated no use of respirators (sheet  
 5938 gasket stampers using N95 respirators is not protective based on OSHA regulations), or respirators with  
 5939 an APF of 10 or 25 (chlor-alkali) and an APF of 10 (gasket use). It is important to note that based on  
 5940 published evidence for asbestos (see Section 2.3.1.2), nominal APF may not be achieved for all  
 5941 respirator users.

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 5943 ONUs were not assumed to use PPE and results show some COUs with cancer risk exceedances for both  
 5944 central and high-end exposure estimates (sheet gasket use and other gasket s [UTV]). For all other  
 5945 COUs, at least one of the ONU scenarios exceeded the cancer risk benchmark. Thus, exceedances were  
 5946 observed for ONUs in every COU.  
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5948 **4.2.2.1 Risk Estimation for Cancer Effects Following Chronic Inhalation Exposures**  
 5949 **for Chlor-alkali Industry**

5950 Exposure data from the chlor-alkali industry were presented for two sampling durations (full shift and  
 5951 short-term) in Table 4-4. and Table 4-5., respectively (taken from Table 2-8). Short term samples were  
 5952 assumed to be approximately 30 minutes in duration. Data on exposure at central tendency (median) and  
 5953 the high-end (95<sup>th</sup> percentile) are presented along with the Excess Lifetime Cancer Risk (ELCR) for  
 5954 each exposure distribution.  
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5956 **Table 4-4. Excess Lifetime Cancer Risk for Chlor-alkali Industry Full Shift Workers and ONUs**  
 5957 **(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 <sup>19</sup>		Asbestos Worker		ONU	
	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end
Producing, handling, and disposing of asbestos diaphragms: <b>Full shift exposure</b>	0.005	0.036	< 0.0025	≤0.008	1.2 E-4	8.4 E-4	5.8 E-5	1.9 E-4

5958 Asbestos Workers: ELCR (Central Tendency) = 0.005 f/cc • 0.2192 • 1.5 • 0.0707 per f/cc

5959 Asbestos Workers: ELCR (High-end) = 0.036 f/cc • 0.2192 • 1.5 • 0.0707 per f/cc

5960 ONU: ELCR (Central Tendency) = 0.0025 f/cc • 0.2192 • 1.5 • 0.0707 per f/cc

5961 ONU: ELCR (High-end) = 0.008 f/cc • 0.2192 • 1.5 • 0.0707 per f/cc

<sup>19</sup> Excel file “Chlor-Alkali – Summary of Area Sampling Data (7-5-2019).xlsx list 15 area samples from Olin. Eleven area samples from one facility all have exposure concentrations of exactly 0.004 f/cc with no mention of detection limit; four area samples from another facility have exposure concentration of exactly 0.008 f/cc and these four samples are labeled ‘Detection limit was 0.008f/cc.’” For the purposes of estimating risks, the sampling values of 0.004 f/cc are used as the measure of central tendency of ONU exposure and the values of 0.008 f/cc at the detection limit are used to represent the high-end of ONU exposure.

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Table 4-4. presents the inhalation cancer risk estimates for chlor-alkali workers and ONUs exposed to asbestos. The exposure values in Table 4-4. were based on monitoring data from 3 chlor-alkali companies. For asbestos workers, the benchmark cancer risk estimate of  $1 \times 10^{-4}$  was exceeded for both high-end and central tendency exposure estimates. For ONUs, the cancer benchmark was exceeded for the high-end exposure value. Estimates exceeding the benchmark are bolded and shaded in pink.

OSHA Standard Number 1910.1001(c)(2) for asbestos describes the 30-minute excursion limit. “The employer shall ensure that no employee is exposed to an airborne concentration of asbestos in excess of 1.0 fiber per cubic centimeter of air (1 f/cc) as averaged over a sampling period of thirty (30) minutes as determined by the method prescribed in Appendix A to this section, or by an equivalent method.” Table 2-4 reports 30-minute short-term personal exposures. As these exposures may not represent chronic exposures, risk estimates were not calculated based on these sample values in isolation. However, workers exposed to these short-term exposure concentrations are likely to be exposed to chrysotile asbestos at other times during their full-shift period. As these short-term exposure concentrations exceed the full shift exposure concentrations, averaging the 30-minutes values into a full 8-hour shift would result in an increased 8-hour TWA exposure concentration with increased risks. Table 4-5 uses 30 minutes as the short-term exposure concentration averaged with 7.5 hours at the full shift exposure concentration. The 30-minute values are provided for asbestos workers at the central tendency and at the high-end, but risks are not calculated just for them. The revised 8-hour TWA for a full shift containing one 30-minute exposure value per day is provided along with the risk associated with that revised full-shift exposure value.

There are no short-term values for ONUs, presumably because the short-term sampling is specifically limited to asbestos workers.

**Table 4-5. Excess Lifetime Cancer Risk for Chlor-alkali Industry Workers (Short-Term Personal Samples from Table 2-4, 8-hour full shift) 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
Producing, handling, and disposing of asbestos diaphragms: <b>Short-term exposures</b> (exactly 30-minutes); and 30-minute short term samples within a full shift)*.	30 min value: 0.026	0.35	N/A	N/A	---	---	---	---
	8-hr TWA: 0.0063*	0.056**	N/A	N/A	<b>1.5 E-4</b>	<b>1.3 E-3</b>	---	---

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\* This 8-hour TWA includes the 30-minute short-term exposure within an 8-hour full shift and is calculated as follows:

$$\{[(0.5 \text{ hour}) \cdot (0.026 \text{ f/cc}) + (7.5 \text{ hours}) \cdot (0.005 \text{ f/cc from Table 4-2})] / 8 \text{ hours}\} = 0.0063 \text{ f/cc}$$

\*\* This 8-hour TWA includes the 30-minute short-term exposure within an 8-hour full shift and is calculated as follows:

$$\{[(0.5 \text{ hour}) \cdot (0.35 \text{ f/cc}) + (7.5 \text{ hours}) \cdot (0.036 \text{ f/cc from Table 4-2})] / 8 \text{ hours}\} = 0.056 \text{ f/cc.}$$

$$\text{ELCR}_{(\text{Central Tendency})} = \{[(0.5 \text{ hour}) \cdot \text{EPC}_{(30 \text{ minute})} + (7.5 \text{ hours}) \cdot \text{EPC}_{(\text{Full Shift})}] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0707.$$

$$\text{ELCR}_{(\text{High-end})} = \{[(0.5 \text{ hour}) \cdot \text{EPC}_{(30 \text{ minute})} + (7.5 \text{ hours}) \cdot \text{EPC}_{(\text{Full Shift})}] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0707.$$

$$\text{ELCR}_{(\text{Central Tendency})} = \{[(0.5 \text{ hour}) \cdot 0.026 + (7.5 \text{ hours}) \cdot 0.005] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0707.$$

$$\text{ELCR}_{(\text{High-end})} = \{[(0.5 \text{ hour}) \cdot 0.35 + (7.5 \text{ hours}) \cdot 0.036] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0707.$$

The results in Table 4-5 show that when a 30-minute high exposure short-term exposure concentration is included as part of a full shift exposure estimation, the result is that workers are likely exposed at

higher concentrations than other full-shift workers who are not exposed to short-term exposures monitored for OSHA compliance, thereby posing an even higher excess lifetime cancer risk. Note that this will be true regardless of the frequency at which they may be exposed to those 30-minute short-term sample values within the 8-hour TWA, as the inclusion of high 30-minute exposures will always be higher than the standard full-shift TWA.

**Applying APFs to Data from Both Full Shift Work and Short-Term Work**

ELCRs for chlor-alkali workers that assumes that they will be wearing PPE with APFs of 10 and 25 for 8-hour TWAs and various combinations of 30 minutes and 7.5 hour exposures are presented in Table 4-6, Table 4-7, Table 4-8, Table 4-9 and Table 4-10.

**Table 4-6. Excess Lifetime Cancer Risk for Chlor-alkali Industry Full Shift Workers and ONUs (from Table 4-4) after consideration of PPE with APF=10 for all workers (excluding ONUs)**

Occupational Exposure Scenario	Asbestos Worker	
	Central Tendency	High-end
	Producing, handling, and disposing of asbestos diaphragms: <b>Full shift exposure</b>	1.2 E-5

**Table 4-7. Excess Lifetime Cancer Risk for Chlor-alkali Industry Full Shift Workers and ONUs (from Table 4-4) after consideration of PPE with APF=25 for all workers (excluding ONUs)**

Occupational Exposure Scenario	ELCR (40 yr exposure starting at age 16 years)	
	Asbestos Worker	
	Central Tendency	High-end
Producing, handling, and disposing of asbestos diaphragms: <b>Full shift exposure</b>	4.8 E-6	3.4 E-5

Table 4-6 and Table 4-7 show the risk estimates when an APF of 10 or 25 is applied to all full shift worker exposures. In both scenarios, the risk estimates for the workers are below the benchmark of  $10^{-4}$  ( $1 \text{ E-}4$ ). 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  $1 \times 10^{-4}$  was exceeded for ONUs for high-end exposures). Table 4-3. indicated the respirators that ACC reported to EPA are currently used by chlor-alkali workers and both APF of 10 and 25 are used depending on the activity being performed. It is not clear whether the workers monitored for either short-term or full shift exposures were wearing respirators at the time of the collection of air samples.

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**Table 4-8. Excess Lifetime Cancer Risk for Chlor-alkali Industry Short-Term Personal Samples (from Table 4-5) after consideration of PPE with APF=25 for short-term workers for 0.5 hours (excluding ONUs)**

Occupational Exposure Scenario	ELCR (40 yr exposure starting at age 16 years)	
	Asbestos Worker	
	Central Tendency	High-end
Producing, handling, and disposing of asbestos diaphragms: <b>Short-term exposures</b> (exactly 30-minutes); and 30-minute short term samples within a full shift)	1.1 E-4	8.1 E-4

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The central risks for 7.5 hours at 0.005 f/cc with no APF were calculated and added to the 0.5 hour risk at 0.026 f/cc and APF=25 and then the sum divided by 8 hours. The high-end risks for 7.5 hours at 0.005 f/cc were calculated and added to the 0.5 hour risk at 0.35 f/cc and APF=25 and then sum divided by 8 hours.

Central: Risk for 7.5 hours =  $0.005 \text{ f/cc} \cdot 0.2192 \cdot 1.5 \cdot 0.0707 = 1.2 \text{ E-4}$   
 Risk for 0.5 hours =  $0.026 \text{ f/cc} \cdot 0.2192 \cdot 1.5 \cdot 0.0707 / (\text{APF of } 25) = 2.4 \text{ E-5}$   
 Risk for 8 hours =  $[7.5 \cdot 1.2 \text{ E-4} + 0.5 \cdot 2.4 \text{ E-5}] / 8 = 1.1 \text{ E-4}$

High-end: Risk for 7.5 hours =  $0.036 \text{ f/cc} \cdot 0.2192 \cdot 1.5 \cdot 0.0707 = 8.4 \text{ E-4}$   
 Risk for 0.5 hours =  $0.35 \text{ f/cc} \cdot 0.2192 \cdot 1.5 \cdot 0.0707 / (\text{APF of } 25) = 3.3 \text{ E-4}$   
 Risk for 8 hours =  $[7.5 \cdot 8.4 \text{ E-4} + 0.5 \cdot 3.3 \text{ E-4}] / 8 = 8.1 \text{ E-4}$

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**Table 4-9. Excess Lifetime Cancer Risk for Chlor-alkali Industry Short-Term Personal Samples (from Table 4-5) after consideration of PPE and with APF=10 for full-shift workers and with APF=25 for short-term workers (excluding ONUs)**

Occupational Exposure Scenario	ELCR (40 yr exposure starting at age 16 years)	
	Asbestos Worker	
	Central Tendency	High-end
Producing, handling, and disposing of asbestos diaphragms: <b>Short-term exposures</b> (exactly 30-minutes); and 30-minute short term samples within a full shift).	1.3 E-5	9.9 E-5

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The central risks for 7.5 hours at 0.005 f/cc and APF=10 were calculated and added to the 0.5 risk at 0.026 f/cc and APF=25 and then sum divided by 8 hours. The high-end risks for 7.5 hours at 0.005 f/cc and APF=10 were calculated and added to the 0.5 risk at 0.026 f/cc and APF=25 and then sum divided by 8 hours.

Central : Risk for 7.5 hours =  $0.005 \text{ f/cc} \cdot 0.2192 \cdot 1.5 \cdot 0.0707 / (\text{APF of } 10) = 1.2 \text{ E-5}$   
 Risk for 0.5 hours =  $0.026 \text{ f/cc} \cdot 0.2192 \cdot 1.5 \cdot 0.0707 / (\text{APF of } 25) = 2.4 \text{ E-5}$   
 Risk for 8 hours =  $[7.5 \cdot 1.2 \text{ E-5} + 0.5 \cdot 2.4 \text{ E-5}] / 8 = 1.3 \text{ E-5}$

High-end: Risk for 7.5 hours =  $0.036 \text{ f/cc} \cdot 0.2192 \cdot 1.5 \cdot 0.0707 / (\text{APF of } 10) = 8.4 \text{ E-5}$   
 Risk for 0.5 hours =  $0.35 \text{ f/cc} \cdot 0.2192 \cdot 1.5 \cdot 0.0707 / (\text{APF of } 25) = 3.3 \text{ E-4}$   
 Risk for 8 hours =  $[7.5 \cdot 8.4 \text{ E-5} + 0.5 \cdot 3.3 \text{ E-4}] / 8 = 9.9 \text{ E-5}$

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**Table 4-10. Excess Lifetime Cancer Risk for Chlor-alkali Industry Short-Term Personal Samples (from Table 4-5) after consideration of PPE and with APF=25 for full-shift workers and with APF=25 for short-term workers (excluding ONUs)**

Occupational Exposure Scenario	ELCR (40 yr exposure starting at age 16 years)	
	Asbestos Worker	
	Central Tendency	High-end
Producing, handling, and disposing of asbestos diaphragms: <b>Short-term exposures</b> (exactly 30-minutes); and 30-minute short term samples within a full shift).	6.0 E-6	5.2 E-5

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Here the method is simply to divide the risks in Table 4-5 by 25:  
 Central Risk from Table 4-5 = 1.5E-4/25 = 6.0E-6  
 High Risk from Table 4-5 = 1.3E-3/25 = 5.2E-5

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Table 4-8, Table 4-9, and Table 4-10 present the ELCR for short-term exposures for chlor-alkali workers. The three scenarios represented are: (1) APF of 25 for short-term (30-minute exposure) and no APF for 7.5 hours; (2) APF of 25 for short-term exposures and APF of 10 for the remaining 7.5 hours; and (3) APF of 25 for both short-term and remaining 7.5 hours. The central tendency and high-end risk estimates exceeded the benchmark for workers in only the first of the three scenarios presented. None of the other combinations of APFs exceeded the benchmark. Note that APFs do not apply to ONU scenarios.

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

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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.

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**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

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

6083

6084 Table 4-11. presents the inhalation cancer risk estimates for workers stamping asbestos-containing sheet  
 6085 gaskets and for ONUs exposed to asbestos. For asbestos workers, the benchmark cancer risk estimate of  
 6086  $1 \times 10^{-4}$  was exceeded for both central tendency and high-end exposure estimates. For ONUs, the cancer  
 6087 benchmark was exceeded for the high-end exposure values. Estimates exceeding the benchmark are  
 6088 shaded in pink and bolded.

6089

6090 Table 4-12 presents the inhalation cancer risk estimates for workers stamping sheet gaskets and for  
 6091 ONUs exposed to asbestos, using an averaging of short-term exposures (assuming 30 minutes) and full  
 6092 shift exposures (7.5 hours per day of the full shift TWA exposure) based on monitoring data. The central  
 6093 tendency short-term exposure value for workers (0.024 fibers/cc) is the arithmetic mean of ten short-  
 6094 term measurements reported in a study of one worker at a company that stamps sheet gaskets containing  
 6095 asbestos. The high-end short-term exposure value for workers (0.059 fibers/cc) is the highest measured  
 6096 short-term exposure value from the available monitoring data. This exposure value occurred during a  
 6097 30-minute sample.

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6099 **Table 4-12. Excess Lifetime Cancer Risk for Sheet Gasket Stamping Short-term Exposures within**  
 6100 **an 8-hour Full Shift (from Table 2-10, Personal Samples) before consideration of PPE and any**  
 6101 **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: <b>Short-term exposures</b> (~30- minute; and ~30-minute short term samples within a full shift)*.	30 min value: 0.024	0.059	0.0042	0.010	---	---	---	---
	8-hr TWA: 0.015*	0.059*	0.0025*	0.010*	<b>3.5 E-4</b>	<b>1.4 E-3</b>	5.6 E-5	<b>2.3 E-4</b>

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\*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.

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

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$$\text{Asbestos Worker: } ELCR_{(Central Tendency)} = \{[(0.5 \text{ hour}) \cdot 0.024 + (7.5 \text{ hours}) \cdot 0.014] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0707.$$

6105

$$\text{Asbestos Worker: } ELCR_{(High-end)} = \{[(0.5 \text{ hour}) \cdot 0.059 + (7.5 \text{ hours}) \cdot 0.059] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0707.$$

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6109 For asbestos workers, the benchmark cancer risk estimate of  $1 \times 10^{-4}$  was exceeded for both central  
 6110 tendency and high-end exposure estimates. For ONUs, the cancer benchmark was exceeded for the high-  
 6111 end exposure values. Estimates exceeding the benchmark are shaded in pink and bolded.

6112 **Applying APFs to Data from Both Full Shift Work and Short-Term Work**

6113 ELCRs for workers who stamp sheet gaskets using PPE with hypothetical APFs of 10 and 25 applied for  
 6114 8-hour TWAs and various combinations of 30 minutes and 7.5 hour exposures are presented in Table  
 6115 4-13, Table 4-14., Table 4-15, and Table 4-16.

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**Table 4-13. Excess Lifetime Cancer Risk for Sheet Gasket Stamping Full Shift Workers and ONUs (from Table 4-11) after consideration of PPE using an APF=10 (excluding ONUs)**

Occupational Exposure Scenario	ELCR (40 yr exposure starting at age 16 years)	
	Asbestos Worker	
	Central Tendency	High-end
Sheet gasket stamping: 8-hr TWA exposure	3.3 E-5	1.4 E-4

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**Table 4-14. Excess Lifetime Cancer Risk for Sheet Gasket Stamping Full Shift Workers and ONUs (from Table 4-11) after consideration of PPE using an APF=25 (excluding ONUs)**

Occupational Exposure Scenario	ELCR (40 yr exposure starting at age 16 years)	
	Asbestos Worker	
	Central Tendency	High-end
Sheet gasket stamping: 8-hr TWA exposure	1.3 E-5	5.6 E-5

6123

6124 For full shift worker scenarios, the benchmark cancer risk estimate of  $1 \times 10^{-4}$  was exceeded for workers  
6125 with high-end exposures when a hypothetical APF of 10 was applied; all other worker scenarios were  
6126 below the benchmark (central tendency for hypothetical APFs of 10 and 25 and high-end exposures with  
6127 an APF of 25. Since the assumption is that ONUs do not wear respirators, application of APFs do not  
6128 apply and so their risk estimates do not change (i.e., the benchmark cancer risk estimate of  $1 \times 10^{-4}$  was  
6129 exceeded for ONUs for high-end exposures).  
6130

6131 **Table 4-15. Excess Lifetime Cancer Risk for Sheet Gasket Stamping Short-term Exposures within**  
6132 **an 8-hour Full Shift (from Table 4-12) after consideration of PPE using an APF=10 for both full-**  
6133 **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

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**Table 4-16. 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=25 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: <b>Short-term exposures</b>	1.4 E-5	5.6 E-5

6143

6144 Tables 4-15 and 4-16 present the ELCR for short-term exposures for sheet gasket stamping workers. The  
6145 two scenarios represented are (all hypothetical applications of an APF): (1) APF of 10 for short-term  
6146 (30-minute exposure) and an APF of 10 for 7.5 hours; and (2) APF of 25 for both short-term and  
6147 remaining 7.5 hours. The central tendency and high-end risk estimates exceeded the benchmark for  
6148 workers in only the first of scenario presented. None of the other combinations of hypothetical APFs  
6149 exceeded the benchmark. And again, APFs do not apply to ONU scenarios.

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**4.2.2.3 Risk Estimation for Cancer Effects Following Chronic Inhalation Exposures for Sheet Gasket Use in Chemical Production**

6152 Exposure data from sheet gasket use (replacing gaskets) – using titanium dioxide production as an  
6153 example - were presented for 8-hour full shift exposures in Table 2-11. These data are based on reports  
6154 from ACC for gasket removal/replacement at titanium dioxide facilities. The 8-hour TWA exposures  
6155 assume that the workers removed gaskets throughout the day during maintenance. Data on the exposure  
6156 at the central and high-end estimates are presented along with the ELCR for each exposure distribution  
6157 in Table 4-6. The high-end value for 8-hr TWA worker exposure (0.094) is based on the highest  
6158 exposure measurement (see Section 2.3.1.4.5). No data are available for evaluating worker short-term  
6159 exposures for this COU (see 2.3.1.4.5).  
6160

6161 **Table 4-17. Excess Lifetime Cancer Risk for Sheet Gasket Use in Chemical Production (using data**  
6162 **from titanium dioxide production), 8-hour TWA (from Table 2-11., Personal Samples) before**  
6163 **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 use: <b>8-hr TWA exposure</b>	0.026	0.094	0.005	0.016	6.0 E-4	2.2 E-3	1.2 E-4	3.7 E-4

6164 Asbestos Workers:  $ELCR_{(Central\ Tendency)} = 0.026\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0707\ per\ f/cc$   
6165 Asbestos Workers:  $ELCR_{(High-end)} = 0.094\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0707\ per\ f/cc$   
6166 ONU:  $ELCR_{(Central\ Tendency)} = 0.005\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0707\ per\ f/cc$   
6167 ONU:  $ELCR_{(High-end)} = 0.016\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0707\ per\ f/cc$   
6168

6169 Table 4-17. presents the inhalation cancer risk estimates based on data for workers replacing sheet  
6170 gaskets in titanium dioxide production and for ONUs exposed to asbestos. For asbestos workers, the  
6171 benchmark cancer risk estimate of  $1 \times 10^{-4}$  was exceeded for both central tendency and high-end exposure

estimates. For ONUs, the cancer benchmark was also exceeded for both the central tendency and the high-end exposure values. Estimates exceeding the benchmark are shaded in pink and bolded.

**Applying APFs**

ELCRs for workers who repair/replace sheet gaskets and ONUs exposed to asbestos using PPE with hypothetical APFs of 10 and 25 applied for 8-hour TWAs are presented in Table 4-18. and Table 4-19. Based on data received from ACC, the current APF used for these activities is 10.

**Table 4-18. Excess Lifetime Cancer Risk for Sheet Gasket Use in Chemical Production, 8-hour TWA (from Table 4-6) after consideration of PPE using the APF=10 reflecting the current use of respirators (excluding ONUs)**

Occupational Exposure Scenario	ELCR (40 yr exposure starting at age 16 years)	
	Asbestos Worker	
	Central Tendency	High-end
Sheet gasket use: <b>8-hr TWA exposure</b>	6.0 E-5	<b>2.2 E-4</b>

**Table 4-19. Excess Lifetime Cancer Risk for Sheet Gasket Use in Chemical Production, 8-hour TWA (from Table 4-6) after consideration of PPE using an APF=25 (excluding ONUs)**

Occupational Exposure Scenario	ELCR (40 yr exposure starting at age 16 years)	
	Asbestos Worker	
	Central Tendency	High-end
Sheet gasket use: <b>8-hr TWA exposure</b>	2.4 E-5	8.8 E-5

In both scenarios, the risk estimates for the workers are below the benchmark of  $1 \times 10^{-4}$  for the central tendency risk estimate and it exceeds the benchmark when a hypothetical APF of 10 is used for the high-end scenario; but not when the APF of 25 is applied to the high-end scenario. As shown in Table 4-3., ACC reported that titanium dioxide sheet gasket workers use respirators with an APF of 10. Since the assumption is that ONUs do not wear respirators, application of APFs do not apply and so their risk estimates do not change. Estimates exceeding the benchmark are shaded in pink and bolded.

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

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. In an effort to provide a risk estimate for this activity, estimated exposures from Table 2-13 were used to represent the central tendencies of exposures for workers and ONUs; there is no estimate for high-end exposures. More information on the limitations of these data is provided in Section 2.3.1.5.3.

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**Table 4-20. Excess Lifetime Cancer Risk for Oil Field Brake Block Use, 8-hour TWA (from Table 2-13 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
Brake Block use: 8-hr TWA exposure	0.03	---	0.02	---	<b>7.0 E-4</b>	---	<b>4.6 E-4</b>	---

6207 Asbestos Workers:  $ELCR_{(Central\ Tendency)} = 0.03\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0707$  per f/cc  
 6208 ONU:  $ELCR_{(Central\ Tendency)} = 0.02\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0707$  per f/cc

6209

6210 Table 4-20. presents the inhalation cancer risk estimates for workers around brake block use and for  
 6211 ONUs exposed to asbestos. For workers and ONUs, the benchmark cancer risk estimate of  $1 \times 10^{-4}$  was  
 6212 exceeded for central tendency. No high-end exposures were available for this activity. Estimates  
 6213 exceeding the benchmark are shaded in pink and bolded.

6214

**Applying APFs**

6215 ELCRs for workers who work near oil field brake blocks exposed to asbestos using PPE with  
 6216 hypothetical APFs of 10 and 25 applied for 8-hour TWAs are presented in Table 4-21. and Table 4-22..

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**Table 4-21. Excess Lifetime Cancer Risk for Oil Field Brake Block Use, 8-hour TWA (from Table 4-20) after consideration of PPE using an APF=10 (excluding ONUs)**

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Occupational Exposure Scenario	ELCR (40 yr exposure starting at age 16 years)	
	Asbestos Worker	
	Central Tendency	High-end
Brake Block use: 8-hr TWA exposure	7.0 E-5	---

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**Table 4-22. Excess Lifetime Cancer Risk for Oil Field Brake Block Use, 8-hour TWA (from Table 4-20) after consideration of PPE using an APF=25 (excluding ONUs)**

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Occupational Exposure Scenario	ELCR (40 yr exposure starting at age 16 years)	
	Asbestos Worker	
	Central Tendency	High-end
Brake Block use: 8-hr TWA exposure	2.8 E-5	---

6224

6225 In both scenarios, the risk estimates for the workers using either the hypothetical APF of 10 or 25 are  
 6226 below the benchmark of  $1 \times 10^{-4}$ . Since the assumption is that ONUs do not wear respirators, application  
 6227 of APFs do not apply and so their risk estimates do not change.

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

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.

**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	<b>1.4 E-4</b>	<b>2.2 E-3</b>	1.6 E-5	<b>2.6 E-4</b>

Asbestos Workers:  $ELCR_{(Central\ Tendency)} = 0.006\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0707\ per\ f/cc$

Asbestos Workers:  $ELCR_{(High-end)} = 0.094\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0707\ per\ f/cc$

ONU:  $ELCR_{(Central\ Tendency)} = 0.0007\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0707\ per\ f/cc$

ONU:  $ELCR_{(High-end)} = 0.011\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0707\ per\ f/cc$

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  $1 \times 10^{-4}$  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.

Table 4-24. presents the inhalation cancer risk estimates for workers repairing or replacing aftermarket auto brakes and clutches and for ONUs exposed to asbestos, using an averaging of short-term exposures (assuming 30 minutes per day) and full shift exposures (7.5 hours per day of the full shift TWA exposure) based on 7 studies located in the literature. For asbestos workers, the benchmark cancer risk estimate of  $1 \times 10^{-4}$  was exceeded for both central tendency and high-end exposure estimates. For ONUs, the cancer benchmark was exceeded for the high-end exposure values. Estimates exceeding the benchmark are shaded in pink and bolded.

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**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	0.836	0.0007	0.100	---	---	---	---
	8-hr TWA: 0.006*	0.140*	0.0007*	0.011*	1.4 E-4	3.3 E-3	1.6 E-5	2.6 E-4

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\*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.  
 $ELCR = \{[(0.5 \text{ hour}) * EPC_{(30 \text{ minute})} + (7.5 \text{ hours}) * EPC_{(Full Shift)}] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0707$ .  
 Asbestos Worker:  $ELCR_{(Central Tendency)} = \{[(0.5 \text{ hour}) * EPC_{(30 \text{ minute})} + (7.5 \text{ hours}) * EPC_{(Full Shift)}] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0707$ .  
 Asbestos Worker:  $ELCR_{(High-end)} = \{[(0.5 \text{ hour}) * EPC_{(30 \text{ minute})} + (7.5 \text{ hours}) * EPC_{(Full Shift)}] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0707$ .  
 Asbestos Worker:  $ELCR_{(Central Tendency)} = \{[(0.5 \text{ hour}) * 0.006 + (7.5 \text{ hours}) * 0.006] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0707$ .  
 Asbestos Worker:  $ELCR_{(High-end)} = \{[(0.5 \text{ hour}) * 0.836 + (7.5 \text{ hours}) * 0.094] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0707$ .  
 ONU:  $ELCR_{(Central Tendency)} = \{[(0.5 \text{ hour}) * 0.0007 + (7.5 \text{ hours}) * 0.0007] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0707$ .  
 ONU:  $ELCR_{(High-end)} = \{[(0.5 \text{ hour}) * 0.1 + (7.5 \text{ hours}) * 0.011] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0707$ .

**Applying APFs to Data from Both Full Shift Work and Short-Term Work**

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ELCRs for workers who repair/replace auto brakes and clutches exposed to asbestos using PPE with hypothetical APFs of 10 and 25 applied for 8-hour TWAs and various combinations of 30 minutes and 7.5 hour exposures are presented in: Table 4-26., Table 4-27., Table 4-27 and Table 4-28.

**Table 4-25. Excess Lifetime Cancer Risk for Repairing or Replacing Aftermarket Auto Brakes and Clutches in an Occupational Setting, 8-hour TWA Exposure (from Table 4-23) after consideration of PPE with APF=10 (excluding ONUs)**

Occupational Exposure Scenario	ELCR (40 yr exposure starting at age 16 years)	
	Asbestos Worker	
	Central Tendency	High-end
Repairing or replacing brakes with asbestos-containing aftermarket automotive parts: 8-hour TWA exposure	1.4 E-5	2.2 E-4

**Table 4-26. Excess Lifetime Cancer Risk for Repairing or Replacing Aftermarket Auto Brakes and Clutches in an Occupational Setting, 8-hour TWA Exposure (from Table 4-24.) after consideration of PPE with APF=25 (excluding ONUs)**

Occupational Exposure Scenario	ELCR (40 yr exposure starting at age 16 years)	
	Asbestos Worker	
	Central Tendency	High-end
Repairing or replacing brakes with asbestos-containing aftermarket automotive parts: 8-hour TWA exposure	5.6 E-6	8.8 E-5

For asbestos workers wearing a hypothetical respirator at APF 10, the benchmark cancer risk estimate of  $1 \times 10^{-4}$  was exceeded for high-end exposure estimates; all other scenarios (hypothetical APF of 10 for central tendency and hypothetical APF of 25 for both central and high-end exposures) had risk estimates below the benchmark. Since the assumption is that ONUs do not wear respirators, application of APFs do not apply and so their risk estimates do not change. Estimates exceeding the benchmark are shaded in pink and bolded.

**Table 4-27. 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 4-24) after consideration of PPE with APF=10 (excluding ONUs)**

Occupational Exposure Scenario	ELCR (40 yr exposure starting at age 16 years)	
	Asbestos Worker	
	Central Tendency	High-end
Repairing or replacing brakes with asbestos-containing aftermarket automotive parts: short-term exposure	1.4 E-5	<b>3.3 E-4</b>

**Table 4-28. 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 4-24) after consideration of PPE with APF=25 (excluding ONUs)**

Occupational Exposure Scenario	ELCR (40 yr exposure starting at age 16 years)	
	Asbestos Worker	
	Central Tendency	High-end
Repairing or replacing brakes with asbestos-containing aftermarket automotive parts: short-term exposure	5.6 E-6	<b>1.3 E-4</b>

6307 Table 4-27. and Table 4-28. display the ELCRs for short-term exposures for workers repairing or  
 6308 replacing auto brakes and using hypothetical APFs of 10 and 25. For asbestos workers exposed to  
 6309 asbestos, the benchmark cancer risk estimate of  $1 \times 10^{-4}$  was exceeded for high-end exposures, but not  
 6310 central tendency exposures, after consideration of both hypothetical APF 10 and APF 25. Estimates  
 6311 exceeding the benchmark are shaded in pink and bolded. And again, APFs do not apply to ONU  
 6312 scenarios.

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

6315 As discussed in Section 2.3.1.8, EPA is using the exposure estimates for aftermarket auto brakes and  
 6316 clutches for the other vehicle friction products COU. Therefore, the risk estimates will mimic those for  
 6317 the aftermarket auto brakes scenarios. Exposure data from aftermarket auto brakes and clutches were  
 6318 presented for two sampling durations (8-hour TWA and short-term) in Table 2-15. The exposure levels  
 6319 are based on an 8-hour TWA from Table 2-15., which are based on 7 studies found in the literature.  
 6320 ELCRs for short-term data from Table 2-15. are also presented.

6322 In addition, as noted in Section 2.3.1.8, there is a limited use of asbestos-containing brakes for a special,  
 6323 large transport plane (the “Super-Guppy”) by the National Aeronautics and Space Administration  
 6324 (NASA) that EPA has recently learned about. In this public draft risk evaluation, EPA is providing  
 6325 preliminary information for public input and the information is provided in a brief format.

**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	<b>1.4 E-4</b>	<b>2.2 E-3</b>	1.6 E-5	<b>2.6 E-4</b>

6330 Asbestos Workers:  $ELCR_{(Central\ Tendency)} = 0.006\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0707\ per\ f/cc$

6331 Asbestos Workers:  $ELCR_{(High-end)} = 0.094\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0707\ per\ f/cc$

6332 ONU:  $ELCR_{(Central\ Tendency)} = 0.0007\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0707\ per\ f/cc$

6333 ONU:  $ELCR_{(High-end)} = 0.011\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0707\ per\ f/cc$

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6335 Table 4-23. presents the inhalation cancer risk estimates for workers repairing and replacing auto brakes  
 6336 and clutches and for ONUs exposed to asbestos. For workers, the benchmark cancer risk estimate of  
 6337  $1 \times 10^{-4}$  was exceeded for central tendency and high-end. For ONUs, the cancer benchmark was exceeded  
 6338 for the high-end only. Estimates exceeding the benchmark are shaded in pink and bolded.

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**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	0.836	0.0007	0.100	---	---	---	---
	8-hr TWA: 0.006*	0.140*	0.0007*	0.011*	<b>1.4 E-4</b>	<b>3.3 E-3</b>	1.6 E-5	<b>2.6 E-4</b>

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\*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.  $ELCR = \{[(0.5 \text{ hour}) * EPC_{(30 \text{ minute})} + (7.5 \text{ hours}) * EPC_{(Full Shift)}] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0673$ .

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

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

Asbestos Worker:  $ELCR_{(Central Tendency)} = \{[(0.5 \text{ hour}) * 0.006 + (7.5 \text{ hours}) * 0.006] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0707$ .

Asbestos Worker:  $ELCR_{(High-end)} = \{[(0.5 \text{ hour}) * 0.836 + (7.5 \text{ hours}) * 0.094] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0707$

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Table 4-24. presents the inhalation cancer risk estimates for workers repairing or replacing aftermarket auto brakes and clutches and for ONUs exposed to asbestos, using an averaging of short-term exposures (assuming 30 minutes per day) and full shift exposures (7.5 hours per day of the full shift TWA exposure) based on 7 studies located in the literature. For asbestos workers, the benchmark cancer risk estimate of  $1 \times 10^{-4}$  was exceeded for both central tendency and high-end exposure estimates. For ONUs, the cancer benchmark was exceeded for the high-end exposure values. Estimates exceeding the benchmark are shaded in pink and bolded.

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**Applying APFs to Data from Both Full Shift Work and Short-Term Work**

ELCRs for workers who repair/replace auto brakes and clutches exposed to asbestos using PPE with hypothetical APFs of 10 and 25 applied for 8-hour TWAs and various combinations of 30 minutes and 7.5 hour exposures are presented in Table 4-26., Table 4-27. Table 4-33 and Table 4-28.

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**Table 4-31. Excess Lifetime Cancer Risk for Installing Brakes and Clutches in Exported Cars in an Occupational Setting, 8-hour TWA Exposure (from Table 4-29) after consideration of PPE with APF=10 (excluding ONUs)**

Occupational Exposure Scenario	ELCR (40 yr exposure starting at age 16 years)	
	Asbestos Worker	
	Central Tendency	High-end
Installing brakes with asbestos-containing automotive parts: 8-hour TWA exposure	1.4 E-5	<b>2.2 E-4</b>

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**Table 4-32. Excess Lifetime Cancer Risk for Installing Brakes and Clutches in Exported Cars in an Occupational Setting, 8-hour TWA Exposure (from Table 4-24.) after consideration of PPE with APF=25 (excluding ONUs)**

Occupational Exposure Scenario	ELCR (40 yr exposure starting at age 16 years)	
	Asbestos Worker	
	Central Tendency	High-end
Installing brakes with asbestos-containing aftermarket automotive parts: 8-hour TWA exposure	5.6 E-6	8.8 E-5

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For asbestos workers wearing a hypothetical respirator at APF 10, the benchmark cancer risk estimate of  $1 \times 10^{-4}$  was exceeded for high-end exposure estimates; all other scenarios (hypothetical APF of 10 for central tendency and hypothetical APF of 25 for both central and high-end exposures) had risk estimates below the benchmark. Since the assumption is that ONUs do not wear respirators, application of APFs do not apply and so their risk estimates do not change. Estimates exceeding the benchmark are shaded in pink and bolded.

**Table 4-33. 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 4-30) after consideration of PPE with APF=10 (excluding ONUs)**

Occupational Exposure Scenario	ELCR (40 yr exposure starting at age 16 years)	
	Asbestos Worker	
	Central Tendency	High-end
Installing brakes with asbestos-containing aftermarket automotive parts: short-term exposure	1.4 E-5	<b>3.3 E-4</b>

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**Table 4-34. 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 4-30) after consideration of PPE with APF=25 (excluding ONUs)**

Occupational Exposure Scenario	ELCR (40 yr exposure starting at age 16 years)	
	Asbestos Worker	
	Central Tendency	High-end
Installing brakes with asbestos-containing aftermarket automotive parts: short-term exposure	5.6 E-6	<b>1.3 E-4</b>

6400

6401 Table 4-27. and Table 4-28. display the ELCRs for short-term exposures for workers repairing or  
6402 replacing auto brakes and using hypothetical APFs of 10 and 25. For asbestos workers exposed to  
6403 asbestos, the benchmark cancer risk estimate of  $1 \times 10^{-4}$  was exceeded for high-end exposures, but not  
6404 central tendency exposures, after consideration of both hypothetical APF 10 and APF 25. Estimates  
6405 exceeding the benchmark are shaded in pink and bolded. And again, APFs do not apply to ONU  
6406 scenarios.

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6408 *Other Vehicle Friction Product – Preliminary Risk Estimates for the NASA Large Transport Plane*

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The following exposure values have been estimated for this use (see Section 2.3.1.8):

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6411 Full Shift: Central Tendency –  $<0.003$  f/cc

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6412 Full Shift: High-End –  $<0.0089$  f/cc

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6413 Short-Term: Central Tendency –  $<0.022$  f/cc

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6414 Short-Term: High-End –  $<0.045$  f/cc

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6416 Given this information, and assuming 12 hours of brake changes every year starting at age 26 years with  
6417 20 years exposure, the Excess Lifetime Cancer Risk for Super Guppy Brake/Repair Replacement for  
6418 Workers is<sup>20</sup>:

6419

<sup>20</sup>FULL SHIFT:

$$TWF_{\text{USER Brakes (2-hours on 4 days every year)}} = (3.3 \text{ hours} / 24 \text{ hours}) \cdot (3.6 \text{ days} / 365 \text{ days}) = 0.001356$$

$$IUR_{(26,20)} = 0.0318$$

$$\text{User: ELCR}_{(\text{Central Tendency})} = 0.003 \text{ f/cc} \cdot 0.001356 \cdot 1.5 \cdot 0.0318 \text{ per f/cc}$$

$$\text{User: ELCR}_{(\text{High-end})} = 0.0089 \text{ f/cc} \cdot 0.001356 \cdot 1.5 \cdot 0.0318 \text{ per f/cc}$$

SHORT TERM:

Central Tendency Exposure includes the 30-minute short-term exposure within each 3.3 hour brake change as follows:

$$\{[(0.5 \text{ hour}) \cdot (0.022 \text{ f/cc}) + (2.8 \text{ hours}) \cdot (0.002 \text{ f/cc from Section 2.3.18})] / 3.3 \text{ hours}\} = 0.005 \text{ f/cc}$$

High End Exposure includes the 30-minute short-term exposure within each 3.3 hour brake change as follows:

$$\{[(0.5 \text{ hour}) \cdot (0.045 \text{ f/cc}) + (2.8 \text{ hours}) \cdot (0.0089 \text{ f/cc from Section 2.3.1.8})] / 3.3 \text{ hours}\} = 0.014 \text{ f/cc}$$

6420 Full Shift: Central Tendency – 1.9 E-7  
 6421 Full Shift: High-End – 5.8 E-7  
 6422 Short-Term: Central Tendency – 3.2 E-7  
 6423 Short-Term: High-End – 9.1 E-7  
 6424  
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6426 **4.2.2.7 Risk Estimation for Cancer Effects Following Inhalation Exposures for**  
 6427 **Gasket Installation/Service in UTVs**

6428 Multiple publications (see Section 2.3.2.2) report on occupational exposures associated with installing  
 6429 and servicing gaskets in automobiles. The exposure data used for this COU are presented in Table 2-23.  
 6430 Data on the exposure at the central and high-end estimates are presented along with the ELCR for each  
 6431 exposure distribution in Table 4-35.  
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6434 **Table 4-35. Excess Lifetime Cancer Risk for UTV Gasket Installation/Service in an Occupational**  
 6435 **Setting, 8-hour TWA Exposure (from Table 2-23.) before consideration of PPE and any relevant**  
 6436 **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	<b>5.6 E-4</b>	<b>1.5 E-3</b>	<b>1.2 E-4</b>	<b>3.5 E-4</b>

6437 Asbestos Workers:  $ELCR_{(Central\ Tendency)} = 0.024\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0707\ per\ f/cc$   
 6438 Asbestos Workers:  $ELCR_{(High-end)} = 0.066\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0707\ per\ f/cc$   
 6439 ONU:  $ELCR_{(Central\ Tendency)} = 0.005\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0707\ per\ f/cc$   
 6440 ONU:  $ELCR_{(High-end)} = 0.015\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0707\ per\ f/cc$   
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6442 Table 4-35. presents the inhalation cancer risk estimates for workers installing and/or servicing gaskets  
 6443 in utility vehicles and for ONUs exposed to asbestos. For both workers and ONUs, the benchmark  
 6444 cancer risk estimate of  $1 \times 10^{-4}$  was exceeded for both central tendency and high-end exposures. Estimates  
 6445 exceeding the benchmark are shaded in pink and bolded.  
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6448 **Applying APFs**

6449 ELCRs for workers who install/service gaskets in UTVs exposed to asbestos using PPE with  
 6450 hypothetical APFs of 10 and 25 applied for 8-hour TWAs are presented in Table 4-36. and Table 4-37.  
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$TWF_{USER\ Brakes} = (3.3\ hours / 24\ hours) \cdot (3.6\ days / 365\ days) = 0.001356$   
 $IUR_{(26,20)} = 0.0318$   
 Worker:  $ELCR_{(Central\ Tendency)} = 0.005\ f/cc \cdot 0.001356 \cdot 1.5 \cdot 0.0318\ per\ f/cc$   
 Worker:  $ELCR_{(High-end)} = 0.014\ f/cc \cdot 0.001356 \cdot 1.5 \cdot 0.0318\ per\ f/cc$

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**Table 4-36. Excess Lifetime Cancer Risk for UTV Gasket Installation/Service in an Occupational Setting, 8-hour TWA Exposure (from Table 4-35) after consideration of PPE with APF=10 (excluding ONUs)**

Occupational Exposure Scenario	ELCR (40 yr exposure starting at age 16 years)	
	Asbestos Worker	
	Central Tendency	High-end
UTV (based on gasket repair/replacement in vehicles: 8-hr TWA exposure)	5.6 E-5	<b>1.5 E-4</b>

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**Table 4-37. Excess Lifetime Cancer Risk for UTV Gasket Installation/Service in an Occupational Setting, 8-hour TWA Exposure (from Table 4-35) after consideration of PPE with APF=25 (excluding ONUs)**

Occupational Exposure Scenario	ELCR (40 yr exposure starting at age 16 years)	
	Asbestos Worker	
	Central Tendency	High-end
UTV (based on sheet gasket use in chemical production: 8-hr TWA exposure)	2.2 E-5	6.0 E-5

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For asbestos workers using respirators with a hypothetical APF of 10, the benchmark cancer risk estimate of  $1 \times 10^{-4}$  was exceeded for the high-end exposure estimate; all other scenarios (hypothetical APF of 10 for central tendency and hypothetical APF of 25 for both central and high-end exposures) had risk estimates below the benchmark. Since the assumption is that ONUs do not wear respirators, application of APFs do not apply and so their risk estimates do not change. Estimates exceeding the benchmark are shaded in pink and bolded.

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**4.2.2.8. Summary of Risk Estimates for Cancer Effects for Occupational Inhalation Exposure Scenarios for All COUs**

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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  $1 \times 10^{-4}$  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.

**Table 4-38. Summary of Risk Estimates for Inhalation Exposures to Workers and ONUs by COU**

COU	Population	Exposure Duration and Level	Cancer Risk Estimates (before applying PPE)	Cancer Risk Estimates (with APF=10 <sup>c</sup> )	Cancer Risk Estimates (with APF=25 <sup>c</sup> )
	Worker	Central Tendency (8-hr)	<b>1.2 E-4</b>	1.2 E-5	4.8 E-6

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Diaphragms for chlor-alkali industry Section 4.2.2.1.		High-end (8-hr)	<b>8.4 E-4</b>	8.4 E-5	3.4 E-5
		Central Tendency short term	<b>1.5 E-4</b> <b>1.1 E-4<sup>a</sup></b>	1.3 E-5 <sup>d</sup>	6.0 E-6 <sup>b</sup>
		High-end short term	<b>1.3 E-3</b> <b>8.1 E-4<sup>a</sup></b>	9.9 E-5 <sup>d</sup>	5.2 E-5 <sup>b</sup>
	ONU	Central Tendency (8-hr)	5.8 E-5	N/A	N/A
		High-end (8-hr)	<b>1.9 E-4</b>	N/A	N/A
Asbestos Sheets – Gasket Stamping Section 4.2.2.2	Worker	Central Tendency (8-hr)	<b>3.3 E-4</b>	3.3 E-5	1.3 E-5
		High-end (8-hr)	<b>1.4 E-3</b>	<b>1.4 E-4</b>	5.6 E-5
		Central Tendency short term	<b>3.5 E-4</b>	3.5 E-5 <sup>e</sup>	1.4 E-5 <sup>f</sup>
		High-end short term	<b>1.4 E-3</b>	<b>1.4 E-4<sup>e</sup></b>	5.6 E-5 <sup>f</sup>
	ONU	Central Tendency (8-hr)	5.6 E-5	N/A	N/A
		High-end (8-hr)	<b>2.3 E-4</b>	N/A	N/A
		Central Tendency short term	5.6 E-5	N/A	N/A
		High-end short term	<b>2.3 E-4</b>	N/A	N/A
Asbestos Sheet Gaskets – use (based on repair/replacement data from TiO <sub>2</sub> industry) Section 4.2.2.3	Worker	Central Tendency (8-hr)	<b>6.0 E-4</b>	6.0 E-5	2.4 E-5
		High-end (8-hr)	<b>2.2 E-3</b>	<b>2.2 E-4</b>	8.8 E-5
	ONU	Central Tendency (8-hr)	<b>1.2 E-4</b>	N/A	N/A
		High-end (8-hr)	<b>3.7 E-4</b>	N/A	N/A
Oil Field Brake Blocks Section 4.2.2.4	Worker	Central Tendency (8-hr)	<b>7.0 E-4</b>	7.0 E-5	2.8 E-5
	ONU	Central Tendency (8-hr)	<b>4.6 E-4</b>	N/A	N/A
Aftermarket Auto Brakes Section 4.2.2.5	Worker	Central Tendency (8-hr)	<b>1.4 E-4</b>	1.4 E-5	5.6 E-6
		High-end (8-hr)	<b>2.2 E-3</b>	<b>2.2 E-4</b>	8.8 E-5
		Central Tendency short-term	<b>1.4 E-4</b>	1.4 E-5 <sup>e</sup>	5.6 E-6 <sup>f</sup>
		High-end short-term	<b>3.3 E-3</b>	<b>3.3 E-4<sup>e</sup></b>	<b>1.3 E-4<sup>f</sup></b>
	ONU	Central Tendency (8-hr)	1.6 E-5	N/A	N/A
		High-end (8-hr)	<b>2.6 E-4</b>	N/A	N/A
		Central Tendency short-term	1.6 E-5	N/A	N/A
		High-end short-term	<b>2.6 E-4</b>	N/A	N/A
Other Vehicle Friction Products Section 4.2.2.6	Worker	Central Tendency (8-hr)	<b>1.4 E-4</b>	1.4 E-5	5.6 E-6
		High-end (8-hr)	<b>2.2 E-3</b>	<b>2.2 E-4</b>	8.8 E-5
		Central Tendency short term	<b>1.4 E-4</b>	1.4 E-5 <sup>e</sup>	5.6 E-6 <sup>f</sup>
		High-end w short term	<b>3.3 E-3</b>	<b>3.3 E-4<sup>e</sup></b>	<b>1.3 E-4<sup>f</sup></b>
	ONU	Central Tendency (8-hr)	1.6 E-5	N/A	N/A
		High-end (8-hr)	<b>2.6 E-4</b>	N/A	N/A
		Central Tendency short-term	1.6 E-5	N/A	N/A
		High-end short-term	<b>2.6 E-4</b>	N/A	N/A

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Other Gaskets – Utility Vehicles Section 4.2.2.7	Worker	Central Tendency (8-hr)	<b>5.6 E-4</b>	5.6 E-5	2.2 E-5
		High-end (8-hr)	<b>1.5 E-3</b>	<b>1.5 E-4</b>	6.0 E-5
	ONU	Central Tendency (8-hr)	<b>1.2 E-4</b>	N/A	N/A
		High-end (8-hr)	<b>3.5 E-4</b>	N/A	N/A

6478 N/A: Not Assessed; ONUs are not assumed to wear respirators

6479 <sup>a</sup>No APF applied for 7.5 hours, APF of 25 applied for 30 minutes.

6480 <sup>b</sup>APF 25 applied for both 30 mins and 7.5 hours

6481 <sup>c</sup> As shown in Table 4-3, EPA has information suggesting use of respirators for two COUs (chlor-alkali: APF of 10 or 25; and sheet gasket use: APF of 10 only). Application of all other APFs is hypothetical.

6482 <sup>d</sup> APF 25 for 30 minutes, APF 10 for 7.5 hours

6483 <sup>e</sup> APF 10 for 30 minutes, APF 10 for 7.5 hours

6484 <sup>f</sup> APF 25 for 30 minutes, APF 25 for 7.5 hours

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6487 For workers, cancer risks were indicated for all conditions of use under high-end and central tendency  
6488 exposure scenarios when PPE was not used. With the use of PPE at APF of 10, most risks were reduced  
6489 but still persisted for chlor-alkali (for both central and high-end estimates when short-term exposures  
6490 were considered), sheet gasket stamping (high-end only), sheet gasket use (high-end only), auto brake  
6491 replacement (high-end only for 8-hour and high-end estimates when short-term exposures are  
6492 considered), and UTV gasket replacement (high-end only). When an APF of 25 was applied, risk was  
6493 still indicated for the auto brakes high-end short-term exposure scenario.

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6495 For ONUs – in which no PPE is assumed to be worn – the benchmark for risk is exceeded for all high-  
6496 end estimates and most central tendency estimates. The exceptions for central tendency exceedances are  
6497 for the following COUs: choralkali (8-hour), sheet gasket stamping (8-hour), and auto brake  
6498 replacement (8-hour and short-term exposure scenarios).

6499 **4.2.3 Risk Estimation for Consumers: Cancer Effects by Conditions of Use**

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6501 **4.2.3.1 Risk Estimation for Cancer Effects Following Episodic Inhalation Exposures**  
6502 **for DIY Brake Repair/Replacement**

6503 EPA assessed chronic chrysotile exposures for the DIY (consumer) and bystander brake repair/  
6504 replacement scenario based on repeated exposures resulting from recurring episodic exposures from  
6505 active use of chrysotile asbestos related to DIY brake-related activities. These activities include  
6506 concomitant exposure to chrysotile asbestos fibers which are reasonably anticipated to remain within  
6507 indoor and outdoor use facilities. It is well-understood that asbestos fibers in air will settle out in dust  
6508 and become re-entrained in air during any changes in air currents or activity within the indoor and  
6509 outdoor use facilities. On the other hand, in occupational settings, regular air sampling would capture  
6510 both new and old fibers and have industrial hygiene practices in place to reduce exposures.

6511

6512 EPA used the following data on exposure frequency and duration, making assumptions when needed:

6513

- 6514 • Exposure frequency of active use of chrysotile asbestos related to DIY brake repair and  
6515 replacement of 3 hours on 1 day every 3 years or 0.33 days per year. This is based on the  
6516 information that brakes are replaced every 35,000 miles, and an average number of miles driven  
6517 per year per driver in the U.S. of 13,476 miles/year ([U.S. DOT, 2018](#)).

6518

- 6519 • An estimate assuming a single brake change at age 16 years old is presented.

6520

- Estimates for exposure duration of 62 years and assuming exposure for a DIY mechanic starting at 16 years old and continuing through their lifetime (78 years) is presented. EPA also did a sensitivity analyses with different ages at first exposure and different exposure durations (see Appendix L and the uncertainties Section 4.3.7).
- Exposure frequency of concomitant exposure to chrysotile asbestos resulting from COUs was based on data in the EPA Exposure Factor Handbook ([U.S. EPA, 2011](#)). ‘Doers’ are the respondents who engage or participated in the activity.<sup>21</sup> According to Table 16-16 of the Handbook, the median time ‘Doers’ spent in garages is approximately one hour per day. The 95<sup>th</sup> percentile of time ‘Doers’ spent in garages is approximately 8 hours. According to Table 16-57 of the Handbook, the median time spent near outdoor locations is 5 minutes, and the 95<sup>th</sup> percentile of time is 30 minutes.
- Over the interval of time between the recurring episodic exposures of active COUs, the fraction of the exposure concentrations from active use of chrysotile asbestos is unknown, however some dispersion of fibers can reasonably be expected to occur over time. For example, if 50% of fibers were removed from garages each year, the concentration at the end of the first year would be 50%, at the end of the second year would 25%, and at the end of the third year would be 13%. In this example, the mean exposure over the 3-year interval would be approximately 30% of the active COUs. In order to estimate the chrysotile asbestos concentration over of the interval of time between the recurring episodic exposures of active COUs in the garages, EPA simply assumed approximate concentrations of 30% of the active COUs over the 3-year interval. In order to estimate the chrysotile asbestos concentration over of the interval of time between the recurring episodic exposures of active COUs in outdoor driveways, EPA simply assumed approximate concentrations of 2% of the active COUs over the 3-year interval based on 95% reduction of fibers each year.
- Exposure frequency of bystander exposures are similar to those of active user (i.e., Doers) and may occur at any age and exposure durations are assumed to continue for a lifetime; with an upper-bound estimate of 78 years of exposure (i.e., ages 0-78) No reduction factor was applied for indoor DIY brake work inside residential garages. A reduction factor of 10 was applied for outdoor DIY brake work<sup>22</sup>. A sensitivity analysis is presented in Appendix L which includes a lower-bound estimate for a bystander of 20 years (ages 0-20) (see the uncertainties Section 4.3.7).

### **Excess lifetime cancer risk for people engaging in DIY brake repair (consumers) and replacement**

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<sup>21</sup> This RE uses the term “consumer” or Do-It-Yourselfer (DIY) or DIY mechanic to refer to the “doer” referenced in the Exposure Factor Handbook.

<sup>22</sup> As explained in Section 2.3.1.2, EPA evaluated consumer bystander exposure for the DIY brake outdoor scenario by applying a reduction factor of 10 to the PBZ value measured outdoors for the consumer user. The reduction factor of 10 was chosen based on a comparison between the PBZ and the < 3meter from automobile values measured indoors across all activities identified in the study data utilized from Blake (a ratio of 6.5). The ratio of 6.5 was rounded up to 10, to account for an additional reduction in concentration to which a bystander may be exposed in the outdoor space based on the high air exchange rates and volume in the outdoors.



$$ELCR_{DIY\ Brakes} = EPC_{DIY\ Brakes} \cdot TWF_{DIY\ Brakes} \cdot IUR_{LTL(DIY\ Brakes)} +$$

$$EPC_{Concomitant\ Exposures} \cdot TWF_{Concomitant\ Exposures} \cdot IUR_{LTL(Concomitant\ Exposures)}$$

$$TWF_{DIY\ Brakes\ (3\text{-hours\ on\ 1\ day\ every\ 3\ years)} = (3/24) \cdot (1/3) \cdot (1/365) = 0.0001142$$

$$IUR_{LTL(DIY\ Brakes)} = IUR_{(16,62)} = 0.0768\ \text{per\ f/cc}$$

$$TWF_{Concomitant\ Exposures\ (1\text{-hour\ per\ day\ every\ day)} = (1/24) \cdot (365/365) = 0.04167$$

$$IUR_{LTL(Concomitant\ Exposures)} = IUR_{(16,62)} = 0.0768\ \text{per\ f/cc}$$

**Excess lifetime cancer risk for bystanders to DIY brake repair and replacement**

$$ELCR_{Bystander} = EPC_{Bystander\ to\ DIY\ brake\ work} \cdot TWF_{Bystander\ to\ DIY\ brake\ work} \cdot IUR_{Lifetime} +$$

$$EPC_{Bystander\ to\ Concomitant\ Exposures} \cdot TWF_{Bystander\ to\ Concomitant\ Exposures} \cdot IUR_{Lifetime}$$

$$TWF_{Bystander\ to\ DIY\ brakes\ work\ (3\text{-hours\ on\ 1\ day\ every\ 3\ years)} = (3/24) \cdot (1/3) \cdot (1/365) = 0.0001142$$

$$IUR_{Lifetime} = 0.16\ \text{per\ f/cc}$$

$$TWF_{Bystander\ to\ Concomitant\ Exposures\ (1\text{-hour\ per\ day\ every\ day)} = (1/24) \cdot (365/365) = 0.04167$$

Exposure values from Table 2-32 were used to represent indoor brake work (with compressed air) and are the basis for the exposure levels used in Tables 4-39 through 4-42, EPA then assumed that the concentration of chrysotile asbestos in the interval between brake work (every 3 years) is 30% of that during measured active use.

Consumers and bystanders were assumed to spend one hour per day in their garages based on the 50<sup>th</sup> percentile estimate in the EPA Exposure Factors Handbook. Based on these assumptions, the consumer risk estimate was exceeded for central and high-end exposures based on replacing breaks every 3 years (Table 4-39). Estimates exceeding the benchmark are shaded in pink and bolded.

Tables 4-40 and 4-41 used the alternative assumptions for age at first exposure (16 years old) and exposure duration (40 years) for the DIY user; and the assumptions for the exposure duration of the bystander (lifetime). Table 4-41 presents another alternative estimate for both the DIY user (performing work from ages 16-36, and a bystander being present from ages 0-20) for the one-hour/day scenario (i.e., Table 4-40). The risk estimates note that the benchmark is exceeded for both these alternative estimates.

**Table 4-39. Excess Lifetime Cancer Risk for Indoor DIY Brake/Repair Replacement with Compressed Air Use for Consumers and Bystanders (exposures from Table 2-32 without a reduction factor) with Exposures at 30% of 3-hour User Concentrations between Brake/Repair Replacement (Consumers 1 hour/day spent in garage; Bystanders 1 hour/day)**

Consumer Exposure Scenario	Exposure Levels (fibers/cc)	ELCR (62 yr exposure starting at age 16 years)	ELCR (Lifetime exposure)
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	DIY User		DIY Bystander		DIY User		DIY Bystander	
	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end
Aftermarket automotive parts – brakes (3-hour TWA indoors every 3 years with compressed air)	0.0445	0.4368	0.0130	0.0296	<b>4.3 E-5</b>	<b>4.2 E-4</b>	<b>2.6 E-5</b>	<b>6.0 E-5</b>

6606  $TWF_{Concomitant\ Exposures\ (1\ hour\ per\ day\ every\ day)} = (1/24) * (365/365) = 0.04167$   
 6607  $IUR_{(16,62)} = 0.0768; IUR_{(Lifetime)} = 0.16$   
 6608 DIY User:  $ELCR_{(Central\ Tendency)} = 0.0445\ f/cc \cdot 0.0001142 \cdot 0.0768\ per\ f/cc + 0.0445 \cdot 0.3 \cdot 0.04167 \cdot 0.0768$   
 6609 DIY User:  $ELCR_{(High-end)} = 0.4368\ f/cc \cdot 0.0001142 \cdot 0.0768\ per\ f/cc + 0.4368 \cdot 0.3 \cdot 0.04167 \cdot 0.0768$   
 6610 DIY Bystander:  $ELCR_{(Central\ Tendency)} = 0.013\ f/cc \cdot 0.0001142 \cdot 0.16\ per\ f/cc + 0.013 \cdot 0.3 \cdot 0.04167 \cdot 0.16$   
 6611 DIY Bystander:  $ELCR_{(High-end)} = 0.0296\ f/cc \cdot 0.0001142 \cdot 0.16\ per\ f/cc + 0.0296 \cdot 0.3 \cdot 0.04167 \cdot 0.16$   
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6614 **Table 4-40. Excess Lifetime Cancer Risk for Indoor DIY Brake/Repair Replacement with**  
 6615 **Compressed Air Use for Consumers for 20 year duration (exposures from Table 2-32 without a**  
 6616 **reduction factor) with Exposures at 30% of 3-hour User Concentrations between Brake/Repair**  
 6617 **Replacement (Consumers 1 hour/day spent in garage)**

Consumer Exposure Scenario	Exposure Levels (fibers/cc)				ELCR (20 yr exposure starting at age 16 years)		ELCR ((20 yr exposure starting at age 0 years))	
	DIY User		DIY Bystander		DIY User		DIY Bystander	
	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end
Aftermarket automotive parts – brakes (3-hour TWA indoors every 3 years with compressed air)	0.0445	0.4368	0.0130	0.0296	<b>2.8 E-5</b>	<b>2.7 E-4</b>	<b>1.7 E-5</b>	<b>3.8 E-5</b>

6618  $TWF_{Concomitant\ Exposures\ (1\ hour\ per\ day\ every\ day)} = (1/24) * (365/365) = 0.04167$   
 6619  $IUR_{(16,36)} = 0.0499; IUR_{(0,20)} = 0.101$   
 6620 DIY User:  $ELCR_{(Central\ Tendency)} = 0.0445\ f/cc \cdot 0.0001142 \cdot 0.0499\ per\ f/cc + 0.0445 \cdot 0.3 \cdot 0.04167 \cdot 0.0499$   
 6621 DIY User:  $ELCR_{(High-end)} = 0.4368\ f/cc \cdot 0.0001142 \cdot 0.0499\ per\ f/cc + 0.4368 \cdot 0.3 \cdot 0.04167 \cdot 0.0499$   
 6622 DIY Bystander:  $ELCR_{(Central\ Tendency)} = 0.013\ f/cc \cdot 0.0001142 \cdot 0.101\ per\ f/cc + 0.013 \cdot 0.3 \cdot 0.04167 \cdot 0.101$   
 6623 DIY Bystander:  $ELCR_{(High-end)} = 0.0296\ f/cc \cdot 0.0001142 \cdot 0.101\ per\ f/cc + 0.0296 \cdot 0.3 \cdot 0.04167 \cdot 0.101$   
 6624

6625 For Table 4-41, users were assumed to spend eight hours per day in their garages based on the 95<sup>th</sup>  
 6626 percentile estimate in the EPA Exposure Factors Handbook (Table 16-16 in the Handbook). Bystanders  
 6627 were assumed to spend one hour per day in their garages. Based on these assumptions, both the  
 6628 consumer and the bystander risk estimates were exceeded for central tendency and high-end exposures.  
 6629 Estimates exceeding the benchmark are shaded in pink and bolded.

6630 **Table 4-41. Excess Lifetime Cancer Risk for Indoor DIY Brake/Repair Replacement with**  
 6631 **Compressed Air Use for Consumers and Bystanders (exposures from Table 2-32 without a**  
 6632 **reduction factor) with Exposures at 30% of 3-hour User Concentrations between Brake/Repair**  
 6633 **Replacement (Consumers 8 hours/day spent in garage; Bystanders 1 hour/day)**

Consumer Exposure Scenario	Exposure Levels (Fibers/cc)		ELCR (62 yr exposure starting at age 16 years)		ELCR (Lifetime exposure)	
	DIY User	DIY Bystander	DIY User	DIY Bystander	DIY User	DIY Bystander

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	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end
Aftermarket automotive parts – brakes (3-hour TWA indoors with compressed air)	0.0445	0.4368	0.0130	0.0296	3.4 E-4	3.4 E-3	2.6 E-5	6.0 E-5

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TWF<sub>Concomitant Exposures (8 hours per day every day)</sub> = (8/24)\*(365/365) = 0.3333  
 IUR<sub>(16,62)</sub>=0.0768; IUR<sub>(Lifetime)</sub>=0.16  
 DIY User: ELCR<sub>(Central Tendency)</sub> = 0.0445 f/cc • 0.0001142 • 0.0768 per f/cc + 0.0445 • 0.3 • 0.3333 • 0.0768  
 DIY User: ELCR<sub>(High-end)</sub> = 0.4368 f/cc • 0.0001142 • 0.0768 per f/cc + 0.4368 • 0.3 • 0.3333 • 0.0768  
 DIY Bystander: ELCR<sub>(Central Tendency)</sub> = 0.013 f/cc • 0.0001142 • 0.16 per f/cc + 0.013 • 0.3 • 0.04167 • 0.16  
 DIY Bystander: ELCR<sub>(High-end)</sub> = 0.0296 f/cc • 0.0001142 • 0.16 per f/cc + 0.0296 • 0.3 • 0.04167 • 0.16

In Table 4-42 the assumption is that DIY brake/repair replacement with compressed air is limited to a single brake change at age 16 years. EPA then assumed that the concentration of chrysotile asbestos following this COU decreases 50% each year as was assumed in all the indoor exposure scenarios. EPA then assumed that both the DIYer and the bystander would remain in the house for 10 years. Risks were determined for the 10-year period by calculating the risk with the appropriate partial lifetime IUR and re-entrainment exposure over 10 years, averaging 10% of the brake/repair concentrations each year (total 10-year cumulative exposure is 50% in first year plus 25% in second year is for all practical purposes equals a limit of one year at the 3-hour concentration divided by 10 years).

**Table 4-42. Risk Estimate using one brake change at age 16 years with 10 years further exposure. Excess Lifetime Cancer Risk for Indoor DIY Brake/Repair Replacement with Compressed Air Use for Consumers and Bystanders (exposures from Table 2-32 without a reduction factor) (Consumers 1 hour/day spent in garage; Bystanders 1 hour/day)**

Consumer Exposure Scenario	Exposure Levels (fibers/cc)				ELCR (62 yr exposure starting at age 16 years)		ELCR (Lifetime exposure)	
	DIY User		DIY Bystander		DIY User		DIY Bystander	
	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end
Aftermarket automotive parts – brakes (3-hour TWA indoors once at 16 yrs old; with compressed air)	0.0445	0.4368	0.0130	0.0296	5.6 E-6	5.5 E-5	3.2 E-6	7.3 E-6

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TWF<sub>Concomitant Exposures (1 hour per day every day)</sub> = (1/24)\*(365/365) = 0.04167  
 IUR<sub>(16,10)</sub>=0.0300; IUR<sub>(0,10)</sub>=0.0595  
 DIY User: ELCR<sub>(Central Tendency)</sub> = 0.0445 f/cc • 0.000005524 • 0.0300 per f/cc + 0.0445 • 0.1 • 0.04167 • 0.0300  
 DIY User: ELCR<sub>(High-end)</sub> = 0.4368 f/cc • 0.000005524 • 0.0300 per f/cc + 0.4368 • 0.1 • 0.04167 • 0.0300  
 DIY Bystander: ELCR<sub>(Central Tendency)</sub> = 0.013 f/cc • 0.000005524 • 0.0595 per f/cc + 0.013 • 0.1 • 0.04167 • 0.0595  
 DIY Bystander: ELCR<sub>(High-end)</sub> = 0.0296 f/cc • 0.000005524 • 0.0595 per f/cc + 0.0296 • 0.1 • 0.04167 • 0.0595

Exposure Levels in Table 4-43 are from Table 2-32 and the assumption is used that the concentration of chrysotile asbestos in the interval between brake works is 2% of that during measured active use. Users and bystanders were assumed to spend 5 minutes per day in the driveway each day based on the 50<sup>th</sup> percentile estimate in the EPA Exposure Factors Handbook (in Table 16-57 in the Handbook). The

6667 reduction factor is 10 for bystanders<sup>23</sup>. The risk estimates for the DIY consumer exceeded the risk  
 6668 benchmark for the high-end exposure only, whereas the risk estimates were not exceeded for either  
 6669 scenario for the bystanders.  
 6670

6671 **Table 4-43. Excess Lifetime Cancer Risk for Outdoor DIY Brake/repair Replacement for**  
 6672 **Consumers and Bystanders (5 minutes per day in driveway) (from Table 2-32 with a reduction**  
 6673 **factor of 10)**

Consumer Exposure Scenario	Exposure Levels (Fibers/cc)				ELCR (62 yr exposure starting at age 16 years)		ELCR (Lifetime exposure)	
	DIY User		DIY Bystander		DIY User		DIY Bystander	
	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end
Aftermarket automotive parts – brakes (3-hour TWA outdoors)	0.007	0.0376	0.0007	0.0038	9.9 E-8	5.3 E-7	2.1 E-8	1.1 E-7

6674  $TWF_{\text{Concomitant Exposures (0.0833 hours per day every day)}} = (0.08333/24) \cdot (365/365) = 0.003472$   
 6675  $IUR_{(16,62)} = 0.0768$ ;  $IUR_{(\text{Lifetime})} = 0.16$   
 6676 DIY User:  $ELCR_{(\text{Central Tendency})} = 0.007 \text{ f/cc} \cdot 0.0001142 \cdot 0.0768 \text{ per f/cc} + 0.007 \cdot 0.02 \cdot 0.003472 \cdot 0.0768$   
 6677 DIY User:  $ELCR_{(\text{High-end})} = 0.0376 \text{ f/cc} \cdot 0.0001142 \cdot 0.0768 \text{ per f/cc} + 0.0376 \cdot 0.02 \cdot 0.003472 \cdot 0.0768$   
 6678 DIY Bystander:  $ELCR_{(\text{Central Tendency})} = 0.0007 \text{ f/cc} \cdot 0.0001142 \cdot 0.16 \text{ per f/cc} + 0.0007 \cdot 0.02 \cdot 0.003472 \cdot 0.16$   
 6679 DIY Bystander:  $ELCR_{(\text{High-end})} = 0.0038 \text{ f/cc} \cdot 0.0001142 \cdot 0.16 \text{ per f/cc} + 0.0038 \cdot 0.02 \cdot 0.003472 \cdot 0.16$   
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6682 **Table 4-44. Excess Lifetime Cancer Risk for Outdoor DIY Brake/Repair Replacement for**  
 6683 **Consumers and Bystanders (30 minutes per day in driveway) (from Table 2-32 with a reduction**  
 6684 **factor of 10)**

Occupational Exposure Scenario	Exposure Levels (Fibers/cc)				ELCR (62 yr exposure starting at age 16 years)		ELCR (Lifetime exposure)	
	DIY User		DIY Bystander		DIY User		DIY Bystander	
	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end
Aftermarket automotive parts – brakes (3-hour TWA outdoors)	0.007	0.0376	0.0007	0.0038	2.9 E-7	1.5 E-6	5.9 E-8	3.2 E-7

6685  $TWF_{\text{Concomitant Exposures (0.5 hours per day every day)}} = (0.5/24) \cdot (365/365) = 0.02083$   
 6686  $IUR_{(16,62)} = 0.0768$ ;  $IUR_{(\text{Lifetime})} = 0.16$   
 6687 DIY User:  $ELCR_{(\text{Central Tendency})} = 0.007 \text{ f/cc} \cdot 0.0001142 \cdot 0.0768 \text{ per f/cc} + 0.007 \cdot 0.02 \cdot 0.02083 \cdot 0.0768$   
 6688 DIY User:  $ELCR_{(\text{High-end})} = 0.0376 \text{ f/cc} \cdot 0.0001142 \cdot 0.0768 \text{ per f/cc} + 0.0376 \cdot 0.02 \cdot 0.02083 \cdot 0.0768$   
 6689 DIY Bystander:  $ELCR_{(\text{Central Tendency})} = 0.0007 \text{ f/cc} \cdot 0.0001142 \cdot 0.16 \text{ per f/cc} + 0.0007 \cdot 0.02 \cdot 0.02083 \cdot 0.16$   
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<sup>23</sup> As explained in Section 2.3.1.2, EPA evaluated consumer bystander exposure for the DIY brake outdoor scenario by applying a reduction factor of 10 to the PBZ value measured outdoors for the consumer user. The reduction factor of 10 was chosen based on a comparison between the PBZ and the < 3meter from automobile values measured indoors across all activities identified in the study data utilized from Blake (a ratio of 6.5). The ratio of 6.5 was rounded up to 10, to account for an additional reduction in concentration to which a bystander may be exposed in the outdoor space based on the high air exchange rates and volume in the outdoors.

6691 DIY Bystander:  $ELCR_{(High-end)} = 0.0038 \text{ f/cc} \cdot 0.0001142 \cdot 0.16 \text{ per f/cc} + 0.0038 \cdot 0.02 \cdot 0.02083 \cdot 0.16$   
 6692  
 6693

6694 Exposure Levels from Table 2-32 are used in Table 4-44. The assumption that the concentration of  
 6695 chrysotile asbestos in the interval between brake works is 2% of that during measured active use. Users  
 6696 and bystanders were assumed to spend 30 minutes per day walking to their cars in the driveway each  
 6697 day based on the 95<sup>th</sup> percentile estimate in the EPA Exposure Factors Handbook (in Table 16-57 in the  
 6698 Handbook). The reduction factor is 10 for bystanders. Neither of the risk estimates for consumers or  
 6699 bystanders in Table 4-44 exceeded the risk benchmark for central tendency and the DIY user exceeded  
 6700 for the high-end but the bystander did not.  
 6701

**4.2.3.2 Risk Estimation for Cancer Effects following Episodic Inhalation Exposures  
 for UTV Gasket Repair/replacement**

6704 EPA assessed chrysotile exposures for the DIY (consumer) and bystander UTV gasket  
 6705 repair/replacement scenario based on aggregated exposures resulting from recurring episodic exposures  
 6706 from active use of chrysotile asbestos related to DIY brake-related activities. These activities include  
 6707 concomitant exposure to chrysotile asbestos fibers which are reasonably anticipated to remain within  
 6708 indoor use facilities. It is well-understood that asbestos fibers in air will settle out in dust and become re-  
 6709 entrained in air during any changes in air currents or activity indoors. On the other hand, in occupational  
 6710 settings, regular air sampling would capture both new and old fibers and have industrial hygiene  
 6711 practices in place to reduce exposures.  
 6712

6713 For the risk estimations for the UTV gasket COU, EPA used the same data/assumptions identified in  
 6714 Section 4.2.3.1 for brakes for exposure frequency and duration; with the exception that there is no  
 6715 outdoor exposure scenario. A sensitivity analysis is presented which includes a lower-bound estimate for  
 6716 a bystander of 20 years (ages 0-20) (see Appendix L and the uncertainties Section 4.3.7).  
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6718 In Table 4-45, the assumption is that DIY UTV gasket replacement is limited to a single gasket change  
 6719 at age 16 years. EPA then assumed that the concentration of chrysotile asbestos in following this COU  
 6720 decreases 50% each year as was assumed in all the indoor exposure scenarios. EPA then assumed that  
 6721 both the DIYer and the bystander would remain in the house for 10 years. Risks were determined for the  
 6722 10-year period by calculating the risk with the appropriate partial lifetime IUR.  
 6723

6724 Based on these assumptions, the consumer risk estimate was exceeded for high-end exposures based on  
 6725 a single UTV gasket change and remaining in the house for 10 years (Table 4-45). Estimates exceeding  
 6726 the benchmark are shaded in pink and bolded.  
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**Table 4-45. Risk Estimate using one UTV gasket change at age 16 years with 10 years further exposure. Excess Lifetime Cancer Risk for Indoor DIY UTV gasket change for Consumers and Bystanders (exposures from Table 2-32 without a reduction factor) (Consumers 1 hour/day spent in garage; Bystanders 1 hour/day)**

Consumer Exposure Scenario	Exposure Levels (fibers/cc)				ELCR (62 yr exposure starting at age 16 years)		ELCR (Lifetime exposure)		
	DIY User		DIY Bystander		DIY User		DIY Bystander		
	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end	

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Aftermarket automotive parts – brakes (3-hour TWA once, indoors)	0.024	0.066	0.012	0.03	4.6 E-7	<b>1.3 E-6</b>	1.7 E-7	9.2 E-7
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TWF<sub>Concomitant Exposures (1 hour per day every day)</sub> = (1/24)\*(365/365) = 0.04167  
 IUR<sub>(16,10)</sub>=0.0300; IUR<sub>(0,10)</sub>=0.0595  
 DIY User: ELCR<sub>(Central Tendency)</sub> = 0.024 f/cc • 0.000005524 • 0.0300 per f/cc + 0.024 • 0.1 • 0.04167 • 0.0300  
 DIY User: ELCR<sub>(High-end)</sub> = 0.066 f/cc • 0.000005524 • 0.0300 per f/cc + 0.066 • 0.1 • 0.04167 • 0.0300  
 DIY Bystander: ELCR<sub>(Central Tendency)</sub> = 0.012 f/cc • 0.000005524 • 0.0595 per f/cc + 0.012 • 0.1 • 0.04167 • 0.0595  
 DIY Bystander: ELCR<sub>(High-end)</sub> = 0.03 f/cc • 0.000005524 • 0.0595 per f/cc + 0.03 • 0.1 • 0.04167 • 0.0595

**Table 4-46. Excess Lifetime Cancer Risk for Indoor DIY UTV Gasket /Repair Replacement for Consumers and Bystanders (exposures from Table 2-32) (Users 1 hour/day spent in garage; Bystanders 1 hour/day)**

Consumer Exposure Scenario	Exposure Levels (Fibers/cc)				ELCR (62 yr exposure starting at age 16 years)		ELCR (Lifetime exposure)	
	DIY User		DIY Bystander		DIY User		DIY Bystander	
	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end
Aftermarket UTV parts – gaskets (indoors every 3 years)	0.024	0.066	0.012	0.030	<b>2.3 E-5</b>	<b>6.4 E-5</b>	<b>2.4 E-5</b>	<b>6.1 E-5</b>

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TWF<sub>Concomitant Exposures (1 hour per day every day)</sub> = (1/24)\*(365/365) = 0.04167  
 IUR<sub>(16,62)</sub>=0.0768; IUR<sub>(Lifetime)</sub>=0.16  
 DIY User: ELCR<sub>(Central Tendency)</sub> = 0.024 f/cc • 0.0001142 • 0.0768 per f/cc + 0.024 • 0.3 • 0.04167 • 0.0768  
 DIY User: ELCR<sub>(High-end)</sub> = 0.066 f/cc • 0.0001142 • 0.0768 per f/cc + 0.066 • 0.3 • 0.04167 • 0.0768  
 DIY Bystander: ELCR<sub>(Central Tendency)</sub> = 0.012 f/cc • 0.0001142 • 0.16 per f/cc + 0.012 • 0.3 • 0.04167 • 0.16  
 DIY Bystander: ELCR<sub>(High-end)</sub> = 0.030 f/cc • 0.0001142 • 0.16 per f/cc + 0.030 • 0.3 • 0.04167 • 0.16

The exposure values from Table 2-32 were used to estimate ELCRs in Table 4-46 for indoor DIY gasket repair/replacement (one-hour/day assumption). The assumption is that the concentration of chrysotile asbestos in the interval between gasket work (every 3 years) is 30% of that during measured active use. Consumers and bystanders were assumed to spend one hour per day in their garages based on the 50<sup>th</sup> percentile estimate in the EPA Exposure Factors Handbook (in Table 16-16 in the Handbook). Based on these assumptions, both the consumer and the bystander risk estimates were exceeded for central tendency and high-end exposures. Estimates exceeding the benchmark are shaded in pink and bolded.

**Table 4-47. Excess Lifetime Cancer Risk for Indoor DIY Gasket/Repair Replacement for Consumers and Bystanders (exposures from Table 2-32) (Consumers 8 hours/day spent in garage; Bystanders 1 hour/day)**

Consumer Exposure Scenario	Exposure Levels (Fibers/cc)				ELCR (62 yr exposure starting at age 16 years)		ELCR (Lifetime exposure)	
	DIY User		DIY Bystander		DIY User		DIY Bystander	
	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end

Aftermarket automotive parts – brakes (indoors every three years)	0.024	0.066	0.012	0.030	<b>1.8 E-4</b>	<b>5.1 E-4</b>	<b>2.4 E-5</b>	<b>6.1 E-5</b>
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TWF<sub>Concomitant Exposures (8 hours per day every day)</sub> = (8/24)\*(365/365) = 0.3333  
 IUR<sub>(16,62)</sub>=0.0768; IUR<sub>(Lifetime)</sub>=0.16  
 DIY User: ELCR<sub>(Central Tendency)</sub> = 0.024 f/cc • 0.0001142 • 0.0768 per f/cc + 0.024 • 0.3 • 0.3333 • 0.0768  
 DIY User: ELCR<sub>(High-end)</sub> = 0.066 f/cc • 0.0001142 • 0.0768 per f/cc + 0.066 • 0.3 • 0.3333 • 0.0768  
 DIY Bystander: ELCR<sub>(Central Tendency)</sub> = 0.012 f/cc • 0.0001142 • 0.16 per f/cc + 0.012 • 0.3 • 0.04167 • 0.16  
 DIY Bystander: ELCR<sub>(High-end)</sub> = 0.030 f/cc • 0.0001142 • 0.16 per f/cc + 0.030 • 0.3 • 0.04167 • 0.16

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The exposure values from Table 2-32 were used to estimate ELCRs in Table 4-47 for indoor DIY gasket repair/replacement (eight hours/day assumption). The assumption is that the concentration of chrysotile asbestos in the interval between replacement is 30% of that during measured active use. Users were assumed to spend eight hours per day in their garages based on the 95<sup>th</sup> percentile estimate in the EPA Exposure Factors Handbook. Bystanders were assumed to spend one hour per day in their garages. Based on these assumptions, both the consumer and the bystander risk estimates were exceeded for central tendency and high-end exposures. Estimates exceeding the benchmark are shaded in pink and bolded.

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#### **4.2.3.3 Summary of Consumer and Bystander Risk Estimates by COU for Cancer Effects Following Inhalation Exposures**

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Table 4-48 summarizes the risk estimates for inhalation exposures for all consumer exposure scenarios. Risk estimates that exceed the benchmark (i.e., cancer risks greater than the cancer risk benchmark) are shaded and in bold.

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Ranging from using an estimate for a single brake job at 16 years of age, and estimates for age at first exposure (16 years old for DIY users and 0 years for bystanders) and exposure duration (62 years for DIY users and 78 years for bystanders), for all COUs that were assessed, there were risks to consumers (DIY) and bystanders for all high-end exposures with the following exceptions: outdoor brake repairs (5 minutes/day in the driveway – benchmark not exceeded for high-end for both DIY and bystanders) and outdoor brake repairs (30 minutes/day in the driveway – benchmark not exceeded for high-end exposures for the bystander only). In addition, risks were noted for central tendency estimates for all COUs (brake and UTV gasket repair/replacement) for both consumers (DIY) and bystanders except for the outdoor exposure scenarios. Outdoor exposure scenarios for brake repair/replacement for 5 minutes in the driveway was the only scenario that did not exceed the benchmark for consumers (DIY) and bystanders. For outdoor exposures of 30 minutes/day once every 3 years, there were no exceedances for either the DIY or bystander for the central tendency exposure scenario.

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To evaluate sensitivity to the age at first exposure and exposure duration assumptions, EPA conducted multiple sensitivity analyses assuming that exposure of DIY users was limited to a single brake change at age 16 years as well as durations of exposure as short as 20 years with different ages of first exposure. Section 4.3.7 provides a summary of the detailed analyses in Appendix L. These sensitivity analyses show that in four of the five scenario pairings different durations and age of first exposure, only one of 24 possible scenarios changed from exceeding the benchmark cancer risk level of 1x10<sup>-6</sup> to no exceedance (DIY user, brake repair outdoors, 30 minutes/ day, high-end only). In the fifth scenario (Sensitivity Analysis 2), there was no change in any of the 24 scenarios exceeding risk benchmarks. All analyses are in Appendix L.

6811 **Table 4-48. Summary of Risk Estimates for Inhalation Exposures to Consumers and Bystanders**  
 6812 **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 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	3.0 E-6
				High-end	7.1 E-6
	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	
Imported Asbestos Products	Gaskets Repair/replacement in UTVs Indoor, 1 hour/d, 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 4.2.3.2	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
		Section 4.2.3.2	DIY	Central Tendency	1.8 E-4



Life Cycle Stage/Category	Subcategory	Consumer Exposure Scenario	Population	Exposure Duration and Level	Cancer Risk Estimates
	Gaskets Repair/ replacement in UTVs 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	High-end	5.1 E-4
				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
High-end	7.16 E-6				

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### 4.3 Assumptions and Key Sources of Uncertainty

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#### 4.3.1 Key Assumptions and Uncertainties in the Uses of Asbestos in the U.S.

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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 14<sup>th</sup>) 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. EPA received voluntary acknowledgement of asbestos import and use from a handful of industries that fall under these COU categories. Some of the COUs are very specialized, and with the exception of the chlor-alkali industry, there are many uncertainties with respect to the extent of use, the number of workers and consumers involved and the exposures that might occur from each activity. For example, the number of consumers who might change out their brakes on their cars with asbestos-containing brakes ordered on the Internet or the number of consumers who might change out the asbestos gaskets in the exhaust system of their UTVs is unknown.

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On April 25, 2019, EPA finalized an Asbestos Significant New Use Rule (SNUR) under TSCA section 5 that prohibits any manufacturing (including import) or processing for discontinued uses of asbestos from restarting without EPA having an opportunity to evaluate each intended use for risks to health and the environment and to take any necessary regulatory action, which may include a prohibition. By finalizing the asbestos SNUR to include manufacturing (including import) or processing discontinued uses not already banned under TSCA, EPA is highly certain that manufacturing (including import), processing, or distribution of asbestos is not intended, known or reasonably foreseen beyond the 6 product categories identified herein.

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EPA will consider legacy uses and associated disposal in subsequent supplemental documents.

### 4.3.2 Key Assumptions and Uncertainties in the Environmental (Aquatic) Assessment

While the EPA has identified reasonably available aquatic toxicity data to characterize the overall environmental hazards of chrysotile asbestos, there are uncertainties and data limitations regarding the analysis of environmental hazards of chrysotile asbestos in the aquatic compartment. Limited data are available to characterize effects caused by acute exposures of chrysotile asbestos to aquatic organisms. Only one short-term aquatic invertebrate study was identified (Belanger et al., 1986b). In addition, the reasonably available data characterizes the effects of chronic exposure to waterborne chrysotile asbestos in fish and clams. While these species are assumed to be representative for aquatic species, without additional data to characterize the effects of asbestos to a broader variety of taxa, the broader ecosystem-level effects of asbestos are uncertain. The range of endpoints reported in the studies across different life stages meant that a single definitive, representative endpoint could not be determined, and the endpoints needed to be discussed accordingly. Several of the effects reported by Belanger *et al.* (e.g., gill tissue altered, fiber accumulation, and siphoning activity) are not directly related to endpoints like mortality or reproductive effects and therefore the biological relevance is unclear. Lastly, the effect concentrations reported in these studies may misrepresent the actual effect concentrations due to the inconsistent methodologies for determining aquatic exposure concentrations of asbestos measured in different laboratories.

During development of the PF, EPA was still in the process of identifying potential asbestos water releases for the COUs. After the PF was released, EPA continued to search EPA databases as well as the literature and either engaged in a dialogue with industries or reached out for a dialogue to shed light on potential releases to water. In addition to the Belanger et al. studies, EPA evaluated the following lines of evidence that suggested there is minimal or no releases of chrysotile asbestos to water: (1) 96% of ~14,000 samples from drinking water sources are below the minimum reporting level of 0.2 MFL and less than 0.2% are above the MCL of 7 MFL for humans; (2) the source of the asbestos fibers is not known to be from a TSCA condition of use in this draft risk evaluation; and (3) TRI data have not shown releases of asbestos to water (Section 2.2.1.). The available information indicated that there were surface water releases of asbestos; however, not all releases are subject to reporting (e.g., effluent guidelines) or are applicable (e.g., friability). Based on the reasonably available information in the published literature, provided by industries using asbestos, and reported in EPA databases, there is minimal or no releases of asbestos to surface water associated with the COUs that EPA is evaluating in this risk evaluation. Therefore, EPA concludes there is no unreasonable risk to aquatic or sediment-dwelling environmental organisms. While this does introduce some uncertainty, EPA views it as low and has confidence in making a determination of no exposure regarding potential releases to water for the COUs in this risk evaluation. This conclusion is also based on the information in Section 2.3 in which, for the major COUs (i.e., chlor-alkali, sheet gasket stamping and sheet gasket use), there is documentation of collecting asbestos waste for disposal via landfill. In addition, there are no reported releases of asbestos to water from TRI.

### 4.3.3 Key Assumptions and Uncertainties in the Occupational Exposure Assessment

The method of identifying asbestos in this RE is based on fiber counts made by phase contrast microscopy (PCM). PCM measurements made in occupational environments were used both in the exposure studies and in the studies used to support the derivation of the chrysotile IUR. PCM detects only fibers longer than 5  $\mu\text{m}$  and  $>0.4 \mu\text{m}$  in diameter, while transmission electron microscopy (TEM), often found in environmental monitoring measurements, can detect much smaller fibers. Most of the studies used in the RE have reported asbestos concentrations using PCM.

6889 In general, when enough data were reasonably available, the 95th and 50th percentile exposure  
6890 concentrations were calculated using reasonably available data (i.e., the chlor-alkali worker monitoring  
6891 data). In other instances, EPA had very little monitoring data available on occupational exposures for  
6892 certain COUs (e.g., sheet gasket stamping and brake blocks) or limited exposure monitoring data in the  
6893 published literature as well. Where there are few data points available, it is unlikely the results will be  
6894 representative of worker exposure across the industry depending on the sample collection location (PBZ  
6895 or source zone) and timing of the monitoring.

6896  
6897 EPA acknowledges that the reported inhalation exposure concentrations for the industrial scenario uses  
6898 may not be representative for the exposures in all companies within that industry. For example, there are  
6899 only three chlor-alkali companies who own a total of 15 facilities in the U.S. that use chrysotile  
6900 diaphragms, but their operations are different, where some of them hydroblast and reuse their chrysotile  
6901 asbestos-containing diaphragms and others replace them. The exposures to workers related to these two  
6902 different activities are expected to be different.

6903  
6904 EPA also received data from one company that fabricates sheet gaskets and one company that uses sheet  
6905 gaskets. These data were used, even though there are limitations, such as the representativeness of  
6906 practices in their respective industries.

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6908 All the raw chrysotile asbestos imported into the U.S. is used by the chlor-alkali industry for use in  
6909 asbestos diaphragms. The number of chlor-alkali plants in the U.S. is known and therefore the number  
6910 of workers potentially exposed is fairly certain. In addition, estimates of workers employed in this  
6911 industry were provided by the chlor-alkali facilities. However, the number of workers potentially  
6912 exposed during other COUs is very limited. Only two workers were identified for stamping sheet  
6913 gaskets, and two titanium dioxide manufacturing facilities were identified in the U.S. who use asbestos-  
6914 containing gaskets. However, EPA is not certain if asbestos-containing sheet gaskets are used in other  
6915 industries and to what extent. For the other COUs, no estimates of the number of potentially exposed  
6916 workers were submitted to EPA by industry or its representatives, so estimates were used. Therefore,  
6917 numbers of workers potentially exposed were estimated; and these estimates could equally be an over-  
6918 estimate or an under-estimate.

6919  
6920 Finally, there is uncertainty in how EPA categorized the exposure data. Each PBZ and area data point  
6921 was classified as either “worker” or “occupational non-user.” The categorizations are based on  
6922 descriptions of worker job activity as provided in worker monitoring data, in the literature and EPA’s  
6923 judgment. In general, PBZ samples were categorized as “worker” and area samples were categorized as  
6924 “occupational non-user.” Exposure data for ONUs were not available for most scenarios. EPA assumes  
6925 that these exposures are expected to be lower than worker exposures, since ONUs do not typically  
6926 directly handle asbestos nor are in the immediate proximity of asbestos.

#### 6927 4.3.4 Key Assumptions and Uncertainties in the Consumer Exposure Assessment

6928 Due to lack of specific information on DIY consumer exposures, the consumer assessment relies on  
6929 available occupational data obtained under certain environmental conditions expected to be more  
6930 representative of a DIY consumer user scenario (no engineering controls, no PPE, residential garage).  
6931 However, the studies utilized still have uncertainties associated with the environment where the work  
6932 was done. In Blake et al. (2003), worker exposures were measured at a former automobile repair facility  
6933 which had an industrial sized and filtered exhaust fan unit to ventilate the building during testing while  
6934 all doors were closed. A residential garage is not expected to have a filtered exhaust fan installed and  
6935 operating during DIY consumer brake repair/replacement activities.

6937 The volume of a former automobile repair facility is considerably larger than a typical residential garage  
6938 and will have different air exchange rates. While this could raise some uncertainties related to the  
6939 applicability of the measured data to a DIY consumer user environment, the locations of the  
6940 measurements utilized for this evaluation minimize that uncertainty.

6941  
6942 There is some uncertainty associated with the length of time EPA assumes the brake repair/replacement  
6943 work takes. The EPA assumed it takes a DIY consumer user about three hours to complete brake  
6944 repair/replacement work. This is two times as long as a professional mechanic. While it is expected to  
6945 take a DIY consumer longer, it is also expected DIY consumer users who do their own brake  
6946 repair/replacement work would, over time, develop some expertise in completing the work as they  
6947 continue to do it every three years.

6948  
6949 There is also some uncertainty associated with the assumption that a bystander would remain within  
6950 three meters from the automobile on which the brake repair/replacement work is being conducted for the  
6951 entire three-hour period EPA assumes it takes the consumer user to complete the work. However,  
6952 considering a residential garage with the door closed is relatively close quarters for car repair work, it is  
6953 likely anyone observing (or learning) the brake repair/replacement work would not be able to stay much  
6954 further away from the car than three meters. Remaining within the garage for the entire three hours also  
6955 has some uncertainty, although it is expected anyone observing (or learning) the brake  
6956 repair/replacement work would remain for the entire duration of the work or would not be able to  
6957 observe (or learn) the task.

6958  
6959 While industry practices have drifted away from the use of compressed air to clean brake drums/pads,  
6960 no information was found in the literature indicating consumers have discontinued such work practices.  
6961 To consider potential consumer exposure to asbestos resulting from brake repair/replacement activities,  
6962 EPA uses data which included use of compressed air. However, EPA recognizes this may be a more  
6963 conservative estimate because use of compressed air typically could cause considerable dust/fibers to  
6964 become airborne if it is the only method used.

6965  
6966 There were no data identified through systematic review providing consumer specific monitoring for  
6967 UTV exhaust system gasket repair/replacement activities. Therefore, this evaluation utilized published  
6968 monitoring data obtained in an occupational setting, by professional mechanics, as a surrogate for  
6969 estimating consumer exposures associated with UTV gasket removal/replacement activities. There is  
6970 some uncertainty associated with the use of data from an occupational setting for a consumer  
6971 environment due to differences in building volumes, air exchange rates, available engineering controls,  
6972 and the potential use of PPE. As part of the literature review, EPA considered these differences and  
6973 utilized reasonably available information which was representative of the expected consumer  
6974 environment.

6975  
6976 There is some uncertainty associated with the use of an automobile exhaust system gasket  
6977 repair/replacement activity as a surrogate for UTV exhaust system gasket repair/replacement activity  
6978 due to expected differences in the gasket size, shape, and location. UTV engines and exhaust systems  
6979 are expected to be smaller than a full automobile engine and exhaust system, therefore the use of an  
6980 automobile exhaust system gasket repair may slightly overestimate exposure to the consumer. At the  
6981 same time, the smaller engine and exhaust system of a UTV could make it more difficult to access the  
6982 gaskets and clean the surfaces where the gaskets adhere therefore increasing the time needed to clean  
6983 and time of exposure resulting from cleaning the surfaces which could underestimate consumer  
6984 exposure.

6985

6986 There is some uncertainty associated with the assumption that UTV exhaust system gasket  
6987 repair/replacement activities would take a consumer a full three hours to complete. While there was no  
6988 published information found providing consumer specific lengths of time to complete a full  
6989 repair/replacement activity. The time needed for a DIY consumer to complete a full UTV exhaust  
6990 system gasket repair/replacement activity can vary depending on several factors including location of  
6991 gaskets, number of gaskets, size of gasket, and adherence once the system is opened up and the gasket  
6992 removed. Without published information, EPA assumes this work takes about three hours and therefore  
6993 utilized the three-hour TWA's to estimate risks for this evaluation.  
6994

6995 Finally, EPA has made some assumptions regarding both age at start of exposure and duration of  
6996 exposure for both the DIY users and bystanders for both the brake and UTV gasket scenarios. Realizing  
6997 there is uncertainty around these assumptions, specifically that they may over-estimate exposures, EPA  
6998 developed a sensitivity analysis approach specifically for the consumer exposure/risk analysis (see  
6999 appropriate part of Section 4.3.8 below) and also performed a sensitivity analysis using five different  
7000 scenarios (Appendix L).  
7001

#### 7002 4.3.5 Key Assumptions and Uncertainties in the Human Health IUR Derivation

7003 The analytical method used to measure exposures in the epidemiology studies is important in  
7004 understanding and interpreting the results as they were used to develop the IUR. As provided in more  
7005 detail in Section 3, the IUR for "current use" asbestos (i.e., chrysotile) is based solely on studies of PCM  
7006 measurement as TEM-based risk data are limited in the literature and the available TEM results for  
7007 chrysotile lack modeling results for mesothelioma. In TEM studies of NC and SC ([Loomis et al., 2010](#);  
7008 [Stayner et al., 2008](#)), models that fit PCM vs TEM were generally equivalent (about 2 AIC units),  
7009 indicating that fit of PCM is similar to the fit of TEM (for these two cohorts), providing confidence in  
7010 those PCM measurements for SC and NC. Given that confidence in the PCM data and the large number  
7011 of analytical measurements, exposure uncertainty is considered low in the cohorts used for IUR  
7012 derivation.  
7013

7014 There is evidence that other cancer endpoints may also be associated with exposure to the commercial  
7015 forms of asbestos. IARC concluded that there was sufficient evidence in humans that commercial  
7016 asbestos (chrysotile, crocidolite, amosite, tremolite, actinolite, and anthophyllite) was causally  
7017 associated with lung cancer and mesothelioma, as well as cancer of the larynx and the ovary ([Straif et  
7018 al., 2009](#)). The lack of sufficient numbers of workers to estimate risks of ovarian and laryngeal cancer is  
7019 a downward bias leading to lower IUR estimates in an overall cancer health assessment; however, the  
7020 selected IUR was chosen to compensate for this bias.  
7021

7022 The endpoint for both mesothelioma and lung cancer was mortality, not incidence. Incidence data are  
7023 not available for any of the cohorts. Nevertheless, mortality rates approximate incidence rates for  
7024 cancers such as lung cancer and mesothelioma because the survival time between cancer incidence and  
7025 cancer mortality is short. Therefore, while the absolute rates of lung cancer mortality at follow-up may  
7026 underestimate the rates of lung cancer incidence, the uncertainty for lung cancer is low. For  
7027 mesothelioma, the median length of survival with mesothelioma is less than 1 year for males, with less  
7028 than 20% surviving after 2-years and less than 6% surviving after 5-years. Because the mesothelioma  
7029 model is absolute risk, this leads to an under-ascertainment on mesothelioma risk, however, the selected  
7030 IUR was chosen to compensate this bias.

7031  
7032 The IUR only characterizes cancer risk. It does not include any risks that may be associated with non-  
7033 cancer health effects. Pleural and pulmonary effects from asbestos exposure (e.g., asbestosis and pleural  
7034 thickening) are well documented ([U.S. EPA, 1988b](#)), although there is no reference concentration (RfC)  
7035 for these non-cancer health effects specifically for chrysotile. During the Problem Formulation step for  
7036 TSCA's risk evaluation of asbestos, EPA considered risks of 1 cancer per 1,000,000 people, and at that  
7037 level of risk, cancer was considered to be a risk driver for the overall health risk of asbestos. The IRIS  
7038 IUR for general asbestos is 0.23 per fiber/cc. The IRIS assessment of Libby amphibole asbestos ([U.S.  
7039 EPA, 2014b](#)) derived a RfC for non-cancer health effects, and at that concentration (9 E-5 fibers/cc), the  
7040 risk of cancer for general asbestos fibers (including chrysotile, actinolite, amosite, anthophyllite,  
7041 crocidolite, and tremolite) was 2 E-5 [IUR\*RfC = (0.23 per fiber/cc)\*(9 E-5 fibers/cc)]. Thus, at a target  
7042 risk of 1 cancer per 1,000,000 people (1E-6), the existing EPA general asbestos cancer toxicity value  
7043 appeared to be the clear risk driver as meeting that target risk would result in lower non-cancer risks  
7044 than at the RfC.

7045  
7046 However, in occupational settings, with workers and ONUs exposed in a workplace, EPA considered  
7047 risks of cancer per 10,000 people. At this risk level, if the non-cancer effects of chrysotile are similar to  
7048 Libby amphibole asbestos, the non-cancer effects of chrysotile are likely to contribute additional risk to  
7049 the overall health risk of asbestos beyond the risk of cancer. Thus, the overall health risks of asbestos  
7050 based on cancer alone are underestimated.

7051  
7052 The POD associated with the only non-cancer toxicity value is 0.026 fibers/cc ([U.S. EPA,  
7053 2014b](#)). Although the non-cancer toxicity of chrysotile may be different from Libby amphibole asbestos,  
7054 there is uncertainty that the IUR for chrysotile asbestos may not fully encompasses the health risks  
7055 associated with chrysotile exposure. Several of the COU-related exposures evaluated for human health  
7056 risks in section 4.2 are at or greater than the POD for non-cancer effects associated with exposure to  
7057 Libby amphibole asbestos.

#### 7059 **4.3.6 Key Assumptions and Uncertainties in the Cancer Risk Values**

7060 Although direct comparison of cancer slopes for PCM and TEM fibers is impossible because different  
7061 counting rules for these methods result in qualitatively and quantitatively different estimates of asbestos  
7062 exposure, comparing the fit of models based on different analytical methods is possible. In TEM studies  
7063 of NC and SC ([Loomis et al., 2010](#); [Stayner et al., 2008](#)), models that fit PCM vs TEM were generally  
7064 equivalent (about 2 AIC units), indicating that fit of PCM is similar to the fit of TEM (for these two  
7065 cohorts), providing confidence in those PCM measurements for SC and NC, whose data is the basis for  
7066 chrysotile IUR.

7067  
7068 Another source of uncertainty in the exposure assessment is that early measurements of asbestos fiber  
7069 concentrations were based on an exposure assessment method (midget impinger) that estimated the  
7070 combined mass of fibers and dust, rather than on counting asbestos fibers. The best available  
7071 methodology for conversion of mass measurements to fiber counts is to use paired and concurrent  
7072 sampling by both methods to develop factors to convert the mass measurements to estimated fiber  
7073 counts for specific operations. There is uncertainty in these conversion factors, but it is minimized in the  
7074 studies of SC and NC chrysotile textile workers due to the availability of an extensive database of paired  
7075 and concurrent samples and the ability to develop operation-specific conversion factors. Uncertainty in  
7076 the estimation of these conversion factors and their application to estimate chrysotile exposures will not  
7077 be differential with respect to disease.

Given the high confidence in the PCM data and the large number of analytical measurements, exposure uncertainty is overall low in the SC and NC cohorts, as very high-quality exposure estimates are available for both cohorts. Statistical error in estimating exposure levels is random and not differential with respect to disease. Therefore, to the extent that such error exists, it is likely to produce either no bias or bias toward the null under most circumstances (e.g., (Kim et al., 2011; Armstrong, 1998)).

Epidemiologic studies are observational and as such are potentially subject to confounding and selection biases. 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. In particular, the NC and SC studies of textile workers, which were chosen as the most informative studies, did not have this information. However, the bias related to this inability to control for smoking is believed to be small because the exposure-response analyses for lung cancer were based on internal comparisons and for both studies the regression models included birth cohort, thus introducing some control for the changing smoking rates over time. It is unlikely that smoking rates among workers in these facilities differed substantially enough with respect to their cumulative chrysotile exposures to induce important confounding in risk estimates for lung cancer. Mesothelioma is not related to smoking and thus smoking could not be a confounder for mesothelioma.

For the purpose of combining risks, it is assumed that the unit risks of mesothelioma and lung cancer mortality are normally distributed. Because risks were derived from a large epidemiological cohort, this is a reasonable assumption supported by the statistical theory and the independence assumption has been investigated and found a reasonable assumption (U.S. EPA, 2014c).

#### 4.3.7 Confidence in the Human Health Risk Estimations

##### Workers/Occupational Non-Users

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. If a worker changes jobs during their career and are no longer exposed to asbestos, this may overestimate exposures. However, if the worker stays employed after age 56, it would underestimate exposures.

The concentration-response functions on which the chrysotile asbestos IUR is based varies as a function of time since first exposure. Consequently, estimates of cancer risk depend not only on exposure concentration, frequency and duration, but also on age at first exposure. To approximate the impact of different assumptions for occupational exposures, Table 4-49 can be used to understand what percentage of the risk in the baseline occupational exposure scenario remains for different ages at first exposure and different durations of exposure

**Table 4-49. Ratios of risks for alternative exposure scenarios using scenario-specific partial lifetime IURs from Appendix K by age at first exposure and duration of exposure compared to baseline occupational exposure scenarios (baseline scenario: first exposure at 16 years for 40 years duration)**

	Duration of exposure (years)
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Age at first exposure (years)	20	40
16	$0.0499/0.0707 = 0.71$	$0.0707/0.0707 = 1$
20	$0.0416/0.0707 = 0.59$	$0.0591/0.0707 = 0.84$
30	$0.0267/0.0707 = 0.38$	$0.0374/0.0707 = 0.53$

Other occupational exposure scenario can be evaluated by selecting different values for the age at first exposure and the duration of exposure from the table of partial lifetime IUR values in Appendix K.

Exposures for ONUs can vary substantially. Most data sources do not sufficiently describe the proximity of these employees to the exposure source. As such, exposure levels for the ONU category will vary depending on the work activity. It is unknown whether these uncertainties overestimate or underestimate exposures.

Cancer risks were indicated for all of the worker COUs and most of the consumer/bystander COUs. If additional factors were not considered in the RE, such as exposures from other sources (e.g., legacy asbestos sources), the risks could be underestimated. Legacy asbestos is not evaluated in the RE at this time, but EPA will consider legacy uses and associated disposal in subsequent supplemental documents.

In addition, several subpopulations (e.g., smokers, genetically predisposed individuals, COU workers who change their own asbestos-containing brakes, etc.) may be more susceptible than others to health effects resulting from exposure to asbestos. These conditions are discussed in more detail for potentially exposed or susceptible subpopulations and aggregate exposures in Section 4.4 and Section 4.5.

Consumer DIY/Bystanders

Similarly, for consumers/bystanders risks could be under- or over-estimated for their COU. Unlike occupational scenarios, there are no standard assumptions for consumers and bystanders, EPA conducted sensitivity analyses to evaluate some alternative scenarios for consumers/bystanders as described below.

For consumers (see Table 4-48) EPA considered age at first exposure of 16 years with duration of exposure 62 years and for bystanders EPA considered age at first exposure of 0 years with lifetime duration (78 years). To evaluate sensitivity to these assumptions, EPA conducted multiple sensitivity analyses assuming that duration of exposure as short as 10 years with different ages of first exposure. Tables 4-50 and 4-51 below show the different scenarios covered in the sensitivity analysis and the associated adjustment factor that may be used to calculate a different risk number. In Table 4-50, DIY exposures with different ages at start of exposure (16, 20 or 30 years old) are paired with different durations of exposure (20, 40 or 62) and Table 4-51 shows the same for bystanders (age at start is always zero but the three exposure durations are 20, 40 and 78). All analyses are presented in Appendix L and show that using the ratios in both Tables 4-49 and 4-50 does not change the overall risk picture in almost all scenarios (see Table 4-51).

**Table 4-50. Ratios of risks for alternative exposure scenarios using scenario-specific partial lifetime IURs from Appendix K by age at first exposure and duration of exposure compared to baseline consumer DIY exposure scenarios (baseline scenario: first exposure at 16 years for 62 years duration)**

	Duration of exposure (years)
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Age at first exposure (years)	20	40	62
16	0.0499/0.0768 = 0.65	0.0707/0.0768 = 0.92	0.0768/0.0768 = 1
20	0.0416/0.0768 = 0.54	0.0591/0.0768 = 0.77	-
30	0.0267/0.0768 = 0.35	0.0374/0.0768 = 0.49	-

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**Table 4-51. Ratios of risks for alternative exposure scenarios using scenario-specific partial lifetime IURs from Appendix K by age at first exposure and duration of exposure compared to baseline consumer bystander exposure scenarios (baseline scenario: first exposure at 0 years for 78 years duration)**

Age at first exposure (years)	Duration of exposure (years)		
	20	40	78
0	0.101/0.16 = 0.63	0.144/0.16 = 0.90	0.16/0.16 = 1

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Table 4-52 provides a summary of the detailed analyses in Appendix L. These sensitivity analyses show that in four of the five scenario pairings, only one of 24 possible scenarios changed from exceeding the benchmark cancer risk level of  $1 \times 10^{-6}$  to no exceedance (DIY user, brake repair outdoors, 30 minutes/day, high-end only). In the fifth scenario (Sensitivity Analysis 2), there was no change in any of the 24 scenarios. All analyses are in Appendix L.

**Table 4-52. Results of Sensitivity Analysis of Exposure Assumptions for Consumer DIY/Bystander Episodic Exposure Scenarios**

Sensitivity Analysis <sup>1</sup>	DIY (age at start and age at end of duration)	Bystander (age at start and age at end of duration)	Change in Risk from Exceedance to No Exceedance	Scenario Affected
Baseline	16-78	0-78	None	17/24 Exceed Benchmarks
1	16-36	0-20	1/24	DIY user, Brake repair, 30 min/day, high-end
2	20-60	0-40	0/24	None
3	20-40	0-40	1/24	DIY user, Brake repair, 30 min/day, high-end
4	30-70	0-40	1/24	DIY user, Brake repair, 30 min/day, high-end

5	30-50	0-20	1/24	DIY user, Brake repair, 30 min/day, high-end
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<sup>1</sup> Includes all brake repair/replacement and gasket repair replacement scenarios – a total of 24. See Table 4-45

Assumptions About Bystanders

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.

The total number of respondents to the survey question on time spent in the garage was 193 and the minimum and maximum reported times were one minute and 790 minutes (~13 hours). Again, these respondents are “doers”, defined as people who reported being in that location (i.e., the garage). In this analysis, it was assumed that the 50<sup>th</sup> percentile would represent a central tendency estimate for being present in the garage (one hour/day) and the 95<sup>th</sup> percentile would represent a high-end estimate for being present in the garage (8 hours).

EPA understands that a bystander in this exposure situation (DIY automotive and UTV repair) is most likely to be a family member (minor or adult relative) with repeated access to the garage used to repair vehicles. As a familial bystander, and not a neighbor or someone visiting, EPA considered that these bystanders would have similar exposures to the garage, and thus to any chrysotile fibers in the same garage environment as the DIY user. EPA used the same median time of one hour per day as the bystander’s estimated central tendency and the same estimate of high end exposures. EPA noted that the younger doers appear to spend somewhat more time in the garage (EFH Table 16-16). In the same table of time spent per day in the garage, some data on doers is shown for ages 1-17 years (children) which can be aggregated to find the mean time spent in a garage. The mean for these children is 77 minutes per day based on 22 young doers, which is similar to the one hour median based on all 193 doers. EPA also noted that male doers had a median of 94 minutes compared to female doers who had a median of 30 minutes per day in the garage. It is possible that familial bystanders are unlike the DIY users and spend little time in the garage. If this were true, then with little or no time spent in the garage, their risks would be limited.

Finally, as part of the sensitivity analysis, understanding that a bystander in a doer family may spend somewhat less time in the garage than the 50<sup>th</sup> percentile time of one hour (60 minutes/day), Table 4-53 below shows the data available in the Exposure Factors Handbook that present other percentiles broken down by age and gender. In its original analysis, EPA used 60 minutes/day. If 10 minutes/day were used for the bystander and in keeping with deriving a risk estimate following a single brake or gasket change and a time-in-residence of only 10 years, the calculated risk values would be:

At 10 minutes/day in the garage following a single brake change and the next 10 years in the house, the by-stander risks would be 6.9 E-8 for the central tendency and 1.6 E-7 for the high-end estimates.

At 10 minutes/day in the garage following a single UTV gasket change and the next 10 years in the house, the by-stander risks would be 6.4 E-8 for the central tendency and 1.6 E-7 for the high-end estimates.

**Table 4-53. Time Spent (minutes/day) in Garage, Doers Only (Taken from Table 16-16 in EFH, 2011)**

Gender and Age Range	Percentiles in the Distribution of Survey Respondents				
	5 <sup>th</sup>	25 <sup>th</sup>	50 <sup>th</sup>	75 <sup>th</sup>	95 <sup>th</sup>
All ages	5	20	60	150	480
Men	10	30	94	183	518
Women	5	15	30	120	240
1-4 yrs old	15	52	100	115	120
5 to 11	10	25	30	120	165
12-17	10	20	51	148	240

Potential Number of Impacted Individuals

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.

The following text accompanies the estimates presented in Table 4-54:

*Chlor-Alkali Workers and ONUs*

There is a total of 3,050 employees at the 15 chlor-alkali plants we have identified as using diaphragms; with approximately 75-148 potentially exposed to asbestos during various activities associated with constructing, using and deconstructing asbestos diaphragms. Subtracting the 75 to 148 workers potentially exposed to asbestos results in approximately 2,900 to 3,000 other employees who work at the same or adjoining plant. This is an upper bound estimate of the number of ONUs and only an unknown subset of these workers may be ONUs. EPA has low certainty in this number because some of these sites are very large and make different products in different parts of the facility (one site is 1,100 acres and has 1,300 employees). Thus, this approach may overestimate the number of ONUs for asbestos diaphragms.

*Sheet Gaskets – Stamping (Workers and ONUs)*

EPA found only two gasket sampling sites handling asbestos containing sheet gasket; one worker and two ONUs per site. However, there may be more gasket stamping sites processing asbestos containing sheet gasket in US. Thus, the uncertainty in this number of impacted individuals is high.

*Sheet Gaskets – Use (Workers and ONUs)*

The Bureau of Labor Statistics 2016 data for the NAICS code 325180 (Other Basic Inorganic Chemical Manufacturing) indicates an industry-wide aggregate average of 25 directly exposed workers per facility and 13 ONUs per facility. The total number of use sites is unknown.

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*Oilfield Brake Blocks (Workers and ONUs)*

According to 2016 Occupational Employment Statistics data from the Bureau of Labor Statistics (BLS) and 2015 data from the U.S. Census' Statistics of U.S. Businesses. EPA used BLS and Census data for three NAICS codes: 211111, Crude Petroleum and Natural Gas Extraction; 213111, Drilling Oil and Gas Wells; and 213112, Support Activities for Oil and Gas Operations, there are up to 61,695 workers and 66,108 ONU. See Table 2-12 for the breakdown by each category. It is not known how many of these workers are exposed to asbestos.

*Aftermarket Automotic Brakes/Linings/Clutches (Workers and ONUs)*

EPA considers the best current estimate of this worker population to be from the Bureau of Labor Statistics, which estimates that 749,900 workers in the United States were employed as automotive service technicians and mechanics in 2016 ([U.S. BLS, 2019](#)); see Section 2.3.1.7 for more details. This includes workers at automotive repair and maintenance shops, automobile dealers, gasoline stations, and automotive parts and accessories stores. ONU exposures associated with automotive repair work are expected to occur because automotive repair and maintenance tasks often take place in large open bays with multiple concurrent activities. EPA did not locate published estimates for the number of ONUs for this COU. However, consistent with the industry profile statistics from OSHA's 1994 rulemaking (see Section 2.3.1.7), EPA assumes that automotive repair establishments, on average, have two workers who perform automotive repair activities. Accordingly, EPA estimates that this COU has 749,900 ONUs.

*UTV Sheet Gaskets (Workers and ONUs)*

Based on Bureau of Labor Statistics and several assumptions detailed in section 2.3.1.9, EPA estimate 1,500 workers for UTV service technicians and mechanics. It is not known how many of them service and/or repair UTV with asbestos containing gasket.

*Aftermarket Automotic Brakes/Linings/Clutches (Consumers/DIY/Bystanders)*

According to the Census's American Community Survey, 108,357,503 occupied housing units have at least one vehicle available. Of these, 39,472,759 (36%) have one vehicle available, 44,402,282 (41%) have two vehicles available, and 24,482,462 (23%) have three or more vehicles available.<sup>24</sup>

According to a 2001 market research study by the Automotive Aftermarket Industry Association ("The Aftermarket Consumer: Do-it-Yourself or Do-it-For-Me"), nearly half of all U.S. households contain at least one automotive DIYer.<sup>25</sup> While some households may contain more than one automotive DIYer, EPA assumes that the number of automotive DIYers is 50% of the number of households with an automobile.

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<sup>24</sup> American Fact Finder, *2013-2017 American Community Survey 5-Year Estimates*, DP04, U.S. Census Bureau.

<sup>25</sup> The Auto Channel, *AAIA REPORT: Percentage of Auto DIYers Unchanged*, 07-03-01. <https://www.theautochannel.com/news/2001/07/03/024549.html>

7305 According to a 2014 online survey of 2,843 consumers conducted by AutoPartsWarehouse.com, 63% of  
 7306 male DIYers and 35% of female DIYers responded that they replace brake pads. The survey respondents  
 7307 were 85% male and 15% female.<sup>26</sup>  
 7308

7309 Combining this data, (108,357,503 households with at least one vehicle available) x (50% of households  
 7310 contain an automotive DIYer) x ((85% of DIYers are male) x (63% of male DIYers replace brake pads)  
 7311 + (15% of DIYers are female) x (35% of female DIYers replace brake pads)) = 31,857,106 automotive  
 7312 DIYers replace brake pads.  
 7313

7314 EPA estimates that brakes are replaced about once every three years.<sup>27</sup> Combining the Census ACS data  
 7315 on the distribution of vehicles per household; the estimate that 31,857,106 automotive DIYers replace  
 7316 brake pads; and the estimate that brakes are replaced once every three years, results in an estimate that  
 7317 that there are approximately 20 million DIY brake jobs per year.  
 7318

7319 The number of asbestos-containing brakes sold in the aftermarket is not known.  
 7320

7321 *COUs for Which No Estimates May be Made*

7322 EPA could develop an reasonable estimate of potentially impacted individuals for two COUs: other  
 7323 vehicle friction products (workers/ONUs) and UTV gasket replacement/repair (DIY/bystanders).  
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**Table 4-54. Summary of Estimated Number of Exposed Workers and DIY Consumers<sup>a</sup>.**

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) <sup>(c)</sup>	<66,108 (total; number in vicinity of asbestos Unknown) <sup>(c)</sup>	-	-

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<sup>26</sup> *Consumers Continue to Embrace DIY Auto Repair, Attempting More Difficult Jobs and Report Saving Big Bucks*, September 30, 2014 by Auto Parts Warehouse <https://www.autopartswarehouse.com/blog/2014/09/consumers-continue-embrace-diy-auto-repair-attempting-difficult-jobs-report-saving-big-bucks/>

<sup>27</sup> Brakes in cars and small trucks are estimated to require replacement approximately every 35,000 to 60,000 miles (Advance Auto Parts, website accessed on November 12, 2018). The three-year timeline is derived by assuming the need to replace brakes every 35,000 miles, and an average number of annual miles driven per driver in the U.S. of 13,476 miles/year (U.S. DOT, 2018).

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 <sup>(d)</sup> )	Unknown	Unknown	Unknown

<sup>a</sup> See Text for details.

## 4.4 Other Risk-Related Considerations

### 4.4.1 Potentially Exposed or Susceptible Subpopulations

EPA identified workers, ONUs, consumers, and bystanders as potentially exposed populations. EPA provided risk estimates for workers and ONUs at both central tendency and high-end exposure levels for most COUs. EPA determined that bystanders may include lifestages of any age.

For inhalation exposures, risk estimates did not differ between genders or across lifestages because both exposures and inhalation hazard values are expressed as an air concentration. EPA expects that variability in human physiological factors (e.g., breathing rate, body weight, tidal volume) could affect the internal delivered concentration or dose of asbestos.

Workers exposed to asbestos in workplace air, especially if they work directly with asbestos, are most susceptible to the health effects associated with asbestos. Some workers not associated with the COU may experience higher exposures to asbestos, such as, but not limited to, asbestos removal workers, firefighters, demolition workers and construction workers ([Landrigan et al., 2004](#)); and these populations will be considered when EPA evaluates legacy uses in subsequent supplemental documents. Although it is clear that the health risks from asbestos exposure increase with heavier exposure and longer exposure time, investigators have found asbestos-related diseases in individuals with only brief exposures. Generally, those who develop asbestos-related diseases show no signs of illness for a long time after exposure ([ATSDR, 2001a](#)).

A source of variability in susceptibility between people is smoking history or the degree of exposure to other risk factors with which asbestos interacts. In addition, 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](#)). Appendix J of this RE illustrates this point in the IUR values for less than lifetime COUs. For example, the IUR for a one-year old child first exposed to chrysotile asbestos for 40 years is 1.31 E-1 while the IUR for a 20-year old first exposed to asbestos for 40 years is 5.4 E-2. Using the central tendency bystander exposure value of 0.032 f/cc, the resulting risk estimates are 1.7 x E-4 and 7.2 x E-5, respectively. There is also some evidence of genetic predisposition for mesothelioma related to having a germline mutation in BAP1 ([Testa et al., 2011](#)).

Finally, from an environmental receptor perspective, although there is evidence of reproductive and developmental effects in controlled laboratory settings following asbestos exposure to aquatic

7366 organisms. The likelihood these effects would occur in the environment is low due to the lack of  
7367 environmental releases of asbestos to surface water from the COUs in this draft risk evaluation.

#### 7368 **4.4.2 Aggregate and Sentinel Exposures**

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7369 Section 2605(b)(4)(F)(ii) of TSCA requires the EPA, as a part of the risk evaluation, to describe whether  
7370 aggregate or sentinel exposures under the conditions of use were considered and the basis for their  
7371 consideration. The EPA has defined aggregate exposure as “the combined exposures to an individual  
7372 from a single chemical substance across multiple routes and across multiple pathways (40 CFR §  
7373 702.33).”

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7375 Aggregate exposures for asbestos were not assessed by routes of exposure, since only inhalation  
7376 exposure was evaluated in the RE. EPA chose not to employ simple additivity of exposure pathways at  
7377 this time within a condition of use because of the uncertainties present in the current exposure estimation  
7378 procedures. This lack of aggregation may lead to an underestimate of exposure but based on physical  
7379 chemical properties the majority of the exposure pathway is believed to be from inhalation exposures.  
7380

7381 Pathways of exposure were not combined in this RE. Although it is possible that workers exposed to  
7382 asbestos might also be exposed as consumers (e.g., by changing brakes at home), the number of  
7383 workers/uses is potentially small. The individual risk estimates already indicate risk; aggregating the  
7384 pathways would increase the risk.

7385  
7386 In addition, the potential for exposure to legacy asbestos for any populations or subpopulation, due to  
7387 activities such as home or building renovations, as well as occupational or consumer exposures  
7388 identified in this RE, is possible. Legacy asbestos exposure is not considered in the RE at this time  
7389 which could underestimate exposures and thus, risks. This is discussed as an uncertainty in Section 4.3.8  
7390 of the RE. EPA will consider legacy uses and associated disposal in subsequent supplemental  
7391 documents.  
7392

7393 The EPA defines sentinel exposure as “*the exposure to a single chemical substance that represents the*  
7394 *plausible upper bound of exposure relative to all other exposures within a broad category of similar or*  
7395 *related exposures* (40 CFR § 702.33).” In terms of this risk evaluation, the EPA considered sentinel  
7396 exposure the highest exposure given the details of the conditions of use and the potential exposure  
7397 scenarios. EPA considered sentinel exposure for asbestos in the form of a high-end level scenario for  
7398 occupational exposure resulting from inhalation exposures for each COU; sentinel exposures for  
7399 workers are the high-end 8-hour exposures for sheet gasket stamping without any PPE.  
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## 7402 **4.5 Risk Conclusions**

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### 7403 **4.5.1 Environmental Risk Conclusions**

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7404 Based on the reasonably available information in the published literature, provided by industries using  
7405 asbestos, and reported in EPA databases, there is minimal or no releases of asbestos to surface water and  
7406 sediments associated with the COUs in this risk evaluation. Therefore, EPA concludes there is no  
7407 unreasonable risk to aquatic or sediment-dwelling environmental organisms. In addition, terrestrial  
7408 pathways, including biosolids, were excluded from analysis at the PF stage.

4.5.2 Human Health Risk Conclusions to Workers

Table 4-57 provides a summary of risk estimates for workers and ONUs. For workers in all six COUs identified in this risk evaluation, cancer risks were exceeded for all central tendency and high-end exposures (chlor-alkali industry, stamping of sheet gaskets, use of sheet gaskets in the chemical production industry, oil field brake blocks, aftermarket auto brakes/other vehicle friction products installation and UTV gasket repair). In addition, for ONUs, cancer risks were exceeded for high-end exposure estimates in all of the COUs. For central tendency exposure estimates for ONUs, cancer risks were exceeded for sheet gasket use, oilfield brake block use, and UTV gasket repair.

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.

**Table 4-55. Summary of Risk Estimates for Inhalation Exposures to Workers and ONUs by COU (Cancer benchmark is 10<sup>-4</sup>)**

Life Cycle Stage/Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Duration and Level	Cancer Risk Estimates (before applying PPE)	Cancer Risk Estimates (with APF=10 <sup>c</sup> )	Cancer Risk Estimates (with APF=25 <sup>c</sup> )
Import – Raw asbestos	Diaphragms for chlor-alkali industry	Section 2.3.1.3	Worker	Central Tendency (8-hr)	1.2 E-4	1.2 E-5	4.8 E-6
				High-end (8-hr)	8.4 E-4	8.4 E-5	3.4 E-5
				Central Tendency short term	1.5 E-4 1.1 E-4 <sup>a</sup>	1.5 E-5 <sup>d</sup>	6.0 E-6 <sup>b</sup>
				High-end short term	1.3 E-3 8.1 E-4 <sup>a</sup>	9.9 E-5 <sup>d</sup>	5.2 E-5 <sup>b</sup>
			ONU	Central (8-hr)	5.8 E-5	N/A	N/A
				High (8-hr)	1.9 E-4	N/A	N/A
				Central (w/ short-term)	--	N/A	N/A
				High (w/ short-term)	--	N/A	N/A
	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 <sup>e</sup>	1.4 E-5 <sup>f</sup>



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Life Cycle Stage/Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Duration and Level	Cancer Risk Estimates (before applying PPE)	Cancer Risk Estimates (with APF=10 <sup>e</sup> )	Cancer Risk Estimates (with APF=25 <sup>e</sup> )	
Import of asbestos products			ONU	High (w/ short-term)	<b>1.4 E-3</b>	<b>1.4 E-4<sup>e</sup></b>	5.6 E-5 <sup>f</sup>	
				Central (8-hr)	5.6 E-5	N/A	N/A	
				High (8-hr)	<b>2.3 E-4</b>	N/A	N/A	
				Central (w/ short-term)	5.6 E-5	N/A	N/A	
				High (w/ short-term)	<b>2.3 E-4</b>	N/A	N/A	
	Asbestos Sheet Gaskets – use (repair/replacement in TiO <sub>2</sub> industry)	Section 2.3.1.5	Worker	Central Tendency (8-hr)	<b>6.0 E-4</b>	6.0 E-5	2.4 E-5	
				High-end (8-hr)	<b>2.2 E-3</b>	<b>2.2 E-4</b>	8.8 E-5	
				ONU	Central (8-hr)	<b>1.2 E-4</b>	N/A	N/A
				High (8-hr)	<b>3.7 E-4</b>	N/A	N/A	
	Oil Field Brake Blocks	Section 2.3.1.6	Worker	Central Tendency (8-hr)	<b>7.0 E-4</b>	7.0 E-5	2.8 E-5	
				ONU	Central Tendency (8-hr)	<b>4.6 E-4</b>	N/A	N/A
	Aftermarket Auto Brakes	Section 2.3.1.7	Worker	Central Tendency (8-hr)	<b>1.4 E-4</b>	1.4 E-5	5.6 E-6	
				High-end (8-hr)	<b>2.2 E-3</b>	<b>2.2 E-4</b>	8.8 E-5	
				Central (w/ short-term)	<b>1.4 E-4</b>	1.4 E-5 <sup>e</sup>	5.6 E-6 <sup>f</sup>	
				High (w/ short-term)	<b>3.3 E-3</b>	<b>3.3 E-4<sup>e</sup></b>	<b>1.3 E-4<sup>f</sup></b>	
				ONU	Central (8-hr)	1.6 E-5	N/A	N/A
					High (8-hr)	<b>2.6 E-4</b>	N/A	N/A
Central (w/ short-term)					1.6 E-5	N/A	N/A	
High (w/ short-term)					<b>2.6 E-4</b>	N/A	N/A	

**PEER REVIEW DRAFT. DO NOT CITE OR QUOTE**

Life Cycle Stage/Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Duration and Level	Cancer Risk Estimates (before applying PPE)	Cancer Risk Estimates (with APF=10 <sup>e</sup> )	Cancer Risk Estimates (with APF=25 <sup>e</sup> )
	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 <sup>e</sup>	5.6 E-6 <sup>f</sup>
				High (w/ short-term)	3.3 E-3	3.3 E-4 <sup>e</sup>	1.3 E-4 <sup>f</sup>
			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 Gaskets – Utility Vehicles	Section 2.3.1.9	Worker	Central Tendency (8-hr)	5.6 E-4	5.6 E-5	2.2 E-5
				High-end (8-hr)	1.5 E-3	1.5 E-4	6.0 E-5
			ONU	Central (8-hr)	1.2 E-4	N/A	N/A
				High (8-hr)	3.5 E-4	N/A	N/A

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N/A: Not Assessed; ONUs are not assumed to wear respirators  
<sup>a</sup>No APF applied for 7.5 hours, APF of 25 applied for 30 minutes.  
<sup>b</sup>APF 25 applied for both 30 mins and 7.5 hours  
<sup>c</sup> As shown in Table 4-3, EPA has information suggesting use of respirators for two COUs (chlor-alkali: APF of 10 or 25; and sheet gasket use: APF of 10 only). Application of all other APFs is hypothetical.  
<sup>d</sup> APF 25 for 30 minutes, APF 10 for 7.5 hours  
<sup>e</sup> APF 10 for 30 minutes, APF 10 for 7.5 hours  
<sup>f</sup> APF 25 for 30 minutes, APF 25 for 7.5 hours

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**4.5.3 Human Health Risk Conclusions to Consumers**

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.

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**Table 4-56. Summary of Risk Estimates for Inhalation Exposures to Consumers and Bystanders by COU (Cancer benchmark is 10<sup>-6</sup>)**

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

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<b>Life Cycle Stage/Category</b>	<b>Subcategory</b>	<b>Consumer Exposure Scenario</b>	<b>Population</b>	<b>Exposure Duration and Level</b>	<b>Cancer Risk Estimates</b>
	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	<b>2.4 E-5</b>
				High-end	<b>6.1 E-5</b>
	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	<b>3.0 E-6</b>
				High end	<b>8.3 E-6</b>
			Bystander	Central Tendency	<b>3.08 E-6</b>
				High-end	<b>7.16 E-6</b>

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## 5 Risk Determination

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### 5.1 Unreasonable Risk

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#### 5.1.1 Overview

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In each risk evaluation under TSCA § 6(b), EPA determines whether a chemical substance presents an unreasonable risk of injury to health or the environment, under the conditions of use. The determination does not consider costs or other non-risk factors. In making this determination, EPA considers relevant risk-related factors, including, but not limited to: the effects of the chemical substance on health and human exposure to such substance under the conditions of use (including cancer and non-cancer risks); the effects of the chemical substance on the environment and environmental exposure under the conditions of use; the population exposed (including any potentially exposed or susceptible subpopulations); the severity of hazard (including the nature of the hazard, the irreversibility of the hazard); and uncertainties. EPA 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. This approach is in keeping with the Agency's final rule, *Procedures for Chemical Risk Evaluation Under the Amended Toxic Substances Control Act* (82 FR 33726).

Under TSCA, conditions of use are defined as the circumstances, as determined by the Administrator, under which the substance is intended, known, or reasonably foreseen to be manufactured, processed, distributed in commerce, used, or disposed of (TSCA §3(4)).

An unreasonable risk may be indicated when health risks under the conditions of use are identified by comparing the estimated risks with the risk benchmarks and where the risks affect the general population or certain potentially exposed or susceptible subpopulations (PESS), such as consumers. For other PESS, such as workers, an unreasonable risk may be indicated when risks are not adequately addressed through expected use of workplace practices and exposure controls, including engineering controls or use of personal protective equipment (PPE). The risk evaluation for asbestos evaluated the cancer risk to workers and occupational non-users and consumers and bystanders from inhalation exposures only, and in this risk determination of asbestos, respirator PPE (where present) and its effect on mitigating inhalation exposure was considered.

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  $5 \times 10^{-2}$ , which is greater than the benchmarks of  $1 \times 10^{-4}$  to  $1 \times 10^{-6}$ . 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.

The degree of uncertainty surrounding cancer risk is a factor in determining whether or not unreasonable risk is present. Where uncertainty is low and EPA has high confidence in the hazard and exposure characterizations (for example, the basis for the characterizations is measured or monitoring data or a

robust model and the hazards identified for risk estimation are relevant for conditions of use), the Agency has a higher degree of confidence in its risk determination. EPA may also consider other risk factors, such as severity of endpoint, reversibility of effect, or exposure-related considerations such as magnitude or number of exposures, in determining that the risks are unreasonable under the conditions of use. Where EPA has made assumptions in the scientific evaluation and whether or not those assumptions are protective, will also be a consideration. Additionally, EPA considers the central tendency and high-end scenarios when determining unreasonable risk. High-end risk estimates (e.g., 95th percentile) are generally intended to cover individuals or subpopulations with greater exposure, and central tendency risk estimates are generally estimates of average or typical exposure.

Conversely, EPA may make a no unreasonable risk determination for conditions of use where the substance's hazard and exposure potential, or where the risk-related factors described previously, lead EPA to determine that the risks are not unreasonable.

### 5.1.2 Risks to Human Health

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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.,  $1 \times 10^{-6}$  to  $1 \times 10^{-4}$  or also denoted as 1 E-6 to 1 E-4) depending on the subpopulation exposed. Generally, EPA considers benchmarks ranging from  $1 \times 10^{-6}$  to  $1 \times 10^{-4}$  as appropriate for the general population, consumer users, and non-occupational PESS.<sup>28</sup>

For the purposes of this risk determination, EPA uses  $1 \times 10^{-6}$  as the benchmark for consumers (e.g., do-it-yourself mechanics) and bystanders. In addition, consistent with the 2017 NIOSH guidance,<sup>29</sup> EPA uses  $1 \times 10^{-4}$  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  $1 \times 10^{-4}$  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.

#### 5.1.2.1 Determining Cancer Risks

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**General population:** In this risk evaluation for asbestos, EPA did not evaluate hazards or exposures to the general population. Further, as part of the problem formulation for asbestos, EPA identified exposure

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<sup>28</sup> As an example, when EPA's Office of Water in 2017 updated the Human Health Benchmarks for Pesticides, the benchmark for a "theoretical upper-bound excess lifetime cancer risk" from pesticides in drinking water was identified as 1 in 1,000,000 to 1 in 10,000 over a lifetime of exposure (EPA. Human Health Benchmarks for Pesticides: Updated 2017 Technical Document. January 2017. <https://www.epa.gov/sites/production/files/2015-10/documents/hh-benchmarks-techdoc.pdf>). Similarly, EPA's approach under the Clean Air Act to evaluate residual risk and to develop standards is a two-step approach that includes a "presumptive limit on maximum individual lifetime [cancer] risk (MIR) of approximately 1 in 10 thousand" and consideration of whether emissions standards provide an ample margin of safety to protect public health "in consideration of all health information, including the number of persons at risk levels higher than approximately 1 in 1 million, as well as other relevant factors" (54 FR 38044, 38045, September 14, 1989).

<sup>29</sup> NIOSH (2016). Current intelligence bulletin 68: NIOSH chemical carcinogen policy, available at <https://www.cdc.gov/niosh/docs/2017-100/pdf/2017-100.pdf>.

7528 pathways under other environmental statutes, administered by EPA, which adequately assess and  
7529 effectively manage exposures and for which long-standing regulatory and analytical processes exist, i.e.,  
7530 the Clean Air Act (CAA), the Safe Drinking Water Act (SDWA), the Clean Water Act (CWA) and the  
7531 Resource Conservation and Recovery Act (RCRA). The Office of Chemical Safety and Pollution  
7532 Prevention works closely with the offices within EPA that administer and implement the regulatory  
7533 programs under these statutes. EPA believes that the TSCA risk evaluation should focus on those  
7534 exposure pathways associated with TSCA uses that are not subject to the regulatory regimes discussed  
7535 above because these pathways are likely to represent the greatest areas of concern to EPA. Because  
7536 stationary source releases of asbestos to ambient air are adequately assessed and any risks are effectively  
7537 managed when under the jurisdiction of the CAA, EPA did not evaluate emission pathways to ambient  
7538 air from commercial and industrial stationary sources or associated inhalation exposure of the general  
7539 population or terrestrial species in this TSCA evaluation. Based on the reasonably available information  
7540 in the published literature, provided by industries using asbestos, and reported in EPA databases, there is  
7541 no evidence of releases of asbestos to water associated with the conditions of use that EPA evaluated. As  
7542 such, EPA did not evaluate in the risk evaluation the surface water pathway for general population  
7543 exposures during or after land application of biosolids. Therefore, EPA did not evaluate hazards or  
7544 exposures to the general population in the risk evaluation, and there is no risk determination for the  
7545 general population.

### 7546 **5.1.3 Determining Environmental Risk**

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7547 As explained in this risk evaluation, after PF, EPA did not evaluate ecological receptors. EPA believes  
7548 there is low or no potential for environmental risk to aquatic receptors (including sediment-dwelling  
7549 organisms) from the COUs included in this risk evaluation because water releases associated with the  
7550 COUs are not expected and were not identified. The available information indicated that there were  
7551 surface water releases of asbestos; however, not all releases are subject to reporting (e.g., effluent  
7552 guidelines) or are applicable (e.g., friability). Based on the reasonably available information in the  
7553 published literature, provided by industries using asbestos, and reported in EPA databases, there is  
7554 minimal or no releases of asbestos to surface water and sediments associated with the COUs in this risk  
7555 evaluation. Therefore, EPA concludes there is no risk to aquatic or sediment-dwelling organisms.  
7556 Further, as described in the PF and above for the general population, other Agency regulations  
7557 adequately assess and effectively manage exposures to terrestrial organisms from asbestos releases to  
7558 terrestrial, including biosolids, pathways. Although EPA assessed the hazards to aquatic and sediment-  
7559 dwelling organisms in the risk evaluation, since no exposures exist under the COUs, EPA determined  
7560 there is no unreasonable risk for the environment.  
7561

## 7562 **5.2 Risk Determination for Chrysotile Asbestos**

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7563 EPA's determination of unreasonable risk for the conditions of use of chrysotile asbestos is based on  
7564 health risks to workers, occupational non-users (exposed to asbestos indirectly by being in the same  
7565 work area), consumers, and bystanders (exposed indirectly by being in the same vicinity where  
7566 consumer uses are carried out).

7567  
7568 As described in sections 4, significant risk were identified for lung cancer and mesothelioma. Section 26  
7569 of TSCA requires that EPA make decisions consistent with the "best available science." Section 26 also  
7570 requires other scientific considerations including consideration of the "extent of independent  
7571 verification" and "weight of the scientific evidence." As described in EPA's framework rule for risk  
7572 evaluation [82 FR 33726] weight of the scientific evidence includes consideration of the "strengths,  
7573 limitations and relevance of the information." Neither the statute nor the framework rule requires that

7574 EPA choose the lowest number and EPA believes that public health is best served when EPA relies upon  
7575 the highest quality information for which EPA has the greatest confidence.  
7576

7577 During risk evaluation, the only fiber type of asbestos that EPA identified as manufactured (including  
7578 imported), processed, or distributed under the conditions of use is chrysotile, the serpentine variety.  
7579 Chrysotile is the prevailing form of asbestos currently mined worldwide. Therefore, it is reasonable to  
7580 assume that commercially available products fabricated overseas are made with chrysotile. Any asbestos  
7581 being imported into the U.S. in articles for the conditions of use EPA has identified in this document is  
7582 believed to be chrysotile. Based on EPA's determination that chrysotile is the only form of asbestos  
7583 imported into the U.S. as both raw form and as contained in articles, EPA performed a quantitative  
7584 assessment for chrysotile asbestos. The other five forms of asbestos are no longer manufactured,  
7585 imported, or processed in the United States and are now subject to a significant new use rule (SNUR)  
7586 that requires notification (via a Significant New Use Notice (SNUN)) of and review by the Agency  
7587 should any person wish to pursue manufacturing, importing, or processing crocidolite (riebeckite),  
7588 amosite (cummingtonite-grunerite), anthophyllite, tremolite or actinolite (either in raw form or as part of  
7589 articles) for any use (40 CFR 721.11095). Under the final asbestos SNUR, EPA will be made aware of  
7590 manufacturing, importing, or processing for any intended use of the other forms of asbestos. If EPA  
7591 finds upon review of a SNUN that the significant new use presents or may present an unreasonable risk  
7592 (or if there is insufficient information to permit a reasoned evaluation of the health and environmental  
7593 effects of the significant new use), then EPA would take action under TSCA section 5(e) or (f) to the  
7594 extent necessary to protect against unreasonable risk. In this draft risk evaluation, EPA evaluated the  
7595 following categories of conditions of use of chrysotile asbestos: manufacturing; processing; distribution  
7596 in commerce; occupational and consumer uses; and disposal. EPA will consider any legacy uses and  
7597 associated disposal for chrysotile asbestos or other asbestos fiber types in subsequent supplemental  
7598 documents.  
7599

7600 As explained in the problem formulation document and Section 1.4 of this risk evaluation, EPA did not  
7601 evaluate the following: emission pathways to ambient air from commercial and industrial stationary  
7602 sources or associated inhalation exposure of the general population or terrestrial species; the drinking  
7603 water exposure pathway for asbestos; the human health exposure pathway for asbestos in ambient water;  
7604 emissions to ambient air from municipal and industrial waste incineration and energy recovery units; on-  
7605 site releases to land that go to underground injection; or on-site releases to land that go to asbestos  
7606 National Emission Standards for Hazardous Air Pollutants (NESHAP) (40 CFR part 61, subpart M)  
7607 compliant landfills or exposures of the general population (including susceptible populations) or  
7608 terrestrial species from such releases.  
7609

7610 The risk evaluation for chrysotile asbestos describes the physical-chemical characteristics that are  
7611 unique to chrysotile asbestos, such as insolubility in water, suspension and duration in air,  
7612 transportability, the friable nature of asbestos-containing products, which attribute to the potential for  
7613 asbestos fibers to be released, settled, and to again become airborne under the conditions of use (re-  
7614 entrainment<sup>30</sup>). Also unique to asbestos is the impact of the timing of exposure relative to the cancer  
7615 outcome; the most relevant exposures for understanding cancer risk were those that occurred decades  
7616 prior to the onset of cancer and subsequent cancer mortality. In addition to the cancer benchmark, the  
7617 physical-chemical properties and exposure considerations are important factors in considering risk of  
7618 injury to health. To account for the exposures for occupational non-users and, in certain cases

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<sup>30</sup> Settled Asbestos Dust Sampling and Analysis 1st Edition Steve M. Hays, James R. Millette CRC Press 1994



bystanders, EPA derived a distribution of exposure values for calculating the risk for cancer by using area monitoring data (i.e., fixed location air monitoring results) where available for certain conditions of use and when appropriate applied exposure reduction factors when monitoring data was not available, using data from published literature.

The risk determination for each COU in this risk evaluation considers both central tendency and high-end risk estimates for workers, ONUs, consumers and bystanders. Where relevant EPA considered PPE for workers. For many of the COUs both the central tendency and high-end risk estimates exceed the risk benchmark while some only at the high-end for each of the exposed populations evaluated. However, the risk benchmarks do not serve as a bright line for making risk determinations and other relevant risk-related factors and EPA's confidence in the underlying data were considered. In particular, risks associated with previous asbestos exposures are compounded when airborne asbestos fibers settle out and again become airborne where they can cause additional exposures and additional risks. The Agency also considered that the health effects associated with asbestos inhalation exposures are severe and irreversible. These risk-related factors resulted in EPA focusing on the high-end risk estimates rather than central tendency risk estimates to be most protective of workers, ONUs, consumers, and bystanders. Additionally, as discussed in Section 4.5.3, for workers and ONUs exposed in a workplace, EPA considered extra risks of 1 cancer per 10,000 people. At this risk level (1E-4), if the non-cancer effects (e.g., asbestosis and pleural thickening) of chrysotile are similar to Libby amphibole asbestos, the non-cancer effects of chrysotile are likely to contribute additional risk to the overall health risk of asbestos beyond the risk of cancer. Thus, the overall health risks of asbestos are underestimated based on cancer alone and support the Agency's focus on using the high-end risk estimates rather than central tendency risk to be protective of workers and ONUs.

The limited conditions of use of asbestos in conjunction with the extensive regulations safeguarding against exposures to asbestos helped to focus the scope of the risk evaluation on occupational and consumer scenarios where chrysotile asbestos in certain uses and products is known, intended, or reasonably foreseen. EPA did not quantitatively assess each life cycle stage and related exposure pathways as part of this risk evaluation. Existing EPA regulations and standards adequately assess and effectively manage exposure pathways to the general population, terrestrial species and chlor-alkali industry occupational populations (i.e., workers and ONUs) for the asbestos waste pathway (e.g., RCRA and the asbestos NESHAP). As such, the Agency did not evaluate these pathways.

The risk determinations are organized by conditions of use and displayed in a table format. Presented first are those life cycle stages where EPA assumes the absence of asbestos exposure, and the conditions of use that do not present an unreasonable risk are summarized in a table. EPA then presents the preliminary risk determination for the chrysotile asbestos-containing brakes conditions of use for the NASA "Super Guppy." Those conditions were determined not to present an unreasonable risk. The risk determinations for the conditions of use that present an unreasonable risk are depicted in section 5.2.1(Occupational Processing and Use of Chrysotile Asbestos) and section 5.2.2 (Consumer Uses of Chrysotile Asbestos). For each of the conditions of use assessed under the asbestos risk evaluation, a risk determination table is presented based on relevant criteria pertaining to each exposed population (i.e., health only for either workers, occupational non-users, consumers, or bystanders as indicated in table headings) is provided and explained below.

#### **Import, Distribution in Commerce and Disposal of Chrysotile Asbestos**

EPA assumed the absence of exposure to asbestos at certain life cycle stages. Raw asbestos and asbestos-containing products are imported into the U.S. in a manner where exposure to asbestos is not

7667 anticipated to occur. According to information reasonably available to EPA, raw asbestos is imported in  
7668 bags wrapped in plastic where they are contained in securely locked shipping containers. These shipping  
7669 containers remain locked until they reach the chlor-alkali plants (Enclosure B: Asbestos Controls in the  
7670 Chlor-Alkali Manufacturing Process [https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-  
7671 0736-0052](https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0736-0052)). Asbestos articles (or asbestos-containing products) are assumed to be imported and  
7672 distributed in commerce in a non-friable state, enclosed in sealed boxes, where fibers are not expected to  
7673 be released.

7674  
7675 EPA also assumes the absence of asbestos exposure during the occupational disposal of asbestos sheet  
7676 gaskets scraps during gasket stamping and the disposal of spent asbestos gaskets used in chemical  
7677 manufacturing plants. This assumption is based on the work practices followed and discussed in section  
7678 2.3.1 that prevent the release of asbestos fibers.

7679  
7680 Considering these exposure assumptions, EPA finds no unreasonable risk to health or the environment  
7681 for the life cycle stages of import and distribution in commerce of asbestos for all the conditions of use.  
7682 EPA also finds no unreasonable risk to health or the environment for occupational populations for the  
7683 disposal of asbestos sheet gaskets scraps during gasket stamping and the disposal of spent asbestos  
7684 gaskets used in chemical manufacturing plants.

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7686 In addition, there is a limited use of asbestos-containing brakes (categorized under other vehicle friction  
7687 products) for a special, large NASA transport plane (the “Super-Guppy”) that EPA recently learned  
7688 about. In this public draft risk evaluation, EPA is providing preliminary information for public input and  
7689 the information is provided in a brief format (see sections 2.3.1.8.2 and 4.2.2.6).

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7691 EPA calculated risk estimates using occupational exposure monitoring data provided by NASA. EPA  
7692 assumes 12 hours of brake changes occur every year starting at age 26 years with 20 years exposure.  
7693 The Excess Lifetime Cancer Risk for Super Guppy Brake/Repair Replacement for Workers is:

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7695	Full Shift (8-hour):	Central Tendency – 1.9 E-7
7696	Full Shift (8-hour):	High-End – 5.8 E-7
7697	Short Term:	Central Tendency – 3.2 E-7
7698	Short Term:	High-End – 9.1 E-7

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7701 Because the risk estimates fall below the benchmark for both the central tendency and high-end and after  
7702 considering the engineering controls and work practices in place discussed in section 2.3.1.8.2, EPA  
7703 finds these COUs (import/manufacture, distribution, use and disposal) do not present an unreasonable  
7704 risk of injury to health.

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Conditions of Use that Do Not Present an Unreasonable Risk to Health or Environment
<ul style="list-style-type: none"><li>• Import of asbestos and asbestos-containing products</li><li>• Distribution of asbestos-containing products</li><li>• Use of asbestos-containing brakes for a specialized, large NASA transport plane.</li><li>• Disposal of asbestos-containing sheet gaskets processed and/or used in the industrial setting and asbestos-containing brakes for a specialized, large NASA transport plane Distribution of asbestos-containing products</li></ul>

**5.2.1 Occupational Processing and Use of Chrysotile Asbestos**

EPA identified the following conditions of use where asbestos is processed and/or used in occupational settings: asbestos diaphragms in chlor-alkali industry, processed asbestos-containing sheet gaskets, asbestos-containing sheet gaskets in chemical production, asbestos-containing brake blocks in the oil industry, aftermarket automotive asbestos-containing brakes/ linings and other vehicle friction products and other asbestos-containing gaskets. OSHA’s Respiratory Protection Standard (29 CFR § 1910.134) requires employers in certain industries to address workplace hazards by implementing engineering control measures and, if these are not feasible, provide respirators that are applicable and suitable for the purpose intended. Assigned protection factors (APFs) are provided in Table 1 under § 1910.134(d)(3)(i)(A) (see Table 2-3 of the risk evaluation) and refer to the level of respiratory protection that a respirator or class of respirators is expected to provide to employees when the employer implements a continuing, effective respiratory protection program. Where applicable, in the following tables, EPA provides risk estimates with PPE using APFs derived from information provided by industry. However, there is some uncertainty in taking this approach as based on published evidence for asbestos (see Section 2.3.1.2), nominal APF may not be achieved for all respirator users.

Occupational non-users (ONUs) are not expected to wear PPE since they do not directly handle the chemical substance or articles thereof. Additionally, because ONUs are expected to be physically farther away from the chemical substance than the workers who handle it, EPA calculated an exposure reduction factor for ONUs based on the monitoring data (i.e., fixed location air monitoring results) provided by industry and the information available in the published literature (refer to section 2.3.1.3 of the risk evaluation).

As explained in section 5.2, EPA considers the high-end risk estimates for workers, occupational non-users, consumers, and bystanders for this risk determination of asbestos.

**Table 5-1. Risk Determination for Chrysotile Asbestos: Processing and Industrial Use of Asbestos Diaphragms in Chlor-alkali Industry (refer to section 4.2.2.1 for the risk characterization)**

Criteria for Risk Determination	Exposed Population	
	Workers	Occupational Non-Users
Life cycle Stage	Processing and Industrial Use	Processing and Industrial 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 <sup>-4</sup> excess cancer risks	10 <sup>-4</sup> excess cancer risks
Risk Estimates without PPE	<b>8 hour TWA</b> 1.2 E-4 Central Tendency 8.4 E-4 High-end <b>Short Term</b> 1.5 E-4 Central Tendency 1.1 E-4 Central Tendency <sup>a</sup>	<b>8 hour TWA</b> 5.8 E-5 Central Tendency 1.9 E-4 High-end <b>Short Term</b> Not available

Criteria for Risk Determination	Exposed Population	
	Workers	Occupational Non-Users
	1.3 E-3 High-end 8.1 E-4 High-end <sup>a</sup>	
Risk Estimates with PPE	<p><b>APF=10</b>  <b>8 hour TWA</b>            1.2 E-5 Central Tendency            8.4 E-5 High-end  <b>Short Term</b>            1.3 E-5 Central Tendency            9.9 E-5 High-end  <b>APF=25</b>  <b>8 hour TWA</b>            4.8 E-6 Central Tendency            3.4 E-5 High-end  <b>Short Term</b>            6.0 E-6 Central Tendency            5.2 E-5 High-end</p>	Not Assessed; ONUs are not assumed to wear respirators
Risk Considerations	<p>EPA calculated risk estimates using occupational exposure monitoring data provided by industry (Section 2.3.1.3). Without respiratory PPE the risk estimates indicate risk (central tendency and high-end); however, when expected use of respiratory PPE is considered for some worker tasks (APF=10 and APF=25), the risk estimates do not indicate unreasonable risk (central tendency and high-end). As depicted in Table 2-7 and documented by industry<sup>b</sup>, of the eight asbestos-related worker tasks, workers wear respiratory PPE during three tasks (Asbestos Unloading/Transport, Glovebox Weighing and Asbestos Handling, and Hydroblasting), but do not wear respiratory PPE during five of the tasks (Asbestos Slurry, Depositing, Cell Assembly, Cell Disassembly, and Filter Press). Although the use of respiratory PPE during three of the worker tasks reduces asbestos exposure and overall risk to workers, respiratory PPE is not worn throughout an entire 8-hour shift. The industry data depicted in Table 2-7 indicates workers without respiratory PPE are exposed to asbestos fibers</p>	<p>EPA calculated risk estimates using area monitoring data (i.e., fixed location air monitoring results) provided by industry (Section 2.3.1.3), which supports EPA’s expectation that ONU inhalation exposures are lower than inhalation exposures for workers directly handling asbestos materials (Table 2-8). There is some uncertainty in the ONU exposure estimate because much of the reported area monitoring data were reported as “less than” values, which may represent non-detects. One facility did not clearly distinguish whether measurements were area samples or personal breathing zone samples. EPA considered both the high-end and central tendency risk estimates in its determination, and although the high-end exceeds the cancer risk benchmark of <math>1 \times 10^{-4}</math>, both risk estimates are fairly similar. Based on the benchmarks exceedances and considering the physical-chemical properties of asbestos, including the potential for asbestos fibers to be released, settled, and to again become airborne during worker activities, the expected absence of respiratory PPE, and the severe and irreversible health effects associated with asbestos inhalation exposures, these</p>

Criteria for Risk Determination	Exposed Population	
	Workers	Occupational Non-Users
	where the maximum short-term PBZ samples for three tasks (cell assembly, cell disassembly and filter press) are in the range of some tasks, and higher than one task (Asbestos unloading/Transport), where respiratory PPE is used. Considering that respiratory PPE is not worn for all worker tasks where occupational exposure monitoring data indicates the presence of airborne asbestos fibers, the potential for released asbestos fibers to settle and to again become airborne during worker activities, and considering the severe and the irreversible effects associated with asbestos inhalation exposures, these conditions of use (for processing and use) present unreasonable risk to workers.	conditions of use (for processing and use) present unreasonable risk to ONUs.

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<sup>a</sup>No APF applied for 7.5 hours, APF of 25 applied for 30 minutes.

<sup>b</sup>Industry provided descriptions of the PPE used in Enclosure C: Overview of Monitoring Data and PPE Requirements  
<https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0736-0052>

**Table 5-2. Risk Determination for Chrysotile Asbestos: Processing Asbestos-Containing Sheet Gaskets (refer to section 4.2.2.2 for the risk characterization)**

Criteria for Risk Determination	Exposed Population	
	Workers	Occupational Non-Users
Life cycle Stage	Processing	Processing
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 <sup>-4</sup> excess cancer risks	10 <sup>-4</sup> excess cancer risks
Risk Estimates without PPE	<b>8-hour TWA</b> 3.3 E-4 Central Tendency 1.4 E-3 High-end <b>Short Term</b> 3.5 E-4 Central Tendency	<b>8-hour TWA</b> 5.6 E-5 Central Tendency 2.3 E-4 High-end <b>Short Term</b> 5.6 E-5 Central Tendency

Criteria for Risk Determination	Exposed Population	
	Workers	Occupational Non-Users
	1.4 E-3 High-end	2.3 E-4 High-end
Risk Estimates with PPE <sup>b</sup>	<p><b>APF = 1</b> An APF of 1 was assigned to the respiratory PPE provided to workers based on industry information <sup>b</sup></p> <p><b>8-hour TWA</b> 3.3 E-4 Central Tendency 1.4 E-3 High-end</p> <p><b>Short Term</b> 3.5 E-4 Central Tendency 1.4 E-3 High-end</p>	Not Assessed; ONUs are not assumed to wear respirators
Risk Considerations	<p>EPA calculated risk estimates using occupational exposure monitoring data provided by industry and in the published literature (Section 2.3.1.4). The use of N95 respirators was reported by industry<sup>a</sup> to be worn by a worker cutting gaskets. However, the OSHA Asbestos Standard 1910.1001 states that such respirators should not be used to mitigate asbestos exposure. Thus, the N95 respirator has an assigned APF=1 due to ineffectiveness as respiratory PPE for mitigating asbestos exposure. Absent effective respiratory PPE<sup>b</sup> risk estimates for both central tendency and high-end exceeds the benchmark of <math>1 \times 10^{-4}</math>. Based on the benchmarks exceedances and considering the physical-chemical properties of asbestos, including the potential for asbestos fibers to be released, settled, and to again become airborne during worker activities, and the severe and irreversible health effects associated with asbestos inhalation exposures, this condition of use presents unreasonable risk to workers.</p>	<p>EPA calculated risk estimates using monitoring data provided by industry and in the published literature. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling asbestos materials and based on exposure measurements in the published literature comparing workers to non-workers, EPA estimated a reduction factor of 5.75 for ONUs which was applied to the exposure estimate for workers (Section 2.3.1.3). Considering the physical-chemical properties of asbestos including the potential for asbestos fibers to be released, settled, and to again become airborne during worker activities, the expected absence of respiratory PPE, and the severe and irreversible health effects associated with asbestos inhalation exposures, EPA considered the high-end risk estimate appropriate for determining ONU risk. High-end risk estimates exceed the cancer risk benchmark of <math>1 \times 10^{-4}</math>. As such this condition of use presents unreasonable risk to ONUs.</p>

<sup>a</sup>Industry provided description of PPE (ACC, 2017a).

<sup>b</sup>Risk to workers was calculated using hypothetical respirator PPE of APF=10 and APF=25 in the risk evaluation. However, the risk estimates based on the hypothetical APF were not used in the risk determination based on industry description of current respiratory PPE.

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**Table 5-3. Risk Determination for Chrysotile Asbestos: Industrial Use of Asbestos-Containing Sheet Gaskets in Chemical Production**  
**(Titanium Dioxide Example is Representative of this COU; refer to section 4.2.2.3 for the risk characterization)**

Criteria for Risk Determination	Exposed Population	
	Workers	Occupational Non-Users
Life cycle Stage	Industrial Use	Industrial 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 <sup>-4</sup> excess cancer risks	10 <sup>-4</sup> excess cancer risks
Risk Estimates without PPE	<b>8-hour TWA</b> 6.0 E-4 Central Tendency 2.2 E-3 High-end	<b>8-hour TWA</b> 1.2 E-4 Central Tendency 3.7 E-4 High-end
Risk Estimates with current PPE <sup>a</sup>	<b>APF=10</b> <b>8-hour TWA</b> 6.0 E-5 Central Tendency 2.2 E-4 High-end	Not Assessed; ONUs are not assumed to wear respirators
Risk Considerations	EPA calculated risk estimates using occupational exposure monitoring data provided by industry and in the published literature (Section 2.3.1.5). Based on respiratory PPE used according to industry <sup>a</sup> EPA also calculated the risk estimates using an APF of 10; however, even with PPE and considering the physical-chemical properties of asbestos, including the potential for asbestos fibers to be released, settled, and to again become airborne during worker activities and the severe and irreversible health effects associated with asbestos inhalation exposures, high-end risk estimates for this condition of use exceed the benchmark of 1x10 <sup>-4</sup> and presents unreasonable risk to workers.	EPA calculated risk estimates using monitoring data provided by industry and in the published literature. Based on exposure measurements in the published literature, EPA estimated a reduction factor of 5.75 for ONUs (Section 2.3.1.4.). Because asbestos fibers released during the worker activities described in Section 2.3.1.5. can settle and again become airborne where they can be inhaled by ONUs, EPA considered it appropriate to use the high-end estimate when determining ONU risk. Based on the high-end risk estimate exceeding the benchmark of 1x10 <sup>-4</sup> , the expected absence of respiratory PPE and the severe and irreversible effects associated with asbestos inhalation exposures, this condition of use presents unreasonable risk to ONUs.

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<sup>a</sup>Industry provided description of PPE ([ACC, 2017a](#)).

**Table 5-4. Risk Determination for Chrysotile Asbestos: Industrial Use and Disposal of Asbestos-Containing Brake Blocks in Oil Industry (refer to section 4.2.2.4 for the risk characterization)**

Criteria for Risk Determination	Exposed Population	
	Workers	Occupational Non-Users
Life cycle Stage	Industrial Use and Disposal	Industrial Use and Disposal
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 <sup>-4</sup> excess cancer risks	10 <sup>-4</sup> excess cancer risks
Risk Estimates without PPE	<b>8-hour TWA</b> 7.0 E-4	<b>8 hour-TWA</b> 4.6 E-4
Risk Estimates with PPE	<b>APF=1</b> Workers are not assumed to wear respirators	Not Assessed; ONUs are not assumed to wear respirators
Risk Considerations (applies to both workers and ONUs)	The estimated exposure scenario used in the risk evaluation is based on one 1988 study of Norway’s offshore petroleum industry and relevance to today’s use of oil field brake blocks in the United States is uncertain. EPA is aware that brake blocks are imported, distributed, and used in the U.S. although the full extent of use could not be determined. According to industry <sup>a</sup> , Drawworks machineries are always used and serviced outdoors, close to oil wells. Information on processes and worker activities are insufficient to determine the proximity of ONUs to workers. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling asbestos materials. Although EPA has calculated a single conservative risk estimate for workers and for ONUs, EPA does not expect routine use of respiratory PPE. Considering the cancer risk benchmark of 1x10 <sup>-4</sup> is exceeded and the severe and irreversible effects associated with asbestos inhalation exposures, these conditions of use present unreasonable risk for both workers and ONUs.	

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<sup>a</sup> Industry provided data ([Popik, 2018](#))



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**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 <sup>-4</sup> excess cancer risks	10 <sup>-4</sup> excess cancer risks
Risk Estimates without PPE	<b>8-hour TWA</b> 1.4 E-4 Central Tendency 2.2 E-3 High-end <b>Short Term</b> 1.4 E-4 Central Tendency 3.3 E-3 High-end	<b>8-hour TWA</b> 1.6 E-5 Central Tendency 2.6 E-4 High-end <b>Short Term</b> 1.6 E-5 Central Tendency 2.6 E-4 High-end
Risk Estimates with PPE	<b>APF = 1</b> Workers are not assumed to wear respirators; Respirators only required by OSHA if PEL exceeded. <b>8-hour TWA</b> 1.4 E-4 Central Tendency 2.2 E-3 High-end <b>Short Term</b> 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

Criteria for Risk Determination	Exposed Population	
	Workers	Occupational Non-Users
	(for both central tendency and high-end). Based on the exceedance of the benchmark of $1 \times 10^{-4}$ and consideration of the severe and irreversible effects associated with asbestos inhalation exposures, these conditions of use present unreasonable risk to workers.	where they can be inhaled by ONUs, EPA considered it appropriate to use the high-end estimate when determining ONU risk. Based on the exceedance (high-end) of the benchmark of $1 \times 10^{-4}$ , the expected absence of respiratory PPE and the potential severity and irreversible effects associated with inhalation exposures to asbestos, these conditions of use present unreasonable risk to ONUs.

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**Table 5-6. Risk Determination for Chrysotile Asbestos: Commercial Use and Disposal of Other Asbestos-Containing Gaskets**  
**(Commercial Mechanic Gasket Repair/Replacement is Representative for this COU; refer to section 4.2.2.7 for the risk characterization)**

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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	<b>8-hour TWA</b> 5.6 E-4 Central Tendency 1.5 E-3 High-end	<b>8-hour TWA</b> 1.2 E-4 Central Tendency 3.5 E-4 High-end
Risk Estimates with PPE	<b>APF=1</b> Workers are not assumed to wear respirators <b>8-hour TWA</b> 5.6 E-4 Central Tendency 1.5 E-3 High-end	Not Assessed; ONUs are not assumed to wear respiratory PPE.

Criteria for Risk Determination	Exposed Population	
	Workers	Occupational Non-Users
Risk Considerations	EPA calculated risk estimates using exposure scenarios based on occupational monitoring data (breathing zone of workers) for asbestos-containing gasket replacement in vehicles. Although, risk to workers was calculated using hypothetical respirator PPE of APF=10 and APF=25, workers are not expected to wear respiratory PPE during gasket repair and replacement in a commercial setting. Based on the expected absence of PPE and the benchmark of $1 \times 10^{-4}$ is exceeded (for both central tendency and high-end), these conditions of use present unreasonable risk to workers.	EPA calculated risk estimates using exposure scenarios based on occupational monitoring data (work area samples in the vicinity of the workers) for asbestos-containing gasket replacement in vehicles. EPA estimated a reduction factor of 5.75 (Section 2.3.1.9) for ONUs. Due to the severe and irreversible effects associated with asbestos inhalation exposures and that asbestos fibers released during the worker activities described in Section 2.3.1.9 can settle and again become airborne where they can be inhaled by ONUs, EPA considered it appropriate to use the high-end estimate when determining ONU risk. Based on the exceedance of the benchmark of $1 \times 10^{-4}$ (for both central tendency and high-end), and the expected absence of respirators, and the potential severity of effect associated with inhalation exposures to asbestos, these conditions of use present unreasonable risk to ONUs.

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7775 **5.2.2 Consumer Uses of Chrysotile Asbestos**

7776 The consumer uses of asbestos include aftermarket automotive asbestos-containing brakes/linings, and  
7777 other asbestos-containing gaskets. Consumers and bystanders are not assumed to wear respiratory PPE,  
7778 therefore, EPA did not assess risk estimates with PPE the conditions of use for these exposed  
7779 populations.

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**Table 5-7. Risk Determination for Chrysotile Asbestos: Consumer Use and Disposal of Aftermarket Automotive Asbestos-Containing Brakes/Linings (Do-it-Yourself Consumer Brake Repair/Replacement is Representative for both COUs; refer to section 4.2.3.1 for the risk characterization)**

Criteria for Risk Determination	Exposed Population	
	Do-it-Yourself Mechanic	Bystander
Life cycle Stage	Consumer Use	Consumer Use
TSCA Section 6(b)(4)(A) Unreasonable Risk Determination	Presents an unreasonable risk of injury to health (consumers and bystanders)	
Unreasonable Risk Driver	Cancer resulting from chronic inhalation exposure	Cancer resulting from chronic inhalation exposure
Benchmark (Cancer)	10 <sup>-6</sup> excess cancer risks	10 <sup>-6</sup> excess cancer risks
Risk Estimates without PPE	<b><u>Indoor, compressed air</u></b> <b>1 hour/day; once every 3 years for 62 years (starting age 16)</b> Exposures at 30% of active used between uses, 1 hour/d in garage 4.3 E-5 Central Tendency 4.2 E-4 High-end	<b><u>Indoor, compressed air</u></b> <b>1 hour/day; once every 3 years for 62 years (starting age 16)</b> Exposures at 30% of active used between uses, 1 hour/d in garage 2.6 E-5 Central Tendency 6.0 E-5 High-end
	<b><u>Indoor, compressed air</u></b> <b>8 hour/day; once every 3 years for 62 years (starting age 16)</b> Exposures at 30% of active used between uses, 8 hours/d in garage 3.4 E-4 Central Tendency 3.4 E-3 High-end	<b><u>Indoor, compressed air</u></b> <b>1 hour/day; once every 3 years for 62 years (starting age 16)</b> Exposures at 30% of active used between uses, 8 hours/d in garage 2.6 E-5 Central Tendency 6.0 E-5 High-end
	<b><u>Indoor, compressed air</u></b> <b>Indoor, compressed air, once at 16 years, staying in residence for 10 years, 1 hour/d in garage</b> 5.6 E-6 Central Tendency 5.5 E-5 High-end	<b><u>Indoor, compressed air</u></b> <b>Indoor, compressed air, once at 16 years, staying in residence for 10 years, 1 hour/d in garage</b> 3.0 E-6 Central Tendency 7.1 E-6 High-end
	<b><u>Outdoor</u></b> <b>Once every 3 years for 62 years (starting age 16)</b> Exposures at 2% of active used between uses, 5 min/d in driveway 9.9 E-8 Central Tendency 5.3 E-7 High-end	<b><u>Outdoor</u></b> <b>Once every 3 years for 62 years (starting age 16)</b> Exposures at 2% of active used between uses, 5 min/d in driveway 2.1 E-8 Central Tendency 1.1 E-7 High-end

Criteria for Risk Determination	Exposed Population	
	Do-it-Yourself Mechanic	Bystander
	<p><b>Outdoor</b>  <b>Once every 3 years for 62 years (starting age 16)</b>                      Exposures at 2% of active used between uses, 30 min/d in driveway                      2.9 E-7 Central Tendency                      1.5 E-6 High-end</p>	<p><b>Outdoor</b>  <b>Once every 3 years for 62 years (starting age 16)</b>                      Exposures at 2% of active used between uses, 30 min/d in driveway                      5.9 E-8 Central Tendency                      3.2. E-7 High-end</p>
Risk Estimates with PPE	Not Assessed; Consumers are not assumed to wear respiratory PPE	Not Assessed; Bystanders are not assumed to wear respiratory PPE
Risk Considerations	<p>EPA calculated risk estimates are based on data provided in the published literature and surrogate monitoring data from occupational brake repair studies. EPA considered 4 different exposure scenarios with different assumptions on the duration of exposure, whether indoors in a garage using compressed air or outside without compressed air. Although DIY brake and clutch work is more likely to occur outdoors, it may also occur inside a garage. Additionally, considering that many DIY mechanics have access to air compressors, EPA expects that at least some DIY mechanics may use compressed air to clean dust from brakes or clutches and can spend up to a full day (8 hours) in their garage and working three hours specifically on brakes and clutches. Because asbestos fibers released during the DIY (consumer) activities described in Section 2.3.2.1 can settle and again become airborne where they can be inhaled by bystanders, EPA considered it appropriate to use the high-end estimate when determining consumer risk. EPA chose a conservative and protective brake and clutch repair/replacement exposure scenario of 3 hours/day once every 3 years inside a garage using compressed air to account for the possibility that some DIY mechanics</p>	<p>EPA calculated risk estimates are based on data provided in the published literature and surrogate monitoring data from occupational brake repair studies. No reduction factor was applied for indoor DIY brake work inside residential garages due to the expected close proximity of bystanders inside a garage. In the absence of data to estimate a reduction factor for outdoor brake work, EPA assumed a reduction factor of 10 (Section 2.3.2.1). Because asbestos fibers released during the DIY (consumer) activities described in Section 2.3.2.1 can settle and again become airborne where they can be inhaled by bystanders, EPA considered it appropriate to use the high-end estimate when determining bystander risk. EPA also chose a conservative and protective brake repair/replacement exposure scenario of 3 hours/day while inside a garage up to 8 hours once every 3 years, using compressed air to account for the possibility that some bystanders (e.g., children watching parents) may fit this exposure scenario. EPA also used a less conservative brake and clutch repair/replacement exposure scenario of once in a lifetime, 1 hour per day, while inside a garage, using compressed air. As part of the analysis, EPA made some assumptions regarding both age at the start of exposure and the duration of exposure. Realizing there is uncertainty around these assumptions, EPA</p>

Criteria for Risk Determination	Exposed Population	
	Do-it-Yourself Mechanic	Bystander
	<p>may fit this exposure scenario. EPA also used a less conservative brake and clutch repair/replacement exposure scenario of once in a lifetime, 1 hour per day, while inside a garage, using compressed air. As part of the analysis, EPA made some assumptions regarding both age at the start of exposure and the duration of exposure. Realizing there is uncertainty around these assumptions, EPA developed a sensitivity analysis approach specifically for the consumer exposure/risk analysis (see Section 4.3.7 and Appendix L.) Under the chosen indoor exposure scenarios, the cancer benchmark is exceeded (both central tendency and high-end), therefore, these conditions of use present unreasonable risk to consumers.</p>	<p>developed a sensitivity analysis approach specifically for the bystander exposure/risk analysis (see Section 4.3.7 and Appendix L.) Based on the exceedance (both central tendency and high-end) of the benchmark of <math>1 \times 10^{-6}</math> for the chosen indoor exposure scenarios, the expected absence of respiratory PPE, and the potential severity of effects associated with inhalation exposures to asbestos, these conditions of use present unreasonable risk to bystanders.</p>

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**Table 5-8. Risk Determination for Chrysotile Asbestos: Consumer Use and Disposal of Other Asbestos-Containing Gaskets**  
(Do-it-Yourself Consumer Gasket Repair/Replacement is Representative for this COU; refer to section 4.2.3.2 for the risk characterization)

Criteria for Risk Determination	Exposed Population	
	Do-it-Yourself Mechanic	Bystander
Life cycle Stage	Consumer Use	Consumer Use
TSCA Section 6(b)(4)(A) Unreasonable Risk Determination	Presents unreasonable risk of injury to health (consumers and bystanders)	
Unreasonable Risk Driver	Cancer resulting from chronic inhalation exposure	Cancer resulting from chronic inhalation exposure
Benchmark (Cancer)	$10^{-6}$ excess cancer risks	$10^{-6}$ excess cancer risks
Risk Estimates without PPE	<b>Indoor 1 hour/day; once every 3 years for 62 years (starting age 16)</b>	<b>Indoor 1 hour/day; once every 3 years for 62 years (starting age 16)</b>

Criteria for Risk Determination	Exposed Population	
	Do-it-Yourself Mechanic	Bystander
	Exposures at 30% of active used between uses, 1 hour/d in garage 2.3 E-5 Central Tendency 6.4 E-5 High-end	Exposures at 30% of active used between uses, 1 hour/d in garage 2.4 E-5 Central Tendency 6.1 E-5 High-end
	<b>Indoor</b> <b>8 hour/day; once every 3 years for 62 years (starting age 16)</b> Exposures at 30% of active used between uses, 8 hours/d in garage 1.8 E-4 Central Tendency 5.1 E-4 High-end	<b>Indoor</b> <b>1 hour/day; once every 3 years for 62 years (starting age 16)</b> Exposures at 30% of active used between uses, 8 hours/d in garage 2.4 E-5 Central Tendency 6.1 E-5 High-end
	<b>Indoor</b> <b>1 hour/day, once in a lifetime (at age 16), staying in residence for 10 years</b> 3.0 E-6 Central Tendency 8.3 E-6 High-end	<b>Indoor</b> <b>1 hour/day, once in a lifetime (at age 16), staying in residence for 10 years</b> 3.08 E-6 Central Tendency 7.16 E-6 High-end
Risk Estimates with PPE	Not Assessed; Consumers are not assumed to wear respiratory PPE	Not Assessed; Bystanders are not assumed to wear respiratory PPE
Risk Considerations	EPA assumed that the duration of gasket repair activity was 3 hours a day and that a DIY mechanic is likely to perform one gasket repair once every 3 years and can spend up to a full day (8 hours) in their garage. This scenario assumes all the work is conducted indoors (within a garage) and both the consumer and bystander remain in the garage for the entirety of the work. EPA presents this conservative and protective gasket repair/replacement exposure scenario approach to account for the possibility that some DIY mechanics may fit this exposure scenario. EPA also presents a less conservative gasket repair/replacement exposure scenario of 1 hour a day, once in a lifetime gasket repair/replacement at age 16. EPA made some assumptions regarding both age at the start of exposure and the duration of exposure. Realizing there is uncertainty around these assumptions, EPA developed a sensitivity analysis approach specifically for the consumer exposure/risk analysis (see Section 4.3.7	EPA assumed that the duration of bystander exposure was 1 hour a day once every 3 years. EPA also presents a less conservative gasket repair/replacement exposure scenario of 1 hour a day, once in a lifetime gasket repair/replacement at age 16. EPA made some assumptions regarding both age at the start of exposure and the duration of exposure. Realizing there is uncertainty around these assumptions, EPA developed a sensitivity analysis approach specifically for the consumer exposure/risk analysis (see Section 4.3.7 and Appendix L.) Due to the severe and irreversible effects associated with asbestos inhalation exposures and that asbestos fibers released during the DIY activities described in Section 2.3.2.2 can settle and again become airborne where they can be inhaled by bystanders, EPA considered it appropriate to use the high-end estimate when determining bystander risk. Based on the exceedance of the benchmark of $1 \times 10^{-6}$ , at both the central tendency and high-end estimates and the expected absence of respiratory

Criteria for Risk Determination	Exposed Population	
	Do-it-Yourself Mechanic	Bystander
	and Appendix L.) Due to the severe and irreversible effects associated with asbestos inhalation exposures and that asbestos fibers released during the DIY activities described in Section 2.3.2.2, can settle and again become airborne where they can be inhaled EPA considered it appropriate to use the high-end estimates when determining consumer risk. Based on the exceedance of the benchmark of $1 \times 10^{-6}$ , at both the central tendency and high-end estimates and the expected absence of respiratory PPE, these conditions of use present unreasonable risk to consumers.	PPE, these conditions of use present unreasonable risk to bystanders.

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### **5.3 Risk Determination for Five other Asbestiform Varieties**

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For the risk evaluation, EPA adopted the TSCA Title II definition of asbestos which includes the varieties of six fiber types – chrysotile (serpentine), crocidolite (riebeckite), amosite (cummingtonite-grunerite), anthophyllite, tremolite or actinolite. In this document, EPA only assessed the conditions of use of chrysotile. EPA will consider legacy uses and associated disposal (which could include the other five asbestiform varieties) in subsequent supplemental documents.

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## 7 APPENDICES

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### Appendix A Regulatory History

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#### A.1 Federal Laws and Regulations

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The federal laws and regulations applicable to asbestos are listed along with the regulating agencies below. States also regulate asbestos through state laws and regulations, which are also listed within this section.

##### **Toxics Substances Control Act (TSCA), 1976**

[15 U.S.C. §2601 et seq](#)

The Toxic Substances Control Act of 1976 provides EPA with authority to require reporting, record-keeping and testing requirements, and restrictions relating to chemical substances and/or mixtures. Certain substances are generally excluded from TSCA, including, among others, food, drugs, cosmetics and pesticides.

TSCA addresses the production, importation, use and disposal of specific chemicals including [polychlorinated biphenyls \(PCBs\)](#), [asbestos](#), [radon](#) and [lead-based paint](#). The Frank R. Lautenberg Chemical Safety for the 21st Century Act updated TSCA in 2016 <https://www.epa.gov/laws-regulations/summary-toxic-substances-control-act>.

##### **Asbestos Hazard Emergency Response Act (AHERA), 1986**

[TSCA Subchapter II: Asbestos Hazard Emergency Response 15 U.S.C. §2641-2656](#)

Defines asbestos as the asbestiform varieties of— chrysotile (serpentine), crocidolite (riebeckite), amosite (cummingtonite-grunerite), anthophyllite, tremolite or actinolite.

Requires local education agencies (i.e., school districts) to inspect school buildings for asbestos and submit asbestos management plans to appropriate state; management plans must be publicly available and inspectors must be trained and accredited.

Tasked EPA to develop an asbestos Model Accreditation Plan (MAP) for states to establish training requirements for asbestos professionals who do work in school buildings and also public and commercial buildings.

##### **Asbestos-Containing Materials in Schools Rule (per AHERA), 1987**

[40 CFR Part 763, Subpart E](#)

Requires local education agencies to use trained and accredited asbestos professionals to identify and manage asbestos-containing building material and perform asbestos response actions (abatement) in school buildings.

##### **1989 Asbestos: Manufacture, Importation, Processing, and Distribution in Commerce**

**Prohibitions; Final Rule** (also known as Asbestos Ban and Phase-out Rule (Remanded), 1989)

[40 CFR Part 763, Subpart I](#)

[Docket ID: OPTS-62048E; FRL-3269-8](#)

EPA issued a final rule under Section 6 of Toxic Substances Control Act (TSCA) banning most asbestos-containing products.

In 1991, this rule was vacated and remanded by the Fifth Circuit Court of Appeals. As a result, most of the original ban on the manufacture, importation, processing or distribution in commerce for the

8359 majority of the asbestos-containing products originally covered in the 1989 final rule was overturned.  
8360 The following products remain banned by rule under the Toxic Substances Control Act (TSCA):  
8361     o Corrugated paper  
8362     o Rollboard  
8363     o Commercial paper  
8364     o Specialty paper  
8365     o Flooring felt

8366  
8367 In addition, the regulation continues to ban the use of asbestos in products that have not historically  
8368 contained asbestos, otherwise referred to as “new uses” of asbestos (Defined by 40 CFR 763.163 as  
8369 "commercial uses of asbestos not identified in §763.165 the manufacture, importation or processing of  
8370 which would be initiated for the first time after August 25, 1989.”).

8371  
8372 **Restrictions on Discontinued Uses of Asbestos; Significant New Use Rule (SNUR), 2019**

8373 40 CFR Parts 9 and 721 – Restrictions on Discontinued Uses of Asbestos

8374 Docket ID: EPA-HQ-OPPT-2018-0159; FRL 9991-33

8375 This final rule strengthens the Agency’s ability to rigorously review an expansive list of asbestos  
8376 products that are no longer on the market before they could be sold again in the United States. Persons  
8377 subject to the rule are required to notify EPA at least 90 days before commencing any manufacturing,  
8378 importing, or processing of asbestos or asbestos-containing products covered under the rule. These uses  
8379 are prohibited until EPA conducts a thorough review of the notice and puts in place any necessary  
8380 restrictions or prohibits use.

8381  
8382 **Other EPA Regulations:**

8383 *Asbestos Worker Protection Rule, 2000*

8384 [40 CFR Part 763, Subpart G](#)

8385 Extends OSHA standards to public employees in states that do not have an OSHA approved worker  
8386 protection plan (about half the country).

8387  
8388 *Asbestos Information Act, 1988*

8389 [15 U.S.C. §2607\(f\)](#)

8390 Helped to provide transparency and identify the companies making certain types of asbestos-containing  
8391 products by requiring manufacturers to report production to the EPA.

8392  
8393 *Asbestos School Hazard Abatement Act (ASHAA), 1984 and Asbestos School Hazard Abatement*  
8394 *Reauthorization Act (ASHARA), 1990*

8395 [20 U.S.C. 4011 et seq.](#) and [Docket ID: OPTS-62048E; FRL-3269-8](#)

8396 Provided funding for and established an asbestos abatement loan and grant program for school districts  
8397 and ASHARA further tasked EPA to update the MAP asbestos worker training requirements.

8398  
8399 *Emergency Planning and Community Right-to-Know Act (EPCRA), 1986*

8400 [42 U.S.C. Chapter 116](#)

8401 Under Section 313, Toxics Release Inventory (TRI), requires reporting of environmental releases of  
8402 friable asbestos at a concentration level of 0.1%.

8403 Friable asbestos is designated as a hazardous substance subject to an Emergency Release Notification at  
8404 40 CFR §355.40 with a reportable quantity of 1 pound.

8405  
8406 *Clean Air Act, 1970*

8407 [42 U.S.C. §7401 et seq.](#)

8408 Asbestos is identified as a Hazardous Air Pollutant.  
8409  
8410 *Asbestos National Emission Standard for Hazardous Air Pollutants (NESHAP), 1973*  
8411 [40 CFR Part 61, Subpart M of the Clean Air Act](#)  
8412 Specifies demolition and renovation work practices involving asbestos in buildings and other facilities  
8413 (but excluding residences with 4 or fewer dwelling units single family homes).  
8414 Requires building owner/operator notify appropriate state agency of potential asbestos hazard prior to  
8415 demolition/renovation.  
8416 Banned spray-applied surfacing asbestos-containing material for fireproofing/insulating purposes in  
8417 certain applications.  
8418 Requires that asbestos-containing waste material from regulated activities be sealed in a leak-tight  
8419 container while wet, labeled, and disposed of properly in a landfill qualified to receive asbestos waste.  
8420  
8421 *Clean Water Act (CWA), 1972*  
8422 [33 U.S.C. §1251 et seq](#)  
8423 Toxic pollutant subject to effluent limitations per Section 1317.  
8424  
8425 *Safe Drinking Water Act (SDWA), 1974*  
8426 [42 U.S.C. §300f](#)  
8427 Asbestos Maximum Contaminant Level Goals (MCLG) 7 million fibers/L (longer than 10um).  
8428  
8429 *Resource Conservation and Recovery Act (RCRA), 1976*  
8430 [42 U.S.C. §6901 et seq.](#)  
8431 [40 CFR 239-282](#)  
8432 Asbestos is subject to solid waste regulation when discarded; NOT considered a hazardous waste.  
8433  
8434 *Comprehensive Environmental Response, Compensation and Liability Act (CERCLA), 1980*  
8435 [42 U.S.C. §9601 et seq.](#)  
8436 [40 CFR Part 302.4 - Designation of Hazardous Substances and Reportable Quantities](#)  
8437 13 Superfund sites containing asbestos, nine of which are on the National Priorities List (NPL)  
8438 Reportable quantity of friable asbestos is one pound.  
8439  
8440 **Other Federal Agencies:**  
8441 Occupational Safety and Health Administration (OSHA):  
8442 [Public Law 91-596](#) Occupational Safety and Health Act, 1970  
8443 Employee permissible exposure limit (PEL) is 0.1 fibers per cubic centimeter (f/cc) as an 8-hour, time-  
8444 weighted average (TWA) and/or the excursion limit (1.0 f/cc as a 30-minute TWA).  
8445 Asbestos General Standard [29 CFR 1910](#)  
8446 Asbestos Shipyard Standard [29 CFR 1915](#)  
8447 Asbestos Construction Standard [29 CFR 1926](#)  
8448  
8449 Consumer Product Safety Commission (CPSC): Banned several consumer products. Federal Hazardous  
8450 Substances Act (FHSA) [16 CFR 1500](#)  
8451 Food and Drug Administration (FDA): Prohibits the use of asbestos-containing filters in pharmaceutical  
8452 manufacturing, processing and packing. [21 CFR 211.72](#)  
8453  
8454 Mine Safety and Health Administration (MSHA): follows OSHA's safety standards.  
8455 Surface Mines [30 CFR part 56, subpart D](#)  
8456 Underground Mines [30 CFR part 57, subpart D](#)

8457  
8458 Department of Transportation  
8459 Prescribes the requirements for shipping manifests and transport vehicle placarding applicable to  
8460 asbestos [40 CFR part 172](#).

8461  
8462 Non-regulatory information of note:  
8463 NIOSH conducts related research and monitors asbestos exposure through workplace activities in an  
8464 effort to reduce illness and ensure worker health and safety.  
8465

## 8466 A.2 State Laws and Regulations

8467 Pursuant to AHERA, states have adopted through state regulation the EPA's Model Accreditation Plan  
8468 (MAP) for asbestos abatement professionals who do work in schools and public and commercial  
8469 buildings. Thirty-nine (39) states<sup>31</sup> have EPA-approved MAP programs and twelve (12) states<sup>32</sup> have  
8470 also applied to and received a waiver from EPA to oversee implementation of the Asbestos-Containing  
8471 Materials in Schools Rule pursuant to AHERA. States also implement regulations pursuant to the  
8472 Asbestos NESHAP regulations or further delegate those oversight responsibilities to local municipal  
8473 governments. While federal regulations set national asbestos safety standards, states have the authority  
8474 to impose stricter regulations. As an example, many states extend asbestos federal regulations – such as  
8475 asbestos remediation by trained and accredited professionals, demolition notification, and asbestos  
8476 disposal – to ensure safety in single-family homes. Thirty (30) states<sup>33</sup> require firms hired to abate  
8477 asbestos in single family homes to be licensed by the state. Nine (9) states<sup>34</sup> mandate a combination of  
8478 notifications to the state, asbestos inspections, or proper removal of asbestos in single family homes.  
8479 Some states have regulations completely independent of the federal regulations. For example, California  
8480 and Washington regulate products containing asbestos. Both prohibit use of more than 0.1% of asbestos  
8481 in brake pads and require laboratory testing and labeling.

8482  
8483 Below is a list of state regulations that are independent of the federal AHERA and NESHAP  
8484 requirements that states implement. This may not be an exhaustive list.

8485  
8486 **California**  
8487 [Asbestos](#) is listed on [California's Candidate Chemical List](#) as a carcinogen. Under [California's](#)  
8488 [Propositions 65](#), businesses are required to warn Californians of the presence and danger of [asbestos](#) in  
8489 products, home, workplace and environment.

8490  
8491 ***California Brake Friction Material Requirements (Effective 2017)***  
8492 [Division 4.5, California Code of Regulations, Title 22 Chapter 30](#)

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<sup>31</sup> Alabama, Alaska, Arkansas, California, Colorado, Connecticut, Delaware, Florida, Illinois, Indiana, Kentucky, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Mississippi, Missouri, Montana, Nebraska, New Hampshire, New Jersey, New York, North Carolina, North Dakota, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, South Dakota, Texas, Utah, Vermont, Virginia, Washington, West Virginia, and Wisconsin.

<sup>32</sup> Connecticut, Colorado, Illinois, Kentucky, Louisiana, Massachusetts, Maine, New Hampshire, Oklahoma, Rhode Island, Texas, and Utah.

<sup>33</sup> California, Colorado, Connecticut, Delaware, Florida, Georgia, Hawaii, Iowa, Kansas, Maine, Maryland, Massachusetts, Michigan, Minnesota, Nebraska, Nevada, New Hampshire, New Jersey, New Mexico, New York, North Carolina, North Dakota, Oregon, Pennsylvania, Utah, Vermont, Virginia, Washington, West Virginia, and Wisconsin.

<sup>34</sup> Colorado, Connecticut, Georgia, Maine, Massachusetts, New York, Oregon, Vermont, and West Virginia.



8493 Sale of any motor vehicle brake friction materials containing more than 0.1% asbestiform fibers by  
8494 weight is prohibited. All brake pads for sale in the state of California must be laboratory tested, certified  
8495 and labeled by the manufacturer.  
8496

8497 **Massachusetts**

8498 *Massachusetts Toxics Use Reduction Act (TURA)*

8499 Requires companies in Massachusetts to provide annual pollution reports and to evaluate and implement  
8500 pollution prevention plans. Asbestos is included on the [Complete List of TURA Chemicals - March](#)  
8501 [2016](#).

8502

8503 **Minnesota**

8504 *Toxic Free Kids Act* [Minn. Stat. 2010 116.9401 – 116.9407](#)

8505 Asbestos is included on the [2016 Minnesota Chemicals of High Concern List](#) as a known carcinogen.  
8506

8507 **New Jersey**

8508 New Jersey [Right to Know Hazardous Substances](#)

8509 The state of New Jersey identifies hazardous chemicals and products. Asbestos is listed as a known  
8510 carcinogen and talc containing asbestos is identified on the Right to Know Hazardous Substances list.  
8511

8512

8512 **Rhode Island**

8513 *Rhode Island Air Resources – Air Toxics Air Pollution Control Regulation No. 22*

8514 Establishes acceptable ambient air levels for asbestos.  
8515

8516

8516 **Washington**

8517 *Better Brakes Law (Effective 2015)* [Chapter 70.285 RCW Brake Friction Material](#)

8518 Prohibits the sale of brake pads containing more than 0.1% asbestiform fibers (by weight) in the state of  
8519 Washington and requires manufacturer certification and package/product labelling.

8520 [Requirement to Label Building Materials that Contain Asbestos Chapter 70.310 RCW](#)

8521 Building materials that contain asbestos must be clearly labeled as such by manufacturers, wholesalers,  
8522 and distributors.  
8523

8524

### 8524 **A.3 International Laws and Regulations**

8525 Asbestos is also regulated internationally. Nearly 60 nations have some sort of asbestos ban. The  
8526 European Union (EU) will prohibit the use of asbestos in the chlor-alkali industry by 2025 ([Regulation](#)  
8527 [\(EC\) No 1907/2006 of the European Parliament and of the Council, 18 December 2006](#)).

8528

8529 Canada banned asbestos in 2018

8530 *Prohibition of Asbestos and Products Containing Asbestos Regulations: SOR/2018-196*

8531 [Canada Gazette, Part II, Volume 152, Number 21](#)  
8532

8533

8533 In addition, the Rotterdam Convention is considering [adding chrysotile to Annex III](#), and the World  
8534 Health Organization (WHO) has a global campaign to eliminate asbestos-related diseases ([WHO](#)  
8535 [Resolution 60.26](#)).  
8536

## Appendix B List of Supplemental Documents

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List of supplemental documents:

Associated **Supplemental Systematic Review Data Quality Evaluation and Date Extraction**

**Documents** – Provides additional detail and information on individual study evaluations and data extractions including criteria nad scoring results.

Physical-Chemical Properties, Fate and Transport

a. *Draft Risk Evaluation for Asbestos, , Systematic Review Supplemental File: Data Quality Evaluation of Physical-Chemical Properties Studies* ([U.S. EPA, 2019j](#))

b. *Draft Risk Evaluation for Asbestos, Systematic Review Supplemental File: Data Extraction of Environmental Fate and Transport Studies* ([U.S. EPA, 2019e](#))

Occupational Exposures and Releases

c. *Draft Risk Evaluation for Asbestos, Systematic Review Supplemental File: Data Quality Evaluation of Environmental Releases and Occupational Exposure* ([U.S. EPA, 2019f](#))

d. *Draft Risk Evaluation for Asbestos, Systematic Review Supplemental File: Data Quality Evaluation of Environmental Releases and Occupational Exposure Data Common Sources* ([U.S. EPA, 2019g](#))

Consumer and Environmental Exposures

e. *Draft Risk Evaluation for Asbestos, Systematic Review Supplemental File: Data Quality Evaluation of Consumer Exposure* ([U.S. EPA, 2019c](#))

f. *Draft Risk Evaluation for Asbestos, Systematic Review Supplemental File: Data Quality Extraction Tables for Consumer Exposure* ([U.S. EPA, 2019i](#))

Environmental Hazard

g. *Draft Risk Evaluation for Asbestos, Systematic Review Supplemental File: Data Quality Evaluation of Ecological Hazard Studies* ([U.S. EPA, 2019d](#))

Human Health Hazard

h. *Draft Risk Evaluation for Asbestos, Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies: Mesothelioma and Lung Cancer Studies* ([U.S. EPA, 2019h](#))

Associated **Supplemental Information Documents** – Provides additional details and information on exposure.

Occupational Exposures

i. *Draft Risk Evaluation for Asbestos, Supplemental File: Occupational Exposure Calculations (Chlor-Alkali)* ([U.S. EPA, 2019b](#))

Consumer Exposures

j. *Draft Risk Evaluation for Asbestos, Supplemental File: Consumer Exposure Calculations* ([U.S. EPA, 2019a](#))

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## Appendix C Conditions of Use Supplementary Information

EPA identified and verified uses of asbestos throughout the scoping, PF, and risk evaluation stages. As explained in the PF document, EPA believes that most asbestos imports listed by Harmonized Tariff Schedule (HTS) code in government and commercial trade databases are likely misreported and are not ongoing COU. EPA has been working with federal partners to better understand the asbestos-containing product import information. In coordination with Customs and Border Protection (CBP), EPA has reviewed available import information for the following asbestos Harmonized Tariff Schedule (HTS) codes:

- 2524.90.0045 Chrysotile Milled Fibers, Group 4 And 5 Grades
- 2524.90.0055 Chrysotile Milled Fibers, Other
- 6812.92.0000 Asbestos, Fibers, Fabricated, Paper, Millboard and Felt
- 6812.93.0000 Asbestos, Fiber, Compressed, Jointing, in Sheets or Rolls
- 6812.99.0003 Asbestos, Fabricated, Cords and String, whether or not Plaited
- 6812.99.0020 Asbestos, Fibers, Fabricated, Gaskets, Packing and Seals
- 6812.99.0055 Asbestos, Fibers, Fabricated, Other
- 6813.20.0010 Asbestos, Mineral Subst, Friction Mat, Brake Lin/Pad, Civil Air
- 6813.20.0015 Asbestos, Mineral Subst, Friction Mat, Brake Linings And Pads
- 6813.20.0025 Asbestos, Mineral Subst, Friction Mat, Other

CBP provided import data for the above asbestos HTS codes in CBP's Automated Commercial Environment (ACE) system, which provided information for 26 companies that reported the import of asbestos-containing products between 2016 and 2018. EPA contacted these 26 companies in order to verify the accuracy of the data reported in ACE. Of these 26 companies, 22 companies confirmed that the HTS codes were incorrectly entered and one company could not be reached. Three companies confirmed that the HTS codes entered in ACE are correct. EPA received confirmation that the following asbestos-containing products are imported into the United States:

- **Gaskets for use in the exhaust for off-road utility vehicles**
  - 6812.99.0020 Asbestos, Fibers, Fabricated, Gaskets, Packing and Seals
- **Gaskets for sealing pipes and flanges**
  - 6812.93.0000 Asbestos, Fiber, Compressed, Jointing, in Sheets or Rolls
- **Brake linings for use in automobiles that are manufactured and then exported (not sold domestically)**
  - 6813.20.0015 Asbestos, Mineral Subst, Friction Mat, Brake Linings And Pads

Regarding the two HTS codes that represent raw chrysotile, one company imported asbestos as waste but reported it in ACE under the HTS code 2524.90.0055 (Chrysotile Milled Fibers, Other). EPA did not contact the two facilities that reported under HTS code 2524.90.0045 (Chrysotile Milled Fibers, Group 4 And 5 Grades) because these entries were from a chloralkali company, which has already confirmed import and use of raw chrysotile.

## Appendix D Releases and Exposure to the Environment Supplementary Information

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### Toxics Release Inventory Data

A source of information that EPA considered in evaluating exposure is data reported under the Toxics Release Inventory (TRI) program. TRI reporting by subject facilities is required by law to provide information on releases and other waste management activities of Emergency Planning and Community Right-to-Know Act (EPCRA) Section 313 chemicals (i.e., TRI chemicals) to the public for informed decision making and to assist the EPA in determining the need for future regulations. Section 313 of EPCRA and Section 6607 of the Pollution Prevention Act (PPA) require certain facilities to report release and other waste management quantities of TRI-listed chemicals annually when a reporting threshold is triggered, but these statutes do not impose any monitoring burden for determining the quantities.

TRI data are self-reported by the subject facility where some facilities are required to measure or monitor emission or other waste management quantities due to regulations unrelated to the TRI Program, or due to company policies. These existing, readily available data are often used by facilities for TRI reporting purposes. When measured (e.g., monitoring) data are not “readily available,” or are known to be non-representative for TRI reporting purposes, the TRI regulations require that facilities determine release and other waste management quantities of TRI-listed chemicals by making “reasonable estimates.” Such reasonable estimates include a variety of different approaches ranging from published or site-specific emission factors (e.g., AP-42), mass balance calculations, or other engineering estimation methods or best engineering judgement. TRI reports are then submitted directly to EPA on an annual basis and must be certified by a facility’s senior management official that the quantities reported to TRI are reasonable estimates as required by law.

Under EPCRA Section 313, asbestos (friable) is a TRI-reportable substance effective January 1, 1987. For TRI reporting, facilities in covered sectors are required to report releases or other waste management of only the friable form of asbestos, under the general CASRN 1332-21-4. TRI interprets “friable” under EPCRA Section 313, referring to the physical characteristic of being able to be crumbled, pulverized or reducible to a powder with hand pressure, and “asbestos” to include the six types of asbestos as defined under Title II of TSCA<sup>35</sup>. Facilities are required to report if they are in a covered industrial code or federal facility and manufacture (including import) or process more than 25,000 pounds of friable asbestos, or if they otherwise use more than 10,000 pounds of friable asbestos.

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<sup>35</sup> According to 53FR4519 (VII)C(5), “The listing for asbestos is qualified by the term “friable.” This term refers to a physical characteristic of asbestos. EPA interprets “friable” as being crumbled, pulverized, or reducible to a powder with hand pressure. Again, only manufacturing, processing, or use of asbestos in the friable form triggers reporting. Similarly, supplier notification applies only to distribution of friable asbestos.”

Table APXD-1 provides production-related waste management data for friable asbestos reported by facilities in covered sectors to the TRI program from reporting years 2015 to 2018<sup>36</sup>. This is an updated table from that reported in the PF document. In reporting year 2018, 43 facilities reported a total of approximately 32 million pounds of friable asbestos waste managed. Of this total, zero pounds were recovered for energy or recycled, approximately 46,000 pounds were treated, and over 32 million pounds were disposed of or otherwise released into the environment.

**Table APX D-2** provides a summary of asbestos TRI releases to the environment for the same reporting years as Table APXD-1. *There were zero pounds of friable asbestos reported as released to water via surface water discharges*, and a total of 171 pounds of air releases from collective fugitive and stack air emissions reported in 2018. The vast majority of friable asbestos was disposed of to Resource Conservation and Recovery Act (RCRA) Subtitle C landfills and to landfills other than RCRA Subtitle C. Of the 153,947 pounds of friable asbestos reported in 2018 as “other releases”, 90,640 pounds were sent off-site to a waste broker for disposal, 14,760 pounds were sent off-site for storage only, and 48,547 pounds were sent off-site for other off-site management.

**Table APX D-1. Summary of Asbestos TRI Production-Related Waste Managed from 2015-2018 (lbs)**

Year	Number of Facilities	Recycling	Energy Recovery	Treatment	Releases <sup>a,b,c</sup>	Total Production Related Waste
2015	38	0	0	188,437	33,446,648	33,635,084
2016	40	2	0	31,993	25,971,339	26,003,335
2017	38	0	0	179,814	30,434,703	30,616,517
2018	43	0	0	46,106	32,329,759	32,375,865

Data source: 2015-2018 TRI Data (Updated November 2019) ([U.S. EPA, 2017d](#)).

<sup>a</sup> Terminology used in these columns may not match the more detailed data element names used in the TRI public data and analysis access points.

<sup>b</sup> Does not include releases due to one-time events not associated with production such as remedial actions or earthquakes.

<sup>c</sup> Counts all releases including release quantities transferred and release quantities disposed of by a receiving facility reporting to TRI.

While production-related waste managed shown in Table APXD-1. excludes any quantities reported as catastrophic or one-time releases (TRI section 8 data), release quantities shown in **Table APX D-2** include both production-related and non-routine quantities (TRI section 5 and 6 data) for 2015-2018. As a result, release quantities may differ slightly and may further reflect differences in TRI calculation methods for reported release range estimates ([U.S. EPA, 2017d](#)).

<sup>36</sup> Reporting year 2018 is the most recent TRI data available. Data presented were queried using TRI Explorer and uses the 2018 National Analysis data set (released to the public in November 2019). This dataset includes revisions for the years 1988 to 2018 processed by EPA.

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**Table APX D-2. Summary of Asbestos TRI Releases to the Environment from 2015-2018 (lbs)**

Year	Number of Facilities	Air Releases		Water Releases	Land Disposal			Other Releases <sup>a</sup>		Total On- and Off-Site Disposal or Other Releases <sup>b, c</sup>
		Stack Air Releases	Fugitive Air Releases		Class I Under-ground Injection	RCRA Subtitle C Landfills	All other Land Disposal <sup>a</sup>			
Totals 2015	38	101	208	0	0	9,623,957	24,029,820	0		33,654,087
		310			33,653,777					
Totals 2016	40	178	106	0	0	8,759,578	17,826,852	0		26,586,715
		285			26,586,430					
Totals 2017	38	80	67	0	0	6,199,224	24,802,748	0		31,002,120
		147			31,001,972					
Totals 2018	43	96	75	0	0	10,599,587	21,657,453	15,394		32,411,158
		171			32,257,040					

Data source: 2015-2018 TRI Data (Updated November 2019) ([U.S. EPA, 2017d](#)).  
<sup>a</sup> Terminology used in these columns may not match the more detailed data element names used in the TRI public data and analysis access points.  
<sup>b</sup> These release quantities do include releases due to one-time events not associated with production such as remedial actions or earthquakes.  
<sup>c</sup> Counts release quantities once at final disposition, accounting for transfers to other TRI reporting facilities that ultimately dispose of the chemical waste.

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**The Clean Water Act and the Safe Drinking Water Act**

**Background (Numeric Criteria and Reportable Levels)**

The Clean Water Act (CWA) requires that states adopt numeric criteria for priority pollutants for which EPA has published recommended criteria under section 304(a). States may adopt criteria that EPA approves as part of the state’s regulatory water quality standards. Once states adopt criteria as water quality standards, the CWA requires that National Pollutant Discharge Elimination System (NPDES) discharge permits include effluent limits as stringent as necessary to meet the standards [CWA section 301(b)(1)(C)]. If state permit writers determine that permit limits are needed, they will determine the level of pollutant allowed to ensure protection of the receiving water for a designated use. This is the process used under the CWA to address risk to human health and aquatic life from exposure to a pollutant in ambient waters.

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EPA develops recommended ambient water quality criteria for pollutants in surface water that are protective of aquatic life or human health designated uses with specific recommendations on the

8717 duration and frequency of those concentrations under section 304(a) of the CWA. These criteria are  
8718 based on priorities of states and others, and a subset of chemicals are identified as “priority pollutants”.  
8719 EPA has identified asbestos as a priority pollutant for which a nationally recommended human health  
8720 water quality criteria for asbestos of 7 MFL has been developed. EPA has not developed a nationally  
8721 recommended water quality criteria for the protection of aquatic life for asbestos, yet EPA may publish  
8722 aquatic life criteria for asbestos in the future if it is identified as a priority under the CWA.

8723 EPA’s National Primary Drinking Water Regulations (NPDWR), established under the Safe Drinking  
8724 Water Act (SDWA), are legally enforceable primary standards and treatment techniques that apply to  
8725 public water systems. Primary standards and treatment techniques protect public health by limiting the  
8726 levels of contaminants in drinking water. The Maximum Contaminant Level (MCL) for asbestos under  
8727 the Safe Drinking Water Act is 7 million fibers per liter, or MFL, for fibers > 10 micrometers. EPA has  
8728 set this level of protection based on the best available science at the time the NPDWR was promulgated  
8729 to prevent potential health problems and considering any limitations in both the feasible treatment  
8730 methods to remove a contaminant and availability of analytical methods to reliably measure the  
8731 occurrence of the contaminant in water. In the case of asbestos, the MCL was set based entirely on the  
8732 health goal since feasible treatment methods and analytical methods were available to achieve the  
8733 protective level of 7 MFL. Public water systems are required to sample each entry point into the  
8734 distribution system for asbestos at least once every 9 years. Transmission electron microscopy is used  
8735 for detection (EPA 800/4-83-043). The detection limit is 0.01 MFL. Here are links to the [analytical](#)  
8736 [standards](#) and the [drinking water regulations](#).

8737  
8738 The Phase II Rule, the regulation for asbestos, became effective in 1992. The Safe Drinking Water Act  
8739 requires EPA to review the national primary drinking water regulation for each contaminant every six  
8740 years and determine if the NPDWR is a candidate for revision, at that time. EPA reviewed asbestos as  
8741 part of the Six Year Review and determined that the 7 MFL for asbestos is still protective of human  
8742 health.

8743  
8744 As discussed in the PF document, because the drinking water exposure pathway for asbestos is currently  
8745 addressed in the SDWA regulatory analytical process for public water systems, this pathway (drinking  
8746 water for human health) will not be evaluated in this draft RE.

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8748 EPA issues Effluent Limitations Guidelines and Pretreatment Standards which are national regulatory  
8749 standards for industrial wastewater discharges to surface waters and publicly owned treatment works, or  
8750 POTWs (municipal sewage treatment plants). EPA issues Effluent Limitations Guidelines and  
8751 Pretreatment Standards for categories of existing sources and new sources under Title III of the [Clean](#)  
8752 [Water Act](#). The standards are technology-based (i.e., they are based on the performance of treatment and  
8753 control technologies); they are not based on risk or impacts upon receiving waters. (See [effluent](#)  
8754 [guidelines](#)).

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8756 The Effluent Limitations Guidelines and Pretreatment Standards for the Asbestos Manufacturing Point  
8757 Source Category (40 CFR Part 427) do not require that industrial facilities monitor asbestos  
8758 concentrations in discharges. Rather, the regulations contain either a zero discharge of pollutants  
8759 standard or require that the discharger not exceed a specified release amount of pollutants including total  
8760 suspended solids (TSS), chemical oxygen demand (COD) and pH. These guidelines were originally  
8761 developed in 1974 and 1975 and were revised in 1995. These guidelines cover legacy uses such as  
8762 manufacture of asbestos cement pipe, asbestos cement sheet, roofing, paper, etc. and may not be  
8763 particularly useful to the COU of asbestos. Additionally, there are effluent guidelines for the chlor-alkali  
8764 industry under 40 CFR Part 415 that cover pollutants such as chlorine, mercury, and lead, but they are

not specific to asbestos. *The EPA Industrial Waste Water Treatment Technology Database does not currently include any data for asbestos ([link to database](#)).*

**Reasonably Available Data from Water Release Databases and Other Information**

EPA investigated industry sector, facility, operational, and permit information regulated by NPDES under the Clean Water Act to identify any permit limits, monitoring and reporting requirements, and any discharge provisions related to asbestos and its COU. The Clean Water Act section 402 specifies that point source pollutant dischargers into waters of the United States must obtain a permit to regulate that facility's discharge. NPDES permits are issued by states, tribes, or territories that have obtained EPA approval to issue permits or by EPA Regions in areas without such approval. Effluent limitations serve as the primary mechanism in NPDES permits for controlling discharges of pollutants to receiving waters and the NPDES permit data are cataloged into the Integrated Compliance Information System (ICIS) to track permit compliance and enforcement status. NPDES permittees must then submit Discharge Monitoring Reports (DMRs) to the appropriate permitting authority on a periodic basis to ensure compliance with discharge standards for water quality and human health. Note that EPA does not currently have data available on facilities that indirectly discharge wastewater to POTWs.

Available discharge data and permit information was accessed through EPA's Envirofacts and Enforcement Compliance History Online (ECHO) database systems. EPA then investigated these data sources for information pertinent to asbestos COU (chlor-alkali plants, sheet gasket stamping and titanium dioxide plants) to identify if there is evidence of asbestos discharges or concentrations and/or violations of their wastewater permits.

**ICIS-NPDES information.** ICIS-NPDES is an information management system maintained by EPA to track permit compliance and enforcement status of facilities regulated by the NPDES under the Clean Water Act. ICIS-NPDES is designed to support the NPDES program at the state, regional, and national levels, and contains discharge monitoring and permit data from facilities in all point source categories who discharge directly to receiving streams.

EPA identified pollutant parameter codes in ICIS-NPDES specific to asbestos (such as asbestos, fibrous asbestos, asbestos (chrysotile), asbestos (amphibole), asbestos fibers (ambiguous asbestos), and non-chrysotile, non-amphibole asbestos fibers) and identified unique NPDES-permitted facilities, outfalls, and locations for those asbestos parameters. EPA then cross-checked their identified standard industrial codes (SIC) with SIC codes associated with the current asbestos users and COU. *The results were that none of these identified SIC codes were associated with current asbestos COU and were not considered relevant for risk evaluation purposes.*

EPA next did a specific NPDES permit search for facilities that may release asbestos (chlor-alkali and sheet gasket facilities) based on gathered location and addresses for these sites. It was found that most chlor-alkali facilities do have issued NPDES permits for industrial (major and minor permit status) operations and for general stormwater and construction stormwater projects. Yet for the identified permits for these industrial subcategories, none of the NPDES limits/monitoring requirements contained asbestos or asbestos-related parameters codes or any direct effluent screening information for asbestos. *Based on the analysis, EPA found no current surface water releases of asbestos or exceedances in the ICIS-NPDES database.*

**EPA's Water Pollutant Loading Tool.** EPA's Water Pollutant Loading Tool calculates pollutant loadings from NPDES permit and Discharge Monitoring Report (DMR) data from EPA's ICIS-NPDES for industrial and municipal point source dischargers. Data are available from the year 2007 to the



8814 present and also include wastewater pollutant discharge data from EPA's Toxics Release Inventory  
8815 (TRI). The Loading Tool was transitioned into ECHO to increase user access to data and streamline site  
8816 maintenance and EPA retired the legacy site (the Discharge Monitoring Report Loading Tool) on  
8817 January 24, 2018. DMR data identifies the permit conditions or limits for each water discharge location,  
8818 the actual values, identified by the permittee, for each monitored pollutant that was discharged, and  
8819 whether or not the amounts discharged exceeded the permit limits.

8820  
8821 DMR was used to help identify facilities with current uses that discharge asbestos to surface water.  
8822 Information was obtained from the DMR Pollutant loading tool accessed on December 1, 2017.  
8823 Facilities were identified using two different search methods: 1) "EZ Search" which identifies facilities  
8824 that submit Discharge Monitoring Reports (DMRs) and 2) "Toxics Release Inventory (TRI) Search"  
8825 which identifies facilities that report releases to the TRI. Searches were conducted for the two most  
8826 current (and complete) years in the tool: 2015 and 2016 for DMR facilities, and 2014 and 2015 for TRI  
8827 facilities.

8828  
8829 TRI data indicate no releases of asbestos in 2014 and 2015 (only friable asbestos is subject to reporting).  
8830 The DMR database reported just one facility reporting a discharge in 2014 and 2015 (accessed on  
8831 December 1, 2017) and this facility has been identified as a mining facility in Duluth, Minnesota. Later,  
8832 in a subsequent search (October 10, 2018) this facility was no longer identified on the DMR. The DMR  
8833 reported a total of zero pounds released in 2014 and 2015 but did provide maximum and average  
8834 effluent concentrations (mg/L) of allowable asbestos. It is assumed that the entry referred to mining  
8835 runoff, since asbestos has not been mined or otherwise produced in the United States since 2002. *EPA*  
8836 *has currently not identified in the existing literature or through consultation with industry any evidence*  
8837 *of discharge to surface water from DMR or TRI database as to any current uses of asbestos (release*  
8838 *from sheet gaskets, release from working on industrial friction products and/or release from asbestos*  
8839 *diaphragms from chlor-alkali facilities). Based on this database no water dischargers were established.*

8840  
8841 EPA did a search of the database for the parameter description of asbestos and identified three facilities  
8842 reporting actual limit values of discharge of asbestos to surface water. One of facilities was the mining  
8843 facility identified earlier on DMR and the other was a quarry. The third was an electric facility. Two  
8844 other electric facilities were also reported. These facilities were not directly related to the current uses of  
8845 asbestos mentioned earlier.

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8847 **STORET.** STORET refers overall to "STORage and RETrieval", an electronic data system for water  
8848 quality monitoring data developed by EPA. Since about 2000, STORET has referred to a local data  
8849 management system ("Modernized STORET") as well as data repository ("STORET Data Warehouse")  
8850 developed for purposes of assisting data owners to manage data locally and share data nationally. Until  
8851 September 2009, the distributed STORET database has been used to compile data at the national level in  
8852 the STORET Data Warehouse. As of September 2009, the Water Quality Exchange, or WQX  
8853 framework, provides the main mechanism for submitting data to the STORET Data Warehouse.

8854  
8855 *EPA did not identify in STORET any evidence of discharge to surface water for the COUs of asbestos.*  
8856 *EPA also did not identify in the existing literature or through consultation with industry any evidence of*  
8857 *discharge to surface water.*

## Appendix E Ecological Data Extraction Tables

The EPA has reviewed acceptable ecotoxicity studies for Chrysotile Asbestos according to the data quality evaluation criteria found in the *Application of Systematic Review in TSCA Risk Evaluations* (U.S. EPA, 2018a). The ten “on-topic” ecotoxicity studies for asbestos included data from aquatic organisms (i.e., vertebrates, invertebrates, and plants) and terrestrial species (i.e., fungi and plants). Following the data quality evaluation, EPA determined that four “on-topic” aquatic vertebrates and invertebrate studies were acceptable while the two “on-topic” aquatic plants studies were unacceptable as summarized in the Table APX E-1 below. In the PF, it was determined that the terrestrial exposure pathways, including biosolids, to environmental receptors was not within the scope of this assessment. As a result, EPA excluded three studies on terrestrial species from further analysis as terrestrial exposures were not expected under the conditions of use for asbestos. One amphibian study was excluded from further review because it was not conducted on chrysotile asbestos. Ultimately four aquatic toxicity studies were used to characterize the effects of chronic exposure of chrysotile asbestos to aquatic vertebrates and invertebrates, as summarized in Table 3-1 Environmental Hazard Characterization of Chrysotile Asbestos.

The results of these ecotoxicity study evaluations can be found in *Chrysotile Asbestos (CASRN 1332-21-4) Systematic Review: Supplemental File for the TSCA Risk Evaluation Document*. The data quality evaluation indicated these studies are of high confidence and are used to characterize the environmental hazards of Chrysotile Asbestos. The results of these studies indicate that there are adverse effects to aquatic organisms following exposure to chrysotile asbestos.

**Table APX E-1. Summary Table On-topic Aquatic Toxicity Studies That Were Evaluated for Chrysotile Asbestos.**

Species	Freshwater/ Salt Water	Duration	End- point	Concentration (s)  (MFL= Millions of fibers per liter)	Effect(s)	Reference	Data Quality Evaluation Rating
Asiatic Clams ( <i>Corbicula</i> sp.)	Freshwater	30d	LOEC ≤ 10 <sup>8</sup> fibers/L  (100 MFL)	10 <sup>8</sup> fibers/L  100 MFL	Gill Tissue Altered	<a href="#">(Belanger et al., 1986b)</a>	High
		30d	Reproduct ive LOEC = 10 <sup>4</sup> fibers/L  (0.01MFL )	10 <sup>4</sup> -10 <sup>8</sup> fibers/L  0.01-100 MFL	Increase in Larvae mortality/ decrease in larvae released		
		96hr-30d	No mortality observed; NOEC >10 <sup>8</sup> fibers/L	10 <sup>2</sup> -10 <sup>8</sup> fibers/L  0.0001-100 MFL	Mortality		

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Species	Freshwater/ Salt Water	Duration	End-point	Concentration (s)  (MFL= Millions of fibers per liter)	Effect(s)	Reference	Data Quality Evaluation Rating
			(>100 MFL)				
		30d	LOEC= 10 <sup>8</sup> fibers/L  (100 MFL)	10 <sup>2</sup> -10 <sup>8</sup> fibers/L  0.0001-100 MFL	Growth		
		30d	NOEC < 10 <sup>8</sup> fibers/L (<100 MFL)  LOEC = 10 <sup>8</sup> fibers/L  (100 MFL)	10 <sup>2</sup> -10 <sup>8</sup> fibers/L  0.0001-100 MFL	Fiber Accumulation		
		96hr-30d	LOEC = 10 <sup>2</sup> fibers/L (0.0001 MFL)	10 <sup>2</sup> -10 <sup>8</sup> fibers/L  0.0001-100 MFL	Siphoning Activity		
Asiatic Clams ( <i>Corbicula fluminea</i> )	Freshwater	30d	LOEC ≤ 10 <sup>2</sup> fibers/L  (≤0.0001 MFL)	10 <sup>2</sup> -10 <sup>8</sup> fibers/L  0.0001-100 MFL	Reduction in siphoning activity	( <a href="#">Belanger et al., 1986a</a> )	High
		30d	LOEC ≤ 10 <sup>8</sup> fibers/L  (≤ 100 MFL)	10 <sup>8</sup> fibers/L  100 MFL	Presence of asbestos in tissues		
Coho Salmon ( <i>Onchorhynchus kisutch</i> )	Saltwater and freshwater	40-86d	NOEC = 1.5x10 <sup>6</sup> fibers/L (1.5 MFL)  LOEC = 3.0x10 <sup>6</sup> fibers/L  (3 MFL)	1.5x10 <sup>6</sup> fibers/L, 3.0x10 <sup>6</sup> fibers/L  1.5 MFL, 3MFL	Behavioral stress (aberrant swimming, loss of equilibrium)  Sublethal effects including: epidermal hypertrophy superimposed on hyperplasia, necrotic epidermis, lateral line degradation, and lesions near the branchial region	( <a href="#">Belanger et al., 1986c</a> )	High

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Species	Freshwater/ Salt Water	Duration	End- point	Concentration (s)  (MFL= Millions of fibers per liter)	Effect(s)	Reference	Data Quality Evaluation Rating
		40-86d	No significant Mortality;  NOEC >3.0x10 <sup>6</sup> fibers/L  (>3 MFL)	1.5x10 <sup>6</sup> fibers/L, 3.0x10 <sup>6</sup> fibers/L  1.5 MFL, 3MFL	Mortality		
		40-86d	No Significant effect; NOEC >3.0x10 <sup>6</sup> fibers/L  (>3 MFL)	1.5x10 <sup>6</sup> fibers/L, 3.0x10 <sup>6</sup> fibers/L  1.5 MFL, 3MFL	Growth		
Green Sunfish ( <i>Lepomis cyanellus</i> )	Freshwater	52-67d	NOEC <1.5x10 <sup>6</sup> fibers/L (<1.5 MFL)  LOEC = 1.5x10 <sup>6</sup> fibers/L  (1.5 MFL)	1.5x10 <sup>6</sup> fibers/L,	Behavioral stress  (aberrant swimming, loss of equilibrium)  Sublethal effects including: epidermal hypertrophy superimposed on hyperplasia, necrotic epidermis, lateral line degradation, and lesions near the branchial region		
		40-86d	No significant Mortality;  NOEC >3.0x10 <sup>6</sup> fibers/L  (3 MFL)	1.5x10 <sup>6</sup> fibers/L, 3.0x10 <sup>6</sup> fibers/L  1.5 MFL, 3MFL	Mortality		
Japanese Medaka ( <i>Oryzias latipes</i> )	Saltwater and freshwater	13-21d	No significant effects; NOEC >10 <sup>6</sup> fibers/L	10 <sup>6</sup> -10 <sup>10</sup> fibers/L  1 MFL-10,000 MFL	Egg development, hatchability, survival.	( <a href="#">Belanger et al., 1990</a> )	High

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Species	Freshwater/ Salt Water	Duration	End- point	Concentration (s)  (MFL= Millions of fibers per liter)	Effect(s)	Reference	Data Quality Evaluation Rating
			(>1 MFL)				
		28d	LOEC = 10 <sup>6</sup> fibers/L  (1 MFL)  NOEC = 10 <sup>4</sup> fibers/L  (0.01 MFL)	10 <sup>6</sup> -10 <sup>10</sup> fibers/L  1 MFL-10,000 MFL	Significant reduction in growth of larval individuals		
		7w	Not statistical ly analyzed	10 <sup>4</sup> -10 <sup>8</sup> fibers/L  0.01-100 MFL	Reproductive performance (viable eggs/day, nonviable eggs/day)		
		49d	LC <sub>100</sub> =10 <sup>10</sup> fibers/L	10 <sup>10</sup> fibers/L  10,000 MFL	100% Larval mortality		
Duckweed ( <i>Lemna gibba</i> )	Freshwater	28d	LOEC = 0.5µg chrysotile /frond  NOEC < 0.5µg chrysotile /frond	0.5-5.0 µg chrysotile/frond  0.5-5.0 µg chrysotile/frond  0.5-5.0 µg chrysotile/frond  0.5-5.0 µg chrysotile/frond  0.5-5.0 µg chrysotile/frond  0.5-5.0 µg chrysotile/frond  0.5-5.0 µg chrysotile/frond  0.5-5.0 µg chrysotile/frond  0.5-5.0 µg chrysotile/frond  0.5-5.0 µg chrysotile/frond  0.5-5.0 µg chrysotile/frond	Decreased # fronds  Decreased Root length  Decreased Chlorophyll Content  Decreased Carotenoid content  Decrease in biomass/ frond  Decreased Protein content (mg/g fresh wt)  Decreased Free sugar (mg/g fresh wt)  Decreased Starch (mg/g fresh wt)  Decreased photosynthetic pigments  Increased lipid peroxidation  Increased cellular hydrogen peroxide levels	(2007; Trivedi et al., 2004)	Unacceptable

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Species	Freshwater/ Salt Water	Duration	End- point	Concentration (s)  (MFL= Millions of fibers per liter)	Effect(s)	Reference	Data Quality Evaluation Rating
				0.5-5.0 µg chrysotile/mL	Increase in catalase activity		

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## Appendix F Environmental Fate Data Extraction Table

### Environmental Fate Study Summary for Asbestos

**Table\_APX F-1. Other Fate Endpoints Study Summary for Asbestos**

System	Study Type (year)	Results	Comments	Affiliated Reference	Data Quality Evaluation Results of Full Study Report
Non guideline, experimental study; the effect of lichen colonization on chrysotile structure is investigated by analyzing the composition of both colonized and uncolonized field samples. The effect of oxalic acid exposure on chrysotile structure is also investigated at various concentrations.	Chrysotile fibers were incubated in oxalic acid solutions for 35 days to observe its effect on MgO content. Chrysotile (both uncolonized or colonized by lichens) from 3 serpentinite outcrops and one asbestos cement roof were collected.	In the three asbestos outcrops and asbestos-cement roof, MgO content (wt %) was lower by 15-20% in lichen colonized chrysotile than in uncolonized chrysotile. Incubation in 50 mM oxalic acid transformed chrysotile fibers into "an amorphous powdery material, consisting mainly of pure silica", and without fibrous nature.	The reviewer agreed with this study's overall quality level.	<a href="#">(Favero-Longo et al., 2005)</a>	High
Non guideline, experimental study; oxalic acid and citric acid leaching of asbestos rich sediment	Asbestos rich sediment and a serpentine bedrock sample underwent leaching in 0.025 M oxalic acid and 0.017 M citric acid. Total elemental analysis was performed using inductively coupled plasma spectrometry (ICPS), individual fiber analysis was done using energy dispersive x-ray analysis (EDX) and a scanning and transmission electron microscope (STEM).	ICPS results showed citric acid was slightly more effective at removing most metals from the sediment samples than oxalic acid; however, EDX analysis of individual fibers showed Mg/Si ratios were reduced from 0.68-0.69 to 0.07 by oxalic acid and only to 0.38 by citric acid.	The reviewer agreed with this study's overall quality level.	<a href="#">(Schreier et al., 1987)</a>	High
Non-guideline, experimental study; decomposition study of asbestos in 25% acid or caustic solutions	Chrysotile, crocidolite, amosite, anthophyllite, actinolite, and tremolite asbestos fibers were dissolved in 25% acid or NaOH solution	Degradation in 25% HCl, acetic acid, H <sub>3</sub> PO <sub>4</sub> , H <sub>2</sub> SO <sub>4</sub> and NaOH, respectively was reported for Chrysotile (55.69, 23.42, 55.18, 55.75 and 0.99%), Crocidolite (4.38, 0.91, 4.37, 3.69 and 1.35%), Amosite	Due to limited information assessing the results were challenging.	<a href="#">(Speil and Leineweber, 1969)</a>	Unacceptable

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		(12.84, 2.63, 11.67, 11.35 and 6.97%), Anthophyllite (2.66, 0.60, 3.16, 2.73 and 1.22%), Actinolite (20.31, 12.28, 20.19, 20.38 and 9.25%) and Tremolite (4.77, 1.99, 4.99, 4.58 and 1.80%).			
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Table APX F-2. Hydrolysis Study Summary for Asbestos

Study Type (year)	pH	Temperature	Duration	Results	Comments	Affiliated Reference	Data Quality Evaluation Results of Full Study Report
Non-guideline, experimental study; dissolution of asbestos in water at various pH and temperatures.	7, 7, 7, 9, and 4 for experiments 1-5, respectively	44, 6, 25, 25, and 25°C for experiments 1-5, respectively	170 or 1024 hours	<p>170-hour study results evaluating Mg removal from Chrysotile (proportion of 1 layer): Experiments 1-4: 0.32-0.94. Experiment 5 (pH 4, 25°C): 8.84</p> <p>170-hour study results evaluating Si removal from Chrysotile (proportion of 1 layer): Experiments 1-4: 0.5-0.25. Experiment 5: 5.05.</p> <p>170-hour study results evaluating Mg removal from Crocidolite (proportion of 1 layer): Experiments 1-5: 0.42-1.80.</p> <p>170-hour study results evaluating Si removal from Crocidolite (proportion of 1 layer): 0.03-0.56.</p> <p>1024-hour results (proportion of one layer removed) for experiment 3 only: Chrysotile, Mg: 0.94; Si: 0.36 Crocidolite, Mg: 1.42; Si: 0.37</p>	The reviewer agreed with this study's overall quality level.	<a href="#">(Gronow, 1987)</a>	High
Non-guideline; dissolution study; sample size, temperature and pH evaluated; pH change over time compared for asbestos minerals, amosite and crocidolite and chrysotile	5.9-6.1 (initial)	5 to 45 °C	20 min; 1000 hours	Rate of dissolution is a function of surface area and temperature. Mg <sup>2+</sup> may be continuously liberated from fibers leaving a silica skeleton. The rate-controlling step was determined to be removal of brucite layer. Smaller particles liberated more magnesium.	The reviewer agreed with this study's overall quality level.	<a href="#">(Choi and Smith, 1972)</a>	High
Non guideline; experimental study; a particle	Not reported but	Not reported but held constant	3-5 days	Chrysotile in natural water acquires a negative surface charge by rapid	The reviewer agreed	<a href="#">(Bales and Morgan, 1985)</a>	High

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Study Type (year)	pH	Temperature	Duration	Results	Comments	Affiliated Reference	Data Quality Evaluation Results of Full Study Report
electrophoresis apparatus was used to monitor absorption properties of chrysotile asbestos aging in water	held constant			adsorption of natural organic matter (<1 day). Positively charged >Mg-OH <sup>2+</sup> sites are removed by dissolution in the outer brucite sheet resulting in exposure of underlying >SiO <sup>-</sup> sites.	with this study's overall quality level.		

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Table APX F-3. Aquatic Bioconcentration Study Summary for Asbestos

Study Type (year)	Initial Concentration	Species	Duration	Result	Comments	Affiliated Reference	Data Quality Evaluation Results of Full Study Report
Non-guideline; experimental study; uptake monitoring of chrysotile asbestos in Coho and juvenile green sunfish	$1.5 \times 10^6$ and $3.0 \times 10^6$ fibers/L	Coho salmon ( <i>Oncorhynchus kisutch</i> ) and juvenile green sunfish ( <i>Lepomis cyanellus</i> )	Coho salmon: 86 and 40 days; Green sunfish: 67 and 52 days	Asbestos fibers were found in the asbestos-treated fish by transmission electron microscopy (TEM); however total body burdens were not calculated. Sunfish lost scales and had epidermal tissue erosion. Asbestos fibers were not identified in control or blank samples.	The reviewer agreed with this study's overall quality level.	( <a href="#">Belanger et al., 1986c</a> )	High
Non-guideline; experimental study; uptake monitoring of chrysotile by Asiatic clams	$2.5 \times 10^8$ - $8.8 \times 10^9$ fibers/L	Asiatic clams ( <i>Corbicula</i> sp.)	96-hours and 30-days	Chrysotile asbestos was detected in clams at $69.1 \pm 17.1$ fibers/mg whole body homogenate after 96 hours of exposure to $10^8$ fibers/L and food. Chrysotile asbestos was detected in clams after 30 days of exposure to $10^8$ fibers/L at $147.3 \pm 52.6$ fibers/mg dry weight gill tissue and $903.7 \pm 122.9$ fibers/mg dry weight visceral tissue. Chrysotile asbestos was not detected in clams after 96 hours at all asbestos exposure concentrations tested with no food.	The reviewer agreed with this study's overall quality level.	( <a href="#">Belanger et al., 1986b</a> )	High
Non-guideline; experimental study; measuring uptake of chrysotile asbestos by Asiatic clams	0, $10^4$ , and $10^8$ fibers/L	Asiatic clams ( <i>Corbicula</i> sp., collected in winter and summer)	30-days	Fibers were not detected in clams from blank control groups and after exposure to $10^4$ fiber/L groups for 30 days. Asbestos concentration in tissue after exposure to $10^8$ fiber/L for 30 days	The reviewer agreed with this study's overall quality level.	( <a href="#">Belanger et al., 1986a</a> )	High

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				(fibers/mg dry weight tissue) in winter samples: Gills: 132.1±36.4; Viscera: 1055.1±235.9 and summer samples: Gill: 147.5±30.9; Viscera: 1127.4±190.2.			
Non-guideline; experimental study; BCF determination of asbestos in the Asiatic clam	0, 10 <sup>4</sup> , and 10 <sup>8</sup> fibers/L	Asiatic clam ( <i>corbicula</i> sp.)	30 day and field exposed	BCF = 0.308 in gill tissue, 1.89 in viscera tissue, and 1.91 in whole clam homogenates after 30-days exposure to 10 <sup>8</sup> fibers/L. Field exposed BCFs = 0.16-0.19 in gills, 64.9-102 in viscera, 1,442-5,222 in whole clams.	The reviewer agreed with this study's overall quality level.	<a href="#">(Belanger et al., 1987)</a>	High
Non-guideline; experimental study; chrysotile asbestos uptake study in Japanese Medaka	5.1±2.8×10 <sup>6</sup> , 7.6±8.1×10 <sup>8</sup> fibers/L	Japanese Medaka ( <i>Oryzias latipes</i> )	13 weeks	After 28 days of exposure to chrysotile asbestos at 10 <sup>10</sup> fibers/L concentrations, fish total body burden was 375.7 fibers/mg. After 3 months of exposure to chrysotile asbestos at 10 <sup>8</sup> fibers/L concentrations, fish total body burden was 486.4±47.9 fibers/mg.	The reviewer agreed with this study's overall quality level.	<a href="#">(Belanger et al., 1990)</a>	High

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## Appendix G SAS Codes for Estimating $K_L$ and $K_M$ from Grouped Data

---

```

8904
8905 /*This SAS code estimates a value for lung cancer potency (KL) using Poisson maximum likelihood
8906 estimation (MLE), along with the 90% confidence interval (CI) generated using the likelihood profile
8907 method. The basic model is  $RR = 1 + CE10 * KL$ .
8908
8909 This code was created by Rebekha Shaw and Bill Thayer at SRC Inc. This is version 1.0 */
8910
8911 /*This is where the code begins execution. */
8912 /*The first step is to create a data table */
8913 data Data_Table;
8914 input CE10_min CE10_max CE10_mid Observed Expected RR;
8915
8916 /*enter data here */
8917 datalines;
8918 0 20 10.0 6 5.75 1.04
8919 20 100 60.0 12 2.82 4.25
8920 100 450 275.0 17 1.57 10.82
8921 450 1097 773.5 21 1.23 17.07
8922 ;
8923
8924 /* Enter text string to identify data source */
8925 title "Wang et al 2013";
8926
8927 /*model*/
8928
8929 proc nlmixed data=Data_Table;
8930 parms KLE2 10; /* KLE2 =  $KL * 1E+02$ . The initial guess is 10. This can be changed if a solution is not
8931 found (unlikely). */
8932
8933 Predicted = (1+CE10_mid*KLE2/100)* Expected; /*equation to calculate predicted number of lung cancer
8934 cases*/
8935
8936 LL=LogPDF("POISSON",Observed,Predicted); /*LogPDF function Returns the logarithm of a probability
8937 density (mass) function. Poisson distribution is specified. */
8938
8939 model Observed ~ general(LL);
8940
8941 estimate 'KLE2' KLE2 ALPHA=0.1; /*generates "Additional Estimates" table in the Results tab with Wald 90%
8942 CI's*/
8943 predict Predicted out=Predicted alpha=0.1; /*generates SAS data table with predicted values and CI's
8944 titled "Predicted"*/
8945 ods output FitStatistics = FitStats;
8946 ods output ParameterEstimates = ModelParams;
8947
8948 Proc print data=Predicted; /*Prints the "Predicted" table in the Results tab*/
8949 run;
8950
8951 data _null_;
8952 set Fitstats;
8953 if _n_ =1;
8954 LLTarget = (Value/-2)-1.353; /*calculates LL_target - needed to run macro PoissonLLBounds*/
8955
8956 call symputx("LLTarget",LLTarget); /*creates macro variable*/
8957 run;
8958 data null ;
8959 set ModelParams;
8960
8961 KLMLE = Estimate*1e-02; /*variable KL_MLE in macro PoissonLLBounds*/
8962 KLINITLB= Estimate*1e-02/10; /*Calculates the initial guess for the lower bound - variable KL_itit_LB in
8963 macro poissonLLBounds*/
8964 KLINITUB= Estimate*1e-02*10; /*Calculates the initial guess for the upper bound - variable KL_itit_LB in
8965 macro PoissonLLBounds*/
8966 call symputx("KLMLE", KLMLE); /*creates macro variable*/
8967 call symputx("KLINITLB", KLINITLB); /*creates macro variable*/
8968 call symputx("KLINITUB", KLINITUB); /*creates macro variable*/

```

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```

8969 run;
8970
8971 /*This is the macro which calculates the 90% confidence interval using the likelihood profile method. It
8972 is executed after the MLE solution has been found */
8973 %macro PoissonLLBounds(inputData=, KL_MLE=, KL_Init_LB=, KL_Init_UB=,
8974 conv_criterion=, LL_target=, max_iteration=);
8975
8976 %let dsid=%sysfunc(open(&inputdata)); * open the input data file;
8977 %let NumSamples=%sysfunc(attrn(&dsid,nobs)); * get the number of observations;
8978 %let rc=%sysfunc(close(&dsid)); * close the data file;
8979
8980 %do j=1 %To 2; * one for upper bound and one for lower bound;
8981
8982 %If %eval(&J=1) %then %let KL=&KL_init_LB;
8983 %If %eval(&J=2) %then %let KL=&KL_Init_UB;
8984
8985 %let i=1; * first time through loop;
8986
8987 %let ConvFactor = 10;
8988 %let ConvRate = %sysevalf(((&KL_MLE-&KL)/&KL_MLE)/10);
8989
8990 %let ConvDirect = -1;
8991 /* negative=from the left and positive=from the right. For lower bound, the initial guess is less than
8992 the target LL so the initial value of convdirect is -1 */
8993
8994 %let KLAdjust=%Sysevalf(-1*&ConvDirect*&KL*&ConvRate);
8995
8996 %do %Until (%sysevalf(&DeltaLL < &conv_criterion) OR %sysevalf(&i > &max_iteration));
8997
8998 Data tempDataLLBound; Set &InputData;
8999 Predicted = (1 + CE10_Mid * &KL) * Expected;
9000 LL=(LogPDF("POISSON",Observed,Predicted)); * likelihood for each
9001 observation;
9002 LL_sum+LL;
9003 output;
9004 Run;
9005
9006 Data TempDataLLBound2; Set tempDataLLBound;
9007 If _N_ = &NumSamples;
9008 NumLoops=&i;
9009 thisKL=&KL;
9010 ConvRateVar=&ConvRate;
9011 ConvFactorVar=&ConvFactor;
9012 ConvDirectVar= %eval(&ConvDirect);
9013
9014 KLAdjustVar=(-1*ConvDirectVar)*thisKL*ConvRateVar;
9015 If &ConvDirect=-1 then DiffLL=abs(LL_sum)-abs(&LL_Target);
9016 Else DiffLL=abs(&LL_Target)-abs(LL_Sum);
9017
9018 /* Test if we have changed direction on the convergence. If we have, change direction
9019 (subtract from current value if we were adding before...) and decrease the convergence rate
9020 (ConvRate) by a factor = ConvFactor. */
9021
9022 if DiffLL<0 then
9023 do; /* need to change directions and make conv rate more gradual */
9024 ConvDirectVar= %eval(-1*&ConvDirect);
9025 ConvRateVar=%sysevalf(&convRate/&ConvFactor);
9026 KLAdjustVar=(-1*ConvDirectVar)*thisKL*ConvRateVar;
9027 call symput('KLAdjust',KLAdjustVar);
9028 call symput('ConvDirect',ConvDirectVar);
9029 call symput('convRate',ConvRateVar);
9030 end;
9031 AbsDiffLL=abs(DiffLL);
9032
9033 call symput('DeltaLL',AbsDiffLL);
9034
9035 output;
9036 Run;
9037
9038 Data tempAllOutput; if _N_=1 then Set TempDataLLBound2; Set tempDataLLBound; Run;
9039 %If %eval(&i=1) %then %do; Data AllOutput; Set tempAllOutput; Run; %end;
9040
9041 %If %eval(&i>1) %then %do; Proc Append base=AllOutput data=tempAllOutput; Run;
9042
9043 %End;

```

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```
9044
9045           %Let i=%eval(&i+1);
9046
9047           %Let KL=%sysevalf(&KL + &KLAdjust);
9048
9049           %End;
9050
9051           %If %eval(&J=1) %then
9052               %Do;
9053                   Data tempout1; length limit $5; Set TempDataLLBound2; limit='lower';
9054 estimate=thisKL; LogLikelihood=LL_sum; loops=numloops; Run;
9055               %End;
9056           %If %eval(&J=2) %then
9057               %Do;
9058                   Data tempout2; length limit $5; Set TempDataLLBound2; limit='upper';
9059 estimate=thisKL; LogLikelihood=LL_sum; loops=numloops; Run;
9060               %End;
9061 %End;
9062
9063           Data PrntOutput; Set tempout1 tempout2; run;
9064
9065           Proc print data=PrntOutput; var limit estimate LogLikelihood Loops ; Run;
9066
9067 %Mend;
9068
9069 /*run macro PoissonLLBounds*/
9070           %PoissonLLBounds(inputData=Data_Table,
9071                           KL_MLE=&KLMLE,
9072                           KL_Init_LB=&KLINITLB,
9073                           KL_Init_UB=&KLINITUB,
9074                           conv_criterion=0.001,
9075                           LL_target=&LLTarget,
9076                           max_iteration=100);
9077 run;
9078
9079 /*the following code creates a summary table with the MLE KLE and confidence bounds*/
9080 PROC SQL;
9081     CREATE TABLE WORK.MLEKL AS
9082     SELECT ("MLE KLE") AS Parameter,
9083            (t1.Estimate*1e-2) AS Value
9084     FROM WORK.MODELPARAMS t1;
9085 QUIT;
9086
9087 PROC SQL;
9088     CREATE TABLE WORK.LBKLUBKL AS
9089     SELECT (case
9090            when t1.limit="lower" then "5% LB KL"
9091            else "95% UB KL"
9092            end) AS Parameter,
9093            t1.estimate AS Value
9094     FROM WORK.PRNTOUTPUT t1;
9095 QUIT;
9096
9097 PROC SQL;
9098     CREATE TABLE WORK.Parameter_Values AS
9099     SELECT * FROM WORK.MLEKL
9100     OUTER UNION CORR
9101     SELECT * FROM WORK.LBKLUBKL
9102     ;
9103 Quit;
9104
9105 Proc print data=Work.Parameter_values;
9106 run;
9107
```

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```
9108 /*This SAS code estimates a value for mesothelioma potency (KM) using Poisson maximum likelihood estimation (MLE), along with the 90% confidence interval (CI)
9109 generated using the likelihood profile method.
9110 This code was created by Rebekha Shaw and Bill Thayer at SRC Inc.
9111 This is version 1.0*/
9112
9113 /*This is where the code begins execution. */
9114 data Data_Table;
9115 input TSFE_Min TSFE_Max TSFE_Mid Duration Conc PY Obs ;
9116 /*The values of TSFE_Mid and Duration are used to calculate a parameter called Q. */
9117 if TSFE_Mid=. then Q = .;
9118 else if TSFE_Mid<10 then Q = 0;
9119 else if TSFE_Mid>(10+duration) then
9120     Q = (TSFE_Mid-10)**3-(TSFE_Mid-10-duration)**3;
9121 else Q = (TSFE_Mid-10)**3;
9122
9123 /*enter data here. The contents of the columns are as follows:
9124
9125 TSFE_Min (years)
9126 SFE_Max (years)
9127 TSFE_Mid (years)
9128 Duration (years)
9129 Conc (f/cc)
9130 Person Years (PY)
9131 Observed cases(Obs)
9132
9133 */
9134 datalines;
9135 20 30 27.7 1.00 6.5 1926 0
9136 30 40 33.9 2.10 8.7 6454 0
9137 40 50 43.1 3.00 14.6 3558 2
9138 50 72 53.56 5.78 31.4 1080 2
9139 ;
9140
9141 /*enter the name of the data set*/
9142 title "North Carolina Sub Co-hort (1999-2003;4 groups)";
9143 run;
9144 /*model*/
9145 proc nlmixed data= Data_Table;
9146 parms KME8 10; /*KME8 is equal to KM*1E+08. The starting guess is 10. This can be changed in the unexpected case where a solution is not found*/
9147 Pred = Conc*Q*PY*KME8/1e+08; /*equation to calculate predicted values*/
9148 LL=LogPDF("POISSON",Obs,Pred); /*LogPDF function Returns the logarithm of a probability density (mass) function. Poisson distribution is specified.*/
9149 model Obs ~ general(11);
9150
9151 estimate 'KME8' KME8 ALPHA=0.1; /*generates "Additional Estimates" table in the Results tab with 90% Wald CI's - this can be deleted if we do not want the Wald CIs
9152 displayed in the SAS output */
9153 predict Pred out=Predicted alpha=0.1; /*generates SAS data table with predicted values and CI's titled "Predicted"*/
9154 ods output FitStatistics = FitStats;
9155 ods output ParameterEstimates = ModelParams;
9156 run;
9157 Proc print data=Predicted; /*Prints the "Predicted" table in the Results tab*/
9158 OPTIONS MPRINT SYMBOLGEN ; /*this prints in the log what value is used for each variable in the macro*/
9159 run;
9160 data _null_ ;
9161 set Fitstats;
9162 if _n_ =1;
9163 LLTarget = (Value/-2)-1.353; /*calculates LL_target - needed to run macro PoissonLLBounds*/
9164
9165 call symputx("LLTarget",LLTarget); /*creates macro variable*/
9166 run;
9167 data _null_ ;
9168 set ModelParams;
9169
9170 KM_MLE = Estimate*1e-8; /*scales back the KM MLE value generated by Proc nlmixed - variable KM_MLE in macro PoissonLLBounds*/
9171 KMINITLB= Estimate*1e-8/10; /*Calculates the initial guess for the lower bound - variable KM_init_LB in macro poissonLLBounds*/
9172 KMINITUB= Estimate*1e-8*10; /*Calculates the initial guess for the upper bound - variable KM_init_LB in macro PoissonLLBounds*/
9173 call symputx("KM_MLE", KM_MLE); /*creates macro variable*/
9174 call symputx("KMINITLB", KMINITLB); /*creates macro variable*/
9175 call symputx("KMINITUB", KMINITUB); /*creates macro variable*/
9176 run;
9177
9178 /*This is the macro which calculates the 90% confidence interval using the likelihood profile method. It is executed after the MLE solution has been found */
9179
9180 %macro PoissonLLBounds(inputData=, KM_MLE=, KM_Init_LB=, KM_Init_UB=,
9181 conv_criterion=, LL_target=, max_iteration=);
```



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```

9183 %Let dsid=%sysfunc(open(&inputdata)); * open the input data file;
9184 %Let NumSamples=%sysfunc(attrn(&dsid,nobs)); * get the number of observations;
9185 %Let rc=%sysfunc(close(&dsid)); * close the data file;
9186
9187
9188
9189 %Do j=1 %To 2; * one for upper bound and one for lower bound;
9190
9191 %If %eval(&J=1) %then %Let KM=&KM_init_LB;
9192 %If %eval(&J=2) %then %Let KM=&KM_init_UB;
9193
9194 %Let i=1; * first time through loop;
9195
9196 %Let ConvFactor = 10;
9197 %Let ConvRate = %syssevalf(((&KM_MLE-&KM)/&KM_MLE)/10);
9198
9199 %Let ConvDirect = -1;
9200 /* negative=from the left and positive=from the right. For lower bound, the initial guess is less than the target LL so the initial value of convdirect is -1 */
9201
9202 %Let KMadjust=%Syssevalf(-1*&ConvDirect*&KM*&ConvRate);
9203
9204 %Do %Until (%syssevalf(&DeltaLL < &conv_criterion) OR %syssevalf(&i > &max_iteration));
9205
9206 Data tempDataLLBound; Set &InputData;
9207 E = Conc * Q * PY * &KM;
9208 LL=(LogPDF("POISSON",Obs,E)); * likelihood for each observation;
9209 LL_sum+LL;
9210 output;
9211 Run;
9212
9213 Data TempDataLLBound2; Set tempDataLLBound;
9214 If _N_ = &NumSamples;
9215 NumLoops=&i;
9216 thisKM=&KM;
9217 ConvRateVar=&ConvRate;
9218 ConvFactorVar=&ConvFactor;
9219 ConvDirectVar= %eval(&ConvDirect);
9220
9221 KMadjustVar=(-1*&ConvDirectVar)*thisKM*ConvRateVar;
9222 If &ConvDirect=-1 then DiffLL=abs(LL_sum)-abs(&LL_Target);
9223 Else DiffLL=abs(&LL_Target)-abs(LL_Sum);
9224
9225 /* Test if we have changed direction on the convergence. If we have, change direction (subtract from current value if we were adding before...)
9226 and decrease the convergence rate (ConvRate) by a factor = ConvFactor.*/
9227
9228 if DiffLL<0 then
9229 do; /* need to change directions and make conv rate more gradual */
9230 ConvDirectVar= %eval(-1*&ConvDirect);
9231 ConvRateVar=%syssevalf(&convRate/&ConvFactor);
9232 KMadjustVar=(-1*&ConvDirectVar)*thisKM*ConvRateVar;
9233 call symput('KMadjust',KMadjustVar);
9234 call symput('ConvDirect',ConvDirectVar);
9235 call symput('convRate',ConvRateVar);
9236 end;
9237 AbsDiffLL=abs(DiffLL);
9238
9239 call symput('DeltaLL',AbsDiffLL);
9240
9241 output;
9242 Run;
9243
9244 Data tempAllOutput; if _N_=1 then Set TempDataLLBound2; Set tempDataLLBound; Run;
9245
9246 %If %eval(&i=1) %then %do; Data AllOutput; Set tempAllOutput; Run; %end;
9247
9248 %If %eval(&i>1) %then %do; Proc Append base=AllOutput data=tempAllOutput; Run; %End;
9249
9250 %Let i=%eval(&i+1);
9251
9252 %Let KM=%syssevalf(&KM + &KMadjust);
9253
9254 %End;
9255
9256 %If %eval(&J=1) %then
9257 %Do;

```

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```
9258                                     Data tempout1; length limit $5; Set TempDataLLBound2; limit='lower'; estimate=thisKM; LogLikelihood=LL_sum;
9259 loops=numloops; Run;
9260                                     %End;
9261                                     %If %eval(&J=2) %then
9262                                     %Do;
9263                                     Data tempout2; length limit $5; Set TempDataLLBound2; limit='upper'; estimate=thisKM; LogLikelihood=LL_sum;
9264 loops=numloops; Run;
9265                                     %End;
9266 %End;
9267
9268 Data PrntOutput; Set tempout1 tempout2; run;
9269
9270 Proc print data=PrntOutput; var limit estimate LogLikelihood Loops ; Run;
9271
9272 %Mend;
9273
9274 /*run macro PoissonLLBounds*/
9275 %PoissonLLBounds(inputData=Data_Table,
9276
9277                                     KM_MLE=%KMMLE,
9278                                     KM_Init_LB=%KMINITLB,
9279                                     KM_Init_UB=%KMINITUB,
9280                                     conv_criterion=0.001,
9281                                     LL_target=%LLTarget,
9282                                     max_iteration=100);
9283 run;
9284
9285
9286
9287
9288
```

## Appendix H BEIR IV Equations for Life Table Analysis

---

### Lung Cancer

Let  $e_i$  be the calculated excess relative risk of lung cancer in an exposed individual at age  $i$ .

Then:

$$\text{Excess Lifetime Risk} = \text{Re}_{lt} - \text{R0}_{lt}$$

$$\text{R0}_{lt} = \sum_{i=1}^{110} \text{R0}_i$$

$$\text{Re}_{lt} = \sum_{i=1}^{110} \text{Re}_i$$

$$\text{R0}_i = \frac{h_i}{h_i^*} S_{1,i} (1 - q_i)$$

$$\text{Re}_i = \frac{he_i}{he_i^*} Se_{1,i} (1 - qe_i)$$

$$he_i = h_i (1 + e_i)$$

$$he_i^* = h_i^* + h_i e_i$$

$$q_i = \exp(-h_i^*)$$

$$qe_i = \exp(-he_i^*)$$

$$S_{1,i} = \prod_{j=1}^{i-1} q_j$$

$$Se_{1,i} = \prod_{j=1}^{i-1} qe_j$$

where:

$i$  and  $j$  = Year index (1 = year 0-1, 2 = year 1-2, etc.)

$\text{R0}_{lt}$  = Lifetime risk of lung cancer in the absence of exposure

$\text{Re}_{lt}$  = Lifetime risk of lung cancer in the presence of exposure

$\text{R0}_i$  = Risk of lung cancer in the absence of exposure in year  $i$

$\text{Re}_i$  = Risk of lung cancer the presence of exposure in year  $i$

$h_i$  = Lung cancer mortality rate in the absence of exposure in year  $i$

$h_i^*$  = All-cause mortality rate in the absence of exposure in year  $i$

$q_i$  = Probability of surviving year  $i$ , all causes acting (no exposure)

$qe_i$  = Probability of surviving year  $i$ , all causes acting (with exposure)

$S_{1,i}$  = Probability of surviving up to start of year  $i$ , all causes acting (no exposure)

$Se_{1,i}$  = Probability of surviving up to start of year  $i$ , all causes acting (with exposure)

9327 Mesothelioma

9328

9329 The same basic approach is followed for calculating lifetime risk of mesothelioma, except that the  
9330 baseline (un-exposed) risk is so small that it is generally assumed to be zero. Thus, the equations for  
9331 calculating lifetime mesothelioma risk are the same as above, except as follows:

9332

9333  $m_i =$  risk of mesothelioma in an exposed individual at age  $i$

9334

9335 
$$Re_t = \sum_{i=1}^{110} Re_i$$

9336

9337 
$$Re_i = \frac{m_i}{he_i^*} Se_{1,i}(1 - qe_i)$$

9338

9339

## Appendix I SAS Code for Life Table Analysis

### Lung Cancer Lifetable

```

9340
9341
9342
9343 /*
9344 This program calculates the risk of lung cancer from inhalation exposure to asbestos,
9345 using a lifetable approach. The basic model is  $RR = 1 + CE10 * KL$ .
9346
9347 The basic code for the lifetable calculations were developed and provided to EPA
9348 by Randall Smith at NIOSH. The code from NIOSH calculates the baseline risk (R0) and the exposed risk
9349 (Rx)
9350 from exposure to an exposure concentration of X_Level using NIOSH Model 2:  $Rx = R0 * (1 + COEF * X\_Level)$ .
9351 X_Level).
9352
9353 EPA has modified the NIOSH as follows:
9354 1) The all-cause and cause-specific (lung cancer) mortality data tables have been updated
9355 2) The NIOSH equation for X_Time has been corrected so all values are for the mid-point of the year:
9356 X_Time = min(max(0,(age+0.5-&Age1st_x-&Lag)),&Duration - 0.5 )
9357 3) An equation has been added to calculate extra risk: Extra_Risk = (Rx - R0) / ( 1 - R0)
9358 4) A macro has been added to find the exposure level (X_Level) that yields an extra risk of 0.01 (1%).
9359 This is referred to as EC1%, which may then be used to calculate the unit risk: UR = 0.01 / EC1%
9360 */
9361 /* .\Beta Version.sas 19jan00, 26jul00, 25oct01, 06dec05, 30nov18
9362 -----
9363 Experimental version
9364 ----- */
9365 title "Excess Risks using BEIR IV method to account for competing risks";
9366 title2 "Effects of airborne exposure to asbestos on lung cancer mortality rates";
9367 title3 "under a linear relative rate model ";
9368
9369 /*-----+
9370 | Compute excess risk by the BEIR IV method using SAS datasteps. |
9371 |
9372 | These programs compute the risk of a cause-specific |
9373 | death in the presence of competing risks, where the cause- |
9374 | specific death-rate is modeled either as a relative rate |
9375 | [h=h0*f(Coef*X)] or as an absolute rate [h=h0+f(Coef*X)] |
9376 | where |
9377 | h denotes the cause-specific death-rate, |
9378 | X denotes cumulative occupational exposure (with Lag) |
9379 | Coef denotes the coefficient for the effect of exposure and |
9380 | h0 is the corresponding rate at baseline (X=0). |
9381 | (Except for Coef, these are functions of age.) |
9382 |
9383 | A few simple models of f(Coef*X) are easily specified as |
9384 | described below. More complicated models can be specified with |
9385 | a little more work. (For a more complicated example, |
9386 | see \_GENERAL.LIB\PROGRAMS\SAS\BEIR-4.Method\BEIR4ex2.SAS). |
9387 |
9388 +Reference: +
9389 | Health Risks of Radon and Other Internally Deposited Alpha- |
9390 | Emitters (BEIR IV). Committee on the Biologic Effects of |
9391 | Ionizing Radiations. National Academy Press. Wash. DC (1988). |
9392 | See especially pages 131-136. |
9393 |
9394 +USER-SUPPLIED ASSIGNMENTS: +
9395 |
9396 |> The following macro variables are assigned using "%LET" state- |
9397 | ments: MODEL, COEF, LAG, AGE1ST_X, DURATION, LASTAGE. |
9398 | Further information appears below. |
9399 |> Exposure concentrations for computing risk are defined |
9400 | in the dataset "X_LEVELS." |
9401 |> All-cause mortality information is entered as a life-table in |
9402 | the data step "ALLCAUSE," and converted to rates per individual. |
9403 |> Cause-specific mortality information for unexposed referents is |
9404 | entered as rates per 100,000 and converted to rates per |
9405 | individual in the data step "CAUSE." |
9406 |
9407 +NOTES: +
9408 |> Datastep "EX_RISK" is where the desired risks are computed. |
9409 |

```

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```
9410 |> If the unexposed(referent) cause-specific mortality rate is from|
9411 | a model then datastep "CAUSE" with variables AGE and RATE as |
9412 | modeled can be modified to incorporate this. However, care |
9413 | must be taken in calculating confidence limits since imprecision|
9414 | in the estimates of all of the parameters of the model |
9415 | contributes to the imprecision of excess risk estimates. |
9416 | |
9417 |> This program is currently set up to apply the Linear Rel. Rate |
9418 | model (Lag= 0) and accumulation of excess risk is over the |
9419 | rates in ALLCAUSE and CAUSE unless truncated at a younger age. |
9420 | (See LASTAGE below.) |
9421 | |
9422 | |
9423 | + SAS Programmer: Randall Smith +
9424 | | The Nat'l Inst. for Occupational Safety & Health |
9425 | | 26jul2000, 23jul2001, 25oct2001, 18nov2018 |
9426 | + Modifications: +
9427 | +
9428 | | 26jul00 Fix the procedure bug causing it to report incorrectly |
9429 | | the age at which accumulation of risk was stopped |
9430 | | whenever the age-specific rates included ages |
9431 | | before the value of &Agelst_X. (&Agelst_X is a macro |
9432 | | expression defining the age exposure begins.) |
9433 | |
9434 | | 23jul01 Make changes to facilitate multiple applications of |
9435 | | BEIR4 algorithm, i.e., MLE(Excess Risk), UCL(ExcessRisk), |
9436 | | searching for concentrations for a fixed risk. These |
9437 | | changes involve defining Macros named BEIR4 and SEARCH |
9438 | | given below with code illustrating these uses for the |
9439 | | linear relative rate model. |
9440 | |
9441 | | 25oct01 Modified to add Macro variable EnvAdj for whether to |
9442 | | increase inhaled dose from intermittent occupational |
9443 | | exposures to continuous environmental exposures |
9444 | | and update US rates for Gibb et al. cohort. |
9445 | |
9446 | | 30nov18 A bug that prevented the calculation of excess risks |
9447 | | after incorporating an adjustment from intermittent |
9448 | | occupational exposures to continuous exposures is fixed. |
9449 | |
9450 | | +---|
9451 | | March 2019: BT (SRC) Added macro CONVERGE_BEIR4 which iteratively |
9452 | | runs macro BEIR4 until the EXPOSURE_CONCENTRATION corresponds to an |
9453 | | extra_risk=0.01 (the point of departure [POD]). |
9454 | |
9455 | | |
9456 | | Macro CONVERGE_BEIR4 works with one value for the exposure |
9457 | | variable XLevel (i.e., when the data C_Levels includes one record.) |
9458 | |
9459 | | |
9460 | | The intent was to make as few changes to BEIR4 as possible. The data |
9461 | | X_LEVELS and variable XLevel are retained but the initial value of |
9462 | | XLevel is provided in the call to macro CONVERGE BEIR4 (the value |
9463 | | of Xlevel in the cards statement is not used in the calculations. |
9464 | | Changes to the BEIR4 macro are in Part III and Part IV, and are |
9465 | | indicated by the letters BT. |
9466 | |
9467 | | |
9468 | | |
9469 | | In addition to the parameter values that are specified by the user |
9470 | | in PART 1, and the user-provided data entered in Part II, parameters |
9471 | | for the new macro CONVERGE_BEIR4 are specified in the call to the |
9472 | | macro CONVERGE_BEIR4 (see end of this SAS program file below). |
9473 | | +-----*/
9474 |
9475 | /* PART I. USER-SUPPLIED ASSIGNMENTS (Macro variables):
9476 | | /-----+
9477 | | Model of cumulative exposure effects: |
9478 | | 1 => Loglinear Relative rate |
9479 | | R=R0*exp(COEF*X) |
9480 | | 2 => Linear Relative rate, |
9481 | | R=R0*(1+COEF*X) |
9482 | | 3 => Absolute rate, |
9483 | | R=R0+COEF*X |
9484 | | 4 => Power relative rate |
```

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```

9485 |           R=R0*(1+X)^COEF           |
9486 |           0 => User Defined & programmed |
9487 |           in datastep Ex_Risk below |
9488 |                                         */ %Let Model = 2;
9489 /* |                                         |
9490 | Cumulative exposure parameter:         */ %Let COEF = 0.01;
9491 /* |                                         |
9492 | Lag or delay between exposure and effect: */ %Let Lag = 10;
9493 /* |                                         |
9494 | Age exposure begins:                   */ %Let Age1st_x = 0;
9495 /* Exposure duration (years):           */ %Let Duration = 85;
9496 /* Adjust dose from occupational to     |
9497 | continuous environmental exposures (Y/N)? */ %Let EnvAdj = Yes;
9498 /* Age to stop accumulating excess risk |
9499 | (supposing rates are available for     |
9500 | ages >= &LastAge); otherwise use all of |
9501 | the supplied rate information:         */ %Let LastAge =85;
9502 /*-----*/
9503
9504 /* PART II. USER-SUPPLIED ASSIGNMENTS (Datesets AllCause, Cause, X_Levels ): */
9505
9506 data AllCause (label="Unxposeds' age-spec mortality rates (all)"
9507               drop=Lx rename=(BLx=Lx) );
9508 /*-----+
9509 | Input lifetable and calculate the corresponding age-specific |
9510 | (all-causes) mortality rate (AllCause) and conditional survival |
9511 | probability for each year of age (qi) together with |
9512 | the corresponding values of age (Age). |
9513 +-----*/
9514 Label Age = "Age at start of year (Age=i)"
9515 BLx = "Number alive at start of year"
9516 Lx = "Number alive at end of year"
9517 CndPrDth = "Pr[Death before age i+1 | alive at age i]"
9518 qi = "Pr[Survive to age i+1 | Alive at age i]"
9519 AllCause = "Age-spec mortality rate (all causes)";
9520
9521 if _n_=1 then input age //// @1 BLx @;
9522 input Lx @@;
9523 CndPrDth = (BLx - Lx)/BLx;
9524
9525 qi = 1-CndPrDth;
9526 if qi <= 0 then AllCause = 1e+50;
9527 else AllCause = - log(qi);
9528
9529 if age < &LastAge then output; else STOP;
9530 BLx=Lx;
9531 age+1;
9532 retain age BLx;
9533 cards;
9534 0 = Life-table starting age. (Required: Values must begin 4 lines down!)
9535 The following are 2016 Life-table values of US population
9536 starting at birth and ending at age 85.
9537 (Source: Nat.Vital Statistics Reports 2017 Vol 66 No 3, Table 1)
9538 100000 99404 99362 99337 99318 99303 99288 99275 99264 99254
9539 99244 99235 99225 99213 99197 99174 99145 99110 99066 99014
9540 98953 98883 98805 98720 98632 98542 98450 98357 98262 98164
9541 98062 97957 97848 97735 97620 97500 97377 97247 97110 96965
9542 96811 96646 96470 96280 96073 95848 95601 95332 95036 94710
9543 94352 93962 93539 93084 92592 92062 91491 90879 90224 89527
9544 88788 88003 87169 86282 85341 84343 83284 82159 80961 79681
9545 78308 76833 75245 73539 71713 69764 67694 65481 63109 60575
9546 57879 55026 52028 48886 45607 0
9547 ;
9548
9549 data CAUSE (label="Unxposeds' age-cause-spec mortality rates");
9550 /*-----+
9551 | Specify unexposeds' age-specific mortality rates (per year) |
9552 | from specific cause. |
9553 +-----*/
9554 label Age = "Age"
9555 Rate_e5 = "Age,cause-specific rate per 100,000"
9556 Rate = "Age,cause-specific rate per individual";
9557
9558 if _n_ = 1 then input age /* input starting age */
9559 ///; /* // => skip next 3 lines */

```

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```

9560 input Rate_e5 @@;
9561
9562 Rate = Rate_e5 * 1e-5; /* Convert to rate per individual */
9563
9564 if age <= 4
9565 then DO; output; age+1; END;
9566 else DO i = 0,1,2,3,4; /*-----*/
9567 if age < &LastAge /* Fill out into yearly intervals from */
9568 then output; /* inputted five year intervals after age 4*/
9569 age+1; /*-----*/
9570 END;
9571
9572 cards;
9573 0 = Start age of cause-specific rate (Required: Rates begin 3 lines down!)
9574 The following are 2013 ICD10 = 113 death rates per 100,000 for US pop'n starting at birth.
9575 For ages 5 and above, each rate holds for the age thru age+4 years.
9576 Source: CDC Wonder
9577 0.0 0.0 0.0 0.0 0.0
9578 0.0 0.0 0.0 0.1 0.1 0.4 1.2 3.2 9.6 27.1 57.8
9579 90.7 136.6 212.5 277.3 321.2
9580 ;
9581
9582 data X_LEVELS (label= "Exposure levels (e.g., concentrations)" );
9583 /*-----+
9584 | Specify environmental exposure levels |
9585 | and update label for the variable, XLevel, if necessary: |
9586 +-----*/
9587 /*-----+
9588 | BT 3/8/19: Add macro CONVERGE_BEIR4 which iteratively runs macro |
9589 | BEIR4 until the EXPOSURE_CONCENTRATION corresponds to extra_risk=0.01|
9590 | |
9591 | |
9592 | |
9593 | The intent was to make as few changes to BEIR4 as possible. The data |
9594 | X_LEVELS and variable XLevel are retained but the initial value of |
9595 | XLevel is provided in the call to macro CONVERGE_BEIR4 (the value |
9596 | of Xlevel in the cards statement is not used in the calculations. |
9597 +-----*/
9598
9599 input XLevel @@;
9600 label XLevel= "Asbestos exposure (F/ml)";
9601 cards;
9602 0.0383
9603 ;
9604
9605 %Macro BEIR4;
9606 /* March 2019 - BT (SRC): Macro BEIR4 is now called by macro CONVERGE_BEIR4.
9607 */
9608 /* 23jul01 modification */
9609 /* Enclose the actual calculations and printed results in a macro */
9610 /* to facilitate multiple applications of the algorithm. */
9611
9612 /* PART III. Perform calculations: */
9613
9614 data EX_RISK (label = "Estimated excess risks [Method=BEIR IV]"
9615 /*keep = XLevel Rx ex_risk RskRatio R0 extra_Risk */
9616 rename = (Rx=Risk));
9617 /*-----+
9618 | Calculate risk and excess risk for each exposure concentration|
9619 | in work.X_Level by BEIR IV method using information in |
9620 | work.AllCause and work.Cause to define referent population: |
9621 +-----*/
9622
9623 length XLevel 8.;
9624 label Age = "Age at start of year (Age=i)"
9625 XTime = "Exposure duration midway between i & i+1"
9626 XDose = "Cumulative exposure midway betw. i & i+1"
9627
9628 R0 = "Unexposed's risk"
9629 Rx = "Exposed's risk (Rx)"
9630 Ex_Risk = "Excess risk (Rx-Ro)"
9631 RskRatio = "Ratio of risks (Rx/Ro)"
9632
9633 hi = "Unexposed's hazard rate at age i"
9634 hix = "Exposed's hazard rate at age i"

```



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9709

```

hstari = "Unexposeds all causes hazard rate(age=i)"
hstarix = "Exposed's all causes hazard rate(age=i)"
qi = "Pr[Survive to i+1 | Surv. to i,unexposed]"
S_li = "Pr[Survive to age=i | unexposed]"
S_lix = "Pr[Survive to age=i | exposed]";

/* BT 3/8/19: Calculation of unexposed's risk (following DO LOOP) could be omitted from
the iteration
but may require further changes to BEIR4(?).
*e.g., %if i=1 %then %do;*/

if _n_=1 then DO;
/* Calculate unexposed's risk (R0) to be retained */
/* based on equation 2A-21 (pg. 131) of BEIR IV: */

/* Initialize: */ S_li = 1; R0 = 0;

DO pointer = 1 to min(n_all,n_cause) until (age>=&LastAge-1);
set allcause (keep=age AllCause rename=(AllCause=hstari))
point=pointer nobs=n_all;
set cause (keep=age Rate rename=(age=ageCause Rate=hi))
point=pointer nobs=n_cause;

if Age NE AgeCause then
put "*** WARNING: Age values in datasets ALLCAUSE and CAUSE don't conform ***"
/ @13 "Rates misaligned on age could give incorrect results"
/ @13 Pointer=
+2 "Age(ALLCAUSE)=" Age +2 "Age(CAUSE)=" AgeCause /;

qi = exp(-hstari);
R0 = R0 + ( hi/hstari * S_li * (1-qi) );
S_li = S_li * qi;
END;
END; /* End of 'if _n_=1 then DO;' stmt */

retain R0;

/* Calculate exposed's risk (Rx) for each exposure level */
/* ultimately based on equation 2A-22 (pg. 132) of BEIR IV */
/* but re-expressed in a form similar to equation 2A-21: */

* BT 3/20/19. This version of CONVERGE_BEIR4 will work when there is
one concentration in data set x_levels -
i.e., one value for xlevel.
The Do loop for X_levels is commented out;
*DO pointX = 1 to No_of_Xs;
* set x_levels point=pointX nobs=No_of_Xs; /* BT 3/8/19: determines when to
end the loop. Nobs is set at compilation,
so the value of nobs is available at first run through loop -
just one record and one variable (XLevel) in dataset x_levels. */

/* BT 3/20/19: added the next lint to set the exposure
concentration = current value of &exposure_conc. */
xlevel = &exposure_conc;

/* Initialize : */ S_lix = 1; Rx = 0;

DO pointer = 1 to min(n_all,n_cause) until (age>=&LastAge-1);
set allcause (keep=age AllCause rename=(AllCause=hstari))
point=pointer nobs=n_all;
set cause (keep=Rate rename=(Rate=hi))
point=pointer nobs=n_cause;

XTime = min( max(0, (age+0.5-&Agelst_x-&Lag))
, &Duration - 0.5 );

if UpCase("&EnvAdj") = "YES" /* Occupational to Environmental Conversion
*/
then XDose = XLevel
* 365/240 /* Days per year */
* 20/10 /* Ventilation (L) per day */
* XTime;
ELSE if UpCase("&EnvAdj") = "NO" /* 30nov2018 ('ELSE') */

```

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```

9710         then XDose = XLevel*XTime;
9711         else DO; put //"Macro variable ENVADJ incorrectly specified."
9712                 //"It should be either YES or NO. Value specified is:
9713 &ENVADJ"
9714                 /;
9715                 STOP;
9716         END;
9717         hix=.;
9718         if &Model = 1 then hix = hi * exp(&COEF*XDose); else
9719         if &Model = 2 then hix = hi * (1 + &COEF*XDose); else
9720         if &Model = 3 then hix = hi + &COEF*XDose; else
9721         if &Model = 4 then hix = hi * (1 + XDose)**&COEF; else
9722         if &Model = 0 then DO;
9723             hix = -99999; /* Code for user-defined model goes here. */
9724         END;
9725
9726         hstarix = hstari          /* hi=backgrd rate is included in hstari */
9727                   + (hix - hi); /* so that adding in the excess */
9728                               /* from exposure (hix-hi) gives the */
9729                               /* total rate of the exposed. */
9730
9731         qix = exp(-hstarix);
9732         Rx = Rx + ( hix/hstarix * S_lix * ( 1-qix ) );
9733         S_lix = S_lix * qix;
9734         output;
9735
9736     END;
9737     Ex_Risk = Rx - R0; * Rx = risk in exposed population;
9738     RskRatio = Rx / R0; * R0 = from cancer;
9739     Extra_risk = Ex_Risk/(1-R0);
9740
9741     /* BT 3/20/19 added:*/
9742     call symput('Extra_Riskm',Extra_Risk);
9743
9744     /*BT 4/24/19 replaced the next line
9745     Diff_Ex_Risk = abs(&ex_risk_target-Ex_Risk); */
9746     Diff_Ex_Risk = abs(&ex_risk_target-Extra_Risk);
9747     call symput('Delta_Ex_Risk',Diff_Ex_Risk);
9748
9749     output;
9750
9751     * END; * corresponds to X_Levels;
9752 STOP;
9753
9754 run;
9755
9756 %Mend BEIR4;
9757
9758
9759 /* -----
9760          BT: March 2019: parameters for the convergence that are used
9761          in the modified version of the BEIR4 macro.
9762 -----*/
9763
9764 %macro Converge_BEIR4 (init_exposure_conc=, ex_risk_target=, conv_criterion=, max_iteration=);
9765
9766     %Let Delta_Ex_Risk = 1; * initial high value to make sure loop is run at least once
9767                               (i.e., macro BEIR4 is called at least
9768 once);
9769     /* BT 4/15/19: added next line to avoid error during compiling of BEIR4*/
9770     %Let Extra_Riskm = 1;
9771
9772     %Let i=1; * first time through loop;
9773
9774     %Do %Until (%sysevalf(&Delta_Ex_risk < &conv_criterion) OR %sysevalf(&i > &max_iteration));
9775
9776         * first time through loop, set exposure_conc=init_exposure_conc;
9777         %If &i=1 %Then
9778             %Do;
9779                 %Let exposure_conc=&init_exposure_conc;
9780
9781             %End;
9782         %If &i>1 %Then
9783             %Do;
9784

```

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```

9785         data tempBEIRCONVERGE;
9786
9787         exposure_conc
9788
9789         (=Extra_Risk);
9790
9791         NumLoops=&i;
9792         thisExposureConc=&exposure_conc;
9793
9794         /* BT 4/15/19: replaced all of the convergence code with the same
9795 code that we used
9796
9797         in the meso code.*/
9798
9799         numvar=&ex_risk_target;
9800         denvar=&Extra_Riskm;
9801
9802         thisexposureconc = thisexposureconc * (numvar/denvar); *update the
9803 concentration;
9804         call symput('exposure_conc',thisexposureconc);
9805
9806         output;
9807
9808         Run;
9809
9810         %End; *Corresponds to If i>1 statement;
9811
9812         %BEIR4;
9813
9814         %Let i=%eval(&i+1);
9815
9816         %End;
9817
9818         %Let EC_1Percent = &exposure_conc;
9819
9820         /*-----+
9821         | Report results if convergence criterion met:
9822         +-----*/
9823
9824         %If %sysevalf(&Delta_Ex_risk < &conv_criterion) %then %do;
9825         data _null_; /* Modified 26-july-00 */
9826         pointer=1;
9827         set allcause (keep=age
9828                     rename=(age=ageall0)) point=pointer nobs=n_all;
9829         set cause (keep=age
9830                  rename=(age=ageCs0)) point=pointer nobs=n_cause;
9831         pointer=n_all;
9832         set allcause (keep=age
9833                     rename=(age=ageall1)) point=pointer nobs=n_all;
9834         pointer=n_cause;
9835         set cause (keep=age
9836                  rename=(age=ageCs1)) point=pointer nobs=n_cause;
9837
9838         Tmp = sum(min(AgeAll1, AgeCs1, (&Lastage-1)), 1);
9839         file PRINT;
9840
9841         if ageall0 NE ageCs0 then DO;
9842         put /"ERROR: The initial age for all-causes rate differs from the"
9843           /" initial age for the cause-specific rate.";
9844         END;
9845         else DO;
9846         put / "Values of macro variables used in this computation: "
9847           // @3 "Value" @17 "Macro_Var" @29 "Description"
9848           / @3 "-----" @17 "-----" @29 "-----"
9849           // @3 "&Model" @17 "MODEL" @29 "1 = Loglinear Relative Rate,"
9850           / @29 "2 = Linear Relative Rate, "
9851           / @29 "3 = Linear Absolute Rate, "
9852           / @29 "4 = 'Power' Relative Rate, "
9853           / @29 "0 = User defined. "
9854           // @3 "&Coef" @17 "COEF" @29 "Exposure parameter estimate"
9855           // @3 "&Lag" @17 "LAG" @29 "Exposure Lag "
9856           // @3 "&Age1st_x" @17 "AGE1ST_X" @29 "Age exposure begins"
9857           / @3 "&Duration" @17 "DURATION" @29 "Duration of exposure"
9858           / @3 "&EnvAdj" @17 "ENVADJ" @29 "Adjust dose from intermittent"
9859           / @29 "occupational exposures to "

```

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```
9860 / @29 "continuous environmental exposures"
9861 / @3 "-----" @17 "-----" @29 "-----"
9862 // "-----"
9863 // @3 "EC1% = " @10 "&EC_1Percent" @25 "(f/ml); Rx = " @39 "&Extra_Riskm"
9864 // "-----"
9865
9866 /"The risks are calculated from age " ageall0 " up to age " Tmp "."
9867 // ;
9868
9869 if ageall1 NE ageCsl then
9870 put /"WARNING: The last age for the all-causes rates differs from"
9871 /" the last age for the cause-specific rates, suggesting"
9872 /" the possibility that the rates weren't entered as desired."
9873 /;
9874 END;
9875 Stop;
9876 run;
9877 proc print data=ex_risk label noobs;
9878 format risk E11. ex_risk E11. Xlevel E11.;*RskRatio 6.4;
9879 run;
9880 %End; *end of the If statement that tests if convergence was met;
9881
9882 %Mend Converge_BEIR4;
9883
9884 /* -----+
9885 | March 2019: BT (SRC) Added macro CONVERGE_BEIR4 which iteratively |
9886 | runs macro BEIR4 until the EXPOSURE_CONCENTRATION corresponds to an |
9887 | extra_risk=0.01 (the point of departure [POD]). |
9888 |
9889 |
9890 |
9891 | In addition to the parameter for CONVERGE_BEIR4, the user should also|
9892 | review parameters and data that are assigned/entered in Part I and |
9893 | Part II (see above). Parameters for CONVERGE_BEIR4 are defined below |
9894 +-----*/
9895 title5 " test of converge_BEIR4, based on MLE(Coef)=&COEF and LastAge=&LastAge";
9896 *%BEIR4; * originally called macr BEIR4 directly. Now BEIR4 is called by Converge_BEIR4;
9897
9898 %Converge_BEIR4(init_exposure_conc=1, /* initial exposure concentration (initial guess) */
9899 ex_risk_target=0.01000000, /* the point of departure (POD) - the
9900 target extra risk */
9901 conv_criterion=0.00000001,
9902 max_iteration=200); /* to avoid excessively long run
9903 times */
9904
9905 Run;
```

**Mesothelioma Lifetable**

```

*
This program calculates the risk of mesothelioma from inhalation exposure to asbestos,
using a lifetable approach. The basic model is  $I_m = C * KM * Q$ .

The basic code for the lifetable calculations were developed and provided to EPA
by Randall Smith at NIOSH.

For mesothelioma, calculations are based on NIOSH Model 3:  $R_x = R_0 + COEF * X\_Dose$ 
For mesothelioma,  $R_0$  is assumed to be zero.

EPA has modified the NIOSH as follows:
1) The all-cause and cause-specific (mesothelioma) mortality data tables have been updated.
2) Code has been added to calculate  $X\_Dose = X\_Level * Q$ , where Q is a function of TSFE and exposure
duration.
2) An equation has been added to calculate extra risk:  $Extra\_Risk = (R_x - R_0) / (1 - R_0)$ 
3) A macro has been added to find the exposure concentration ( $X\_Level$ ) that yields an extra risk of 1%.
This is referred to as EC.
This value may then be used to calculate the unit risk:  $UR = 0.01 / EC$ 

*/

/* .\Beta Version.sas 19jan00, 26jul00, 25oct01, 06dec05, 30nov18
-----
Experimental version
----- */
title "Excess Risks using BEIR IV method to account for competing risks";
title2 "Effects of airborne exposure to asbestos on mesothelioma mortality rates";
title3 "under a linear absolute rate model .";

/*-----+
| Compute excess risk by the BEIR IV method using SAS datasteps. |
| These programs compute the risk of a cause-specific |
| death in the presence of competing risks, where the cause- |
| specific death-rate is modeled either as a relative rate |
| [ $h=h_0*f(Coef*X)$ ] or as an absolute rate [ $h=h_0+f(Coef*X)$ ] |
| where |
| h denotes the cause-specific death-rate, |
| X denotes cumulative occupational exposure (with Lag) |
| Coef denotes the coefficient for the effect of exposure and |
| h0 is the corresponding rate at baseline (X=0). |
| (Except for Coef, these are functions of age.) |
| A few simple models of f(Coef*X) are easily specified as |
| described below. More complicated models can be specified with |
| a little more work. (For a more complicated example, |
| see \_GENERAL.LIB\PROGRAMS\SAS\BEIR-4.Method\BEIR4ex2.SAS). |
+Reference: +
| Health Risks of Radon and Other Internally Deposited Alpha- |
| Emitters (BEIR IV). Committee on the Biologic Effects of |
| Ionizing Radiations. National Academy Press. Wash. DC (1988). |
| See especially pages 131-136. |
+USER-SUPPLIED ASSIGNMENTS: +
|
|> The following macro variables are assigned using "%LET" state- |
| ments: MODEL, COEF, LAG, AGE1ST_X, DURATION, LASTAGE. |
| Further information appears below. |
|> Exposure concentrations for computing risk are defined |
| in the dataset "X_LEVELS." |
|> All-cause mortality information is entered as a life-table in |
| the data step "ALLCAUSE," and converted to rates per individual. |
|> Cause-specific mortality information for unexposed referents is |
| entered as rates per 100,000 and converted to rates per |
| individual in the data step "CAUSE." |

```

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```
9982 | | |
9983 | | |
9984 | +NOTES: | +
9985 | |> Datastep "EX_RISK" is where the desired risks are computed. |
9986 | | |
9987 | |> If the unexposed(referent) cause-specific mortality rate is from |
9988 | | a model then datastep "CAUSE" with variables AGE and RATE as |
9989 | | modeled can be modified to incorporate this. However, care |
9990 | | must be taken in calculating confidence limits since imprecision |
9991 | | in the estimates of all of the parameters of the model |
9992 | | contributes to the imprecision of excess risk estimates. |
9993 | | |
9994 | |> This program is currently set up to apply the Linear Rel. Rate |
9995 | | model (Lag= 0) and accumulation of excess risk is over the |
9996 | | rates in ALLCAUSE and CAUSE unless truncated at a younger age. |
9997 | | (See LASTAGE below.) |
9998 | | |
9999 | | |
10000 | + SAS Programmer: Randall Smith | +
10001 | | The Nat'l Inst. for Occupational Safety & Health |
10002 | | 26jul2000, 23jul2001, 25oct2001, 18nov2018 |
10003 | + Modifications: | +
10004 | | |
10005 | | 26jul00 Fix the procedure bug causing it to report incorrectly |
10006 | | the age at which accumulation of risk was stopped |
10007 | | whenever the age-specific rates included ages |
10008 | | before the value of &Agelst_X. (&Agelst_X is a macro |
10009 | | expression defining the age exposure begins.) |
10010 | | |
10011 | | 23jul01 Make changes to facilitate multiple applications of |
10012 | | BEIR4 algorithm, i.e., MLE(Excess Risk), UCL(ExcessRisk), |
10013 | | searching for concentrations for a fixed risk. These |
10014 | | changes involve defining Macros named BEIR4 and SEARCH |
10015 | | given below with code illustrating these uses for the |
10016 | | linear relative rate model. |
10017 | | |
10018 | | 25oct01 Modified to add Macro variable EnvAdj for whether to |
10019 | | increase inhaled dose from intermittent occupational |
10020 | | exposures to continuous environmental exposures |
10021 | | and update US rates for Gibb et al. cohort. |
10022 | | |
10023 | | 30nov18 A bug that prevented the calculation of excess risks |
10024 | | after incorporating an adjustment from intermittent |
10025 | | occupational exposures to continuous exposures is fixed. |
10026 | | |
10027 | | +---|
10028 | | April 2019: BT (SRC) Added maxro CONVERGE_BEIR4 which iteratively |
10029 | | runs macro BEIR4 until the EXPOSURE_CONCENTRATION corresponds to an |
10030 | | extra_risk=0.01 (the point of departure [POD]). |
10031 | | |
10032 | | |
10033 | | Macro CONVERGE_BEIR4 works with one value for the exposure |
10034 | | variable XLevel (i.e., when the data C Levels includes one record.) |
10035 | | |
10036 | | |
10037 | | The intent was to make as few changes to BEIR4 as possible. The data |
10038 | | X_LEVELS and variable XLevel are retained but the initial value of |
10039 | | XLevel is provided in the call to macro CONVERGE_BEIR4 (the value |
10040 | | of Xlevel in the cards statement is not used in the calculations. |
10041 | | Changes to the BEIR4 macro are in Part III and Part IV, and are |
10042 | | indicated by the letters BT. |
10043 | | |
10044 | | |
10045 | | |
10046 | | In addition to the parameter values that are specified by the user |
10047 | | in PART 1, and the user-provided data entered in Part II, parameters |
10048 | | for the new macro CONVERGE_BEIR4 are specified in the call to the |
10049 | | macro CONVERGE_BEIR4 (see end of this SAS program file below). |
10050 | | +-----*/
10051 | |
10052 | |
10053 | |
10054 | |
10055 | /* PART I. USER-SUPPLIED ASSIGNMENTS (Macro variables):
10056 | /*-----+
```

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```

10057 | Model of cumulative exposure effects: |
10058 |     1 => Loglinear Relative rate |
10059 |         R=R0*exp(COEF*X) |
10060 |     2 => Linear Relative rate, |
10061 |         R=R0*(1+COEF*X) |
10062 |     3 => Absolute rate, |
10063 |         R=R0+COEF*X |
10064 |     4 => Power relative rate |
10065 |         R=R0*(1+X)^COEF |
10066 |     0 => User Defined & programmed |
10067 |         in datastep Ex_Risk below |
10068 |                                     */ %Let Model = 3;
10069 /*
10070 | Cumulative exposure parameter: */ %Let COEF = 0.000000015;
10071 /*
10072 | Lag or delay between exposure and effect: */ %Let Lag = 10; /* Lag is built into Q, so this
10073 value is ignored */
10074 /*
10075 | Age exposure begins: */ %Let Age1st_x = 0;
10076 /* Exposure duration (years): */ %Let Duration = 85;
10077 /* Adjust dose from occupational to
10078 | continuous environmental exposures (Y/N)? */ %Let EnvAdj = Yes;
10079 /* Age to stop accumulating excess risk
10080 | (supposing rates are available for
10081 | ages >= &LastAge); otherwise use all of
10082 | the supplied rate information: */ %Let LastAge =85;
10083 /*-----*/
10084
10085
10086 /* PART II. USER-SUPPLIED ASSIGNMENTS (Datesets AllCause, Cause, X_Levels ): */
10087
10088
10089 data AllCause (label="Unxposeds' age-spec mortality rates (all)"
10090 drop=Lx rename=(BLx=Lx) );
10091
10092 /*-----+
10093 | Input lifetable and calculate the corresponding age-specific |
10094 | (all-causes) mortality rate (AllCause) and conditional survival |
10095 | probability for each year of age (qi) together with |
10096 | the corresponding values of age (Age). |
10097 +-----*/
10097 Label Age = "Age at start of year (Age=i)"
10098 BLx = "Number alive at start of year"
10099 Lx = "Number alive at end of year"
10100 CndPrDth = "Pr[Death before age i+1 | alive at age i]"
10101 qi = "Pr[Survive to age i+1 | Alive at age i]"
10102 AllCause = "Age-spec mortality rate (all causes)";
10103
10104 if _n_=1 then input age /// @1 BLx @;
10105 input Lx @@;
10106 CndPrDth = (BLx - Lx)/BLx;
10107
10108 qi = 1-CndPrDth;
10109 if qi <= 0 then AllCause = 1e+50;
10110 else AllCause = - log(qi);
10111
10112 if age < &LastAge then output; else STOP;
10113 BLx=Lx;
10114 age+1;
10115 retain age BLx;
10116 cards;
10117 0 = Life-table starting age. (Required: Values must begin 4 lines down!)
10118 The following are 2013 Life-table values of US population
10119 starting at birth and ending at age 85.
10120 (Source: Nat.Vital Statistics Reports 2017 Vol 66 No 3, Table 1)
10121 100000 99404 99362 99337 99318 99303 99288 99275 99264 99254
10122 99244 99235 99225 99213 99197 99174 99145 99110 99066 99014
10123 98953 98883 98805 98720 98632 98542 98450 98357 98262 98164
10124 98062 97957 97848 97735 97620 97500 97377 97247 97110 96965
10125 96811 96646 96470 96280 96073 95848 95601 95332 95036 94710
10126 94352 93962 93539 93084 92592 92062 91491 90879 90224 89527
10127 88788 88003 87169 86282 85341 84343 83284 82159 80961 79681
10128 78308 76833 75245 73539 71713 69764 67694 65481 63109 60575
10129 57879 55026 52028 48886 45607 0
10130 ;
10131

```

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```

10132
10133 data CAUSE (label="Unexposeds' age-cause-spec mortality rates");
10134 /*-----+
10135 | Specify unexposeds' age-specific mortality rates (per year) |
10136 | from specific cause. |
10137 +-----*/
10138
10139 label Age = "Age"
10140 Rate_e5 = "Age,cause-specific rate per 100,000"
10141 Rate = "Age,cause-specific rate per individual";
10142
10143 if _n_ = 1 then input age /* input starting age */
10144 /* //; /* // => skip next 3 lines */
10145 input Rate_e5 @@;
10146
10147 Rate = Rate_e5 * 1e-5; /* Convert to rate per individual */
10148
10149 if age <= 4
10150 then DO; output; age+1; END;
10151 else DO i = 0,1,2,3,4; /*-----*/
10152 if age < &LastAge /* Fill out into yearly intervals from */
10153 then output; /* inputted five year intervals after age 4*/
10154 age+1; /*-----*/
10155 END;
10156 cards;
10157 0 = Start age of cause-specific rate (Required: Rates begin 3 lines down!)
10158 The following are 2013 ICD10 = 113 death rates per 100,000 for US pop'n starting at birth.
10159 For ages 5 and above, each rate holds for the age thru age+4 years.
10160 Source: CDC Wonder
10161 0.0 0.0 0.0 0.0 0.0 0.0
10162 0.0 0.0 0.0 0.0 0.0 0.0 0.0 0.0 0.0 0.0 0.0 0.0
10163 0.0 0.0 0.0 0.0 0.0
10164
10165 ;
10166
10167 run;
10168
10169 data X_LEVELS (label= "Exposure levels (e.g., concentrations) " );
10170 /*-----+
10171 | Specify environmental exposure levels |
10172 | and update label for the variable, XLevel, if necessary: |
10173 +-----*/
10174
10175 input XLevel @@;
10176 label XLevel= "Asbestos exposure (F/ml)";
10177 cards;
10178 0.001
10179 ;
10180
10181 %Macro BEIR4;
10182 /* April 2 2019 - BT (SRC): Macro BEIR4 is now called by macro CONVERGE_BEIR4.*/
10183 /* 23jul01 modification */
10184 /* Enclose the actual calculations and printed results in a macro */
10185 /* to facilitate multiple applications of the algorithm. */
10186
10187 /* PART III. Perform calculations: */
10188
10189 data EX_RISK (label = "Estimated excess risks [Method=BEIR IV]"
10190 /*keep = XLevel Rx ex_risk RskRatio */
10191 rename= (Rx=Risk));
10192 /*-----+
10193 | Calculate risk and excess risk for each exposure concentration|
10194 | in work.X_Level by BEIR IV method using information in |
10195 | work.AllCause and work.Cause to define referent population: |
10196 +-----*/
10197 length XLevel 8.;
10198 label Age = "Age at start of year (Age=i)"
10199 XTime = "Exposure duration midway between i & i+1"
10200 XDose = "Cumulative exposure midway betw. i & i+1"
10201
10202 R0 = "Unexposed's risk"
10203 Rx = "Exposed's risk (Rx)"
10204 Ex_Risk = "Excess risk (Rx-Ro)"
10205 RskRatio = "Ratio of risks (Rx/Ro)"
10206

```



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```

10207 hi = "Unexposed's hazard rate at age i"
10208 hix = "Exposed's hazard rate at age i"
10209 hstari = "Unexposeds all causes hazard rate(age=i)"
10210 hstarix = "Exposed's all causes hazard rate(age=i)"
10211 qi = "Pr[Survive to i+1 | Surv. to i,unexposed]"
10212 S_li = "Pr[Survive to age=i | unexposed]"
10213 S_lix = "Pr[Survive to age=i | exposed]"
10214 XLevel = "EC1%";
10215
10216 /* BT 3/8/19: Calculation of unexposed's risk (following DO LOOP) could be omitted from
10217 the iteration
10218 but may require further changes to BEIR4(?).
10219 *e.g., %if i=1 %then %do;*/
10220
10221 if _n_=1 then DO;
10222 /* Calculate unexposed's risk (R0) to be retained */
10223 /* based on equation 2A-21 (pg. 131) of BEIR IV: */
10224
10225 /* Initialize: */ S_li = 1; R0 = 0;
10226
10227 DO pointer = 1 to min(n_all,n_cause) until (age>=&LastAge-1);
10228 set allcause (keep=age AllCause rename=(AllCause=hstari))
10229 point=pointer nobs=n_all;
10230 set cause (keep=age Rate rename=(age=ageCause Rate=hi))
10231 point=pointer nobs=n_cause;
10232
10233 if Age NE AgeCause then
10234 put "*** WARNING: Age values in datasets ALLCAUSE and CAUSE don't conform ***"
10235 / @13 "Rates misaligned on age could give incorrect results"
10236 / @13 Pointer=
10237 +2 "Age (ALLCAUSE)=" Age +2 "Age (CAUSE)=" AgeCause /;
10238
10239 qi = exp(-hstari);
10240 R0 = R0 + ( hi/hstari * S_li * (1-qi) );
10241 S_li = S_li * qi;
10242 END;
10243 END; /* End of 'if _n_=1 then DO;' stmt */
10244
10245 retain R0;
10246
10247
10248 /* Calculate exposed's risk (Rx) for each exposure level */
10249 /* ultimately based on equation 2A-22 (pg. 132) of BEIR IV */
10250 /* but re-expressed in a form similar to equation 2A-21: */
10251
10252 * BT 3/20/19. This version of CONVERGE_BEIR4 will work when there is
10253 one concentration in data set x_levels - i.e., one value for
10254 xlevel.
10255 The Do loop for X_levels is commented out;
10256 *DO pointX = 1 to No_of_Xs;
10257 * set x_levels point=pointX nobs=No_of_Xs; /* BT 3/8/19: determines when to end the
10258 loop. Nobs is set at compilation,
10259
10260 so the value of nobs is available at first run through loop -
10261
10262 just one record and one variable (XLevel) in dataset x_levels. */
10263
10264
10265 xlevel = &exposure_conc;
10266
10267 /* Initialize : */ S_lix = 1; Rx = 0;
10268
10269 DO pointer = 1 to min(n_all,n_cause) until (age>=&LastAge-1);
10270 set allcause (keep=age AllCause rename=(AllCause=hstari))
10271 point=pointer nobs=n_all;
10272 set cause (keep=Rate rename=(Rate=hi))
10273 point=pointer nobs=n_cause;
10274
10275 /*
10276 XTime = min( max(0, (age+0.5-&Age1st_x-&Lag))
10277 , &Duration );
10278
10279
10280 Q = .;
10281 If Age < 10 then Q = 0;

```

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```

10282         If Age >= (XTime +10) then Q = ((Age-10)**3)-((-10-XTime)**3);
10283         Else Q = (XTime-10)**3;
10284
10285     */
10286
10287         TSFE=.;
10288     If Age < &Age1st_x then TSFE = 0;
10289     Else TSFE = Age - &Age1st_x + 0.5;
10290
10291
10292         d = .;
10293         If Age < &Age1st_x then d = 0; else
10294         If Age >= &Age1st_x + &Duration then d = &Duration - 0.5;
10295         Else d = Age-&Age1st_x + 0.5;
10296
10297
10298         Q=.;
10299         If TSFE < 10 then Q = 0; else
10300         If TSFE >= d+10 then Q = (TSFE-10)**3-(TSFE-10-d)**3;
10301         Else Q = (TSFE-10)**3;
10302
10303     if UpCase("&EnvAdj") = "YES" /* Occupational to Environmental Conversion */
10304     then XDose = XLevel
10305         * 365/240 /* Days per year */
10306         * 20/10 /* Ventilation (L) per day */
10307         * Q; /* BT: in lung cancer program, this line has just
10308 XTime (instead of Q) */
10309     ELSE if UpCase("&EnvAdj") = "NO" /* 30nov2018 ('ELSE') */
10310     then XDose = XLevel*XTime;
10311     else DO; put //"Macro variable ENVADJ incorrectly specified."
10312             //"It should be either YES or NO. Value specified is: &ENVADJ"
10313             /;
10314     STOP;
10315     END;
10316
10317     hix=.;
10318     if &Model = 1 then hix = hi * exp(&COEF*XDose); else
10319     if &Model = 2 then hix = hi * (1 + &COEF*XDose); else
10320     if &Model = 3 then hix = hi + &COEF*XDose; else
10321     if &Model = 4 then hix = hi * (1 + XDose)**&COEF; else
10322     if &Model = 0 then DO;
10323     hix = -99999; /* Code for user-defined model goes here. */
10324     END;
10325
10326     hstarix = hstari /* hi=backgrd rate is included in hstari */
10327     + (hix - hi); /* so that adding in the excess */
10328     /* from exposure (hix-hi) gives the */
10329     /* total rate of the exposed. */
10330
10331     qix = exp(-hstarix);
10332     Rx = Rx + ( hix/hstarix * S_lix * ( 1-qix ) );
10333     S_lix = S_lix * qix;
10334
10335     output;
10336
10337     END;
10338     Ex Risk = Rx - R0; /* BT 4/2/19: was Ex_Risk = Rx - R0; */
10339     * RskRatio = Rx / R0;
10340     output;
10341
10342     /* BT 4/14/19: the macro variables for risk and difference between the
10343     calculated risk
10344     and the target risk were moved from Converge_BEIR4 to BEIR4
10345     */
10346     call symput('Extra_Riskm',Ex_Risk);
10347
10348     Diff_Ex_Risk = abs(&ex_risk_target-Ex_Risk);
10349     call symput('Delta_Ex_Risk',Diff_Ex_Risk);
10350
10351     * END; * corresponds to X_Levels;
10352
10353     STOP;
10354     run;
10355
10356 %Mend BEIR4;
10357
10358 /* -----
10359 BT: March 2019: parameters for the convergence that are used
10360 in the modified version of the BEIR4 macro.

```

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```

-----*/
10357
10358
10359 %macro Converge_BEIR4 (init_exposure_conc=, ex_risk_target=, conv_criterion=, max_iteration=);
10360
10361
10362     %Let Extra_Riskm = 1;
10363
10364     %Let Delta_Ex_Risk = 1; * initial high value to make sure loop is run at least once
10365                               (i.e., macro BEIR4 is called at least
10366 once);
10367
10368     %Let i=1; * first time through loop;
10369
10370
10371
10372     %Do %Until (%sysevalf(&Delta_Ex_risk < &conv_criterion) OR %sysevalf(&i > &max_iteration));
10373
10374         * first time through loop, set exposure_conc=init_exposure_conc;
10375
10376         %If &i=1 %Then
10377             %Do;
10378                 %Let exposure_conc=&init_exposure_conc;
10379
10380             %End;
10381         %If &i>1 %Then
10382             %Do;
10383
10384                 data tempBEIRCONVERGE;
10385                               /* BT March 2019: BEIR4 has run at least
10386 once. Adjust exposure_conc                               Extra_Riskm is created in BEIR4
10387                               (=Ex_Risk)*/
10388                               NumLoops=&i;
10389                               thisExposureConc=&exposure_conc; *set equal to concentration in
10390 loop i-1;
10391                               numvar=&ex_risk_target;
10392                               denvar=&Extra_Riskm;
10393                               thisexposureconc = thisexposureconc * (numvar/denvar); *update the
10394 concentration;
10395                               call symput('exposure_conc',thisexposureconc);
10396                               output;
10397                               Run;
10398
10399                 %End; *Corresponds to If i>1 statement;
10400
10401             %BEIR4;
10402
10403             %Let i=%eval(&i+1);
10404
10405         %End;
10406
10407     %Let EC_1Percent = &exposure_conc);
10408
10409
10410
10411
10412
10413
10414 /*-----+
10415 | Report results if convergence criterion met:
10416 +-----*/
10417
10418 %If %sysevalf(&Delta_Ex_risk < &conv_criterion) %then %do;
10419     data _null_; /* Modified 26-july-00 */
10420         pointer=1;
10421         set allcause (keep=age
10422                     rename=(age=ageall0)) point=pointer nobs=n_all;
10423         set cause (keep=age
10424                  rename=(age=ageCs0)) point=pointer nobs=n_cause;
10425         pointer=n_all;
10426         set allcause (keep=age
10427                     rename=(age=ageall1)) point=pointer nobs=n_all;
10428         pointer=n_cause;
10429         set cause (keep=age
10430                  rename=(age=ageCs1)) point=pointer nobs=n_cause;
10431

```

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```

10432 Tmp = sum(min(AgeAll1, AgeCs1, (&Lastage-1)), 1);
10433 file PRINT;
10434
10435 if ageall0 NE ageCs0 then DO;
10436   put /"ERROR: The initial age for all-causes rate differs from the"
10437     /"           initial age for the cause-specific rate.";
10438 END;
10439 else DO;
10440   put / "Values of macro variables used in this computation:      "
10441     // @3 "Value"      @17 "Macro_Var" @29 "Description"
10442     // @3 "-----"    @17 "-----"  @29 "-----"
10443     // @3 "&Model"    " @17 "MODEL"    @29 "1 = Loglinear Relative Rate,"
10444     //                                     @29 "2 = Linear Relative Rate, "
10445     //                                     @29 "3 = Linear Absolute Rate,  "
10446     //                                     @29 "4 = 'Power' Relative Rate,  "
10447     //                                     @29 "0 = User defined.          "
10448     // @3 "&Coef"     " @17 "COEF"      @29 "Exposure parameter estimate"
10449     // @3 "&Lag"      " @17 "LAG"       @29 "Exposure Lag "
10450     // @3 "&Age1st_x" @17 "AGE1ST_X"   @29 "Age exposure begins"
10451     // @3 "&Duration" @17 "DURATION"   @29 "Duration of exposure"
10452     // @3 "&EnvAdj"   @17 "ENVADJ"    @29 "Adjust dose from intermittent"
10453     //                                     @29 "occupational exposures to "
10454     //                                     @29 "continuous environmental exposures"
10455     //                                     / @3 "-----" @17 "-----" @29 "-----"
10456     // "-----" // @3 "EC1% = " @10 "&EC_1Percent" @20 " (f/ml); Rx = " @34
10457 "&Extra_Riskm" // "-----"
10458 // "-----"
10459 // "-----"
10460 // "-----"
10461 // "The risks are calculated from age " ageall0 " up to age " Tmp "."
10462 // ;
10463
10464 if ageall1 NE ageCs1 then
10465   put /"WARNING: The last age for the all-causes rates differs from"
10466     /"           the last age for the cause-specific rates, suggesting"
10467     /"           the possibility that the rates weren't entered as desired."
10468     /;
10469 END;
10470 Stop;
10471 run;
10472 proc print data=ex_risk label noobs;
10473   format risk E11. ex_risk E11. Xlevel E11.; *RskRatio 6.4;
10474 run;
10475 %End; *end of the If statement that tests if convergence was met;
10476
10477 %Mend Converge_BEIR4;
10478
10479
10480
10481 /* the following options are for debugging - comment out after code is running as expected*/
10482 Options mlogic mprint symbolgen;
10483
10484
10485 /*
10486   %Let LastAge =85;
10487   %LET LAG     = 10;
10488   %Let MODEL   = 3;
10489   %Let COEF    = 0.000000015;
10490 */
10491
10492 /* -----+
10493 | April 2019: BT (SRC) Added macro CONVERGE_BEIR4 which iteratively |
10494 | runs macro BEIR4 until the EXPOSURE_CONCENTRATION corresponds to an |
10495 | extra_risk=0.01 (the point of departure [POD]). |
10496 | |
10497 | |
10498 | At the second iteration of the Converge_BEIR4 macro, the exposure |
10499 | concentration is adjusted by a factor equal to the initial |
10500 | concentration x ConvRate. It is recommended to use a convrate equal |
10501 | to 0.1, which produces an adjustment of approximately 10% of the |
10502 | initial concentration value. The conversion rate is adjusted in |
10503 | later iterations (to smaller adjustments) as needed to converge. |
10504 | |
10505 | |
10506 | |

```

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```
10507 | In addition to the parameter for CONVERGE_BEIR4, the user should also|
10508 | review parameters and data that are assigned/entered in Part I and |
10509 | Part II (see above). Parameters for CONVERGE_BEIR4 are defined below |
10510 +-----*/
10511 title5 "based on MLE(Coef)=&COEF and LastAge=&LastAge";
10512 *%BEIR4; * originally called macr BEIR4 directly. Now BEIR4 is called by Converge_BEIR4;
10513
10514
10515
10516 %Converge_BEIR4(init_exposure_conc=0.1, /* initial exposure concentration (initial guess) */
10517 ex_risk_target= 0.0100, /* the point of departure (POD) - the
10518 target extra risk */
10519 conv_criterion=0.00000001,
10520 max_iteration=300); /* to avoid excessively long run
10521 times */
10522
10523
10524 Run;
10525
10526
10527
```

## Appendix J Results of Modeling for IUR Derivation

10528  
10529  
10530  
10531

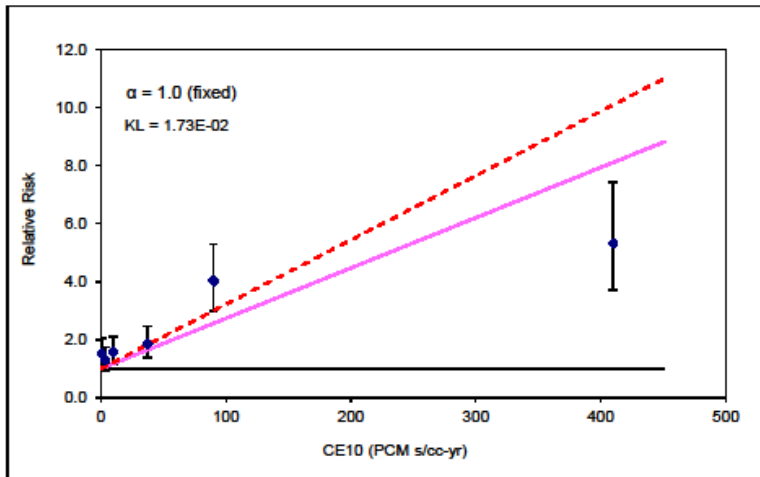
### Section 1

Hein et al. (2007)

#### EPA Modeling of Hein et al. (2007) Grouped Lung Cancer Data

Cohort: South Carolina  
Citation: Hein et al. 2007  
Data: Table 3

CE10 (PCM s/cc-yrs)			Lung Cancer Deaths		
Min	Max	Mid	Obs	Exp	RR
0	1.5	0.75	34	22.10	1.54
1.5	5	3.25	33	25.30	1.30
5	15	10	34	21.70	1.57
15	60	37.5	35	18.80	1.86
60	120	90	37	9.20	4.02
120	699.8	409.9	25	4.70	5.32
			198	101.8	1.94



Value	Alpha	KL	AIC
MLE	1.00	1.73E-02	54.29
UB	1.00	2.22E-02	—

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10545

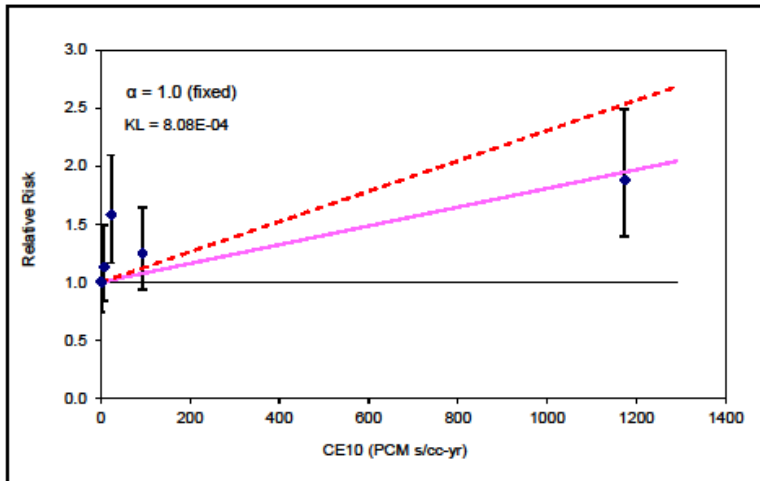
**Section 2**

Loomis et al. (2009)

**EPA Modeling of Loomis et al. (2009) Grouped Lung Cancer Data**

Cohort: North Carolina  
Citation: Loomis et al 2009  
Data: Table 5

CE10 (PCM s/cc-yrs)			Lung Cancer Deaths		
Min	Max	Mid	Obs	Exp	RR
0	2.3	1.15	37	37.00	1.00
2.3	11.5	6.9	37	32.74	1.13
11.5	34.8	23.15	35	22.15	1.58
34.8	152.7	93.75	37	29.60	1.25
152.7	2194	1173.35	35	18.62	1.88
			181	140.1	1.29



Value	Alpha	KL	AIC
MLE	1.00	8.08E-04	35.33
UB	1.00	1.31E-03	—

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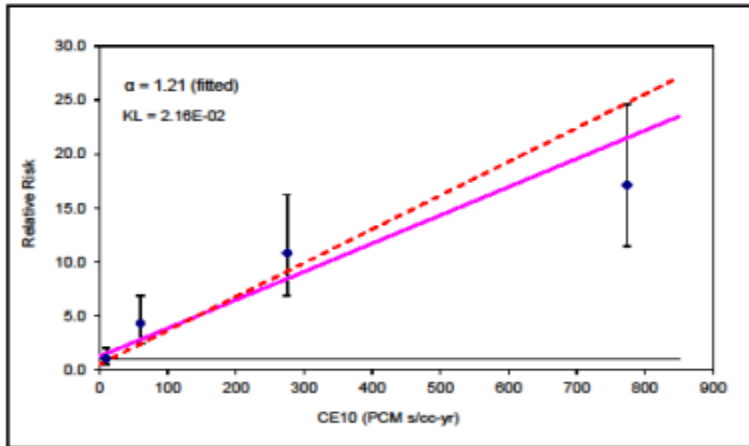
**Section 3**

Wang et al. (2013b)

**EPA Modeling of Wang et al. (2013) Grouped Lung Cancer Data**

Cohort: Chinese miners  
 Citation: Wang et al. 2013  
 Data: Table 5 + 6

CE10 (PCM s/cc-yrs)			Lung Cancer Deaths		
Min	Max	Mid	Obs	Exp	RR
0	20	10	6	5.75	1.04
20	100	60	12	2.82	4.25
100	450	275	17	1.57	10.82
450	1097	773.5	21	1.23	17.07
			56	11.4	4.92



Value	Alpha	KL	AIC
MLE	1.21	2.16E-02	24.44
UB	0.48	6.47E-02	--

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 10591



**Appendix K Less Than Lifetime (or Partial lifetime) IUR**

**Table\_Apx K-1. (LTL) Chrysotile Asbestos Inhalation Unit Risk Values for Less Than Lifetime Condition of Use**

Age at first exposure (years)	Duration of exposure (years)										
	1	5	10	15	20	25	30	35	40	62	78
0	4.06E-03	3.12E-02	5.95E-02	8.25E-02	1.01E-01	1.15E-01	1.27E-01	1.36E-01	1.44E-01	1.62E-01	1.64E-01
1	3.91E-03	3.00E-02	5.72E-02	7.91E-02	9.67E-02	1.11E-01	1.22E-01	1.31E-01	1.38E-01	1.55E-01	1.57E-01
2	3.78E-03	2.89E-02	5.49E-02	7.59E-02	9.27E-02	1.06E-01	1.17E-01	1.25E-01	1.32E-01	1.48E-01	1.50E-01
3	3.64E-03	2.77E-02	5.27E-02	7.28E-02	8.89E-02	1.02E-01	1.12E-01	1.20E-01	1.26E-01	1.42E-01	1.43E-01
4	3.51E-03	2.66E-02	5.06E-02	6.98E-02	8.51E-02	9.73E-02	1.07E-01	1.15E-01	1.21E-01	1.35E-01	1.37E-01
5	3.39E-03	2.56E-02	4.85E-02	6.69E-02	8.15E-02	9.31E-02	1.02E-01	1.10E-01	1.15E-01	1.30E-01	1.31E-01
6	3.27E-03	2.45E-02	4.65E-02	6.41E-02	7.81E-02	8.91E-02	9.79E-02	1.05E-01	1.10E-01	1.24E-01	1.25E-01
7	3.15E-03	2.35E-02	4.46E-02	6.14E-02	7.47E-02	8.53E-02	9.37E-02	1.00E-01	1.06E-01	1.18E-01	1.19E-01
8	3.04E-03	2.26E-02	4.27E-02	5.87E-02	7.15E-02	8.16E-02	8.96E-02	9.60E-02	1.01E-01	1.13E-01	
9	2.93E-03	2.17E-02	4.09E-02	5.62E-02	6.84E-02	7.80E-02	8.57E-02	9.18E-02	9.67E-02	1.08E-01	
10	2.82E-03	2.08E-02	3.91E-02	5.38E-02	6.54E-02	7.46E-02	8.19E-02	8.78E-02	9.25E-02	1.03E-01	
11	2.72E-03	1.99E-02	3.75E-02	5.15E-02	6.25E-02	7.13E-02	7.83E-02	8.39E-02	8.85E-02	9.80E-02	
12	2.62E-03	1.91E-02	3.59E-02	4.92E-02	5.98E-02	6.82E-02	7.49E-02	8.03E-02	8.46E-02	9.34E-02	
13	2.52E-03	1.82E-02	3.43E-02	4.71E-02	5.72E-02	6.52E-02	7.16E-02	7.67E-02	8.09E-02	8.90E-02	
14	2.43E-03	1.75E-02	3.28E-02	4.50E-02	5.46E-02	6.23E-02	6.84E-02	7.34E-02	7.73E-02	8.48E-02	
15	2.34E-03	1.67E-02	3.14E-02	4.30E-02	5.22E-02	5.95E-02	6.54E-02	7.01E-02	7.39E-02	8.07E-02	
16	2.26E-03	1.60E-02	3.00E-02	4.11E-02	4.99E-02	5.69E-02	6.25E-02	6.71E-02	7.07E-02	7.68E-02	
17	2.17E-03	1.53E-02	2.87E-02	3.93E-02	4.77E-02	5.44E-02	5.98E-02	6.41E-02	6.76E-02	7.31E-02	
18	2.09E-03	1.46E-02	2.74E-02	3.75E-02	4.55E-02	5.20E-02	5.71E-02	6.13E-02	6.46E-02	6.96E-02	
19	2.02E-03	1.40E-02	2.62E-02	3.58E-02	4.35E-02	4.97E-02	5.46E-02	5.86E-02	6.18E-02	6.62E-02	
20	1.94E-03	1.34E-02	2.50E-02	3.42E-02	4.16E-02	4.75E-02	5.22E-02	5.61E-02	5.91E-02	6.29E-02	
21	1.87E-03	1.28E-02	2.39E-02	3.27E-02	3.97E-02	4.54E-02	5.00E-02	5.36E-02	5.65E-02	5.99E-02	
22	1.81E-03	1.22E-02	2.28E-02	3.12E-02	3.80E-02	4.34E-02	4.78E-02	5.13E-02	5.40E-02	5.69E-02	
23	1.74E-03	1.17E-02	2.18E-02	2.99E-02	3.63E-02	4.15E-02	4.57E-02	4.91E-02	5.16E-02	5.41E-02	
24	1.68E-03	1.12E-02	2.08E-02	2.85E-02	3.47E-02	3.97E-02	4.38E-02	4.70E-02	4.94E-02		
25	1.62E-03	1.07E-02	1.99E-02	2.73E-02	3.32E-02	3.80E-02	4.19E-02	4.50E-02	4.72E-02		
26	1.57E-03	1.02E-02	1.90E-02	2.61E-02	3.18E-02	3.64E-02	4.01E-02	4.30E-02	4.51E-02		
27	1.51E-03	9.78E-03	1.82E-02	2.50E-02	3.04E-02	3.49E-02	3.84E-02	4.12E-02	4.30E-02		
28	1.46E-03	9.36E-03	1.74E-02	2.39E-02	2.91E-02	3.34E-02	3.68E-02	3.94E-02	4.11E-02		
29	1.41E-03	8.96E-03	1.67E-02	2.29E-02	2.79E-02	3.20E-02	3.53E-02	3.77E-02	3.92E-02		
30	1.37E-03	8.57E-03	1.59E-02	2.19E-02	2.67E-02	3.07E-02	3.38E-02	3.61E-02	3.74E-02		
31	1.33E-03	8.21E-03	1.53E-02	2.10E-02	2.57E-02	2.94E-02	3.24E-02	3.45E-02	3.56E-02		
32	1.28E-03	7.87E-03	1.46E-02	2.01E-02	2.46E-02	2.82E-02	3.10E-02	3.30E-02	3.39E-02		

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Age at first exposure (years)	Duration of exposure (years)										
	1	5	10	15	20	25	30	35	40	62	78
33	1.25E-03	7.54E-03	1.40E-02	1.93E-02	2.36E-02	2.71E-02	2.97E-02	3.15E-02	3.23E-02		
34	1.21E-03	7.23E-03	1.35E-02	1.85E-02	2.27E-02	2.60E-02	2.85E-02	3.01E-02	3.07E-02		
35	1.18E-03	6.94E-03	1.29E-02	1.78E-02	2.18E-02	2.50E-02	2.73E-02	2.87E-02	2.91E-02		
36	1.14E-03	6.67E-03	1.24E-02	1.71E-02	2.09E-02	2.40E-02	2.61E-02	2.73E-02	2.76E-02		
37	1.11E-03	6.41E-03	1.19E-02	1.65E-02	2.01E-02	2.30E-02	2.50E-02	2.60E-02	2.61E-02		
38	1.08E-03	6.17E-03	1.15E-02	1.58E-02	1.94E-02	2.21E-02	2.39E-02	2.47E-02	2.48E-02		
39	1.06E-03	5.94E-03	1.10E-02	1.52E-02	1.86E-02	2.12E-02	2.28E-02	2.34E-02	2.34E-02		
40	1.03E-03	5.72E-03	1.06E-02	1.47E-02	1.79E-02	2.03E-02	2.17E-02	2.21E-02	2.21E-02		
41	1.01E-03	5.51E-03	1.02E-02	1.41E-02	1.72E-02	1.94E-02	2.06E-02	2.09E-02	2.09E-02		
42	9.81E-04	5.32E-03	9.87E-03	1.36E-02	1.65E-02	1.86E-02	1.96E-02	1.98E-02	1.98E-02		
43	9.59E-04	5.13E-03	9.52E-03	1.31E-02	1.59E-02	1.77E-02	1.86E-02	1.86E-02	1.86E-02		
44	9.38E-04	4.95E-03	9.18E-03	1.26E-02	1.52E-02	1.69E-02	1.75E-02	1.75E-02	1.75E-02		
45	9.16E-04	4.78E-03	8.85E-03	1.21E-02	1.46E-02	1.60E-02	1.65E-02	1.65E-02	1.65E-02		
46	8.93E-04	4.62E-03	8.53E-03	1.17E-02	1.39E-02	1.52E-02	1.55E-02	1.55E-02			
47	8.71E-04	4.46E-03	8.23E-03	1.12E-02	1.33E-02	1.43E-02	1.45E-02	1.45E-02			
48	8.50E-04	4.31E-03	7.92E-03	1.07E-02	1.26E-02	1.35E-02	1.36E-02	1.36E-02			
49	8.31E-04	4.16E-03	7.63E-03	1.03E-02	1.20E-02	1.26E-02	1.27E-02	1.27E-02			
50	8.10E-04	4.02E-03	7.34E-03	9.81E-03	1.13E-02	1.18E-02	1.18E-02	1.18E-02			
51	7.87E-04	3.88E-03	7.04E-03	9.33E-03	1.06E-02	1.09E-02	1.09E-02				
52	7.65E-04	3.74E-03	6.75E-03	8.85E-03	9.94E-03	1.01E-02	1.01E-02				
53	7.44E-04	3.60E-03	6.44E-03	8.36E-03	9.25E-03	9.33E-03	9.33E-03				
54	7.24E-04	3.46E-03	6.13E-03	7.86E-03	8.55E-03	8.57E-03	8.57E-03				
55	7.00E-04	3.31E-03	5.82E-03	7.34E-03	7.84E-03	7.84E-03	7.84E-03				
56	6.74E-04	3.17E-03	5.49E-03	6.82E-03	7.14E-03	7.14E-03					
57	6.49E-04	3.02E-03	5.16E-03	6.29E-03	6.47E-03	6.47E-03					
58	6.24E-04	2.86E-03	4.81E-03	5.74E-03	5.82E-03	5.82E-03					
59	6.00E-04	2.70E-03	4.46E-03	5.19E-03	5.21E-03	5.21E-03					
60	5.71E-04	2.53E-03	4.10E-03	4.62E-03	4.62E-03	4.62E-03					
61	5.37E-04	2.36E-03	3.73E-03	4.07E-03	4.07E-03						
62	5.04E-04	2.18E-03	3.36E-03	3.55E-03	3.55E-03						
63	4.72E-04	2.00E-03	2.98E-03	3.07E-03	3.07E-03						
64	4.40E-04	1.81E-03	2.59E-03	2.62E-03	2.62E-03						
65	4.05E-04	1.63E-03	2.20E-03	2.20E-03	2.20E-03						
66	3.67E-04	1.44E-03	1.81E-03	1.81E-03							
67	3.29E-04	1.25E-03	1.47E-03	1.47E-03							
68	2.93E-04	1.06E-03	1.16E-03	1.16E-03							
69	2.58E-04	8.61E-04	8.91E-04	8.91E-04							
70	2.21E-04	6.53E-04	6.53E-04	6.53E-04							

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For calculation of Table \_Apx K-1, the following procedure was used. For each cell of the table, the lung cancer and mesothelioma partial lifetime risk corresponding to the age at first exposure and duration of exposure was calculated using selected models for lung cancer and mesothelioma and

10599 potency factors from Table 3-9 and 3-10, Then lung cancer and mesothelioma risks were statistically  
 10600 combined using the same procedure as described in Section 3.2.4.6.

## Appendix L Sensitivity Analysis of Exposures for DIY/Bystander Episodic Exposure Scenarios

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 10604

10605 As presented in Section 4.3.8, there are some uncertainties pertaining to the assumptions made for  
 10606 exposure durations for both DIY users and bystanders for the brake repair/replacement scenarios. This  
 10607 Appendix provides a more detailed analyses using various combinations of age at start of first exposure  
 10608 and duration of exposure for both the DIYers and the bystanders for both the brake repair/replacement  
 10609 and the UTV gasket repair/replacement scenarios.

10610  
 10611 In Table L-1, the assumption is that DIY brake/repair replacement with compressed air begins at age 16  
 10612 years and continues for 20 years instead of for 62 years.

10614 Here, the unit risk for Users is:  $IUR_{LTL}(DIY\ Brakes) = IUR(16,20) = 0.0499$  per f/cc  
 10615 The unit risk for Bystanders is:  $IUR_{LTL}(DIY\ Bystanders) = IUR(0,20) = 0.101$  per f/cc  
 10616

10617 **Table\_Apx L-1. Excess Lifetime Cancer Risk for Indoor DIY Brake/Repair Replacement with**  
 10618 **Compressed Air Use for Consumers for 20 year duration (exposures from Table 2-32 without a**  
 10619 **reduction factor) (Consumers 1 hour/day spent in garage).**

Consumer Exposure Scenario	Exposure Levels (fibers/cc)				ELCR (20 yr exposure starting at age 16 years)		ELCR ((20 yr exposure starting at age 0 years))	
	DIY User		DIY Bystander		DIY User		DIY Bystander	
	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end
Aftermarket automotive parts – brakes (3-hour TWA indoors every 3 years with compressed air)	0.0445	0.4368	0.0130	0.0296	<b>2.8 E-5</b>	<b>2.7 E-4</b>	<b>1.7 E-5</b>	<b>3.8 E-5</b>

10620  $TWF_{Concomitant\ Exposures\ (1\ hour\ per\ day\ every\ day)} = (1/24) * (365/365) = 0.04167$   
 10621 DIY User:  $ELCR_{(Central\ Tendency)} = 0.0445\ f/cc \cdot 0.0001142 \cdot 0.0499\ per\ f/cc + 0.0445 \cdot 0.3 \cdot 0.04167 \cdot 0.0499$   
 10622 DIY User:  $ELCR_{(High-end)} = 0.4368\ f/cc \cdot 0.0001142 \cdot 0.0499\ per\ f/cc + 0.4368 \cdot 0.3 \cdot 0.04167 \cdot 0.0499$   
 10623 DIY Bystander:  $ELCR_{(Central\ Tendency)} = 0.013\ f/cc \cdot 0.0001142 \cdot 0.101\ per\ f/cc + 0.013 \cdot 0.3 \cdot 0.04167 \cdot 0.101$   
 10624 DIY Bystander:  $ELCR_{(High-end)} = 0.0296\ f/cc \cdot 0.0001142 \cdot 0.101\ per\ f/cc + 0.0296 \cdot 0.3 \cdot 0.04167 \cdot 0.101$   
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10626 Exposure values from Table 2-32 were used to represent indoor brake work (with compressed air) and  
 10627 are the basis for the exposure levels used in Table\_Apx L-1. EPA then assumed that the concentration of  
 10628 chrysotile asbestos in the interval between brake work (every 3 years) is 30% of that during measured  
 10629 active use. Consumers were assumed to spend one hour per day in their garages based on the 50<sup>th</sup>  
 10630 percentile estimate in the EPA Exposure Factors Handbook. Based on these assumptions, the consumer  
 10631 risk estimates were exceeded for central tendency and high-end exposures (L-1). Estimates exceeding  
 10632 the benchmark are shaded in pink and bolded.

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 10634 Comparing these results with those of Table 4-38, we see that the ratio of the risks for the DIY User  
 10635 based on 20 years exposure compared to 40 years of exposures is equal to the ratio of the less than  
 10636 lifetime inhalation unit risks:

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DIY Users: [IUR(16,20) = 0.0499 per f/cc] / [IUR(16,62) = 0.0768 per f/cc] = **0.65**  
 DIY Users: [20 yr risk (Central) = 2.80 E-5] / [62 yr risk (Central) = 4.31 E-5] = **0.65**  
 DIY Users: [20 yr risk (High) = 2.74 E-4] / [62 yr risk (High) = 4.23 E-4] = **0.65**

Similarly for bystanders, the ratio of the risk based on 20 years exposure compared to 62 years exposure is equal to the ratio of the 20-year less than lifetime risk to the lifetime unit risk:

DIY Bystanders: [IUR(0,20) = 0.101 per f/cc] / [IUR(Lifetime) = 0.16 per f/cc] = **0.63**  
 DIY Bystanders: [20 yr risk (Central) = 1.66 E-5] / [78 yr risk (Central) = 2.62 E-5] = **0.63**  
 DIY Bystanders: [20 yr risk (High) = 3.77 E-5] / [78 yr risk (High) = 5.97 E-5] = **0.63**

Using this approach, and relying on the ratios presented in Table 4-49, Table\_Apx L-2 provides and ratios for five different sensitivity pairings.

**Table\_Apx L-2. Ratios of risk for alternative exposure scenarios compared to DIY User and Bystander exposure scenario assuming DIY User is first exposed at age 16 years for 62 years duration and DIY Bystander is exposed from age 0-78 years.**

Exposure scenario		Age at first exposure (years)	Duration (years)	Baseline partial lifetime IUR	Exposure scenario partial lifetime IUR	Ratio of risks for exposure scenario
Baseline	DIY User	16	62	0.0768	0.0768	1
	Bystander	0	78	0.16	0.16	1
Sensitivity #1	DIY User	16	20	0.0768	0.0499	0.65
	Bystander	0	20	0.16	0.101	0.63
Sensitivity #2	DIY User	20	40	0.0768	0.0591	0.77
	Bystander	0	40	0.16	0.144	0.90
Sensitivity #3	DIY User	20	20	0.0768	0.0416	0.54
	Bystander	0	20	0.16	0.101	0.63
Sensitivity #4	DIY User	30	40	0.0768	0.0374	0.49
	Bystander	0	40	0.16	0.144	0.90
Sensitivity #5	DIY User	30	20	0.073	0.0267	0.37
	Bystander	0	20	0.16	0.101	0.63

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10657 Table\_Apx L-3through Table\_Apx L-7 below show the results of applying these ratios to all of the  
 10658 possible scenarios presented in Table 4-48 using the five sensitivity analyses pairings in Table\_Apx L-2.  
 10659 Table\_Apx L-8 at the end summarizes the results to show how only one of 24 scenarios changes from an  
 10660 exceedence to no exceedence for four (1, 3, 4, 5) of the five sensitivity analyses (DIY user, Brakes  
 10661 Repair/ replacement, Outdoor, once every 3 years, 30 min/d in driveway, high-end only).  
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10663 **Table\_Apx L-3. Sensitivity Analysis #1: Summary of Risk Estimates for Inhalation Exposures to**  
 10664 **Consumers and Bystanders by COU (Cancer benchmark is 10<sup>-6</sup>) Comparing the Baseline**  
 10665 **Exposure Scenario from Table 4-45 with Risks Assuming DIY Users Are Exposed From Age 16-36**  
 10666 **years and Bystanders Are Exposed Age 0-20 years.**  
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Life Cycle Stage/Category	Subcategory	Consumer Exposure Scenario	Population	Exposure Duration and Level	Cancer Risk Estimates (from Table 4-45)	Cancer Risk Estimates Users age 16-36 (*0.65) and Bystanders 0-20 (*0.63)
Imported asbestos products	Brakes Repair/replacement Indoor, compressed air, 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 4.2.3.1	DIY	Central Tendency	4.3 E-5	2.8 E-5
				High-end	4.2 E-4	2.7 E-5
			Bystander	Central Tendency	2.6 E-5	1.6 E-5
				High-end	6.0 E-5	3.8 E-5
	Brakes Repair/ replacement Indoor, compressed air, once every 3 years for 62/20 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	2.2 E-4
				High-end	3.4 E-3	2.2 E-3
			Bystander	Central Tendency	2.6 E-5	1.6 E-5
				High-end	6.0 E-5	3.8 E-5
	Brakes Repair/ replacement Outdoor, once every 3 years for 62/20 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	6.4 E-8
				High-end	5.3 E-7	3.4 E-7
			Bystander	Central Tendency	2.1 E-8	1.3 E-8
				High-end	1.1 E-7	6.9 E-8
Brakes Repair/ replacement Outdoor, once every 3 years for 62/20 years starting at 16 years, exposures at 2% of active used between	Section 4.2.3.1	DIY	Central Tendency	2.9 E-7	1.9 E-7	
			High-end	1.5 E-6	9.8 E-7	
		Bystander	Central Tendency	5.9 E-8	3.7 E-8	

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<b>Life Cycle Stage/Category</b>	<b>Subcategory</b>	<b>Consumer Exposure Scenario</b>	<b>Population</b>	<b>Exposure Duration and Level</b>	<b>Cancer Risk Estimates (from Table 4-45)</b>	<b>Cancer Risk Estimates Users age 16-36 (*0.65) and Bystanders 0-20 (*0.63)</b>
	uses, 30 min/d in driveway			High-end	3.2 E-7	2.0 E-7
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 4.2.3.2	DIY	Central Tendency	<b>2.3 E-5</b>	<b>1.5 E-5</b>
				High-end	<b>6.4 E-5</b>	<b>4.2 E-5</b>
			Bystander	Central Tendency	<b>2.4 E-5</b>	<b>1.5 E-5</b>
				High-end	<b>6.1 E-5</b>	<b>3.8 E-5</b>
	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, 8 hour/d in garage	Section 4.2.3.2	DIY	Central Tendency	<b>1.8 E-4</b>	<b>1.2 E-4</b>
				High-end	<b>5.1 E-4</b>	<b>3.3 E-4</b>
			Bystander	Central Tendency	<b>2.4 E-5</b>	<b>1.5 E-5</b>
				High-end	<b>6.1 E-5</b>	<b>3.8 E-5</b>

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**Table\_Apx L-4. Sensitivity Analysis #2: Summary of Risk Estimates for Inhalation Exposures to Consumers and Bystanders by COU (Cancer benchmark is 10<sup>-6</sup>) Comparing the Baseline Exposure Scenario from Table 4-45 with Risks Assuming DIY Users Are Exposed From Age 20-60 years and Bystanders Are Exposed Age 0-40 years.**

Life Cycle Stage/Category	Subcategory	Consumer Exposure Scenario	Population	Exposure Duration and Level	Cancer Risk Estimates (from Table 4-45)	Cancer Risk Estimates Users age 20-60 (*0.77) and Bystanders 0-40 (*0.90)
Imported asbestos products	Brakes Repair/replacement Indoor, compressed air, once every 3 years for 62/40 years starting at 16/20 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	3.3 E-5
				High-end	4.2 E-4	3.2 E-4
			Bystander	Central Tendency	2.6 E-5	2.3 E-5
				High-end	6.0 E-5	5.4 E-5
	Brakes Repair/replacement Indoor, compressed air, once every 3 years for 62/40 years starting at 16/20 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	2.6 E-4
				High-end	3.4 E-3	2.6 E-3
			Bystander	Central Tendency	2.6 E-5	2.3 E-5
				High-end	6.0 E-5	5.4 E-5
	Brakes Repair/replacement Outdoor, once every 3 years for 62/40 years starting at 16/20 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	7.6 E-8
				High-end	5.3 E-7	4.1 E-7
			Bystander	Central Tendency	2.1 E-8	1.9 E-8
				High-end	1.1 E-7	9.9 E-8
Brakes Repair/replacement Outdoor, once every 3 years for 62/40 years starting at 16/20 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	2.2 E-7	
			High-end	1.5 E-6	1.2 E-6	
		Bystander	Central Tendency	5.9 E-8	5.3 E-8	
			High-end	3.2 E-7	2.9 E-7	
Imported Asbestos Products	Gaskets Repair/replacement in UTVs	Section 4.2.3.2	DIY	Central Tendency	2.3 E-5	1.8 E-5
				High-end	6.4 E-5	4.9 E-5

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Life Cycle Stage/Category	Subcategory	Consumer Exposure Scenario	Population	Exposure Duration and Level	Cancer Risk Estimates (from Table 4-45)	Cancer Risk Estimates Users age 20-60 (*0.77) and Bystanders 0-40 (*0.90)
	Indoor, 1 hour/d, once every 3 years for 62/40 years starting at 16/20 years exposures at 30% of active used between uses, 1 hour/d in garage		Bystander	Central Tendency	2.4 E-5	2.2 E-5
				High-end	6.1 E-5	5.5 E-5
	Gaskets Repair/ replacement in UTVs Indoor, 1 hour/d, once every 3 years for 62/40 years starting at 16/20 years exposures at 30% of active used between uses, 8 hour/d in garage	Section 4.2.3.2	DIY	Central Tendency	1.8 E-4	1.4 E-4
				High-end	5.1 E-4	3.9 E-4
			Bystander	Central Tendency	2.4 E-5	2.2 E-5
				High-end	6.1 E-5	5.5 E-5

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**Table\_Apx L-5. Sensitivity Analysis #3: Summary of Risk Estimates for Inhalation Exposures to Consumers and Bystanders by COU (Cancer benchmark is 10<sup>-6</sup>) Comparing the Baseline Exposure Scenario from Table 4-45 with Risks Assuming DIY Users Are Exposed From Age 20-40 years and Bystanders Are Exposed Age 0-20 years.**

Life Cycle Stage/Category	Subcategory	Consumer Exposure Scenario	Population	Exposure Duration and Level	Cancer Risk Estimates (from Table 4-45)	Cancer Risk Estimates Users age 20-40 (*0.54) and Bystanders 0-20 (*0.63)
Imported asbestos products	Brakes Repair/replacement	Section 4.2.3.1	DIY	Central Tendency	4.3 E-5	2.3 E-5
				High-end	4.2 E-4	2.3 E-4



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<b>Life Cycle Stage/Category</b>	<b>Subcategory</b>	<b>Consumer Exposure Scenario</b>	<b>Population</b>	<b>Exposure Duration and Level</b>	<b>Cancer Risk Estimates (from Table 4-45)</b>	<b>Cancer Risk Estimates Users age 20-40 (*0.54) and Bystanders 0-20 (*0.63)</b>
	Indoor, compressed air, once every 3 years for 62/20 years starting at 16/20 years, exposures at 30% of active used between uses, 1 hour/d in garage		Bystander	Central Tendency	2.6 E-5	1.6 E-5
				High-end	6.0 E-5	3.8 E-5
	Brakes Repair/ replacement Indoor, compressed air, once every 3 years for 62/20 years starting at 16/20 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	1.8 E-4
				High-end	3.4 E-3	1.8 E-3
			Bystander	Central Tendency	2.6 E-5	1.6 E-5
				High-end	6.0 E-5	3.8 E-5
	Brakes Repair/ replacement Outdoor, once every 3 years for 62/20 years starting at 16/20 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	5.3 E-8
				High-end	5.3 E-7	2.8 E-7
			Bystander	Central Tendency	2.1 E-8	1.3 E-8
				High-end	1.1 E-7	6.9 E-8
	Brakes Repair/ replacement Outdoor, once every 3 years for 62/20 years starting at 16/20 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	1.6 E-7
				High-end	1.5 E-6	8.1 E-7
Bystander			Central Tendency	5.9 E-8	3.7 E-8	
			High-end	3.2 E-7	2.0 E-7	
Imported Asbestos Products	Gaskets Repair/ replacement in UTVs Indoor, 1 hour/d, once every 3 years for 62/20 years starting at 16/20 years	DIY	Central Tendency	2.3 E-5	1.2 E-5	
			High-end	6.4 E-5	3.5 E-5	
		Bystander	Central Tendency	2.4 E-5	1.5 E-5	

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<b>Life Cycle Stage/Category</b>	<b>Subcategory</b>	<b>Consumer Exposure Scenario</b>	<b>Population</b>	<b>Exposure Duration and Level</b>	<b>Cancer Risk Estimates (from Table 4-45)</b>	<b>Cancer Risk Estimates Users age 20-40 (*0.54) and Bystanders 0-20 (*0.63)</b>
	exposures at 30% of active used between uses, 1 hour/d in garage			High-end	<b>6.1 E-5</b>	<b>3.8 E-5</b>
	Gaskets Repair/ replacement in UTVs Indoor, 1 hour/d, once every 3 years for 62/20 years starting at 16/20 years exposures at 30% of active used between uses, 8 hour/d in garage	Section 4.2.3.2	DIY	Central Tendency	<b>1.8 E-4</b>	<b>9.7 E-5</b>
				High-end	<b>5.1 E-4</b>	<b>2.8 E-4</b>
			Bystander	Central Tendency	<b>2.4 E-5</b>	<b>1.5 E-5</b>
				High-end	<b>6.1 E-5</b>	<b>3.8 E-5</b>

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**Table\_Apx L-6. Sensitivity Analysis #4: Summary of Risk Estimates for Inhalation Exposures to Consumers and Bystanders by COU (Cancer benchmark is 10<sup>-6</sup>) Comparing the Baseline Exposure Scenario from Table 4-45 with Risks Assuming DIY Users Are Exposed From Age 30-70 years and Bystanders Are Exposed Age 0-40 years.**

Life Cycle Stage/Category	Subcategory	Consumer Exposure Scenario	Population	Exposure Duration and Level	Cancer Risk Estimates (from Table 4-45)	Cancer Risk Estimates Users age 30-70 (*0.49) and Bystanders 0-40 (*0.90)
Imported asbestos products	Brakes Repair/replacement Indoor, compressed air, once every 3 years for 62/40 years starting at 16/30 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	2.1 E-5
				High-end	4.2 E-4	2.1 E-4
			Bystander	Central Tendency	2.6 E-5	2.3 E-5
				High-end	6.0 E-5	5.4 E-5
	Brakes Repair/replacement Indoor, compressed air, once every 3 years for 62/40 years starting at 16/30 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	1.7 E-4
				High-end	3.4 E-3	1.7 E-3
			Bystander	Central Tendency	2.6 E-5	2.3 E-5
				High-end	6.0 E-5	5.4 E-5
	Brakes Repair/replacement Outdoor, once every 3 years for 62/40 years starting at 16/30 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	4.9 E-8
				High-end	5.3 E-7	2.6 E-7
			Bystander	Central Tendency	2.1 E-8	1.9 E-8
				High-end	1.1 E-7	9.9 E-8
Brakes Repair/replacement Outdoor, once every 3 years for 62/40 years starting at 16/30 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	1.4 E-7	
			High-end	1.5 E-6	7.4 E-7	
		Bystander	Central Tendency	5.9 E-8	53 E-8	
			High-end	3.2 E-7	2.9 E-7	
Imported Asbestos Products	Gaskets Repair/replacement in UTVs	Section 4.2.3.2	DIY	Central Tendency	2.3 E-5	1.1 E-5
				High-end	6.4 E-5	3.1 E-5

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<b>Life Cycle Stage/Category</b>	<b>Subcategory</b>	<b>Consumer Exposure Scenario</b>	<b>Population</b>	<b>Exposure Duration and Level</b>	<b>Cancer Risk Estimates (from Table 4-45)</b>	<b>Cancer Risk Estimates Users age 30-70 (*0.49) and Bystanders 0-40 (*0.90)</b>
	Indoor, 1 hour/d, once every 3 years for 62/40 years starting at 16/30 years exposures at 30% of active used between uses, 1 hour/d in garage		Bystander	Central Tendency	<b>2.4 E-5</b>	<b>2.2 E-5</b>
				High-end	<b>6.1 E-5</b>	<b>5.5 E-5</b>
	Gaskets Repair/ replacement in UTVs Indoor, 1 hour/d, once every 3 years for 62/40 years starting at 16/30 years exposures at 30% of active used between uses, 8 hour/d in garage	Section 4.2.3.2	DIY	Central Tendency	<b>1.8 E-4</b>	<b>8.8 E-5</b>
				High-end	<b>5.1 E-4</b>	<b>2.5 E-4</b>
			Bystander	Central Tendency	<b>2.4 E-5</b>	<b>2.2 E-5</b>
				High-end	<b>6.1 E-5</b>	<b>5.5 E-5</b>

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**Table\_Apx L-7. Sensitivity Analysis #5: Summary of Risk Estimates for Inhalation Exposures to Consumers and Bystanders by COU (Cancer benchmark is 10<sup>-6</sup>) Comparing the Baseline Exposure Scenario from Table 4-45 with Risks Assuming DIY Users Are Exposed From Age 30-50 years and Bystanders Are Exposed Age 0-20 years.**

Life Cycle Stage/Category	Subcategory	Consumer Exposure Scenario	Population	Exposure Duration and Level	Cancer Risk Estimates (from Table 4-45)	Cancer Risk Estimates Users age 30-50 (*0.37) and Bystanders 0-20 (*0.63)
Imported asbestos products	Brakes Repair/replacement Indoor, compressed air, once every 3 years for 62/20 years starting at 16/30 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	1.6 E-5
				High-end	4.2 E-4	1.6 E-4
			Bystander	Central Tendency	2.6 E-5	1,6 E-5
				High-end	6.0 E-5	3.8 E-5
	Brakes Repair/replacement Indoor, compressed air, once every 3 years for 62/20 years starting at 16/30 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	1.3 E-4
				High-end	3.4 E-3	1.3 E-3
			Bystander	Central Tendency	2.6 E-5	1.6 E-5
				High-end	6.0 E-5	3.8 E-5
	Brakes Repair/replacement Outdoor, once every 3 years for 62/20 years starting at 16/30 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	3.7 E-8
				High-end	5.3 E-7	2.0 E-7
			Bystander	Central Tendency	2.1 E-8	1.3 E-8
				High-end	1.1 E-7	6.9 E-8
Brakes Repair/replacement Outdoor, once every 3 years for 62/20 years starting at 16/30 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	1.1 E-8	
			High-end	1.5 E-6	5.6 E-7	
		Bystander	Central Tendency	5.9 E-8	3.7 E-8	
			High-end	3.2 E-7	2.0 E-7	
Imported Asbestos Products	Gaskets Repair/replacement in UTVs	Section 4.2.3.2	DIY	Central Tendency	2.3 E-5	8.5 E-6
				High-end	6.4 E-5	2.4 E-5

Life Cycle Stage/Category	Subcategory	Consumer Exposure Scenario	Population	Exposure Duration and Level	Cancer Risk Estimates (from Table 4-45)	Cancer Risk Estimates Users age 30-50 (*0.37) and Bystanders 0-20 (*0.63)
	Indoor, 1 hour/d, once every 3 years for 62/20 years starting at 16/30 years exposures at 30% of active used between uses, 1 hour/d in garage		Bystander	Central Tendency	2.4 E-5	1.5 E-5
High-end				6.1 E-5	3.8 E-5	
	Gaskets Repair/ replacement in UTVs Indoor, 1 hour/d, once every 3 years for 62/20 years starting at 16/30 years exposures at 30% of active used between uses, 8 hour/d in garage	Section 4.2.3.2	DIY	Central Tendency	1.8 E-4	6.7 E-5
High-end				5.1 E-4	1.9 E-4	
Bystander			Central Tendency	2.4 E-5	1.5 E-5	
			High-end	6.1 E-5	3.8 E-5	

**Table\_Apx L-8: Results of 24 Sensitivity Analysis of Exposure Assumptions for Consumer DIY/Bystander Episodic Exposure Scenarios**

Sensitivity Analysis	DIY (age at start and age at end of duration)	Bystander (age at start and age at end of duration)	Change in Risk from Exceedence to No Exceedence	Scenario Affected
Baseline	16-78	0-78	None	17/24 Exceed Benchmarks
1	16-36	0-20	1/24	DIY user, Brake repair, 30 min/day, high-end
2	20-60	0-40	0/24	None
3	20-40	0-40	1/24	DIY user, Brake repair, 30 min/day, high-end
4	30-70	0-40	1/24	DIY user, Brake repair, 30 min/day, high-end
5	30-50	0-20	1/24	DIY user, Brake repair, 30 min/day, high-end