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Risk Evaluation for Asbestos Part I: Chrysotile Asbestos

NOTICE: experts including members of AIHA, the American Industrial Hygienists Association, do not agree with the U.S. EPA's conclusions in this document.

The overall logic of the U.S. EPA rulemaking is based on the erroneous assumption about the cancer risk for chrysotile asbestos exposure being substantial, while numerous scientific sources prove the opposite to be true.

CLICK ANYWHERE on THIS PAGE to RETURN TO ASBESTOS HAZARD INFORMATION at InspectApedia.com (e.g. Drywall dust hazards from Amphible & Chrysotile Asbestos)

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PREAMBLE

In this preamble, the Agency describes its approach to completing the Risk Evaluation for Asbestos under TSCA Section 6(a). The risk evaluation will be issued in two parts:

- Risk Evaluation for Asbestos Part 1: Chrysotile Asbestos (published with this preamble)
- Risk Evaluation for Asbestos Part 2: Legacy Uses and Associated Disposals of Asbestos (forthcoming)

Figure P-1 shows a timeline for the development of the risk evaluation for asbestos. It starts with the identification of asbestos as one of the First 10 Chemicals for risk evaluation under the Toxic Substances Control Act (TSCA) in December of 2016. A Scope document and a Problem Formulation document were then developed (2017 and 2018, respectively) and a draft risk evaluation (RE) was released to the public in March of 2020. In late 2019, the court in *Safer Chemicals, Healthy Families v. EPA*, 943 F.3d 397 (9th Cir. 2019) held that EPA's Risk Evaluation Rule, 82 FR 33726 (July 20, 2017), should not have excluded "legacy uses" (*i.e.*, uses without ongoing or prospective manufacturing, processing, or distribution) or "associated disposals" (*i.e.*, future disposal of legacy uses) from the definition of conditions of use, although the court upheld EPA's exclusion of "legacy disposals" (*i.e.*, past disposal). Due to the court ruling, in the March 2020 draft risk evaluation, EPA had signaled the inclusion of other fiber types, in addition to chrysotile, as well as consideration of legacy uses and associated disposal for the asbestos risk evaluation in a supplemental scope document and supplemental risk evaluation when these activities are known, intended, or reasonably foreseen. This was supported by both public comment and the SACC during the SACC Peer Review (virtual) meeting.

Figure P-2 is a text box with definitions for terms and documents important to understanding the shift in the development of the risk evaluation for asbestos from 2016 to the present (2020).

The Path to Finalizing the Risk Evaluation for Asbestos: Parts 1 and 2

After considering SACC recommendations and public comments on the March 2020 draft risk evaluation of asbestos, EPA decided to divide the risk evaluation into two parts: Part 1 on chrysotile asbestos (herein) and Part 2 on legacy uses and associated disposal of asbestos (forthcoming). Together, the documents will make up the risk evaluation for asbestos under TSCA Section 6.

Part 1, which accompanies this Preamble, completes the evaluation of chrysotile asbestos imported, processed and distributed for use in the United States. EPA is confident that the chrysotile asbestos conditions of use (COUs) represent all intended, known, or reasonably foreseen import, processing, and distribution of chrysotile asbestos; uses of chrysotile asbestos that have been imported, processed, and distributed; and disposal of such chrysotile asbestos uses.

In finalizing the risk evaluation Part 1 (chrysotile asbestos), EPA made appropriate and necessary changes to update the document to reflect the best available science (following the standard in TSCA section 26(h)) to support the risk determination and inform risk management decision for the conditions of use evaluated in this document based on recommendations from the SACC and public comment. These changes are reflected in the accompanying *Response to Comments* document. However, some recommendations and comments that were identified in the SACC report are more relevant to what EPA will address in Part 2 of the risk evaluation for asbestos (*i.e.*, for legacy uses, including chrysotile and other fiber types of asbestos).

EPA has initiated the process for Part 2 and is currently identifying the relevant information available. EPA will describe the COUs and the fiber types to be examined in a scope document that is currently under development and will be made available for public comment. After review and consideration of public comments, EPA will revise, where appropriate, and publish a final scope document. The legacy uses and associated disposals of chrysotile asbestos were excluded from the Scope document for Part 1 and will be included in Part 1. Thus, the COUs included in Part 1 and those to be included in Part 2 will not overlap. Subsequent to finalizing the Scope, EPA will develop Part 2 of the risk evaluation for asbestos.

Figure P-1: Schematic of the TSCA Risk Evaluation Timeline

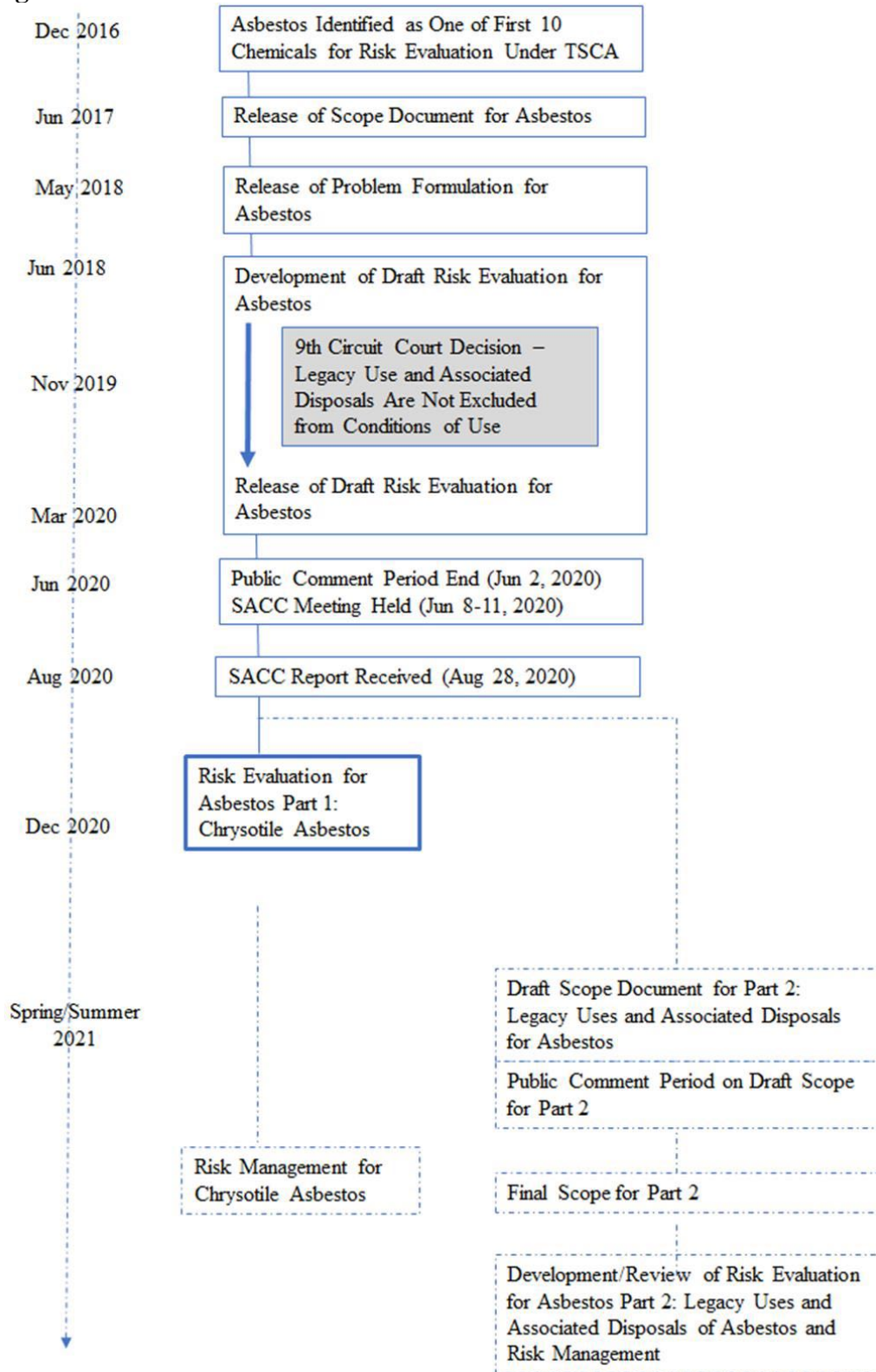


Figure P-2: Important Definitions for the Risk Evaluation for Asbestos

Definitions

Asbestos. For the purposes of the Risk Evaluation for asbestos under TSCA Section 6(a), EPA is using the TSCA Title II (added to TSCA in 1986), Section 202 definition; which is - “asbestiform varieties of six fiber types – chrysotile (serpentine), crocidolite (riebeckite), amosite (cummingtonite-grunerite), anthophyllite, tremolite or actinolite.” The latter five fiber types are amphibole varieties. This definition was previously defined in the scope document and has consistently been applied in this risk evaluation process.

Chrysotile Asbestos. One of the six fiber types of asbestos as defined above. Chrysotile asbestos is the only fiber type currently being imported, processed, or distributed in the United States. These activities, along with the ensuing uses and disposals, encompass the Conditions of Use (COUs) presented in Part 1 of the Risk Evaluation for asbestos.

Draft Risk Evaluation for Asbestos. The title of the March 2020 publicly released draft risk evaluation. Although the draft was focused on chrysotile asbestos, the title and contents of the document generated some confusion as was evident by peer review and public comments received. Throughout this document (*i.e.*, Risk Evaluation for Asbestos Part 1: Chrysotile Asbestos), the term is used only to refer to the March 2020 *draft* risk evaluation.

Risk Evaluation for Asbestos. The risk evaluation for asbestos will consist of two Parts: Part 1 is on chrysotile asbestos (finalized December 2020) and Part 2 will be on legacy uses and associated disposal, including the five other fiber types of asbestos (scope and risk evaluation are forthcoming).

Risk Evaluation for Asbestos: Part 1 - Chrysotile Asbestos. The December 2020 risk evaluation of the asbestos fiber type (chrysotile) currently imported, processed and distributed for use in the United States. Hereafter, referred to as Part 1 or Part 1 of the risk evaluation.

Risk Evaluation for Asbestos: Part 2 – Legacy uses and associated disposals of asbestos. The forthcoming risk evaluation for the legacy uses and associated disposals, including the

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Docket

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

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ABBREVIATIONS

ABPO	1989 Asbestos Ban and Phase Out Rule
ACC	American Chemistry Council
ADC	Average Daily Concentration
AHERA	Asbestos Hazard Emergency Response Act
AIC	Akaike Information Criterion
ASHAA	Asbestos School Hazard Abatement Act
ASHARA	Asbestos School Hazard Abatement Reauthorization Act
ATSDR	Agency for Toxic Substances and Disease Registry
CAA	Clean Air Act
CASRN	Chemical Abstracts Service Registry Number
CBI	Confidential Business Information
CDR	Chemical Data Reporting
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act
COU	Condition of Use
CPSC	Consumer Product Safety Commission
CWA	Clean Water Act
DIY	Do-It-Yourself
DPT	Diffuse Pleural Thickening
EG	Effluent Guideline
ELCR	Excess Lifetime Cancer Risk
EMP	Elongated Mineral Particle
EPA	Environmental Protection Agency
EPCRA	Emergency Planning and Community Right-to-Know Act
EU	European Union
FDA	Food and Drug Administration
f/cc	Fibers per cubic centimeter
FHSA	Federal Hazardous Substance Act
g	Gram(s)
HAP	Hazardous Air Pollutant
HEPA	High-Efficiency Particulate Air
HTS	Harmonized Tariff Schedule
IARC	International Agency for Research on Cancer
IRIS	Integrated Risk Information System
IUR	Inhalation Unit Risk
K _l	Lung cancer potency factor
K _m	Mesothelioma potency factor
LADC	Lifetime Average Daily Concentration
lb	Pound
LTL	Less Than Lifetime
LOEC	Lowest Observable Effect Concentration
MAP	Model Accreditation Plan
MCLG	Maximum Contaminant Level Goal
µm	Micrometers
MFL	Million Fibers per Liter
mppcf	million particles per cubic foot of air
mg	Milligram(s)
MPa	Megapascal
MSHA	Mine Safety and Health Administration

mV	Millivolt
NAICS	North American Industry Classification System
ND	Non-detects (value is < analytical detection limit)
NEI	National Emissions Inventory
NESHAP	National Emission Standard for Hazardous Air Pollutants
NIH	National Institutes of Health
NIOSH	National Institute for Occupational Safety and Health
NMRD	Non-Malignant Respiratory Disease
NPL	National Priorities List
NTP	National Toxicology Program
OCSPP	Office of Chemical Safety and Pollution Prevention
OEM	Original Equipment Manufacturer
ONU	Occupational Non-User
OPPT	Office of Pollution Prevention and Toxics
OSHA	Occupational Safety and Health Administration
PCM	Phase Contrast Microscopy
PECO	Population, Exposure, Comparator and Outcome
PEL	Permissible Exposure Limit
PESO	Pathways/Processes, Exposure, Setting and Outcomes
PF	Problem Formulation
POD	Point of Departure
POTW	Publicly Owned Treatment Works
PPE	Personal Protective Equipment
ppm	Part(s) per Million
RCRA	Resource Conservation and Recovery Act
RA	Risk Assessment
RESO	Receptors, Exposure, Setting/Scenario and Outcomes
RfC	Reference Concentration
RIA	Regulatory Impact Analysis
RR	Relative Risk
SACC	Science Advisory Committee on Chemicals
SDS	Safety Data Sheet
SDWA	Safe Drinking Water Act
SMR	Standardized Mortality Ratio
SNUN	Significant New Use Notice
SNUR	Significant New Use Rule
TSFE	Time Since First Exposure
TCCR	Transparent, Clear, Consistent, and Reasonable
TEM	Transmission Electron Microscopy
TRI	Toxics Release Inventory
TSCA	Toxic Substances Control Act
TURA	Toxics Use Reduction Act
TWA	Time Weighted Average
UB	Upper Bound
U.S.	United States
USGS	United States Geological Survey
UTV	Utility vehicle
WHO	World Health Organization

1 EXECUTIVE SUMMARY

2 This Risk Evaluation for Asbestos, Part 1: Chrysotile Asbestos (hereafter referred to as “Part 1” or “Part
3 1 of the risk evaluation”) for imported, processed and distributed uses of chrysotile asbestos¹ was
4 performed in accordance with the Frank R. Lautenberg Chemical Safety for the 21st Century Act and is
5 being issued following public comment and peer review. The Frank R. Lautenberg Chemical Safety for
6 the 21st Century Act amended the Toxic Substances Control Act (TSCA), the Nation’s primary
7 chemicals management law, in June 2016. Under the amended statute, EPA is required, under TSCA
8 Section 6(b), to conduct risk evaluations to determine whether a chemical substance presents an
9 unreasonable risk of injury to health or the environment, under the conditions of use, without
10 consideration of costs or other non-risk factors, including an unreasonable risk to potentially exposed or
11 susceptible subpopulations identified as relevant to the Risk Evaluation. Also, as required by TSCA
12 Section 6(b), EPA established, by rule, a process to conduct these Risk Evaluations, *Procedures for*
13 *Chemical Risk Evaluation Under the Amended Toxic Substances Control Act* (82 FR 33726) (Risk
14 Evaluation Rule). Part 1 of the risk evaluation is in conformance with TSCA Section 6(b) and the Risk
15 Evaluation Rule and is to be used to inform risk management decisions. In accordance with TSCA
16 Section 6(b), if EPA finds unreasonable risk from a chemical substance under its conditions of use in
17 any final Risk Evaluation, the Agency will propose actions to address those risks within the timeframe
18 required by TSCA. However, any proposed or final determination that a chemical substance presents
19 unreasonable risk under TSCA Section 6(b) is not the same as a finding that a chemical substance is
20 “imminently hazardous” under TSCA Section 7. The conclusions, findings, and determinations in Part 1
21 are for the purpose of identifying whether the chemical substance presents unreasonable risk under the
22 conditions of use, in accordance with TSCA section 6, and are not intended to represent any findings
23 under TSCA section 7.

24 TSCA § 26(h) and (i) require EPA, when conducting Risk Evaluations, to use scientific information,
25 technical procedures, measures, methods, protocols, methodologies and models consistent with the best
26 available science and base its decisions on the weight of the scientific evidence². To meet these TSCA §
27 26 science standards, EPA used the TSCA systematic review process described in the *Application of*
28 *Systematic Review in TSCA Risk Evaluations* document ([U.S. EPA, 2018a](#)). The data collection,
29 evaluation, and integration stages of the systematic review process are used to develop the exposure, fate
30 and hazard assessments for the risk evaluations. To satisfy requirements in TSCA Section 26(j)(4) and
31 40 CFR 702.51(e), EPA has provided a list of studies considered in carrying out Part 1 and the results of
32 those studies are included in the *Systematic Review Data Quality Evaluation Documents* (see Appendix
33 B).

34 Asbestos is subject to federal and state regulations and reporting requirements. Asbestos is reportable to
35 the Toxics Release Inventory (TRI) under Section 313 of the Emergency Planning and Community

¹ As noted in the **PREAMBLE**, this document is Part 1 of the final Risk Evaluation for asbestos and is limited to chrysotile asbestos and the conditions of use (COUs) defined in this document. Part 2 is forthcoming and will be on legacy uses and associated disposal of asbestos.

² Weight of the scientific evidence is defined in EPA regulations 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 CFR 702.33.

36 Right-to-Know Act (EPCRA) but is only reportable in the friable³ form at concentration levels of 0.1%
37 or greater. It is designated a Hazardous Air Pollutant (HAP) under the Clean Air Act (CAA), and is a
38 hazardous substance under the Comprehensive Environmental Response, Compensation and Liability
39 Act (CERCLA). Asbestos is subject to National Primary Drinking Water Regulations (NPDWR) under
40 the Safe Drinking Water Act (SDWA) and designated as a toxic pollutant under the Clean Water Act
41 (CWA) and as such is subject to effluent limitations. Under TSCA, EPA has promulgated several
42 regulations for asbestos, including the Asbestos Ban and Phase Out rule of 1989, which was then largely
43 vacated in 1991, and under the Asbestos Hazard Emergency Response Act (AHERA), which requires
44 inspection of schools for asbestos. On April 25, 2019, EPA finalized an Asbestos Significant New Use
45 Rule (SNUR) under TSCA Section 5 that prohibits manufacture (including import) or processing of
46 discontinued uses of asbestos from restarting without EPA having an opportunity to evaluate each
47 intended use for risks to health and the environment and to take any necessary regulatory action, which
48 may include a prohibition.

49 Asbestos has not been mined or otherwise produced in the U.S. since 2002. Although there are several
50 known types of asbestos, the only form of asbestos known to be imported, processed, or distributed for
51 use in the United States is chrysotile asbestos. As a naturally occurring mineral, chrysotile can co-occur
52 with other minerals, including amphibole forms of asbestos. Trace amounts of these minerals may
53 remain in chrysotile as it is used in commerce. This commercial chrysotile, rather than theoretically
54 “pure” chrysotile, is therefore the substance of concern for this assessment. Raw chrysotile asbestos
55 currently imported into the U.S. is used exclusively by the chlor-alkali industry. The total amount of raw
56 chrysotile asbestos imported into the U.S. in 2019 was 100 metric tons. EPA has also identified the
57 importation of asbestos-containing products; however, the import volumes of those products are not
58 fully known. The asbestos-containing products that EPA has identified as being imported and used are
59 sheet gaskets, brake blocks, aftermarket automotive brakes/linings, other vehicle friction products, and
60 other gaskets. In Part 1 of the asbestos risk evaluation, EPA evaluated the following categories of
61 conditions of use (COU): importing; processing; distribution in commerce; occupational and consumer
62 uses; and disposal.

63 Approach

64 EPA used reasonably available information (defined in 40 CFR 702.33 as “*information that EPA*
65 *possesses or can reasonably generate, obtain, and synthesize for use in risk evaluations, considering the*
66 *deadlines specified in TSCA section 6(b)(4)(G) for completing such evaluation*”), in a fit-for-purpose
67 approach, to develop a document that relies on the best available science and is based on the weight of
68 the scientific evidence. EPA used previous analyses as a starting point for identifying key and
69 supporting studies to inform the exposure, fate, and hazard assessments. EPA also evaluated other
70 studies published since the publication of previous analyses. EPA reviewed the information and
71 evaluated the quality of the methods and reporting of results of the individual studies using the
72 evaluation strategies described in *Application of Systematic Review in TSCA Risk Evaluations* ([U.S.](#)
73 [EPA, 2018a](#)).

74 During development of this Part 1 of the risk evaluation for asbestos, the only asbestos fiber type that
75 EPA identified as imported, processed, or distributed under the COUs in the United States is chrysotile,
76 the serpentine variety. Chrysotile is the prevailing form of asbestos currently mined worldwide, and so it
77 is assumed that a majority of commercially available products fabricated overseas that contain asbestos

³ The TRI listing has the following definition for 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.” (40 CFR Part 372).

78 are made with chrysotile. Any asbestos being imported into the U.S. in articles is believed to be
79 chrysotile. The other five forms of asbestos are now subject to a SNUR⁴.

80 EPA evaluated the following categories of COU of chrysotile asbestos in this Part 1 of the risk
81 evaluation for asbestos: importing; processing; distribution in commerce; occupational and consumer
82 uses (use of diaphragms in the chlor-alkali industry, sheet gaskets in chemical production facilities,
83 oilfield brake blocks, aftermarket automotive brakes/linings, other vehicle friction products, and other
84 gaskets); and disposal. EPA reviewed the court decision in *Safer Chemicals Healthy Families v. EPA*,
85 943 F.3d 397 (9th Cir. 2019). This Part 1 of the risk evaluation for asbestos does not reflect
86 consideration of any legacy uses and associated disposal for chrysotile asbestos or other asbestos fiber
87 types as a result of that decision. EPA intends to consider legacy uses and associated disposal and other
88 fiber types in Part 2 of the asbestos risk evaluation.

89 In the [problem formulation](#) (U.S. EPA, 2018d) (PF), EPA identified the COUs and presented three
90 conceptual models and an analysis plan. These have been carried into this document where EPA has
91 quantitatively evaluated the risk to human health using monitoring data submitted by industry and found
92 in the scientific literature through systematic review for the COUs (identified in Section 1.4.3 of this
93 Part 1 of the risk evaluation for asbestos).

94 During the PF phase of the Risk Evaluation, EPA was still in the process of identifying potential
95 chrysotile asbestos water releases for the TSCA COUs to determine the need to evaluate risk to aquatic
96 and sediment-dwelling organisms. In the draft Risk Evaluation released in March 2020, EPA concluded
97 that, based on the reasonably available information in the published literature, provided by industries
98 using asbestos, and reported in EPA databases, there were minimal or no releases of asbestos to surface
99 water associated with the COUs that EPA is evaluating in Part 1. EPA has considered peer review and
100 public comments on this conclusion and has retained the finding in the draft Risk Evaluation that there is
101 low or no potential for environmental risk to aquatic or sediment-dwelling receptors from the COUs
102 included in this Part 1 of the risk evaluation for asbestos. This is because EPA is confident that the
103 minimal water release data cannot be attributed to chrysotile asbestos from the COUs in this document.
104 However, in Part 2 of the Risk Evaluation for Asbestos that will examine legacy uses and associated
105 disposals of asbestos, EPA expects to address the issue of releases to surface water based on those other
106 asbestos uses (See Section 4.1).

107 In occupational settings, EPA evaluated inhalation exposures to workers and occupational non-users, or
108 ONUs. EPA used inhalation monitoring data submitted by industry and literature sources, where
109 reasonably available and that met TSCA systematic review data evaluation criteria, to estimate potential
110 inhalation exposures. In consumer settings, EPA evaluated inhalation exposures to both consumers (Do-
111 it-Yourselfers or DIY mechanics) and bystanders and used estimated inhalation exposures, from
112 literature sources where reasonably available and that met data evaluation criteria, to estimate potential
113 exposures using a range of user durations. These analyses are described in Section 2.3.

114 EPA evaluated reasonably available information for human health hazards and identified hazard

⁴ 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.

115 endpoints for cancer. EPA used the *Framework for Human Health Risk Assessment to Inform Decision*
116 *Making* ([U.S. EPA, 2014a](#)) to evaluate, extract, and integrate asbestos' dose-response information. EPA
117 evaluated the large database of health effects associated with asbestos exposure cited in numerous U.S.
118 and international data sources. Many authorities have established that there are causal associations
119 between asbestos exposures and cancer ([NTP, 2016](#); [IARC, 2012b](#); [ATSDR, 2001a](#); [U.S. EPA, 1988b](#);
120 [IARC, 1987](#); [U.S. EPA, 1986](#); [IARC, 1977](#)).

121 EPA evaluated inhalation exposures to chrysotile asbestos in occupational and consumer settings in this
122 Part 1 of the risk evaluation for asbestos. Dermal exposures were identified as a possible route of
123 exposure in the PF but were not included in the evaluation since the only reported effects were dermal-
124 specific lesions, and the major hazard concern is development of cancer via inhalation. Chrysotile
125 asbestos is a fiber and is not expected to be absorbed into the body through the exterior skin surfaces and
126 be distributed to the lungs. Furthermore, as also described in the PF, non-cancer hazards from inhalation
127 exposures were identified for consideration at that time, but risks associated with non-cancer effects
128 were not quantified. Both the SACC and public comments suggested that EPA consider non-cancer
129 effects in Part 1 of the risk evaluation for asbestos; however, EPA maintains that the evaluation of
130 cancer effects and subsequent risk determinations, that consider non-cancer risks, are health protective
131 for the evaluated COUs for chrysotile asbestos. In Part 2 of the risk evaluation for asbestos that will
132 examine legacy uses and associated disposals, EPA will consider the reasonably available information
133 for cancer and non-cancer hazards.

134 Given the well-established carcinogenicity of asbestos for cancer, EPA, in its PF document, decided to
135 limit the scope of its systematic review to cancer and to inhalation exposures with the goal of updating,
136 or reaffirming, the existing 1988 EPA inhalation unit risk (IUR) for general asbestos ([U.S. EPA,](#)
137 [1988b](#)). Therefore, the literature was reviewed to determine whether a new IUR needed to be
138 developed. The IUR for asbestos developed in 1988 was based on 14 epidemiologic studies that
139 included occupational exposure to chrysotile, amosite, or mixed-mineral exposures [chrysotile, amosite,
140 crocidolite]. However, EPA's research to identify COUs indicated that only chrysotile asbestos is
141 currently being imported in the raw form or imported in products. Therefore, studies of populations
142 exposed only to chrysotile provide the most informative data for developing the TSCA risk estimates
143 for the COUs presently considered for chrysotile asbestos, and EPA decided to focus on studies where
144 the exposure was limited to chrysotile asbestos. EPA will consider legacy uses and associated disposals
145 for all 6 fiber types including in the AHERA Title II definition in Part 2 of the risk evaluation for
146 asbestos.

147 As stated in Section 3.2, epidemiological studies on mesothelioma and lung cancer in cohorts of workers
148 using chrysotile in commerce were identified that could inform the estimation of an exposure-response
149 function allowing for the derivation of a chrysotile asbestos IUR. EPA could not find any recent risk
150 values in the literature for chrysotile asbestos following the derivation of the IRIS IUR value from the
151 1980s.

152 Cancer potency values were either extracted from published epidemiology studies or derived from the
153 data within those studies. Once the cancer potency values were obtained, they were adjusted for
154 differences in air volumes between workers and other populations so that those values can be applied to
155 the U.S. population, as a whole, in standard EPA life-table analyses. The life-table methodology allows
156 the estimation of an exposure concentration associated with a specific extra risk of cancer incidence
157 caused by chrysotile asbestos. The risk of mesothelioma was adjusted to compensate for
158 underascertainment of mesothelioma. The risk of lung cancer was adjusted to account for the risk of

159 other established cancer endpoints (*i.e.*, cancers of larynx and ovary)⁵. According to standard
160 practice, the lifetime unit risks for lung cancer and mesothelioma were estimated separately and then
161 statistically combined to yield the cancer inhalation unit risk. Less-than-lifetime or partial lifetime unit
162 risks were also derived for a range of exposure scenarios based on different ages of first exposure and
163 different durations of exposure (*e.g.*, 20 years old and 40 years of exposure) (Section 3.2: Human Health
164 Hazards).

165 Risk Characterization

166 Environmental Risk: Based on the reasonably available information in the published literature,
167 provided by industries using chrysotile asbestos, and reported in EPA databases, there is minimal or no
168 releases of chrysotile asbestos to surface water associated with the COUs that EPA is evaluating in this
169 Part 1 of the risk evaluation for asbestos. Thus, EPA believes there is low or no potential for
170 environmental risk to aquatic or sediment-dwelling receptors from the COUs included in this
171 document because water releases associated with the COUs were not identified and not expected.
172 Similarly, EPA expects low or no risk to terrestrial species from water pathways, including biosolids,
173 as discussed in the problem formulation.

174 Human Health Risks: EPA identified cancer risks from inhalation exposure to chrysotile asbestos.

175 For workers and ONUs, EPA estimated cancer risk from inhalation exposures to chrysotile asbestos
176 using IUR values and exposures for each COU. EPA estimated risks using several occupational
177 exposure scenarios related to the central and high-end estimates of exposure without the use of
178 personal protective equipment (PPE), and with potential PPE for workers using chrysotile asbestos.
179 Industry submissions indicated that some workers used respirators for certain tasks, but not others, and
180 some workers used ineffective respirators (sheet gasket stampers). Sheet gasket stampers using N95
181 respirators are not protected as OSHA's Asbestos standards prohibit the use of filtering facepiece
182 respiratory for protection against asbestos fibers (OSHA asbestos standards do not specifically regulate
183 N95 respirators). Although hypothetical respirator usage with an applied protection factor (APF) of 10
184 and 25 was calculated for all COUs, actual respirator use was limited to an APF of 10 (the use of sheet
185 gaskets) and APFs of 10 and 25, in some cases, for chlor-alkali use. No other APFs were indicated for
186 any other COU. For asbestos, nominal APFs (*e.g.*, 25) may not be achieved for all PPE users. More
187 information on respiratory protection, including EPA's approach regarding the occupational exposure
188 scenarios for asbestos, is in Section 2.3.1.2.

189 For workers, cancer risks in excess of the benchmark of 1 death per 10,000 (or 1×10^{-4}) were indicated
190 for virtually all quantitatively assessed COUs (except the Super Guppy scenario) under high-end and
191 central tendency exposure scenarios when PPE was not used. Risks were below the benchmark for
192 chlor-alkali workers (full-shift, central tendency exposure estimate only) and the specialized brake pad
193 work for the NASA Super Guppy aircraft (both for central and high-end exposure estimates). With the
194 hypothetical use of PPE at APF of 10 (except for chlor-alkali processing and use [short-term⁶] and sheet
195 gasket use), most risks were reduced for central tendency estimates but still persisted for sheet gasket
196 stamping, auto brake replacement, other vehicle friction products and utility vehicle (UTV use and
197 disposal) gasket replacement for high-end exposure estimates (both 8-hour and short-term durations).
198 Although not expected to be worn given the reasonably available information, when PPE with an APF

⁵ The methodology involved in risk characterization has evolved over time and the existing EPA IURs for other asbestos fiber types [U.S. EPA \(2014b, 1986\)](#) estimated risks of cancer mortality and did not account for the risk of other cancers, and the 1986 IUR did not adjust for mesothelioma underascertainment.

⁶ Short-term means accounting for higher exposures during short periods of time during the work shift. See Section 2.3.1 for more information.

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

202 For ONUs, cancer risks in excess of the benchmark of 1 death per 10,000 (or 1×10^{-4}) were indicated for
203 both central tendency and high-end exposures for sheet gasket use (in chemical production) and UTV
204 gasket replacement. In addition, cancer risks for ONUs were indicated for high-end exposures only for
205 chlor-alkali, sheet gasket stamping. ONUs are not assumed to be using PPE to reduce exposures to
206 chrysotile asbestos used in their vicinity. EPA's estimates for ONU risks for each occupational exposure
207 scenario are presented by each COU in Section 4.2.2 and summarized in Table 4-38.

208 For consumers (Do-it-Yourselfers, or DIY) and bystanders of consumer use, EPA estimated cancer
209 risks resulting from inhalation exposures with a range of user durations, described in detail in Section
210 4.2.3. EPA assumed that consumers or bystanders would not use PPE.

211 For consumers and bystanders, cancer risks in excess of the benchmark of 1 death per 1,000,000 (or 1
212 $\times 10^{-6}$) were indicated for most COUs for consumer exposure scenarios. Risks were indicated for all
213 high-end exposures for both consumers and bystanders for brake and UTV gasket indoor scenarios;
214 and the high-end consumer outdoor scenarios (for 30-minute exposures). EPA's estimates for
215 consumer and bystander risks for each consumer use exposure scenario are presented in Section 4.2.3
216 and summarized in Table 4-48.

217 Uncertainties: Uncertainties have been identified and discussed after each section in this Part 1 of the
218 risk evaluation for asbestos. In addition, Section 4.3 summarizes the major assumptions and key
219 uncertainties by major topic: uses of asbestos, occupational exposure, consumer exposure,
220 environmental risk, IUR derivation, cancer risk value and human health risk estimates.

221 Beginning with the February, 2017 request for information on uses of asbestos ([see 2017 Public](#)
222 [Meeting](#)) and followed by the Scope document (June [2017d](#)), Problem Formulation (June [2018d](#)), and
223 draft [Risk Evaluation \(2020\)](#), EPA has refined its understanding of the current conditions of use of
224 chrysotile asbestos in the U.S. Chrysotile asbestos was the only fiber type imported, processed, or
225 distributed in commerce for use in 2019 (from the latest import records). All the raw asbestos imported
226 into the U.S. is used by the chlor-alkali industry for use in asbestos diaphragms. The remaining COUs
227 involve use and disposal of articles that contain chrysotile asbestos. EPA received voluntary
228 acknowledgement of these uses/disposals from a handful of industries that fall under these COU
229 categories.

230 By finalizing the asbestos SNUR on April 25, 2019 to include manufacturing (including import) or
231 processing of discontinued uses not already banned under TSCA, EPA is highly certain that
232 manufacturing (including import), processing, or distribution of asbestos is not intended, known or
233 reasonably foreseen for uses beyond use in the six product categories in this Part 1 of the risk
234 evaluation for asbestos. EPA will consider legacy uses and associated disposals of asbestos in Part 2 of
235 the risk evaluation for asbestos.

236 For occupational exposures, the number of chlor-alkali plants in the U.S. is known and therefore the
237 number of workers potentially exposed from chlor-alkali activities is reasonably certain. The number of
238 workers potentially exposed for other COUs is less certain. Only two workers were identified for
239 stamping sheet gaskets, and two TiO₂ manufacturing facilities were identified in the U.S. that use
240 chrysotile asbestos-containing gaskets. However, EPA is not certain if chrysotile asbestos-containing
241 sheet gaskets are used in other industries and to what extent. For the other COUs, no estimates of the

242 number of potentially exposed workers were submitted to EPA by industry or its representatives, so
243 estimates were used and were based on market estimates for that work category. However; no
244 information on the market share for asbestos containing products, with the exception of aftermarket
245 automotive brakes/linings, is reasonably available. Based on peer review and public comments received
246 on the draft Risk Evaluation, EPA adjusted its estimates for the number of potentially affected
247 individuals who may purchase and use chrysotile asbestos aftermarket automotive brakes/linings (see
248 Section 4.3.7). Therefore, numbers of workers potentially exposed were estimated and, based on the
249 COU, these estimations have a range of uncertainty from low (chlor-alkali) to high (sheet gasket use,
250 oilfield brake blocks, aftermarket automotive brakes/linings, other vehicle friction products and other
251 gaskets).

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

256 A review of reasonably available literature for consumer exposure estimates related to brake
257 repair/replacement activities by a DIY consumer was limited and no information for consumer exposure
258 estimates related to UTV exhaust system gasket repair/replacement activities was found. This absence of
259 scenario-specific exposure information required EPA to use surrogate monitoring data from
260 occupational studies to evaluate consumer risk resulting from exposure to asbestos during these two
261 activities. The surrogate occupational studies tended to be based on older studies that may or may not
262 reflect current DIY consumer activities, including best practices for removing asbestos containing
263 materials. In addition, EPA is uncertain about the number of chrysotile asbestos containing brakes that
264 are being purchased online and installed in cars (classic cars or newer cars) or gaskets that are being
265 replaced in UTVs.

266 After the PF was released, EPA continued to search EPA databases and all publicly available literature
267 and contact industries to shed light on potential releases to water from the COUs in this Part 1 of the risk
268 evaluation for asbestos for the purpose of evaluating risk to aquatic or sediment-dwelling organisms.
269 EPA found minimal or no releases of asbestos to surface water associated with the COUs in this risk
270 evaluation. In addition, there are no reported releases of asbestos to water from TRI. Despite the fact that
271 the comprehensive efforts put forth have not identified releases of chrysotile asbestos into water from
272 COUs, EPA acknowledges that uncertainty remains.

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

281 Depending on the variations in the exposure profile of the workers/occupational non-users and
282 consumers/bystanders, risks could be under- or over-estimated for all COUs. The estimates for extra
283 cancer risk were based on the EPA-derived IUR for chrysotile asbestos. The occupational exposure
284 assessment made standard assumptions of 240 days per year, 8 hours per day over 40 years starting at

285 age 16 years.⁷ This assumes the workers and ONUs are regularly exposed until age 56. If a worker
286 changes jobs during their career and is no longer exposed to chrysotile asbestos, this may overestimate
287 exposures. However, if the worker stays employed after age 56, it would underestimate exposures.

288 EPA's assessments, risk estimations, and risk determinations accounted for uncertainties throughout this
289 Part 1 of the risk evaluation for asbestos. EPA used reasonably available information, in a fit-for-
290 purpose approach, to develop a document that relies on the best available science and is based on the
291 weight of the scientific evidence. For instance, systematic review was conducted to identify reasonably
292 available information related to chrysotile asbestos hazards and exposures. The consideration of
293 uncertainties supports the Agency's risk determinations, each of which is supported by substantial
294 evidence, as set forth in detail in later sections of this Part 1 of the risk evaluation for asbestos.

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

304 EPA identified certain human subpopulations who may be more susceptible to exposure to chrysotile
305 asbestos than others. Workers exposed to chrysotile asbestos in workplace air, especially if they work
306 directly with chrysotile asbestos, are most susceptible to the health effects associated with chrysotile
307 asbestos. Although it is clear that the health risks from chrysotile asbestos exposure increases with
308 higher exposure and longer exposure time, investigators have found asbestos-related diseases in
309 individuals with only brief exposures. Generally, those who develop asbestos-related diseases could
310 show no signs of illness for decades after exposure.

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

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

322 EPA based its risk determinations on the high-end exposure estimates for workers, consumers, and
323 bystanders in order to capture individuals who may be PESS.

324 Aggregate and Sentinel Exposures: Section 6(b)(4)(F)(ii) of TSCA requires the EPA, as a part of the risk
325 evaluation, to describe whether aggregate or sentinel exposures under the conditions of use were

⁷ The Fair Labor Standards Act of 1938 allows adolescents to work an unrestricted number of hours at age 16 years.

326 considered and the basis for their consideration. The EPA has defined aggregate exposure as “*the*
327 *combined exposures to an individual from a single chemical substance across multiple routes and*
328 *across multiple pathways* (40 CFR § 702.33).” Exposures to chrysotile asbestos were evaluated by the
329 inhalation route only. Inhalation, dermal, and oral exposures could occur simultaneously for workers and
330 consumers. EPA chose not to employ simple additivity of exposure pathways within a COU since the
331 most critical exposure pathway is inhalation and the target being assessed is combined lung cancer and
332 mesothelioma. Furthermore, EPA recognizes it is possible that workers exposed to chrysotile asbestos at
333 work might also be exposed as consumers (*e.g.*, by changing asbestos-containing brakes at home) or
334 may cause unintentional exposure to individuals in their residence due to take-home exposure from
335 contaminated clothing or other items. While adding such exposures could increase risks to the worker,
336 ONU, consumer, or bystander, which already individually exceed the cancer benchmarks in virtually
337 every scenario evaluated, these additional pathways are not evaluated together because EPA did not
338 identify or receive information which could inform developing such an exposure scenario and does not
339 have models which could adequately evaluate and address such combined scenarios.

340 The EPA defines sentinel exposure as “*the exposure to a single chemical substance that represents the*
341 *plausible upper bound of exposure relative to all other exposures within a broad category of similar or*
342 *related exposures* (40 CFR § 702.33).” In this Part 1 of the risk evaluation for asbestos, the EPA
343 considered sentinel exposure the highest exposure given the details of the COU and the potential
344 exposure scenarios. EPA considered sentinel exposures by considering risks to populations who may
345 have upper bound (*e.g.*, high-end) exposures. EPA’s decisions for unreasonable risk are based on high-
346 end exposure estimates to capture individuals who may receive sentinel exposure.

347 Risk Determination

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

360 Environmental Risk: As described in the problem formulation ([U.S. EPA, 2018d](#)), EPA found that
361 exposures to terrestrial species may occur from the conditions of use due to releases to air, water or land.
362 During the course of developing the draft risk evaluation for asbestos, OPPT worked closely with the
363 offices within EPA that administer and implement regulatory programs under the Clean Air Act (CAA),
364 the Safe Drinking Water Act (SDWA), and the Clean Water Act (CWA. Through this intra-agency
365 coordination, EPA determined that exposures to terrestrial species via surface water, ambient air and
366 disposal pathways fall under the jurisdiction of other environmental statutes administered by EPA, *i.e.*,
367 CAA, SDWA, and the CWA. As explained in more detail in Section 1.4.2, EPA believes it is both
368 reasonable and prudent to tailor TSCA risk evaluations when other EPA offices have expertise and
369 experience to address specific environmental media, rather than attempt to evaluate and regulate
370 potential exposures and risks from those media under TSCA. EPA believes that coordinated action on
371 exposure pathways and risks addressed by other EPA-administered statutes and regulatory programs is

372 consistent with statutory text and legislative history, particularly as they pertain to TSCA’s function as a
373 “gap-filling” statute, and also furthers EPA aims to efficiently use Agency resources, avoid duplicating
374 efforts taken pursuant to other Agency programs, and meet the statutory deadline for completing risk
375 evaluations. EPA has therefore tailored the scope of this Part 1 of the risk evaluation for asbestos using
376 authorities in TSCA sections 6(b) and 9(b)(1). EPA did not evaluate hazards or exposures from
377 chrysotile asbestos releases to terrestrial pathways for terrestrial organisms, and as such the
378 unreasonable risk determinations for relevant conditions of use do not account for exposures to
379 terrestrial organisms.

380 After the PF was released, EPA continued to search EPA databases as well as the literature and
381 contacted industries to shed light on potential releases of chrysotile asbestos to water from the TSCA
382 COUs. Based on the reasonably available information in the published literature, provided by industries
383 using asbestos, and reported in EPA databases, there is minimal or no releases of chrysotile asbestos to
384 surface water associated with the COUs in this Part 1 of the risk evaluation for asbestos. EPA has
385 considered peer review and public comments on this conclusion and EPA is confident that the minimal
386 water release data available cannot be attributed to chrysotile asbestos from the COUs in this document.
387 Therefore, EPA concludes there is no unreasonable risk to aquatic organisms (including sediment-
388 dwelling organisms) from the COUs in this Part 1 of the risk evaluation for asbestos. Details are
389 provided in Section 4.1.

390 Risk of Injury to Health: EPA’s determination of unreasonable risk for specific COUs of chrysotile
391 asbestos listed below are based on health risks to workers, occupational non-users, consumers, or
392 bystanders from consumer use. The health effect driver for EPA’s determination of unreasonable risk is
393 cancer from inhalation exposure. As described below, risks to the general population were not evaluated
394 for these conditions of use.

395 There are physical-chemical characteristics that are unique to asbestos, such as insolubility in water,
396 opportunity for suspension and extended duration in air, transportability and the friable nature of
397 asbestos-containing products. These attributes allow asbestos fibers to be released, settled, and to again
398 become airborne (“re-entrainment”) under certain conditions of use. Also unique to asbestos is the
399 impact of the timing of exposure relative to the cancer outcome; the most relevant exposures for
400 understanding cancer risk were those that occurred decades prior to the onset of cancer. In addition to
401 the cancer benchmark, the physical-chemical properties and exposure considerations are important
402 factors in considering risk of injury to health. To account for the exposures for ONUs and, in certain
403 cases bystanders, EPA derived a distribution of exposure values for calculating the risk for cancer by
404 using area monitoring data (*i.e.*, fixed location air monitoring results) where available for certain
405 conditions of use and when appropriate applied exposure reduction factors, using data from published
406 literature (see Sections 2.3.1 and 2.3.2 for details on ONU and bystander methods, respectively). The
407 risk determination for each COU in this Part 1 of the risk evaluation for asbestos considers both central
408 tendency and high-end risk estimates for workers, ONUs, consumers and bystanders. Where relevant
409 EPA considered PPE for workers. For many of the COUs both the central tendency and high-end risk
410 estimates exceed the risk benchmark for each of the exposed populations evaluated. However, the risk
411 benchmarks do not serve as a bright line for making risk determinations and other relevant risk-related
412 factors were considered. EPA focused on the high-end risk estimates rather than central tendency risk
413 estimates to be protective of workers, ONUs, consumers, and bystanders. Additionally, EPA’s
414 confidence in the data used in the risk estimate is considered.

415 Risk to the General Population: As part of the problem formulation for asbestos, EPA found that
416 exposures to the general population may occur from the conditions of use due to releases to air, water or
417 land. During the course of developing the draft risk evaluation for asbestos, OPPT worked closely with

418 the offices within EPA that administer and implement regulatory programs under the Clean Air Act
419 (CAA), the Safe Drinking Water Act (SDWA), and the Clean Water Act (CWA). Through this intra-
420 agency coordination, EPA determined that exposures to the general population via surface water,
421 drinking water, ambient air and disposal pathways falls under the jurisdiction of other environmental
422 statutes administered by EPA, (*i.e.*, CAA, SDWA, and the CWA). As explained in more detail in
423 Section 1.4.2, EPA believes it is both reasonable and prudent to tailor TSCA risk evaluations when other
424 EPA offices have expertise and experience to address specific environmental media, rather than attempt
425 to evaluate and regulate potential exposures and risks from those media under TSCA. EPA believes that
426 coordinated action on exposure pathways and risks addressed by other EPA-administered statutes and
427 regulatory programs is consistent with statutory text and legislative history, particularly as they pertain
428 to TSCA’s function as a “gap-filling” statute, and also furthers EPA aims to efficiently use Agency
429 resources, avoid duplicating efforts taken pursuant to other Agency programs, and meet the statutory
430 deadline for completing risk evaluations. EPA has therefore tailored the scope of this Part 1 of the risk
431 evaluation for asbestos using authorities in TSCA sections 6(b) and 9(b)(1). Therefore, EPA did not
432 evaluate hazards or exposures to the general population in this document, and as such the unreasonable
433 risk determinations for the relevant conditions of use do not account for exposures to the general
434 population.

435 Risk to Workers: The conditions of use of asbestos that present an unreasonable risk to workers include
436 processing and industrial use of chrysotile asbestos-containing diaphragms, processing and industrial use
437 of chrysotile asbestos-containing sheet gaskets and industrial use of chrysotile asbestos-containing brake
438 blocks, aftermarket automotive chrysotile asbestos-containing brakes/linings, other vehicle friction
439 products, and other chrysotile asbestos-containing gaskets. A full description of EPA’s determination for
440 each condition of use is in Section 5.2.

441 Risk to Occupational Non-Users (ONUs): EPA determined that the conditions of use that present
442 unreasonable risks for ONUs include processing and industrial use of chrysotile asbestos-containing
443 diaphragms, processing and industrial use of chrysotile asbestos-containing sheet gaskets and industrial
444 use of chrysotile asbestos-containing brake blocks, other vehicle friction products, and other chrysotile
445 asbestos-containing gaskets. A full description of EPA’s determination for each condition of use is in
446 Section 5.2.

447 EPA generally assumes compliance with OSHA requirements for protection of workers, including the
448 implementation of the hierarchy of controls. In support of this assumption, EPA used reasonably
449 available information indicating that some employers, particularly in the industrial setting, are providing
450 appropriate engineering, or administrative controls, or PPE to their employees consistent with OSHA
451 requirements. While EPA does not have reasonably available information to either support or contradict
452 this assumption for each condition of use, EPA does not believe that the Agency must presume, in the
453 absence of such information, a lack of compliance with existing regulatory programs and practices.
454 Rather, EPA assumes there is compliance with worker protection standards unless case-specific facts
455 indicate otherwise, and therefore existing OSHA regulations for worker protection and hazard
456 communication will result in use of appropriate PPE in a manner that achieves the stated APF or PF.
457 EPA’s decisions for unreasonable risk to workers are based on high-end exposure estimates, in order to
458 account for the uncertainties related to whether or not workers are using PPE. EPA believes this is a
459 reasonable and appropriate approach that accounts for reasonably available information and professional
460 judgement related to worker protection practices, and addresses uncertainties regarding availability and
461 use of PPE.

462 Risk to Consumers: For consumers, EPA determined that the conditions of use that present an
463 unreasonable risk are use of aftermarket automotive chrysotile asbestos-containing brakes/linings and

464 other chrysotile asbestos-containing gaskets. A full description of EPA’s determination for each
465 condition of use is in Section 5.2.

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

470 Summary of Risk Determinations for the Risk Evaluation for Asbestos Part 1: Chrysotile Asbestos: In
471 conducting risk evaluations, “EPA will determine whether the chemical substance presents an
472 unreasonable risk of injury to health or the environment under each condition of use [] within the scope
473 of the risk evaluation, either in a single decision document or in multiple decision documents...” 40
474 CFR 702.47. Under EPA’s implementing regulations, “[a] determination by EPA that the chemical
475 substance, under one or more of the conditions of use within the scope of the risk evaluation, does not
476 present an unreasonable risk of injury to health or the environment will be issued by order and
477 considered to be a final Agency action, effective on the date of issuance of the order.” 40 CFR
478 702.49(d).

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

481 EPA has determined that the following conditions of use of chrysotile asbestos present an unreasonable
482 risk of injury to health to workers (including, in some cases, occupational non-users) or to consumers
483 (including, in some cases, bystanders).

484 Pursuant to TSCA section 6(i)(2), the unreasonable risk determinations for these conditions of use are
485 not considered final agency action. EPA will initiate TSCA section 6(a) risk management actions on
486 these conditions of use as required under TSCA section 6(c)(1).⁸ The details of these determinations are
487 presented in Section 5.2.

Occupational Conditions of Use that Present an Unreasonable Risk
<ul style="list-style-type: none">• Processing and Industrial use of Chrysotile Asbestos Diaphragms in the Chlor-alkali Industry• Processing and Industrial Use of Chrysotile Asbestos-Containing Sheet Gaskets in Chemical Production• Industrial Use and Disposal of Chrysotile Asbestos-Containing Brake Blocks in Oil Industry• Commercial Use and Disposal of Aftermarket Automotive Chrysotile Asbestos-Containing Brakes/Linings• Commercial Use and Disposal of Other Chrysotile Asbestos-Containing Vehicle Friction Products• Commercial Use and Disposal of Other Asbestos-Containing Gaskets

488

⁸ 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.

Consumer Uses and Disposal that Present an Unreasonable Risk
<ul style="list-style-type: none">• Aftermarket Automotive Chrysotile Asbestos-Containing Brakes/Linings• Other Chrysotile Asbestos-Containing Gaskets

489 EPA has determined that the following conditions of use of chrysotile asbestos do not present an
490 unreasonable risk of injury to health. These determinations are considered final agency action and are
491 being issued by order pursuant to TSCA section 6(i)(1), and the TSCA section 6(i)(1) order is contained
492 in Section 5.3.1 of Part 1 of the risk evaluation for asbestos. The details of these determinations are
493 presented in Section 5.2.
494

Conditions of Use that Do Not Present an Unreasonable Risk
<ul style="list-style-type: none">• Import of chrysotile asbestos and chrysotile asbestos-containing products• Distribution of chrysotile asbestos-containing products• Use of chrysotile asbestos-containing brakes for a specialized, large NASA transport plane• Disposal of chrysotile asbestos-containing sheet gaskets processed and/or used in the industrial setting and asbestos-containing brakes for a specialized, large NASA transport plane

495

1 INTRODUCTION

497 This document presents Part 1 (chrysotile asbestos) of the risk evaluation of asbestos⁹ under the Frank
498 R. Lautenberg Chemical Safety for the 21st Century Act which amended the Toxic Substances Control
499 Act (TSCA), the Nation’s primary chemicals management law, in June 2016.

500 The Agency published the *Scope of the Risk Evaluation for Asbestos* ([U.S. EPA, 2017d](#)) in June 2017,
501 and the *Problem Formulation* in June 2018 [U.S. EPA \(2018d\)](#), which represented the analytical phase of
502 risk evaluation in which “the purpose for the assessment is articulated, the problem is defined, and a
503 plan for analyzing and characterizing risk is determined” as described in Section 2.2 of the [Framework
504 for Human Health Risk Assessment to Inform Decision Making](#). EPA received comments on the
505 published Problem Formulation for asbestos and has considered the comments specific to asbestos, as
506 well as more general comments regarding EPA’s Risk Evaluation approach for developing the Risk
507 Evaluations for the first 10 chemicals EPA is evaluating; including this Part 1 of the risk evaluation for
508 asbestos.

509 In the [problem formulation U.S. EPA \(2018d\)](#), EPA identified the conditions of use (COUs) and
510 presented three conceptual models and an analysis plan. Based on EPA’s analysis of the COUs,
511 physical-chemical and fate properties, environmental releases and exposure pathways, the problem
512 formulation preliminarily concluded that further analysis was necessary for exposure pathways to
513 workers (including ONUs), consumers (including bystanders), and surface water, based on a qualitative
514 assessment of the physical-chemical properties and fate of asbestos in the environment. After the
515 problem formulation was released, there were two major developments that warranted changes/updates
516 prior to release of the draft Risk Evaluation. First, EPA continued to search EPA databases as well as the
517 literature and either engaged in a dialogue with industries or reached out for a dialogue to shed light on
518 potential releases to water for chrysotile asbestos. It was concluded there were no water releases for the
519 COUs associated with chrysotile asbestos based on the collected information. Second, a new COU was
520 discovered within the vehicle friction products category (*i.e.*, the use of brakes/friction products in a
521 large aircraft operated by NASA). Both of these were included in the subsequently published draft Risk
522 Evaluation for asbestos for which EPA has taken public and peer review comments.

523 As per EPA’s final rule, [Procedures for Chemical Risk Evaluation Under the Amended Toxic
524 Substances Control Act](#) (82 Fed. Reg. 33726 (July 20, 2017)), the draft Risk Evaluation for asbestos was
525 subject to both public comment and peer review; which are distinct but related processes. EPA provided
526 60 days for public comment on any and all aspects of the draft Risk Evaluation, including the
527 submission of any additional information that might be relevant to the science underlying the document
528 and the outcome of the systematic review associated with chrysotile asbestos. This satisfies TSCA (15
529 U.S.C. 2605(b)(4)(H)), which requires EPA to provide public notice and an opportunity for comment on
530 a draft Risk Evaluation prior to publishing a final Risk Evaluation.

531 Peer review was conducted in accordance with EPA’s regulatory procedures for chemical Risk
532 Evaluations, including using the EPA Peer Review Handbook ([U.S. EPA, 2015b](#)) and other methods
533 consistent with the science standards laid out in Section 26 of TSCA (*See* 40 CFR 702.45). As explained
534 in the Risk Evaluation Rule ([U.S. EPA, 2017c](#)), the purpose of peer review is for the independent review
535 of the science underlying the risk assessment. Peer review addressed aspects of the underlying science as

⁹ As noted in the **PREAMBLE**, this is Part 1 of the final Risk Evaluation for asbestos. Part 1 includes the imported, processed, and distributed uses of chrysotile asbestos in the United States. Part 2 will be on legacy uses and associated disposals of asbestos. Please see Figure P-2 for definitions and terms used throughout this document and note that occasionally the term “asbestos” is used (depending on context), but the focus of this Part 1 document is chrysotile asbestos.

536 outlined in the charge to the peer review panel such as hazard assessment, assessment of dose-response,
537 exposure assessment, and risk characterization.

538 As explained in the [Risk Evaluation Rule](#) (82 Fed. Reg. 33726 (July 20, 2017)), it is important for peer
539 reviewers to consider how the underlying risk evaluation analyses fit together to produce an integrated
540 risk characterization, which forms the basis of an unreasonable risk determination. EPA believed peer
541 reviewers were most effective in this role if they received the benefit of public comments on draft risk
542 evaluations prior to peer review. For this reason, and consistent with standard Agency practice, the
543 public comment period preceded peer review. In response to public comments received on the draft Risk
544 Evaluation and/or in response to peer review, the overall approach to finalizing the Risk Evaluation for
545 asbestos changed as described in the Preamble by dividing the Risk Evaluation into two parts.
546 Furthermore, EPA responded to public and [peer review comments](#) received on the draft Risk Evaluation
547 in the response to comments document and, where appropriate, made revisions in response to those
548 comments in Part 1 of the risk evaluation.

549 The conclusions, findings, and determinations in Part 1 of the risk evaluation are for the purpose of
550 identifying whether exposure to chrysotile asbestos presents unreasonable risk or no unreasonable risk
551 under the conditions of use, in accordance with TSCA Section 6, and are not intended to represent any
552 findings under TSCA section 7.

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

559 The COUs in this Part 1 of the risk evaluation for asbestos are limited to only a few categories of
560 ongoing and prospective uses, and chrysotile is the only type of asbestos fiber identified for these COUs.
561 Ongoing uses of asbestos in the U.S. were difficult to identify despite using an extensive list of
562 resources. To determine the COUs of asbestos and inversely, activities that do not qualify as COUs,
563 EPA conducted extensive research and outreach. EPA identified activities that include import of raw
564 chrysotile asbestos, used solely in the chlor-alkali industry, and import and use of chrysotile asbestos-
565 containing products. The COUs included in this Part 1 of the risk evaluation for asbestos that EPA
566 considers to be known, intended, or reasonably foreseen are the import, use, distribution and disposal of
567 chrysotile asbestos diaphragms, sheet gaskets, other gaskets, oilfield brake blocks, aftermarket
568 automotive brakes/linings, and other vehicle friction products and the processing, distribution and
569 disposal of chrysotile asbestos diaphragms and sheet gaskets. Some of these COUs are very specialized.
570 Since the Problem Formulation, several conditions of use were removed from the scope of the draft Risk
571 Evaluation based on further investigation (see Section 1.4.4); these COUs pertain to woven products,
572 cement products, and packings (from “gaskets and packings”). EPA determined that there is no evidence
573 to indicate manufacture (including import), processing, or distribution of asbestos-containing woven
574 products, cement products, or packings. These conditions of use were added to the Significant New Use
575 Rule (SNUR) for asbestos (40 CFR 721.11095). The Asbestos SNUR is an Agency action
576 complementary to the Risk Evaluation for asbestos and taken under TSCA section 5 to prohibit any
577 manufacturing (including import) or processing for discontinued uses of asbestos from restarting without
578 EPA having an opportunity to evaluate them to determine risks to health or the environment and take
579 any necessary regulatory action, which may include a prohibition. The final asbestos SNUR ensures that
580 any manufacturing (including import) and processing for all discontinued uses and types of asbestos that
581 are not already banned are restricted from re-entering the U.S. marketplace without notification to EPA

582 and review and any necessary regulatory action by the Agency. Thus, should any person wish to
583 manufacture, import, or process asbestos for an activity that is not a COU identified in this document or
584 subject to an existing ban, then EPA would review the risk of the activity associated with such a use in
585 accordance with TSCA section 5¹⁰.

586 During the investigation of the COUs, EPA also determined that asbestos is no longer mined in the U.S.,
587 and that only chrysotile asbestos is being imported. The other five forms of asbestos identified for the
588 Risk Evaluation, including crocidolite (riebeckite), amosite (cummingtonite-grunerite), anthophyllite,
589 tremolite or actinolite, are no longer manufactured, imported, or processed in the United States and are
590 also now subject to the SNUR. After EPA confirmed that chrysotile asbestos is the only type of asbestos
591 still being imported into the U.S. either in raw form or in products, EPA developed a chrysotile IUR¹¹ to
592 be used in Part 1 of the risk evaluation. The IUR for asbestos developed in 1988 was based on 14
593 epidemiologic studies that included occupational exposure to chrysotile, amosite, or mixed-mineral
594 exposures (chrysotile, amosite, crocidolite). As a naturally occurring mineral, chrysotile asbestos can co-
595 occur with other minerals, including amphibole forms of asbestos. Trace amounts of these minerals may
596 remain in chrysotile asbestos as it is used in commerce. The epidemiologic studies that are reasonably
597 available include populations exposed to chrysotile asbestos, which may contain small, but variable
598 amounts of amphibole asbestos. Because the only form of asbestos imported, processed, or distributed
599 for use in the United States today is chrysotile asbestos, studies of populations exposed only to
600 chrysotile asbestos provide the most informative data for the purpose of updating the TSCA risk
601 estimates for the COUs for chrysotile asbestos in this document. EPA will consider legacy uses and
602 associated disposals of asbestos in Part 2 of the risk evaluation for asbestos (as noted in the Preamble).

603 EPA stated in the Problem Formulation that the asbestos Risk Evaluation would focus on
604 epidemiological data on lung cancer and mesothelioma. The 1988 IUR identified asbestos as a
605 carcinogen causing both lung cancer and mesothelioma from inhalation exposures and derived a unit
606 risk to address both cancers and cover all asbestos fiber types (for all TSCA Title II fiber types – see
607 Section 1.1). Over 24,000 studies were initially identified for consideration during the Systematic
608 Review process to determine whether the existing IRIS 1988 IUR was appropriate for TSCA purposes.
609 Once EPA determined that only conditions of use of chrysotile asbestos were going to be evaluated for
610 the draft risk evaluation, the focus turned to whether a chrysotile-specific IUR could be derived. EPA is
611 not aware of any other chrysotile asbestos-specific IUR or any other risk-based values having been
612 estimated for other types of cancer for asbestos fiber types by either EPA or other government agencies.
613 For the derivation of a chrysotile asbestos IUR, epidemiological studies on mesothelioma and lung
614 cancer in cohorts of workers using chrysotile asbestos in commerce were identified to inform the
615 estimation of an exposure-response function.

616 Related to the focus on chrysotile asbestos is the method of identifying asbestos in studies used to
617 develop the IUR. The IUR is based on fiber counts made by phase contrast microscopy (PCM) and
618 should not be applied directly to measurements made by other analytical techniques. PCM
619 measurements made in occupational environments were used in the studies that support the derivation of
620 the chrysotile asbestos IUR. PCM detects only fibers longer than 5 µm and >0.4 µm in diameter, while
621 transmission electron microscopy (TEM), often found in environmental monitoring measurements, can
622 detect much smaller fibers. In developing a PCM-based IUR in this Part 1 of the risk evaluation for

¹⁰ As of December 2020, EPA has not received any SNUNs for asbestos.

¹¹ Inhalation Unit risk (IUR) is typically defined as a plausible upper bound on the estimate of cancer risk per µg/m³ 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).

623 asbestos, several TEM papers modeling risk of lung cancer were found, but because there was no TEM-
624 based modeling of mesothelioma risk, TEM data could not be used to derive a TEM-based IUR.

625 EPA derived an IUR for chrysotile asbestos using five epidemiological study cohorts analyzing lung
626 cancer and mesothelioma. EPA derived cancer-specific unit risks using lifetables. Different modeling
627 choices and combinations of cancer-specific unit risks yielded candidate IUR values ranging from 0.15
628 to 0.45 per f/cc, indicating low model-based uncertainty. The IUR chosen is 0.16 per f/cc and it was
629 applied to the COUs to calculate lifetime risks for workers and consumers.

630 EPA estimated risks for workers, occupational non-users (ONUs), consumers (do-it-yourself [DIY]
631 mechanics) and bystanders for the COUs identified. Inhalation exposure scenarios were used to estimate
632 risks for cancer based on the EPA-derived IUR for chrysotile asbestos. This assessment is unique with
633 respect to the timing of exposure relative to the cancer outcome as the time since first exposure plays a
634 dominant role in modeling risk. Occupational exposures assumed 240 days/year for 8-hour workdays for
635 40 years starting at 16 years old; with other starting ages and exposure durations also presented.
636 Occupational exposures for chlor-alkali and sheet gasket workers and ONUs were based on monitoring
637 data supplied by companies performing the work. Consumer exposures were based on study data
638 provided in the literature for gasket replacement and brakes. Consumer exposures assumed that DIY
639 mechanics for both COUs changed brakes or gaskets once every three years (the task taking three hours)
640 over a lifetime and that exposures lingered between the episodic exposures.

641 Section 1 presents the basic physical-chemical characteristics of chrysotile asbestos, as well as a
642 background on regulatory history, COUs, and conceptual models, with particular emphasis on any
643 changes since the publication of the [draft Risk Evaluation](#). Section 1 also includes a discussion of the
644 systematic review process utilized in this document. Section 2 provides a discussion and analysis of the
645 exposures, both health and environmental, that can be expected based on the COUs for chrysotile
646 asbestos. Section 3 discusses the environmental and health hazards of chrysotile asbestos. Section 4
647 presents the risk characterization, where EPA integrates and assesses reasonably available information
648 on health and environmental hazards and exposures, as required by TSCA (15 U.S.C. 2605(b)(4)(F)).
649 This section also includes a discussion of any uncertainties and how they impact this document. Section
650 5 presents the risk determination of whether risks posed by the chemical substance under the COUs are
651 “unreasonable” as required under TSCA (15 U.S.C. 2605(b)(4)).

652 **1.1 Physical and Chemical Properties and Environmental Fate**

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

659 TSCA Title II (added to TSCA in 1986), Section 202 defines asbestos as the “asbestiform varieties of
660 six fiber types – chrysotile (serpentine), crocidolite (riebeckite), amosite (cummingtonite-grunerite),
661 anthophyllite, tremolite or actinolite.” The latter five fiber types are amphibole varieties. In the *Problem*
662 *Formulation of the Risk Evaluation for Asbestos* (EPA-HQ-OPPT-2016-0736-0131) ([U.S. EPA, 2018d](#)),
663 physical and chemical properties of all six fiber types were presented. As discussed in more detail in
664 Section 1.4, the risk evaluation for asbestos Part 1 has focused on chrysotile asbestos given EPA’s

665 knowledge of the COUs of chrysotile asbestos, and EPA will consider legacy uses and associated
 666 disposals of asbestos in Part 2 of the final Risk Evaluation for asbestos (see Preamble).

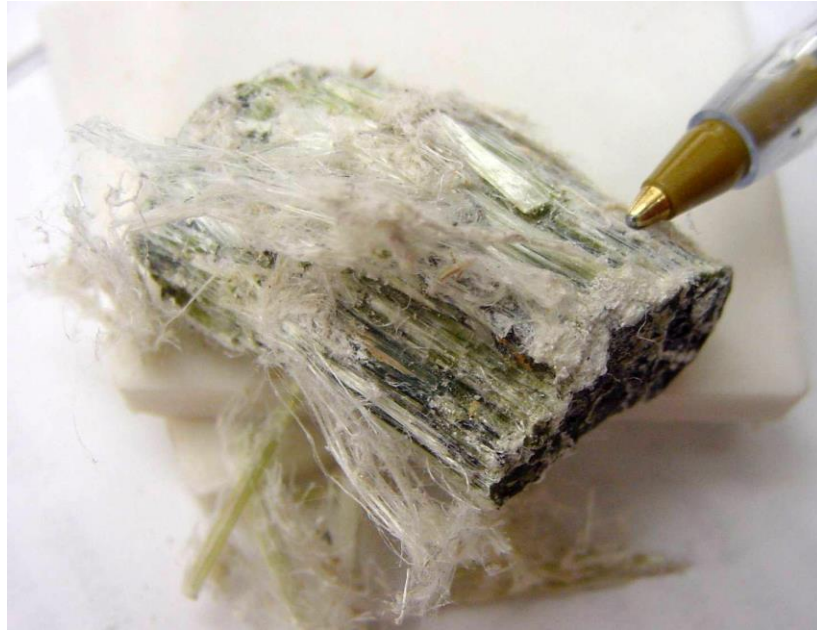
667 Table 1-1. shows the physical and chemical properties for chrysotile asbestos, a hydrated magnesium
 668 silicate mineral, with relatively long and flexible crystalline fibers that are capable of being woven.
 669 Chrysotile asbestos fibers used in most commercial applications consist of aggregates and usually
 670 contain a broad distribution of fiber lengths. Chrysotile asbestos fiber bundle lengths usually range from
 671 a fraction of a millimeter to several centimeters, and diameters range from 0.1 to 100 μm ([Virta, 2002](#)).
 672 Chrysotile asbestos fibers have a net positive surface charge and form a stable suspension in water.
 673
 674

Table 1-1. Physical and Chemical Properties of Chrysotile Asbestos Fibers^a

Property	Description
Essential composition	Mg silicate with some water
Color	White, gray, green, yellowish
Surface area ^b . (m^2/g)	13.5-22.4
Hardness (Mohs)	2.5-4.0
Specific gravity	2.4-2.6
Flexibility	High
Texture	Silky, soft to harsh
Spinnability	Very good
Fiber size, median true diameter (μm) ^c	0.06 ^e
Fiber size, median true length (μm) ^d	0.55 ^e
Resistance to Acids	Weak, undergoes fairly rapid attack
Resistance to Bases	Very good
Zeta potential (mV) ^d	+13.6 to +54
Decomposition temperature ($^{\circ}\text{C}$)	600-850
^a Badollet (1951) ^b Addison et al. (1966) ^c Hwang (1983) ^d Virta (2011) ^e 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.”	

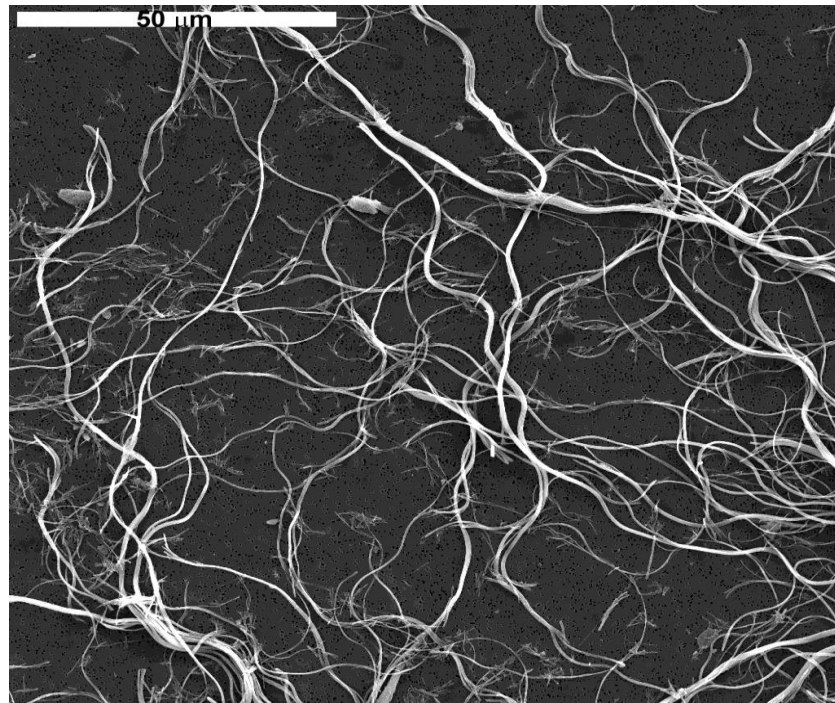
675

676 Figure 1-1 shows two pictures of chrysotile asbestos; one at the “macro” level and one at the
677 microscopic level.
678
679



A

680
681
682



B

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685
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688

Figure 1-1. Chrysotile Asbestos.
Both photographs are from the USGS. [A \(top\)](#) and [B \(bottom\)](#)

1.2 Uses and Production Volume

690 The only form of asbestos manufactured (including imported), processed, or distributed for use in the
691 United States today is chrysotile asbestos. The United States Geological Survey (USGS) estimated that
692 100 metric tons of raw chrysotile asbestos were imported into the U.S. in 2019 ([USGS, 2020](#)). This raw
693 asbestos is used exclusively by the chlor-alkali industry and imported amounts tend to range between
694 100 and 800 metric tons during a given year.

695 In addition to the use of raw imported chrysotile asbestos by the chlor-alkali industry, EPA is also aware
696 of imported asbestos-containing products; however, the import volumes of those products are not fully
697 known. The asbestos-containing products that EPA has identified as being imported and used are sheet
698 gaskets, brake blocks, aftermarket automotive brakes/linings, other vehicle friction products, and other
699 gaskets. More information about the uses of chrysotile asbestos and EPA's methodology for identifying
700 COUs is provided in Section 1.4.1 of this document. EPA will consider legacy uses and other types of
701 asbestos fibers in Part 2 of the risk evaluation of asbestos (see Preamble).

702 1.3 Regulatory and Assessment History

703 EPA conducted a search of existing domestic and international laws, regulations and assessments
704 pertaining to asbestos. EPA compiled this summary from data available from federal, state, international
705 and other government sources, as cited in Appendix A (Regulatory History). EPA evaluated and
706 considered the impact of at least some of these existing laws and regulations to determine what, if any
707 further analysis might be necessary as part of the risk evaluation for asbestos. Consideration of the nexus
708 between these regulations and the TSCA COUs evaluated in Part 1 of the risk evaluation for asbestos
709 were developed and described in the Problem Formulation document and are further described in
710 Section 1.4.2.

711 *Federal Laws and Regulations*

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

715 *State Laws and Regulations*

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

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

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

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

Table 1-2. Assessment History of Asbestos

Authoring Organization	Assessment
EPA assessments	
EPA, Integrated Risk Information System (IRIS)	IRIS Assessment on Asbestos (1988b)
EPA, Integrated Risk Information System (IRIS)	IRIS Assessment on Libby Amphibole Asbestos (2014c)
EPA, Region 8	Site-Wide Baseline Ecological Risk Assessment, Libby Asbestos Superfund Site, Libby Montana U.S. EPA (2014b)
EPA, Drinking Water Criteria Document	U.S. EPA Drinking Water Criteria Document for Asbestos (1985)
EPA, Ambient Water Quality Criteria for Asbestos	Asbestos: Ambient Water Quality Criteria (1980)
EPA, Final Rule (40 CFR Part 763)	Asbestos; Manufacture, Importation, Processing and Distribution in Commerce Prohibitions (1989)
EPA, Asbestos Modeling Study	Final Report; Asbestos Modeling Study U.S. EPA (1988a)
EPA, Asbestos Exposure Assessment	Revised Report to support ABPO rule (1988)
EPA, Nonoccupational Exposure Report	Revised Draft Report, Nonoccupational Asbestos Exposure Versar (1987)
EPA, Airborne Asbestos Health Assessment Update	Support document for NESHAP review (1986)
Other U.S.-based organizations	
National Institute for Occupational Safety and Health (NIOSH)	Asbestos Fibers and Other Elongate Mineral Particles: State of the Science and Roadmap for Research (2011b)
Agency for Toxic Substances and Disease Registry (ATSDR)	Toxicological Profile for Asbestos (2001a)
National Toxicology Program (NTP)	Report on Carcinogens, Fourteenth Edition (2016)
CA Office of Environmental Health Hazard Assessment (OEHHA), Pesticide and Environmental Toxicology Section	Public Health Goal for Asbestos in Drinking Water (2003)
International	
International Agency for Research on Cancer (IARC)	IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Arsenic, Metals, Fibres, and Dusts. Asbestos (Chrysotile, Amosite, Crocidolite, Tremolite, Actinolite, and Anthophyllite) (2012b)
World Health Organization (WHO)	World Health Organization (WHO) Chrysotile Asbestos (2014)

Authoring Organization	Assessment
Environment and Climate Change Canada	Prohibition of Asbestos and Products Containing Asbestos Regulations https://www.canada.ca/content/dam/eccc/documents/pdf/pollution-waste/asbestos-amiante/general%20factsheet%20_EN.pdf

726

727

1.4 Scope of the Evaluation

728

1.4.1 Refinement of Asbestos Fiber Type Considered in the Risk Evaluation for Asbestos Part 1: Chrysotile Asbestos

729

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

742

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

749

750 Chrysotile asbestos is the prevailing form of asbestos currently mined worldwide, and therefore;
 751 commercially available products fabricated overseas are made with chrysotile asbestos. Any asbestos
 752 being imported into the U.S. in articles for the COUs EPA has identified is believed to be chrysotile
 753 asbestos. Based on EPA’s determination that chrysotile asbestos is the only form of asbestos imported
 754 into the U.S. as both raw form and as contained in articles, EPA is performing a quantitative evaluation
 755 for chrysotile asbestos in this Part 1 of the risk evaluation for asbestos. EPA will consider legacy uses
 756 and associated disposals of asbestos in Part 2. Together, Parts 1 and 2 will constitute the final Risk
 757 Evaluation for asbestos.

758

1.4.2 Conditions of Use Included in the Risk Evaluation for Asbestos Part 1: Chrysotile Asbestos

759

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

764 To determine the COUs of asbestos and inversely, activities that do not qualify as a COU, EPA
 765 conducted extensive research and outreach. This included EPA’s review of published literature and
 766 online databases including the most recent data available from EPA’s Chemical Data Reporting (CDR)
 767 and Toxics Release Inventory (TRI) programs, Safety Data Sheets (SDSs), the U.S. Geological Survey’s
 768 Mineral Commodities Summary and Minerals Yearbook, the U.S. International Trade Commission’s
 769 DataWeb and government and commercial trade databases. EPA also reviewed company websites of
 770 potential manufacturers, importers, distributors, retailers, or other users of asbestos. EPA also received
 771 comments on the *Scope of the Risk Evaluation for Asbestos* (EPA-HQ-OPPT-2016-0736-0086, [\(2017c\)](#))
 772 that were used to inform the COUs. In addition, prior to the June 2017 publication of the scope
 773 document, EPA convened meetings with companies, industry groups, chemical users, and other
 774 stakeholders to aid in identifying COUs, and verifying COUs identified by EPA.

775 EPA has removed from this Part 1 of the risk evaluation for asbestos any activities that EPA has
 776 concluded do not constitute COUs – for example, because EPA has insufficient information to find
 777 certain activities are circumstances under which the chemical is actually “intended, known, or
 778 reasonably foreseen to be manufactured, processed, distributed in commerce, used or disposed of.”

779 Since the PF document was published in June 2018 ([U.S. EPA, 2018d](#)), EPA has further refined the
 780 COU of asbestos as described in the [draft Risk Evaluation](#). In that document, EPA determined that
 781 packings, woven products, and cement products are not current COUs. Asbestos “packings” are listed
 782 under a broader category of “gaskets, packings, and seals” and more detailed data revealed that only
 783 imported gaskets, not packings, contain asbestos. EPA concluded that “woven and knitted fabrics,”
 784 which are reported in USGS’s 2016 Minerals Yearbook under Harmonized Tariff Schedule (HTS) code
 785 6812.99.0004 are misreported (see Appendix C for further explanation). Upon further review, EPA
 786 determined that woven products are not a COU but are precursors to asbestos-containing products or
 787 physical attributes of the asbestos. EPA contacted potential foreign exporters of asbestos woven
 788 products and asbestos cement products, and these foreign companies informed EPA that they do not
 789 have customers in the United States ([2018b, c](#)). The Agency has not found any evidence to suggest that
 790 woven products (other than those that are already covered under a distinct COU such as brake blocks
 791 used in draw works) or cement products imported into the United States contain asbestos. Furthermore,
 792 EPA discussed the use of asbestos in cement pipe with a trade organization, who indicated that domestic
 793 production, importation, or distribution for such a use is neither known to be currently ongoing nor
 794 foreseeable ([AWWA, 2019](#)). Based on outreach activity and lack of evidence, EPA does not believe
 795 asbestos packings, asbestos woven products (that are not already covered under a separate and ongoing
 796 COU), or asbestos cement products are manufactured (including imported), processed, or distributed in
 797 the United States, and therefore, packings, woven products, and cement products are no longer under
 798 consideration for this Part 1 of the risk evaluation on asbestos which is focused on chrysotile asbestos
 799 and are now subject to the asbestos SNUR under TSCA section 5. Table 1-3. represents the activities
 800 that have been removed from the scope of this risk evaluation for chrysotile asbestos (Part 1 of the risk
 801 evaluation for asbestos). EPA will consider legacy uses and other asbestos fiber types in Part 2 of the
 802 risk evaluation for asbestos.

803 **Table 1-3. Categories Determined Not to be Manufactured (Including Imported), Processed, or**
 804 **Distributed for the Risk Evaluation for Asbestos Part 1: Chrysotile Asbestos**

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

805

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

813 Based on the above discussion, the COUs that are included in this Part 1 of the risk evaluation for
814 asbestos are described in Table 1-4.

815 The life cycle diagram is presented in Figure 1-2. Chrysotile Asbestos.

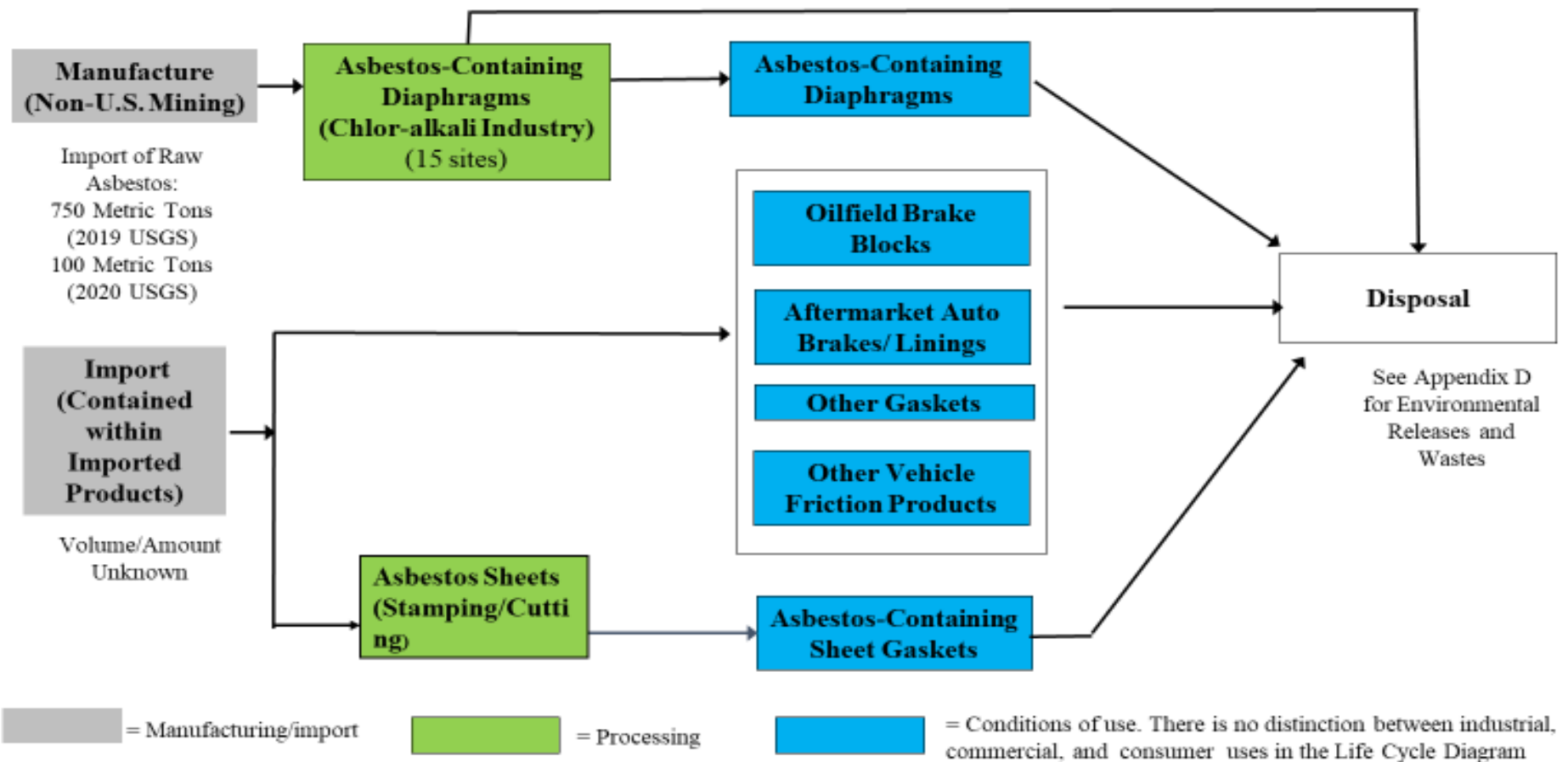
816

817 **Table 1-4. Categories of Conditions of Use Included in this Risk Evaluation for Asbestos Part 1:**
818 **Chrysotile Asbestos**

Product Category	Example
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

819

MANUFACTURE/IMPORT PROCESSING INDUSTRIAL, COMMERCIAL, CONSUMER USES RELEASES/WASTE DISPOSAL



820

821 **Figure 1-2. Chrysotile Asbestos Life Cycle Diagram**

822 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
 823 of woven products, cement products, and packing (see Section 1.4.3) as well as using the 2019 import volume of raw asbestos (reported in
 824 2020).

1.4.3 Refinement of Evaluation of Releases to Surface Water

826 EPA did not evaluate the risk to aquatic species from exposure to surface water in its PF. During the PF
827 phase of the Risk Evaluation, EPA was still in the process of identifying potential asbestos water
828 releases for the TSCA COUs for chrysotile asbestos. After the PF was released, EPA continued to search
829 EPA databases as well as the literature and attempted to contact industries to shed light on potential
830 releases to water. The available information indicated that there were surface water releases of asbestos;
831 however, it is unclear of the source of the asbestos and the fiber type present. In the draft Risk
832 Evaluation, EPA concluded that, based on the reasonably available information in the published
833 literature, provided by industries using asbestos, and reported in EPA databases, there were minimal or
834 no releases of asbestos to surface water associated with the COUs that EPA is evaluating (see Appendix
835 D).

836 EPA has considered peer review and public comments on this conclusion and has decided to keep the
837 finding made in the draft Risk Evaluation (*i.e.*, that there were minimal or no releases of asbestos to
838 surface water associated with the COUs that EPA is evaluating in this Part 1 of the risk evaluation for
839 asbestos). This is because EPA is confident that the minimal water release data available and reported
840 more fully in the PF – and now presented again in Appendix D – cannot be attributed to chrysotile
841 asbestos from the COUs in this Part 1 of the risk evaluation for asbestos. Assessing possible risk to
842 aquatic organisms from the exposures described would not be reasonably attributed to the COUs.
843 However, based on the decision to develop a scope and risk evaluation for legacy uses and associated
844 disposals of asbestos (Part 2 of the final Risk Evaluation for asbestos), EPA expects to address the issue
845 of releases to surface water based on those other uses.

846

1.4.4 Exposure Pathways and Risks Addressed by Other EPA-Administered Statutes

847 In its TSCA Section 6(b) risk evaluations, EPA is coordinating action on certain exposure pathways and
848 risks falling under the jurisdiction of other EPA-administered statutes or regulatory programs. More
849 specifically, EPA is exercising its TSCA authorities to tailor the scope of its risk evaluations, rather than
850 focusing on environmental exposure pathways addressed under other EPA-administered statutes or
851 regulatory programs or risks that could be eliminated or reduced to a sufficient extent by actions taken
852 under other EPA-administered laws. EPA considers this approach to be a reasonable exercise of the
853 Agency's TSCA authorities, which include:

- 854 • TSCA Section 6(b)(4)(D): “The Administrator shall, not later than 6 months after the initiation
855 of a risk evaluation, publish the scope of the risk evaluation to be conducted, including the
856 hazards, exposures, conditions of use, and the potentially exposed or susceptible subpopulations
857 the Administrator expects to consider...”
- 858 • TSCA Section 9(b)(1): “The Administrator shall coordinate actions taken under this chapter with
859 actions taken under other Federal laws administered in whole or in part by the Administrator. If
860 the Administrator determines that a risk to health or the environment associated with a chemical
861 substance or mixture could be eliminated or reduced to a sufficient extent by actions taken under
862 the authorities contained in such other Federal laws, the Administrator shall use such authorities
863 to protect against such risk unless the Administrator determines, in the Administrator's
864 discretion, that it is in the public interest to protect against such risk by actions taken under this
865 chapter.”
- 866 • TSCA Section 9(e): “...[I]f the Administrator obtains information related to exposures or
867 releases of a chemical substance or mixture that may be prevented or reduced under another
868 Federal law, including a law not administered by the Administrator, the Administrator shall

- 869 make such information available to the relevant Federal agency or office of the Environmental
870 Protection Agency.”
- 871 • TSCA Section 2(c): “It is the intent of Congress that the Administrator shall carry out this
872 chapter in a reasonable and prudent manner, and that the Administrator shall consider the
873 environmental, economic, and social impact of any action the Administrator takes or proposes as
874 provided under this chapter.”
 - 875 • TSCA Section 18(d)(1): “Nothing in this chapter, nor any amendment made by the Frank R.
876 Lautenberg Chemical Safety for the 21st Century Act, nor any rule, standard of performance,
877 risk evaluation, or scientific assessment implemented pursuant to this chapter, shall affect the
878 right of a State or a political subdivision of a State to adopt or enforce any rule, standard of
879 performance, risk evaluation, scientific assessment, or any other protection for public health or
880 the environment that— (i) is adopted or authorized under the authority of any other Federal law
881 or adopted to satisfy or obtain authorization or approval under any other Federal law...”

882 TSCA authorities supporting tailored risk evaluations and intra-agency referrals

883 *TSCA Section 6(b)(4)(D)*

884 TSCA Section 6(b)(4)(D) requires EPA, in developing the scope of a risk evaluation, to identify the
885 hazards, exposures, conditions of use, and potentially exposed or susceptible subpopulations the Agency
886 “expects to consider” in a risk evaluation. This language suggests that EPA is not required to consider
887 all conditions of use, hazards, or exposure pathways in risk evaluations.

888 In the problem formulation documents for many of the first 10 chemicals undergoing risk evaluation,
889 EPA applied this authority and rationale to certain exposure pathways, explaining that “EPA is planning
890 to exercise its discretion under TSCA 6(b)(4)(D) to focus its analytical efforts on exposures that are
891 likely to present the greatest concern and consequently merit a risk evaluation under TSCA, by
892 excluding, on a case-by-case basis, certain exposure pathways that fall under the jurisdiction of other
893 EPA-administered statutes.” This approach is informed by the legislative history of the amended TSCA,
894 which supports the Agency’s exercise of discretion to focus the risk evaluation on areas that raise the
895 greatest potential for risk. See June 7, 2016 Cong. Rec., S3519-S3520. Consistent with the approach
896 articulated in the problem formulation documents, and as described in more detail below, EPA is
897 exercising its authority under TSCA to tailor the scope of exposures evaluated in TSCA risk evaluations,
898 rather than focusing on environmental exposure pathways addressed under other EPA-administered,
899 media-specific statutes and regulatory programs.

900 *TSCA Section 9(b)(1)*

901 In addition to TSCA Section 6(b)(4)(D), the Agency also has discretionary authority under the first
902 sentence of TSCA Section 9(b)(1) to “coordinate actions taken under [TSCA] with actions taken under
903 other Federal laws administered in whole or in part by the Administrator.” This broad, freestanding
904 authority provides for intra-agency coordination and cooperation on a range of “actions.” In EPA’s
905 view, the phrase “actions taken under [TSCA]” in the first sentence of Section 9(b)(1) is reasonably read
906 to encompass more than just risk management actions, and to include actions taken during risk
907 evaluation as well. More specifically, the authority to coordinate intra-agency actions exists regardless
908 of whether the Administrator has first made a definitive finding of risk, formally determined that such
909 risk could be eliminated or reduced to a sufficient extent by actions taken under authorities in other
910 EPA-administered Federal laws, and/or made any associated finding as to whether it is in the public
911 interest to protect against such risk by actions taken under TSCA. TSCA Section 9(b)(1) therefore
912 provides EPA authority to coordinate actions with other EPA offices without ever making a risk finding
913 or following an identification of risk. This includes coordination on tailoring the scope of TSCA risk

914 evaluations to focus on areas of greatest concern rather than exposure pathways addressed by other
915 EPA-administered statutes and regulatory programs, which does not involve a risk determination or
916 public interest finding under TSCA Section 9(b)(2).

917 In a narrower application of the broad authority provided by the first sentence of TSCA Section 9(b)(1),
918 the remaining provisions of Section 9(b)(1) provide EPA authority to identify risks and refer certain of
919 those risks for action by other EPA offices. Under the second sentence of Section 9(b)(1), “[i]f the
920 Administrator determines that a risk to health or the environment associated with a chemical substance
921 or mixture could be eliminated or reduced to a sufficient extent by actions taken under the authorities
922 contained in such other Federal laws, the Administrator shall use such authorities to protect against such
923 risk unless the Administrator determines, in the Administrator’s discretion, that it is in the public interest
924 to protect against such risk by actions taken under [TSCA].” Coordination of intra-agency action on
925 risks under TSCA Section 9(b)(1) therefore entails both an identification of risk, and a referral of any
926 risk that could be eliminated or reduced to a sufficient extent under other EPA-administered laws to the
927 EPA office(s) responsible for implementing those laws (absent a finding that it is in the public interest to
928 protect against the risk by actions taken under TSCA).

929 Risk may be identified by OPPT or another EPA office, and the form of the identification may vary. For
930 instance, OPPT may find that one or more conditions of use for a chemical substance present(s) a risk to
931 human or ecological receptors through specific exposure routes and/or pathways. This could involve a
932 quantitative or qualitative assessment of risk based on reasonably available information (which might
933 include, *e.g.*, findings or statements by other EPA offices or other federal agencies). Alternatively, risk
934 could be identified by another EPA office. For example, another EPA office administering non-TSCA
935 authorities may have sufficient monitoring or modeling data to indicate that a particular condition of use
936 presents risk to certain human or ecological receptors, based on expected hazards and exposures. This
937 risk finding could be informed by information made available to the relevant office under TSCA Section
938 9(e), which supports cooperative actions through coordinated information-sharing.

939 Following an identification of risk, EPA would determine if that risk could be eliminated or reduced to a
940 sufficient extent by actions taken under authorities in other EPA-administered laws. If so, TSCA
941 requires EPA to “use such authorities to protect against such risk,” unless EPA determines that it is in
942 the public interest to protect against that risk by actions taken under TSCA. In some instances, EPA may
943 find that a risk could be sufficiently reduced or eliminated by future action taken under non-TSCA
944 authority. This might include, *e.g.*, action taken under the authority of the Safe Drinking Water Act to
945 address risk to the general population from a chemical substance in drinking water, particularly if the
946 Office of Water has taken preliminary steps such as listing the subject chemical substance on the
947 Contaminant Candidate List. This sort of risk finding and referral could occur during the risk evaluation
948 process, thereby enabling EPA to use more a relevant and appropriate authority administered by another
949 EPA office to protect against hazards or exposures to affected receptors.

950 Legislative history on TSCA Section 9(b)(1) supports both broad coordination on current intra-agency
951 actions, and narrower coordination when risk is identified and referred to another EPA office for action.
952 A Conference Report from the time of TSCA’s passage explained that Section 9 is intended “to assure
953 that overlapping or duplicative regulation is avoided while attempting to provide for the greatest
954 possible measure of protection to health and the environment.” S. Rep. No. 94-1302 at 84. See also H.
955 Rep. No. 114-176 at 28 (stating that the 2016 TSCA amendments “reinforce TSCA’s original purpose of
956 filling gaps in Federal law,” and citing new language in Section 9(b)(2) intended “to focus the
957 Administrator’s exercise of discretion regarding which statute to apply and to encourage decisions that
958 avoid confusion, complication, and duplication”). Exercising TSCA Section 9(b)(1) authority to
959 coordinate on tailoring TSCA risk evaluations is consistent with this expression of Congressional intent.

960 Legislative history also supports a reading of Section 9(b)(1) under which EPA coordinates intra-agency
961 action, including information-sharing under TSCA Section 9(e), and the appropriately positioned EPA
962 office is responsible for the identification of risk and actions to protect against such risks. See, e.g.,
963 Senate Report 114-67, 2016 Cong. Rec. S3522 (under TSCA Section 9, “if the Administrator finds that
964 disposal of a chemical substance may pose risks that could be prevented or reduced under the Solid
965 Waste Disposal Act, the Administrator should ensure that the relevant office of the EPA receives that
966 information”); H. Rep. No. 114-176 at 28, 2016 Cong. Rec. S3522 (under Section 9, “if the
967 Administrator determines that a risk to health or the environment associated with disposal of a chemical
968 substance could be eliminated or reduced to a sufficient extent under the Solid Waste Disposal Act, the
969 Administrator should use those authorities to protect against the risk”). Legislative history on Section
970 9(b)(1) therefore supports coordination with and referral of action to other EPA offices, especially when
971 statutes and associated regulatory programs administered by those offices could address exposure
972 pathways or risks associated with conditions of use, hazards, and/or exposure pathways that may
973 otherwise be within the scope of TSCA risk evaluations.

974 *TSCA Sections 2(c) & 18(d)(1)*

975 Finally, TSCA Sections 2(c) and 18(d) support coordinated action on exposure pathways and risks
976 addressed by other EPA-administered statutes and regulatory programs. Section 2(c) directs EPA to
977 carry out TSCA in a “reasonable and prudent manner” and to consider “the environmental, economic,
978 and social impact” of its actions under TSCA. Legislative history from around the time of TSCA’s
979 passage indicates that Congress intended EPA to consider the context and take into account the impacts
980 of each action under TSCA. S. Rep. No. 94-698 at 14 (“the intent of Congress as stated in this
981 subsection should guide each action the Administrator takes under other sections of the bill”).

982 Section 18(d)(1) specifies that state actions adopted or authorized under any Federal law are not
983 preempted by an order of no unreasonable risk issued pursuant to TSCA Section 6(i)(1) or a rule to
984 address unreasonable risk issued under TSCA Section 6(a). Thus, even if a risk evaluation were to
985 address exposures or risks that are otherwise addressed by other federal laws and, for example,
986 implemented by states, the state laws implementing those federal requirements would not be preempted.
987 In such a case, both the other federal and state laws, as well as any TSCA Section 6(i)(1) order or TSCA
988 Section 6(a) rule, would apply to the same issue area. See also TSCA Section 18(d)(1)(A)(iii). In
989 legislative history on amended TSCA pertaining to Section 18(d), Congress opined that “[t]his approach
990 is appropriate for the considerable body of law regulating chemical releases to the environment, such as
991 air and water quality, where the states have traditionally had a significant regulatory role and often have
992 a uniquely local concern.” Sen. Rep. 114-67 at 26.

993 EPA’s careful consideration of whether other EPA-administered authorities are available and more
994 appropriate for addressing certain exposures and risks is consistent with Congress’ intent to maintain
995 existing federal requirements and the state actions adopted to locally and more specifically implement
996 those federal requirements, and to carry out TSCA in a reasonable and prudent manner. EPA believes it
997 is both reasonable and prudent to tailor TSCA risk evaluations in a manner reflective of expertise and
998 experience exercised by other EPA and State offices to address specific environmental media, rather
999 than attempt to evaluate and regulate potential exposures and risks from those media under TSCA. This
1000 approach furthers Congressional direction and EPA aims to efficiently use Agency resources, avoid
1001 duplicating efforts taken pursuant to other Agency and State programs, and meet the statutory deadline
1002 for completing risk evaluations.

1003 EPA-administered statutes and regulatory programs that address specific exposure pathways and/or risks

1004 During the course of the risk evaluation process for asbestos, Part 1 (chrysotile asbestos), OPPT worked
1005 closely with the offices within EPA that administer and implement regulatory programs under the Clean
1006 Air Act (CAA), the Safe Drinking Water Act (SDWA), and the Clean Water Act (CWA). Through intra-
1007 agency coordination, EPA determined that specific exposure pathways are well-regulated by the EPA
1008 statutes and regulations described in the following paragraphs.

1009 *Ambient Air Pathway*

1010 The CAA contains a list of hazardous air pollutants (HAP) and provides EPA with the authority to add
1011 to that list pollutants that present, or may present, a threat of adverse human health effects or adverse
1012 environmental effects. For stationary source categories emitting HAP, the CAA requires issuance of
1013 technology-based standards and, if necessary, additions or revisions to address developments in
1014 practices, processes, and control technologies, and to ensure the standards adequately protect public
1015 health and the environment. The CAA thereby provides EPA with comprehensive authority to regulate
1016 emissions to ambient air of any hazardous air pollutant.

1017 Asbestos was designated as a HAP on March 31, 1971 (36 FR 5931) and remains listed as a HAP under
1018 Section 112 of the CAA. See 42 U.S.C. § 7412. EPA has issued a number of standards for source
1019 categories that emit pollutants designated as a HAP prior to the 1990 Clean Air Act Amendments as
1020 well as technology-based standards for source categories that emit pollutants listed as a HAP under
1021 Section 112 of the CAA. The National Emission Standard for Asbestos includes standards for multiple
1022 sources or potential sources of asbestos releases to the ambient air including asbestos mills, roadways,
1023 manufacturing, demolition and renovation, insulating materials, waste disposal, and operations that
1024 convert asbestos-containing waste material into non-asbestos (asbestos-free) material, among others. See
1025 40 CFR part 61, subpart M. Because stationary source releases of asbestos to ambient air are addressed
1026 under the CAA, EPA is not evaluating emissions to ambient air from commercial and industrial
1027 stationary sources or associated inhalation exposure of the general population or terrestrial species in
1028 this Part 1 of the risk evaluation for asbestos.

1029 *Drinking Water Pathway*

1030 EPA has regular analytical processes to identify and evaluate drinking water contaminants of potential
1031 regulatory concern for public water systems under the Safe Drinking Water Act (SDWA). Under
1032 SDWA, EPA must also review existing national primary drinking water regulations every 6 years, and
1033 subsequently revise them as appropriate.

1034 EPA has promulgated National Primary Drinking Water Regulations (NPDWRs) for asbestos under
1035 SDWA. See 40 CFR part 141; Appendix A. EPA has set an enforceable Maximum Contaminant Level
1036 (MCL) as close as feasible to a health based, non-enforceable Maximum Contaminant Level Goal
1037 (MCLG). Feasibility refers to both the ability to treat water to meet the MCL and the ability to monitor
1038 water quality at the MCL, SDWA Sections 1412(b)(4)(D) and 1401(1)(C)(i), Public water systems are
1039 required to monitor for the regulated chemical based on a standardized monitoring schedule to ensure
1040 compliance with the MCL. The MCL for asbestos in water is 7 million fibers/liter, or 7 MFL.

1041 Hence, because the drinking water exposure pathway for asbestos is currently addressed in the NPDWR,
1042 EPA is not evaluating exposures to the general population from the drinking water exposure pathway in
1043 in this Part 1 of the risk evaluation for asbestos.

1044 *Ambient Water Pathway*

1045 EPA develops recommended water quality criteria under Section 304(a) of the CWA for pollutants in
1046 surface water that are protective of aquatic life or human health designated uses. EPA develops and
1047 publishes water quality criteria based on priorities of states and others that reflect the latest scientific
1048 knowledge. A subset of these chemicals is identified as “priority pollutants” (103 human health and 27
1049 aquatic life). The CWA requires states adopt numeric criteria for priority pollutants for which EPA has
1050 published recommended criteria under Section 304(a), the discharge or presence of which in the affected
1051 waters could reasonably be expected to interfere with designated uses adopted by the state. When states
1052 adopt criteria that EPA approves as part of state’s regulatory water quality standards, exposure is
1053 considered when state permit writers determine if permit limits are needed and at what level for a
1054 specific discharger of a pollutant to ensure protection of the designated uses of the receiving water. Once
1055 states adopt criteria as water quality standards, the CWA requires that National Pollutant Discharge
1056 Elimination System (NPDES) discharge permits include effluent limits as stringent as necessary to meet
1057 standards. CWA Section 301(b)(1)(C). This is the process used under the CWA to address risk to human
1058 health and aquatic life from exposure to a pollutant in ambient waters.

1059 EPA has identified asbestos as a priority pollutant and EPA has developed recommended water quality
1060 criteria for protection of human health for asbestos which are available for adoption into state water
1061 quality standards for the protection of human health and are available for use by NPDES permitting
1062 authorities in deriving effluent limits to meet state narrative criteria. See, *e.g.*, 40 CFR part 423,
1063 Appendix A; 40 CFR 131.11(b)(1); 40 CFR 122.44(d)(1)(vi); and 40 CFR 131.36(b)(1), 131.38(b)(1),
1064 and 40 CFR part 122, Appendix D, Table V. As such, EPA is not evaluating exposures to the general
1065 population from the surface water exposure pathway in this Part 1 of the risk evaluation for asbestos.

1066 EPA has not developed CWA section 304(a) recommended water quality criteria for the protection of
1067 aquatic life for asbestos, so there are no national recommended criteria for this use available for
1068 adoption into state water quality standards and available for use in NPDES permits.

1069 *On-site Releases to Superfund Sites*

1070 The Comprehensive Environmental Response, Compensation, and Liability Act – otherwise known as
1071 CERCLA or Superfund – provides EPA with broad authority to address uncontrolled or abandoned
1072 hazardous-waste sites as well as accidents, spills, and other releases of hazardous substances, pollutants
1073 and contaminants into the environment. Through CERCLA, EPA is provided authority to conduct a
1074 response action and seek reimbursement of cleanup costs from potentially responsible parties, or in
1075 certain circumstances, order a potentially responsible party to conduct a cleanup.

1076 CERCLA Section 101(14) defines “hazardous substance” by referencing other environmental statutes,
1077 including toxic pollutants listed under CWA Section 307(a); hazardous substances designated pursuant
1078 to CWA Section 311(b)(2)(A); hazardous air pollutants listed under CAA Section 112; imminently
1079 hazardous substances with respect to which EPA has taken action pursuant to TSCA Section 7; and
1080 hazardous wastes having characteristics identified under or listed pursuant to RCRA Section 3001. See
1081 40 CFR 302.4. CERCLA Section 102(a) also authorizes EPA to promulgate regulations designating as
1082 hazardous substances those substances which, when released into the environment, may present
1083 substantial danger to the public health or welfare or the environment. EPA must also promulgate
1084 regulations establishing the quantity of any hazardous substance the release of which must be reported
1085 under Section 103. Section 103 requires persons in charge of vessels or facilities to report to the
1086 National Response Center if they have knowledge of a release of a hazardous substance above the
1087 reportable quantity threshold.

1088 Asbestos is a hazardous substance under CERCLA. Releases of friable asbestos in excess of 1 pound
1089 within a 24-hour period must be reported (40 CFR 302.4, 302.6). This Part 1 of the risk evaluation for
1090 asbestos does not include on-site releases to the environment of asbestos at Superfund sites and
1091 subsequent exposure of the general population or non-human species.

1092 *Disposal Pathways*

1093 Asbestos is not regulated as a RCRA hazardous waste under RCRA Subtitle C; therefore, asbestos solid
1094 wastes are not required to be disposed of in Subtitle C hazardous waste landfills. However, it is possible
1095 that asbestos wastes could be disposed this way due to other characteristics of an asbestos-containing
1096 solid waste mixture. EPA is not evaluating on-site releases to land from RCRA Subtitle C hazardous
1097 waste landfills or exposures of the general population or terrestrial species from such releases in this Part
1098 1 of the risk evaluation for asbestos. Design standards for Subtitle C landfills require double liner,
1099 double leachate collection and removal systems, leak detection system, run on, runoff, and wind
1100 dispersal controls, and a construction quality assurance program. They are also subject to closure and
1101 post-closure care requirements including installing and maintaining a final cover, continuing operation
1102 of the leachate collection and removal system until leachate is no longer detected, maintaining and
1103 monitoring the leak detection and groundwater monitoring system. Bulk liquids may not be disposed in
1104 Subtitle C landfills. Subtitle C landfill operators are required to implement an analysis and testing
1105 program to ensure adequate knowledge of waste being managed, and to train personnel on routine and
1106 emergency operations at the facility. Hazardous waste being disposed in Subtitle C landfills must also
1107 meet RCRA waste treatment standards before disposal. See 40 CFR part 264; Appendix A.

1108 In addition, landfills have special requirements for handling and securing the asbestos-containing waste
1109 regulated under the National Emission Standard for Asbestos (40 C.F.R. Part 61, Subpart M) to prevent
1110 releases of asbestos into the air. This regulation requires regulated asbestos-containing waste material be
1111 sealed in a leak-tight container while wet, labeled, and disposed of properly in a landfill qualified to
1112 receive asbestos waste. Landfills have special requirements for handling and securing the asbestos
1113 containing waste to prevent releases of asbestos into the air. Transportation vehicles that move the waste
1114 from the point of generation to the asbestos landfill have special labeling requirements and waste
1115 shipment recordkeeping requirements. EPA is not evaluating emissions from the asbestos waste pathway
1116 from the processing and use of chrysotile asbestos diaphragms at chlor-alkali facilities. The National
1117 Emission Standard for Asbestos specifically addresses this asbestos waste pathway. See 40 CFR §§
1118 61.144(a)(9), 61.150. Finally, asbestos fibers (all six types) are not likely to be leached out of a landfill.

1119 EPA is not evaluating on-site releases to land from RCRA Subtitle D municipal solid waste (MSW)
1120 landfills or exposures of the general population or terrestrial species from such releases in this Part 1 of
1121 the risk evaluation for asbestos. While permitted and managed by the individual states, municipal solid
1122 waste landfills are required by federal regulations to implement some of the same requirements as
1123 Subtitle C landfills. MSW landfills generally must have a liner system with leachate collection and
1124 conduct groundwater monitoring and corrective action when releases are detected. MSW landfills are
1125 also subject to closure and post-closure care requirements and must have financial assurance for funding
1126 of any needed corrective actions. MSW landfills have also been designed to allow for the small amounts
1127 of hazardous waste generated by households and very small quantity waste generators (less than 220 lbs
1128 per month). Finally, asbestos fibers (all six types) are not likely to be leached out of a landfill.

1129 EPA is not evaluating on-site releases to land from industrial non-hazardous waste and
1130 construction/demolition waste landfills or associated exposures to the general population or terrestrial
1131 species in this Part 1 of the risk evaluation for asbestos. Industrial non-hazardous and
1132 construction/demolition waste landfills are primarily regulated under authorized state regulatory

1133 programs. States must also implement limited federal regulatory requirements for siting, groundwater
1134 monitoring and corrective action and a prohibition on open dumping and disposal of bulk liquids. States
1135 may also establish additional requirements such as for liners, post-closure and financial assurance, but
1136 are not required to do so. See, *e.g.*, RCRA Section 3004(c), 4007; 40 CFR part 257.

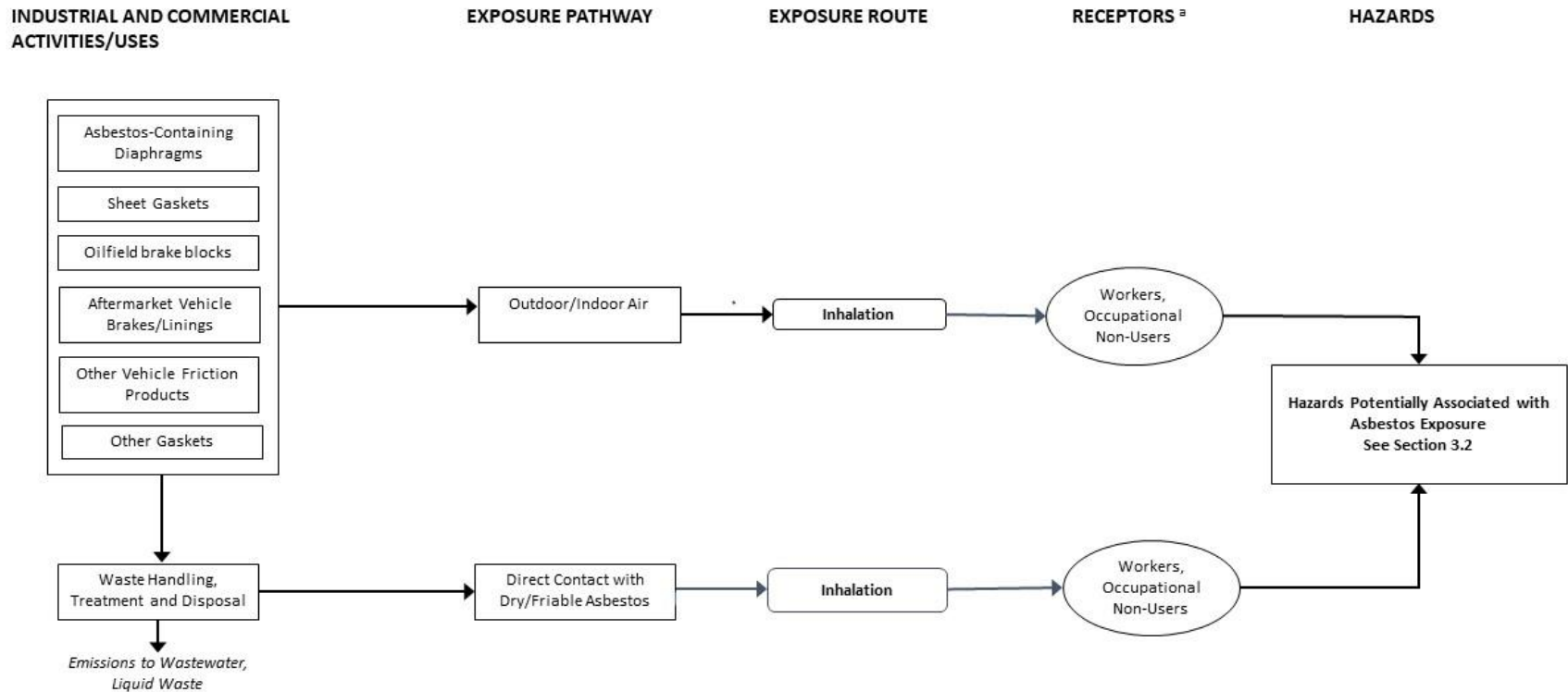
1137 EPA is not evaluating emissions to ambient air from municipal and industrial waste incineration and
1138 energy recovery units or associated exposures to the general population or terrestrial species in this Part
1139 1 of the risk evaluation for asbestos, as these emissions are regulated under Section 129 of the Clean Air
1140 Act. CAA Section 129 requires EPA to review and, if necessary, add provisions to ensure the standards
1141 adequately protect public health and the environment. Thus, combustion by-products from incineration
1142 treatment of asbestos wastes would be subject to these regulations, as would asbestos burned for energy
1143 recovery. See 40 CFR part 60.

1144 EPA is not evaluating on-site releases to land that go to underground injection or associated exposures to
1145 the general population or terrestrial species in this Part 1 of the risk evaluation for asbestos.
1146 Environmental disposal of asbestos injected into Class I hazardous well types are covered under the
1147 jurisdiction of the SDWA and disposal of asbestos via underground injection is not likely to result in
1148 environmental and general population exposures. See 40 CFR part 144.

1149 **1.4.5 Conceptual Models**

1150 The conceptual models have been modified to reflect the refined COUs of chrysotile asbestos described
1151 in Section 1.4.1. Figure 1-3. Chrysotile Asbestos and Figure 1-4. Commercial Chrysotile Asbestos
1152 present the conceptual models for industrial and commercial uses and consumer uses, respectively.-The
1153 chrysotile asbestos conceptual model for environmental releases and wastes from the refined COUs was
1154 removed and is discussed in Releases and Exposure to the Environment Supplementary Information
1155 Appendix D.

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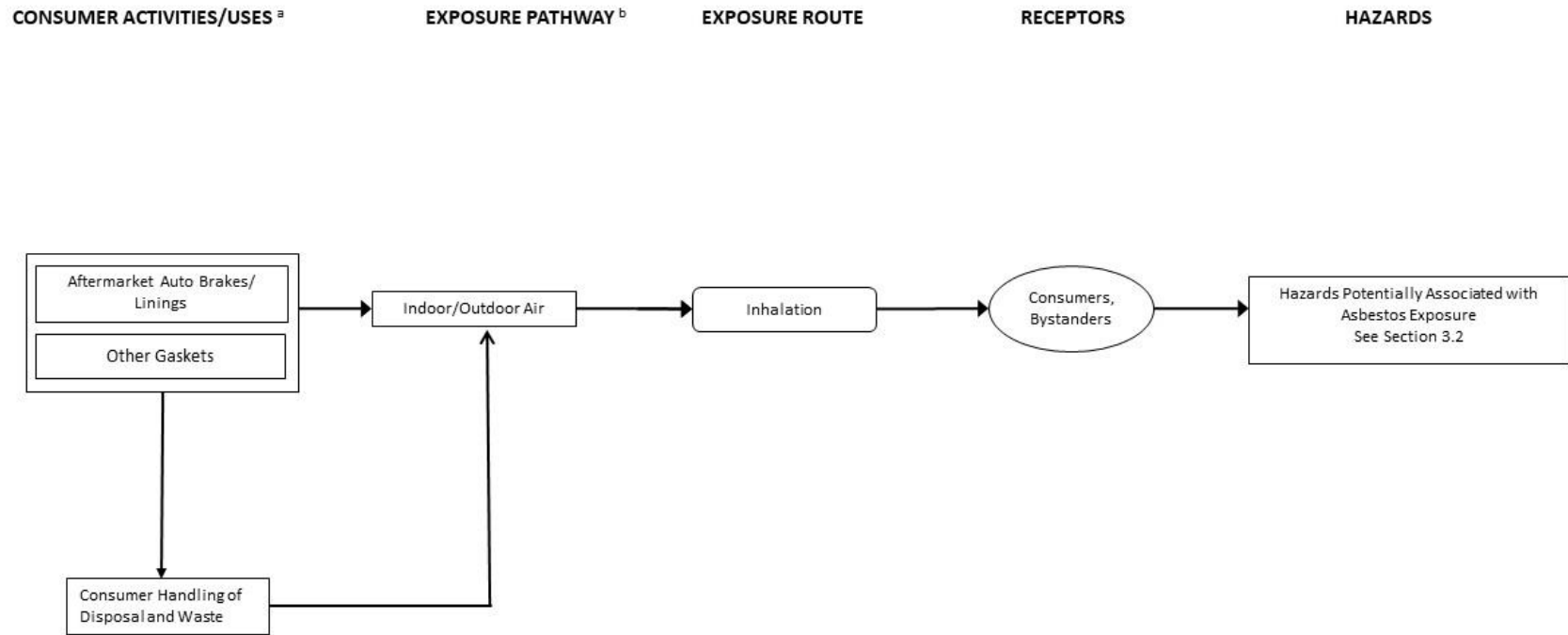


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Figure 1-3. Chrysotile Asbestos Conceptual Model for Industrial and Commercial Activities and Uses: Potential Exposures and Hazards

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

^a Receptors include PESS.



1164

1165 **Figure 1-4. Chrysotile Asbestos Conceptual Model for Consumer Activities and Uses: Potential Exposures and Hazards**

1166 ^aWoven products were removed from this model after the PF was published. Utility vehicle gaskets were added.

1167 ^bProducts may be used during indoor and outdoor activities.

1168 ^cReceptors include PESS.

1.5 Systematic Review

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

EPA planned and conducted a comprehensive literature search based on key words related to the different discipline-specific evidence supporting this Part 1 of the risk evaluation for asbestos (*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 this Part 1 of the risk evaluation for asbestos. Screening decisions were made based on eligibility criteria documented in the form of the populations, exposures, comparators, and outcomes (PECO) framework or a modified framework.¹²

¹² 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.

Data sources that met the criteria were carried forward to the data evaluation stage. The inclusion and exclusion criteria for full text screening for asbestos are available in Appendix D of the *Problem Formulation of the Risk Evaluation for Asbestos* ([U.S. EPA, 2018d](#)).

Although EPA conducted a comprehensive search and screening process as described above, EPA made the decision to leverage the literature published in previous assessments¹³ when identifying relevant key and supporting data¹⁴ and information for developing this Part 1 of the risk evaluation for asbestos. This is discussed in the [Strategy for Conducting Literature Searches for Asbestos: Supplemental Document to the TSCA Scope Document](#) (EPA-HQ-OPPT-2016-0736). In general, many of the key and supporting data sources were identified in the comprehensive *Asbestos Bibliography: Supplemental File for the TSCA Scope Document* ([U.S. EPA, 2017a, b](#)). However, there were instances in which EPA missed relevant references that were not captured in the initial categorization of the on-topic references. EPA found additional relevant data and information using backward reference searching, which is a technique that will be included in future search strategies. This issue is discussed in Section 4 of the [Application of Systematic Review for TSCA Risk Evaluations](#) U.S. EPA (2018a). Other relevant key and supporting references were identified through targeted supplemental searches to support the analytical approaches and methods in this Part 1 of the risk evaluation for asbestos (*e.g.*, to locate specific information for exposure modeling) or to identify new data and information published after the date limits of the initial search.

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

This pragmatic approach allowed EPA to maximize the scientific and analytical efforts of other regulatory and non-regulatory agencies by accepting, for the most part, the relevant scientific knowledge gathered and analyzed by others except for influential information sources that may have an impact on the weight of the scientific evidence and ultimately the risk findings. The influential information (*i.e.*, key/supporting) came from a smaller pool of sources subject to the rigor of the TSCA systematic review process to ensure that this Part 1 of the risk evaluation for asbestos used the best available science and the weight of the scientific evidence.

Figure 1-5. to Figure 1-9. depict the literature flow diagrams illustrating the results of this process for each scientific discipline-specific evidence supporting this Part 1 of the risk evaluation for asbestos. Each diagram provides the total number of references at the start of each systematic review stage (*i.e.*,

¹³ 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).

¹⁴ Key and supporting data and information are those that support key analyses, arguments, and/or conclusions in this Part 1 of the risk evaluation for asbestos.

data search, data screening, data evaluation, data extraction/data integration) and those excluded based on criteria guiding the screening and data quality evaluation decisions.

EPA bypassed the data screening step for data sources that were highly relevant to this Part 1 of the risk evaluation for asbestos and moved these sources directly to the data quality evaluation step, as described above. These data sources are depicted as “key/supporting data sources” in the literature flow diagrams. Note that the number of “key/supporting data sources” were excluded from the total count during the data screening stage and added, for the most part, to the data evaluation stages depending on the discipline-specific evidence. The exception was the releases and occupational exposure data sources that were subject to a combined data extraction and evaluation step as shown in Figure 1-6.

EPA did not have a previous, recent toxicity assessment for general asbestos on which to build; therefore, initially the Systematic Review included a very large number of papers for all areas. Initially, studies were limited to those published after 1987, containing at least one of the six fiber types identified under TSCA. In terms of evaluating human health, only observational human studies were identified for searching; however, the scope of the risk evaluation was further refined to identify studies pertaining to only mesothelioma and lung cancer as health outcomes¹⁵, as well as studies containing information specific to chrysotile asbestos only.

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

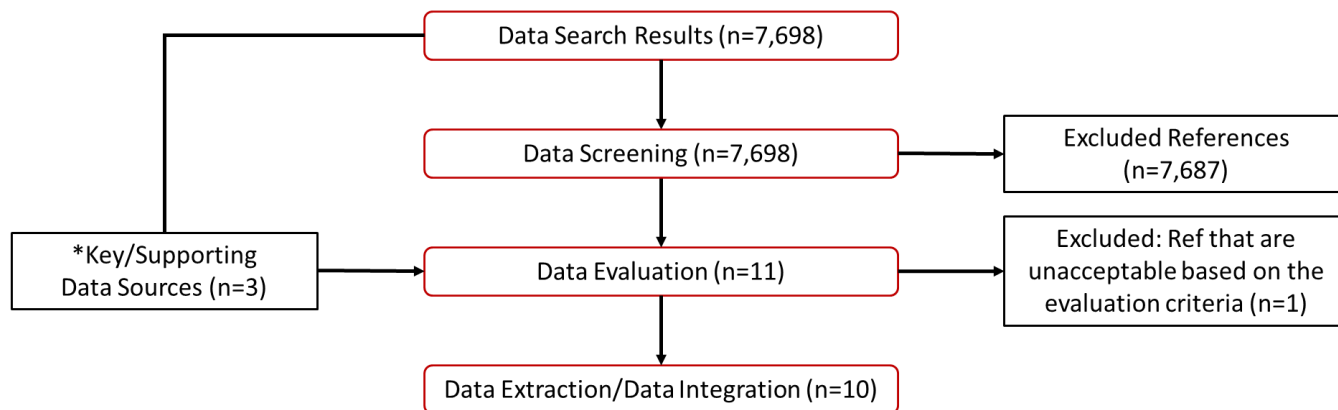
Information for fate assessment for the first 10 chemical risk evaluations considered the physical chemical properties of the chemical and environmental endpoints. For the first 10 chemicals, EPA assessed chemical fate as defined by traditional fate endpoints, for example, solubility, partitioning coefficients, biodegradation and bioaccumulation – properties that do not apply to asbestos minerals. As such, there were few discipline-specific papers identified in the fate systematic review of asbestos literature (Figure 1-5).

¹⁵ See Appendix M for an exception in response to peer review and public comments.

Figure 1-5. 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.

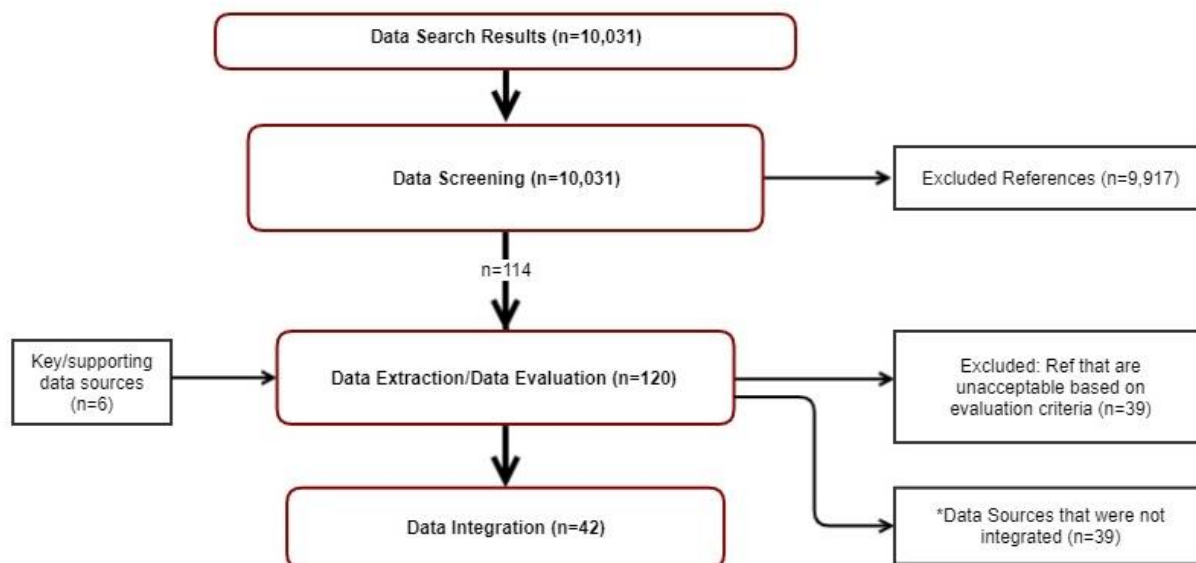
Note 2: Data sources identified relevant to physical-chemical properties were not included in this literature flow diagram. The data quality evaluation of physical-chemical properties studies can be found in the supplemental document, *Data Quality Evaluation of Physical-Chemical Properties Studies* ([U.S. EPA, 2020j](#)) 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.

Figure 1-6. Key/Supporting Data Sources for Engineering Releases and Occupational Exposure

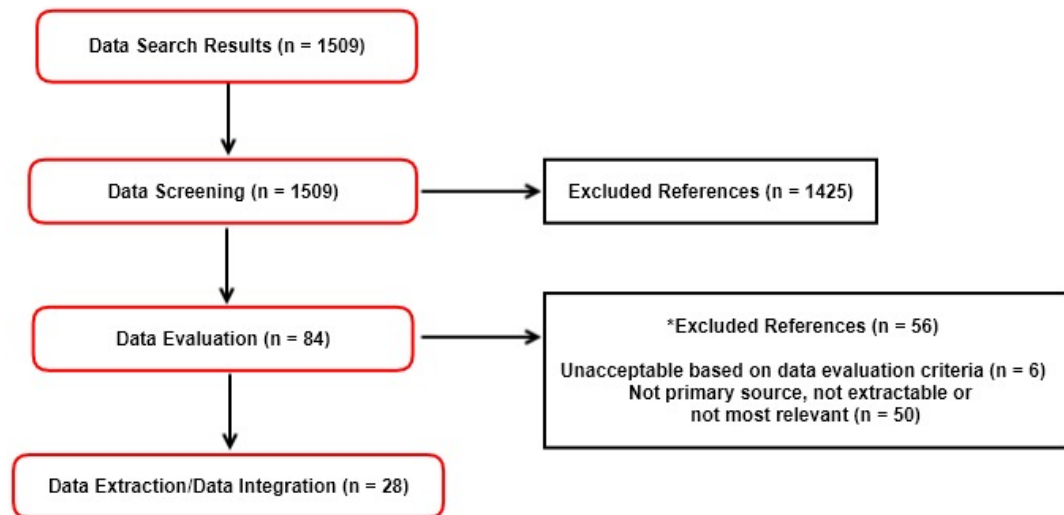
Note: Literature search results for environmental release and occupational exposure yielded 10,031 data sources. Of these data sources, 114 were determined to be relevant for this Part 1 of the risk evaluation for asbestos through the data screening process. These relevant data sources were entered into 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).



*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.

Figure 1-7. 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 this Part 1 of the risk evaluation for asbestos. The data evaluation and data extraction files are provided as separate files (See Appendix B).



*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.

Figure 1-8. 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).

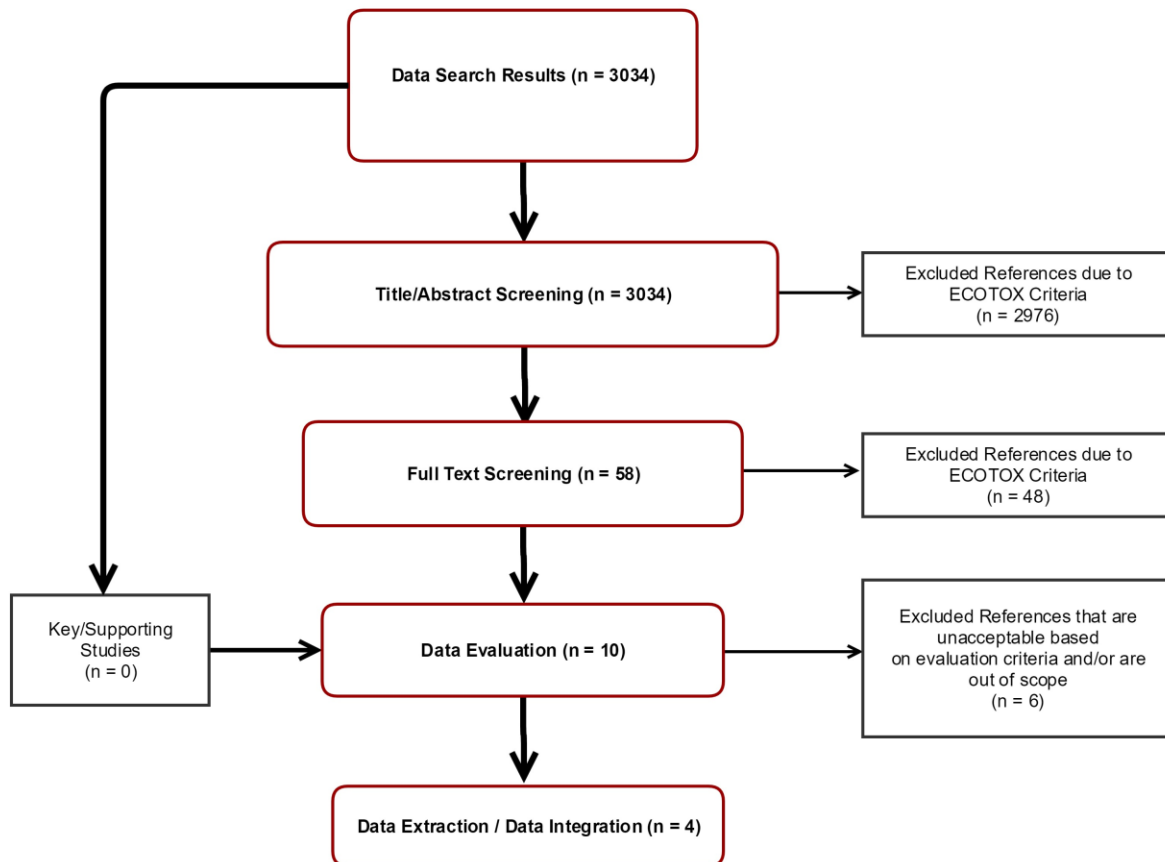
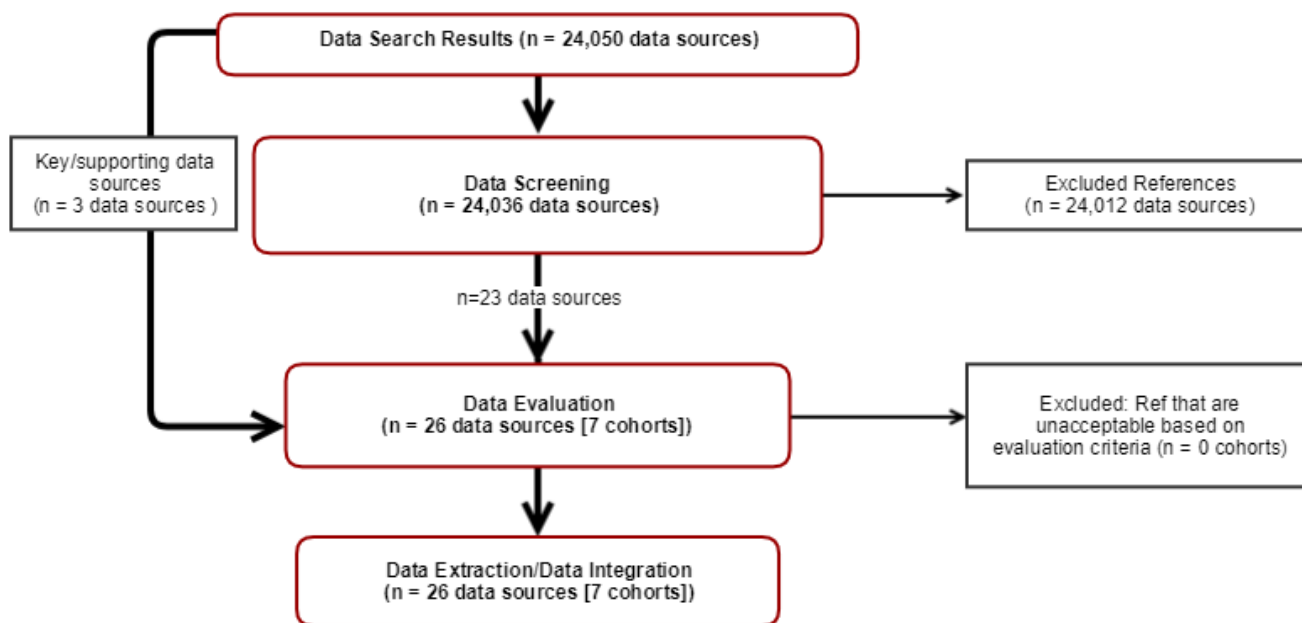


Figure 1-9. Key/Supporting Data Sources for Human Health Hazard

Note: Studies were restricted to only mesothelioma and lung cancer as health outcomes¹⁶, 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).



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.

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 chrysotile asbestos exposure and risk of lung cancer and 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.

¹⁶ An exception is for studies for laryngeal and ovarian cancer that informed the development of an adjustment factor for the IUR (see Appendix M). These studies were additionally included based on peer review and public comments.

2 EXPOSURES

This section describes EPA's approach to assessing environmental and human exposures. First, the fate and transport of chrysotile asbestos in the environment is characterized. Then, releases of chrysotile asbestos to the environment to evaluate environmental receptors are assessed. Finally, an evaluation of exposures to humans (occupational and consumers; including potentially exposed or susceptible subpopulations (PESS)) is presented. For all exposure-related disciplines, EPA screened, evaluated, extracted and integrated available empirical (*i.e.*, monitoring) data.

Exposure equations and selected values used in the exposure assessment are presented in the following sections. More specific information is provided in supplemental files identified in Appendix B: List of Supplemental Documents.

2.1 Fate and Transport

Although Part 1 of the risk evaluation on asbestos is focused on the chrysotile asbestos fiber type, some of the information in this section is taken from and pertains to asbestos fibers in general. Asbestos is a persistent mineral fiber that can be found in soils, sediments, lofted in air and windblown dust, surface water, ground water and biota ([ATSDR, 2001b](#)). Asbestos fibers are largely chemically inert in the environment. They may undergo minor physical changes, such as changes in fiber length or leaching of surface minerals, but do not react or dissolve in most environmental conditions ([Favero-Longo et al., 2005](#); [Gronow, 1987](#); [Schreier et al., 1987](#); [Choi and Smith, 1972](#)).

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

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

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

Chrysotile asbestos, which is the focus of the Risk Evaluation for Asbestos Part 1, may be released to the environment through industrial or commercial activities, such as processing raw chrysotile asbestos, fabricating/processing asbestos containing products, or the lofting of friable chrysotile asbestos during use, disturbance and disposal of asbestos containing products.

2.2 Releases to Water

2.2.1 Water Release Assessment Approach and Methodology

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

As discussed in the PF document and *Exposure Pathways and Risks Addressed by Other EPA-Administered Statutes* (Section 1.4.4), because the drinking water exposure pathway for asbestos is currently addressed in the Safe Drinking Water Act (SDWA) via a NPDWR for asbestos, this pathway (drinking water for human health) was not evaluated in this Part 1 of the risk evaluation for asbestos. The Office of Water does not have an ambient water quality criterion for asbestos for aquatic life. Thus, potential releases from industrial and commercial activities associated with the TSCA COUs to surface water were considered in this Part 1 of the risk evaluation for asbestos. However, identifying or estimating asbestos concentrations in water to evaluate risk to environmental receptors has been challenging. During the PF phase of the RE, EPA was still in the process of identifying potential asbestos water releases for the TSCA COUs. After the PF was released, EPA continued to search other sources of data including TRI data, EPA environmental and compliance monitoring databases, including permits, industry responses to EPA questions, and other EPA databases. Details of these investigations are included in Appendix D and summarized below.

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

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

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 for 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 Part 1 of the risk evaluation for asbestos. 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 \geq 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 (<0.2%)
MRL = Minimum Reporting Level MFL = million fibers per liter MCL = maximum contaminant level			

2.2.2 Water Releases Reported by Conditions of Use

2.2.2.1 Processing and Industrial Use of Chrysotile 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 chrysotile asbestos diaphragms are 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 μ m 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. EPA acknowledges there is some uncertainty in this conclusion in the absence of monitoring data to confirm assumptions; however, EPA believes this uncertainty is low.

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

2.2.2.2 Processing Chrysotile Asbestos-Containing Sheet Gaskets

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

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

2.2.2.3 Industrial Use of Sheet Gaskets at Chemical Production Plants

Based on reasonably available process information for the titanium dioxide (TiO₂) production facility - the example used in this Part 1 of the risk evaluation for asbestos for chemical production plants - described by ACC ([ACC, 2017b](#)) and EPA knowledge of the titanium manufacturing process, the purpose of the gasket is to seal equipment components. The information indicates that after maintenance workers remove a gasket from a flange, he or she will double-bag and seal the gasket and label the bag "asbestos," and place it in special containers for disposal in a landfill permitted to accept asbestos wastes. It appears that there are no water releases during use of asbestos gaskets or disposal, and water is not used as an exposure control method; therefore, releases to water are not anticipated. However, it is not possible to rule out incidental releases of asbestos fibers in wastewater at other facilities if different methods are used, but any amounts of release cannot be quantified.

2.2.2.4 Industrial Use and Disposal of Chrysotile Asbestos-Containing Brake Blocks in Oil Industry

EPA attempted to evaluate potential water releases of asbestos from use in oil field brake blocks. EPA found no reasonably available data or publications documenting asbestos releases from the use of oil field brake blocks to water. The only relevant information obtained was an industry contact's remark

that workers wash down drawworks before removing used brake blocks and installing new ones ([Popik, 2018](#)) – a comment that suggests some asbestos fibers may be released into water during this practice. The TRI reporting requirements do not apply to the three NAICS codes believed to best represent the industries that use oil field brake blocks. No other reasonably available data, such as relevant sampling data, publications, or other quantitative insights were found to inform the release assessment. The reasonably available information currently available for this COU is insufficient for deriving water release estimates.

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

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

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

2.2.3 Summary of Water Releases and Exposures

During the PF phase of the RE, EPA was still in the process of identifying potential asbestos water releases for the TSCA COUs in this document. After the PF was released, EPA continued to search EPA databases as well as the literature and attempted to contact industries to shed light on potential releases to water. Very little information was located that indicated that there were surface water releases of asbestos; however, it is unclear of the source of the asbestos and the fiber type present. In the draft Risk Evaluation, EPA concluded that, based on the reasonably available information in the published literature, provided by industries using asbestos, and reported in EPA databases, there were minimal or no releases of asbestos to surface water associated with the COUs that EPA is evaluating (see Appendix D). EPA has considered peer review and public comments on this conclusion and has decided to keep the finding made in the draft Risk Evaluation (*i.e.*, that there were minimal or no releases of asbestos to surface water associated with the COUs that EPA is evaluating in this Part 1 of the risk evaluation for asbestos). This is because EPA is confident that the minimal water release data available cannot be attributed to chrysotile asbestos from the COUs in this Part 1 of the risk evaluation for asbestos. Assessing possible risk to aquatic organisms from the exposures described would not be reasonably attributed to the COUs. However, based on the decision to develop a scope and risk evaluation for legacy uses and associated disposals of asbestos (Part 2 of the final Risk Evaluation for asbestos), EPA expects to address the issue of releases to surface water based on those other uses (See Section 4.1).

2.3 Human Exposures

EPA evaluated both occupational and consumer scenarios for each COU. The following table provides a description of the COUs and the scenario (occupational or consumer) evaluated in this Part 1 of the risk evaluation for asbestos.

Table 2-2. Crosswalk of Conditions of Use and Occupational and Consumer Scenarios Assessed in the Risk Evaluation for Asbestos Part 1: Chrysotile Asbestos

COU	Scenario	Form of Chrysotile Asbestos ¹
Diaphragms for Chlor-Alkali Industry (Processing and Use)	Occupational	Imported raw asbestos (used to fabricate diaphragms)
Brake Block Use (Use)	Occupational	Imported article
Sheet Gaskets Stamping (Processing)	Occupational	Imported sheets
Sheet Gaskets In chemical production (Use)	Occupational	Gaskets imported or purchased in US
Brakes Installation in exported cars (Use)	Occupational	Imported brakes
Brakes Repair/replacement (Use and Disposal)	Occupational (repair shops)	Imported brakes
Brakes Repair/replacement (Use and Disposal)	Consumer (DIY)	Imported brakes
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

¹ EPA understands that, with the exception of chlor-alkali and possibly sheet gaskets, these products could be purchased through the internet.

2.3.1 Occupational Exposures

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 who are ≥ 16 years of age. This section summarizes the key occupational acute and chronic inhalation exposure concentrations for asbestos.

EPA only evaluated inhalation exposures to workers and occupational non-users (ONUs) in association with chrysotile asbestos manufacturing (import), processing, distribution and use in industrial applications and products in this Part 1 of the risk evaluation for asbestos. The physical condition of chrysotile 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, the products can be made friable due to physical and chemical wear over time. 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.

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 chrysotile asbestos or chrysotile 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.

EPA considered two issues unique to asbestos, when compared to other chemicals for which EPA has developed TSCA risk evaluations. One issue is the possibility of asbestos fibers settling to surfaces and subsequently becoming resuspended into the workplace air. The extent to which this process occurs is presumably reflected in the sampling data that EPA considered for each COU. The second unique issue for asbestos is that it can be found in friable and non-friable materials; and the friability of the materials has direct bearing on asbestos releases to the air. This issue is also presumably reflected in the sampling data (*i.e.*, asbestos in friable materials has a greater likelihood of being detected in the air samples, as compared to asbestos in non-friable materials).

Components of the Occupational Exposure Assessment

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

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

acknowledges the limitations associated with these exposure estimates.

- **Inhalation Exposure Results for Use in the Part 1 of the risk evaluation for Asbestos:** Central tendency and high-end estimates of inhalation exposure to workers and ONUs.

2.3.1.1 Occupational Exposures Approach and Methodology

EPA reviewed reasonably available information from OSHA, NIOSH, the peer-reviewed literature, industries using asbestos or asbestos-containing products, and trade associations that represent this industry (*e.g.*, ACC) to identify relevant occupational inhalation exposure data. The data provided by OSHA included sampling results in states with federal OSHA oversight; data from “state plan states” were not included in OSHA’s data submittal to EPA. Quantitative data obtained during Systematic Review were used to build appropriate exposure scenarios when monitoring data were not reasonably available to develop exposure estimates. For uses with limited available exposure data the assessment used similar occupational data and best professional judgment to estimate exposures. In these cases, EPA used assumptions to evaluate risk.

General Inhalation Exposures Approach and Methodology

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

2.3.1.2 Consideration of Engineering Controls and Personal Protective Equipment

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

OSHA Respiratory Protection and Asbestos 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 for general industry (29 CFR § 1910.1001) construction (29 CFR § 1926.1101), and shipyards (29 CFR § 1915.1001). These standards have multiple provisions that are highlighted below.

OSHA’s Respiratory Protection Standard (29 CFR § 1910.134) requires employers to provide respiratory protection whenever it is necessary to protect the health of the employee from contaminated

or oxygen deficient air. This includes situations where respirators are necessary to protect employees in an emergency. Employers must follow the hierarchy of controls which requires the use of engineering and work practice controls where feasible. Only if such controls are not feasible or while they are being implemented may an employer rely on a respirator to protect employees. 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^{eg}

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

^a 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.

^b 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.

^c This APF category includes filtering facepieces and half masks with elastomeric facepieces.

^d 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.

^e 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).

^f These respirators are not common.

^g 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 asbestos standards also include respiratory protection provisions found at 29 CFR § 1910.1001(g) for general industry, 29 CFR § 1926.1101(h) for construction, and 29 CFR § 1915.1001(g) for shipyards. The respiratory protection provisions in these standards require employers to provide each employee an appropriate respirator that complies with the requirements outlined in the provision. In the general industry standard, paragraph (g)(2)(ii) requires employers to provide an employee with a tight-fitting, powered air-purifying respirator (PAPR) instead of a negative pressure respirator selected according to paragraph (g)(3) when the employee chooses to use a PAPR and it provides adequate protection to the employee. In addition, paragraph (g)(3) of the general industry standard 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 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 if the industrial hygiene program in place is poorly maintained. An inadequate respiratory protection program could lead to inadequate respirator fit tests and poor maintenance of respirators which could affect APF. 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 protective 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 a 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 asbestos) 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). Because it is chemically inert and able to effectively separate the anode and cathode chemicals in electrolytic cells ([USGS, 2017](#)), the incorporation of asbestos can be viewed as vital. Figure 2-1. below shows a typical diaphragm after it has been formed.



**Figure 2-1. Closeup of a Chrysotile Asbestos 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 were generally consistent with details of the process descriptions provided by industry and described below. Other citations are included in the following paragraphs only for specific details not covered in the main docket reference ([EPA-HQ-OPPT-2016-0763-0052](#)).

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

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

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

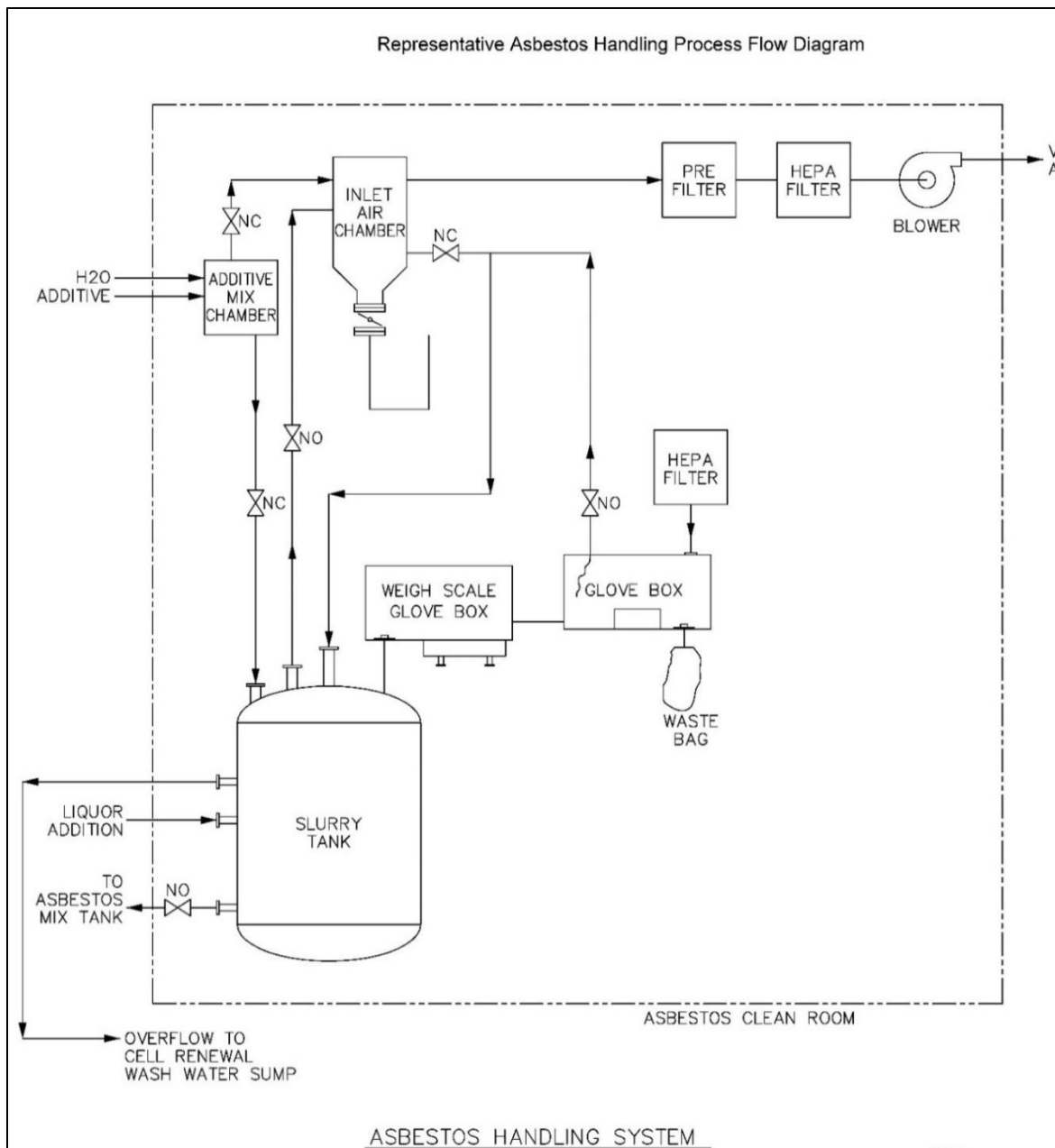


Figure 2-2. Process Flow Diagram of an Asbestos Handling System and Slurry Mix Tank Image Courtesy of the American Chemistry Council Source: EPA-HQ-OPPT-2016-0736-0106

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, and the asbestos fuses to the screen or plate; and the product material is 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

area of 70 m² and each cell typically has 20 chrysotile asbestos diaphragms within it, although cell sizes vary ([EPA Preliminary Information](#)).

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

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

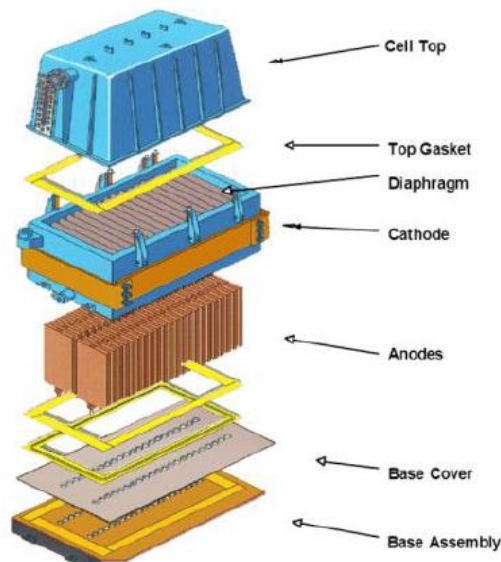


Figure 2-3. Electrolytic Cell Construction
Image courtesy of the American Chemistry Council
Source: ([See Enclosure B](#))

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](#)). Industry reports that chlor-alkali facilities rarely receive damaged bags of chrysotile asbestos. According to Occidental, the last time the company's facilities reported receiving a broken bag was between 4 and 10 years ago and the range in this estimate reflects observations from different Occidental facilities ([Occidental, Volume 2, p. 27](#)).

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 f/cc at 50th percentile and as high as 0.04 f/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 ([See Enclosure C](#)).

For cell assembly, 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 diaphragms to remove the asbestos coating. For disassembly, workers typically wear impermeable gloves, boots, goggles, and disposable particulate suits but do not wear respirators even though the short

term (15-minute sampling time) ambient air concentrations were reported to be 0.016 f/cc at 50th percentile and as high as 0.45 f/cc ([See Enclosure C](#)). For hydroblasting, workers wear a supplied air respirator hood, a waterproof suit, impermeable gloves, and boots ([See Enclosure C](#)). This activity occurs in blasting rooms, and workers (while wearing PPE) may be present in these rooms during hydroblasting activity ([Axiall-Westlake, 2017](#)).

For one site EPA visited, the hydroblasting itself was not enclosed but was conducted in a dedicated area. The asbestos handling area (slurry mixing, oven, diaphragm disassembly, and hydroblasting area) was walled off on three sides with a series of giant pull down doors. The fourth side wall did not extend to the ceiling. The layout of such areas may be different at other sites. The frequency with which workers conduct hydroblasting varies from one facility to the next. Some facilities do not hydroblast spent diaphragms at all; while others may conduct this activity up to five times per week, with each hydroblasting event lasting up to 90 minutes.

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

2.3.1.3.3 Number of Sites and Potentially Exposed Workers – Asbestos Diaphragms

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

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

In 2016 CDR, Olin reported a total of at least 25 and fewer than 50 workers who are likely exposed to asbestos across all of the company's chlor-alkali facilities, and Axiall-Westlake reported a total of at least 50 and fewer than 100 workers who are likely exposed to asbestos across all of the company's chlor-alkali facilities. This results in an estimate of at least 75 (25 plus 50) and fewer than 148 (49 plus

99) workers likely exposed, although this estimate does not include Occidental facilities. As noted previously, Occidental facilities did not report to CDR.

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

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

2.3.1.3.4 Occupational Inhalation Exposures – Chrysotile Asbestos Diaphragms

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

Analysis of Exposed Workers

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

To assess occupational inhalation exposures, EPA used exposure monitoring data provided by industry. Data were provided by the three companies that currently use chrysotile asbestos in the United States chlor-alkali industry. Occidental provided exposure monitoring data for six facilities for 1996 to 2016 ([Occidental Data, see Volume 2](#)); Axiall-Westlake provided data for 2016 from a single facility ([Axiall, Attachments 1 and 2](#)); and Olin provided data for 2012 to 2019 from three chlor-alkali facilities and a fourth facility that reprocesses anodes ([Olin Corp, 2017](#)). ACC also provided data for 1996 to 2016 ([ACC Data](#)) but those data were duplicative of the data submissions from the individual companies..

EPA also reviewed information published by European Union (EU) agencies ([EC, 2014](#); [ECHA, 2014](#)). The limitation with these publications is that exposure data from EU facilities may not be representative of the U.S. manufacturing environment, due to differences in process design, production levels, ventilation practices, regulatory frameworks, and other factors.

The following tables summarize occupational exposure results of different exposure durations for the fabrication, use, and disposal of chrysotile asbestos diaphragms in the chlor-alkali industry. The exposure durations considered are full-shift samples, 30-minute average samples, and additional samples of other durations. The tables summarize 759 personal breathing zone sampling results based on the combined data from Axiall-Westlake, Occidental, and Olin; which included a numerical sample duration for each sample. 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.

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. EPA notes that more than half of the samples were non-detectable.

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. (Note: The data in Table 2-7 were provided by ACC. These data were not included in the tallies in the other tables, because ACC informed EPA that the data it provided were duplicates of data from the three companies.)

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	2001 to 2017	58	11**	0.024	0.512

*Data from Olin and Occidental, 47 percent of these samples were non-detects

**Note: The maximum concentration in this table (11 f/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 f/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	357	0.41	0.0049	0.034

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

**Data from Axiall-Westlake, Occidental, and Olin. 57 percent of these were non-detects

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)	50th Percentile (f/cc)	95th Percentile (f/cc)
PBZ	2004 to 2019	344	1.78	0.024	0.514

*Data from Axiall-Westlake, Occidental, and Olin, 53 percent of these were non-detects

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](#))

Analysis of ONUs

At chlor-alkali facilities, ONU exposures to chrysotile 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 PPE. 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.

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 came from Olin’s facilities located in Arkansas and Louisiana. These data include 15 full-shift asbestos samples collected at fixed locations. The asbestos concentration levels are reported as either 0.004 f/cc [N=11] or 0.008 f/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 f/cc state “detection limit was 0.008 f/cc.”

EPA followed the same approach as noted above for non-detect observations, which in this case is replacing the observation by the limit of detection (LOD) divided by two. Therefore, for deriving exposure estimates, the 15 area samples were assigned numerical values of 0.002 f/cc [N=11] and 0.004 f/cc [N=4]. The central tendency ONU concentration used in EPA’s analysis was 0.0025 f/cc (*i.e.*, the arithmetic mean of the 15 data points), and the high end ONU concentration used in EPA’s analysis was <0.008 f/cc.

2.3.1.3.5 Exposure Results for Use in the Risk Evaluation for Asbestos: Part 1– Chlor-Alkali

Table 2-8 presents chrysotile asbestos exposure data that EPA used in this Part 1 of the risk evaluation for asbestos 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 Chrysotile Asbestos Exposures During Processing and Use in the Chlor-Alkali Industry Used in EPA’s Risk Evaluation for Asbestos Part 1: Chrysotile Asbestos

Occupational Exposure Scenario	Exposure Levels (f/cc)					
	Workers			ONUs		
	Central Tendency	High-end (95 th percentile)	Confidence Rating	Central Tendency	High-end	Confidence Rating
Producing, handling, and disposing of asbestos diaphragms: full-shift TWA exposure	0.0049	0.034	High	0.0025	0.008	Medium
Producing, handling, and disposing of asbestos diaphragms: short-term TWA exposure (30 mins)	0.024	0.512	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 chrysotile 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, 2020b](#)):

- Table 2-8 lists the full-shift TWA exposure levels that EPA used in this Part 1 of the risk evaluation for asbestos. The central tendency value for workers (0.0049 f/cc) is the median value of the full-shift exposure samples provided by Axiall-Westlake, Olin, and Occidental, and the high-end value (0.034 f/cc) is the calculated 95th percentile (see Table 2-5).
- For ONU exposure estimates area samples were used. Two chlor-alkali facilities provided a total of 15 area samples that were all below the limit of detection (LOD). There were two different detection limits in the two submissions. Central tendency exposure concentrations were calculated as the arithmetic mean of the individual observations, using one-half the detection limit for individual samples; and the high-end concentration is 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 (0.024 f/cc) is the median value of all 30-minute personal samples submitted. The high-end short-term TWA exposure value for workers (0.512 f/cc) is the calculated 95th percentile value for the compiled industry short-term exposure data. These values are based on all employee tasks combined. Refer to Table

2-7 for specific employee tasks (e.g., asbestos handling, filter press operation) with higher short-term exposure levels.

2.3.1.3.6 Data Assumptions, Uncertainties and Level of Confidence

The exposure data shown in Table 2-8 are based on monitoring results from the chlor-alkali industry. Worker exposure sampling data are available from all three companies (i.e., Axiall-Westlake, Occidental, Olin) that currently operate the entire inventory of chlor-alkali facilities nationwide and the overall confidence ratings from systematic review for these data were all rated high. Tables 2-4 through 2-7 summarize 759 individual exposure sampling results, which represent extensive coverage of the estimated 100 directly exposed workers. Each company submission of monitoring data includes a variety of worker activities. Therefore, this collection of monitoring data likely captures the variability in exposures across the different chlor-alkali sites and likely captures the variability in exposures during normal operations within a single site.

EPA notes the monitoring data cover all of the chlor-alkali companies that use chrysotile asbestos. However, it is uncertain if some infrequent and high-exposure activities are captured in this dataset, such as exposures when cleaning spilled asbestos within a container from damaged bags. The high-end estimates presented in the table 2-8 are applicable to an unknown fraction of the workers.

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 from multiple sites, which is the highest level of the inhalation exposure assessment approach hierarchy. One notable limitation is the considerable portion of non-detectable observations. EPA investigated different approaches to evaluating the non-detect observations (e.g., substitution with zero, substitution with the full detection limit) and continues to base its estimated concentrations on the non-detect substitution methods discussed earlier in this section.

Based on these strengths and limitations of the data, the overall confidence for EPA's assessment of occupational inhalation exposures for this scenario is high both for 8-hour and short-term durations.

For the ONU data, all of the area monitoring results showed non-detectable levels. In addition, it may be that ONUs may be exposed at less than a full shift, every workday. Overall, there is medium confidence for this set of data.

2.3.1.4 Sheet Gaskets

This section describes how chrysotile 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 ([EPA-HQ-OPPT-2016-0736-0119](#)). 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 0.6167 pounds per square foot ([ACC, 2018](#)). Branham employees stamp and cut gaskets to customer size specifications in a production area. Various other operations occur simultaneously at the Branham facilities to include stamping of non-asbestos gaskets using similar stamping machines. These other operations occur approximately 20 feet away from the stamping machines used to make asbestos-containing gaskets ([EHM, 2013](#)). As noted later in this section, EPA considers the workers supporting other nearby operations to be ONUs for this risk evaluation.

At the Branham facility visited by EPA, workers used three stamping machines to cut the imported asbestos-containing sheets into desired sizes. The facility reportedly does not saw gasket material ([Branham, 2018](#)), and EPA did not see evidence of this practice during its site visit. The stamping machines can be adjusted to make products of varying diameters, from 4 inches to 4 feet. Figure 2-5. shows a worker wearing a face mask while operating one of the stamping machines, which uses round

headed dies attached to a blade. Blades are not removed from the dies, and the dies are seldom “re-ruled” (where the rule blade would be pressed back into the wooden die frame).



Figure 2-5. Chrysotile Asbestos-Containing Stamping Operation
Photographs courtesy of Branham Corporation and used with Branham’s permission

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



Figure 2-6. Rule Blade for Stamping Machine
Photographs courtesy of Branham Corporation and used with Branham’s permission

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 sheet gasket material is not friable; they also noted that asbestos fibers are 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 PCM-detected 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 chrysotile 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, 2017b](#)):

- 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.

2.3.1.4.2 Worker Activities – Cutting of Asbestos-containing Sheet Gaskets

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

The amount of time that workers conduct cutting asbestos-containing sheets varies with production demand and other factors. EPA received one month of worker activity data for Branham’s Mississippi

facility, and these data indicated that, in May 2018, the worker spent no more than 70 minutes per day processing asbestos-containing gaskets ([Branham, 2018](#)). Branham informed EPA that the worker at the Kentucky facility perform asbestos-containing gasket stamping activity two to three days per week ([Branham, 2018](#)). The worker exposure levels from the Kentucky facility will be used in Part 1 because Branham officials informed EPA that they do not anticipate considerable increases or decreases in production demand for asbestos-containing sheet gaskets.

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

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

Branham operates two facilities that process asbestos-containing gaskets, with one worker at each facility who stamps the asbestos-containing sheet gaskets. During its site visit to one facility, EPA observed that stamping of asbestos-containing sheeting occurs in a 5,500 square foot open floor area. Other employees work in this open space, typically at least 20 feet away from where asbestos-containing gaskets are processed. EPA considers these other employees to be ONUs. The facility also included a fully-enclosed air-conditioned office space, where other employees worked; but those office workers were not considered to be ONUs.

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

These estimates are based on the one company known to stamp asbestos-containing sheet gaskets. It is unknown if other U.S. companies perform this same stamping activity. EPA attempted to identify other companies that cut/stamp asbestos-containing sheet gaskets in the United States but could not locate any. Therefore, based on reasonably available information, EPA concluded that there are no other additional facilities that cut or stamp imported asbestos-containing sheet gaskets.

2.3.1.4.4 Occupational Inhalation Exposure Results – Sheet Gasket Stamping

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

EPA considered the 2011 to 2016 nationwide exposure data provided by OSHA and the history of NIOSH HHEs. EPA also considered the published literature on asbestos exposures associated with sheet

gasket stamping. This search identified two studies that presented original worker exposure monitoring data. One was a 1998 study of sheet gasket production in Bulgaria ([Stroková et al., 1998](#)). The other was a 2000 publication as part of litigation support that examined exposures in a simulated work environment ([Fowler, 2000](#)).

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

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

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

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

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

Duration (minutes)	Result (f/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

2.3.1.4.5 Exposure Data for Use in the Risk Evaluation for Asbestos: Part 1 – Chrysotile Asbestos; Sheet Gasket Stamping

Table 2-10 presents the asbestos exposure data that EPA used in this Part 1 of the risk evaluation for asbestos 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. The following assumptions were made in compiling these data:

- The central tendency 8-hour TWA exposure value reported for workers (0.014 f/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) 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 f/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 f/cc) is the highest measured short-term exposure of the Branham worker. This exposure value occurred during a 30-minute sample ([EHM, 2013](#)).
- 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 is consistent with concentration reduction data reported in other studies in the peer-reviewed literature (*e.g.*, [Donovan et al., 2011](#)).

Table 2-10. Summary of Asbestos Exposures During Sheet Gasket Stamping Used in EPA’s Risk Evaluation for Asbestos Part 1: Chrysotile Asbestos

Occupational Exposure Scenario	Full-Shift Exposures (f/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

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 because it was the only study available that provided direct observations for chrysotile 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 level 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 chrysotile 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 chrysotile 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.

The ONU exposure estimate is less certain because no relevant ONU concentration estimates were available for the Branham facilities. EPA used a concentration reduction factor approach to fill this gap. As a result, the ONU exposure concentration estimate has greater uncertainty. In addition, ONUs may not be exposed at full shift, every workday.

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

2.3.1.5 Use of Gaskets in Chemical Production

2.3.1.5.1 Process Description – Sheet Gasket Use

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

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

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

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

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

Though Chemours has an active program to replace asbestos-containing gaskets with asbestos-free alternatives and this program has resulted in considerable decreases in asbestos-containing gasket use ([EPA-HQ-OPPT-2016-0736-0067](#)), gaskets formulated from non-friable chrysotile asbestos-containing sheeting continue to be the only product proven capable of withstanding certain extreme operating

conditions and therefore provide a greater degree of process safety and integrity than unproven alternatives according to industry ([ACC, 2017b](#)). A single titanium dioxide manufacturer can have approximately 4,000 gaskets of various sizes distributed throughout the plant which are periodically replaced during facility shutdowns.

2.3.1.5.2 Worker Activities – Sheet Gasket Use

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

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

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

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

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

2.3.1.5.4 Occupational Inhalation Exposures – Sheet Gasket Use

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

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

EPA also considered the published literature on worker asbestos exposure attributed to gasket removal. This search did not identify publications that specifically addressed asbestos-containing gasket use in the titanium dioxide manufacturing industry. However, two peer-reviewed publications measured worker 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 f/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 f/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](#)). Further, EPA compiled and reviewed a large number of additional studies that characterized worker exposures during gasket removal. These studies reported a broad range of worker asbestos exposure levels. However, EPA ultimately chose to base this COU's worker exposure estimates on data provided by industry, given that the one company known to use the chrysotile asbestos-containing sheet gaskets provided exposure data (through ACC) for its gasket servicing workers. EPA viewed these direct observations as most representative for this COU, rather than using surrogate values based on workers in other industries who may use different gasket removal practices.

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 f/cc, with an average of 0.026 f/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 the Risk Evaluation for Asbestos Part 1: Chrysotile Asbestos – Sheet Gasket Use

Table 2-11. presents the worker exposure concentrations that EPA is using in this Part 1 of the risk evaluation for asbestos for use of chrysotile 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 f/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 f/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 full-shift data points apply to short-term durations would understate the highest short-term exposures. That is because short-term data within a shift generally span a range of concentrations, and the corresponding full-shift concentration for that shift would fall within that range (and be lower than the highest short-term result). Therefore, EPA has determined that this COU has no reasonably available data for evaluating worker short-term exposures.
- EPA considered multiple options for estimating ONU exposure concentrations. First, EPA revisited existing data sources in an attempt to identify direct measurements; however, the data from facilities that stamp and use sheet gaskets do not have any information relevant to ONUs. Second, EPA considered assuming ONU exposures are the same as worker exposures. EPA did not pursue this option, given that ONU exposures are likely less than worker exposures for the gasket-related conditions of use (*i.e.*, EPA found no instances where ONUs are in very close proximity to process areas where asbestos-containing gaskets are removed). The third option was to derive ONU exposures based on a calculated “decay factor.” EPA is using this third approach to estimate ONU exposures. Specifically, the literature includes “bystander” exposure studies that EPA used to estimate ONU exposures. 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.

Table 2-11. Summary of Asbestos Exposures During Sheet Gasket Use Used in the Risk Evaluation for Asbestos Part 1: Chrysotile Asbestos

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

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 occupational settings and based on other worker practices. Moreover, the central tendency concentration shown in Table 2-11.1 falls within the range of results from the relevant literature that EPA reviewed, suggesting that the data source considered ([ACC, 2017a](#)) does not understate exposures.

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 level of the inhalation exposure assessment approach hierarchy. The overall confidence rating from systematic review for these data was rated medium. These monitoring data were provided to EPA by industry and represent actual measurements made during asbestos-containing sheet gasket removal at a titanium dioxide manufacturing facility in the United States. However, based on reasonably available information, EPA is not aware of any additional facilities that use asbestos-containing sheet gaskets, and EPA could not determine if the industry-provided monitoring data are representative of all U.S. facilities that use asbestos-containing sheet gaskets. The monitoring data were collected from 2009 through 2017; therefore, the data likely capture temporal variability in the facility’s operations.

The ONU exposure estimates are based on “decay factors” observed for gasket removal operations. These ONU estimates are therefore uncertain. The uncertainty cannot be reduced with the data currently available to EPA. In addition, ONU may not be exposed at full shift, every workday.

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

2.3.1.6 Oil Field Brake Blocks

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

2.3.1.6.1 Process Description – Oil Field Brake Blocks

The rotary drilling rig of an oil well uses a drawworks hoisting machine to raise and lower the traveling blocks during drilling. The drawworks is a permanently installed component of a mobile drilling rig package, which can be either “trailerized” or self-propelled. Therefore, there is no on-site assembly of

the drawworks. Except for initial fabrication and assembly prior to installation on a new rig, the drawworks is not set or installed in an enclosed building ([Popik, 2018](#)).

This section focuses on oil field operations involving asbestos-containing brake blocks. EPA acknowledges that many of today's rigs use electromagnetic braking systems that reportedly do not contain asbestos and some use braking systems that do not use friction pads during normal operation. However, a precise count of the rigs with and without asbestos-containing brakes is not available. The remainder of this section summarizes information EPA compiled on the operations involving the asbestos-containing brakes.

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

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

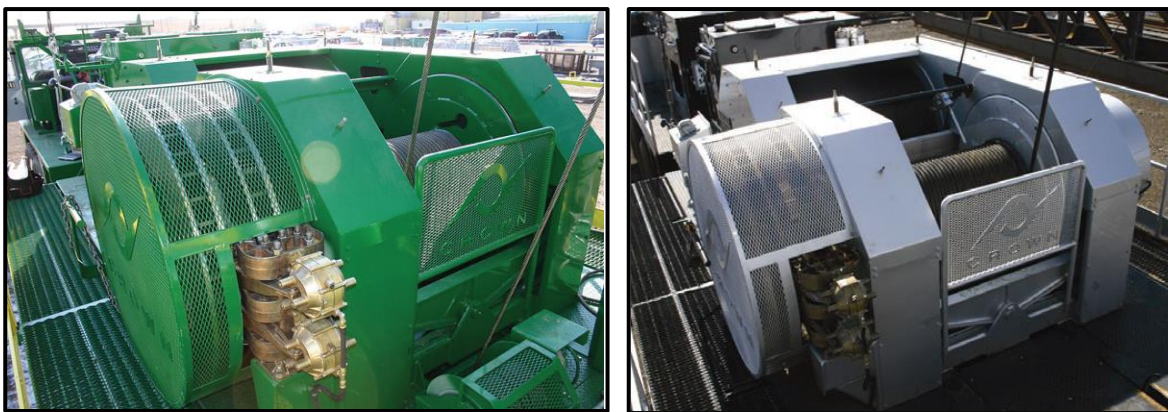


Figure 2-8. Photographs of Typical Oil Field Drawworks

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 chrysotile 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 containing 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 chrysotile 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. EPA notes that workers in the vicinity of brake replacement activity may be exposed due to brake block wear; and these workers were considered to be ONUs.

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

EPA identified one U.S. facility that imports chrysotile 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, for which extensive information is available to estimate numbers of potentially exposed workers, EPA found no direct accounts of the number of workers who use asbestos-containing oil field brake blocks. The lack of information necessitated the use of other established methods to estimate the number of potentially exposed workers.

To derive these estimates, 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 high-end estimates of the number of establishments and potentially exposed workers and ONUs. The actual number of potentially exposed workers and ONUs is likely lower than EPA's estimates.

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 Gas Operations	9,591	11,880	314,589	26	36,059	26,007	3	2
All NAICS		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

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

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

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

2.3.1.6.5 Exposure Results for Use in the Risk Evaluation for Asbestos: Part 1 Chrysotile Asbestos – Oil Field Brake Blocks

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

As noted previously, ONUs for this COU include workers in the vicinity of brake blocks, but whose job duties do not involve repair or servicing of the brake blocks, 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.

Table 2-13. Summary of Asbestos Exposures During Use in Brake Blocks for the Risk Evaluation for Asbestos Part 1: Chrysotile Asbestos

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

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 to 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. For this COU, ONU may not be exposed at full shift, every workday.

These are significant uncertainties in the assessment, but the uncertainties cannot be reduced through review of other available information. EPA is not aware of published accounts of worker exposure concentrations in the United States to chrysotile asbestos from oil field brake blocks.

EPA considered asbestos sampling data from hoist crane operations as a surrogate for this COU ([Spencer et al., 1999](#)), but ultimately believes the one study of brake blocks on an oil rig is more representative of this COU than measurements from a hoist crane in an industrial setting. EPA believes the values in Table 2-13 represent the best available information, but there is also reason to believe these values might overstate actual exposures.

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

2.3.1.7 Aftermarket Automotive Brakes/Linings and Clutches

The use of chrysotile 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

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](#)). Up through the 1990s many new automobiles manufactured in the United States had brake assemblies with asbestos-containing components. However, by 2000, asbestos was no longer used in the brakes of virtually all automobiles sold domestically ([Paustenbach et al., 2004](#)). 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](#)).

The two primary types of automobile brakes are drum brakes and disc brakes, and chrysotile asbestos has been found both in linings for drum brake assemblies and pads in disc brake assemblies (see Figure 2-9.). Drum brakes were more prevalent than disc brakes in older vehicles. When the vehicle operator engages drum brakes, the brake shoes (which contain friction materials) contact the rotating brake drum, and this contact slows the vehicle. Disc brakes are much more common today than drum brakes, and they function by applying brake pads (which contain friction materials) to the surface of the revolving brake disc, and this contact slows the vehicle. [Richter et al. \(2009\)](#) state that by the mid-1990s, material and design improvement led to most cars being manufactured with disc brakes, effectively phasing out drum brakes in passenger automobiles. However, further investigation online by EPA into the use of disc/drum brakes indicate that while front brakes appear to mostly have been converted to disc brakes in front wheel drive vehicles, many passenger vehicles have a combination of disc brakes for the front wheels and drum brakes for the rear wheels.

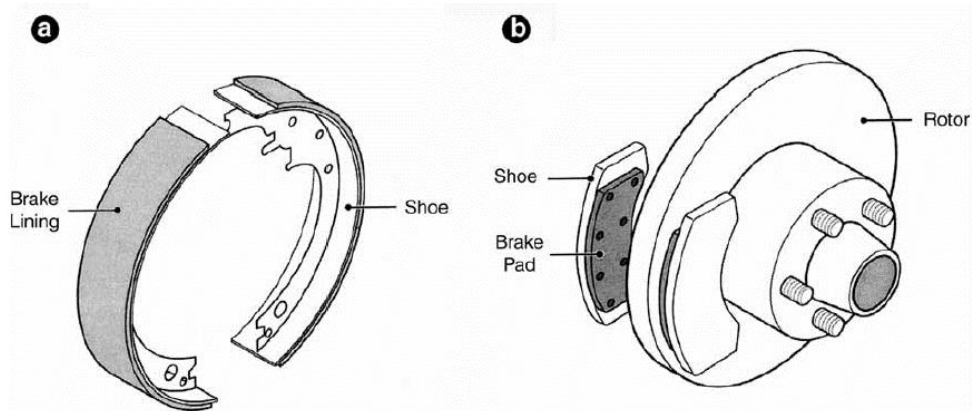


Figure 2-9. Illustrations of brake assembly components: (a) a brake lining designed to be used with an internal drum brake and (b) a brake pad designed for use with a disc brake.
 Source: [Paustenbach et al. \(2004\)](#).

Use of asbestos-containing braking systems began to decline in the 1970s due to many factors, including toxicity concerns, rising insurance costs, regulatory scrutiny, challenges associated with disposing of asbestos-containing waste, and availability of asbestos-free substitutes ([Paustenbach et al., 2004](#)). In 1989, EPA issued a final rule that banned the manufacturing and importing of many asbestos-containing products, including automobile brake pads and linings (54 FR 29460). While the United States Court of Appeals for the Fifth Circuit vacated most of this ban¹⁷ in 1991, many manufacturers had already begun to phase out asbestos-containing materials and develop alternatives, including the non-asbestos organic fibers that are almost universally used in automobile brake assemblies today ([Paustenbach et al., 2004](#)). By 2000, domestic manufacturers had eliminated asbestos from virtually all brake assemblies in automobiles ([Paustenbach et al., 2004](#)). EPA is not aware of any automobile manufacturers that currently use asbestos products in brake assemblies for U.S. vehicles. In fact, the Agency received verification from five major vehicle manufacturers that asbestos-containing automotive parts are no longer used and import data has been misreported under the wrong Harmonized Tariff Schedule (HTS) code. However, the Agency knows of at least one company that imports asbestos-containing friction products for use in cars assembled in the U.S., but those vehicles are exported for sale and are not sold domestically. The COU identified for this scenario is specified as “other vehicle friction products” in Table 1-34, and the exposure values are based on aftermarket auto brakes (see Section 2.3.1.8).

The history of asbestos in aftermarket brake products has followed a similar pattern. For decades, asbestos was found in various aftermarket brake replacement parts (*e.g.*, pads, linings, and shoes); but the same factors listed in the previous paragraph led to a significant decline in the use of asbestos in aftermarket vehicle friction products. Nonetheless, the literature indicates that asbestos-containing replacement brake materials continued to be available from parts suppliers into the 2000s; researchers were able to purchase these materials in 2008 from a vintage auto parts facility ([Madl et al., 2008](#)). Today, individual consumers can find aftermarket automotive products marketed as containing asbestos through online retailers.

In more recent years, state laws and regulations have limited sales of asbestos-containing aftermarket brake parts, even among existing stockpiles. In 2010, for instance, the state of Washington passed its “Better Brakes Law,” which prohibits manufacturers, retailers, wholesalers, and distributors from selling brake friction material that contains more than 0.1 percent asbestiform fibers ([Washington State, 2010](#)).

¹⁷ Federal Register notice - <https://www.govinfo.gov/content/pkg/FR-1994-06-28/html/94-15676.htm>

In the same year, the state of California passed legislation with similar requirements. The not-to-exceed limit of 0.1 percent asbestiform fibers in aftermarket brake parts now essentially extends nationwide, due to a memorandum of understanding between EPA and multiple industry stakeholders (e.g., Motor and Equipment Manufacturers Association, Automotive Aftermarket Suppliers Association, Brake Manufacturers Council) ([U.S. EPA, 2015a](#)).

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

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

Automobile Clutches

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

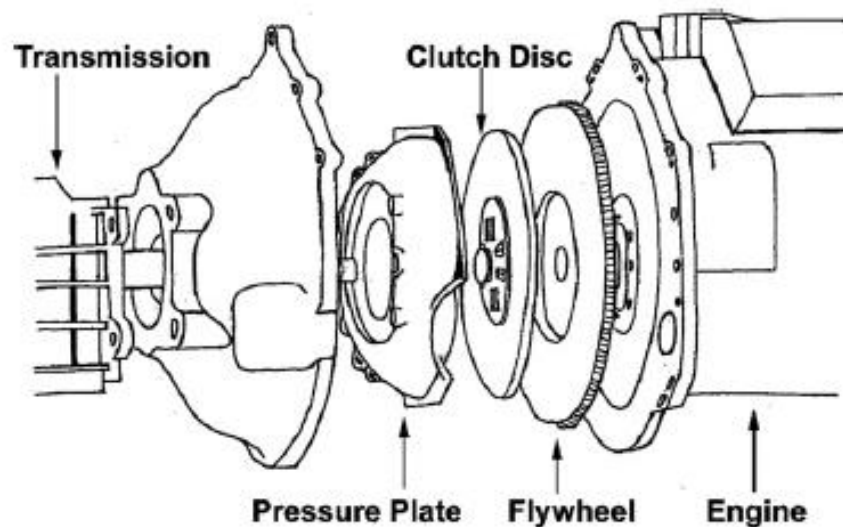


Figure 2-10. Schematic of a clutch assembly. The clutch disc is made of friction material, which may contain asbestos.

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

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](#)).

EPA is not aware of any car manufacturers that currently import asbestos-containing clutch assemblies. 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.

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.

2.3.1.7.2 Worker Activities – Aftermarket Automotive Brakes/Linings and Clutches

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.

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 move to locations throughout the workplace.

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. Additionally, OSHA amended its asbestos standards in 1994. Major revisions in these standards included a reduced time-weighted-average permissible exposure limit (PEL) of 0.1 fiber per cubic centimeter (f/cc) for all asbestos work in all industries, a new classification scheme for asbestos construction and shipyard industry work which ties mandatory work practices to work classification, a presumptive asbestos identification requirement for “high hazard” asbestos containing building materials, limited notification requirements for employers

who use unlisted compliance methods in high risk asbestos abatement work, and mandatory methods of control for brake and clutch repair (Federal Register, 1994). The requirements specific to brake and clutch repair are in Appendix F of the general industry standard (see: <https://www.osha.gov/laws-regs/regulations/standardnumber/1910/1910.1001AppF>). The updated standards are 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](#)). Chemical changes also occur, such as transformation into forsterite (a deformation product of chrysotile), or to transition series fibers (chrysotile/forsterite), but chemical changes are thought as less important than physical changes for biological outcomes ([OSHA, 2006](#)).

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](#)).

However, clutch repair and replacement differ from brake work in two important ways. First, clutches generally do not need to be repaired as frequently. By estimates made in 2008, clutches typically last three times longer than brake linings ([Cohen and Van Orden, 2008](#)). Second, a common clutch repair method is to remove and replace the entire clutch assembly, rather than replacing the clutch disc component ([Cohen and Van Orden, 2008](#)). Finally, vehicles have only one clutch assembly and up to four brakes; therefore, clutch servicing only involves repair of one apparatus, while brake servicing involves multiple components. These three and other factors likely result in clutch repair asbestos exposures being lower than comparable brake repair asbestos exposures.

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

EPA considered several data sources when estimating the number of workers directly exposed to asbestos when working with aftermarket automotive products. In the late 1980s, NIOSH conducted a series of industrial hygiene surveys on brake repair facilities, and the Agency estimated that 155,000 brake mechanics and garage workers in the United States were potentially exposed to asbestos ([OSHA, 2006](#)). In 1994, OSHA estimated as part of its updated asbestos rulemaking that 676,000 workers performed automotive repair activities, and these workers were found in 329,000 establishments (*i.e.*, approximately two workers per establishment) (59 FR 40964). Additionally the Bureau of Labor Statistics estimated that 749,900 workers in the United States were employed as automotive service technicians and mechanics in 2016 ([U.S. BLS, 2019](#)). 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, EPA assumes that automotive repair establishments, on average, have two workers who perform automotive repair activities and therefore EPA assumes an equal number of exposed workers and ONUs for this COU.

EPA estimated the number of potential individuals exposed to asbestos using the limited available information on the potential market share of asbestos brakes. Details are provided in Section 4.3.7: Confidence in the Human Health Risk Estimations. EPA assumes that asbestos brakes may represent only approximately 0.05% of aftermarket automotive brakes. By applying this factor (0.05%) to the universe of automotive service technicians and mechanics (749,900), EPA's estimate of potentially exposed workers is 375. For the same reasons noted above, EPA assumes an equal number (375) of ONUs for this COU.

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

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

Automobile Brake Repair and Replacement

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

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 f/cc). The other two studies reported average (arithmetic mean) asbestos concentrations over the brake job duration of 0.006 f/cc and 0.007 f/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. While EPA is aware of and thoroughly reviewed studies of asbestos exposure among brake mechanics in various foreign countries (*e.g.*, Australia, Colombia, Iran, Norway), EPA focused on U.S. business establishments due to the availability of measurements and the fact that OSHA’s asbestos standard mandates controls and other safe work practices that do not apply in other countries. Further, the profile of brakes encountered in U.S. vehicles differs from what is seen in other countries.

The following peer-reviewed publications met EPA’s selection criteria (and all were given a high rating in the data evaluation; see supplemental file [\(U.S. EPA, 2020f\)](#)):

- 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 f/cc to 0.4368 f/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. While arc grinding during brake replacement is not believed to be a common practice today, EPA conducted web searches that identified recent videos showing individuals using arc

grinding during brake repair. Given the evidence of the ongoing activity, even if uncommon, EPA retained this study in the exposure assessment.

- 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 f/cc to 0.836 f/cc, with the highest exposure associated with unpacking and packing 16 boxes of asbestos-containing brake pads over approximately 30 minutes. EPA acknowledges that this study did not characterize actual brake repair or servicing activities. However, workers must handle aftermarket parts (*i.e.*, open and close boxes) as part of their overall repair jobs. For this reason, EPA continued to include this study in its estimates of worker and ONU exposures.
- 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 for this COU) 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 personal breathing zone asbestos concentrations while repairing or replacing asbestos-containing clutches are comparable to the concentrations for brake repair and replacement activity. However, the frequency of workers performing this task is expected to be lower than the brake. 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 even though it is clear that the brake-related exposure concentrations may overstate exposures that occur during clutch repair.

2.3.1.7.5 Exposure Data for Use in the Risk Evaluation for Asbestos Part 1: Chrysotile Asbestos – Aftermarket Auto Brakes/Linings and Clutches

Table 2-15. presents the asbestos exposure data that EPA used in this Part 1 of the risk evaluation for asbestos 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 the Risk Evaluation for Asbestos Part 1: Chrysotile Asbestos

Occupational Exposure Scenario	Exposure Levels (f/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	0.001	0.002	Medium
Repairing or replacing brakes with asbestos-containing aftermarket automotive parts: short-term exposure	0.006	0.836	Medium	0.001	0.002	Medium

Worker Exposures

- 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 f/cc) is the median of those central tendencies from those seven studies. Thus, three of the studies reported central tendency concentrations lower than 0.006 f/cc, one reported a central tendency concentration of 0.006 f/cc, and the other three studies reported higher exposure concentrations.
- 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 f/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 f/cc) is the highest short-term personal breathing zone observation among the seven studies that met the review criteria ([Madl et al., 2008](#)). The highest concentration was from a 15-minute average sample and therefore might overstate (by no more than a factor of two) the 30-minute concentration. The high-end 8-hour exposure value for workers (0.094 f/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.

ONU Exposures

EPA used area sampling results from the five NIOSH studies cited above to derive ONU exposure estimates for this condition of use. In each study, NIOSH collected area samples at the fender and at the axle of the vehicle as its brakes were being serviced. EPA considered these area samples to be representative of ONU exposures, because other workers may conduct other tasks at these locations during brake servicing. The duration of the area sample was the time needed to replace a vehicle’s

brakes or two hours, whichever was longer. Across the five studies, more than 70 area samples were collected at these locations. The area samples were tested for asbestos using PCM, and all were non-detect. NIOSH reported arithmetic mean concentrations for these samples as <0.002 f/cc. Based on these data, EPA assumed the ONU central tendency exposure concentration to equal one-half the detection limit, or 0.001 f/cc; and EPA assumed the ONU high-end exposure concentration to equal the detection limit for most samples, or 0.002 f/cc. These values were applied to both 8-hour TWA exposure and short-term exposure. It is possible that ONU may not be exposed at full shift, every workday.

2.3.1.7.6 Data Assumptions, Uncertainties and Level of Confidence

The universe of automotive repair establishments in the United States is expected to have large variability in the determinants of exposure to asbestos during brake repair. These exposure determinants include, but are not limited to, vehicle age, type of brake assembly (disc vs. drum), asbestos content of used and replacement parts, dust control measures used, brake servicing techniques (*e.g.*, use of arc grinding), number of vehicles serviced per day, and duration of individual repair jobs. It is uncertain if the studies EPA cited and used fully capture the distribution of determinants of exposure of current automotive brake jobs, and some of the studies reviewed for this Part 1 of the risk evaluation for asbestos are based on practices that are not widely used today.

PCM-based personal exposure measurement in an automotive repair facility may overstate asbestos exposures, which some studies have demonstrated through TEM analyses of filter samples ([Blake et al., 2003](#); [Weir et al., 2001](#)). PCM measurements are based entirely on dimensional criteria and do not confirm the presence of asbestos, as can be done through supplemental analyses by TEM or another confirmatory method. Automotive repair facilities involve many machining operations that can release non-asbestos airborne fibers, such as cellulose fibers from brushes and metal and plastic fragments from body repair ([Blake et al., 2008](#)).

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 level of the inhalation exposure assessment approach hierarchy. The overall confidence ratings from systematic review for these data were high. The monitoring data were all collected from U.S.-based vehicle maintenance and repair shops. While these studies were conducted after the implementation of the OSHA rule, many of the studies were conducted in the late 1980s and may not be representative of current operations. This is particularly true for the study that evaluated arc grinding. However, that study's results are directly reflected only in the high-end exposure estimate. (Note: The central tendency value in the table comes from one of the NIOSH studies.) EPA believes it is appropriate to consider arc grinding in the high-end exposure category, given evidence that this work practice continues today, albeit uncommonly.

The ONU exposure estimates are based on a dataset comprised entirely of non-detect observations and therefore are uncertain. The uncertainty cannot be reduced using other sampling results that EPA considered for this analysis. EPA assigns a "medium" confidence factor for these exposure concentration estimates.

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

2.3.1.8 Other Vehicle Friction Products

While EPA has verified that U.S. automotive manufacturers are not installing asbestos brakes on new cars for domestic distribution, EPA has identified a company that is importing asbestos-containing brakes and installing them in their cars in the United States. These cars are exported and not sold domestically. In addition, there is a limited use of asbestos-containing brakes for a special, large transport plane (the “Super-Guppy”) by the National Aeronautics and Space Administration (NASA).

2.3.1.8.1 Installing New Brakes on New Cars for Export Only

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

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

Table 2-16. Other Vehicle Friction Products Exposure Levels (from Aftermarket Automotive Parts exposure levels) Used in the Risk Evaluation for Asbestos Part 1: Chrysotile Asbestos

Occupational Exposure Scenario	Exposure Levels (f/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	0.001	0.002	Low
Installing brakes with asbestos-containing automotive parts: short-term exposure	0.006	0.836	Low	0.001	0.002	Low

Data Assumptions, Uncertainties and Level of Confidence

The assumptions and uncertainties described above under Section 2.3.1.7.6 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, although we do not have the data, the actual exposure could be lower than estimated here. For this COU, ONU may not be exposed at full shift, every workday.

Based on these strengths and limitations of the underlying data described above and in Section 2.3.1.7.6, the overall confidence for EPA’s assessment of occupational and ONU 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)

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. This section is based on information provided by NASA.



Figure 2-11. NASA Super Guppy Turbine Aircraft

Photograph courtesy of NASA

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

The SGT aircraft has eight landing gear systems, and each system has 32 brake blocks. The individual blocks (Figure 2-12) contain 43 percent chrysotile asbestos; and they are 4 inches long, 4 inches wide, and 1 inch thick ([NASA, 2020b](#)). Each brake block weighs approximately 12.5 ounces.



Figure 2-12. Brakes for NASA Super Guppy Turbine Aircraft
Photograph courtesy of NASA

Worker Activities

Replacing asbestos-containing brake blocks is the principal worker activity potentially associated with asbestos exposure, and this task is performed by four certified technicians. According to NASA, the brake blocks are not replaced due to excessive wear; rather, they are typically replaced because they have become separated from the brake system or because they have become covered with hydraulic fluid or other substances ([NASA, 2020a](#)). This is an important observation, because in EPA's judgment, worn brake blocks would be more likely to contain dusts to which workers would be exposed.

In materials provided to EPA, NASA described the process by which workers replace brake blocks. This process begins by removing the brakes from the landing gear. To do so, the SGT aircraft is raised at the axle pads, and the landing gear is opened to allow workers access to the individual brake systems. The workers remove the brakes from the aircraft and clean the brakes at an outdoor wash facility.

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



Figure 2-13. Ventilated Walk-in Booth Where Brakes Pads Are Replaced
Photograph courtesy of NASA

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

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

Number of Sites and Potentially Exposed Workers

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

Over the course of a year, only four certified technicians at this location perform brake pad replacement; and one or two of these technicians will perform individual brake pad replacements ([NASA, 2020b](#)). Because the brake replacement work occurs in a ventilated walk-in booth, asbestos fibers likely are not released into the general workspace where ONUs may be exposed.

Therefore, for this condition of use, EPA assumes four workers may be exposed, and no ONUs are exposed.

Worker Inhalation Exposures

EPA's estimate of occupational inhalation exposures for this condition of use are based on five worker exposure samples that NASA collected in 2014 ([NASA, \(020a\)](#)). The sampling was conducted according to NIOSH Method 7400, and asbestos was not found above the detection limit in any of the samples. 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, based on sampling durations of 83, 17, and 85 minutes, were: <0.003 f/cc, <0.006 f/cc, and <0.0089 f/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 values. By this approach, EPA calculated a central tendency concentration of <0.003 f/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 f/cc and <0.045 f/cc. Following the same approach that was used for full shift exposures, EPA estimated a central tendency short-term exposure of <0.022 f/cc and a high-end short-term exposure of <0.045 f/cc.
- According to NASA (NASA, 2020c), records from a recent 36-month period indicate 3.6 brakes were changed each year with an average time of 3.3 hours per brake change.

Based on these assumptions, EPA will use the exposure values in Table 2-17.

Table 2-17. Summary of Asbestos Exposures During Replacement of Brake Pads/Blocks in the NASA Super Guppy Used in the Risk Evaluation for Asbestos Part 1: Chrysotile Asbestos

Occupational Exposure Scenario	Exposure Levels (f/cc)					
	Workers			ONUs		
	Central Tendency	High-end	Confidence Rating	Central Tendency	High-end	Confidence Rating
Replacing brake pads: 8-hour TWA exposure	<0.003	<0.0089	High	Not expected		High
Replacing brake pads: short-term exposure (30 minutes)	<0.022	<0.045	High			

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 Part 1 of the risk evaluation for asbestos.

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)

2.3.1.9.1 Process Description – UTV Gasket installation/Serviceing

EPA has identified the use of chrysotile 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 Part 1 of the risk evaluation for asbestos, 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.

2.3.1.9.2 Worker Activities – UTV Gasket Installation/Serviceing

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.

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).

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

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.¹⁸ However, no NAICS codes are specific to UTV dealers. These establishments are classified under the 4-digit NAICS 4412, “Other Motor Vehicle Dealers.” Table 2-188. 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.

Table 2-18. Number of Other Motor Vehicle Dealers

2012 NAICS code	2012 NAICS Code Description	Number of Establishments
4412	Other motor vehicle dealers	14,249
44121	Recreational vehicle dealers	2,605
441222	Boat dealers	4,645
441228	<i>Motorcycle, ATV, and all other motor vehicle dealers</i>	6,999
4412281	Motorcycle, ATV, and personal watercraft dealers	5,098
4412282	All other motor vehicle dealers	1,901

Source: ([U.S. Census Bureau, 2016a](#)).

¹⁸ 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.

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 2-199. 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) sell and repair UTVs and ATVs, and that the other half specialize in personal watercraft.

Table 2-19. 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: (U.S. Census Bureau, 2016b).				

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 4412.¹⁹

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
Recreational Vehicle Service Technicians (493092)	11,260
Total	41,930
Source: (U.S. BLS, 2019).	

Based on the estimates for NAICS 4412 in Table 2-18. 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.

¹⁹ This count excludes occupations in NAICS 4412 that are less likely to engage in engine repair involving gaskets similar to those found in UTVs. These would be 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.

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

Description	Number
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
Estimated average number of employees per establishment that may repair motor vehicle engines (calculated as 41,930 divided by 14,429)	~3 employees per establishment

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 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	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 UTVs	4,500

2.3.1.9.4 Occupational Inhalation Exposures for Use in the Risk Evaluation for Asbestos Part 1: Chrysotile Asbestos for UTV Gasket Installation/Service

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.

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 according to the study ([Paustenbach et al., 2006](#)), workers reportedly spent less than one minute for removal of each gasket. It often took the worker more time to access the gasket due to rusted

bolts than to remove the gasket. Workers reportedly spent less than one minute on the removal of each gasket. It often took the worker more time to access the gasket due to rusted bolts than to remove the gasket. 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 f/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 f/cc and a maximum concentration of 0.066 f/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 f/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).

ONU Exposures

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

Table 2-23. UTV Gasket Installation/Service Exposure Levels for the Risk Evaluation for Asbestos Part 1: Chrysotile Asbestos

Occupational Exposure Scenario	8-hr TWA Exposure Levels (f/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

2.3.1.9.5 Data Assumptions, Uncertainties and Level of Confidence

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

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

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

Additionally, EPA identified two sources of uncertainty pertaining to the data analysis. One pertains to the uncertainties associated with non-detect observations. For the average worker exposure concentration, 74 percent of the samples were non-detects; and the study authors replaced these observations with one-half the detection limit when calculating average concentrations. Similarly, for the area sampling results used for ONU exposures, 76 percent of the samples were non-detects.

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

It is possible that ONUs may not be exposed at a full shift, every workday. Based on these strengths and limitations of the data, the overall confidence for EPA's assessment of occupational and ONU inhalation exposures for this scenario is medium.

2.3.1.10 Summary of Inhalation Occupational Exposure Assessment

Table 2-24. summarizes the inhalation exposure estimates for all COUs that EPA evaluated. Where statistics can be calculated, the central tendency estimate represents the 50th percentile exposure level of the available data set, and the high-end estimate represents the 95th percentile exposure level. The central tendency and high-end exposures for ONU are derived separately from workers, often by using either a reduction factor or the analytical limit of detection. See the footnotes for an explanation of the concentrations used for each COU.

Table 2-24. Summary of Occupational Inhalation Exposures

Condition of Use	Duration	Type	TWA Exposures, f/cc (see footnotes)		
			Central Tendency	High-end	Confidence Rating
Diaphragms for Chlor-Alkali Industry (Processing and Use)	Full Shift	Worker	0.0049 (a)	0.034 (a)	High
		ONU	0.0025 (b)	0.008 (b)	High
	Short-term	Worker	0.024 (a)	0.512 (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	0.001 (h)	0.002 (h)	Medium
	Short-term	Worker	0.006 (g)	0.836 (g)	Medium
		ONU	0.001 (h)	0.002 (h)	Medium
Other Vehicle Friction Products (brakes installed in exported cars) (Use)	Full Shift	Worker	0.006 (g)	0.094 (g)	Medium
		ONU	0.001 (h)	0.002 (h)	Medium
	Short-term	Worker	0.006 (g)	0.836 (g)	Medium
		ONU	0.001 (h)	0.002 (h)	Medium
Replacing brake pads in NASA Super Guppy	Full Shift	Worker	<0.003	<0.0089	High
		ONU	Not Expected	Not Expected	High
	Short-Term	Worker	<0.022	0.045	High
		ONU	Not Expected	Not Expected	High
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	-

- (a) Full-shift exposure concentrations for the chlor-alkali industry are based on worker exposure monitoring data. Central tendency concentrations are 50th percentile values and high-end concentrations are 95th percentile values. 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 (0.024 f/cc) is the median value of all 30-minute personal samples submitted. The high-end short-term TWA exposure value for workers (0.512 f/cc) is the calculated 95th percentile value for the compiled industry short-term exposure data.
- (b) ONU exposure concentrations for the chlor-alkali industry are based on area monitoring data with all samples being non-detect observations that were replaced with surrogate values. Central tendency exposure concentrations were calculated as the arithmetic mean of the individual observations, using one-half the detection limit for individual samples; and the high-end concentration is the highest detection limit provided.
- (c) Concentrations for sheet gasket stampers are based on worker exposure monitoring data (10 samples). Central tendency is the single full-shift TWA data point available; and high-end assumes the highest observed short-term exposure persists over an entire shift. For short-term exposures, central tendency is the median concentration observed, and high-end is the highest concentration observed.
- (d) Concentrations for ONUs at sheet gasket stamping facilities and sheet gasket use facilities were estimated by EPA using a concentration-decay factor for bystander exposures derived from the literature.
- (e) Concentrations for sheet gasket use are based on descriptive statistics provided to EPA of 34 worker exposure monitoring samples. The central tendency concentration is the arithmetic mean and the high-end concentration is the highest measured value.
- (f) Concentrations for oil field brake blocks are based on two data points—arithmetic mean exposure for different worker activities—reported in the scientific literature.
- (g) Concentrations for aftermarket automotive parts are based on worker exposure monitoring data documented in seven studies. For full shift exposures, the central tendency concentration is the median of the arithmetic mean exposure values reported across the seven studies; and the high-end concentration is the highest TWA exposure concentration reported. For short-term exposures, the same data set was used but data were summarized for individual observations, not the full-shift TWA values.
- (h) Concentrations for ONUs at auto repair facilities were estimated using more than 70 area samples that NIOSH collected at bystander sampling locations.
- (i) Asbestos air measurements from (Paustenbach et al., [2006](#)): Removal and replacement of exhaust system gaskets from vehicles manufactured before 1974 with original and old exhaust systems.

2.3.2 Consumer Exposures

This section summarizes the data used for estimating consumer inhalation exposures to chrysotile asbestos for two potential do-it-yourself (DIY) scenarios: (1) automobile brake repair/replacement and (2) gasket repair/replacement in Utility Vehicles (UTVs). Specifically, the brake repair/replacement scenario involves repair or installation of imported aftermarket automobile brake pads (disc brakes) or brake shoes (drum brakes) containing asbestos. The gasket repair/replacement in the UTV scenario involves removal or installation of aftermarket gaskets for UTV exhaust systems containing asbestos. In response to peer review and public comments received on the draft Risk Evaluation, EPA recognizes brake repair/replacement work and gasket repair/replacement work may occur on other vehicle types (*i.e.*, motorcycles, snowmobiles, tractors). However, EPA did not identify or receive data which could either inform exposures during such “other vehicle” repair/replacement activities or inform methodology to extrapolate from automobile specific data to such “other vehicle” activities. Therefore, EPA did not evaluate other vehicle repair/replacement activities. Additionally, EPA recognizes exposure to bystanders may occur via take-home/take-in (from garage) exposures, depending on personal hygiene practices of a DIY consumer, however, EPA did not identify or receive data which could inform exposures or methodologies to extract such data. Therefore, EPA did not evaluate take-home/take-in (from garage) exposures to chrysotile asbestos for either brake repair/replacement work or gasket repair/replacement work.

Inhalation exposures are evaluated for both automobile brake repair/replacement and UTV exhaust system gasket repair/replacement activities for the individual doing the repair/replacement work and a

potential bystander observing the work within the immediate area. For each scenario, it is assumed that consumers and bystanders will not be wearing any personal protective equipment.

Dermal exposures are not assessed for consumers in this Part 1 of the risk evaluation for asbestos. The basis for excluding this route is the expected state of asbestos being only solid/fiber phase. While asbestos may deposit on open/unprotected skin, it will not absorb into the body through the protective outer skin layers. Therefore, a dermal dose resulting from dermal exposure is not expected.

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

EPA has found no reasonably available information to suggest that chrysotile asbestos-containing brakes are manufactured in the United States, and based on stakeholder outreach, the Agency does not believe that any domestic car manufacturer installs asbestos-containing brakes in new cars sold domestically.²⁰ However, general online searches have indicated brakes and gaskets identified to contain asbestos are available for consumers to purchase as aftermarket replacement parts for cars and UTVs. EPA recognizes that while an aftermarket product may be labelled to contain asbestos (in particular products manufactured outside the United States) such labelling is not a guarantee the product actually contains asbestos. Similarly, it should be recognized that even though a product is not labelled to contain asbestos (in particular products manufactured outside the United States) such products may contain asbestos but have no requirement to label as containing asbestos. Based on these possibilities, and to ensure potential exposure to asbestos during brake repair/replacement activities or UTV exhaust system gasket repair/replacement activities is adequately evaluated, EPA assumes DIY consumers do these repair/replacement activities with aftermarket products containing asbestos. This assumption does have some uncertainties which are discussed in the uncertainties section.

The number of consumers impacted by these COUs is unknown because EPA did not identify or receive data which can inform the actual number of individuals doing DIY repair/replacement activities (including potential shade mechanics²¹ or consumers working on more than one car), the actual number of those doing the repair/replacement activities with products containing asbestos, and the actual number of products which contain asbestos purchased for consumer use. This is discussed in more detail in the uncertainties section (Section 4.3.7: Confidence in the Human Health Risk Estimations).

²⁰ 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.

²¹ A term used for hobbyist mechanics; or one who works on their own vehicle.

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

This consumer assessment addresses potential scenarios in which a DIY consumer installs, repairs or replaces existing automobile brakes with imported aftermarket brake pads or shoes which may contain asbestos; including brake linings. While peer-reviewed literature indicates much of the asbestos brake pad or shoe use has been phased out and the majority of existing cars on the road do not have asbestos brakes ([Finley et al., 2007](#)), asbestos-containing brakes and shoes can still be purchased in the United States and from sources outside the United States. This scenario evaluates potential consumer inhalation exposure to asbestos during removal of the old brakes or shoes containing asbestos, cleaning of the brake housing, shoes, and wheel assembly, as well as installation and grinding of the newly installed brakes or shoes containing asbestos. While grinding of brakes or shoes may not be common to all DIY consumers, there is readily available grinding equipment which consumers can purchase for DIY projects which fit in a residential garage. Additionally, certain DIY consumers (in particular classic cars hobbyists but also others) may be required to grind brakes or shoes in order for the aftermarket product to properly fit the brake assembly. Considering these possibilities, EPA includes grinding activity as part of its evaluation of asbestos exposure to the DIY consumer during brake repair/replacement activities while acknowledging the associated uncertainties (discussed in Section 2.3.2.1.4 Data Assumptions, Uncertainties and Level of Confidence).

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

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

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

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

- Location: EPA presents an indoor and an outdoor scenario for brake repair and replacement work. The indoor scenario assumes the DIY brake repair/replacement is performed in the consumer's residential garage with the garage door closed. It also assumes the additional work associated with this brake work is arc grinding and occurs within the garage with the garage door closed. The outdoor scenario assumes the DIY brake repair/replacement work is performed in the consumer's residential driveway. It also assumes the additional work associated with this brake work is brake filing and occurs in the residential driveway.
- Duration of Activity: Available literature indicates a typical "brake job" for a professional brake mechanic for a single vehicle takes between one and two hours ([Paustenbach et al., 2003](#)). No data were found in existing literature on the length of time needed for a DIY consumer to perform a brake job. EPA assumes a consumer DIY brake repair/replacement event could take twice as long as a professional mechanic, or about three hours (double the mean of time found in the literature for professional mechanics).
- 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 for this evaluation. 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](#)). There are several factors which can affect this assumption which are discussed in Section 2.3.2.1.4 Data Assumptions, Uncertainties and Level of Confidence but include driving patterns, driving frequency, distances driven, a DIY consumer is a shade tree mechanic, owns and works on more than one car within a family, and works on vintage cars.

- **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)
Sheehy et al. (1989)	Yes	Yes	Medium (1.7)
Blake et al. (2003)	Yes	No	Medium (1.8)
Paustenbach et al. (2003)	Yes	No	High (1.0)
Yeung et al. (1999)	Yes	No	Medium (2.0)
Kakooei et al. (2011)	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 historical DIY consumer practices (including use of compressed air and other cleaning practices along with potential grinding activities) and 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 driveway, 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 m³) 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 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 (f/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 ^a	0.007	Not monitored ^b	Outdoors	Medium

^a No monitoring duration was provided within the study.

^b This study did not include outdoor area monitoring which could be applied to the bystander

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.

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 outdoor²².

DIY Consumer User

Indoor Scenario

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 Part 1 of the risk evaluation for asbestos, 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 measured during brake repair/replacement activities only (no additional work like grinding/filing) and do include the use of compressed air. However, compressed air was only used to blow out residual dust from brake drums after the majority of residual dust is broken out by placing the brakes on the floor with a shock to knock off loose material. While the use of compressed air is not a recommended practice, no reasonably available information was found that surveyed actual cleaning methods used or preferred by DIY consumers for this scenario. Additionally, compressed air systems (either cans or mechanical air compressors) are readily available and used by consumers for multiple DIY activities. EPA therefore utilized these values to evaluate consumer inhalation exposure with the understanding that they may represent a more conservative exposure concentration value.

Outdoor Scenario

EPA utilized the personal breathing zone concentration from the [Blake et al. \(2003\)](#) study obtained during filing of brakes for the high-end exposure concentration for the consumer user under the outdoor scenario. Although this value was obtained in an indoor environment it is a potential additional work activity that could also be performed outside and more readily expected to occur outdoors than arc grinding. Additionally, it is expected that filing of brakes would place a consumer's personal breathing zone very close to the brakes being filed. Such close proximity is expected to minimize potential impact of the higher air exchange rates and outdoor volumes on exposure to asbestos in the personal breathing zone and therefore using the indoor measurements for an outdoor scenario is a feasible exposure condition.

EPA used the average monitored outdoor concentration measured in the personal breathing zone from the [Sheehy et al. \(1989\)](#) study to represent the central tendency value for the consumer user under the outdoor scenario. The [Sheehy et al. \(1989\)](#) study is the only study identified through the systematic review process which included PBZ monitoring data for a DIY consumer user during outdoor brake repair/replacement work. The duration of the monitoring in [Sheehy et al. \(1989\)](#) was not specified for the outdoor work, EPA assumes monitoring occurred for the entire expected duration for the DIY consumer user to complete the work. As the study describes, the DIY consumer user utilized various

²² Although exposures would be very low and are not quantified here, an assumption is made in Section 4.2.3.1 to allow for cancer risk estimation for bystanders from outdoor brake replacement.

wetting techniques on the brakes to clean grease, dirt, and flush the surface of the drums. Considering these methods were utilized, EPA assumes compressed air was not used for the outdoor scenario.

Bystander

Indoor Scenario

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

Outdoor Scenario:

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

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 for Risk Evaluation for Asbestos Part 1: Chrysotile Asbestos - DIY Brake Repair/Replacement Scenario

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 ^a	0.0296 ^a	Medium
Aftermarket Automotive Parts-Brakes (Outdoor)	0.007	0.0376	0.0007 ^b	0.0038 ^b	Medium (DIY) Medium-Low (Bystander)

^a Based on area samples, see section 2.3.2.1.2.

^b Reduction factor of 10 used, see section 2.3.2.1.2 .

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.
- To address potential uncertainties surrounding EPA's use of 78-year lifetime and ongoing DIY brake repair work every 3 years for the entire 62 years, EPA also estimated exposure for a single brake repair/replacement activity within a lifetime.

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

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

There is some uncertainty associated with the assumed length of time the brake repair/replacement work takes. EPA assumes it takes a DIY consumer user about three hours to complete brake repair/replacement work. This is two times as long as a professional mechanic. While it is expected to take a DIY consumer longer, it is also expected DIY consumer users who do their own brake

repair/replacement work would, over time, develop some expertise in completing the work as they continue to do it every three years.

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

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

EPA did not purchase brake products which may contain asbestos, or are advertised to contain asbestos, from online vendors or conduct testing of such products to confirm whether available brake repair/replacement products did or did not contain asbestos (due largely to cost and resource constraints). Instead, EPA assumes DIY consumers purchase and use aftermarket brake products which contain asbestos for this evaluation. However, there is some uncertainty associated with whether purchased aftermarket brake products installed by DIY consumers for brake repair/replacement work contain or do not contain asbestos. While some products manufactured and purchased outside of the United States may be labelled as containing asbestos, the product may not actually contain asbestos as such labelling could be intended to encourage purchase of the products based on a belief that asbestos containing products are better than non-asbestos containing products. Similarly, certain products manufactured and purchased from outside the United States may not be labelled as containing asbestos, due to the absence of labeling requirements, but may contain asbestos. Finally, if some products do contain asbestos, there is additional uncertainty that consumers purchase and use those specific asbestos containing products.

As mentioned earlier, EPA did not evaluate asbestos exposure resulting from brake repair/replacement work on "other vehicles" like motorcycles, snowmobiles or tractors. The reason these "other vehicles" were not evaluated is the absence of data to inform asbestos exposure resulting from such "other vehicle" activities or inform methodology to extrapolate from automobile specific activities to such "other vehicles". Considering the wide variation in size and accessibility of "other vehicles" and the absence of data to inform "other vehicle" analyses, the uncertainties could be considerable across multiple factors including, but not limited to, frequency, duration of work, and exposures resulting from "other vehicle" brake repair/replacement activities.

EPA recognizes the uncertainty associated with identifying the actual number of consumers and bystanders receiving an exposure to asbestos from automobile brake repair/replacement activities. In the draft Risk Evaluation, EPA did not identify data which could inform the actual number of DIY consumers within the United States involved with DIY brake repair replacement activities that use brake products containing asbestos. However, EPA provided an estimate of the number of brake repairs conducted by DIY consumers in the United States as approximately 31 million.

Both peer review and public commenters questioned this estimate; which was based on assuming that 100% of DIYers replace brake pads and that it is likely that the asbestos-containing brakes is a much lower percentage of the available brakes on the market. EPA agreed and updated the estimated number of individuals exposed by the limited available information on the potential market share of asbestos brakes. Details are provided in Section 4.3.7. EPA's updated estimate for number of DIYers assumes that asbestos brakes may represent approximately 0.05% of aftermarket automotive brakes. By applying this factor (0.05%) to the universe of DIYers (over 31,000,000), EPA's estimate of potentially exposed DIYers is a little over 15,900.

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

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

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

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

2.3.2.2 Consumer Exposures Approach and Methodology – DIY Gaskets in UTVs

This exposure assessment looks at a potential consumer exposure scenario where a DIY consumer removes, cleans, handles, and replaces gaskets associated with exhaust systems on UTVs which may contain chrysotile asbestos. This scenario falls under the "other gaskets" COU in Table 1-4 of this risk evaluation. Asbestos exposure is estimated for the DIY consumer user (the individual performing the gasket repair work) as well as a bystander who may observe the gasket work. This scenario also 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.

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

Thirty studies relating to gasket repair/replacement were identified and reviewed as part of the systematic review process for exposure. These studies were compared against a series of criteria to evaluate how representative the studies are for DIY consumer exhaust system gasket repair/replacement activity. The first two criteria involved identifying whether the studies were automotive in nature and whether there was enough information about automotive gaskets within the study. EPA also focused on primary sources of data and not secondary or supplemental sources. The final criterion was to review the studies to ensure they were consistent with an expected DIY consumer scenario of removal, cleaning, and replacing gaskets. For example, studies involving machining or processing of gaskets were not considered as it is unlikely a DIY consumer gasket repair/replacement activity would involve machining and gasket processing. When compared to these criteria, three of the thirty studies were fully evaluated; a 2006 study by [Blake et al. \(2006\)](#), a 2005 study by [Liukonen and Weir \(2005\)](#), and a 2006 study by [Paustenbach et al. \(2006\)](#), as shown in Table 2-28.

Table 2-28. Summary of Studies Satisfying Factors Applied to Identified Literature

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

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.

Relevant data from each of the three studies identified in Table 2-28 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, 2020a](#)). 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 2-29 summarizes the data based on the methodologies described here.

Table 2-29. Summary Results of Asbestos Exposures in Gasket Repair Studies

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

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).

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.

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.

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 (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

EPA used the PBZ values from [Paustenbach et al. \(2006\)](#) identified in Table 2-29 for the DIY consumer. The maximum concentration was used as the high-end estimated concentration for the consumer and the mean concentration was used as the central tendency concentration.

Bystander

EPA used the bystander values from [Paustenbach et al. \(2006\)](#) identified in Table 2-29 for the bystander. The bystander values from [Paustenbach et al. \(2006\)](#) represent area monitoring obtained within four feet of the automobile on which the exhaust system work was being performed. EPA believes this distance is a reasonable distance at which a bystander observing gasket work may be located within a residential garage during the gasket work. The maximum concentration from Table 2-29 was utilized as the high-end estimated concentration for the 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. EPA recognizes that frequency can vary depending on a variety of factors including the location of the gasket and the number of gaskets needing change-out at any one time. Additional variability may occur based on the consumer use patterns for a given UTV in that limited frequency and duration of use may affect the frequency at which a gasket needs to be changed. Some gasket work may not be needed but performed by a DIY consumer to increase speed or other factors related to a UTV’s performance. 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. Since UTV specific data was neither identified and evaluated as part of EPA’s systematic review process nor provided as part of comments, 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 starts at 16 years old and continues through the average adult lifetime of 78 years. Uncertainties associated with this assumption are discussed in Section 2.3.2.2.3. Table 2-30 provides a summary of the data utilized for this evaluation. Additional exposure durations were evaluated for reference or comparison. These are presented in Section 4.2.3.2.

Table 2-30. Estimated Exposure Concentrations for UTV Gasket Repair/Replacement Scenario – DIY Mechanic and Bystander for Use in the Risk Evaluation for Asbestos Part 1: Chrysotile Asbestos

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

2.3.2.2.3 Data Assumptions, Uncertainties and Level of Confidence

There was 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.

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.

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 timeframes 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.

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

There is uncertainty associated with the assumption that an individual would be associated with UTV use or UTV exhaust system gasket repair/replacement activities for the entire average adult lifetime of 78 years beginning at 16 years of age. It is possible certain individuals may be involved with UTV work prior to 16 years of age. While older individuals may not be associated with their personal UTV and related gasket work up to age 78, they may provide assistance on gasket work or perhaps change from a consumer to a bystander.

The EPA has an overall medium confidence rating for the *literature, studies, and data* utilized for the Consumer DIY UTV Exhaust System Gasket Repair/Replacement COU. This is based on the existence

of monitoring data in both the personal breathing zone and area sampling associated directly with gasket repair/replacement activities. The studies utilized are also representative of expected consumer working conditions for a DIY consumer. Both factors would indicate a high confidence in the studies and data used. However, since the data utilized is based on a professional mechanic performing the gasket repair/replacement work rather than a DIY consumer, and the use of a study involving automobile exhaust system gasket repair/replacement activities as a surrogate for UTV exhaust system work, the overall confidence is medium.

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

The EPA has an overall low confidence rating *for the frequency of gasket repair/replacement activities* (once every 3 years). This is based on the absence of data specific to frequency of UTV exhaust system gasket repair/replacement work. Additionally, the need for such repair/replacement work is expected to be heavily reliant on the frequency an individual uses the UTV, and the degree to which the UTV is pushed during use (heavy use, in extreme conditions could require more frequent work while limited use, in relatively tranquil conditions could require less frequent work).

The EPA has an overall low confidence rating *for the lifetime association of an individual with UTV exhaust system gasket repair/replacement work* (16-78 years of age). This is based on the absence of data on the age distribution of UTV ownership and self-repair/replacement work of exhaust system gaskets on UTVs. As discussed in the uncertainties, however, while a particular DIY consumer may not own a UTV for their entire lifetime, they could be involved with UTV exhaust system gasket repair/replacement work in different ways throughout their life (learning how to do the work early in life, then doing the work, then observing others/or training others to do such work).

2.3.2.3 Summary of Inhalation Data Supporting the Consumer Exposure Assessment

Table 2-31 contains a summary of the consumer inhalation exposure data used to calculate the risk estimates in Section 4.2.3.

Table 2-31. Summary of Consumer Inhalation Exposures

Condition of Use	Duration	Type	Exposure Concentrations, f/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

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 3.245

In developing this Part 1 of the risk evaluation for asbestos, 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 Part 1 of the risk evaluation for asbestos, workers who use asbestos as part of their work, and groups who have higher age- and route-specific inhalation 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 chrysotile asbestos and considered them in this Part 1 of the risk evaluation for asbestos:

- Workers and occupational non-users for the COUs in this Part 1 of the risk evaluation for asbestos (chlor-alkali, sheet gaskets, oilfield brake blocks, aftermarket automotive brakes and linings, other friction 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 who age ≥ 16 years of age exposed to chrysotile asbestos as well as non-users or workers exposed to chrysotile asbestos indirectly by being in the same work area of the building. Also, adolescents were considered as a potentially exposed or susceptible subpopulations.
- Consumers and bystanders associated with consumer (DIY) use. Chrysotile 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).

Table 2-32 presents the percentage of employed workers and ONUs who may be susceptible subpopulations within select industry sectors relevant to the chrysotile asbestos COUs. The percentages were calculated using Current Population Survey (CPS) data for 2017. CPS is a monthly survey of households conducted by the Bureau of Census for the Bureau of Labor Statistics and provides a comprehensive body of data on the labor force characteristics. Statistics for the following subpopulations of workers and ONUs are provided: adolescents, adult men and women. As shown in Table 2-32, men make up the majority of the workforce in the chrysotile asbestos COUs. In other sectors, women (including those of reproductive age and elderly women) make up a larger portion of wholesale and retail trade.

Table 2-32. Percentage of Employed Persons by Age, Sex, and Industry Sector (2017 and 2018 worker demographics from BLS)

Age Group	Sex	Mining, quarrying, and oil and gas extraction	Manufacturing	Wholesale and retail trade
		COU: Oilfield Brake Block	COU: Chlor-Alkali; Gasket stamping; Gasket use in chemical plants	COU: Auto brake; UTV
Adolescent ²³ (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%

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

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

Adolescents, or persons between 16 and 19 years in age, are generally a small part of the total workforce. Table 2-33 presents further breakdown on the percentage of employed adolescents by industry subsectors. As shown in the table, they comprise less than 2 percent of the workforce, with the exception of wholesale and retail trade subsector where asbestos may be used in UTV gaskets and auto brakes.

²³ Note that while BLS defines adolescents as 16-19 years old, EPA defines adolescents as 16 to < 21 years old.

Table 2-33. 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 (≥ 16 years of age). Bystanders could be any age group ranging from infants to adults. EPA estimates bystander risks, including infants, by applying a specific IUR for age at first exposure and duration of exposure and provides these calculations in Section 4.2.3.

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 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, 2020d](#)) 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 \(2012b\)](#) 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 asbestos 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 asbestos until hatching for 13-21 days, larvae-juvenile fish were exposed to chrysotile asbestos for 13 weeks, and juvenile-adult fish were exposed to chrysotile asbestos 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.

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 chrysotile 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 chrysotile 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 Chrysotile Asbestos

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

^aNOEC, No Observable Effect Concentration.

^bLOEC, Lowest Observable Effect Concentration.

^cValues 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.

^dMFL, Million Fibers/Liter.

^eData 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 10⁴-10⁸ 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 10²-10⁸ fibers/L to clams demonstrated reduced siphoning activity.

3.2 Human Health Hazards from Inhalation of Chrysotile Asbestos

Many authorities have established that there are causal associations between asbestos exposures and lung cancer and mesotheliomas ([NTP, 2016](#); [IARC, 2012b](#); [ATSDR, 2001a](#); [U.S. EPA, 1988b](#); [IARC, 1987](#); [U.S. EPA, 1986](#); [IARC, 1977](#)). EPA also noted in the scope that there is a causal association

between exposure to asbestos and cancer of the larynx and cancer of the ovary ([IARC, 2012b](#)). Additionally, there is a causal association between asbestos exposures and non-cancer health effects including respiratory effects (*e.g.*, asbestosis, non-malignant respiratory disease, deficits in pulmonary function, diffuse pleural thickening and pleural plaques) as well as some evidence of immunological and lymphoreticular effects ([ATSDR, 2001a](#)). Given the well-established carcinogenicity of asbestos for lung cancer and mesothelioma and the existence of an IUR for asbestos, 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 Part 1 of the risk evaluation for asbestos is chrysotile asbestos. Thus, the EPA-derived chrysotile asbestos IUR described in Section 3.2.4 is used 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](#)).

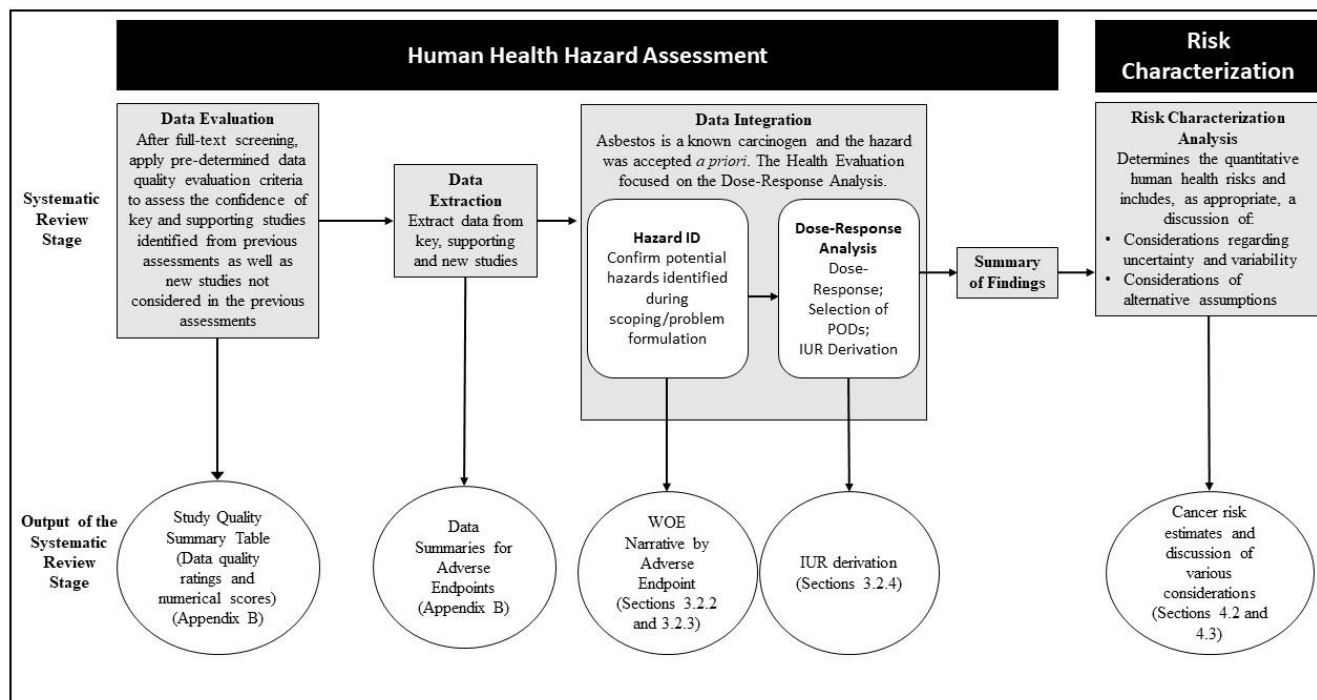


Figure 3-1. EPA Approach to Hazard Identification, Data Integration, and Dose-Response Analysis for Chrysotile Asbestos

In the PF document, it was stated that the asbestos Risk Evaluation would focus on epidemiological inhalation data on lung cancer and mesothelioma for all TSCA Title II fiber types, just as stated in the 1988 EPA IRIS Assessment on Asbestos ([U.S. EPA, 1988b](#)). This was based on the large database on the health effects associated with asbestos exposure which has been cited in numerous U.S. and international data sources. These data sources included, but were not limited to, EPA IRIS Assessment [IRIS Assessment on Asbestos \(1988b\)](#), [IRIS Assessment on Libby Amphibole Asbestos \(2014c\)](#), National Toxicology Program (NTP) [Report on Carcinogens, Fourteenth Edition \(2016\)](#), NIOSH

[Asbestos Fibers and Other Elongate Mineral Particles: State of the Science and Roadmap for Research \(2011b\)](#), [ATSDR Toxicological Profile for Asbestos \(2001a\)](#), [IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Arsenic, Metals, Fibres, and Dusts. Asbestos \(Chrysotile, Amosite, Crocidolite, Tremolite, Actinolite, and Anthophyllite\) \(2012b\)](#), and [World Health Organization \(WHO\) Chrysotile Asbestos \(2014\)](#).

EPA conducted comprehensive searches for reasonably available information on health 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). The relevant studies were evaluated using the data quality criteria in the *Application of Systemic Review in TSCA Risk Evaluations* document ([U.S. EPA, 2018a](#)). The process and results of this systematic review are available in a supplemental document (see *Systematic Review Supplemental File: Data Quality Evaluation and Data Extraction of Human Health Hazard Studies*).

This EPA human health hazard assessment consists of hazard identification and dose-response assessment as described in EPA's *Framework for Human Health Risk Assessment to Inform Decision Making* ([U.S. EPA, 2014a](#)). Hazards were identified from consensus documents. EPA integrated 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 other legacy uses and associated disposals of asbestos in a separate and forthcoming Part 2 of the risk evaluation for asbestos (as noted in the Preamble). 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 asbestos in commerce. To identify studies with the potential to be used to derive an 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 for the study domains of *exposure, outcome, study participation, potential confounding, and analysis* which were tailored to the specific needs of evaluating asbestos studies for their potential to provide information on the exposure-response relationship between asbestos exposure and risk of lung cancer and from mesothelioma. (see Section 3.2.4 and *Systematic Review Supplemental File: Data Quality Evaluation and Data Extraction of Human Health Hazard Studies* ([U.S. EPA, 2020h](#))). 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 *Supplemental File* 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 from Inhalation of Chrysotile Asbestos

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 this Part 1 of 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 all types of asbestos – including chrysotile 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 risk of lung cancer and from mesothelioma.

During scoping and PF, EPA reviewed the existing EPA IRIS health assessments to ascertain the established health hazards and any known toxicity values. EPA had previously, in the IRIS assessment on asbestos ([U.S. EPA, 1988b](#)), identified asbestos as a carcinogen causing both lung cancer and mesothelioma from inhalation exposures and derived an IUR to address both cancers. No toxicity values or IURs have yet been estimated for other cancers that have been identified by the International Agency for Research on Cancer (IARC) and other government agencies. Given the well-established carcinogenicity of asbestos for lung cancer and mesothelioma, EPA, in its PF document, had 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 unit risk. As explained in Section 1.4, the only COUs of asbestos or asbestos containing products assessed in this risk evaluation are for chrysotile asbestos. Thus, an IUR value for chrysotile asbestos only was developed.

3.2.2.1 Non-Cancer Hazards from Inhalation of Chrysotile Asbestos

Asbestos exposure is known to cause various non-cancer health outcomes including respiratory and cardiovascular effects. Respiratory effects of asbestos are well-documented and include asbestosis, non-malignant respiratory disease (NMRD), deficits in pulmonary function, diffuse pleural thickening (DPT), and pleural plaques. Various immunological and lymphoreticular effects are suggested, but not well-established ([ATSDR, 2001a](#); [U.S. EPA, 1988b](#))

These non-cancer effects are adverse. Asbestosis and NMRD have been observed to be a cause of mortality in many asbestos exposed cohorts. DPT and pleural plaques decrease pulmonary function ([U.S. EPA, 2014c](#)). Pulmonary deficits are considered to be adverse for an asbestos-exposed population, because a shift in distribution of pulmonary function in an exposed population results in a considerable increase in the proportion of individuals with a significant degree of pulmonary impairment below a clinically adverse level.

There is no RfC for general asbestos (*i.e.*, actinolite, amosite, anthophyllite, chrysotile, crocidolite,

tremolite), derived by EPA or any of the consensus organizations; only Libby Amphibole asbestos has a RfC which is based on pleural plaques ([U.S. EPA, 2014c](#)).

3.2.2.2 Cancer Hazards from Inhalation of Chrysotile Asbestos

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

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

3.2.2.3 Mode of Action (MOA) Considerations for Chrysotile Asbestos

EPA evaluated the evidence supporting plausible modes of action (MOA) of chrysotile asbestos carcinogenicity for specific tumor locations using the modified Hill criteria for MOA analysis described in EPA's [Guidelines for Carcinogen Risk Assessment](#) ([U.S. EPA, 2005](#)). EPA considered available evidence from animal cancer bioassays, genotoxicity studies, specific MOAs proposed in the literature, and the analysis previously presented in the IRIS Toxicological Review of Libby Amphibole Asbestos ([2014c](#)) and the International Agency for Research on Cancer (IARC) proposed a mechanism for the carcinogenicity of asbestos fibers [see Figure 4-2 in [IARC \(2012b\)](#)].

EPA specifically considered MOAs for lung carcinogenicity and mesothelioma of chrysotile asbestos. There is insufficient chemical-specific information about larynx, ovary, pharynx, stomach and colorectum tumors to support MOA analysis for these tumor types.

Potential Modes of Action for Chrysotile Asbestos Lung Carcinogenicity and Mesothelioma

Physicochemical properties of chrysotile fibers

Chrysotile asbestos falls into the serpentine asbestos mineral group. The chrysotile crystal structure results from the association of a tetrahedral silicate sheet with an octahedral brucite-like sheet. These

two sheets form a silicate layer with a slight misfit that causes curling to form concentric cylinders with the silicate layer on the inside and brucite-like layer on the outside of the cylinder. The fibrils are held together by Van der Waals interparticle forces so that when the chrysotile fiber is broken up large numbers of smaller fibers are generated ([Fubini and Arean, 1999](#)).

It has been proposed that the pathogenic potential of asbestos fibers depends on their aspect ratio and fiber size ([IARC, 2012b](#)). While some shorter asbestos fibers have been shown to be cleared by the system more efficiently, evidence from in vitro genotoxicity studies in Chinese hamster lung cells suggests that short and intermediate chrysotile fibers may induce micronuclei formation and sister chromatid exchange ([Lu et al., 1994](#)). There is some evidence that aspect ratio and size may play differing roles in the onset and progression of lung cancer and mesothelioma. NIOSH ([2008](#)) reported an association of lung cancer with fibers longer than 10 μm and thicker than 0.15 μm , while mesothelioma was more closely associated with shorter, thinner fibers (~5 μm long and 0.1 μm thin). However, this evidence is equivocal as multiple epidemiologic studies [summarized in the IARC monograph, [IARC \(2012b\)](#)] have reported the presence of short fibers (<5 μm , typically associated with fibrosis) in the lung and pleural tissue of malignant mesothelioma patients.

Fiber aerodynamic diameter is a key determinant of the extent of deposition and penetration to different parts of the respiratory tract ([IOM, 2006](#)). Fibers with an aerodynamic diameter less than 3 μm , which includes chrysotile asbestos, are capable of penetration into the deep pulmonary region ([NIOSH, 2011a](#)).

Generation of reactive oxygen and reactive nitrogen species

In addition to aspect ratio and size, the surface of asbestos fibers has reactivity that may generate reactive oxygen species (ROS), reactive nitrogen species (RNS) and lead to iron mobilization and or biodeposition as reviewed in [Miller et al. \(2014\)](#). The surface reactivity of asbestos fibers, including chrysotile asbestos, has been implicated in the pathogenesis of lung cancer and mesothelioma. The ability of chrysotile, and other asbestos fibers, to produce ROS and RNS depends on the presence of iron ions on the surface of the fibers ([Gazzano et al., 2005](#)). While chrysotile asbestos is a low iron containing asbestos fiber, it has been shown to produce ROS ([Wang et al., 2019](#); [Miller et al., 2014](#); [Kopnin et al., 2004](#)). It has been postulated that lung macrophages encounter inhaled asbestos fibers and proceed to undergo phagocytosis but are unable to complete the process leading to “frustrated phagocytosis²⁴” resulting in oxidative stress. Chrysotile fiber, and other asbestos fiber, derived-oxidative stress has been shown to damage cellular macromolecules, such as proteins, lipids and nucleic acids ([Miller et al., 2014](#); [Gulumian, 1999](#); [Ghio et al., 1998](#)) and apoptosis ([Upadhyay and Kamp, 2003](#); [Simeonova and Luster, 1995](#)) which may then contribute and play key roles in the onset and progression of asbestos related diseases, such as lung cancer and mesothelioma.

Overall MOA conclusions

Evidence from both in vitro and in vivo studies strongly suggest that the physicochemical properties of chrysotile asbestos fiber along with the reactive oxidants generated by these are key in the pathogenesis of asbestos related diseases such as lung cancer and mesothelioma. However, there is currently insufficient information to determine the MOA for either chrysotile lung carcinogenicity or mesothelioma. Chrysotile asbestos mesothelioma and lung carcinogenicity may be mediated by different underlying complex mechanisms that have yet to be fully elucidated. In the absence of other information about MOA, EPA often takes the health-protective approach of assuming a linear no-threshold risk model consistent with a mutagenic MOA ([U.S. EPA, 2005](#)).

²⁴ Frustrated phagocytosis: When a phagocyte fails to engulf its target, in this case the asbestos fiber, and the toxic agent (asbestos) results in the target being released or spread into the environment ([Mularski et al., 2018](#)).

3.2.3 Derivation of a Chrysotile Asbestos Inhalation Unit Risk

3.2.3.1 Considerations in Derivation of a Chrysotile Asbestos Inhalation Unit Risk

EPA did not have a previous, recent risk assessment of asbestos on which to build; therefore, the literature was reviewed to determine whether a new IUR needed to be developed. As the RE process progressed, several decisions were made that refined and narrowed the scope of the RE. It was determined during PF that the RE would focus on epidemiologic data on mesothelioma and lung cancer by the inhalation route. The existing EPA IUR for asbestos was developed in 1988 was based on 14 epidemiologic studies that included occupational exposure to chrysotile, amosite, or mixed-mineral asbestos exposures (chrysotile, amosite, crocidolite). However, EPA's research to identify COUs indicated that only chrysotile asbestos is currently being imported in the raw form or imported in products. The other five forms of asbestos identified for this risk evaluation are no longer manufactured, imported, processed, or distributed in the United States. This commercial chrysotile asbestos is therefore the substance of concern for this quantitative assessment and thus EPA sought to derive an IUR specific to chrysotile asbestos. The epidemiologic studies available for risk assessment all include populations exposed to commercial chrysotile asbestos, which may contain small, but variable amounts of amphibole asbestos. Because chrysotile asbestos is the only form of asbestos in the United States with COUs in this document, studies of populations exposed only to chrysotile asbestos provide the most informative data for the purpose of developing the TSCA risk estimates for the COUs for chrysotile asbestos. EPA will consider legacy uses and associated disposals of asbestos in a separate and forthcoming Part 2 of the risk evaluation for asbestos.

3.2.3.2 Rationale for Asbestos-Specific Data Evaluation Criteria

For the first 10 TSCA REs, a general set of study evaluation criteria was developed. These data evaluation criteria were not tailored to any specific exposure or outcome. In the PF step of the asbestos assessment, it was accepted that exposure to asbestos was a known cause of lung cancer and mesothelioma, and that the purpose of the systematic review would be the identification of studies which could inform the estimation of an exposure-response function allowing for the derivation of an asbestos inhalation unit risk for lung cancer and mesothelioma combined. The study domains of *exposure*, *outcome*, *study participation*, *potential confounding*, and *analysis* were further tailored to the specific needs of evaluating asbestos studies for their potential to provide information on the exposure-response relationship between chrysotile asbestos exposure and risk of lung cancer and mesothelioma ([U.S. EPA, 2020h](#)).

In terms of evaluating *exposure* information, asbestos is unique among these first 10 TSCA chemicals as it is a fiber and has a long history of different exposure assessment methodologies. For mesothelioma, this assessment is also 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. Asbestos measurement methodologies have changed over those decades, from early measurement of total dust particles measured in units of million particles per cubic foot of air (mppcf) by samplers called midget impingers to fibers per milliliter (f/ml), or the equivalent fibers per cubic centimeter (f/cc), where fiber samples were collected on membrane filters and the fiber count per volume of air was measured by analyzing the filters using phase contrast microscopy (PCM). In several studies encompassing several decades of asbestos exposures, matched samples from midget impingers and membrane filters were compared to derive job- (or location-) specific factors allowing for the conversion of earlier midget impinger measurements to estimate PCM measurement of asbestos air concentrations. While some studies were able to provide these factors for specific locations and jobs,

other studies were only able to derive one factor for all jobs and locations. The use of such data has allowed asbestos researchers to investigate the risk of asbestos and successfully model lung cancer and mesothelioma mortality over several decades of evaluation ([U.S. EPA, 2014c](#), [1988b](#), [1986](#)). Thus, the general exposure evaluation criteria were adjusted to be specific to exposure assessment methodologies such as midget impingers and PCM with attention to the use of job-exposure-matrices (JEMs) to reconstruct workers' exposure histories and the reporting of key metrics needed to derive exposure-response functions for lung cancer and mesothelioma.

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

Mesothelioma is a very rare cancer. As noted by U.S. EPA ([2014c](#)), the “Centers for Disease Control and Prevention estimated the death rate from mesothelioma, using 1999 to 2005 data, as approximately 23.2 per million per year in males and 5.1 per million per year in females ([CDC, 2009](#)).” While very rare, the overwhelmingly dominant cause of mesothelioma is asbestos exposure ([Tossavainen, 1997](#)), making the observance of mesothelioma in a population a very specific indicator for asbestos exposure. The prevailing risk model for mesothelioma is an absolute risk model, which assumes there is no risk at zero exposure ([U.S. EPA, 1986](#); [Peto et al., 1982](#); [Peto, 1978](#)). This use of an absolute risk model differs from the standard use of a relative risk model for lung and other cancers. For the relative risk model, the risk of lung cancer in an asbestos exposed population multiplies a background risk in an unexposed population. Thus, an important consideration of study quality is the evaluation of that comparison population. However, for mesothelioma, no comparison population is needed to estimate the absolute risk among people exposed to asbestos, and therefore the criteria in the *study participation* domain (that includes comparison population) were adjusted for mesothelioma.

In terms of evaluating *potential confounding*, the generic potential confounding section was adapted to recognize that there are both direct and indirect methods for controlling for some confounders. Specifically, methodologies that involve only internal comparisons within a working population may indirectly control for smoking and other factors assuming these factors do not vary with asbestos exposure concentrations in the workplace. In contrast, mesothelioma is much simpler to evaluate for potential confounding as diagnostic X-ray contrast medium “Thorotrast” and external beam radiotherapy are the only other known non-fibrous risk factors for mesothelioma, and these are unlikely to be confounders because these rare procedures are not routinely done on healthy workers. Screening programs typically X-ray all workers – regardless of their cumulative asbestos exposure. There are other fibrous risk factors for mesothelioma such as fluoroedenite ([Grosse et al., 2014](#)) and erionite ([IARC, 2012a](#)), but exposures to these materials which are not used in conjunction with chrysotile asbestos in COUs.

In terms of evaluating *analysis*, the evaluation criteria were adapted for both mesothelioma and lung cancer. For mesothelioma, the Peto model ([Peto et al., 1982](#); [Peto, 1978](#)) has traditionally been used for summary data published in the literature ([U.S. EPA, 1986](#)) but also has been used with individual-level data (*e.g.*, [Berman and Crump \(2008a\)](#)), so studies were considered acceptable if the authors reported

sufficient information on the results of using the Peto model or presented sufficient information to fit the Peto model *post hoc*. For lung cancer, a wider selection of statistical models was acceptable, with the preference generally given to modeling that used individual data in the analysis.

3.2.3.3 Additional considerations for final selection of studies for exposure-response

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

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

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

Studies were deemed informative for lung cancer risk assessment if either the relative risk of lung cancer per unit of cumulative chrysotile asbestos exposure in fibers per cc-year (f/cc-yrs) were available from fitting log-linear or additive relative risk models or the data needed to fit such models as described below. The group of Balangero, Italy cohort studies including Pira et al. ([2009](#)) was excluded for lack of results from models using a continuous measure of exposure. Studies that presented lung cancer risks only in relation to impinger total dust exposure were excluded from consideration unless they provided at least a data-based, study-specific factor for converting concentrations from mppcf to f/cc.

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

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

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

Study Cohort	Author, Year	Study Quality**
South Carolina, US	Berman and Crump (2008b)	Lung Cancer 1.6 High Mesothelioma 1.7 Medium
	Brown et al. (1994)	
	Cole et al. (2013)	
	Dement et al. (1983b)	
	Dement et al. (1994)	
	Dement and Brown (1994)	
	Edwards et al. (2014)	
	Elliott et al. (2012)	
	Hein et al. (2007)	
	Loomis et al. (2012)	
	SRC (2019c)	
	Stayner et al. (1997)	
Stayner et al. (2008)		
Qinghai, China - miners	Wang et al. (2012)	Lung Cancer 1.6 High
	Wang et al. (2013b)	
	Wang et al. (2014)	
Balangero, Italy*	Piolatto et al. (1990)	NA
	Pira et al. (2009)	
	Pira et al. (2017)	
	Rubino et al. (1979)	
North Carolina, US	Dement et al. (2008)	Lung Cancer 1.7 Medium Mesothelioma 1.5 High
	Elliott et al. (2012)	
	Loomis et al. (2009)	
	Loomis et al. (2010)	
	Loomis et al. (2012)	
	Loomis et al. (2019)	
SRC (2019a)		
Salonit Anhovo, Slovenia*	Dodic Fikfak (2003)	NA
	Dodic Fikfak et al. (2007)	
Quebec, Canada	Berman and Crump (2008b)	Lung Cancer*** Low*** Mesothelioma Medium***
	Gibbs and Lachance (1972)	
	Liddell et al. (1997)	
	Liddell et al. (1998)	
	Liddell and Armstrong (2002)	
	Mcdonald et al. (1993a)	
	Mcdonald et al. (1993b)	
	SRC (2019b)	
	Vacek (1998)	
Chongqing, China – asbestos products factory including textiles	Courtice et al. (2016)	Lung Cancer 1.4 High
	Deng et al. (2012)	
	Wang et al. (2014)	
	Wang et al. (2013a)	
	Yano et al. (2001)	

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

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

*** Study quality was downgraded for this cohort during conflict resolution between primary review and QA/QC review. Downgrading was due to lack of PCM or TEM-equivalent exposure estimates and potentially significant co-exposure to tremolite or other amphiboles.

3.2.3.4 Statistical Methodology

The first step in deriving a cancer unit risk for risk estimation is to identify potency factors for lung cancer and mesothelioma. Cancer potency values are either extracted from published epidemiology studies or derived from the data within those studies. Once the cancer potency values have been obtained, they are adjusted for differences in air volumes between workers and other populations (Section 3.3.3.4.3). Those adjusted values can be applied to the U.S. population as a whole in the standard EPA life-table analyses. These life-table analyses allow for the estimation of an exposure concentration associated with a specific extra risk of cancer incidence caused by asbestos. The unit risks for lung cancer and mesothelioma are estimated separately and then combined to yield the cancer inhalation unit risk.

3.2.3.4.1 Cancer Risk Models for Asbestos Exposures

A cancer risk model predicts the probability of cancer in an individual with a specified history of exposure to a cancer-causing agent. In the case of inhalation exposure to chrysotile 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 from epidemiologic studies from exposure to asbestos is usually quantified using a linear relative risk model of the following form ([Berman and Crump, 2008b](#); [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 CE10 to represent the 10-year lag period.

K_L = Lung cancer potency factor (f/cc-yrs)⁻¹.

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 $\alpha = \exp(\beta_0)$ 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_L) and were carried forward for further consideration.

Mesothelioma

For mesothelioma, the risk model is usually an absolute risk model that gives the risk of 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 below was originally proposed by [Peto et al. \(1982\)](#) and [Peto \(1978\)](#) and was subsequently used by U.S. EPA ([1986](#)). Berman and Crump ([2008b](#)) adapted this model for variable exposure, which is used in this Part 1 of the risk evaluation for asbestos.

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

where:

I_m = Rate of mesothelioma (cases per person year)

C = Concentration of asbestos (f/cc)

K_M = Mesothelioma potency factor (f/cc-yrs³)⁻¹

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 $10 \leq \text{TSFE} < d + 10$ $Q = (\text{TSFE} - 10)^3$
- for TSFE $\geq d + 10$ $Q = (\text{TSFE} - 10)^3 - (\text{TSFE} - 10 - d)^3$

3.2.3.4.2 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 summarized into groups according to a key exposure metric (CE10 for lung cancer, TSFE for mesothelioma), and the mid-point of the range for each exposure metric is usually used in the fitting, unless means/medians of exposure metric were available. In cases where the upper bound of the highest exposure category was not reported in the publication, the value for the upper bound of the highest exposure category was assumed to be the maximum exposure reported in the publication. Background parameter α in lung cancer model was both assumed fixed at 1 and fitted. Results with lower AIC are shown in the tables below. Full modeling results for both cases are shown in Appendix J.

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 data summarized into groups, 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 three 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. The inhalation unit risk (see below) is derived from the one-sided 95% upper bound (which is equivalent to the upper bound of the two-sided 90% upper bound). In the literature, authors typically report two-sided 95% confidence intervals (*i.e.*, from the 2.5% to the 97.5% bounds). In these instances, EPA computed the standard error of the effect estimate from the published results and used that value to estimate the 5% and 95% 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.

3.3.3.4.3 Extrapolation from Workers to the General Population to Derive an 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 lifetime²⁵, 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 exposure pattern (*e.g.*, a bystander to consumer/DIY COU). The extrapolation is based on the assumption that the ratio of the risk of cancer in one population compared to another (both exposed to the same level of asbestos in air) is related to the ratio of the amount of asbestos-contaminated air that is inhaled per unit time (*e.g.*, per year).

For workers, EPA assumes a breathing rate of 10 m³ of air per 8-hour work day ([U.S. EPA, 2009](#)). If workplace exposure is assumed to occur 240 workdays/year, the volume of air inhaled in a year is calculated as follows:

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

For a resident, EPA usually assumes a breathing rate of 20 m³/day ([U.S. EPA, 2009](#)). If exposure is assumed to be continuous (24 hours per day, 365 days per year), the volume inhaled in a year is calculated as follows:

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

In this case, the extrapolation factor from worker to resident is:

²⁵ Note that the lifetime inhalation unit risk is then applied to specific environmental exposure scenarios applicable to current chrysotile asbestos uses; for specific worker exposure scenarios, the extrapolation factor described may not be applied.

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

In the tables below (Section 3.2.4), the potencies are shown as calculated from epidemiological studies, and the worker to other populations extrapolation factor is applied in the life-table analyses so that the unit risks and IUR incorporate that extrapolation factor.

3.2.3.4.4 Life-Table Analysis and Derivation of Inhalation Unit Risk

Potency factors are not analogous to lifetime unit risks or cancer slope factors, and do not directly predict the excess risk of lung cancer or mesothelioma in an exposed individual. Rather, the potency factors are used in lifetable analyses for lung cancer and mesothelioma to predict the risk of cancer as a result of the exposure in a specified year of life. However, it is important to recognize that cancer risk in a particular year of life is conditional on the assumption that the individual is alive at the start of the year. Consequently, the risk of a chrysotile asbestos-related cancer within a specified year of life is calculated as a function of the probability of being alive at the start of the year, the background probability of getting cancer, and the increased risk of getting cancer from chrysotile asbestos exposure within the specified year. The lifetime risk is then the sum of all the yearly risks. This procedure is performed to calculate the lifetime risk both for an unexposed individual (R_0) and for an individual with exposure to chrysotile asbestos (R_x).

“Extra risk” for cancer is a calculation of risk which adjusts for background incidence rates of the same type of cancer, by estimating risk at a specified exposure level and is calculated as follows ([U.S. EPA, 2012](#)):

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

For mesothelioma, because background risk (R_0) is assumed to be zero, extra risk is the same as absolute risk (R_x).

The unit risk is risk of incident cancer per unit asbestos concentration (fiber/cc or f/cc) in inhaled air. The unit risk is calculated by using life table analysis to find the exposure concentration (EC) that yields a 1% (0.01) extra risk of cancer. The 1% value is referred to as the Benchmark Response (BMR). This value is used because it represents a cancer response level that is near the low end of the observable range ([U.S. EPA, 2012](#)).

As described in Section 3.2.2.3, because MOA for chrysotile asbestos is uncertain, following the recommendations of the Guidelines for Carcinogen Risk Assessment ([U.S. EPA, 2005](#)) a linear extrapolation to low doses was used. Given the EC at 1% extra risk (EC_{01}), the unit risk is the slope of a linear exposure-response line from the origin through the EC_{01} :

$$\text{Unit risk} = 0.01 / EC_{01}$$

A unit risk value may be calculated based on both the best estimate and the 95% upper confidence bound (UB) on the potency factor. The value based on the upper 95% confidence bound is normally used for decision-making, since it corresponds to a lower 5% confidence bound (LB) on the exposure level yielding 1% extra risk (LEC_{01}). Inhalation unit risk is derived by statistically combining risks of lung cancer and mesothelioma. This procedure is described below in the section on combining unit risks.

Life table calculations require as input the all-cause mortality rates and cause-specific cancer incidence rate for the general population in each year of life. The all-cause mortality data were obtained from the

National Vital Statistics Report Vol 68 No 7 Table 1 ([2017](#)), which provides data from the U.S. population in 2017. Lung-cancer incidence rates were obtained by downloading 2017 data for malignant neoplasms of bronchus and lung (ICD-10 C33-C34) from CDC Wonder (<http://wonder.cdc.gov/ucd-icd10.html>). Because cause-specific rates were given for 5-year intervals, the cause-specific rate for each 5-year interval was applied to each age within the interval. For mesothelioma, the incidence rate in the absence of asbestos exposure was assumed to be zero.

The detailed equations for calculating lifetime excess cancer risk for a specified exposure concentration in the presence of competing risks are based on the approach used by NRC ([1988](#)) for evaluating lung cancer risks from radon. The equations are detailed in Appendix H. The SAS code for lung cancer life table analysis was provided to EPA by NIOSH²⁶ and was adapted for use by a) entering the data noted above, b) adding an equation to compute extra risk, and c) adding a macro to solve for the EC. The SAS code for mesothelioma was created by inserting user-defined equations for the mesothelioma risk model into the NIOSH code. The SAS codes for performing the mesothelioma and lung cancer life table calculations are provided in Appendix I. As a quality check, life table calculations were also performed using Microsoft Excel. Both methods yielded the same results to three or more significant figures.

3.2.3.5 Study Descriptions and Model Fitting Results

The asbestos exposure data and exposure assessment methods in studies of the Charleston, South Carolina textile plant ([Elliott et al., 2012](#); [Hein et al., 2007](#)) are exceptionally detailed compared to most asbestos studies. The methods used were innovative at the time, a large number of exposure measurements cover the relevant study period, and detailed process and work history information were available and utilized in estimating exposures. The exposure data used in studies of North Carolina plants ([Loomis et al., 2019](#); [Elliott et al., 2012](#)) are also high quality. The methods were similar to those developed for the studies of the South Carolina plant. However, relative to the South Carolina study, the number of exposure measurements is smaller, and the historical process and work-history data are less detailed. Nevertheless, the exposure data are of higher quality than those utilized in other studies of occupational cohorts exposed to chrysotile asbestos. For both U.S. textile cohorts, the exposure assessment methods and results have been published in full detail ([Dement et al., 1983b](#); [Dement et al., 2009](#)).

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

Information about the assessment of exposures for the Québec asbestos mining and milling cohorts is presented in several papers ([Liddell and Armstrong, 2002](#); [1998](#); [Vacek, 1998](#); [Liddell et al., 1997](#); [1993a](#); [1980a](#); [McDonald et al., 1980b](#)), but the reports are lacking important details and are sometimes in conflict. Nevertheless, it is evident that exposure measurements do not cover the entire study period. The number of measurements is not consistently reported but appears to be smaller than for either of the U.S. textile cohorts, while the number of distinct jobs was larger. Moreover, all the reported measurements were of total dust, rather than fibers. Some reports have suggested or used a conversion factor, but the use of single factor for all operations is likely to introduce substantial exposure

²⁶ Beta Version. SAS 30NOV18, provided by Randall Smith, National Institute for Occupational Safety & Health.

misclassification since the relationship between total dust and fiber counts has been shown to vary considerably by process.

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

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

3.2.3.5.1 Highest quality cohorts with results carried forward for IUR derivation

South Carolina asbestos textile plant

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

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

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

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

Hein et al. (2007) did not report exposure-response analysis or detailed data for mesothelioma in the Charleston cohort. All death certificates for deaths before ICD-10 in 1999 were investigated (Hein, personal communication) for mention of mesothelioma (3 deaths), no mesothelioma deaths after 1999 were observed. Berman & Crump (2008b) estimated K_M for the cohort from analyses of the original data obtained from the study investigators (see Table 3-3). They did not reject the hypothesis of linearity in the variable exposure Peto model, so results of the linear model are shown.

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

Endpoint	Source	Table in original publication	Potency Factor K_L or K_M		Exposure Concentration associated with BMR (1% Extra Risk) (f/cc)		Lifetime Unit Risk (per f/cc)	
			MLE	95% UB	EC ₀₁ MLE	LEC ₀₁ 5% LB	MLE	95% UB
Lung Cancer	Hein et al. (2007) linear	Table 5	1.98E-2	2.80E-2	4.67E-2	3.30E-2	2.14E-1	3.03E-1
	EPA modeling of Hein et al. (2007) grouped data linear	NIOSH	1.76E-02	2.64E-02	5.25E-02	3.50E-02	1.90E-01	2.86E-01
	Elliott et al. (2012) linear	Table 2	2.35E-2	3.54E-2	3.93E-2	2.61E-2	2.54E-1	3.83E-1
	Elliott et al. (2012) exponential	Table 2	5.10E-3	6.36E-3	1.67E-1	1.34E-1	5.99E-2	7.47E-2
Mesothelioma	Berman and Crump (2008b) using the data from Hein et al. (2007) variable exposure Peto	Table 4	1.5E-9	3.3E-9	3.9E-1	1.8E-1	2.6E-2	5.7E-2

- 1) Data summarized by NIOSH from the data of Hein et al. (2007) as well as details for the modeling for lung cancer are provided in Appendix J, Section 1. Details for the modeling of mesothelioma are provided in Berman and Crump (2008b)
- 2) In EPA modeling of Hein et al. (2007) grouped data, $\alpha=1.35$ 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 (2008b) 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 (2008b) are available, and those are carried forward for the unit risk derivation.

North Carolina asbestos textile plants

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 asbestos 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.

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. Results of modeling with lag of 10 years are shown in Table 3-4.

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 in 1999, 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 used variable exposure Peto model for the post-1999 subcohort reported in that publication (see Table 3-4).

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

Endpoint	Source	Table in Original Publication	Potency Factor K _L or K _M		Exposure Concentration associated with BMR (1% Extra Risk) (f/cc)		Lifetime Unit Risk (per f/cc)	
			MLE	95% UB	EC ₀₁ MLE	LEC ₀₁ 5% LB	MLE	95% UB
Lung Cancer	Elliott et al. (2012) linear	Table 2	1.20E-3	2.71E-3	7.70E-1	3.41E-1	1.30E-2	2.93E-2
	Elliott et al. (2012) exponential	Table 2	9.20E-4	1.40E-3	9.25E-1	6.08E-1	1.08E-2	1.64E-2
	Loomis et al. (2009) exponential	Table 6	1.01E-3	1.47E-3	8.43E-1	5.79E-1	1.19E-2	1.73E-2
	EPA modeling of Loomis et al. (2009) grouped data linear	Table 5	5.15E-4	1.02E-3	1.79	9.06E-1	5.57E-3	1.10E-2
Mesothelioma	Loomis et al. (2019), variable exposure Peto	Text, page 475	2.96E-9	5.87E-9	1.97E-1	9.92E-2	5.08E-2	1.01E-1

- 1) Details for the modeling are provided in Appendix J, Section 2.
- 2) In EPA modeling of the Loomis et al. (2009) lung cancer grouped data, fitted alpha=1.18 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.

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.

3.2.3.5.2 Other cohorts with results not carried forward for IUR 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., 2013a; Deng et al., 2012). The 2008 follow-up of the cohort added 279 women employed between 1970 and 1972 (Wang et al., 2013a).

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 asbestos 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 asbestos 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).

Exposure-response data for lung cancer in the Chongqing cohort have been reported in several papers. Deng et al. (2012) analyzed data for 586 men and women followed to 2006 and reported quantitative risk estimates for cumulative chrysotile asbestos exposure obtained by fitting log-linear and additive relative rate models with adjustment for age, smoking and calendar period. Wang et al. (2014) published additional analyses of the same study population but truncated the follow-up period from 1981 to 2006 to make it more comparable with a study of Chinese asbestos miners (described below). The vital status of this cohort was updated to 2008 and an analysis including follow-up from 1972 to 2008 was published by Courtice et al. (2016). The latter papers provide quantitative risk estimates from internal analyses with log-linear relative rate models. Papers on the Chongqing cohort provide informative exposure-response information in units of f/cc-years from Cox or Poisson regression analyses. However,

there is potential for misclassification of exposures due to the relatively small number of exposure measurements, the lack of fiber measurements before 1999 and use of area rather than personal sampling (Deng et al., 2012). Modeling results from Deng et al. (2012) are provided in Table 3-5. Result of modeling with lag of 10 years are shown.

Table 3-5. Model Fitting Results for the Chongqing China Cohort

Endpoint	Source	Table in Original Publication	Potency Factor K_L		Exposure Concentration associated with BMR (1% Extra Risk) (f/cc)		Lifetime Unit Risk (per f/cc)	
			MLE	95% UB	EC ₀₁ MLE	LEC ₀₁ 5% LB	MLE	95% UB
Lung Cancer	Deng et al. (2012) exponential	Table 3	2.08E-3	3.02E-3	4.09E-1	2.82E-1	2.44E-2	3.55E-2
	Deng et al. (2012) Linear	Table 3	4.21E-3	4.56E-3	2.19E-1	2.03E-1	4.56E-2	4.94E-2

Details for the modeling are provided in Deng et al. (2012)

Data for mesothelioma were reported for follow-up through 2008 of the expanded cohort including women (Wang et al., 2013a). Three deaths coded as mesothelioma according to the ICD-10 (2 among men and 1 among women) were recognized and only SMRs were reported separately for men and women (Wang et al., 2013a). Data on the exposure levels of the mesothelioma cases are not available, however, so model fitting was not possible. No other analyses of mesothelioma have been reported for the Chongqing cohort.

Québec, Canada Asbestos Mines and Mills

Data from studies of miners, millers and asbestos products factory workers at several facilities in Québec, Canada are reported in multiple publications (Liddell and Armstrong, 2002; 1998; Vacek, 1998; Liddell et al., 1997; 1993a; 1980a; McDonald et al., 1980b). The earliest publication, McDonald et al. (1980b), included 11,379 miners and millers from Québec, Canada who were born between 1891 and 1920 and had worked for at least a month in the mines and mills and were followed to 1975. Additional findings based on follow-up of the cohort to 1988 were reported by McDonald et al. (1993a), and further extended to 1992 by Liddell et al. (1997). Trace amounts of tremolite have been reported in samples from the Canadian mines (IARC, 2012b), with the amounts varying between mines (Liddell et al., 1997).

The most detailed description of exposure assessment methods used in the Québec studies is given by Gibbs and Lachance (1972). Additional details and updates are given in later publications (e.g., Liddell et al. (1997); McDonald et al. (1980b)). Total dust concentrations (in mppcf) were estimated using midjet impinger measurements taken from 1948 to 1966 (Gibbs and Lachance, 1972). Several different figures are reported for the total number of dust measurements used to estimate exposures: Gibbs and Lachance (1972) reported 3096; McDonald et al. (1980b) reported “well over 4000,” and McDonald et al. (1980a) reported 10,205. Annual dust concentrations for 5783 unique jobs were assigned according a 13-point scale with categories of 0.5, 2, 7, 12, 17, 22, 27, 32, 37, 42, 47, 70 and 140 mppcf. The authors describe the categories as “approximating to the mean,” but the methods of analyzing the exposure measurements and developing the categories are not reported. Different approaches were used to estimate exposures in earlier and later years when dust data were judged to be inadequate; exposures in years before 1948 were reportedly estimated by expert assessment based on interviews with workers and

company personnel, while those after 1966 were estimated by extrapolation from the previously measured levels (Liddell et al., 1997). Cumulative dust exposure (in mppcf-years) for each worker was estimated from the assigned dust concentrations and individual work histories; estimated exposures in years before 1938 were multiplied by 1.65 to account for longer work weeks at that time (Liddell et al., 1997). Fibers reportedly accounted for 8-15% of total dust (Gibbs and Lachance, 1972). Most exposure-response analyses for the cohort were reported relative to cumulative dust exposure in mppcf. However, in a case-control study of lung cancer, McDonald et al. (1980a) adopted an overall conversion factor of 3.14 f/cc per mppcf, citing 11,819 fiber measurements (methods of measurement and analysis not described), “unfortunately with little overlap” with the dust data. In another publication, McDonald et al. (1980b) suggested fiber concentrations per cc would be between 1 and 7 per mppcf. Liddell et al. (1984) subsequently reported conversion factors ranging from 3.44 to 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 D is the dust concentration measured by impinger, for the ratio of fibers to dust (units not specified). Gibbs and Lachance (1972), reported that the correlation between midget impinger and membrane filter counts (0.32) was poor and suggested that “no single conversion factor was justified.” Berman (2010) performed an analysis of dust samples from the Québec mines and found that one third of the PCM structures samples in the dust were not asbestos, and that about one third of structures counted by PCM were also counted by TEM. These findings along with the uncertainties concerning what is an appropriate conversion factor raise significant concerns about the accuracy of the f/cc estimates of exposure from the Québec studies.

Most analyses of the Québec cohort compared workers’ mortality to the general population using SMRs (e.g., Liddell et al. (1997; 1993a); McDonald et al. (1980b)). Liddell et al. (1998) conducted a nested case-control study of lung cancer in a subset of workers at the mines and mills that were included in the previous cohort studies and workers from an asbestos products factory. Subsequent publications by Vacek et al. (1998), and Liddell and Armstrong (2002) presented more detailed analyses on a subset of the cohort to examine the role of intensity and timing of exposure, and of potential effect modification by cigarette smoking. All exposure-response analyses of lung cancer in the Québec studies utilized total dust exposure expressed in mppcf. Estimates of K_L or analogous additive relative risk measures have not been reported for these studies.

Berman and Crump (2008b) estimated K_L for the Québec cohort using summarized data in Liddell et al. (1997). A single conversion factor for all operations of 3.14 f/cc per mppcf was assumed in this analysis (and mesothelioma analysis below). Results of lung cancer modeling with lag of 10 years are presented in Table 3-6.

Liddell et al. (1997) reported 38 cases of mesothelioma in the last follow-up through 1992. There is a considerable uncertainty about potency (K_M) estimates in this cohort. Berman and Crump (2008b) conducted testing of linearity in the Québec cohort (35 cases of mesothelioma were used in their analysis) using the variable exposure Peto model and statistically rejected linearity ($p < 0.00001$) resulting in sublinearity and thus estimated values of K_M from the non-linear model that were one and a half orders of magnitude higher than in linear model ($0.02E-8$ vs $0.72E-8$). Because no confidence interval was reported for the non-linear model, only the linear model result for the “Mines and mills at Asbestos” (based on eight cases) is shown in the Table 3-6 because it was specific to mining and linearity was not rejected.

Table 3-6. Model Fitting Results for the Québec, Canada Cohort

Endpoint	Source	Table in Original publication	Potency Factor K_L		Exposure Concentration associated with BMR (1% Extra Risk) (f/cc)		Lifetime Unit Risk (per f/cc)	
			MLE	95% UB	EC ₀₁ MLE	LEC ₀₁ 5% LB	MLE	95% UB
Lung Cancer	Berman and Crump (2008b) modeling of grouped data linear	Table B1	2.9E-4	4.1E-4	3.2	2.3	3.1E-3	4.4E-3
Mesothelioma	Berman and Crump (2008b) variable exposure Peto model	Table 1	1.2E-10	2.1E-10	4.9	2.8	2.1E-3	3.6E-3

1. Details for the modeling are provided in Berman and Crump (2008b).
2. In Berman and Crump (2008b) modeling of the lung cancer, $\alpha=1.15$ was fitted.
3. Berman and Crump (2008b) reported lung cancer and mesothelioma potency numbers (K_L and K_M) with 2 significant digits.

Qinghai, China Asbestos Mine

Wang et al. (2014; 2013a; 2012) reported findings from exposure-response analyses of a cohort of 1539 workers at a chrysotile asbestos mine in Qinghai Province, China who were on the registry January 1, 1981 and had been employed for at least one year. The cohort was followed for vital status from 1981 to 2006.

The mine opened in 1958 (no closing date reported) and produced commercial chrysotile asbestos with no detectable tremolite asbestos content (LOD 0.1%, Wang et al. (2012)). Total dust concentrations in the mine were measured periodically between 1984 and 1995 by area sampling in fixed locations (Wang et al., 2012). Sampling was performed according to Chinese national standards. The number of measurements during this period is not reported. An additional 28 measurements were taken in 2006 in 8 different workshops. Dust concentrations in mg/m³ were converted to f/cc using a linear regression model based on 35 paired measurements taken in 1991. Fiber concentrations were estimated by workshop and job title for the period 1984-2006, apparently using a single conversion factor. The estimation methods are not described in detail in English-language publications, but further details may be available in Chinese-language publications referenced by Wang et al. (2013b; 2012), but not reviewed here. As recognized by the authors Wang et al. (2013b), there is potential for exposure measurement error due to the conversion from mppcf to f/cc-yrs which was based on 35 paired samples that were collected in only one year, for an unspecified number of operations.

Wang et al. (2013b) report estimates of SMRs and standardized rate ratios (SRRs) for lung cancer by categorical levels of f/cc-yrs, stratified by smoking status. EPA used these combined data for smokers and non-smokers to estimate a value and confidence interval for K_L based on the linear relative risk model.

Wang et al. (2014) presented rate ratios for categorical and continuous exposure variables using log-linear Cox proportional hazards models adjusted for age and smoking. The findings from the Cox model

are useful for risk assessment in that asbestos exposure is modeled as a continuous variable using individual level data, which generally provides a more statistically powerful examination of exposure-response relationships than a grouped analysis. Furthermore, the Cox PH analyses by Wang et al. (2014) adjusted for smoking, whereas the earlier SMR and SRR analyses (Wang et al., 2013b) did not. Modeling results with lag of 10 years are shown in Table 3-7.

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 K_L		Exposure Concentration associated with BMR (1% Extra Risk) (f/cc)		Lifetime Unit Risk (per f/cc)	
			MLE	95% UB	EC ₀₁ MLE	LEC ₀₁ 5% LB	MLE	95% UB
Lung Cancer	EPA modeling of Wang et al. (2013b) grouped data linear	Tables 5 and 6	2.72E-2	3.51E-2	3.40E-2	2.63E-2	2.94E-1	3.80E-1
	Wang et al. (2014) exponential	Table 3	1.82E-3	2.63E-3	4.68E-1	3.24E-1	2.14E-2	3.09E-2

- 1) Details for the modeling are provided in Appendix J, Section 3.
- 2) In EPA modeling of the Wang et al. (2013b) grouped data, alpha was fixed 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 1.2 and it was used to calculate the potency factor as follows: potency factor = $\ln(1.2) / 100$.

3.2.3.6 Lung Cancer and Mesothelioma Potencies Ranges by Industry

Historically, it has been proposed in the asbestos literature, that lung cancer and mesothelioma potencies may differ by industry (e.g., U.S. EPA (1986), Berman and Crump (2008b) and references therein). The estimated potencies of lung cancer (K_L) are available from both North and South Carolina cohorts and from two other cohorts (Québec, Canada; Qinghai, China). Regarding mesothelioma, estimated potency estimates (K_M) are available from both North Carolina and South Carolina cohorts, and Québec, Canada cohort. These results allow comparison of lung cancer and mesothelioma potencies by industry (textile vs. mining); one remaining cohort included multiple industries and was not included in the comparison (Chongqing, China). Because there are at most two cohorts in each industry category, only a rough comparison is possible by looking at range of K_L and K_M for each industry. Results are in Table 3-8 below. It is clear that the range of potencies in each cell is very wide; however, this limited data indicates that among these cohorts exposed only to chrysotile asbestos, there is no evidence that the potencies of lung cancer and mesothelioma are different between textile and mining industries.

Table 3-8. Comparison of Cancer Potencies (K_L and K_M) by Industry

Industry	Cancer Outcome	Cancer Potencies (K _L or K _M)	
		MLE	95% UB
Textiles	Lung Cancer (K _L)	5.15E-4 – 2.95E-2	1.02E-3 – 3.54E-2
Mining		2.9E-4 – 2.72E-2	4.1E-4 – 3.51E-2
Textiles	Mesothelioma (K _M)	1.95E-9 – 2.96E-9	3.3E-9 – 5.87E-9
Mining		1.2E-10-7.2E-9	2.1E-10 - NA

Textiles cohorts (Loomis et al., 2009; Hein et al., 2007); Mining cohorts (Québec, Canada (95% UB for non-linear model K_m is N/A); Qinghai, China (only for lung cancer)). The cohort from Chongqing, China (lung cancer only) was not included here, but its lung cancer potencies are intermediate and would not change the lung cancer ranges provided in the Table.

3.2.3.7 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 K _L or K _M		Exposure Concentration associated with BMR (1% Extra Risk) (f/cc)		Lifetime Unit Risk (per f/cc)	
			MLE	95% UB	EC ₀₁ MLE	LEC ₀₁ 5% LB	MLE	95% UB
South Carolina	Lung Cancer	Hein et al. (2007) linear	1.98E-2	2.80E-2	4.67E-2	3.30E-2	2.14E-1	3.03E-1
		Elliott et al. (2012) linear	2.35E-2	3.54E-2	3.93E-2	2.61E-2	2.54E-1	3.83E-1
		Elliott et al. (2012) exponential	5.10E-3	6.36E-3	3.67E-1	1.34E-1	5.99E-2	7.47E-2
	Mesothelioma	Berman and Crump (2008b) using the data from Hein et al. (2007) variable exposure Peto	1.5E-9	3.3E-9	3.9E-1	1.8E-1	2.6E-2	5.7E-2
North Carolina	Lung Cancer	Elliott et al. (2012) linear	1.20E-3	2.71E-3	7.70E-1	3.41E-1	1.30E-2	2.93E-2
		Elliott et al. (2012) exponential	9.20E-4	1.40E-3	9.25E-1	6.08E-1	1.08E-2	1.64E-2
		Loomis et al. (2009) exponential	1.01E-3	1.47E-3	8.43E-1	5.79E-1	1.19E-2	1.73E-2
	Mesothelioma	Loomis et al. (2019) variable exposure Peto	2.96E-9	5.87E-9	1.97E-1	9.92E-2	5.08E-2	1.01E-1

3.2.3.8 Derivation of Inhalation Unit Risk of Cancer Incidence: Issues to Consider

3.2.3.8.1 Biases in the Cancer Risk Values

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](#)).

There is no single or set of morphological criteria that are entirely specific for mesothelioma ([Whitaker, 2000](#)). Peritoneal mesothelioma diagnosis is challenging, because mesothelioma and ovarian or peritoneal serous carcinoma have a common histogenesis, and may be difficult to differentiate morphologically ([Davidson, 2011](#)). To account for various sources of underascertainment of mesothelioma deaths, U.S. EPA ([2014c](#)), following Kopylev et al. ([2011](#)), developed a multiplier of risk for mesothelioma deaths before and after introduction of ICD-10. Although this procedure was developed based on the Libby Worker cohort, the problematic diagnostic issues described above are agnostic to the fiber type exposure. The developed multiplier ([U.S. EPA, 2014c](#)) is 1.39 with confidence interval (0.80, 2.17). Table 3-10 shows the mesothelioma unit risks adjusted for underascertainment.

Table 3-10. Addressing Underascertainment of Mesothelioma

Cohort	Source	Mesothelioma MLE Unit risk (per f/cc)	Mesothelioma UB unit risk (per f/cc)	Adjusted Mesothelioma MLE Unit Risk (per f/cc)	Adjusted Mesothelioma UB risk (per f/cc)
South Carolina	Berman and Crump (2008b) using the data from Hein et al. (2007) variable exposure Peto	2.6E-2	5.7E-2	3.6E-2	7.9E-2
North Carolina	Loomis et al. (2019) variable exposure Peto	5.08E-2	1.01E-1	7.06E-2	1.40E-1

Addressing inhalation cancer risks 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. Failing to account for those risks in the IUR necessarily underestimates the total cancer risk associated with chrysotile asbestos.

An adjustment factor for these other cancers has been developed by comparing the excess deaths from lung cancer with the number of excess deaths from other cancers.

Adjustment factor = (excess lung cancer + excess other cancer)/(excess lung cancer)

This approach has been applied to estimate adjustment factors for laryngeal and ovarian cancers using data from studies of chrysotile asbestos exposed workers that reported findings for these sites (see Appendix M). The adjustment factor for laryngeal cancer is 1.02 and the adjustment factor for ovarian cancer is 1.04. The combined adjustment factor for lung cancer to address other cancers is 1.06. Table 3-11 shows the lung cancer unit risks adjusted for other cancers.

Table 3-11. Addressing Risk of Other Cancers

Cohort	Source	Lung Cancer MLE Unit Risk (per f/cc)	Lung Cancer UB Unit Risk (per f/cc)	Adjusted Lung Cancer MLE Unit Risk (per f/cc)	Adjusted Lung Cancer UB Unit Risk (per f/cc)
South Carolina	Hein et al. (2007) linear	2.14E-1	3.03E-1	2.27E-1	3.21E-1
	Elliott et al. (2012) linear	2.54E-1	3.83E-1	2.69E-1	4.06E-1
	Elliott et al. (2012) exponential	5.99E-2	7.47E-2	6.35E-2	7.92E-2
North Carolina	Elliott et al. (2012) linear	1.30E-2	2.93E-2	1.38E-2	3.11E-2
	Elliott et al. (2012) exponential	1.08E-2	1.64E-2	1.14E-2	1.74E-2
	Loomis et al. (2009) exponential	1.19E-2	1.73E-2	1.26E-2	1.84E-2

3.2.3.8.2 Combining Lung Cancer Unit Risk and Mesothelioma Unit Risk

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

Because the estimated risks for mesothelioma and lung cancer were derived using maximum likelihood estimation, it follows from statistical theory that each of these estimates of risk is approximately normally distributed. For independent normal random variables, a standard deviation for a sum is easily derived from individual standard deviations, which are estimated from confidence intervals: standard deviation = (upper bound – central estimate) ÷ $Z_{0.95}$, where $Z_{0.95}$ is a standard normal quantile equal to 1.645. For normal random variables, the standard deviation of a sum is the square root of the sum of the squares of individual standard deviations. It is important to mention here that assumption of independence above is a theoretical assumption, but U.S. EPA (2014c) conducted an empirical evaluation and found that the assumption of independence in this case does not introduce substantial error.

In order to combine the unit risks, first obtain an estimate of the standard deviation (SD) of the sum of the individual unit risks as:

$$SD = \sqrt{ [((UB_{LC} - CE_{LC}) \div 1.645)^2 + ((UB_M - CE_M) \div 1.645)^2] }$$

Where,

UB – upper bound unit risk; CE – central estimate of unit risk; LC – lung cancer

M – mesothelioma

Then, the combined central estimate of risk (CCE) of either mesothelioma or lung cancer is $CCE = (CE_{LC} + CE_M)$ per f/cc, and the combined IUR is $CCE + SD \times 1.645$ per f/cc.

3.2.3.9 Derivation of Inhalation Unit Risk of Cancer Incidence

To illustrate the range of estimates in the estimates of the cancer incidence IUR, central risks and upper bounds for the combined IUR for North and South Carolina cohorts are presented in Table 3-12.

Table 3-12. Range of Estimates of Estimated Central Unit Risks and IURs for North and South 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)
North Carolina Cohort							
Elliott et al. (2012) Linear	1.38E-2	3.11E-2	Loomis et al. (2019) variable exposure Peto	7.06E-2	1.40E-1	0.084	0.16
Elliott et al. (2012) Exponential	1.14E-2	1.74E-2	Loomis et al. (2019) variable exposure Peto	7.06E-2	1.40E-1	0.082	0.15
Loomis et al. (2009) Exponential	1.26E-2	1.84E-2	Loomis et al. (2019) variable exposure Peto	7.06E-2	1.40E-1	0.083	0.15
South Carolina Cohort							
Hein et al. (2007) Linear	2.3E-1	3.2E-1	Berman and Crump (2008b) using the data from Hein et al. (2007) variable exposure Peto	3.6E-2	7.9E-2	0.26	0.37
Elliott et al. (2012) Linear	2.7E-1	4.1E-1	Berman and Crump (2008b) using the data from Hein et al. (2007) variable exposure Peto	3.6E-2	7.9E-2	0.31	0.45
Elliott et al. (2012) Exponential	6.35E-2	7.92E-2	Berman and Crump (2008b) using the data from Hein et al. (2007) variable exposure Peto	3.6E-2	7.9E-2	0.10	0.15

The values of the estimated IURs range from 0.15 per f/cc to 0.45 per f/cc (Table 3-12). There is a three-fold difference between lowest and highest IUR estimates – a very low range of model uncertainty in risk assessment. Because of low model uncertainty, EPA selected a median IUR value. Because there are six IUR values, the median is 0.155 per f/cc, which is between values 0.15 per f/cc and 0.16 per f/cc. Rounding to two significant digits, EPA selected 0.16 per f/cc based on modeling of North Carolina cohort (linear model for lung cancer and variable exposure Peto model for mesothelioma) as the chrysotile asbestos lifetime cancer incidence IUR, shown in Table 3-13.

Table 3-13. Estimates of Selected Central Risk and IUR of Cancer Incidence 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 Incidence Unit Risk (Lung Cancer + Meso)	Lifetime Cancer Incidence IUR (per f/cc)
NC Elliott et al. (2012) Linear	1.38E-2	3.11E-2	NC Loomis et al. (2019) variable exposure Peto	7.06E-2	1.40E-1	0.08	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 K.

Uncertainties in the cancer risk values are presented in Section 4.3.5 and 4.3.6.

3.2.4 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 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 susceptibility*. EPA addresses the subpopulations identified as relevant based on *greater exposure* in Section 2.3.3.

Factors affecting susceptibility examined in the available studies on asbestos include lifestage, gender, genetic polymorphisms and lifestyle factors. Additional susceptible subpopulations may include pregnant workers and children exposed prenatally. There is some evidence of genetic predisposition for mesothelioma related to having a germline mutation in BAP1 (Testa et al., 2011). Cigarette smoking in an important risk factor for lung cancer in the general population. In addition, lifestage is important relative to when the first exposure occurs. The long-term retention of asbestos fibers in the lung and the long latency period for the onset of asbestos-related respiratory diseases suggest that individuals

exposed earlier in life may be at greater risk to the eventual development of respiratory problems than those exposed later in life ([ATSDR, 2001a](#)). Appendix K of this RE illustrates this point in the IUR values for less than lifetime COUs. For example, the IUR for a two-year old child first exposed to chrysotile asbestos for 40 years is 1.30 E-1 while the IUR for a 20-year old first exposed to asbestos for 40 years is 4.86 E-2.

4 RISK CHARACTERIZATION

4.1 Environmental Risk

EPA made refinements to the conceptual models during the PF that resulted in the elimination of the terrestrial exposure, including biosolids, pathways. Thus, environmental hazard data sources on terrestrial organisms were determined to be out of scope and excluded from data quality evaluation and further consideration in the risk evaluation process.

In the PF, EPA identified the need to better determine whether there were releases to surface water and sediments from the COUs in this risk evaluation and whether risk estimates for aquatic (including sediment-dwelling) organisms should be included in the risk evaluation. Thus, reasonably available environmental hazard data/information on aquatic toxicity was carried through the systematic review process (data evaluation, data extraction and data integration).

EPA reviewed reasonably available information on environmental hazards posed by chrysotile asbestos. A total of four on-topic and in scope environmental hazard studies were identified for chrysotile asbestos and were determined to have acceptable data quality with overall high data quality (Appendix E). In addition, the *Systematic Review Supplemental File: Asbestos Data Quality Evaluation of Environmental Hazard Studies* presents details of the data evaluations for each study, including scores for each metric and the overall study score. These laboratory studies indicated reproductive, development, and sublethal effects at a concentration range of 10^4 - 10^8 fibers/L, which is equivalent to 0.01 to 100 MFL, to aquatic environmental receptors following chronic exposure to chrysotile asbestos.

On the exposure side of the equation, Table 2-1 presents asbestos monitoring results from the last two six-year Office of Water sampling programs (encompassing 1998 through 2011). Results of the next six-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 Part 1 of the risk evaluation for asbestos 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 COUs to determine the need to evaluate risk to aquatic and sediment-dwelling organisms. EPA continued to search EPA databases as well as the literature and engaged in a dialogue with industries to shed light on potential releases to water. The available information indicated that there were surface water releases of asbestos; however, it is unclear of the source of the asbestos and the fiber type present. In the draft Risk Evaluation, EPA concluded that, based on the reasonably available information in the published literature, provided by industries using asbestos, and reported in EPA databases, there were minimal or no releases of asbestos to surface water associated with the COUs that EPA is evaluating (see Appendix D). Therefore, in the draft Risk Evaluation, EPA concluded there was no unreasonable risk to aquatic or sediment-dwelling environmental organisms.

EPA has considered peer review and public comments on this conclusion and has decided to retain the finding made in the draft Risk Evaluation (*i.e.*, that there were minimal or no releases of asbestos to surface water associated with the COUs that EPA is evaluating in this Part 1 of the risk evaluation for

asbestos). EPA is confident that the minimal water release data available and reported more fully in the PF – and now presented again in Appendix D – cannot be attributed to chrysotile asbestos from the COUs in this Part 1 of the risk evaluation for asbestos. Assessing possible risk to aquatic organisms from the exposures described would not be reasonably attributed to the COUs. However, based on the decision to develop a scope and risk evaluation for legacy uses and associated disposals of asbestos (Part 2 of the final Risk Evaluation for asbestos), EPA expects to address the issue of releases to surface water based on those other uses.

Therefore, EPA concludes there is low or no risk to aquatic or sediment-dwelling organisms from exposure to chrysotile asbestos. In addition, terrestrial pathways, including biosolids, were excluded from analysis at the PF stage.

4.2 Human Health Risk

4.2.1 Risk Estimation Approach

EPA usually estimates extra cancer risks for repeated exposures to a chemical using an equation where $\text{Risk} = \text{Human Exposure (e.g., LADC)} \times \text{IUR}$. 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 progression. 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:

$$\text{ELCR} = \text{EPC} \cdot \text{TWF} \cdot \text{IUR}_{\text{LTL}}$$

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_{LTL} = Less than lifetime Inhalation Unit Risk per f/cc

[For example: the notation for the less than lifetime IUR could start at age 16 with 40 years duration $\text{IUR}_{(16,40)}$. 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 exposure²⁷, 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 microscopy²⁸.
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 m³ air during an 8-hour shift and non-workers breath 20 m³ in 24 hours. The hourly ratio of those breathing volumes is the volumetric adjustment factor for workers ($V_{\text{(worker)}}$) [(10/8) / (20/24) = 1.5]. Thus, for workers, the formula, $ELCR = EPC \cdot TWF \cdot IUR_{LTL}$, is extended as $ELCR = EPC \cdot TWF \cdot V \cdot IUR_{LTL}$.

$$TWF_{\text{(worker)}} = (8 \text{ hours} / 24 \text{ hours}) \cdot (240 \text{ days} / 365 \text{ days}) = 0.2192, \text{ and}$$

$$V_{\text{(worker)}} = 1.5$$

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:

$$IUR_{(16,40)} = 0.0612 \text{ per f/cc}$$

Based on these two factors, the excess lifetime cancer risk would be computed as:

²⁷ See U.S. EPA (1994) and Part F update to RAGS inhalation guidance [U.S. EPA \(2009\)](#).

²⁸ PCM-equivalent (PCMe) concentrations measured using TEM could also be used.

$$\text{ELCR} = \text{EPC in f/cc} \cdot 0.2192 \cdot 1.5 \cdot (0.0612 \text{ per f/cc})$$

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 0: Sensitivity Analysis of Exposures for DIY/Bystander Scenarios

For example:

- First exposure at age 0 with 78 years exposure: $\text{IUR}_{(0,78)} = 0.16$ per f/cc
- First exposure at age 16 with 62 years exposure: $\text{IUR}_{(16,62)} = 0.0641$ per f/cc
- First exposure at age 16 with 40 years exposure: $\text{IUR}_{(16,40)} = 0.0612$ per f/cc
- First exposure at age 16 with 20 years exposure: $\text{IUR}_{(16,20)} = 0.0468$ per f/cc
- First exposure at age 16 with 10 years exposure: $\text{IUR}_{(16,10)} = 0.0292$ 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 50th 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 50th 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 95th percentile. If the 95th percentile was not available, or if the full distribution was not known and the preferred statistics were not available, EPA estimated a maximum estimate in lieu of the high-end. Refer to **Table 2-24** and **Table 2-25** for occupational and consumer exposures, respectively.

EPA received occupational monitoring data for some of the COUs (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 1×10^{-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 1×10^{-4} for workers/ONUs and 1×10^{-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
Population of Interest and Exposure Scenario: <i>Workers</i>	Adult and adolescent workers (≥ 16 years old) exposed to chrysotile asbestos 8-hours/day for 240 days/year for working 40 years
Population of Interest and Exposure Scenario: <i>Occupational Non-Users (ONUs)</i>	Adults and adolescents of both sexes (≥ 16 years old) indirectly exposed to chrysotile asbestos while being in the same building during product use.
Health Effects of Concern, Concentration and Time Duration	<p><i>Cancer Health Effects: Cancer Incidence</i> Chrysotile Asbestos Cancer IUR (see Section 3.2.4)</p> <ul style="list-style-type: none"> • Lifetime Inhalation Unit Risk per f/cc (from Table 3-13) <ul style="list-style-type: none"> ○ Incidence of Cancer ○ 0.16 per f/cc • <u>Less than Lifetime</u> Inhalation Unit Risk per f/cc (IUR_{LTL}) <ul style="list-style-type: none"> ○ Uses values from life tables for different combination of starting age of exposure and duration (see Table APX-K-1) <p>Uses a Time Weighting Factor, this factor accounts for less-than-continuous exposure during a one-year exposure</p>
<p>Notes: Adult workers (≥ 16 years old) include both healthy female and male workers.</p>	

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
Population of Interest and Exposure Scenario: <i>Users (or Do-It-Yourselfers; DIY)</i>	<i>Consumer Users:</i> Adults and adolescents of both sexes (≥ 16 years old) exposed to chrysotile asbestos
Population of Interest and Exposure Scenario: <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
Health Effects of Concern, Concentration and Time Duration	<p><i>Cancer Health Effects: Incidence of Cancer</i> Chrysotile Asbestos Cancer IUR (see Section 3.2.4)</p> <ul style="list-style-type: none"> • Lifetime Inhalation Unit Risk per f/cc (from Table 3-13) <ul style="list-style-type: none"> ○ Incidence of Cancer ○ 0.16 per f/cc • <u>Less than Lifetime</u> Inhalation Unit Risk per f/cc (IUR_{LTL}) <ul style="list-style-type: none"> ○ Uses values from life tables for different combination of starting age of exposure and duration (see Table APX-K-1) <p>Uses a Time Weighting Factor, this factor accounts for less-than-continuous exposure during a one-year exposure</p>
<p>Re-entrainment²⁹ of asbestos can occur indoors in a garage. Both users and bystanders can be exposed.</p>	

²⁹ Settled Asbestos Dust Sampling and Analysis 1st Edition Steve M. Hays, James R. Millette CRC Press 1994

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 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), the 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 ³ APF to use for the risk calculation: 10 to 25
Sheet gasket stamping	Yes, provided by industry	Workers wear N95 filtering facepiece masks. A site-specific industrial hygiene evaluation determined that asbestos exposures were not high enough to require employee respirator use. (Note: the EPA risk estimates indicate that these workers should be wearing appropriate respirators, which is not an N95 mask. See footnote 1).	Half mask with N95 ¹ Hypothetical APF to use for the risk calculation: 10 to 25
Sheet gasket use (Chemical Production)	Yes, provided by industry	When replacing or servicing asbestos-containing sheet gaskets, workers in the titanium dioxide industry wear respirators, either airline respirators or cartridge respirators with P-100 HEPA filters.	Cartridge respirators with P-100 HEPA filters APF 10 Airline respirators: APF 10 APF to use for the risk calculation: 10
Oilfield brake blocks	Yes, from the literature	No information is reasonably available on respirator use for this COU. A safety data sheet obtained by EPA did not list respirator use (see Section 2.3.1.6.1).	Hypothetical APF to use for the risk calculation: 10 to 25
Aftermarket automotive	Yes, provided in	An unknown amount of respirator use occurs among these workers. OSHA’s asbestos standard	Hypothetical APF to use for the risk

brakes and clutches	literature	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.	calculation: 10 to 25
Other gasket vehicle friction product (UTV)	No ²	No information is reasonably available on respirator use for this COU, but worker activities are expected to be similar to those for aftermarket automotive brakes and clutches.	Hypothetical APF to use for the risk calculation: 10 to 25

¹ OSHA Asbestos Standard 1910.1001 states that negative pressure and filtering masks should not be used for asbestos exposure. The N95 is a negative pressure mask.

² EPA is using worker exposure data from the sheet gasket replacement in the chemical manufacturing industry as a surrogate for the exposures that may occur when workers service UTV friction products.

Source: OSHA (2006). Asbestos-Automotive Brake and Clutch Repair Work: Safety and Health Information Bulletin. SHIB 07-26-06. Available online at: <https://www.osha.gov/dts/shib/shib072606.html>.

³ See Table 2-7.

4.2.2 Risk Estimation for Workers: Cancer Effects Following Less than Lifetime Inhalation Exposures by Conditions of Use

This section presents the risk estimates for workers and ONUs exposed to chrysotile asbestos for the COUs included in this Part 1 of the risk evaluation for asbestos. EPA typically uses a benchmark cancer risk level of 1×10^{-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. Before presenting the estimates, discussion of how personal protective equipment (PPE) is considered is warranted.

For all COUs that were quantitatively assessed (except the Super Guppy scenario), there were risks to workers without respirators as PPE for both central and high-end exposure estimates; including those scenarios for which short-term exposure concentrations were available to include in the analysis. When PPE were applied (some known, some hypothetical), risks were not exceeded for some COUs (chlor-alkali and oilfield brake blocks) but they were exceeded for others (sheet gasket stamping – central and high-end, short-term exposure estimates; sheet gasket use – high-end exposure estimate; aftermarket auto brakes and other vehicle friction products – high-end and high-end short-term exposure estimates; and other gaskets [UTV] – high-end exposure estimates). Industry submissions indicated no use of respirators (*e.g.*, sheet gasket stampers using N95 respirators is not protective based on OSHA regulations), or respirators with an APF of 10 or 25 (chlor-alkali) and an APF of 10 (gasket use). It is important to note that based on published evidence for asbestos, nominal APF may not be achieved for all respirator users (see Section 2.3.1.2).

ONUs were not assumed to use PPE, so APFs do not apply in estimated risks to ONUs. Results show some COUs with cancer risk exceedances for ONUs for both central and high-end exposure estimates (sheet gasket use and other gaskets [UTV]). For all other quantitatively assessed COUs (except the Super Guppy scenario), at least one of the ONU scenarios exceeded the cancer risk benchmark. Thus, exceedances were observed for ONUs in every quantitatively assessed COU (except the Super Guppy scenario).

4.2.2.1 Risk Estimation for Cancer Effects Following Chronic Inhalation Exposures for Chlor-alkali Industry

Exposure data from the chlor-alkali industry were presented for two sampling durations (full shift and short-term) in Table 4-4. and Table 4-5., respectively (taken from Table 2-8). Short term samples were assumed to be approximately 30 minutes in duration. Data on exposure at central tendency (median) and the high-end (95th percentile) are presented along with the Excess Lifetime Cancer Risk (ELCR) for each exposure distribution.

Table 4-4. Excess Lifetime Cancer Risk for Chlor-alkali Industry Full Shift Workers and ONUs (Personal Samples) before consideration of PPE and any relevant APF

Occupational Exposure Scenario	Exposure Levels (f/cc)				ELCR (40 yr exposure starting at age 16 years)			
	Asbestos Worker		ONU ³⁰		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: Full shift exposure	0.0049	0.034	< 0.0025	≤0.008	9.9 E-5	6.8 E-4	5.0 E-5	1.6 E-4

Asbestos Workers: ELCR (Central Tendency) = 0.0049 f/cc • 0.2192 • 1.5 • 0.0612 per f/cc

Asbestos Workers: ELCR (High-end) = 0.034 f/cc • 0.2192 • 1.5 • 0.0612 per f/cc

ONU: ELCR (Central Tendency) = 0.0025 f/cc • 0.2192 • 1.5 • 0.0612 per f/cc

ONU: ELCR (High-end) = 0.008 f/cc • 0.2192 • 1.5 • 0.0612 per f/cc

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 1x10⁻⁴ was exceeded for the high-end exposure estimate; but not the central tendency exposure estimate. 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 29 CFR 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-minute values into a full 8-hour shift would

³⁰ 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 0.0025 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.

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 (f/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: Short-term exposures (exactly 30-minutes); and 30-minute short term samples within a full shift)*.	30 min value: 0.024	0.512	N/A	N/A	---	---	---	---
	8-hr TWA: 0.0061*	0.0639**	N/A	N/A	1.2 E-4	1.3 E-3	---	---

* This 8-hour TWA includes the 30-minute short-term exposure within an 8-hour full shift and is calculated as follows:

$$\{[(0.5 \text{ hour}) \cdot (0.024 \text{ f/cc}) + (7.5 \text{ hours}) \cdot (0.0049 \text{ f/cc from Table 4-2})] / 8 \text{ hours}\} = 0.0061 \text{ f/cc}$$

** This 8-hour TWA includes the 30-minute short-term exposure within an 8-hour full shift and is calculated as follows:

$$\{[(0.5 \text{ hour}) \cdot (0.512 \text{ f/cc}) + (7.5 \text{ hours}) \cdot (0.034 \text{ f/cc from Table 4-2})] / 8 \text{ hours}\} = 0.0639 \text{ f/cc.}$$

$$\text{ELCR}_{(\text{Central Tendency})} = \{[(0.5 \text{ hour}) \cdot \text{EPC}_{(30 \text{ minute})} + (7.5 \text{ hours}) \cdot \text{EPC}_{(\text{Full Shift})}] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0612.$$

$$\text{ELCR}_{(\text{High-end})} = \{[(0.5 \text{ hour}) \cdot \text{EPC}_{(30 \text{ minute})} + (7.5 \text{ hours}) \cdot \text{EPC}_{(\text{Full Shift})}] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0612.$$

$$\text{ELCR}_{(\text{Central Tendency})} = \{[(0.5 \text{ hour}) \cdot 0.024 + (7.5 \text{ hours}) \cdot 0.0049] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0612.$$

$$\text{ELCR}_{(\text{High-end})} = \{[(0.5 \text{ hour}) \cdot 0.512 + (7.5 \text{ hours}) \cdot 0.034] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0612.$$

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: Full shift exposure	9.9 E-6	6.8 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: Full shift exposure	3.9 E-6	2.7 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 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 1×10^{-4} was exceeded for ONUs for high-end exposures). Table 4-3 indicated the respirators that ACC reported to EPA are currently used by some 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.

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: Short-term exposures (exactly 30-minutes); and 30-minute short term samples within a full shift)	9.4 E-5	6.7 E-4

The central risks for 7.5 hours at 0.0049 f/cc with no APF were calculated and added to the 0.5 hour risk at 0.024 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 = $0.0049 \text{ f/cc} \cdot 0.2192 \cdot 1.5 \cdot 0.0612$
 Risk for 0.5 hours = $0.024 \text{ f/cc} \cdot 0.2192 \cdot 1.5 \cdot 0.0612 / (\text{APF of } 25)$
 Risk for 8 hours = $[7.5 \cdot 1.2 \text{ E-4} + 0.5 \cdot 2.4 \text{ E-5}] / 8$

High-end:
 Risk for 7.5 hours = $0.034 \text{ f/cc} \cdot 0.2192 \cdot 1.5 \cdot 0.0612$
 Risk for 0.5 hours = $0.512 \text{ f/cc} \cdot 0.2192 \cdot 1.5 \cdot 0.0612 / (\text{APF of } 25)$
 Risk for 8 hours = $[7.5 \cdot 8.4 \text{ E-4} + 0.5 \cdot 3.3 \text{ E-4}] / 8$

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: Short-term exposures (exactly 30-minutes); and 30-minute short term samples within a full shift).	1.0 E-5	9.0 E-5

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 0.024 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 0.024 f/cc and APF=25 and then sum divided by 8 hours.

Central : Risk for 7.5 hours = $0.005 \text{ f/cc} \cdot 0.2192 \cdot 1.5 \cdot 0.0612 / (\text{APF of } 10)$
 Risk for 0.5 hours = $0.024 \text{ f/cc} \cdot 0.2192 \cdot 1.5 \cdot 0.0612 / (\text{APF of } 25)$
 Risk for 8 hours = $[7.5 \cdot 1.2 \text{ E-}5 + 0.5 \cdot 2.4 \text{ E-}5]/8$

High-end: Risk for 7.5 hours = $0.034 \text{ f/cc} \cdot 0.2192 \cdot 1.5 \cdot 0.0612 / (\text{APF of } 10)$
 Risk for 0.5 hours = $0.512 \text{ f/cc} \cdot 0.2192 \cdot 1.5 \cdot 0.0612 / (\text{APF of } 25)$
 Risk for 8 hours = $[7.5 \cdot 8.4 \text{ E-}5 + 0.5 \cdot 3.3 \text{ E-}4]/8$

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: Short-term exposures (exactly 30-minutes); and 30-minute short term samples within a full shift).	4.9 E-6	5.1 E-5

Here the method is simply to divide the risks in Table 4-5 by 25:

Central Risk from Table 4-5 = $1.5\text{E-}4/25$
 High Risk from Table 4-5 = $1.3\text{E-}3/25$

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 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.

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

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 f/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.

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 (f/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	2.8 E-4	1.2 E-3	4.8 E-5	2.0 E-4

Asbestos Workers: $ELCR_{(Central\ Tendency)} = 0.014\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0612\ per\ f/cc$

Asbestos Workers: $ELCR_{(High-end)} = 0.059\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0612\ per\ f/cc$

ONU: $ELCR_{(Central\ Tendency)} = 0.0024\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0612\ per\ f/cc$

ONU: $ELCR_{(High-end)} = 0.01\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0612\ per\ f/cc$

Table 4-11. presents the inhalation cancer risk estimates for workers stamping asbestos-containing sheet gaskets and for ONUs exposed to asbestos. For asbestos workers, the benchmark cancer risk estimate of 1×10^{-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.

Table 4-12 presents the inhalation cancer risk estimates for workers stamping sheet gaskets and for ONUs exposed to asbestos, using an averaging of short-term exposures (assuming 30 minutes) and full shift exposures (7.5 hours per day of the full shift TWA exposure) based on monitoring data. The central tendency short-term exposure value for workers (0.024 f/cc) is the arithmetic mean of ten short-term measurements reported in a study of one worker at a company that stamps sheet gaskets containing asbestos. The high-end short-term exposure value for workers (0.059 f/cc) is the highest measured short-term exposure value from the available monitoring data. This exposure value occurred during a 30-minute sample.

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

Occupational Exposure Scenario	Exposure Levels (f/cc)				ELCR (40 yr exposure starting at age 16 years)			
	Asbestos Worker		ONU		Asbestos Worker		ONU	
	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end	Central Tendency	High-end
Sheet gasket stamping: Short-term exposures (~30- minute; and ~30-minute short term samples within a full shift)*.	30 min value: 0.024	0.059	0.0042	0.010	---	---	---	---
	8-hr TWA: 0.0146*	0.059*	0.0025*	0.010*	2.9 E-4	1.2 E-3	4.8 E-5	2.0 E-4

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

$$ELCR = \{[(0.5 \text{ hour}) \cdot EPC_{(30 \text{ minute})} + (7.5 \text{ hours}) \cdot EPC_{(Full Shift)}] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0612.$$

$$\text{Asbestos Worker: } ELCR_{(Central Tendency)} = \{[(0.5 \text{ hour}) \cdot 0.024 + (7.5 \text{ hours}) \cdot 0.014] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0612.$$

$$\text{Asbestos Worker: } ELCR_{(High-end)} = \{[(0.5 \text{ hour}) \cdot 0.059 + (7.5 \text{ hours}) \cdot 0.059] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0612.$$

For asbestos workers, the benchmark cancer risk estimate of 1×10^{-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.

Applying APFs to Data from Both Full Shift Work and Short-Term Work

ELCRs for workers who stamp sheet gaskets 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 4-13, Table 4-14., Table 4-15, and Table 4-16.

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	2.8 E-5	1.2 E-4

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	1.1 E-5	4.7 E-5

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

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

Occupational Exposure Scenario	ELCR (40 yr exposure starting at age 16 years)	
	Asbestos Worker	
	Central Tendency	High-end
Sheet gasket stamping: Short-term exposures	2.9 E-5	1.2 E-4

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: Short-term exposures	1.2 E-5	4.7 E-5

Tables 4-15 and 4-16 present the ELCR for short-term exposures for sheet gasket stamping workers. The two scenarios represented are (all hypothetical applications of an APF): (1) APF of 10 for short-term (30-minute exposure) and an APF of 10 for 7.5 hours; and (2) APF of 25 for both short-term and remaining 7.5 hours. The high-end risk estimates exceeded the benchmark for workers in only the first scenario presented. None of the other combinations of hypothetical APFs exceeded the benchmark.

4.2.2.3 Risk Estimation for Cancer Effects Following Chronic Inhalation Exposures for Sheet Gasket Use in Chemical Production

Exposure data from sheet gasket use (replacing gaskets) – using titanium dioxide production as an example - were presented for 8-hour full shift exposures in Table 2-11. These data are based on reports from ACC for gasket removal/replacement at titanium dioxide facilities. The 8-hour TWA exposures assume that the workers removed gaskets throughout the day during maintenance. Data on the exposure at the central and high-end estimates are presented along with the ELCR for each exposure distribution in Table 4-6. The high-end value for 8-hr TWA worker exposure (0.094) is based on the highest

exposure measurement (see Section 2.3.1.4.5). No data are available for evaluating worker short-term exposures for this COU (see 2.3.1.4.5).

Table 4-17. Excess Lifetime Cancer Risk for Sheet Gasket Use in Chemical Production (using data from titanium dioxide production), 8-hour TWA (from Table 2-11., Personal Samples) before consideration of PPE and any relevant APF

Occupational Exposure Scenario	Exposure Levels (f/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: 8-hr TWA exposure	0.026	0.094	0.005	0.016	5.2 E-4	1.9 E-3	1.0 E-4	3.2 E-4

Asbestos Workers: $ELCR_{(Central\ Tendency)} = 0.026\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0612\ per\ f/cc$

Asbestos Workers: $ELCR_{(High-end)} = 0.094\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0612\ per\ f/cc$

ONU: $ELCR_{(Central\ Tendency)} = 0.005\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0612\ per\ f/cc$

ONU: $ELCR_{(High-end)} = 0.016\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0612\ per\ f/cc$

Table 4-17. presents the inhalation cancer risk estimates based on data for workers replacing sheet gaskets in titanium dioxide production and for ONUs exposed to asbestos. For asbestos workers, the benchmark cancer risk estimate of 1×10^{-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: 8-hr TWA exposure	5.2 E-5	1.9 E-4

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: 8-hr TWA exposure	2.1 E-5	7.6 E-5

In both scenarios, the risk estimates for the workers are below the benchmark of 1×10^{-4} for the central tendency risk estimate. The benchmark is exceeded 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. 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 one 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.

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 (f/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	---	6.0 E-4	---	4.0 E-4	---

Asbestos Workers: $ELCR_{(Central\ Tendency)} = 0.03\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0612\ per\ f/cc$

ONU: $ELCR_{(Central\ Tendency)} = 0.02\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0612\ per\ f/cc$

Table 4-20. presents the inhalation cancer risk estimates for workers around brake block use and for ONUs exposed to asbestos. For workers and ONUs, the benchmark cancer risk estimate of 1×10^{-4} was exceeded for central tendency. No high-end exposures were available for this activity. Estimates exceeding the benchmark are shaded in pink and bolded.

Applying APFs

ELCRs for workers who work near oil field brake blocks exposed to asbestos using PPE with hypothetical APFs of 10 and 25 applied for 8-hour TWAs are presented in Table 4-21. and Table 4-22.

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)

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	6.0 E-5	---

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)

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	2.4 E-5	---

In both scenarios, the risk estimates for the workers using either the hypothetical APF of 10 or 25 are below the benchmark of 1 E-4.

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 (f/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	0.001	0.002	1.2 E-4	1.9 E-3	2.0 E-5	4.0 E-5

Asbestos Workers: ELCR (Central Tendency) = 0.006 f/cc • 0.2192 • 1.5 • 0.0612 per f/cc

Asbestos Workers: ELCR (High-end) = 0.094 f/cc • 0.2192 • 1.5 • 0.0612 per f/cc

ONU: ELCR (Central Tendency) = 0.001 f/cc • 0.2192 • 1.5 • 0.0612 per f/cc

ONU: ELCR (High-end) = 0.002 f/cc • 0.2192 • 1.5 • 0.0612 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 1×10^{-4} was exceeded for central tendency and high-end. For ONUs, the benchmark of 1 E-4 was not exceeded. 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 1×10^{-4} was exceeded for both central tendency and high-end exposure estimates. For ONUs, the cancer benchmark was not exceeded. Estimates exceeding the benchmark are shaded in pink and bolded.

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 (f/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	0.001	0.002	---	---	---	---
	8-hr TWA: 0.006*	0.140*	0.001*	0.002*	1.2 E-4	2.8 E-3	2.0 E-5	4.0 E-5

*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.

$$ELCR = \{[(0.5 \text{ hour}) \cdot EPC_{(30 \text{ minute})} + (7.5 \text{ hours}) \cdot EPC_{(Full Shift)}] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0612.$$

$$\text{Asbestos Worker: } ELCR_{(Central Tendency)} = \{[(0.5 \text{ hour}) \cdot EPC_{(30 \text{ minute})} + (7.5 \text{ hours}) \cdot EPC_{(Full Shift)}] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0612.$$

$$\text{Asbestos Worker: } ELCR_{(High-end)} = \{[(0.5 \text{ hour}) \cdot EPC_{(30 \text{ minute})} + (7.5 \text{ hours}) \cdot EPC_{(Full Shift)}] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0612.$$

$$\text{Asbestos Worker: } ELCR_{(Central Tendency)} = \{[(0.5 \text{ hour}) \cdot 0.006 + (7.5 \text{ hours}) \cdot 0.006] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0612.$$

$$\text{Asbestos Worker: } ELCR_{(High-end)} = \{[(0.5 \text{ hour}) \cdot 0.836 + (7.5 \text{ hours}) \cdot 0.094] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0612.$$

$$\text{ONU: } ELCR_{(Central Tendency)} = \{[(0.5 \text{ hour}) \cdot 0.001 + (7.5 \text{ hours}) \cdot 0.001] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0612.$$

$$\text{ONU: } ELCR_{(High-end)} = \{[(0.5 \text{ hour}) \cdot 0.002 + (7.5 \text{ hours}) \cdot 0.002] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0612.$$

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 4-25, Table 4-26, 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	1.2 E-5	1.9 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	4.8 E-6	7.6 E-5

For asbestos workers wearing a hypothetical respirator at APF 10, the benchmark cancer risk estimate of 1×10^{-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. 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	1.2 E-5	2.8 E-4

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	4.8 E-6	1.1 E-4

Table 4-27. and Table 4-28. display the ELCRs for short-term exposures for workers repairing or replacing auto brakes and using hypothetical APFs of 10 and 25. For asbestos workers exposed to asbestos, the benchmark cancer risk estimate of 1×10^{-4} was exceeded for high-end exposures, but not central tendency exposures, after consideration of both hypothetical APF 10 and APF 25. Estimates exceeding the benchmark are shaded in pink and bolded.

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

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

In addition, as noted in Section 2.3.1.8.2, there is a limited use of asbestos-containing brakes for a special, large transport plane (the “Super-Guppy”) by the National Aeronautics and Space Administration (NASA).

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 (f/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 in exported cars: 8-hour TWA exposure	0.006	0.094	0.001	0.002	1.2 E-4	1.9 E-3	2.0 E-5	4.0 E-5

Asbestos Workers: $ELCR_{(Central\ Tendency)} = 0.006\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0612\ per\ f/cc$

Asbestos Workers: $ELCR_{(High-end)} = 0.094\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0612\ per\ f/cc$

ONU: $ELCR_{(Central\ Tendency)} = 0.001\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0612\ per\ f/cc$

ONU: $ELCR_{(High-end)} = 0.002\ f/cc \cdot 0.2192 \cdot 1.5 \cdot 0.0612\ per\ f/cc$

Table 4-29 presents the inhalation cancer risk estimates for workers repairing and replacing auto brakes and clutches in exported cars and for ONUs exposed to asbestos. For workers, the benchmark cancer risk estimate of 1×10^{-4} was exceeded for central tendency and high-end. For ONUs, the benchmark of 1×10^{-4} was not exceeded. Estimates exceeding the benchmark are shaded in pink and bolded.

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 (f/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 in exported cars: short-term exposure (~30-minute; and ~30-minute short term samples within a full shift)*.	30 min value: 0.006	0.836	0.001	0.002	---	---	---	---
	8-hr TWA: 0.006*	0.140*	0.001*	0.002*	1.2 E-4	2.8 E-4	2.0 E-5	4.0 E-5

*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: $ELCR_{(Central Tendency)} = \{[(0.5 \text{ hour}) * EPC_{(30 \text{ minute})} + (7.5 \text{ hours}) * EPC_{(Full Shift)}] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0612$.

Asbestos Worker: $ELCR_{(High-end)} = \{[(0.5 \text{ hour}) * EPC_{(30 \text{ minute})} + (7.5 \text{ hours}) * EPC_{(Full Shift)}] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0612$

Asbestos Worker: $ELCR_{(Central Tendency)} = \{[(0.5 \text{ hour}) * 0.006 + (7.5 \text{ hours}) * 0.006] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0612$

Asbestos Worker: $ELCR_{(High-end)} = \{[(0.5 \text{ hour}) * 0.836 + (7.5 \text{ hours}) * 0.094] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0612$

ONU: $ELCR_{(Central Tendency)} = \{[(0.5 \text{ hour}) * 0.001 + (7.5 \text{ hours}) * 0.001] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0612$

ONU: $ELCR_{(High-end)} = \{[(0.5 \text{ hour}) * 0.002 + (7.5 \text{ hours}) * 0.002] / 8 \text{ hours}\} \cdot 0.2192 \cdot 1.5 \cdot 0.0612$

Table 4-30 presents the inhalation cancer risk estimates for workers repairing or replacing aftermarket auto brakes and clutches in exported cars 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 1×10^{-4} was exceeded for both central tendency and high-end exposure estimates. For ONUs, the benchmark of 1×10^{-4} was not exceeded. Estimates exceeding the benchmark are shaded in pink and bolded.

Applying APFs to Data from Both Full Shift Work and Short-Term Work

ELCRs for workers who repair/replace auto brakes and clutches in exported cars 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 4-31, Table 4-32, Table 4-33 and Table 4-34.

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 in exported cars: 8-hour TWA exposure	1.2 E-5	1.9 E-4

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 in exported cars: 8-hour TWA exposure	4.8 E-6	7.6 E-5

For asbestos workers wearing a hypothetical respirator at APF 10, the benchmark cancer risk estimate of 1×10^{-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 in exported cars: short-term exposure	1.2 E-5	2.8 E-4

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 in exported cars: short-term exposure	4.8 E-6	1.1 E-4

Table 4-33 and Table 4-34 display the ELCRs for short-term exposures for workers repairing or replacing auto brakes in exported cars and using hypothetical APFs of 10 and 25. For asbestos workers exposed to asbestos, the benchmark cancer risk estimate of 1×10^{-4} was exceeded for high-end exposures, but not central tendency exposures, after consideration of both hypothetical APF 10 and APF 25. Estimates exceeding the benchmark are shaded in pink and bolded.

4.2.2.7 Risk Estimation for Cancer Effects Following Chronic Exposures for Replacing Brakes on the NASA Large Transport Plane (i.e., Super Guppy)

Table 4-35. Excess Lifetime Cancer Risk for Replacing Brakes on the NASA Large Transport Plane (i.e., Super Guppy) in an Occupational Setting, 8-hour TWA Exposure (from Table 2-17) before consideration of PPE and any relevant APF

Occupational Exposure Scenario	Exposure Levels (f/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 aircraft parts in the NASA Large Transport Plane: 8-hour TWA exposure	0.003	0.0089	N/A	N/A	3.7 E-7	1.1 E-6	N/A	N/A

$TWF_{\text{USER Brakes}} (3.3\text{-hours on } 3.6 \text{ days every year}) = (3.3 \text{ hours} / 24 \text{ hours}) \cdot (3.6 \text{ days} / 365 \text{ days}) = 0.001356$

User: $ELCR_{\text{(Central Tendency)}} = 0.003 \text{ f/cc} \cdot 0.001356 \cdot 1.5 \cdot 0.0612 \text{ per f/cc}$

User: $ELCR_{\text{(High-end)}} = 0.0089 \text{ f/cc} \cdot 0.001356 \cdot 1.5 \cdot 0.0612 \text{ per f/cc}$

Table 4-36. Excess Lifetime Cancer Risk for Replacing Brakes on the NASA Large Transport Plane (i.e., Super Guppy) in an Occupational Setting, Short-term Exposures Within an 8-hour TWA (from Table 2-17) before consideration of PPE and any relevant APF

Occupational Exposure Scenario	Exposure Levels (f/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 aircraft parts: short-term exposure (~30-minute; and ~30-minute short term samples within a full shift)*.	30 min value: 0.022 8-hr TWA: 0.0059*	0.045 0.014*	N/A	N/A	--- 7.3 E-7	--- 1.7 E-6	N/A	N/A

Central Tendency Exposure includes the 30-minute short-term exposure within each 3.3 hour brake change as follows:

$$\{[(0.5 \text{ hour}) \cdot (0.022 \text{ f/cc}) + (2.8 \text{ hours}) \cdot (0.003 \text{ f/cc})]/3.3 \text{ hours}\} = 0.0059 \text{ f/cc}$$

High End Exposure includes the 30-minute short-term exposure within each 3.3 hour brake change as follows:

$$\{[(0.5 \text{ hour}) \cdot (0.045 \text{ f/cc}) + (2.8 \text{ hours}) \cdot (0.0089 \text{ f/cc})]/3.3 \text{ hours}\} = 0.014 \text{ f/cc}$$

$$TWF_{\text{USER Brakes}} = (3.3 \text{ hours} / 24 \text{ hours}) \cdot (3.6 \text{ days} / 365 \text{ days}) = 0.001356$$

$$\text{Worker: ELCR}_{(\text{Central Tendency})} = 0.0059 \text{ f/cc} \cdot 0.001356 \cdot 1.5 \cdot 0.0612 \text{ per f/cc}$$

$$\text{Worker: ELCR}_{(\text{High-end})} = 0.014 \text{ f/cc} \cdot 0.001356 \cdot 1.5 \cdot 0.0612 \text{ per f/cc}$$

These risk estimates fall below the benchmark for both the central tendency and high-end. Respirator usage is also not required by NASA because measured exposures were below applicable occupational exposure limits (NASA, 2020a) and the work is performed in a special, ventilated walk-in booth specifically built for this activity (see Section 2.3.1.8.2). Because the risk estimates already do not show exceedances, there is no reason to consider or incorporate hypothetical PPE and an APF. Despite respiratory protection not being required, NASA informed EPA that some certified technicians choose to use half mask air-purifying respirator with P-100 particulate filters when replacing the brake pads.

4.2.2.8 Risk Estimation for Cancer Effects Following Inhalation Exposures for Gasket Installation/Service in UTVs

Multiple publications (see Section 2.3.2.2) report on occupational exposures associated with installing and servicing gaskets in automobiles. The exposure data used for this COU are presented in Table 2-23. Data on the exposure at the central and high-end estimates are presented along with the ELCR for each exposure distribution in Table 4-35.

Table 4-37. Excess Lifetime Cancer Risk for UTV Gasket Installation/Service in an Occupational Setting, 8-hour TWA Exposure (from Table 2-23.) before consideration of PPE and any relevant APF

Occupational Exposure Scenario	Exposure Levels (f/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	4.8 E-4	1.3 E-3	1.0 E-4	3.0 E-4

Asbestos Workers: ELCR (Central Tendency) = 0.024 f/cc • 0.2192 • 1.5 • 0.0612 per f/cc

Asbestos Workers: ELCR (High-end) = 0.066 f/cc • 0.2192 • 1.5 • 0.0612 per f/cc

ONU: ELCR (Central Tendency) = 0.005 f/cc • 0.2192 • 1.5 • 0.0612 per f/cc

ONU: ELCR (High-end) = 0.015 f/cc • 0.2192 • 1.5 • 0.0612 per f/cc

Table 4-35. presents the inhalation cancer risk estimates for workers installing and/or servicing gaskets in utility vehicles and for ONUs exposed to asbestos. For both workers and ONUs, the benchmark cancer risk estimate of 1×10^{-4} was exceeded for both central tendency and high-end exposures. Estimates exceeding the benchmark are shaded in pink and bolded.

Applying APFs

ELCRs for workers who install/service gaskets in UTVs exposed to asbestos using PPE with hypothetical APFs of 10 and 25 applied for 8-hour TWAs are presented in Table 4-36. and Table 4-37.

Table 4-38. 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)	4.8 E-5	1.3 E-4

Table 4-39. 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 gasket repair/replacement in vehicles: 8-hr TWA exposure)	1.9 E-5	5.3 E-5

For asbestos workers using respirators with a hypothetical APF of 10, the benchmark cancer risk estimate of 1×10^{-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. Estimates exceeding the benchmark are shaded in pink and bolded.

4.2.2.8. Summary of Risk Estimates for Cancer Effects for Occupational Inhalation Exposure Scenarios for All COUs

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 1×10^{-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 4-40. 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 ^e)	Cancer Risk Estimates (with APF=25 ^e)
Diaphragms for chlor-alkali industry Section 4.2.2.1.	Worker	Central Tendency (8-hr)	9.9 E-5	9.9 E-6	3.9 E-6
		High-end (8-hr)	6.8 E-4	6.8 E-5	2.7 E-5
		Central Tendency short term	1.2 E-4 9.4 E-5^a	1.0 E-5 ^d	4.9 E-6 ^b
		High-end short term	1.3 E-3 6.7 E-4^a	9.0 E-5 ^d	5.1 E-5 ^b
	ONU	Central Tendency (8-hr)	5.0 E-5	N/A	N/A
		High-end (8-hr)	1.6 E-4	N/A	N/A
Asbestos Sheets – Gasket Stamping Section 4.2.2.2	Worker	Central Tendency (8-hr)	2.8 E-4	2.8 E-5	1.1 E-5
		High-end (8-hr)	1.2 E-3	1.2 E-4	4.7 E-5
		Central Tendency short term	2.9 E-4	2.9 E-5 ^e	1.2 E-5 ^f
		High-end short term	1.2 E-3	1.2 E-4^e	4.7 E-5 ^f
	ONU	Central Tendency (8-hr)	4.8 E-5	N/A	N/A
		High-end (8-hr)	2.0 E-4	N/A	N/A
		Central Tendency short term	5.1 E-5	N/A	N/A
		High-end short term	2.0 E-4	N/A	N/A
Asbestos Sheet Gaskets – use (based on repair/replacement data from TiO ₂ industry) Section 4.2.2.3	Worker	Central Tendency (8-hr)	5.2 E-4	5.2 E-5	2.1 E-5
		High-end (8-hr)	1.9 E-3	1.9 E-4	7.6 E-5
	ONU	Central Tendency (8-hr)	1.0 E-4	N/A	N/A
		High-end (8-hr)	3.2 E-4	N/A	N/A
Oil Field Brake Blocks Section 4.2.2.4	Worker	Central Tendency (8-hr)	6.0 E-4	6.0 E-5	2.4 E-5
	ONU	Central Tendency (8-hr)	4.0 E-4	N/A	N/A
Aftermarket Auto Brakes Section 4.2.2.5	Worker	Central Tendency (8-hr)	1.2 E-4	1.2 E-5	4.8 E-6
		High-end (8-hr)	1.9 E-3	1.9 E-4	7.6 E-5
		Central Tendency short-term	1.2 E-4	1.2 E-5 ^e	4.8 E-6 ^f
		High-end short-term	2.8 E-3	2.8 E-4^e	1.1 E-4^f
	ONU	Central Tendency (8-hr)	2.0 E-5	N/A	N/A

		High-end (8-hr)	4.0 E-5	N/A	N/A
		Central Tendency short-term	2.0 E-5	N/A	N/A
		High-end short-term	4.0 E-5	N/A	N/A
Other Vehicle Friction Products Section 4.2.2.6	Worker	Central Tendency (8-hr)	1.2 E-4	1.2 E-5	4.8 E-6
		High-end (8-hr)	1.9 E-3	1.9 E-4	7.6 E-5
		Central Tendency short term	1.2 E-4	1.2 E-5 ^e	4.8 E-6 ^f
		High-end w short term	2.8 E-3	2.8 E-4^e	1.1 E-4^f
	ONU	Central Tendency (8-hr)	2.0 E-5	N/A	N/A
		High-end (8-hr)	4.0 E-5	N/A	N/A
		Central Tendency short-term	2.0 E-5	N/A	N/A
		High-end short-term	4.0 E-5	N/A	N/A
Other Vehicle Friction Products: Super Guppy Section 4.2.2.6	Worker	Central Tendency (8-hr)	3.7 E-7	N/A	N/A
		High-end (8-hr)	1.1 E-6	N/A	N/A
		Central Tendency (short-term)	7.3 E-7	N/A	N/A
		High-end (short-term)	1.7 E-7	N/A	N/A
Other Gaskets – Utility Vehicles Section 4.2.2.7	Worker	Central Tendency (8-hr)	4.8 E-4	4.8 E-5	1.9 E-5
		High-end (8-hr)	1.3 E-3	1.3 E-4	5.3 E-5
	ONU	Central Tendency (8-hr)	1.0 E-4	N/A	N/A
		High-end (8-hr)	3.0 E-4	N/A	N/A

N/A: Not Assessed; ONUs are not assumed to wear respirators

^aNo APF applied for 7.5 hours, APF of 25 applied for 30 minutes.

^bAPF 25 applied for both 30 mins and 7.5 hours

^c 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.

^d APF 25 for 30 minutes, APF 10 for 7.5 hours

^e APF 10 for 30 minutes, APF 10 for 7.5 hours

^f APF 25 for 30 minutes, APF 25 for 7.5 hours

For workers, with the exceptions of the central tendency, full shift chlor-alkali worker and all scenarios assessed for brake pad replacements for the NASA Super Guppy, cancer risks were indicated for all quantitatively assessed conditions of use under high-end and central tendency exposure scenarios when PPE was not used. With the use of PPE at APF of 10, most risks were reduced but still persisted for chlor-alkali (for high-end estimates when short-term exposures were considered), sheet gasket stamping (high-end only), sheet gasket use (high-end only), auto brake replacement (high-end only for 8-hour and central and high-end estimates when short-term exposures are considered), and UTV gasket replacement (high-end only). When an APF of 25 was applied, risk was still indicated for the auto brakes and other vehicle friction products high-end short-term exposure scenarios.

For ONUs, the benchmark for risk is exceeded for most high-end estimates and most central tendency estimates. The exceptions for central tendency exceedances are for the following COUs: chlor-alkali (8-hour), sheet gasket stamping (8-hour), and auto brake replacement (8-hour and short-term exposure scenarios). The exceptions for high-end exceedances are for the aftermarket auto brakes and other vehicle friction products scenarios.

4.2.3 Risk Estimation for Consumers: Cancer Effects by Conditions of Use

4.2.3.1 Risk Estimation for Cancer Effects Following Episodic Inhalation Exposures for DIY Brake Repair/Replacement

EPA assessed chronic chrysotile exposures for the DIY (consumer) and bystander brake repair/replacement scenario based on repeated exposures resulting from recurring episodic exposures from active use of chrysotile asbestos related to DIY brake-related activities. These activities include concomitant exposure to chrysotile asbestos fibers which are reasonably anticipated to remain within indoor and outdoor use facilities. It is well-understood that asbestos fibers in air will settle out in dust and become re-entrained in air during any changes in air currents or activity within the indoor and outdoor use facilities. On the other hand, in occupational settings, regular air sampling would capture both new and old fibers and have industrial hygiene practices in place to reduce exposures.

EPA used the following data on exposure frequency and duration, making assumptions when needed:

- Exposure frequency of active use of chrysotile asbestos related to DIY brake repair and replacement of 3 hours on 1 day every 3 years or 0.33 days per year. This is based on the information that brakes are replaced every 35,000 miles, and an average number of miles driven per year per driver in the U.S. of 13,476 miles/year ([U.S. DOT, 2018](#)).
- An estimate assuming a single brake change at age 16 years old is presented.
- 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.³¹ According to Table 16-16 of the Handbook, the median time ‘Doers’ spent in garages is approximately one hour per day. The 95th 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 95th 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 be 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 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 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.

³¹ This Part 1 of the risk evaluation for asbestos uses the term “consumer” or Do-It-Yourselfer (DIY) or DIY mechanic to refer to the “doer” referenced in the Exposure Factor Handbook.

- 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 work³². 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

$$ELCR_{DIY\ Brakes} = EPC_{DIY\ Brakes} \cdot TWF_{DIY\ Brakes} \cdot IUR_{LTL(DIY\ Brakes)} +$$

$$EPC_{Concomitant\ Exposures} \cdot TWF_{Concomitant\ Exposures} \cdot IUR_{LTL(Concomitant\ Exposures)}$$

$$TWF_{DIY\ Brakes\ (3\text{-hours\ on\ 1\ day\ every\ 3\ years)} = (3/24) \cdot (1/3) \cdot (1/365) = 0.0001142$$

$$IUR_{LTL(DIY\ Brakes)} = IUR_{(16,62)} = 0.0641\ \text{per f/cc}$$

$$TWF_{Concomitant\ Exposures\ (1\text{-hour\ per\ day\ every\ day)} = (1/24) \cdot (365/365) = 0.04167$$

$$IUR_{LTL(Concomitant\ Exposures)} = IUR_{(16,62)} = 0.0641\ \text{per f/cc}$$

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 2-31 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 50th 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 brakes every 3 years (Table 4-39). Estimates exceeding the benchmark are shaded in pink and bolded.

³² 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.

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 (Table 4-40). The risk estimates note that the benchmark is exceeded for both these alternative estimates.

Table 4-41. Excess Lifetime Cancer Risk for Indoor DIY Brake/Repair Replacement with Compressed Air Use for Consumers and Bystanders (exposures from Table 2-31 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 (f/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 every 3 years with compressed air)	0.0445	0.4368	0.0130	0.0296	3.6 E-5	3.5 E-4	2.6 E-5	6.0 E-5

$$TWF_{\text{Concomitant Exposures (1 hour per day every day)}} = (1/24) \cdot (365/365) = 0.04167$$

$$IUR_{(16,62)} = 0.0641; IUR_{(\text{Lifetime})} = 0.16$$

$$\text{DIY User: ELCR}_{(\text{Central Tendency})} = 0.0445 \text{ f/cc} \cdot 0.0001142 \cdot 0.0641 \text{ per f/cc} + 0.0445 \cdot 0.3 \cdot 0.04167 \cdot 0.0641$$

$$\text{DIY User: ELCR}_{(\text{High-end})} = 0.4368 \text{ f/cc} \cdot 0.0001142 \cdot 0.0641 \text{ per f/cc} + 0.4368 \cdot 0.3 \cdot 0.04167 \cdot 0.0641$$

$$\text{DIY Bystander: ELCR}_{(\text{Central Tendency})} = 0.013 \text{ f/cc} \cdot 0.0001142 \cdot 0.16 \text{ per f/cc} + 0.013 \cdot 0.3 \cdot 0.04167 \cdot 0.16$$

$$\text{DIY Bystander: ELCR}_{(\text{High-end})} = 0.0296 \text{ f/cc} \cdot 0.0001142 \cdot 0.16 \text{ per f/cc} + 0.0296 \cdot 0.3 \cdot 0.04167 \cdot 0.16$$

Table 4-42. Excess Lifetime Cancer Risk for Indoor DIY Brake/Repair Replacement with Compressed Air Use for Consumers for 20-year duration (exposures from Table 2-31 without a reduction factor) with Exposures at 30% of 3-hour User Concentrations between Brake/Repair Replacement (Consumers 1 hour/day spent in garage)

Consumer Exposure Scenario	Exposure Levels (f/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	2.6 E-5	2.6 E-4	1.7 E-5	3.9 E-5

$$TWF_{\text{Concomitant Exposures (1 hour per day every day)}} = (1/24) \cdot (365/365) = 0.04167$$

$$IUR_{(16,20)} = 0.0468; IUR_{(0,20)} = 0.1057$$

$$\text{DIY User: ELCR}_{(\text{Central Tendency})} = 0.0445 \text{ f/cc} \cdot 0.0001142 \cdot 0.0468 \text{ per f/cc} + 0.0445 \cdot 0.3 \cdot 0.04167 \cdot 0.0468$$

$$\text{DIY User: ELCR}_{(\text{High-end})} = 0.4368 \text{ f/cc} \cdot 0.0001142 \cdot 0.0468 \text{ per f/cc} + 0.4368 \cdot 0.3 \cdot 0.04167 \cdot 0.0468$$

$$\text{DIY Bystander: ELCR}_{(\text{Central Tendency})} = 0.013 \text{ f/cc} \cdot 0.0001142 \cdot 0.1057 \text{ per f/cc} + 0.013 \cdot 0.3 \cdot 0.04167 \cdot 0.1057$$

$$\text{DIY Bystander: ELCR}_{(\text{High-end})} = 0.0296 \text{ f/cc} \cdot 0.0001142 \cdot 0.1057 \text{ per f/cc} + 0.0296 \cdot 0.3 \cdot 0.04167 \cdot 0.1057$$

For Table 4-41, users were assumed to spend eight hours per day in their garages based on the 95th percentile estimate in the EPA Exposure Factors Handbook (Table 16-16 in the 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 during use of compressed air. Estimates exceeding the benchmark are shaded in pink and bolded.

Table 4-43. Excess Lifetime Cancer Risk for Indoor DIY Brake/Repair Replacement with Compressed Air Use for Consumers and Bystanders (exposures from Table 2-31 without a reduction factor) with Exposures at 30% of 3-hour User Concentrations between Brake/Repair Replacement (Consumers 8-hours/day spent in garage; Bystanders 1 hour/day)

Consumer Exposure Scenario	Exposure Levels (f/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 with compressed air)	0.0445	0.4368	0.0130	0.0296	2.9 E-4	2.8 E-3	2.6 E-5	6.0 E-5

$$TWF_{\text{Concomitant Exposures (8-hours per day every day)}} = (8/24) \cdot (365/365) = 0.3333$$

$$IUR_{(16,62)} = 0.0641; IUR_{(\text{Lifetime})} = 0.16$$

$$\text{DIY User: ELCR}_{(\text{Central Tendency})} = 0.0445 \text{ f/cc} \cdot 0.0001142 \cdot 0.0641 \text{ per f/cc} + 0.0445 \cdot 0.3 \cdot 0.3333 \cdot 0.0641$$

$$\text{DIY User: ELCR}_{(\text{High-end})} = 0.4368 \text{ f/cc} \cdot 0.0001142 \cdot 0.0641 \text{ per f/cc} + 0.4368 \cdot 0.3 \cdot 0.3333 \cdot 0.0641$$

$$\text{DIY Bystander: ELCR}_{(\text{Central Tendency})} = 0.013 \text{ f/cc} \cdot 0.0001142 \cdot 0.16 \text{ per f/cc} + 0.013 \cdot 0.3 \cdot 0.04167 \cdot 0.16$$

$$\text{DIY Bystander: ELCR}_{(\text{High-end})} = 0.0296 \text{ f/cc} \cdot 0.0001142 \cdot 0.16 \text{ per f/cc} + 0.0296 \cdot 0.3 \cdot 0.04167 \cdot 0.16$$

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 equal to a limit of one year at the 3-hour concentration divided by 10 years).

Table 4-44. 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 2-31 without a reduction factor) (Consumers 1 hour/day spent in garage; Bystanders 1 hour/day)

Consumer Exposure Scenario	Exposure Levels (f/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	5.4 E-6	5.3 E-5	3.4 E-6	7.8 E-6

$$TWF_{\text{Concomitant Exposures (1 hour per day every day)}} = (1/24) \cdot (365/365) = 0.04167$$

$$IUR_{(16,10)} = 0.0292; IUR_{(0,10)} = 0.0634$$

$$\text{DIY User: ELCR}_{(\text{Central Tendency})} = 0.0445 \text{ f/cc} \cdot 0.000005524 \cdot 0.0292 \text{ per f/cc} + 0.0445 \cdot 0.1 \cdot 0.04167 \cdot 0.0292$$

DIY User: ELCR (High-end) = 0.4368 f/cc • 0.000005524 • 0.0292 per f/cc + 0.4368 • 0.1 • 0.04167 • 0.0292
 DIY Bystander: ELCR (Central Tendency) = 0.013 f/cc • 0.000005524 • 0.0634 per f/cc + 0.013 • 0.1 • 0.04167 • 0.0634
 DIY Bystander: ELCR (High-end) = 0.0296 f/cc • 0.000005524 • 0.0634 per f/cc + 0.0296 • 0.1 • 0.04167 • 0.0634

Exposure Levels in Table 4-43 are from Table 2-31 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 50th percentile estimate in the EPA Exposure Factors Handbook (in Table 16-57 in the Handbook). The reduction factor is 10 for bystanders³³. Neither of the risk estimates for consumers or bystanders in Table 4-43 exceeded the risk benchmark for either the central tendency or high-end estimates.

Table 4-45. Excess Lifetime Cancer Risk for Outdoor DIY Brake/repair Replacement for Consumers and Bystanders (5 minutes per day in driveway) (from Table 2-31 with a reduction factor of 10)

Consumer Exposure Scenario	Exposure Levels (f/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	8.2 E-8	4.4 E-7	2.1 E-8	1.1 E-7

$TWF_{\text{Concomitant Exposures (0.0833 hours per day every day)}} = (0.08333/24) \cdot (365/365) = 0.003472$

$IUR_{(16,62)} = 0.0641$; $IUR_{(\text{Lifetime})} = 0.16$

DIY User: ELCR (Central Tendency) = 0.007 f/cc • 0.0001142 • 0.0641 per f/cc + 0.007 • 0.02 • 0.003472 • 0.0641

DIY User: ELCR (High-end) = 0.0376 f/cc • 0.0001142 • 0.0641 per f/cc + 0.0376 • 0.02 • 0.003472 • 0.0641

DIY Bystander: ELCR (Central Tendency) = 0.0007 f/cc • 0.0001142 • 0.16 per f/cc + 0.0007 • 0.02 • 0.003472 • 0.16

DIY Bystander: ELCR (High-end) = 0.0038 f/cc • 0.0001142 • 0.16 per f/cc + 0.0038 • 0.02 • 0.003472 • 0.16

³³ 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 < 3 meter from automobile values measured indoors across all activities identified in the study data utilized from Blake (a ratio of 6.5). The ratio of 6.5 was rounded up to 10, to account for an additional reduction in concentration to which a bystander may be exposed in the outdoor space based on the high air exchange rates and volume in the outdoors.

Table 4-46. Excess Lifetime Cancer Risk for Outdoor DIY Brake/Repair Replacement for Consumers and Bystanders (30 minutes per day in driveway) (from Table 2-31 with a reduction factor of 10)

Occupational Exposure Scenario	Exposure Levels (f/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	2.4 E-7	1.3 E-6	5.9 E-8	3.2 E-7

$TWF_{\text{Concomitant Exposures (0.5 hours per day every day)}} = (0.5/24) \cdot (365/365) = 0.02083$

$IUR_{(16,62)} = 0.0641$; $IUR_{(\text{Lifetime})} = 0.16$

DIY User: $ELCR_{(\text{Central Tendency})} = 0.007 \text{ f/cc} \cdot 0.0001142 \cdot 0.0641 \text{ per f/cc} + 0.007 \cdot 0.02 \cdot 0.02083 \cdot 0.0641$

DIY User: $ELCR_{(\text{High-end})} = 0.0376 \text{ f/cc} \cdot 0.0001142 \cdot 0.0641 \text{ per f/cc} + 0.0376 \cdot 0.02 \cdot 0.02083 \cdot 0.0641$

DIY Bystander: $ELCR_{(\text{Central Tendency})} = 0.0007 \text{ f/cc} \cdot 0.0001142 \cdot 0.16 \text{ per f/cc} + 0.0007 \cdot 0.02 \cdot 0.02083 \cdot 0.16$

DIY Bystander: $ELCR_{(\text{High-end})} = 0.0038 \text{ f/cc} \cdot 0.0001142 \cdot 0.16 \text{ per f/cc} + 0.0038 \cdot 0.02 \cdot 0.02083 \cdot 0.16$

Exposure Levels from Table 2-31 are used in Table 4-44. The assumption 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 30 minutes per day in the driveway each day based on the 95th percentile estimate in the EPA Exposure Factors Handbook (in Table 16-57 in the Handbook). The reduction factor is 10 for bystanders. The risk estimates for the DIY consumer exceeded the risk benchmark for the high-end exposure only, whereas the risk estimates were not exceeded for either scenario for the bystanders.

4.2.3.2 Risk Estimation for Cancer Effects following Episodic Inhalation Exposures for UTV Gasket Repair/replacement

EPA assessed chrysotile exposures for the DIY (consumer) and bystander UTV gasket repair/replacement scenario based on aggregated exposures resulting from recurring episodic exposures from active use of chrysotile asbestos related to DIY brake-related activities. These activities include concomitant exposure to chrysotile asbestos fibers which are reasonably anticipated to remain within indoor use facilities. It is well-understood that asbestos fibers in air will settle out in dust and become re-entrained in air during any changes in air currents or activity indoors. On the other hand, in occupational settings, regular air sampling would capture both new and old fibers and have industrial hygiene practices in place to reduce exposures.

For the risk estimations for the UTV gasket COU, EPA used the same data/assumptions identified in Section 4.2.3.1 for brakes for exposure frequency and duration; with the exception that there is no outdoor exposure scenario. A sensitivity analysis is presented which includes a lower-bound estimate for a bystander of 20 years (ages 0-20) (see Appendix L and the uncertainties Section 4.3.7).

The assumption is that DIY UTV gasket replacement is limited to a single gasket change at age 16 years. EPA then assumed that the concentration of chrysotile asbestos in 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.

Based on these assumptions, the consumer and bystander risk estimates were exceeded for both central and high-end exposures based on a single UTV gasket change and remaining in the house for 10 years. Estimates exceeding the benchmark are shaded in pink and bolded.

Table 4-47. 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 2-31 without a reduction factor) (Consumers 1 hour/day spent in garage; Bystanders 1 hour/day)

Consumer Exposure Scenario	Exposure Levels (f/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 once, indoors)	0.024	0.066	0.012	0.03	2.9 E-6	8.0 E-6	3.2 E-6	7.9 E-6

$$TWF_{\text{Concomitant Exposures (1 hour per day every day)}} = (1/24) \cdot (365/365) = 0.04167$$

$$IUR_{(16,10)} = 0.0292; IUR_{(0,10)} = 0.0634$$

$$\text{DIY User: ELCR}_{(\text{Central Tendency})} = 0.024 \text{ f/cc} \cdot 0.000005524 \cdot 0.0292 \text{ per f/cc} + 0.024 \cdot 0.1 \cdot 0.04167 \cdot 0.0292$$

$$\text{DIY User: ELCR}_{(\text{High-end})} = 0.066 \text{ f/cc} \cdot 0.000005524 \cdot 0.0292 \text{ per f/cc} + 0.066 \cdot 0.1 \cdot 0.04167 \cdot 0.0292$$

$$\text{DIY Bystander: ELCR}_{(\text{Central Tendency})} = 0.012 \text{ f/cc} \cdot 0.000005524 \cdot 0.0634 \text{ per f/cc} + 0.012 \cdot 0.1 \cdot 0.04167 \cdot 0.0634$$

$$\text{DIY Bystander: ELCR}_{(\text{High-end})} = 0.03 \text{ f/cc} \cdot 0.000005524 \cdot 0.0634 \text{ per f/cc} + 0.03 \cdot 0.1 \cdot 0.04167 \cdot 0.0634$$

Table 4-48. Excess Lifetime Cancer Risk for Indoor DIY UTV Gasket /Repair Replacement for Consumers and Bystanders (exposures from Table 2-31) (Users 1 hour/day spent in garage; Bystanders 1 hour/day)

Consumer Exposure Scenario	Exposure Levels (f/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	1.9 E-5	5.3 E-5	2.4 E-5	6.1 E-5

$$TWF_{\text{Concomitant Exposures (1 hour per day every day)}} = (1/24) \cdot (365/365) = 0.04167$$

$$IUR_{(16,62)} = 0.0641; IUR_{(\text{Lifetime})} = 0.16$$

$$\text{DIY User: ELCR}_{(\text{Central Tendency})} = 0.024 \text{ f/cc} \cdot 0.0001142 \cdot 0.0641 \text{ per f/cc} + 0.024 \cdot 0.3 \cdot 0.04167 \cdot 0.0641$$

$$\text{DIY User: ELCR}_{(\text{High-end})} = 0.066 \text{ f/cc} \cdot 0.0001142 \cdot 0.0641 \text{ per f/cc} + 0.066 \cdot 0.3 \cdot 0.04167 \cdot 0.0641$$

$$\text{DIY Bystander: ELCR}_{(\text{Central Tendency})} = 0.012 \text{ f/cc} \cdot 0.0001142 \cdot 0.16 \text{ per f/cc} + 0.012 \cdot 0.3 \cdot 0.04167 \cdot 0.16$$

$$\text{DIY Bystander: ELCR}_{(\text{High-end})} = 0.030 \text{ f/cc} \cdot 0.0001142 \cdot 0.16 \text{ per f/cc} + 0.030 \cdot 0.3 \cdot 0.04167 \cdot 0.16$$

The exposure values from Table 2-31 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 50th 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 4-49. Excess Lifetime Cancer Risk for Indoor DIY Gasket/Repair Replacement for Consumers and Bystanders (exposures from Table 2-31) (Consumers 8-hours/day spent in garage; Bystanders 1 hour/day)

Consumer Exposure Scenario	Exposure Levels (f/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	1.5 E-4	4.2 E-4	2.4 E-5	6.1 E-5

$$TWF_{\text{Concomitant Exposures (8-hours per day every day)}} = (8/24) \cdot (365/365) = 0.3333$$

$$IUR_{(16,62)} = 0.0641; IUR_{(\text{Lifetime})} = 0.16$$

$$\text{DIY User: ELCR}_{(\text{Central Tendency})} = 0.024 \text{ f/cc} \cdot 0.0001142 \cdot 0.0641 \text{ per f/cc} + 0.024 \cdot 0.3 \cdot 0.3333 \cdot 0.0641$$

$$\text{DIY User: ELCR}_{(\text{High-end})} = 0.066 \text{ f/cc} \cdot 0.0001142 \cdot 0.0641 \text{ per f/cc} + 0.066 \cdot 0.3 \cdot 0.3333 \cdot 0.0641$$

$$\text{DIY Bystander: ELCR}_{(\text{Central Tendency})} = 0.012 \text{ f/cc} \cdot 0.0001142 \cdot 0.16 \text{ per f/cc} + 0.012 \cdot 0.3 \cdot 0.04167 \cdot 0.16$$

$$\text{DIY Bystander: ELCR}_{(\text{High-end})} = 0.030 \text{ f/cc} \cdot 0.0001142 \cdot 0.16 \text{ per f/cc} + 0.030 \cdot 0.3 \cdot 0.04167 \cdot 0.16$$

The exposure values from Table 2-31 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 95th percentile estimate in the EPA Exposure Factors Handbook ([U.S. EPA, 2011](#)). 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.

4.2.3.3 Summary of Consumer and Bystander Risk Estimates by COU for Cancer Effects Following Inhalation Exposures

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.

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 quantitatively assessed, there were risks to consumers (DIY) and bystanders for all high-end and central tendency exposures from brake repair/replacement and UTV gasket repair/replacement scenarios except outdoor brake scenarios (outdoor scenario was not evaluated for gasket replacement). One outdoor brake scenario showed risks to the DIY consumer for the high-end exposure scenario (30 minutes/day in the driveway).

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 1×10^{-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 exceeding risk benchmarks. All analyses are in Appendix L.

Table 4-50. Summary of Risk Estimates for Inhalation Exposures to Consumers and Bystanders by COU (Cancer benchmark is 10⁻⁶)

Life Cycle Stage/Category	Subcategory	Consumer Exposure Scenario	Population	Exposure Duration and Level	Cancer Risk Estimates
Imported asbestos products	Brakes Repair/replacement Indoor, compressed air, once every 3 years for 62 years starting at 16 years, exposures at 30% of active used between uses, 1 hour/d in garage	Section 4.2.3.1	DIY	Central Tendency	3.6 E-5
				High-end	3.5 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	2.6 E-4
				High-end	2.6 E-3
			Bystander	Central Tendency	1.7 E-5
				High-end	3.9 E-5
	Brakes Repair/replacement Indoor, compressed air, once at 16 years, staying in residence for 10 years, exposures at 10% of active use, 1 hour/d in garage	Section 4.2.3.1	DIY	Central Tendency	5.4 E-6
				High End	5.3 E-5
			Bystander	Central Tendency	3.4 E-6
				High-end	7.8 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	8.2 E-8
				High-end	4.4 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	2.4 E-7
				High-end	1.3 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	1.9 E-5
				High-end	5.3 E-5
			Bystander	Central Tendency	2.4 E-5
				High-end	6.1 E-5
	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	Section 4.2.3.2	DIY	Central Tendency	1.5 E-4
				High-end	4.2 E-4
			Bystander	Central Tendency	2.4 E-5
				High-end	6.1 E-5
	Gasket Repair Repair/replacement Indoor, once at 16 years, staying in residence for 10 years, exposures at 10% of active use, 1 hour/d in garage	Section 4.2.3.2	DIY	Central Tendency	2.9 E-6
				High end	8.0 E-6
			Bystander	Central Tendency	3.2 E-6
				High-end	7.9 E-6

4.3 Assumptions and Key Sources of Uncertainty

4.3.1 Key Assumptions and Uncertainties in the Uses of Asbestos in the U.S.

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 ([see 2017 Public Meeting](#)) on uses of asbestos and followed by both the Scope document (June [2017d](#)) 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.

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.

EPA will consider legacy uses and associated disposal and other fiber types of asbestos in Part 2 of the final Risk Evaluation on asbestos (see Preamble).

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 differ from 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 engaged in a dialogue with industries 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 described in the 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, it is unclear of the source of the asbestos and the fiber type present. In the draft Risk Evaluation, EPA concluded that, based on the reasonably available information in the published literature, provided by industries using asbestos, and reported in EPA databases, there were minimal or no releases of asbestos to surface water associated with the COUs that EPA is evaluating (see Appendix D). Therefore, EPA concluded there is no unreasonable risk to aquatic or sediment-dwelling environmental organisms.

EPA has considered peer review and public comments on this conclusion and has decided to keep the finding made in the draft Risk Evaluation (*i.e.*, that there were minimal or no releases of asbestos to surface water associated with the COUs that EPA is evaluating in this Part 1 of the risk evaluation for asbestos). This is because EPA is confident that the minimal water release data available and reported more fully in the PF – and now presented again in Appendix D – cannot be attributed to chrysotile asbestos from the COUs in this Part 1 of the risk evaluation for asbestos. Assessing possible risk to

aquatic organisms from the exposures described would not be reasonably attributed to the COUs. However, based on the decision to develop a scope and risk evaluation for legacy uses and associated disposals of asbestos (Part 2 of the final Risk Evaluation for asbestos), EPA expects to address the issue of releases to surface water based on those other uses.

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 Part 1 of the risk evaluation for asbestos. 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. Finally, 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 Part 1 of the risk evaluation for asbestos 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 asbestos IUR. PCM detects only fibers longer than 5 μm 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 this Part 1 of the risk evaluation for asbestos have reported asbestos concentrations using PCM.

In general, when enough data were reasonably available, the 95th and 50th percentile exposure concentrations were calculated using reasonably available data (*i.e.*, the chlor-alkali worker monitoring data). In other instances, EPA had very little monitoring data available on occupational exposures for certain COUs (*e.g.*, sheet gasket stamping and brake blocks) or limited exposure monitoring data in the published literature as well. Where there are few data points available, it is unlikely the results will be representative of worker exposure across the industry depending on the sample collection location (PBZ or source zone) and timing of the monitoring.

EPA acknowledges that the reported inhalation exposure concentrations for the industrial scenario uses may not be representative for the exposures in all companies within that industry. For example, there are only three chlor-alkali companies who own a total of 15 facilities in the U.S. that use chrysotile asbestos diaphragms, but their operations are different, where some of them hydroblast and reuse their chrysotile asbestos-containing diaphragms and others replace them. The exposures to workers related to these two different activities are different.

EPA also received data from one company that fabricates sheet gaskets and one company that uses sheet gaskets. These data were used, even though there are limitations, such as the representativeness of practices in their respective industries.

All the raw chrysotile asbestos imported into the U.S. is used by the chlor-alkali industry for use in asbestos diaphragms. The number of chlor-alkali plants in the U.S. is known and therefore the number of workers potentially exposed is reasonably certain. In addition, estimates of workers employed in this industry were provided by the chlor-alkali facilities. However, the number of workers potentially exposed during other COUs is very limited. Only two workers were identified for stamping sheet gaskets, and two titanium dioxide manufacturing facilities were identified in the U.S. who use asbestos-containing gaskets. However, EPA is not certain if asbestos-containing sheet gaskets are used in other industries and to what extent. For the other COUs, no estimates of the number of potentially exposed workers were submitted to EPA by industry or its representatives, so estimates were used. Therefore,

numbers of workers potentially exposed were estimated; and these estimates could equally be an over-estimate or an under-estimate.

Finally, there is uncertainty in how EPA categorized the exposure data. Each PBZ and area data point was classified as either “worker” or “occupational non-user.” The categorizations are based on descriptions of worker job activity as provided in worker monitoring data, in the literature and EPA’s judgment. In general, PBZ samples were categorized as “worker” and area samples were categorized as “occupational non-user.” Exposure data for ONUs were not available for most scenarios. EPA assumes that these exposures are expected to be lower than worker exposures, since ONUs do not typically directly handle asbestos nor are in the immediate proximity of asbestos.

4.3.4 Key Assumptions and Uncertainties in the Consumer Exposure Assessment

Due to lack of specific information on DIY consumer exposures, the consumer assessment relies on available occupational data obtained under certain environmental conditions expected to be more representative of a DIY consumer scenario (no engineering controls, no PPE, residential garage). However, the studies utilized still have uncertainties associated with the environment 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.

The volume of a former automobile repair facility is considerably larger than a typical residential garage and will have different air exchange rates. While this could raise some uncertainties related to the applicability of the measured data to a DIY consumer environment, the locations of the measurements utilized for this evaluation minimize that uncertainty.

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

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

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

There were no data 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, performed by professional mechanics, on automobile exhaust systems 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 which was representative of the expected consumer environment.

There is 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.

There is uncertainty associated with the assumption that UTV exhaust system gasket repair/replacement activities would take a consumer a full three hours to complete. While there was no published information found providing consumer specific lengths of time to complete a full repair/replacement activity. 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 once the system is opened up and the gasket removed. Without published information, EPA assumes this work takes about three hours and therefore three-hour time frames to estimate risks for this evaluation.

Finally, EPA has made some assumptions regarding both age at start of exposure and duration of exposure for both the DIY users and bystanders for both the brake and UTV gasket scenarios. Realizing there is uncertainty around these assumptions, specifically that they may over-estimate exposures, EPA developed a sensitivity analysis approach specifically for the consumer exposure/risk analysis (see appropriate part of Section 4.3.8 below) and also performed a sensitivity analysis using five different scenarios (Appendix L), including a single brake change (or UTV gasket) repair/replacement activity at 16 years of age.

4.3.5 Key Assumptions and Uncertainties in the Human Health IUR Derivation

The analytical method used to measure exposures in the epidemiology studies is important in understanding and interpreting the results as they were used to develop the IUR. As provided in more detail in Section 3, the IUR for “current use” asbestos (*i.e.*, chrysotile) is based solely on studies of PCM measurement as TEM-based risk data are limited in the literature and the available TEM results for chrysotile asbestos lack modeling results for mesothelioma. In TEM studies of NC and SC ([Loomis et al., 2010](#); [Stayner et al., 2008](#)), models that fit PCM vs. TEM were generally equivalent (about 2 AIC units), indicating that fit of PCM is similar to the fit of TEM (for these two cohorts), providing confidence in those PCM measurements for SC and NC. Given that confidence in the PCM data and the large number of analytical measurements, exposure uncertainty is considered low in the cohorts used for IUR derivation.

The endpoint for both mesothelioma and lung cancer was mortality, not incidence. Incidence data are not available for any of the cohorts. However, for lung cancer, EPA was able to use background rates of incidence data in lifetables (Appendix H) as a way to address this bias of using mortality data for risk estimates instead of incidence data. For mesothelioma, this adjustment in lifetable methodology was not possible, because the mesothelioma model uses an absolute risk. Thus, using mortality data for mesothelioma remains a downward bias in the selected IUR value. However, for mesothelioma, the median length of survival with mesothelioma is less than 1 year for males, with less than 20% surviving after 2-years and less than 6% surviving after 5-years.

By definition, the IUR only characterizes cancer risk. It does not include any risks that may be associated with non-cancer health effects. Pleural and pulmonary effects from asbestos exposure (*e.g.*, asbestosis and pleural thickening) are well-documented ([U.S. EPA, 1988b](#)), although there is no reference concentration (RfC) for these non-cancer health effects specifically for chrysotile asbestos. The IUR for chrysotile asbestos is 0.16 per f/cc (Section 3.2.4). Based on this IUR, the chrysotile asbestos exposure concentration, over a lifetime, that would be expected to cause 1 cancer per 1,000,000 people (1E-6) in the general population is 6E-6 f/cc. The IRIS assessment of Libby amphibole asbestos ([U.S. EPA, 2014b](#)) derived a RfC for non-cancer health effects, and at that concentration (9 E-5 f/cc), the risk of cancer for chrysotile asbestos was 1 E-5 [IUR*RfC = (0.16 per f/cc)*(9 E-5 f/cc)]. Thus, at a target risk of 1 cancer per 1,000,000 people (1E-6) and exposures at or below 6E-6 f/cc to meet that target risk, the chrysotile asbestos cancer toxicity value appeared to be the clear risk driver as meeting that target risk for the general population (including consumers, DIY and bystanders) would result in lower non-cancer risks than at the Libby Amphibole asbestos RfC (*i.e.*, 6E-6 f/cc < 9E-5 f/cc).

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

4.3.6 Key Assumptions and Uncertainties in the Cancer Risk Values

Although direct comparison of cancer slopes for PCM and TEM fibers is impossible because different counting rules for these methods result in qualitatively and quantitatively different estimates of asbestos exposure, comparing the fit of models based on different analytical methods is possible. In TEM studies of NC and SC ([Loomis et al., 2010](#); [Stayner et al., 2008](#)), models that fit PCM vs. TEM were generally equivalent (about 2 AIC³⁴ units), indicating that fit of PCM is similar to the fit of TEM (for these two cohorts), providing confidence in those PCM measurements for SC and NC, whose data is the basis for chrysotile asbestos IUR.

Another source of uncertainty in the exposure assessment is that early measurements of asbestos fiber concentrations were based on an exposure assessment method (midget impinger) that estimated the combined mass of fibers and dust, rather than on counting asbestos fibers. The best available methodology for conversion of mass measurements to fiber counts is to use paired and concurrent sampling by both methods to develop factors to convert the mass measurements to estimated fiber counts for specific operations. There is uncertainty in these conversion factors, but it is minimized in the

³⁴ AIC stands for Akaike Information Criterion; a measure of relative quality used for evaluation of statistical models.

studies of SC and NC chrysotile asbestos textile workers due to the availability of an extensive database of paired and concurrent samples and the ability to develop operation-specific conversion factors. Uncertainty in the estimation of these conversion factors and their application to estimate chrysotile asbestos exposures will not be differential with respect to disease because this uncertainty is independent of cancer status.

As noted in Section 3.2.3.3 the exposure data from the SC and NC cohorts are of higher quality than those utilized in other studies of occupational cohorts exposed to chrysotile asbestos. Given the confidence in the PCM data and the large number of analytical measurements, exposure uncertainty is overall low in the SC and NC cohorts, as high-quality exposure estimates are available for both cohorts. Statistical error in estimating exposure levels is random and not differential with respect to disease (*i.e.*, independent). 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 ([Blair et al., 2007](#); [Siemiatycki et al., 1988](#)) 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 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 years³⁵. 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

³⁵ The Fair Labor Standards Act of 1938 allows adolescents to work an unrestricted number of hours at age 16 years.

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 4-51. 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 occupational exposure scenarios (baseline scenario: first exposure at 16 years for 40 years duration)

Age at first exposure (years)	Duration of exposure (years)	
	20	40
16	0.0468/0.0612 = 0.76	0.0612/0.0612 = 1
20	0.0374/0.0612 = 0.61	0.0486/0.0612 = 0.79
30	0.0209/0.0612 = 0.34	0.0269/0.0612 = 0.44

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 uses and associated disposals of asbestos will be considered in Part 2 of the risk evaluation for asbestos (see Preamble).

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, risks for consumers/bystanders 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 do not change the overall risk picture in almost all scenarios. Table 4-52 provides a summary of the detailed analyses in Appendix L.

Table 4-52. 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)

Age at first exposure (years)	Duration of exposure (years)		
	20	40	62
16	0.0468/0.0641 = 0.73	0.0612/0.0641 = 0.95	0.0641/0.0641 = 1
20	0.0374/0.0641 = 0.58	0.0486/0.0641 = 0.76	-
30	0.0209/0.0641 = 0.33	0.0269/0.0641 = 0.42	-

Table 4-53. 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	0.106/0.16 = 0.66	0.144/0.16 = 0.90	0.16/0.16 = 1

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 1×10^{-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 4-54. Results of Sensitivity Analysis of Exposure Assumptions for Consumer DIY/Bystander Episodic Exposure Scenarios

Sensitivity Analysis ¹	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

¹ 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 Part 1 of the risk evaluation for asbestos, 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 50th percentile would represent a central tendency estimate for being present in the garage (one hour/day) and the 95th 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 asbestos 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 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 50th 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 4-55. 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 th	25 th	50 th	75 th	95 th
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 yrs old	10	25	30	120	165
12-17 yrs old	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 reasonably certain about the number of chlor-alkali workers given the information submitted by industry. For some of the other COUs, the estimated numbers presented in the draft risk evaluation were modified based on peer review and public comments.

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.

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 Automobile Brakes/Linings/Clutches (Workers and ONUs)

As discussed in Sections 2.3.1.7.2, EPA has changed its estimates of the number of workers/ONUs who may be exposed to chrysotile asbestos from replacing aftermarket automobile brakes/linings/clutches.

The Draft Risk Evaluation estimated that there are 749,900 automotive service technicians and mechanics that may be exposed as workers to aftermarket automotive brakes/linings, and clutches, and another 749,900 that may be exposed as ONUs.

Both peer review and public commenters questioned this estimate; which was based on assuming that 100% of workers in automotive repair could potentially be exposed to a much lower percentage of asbestos-containing brakes on the market. [One commenter](#) suggested that, at worst case, asbestos brakes represent 0.002% of the market, and that it is not rational to conclude that 100% of workers in automotive repair could potentially be exposed to 0.002% of brakes on the market. While EPA disagreed with some of the specifics in the calculation, EPA does agree such an adjustment is warranted.

Government data on imports are characterized by Harmonized Tariff Schedule (HTS) code. Most friction materials are reported under HTS code 6813.³⁶ Products containing asbestos should be reported under HTS 6813.20. Within HTS 6813.20, the 8-digit codes 6813.20.10 and 6813.20.15 represent brakes linings and pads for use in civil aircraft and in other vehicles (*e.g.*, cars), respectively. Although some of the shipments coded with an HTS code for asbestos may be misclassified and may not contain asbestos,

³⁶ HTS 6813 is defined as “Friction material and articles thereof (for example, sheets, rolls, strips, segments, discs, washers, pads), not mounted, for brakes, for clutches or the like, with a basis of asbestos, of other mineral substances or of cellulose, whether or not combined with textile or other materials.”

it is also possible that some of the shipments coded with a non-asbestos HTS code are also misclassified and actually do contain asbestos. The figures for imports of brakes in HTS code 6813.20.15 seem to be the best option available for estimating the potential market share of asbestos brakes.

According to U.S. Census Bureau data, the average annual value of imports in HTS 6813.20.15 during the period from 2010 to 2019 was \$1,949,006.³⁷ According to the web page of a market research group, the demand for aftermarket automotive brake is approximately \$4.3 billion per year for all of North America.³⁸ Based on this data, asbestos brakes may represent approximately 0.05% of aftermarket automotive brakes.

Assuming that the number of potentially exposed individuals is equal to the apparent market share of asbestos brakes and applying a 0.05% adjustment factor to the estimates of 749,900 yields a value of 375 for both workers and ONUs (see Table 4-54).

While there would still be uncertainty in these newer estimates, the level of confidence is higher than in the 100% estimates in the draft risk evaluation.

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 Automobile Brakes/Linings/Clutches (Consumers/DIY/Bystanders)

In the draft Risk Evaluation, EPA calculated the number of consumers that could be purchasing and performing DIY brake jobs based on housing units and number of vehicles per household, and the results of a survey suggesting that 50% of all U.S. households have at least one automotive DIYer (see Section 4.3.7 in the [Draft Risk Evaluation for Asbestos](#)). The estimate was that 31,857,106 automotive DIYers replace brake pads.

However, as noted above, both peer review and public commenters questioned this estimate; which was based on assuming that 100% of the estimated DIYers purchased and used asbestos-containing brakes.

Using the same logic previously described (*i.e.*, a market share estimate of 0.05%), and applying a 0.05% adjustment factor to the estimates of 31,857,106 yields a value of 15,929 for DIYers (see Table 4-54).

While there would still be uncertainty in these newer estimates, the level of confidence is higher than in the 100% estimates in the draft risk evaluation.

COUs for Which No Estimates May be Made

EPA could not develop a reasonable estimate of potentially impacted individuals for two COUs: other vehicle friction products (workers/ONUs) and UTV gasket replacement/repair (DIY/bystanders).

³⁷ Source: U.S. International Trade Commission DataWeb (USITC DataWeb), using data retrieved from the U.S. Bureau of the Census (accessed August 12, 2020).

³⁸ Since this figure is not limited to the United States, it may underestimate the fraction of U.S. brakes that may contain asbestos (*i.e.*, the denominator is too large).

Table 4-56. Summary of Estimated Number of Exposed Workers and DIY Consumers^a.

Condition of Use	Industrial and Commercial		DIY	
	Workers	ONU	Consumer	Bystanders
Asbestos diaphragms – chlor-alkali	75-148	<2900-3000	-	-
Sheet gaskets – stamping	≥2	≥4	-	-
Sheet gaskets – use	25/facility (no. of facilities Unknown)	13/facility (no. of facilities Unknown)	-	-
Oilfield brake blocks	<61,695 (total; number exposed to asbestos unknown)	<66,108 (total; number in vicinity of asbestos unknown)	-	-
Aftermarket automotive brakes/linings, clutches	375	375	15,929	Unknown
Other Vehicle Friction Products (brakes installed in exported cars)	Unknown	Unknown	-	-
Other gaskets – UTVs	~1500 (total; number exposed to asbestos unknown)	Unknown	Unknown	Unknown

^a 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 due to higher exposures. Some workers not associated with the COU in this Part 1 of the risk evaluation for asbestos 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 and associated disposals of asbestos in Part 2 of the risk evaluation for asbestos. Although it is clear that the health risks from asbestos exposure increase with greater 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 K of this Part 1 of the risk evaluation for asbestos 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](#)).

4.4.2 Aggregate and Sentinel Exposures

Section 2605(b)(4)(F)(ii) of TSCA requires the EPA, as a part of the risk evaluation, to describe whether aggregate or sentinel exposures under the conditions of use were considered and the basis for their consideration. The EPA has defined aggregate exposure as “the combined exposures to an individual from a single chemical substance across multiple routes and across multiple pathways (40 CFR § 702.33).”

Aggregate exposures for chrysotile asbestos were not assessed by *routes of exposure* in this Part 1 of the risk evaluation for asbestos since only inhalation exposure was evaluated. Although there is the possibility of dermal exposures occurring for the chrysotile asbestos COUs, it is unlikely that they would contribute to mesothelioma and lung cancer. As discussed in the scope and PF documents, the only known hazard associated with dermal exposure to asbestos is the formation of warts. But perhaps most importantly, with risk estimations already exceeding benchmarks from simply inhalation exposures, adding other, different exposures/hazards does not seem pragmatic.

Pathways of exposure were not combined in this Part 1 of the risk evaluation for asbestos. Although it is possible that workers exposed to asbestos might also be exposed as consumers (*e.g.*, by changing brakes at home), the number of workers/users is potentially small. The individual risk estimates already indicate risk; aggregating the pathways would increase the risk.

In addition, the potential for exposure to other uses/fiber types of asbestos (besides chrysotile), legacy asbestos for any populations or subpopulation, due to activities such as home or building renovations, as well as occupational or consumer exposures identified in this Part 1 of the risk evaluation for asbestos, is possible. EPA will consider legacy uses and associated disposals of asbestos in Part 2 of the Risk Evaluation on asbestos (see Preamble).

The EPA defines sentinel exposure as “*the exposure to a single chemical substance that represents the plausible upper bound of exposure relative to all other exposures within a broad category of similar or related exposures* (40 CFR § 702.33).” In terms of this Part 1 of the risk evaluation for asbestos, the EPA considered sentinel exposure the highest exposure given the details of the conditions of use and the potential exposure scenarios. EPA considered sentinel exposure for chrysotile asbestos in the form of a high-end level scenario for occupational, ONU, consumer DIY, and bystander exposures resulting from inhalation exposures for each COU.

4.5 Risk Conclusions

4.5.1 Environmental Risk Conclusions

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 and sediments associated with the COUs in this risk evaluation. Therefore, EPA concludes there is no unreasonable risk to aquatic or sediment-dwelling environmental organisms. In addition, terrestrial pathways, including biosolids, were excluded from analysis at the PF stage.

4.5.2 Human Health Risk Conclusions to Workers

Table 4-38 provides a summary of risk estimates for workers and ONUs. For workers in all of quantitatively assessed COU categories identified in this Part 1 of the risk evaluation for asbestos cancer risks were exceeded for all high-end exposures (except for other vehicle friction products: Super Guppy and high-end exposures were not assessed for oilfield brake blocks) and for most at the central tendency (except for chlor-alkali diaphragms and other vehicle friction products: Super Guppy). In addition, for ONUs, cancer risks were exceeded for both central tendency and high-end exposures for sheet gasket use and UTV gasket replacement. Cancer risks for ONUs were indicated for high-end exposures only for chlor-alkali, sheet gasket stamping. With the assumed use of respirators as PPE at APF of 10, most risks would be reduced but still persisted for high-end exposures for sheet gasket stamping, sheet gasket use, aftermarket auto brake replacement, other vehicle friction products and UTV gasket replacement. When respirators with an APF of 25 was assumed, risk was still indicated for the aftermarket auto brakes and the other vehicle friction products for high-end short-term exposure scenario. 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 are not assumed to be using PPE to reduce exposures to asbestos.

4.5.3 Human Health Risk Conclusions to Consumers

Table 4-48 provides a summary of risk estimates for consumers and bystanders. Cancer risks were exceeded for all consumer and bystander exposure scenarios except for some of the outdoor brake repair/ replacement exposure scenarios. Cancer risk, however, was still exceeded for the consumer/DIYer 30 minutes/day outdoor brake repair/replacement exposure scenario for high-end exposures.

5 Risk Determination

5.1 Unreasonable Risk

5.1.1 Overview

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). This Part 1 of 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 chrysotile asbestos, respirator PPE (where present) and its effect on mitigating inhalation exposure was considered.

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 5×10^{-2} , which is greater than the benchmarks of 1×10^{-4} to 1×10^{-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 sensitivity or 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

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.*, 1×10^{-6} to 1×10^{-4} or also denoted as 1 E-6 to 1 E-4) depending on the subpopulation exposed. Generally, EPA considers benchmarks ranging from 1×10^{-6} to 1×10^{-4} as appropriate for the general population, consumer users, and non-occupational PESS.³⁹

For the purposes of this risk determination, EPA uses 1×10^{-6} as the benchmark for consumers (*e.g.*, do-it-yourself mechanics) and bystanders. In addition, consistent with the 2017 NIOSH guidance,⁴⁰ EPA uses 1×10^{-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 1×10^{-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. When making an unreasonable risk determination based on injury to health of workers, EPA also makes assumptions regarding workplace practices and the implementation of the required hierarchy of controls from OSHA. EPA assumes that feasible exposure controls, including engineering controls, administrative controls, or use of personal protective equipment (PPE) are implemented in the workplace. EPA's decisions for unreasonable risk to workers are based on high-end exposure estimates, in order to capture not only exposures for PESS but also to account for the uncertainties related to whether or not workers are using PPE

EPA did not evaluate risks to the general population from any conditions of use and the unreasonable risk determinations do not account for any risks to the general population. Additional details regarding the general population are in Section 1.4.4.

³⁹ 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).

⁴⁰ NIOSH 2016). Current intelligence bulletin 68: NIOSH chemical carcinogen policy, available at <https://www.cdc.gov/niosh/docs/2017-100/pdf/2017-100.pdf>.

5.1.3 Determining Environmental Risk

In the draft Risk Evaluation, EPA concluded that, based on the reasonably available information in the published literature, provided by industries using asbestos, and reported in EPA databases, there were minimal or no releases of asbestos to surface water associated with the COUs that EPA is evaluating (see Appendix D). Therefore, EPA concluded there was no unreasonable risk to aquatic or sediment-dwelling environmental organisms.

EPA has considered peer review and public comments on this conclusion and has decided to retain the finding made in the draft Risk Evaluation (*i.e.*, that there were minimal or no releases of asbestos to surface water associated with the COUs that EPA is evaluating in this Part 1 of the risk evaluation for asbestos). EPA is confident that the minimal water release data available and reported more fully in the PF – and now presented again in Appendix D – cannot be attributed to chrysotile asbestos from the COUs in this Part 1 of the risk evaluation for asbestos. Assessing possible risk to aquatic organisms from the exposures described would not be reasonably attributed to the COUs. However, based on the decision to develop a scope and risk evaluation for legacy uses and associated disposals of asbestos (Part 2 of the final Risk Evaluation for asbestos), EPA expects to address the issue of releases to surface water based on those other uses. EPA did not evaluate risks to terrestrial organisms from any conditions of use and the unreasonable risk determinations do not account for any risks to terrestrial organisms. Additional details regarding terrestrial organism exposures are in Section 1.4.4.

5.2 Risk Determination for Chrysotile Asbestos

EPA's determination of unreasonable risk for the conditions of use of chrysotile asbestos in this Part 1 of the risk evaluation for asbestos is based on health risks to workers, occupational non-users (exposed to asbestos indirectly by being in the same work area), consumers, and bystanders (exposed indirectly by being in the same vicinity where consumer uses are carried out).

As described in Section 4, significant risk of cancer incidence was identified. Section 26 of TSCA requires that EPA make decisions consistent with the “best available science” and based on “weight of the scientific evidence.” As described in EPA's framework rule for risk evaluation [82 FR 33726] weight of the scientific evidence includes consideration of the “strengths, limitations and relevance of the information.” EPA believes that public health is best served when EPA relies upon the highest quality information for which EPA has the greatest confidence.

The only fiber type of asbestos that EPA identified as manufactured (including imported), processed, or distributed under the conditions of use is chrysotile asbestos, the serpentine variety. Chrysotile asbestos is the prevailing form of asbestos currently mined worldwide. Therefore, it is reasonable to assume that commercially available products fabricated overseas are made with chrysotile asbestos. Any asbestos being imported into the U.S. in articles for the conditions of use EPA has identified in this document is believed to be chrysotile asbestos. Based on EPA's determination that chrysotile asbestos is the only form of asbestos imported into the U.S. as both raw form and as contained in articles, EPA performed a quantitative assessment for chrysotile asbestos. 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 (via a Significant New Use Notice (SNUN)) 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). 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 a 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. In this Part 1 of the risk evaluation for asbestos, EPA evaluated the following categories of conditions of use of chrysotile asbestos: manufacturing; processing; distribution in commerce; occupational and consumer uses; and disposal. EPA will also develop a scope document that will include other asbestos conditions of use, including “legacy” uses (*e.g.*, in situ building materials) and evaluation of additional fiber types (Part 2 of the final risk evaluation for asbestos, see Preamble). As explained in the problem formulation document and Section 1.4 of this Part 1 of the risk evaluation for asbestos, EPA did not evaluate the following: emission pathways to ambient air from commercial and industrial stationary sources or associated inhalation exposure of the general population or terrestrial species; the drinking water exposure pathway for asbestos; the human health exposure pathway for asbestos in ambient water; emissions to ambient air from municipal and industrial waste incineration and energy recovery units; on-site releases to land that go to underground injection; or on-site releases to land that go to asbestos National Emission Standards for Hazardous Air Pollutants (NESHAP) (40 CFR part 61, subpart M) compliant landfills or exposures of the general population (including susceptible populations) or terrestrial species from such releases.

This Part 1 of the risk evaluation on asbestos describes the physical-chemical characteristics that are unique to chrysotile asbestos, such as insolubility in water, suspension and duration in air, transportability, the friable nature of asbestos-containing products, which attribute to the potential for asbestos fibers to be released, settled, and to again become airborne under the conditions of use (re-entrainment⁴¹). Also unique to asbestos is the impact of the timing of exposure relative to the cancer outcome; the most relevant exposures for understanding cancer risk were those that occurred decades prior to the onset of cancer. In addition to the cancer benchmark, the physical-chemical properties and exposure considerations are important factors in considering risk of injury to health. To account for the exposures for occupational non-users and, in certain cases 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 Part 1 of the risk evaluation for asbestos 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 exceeded the risk benchmark 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. Thus, EPA focused 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 asbestos are similar to Libby amphibole asbestos, the non-cancer effects of chrysotile asbestos are likely to contribute additional risk to the overall health risk of chrysotile asbestos beyond the risk of cancer. Several of the COU-related exposures evaluated for human health risks in Section 4.2 are at or greater than the point of departure (POD) for non-cancer effects associated with exposure to Libby amphibole asbestos. Thus, the overall health risks of chrysotile asbestos are underestimated based on cancer risk alone and support the

⁴¹ Settled Asbestos Dust Sampling and Analysis 1st Edition Steve M. Hays, James R. Millette CRC Press 1994

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 this Part 1 of the risk evaluation for asbestos 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 document. Existing EPA regulations and standards address exposure pathways to the general population and terrestrial species as well as exposures to chlor-alkali industry occupational populations (*i.e.*, workers and ONUs) for the asbestos waste pathway (*e.g.*, the asbestos NESHAP, particularly 40 CFR §§ 61.144(a)(9), 61.150). 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 risk determination for the chrysotile asbestos-containing brakes conditions of use for the NASA "Super Guppy." Those conditions of use 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 in this Part 1 of the risk evaluation on asbestos, 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 chrysotile asbestos and asbestos-containing products are imported into the U.S. in a manner where exposure to asbestos is not anticipated to occur. According to information reasonably available to EPA, raw chrysotile asbestos is imported in bags wrapped in plastic where they are contained in securely locked shipping containers. These shipping containers remain locked until they reach the chlor-alkali plants (Enclosure B: Asbestos Controls in the Chlor-Alkali Manufacturing Process <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0736-0052>). Asbestos articles (or asbestos-containing products) are assumed to be imported and distributed in commerce in a non-friable state, enclosed in sealed boxes, where fibers are not expected to be released.

EPA also assumes the absence of asbestos exposure during the occupational disposal of asbestos sheet gaskets scraps during gasket stamping and the disposal of spent asbestos gaskets used in chemical manufacturing plants. This assumption is based on the work practices followed and discussed in Section 2.3.1 that prevent the release of asbestos fibers.

Considering these exposure assumptions, EPA finds no unreasonable risk to health or the environment for the life cycle stages of import and distribution in commerce of chrysotile asbestos for all the conditions of use evaluated in this Part 1 of the risk evaluation on asbestos. EPA also finds no unreasonable risk to health or the environment for occupational populations for the disposal of asbestos sheet gaskets scraps during gasket stamping and the disposal of spent asbestos gaskets used in chemical manufacturing plants.

In addition, there is a limited use of asbestos-containing brakes (categorized under other vehicle friction products) for a special, large NASA transport plane (the “Super-Guppy”). (See sections 2.3.1.8.2 and 4.2.2.6). EPA calculated risk estimates using occupational exposure monitoring data provided by NASA. EPA assumes 12 hours of brake changes occur every year starting at age 26 years with 20 years exposure.

The Excess Lifetime Cancer Risk for Super Guppy Brake/Repair Replacement for Workers is:

Full Shift: Central Tendency – 3.7 E-7

Full Shift: High-End – 1.1 E-6

Short-Term: Central Tendency – 7.3 E-7

Short-Term: High-End – 1.7 E-6

Because these risk estimates fall below the benchmark for both the central tendency and high-end and after considering the engineering controls and work practices in place discussed in section 2.3.1.8.2, EPA finds these COUs (import/manufacture, distribution, use and disposal) do not present an unreasonable risk of injury to health.

Conditions of Use that Do Not Present an Unreasonable Risk to Health or Environment
<ul style="list-style-type: none">• Import of chrysotile asbestos and chrysotile asbestos-containing products• Distribution of chrysotile asbestos-containing products• Use of chrysotile asbestos brakes for a specialized, large NASA transport plane• Disposal of chrysotile asbestos-containing sheet gaskets processed and/or used in the industrial setting and chrysotile asbestos-containing brakes for a specialized, large NASA transport plane

5.2.1 Occupational Processing and Use of Chrysotile Asbestos

EPA identified the following conditions of use where chrysotile 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) 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 APFs 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 (see section 2.3.1.3).

As explained in Section 5.2, EPA considers the high-end risk estimates for risk to workers, occupational non-users, consumers, and bystanders for this risk determination of chrysotile 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 ⁻⁴ excess cancer risks	10 ⁻⁴ excess cancer risks
Risk Estimates without PPE	8-hour TWA 9.9 E-5 Central Tendency 6.8 E-4 High-end Short Term 1.2 E-4 Central Tendency 9.4 E-5 Central Tendency ^a 1.3 E-3 High-end 6.7 E-4 High-end ^a	8-hour TWA 5.0 E-5 Central Tendency 1.6 E-4 High-end Short Term Not available
Risk Estimates with PPE	APF=10 8-hour TWA 9.9 E-6 Central Tendency 6.8 E-5 High-end Short Term 1.0 E-5 Central Tendency 9.0 E-5 High-end APF=25 8-hour TWA 3.9 E-6 Central Tendency 2.7 E-5 High-end Short Term 4.9 E-6 Central Tendency 5.1 E-5 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 (Section 2.3.1.3). Without respiratory PPE the high-end risk estimates exceed the	EPA calculated risk estimates using area monitoring data (<i>i.e.</i> , fixed location air monitoring results) provided by industry (Section 2.3.1.3), which supports EPA's

	<p>10x⁻⁴ risk benchmark; however, when expected use of respiratory PPE is considered for some worker tasks (APF=10 and APF=25), the risk estimates do not exceed the risk benchmark (at the central tendency and high-end). As depicted in Table 2-7 and documented by industry^b, 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 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 physical-chemical properties of asbestos 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.</p> <p>Finally, 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.</p>	<p>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 1x10⁻⁴, both risk estimates are fairly similar. Based on the benchmarks exceedances and considering the physical-chemical properties of asbestos, the expected absence of respiratory PPE, and the severe and irreversible health effects associated with asbestos inhalation exposures, these conditions of use (for processing and use) present unreasonable risk to ONUs.</p>
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	<p>At this risk level (1E-4), if the non-cancer effects (<i>e.g.</i>, asbestosis and pleural thickening) of chrysotile asbestos are similar to Libby amphibole asbestos, the non-cancer effects of chrysotile asbestos are likely to contribute additional risk to the overall health risk of chrysotile asbestos beyond the risk of cancer. Thus, the overall health risks of chrysotile asbestos are underestimated based on cancer risk 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.</p>	
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^aNo APF applied for 7.5 hours, APF of 25 applied for 30 minutes.

^bIndustry 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 ⁻⁴ excess cancer risks	10 ⁻⁴ excess cancer risks
Risk Estimates without PPE	8-hour TWA 2.8 E-4 Central Tendency 1.2 E-3 High-end Short Term 2.9 E-4 Central Tendency 1.2 E-3 High-end	8-hour TWA 4.8 E-5 Central Tendency 2.0 E-4 High-end Short Term 5.1 E-5 Central Tendency 2.0 E-4 High-end
Risk Estimates with PPE ^b	APF = 1 An APF of 1 was assigned to the respiratory PPE provided to workers based on industry information ^b 8-hour TWA 2.8 E-4 Central Tendency 1.2 E-3 High-end	Not Assessed; ONUs are not assumed to wear respirators

	<p>Short Term 2.9 E-4 Central Tendency 1.2 E-3 High-end</p>	
<p>Risk Considerations</p>	<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^a to be worn by a worker cutting gaskets. However, the OSHA Asbestos Standard 29 CFR 1910.1001 states that such respirators should not be used to mitigate asbestos exposure. However, using N95 respirators are not protective as OSHA Asbestos Standard 29 CFR 1910.1001 prohibit the use of filtering facepiece respirators for protection against asbestos fibers. Thus, the N95 respirator has an assigned APF=1 due to ineffectiveness as respiratory PPE for mitigating asbestos exposure. Absent effective respiratory PPE^b risk estimates for both central tendency and high-end exceeds the benchmark of 1×10^{-4}. Based on the benchmark exceedances and considering the physical-chemical properties of asbestos 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).. High-end risk estimates exceed the cancer risk benchmark of 1×10^{-4}. Based on the benchmark exceedances and considering the physical-chemical properties of asbestos and the severe and irreversible health effects associated with asbestos inhalation exposures, this condition of use presents unreasonable risk to ONUs.</p>

^aIndustry provided description of PPE [ACC \(2017a\)](#).

^bRisk 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.

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 ⁻⁴ excess cancer risks	10 ⁻⁴ excess cancer risks
Risk Estimates without PPE	8-hour TWA 5.2 E-4 Central Tendency 1.9 E-3 High-end	8-hour TWA 1.0 E-4 Central Tendency 3.2 E-4 High-end
Risk Estimates with current PPE ^a	APF=10 8-hour TWA 5.2 E-5 Central Tendency 1.9 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 ^a EPA also calculated the risk estimates using an APF of 10; however, even with PPE and considering the physical-chemical properties of asbestos 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 ⁻⁴ 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. EPA considered it appropriate to use the high-end estimate when determining ONU risk; however, both central tendency and high-end estimates showed risk. Based on the central tendency and high-end risk estimates exceeding the benchmark of 1x10 ⁻⁴ , the expected absence of respiratory PPE, the physical-chemical properties of asbestos and the severe and irreversible effects associated with asbestos inhalation exposures, this condition of use presents unreasonable risk to ONUs.

^aIndustry 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 ⁻⁴ excess cancer risks	10 ⁻⁴ excess cancer risks
Risk Estimates without PPE	8-hour TWA 6.0 E-4	8-hour-TWA 4.0 E-4
Risk Estimates with PPE	APF=1 Workers are not assumed to wear respirators	Not Assessed; ONUs are not assumed to wear respirators
Risk Considerations (applies to both workers and ONUs)	<p>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^a, 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 1x10⁻⁴ is exceeded, the physical-chemical properties of asbestos and the severe and irreversible effects associated with asbestos inhalation exposures, these conditions of use present unreasonable risk for both workers and ONUs.</p>	

^a Industry provided data [Popik \(2018\)](#)

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	Does not present an unreasonable risk of injury to health
Unreasonable Risk Driver	Cancer resulting from chronic inhalation exposure	Cancer resulting from chronic inhalation exposure
Benchmark (Cancer)	10 ⁻⁴ excess cancer risks	10 ⁻⁴ excess cancer risks
Risk Estimates without PPE	8-hour TWA 1.2 E-4 Central Tendency 1.9 E-3 High-end Short Term 1.2 E-4 Central Tendency 2.8 E-3 High-end	8-hour TWA 2.0 E-5 Central Tendency 4.0 E-5 High-end Short Term 2.0 E-5 Central Tendency 4.0 E-5 High-end
Risk Estimates with PPE	APF = 1 Workers are not assumed to wear respirators; Respirators only required by OSHA if PEL exceeded. 8-hour TWA 1.2 E-4 Central Tendency 1.9 E-3 High-end Short Term 1.2 E-4 Central Tendency 2.8 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 (for both central tendency and high-end). Based on the exceedance of the benchmark of 1x10 ⁻⁴ and consideration of the physical-chemical	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 where they can be inhaled by ONUs, EPA considered it appropriate to use the high-end estimate when determining ONU risk. Based on no exceedance of the benchmark of 1x10 ⁻⁴ , even with the expected absence

	properties of asbestos and the severe and irreversible effects associated with asbestos inhalation exposures, these conditions of use present unreasonable risk to workers.	of respiratory PPE, these conditions of use do not 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)**

Criteria for Risk Determination	Exposed Population	
	Workers	Occupational Non-Users
Life cycle Stage	Commercial Use	Commercial Use
TSCA Section 6(b)(4)(A) Unreasonable Risk Determination	Presents an unreasonable risk of injury to health (workers and occupational non-users)	
Unreasonable Risk Driver	Cancer resulting from chronic inhalation exposure	Cancer resulting from chronic inhalation exposure
Benchmark (Cancer)	10 ⁻⁴ excess cancer risks	10 ⁻⁴ excess cancer risks
Risk Estimates without PPE	8-hour TWA 4.8 E-4 Central Tendency 1.3 E-3 High-end	8-hour TWA 1.0 E-4 Central Tendency 3.0 E-4 High-end
Risk Estimates with PPE	APF=1 Workers are not assumed to wear respirators 8-hour TWA 4.8 E-4 Central Tendency 1.3 E-3 High-end	Not Assessed; ONUs are not assumed to wear respiratory PPE.
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 1x10 ⁻⁴ is exceeded (for both central tendency and high-end),	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

	<p>the physical-chemical properties of asbestos and the severe and irreversible effects associated with asbestos inhalation exposures, these conditions of use present unreasonable risk to workers.</p>	<p>considered it appropriate to use the high-end estimate when determining ONU risk; however both central tendency and high-end risk estimates presented risk. Based on the exceedance of the benchmark of 1×10^{-4} (for both central tendency and high-end), and the expected absence of respirators, the physical-chemical properties of asbestos and the potential severity of effect associated with inhalation exposures to asbestos, these conditions of use present unreasonable risk to ONUs.</p>
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5.2.2 Consumer Uses of Chrysotile Asbestos

The consumer uses of asbestos include aftermarket automotive asbestos-containing brakes/linings, and other asbestos-containing gaskets. Consumers and bystanders are not assumed to wear respiratory PPE. Therefore, EPA did not assess risk estimates with PPE for the conditions of use for these exposed populations.

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 and Disposal	Consumer Use and Disposal
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^{-6} excess cancer risks	10^{-6} excess cancer risks
Risk Estimates without PPE	<p>Indoor, compressed air 1 hour/day; once every 3 years for 62 years (starting age 16) Exposures at 30% of active used between uses, 1 hour/d in garage 3.6 E-5 Central Tendency 3.5 E-4 High-end</p>	<p>Indoor, compressed air 1 hour/day; once every 3 years for 62 years (starting age 16) 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</p>

	<p><u>Indoor, compressed air</u> 8-hour/day; once every 3 years for 62 years (starting age 16) Exposures at 30% of active used between uses, 8 hours/d in garage 2.6 E-4 Central Tendency 2.6 E-3 High-end</p>	<p><u>Indoor, compressed air</u> 8-hour/day; once every 3 years for 62 years (starting age 16) Exposures at 30% of active used between uses, 8 hours/d in garage 1.7 E-5 Central Tendency 3.9 E-5 High-end</p>
	<p><u>Indoor, compressed air</u> Indoor, compressed air, once at 16 years, staying in residence for 10 years, exposures at 10% of active used between uses, 1 hour/d in garage 5.4 E-6 Central Tendency 5.3 E-5 High-end</p>	<p><u>Indoor, compressed air</u> Indoor, compressed air, once at 16 years, staying in residence for 10 years, exposures at 10% of active used between uses, 1 hour/d in garage 3.4 E-6 Central Tendency 7.8 E-6 High-end</p>
	<p><u>Outdoor</u> Once every 3 years for 62 years (starting age 16) Exposures at 2% of active used between uses, 5 min/d in driveway 8.2 E-8 Central Tendency 4.4 E-7 High-end</p>	<p><u>Outdoor</u> Once every 3 years for 62 years (starting age 16) 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</p>
	<p><u>Outdoor</u> Once every 3 years for 62 years (starting age 16) Exposures at 2% of active used between uses, 30 min/d in driveway 2.4 E-7 Central Tendency 1.3 E-6 High-end</p>	<p><u>Outdoor</u> Once every 3 years for 62 years (starting age 16) 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</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</p>

	<p>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 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). Based on the benchmark exceedances and considering the physical-chemical properties of asbestos and the severe and irreversible health effects associated with asbestos inhalation exposures, this condition of use presents unreasonable risk to consumers.</p>	<p>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 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 1×10^{-6} for the chosen indoor exposure scenarios, the expected absence of respiratory PPE, the physical-chemical properties of asbestos 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 and Disposal	Consumer Use and Disposal
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	Indoor 1 hour/day; once every 3 years for 62 years (starting age 16) Exposures at 30% of active used between uses, 1 hour/d in garage 1.9 E-5 Central Tendency 5.3 E-5 High-end	Indoor 1 hour/day; once every 3 years for 62 years (starting age 16) 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
	Indoor 8-hour/day; once every 3 years for 62 years (starting age 16) Exposures at 30% of active used between uses, 8 hours/d in garage 1.5 E-4 Central Tendency 4.2 E-4 High-end	Indoor 8-hour/day; once every 3 years for 62 years (starting age 16) 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
	Indoor Indoor, compressed air, once at 16 years, staying in residence for 10 years, exposures at 10% of active used between uses, 1 hour/d in garage 2.9 E-6 Central Tendency 8.0 E-6 High-end	Indoor Indoor, compressed air, once at 16 years, staying in residence for 10 years, exposures at 10% of active used between uses, 1 hour/d in garage 3.2 E-6 Central Tendency 7.9 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	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

	<p>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 and Appendix L.) Because 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 1×10^{-6}, at both the central tendency and high-end estimates, the expected absence of respiratory PPE, the physical-chemical properties of asbestos and the severe and irreversible effects associated with asbestos inhalation exposures, these conditions of use present unreasonable risk to consumers.</p>	<p>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.) Because 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 1×10^{-6}, at both the central tendency and high-end estimates, the expected absence of respiratory PPE, the physical-chemical properties of asbestos and the severe and irreversible effects associated with asbestos inhalation exposures, these conditions of use present unreasonable risk to bystanders.</p>
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5.3 Unreasonable Risk Determination Conclusion

5.3.1 No Unreasonable Risk Determinations

TSCA Section 6(b)(4) requires EPA to conduct risk evaluations to determine whether chemical substances present unreasonable risk under their conditions of use. In conducting risk evaluations, “EPA will determine whether the chemical substance presents an unreasonable risk of injury to health or the environment under each condition of use [] within the scope of the risk evaluation, either in a single decision document or in multiple decision documents.” 40 CFR 702.47. Pursuant to TSCA section 6(i)(1), a determination of “no unreasonable risk” shall be issued by order and considered to be final agency action. Under EPA’s implementing regulations, “[a] determination by EPA that the chemical

substance, under one or more of the conditions of use within the scope of the risk evaluations, does not present an unreasonable risk of injury to health or the environment will be issued by order and considered to be a final Agency action, effective on the date of issuance of the order.” 40 CFR 702.49(d).

EPA has determined that the following conditions of use of chrysotile asbestos do not present an unreasonable risk of injury to health or the environment:

- Import of chrysotile asbestos and chrysotile asbestos-containing products (Section 5.2, Section 4, Section 3, Section 2)
- Distribution of chrysotile asbestos-containing products (Section 5.2, Section 4, Section 3, Section 2)
- Use of chrysotile asbestos brakes for a specialized, large NASA transport plane (Section 5.2, Section 4.2.2.6, Section 3, Section 2.3.1.8.2)
- Disposal of chrysotile asbestos-containing sheet gaskets processed and/or used in the industrial setting and asbestos-containing brakes for a specialized, large NASA transport plane (Section 5.2, Section 4.2.2.6, Section 3, Section 2.3.1.8.2)

This subsection of the Part 1 of the risk evaluation for asbestos therefore constitutes the order required under TSCA Section 6(i)(1), and the “no unreasonable risk” determinations in this subsection are considered to be final agency action effective on the date of issuance of this order. All assumptions that went into reaching the determinations of no unreasonable risk for these conditions of use, including any considerations excluded for these conditions of use, are incorporated into this order.

The support for each determination of “no unreasonable risk” is set forth in Section 5.2, “Risk Determination for Chrysotile Asbestos.” This subsection also constitutes the statement of basis and purpose required by TSCA Section 26(f).

5.3.2 Unreasonable Risk Determinations

EPA has determined that the following conditions of use of chrysotile asbestos present an unreasonable risk of injury:

- Processing and Industrial use of Chrysotile Asbestos Diaphragms in the Chlor-alkali Industry
- Processing and Industrial Use of Chrysotile Asbestos-Containing Sheet Gaskets in Chemical Production
- Industrial Use and Disposal of Chrysotile Asbestos-Containing Brake Blocks in Oil Industry
- Commercial and Consumer Use and Disposal of Aftermarket Automotive Chrysotile Asbestos-Containing Brakes/Linings
- Commercial Use and Disposal of Other Chrysotile Asbestos-Containing Vehicle Friction Products
- Commercial and Consumer Use and Disposal of Other Chrysotile Asbestos-Containing Gaskets

EPA will initiate TSCA Section 6(a) risk management actions on these conditions of use as required under TSCA Section 6(c)(1). Pursuant to TSCA Section 6(i)(2), the “unreasonable risk” determinations for these conditions of use are not considered final agency action.

5.4 Risk Determination for Five other Asbestiform Varieties

For the asbestos 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 Part 1 of the risk evaluation for asbestos, EPA only assessed conditions of use for chrysotile asbestos. The Agency will evaluate legacy uses and associated disposals and other fiber types of asbestos in Part 2 of the risk evaluation for asbestos. Part 2 will begin with a draft scope document (see Figure P-1 in the Preamble). Those legacy uses could include the other five asbestiform varieties included in the TSCA Title II definition. As such, risk determinations for conditions of use that include those asbestiform varieties would be made in a subsequent document.

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APPENDICES

Appendix A Regulatory History

A.1 Federal Laws and Regulations

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 majority of the asbestos-containing products originally covered in the 1989 final rule was overturned. The following products remain banned by rule under the Toxic Substances Control Act (TSCA):

- Corrugated paper
- Rollboard
- Commercial paper
- Specialty paper
- Flooring felt

In addition, the regulation continues to ban the use of asbestos in products that have not historically contained asbestos, otherwise referred to as “new uses” of asbestos (Defined by 40 CFR 763.163 as "commercial uses of asbestos not identified in §763.165 the manufacture, importation or processing of which would be initiated for the first time after August 25, 1989.”).

Restrictions on Discontinued Uses of Asbestos; Significant New Use Rule (SNUR), 2019

40 CFR Parts 9 and 721 – Restrictions on Discontinued Uses of Asbestos

Docket ID: EPA-HQ-OPPT-2018-0159; FRL 9991-33

This final rule strengthens the Agency’s ability to rigorously review an expansive list of asbestos products that are no longer on the market before they could be sold again in the United States. Persons subject to the rule are required to notify EPA at least 90 days before commencing any manufacturing, importing, or processing of asbestos or asbestos-containing products covered under the rule. These uses are prohibited until EPA conducts a thorough review of the notice and puts in place any necessary restrictions or prohibits use.

Other EPA Regulations:

Asbestos Worker Protection Rule, 2000

[40 CFR Part 763, Subpart G](#)

Extends OSHA standards to public employees in states that do not have an OSHA approved worker protection plan (about half the country).

Asbestos Information Act, 1988

[15 U.S.C. §2607\(f\)](#)

Helped to provide transparency and identify the companies making certain types of asbestos-containing products by requiring manufacturers to report production to the EPA.

Asbestos School Hazard Abatement Act (ASHAA), 1984 and Asbestos School Hazard Abatement Reauthorization Act (ASHARA), 1990

[20 U.S.C. 4011 et seq.](#) and [Docket ID: OPTS-62048E; FRL-3269-8](#)

Provided funding for and established an asbestos abatement loan and grant program for school districts and ASHARA further tasked EPA to update the MAP asbestos worker training requirements.

Emergency Planning and Community Right-to-Know Act (EPCRA), 1986

[42 U.S.C. Chapter 116](#)

Under Section 313, Toxics Release Inventory (TRI), requires reporting of environmental releases of friable asbestos at a concentration level of 0.1%.

Friable asbestos is designated as a hazardous substance subject to an Emergency Release Notification at 40 CFR §355.40 with a reportable quantity of 1 pound.

Clean Air Act, 1970

[42 U.S.C. §7401 et seq.](#)

Asbestos is identified as a Hazardous Air Pollutant.

Asbestos National Emission Standard for Hazardous Air Pollutants (NESHAP), 1973

[40 CFR Part 61, Subpart M of the Clean Air Act](#)

Specifies demolition and renovation work practices involving asbestos in buildings and other facilities (but excluding residences with 4 or fewer dwelling units single family homes).

Requires building owner/operator notify appropriate state agency of potential asbestos hazard prior to demolition/renovation.

Banned spray-applied surfacing asbestos-containing material for fireproofing/insulating purposes in certain applications.

Requires that asbestos-containing waste material from regulated activities be sealed in a leak-tight container while wet, labeled, and disposed of properly in a landfill qualified to receive asbestos waste.

Clean Water Act (CWA), 1972

[33 U.S.C. §1251 et seq](#)

Toxic pollutant subject to effluent limitations per Section 1317.

Safe Drinking Water Act (SDWA), 1974

[42 U.S.C. §300f et seq](#)

Asbestos Maximum Contaminant Level Goals (MCLG) 7 million fibers/L (longer than 10um).

Resource Conservation and Recovery Act (RCRA), 1976

[42 U.S.C. §6901 et seq.](#)

[40 CFR 239-282](#)

Asbestos is subject to solid waste regulation when discarded; NOT considered a hazardous waste.

Comprehensive Environmental Response, Compensation and Liability Act (CERCLA), 1980

[42 U.S.C. §9601 et seq.](#)

[40 CFR Part 302.4 - Designation of Hazardous Substances and Reportable Quantities](#)

13 Superfund sites containing asbestos, nine of which are on the National Priorities List (NPL)

Reportable quantity of friable asbestos is one pound.

Other Federal Agencies:

Occupational Safety and Health Administration (OSHA):

[Public Law 91-596](#) Occupational Safety and Health Act, 1970

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 (1.0 f/cc as a 30-minute TWA).

Asbestos General Standard [29 CFR 1910](#)

Asbestos Shipyard Standard [29 CFR 1915](#)

Asbestos Construction Standard [29 CFR 1926](#)

Consumer Product Safety Commission (CPSC): Banned several consumer products. Federal Hazardous Substances Act (FHSA) [16 CFR 1500](#)

Food and Drug Administration (FDA): Prohibits the use of asbestos-containing filters in pharmaceutical manufacturing, processing and packing. [21 CFR 211.72](#)

Mine Safety and Health Administration (MSHA): follows OSHA's safety standards.

Surface Mines [30 CFR part 56, subpart D](#)

Underground Mines [30 CFR part 57, subpart D](#)

Department of Transportation

Prescribes the requirements for shipping manifests and transport vehicle placarding applicable to asbestos [40 CFR part 172](#).

Non-regulatory information of note:

NIOSH conducts related research and monitors asbestos exposure through workplace activities in an effort to reduce illness and ensure worker health and safety.

A.2 State Laws and Regulations

Pursuant to AHERA, states have adopted through state regulation the EPA's Model Accreditation Plan (MAP) for asbestos abatement professionals who do work in schools and public and commercial buildings. Thirty-nine (39) states⁴² have EPA-approved MAP programs and twelve (12) states⁴³ have also applied to and received a waiver from EPA to oversee implementation of the Asbestos-Containing Materials in Schools Rule pursuant to AHERA. States also implement regulations pursuant to the Asbestos NESHAP regulations or further delegate those oversight responsibilities to local municipal governments. While federal regulations set national asbestos safety standards, states have the authority to impose stricter regulations. As an example, many states extend asbestos federal regulations – such as asbestos remediation by trained and accredited professionals, demolition notification, and asbestos disposal – to ensure safety in single-family homes. Thirty (30) states⁴⁴ require firms hired to abate asbestos in single family homes to be licensed by the state. Nine (9) states⁴⁵ mandate a combination of notifications to the state, asbestos inspections, or proper removal of asbestos in single family homes. Some states have regulations completely independent of the federal regulations. For example, California and Washington regulate products containing asbestos. Both prohibit use of more than 0.1% of asbestos in brake pads and require laboratory testing and labeling.

Below is a list of state regulations that are independent of the federal AHERA and NESHAP requirements that states implement. This may not be an exhaustive list.

California

[Asbestos](#) is listed on [California's Candidate Chemical List](#) as a carcinogen. Under [California's Propositions 65](#), businesses are required to warn Californians of the presence and danger of [asbestos](#) in products, home, workplace and environment.

California Brake Friction Material Requirements (Effective 2017)

[Division 4.5, California Code of Regulations, Title 22 Chapter 30](#)

⁴² 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.

⁴³ Connecticut, Colorado, Illinois, Kentucky, Louisiana, Massachusetts, Maine, New Hampshire, Oklahoma, Rhode Island, Texas, and Utah.

⁴⁴ 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.

⁴⁵ Colorado, Connecticut, Georgia, Maine, Massachusetts, New York, Oregon, Vermont, and West Virginia.

Sale of any motor vehicle brake friction materials containing more than 0.1% asbestiform fibers by weight is prohibited. All brake pads for sale in the state of California must be laboratory tested, certified and labeled by the manufacturer.

Massachusetts

[Massachusetts Toxics Use Reduction Act \(TURA\)](#)

Requires companies in Massachusetts to provide annual pollution reports and to evaluate and implement pollution prevention plans. Asbestos is included on the [Complete List of TURA Chemicals - March 2016](#).

Minnesota

[Toxic Free Kids Act Minn. Stat. 2010 116.9401 – 116.9407](#)

Asbestos is included on the [2016 Minnesota Chemicals of High Concern List](#) as a known carcinogen.

New Jersey

[New Jersey Right to Know Hazardous Substances](#)

The state of New Jersey identifies hazardous chemicals and products. Asbestos is listed as a known carcinogen and talc containing asbestos is identified on the Right to Know Hazardous Substances list.

Rhode Island

[Rhode Island Air Resources – Air Toxics Air Pollution Control Regulation No. 22](#)

Establishes acceptable ambient air levels for asbestos.

Washington

[Better Brakes Law \(Effective 2015\) Chapter 70.285 RCW Brake Friction Material](#)

Prohibits the sale of brake pads containing more than 0.1% asbestiform fibers (by weight) in the state of Washington and requires manufacturer certification and package/product labelling.

[Requirement to Label Building Materials that Contain Asbestos Chapter 70.310 RCW](#)

Building materials that contain asbestos must be clearly labeled as such by manufacturers, wholesalers, and distributors.

A.3 International Laws and Regulations

Asbestos is also regulated internationally. Nearly 60 nations have some sort of asbestos ban. The European Union (EU) will prohibit the use of asbestos in the chlor-alkali industry by 2025 ([Regulation \(EC\) No 1907/2006 of the European Parliament and of the Council, 18 December 2006](#)).

Canada banned asbestos in 2018

Prohibition of Asbestos and Products Containing Asbestos Regulations: SOR/2018-196

[Canada Gazette, Part II, Volume 152, Number 21](#)

In addition, the Rotterdam Convention is considering [adding chrysotile asbestos to Annex III](#), and the World Health Organization (WHO) has a global campaign to eliminate asbestos-related diseases ([WHO Resolution 60.26](#)).

Appendix B List of Supplemental Documents

Associated **Supplemental Systematic Review Data Quality Evaluation and Data Extraction Documents** – Provides additional detail and information on individual study evaluations and data extractions including criteria and scoring results.

Physical-Chemical Properties, Fate and Transport

a. Final Risk Evaluation for Asbestos: Part 1 Chrysotile Asbestos, Systematic Review Supplemental File: Data Quality Evaluation of Physical-Chemical Properties Studies ([U.S. EPA, 2020j](#))

b. Final Risk Evaluation for Asbestos: Part 1 Chrysotile Asbestos, Systematic Review Supplemental File: Data Extraction of Environmental Fate and Transport Studies ([U.S. EPA, 2020e](#))

Occupational Exposures and Releases

c. Final Risk Evaluation for Asbestos: Part 1 Chrysotile Asbestos, Systematic Review Supplemental File: Data Quality Evaluation of Environmental Releases and Occupational Exposure ([U.S. EPA, 2020f](#))

d. Final Risk Evaluation for Asbestos: Part 1 Chrysotile Asbestos, Systematic Review Supplemental File: Data Quality Evaluation of Environmental Releases and Occupational Exposure Data Common Sources ([U.S. EPA, 2020g](#))

Consumer and Environmental Exposures

e. Final Risk Evaluation for Asbestos: Part 1 Chrysotile Asbestos, Systematic Review Supplemental File: Data Quality Evaluation of Consumer Exposure ([U.S. EPA, 2020c](#))

f. Final Risk Evaluation for Asbestos: Part 1 Chrysotile Asbestos, Systematic Review Supplemental File: Data Quality Extraction Tables for Consumer Exposure ([U.S. EPA, 2020i](#))

Environmental Hazard

g. Final Risk Evaluation for Asbestos: Part 1 Chrysotile Asbestos, Systematic Review Supplemental File: Data Quality Evaluation of Ecological Hazard Studies ([U.S. EPA, 2020d](#))

Human Health Hazard

h. Final Risk Evaluation for Asbestos: Part 1 Chrysotile Asbestos, Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies: Mesothelioma and Lung Cancer Studies ([U.S. EPA, 2020h](#))

i. Final Risk Evaluation for Asbestos Part 1: Chrysotile Asbestos, Systematic Review Supplemental File: Data Quality Evaluation for Epidemiological Studies of Ovarian and Laryngeal Cancers ([U.S. EPA, 2020k](#))

Associated **Supplemental Information Documents** – Provides additional details and information on exposure.

Occupational Exposures

i. Final Risk Evaluation for Asbestos: Part 1 Chrysotile Asbestos, Supplemental File: Occupational Exposure Calculations (Chlor-Alkali) ([U.S. EPA, 2020b](#))

Consumer Exposures

j. Final Risk Evaluation for Asbestos: Part 1 Chrysotile Asbestos, Supplemental File: Consumer Exposure Calculations ([U.S. EPA, 2020a](#))

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 asbestos, 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 chlor-alkali company, which has already confirmed import and use of raw chrysotile asbestos.

Appendix D Releases and Exposure to the Environment Supplementary Information

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 TSCA⁴⁶. 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 during a calendar year.

Table APX D-1 provides production-related waste management data for friable asbestos reported by subject facilities to the TRI program from reporting years 2015 to 2018⁴⁷. This is an updated table from that reported in the PF document. In reporting year 2018, 44 facilities reported a total of approximately 34 million pounds of friable asbestos waste managed. Of this total, zero pounds were recovered for

⁴⁶ 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.”

⁴⁷ Data presented were queried using TRI Explorer and uses the 2019 National Analysis data set (released to the public in October 2020). This dataset includes revisions for the years 1988 to 2018 processed by EPA.

36 energy or recycled, approximately 46,000 pounds were treated, and over 33 million pounds were
 37 disposed of or otherwise released into the environment.

38 **Table APX D-2** provides a summary of asbestos TRI releases to the environment for the same
 39 reporting years as **Table APX D-1**. *There were zero pounds of friable asbestos reported as released to*
 40 *water via surface water discharges*, and a total of 171 pounds of air releases from collective fugitive and
 41 stack air emissions reported in 2018. The vast majority of friable asbestos was disposed of to Resource
 42 Conservation and Recovery Act (RCRA) Subtitle C landfills and to landfills other than RCRA Subtitle
 43 C. Of the 153,947 pounds of friable asbestos reported in 2018 as “other releases”, 90,640 pounds were
 44 sent off-site to a waste broker for disposal, 14,760 pounds were sent off-site for storage only, and 48,547
 45 pounds were sent off-site for other off-site management.

46 **Table APX D-1. Summary of Asbestos TRI Production-Related Waste Managed from 2015-2018**
 47 (lbs)

Year	Number of Facilities	Recycling	Energy Recovery	Treatment	Releases ^{a,b,c}	Total Production Related Waste
2015	39	0	0	188,437	38,197,608	38,386,044
2016	41	2	0	31,993	26,748,379	26,780,375
2017	39	0	0	179,814	30,796,283	30,976,097
2018	44	0	0	46,106	33,888,979	33,935,085

Data source: 2015-2018 TRI Data (Updated October 2020) ([U.S. EPA, 2017e](#)).

^a Terminology used in these columns may not match the more detailed data element names used in the TRI public data and analysis access points.

^b Does not include releases due to one-time events not associated with production such as remedial actions or earthquakes.

^c Counts all releases including release quantities transferred and release quantities disposed of by a receiving facility reporting to TRI.

48 While production-related waste managed shown in **Table APX D-1** excludes any quantities reported as
 49 catastrophic or one-time releases (TRI section 8 data), release quantities shown in **Table APX D-2**
 50 include both production-related and non-routine quantities (TRI section 5 and 6 data) for 2015-2018. As
 51 a result, release quantities may differ slightly and may further reflect differences in TRI calculation
 52 methods for reported release range estimates ([U.S. EPA, 2017e](#)).
 53

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 ^a	Total On- and Off-Site Disposal or Other Releases ^{b, c}
		Stack Air Releases	Fugitive Air Releases		Class I Under-ground Injection	RCRA Subtitle C Landfills	All other Land Disposal ^a		
Totals 2015	39	101	208	0	0	9,623,957	28,780,780	0	38,405,047
		310			38,404,737				
Totals 2016	41	178	106	0	0	8,759,578	18,603,892	0	27,363,755
		285			27,363,470				
Totals 2017	39	80	67	0	0	6,199,224	25,162,328	0	31,361,700
		147			31,361,552				
Totals 2018	44	96	75	0	0	10,599,587	23,216,673	153,947	33,970,378
		171			33,816,260				

Data source: 2015-2018 TRI Data (Updated October 2020) ([U.S. EPA, 2017e](#)).

^a Terminology used in these columns may not match the more detailed data element names used in the TRI public data and analysis access points.

^b These release quantities do include releases due to one-time events not associated with production such as remedial actions or earthquakes.

^c Counts release quantities once at final disposition, accounting for transfers to other TRI reporting facilities that ultimately dispose of the chemical waste.

55 The Clean Water Act and the Safe Drinking Water Act

56 Background (Numeric Criteria and Reportable Levels)

57 The Clean Water Act (CWA) requires that states adopt numeric criteria for priority pollutants for which
58 EPA has published recommended criteria under section 304(a). States may adopt criteria that EPA
59 approves as part of the state's regulatory water quality standards. Once states adopt criteria as water
60 quality standards, the CWA requires that National Pollutant Discharge Elimination System (NPDES)
61 discharge permits include effluent limits as stringent as necessary to meet the standards [CWA section
62 301(b)(1)(C)]. If state permit writers determine that permit limits are needed, they will determine the
63 level of pollutant allowed to ensure protection of the receiving water for a designated use. This is the
64 process used under the CWA to address risk to human health and aquatic life from exposure to a
65 pollutant in ambient waters.

66 EPA develops recommended ambient water quality criteria for pollutants in surface water that are
67 protective of aquatic life or human health designated uses with specific recommendations on the
68 duration and frequency of those concentrations under section 304(a) of the CWA. These criteria are
69 based on priorities of states and others, and a subset of chemicals are identified as "priority pollutants".
70 EPA has identified asbestos as a priority pollutant for which a nationally recommended human health
71 water quality criteria for asbestos of 7 million fibers per liter (MFL) has been developed. EPA has not
72 developed a nationally recommended water quality criteria for the protection of aquatic life for asbestos,
73 yet EPA may publish aquatic life criteria for asbestos in the future if it is identified as a priority under
74 the CWA.

75 EPA's National Primary Drinking Water Regulations (NPDWR), established under the Safe Drinking
76 Water Act (SDWA), are legally enforceable primary standards and treatment techniques that apply to
77 public water systems. Primary standards and treatment techniques protect public health by limiting the
78 levels of contaminants in drinking water. The Maximum Contaminant Level (MCL) for asbestos under
79 the Safe Drinking Water Act is 7 MFL, for fibers > 10 micrometers. EPA has set this level of protection
80 based on the best available science at the time the NPDWR was promulgated to prevent potential health
81 problems and considering any limitations in both the feasible treatment methods to remove a
82 contaminant and availability of analytical methods to reliably measure the occurrence of the
83 contaminant in water. In the case of asbestos, the MCL was set based entirely on the health goal since
84 feasible treatment methods and analytical methods were available to achieve the protective level of 7
85 MFL. Public water systems are required to sample each entry point into the distribution system for
86 asbestos at least once every 9 years. Transmission electron microscopy (TEM) is used for detection
87 (EPA 800/4-83-043). The detection limit is 0.01 MFL. Here are links to the [analytical standards](#) and the
88 [drinking water regulations](#).

89 The Phase II Rule, the regulation for asbestos, became effective in 1992. The Safe Drinking Water Act
90 requires EPA to review the national primary drinking water regulation for each contaminant every six
91 years and determine if the NPDWR is a candidate for revision, at that time. EPA reviewed asbestos as
92 part of the Six Year Review and determined that the 7 MFL for asbestos is still protective of human
93 health.

94 As discussed in the PF document, because the drinking water exposure pathway for asbestos is currently
95 addressed in the SDWA regulatory analytical process for public water systems, this pathway (drinking
96 water for human health) was not evaluated in this Part 1 of the risk evaluation for asbestos.

97 EPA issues Effluent Limitations Guidelines and Pretreatment Standards which are national regulatory
98 standards for industrial wastewater discharges to surface waters and publicly owned treatment works, or
99 POTWs (municipal sewage treatment plants). EPA issues Effluent Limitations Guidelines and
100 Pretreatment Standards for categories of existing sources and new sources under Title III of the [Clean](#)
101 [Water Act](#). The standards are technology-based (*i.e.*, they are based on the performance of treatment and
102 control technologies); they are not based on risk or impacts upon receiving waters. (See [effluent](#)
103 [guidelines](#)).

104 The Effluent Limitations Guidelines and Pretreatment Standards for the Asbestos Manufacturing Point
105 Source Category (40 CFR Part 427) do not require that industrial facilities monitor asbestos
106 concentrations in discharges. Rather, the regulations contain either a zero discharge of pollutants
107 standard or require that the discharger not exceed a specified release amount of pollutants including total
108 suspended solids (TSS), chemical oxygen demand (COD) and pH. These guidelines were originally
109 developed in 1974 and 1975 and were revised in 1995. These guidelines cover legacy uses such as
110 manufacture of asbestos cement pipe, asbestos cement sheet, roofing, paper, etc. and may not be
111 particularly useful to the COU of asbestos. Additionally, there are effluent guidelines for the chlor-alkali
112 industry under 40 CFR Part 415 that cover pollutants such as chlorine, mercury, and lead, but they are
113 not specific to asbestos. *The EPA [Industrial Waste Water Treatment Technology Database](#) does not*
114 *currently include any data for asbestos.*

115 **Reasonably Available Data from Water Release Databases and Other Information**

116 EPA investigated industry sector, facility, operational, and permit information regulated by NPDES
117 under the Clean Water Act to identify any permit limits, monitoring and reporting requirements, and any
118 discharge provisions related to asbestos and its COU. The Clean Water Act section 402 specifies that

119 point source pollutant dischargers into waters of the United States must obtain a permit to regulate that
120 facility's discharge. NPDES permits are issued by states, tribes, or territories that have obtained EPA
121 approval to issue permits or by EPA Regions in areas without such approval. Effluent limitations serve
122 as the primary mechanism in NPDES permits for controlling discharges of pollutants to receiving waters
123 and the NPDES permit data are cataloged into the Integrated Compliance Information System (ICIS) to
124 track permit compliance and enforcement status. NPDES permittees must then submit Discharge
125 Monitoring Reports (DMRs) to the appropriate permitting authority on a periodic basis to ensure
126 compliance with discharge standards for water quality and human health. Note that EPA does not
127 currently have data available on facilities that indirectly discharge wastewater to POTWs.

128 Available discharge data and permit information was accessed through EPA's Envirofacts and
129 Enforcement Compliance History Online (ECHO) database systems. EPA then investigated these data
130 sources for information pertinent to asbestos COU (chlor-alkali plants, sheet gasket stamping and
131 titanium dioxide plants) to identify if there is evidence of asbestos discharges or concentrations and/or
132 violations of their wastewater permits.

133 **ICIS-NPDES information.** ICIS-NPDES is an information management system maintained by EPA to
134 track permit compliance and enforcement status of facilities regulated by the NPDES under the Clean
135 Water Act. ICIS-NPDES is designed to support the NPDES program at the state, regional, and national
136 levels, and contains discharge monitoring and permit data from facilities in all point source categories
137 who discharge directly to receiving streams.

138 EPA identified pollutant parameter codes in ICIS-NPDES specific to asbestos (such as asbestos, fibrous
139 asbestos, asbestos (chrysotile), asbestos (amphibole), asbestos fibers (ambiguous asbestos), and non-
140 chrysotile, non-amphibole asbestos fibers) and identified unique NPDES-permitted facilities, outfalls,
141 and locations for those asbestos parameters. EPA then cross-checked their identified standard industrial
142 codes (SIC) with SIC codes associated with the current asbestos users and COU. *The results were that*
143 *none of these identified SIC codes were associated with current chrysotile asbestos COUs in this Part 1*
144 *of the risk evaluation for asbestos.*

145 EPA next did a specific NPDES permit search for facilities that may release asbestos (chlor-alkali and
146 sheet gasket facilities) based on gathered location and addresses for these sites. It was found that most
147 chlor-alkali facilities do have issued NPDES permits for industrial (major and minor permit status)
148 operations and for general stormwater and construction stormwater projects. Yet for the identified
149 permits for these industrial subcategories, none of the NPDES limits/monitoring requirements contained
150 asbestos or asbestos-related parameters codes or any direct effluent screening information for asbestos.
151 *Based on the analysis, EPA found no current surface water releases of asbestos or exceedances in the*
152 *ICIS-NPDES database.*

153 **EPA's Water Pollutant Loading Tool.** EPA's Water Pollutant Loading Tool calculates pollutant
154 loadings from NPDES permit and Discharge Monitoring Report (DMR) data from EPA's ICIS-NPDES
155 for industrial and municipal point source dischargers. Data are available from the year 2007 to the
156 present and also include wastewater pollutant discharge data from EPA's Toxics Release Inventory
157 (TRI). The Loading Tool was transitioned into ECHO to increase user access to data and streamline site
158 maintenance and EPA retired the legacy site (the Discharge Monitoring Report Loading Tool) on
159 January 24, 2018. DMR data identifies the permit conditions or limits for each water discharge location,
160 the actual values, identified by the permittee, for each monitored pollutant that was discharged, and
161 whether or not the amounts discharged exceeded the permit limits.

162 DMR was used to help identify facilities with current uses that discharge asbestos to surface water.
163 Information was obtained from the DMR Pollutant loading tool accessed on December 1, 2017.
164 Facilities were identified using two different search methods: 1) "EZ Search" which identifies facilities
165 that submit Discharge Monitoring Reports (DMRs) and 2) "Toxics Release Inventory (TRI) Search"
166 which identifies facilities that report releases to the TRI. Searches were conducted for the two most
167 current (and complete) years in the tool: 2015 and 2016 for DMR facilities, and 2014 and 2015 for TRI
168 facilities.

169 TRI data indicate no releases of asbestos in 2014 and 2015 (only friable asbestos is subject to reporting).
170 The DMR database reported just one facility reporting a discharge in 2014 and 2015 (accessed on
171 December 1, 2017) and this facility has been identified as a mining facility in Duluth, Minnesota. Later,
172 in a subsequent search (October 10, 2018) this facility was no longer identified on the DMR. The DMR
173 reported a total of zero pounds released in 2014 and 2015 but did provide maximum and average
174 effluent concentrations (mg/L) of allowable asbestos. It is assumed that the entry referred to mining
175 runoff, since asbestos has not been mined or otherwise produced in the United States since 2002. *EPA*
176 *has currently not identified in the existing literature or through consultation with industry any evidence*
177 *of discharge to surface water from DMR or TRI database as to any current uses of asbestos (release*
178 *from sheet gaskets, release from working on industrial friction products and/or release from chrysotile*
179 *asbestos diaphragms from chlor-alkali facilities). Based on this database no water dischargers were*
180 *established.*

181 EPA did a search of the database for the parameter description of asbestos and identified three facilities
182 reporting actual limit values of discharge of asbestos to surface water. One of facilities was the mining
183 facility identified earlier on DMR and the other was a quarry. The third was an electric facility. Two
184 other electric facilities were also reported. These facilities were not directly related to the current uses of
185 asbestos mentioned earlier.

186 **STORET.** STORET refers overall to "STORAge and RETrieval", an electronic data system for water
187 quality monitoring data developed by EPA. Since about 2000, STORET has referred to a local data
188 management system ("Modernized STORET") as well as data repository ("STORET Data Warehouse")
189 developed for purposes of assisting data owners to manage data locally and share data nationally. Until
190 September 2009, the distributed STORET database has been used to compile data at the national level in
191 the STORET Data Warehouse. As of September 2009, the Water Quality Exchange, or WQX
192 framework, provides the main mechanism for submitting data to the STORET Data Warehouse.

193 *EPA did not identify in STORET any evidence of discharge to surface water for the COUs of chrysotile*
194 *asbestos. EPA also did not identify in the existing literature or through consultation with industry any*
195 *evidence of discharge to surface water.*

Appendix E Ecological Data Extraction Tables

197 The EPA has reviewed acceptable ecotoxicity studies for Chrysotile Asbestos according to the data
 198 quality evaluation criteria found in the *Application of Systematic Review in TSCA Risk Evaluations* ([U.S.
 199 EPA, 2018a](#)). The ten “on-topic” ecotoxicity studies for asbestos included data from aquatic organisms
 200 (*i.e.*, vertebrates, invertebrates, and plants) and terrestrial species (*i.e.*, fungi and plants). Following the
 201 data quality evaluation, EPA determined that four “on-topic” aquatic vertebrates and invertebrate studies
 202 were acceptable while the two “on-topic” aquatic plants studies were unacceptable as summarized in the
 203 Table APX E-1 below. In the PF, it was determined that the terrestrial exposure pathways, including
 204 biosolids, to environmental receptors was not within the scope of this assessment. As a result, EPA
 205 excluded three studies on terrestrial species from further analysis as terrestrial exposures were not
 206 expected under the conditions of use for asbestos. One amphibian study was excluded from further
 207 review because it was not conducted on chrysotile asbestos. Ultimately four aquatic toxicity studies
 208 were used to characterize the effects of chronic exposure of chrysotile asbestos to aquatic vertebrates
 209 and invertebrates, as summarized in Table 3-1 Environmental Hazard Characterization of Chrysotile
 210 Asbestos.

211 The results of these ecotoxicity study evaluations can be found in *Final Risk Evaluation for Asbestos
 212 Part 1: Chrysotile Asbestos, Systematic Review Supplemental File: Data Quality Evaluation of
 213 Ecological Hazard Studies*. The data quality evaluation indicated these studies are of high confidence
 214 and are used to characterize the environmental hazards of Chrysotile Asbestos. The results of these
 215 studies indicate that there are adverse effects to aquatic organisms following exposure to chrysotile
 216 asbestos.

217 **Table APX E-1. Summary Table On-topic Aquatic Toxicity Studies That Were Evaluated for**
 218 **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 \leq 10^8 fibers/L (100 MFL)	10^8 fibers/L 100 MFL	Gill Tissue Altered	Belanger et al. (1986b)	High
		30d	Reproductive LOEC = 10^4 fibers/L (0.01MFL)	10^4 - 10^8 fibers/L 0.01-100 MFL	Increase in Larvae mortality/ decrease in larvae released		
		96hr-30d	No mortality observed; NOEC $>10^8$ fibers/L (>100 MFL)	10^2 - 10^8 fibers/L 0.0001-100 MFL	Mortality		

Species	Freshwater / Salt Water	Duration	End-point	Concentration(s) (MFL= Millions of fibers per liter)	Effect(s)	Reference	Data Quality Evaluation Rating
		30d	LOEC= 10 ⁸ fibers/L (100 MFL)	10 ² -10 ⁸ fibers/L 0.0001-100 MFL	Growth		
		30d	NOEC < 10 ⁸ fibers/L (<100 MFL) LOEC = 10 ⁸ fibers/L (100 MFL)	10 ² -10 ⁸ fibers/L 0.0001-100 MFL	Fiber Accumulation		
		96hr-30d	LOEC = 10 ² fibers/L (0.0001 MFL)	10 ² -10 ⁸ fibers/L 0.0001-100 MFL	Siphoning Activity		
Asiatic Clams (<i>Corbicula fluminea</i>)	Freshwater	30d	LOEC ≤ 10 ² fibers/L (≤0.0001 MFL)	10 ² -10 ⁸ fibers/L 0.0001-100 MFL	Reduction in siphoning activity	Belanger et al. (1986a)	High
		30d	LOEC ≤ 10 ⁸ fibers/L (≤ 100 MFL)	10 ⁸ fibers/L 100 MFL	Presence of asbestos in tissues		
Coho Salmon (<i>Oncorhynchus kisutch</i>)	Saltwater and freshwater	40-86d	NOEC = 1.5x10 ⁶ fibers/L (1.5 MFL) LOEC = 3.0x10 ⁶ fibers/L (3 MFL)	1.5x10 ⁶ fibers/L, 3.0x10 ⁶ fibers/L 1.5 MFL, 3MFL	Behavioral stress (aberrant swimming, loss of equilibrium) Sublethal effects including epidermal hypertrophy superimposed on hyperplasia, necrotic epidermis,	Belanger et al. (1986c)	High

Species	Freshwater / Salt Water	Duration	End-point	Concentration(s) (MFL= Millions of fibers per liter)	Effect(s)	Reference	Data Quality Evaluation Rating
					lateral line degradation, and lesions near the branchial region		
		40-86d	No significant Mortality; NOEC >3.0x10 ⁶ fibers/L (>3 MFL)	1.5x10 ⁶ fibers/L, 3.0x10 ⁶ fibers/L 1.5 MFL, 3MFL	Mortality		
		40-86d	No Significant effect; NOEC >3.0x10 ⁶ fibers/L (>3 MFL)	1.5x10 ⁶ fibers/L, 3.0x10 ⁶ fibers/L 1.5 MFL, 3MFL	Growth		
Green Sunfish (<i>Lepomis cyanellus</i>)	Freshwater	52-67d	NOEC <1.5x10 ⁶ fibers/L (<1.5 MFL) LOEC = 1.5x10 ⁶ fibers/L (1.5 MFL)	1.5x10 ⁶ 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		

Species	Freshwater / Salt Water	Duration	End-point	Concentration(s) (MFL= Millions of fibers per liter)	Effect(s)	Reference	Data Quality Evaluation Rating
					branchial region		
		40-86d	No significant Mortality; NOEC >3.0x10 ⁶ fibers/L (3 MFL)	1.5x10 ⁶ fibers/L, 3.0x10 ⁶ fibers/L 1.5 MFL, 3MFL	Mortality		
Japanese Medaka (<i>Oryzias latipes</i>)	Saltwater and freshwater	13-21d	No significant effects; NOEC >10 ⁶ fibers/L (>1 MFL)	10 ⁶ -10 ¹⁰ fibers/L 1 MFL-10,000 MFL	Egg development, hatchability, survival.	Belanger et al. (1990)	High
		28d	LOEC = 10 ⁶ fibers/L (1 MFL) NOEC = 10 ⁴ fibers/L (0.01 MFL)	10 ⁶ -10 ¹⁰ fibers/L 1 MFL-10,000 MFL	Significant reduction in growth of larval individuals		
		7w	Not statistically analyzed	10 ⁴ -10 ⁸ fibers/L 0.01-100 MFL	Reproductive performance (viable eggs/day, nonviable eggs/day)		
		49d	LC ₁₀₀ =10 ¹⁰ fibers/L	10 ¹⁰ fibers/L 10,000 MFL	100% Larval mortality		
Duckweed (<i>Lemna gibba</i>)	Freshwater	28d	LOEC = 0.5µg	0.5-5.0 µg chrysotile/frond	Decreased # fronds	Trivedi, 2007 ; Trivedi et al. (2004)	Unacceptable
				0.5-5.0 µg chrysotile/frond	Decreased Root length		

Species	Freshwater / Salt Water	Duration	End-point	Concentration(s) (MFL= Millions of fibers per liter)	Effect(s)	Reference	Data Quality Evaluation Rating
			chrysotile/frond	0.5-5.0 µg chrysotile/frond	Decreased Chlorophyll Content		
			NOEC < 0.5µg chrysotile/frond	0.5-5.0 µg chrysotile/frond	Decreased Carotenoid content		
				0.5-5.0 µg chrysotile/frond	Decrease in biomass/ frond		
				0.5-5.0 µg chrysotile/frond	Decreased Protein content (mg/g fresh wt)		
				0.5-5.0 µg chrysotile/frond	Decreased Free sugar (mg/g fresh wt)		
				0.5-5.0 µg chrysotile/frond	Decreased Starch (mg/g fresh wt)		
				0.5-5.0 µg chrysotile/frond	Decreased photosynthetic pigments		
				0.5-5.0 µg chrysotile/frond	Increased lipid peroxidation		
				0.5-5.0 µg chrysotile/frond	Increased cellular hydrogen peroxide levels		
				0.5-5.0 µg chrysotile/mL	Increase in catalase activity		

Appendix F Environmental Fate Data Extraction Table

221 Environmental Fate Study Summary for Chrysotile Asbestos

222 **Table APX F-1. Other Fate Endpoints Study Summary for Chrysotile 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 asbestos structure is investigated by analyzing the composition of both colonized and uncolonized field samples. The effect of oxalic acid exposure on chrysotile asbestos 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.	Favero-Longo et al. (2005)	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.	Schreier et al. (1987)	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 ₃ PO ₄ , H ₂ SO ₄ 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%),	Due to limited information assessing the results were challenging.	Speil and Leineweber (1969)	Unacceptable

System	Study Type (year)	Results	Comments	Affiliated Reference	Data Quality Evaluation Results of Full Study Report
		Amosite (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%).			

Table APX F-2. Hydrolysis Study Summary for Chrysotile 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 chrysotile and crocidolite 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.	Gronow (1987)	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 ²⁺ 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.	Choi and Smith (1972)	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	Bales and	High

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 ²⁺ sites are removed by dissolution in the outer brucite sheet resulting in exposure of underlying >SiO ⁻ sites.	with this study's overall quality level.	Morgan (1985)	

225

Table APX F-3. Aquatic Bioconcentration Study Summary for Chrysotile 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	1.5×10^6 and 3.0×10^6 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.	Belanger et al. (1986c)	High
Non-guideline; experimental study; uptake monitoring of chrysotile by Asiatic clams	2.5×10^8 - 8.8×10^9 fibers/L	Asiatic clams (<i>Corbicula</i> sp.)	96-hours and 30-days	Chrysotile asbestos was detected in clams at 69.1 ± 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 ± 52.6 fibers/mg dry weight gill tissue and 903.7 ± 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.	Belanger et al. (1986b)	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.	Belanger et al. (1986a)	High

				(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 ⁴ , and 10 ⁸ 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 ⁸ 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.	Belanger et al. (1987)	High
Non-guideline; experimental study; chrysotile asbestos uptake study in Japanese Medaka	5.1±2.8×10 ⁶ , 7.6±8.1×10 ⁸ fibers/L	Japanese Medaka (<i>Oryzias latipes</i>)	13 weeks	After 28 days of exposure to chrysotile asbestos at 10 ¹⁰ fibers/L concentrations, fish total body burden was 375.7 fibers/mg. After 3 months of exposure to chrysotile asbestos at 10 ⁸ fibers/L concentrations, fish total body burden was 486.4±47.9 fibers/mg.	The reviewer agreed with this study's overall quality level.	Belanger et al. (1990)	High

Appendix G SAS Codes for Estimating K_L and K_M from Grouped Data

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```
/*This SAS code estimates a value for lung cancer potency (KL) using Poisson maximum likelihood
estimation (MLE), along with the 90% confidence interval (CI) generated using the likelihood profile
method. The basic model is  $RR = 1 + CE10 * KL$ .

This code was created by Rebekha Shaw and Bill Thayer at SRC Inc. This is version 1.0 */

/*This is where the code begins execution. */
/*The first step is to create a data table */
data Data_Table;
input CE10_min CE10_max CE10_mid Observed Expected RR;

/*enter data here */
datalines;
0 20 10.0 6 5.75 1.04
20 100 60.0 12 2.82 4.25
100 450 275.0 17 1.57 10.82
450 1097 773.5 21 1.23 17.07
;

/* Enter text string to identify data source */
title "Wang et al 2013";

/*model*/

proc nlmixed data=Data_Table;
parms KLE2 10; /* KLE2 =  $KL * 1E+02$ . The initial guess is 10. This can be changed if a solution is not
found (unlikely). */

Predicted = (1+CE10_mid*KLE2/100)* Expected; /*equation to calculate predicted number of lung cancer
cases*/

LL=LogPDF("POISSON",Observed,Predicted); /*LogPDF function Returns the logarithm of a probability
density (mass) function. Poisson distribution is specified. */

model Observed ~ general(LL);

estimate 'KLE2' KLE2 ALPHA=0.1; /*generates "Additional Estimates" table in the Results tab with Wald 90%
CI's*/
predict Predicted out=Predicted alpha=0.1; /*generates SAS data table with predicted values and CI's
titled "Predicted"*/
ods output FitStatistics = FitStats;
ods output ParameterEstimates = ModelParams;

Proc print data=Predicted; /*Prints the "Predicted" table in the Results tab*/
run;

data _null_;
set Fitstats;
if _n_ =1;
LLTarget = (Value/-2)-1.353; /*calculates LL_target - needed to run macro PoissonLLBounds*/

call symputx("LLTarget",LLTarget); /*creates macro variable*/
run;
data _null_;
set ModelParams;

KLMLE = Estimate*1e-02; /*variable KL_MLE in macro PoissonLLBounds*/
KLINITLB= Estimate*1e-02/10; /*Calculates the initial guess for the lower bound - variable KL_init_LB in
macro poissonLLBounds*/
KLINITUB= Estimate*1e-02*10; /*Calculates the initial guess for the upper bound - variable KL_init_LB in
macro PoissonLLBounds*/
call symputx("KLMLE", KLMLE); /*creates macro variable*/
call symputx("KLINITLB", KLINITLB); /*creates macro variable*/
call symputx("KLINITUB", KLINITUB); /*creates macro variable*/
run;

/*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 */
%macro PoissonLLBounds(inputData=, KL_MLE=, KL_Init_LB=, KL_Init_UB=,
conv_criterion=, LL_target=, max_iteration=);
```

```

301
302 %Let dsid=%sysfunc(open(&inputdata)); * open the input data file;
303 %Let NumSamples=%sysfunc(attrn(&dsid,nobs)); * get the number of observations;
304 %Let rc=%sysfunc(close(&dsid)); * close the data file;
305
306 %Do j=1 %To 2; * one for upper bound and one for lower bound;
307
308 %If %eval(&J=1) %then %Let KL=&KL_init_LB;
309 %If %eval(&J=2) %then %Let KL=&KL_Init_UB;
310
311 %Let i=1; * first time through loop;
312
313 %Let ConvFactor = 10;
314 %let ConvRate = %sysevalf(((&KL_MLE-&KL)/&KL_MLE)/10);
315
316 %Let ConvDirect = -1;
317 /* negative=from the left and positive=from the right. For lower bound, the initial guess is less than
318 the target LL so the initial value of convdirect is -1 */
319
320 %Let KLAdjust=%Sysevalf(-1*&ConvDirect*&KL*&ConvRate);
321
322 %Do %Until (%sysevalf(&DeltaLL < &conv_criterion) OR %sysevalf(&i > &max_iteration));
323
324 Data tempDataLLBound; Set &InputData;
325 Predicted = (1 + CE10_Mid * &KL) * Expected;
326 LL=(LogPDF("POISSON",Observed,Predicted)); * likelihood for each
327 observation;
328 LL_sum+LL;
329 output;
330 Run;
331
332 Data TempDataLLBound2; Set tempDataLLBound;
333 If _N_ = &NumSamples;
334 NumLoops=&i;
335 thisKL=&KL;
336 ConvRateVar=&ConvRate;
337 ConvFactorVar=&ConvFactor;
338 ConvDirectVar= %eval(&ConvDirect);
339
340 KLAdjustVar=(-1*ConvDirectVar)*thisKL*ConvRateVar;
341 If &ConvDirect=-1 then DiffLL=abs(LL_sum)-abs(&LL_Target);
342 Else DiffLL=abs(&LL_Target)-abs(LL_Sum);
343
344 /* Test if we have changed direction on the convergence. If we have, change direction
345 (subtract from current value if we were adding before...) and decrease the convergence rate
346 (ConvRate) by a factor = ConvFactor. */
347
348 if DiffLL<0 then
349 do; /* need to change directions and make conv rate more gradual */
350 ConvDirectVar= %eval(-1*&ConvDirect);
351 ConvRateVar=%sysevalf(&convRate/&ConvFactor);
352 KLAdjustVar=(-1*ConvDirectVar)*thisKL*ConvRateVar;
353 call symput('KLAdjust',KLAdjustVar);
354 call symput('ConvDirect',ConvDirectVar);
355 call symput('convRate',ConvRateVar);
356 end;
357 AbsDiffLL=abs(DiffLL);
358
359 call symput('DeltaLL',AbsDiffLL);
360
361 output;
362 Run;
363
364 Data tempAllOutput; if _N_=1 then Set TempDataLLBound2; Set tempDataLLBound; Run;
365
366 %If %eval(&i=1) %then %do; Data AllOutput; Set tempAllOutput; Run; %end;
367
368 %If %eval(&i>1) %then %do; Proc Append base=AllOutput data=tempAllOutput; Run;
369
370 %End;
371
372 %Let i=%eval(&i+1);
373
374 %Let KL=%sysevalf(&KL + &KLAdjust);
375
376 %End;

```

```

376
377      %If %eval(&J=1) %then
378          %Do;
379              Data tempout1; length limit $5; Set TempDataLLBound2; limit='lower';
380 estimate=thisKL; LogLikelihood=LL_sum; loops=numloops; Run;
381          %End;
382      %If %eval(&J=2) %then
383          %Do;
384              Data tempout2; length limit $5; Set TempDataLLBound2; limit='upper';
385 estimate=thisKL; LogLikelihood=LL_sum; loops=numloops; Run;
386          %End;
387 %End;
388
389      Data PrntOutput; Set tempout1 tempout2; run;
390
391      Proc print data=PrntOutput; var limit estimate LogLikelihood Loops ; Run;
392
393 %Mend;
394
395 /*run macro PoissonLLBounds*/
396 %PoissonLLBounds(inputData=Data_Table,
397                  KL_MLE=&KLMLE,
398                  KL_Init_LB=&KLINITLB,
399                  KL_Init_UB=&KLINITUB,
400                  conv_criterion=0.001,
401                  LL_target=&LLTarget,
402                  max_iteration=100);
403 run;
404
405 /*the following code creates a summary table with the MLE KLE and confidence bounds*/
406 PROC SQL;
407     CREATE TABLE WORK.MLEKL AS
408     SELECT ("MLE KLE") AS Parameter,
409            (t1.Estimate*1e-2) AS Value
410     FROM WORK.MODELPARAMS t1;
411 QUIT;
412
413 PROC SQL;
414     CREATE TABLE WORK.LBKLUK AS
415     SELECT (case
416            when t1.limit="lower" then "5% LB KL"
417            else "95% UB KL"
418            end) AS Parameter,
419            t1.estimate AS Value
420     FROM WORK.PRNTOUTPUT t1;
421 QUIT;
422
423 PROC SQL;
424     CREATE TABLE WORK.Parameter_Values AS
425     SELECT * FROM WORK.MLEKL
426     OUTER UNION CORR
427     SELECT * FROM WORK.LBKLUK
428     ;
429 Quit;
430
431 Proc print data=Work.Parameter_values;
432 run;
433

```

```

434 /*This SAS code estimates a value for mesothelioma potency (KM) using Poisson maximum likelihood estimation (MLE), along with the 90% confidence interval (CI)
435 generated using the likelihood profile method.
436 This code was created by Rebekha Shaw and Bill Thayer at SRC Inc.
437 This is version 1.0*/
438
439 /*This is where the code begins execution. */
440 data Data_Table;
441 input TSFE_Min TSFE_Max TSFE_Mid Duration Conc PY Obs ;
442 /*The values of TSFE_Mid and Duration are used to calculate a parameter called Q. */
443 if TSFE_Mid=. then Q = .;
444 else if TSFE_Mid<10 then Q = 0;
445 else if TSFE_Mid>(10+duration) then
446     Q = (TSFE_Mid-10)**3-(TSFE_Mid-10-duration)**3;
447 else Q =(TSFE_Mid-10)**3;
448
449 /*enter data here. The contents of the columns are as follows:
450
451 TSFE_Min (years)
452 SFE_Max (years)
453 TSFE_Mid (years)
454 Duration (years)
455 Conc (#/cc)
456 Person Years (PY)
457 Observed cases(Obs)
458
459 */
460 datalines;
461 20 30 27.7 1.00 6.5 1926 0
462 30 40 33.9 2.10 8.7 6454 0
463 40 50 43.1 3.00 14.6 3558 2
464 50 72 53.56 5.78 31.4 1080 2
465 ;
466
467 /*enter the name of the data set*/
468 title "North Carolina Sub Co-hort (1999-2003;4 groups)";
469 run;
470 /*model*/
471 proc nlmixed data= Data_Table;
472 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*/
473 Pred = Conc*Q*PY*KME8/1e+08; /*equation to calculate predicted values*/
474 LL=LogPDF("POISSON",Obs,Pred); /*LogPDF function Returns the logarithm of a probability density (mass) function. Poisson distribution is specified.*/
475 model Obs ~ general(11);
476
477 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
478 displayed in the SAS output */
479 predict Pred out=Predicted alpha=0.1; /*generates SAS data table with predicted values and CI's titled "Predicted"*/
480 ods output FitStatistics = FitStats;
481 ods output ParameterEstimates = ModelParams;
482 run;
483 Proc print data=Predicted; /*Prints the "Predicted" table in the Results tab*/
484 OPTIONS MPRINT SYMBOLGEN ; /*this prints in the log what value is used for each variable in the macro*/
485 run;
486 data _null_ ;
487 set Fitstats;
488 if _n_ =1;
489 LLTarget = (Value/~2)-1.353; /*calculates LL_target - needed to run macro PoissonLLBounds*/
490
491 call symputx("LLTarget",LLTarget); /*creates macro variable*/
492 run;
493 data _null_ ;
494 set ModelParams;
495
496 KMLE = Estimate*1e-8; /*scales back the KM MLE value generated by Proc nlmixed - variable KM_MLE in macro PoissonLLBounds*/
497 KMINITLB= Estimate*1e-8/10; /*Calculates the initial guess for the lower bound - variable KM_init_LB in macro poissonLLBounds*/
498 KMINITUB= Estimate*1e-8*10; /*Calculates the initial guess for the upper bound - variable KM_init_LB in macro PoissonLLBounds*/
499 call symputx("KMLE", KMLE); /*creates macro variable*/
500 call symputx("KMINITLB", KMINITLB); /*creates macro variable*/
501 call symputx("KMINITUB", KMINITUB); /*creates macro variable*/
502 run;
503
504 /*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 */
505
506 %macro PoissonLLBounds(inputData=, KM_MLE=, KM_Init_LB=, KM_Init_UB=,
507 conv_criterion=, LL_target=, max_iteration=);

```

```

509 %Let dsid=%sysfunc(open(&inputdata)); * open the input data file;
510 %Let NumSamples=%sysfunc(attrn(&dsid,nobs)); * get the number of observations;
511 %Let rc=%sysfunc(close(&dsid)); * close the data file;
512
513
514
515 %Do j=1 %To 2; * one for upper bound and one for lower bound;
516
517 %If %eval(&J=1) %then %Let KM=&KM_init_LB;
518 %If %eval(&J=2) %then %Let KM=&KM_init_UB;
519
520 %Let i=1; * first time through loop;
521
522 %Let ConvFactor = 10;
523 %Let ConvRate = %syssevalf(((&KM_MLE-&KM)/&KM_MLE)/10);
524
525 %Let ConvDirect = -1;
526 /* 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 */
527
528 %Let KMadjust=%syssevalf(-1*&ConvDirect*&KM*&ConvRate);
529
530 %Do %Until (%syssevalf(&DeltaLL < &conv_criterion) OR %syssevalf(&i > &max_iteration));
531
532 Data tempDataLLBound; Set &InputData;
533 E = Conc * Q * PY * &KM;
534 LL=(LogPDF("POISSON",Obs,E)); * likelihood for each observation;
535 LL_sum+LL;
536 output;
537
538 Run;
539
540 Data TempDataLLBound2; Set tempDataLLBound;
541 If _N_ = &NumSamples;
542 NumLoops=&i;
543 thisKM=&KM;
544 ConvRateVar=&ConvRate;
545 ConvFactorVar=&ConvFactor;
546 ConvDirectVar= %eval(&ConvDirect);
547
548 KMadjustVar=(-1*&ConvDirectVar)*thisKM*&ConvRateVar;
549 If &ConvDirect=-1 then DiffLL=abs(LL_sum)-abs(&LL_Target);
550 Else DiffLL=abs(&LL_Target)-abs(LL_Sum);
551
552 /* Test if we have changed direction on the convergence. If we have, change direction (subtract from current value if we were adding before...)
553 and decrease the convergence rate (ConvRate) by a factor = ConvFactor.*/
554
555 if DiffLL<0 then
556 do; /* need to change directions and make conv rate more gradual */
557 ConvDirectVar= %eval(-1*&ConvDirect);
558 ConvRateVar=%syssevalf(&convRate/&ConvFactor);
559 KMadjustVar=(-1*&ConvDirectVar)*thisKM*&ConvRateVar;
560 call symput('KMadjust',KMadjustVar);
561 call symput('ConvDirect',ConvDirectVar);
562 call symput('ConvRate',ConvRateVar);
563 end;
564 AbsDiffLL=abs(DiffLL);
565
566 call symput('DeltaLL',AbsDiffLL);
567
568 output;
569 Run;
570
571 Data tempAllOutput; if _N_=&1 then Set TempDataLLBound2; Set tempDataLLBound; Run;
572 %If %eval(&i=1) %then %do; Data AllOutput; Set tempAllOutput; Run; %end;
573
574 %If %eval(&i>1) %then %do; Proc Append base=AllOutput data=tempAllOutput; Run; %End;
575
576 %Let i=%eval(&i+1);
577
578 %Let KM=%syssevalf(&KM + &KMadjust);
579
580 %End;
581
582 %If %eval(&J=1) %then
583 %Do;

```

```

584                                     Data tempout1; length limit $5; Set TempDataLLBound2; limit='lower'; estimate=thisKM; LogLikelihood=LL_sum;
585 loops=numloops; Run;
586                                     %End;
587                                     %If %eval(&J=2) %then
588                                     %Do;
589                                     Data tempout2; length limit $5; Set TempDataLLBound2; limit='upper'; estimate=thisKM; LogLikelihood=LL_sum;
590 loops=numloops; Run;
591                                     %End;
592 %End;
593
594 Data PrntOutput; Set tempout1 tempout2; run;
595
596 Proc print data=PrntOutput; var limit estimate LogLikelihood Loops ; Run;
597
598 %Mend;
599
600 /*run macro PoissonLLBounds*/
601 %PoissonLLBounds(inputData=Data_Table,
602                 KM_MLE=%KMMLE,
603                 KM_Init_LB=%KMINITLB,
604                 KM_Init_UB=%KMINITUB,
605                 conv_criterion=0.001,
606                 LL_target=%LLTarget,
607                 max_iteration=100);
608 run;
609

```


Appendix H BEIR IV Equations for Life Table Analysis

610

611 Lung Cancer

612

613 Let e_i be the calculated excess relative risk of lung cancer in an exposed individual at age i .

614

615 Then:

616

617
$$\text{Excess Lifetime Risk} = \text{Re}_{lt} - \text{R}0_{lt}$$

618

$$\text{R}0_{lt} = \sum_{i=1}^{110} \text{R}0_i$$

619

$$\text{Re}_{lt} = \sum_{i=1}^{110} \text{Re}_i$$

620

$$\text{R}0_i = \frac{h_i}{h_i^*} S_{1,i} (1 - q_i)$$

621

$$\text{Re}_i = \frac{he_i}{he_i^*} Se_{1,i} (1 - qe_i)$$

622

$$he_i = h_i (1 + e_i)$$

623

$$he_i^* = h_i^* + h_i e_i$$

624

$$q_i = \exp(-h_i^*)$$

625

$$qe_i = \exp(-he_i^*)$$

626

$$S_{1,i} = \prod_{j=1}^{i-1} q_j$$

627

$$Se_{1,i} = \prod_{j=1}^{i-1} qe_j$$

628

where:

629

630 i and j = Year index (1 = year 0-1, 2 = year 1-2, etc.)

631 $\text{R}0_{lt}$ = Lifetime risk of lung cancer in the absence of exposure

632 Re_{lt} = Lifetime risk of lung cancer in the presence of exposure

633 $\text{R}0_i$ = Risk of lung cancer in the absence of exposure in year i

634 Re_i = Risk of lung cancer the presence of exposure in year i

635 h_i = Lung cancer incidence rate in the absence of exposure in year i

636 h_i^* = All-cause mortality rate in the absence of exposure in year i

637 q_i = Probability of surviving year i , all causes acting (no exposure)

638 qe_i = Probability of surviving year i , all causes acting (with exposure)

639 $S_{1,i}$ = Probability of surviving up to start of year i , all causes acting (no exposure)

640 $Se_{1,i}$ = Probability of surviving up to start of year i , all causes acting (with exposure)

641 Mesothelioma

642

643 The same basic approach is followed for calculating lifetime risk of mesothelioma, except that the
644 baseline (un-exposed) risk is so small that it is generally assumed to be zero. Thus, the equations for
645 calculating lifetime mesothelioma risk are the same as above, except as follows:

646

647 $m_i =$ risk of mesothelioma in an exposed individual at age i

648

649
$$Re_{it} = \sum_{i=1}^{110} Re_i$$

650

651
$$Re_i = \frac{m_i}{he_i^*} Se_{1,i} (1 - qe_i)$$

Appendix I SAS Code for Life Table Analysis

There are three SAS programs in this appendix:

- **Lung Cancer Lifetable for Linear Models**
- **Lung Cancer Lifetable for Non-Linear Models**
- **Mesothelioma Lifetable**

SAS Lung Cancer Lifetable for Linear Models

```
666 OPTIONS NODATE NONUMBER orientation=landscape linesize=max; *BT added 7/3/19;
667
668 /*
669 This program calculates the risk of lung cancer from inhalation exposure to asbestos,
670 using a lifetable approach based on BEIR IV. The basic exposure-response model is  $RR = 1 + CE10 * KL$ .
671
672 The basic code for the lifetable calculations were developed and provided to EPA
673 by Randall Smith at NIOSH. The code from NIOSH calculates the baseline risk (R0) and the exposed
674 risk (Rx)
675 from exposure to an exposure concentration of X_Level using NIOSH Model 2:  $Rx = R0 * (1 + COEF * X)$ .
676
677 EPA has modified the NIOSH code as follows:
678
679 1) The all-cause mortality and cause-specific (lung cancer) incidence data tables have been
680 updated based on CDC Wonder 2017.
681
682 2) An equation has been added to calculate extra risk:  $Extra\_Risk = (Rx - R0) / (1 - R0)$ 
683
684 3) A macro has been added to find the exposure level (X_Level) that yields an extra risk of 0.01
685 (1%).
686 This is referred to as EC1%, which may then be used to calculate the unit risk:  $UR = 0.01 / EC1\%$ 
687
688 */
689
690 /* .\Beta Version.sas 19jan00, 26jul00, 25oct01, 06dec05, 30nov18
691 -----
692 Experimental version
693 ----- */
694
695 title "Lifetable calculation of lung cancer risk";
696 title2 "under a linear relative rate model";
697
698 /*-----+
699 | Compute excess risk by the BEIR IV method using SAS datasteps. |
700 |
701 | These programs compute the risk of a cause-specific |
702 | death in the presence of competing risks, where the cause- |
703 | specific death-rate is modeled either as a relative rate |
704 |  $[h=h0*f(Coef*X)]$  or as an absolute rate  $[h=h0+f(Coef*X)]$  |
705 | where |
706 | h denotes the cause-specific death-rate, |
707 | X denotes cumulative occupational exposure (with Lag) |
708 | Coef denotes the coefficient for the effect of exposure and |
709 | h0 is the corresponding rate at baseline (X=0). |
710 | (Except for Coef, these are functions of age.) |
711 |
712 | A few simple models of f(Coef*X) are easily specified as |
```

```

713 | described below. More complicated models can be specified with |
714 | a little more work. (For a more complicated example, |
715 | see \_GENERAL.LIB\PROGRAMS\SAS\BEIR-4.Method\BEIR4ex2.SAS). |
716 | |
717 +Reference: |
718 | Health Risks of Radon and Other Internally Deposited Alpha- |
719 | Emitters (BEIR IV). Committee on the Biologic Effects of |
720 | Ionizing Radiations. National Academy Press. Wash. DC (1988). |
721 | See especially pages 131-136. |
722 | |
723 | |
724 +USER-SUPPLIED ASSIGNMENTS: |
725 | |
726 |> The following macro variables are assigned using "%LET" state- |
727 | ments: MODEL, COEF, LAG, AGE1ST_X, DURATION, LASTAGE. |
728 | Further information appears below. |
729 |> Exposure concentrations for computing risk are defined |
730 | in the dataset "X_LEVELS." |
731 |> All-cause mortality information is entered as a life-table in |
732 | the data step "ALLCAUSE," and converted to rates per individual. |
733 |> Cause-specific incidence information for unexposed referents is |
734 | entered as rates per 100,000 and converted to rates per |
735 | individual in the data step "CAUSE." |
736 | |
737 | |
738 +NOTES: |
739 |> Dataset "EX_RISK" is where the desired risks are computed. |
740 | |
741 |> If the unexposed(referent) cause-specific incidence rate is from |
742 | a model then dataset "CAUSE" with variables AGE and RATE as |
743 | modeled can be modified to incorporate this. However, care |
744 | must be taken in calculating confidence limits since imprecision |
745 | in the estimates of all of the parameters of the model |
746 | contributes to the imprecision of excess risk estimates. |
747 | |
748 |> This program is currently set up to apply the Linear Rel. Rate |
749 | model (Lag= 0) and accumulation of excess risk is over the |
750 | rates in ALLCAUSE and CAUSE unless truncated at a younger age. |
751 | (See LASTAGE below.) |
752 | |
753 | |
754 + SAS Programmer: Randall Smith |
755 | The Nat'l Inst. for Occupational Safety & Health |
756 | 26jul2000, 23jul2001, 25oct2001, 18nov2018 |
757 + Modifications: |
758 + |
759 | 26jul00 Fix the procedure bug causing it to report incorrectly |
760 | the age at which accumulation of risk was stopped |
761 | whenever the age-specific rates included ages |
762 | before the value of &Age1st_X. (&Age1st_X is a macro |
763 | expression defining the age exposure begins.) |
764 | |
765 | 23jul01 Make changes to facilitate multiple applications of |
766 | BEIR4 algorithm, i.e., MLE(Excess Risk), UCL(ExcessRisk), |
767 | searching for concentrations for a fixed risk. These |
768 | changes involve defining Macros named BEIR4 and SEARCH |
769 | given below with code illustrating these uses for the |
770 | linear relative rate model. |
771 | |
772 | 25oct01 Modified to add Macro variable EnvAdj for whether to |
773 | increase inhaled dose from intermittent occupational |
774 | exposures to continuous environmental exposures |
775 | and update US rates for Gibb et al. cohort. |
776 | |
777 | 30nov18 A bug that prevented the calculation of excess risks |
778 | after incorporating an adjustment from intermittent |
779 | occupational exposures to continuous exposures is fixed. |
780 | |
781 | +---|
782 | March 2019: BT (SRC) Added macro CONVERGE_BEIR4 which iteratively |
783 | runs macro BEIR4 until the EXPOSURE_CONCENTRATION corresponds to an |

```

```

784 |extra_risk=0.01 (the point of departure [POD]).
785 |
786 |
787 | Macro CONVERGE_BEIR4 works with one value for the exposure
788 | variable XLevel (i.e., when the data C_Levels includes one record.)
789 |
790 |
791 | The intent was to make as few changes to BEIR4 as possible. The data
792 | X_LEVELS and variable XLevel are retained but the initial value of
793 | XLevel is provided in the call to macro CONVERGE_BEIR4 (the value
794 | of Xlevel in the cards statement is not used in the calculations.
795 | Changes to the BEIR4 macro are in Part III and Part IV, and are
796 | indicated by the letters BT.
797 |
798 |
799 |
800 | In addition to the parameter values that are specified by the user
801 | in PART 1, and the user-provided data entered in Part II, parameters
802 | for the new macro CONVERGE_BEIR4 are specified in the call to the
803 | macro CONVERGE_BEIR4 (see end of this SAS program file below).
804 +-----*/
805
806
807 /* PART I. USER-SUPPLIED ASSIGNMENTS (Macro variables):
808 /*-----+
809 | Model of cumulative exposure effects:
810 |     1 => Loglinear Relative rate
811 |           R=R0*exp(COEF*X)
812 |     2 => Linear Relative rate,
813 |           R=R0*(1+COEF*X)
814 |     3 => Absolute rate,
815 |           R=R0+COEF*X
816 |     4 => Power relative rate
817 |           R=R0*(1+X)^COEF
818 |     0 => User Defined & programmed
819 |           in dataset Ex_Risk below
820 |
821 |
822 | Cumulative exposure parameter:
823 |
824 | Lag or delay between exposure and effect:
825 |
826 | Age exposure begins:
827 | Exposure duration (years):
828 | Adjust dose from occupational to
829 | continuous environmental exposures (Y/N)?
830 | Age to stop accumulating excess risk
831 | (supposing rates are available for
832 | ages >= &LastAge); otherwise use all of
833 | the supplied rate information:
834 +-----*/
835
836
837 /* PART II. USER-SUPPLIED ASSIGNMENTS (Datesets AllCause, Cause, X_Levels ): */
838
839
840 data AllCause (label="Unexposeds' age-spec mortality rates (all)"
841               drop=Lx rename=(BLx=Lx) );
842
843 +-----+
844 | Input lifetable and calculate the corresponding age-specific
845 | (all-causes) mortality rate (AllCause) and conditional survival
846 | probability for each year of age (qi) together with
847 | the corresponding values of age (Age).
848 +-----*/
849
850 Label Age = "Age at start of year (Age=i)"
851       BLx = "Number alive at start of year"
852       Lx = "Number alive at end of year"
853       CndPrDth = "Pr[Death before age i+1 | alive at age i]"
854       qi = "Pr[Survive to age i+1 | Alive at age i]"
855       AllCause = "Age-spec mortality rate (all causes)";

```

```

855     if _n_=1 then input age  /// @1 BLx @; /* /// => skip next 4 lines */
856     input Lx @@;
857     CndPrDth = (BLx - Lx)/BLx;
858
859     qi      = 1-CndPrDth;
860     if qi <= 0 then AllCause = 1e+50;
861             else AllCause = - log(qi);
862
863     if age < &LastAge then output; else STOP;
864     BLx=Lx;
865     age+1;
866     retain age BLx;
867 cards;
868     0 = Life-table starting age. (Required: Values must begin 4 lines down!)
869     The following are 2017 Life-table values of US population
870     starting at birth and ending at age 85.
871     (Source: Nat.Vital Statistics Reports 2019 Vol 68 No 7, Table 1,
872     https://www.cdc.gov/nchs/data/nvsr/nvsr68/nvsr68_07-508.pdf)
873     100000 99422 99384 99360 99341 99326 99312 99299 99288 99278
874     99268 99259 99249 99236 99217 99191 99158 99116 99066 99006
875     98937 98858 98770 98674 98573 98466 98355 98241 98122 97999
876     97872 97740 97603 97461 97314 97163 97006 96843 96674 96501
877     96321 96135 95939 95732 95511 95275 95023 94753 94461 94144
878     93797 93419 93008 92560 92070 91538 90963 90345 89684 88978
879     88226 87424 86570 85664 84706 83696 82632 81507 80315 79048
880     77697 76265 74715 73064 71296 69418 67402 65245 62933 60462
881     57839 55053 52123 49035 45771 42382
882
883 ;
884 *run;*BT 7/3/19 added Run statement here;
885
886     data CAUSE (label="Unexposeds' age-cause-spec mortality rates");
887     /*-----+
888     | Specify unexposeds' age-specific mortality rates (per year) |
889     | from specific cause. |
890     +-----*/
891     label Age      = "Age"
892           Rate_e5  = "Age,cause-specific rate per 100,000"
893           Rate     = "Age,cause-specific rate per individual";
894
895     if _n_ = 1 then input age  /* input starting age */
896                       ///;   /* /// => skip next 3 lines */
897     input Rate_e5 @@;
898
899     Rate = Rate_e5 * 1e-5; /* Convert to rate per individual */
900
901     if age <= 4
902     then DO; output; age+1; END;
903     else DO i = 0,1,2,3,4; /*-----*/
904           if age < &LastAge /* Fill out into yearly intervals from */
905           then output; /* inputted five year intervals after age 4*/
906           age+1; /*-----*/
907     END;
908 cards;
909     0 = Start age of cause-specific rate (Required: Rates begin 3 lines down!)
910     The following are 2017 cancer site code 22030 lung and bronchus incidence rates per
911     100,000 for US pop'n starting at birth.
912     For ages 5 and above, each rate holds for the age thru age+4 years.
913     Source: CDC Wonder, https://wonder.cdc.gov/cancer-v2017.HTML
914     0.205 0.100 0.100 0.100 0.100 0.039 0.039 0.104 0.299 0.553 1.267 2.600 6.534 16.528 44.403
915     96.098 149.112 223.906 319.322 391.202 395.215
916
917 ;
918 *run; *BT 7/3/19 added Run statement here;
919
920     data X_LEVELS (label= "Exposure levels (e.g., concentrations)");
921     /*-----+
922     | Specify environmental exposure levels |
923     | and update label for the variable, XLevel, if necessary: |
924     +-----*/
925     /*-----+

```

```

926 | BT 3/8/19: Add macro CONVERGE_BEIR4 which iteratively runs macro |
927 | BEIR4 until the EXPOSURE_CONCENTRATION corresponds to extra_risk=0.01|
928 | |
929 | |
930 | |
931 | The intent was to make as few changes to BEIR4 as possible. The data |
932 | X_LEVELS and variable XLevel are retained but the initial value of |
933 | XLevel is provided in the call to macro CONVERGE_BEIR4 (the value |
934 | of Xlevel in the cards statement is not used in the calculations. |
935 |-----*/
936
937     input XLevel @@;
938     label XLevel= "Asbestos exposure (F/ml)";
939     cards;
940     0.0383
941     ;
942 run;
943
944 %Macro BEIR4;
945 /* March 2019 - BT (SRC): Macro BEIR4 is now called by macro CONVERGE_BEIR4.
946 */
947 /* 23jul01 modification */
948 /* Enclose the actual calculations and printed results in a macro */
949 /* to facilitate multiple applications of the algorithm. */
950
951 /* PART III. Perform calculations: */
952
953     data EX_RISK (label = "Estimated excess risks [Method=BEIR IV]"
954                 /*keep = XLevel Rx ex_risk RskRatio R0 extra_Risk */
955                 rename= (Rx=Risk));
956 /*-----+
957 | Calculate risk and excess risk for each exposure concentration|
958 | in work.X_Level by BEIR IV method using information in |
959 | work.AllCause and work.Cause to define referent population: |
960 |-----*/
961     format rate F15.8 hi F15.8; *BT 7/3/19: added the format statement;
962     length XLevel 8.;
963     label Age = "Age at start of year (i)"
964           XTime = "Exposure duration midway between i & i+1"
965           XDose = "CE10(adj) (f/cc-yrs)"
966
967           R0 = "Cumulative Risk of lung cancer (unexposed) (R0)"
968           Rx = "Cumulative risk of lung cancer (exposed) (Re)"
969           Ex_Risk = "Excess risk (Rx-Ro)"
970           RskRatio = "Ratio of risks (Rx/Ro)"
971
972           hi = "Lung Cancer hazard (unexposed) (hi)"
973           hix = "Lung Cancer hazard (exposed) (hei)"
974           hstari = "All cause hazard (unexposed) (h*i)"
975           hstarix = "All cause hazard (exposed) (he*i)"
976           qi = "Probability of surviving year i assuming alive at start (unexposed)
977 (qi)"
978           S_li = "Probability of surviving to end of year i (unexposed) (S1,i)"
979           S_lix = "Probability of surviving to end of year i (exposed) (S1,i)";
980
981     /* BT 3/8/19: Calculation of unexposed's risk (following DO LOOP) could be omitted
982 from the iteration
983 but may require further changes to BEIR4(?).
984 *e.g., %if i=1 %then %do;*/
985
986     if _n_=1 then DO;
987     /* Calculate unexposed's risk (R0) to be retained */
988     /* based on equation 2A-21 (pg. 131) of BEIR IV: */
989
990     /* Initialize: */ S_li = 1; R0 = 0;
991
992     DO pointer = 1 to min(n_all,n_cause) until (age>=&LastAge-1);
993     set allcause (keep=age AllCause rename=(AllCause=hstari))
994     point=pointer nobs=n_all;
995     set cause (keep=age Rate rename=(age=ageCause Rate=hi))
996     point=pointer nobs=n_cause;

```

```

997
998
999
1000 conform ***
1001
1002 / @13 "Rates misaligned on age could give incorrect results"
1003 / @13 Pointer=
1004 +2 "Age(ALLCAUSE)=" Age +2 "Age(CAUSE)=" AgeCause /;
1005
1006 qi = exp(-hstari);
1007 R0 = R0 + ( hi/hstari * S_li * (1-qi) );
1008 S_li = S_li * qi;
1009
1010 END;
1011 END; /* End of 'if _n_=1 then DO;' stmt */
1012
1013 retain R0;
1014
1015 /* Calculate exposed's risk (Rx, renamed to Risk) for each exposure level
1016 */
1017 /* ultimately based on equation 2A-22 (pg. 132) of BEIR IV */
1018 /* but re-expressed in a form similar to equation 2A-21: */
1019
1020 is * BT 3/20/19. This version of CONVERGE_BEIR4 will work when there
1021
1022 one concentration in data set x_levels -
1023 i.e., one value for xlevel.
1024 The Do loop for X_levels is commented out;
1025 *DO pointX = 1 to No_of_Xs;
1026 * set x_levels point=pointX nobs=No_of_Xs; /* BT 3/8/19: determines
1027 when to end the loop. Nobs is set at compilation,
1028
1029 so the value of nobs is available at first run through loop -
1030
1031 just one record and one variable (XLevel) in dataset x_levels. */
1032
1033 /* BT 3/20/19: added the next line to set the exposure
1034 concentration = current value of &exposure_conc. */
1035 xlevel = &exposure_conc;
1036
1037 /* Initialize : */ S_lix = 1; Rx = 0;S_li=1; R0=0;
1038
1039 DO pointer = 1 to min(n_all,n_cause) until (age>=&LastAge-1);
1040 set allcause (keep=age AllCause rename=(AllCause=hstari))
1041 point=pointer nobs=n_all;
1042 set cause (keep=Rate rename=(Rate=hi))
1043 point=pointer nobs=n_cause;
1044
1045 XTime = min( max(0, (age+0.5-&Agelst_x-&Lag))
1046 , &Duration);
1047
1048 if UpCase("&EnvAdj") = "YES" /* Occupational
1049 to Environmental Conversion */
1050 then
1051 XDose = XLevel
1052 * 365/240 /* Days per year */
1053 * 20/10 /* Ventilation (L) per day */
1054 * XTime;
1055
1056 ELSE if UpCase("&EnvAdj") = "NO" /* 30nov2018 ('ELSE') */
1057 then XDose = XLevel*XTime;
1058 else DO; put //"Macro variable ENVADJ incorrectly specified."
1059 //"It should be either YES or NO. Value specified
1060 is: &ENVADJ"
1061
1062 /;
1063 STOP;
1064 END;
1065
1066 hix=.;
1067 if &Model = 1 then hix = hi * exp(&COEF*XDose); else
1068 if &Model = 2 then hix = hi * (1 + &COEF*XDose); else
1069 if &Model = 3 then hix = hi + &COEF*XDose; else

```



```

1068         if &Model = 4 then hix = hi * (1 + XDose)**&COEF; else
1069         if &Model = 0 then DO;
1070             hix = -99999; /* Code for user-defined model goes here. */
1071         END;
1072
1073         hstarix = hstari          /* hi=backgrd rate is included in hstari
1074     */
1075             + (hix - hi);      /* so that adding in the excess
1076     */
1077                                 /* from exposure (hix-hi) gives the
1078     */
1079                                 /* total rate of the exposed.
1080     */
1081
1082         qix = exp(-hstarix);
1083         Rx = Rx + ( hix/hstari * S_lix * ( 1-qix ) );
1084         S_lix = S_lix * qix;
1085
1086                                 qi = exp(-hstari);
1087         R0 = R0 + ( hi/hstari * S_li * (1-qi) );
1088         S_li = S_li * qi;
1089
1090         output;
1091
1092     END;
1093     Ex_Risk = Rx - R0; * Rx = risk in exposed population;
1094     RskRatio = Rx / R0; * R0 = from cancer;
1095     Extra_risk = Ex_Risk/(1-R0);
1096
1097     /* BT 3/20/19 added:*/
1098     call symput('Extra_Riskm',Extra_Risk);
1099
1100     /*BT 4/24/19 replaced the next line
1101     Diff_Ex_Risk = abs(&ex_risk_target-Ex_Risk); */
1102     Diff_Ex_Risk = abs(&ex_risk_target-Extra_Risk);
1103     call symput('Delta_Ex_Risk',Diff_Ex_Risk);
1104
1105     output;
1106
1107     * END; * corresponds to X_Levels;
1108 STOP;
1109
1110 run;
1111
1112 %Mend BEIR4;
1113
1114
1115
1116 /* -----
1117     BT: March 2019: parameters for the convergence that are used
1118     in the modified version of the BEIR4 macro.
1119     -----*/
1120
1121 %macro Converge_BEIR4 (init_exposure_conc=, ex_risk_target=, conv_criterion=, max_iteration=);
1122
1123     %Let Delta_Ex_Risk = 1; * initial high value to make sure loop is run at least once
1124                             (i.e., macro BEIR4 is called
1125 at least once);
1126
1127     /* BT 4/15/19: added next line to avoid error during compiling of BEIR4*/
1128     %Let Extra_Riskm = 1;
1129
1130     %Let i=1; * first time through loop;
1131
1132     %Do %Until (%sysevalf(&Delta_Ex_risk < &conv_criterion) OR %sysevalf(&i >
1133 &max_iteration));
1134
1135     * first time through loop, set expososure_conc=init_exposure_conc;
1136     %If &i=1 %Then
1137         %Do;
1138         %Let exposure_conc=&init_exposure_conc;

```

```

1139
1140             %End;
1141             %If &i>1 %Then
1142                 %Do;
1143
1144                 data tempBEIRCONVERGE;
1145
1146                 exposure_conc             *BEIR4 has run at least once. Adjust
1147
1148                 BEIR4 (=Extra_Risk);             Extra_Riskm is created in
1149
1150                 NumLoops=&i;
1151                 thisExposureConc=&exposure_conc;
1152
1153                 /* BT 4/15/19: replaced all of the convergence code with
1154                 the same code that we used
1155
1156                 in the meso code.*/
1157
1158                 numvar=&ex_risk_target;
1159                 denvar=&Extra_Riskm;
1160
1161                 thisexposureconc = thisexposureconc * (numvar/denvar);
1162
1163                 *update the concentration;
1164                 call symput('exposure_conc',thisexposureconc);
1165
1166                 output;
1167
1168                 Run;
1169
1170                 %End; *Corresponds to If i>1 statement;
1171
1172                 %BEIR4;
1173
1174                 %Let i=%eval(&i+1);
1175
1176                 %End;
1177
1178                 %Let EC_1Percent = &exposure_conc;
1179
1180                 /*-----+
1181                 | Report results if convergence criterion met:
1182                 +-----*/
1183
1184                 %If %sysevalf(&Delta_Ex_risk < &conv_criterion) %then %do;
1185                 title5 "based on KL = &COEF, Concentration = &EC_1Percent, and LastAge = &LastAge";
1186
1187                 data _null_;             /* Modified 26-july-00 */
1188                 pointer=1;
1189                 set allcause (keep=age
1190                             rename=(age=ageall0)) point=pointer nobs=n_all;
1191                 set cause (keep=age
1192                           rename=(age=ageCs0)) point=pointer nobs=n_cause;
1193                 pointer=n_all;
1194                 set allcause (keep=age
1195                             rename=(age=ageall1)) point=pointer nobs=n_all;
1196                 pointer=n_cause;
1197                 set cause (keep=age
1198                           rename=(age=ageCs1)) point=pointer nobs=n_cause;
1199
1200                 Tmp = sum(min(AgeAll1, AgeCs1, (&Lastage-1)), 1);
1201                 file PRINT;
1202
1203                 if ageall0 NE ageCs0 then DO;
1204                 put /"ERROR: The initial age for all-causes rate differs from the"
1205                     /"          initial age for the cause-specific rate.";
1206                 END;
1207                 else DO;
1208                 put / "Values of macro variables used in this computation:          "
1209                     // @3 "Value"          @17 "Macro_Var" @29 "Description"
1210                     // @3 "-----"          @17 "-----" @29 "-----"
1211                     // @3 "&Model"          @17 "MODEL" @29 "1 = Loglinear Relative Rate,"

```

```

1210 / @29 "2 = Linear Relative Rate, "
1211 / @29 "3 = Linear Absolute Rate, "
1212 / @29 "4 = 'Power' Relative Rate, "
1213 / @29 "0 = User defined. "
1214 / @3 "&Coef" @17 "COEF" @29 "Exposure parameter estimate"
1215 // @3 "&Lag" @17 "LAG" @29 "Exposure Lag "
1216 // @3 "&Agelst_x" @17 "AGE1ST_X" @29 "Age exposure begins"
1217 / @3 "&Duration" @17 "DURATION" @29 "Duration of exposure"
1218 / @3 "&EnvAdj" @17 "ENVADJ" @29 "Adjust dose from intermittent"
1219 / @29 "occupational exposures to "
1220 / @29 "continuous environmental exposures"
1221 / @3 "-----" @17 "-----" @29 "-----"
1222 "
1223 // "-----"
1224 // @3 "EC1% = " @10 "&EC_1Percent" @25 "(f/ml); Rx = " @39
1225 "&Extra_Riskm"
1226 // "-----"
1227 "-"
1228
1229 /"The risks are calculated from age " ageall0 " up to age " Tmp "."
1230 // ;
1231
1232 if ageall1 NE ageCsl then
1233 put /"WARNING: The last age for the all-causes rates differs from"
1234 /" the last age for the cause-specific rates, suggesting"
1235 /" the possibility that the rates weren't entered as desired."
1236 /;
1237 END;
1238 Stop;
1239 run;
1240 /* BT 7/5/19: Start of code that was added to merge variables for unexposed risk
1241 (S_li and S_lix) to the rest of the output, by age;
1242 */
1243
1244 Data newSRCDData(keep=SRC_age SRC_S_li SRC_S_lix);
1245 set ex_Risk;
1246 SRC_age=0; SRC_S_li=1; SRC_S_lix=1;
1247 output;
1248
1249 do obsnum=1 to last-1;
1250 set ex_Risk point=obsnum nobs=last;
1251 if _error_ then abort;
1252 SRC_age=age+1; SRC_S_li=S_li; SRC_S_lix=S_lix;
1253 output;
1254 end;
1255
1256 stop;
1257 run;
1258
1259 * rename variables to enable overwriting the values of S_li and S_lix in ex_risk with the values
1260 in newSRCDData;
1261 * Data file tempSRCDData has age=0-85 while the ex_Risk file has age 0-84, with last two
1262 records
1263 both having age=84.;
1264 Data tempSRCDData; Set newSRCDData(rename=(SRC_Age=age SRC_S_li=S_li SRC_S_lix=S_lix));
1265 if age=&LastAge then age=%sysevalf(&Lastage-1); Else age=age;
1266 Run;
1267
1268 * there are duplicate values for age in both ex_risk and tempSRCDData
1269 which may produce too many records. if that happens, then we use two set
1270 statements;
1271 Data ex_risk; merge ex_risk tempSRCDData; By Age; Run;
1272
1273 /* BT 7/5/19: End of code that was added to merge variables for unexposed risk
1274 (S_li and S_lix) to the rest of the output, by age;
1275 */
1276
1277 *BT 7/3/19: made the these changes to the following Proc Print procedure:
1278 - commented out the label option and added the split, uniform and
1279 width= options
1280 - included all variables to the format statement;

```

```

1281 proc print data=ex_risk /*label*/ noobs split='/' width=FULL;
1282   format age F4. Xdose E11. hi E11. hstari E11. hix E11. hstarix E11. qi E11. qix E11.
1283           S_li E11. S_lix E11. R0 E11. Risk E11. Ex_Risk E11. ;
1284   label Age = "Age at start of year (i)"
1285          XDose = "CE10(adj) (f\cc-yrs)"
1286
1287   R0 = "Cumulative Risk of lung cancer (unexposed) (R0)"
1288   Risk = "Cumulative risk of lung cancer (exposed) (Re)"
1289   Ex_Risk = "Excess risk/[Rx-Ro]/ /(Ex_Risk)"
1290   hi = "Lung Cancer hazard (unexposed) (hi)"
1291   hix = "Lung Cancer hazard (exposed) (hei)"
1292   hstari = "All cause hazard (unexposed) (h*i)"
1293   hstarix = "All cause hazard (exposed) (he*i)"
1294   qi = "Probability of surviving year i assuming alive at start
1295 (unexposed) (qi)"
1296   qix = "Probability of surviving year i assuming alive at start
1297 (exposed) (qei)"
1298   S_li = "Probability of surviving to end of year i (unexposed) (S1,i)"
1299   S_lix = "Probability of surviving to end of year i (exposed) (S1e,i)";
1300
1301   Var Age Xdose hi hstari hix hstarix qi qix S_li S_lix R0 Risk Extra_risk; *BT
1302 7/3/19: Var statement added;
1303   label Extra_risk="Extra Risk (Re - R0)\(1 - R0)";
1304 run;
1305
1306 %End; *end of the If statement that tests if convergence was met;
1307
1308 %Mend Converge_BEIR4;
1309
1310 /* -----+
1311 | March 2019: BT (SRC) Added macro CONVERGE_BEIR4 which iteratively |
1312 | runs macro BEIR4 until the EXPOSURE_CONCENTRATION corresponds to an |
1313 | extra_risk=0.01 (the point of departure [POD]). |
1314 | |
1315 | |
1316 | |
1317 | In addition to the parameter for CONVERGE_BEIR4, the user should also|
1318 | review parameters and data that are assigned/entered in Part I and |
1319 | Part II (see above). Parameters for CONVERGE_BEIR4 are defined below |
1320 +-----*/
1321
1322   *%BEIR4; * originally called macr BEIR4 directly. Now BEIR4 is called by Converge_BEIR4;
1323
1324   %Converge_BEIR4(init_exposure_conc=1, /* initial exposure concentration (initial
1325 guess) */
1326                   ex_risk_target=0.01000000, /* the point of departure
1327 (POD) - the target extra risk */
1328                   conv_criterion=0.00000001,
1329                   max_iteration=200); /* to avoid excessively long
1330 run times */
1331
1332
1333 Run;
1334 SAS Lung Cancer Lifetable for Non-Linear Models
1335 OPTIONS NODATE NONUMBER orientation=landscape linesize=max; *BT added 7/3/19;
1336
1337 /*
1338 This program calculates the risk of lung cancer from inhalation exposure to asbestos,
1339 using a lifetable approach based on BEIR IV. The basic exposure-response model is RR = exp(beta
1340 * CE10).
1341
1342 The basic code for the lifetable calculations were developed and provided to EPA
1343 by Randall Smith at NIOSH. The code from NIOSH calculates the baseline risk (R0) and the exposed
1344 risk (Rx)
1345 from exposure to an exposure concentration of X_Level using NIOSH Model 1: Rx = R0 * exp(COEF *
1346 X_Level).
1347
1348 EPA has modified the NIOSH as follows:
1349 1) The all-cause mortality and cause-specific (lung cancer) incidence data tables have been
1350 updated based on CDC Wonder 2017.

```

```

1351 2) An equation has been added to calculate extra risk: Extra_Risk = (Rx - R0) / ( 1 - R0)
1352 3) A macro has been added to find the exposure level (X_Level) that yields an extra risk of 0.01
1353 (1%).
1354 This is referred to as EC1%, which may then be used to calculate the unit risk: UR = 0.01 /
1355 EC1%
1356
1357 */
1358
1359 /* .\Beta Version.sas 19jan00, 26jul00, 25oct01, 06dec05, 30nov18
1360 -----
1361 Experimental version
1362 ----- */
1363 title "Lifetable calculation of lung cancer risk";
1364 title2 "under a non-linear relative rate model";
1365
1366 /*-----+
1367 | Compute excess risk by the BEIR IV method using SAS datasteps. |
1368 | |
1369 | These programs compute the risk of a cause-specific |
1370 | death in the presence of competing risks, where the cause- |
1371 | specific death-rate is modeled either as a relative rate |
1372 | [h=h0*f(Coef*X)] or as an absolute rate [h=h0+f(Coef*X)] |
1373 | where |
1374 | h denotes the cause-specific death-rate, |
1375 | X denotes cumulative occupational exposure (with Lag) |
1376 | Coef denotes the coefficient for the effect of exposure and |
1377 | h0 is the corresponding rate at baseline (X=0). |
1378 | (Except for Coef, these are functions of age.) |
1379 | |
1380 | A few simple models of f(Coef*X) are easily specified as |
1381 | described below. More complicated models can be specified with |
1382 | a little more work. (For a more complicated example, |
1383 | see \_GENERAL.LIB\PROGRAMS\SAS\BEIR-4.Method\BEIR4ex2.SAS). |
1384 | |
1385 |+Reference: |
1386 | Health Risks of Radon and Other Internally Deposited Alpha- |
1387 | Emitters (BEIR IV). Committee on the Biologic Effects of |
1388 | Ionizing Radiations. National Academy Press. Wash. DC (1988). |
1389 | See especially pages 131-136. |
1390 | |
1391 |+USER-SUPPLIED ASSIGNMENTS: |
1392 | |
1393 |+ The following macro variables are assigned using "%LET" state- |
1394 | ments: MODEL, COEF, LAG, AGE1ST_X, DURATION, LASTAGE. |
1395 | Further information appears below. |
1396 |+ Exposure concentrations for computing risk are defined |
1397 | in the dataset "X_LEVELS." |
1398 |+ All-cause mortality information is entered as a life-table in |
1399 | the data step "ALLCAUSE," and converted to rates per individual. |
1400 |+ Cause-specific mortality information for unexposed referents is |
1401 | entered as rates per 100,000 and converted to rates per |
1402 | individual in the data step "CAUSE." |
1403 | |
1404 |+NOTES: |
1405 | |
1406 |+ Datastep "EX_RISK" is where the desired risks are computed. |
1407 | |
1408 |+ If the unexposed(referent) cause-specific mortality rate is from |
1409 | a model then dataset "CAUSE" with variables AGE and RATE as |
1410 | modeled can be modified to incorporate this. However, care |
1411 | must be taken in calculating confidence limits since imprecision |
1412 | in the estimates of all of the parameters of the model |
1413 | contributes to the imprecision of excess risk estimates. |
1414 | |
1415 |+ This program is currently set up to apply the Linear Rel. Rate |
1416 | model (Lag= 0) and accumulation of excess risk is over the |
1417 | rates in ALLCAUSE and CAUSE unless truncated at a younger age. |
1418 | (See LASTAGE below.) |
1419 | |
1420 | |
1421 | |

```

```

1422 + SAS Programmer: Randall Smith +
1423 | The Nat'l Inst. for Occupational Safety & Health |
1424 | 26jul2000, 23jul2001, 25oct2001, 18nov2018 |
1425 + Modifications: +
1426 +
1427 | 26jul00 Fix the procedure bug causing it to report incorrectly |
1428 | the age at which accumulation of risk was stopped |
1429 | whenever the age-specific rates included ages |
1430 | before the value of &Agelst_X. (&Agelst_X is a macro |
1431 | expression defining the age exposure begins.) |
1432 |
1433 | 23jul01 Make changes to facilitate multiple applications of |
1434 | BEIR4 algorithm, i.e., MLE(Excess Risk), UCL(ExcessRisk), |
1435 | searching for concentrations for a fixed risk. These |
1436 | changes involve defining Macros named BEIR4 and SEARCH |
1437 | given below with code illustrating these uses for the |
1438 | linear relative rate model. |
1439 |
1440 | 25oct01 Modified to add Macro variable EnvAdj for whether to |
1441 | increase inhaled dose from intermittent occupational |
1442 | exposures to continuous environmental exposures |
1443 | and update US rates for Gibb et al. cohort. |
1444 |
1445 | 30nov18 A bug that prevented the calculation of excess risks |
1446 | after incorporating an adjustment from intermittent |
1447 | occupational exposures to continuous exposures is fixed. |
1448 |
1449 | +---|
1450 | March 2019: BT (SRC) Added macro CONVERGE_BEIR4 which iteratively |
1451 | runs macro BEIR4 until the EXPOSURE_CONCENTRATION corresponds to an |
1452 | extra_risk=0.01 (the point of departure [POD]). |
1453 |
1454 |
1455 | Macro CONVERGE_BEIR4 works with one value for the exposure |
1456 | variable XLevel (i.e., when the data C_Levels includes one record.) |
1457 |
1458 |
1459 | The intent was to make as few changes to BEIR4 as possible. The data |
1460 | X_LEVELS and variable XLevel are retained but the initial value of |
1461 | XLevel is provided in the call to macro CONVERGE_BEIR4 (the value |
1462 | of Xlevel in the cards statement is not used in the calculations. |
1463 | Changes to the BEIR4 macro are in Part III and Part IV, and are |
1464 | indicated by the letters BT. |
1465 |
1466 |
1467 |
1468 | In addition to the parameter values that are specified by the user |
1469 | in PART 1, and the user-provided data entered in Part II, parameters |
1470 | for the new macro CONVERGE_BEIR4 are specified in the call to the |
1471 | macro CONVERGE_BEIR4 (see end of this SAS program file below). |
1472 |-----*/
1473
1474
1475 /* PART I. USER-SUPPLIED ASSIGNMENTS (Macro variables):
1476 |-----+
1477 | Model of cumulative exposure effects: |
1478 | 1 => Loglinear Relative rate |
1479 | R=R0*exp(COEF*X) |
1480 | 2 => Linear Relative rate, |
1481 | R=R0*(1+COEF*X) |
1482 | 3 => Absolute rate, |
1483 | R=R0+COEF*X |
1484 | 4 => Power relative rate |
1485 | R=R0*(1+X)^COEF |
1486 | 0 => User Defined & programmed |
1487 | in datastep Ex_Risk below |
1488 | */ %Let Model = 1;
1489 /*
1490 | Cumulative exposure parameter: */ %Let COEF = 1e-2;
1491 /*
1492 | Lag or delay between exposure and effect: */ %Let Lag = 10;

```

```

1493 /* |
1494 | Age exposure begins: */ %Let Agelst_x = 40;
1495 /* Exposure duration (years): */ %Let Duration = 20;
1496 /* Adjust dose from occupational to |
1497 | continuous environmental exposures (Y/N)? */ %Let EnvAdj = Yes;
1498 /* Age to stop accumulating excess risk |
1499 | (supposing rates are available for |
1500 | ages >= &LastAge); otherwise use all of |
1501 | the supplied rate information: */ %Let LastAge = 85;
1502 /*-----*/
1503
1504
1505 /* PART II. USER-SUPPLIED ASSIGNMENTS (Datesets AllCause, Cause, X_Levels ): */
1506
1507
1508 data AllCause (label="Unexposeds' age-spec mortality rates (all)"
1509 drop=Lx rename=(BLx=Lx) );
1510
1511 /*-----+
1512 | Input lifetable and calculate the corresponding age-specific |
1513 | (all-causes) mortality rate (AllCause) and conditional survival |
1514 | probability for each year of age (qi) together with |
1515 | the corresponding values of age (Age). |
1516 +-----*/
1517
1518 Label Age = "Age at start of year (Age=i)"
1519 BLx = "Number alive at start of year"
1520 Lx = "Number alive at end of year"
1521 CndPrDth = "Pr[Death before age i+1 | alive at age i]"
1522 qi = "Pr[Survive to age i+1 | Alive at age i]"
1523 AllCause = "Age-spec mortality rate (all causes)";
1524
1525 if _n_=1 then input age //// @1 BLx @; /* //// => skip next 4 lines */
1526 input Lx @@;
1527 CndPrDth = (BLx - Lx)/BLx;
1528
1529 qi = 1-CndPrDth;
1530 if qi <= 0 then AllCause = 1e+50;
1531 else AllCause = - log(qi);
1532
1533 if age < &LastAge then output; else STOP;
1534 BLx=Lx;
1535 age+1;
1536 retain age BLx;
1537 cards;
1538 0 = Life-table starting age. (Required: Values must begin 4 lines down!)
1539 The following are 2017 Life-table values of US population
1540 starting at birth and ending at age 85.
1541 (Source: Nat.Vital Statistics Reports 2019 Vol 68 No 7, Table 1,
1542 https://www.cdc.gov/nchs/data/nvsr/nvsr68/nvsr68_07-508.pdf)
1543 100000 99422 99384 99360 99341 99326 99312 99299 99288 99278
1544 99268 99259 99249 99236 99217 99191 99158 99116 99066 99006
1545 98937 98858 98770 98674 98573 98466 98355 98241 98122 97999
1546 97872 97740 97603 97461 97314 97163 97006 96843 96674 96501
1547 96321 96135 95939 95732 95511 95275 95023 94753 94461 94144
1548 93797 93419 93008 92560 92070 91538 90963 90345 89684 88978
1549 88226 87424 86570 85664 84706 83696 82632 81507 80315 79048
1550 77697 76265 74715 73064 71296 69418 67402 65245 62933 60462
1551 57839 55053 52123 49035 45771 42382
1552 ;
1553
1554 *run;*BT 7/3/19 added Run statement here;
1555
1556 data CAUSE (label="Unexposeds' age-cause-spec mortality rates");
1557 /*-----+
1558 | Specify unexposeds' age-specific mortality rates (per year) |
1559 | from specific cause. |
1560 +-----*/
1561
1562 label Age = "Age"
1563 Rate_e5 = "Age,cause-specific rate per 100,000"
1564 Rate = "Age,cause-specific rate per individual";
1565
1566 if _n_ = 1 then input age /* input starting age */

```

```

1564                                     ///; /* /// => skip next 3 lines */
1565 input Rate_e5 @@;
1566
1567 Rate = Rate_e5 * 1e-5; /* Convert to rate per individual */
1568
1569 if age <= 4
1570 then DO; output; age+1; END;
1571 else DO i = 0,1,2,3,4; /*-----*/
1572 if age < &LastAge /* Fill out into yearly intervals from */
1573 then output; /* inputted five year intervals after age 4*/
1574 age+1; /*-----*/
1575 END;
1576 cards;
1577 0 = Start age of cause-specific rate (Required: Rates begin 3 lines down!)
1578 The following are 2017 cancer site code 22030 lung and bronchus incidence rates per
1579 100,000 for US pop'n starting at birth.
1580 For ages 5 and above, each rate holds for the age thru age+4 years.
1581 Source: CDC Wonder, https://wonder.cdc.gov/cancer-v2017.HTML
1582 0.205 0.100 0.100 0.100 0.100 0.039 0.039 0.104 0.299 0.553 1.267 2.600 6.534 16.528 44.403
1583 96.098 149.112 223.906 319.322 391.202 395.215
1584 ;
1585 *run; *BT 7/3/19 added Run statement here;
1586
1587 data X_LEVELS (label= "Exposure levels (e.g., concentrations)");
1588 /*-----+
1589 | Specify environmental exposure levels |
1590 | and update label for the variable, XLevel, if necessary: |
1591 +-----*/
1592 /*-----+
1593 | BT 3/8/19: Add macro CONVERGE_BEIR4 which iteratively runs macro |
1594 | BEIR4 until the EXPOSURE_CONCENTRATION corresponds to extra_risk=0.01|
1595 | |
1596 | |
1597 | |
1598 | The intent was to make as few changes to BEIR4 as possible. The data |
1599 | X_LEVELS and variable XLevel are retained but the initial value of |
1600 | XLevel is provided in the call to macro CONVERGE_BEIR4 (the value |
1601 | of Xlevel in the cards statement is not used in the calculations. |
1602 +-----*/
1603
1604 input XLevel @@;
1605 label XLevel= "Asbestos exposure (F/ml)";
1606 cards;
1607 0.0383
1608 ;
1609 run;
1610
1611 %Macro BEIR4;
1612 /* March 2019 - BT (SRC): Macro BEIR4 is now called by macro CONVERGE_BEIR4.
1613 */
1614 /* 23jul01 modification */
1615 /* Enclose the actual calculations and printed results in a macro */
1616 /* to facilitate multiple applications of the algorithm. */
1617
1618 /* PART III. Perform calculations: */
1619
1620 data EX_RISK (label = "Estimated excess risks [Method=BEIR IV]"
1621 /*keep = XLevel Rx ex_risk RskRatio R0 extra_Risk */
1622 rename= (Rx=Risk));
1623 /*-----+
1624 | Calculate risk and excess risk for each exposure concentration|
1625 | in work.X_Level by BEIR IV method using information in |
1626 | work.AllCause and work.Cause to define referent population: |
1627 +-----*/
1628 format rate F15.8 hi F15.8; *BT 7/3/19: added the format statement;
1629 length XLevel 8.;
1630 label Age = "Age at start of year (i)"
1631 XTime = "Exposure duration midway between i & i+1"
1632 XDose = "CE10(adj) (f/cc-yrs)"
1633
1634 R0 = "Cumulative Risk of lung cancer (unexposed) (R0)"

```



```

1635 Rx      = "Cumulative risk of lung cancer (exposed) (Re)"
1636 Ex_Risk = "Excess risk (Rx-Ro)"
1637 RskRatio = "Ratio of risks (Rx/Ro)"
1638
1639 hi      = "Lung Cancer hazard (unexposed) (hi)"
1640 hix     = "Lung Cancer hazard (exposed) (hei)"
1641 hstari  = "All cause hazard (unexposed) (h*i)"
1642 hstarix = "All cause hazard (exposed) (he*i)"
1643 qi      = "Probability of surviving year i assuming alive at start (unexposed)
1644 (qi)"
1645 S_li    = "Probability of surviving to end of year i (unexposed) (S1,i)"
1646 S_lix   = "Probability of surviving to end of year i (exposed) (S1,i)";
1647
1648 /* BT 3/8/19: Calculation of unexposed's risk (following DO LOOP) could be omitted
1649 from the iteration
1650 but may require further changes to BEIR4(?).
1651 *e.g., %if i=1 %then %do;*/
1652
1653 if _n_=1 then DO;
1654 /* Calculate unexposed's risk (R0) to be retained */
1655 /* based on equation 2A-21 (pg. 131) of BEIR IV: */
1656
1657 /* Initialize: */ S_li = 1; R0 = 0;
1658
1659 DO pointer = 1 to min(n_all,n_cause) until (age>=&LastAge-1);
1660 set allcause (keep=age AllCause rename=(AllCause=hstari))
1661 point=pointer nobs=n_all;
1662 set cause (keep=age Rate rename=(age=ageCause Rate=hi))
1663 point=pointer nobs=n_cause;
1664
1665 if Age NE AgeCause then
1666 put "*** WARNING: Age values in datasets ALLCAUSE and CAUSE don't
1667 conform ***"
1668 / @13 "Rates misaligned on age could give incorrect results"
1669 / @13 Pointer=
1670 +2 "Age(ALLCAUSE)=" Age +2 "Age(CAUSE)=" AgeCause /;
1671
1672 qi = exp(-hstari);
1673 R0 = R0 + ( hi/hstari * S_li * (1-qi) );
1674 S_li = S_li * qi;
1675 END;
1676 END; /* End of 'if _n_=1 then DO;' stmt */
1677
1678 retain R0;
1679
1680
1681
1682
1683 /* Calculate exposed's risk (Rx, renamed to Risk) for each exposure level
1684 */
1685 /* ultimately based on equation 2A-22 (pg. 132) of BEIR IV */
1686 /* but re-expressed in a form similar to equation 2A-21: */
1687
1688 * BT 3/20/19. This version of CONVERGE_BEIR4 will work when there
1689 is
1690 one concentration in data set x_levels -
1691 i.e., one value for xlevel.
1692 The Do loop for X_levels is commented out;
1693 *DO pointX = 1 to No_of_Xs;
1694 * set x_levels point=pointX nobs=No_of_Xs; /* BT 3/8/19: determines
1695 when to end the loop. Nobs is set at compilation,
1696
1697 so the value of nobs is available at first run through loop -
1698
1699 just one record and one variable (XLevel) in dataset x_levels. */
1700
1701 /* BT 3/20/19: added the next lint to set the exposure
1702 concentration = current value of &exposure_conc. */
1703 xlevel = &exposure_conc;
1704
1705

```

```

1706 /* Initialize : */ S_lix = 1; Rx = 0;S_li=1; R0=0;
1707
1708 DO pointer = 1 to min(n_all,n_cause) until (age>=&LastAge-1);
1709 set allcause (keep=age AllCause rename=(AllCause=hstari))
1710 point=pointer nob= n_all;
1711 set cause (keep=Rate rename=(Rate=hi))
1712 point=pointer nob= n_cause;
1713
1714 XTime = min( max(0, (age+0.5-&Agelst_x-&Lag))
1715 , &Duration );
1716
1717 if UpCase("&EnvAdj") = "YES" /* Occupational to Environmental
1718 Conversion */
1719 then XDose = XLevel
1720 * 365/240 /* Days per year */
1721 * 20/10 /* Ventilation (L) per day */
1722 * XTime;
1723 ELSE if UpCase("&EnvAdj") = "NO" /* 30nov2018 ('ELSE') */
1724 then XDose = XLevel*XTime;
1725 else DO; put //"Macro variable ENVADJ incorrectly specified."
1726 /*It should be either YES or NO. Value specified
1727 is: &ENVADJ"
1728 /;
1729 STOP;
1730 END;
1731 hix=.;
1732 if &Model = 1 then hix = hi * exp(&COEF*XDose); else
1733 if &Model = 2 then hix = hi * (1 + &COEF*XDose); else
1734 if &Model = 3 then hix = hi + &COEF*XDose; else
1735 if &Model = 4 then hix = hi * (1 + XDose)**&COEF; else
1736 if &Model = 0 then DO;
1737 hix = -99999; /* Code for user-defined model goes here. */
1738 END;
1739
1740 hstarix = hstari /* hi=backgrd rate is included in hstari
1741 */
1742 + (hix - hi); /* so that adding in the excess
1743 */
1744 /* from exposure (hix-hi) gives the
1745 */
1746 /* total rate of the exposed.
1747 */
1748 qix = exp(-hstarix);
1749 Rx = Rx + ( hix/hstarix * S_lix * ( 1-qix ) );
1750 S_lix = S_lix * qix;
1751
1752 qi = exp(-hstari);
1753 R0 = R0 + ( hi/hstari * S_li * (1-qi) );
1754 S_li = S_li * qi;
1755
1756
1757 output;
1758
1759 END;
1760 Ex_Risk = Rx - R0; * Rx = risk in exposed population;
1761 RskRatio = Rx / R0; * R0 = from cancer;
1762 Extra_risk = Ex_Risk/(1-R0);
1763
1764 /* BT 3/20/19 added:*/
1765 call symput('Extra_Riskm',Extra_Risk);
1766
1767 /*BT 4/24/19 replaced the next line
1768 Diff_Ex_Risk = abs(&ex_risk_target-Ex_Risk); */
1769 Diff_Ex_Risk = abs(&ex_risk_target-Extra_Risk);
1770 call symput('Delta_Ex_Risk',Diff_Ex_Risk);
1771
1772 output;
1773
1774 * END; * corresponds to X_Levels;
1775 STOP;
1776

```

```

1777         run;
1778
1779
1780
1781
1782 %Mend BEIR4;
1783
1784
1785
1786 /* -----
1787          BT: March 2019: parameters for the convergence that are used
1788          in the modified version of the BEIR4 macro.
1789 -----*/
1790
1791 %macro Converge_BEIR4 (init_exposure_conc=, ex_risk_target=, conv_criterion=, max_iteration=);
1792
1793     %Let Delta_Ex_Risk = 1; * initial high value to make sure loop is run at least once
1794                               (i.e., macro BEIR4 is called
1795 at least once);
1796
1797     /* BT 4/15/19: added next line to avoid error during compiling of BEIR4*/
1798     %Let Extra_Riskm = 1;
1799
1800     %Let i=1; * first time through loop;
1801
1802     %Do %Until (%sysevalf(&Delta_Ex_risk < &conv_criterion) OR %sysevalf(&i >
1803 &max_iteration));
1804
1805         * first time through loop, set exposure_conc=init_exposure_conc;
1806         %If &i=1 %Then
1807             %Do;
1808                 %Let exposure_conc=&init_exposure_conc;
1809
1810             %End;
1811         %If &i>1 %Then
1812             %Do;
1813
1814                 data tempBEIRCONVERGE;
1815
1816                     exposure_conc           *BEIR4 has run at least once. Adjust
1817
1818                     Extra_Riskm is created in
1819 BEIR4 (=Extra_Risk);
1820
1821                     NumLoops=&i;
1822                     thisExposureConc=&exposure_conc;
1823
1824                     /* BT 4/15/19: replaced all of the convergence code with
1825 the same code that we used
1826
1827                     in the meso code.*/
1828
1829                     numvar=&ex_risk_target;
1830                     denvar=&Extra_Riskm;
1831
1832                     thisexposureconc = thisexposureconc * (numvar/denvar);
1833
1834                     *update the concentration;
1835                     call symput('exposure_conc',thisexposureconc);
1836
1837                     output;
1838
1839                     Run;
1840
1841                 %End; *Corresponds to If i>1 statement;
1842
1843             %BEIR4;
1844
1845             %Let i=%eval(&i+1);
1846
1847         %End;
1848
1849 %Let EC_1Percent = &exposure_conc;

```

```

1848
1849
1850
1851
1852 /*-----+
1853 | Report results if convergence criterion met:
1854 +-----*/
1855
1856 %If %sysevalf(&Delta_Ex_risk < &conv_criterion) %then %do;
1857 title5 "based on beta=&COEF, Concentration=&EC_1Percent, and LastAge=&LastAge";
1858
1859 data _null_;          /* Modified 26-july-00 */
1860     pointer=1;
1861     set allcause (keep=age
1862                 rename=(age=ageall0)) point=pointer nobs=n_all;
1863     set cause    (keep=age
1864                 rename=(age=ageCs0)) point=pointer nobs=n_cause;
1865     pointer=n_all;
1866     set allcause (keep=age
1867                 rename=(age=ageall1)) point=pointer nobs=n_all;
1868     pointer=n_cause;
1869     set cause    (keep=age
1870                 rename=(age=ageCs1)) point=pointer nobs=n_cause;
1871
1872     Tmp = sum(min(AgeAll1, AgeCs1, (&Lastage-1)), 1);
1873     file PRINT;
1874
1875     if ageall0 NE ageCs0 then DO;
1876         put /"ERROR: The initial age for all-causes rate differs from the"
1877             /"         initial age for the cause-specific rate.";
1878     END;
1879     else DO;
1880         put / "Values of macro variables used in this computation:      "
1881             // @3 "Value"          @17 "Macro_Var" @29 "Description"
1882             // @3 "-----"        @17 "-----" @29 "-----"
1883             // @3 "&Model"         @17 "MODEL"      @29 "1 = Loglinear Relative Rate,"
1884             // @29 "2 = Linear Relative Rate,"
1885             // @29 "3 = Linear Absolute Rate,"
1886             // @29 "4 = 'Power' Relative Rate,"
1887             // @29 "0 = User defined."
1888             // @3 "&Coef"          @17 "COEF"        @29 "Exposure parameter estimate"
1889             // @3 "&Lag"           @17 "LAG"         @29 "Exposure Lag "
1890             // @3 "&Age1st_x"      @17 "AGE1ST_X"    @29 "Age exposure begins"
1891             // @3 "&Duration"      @17 "DURATION"    @29 "Duration of exposure"
1892             // @3 "&EnvAdj"        @17 "ENVADJ"      @29 "Adjust dose from intermittent"
1893             // @29 "occupational exposures to "
1894             // @29 "continuous environmental exposures"
1895             // @3 "-----"        @17 "-----" @29 "-----"
1896     "
1897         // "-----"
1898             // @3 "EC1% = " @10 "&EC_1Percent" @25 "(f/ml); Rx = " @39
1899 "&Extra_Riskm"
1900             // "-----"
1901     "-"
1902
1903         /"The risks are calculated from age " ageall0 " up to age " Tmp "."
1904         // ;
1905
1906     if ageall1 NE ageCs1 then
1907         put /"WARNING: The last age for the all-causes rates differs from"
1908             /"         the last age for the cause-specific rates, suggesting"
1909             /"         the possibility that the rates weren't entered as desired."
1910         // ;
1911     END;
1912     Stop;
1913     run;
1914 /* BT 7/5/19: Start of code that was added to merge variables for unexposed risk
1915                (S_li and S_lix) to the rest of the output, by age;
1916 */
1917
1918 Data newSRCData(keep=SRC_age SRC_S_li SRC_S_lix);

```

```

1919         set ex_Risk;
1920         SRC_age=0; SRC_S_li=1; SRC_S_lix=1;
1921         output;
1922
1923         do obsnum=1 to last-1;
1924         set ex_Risk point=obsnum nobs=last;
1925         if _error_ then abort;
1926             SRC_age=age+1; SRC_S_li=S_li; SRC_S_lix=S_lix;
1927         output;
1928         end;
1929
1930     stop;
1931 run;
1932
1933 * rename variables to enable overwriting the values of S_li and S_lix in ex_risk with the values
1934 in newSRCData;
1935 * Data file tempSRCData has age=0-85 while the ex_Risk file has age 0-84, with last two
1936 records
1937     both having age=84.;
1938 Data tempSRCData; Set newSRCData(rename=(SRC_Age=age SRC_S_li=S_li SRC_S_lix=S_lix));
1939     if age=&LastAge then age=%sysvalf(&Lastage-1); Else age=age;
1940 Run;
1941
1942 * there are duplicate values for age in both ex_risk and tempSRCData
1943     which may produce too many records. if that happens, then we use two set
1944 statements;
1945 Data ex_risk; merge ex_risk tempSRCData; By Age; Run;
1946
1947 /* BT 7/5/19: End of code that was added to merge variables for unexposed risk
1948     (S_li and S_lix) to the rest of the output, by age;
1949 */
1950
1951 *BT 7/3/19: made the these changes to the following Proc Print procedure:
1952     - commented out the label option and added the split, uniform and
1953 width= options
1954     - included all variables to the format statement;
1955 proc print data=ex_risk /*label*/ noobs split='/' width=FULL;
1956     format age F4. Xdose E11. hi E11. hstari E11. hix E11. hstarix E11. qi E11. qix E11.
1957         S_li E11. S_lix E11. R0 E11. Risk E11. Ex_Risk E11. ;
1958         label Age = "Age at start of year (i)"
1959             XDose = "CE10(adj) (f\cc-yrs)"
1960
1961             R0 = "Cumulative Risk of lung cancer (unexposed) (R0)"
1962             Risk = "Cumulative risk of lung cancer (exposed) (Re)"
1963             Ex_Risk = "Excess risk/[Rx-Ro]/ /(Ex_Risk)"
1964             hi = "Lung Cancer hazard (unexposed) (hi)"
1965             hix = "Lung Cancer hazard (exposed) (hei)"
1966             hstari = "All cause hazard (unexposed) (h*i)"
1967             hstarix = "All cause hazard (exposed) (he*i)"
1968             qi = "Probability of surviving year i assuming alive at start
1969 (unexposed) (qi)"
1970             qix = "Probability of surviving year i assuming alive at start
1971 (exposed) (qei)"
1972             S_li = "Probability of surviving to end of year i (unexposed) (S1,i)"
1973             S_lix = "Probability of surviving to end of yeari (exposed) (S1,i)";
1974
1975     Var Age Xdose hi hstari hix hstarix qi qix S_li S_lix R0 Risk Extra_risk; *BT
1976 7/3/19: Var statement added;
1977     label Extra_risk="Extra Risk (Re - R0)\(1 - R0)";
1978 run;
1979
1980 %End; *end of the If statement that tests if convergence was met;
1981
1982 %Mend Converge_BEIR4;
1983
1984 /* -----+
1985 | March 2019: BT (SRC) Added maxro CONVERGE_BEIR4 which iteratively |
1986 | runs macro BEIR4 until the EXPOSURE_CONCENTRATION corresponds to an |
1987 | extra_risk=0.01 (the point of departure [POD]). |

```

```

1988 |
1989 |
1990 |
1991 | In addition to the parameter for CONVERGE_BEIR4, the user should also|
1992 | review parameters and data that are assigned/entered in Part I and |
1993 | Part II (see above). Parameters for CONVERGE_BEIR4 are defined below |
1994 |-----*/
1995
1996     *%BEIR4; * originally called macr BEIR4 directly. Now BEIR4 is called by Converge_BEIR4;
1997
1998     %Converge_BEIR4(init_exposure_conc=1,          /* initial exposure concentration (initial
1999 guess) */
2000                                     ex_risk_target=0.01000000,    /* the point of departure
2001 (POD) - the target extra risk */
2002                                     conv_criterion=0.00000001,
2003                                     max_iteration=200);          /* to avoid excessively long
2004 run times */
2005
2006 Run;

```

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2076

SAS Mesothelioma Lifetable

```
OPTIONS NODATE NONUMBER orientation=landscape papersize=legal;

/*
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 "Lifetable calculation of mesothelioma risk";
title2 "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 datastep "X_LEVELS." |
| + |
+-----*/
```

```

2077 |> All-cause mortality information is entered as a life-table in |
2078 | the data step "ALLCAUSE," and converted to rates per individual. |
2079 | Cause-specific mortality information for unexposed referents is |
2080 | entered as rates per 100,000 and converted to rates per |
2081 | individual in the data step "CAUSE." |
2082 | |
2083 | |
2084 +NOTES: |
2085 |> Daststep "EX_RISK" is where the desired risks are computed. |
2086 | |
2087 |> If the unexposed(referent) cause-specific mortality rate is from |
2088 | a model then daststep "CAUSE" with variables AGE and RATE as |
2089 | modeled can be modified to incorporate this. However, care |
2090 | must be taken in calculating confidence limits since imprecision |
2091 | in the estimates of all of the parameters of the model |
2092 | contributes to the imprecision of excess risk estimates. |
2093 | |
2094 |> This program is currently set up to apply the Linear Rel. Rate |
2095 | model (Lag= 0) and accumulation of excess risk is over the |
2096 | rates in ALLCAUSE and CAUSE unless truncated at a younger age. |
2097 | (See LASTAGE below.) |
2098 | |
2099 | |
2100 + SAS Programmer: Randall Smith |
2101 | The Nat'l Inst. for Occupational Safety & Health |
2102 | 26jul2000, 23jul2001, 25oct2001, 18nov2018 |
2103 + Modifications: |
2104 + |
2105 | 26jul00 Fix the procedure bug causing it to report incorrectly |
2106 | the age at which accumulation of risk was stopped |
2107 | whenever the age-specific rates included ages |
2108 | before the value of &Agelst_X. (&Agelst_X is a macro |
2109 | expression defining the age exposure begins.) |
2110 | |
2111 | 23jul01 Make changes to facilitate multiple applications of |
2112 | BEIR4 algorithm, i.e., MLE(Excess Risk), UCL(ExcessRisk), |
2113 | searching for concentrations for a fixed risk. These |
2114 | changes involve defining Macros named BEIR4 and SEARCH |
2115 | given below with code illustrating these uses for the |
2116 | linear relative rate model. |
2117 | |
2118 | 25oct01 Modified to add Macro variable EnvAdj for whether to |
2119 | increase inhaled dose from intermittent occupational |
2120 | exposures to continuous environmental exposures |
2121 | and update US rates for Gibb et al. cohort. |
2122 | |
2123 | 30nov18 A bug that prevented the calculation of excess risks |
2124 | after incorporating an adjustment from intermittent |
2125 | occupational exposures to continuous exposures is fixed. |
2126 | |
2127 | +---|
2128 | April 2019: BT (SRC) Added maxro CONVERGE_BEIR4 which iteratively |
2129 | runs macro BEIR4 until the EXPOSURE_CONCENTRATION corresponds to an |
2130 | extra_risk=0.01 (the point of departure [POD]). |
2131 | |
2132 | |
2133 | Macro CONVERGE_BEIR4 works with one value for the exposure |
2134 | variable XLevel (i.e., when the data C_Levels includes one record.) |
2135 | |
2136 | |
2137 | The intent was to make as few changes to BEIR4 as possible. The data |
2138 | X_LEVELS and variable XLevel are retained but the initial value of |
2139 | XLevel is provided in the call to macro CONVERGE_BEIR4 (the value |
2140 | of Xlevel in the cards statement is not used in the calculations. |
2141 | Changes to the BEIR4 macro are in Part III and Part IV, and are |
2142 | indicated by the letters BT. |
2143 | |
2144 | |
2145 | |
2146 | In addition to the parameter values that are specified by the user |
2147 | in PART 1, and the user-provided data entered in Part II, parameters |

```



```

2148 | for the new macro CONVERGE_BEIR4 are specified in the call to the |
2149 | macro CONVERGE_BEIR4 (see end of this SAS program file below). |
2150 +-----*/
2151
2152
2153
2154
2155 /* PART I. USER-SUPPLIED ASSIGNMENTS (Macro variables):
2156 /*-----+
2157 | Model of cumulative exposure effects: |
2158 | 1 => Loglinear Relative rate |
2159 | R=R0*exp(COEF*X) |
2160 | 2 => Linear Relative rate, |
2161 | R=R0*(1+COEF*X) |
2162 | 3 => Absolute rate, |
2163 | R=R0+COEF*X |
2164 | 4 => Power relative rate |
2165 | R=R0*(1+X)^COEF |
2166 | 0 => User Defined & programmed |
2167 | in dataset Ex_Risk below |
2168 | | */ %Let Model = 3;
2169 /* |
2170 | Cumulative exposure parameter: */ %Let COEF = 2.961e-9;
2171
2172 /* |
2173 | Lag or delay between exposure and effect: */ %Let Lag = 10; /* Lag is built into Q, so
2174 this value is ignored */
2175 /* |
2176 | Age exposure begins: */ %Let Age1st_x = 20;
2177 /* Exposure duration (years): */ %Let Duration = 20;
2178 /* Adjust dose from occupational to
2179 | continuous environmental exposures (Y/N)? */ %Let EnvAdj = Yes;
2180 /* Age to stop accumulating excess risk
2181 | (supposing rates are available for
2182 | ages >= &LastAge); otherwise use all of
2183 | the supplied rate information: */ %Let LastAge =85;
2184 /*-----*/
2185
2186
2187 /* PART II. USER-SUPPLIED ASSIGNMENTS (Datesets AllCause, Cause, X_Levels ): */
2188
2189
2190 data AllCause (label="Unxposeds' age-spec mortality rates (all)"
2191 drop=Lx rename=(BLx=Lx) );
2192 /*-----+
2193 | Input lifetable and calculate the corresponding age-specific |
2194 | (all-causes) mortality rate (AllCause) and conditional survival |
2195 | probability for each year of age (qi) together with |
2196 | the corresponding values of age (Age). |
2197 +-----*/
2198 Label Age = "Age at start of year (Age=i)"
2199 BLx = "Number alive at start of year"
2200 Lx = "Number alive at end of year"
2201 CndPrDth = "Pr[Death before age i+1 | alive at age i]"
2202 qi = "Pr[Survive to age i+1 | Alive at age i]"
2203 AllCause = "Age-spec mortality rate (all causes)";
2204
2205 if _n_=1 then input age /// @1 BLx @;
2206 input Lx @@;
2207 CndPrDth = (BLx - Lx)/BLx;
2208
2209 qi = 1-CndPrDth;
2210 if qi <= 0 then AllCause = 1e+50;
2211 else AllCause = - log(qi);
2212
2213 if age < &LastAge then output; else STOP;
2214 BLx=Lx;
2215 age+1;
2216 retain age BLx;
2217 cards;
2218 0 = Life-table starting age. (Required: Values must begin 4 lines down!)

```

```

2219         The following are 2017 Life-table values of US population
2220         starting at birth and ending at age 85.
2221         (Source: Nat.Vital Statistics Reports 2019 Vol 68 No 7, Table 1,
2222         https://www.cdc.gov/nchs/data/nvsr/nvsr68/nvsr68_07-508.pdf)
2223         100000 99422 99384 99360 99341 99326 99312 99299 99288 99278
2224         99268 99259 99249 99236 99217 99191 99158 99116 99066 99006
2225         98937 98858 98770 98674 98573 98466 98355 98241 98122 97999
2226         97872 97740 97603 97461 97314 97163 97006 96843 96674 96501
2227         96321 96135 95939 95732 95511 95275 95023 94753 94461 94144
2228         93797 93419 93008 92560 92070 91538 90963 90345 89684 88978
2229         88226 87424 86570 85664 84706 83696 82632 81507 80315 79048
2230         77697 76265 74715 73064 71296 69418 67402 65245 62933 60462
2231         57839 55053 52123 49035 45771 42382
2232     ;
2233
2234
2235     data CAUSE (label="Unexposeds' age-cause-spec mortality rates");
2236     /*-----+
2237     | Specify unexposeds' age-specific mortality rates (per year) |
2238     | from specific cause.                                     |
2239     +-----*/
2240
2241         label Age = "Age"
2242         Rate_e5 = "Age,cause-specific rate per 100,000"
2243         Rate = "Age,cause-specific rate per individual";
2244
2245     if _n_ = 1 then input age /* input starting age */
2246                     ///; /* // => skip next 3 lines */
2247     input Rate_e5 @@;
2248
2249     Rate = Rate_e5 * 1e-5; /* Convert to rate per individual */
2250
2251     if age <= 4
2252     then DO; output; age+1; END;
2253     else DO i = 0,1,2,3,4; /*-----*/
2254         if age < &LastAge /* Fill out into yearly intervals from */
2255             then output; /* inputted five year intervals after age 4*/
2256             age+1; /*-----*/
2257     END;
2258     cards;
2259     0 = Start age of cause-specific rate (Required: Rates begin 3 lines down!)
2260     The following are 2013 ICD10 = 113 death rates per 100,000 for US pop'n starting at
2261     birth.
2262     For ages 5 and above, each rate holds for the age thru age+4 years.
2263     Source: CDC Wonder
2264     0.0 0.0 0.0 0.0 0.0
2265     0.0 0.0 0.0 0.0 0.0 0.0 0.0 0.0 0.0 0.0 0.0
2266     0.0 0.0 0.0 0.0 0.0
2267
2268     ;
2269
2270     run;
2271
2272     data X_LEVELS (label= "Exposure levels (e.g., concentrations)");
2273     /*-----+
2274     | Specify environmental exposure levels |
2275     | and update label for the variable, XLevel, if necessary: |
2276     +-----*/
2277
2278     input XLevel @@;
2279     label XLevel= "Asbestos exposure (F/ml)";
2280     cards;
2281     0.001
2282     ;
2283
2284     %Macro BEIR4;
2285     /* April 2 2019 - BT (SRC): Macro BEIR4 is now called by macro CONVERGE_BEIR4.*/
2286     /* 23jul01 modification */
2287     /* Enclose the actual calculations and printed results in a macro */
2288     /* to facilitate multiple applications of the algorithm. */
2289

```

```

2290 /* PART III. Perform calculations: */
2291
2292 data EX_RISK (label = "Estimated excess risks [Method=BEIR IV]"
2293             /*keep = XLevel Rx ex_risk RskRatio */
2294             rename= (Rx=Risk));
2295
2296 /*-----+
2297 | Calculate risk and excess risk for each exposure concentration|
2298 | in work.X_Level by BEIR IV method using information in      |
2299 | work.AllCause and work.Cause to define referent population:  |
2300 +-----*/
2301 length XLevel 8.;
2302 label Age      = "Age at start of year (i)"
2303       XTime    = "Exposure duration midway between i & i+1[Xtime]"
2304       XDose    = "Cumulative exposure midway thru year (C*Q*Adj)[XDose]"
2305
2306       R0       = "Cumulative risk of mesothelioma (unexposed) (R0)"
2307       Rx       = "Cumulative risk of mesothelioma (exposed) (Re)"
2308       Ex_Risk  = "Excess risk (Rx-Ro)"
2309       RskRatio = "Ratio of risks (Rx/Ro)"
2310
2311       hi       = "Mesothelioma hazard (unexposed) (hi)"
2312       hix      = "Mesothelioma hazard (exposed) (hei)"
2313       hstari   = "All cause hazard (unexposed) (h*i)"
2314       hstarix  = "All cause hazard (exposed) (he*i)"
2315       qi       = "Probability of surviving year i assuming alive at start (unexposed)
2316 (qi)"
2317       S_li     = "Probability of surviving to end of year i (unexposed) (S1,i)"
2318       S_lix    = "Probability of surviving to end of year i (exposed) (Sel,i)"
2319               XLevel = "Ecl%";
2320
2321 /* BT 7/5/19:add arrays for writing out the values for Array S_li and
2322 S_lix */
2323       * ARRAY A_S_li[0:85]; *0 corresponds to age=0;
2324       * ARRAY A_S_lix[0:85];
2325
2326       *A_S_li[0]=1;* A_S_lix[0]=1;
2327
2328
2329
2330 /* BT 3/8/19: Calculation of unexposed's risk (following DO LOOP) could be omitted
2331 from the iteration
2332 but may require further changes to BEIR4(?).
2333 *e.g., %if i=1 %then %do;*/
2334
2335 if _n_=1 then DO;
2336 /* Calculate unexposed's risk (R0) to be retained */
2337 /* based on equation 2A-21 (pg. 131) of BEIR IV: */
2338
2339 /* Initialize: */ S_li = 1; R0 = 0; R0rs=0; S_lirs=1;
2340
2341 DO pointer = 1 to min(n_all,n_cause) until (age>=&LastAge-1);
2342 set allcause (keep=age AllCause rename=(AllCause=hstari))
2343 point=pointer nobs=n_all;
2344 set cause (keep=age Rate rename=(age=ageCause Rate=hi))
2345 point=pointer nobs=n_cause;
2346
2347 if Age NE AgeCause then
2348 put "*** WARNING: Age values in datasets ALLCAUSE and CAUSE don't conform ***"
2349 / @13 "Rates misaligned on age could give incorrect results"
2350 / @13 Pointer=
2351 +2 "Age(ALLCAUSE)=" Age +2 "Age(CAUSE)=" AgeCause /;
2352
2353 qi = exp(-hstari);
2354 R0 = R0 + ( hi/hstari * S_li * (1-qi) );
2355 S_li = S_li * qi;
2356
2357
2358
2359
2360 END;

```

```

2361      END;                /* End of 'if _n_=1 then DO;' stmt */
2362
2363      retain R0;
2364
2365
2366      /* Calculate exposed's risk (Rx) for each exposure level          */
2367      /* ultimately based on equation 2A-22 (pg. 132) of BEIR IV        */
2368      /* but re-expressed in a form similar to equation 2A-21:        */
2369
2370      * BT 3/20/19. This version of CONVERGE_BEIR4 will work when there is
2371      one concentration in data set x_levels - i.e., one value
2372 for xlevel.
2373
2374      The Do loop for X_levels is commented out;
2375      *DO pointX = 1 to No_of_Xs;
2376      * set x_levels point=pointX nobs=No_of_Xs; /* BT 3/8/19: determines when to
2377 end the loop. Nobs is set at compilation,
2378
2379      so the value of nobs is available at first run through loop -
2380
2381      just one record and one variable (XLevel) in dataset x_levels. */
2382
2383      xlevel = &exposure_conc;
2384
2385      /* Initialize : */ S_lix = 1; Rx = 0; S_li=1;
2386
2387      DO pointer = 1 to min(n_all,n_cause) until (age>=&LastAge-1);
2388      set allcause (keep=age AllCause rename=(AllCause=hstari))
2389      point=pointer nobs=n_all;
2390      set cause (keep=Rate rename=(Rate=hi))
2391      point=pointer nobs=n_cause;
2392
2393      /*
2394      XTime = min( max(0, (age+0.5-&Agelst_x-&Lag)
2395      , &Duration );
2396
2397
2398      Q = .;
2399      If Age < 10 then Q = 0;
2400      If Age >= (XTime +10) then Q = ((Age-10)**3)-((-10-XTime)**3);
2401      Else Q = (XTime-10)**3;
2402
2403      */
2404
2405      TSFE=.;
2406      If Age < &Agelst_x then TSFE = 0;
2407      Else TSFE = Age - &Agelst_x + 0.5;
2408
2409      d = .;
2410      If Age < &Agelst_x then d = 0; else
2411      If Age >= &Agelst_x + &Duration then d = &Duration - 0.5;
2412      Else d = Age-&Agelst_x + 0.5;
2413
2414
2415      Q=.;
2416      If TSFE < 10 then Q = 0; else
2417      If TSFE >= d+10 then Q = (TSFE-10)**3-(TSFE-10-d)**3;
2418      Else Q = (TSFE-10)**3;
2419
2420      if UpCase("&EnvAdj") = "YES" /* Occupational to Environmental Conversion */
2421      then XDose = XLevel
2422      * 365/240 /* Days per year */
2423      * 20/10 /* Ventilation (L) per day */
2424      * Q; /* BT: in lung cancer program, this line has
2425 just XTime (instead of Q) */
2426      ELSE if UpCase("&EnvAdj") = "NO" /* 30nov2018 ('ELSE') */
2427      then XDose = XLevel*XTime;
2428      else DO; put //"Macro variable ENVADJ incorrectly specified."
2429      /*It should be either YES or NO. Value specified is: &ENVADJ"
2430      /*
2431      STOP;

```

```

2432         END;
2433     hix=.;
2434     if &Model = 1 then hix = hi * exp(&COEF*XDose);   else
2435     if &Model = 2 then hix = hi * (1 + &COEF*XDose); else
2436     if &Model = 3 then hix = hi + &COEF*XDose;     else
2437     if &Model = 4 then hix = hi * (1 + XDose)**&COEF; else
2438     if &Model = 0 then DO;
2439         hix = -99999; /* Code for user-defined model goes here. */
2440     END;
2441
2442
2443
2444         /*start of what RS added */
2445         qi = exp(-hstari);
2446         R0 = R0 + ( hi/hstari * S_li * (1-qi) );
2447     S_li = S_li * qi;
2448
2449         /*end of what RS added */
2450
2451     hstarix = hstari          /* hi=backgrd rate is included in hstari */
2452             + (hix - hi);    /* so that adding in the excess */
2453                             /* from exposure (hix-hi) gives the */
2454                             /* total rate of the exposed. */
2455
2456     qix = exp(-hstarix);
2457     Rx = Rx + ( hix/hstarix * S_lix * ( 1-qix ) );
2458     S_lix = S_lix * qix;
2459
2460
2461         output;
2462     END;
2463     Ex_Risk = Rx - R0; /* BT 4/2/19: was Ex_Risk = Rx - R0; */
2464     * RskRatio = Rx / R0;
2465     output;
2466
2467         /* BT 4/14/19: the macro variables for risk and difference between
2468 the calculated risk
2469                                     and the target risk were moved from Converge_BEIR4
2470 to BEIR4 */
2471         call symput('Extra_Riskm',Ex_Risk);
2472
2473         Diff_Ex_Risk = abs(&ex_risk_target-Ex_Risk);
2474         call symput('Delta_Ex_Risk',Diff_Ex_Risk);
2475
2476     * END; * corresponds to X_Levels;
2477
2478     STOP;
2479     run;
2480
2481
2482 %Mend BEIR4;
2483
2484 /* -----
2485         BT: March 2019: parameters for the convergence that are used
2486         in the modified version of the BEIR4 macro.
2487 -----*/
2488
2489 %macro Converge_BEIR4 (init_exposure_conc=, ex_risk_target=, conv_criterion=, max_iteration=);
2490
2491     %Let Extra_Riskm = 1;
2492
2493     %Let Delta_Ex_Risk = 1; * initial high value to make sure loop is run at least once
2494                             (i.e., macro BEIR4 is called
2495 at least once);
2496
2497     %Let i=1; * first time through loop;
2498
2499
2500
2501

```

```

2502     %Do %Until (%sysevalf(&Delta_Ex_risk < &conv_criterion) OR %sysevalf(&i >
2503 &max_iteration));
2504
2505         * first time through loop, set exposure_conc=init_exposure_conc;
2506
2507         %If &i=1 %Then
2508             %Do;
2509                 %Let exposure_conc=&init_exposure_conc;
2510
2511             %End;
2512         %If &i>1 %Then
2513             %Do;
2514
2515                 data tempBEIRCONVERGE;
2516
2517                 least once. Adjust exposure_conc
2518
2519                 BEIR4 (=Ex_Risk)*/
2520
2521                 NumLoops=&i;
2522                 thisExposureConc=&exposure_conc; *set equal to
2523                 concentration in loop i-1;
2524
2525                 numvar=&ex_risk_target;
2526                 denvar=&Extra_Riskm;
2527
2528                 thisexposureconc = thisexposureconc * (numvar/denvar);
2529
2530                 *update the concentration;
2531                 call symput('exposure_conc',thisexposureconc);
2532
2533                 output;
2534
2535                 Run;
2536
2537                 %End; *Corresponds to If i>1 statement;
2538
2539             %BEIR4;
2540
2541             %Let i=%eval(&i+1);
2542
2543         %End;
2544
2545         %Let EC_1Percent = &exposure_conc;
2546
2547         /*-----+
2548         | Report results if convergence criterion met:
2549         +-----*/
2550
2551         %If %sysevalf(&Delta_Ex_risk < &conv_criterion) %then %do;
2552
2553             title5 "based on KM=&COEF, Concentration=&EC_1Percent, and LastAge=&LastAge";
2554
2555             data _null_;
2556                 /* Modified 26-july-00 */
2557                 pointer=1;
2558                 set allcause (keep=age
2559                             rename=(age=ageall0)) point=pointer nobs=n_all;
2560                 set cause (keep=age
2561                            rename=(age=ageCs0)) point=pointer nobs=n_cause;
2562                 pointer=n_all;
2563                 set allcause (keep=age
2564                             rename=(age=ageall1)) point=pointer nobs=n_all;
2565                 pointer=n_cause;
2566                 set cause (keep=age
2567                           rename=(age=ageCs1)) point=pointer nobs=n_cause;
2568
2569                 Tmp = sum(min(AgeAll1, AgeCs1, (&Lastage-1)), 1);
2570                 file PRINT;
2571
2572                 if ageall0 NE ageCs0 then DO;
2573                     put /"ERROR: The initial age for all-causes rate differs from the"
2574                         /" initial age for the cause-specific rate.";

```

```

2573     END;
2574     else DO;
2575         put / "Values of macro variables used in this computation:      "
2576             // @3 "Value"      @17 "Macro_Var" @29 "Description"
2577             // @3 "-----"    @17 "-----"  @29 "-----"
2578             // @3 "&Model"    " @17 "MODEL"    @29 "1 = Loglinear Relative Rate,"
2579             /                                     @29 "2 = Linear Relative Rate, "
2580             /                                     @29 "3 = Linear Absolute Rate, "
2581             /                                     @29 "4 = 'Power' Relative Rate, "
2582             /                                     @29 "0 = User defined.      "
2583             // @3 "&Coef"     " @17 "COEF"      @29 "Exposure parameter estimate"
2584             // @3 "&Lag"      " @17 "LAG"        @29 "Exposure Lag "
2585             // @3 "&Age1st_x" @17 "AGE1ST_X"    @29 "Age exposure begins"
2586             // @3 "&Duration" @17 "DURATION"    @29 "Duration of exposure"
2587             // @3 "&EnvAdj"   @17 "ENVADJ"      @29 "Adjust dose from intermittent"
2588             /                                     @29 "occupational exposures to "
2589             /                                     @29 "continuous environmental exposures"
2590             / @3 "-----"    @17 "-----"    @29 "-----"
2591 "
2592             // "-----"
2593             // @3 "EC1% = " @10 "&EC_1Percent" @20 " (f/ml); Rx = " @34
2594 "&Extra_Riskm"
2595             // "-----"
2596 "-"
2597
2598             /"The risks are calculated from age " ageall0 " up to age " Tmp "."
2599             // ;
2600
2601     if ageall1 NE ageCsl then
2602         put /"WARNING: The last age for the all-causes rates differs from"
2603           /"         the last age for the cause-specific rates, suggesting"
2604           /"         the possibility that the rates weren't entered as desired."
2605           /;
2606     END;
2607 Stop;
2608 run;
2609
2610 /* BT 7/5/19: Start of code that was added to merge variables for unexposed risk
2611              (S_li and S_lix) to the rest of the output, by age;
2612 */
2613
2614 Data newSRCData(keep=SRC_age SRC_S_li SRC_S_lix);
2615     set ex_Risk;
2616     SRC_age=0; SRC_S_li=1; SRC_S_lix=1;
2617     output;
2618
2619     do obsnum=1 to last-1;
2620         set ex_Risk point=obsnum nobs=last;
2621         if _error_ then abort;
2622         SRC_age=age+1; SRC_S_li=S_li; SRC_S_lix=S_lix;
2623         output;
2624     end;
2625
2626     stop;
2627 run;
2628
2629 * rename variables to enable overwriting the values of S_li and S_lix in ex_risk with the values
2630 in newSRCData;
2631 * Data file tempSRCData has age=0-85 while the ex_Risk file has age 0-84, with last two
2632 records
2633     both having age=84.;
2634 Data tempSRCData; Set newSRCData(rename=(SRC_Age=age SRC_S_li=S_li SRC_S_lix=S_lix));
2635     if age=&LastAge then age=%sysevalf(&Lastage-1); Else age=age;
2636 Run;
2637
2638 * there are duplicate values for age in both ex_risk and tempSRCData
2639     which may produce too many records. if that happens, then we use two set
2640 statements;
2641 Data ex_risk; merge ex_risk tempSRCData; By Age; Run;
2642
2643 /* BT 7/5/19: End of code that was added to merge variables for unexposed risk

```

```

2644                                     (S_li and S_lix) to the rest of the output, by age;
2645 */
2646
2647     proc print data=ex_risk label noobs;
2648         var Age TSFE d Q hix hi hstari hstarix qi qix S_li S_lix R0 Risk;
2649         label d="Exp. duration midway thru year i (yrs)"
2650                TSFE="TSFE midway thru year i (yrs)"
2651                Q="Q (yrs3)"
2652                qix="Probability of surviving year i assuming alive at start (exposed)
2653 (qei)";
2654
2655         run;
2656
2657
2658 %End; *end of the If statement that tests if convergence was met;
2659
2660 %Mend Converge_BEIR4;
2661
2662
2663
2664 /* the following options are for debugging - comment out after code is running as expected*/
2665 Options mlogic mprint symbolgen;
2666
2667
2668 /*
2669     %Let LastAge =85;
2670     %LET LAG = 10;
2671     %Let MODEL = 3;
2672     %Let COEF = 0.000000015;
2673 */
2674
2675 /* -----+
2676 | April 2019: BT (SRC) Added maxro CONVERGE_BEIR4 which iteratively |
2677 | runs macro BEIR4 until the EXPOSURE_CONCENTRATION corresponds to an |
2678 | extra_risk=0.01 (the point of departure [POD]). |
2679 |
2680 |
2681 | At the second iteration of the Converge_BEIR4 macro, the exposure |
2682 | concentration is adjusted by a factor equal to the initial |
2683 | concentration x ConvRate. It is recommended to use a convrate equal |
2684 | to 0.1, which produces an adjustment of approximately 10% of the |
2685 | initial concentration value. The conversion rate is adjusted in |
2686 | later iterations (to smaller adustments) as needed to converge. |
2687 |
2688 |
2689 |
2690 | In addition to the parameter for CONVERGE_BEIR4, the user should also|
2691 | review parameters and data that are assigned/entered in Part I and |
2692 | Part II (see above). Parameters for CONVERGE_BEIR4 are defined below |
2693 +-----*/
2694
2695 *%BEIR4; * originally called macr BEIR4 directly. Now BEIR4 is called by Converge_BEIR4;
2696
2697
2698
2699     %Converge_BEIR4(init_exposure_conc=0.1, /* initial exposure concentration (initial
2700 guess) */
2701
2702                                     ex_risk_target= 0.0100, /* the point of departure
2703 (POD) - the target extra risk */
2704                                     conv_criterion=0.00000001,
2705                                     max_iteration=300); /* to avoid excessively long
2706 run times */
2707
2708 Run;
2709

```


2710

2711

Appendix J Results of Modeling for IUR Derivation

2712

Section 1

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Hein et al. (2007)

2714

2715

SOUTH CAROLINA LUNG CANCER KL FITTING

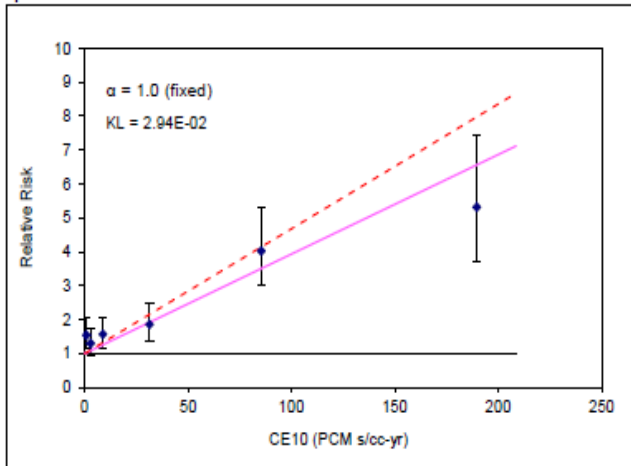
Citation: Hein et al 2007

Cohort: South Carolina

Data: NIOSH

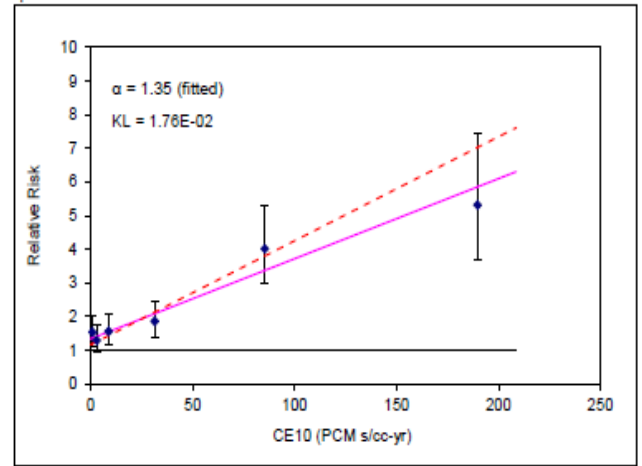
CE10 (PCM s/cc-yrs)			Lung Cancer Deaths		
Min	Max	Mid	Obs	Exp	RR
0	1.5	0.8	34	22.10	1.54
1.5	5	2.9	33	25.30	1.30
5	15	8.7	34	21.70	1.57
15	60	31.3	35	18.80	1.86
60	120	85.3	37	9.20	4.02
120		189.6	25	4.70	5.32
			198	101.80	1.94

Alpha Fixed at 1.00



Value	Alpha	KL	AIC
MLE	1.00	2.94E-02	42.41
UB	1.00	3.68E-02	--

Alpha = Fitted



Value	Alpha	KL	AIC
MLE	1.35	1.76E-02	36.48
UB	1.17	2.64E-02	--

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2717

2718

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 2724
 2725
 2726
 2727
 2728

Section 2

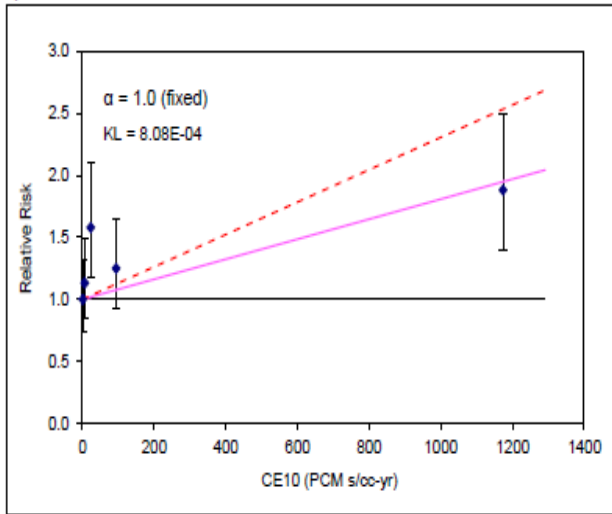
Loomis et al. (2009)

NORTH CAROLINA LUNG CANCER KL FITTING

Citation: Loomis et al 2009
 Cohort: North Carolina
 Data: Table 5

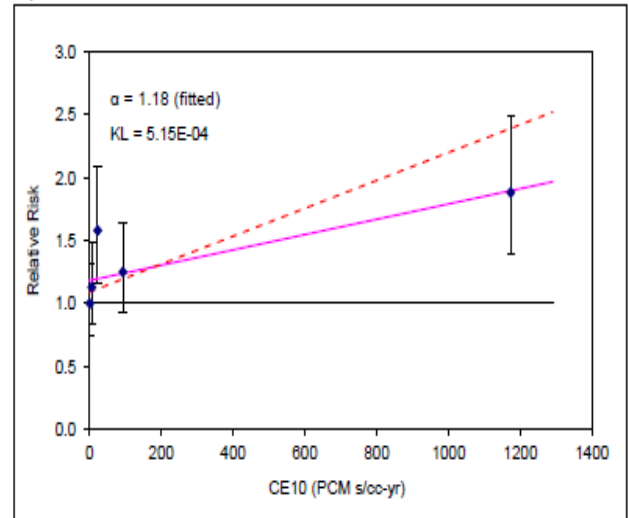
CE10 (PCM s/cc-yrs)			Lung Cancer Deaths		
Min	Max	Mid	Obs	Exp	RR
0	2.3	1.2	37	37.00	1.00
2.3	11.5	6.9	37	32.74	1.13
11.5	34.8	23.2	35	22.15	1.58
34.8	152.7	93.8	37	29.60	1.25
152.7	2194	1173.4	35	18.62	1.88
			181	140.11	1.29

Alpha Fixed at 1.00



Value	Alpha	KL	AIC
MLE	1.00	8.08E-04	35.33
UB	1.00	1.31E-03	--

Alpha = Fitted



Value	Alpha	KL	AIC
MLE	1.18	5.15E-04	32.83
UB	1.09	1.02E-03	--

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 2730
 2731
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Section 3

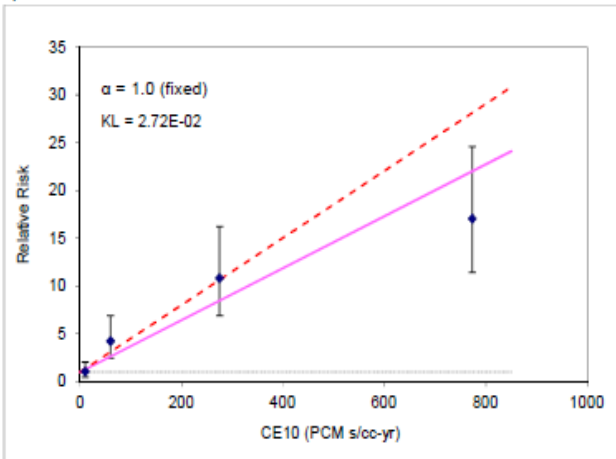
Wang et al. (2013b)

QINGHAI, CHINA LUNG CANCER KL FITTING

Citation: Wang et al. 2013
Cohort: Chinese miners (all)
Data: Table 5 + 6

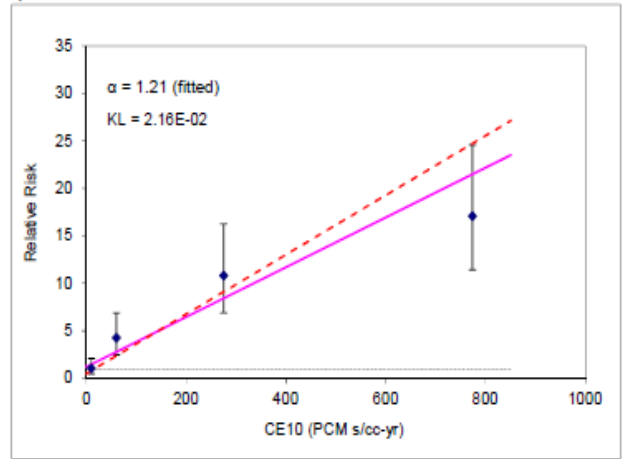
CE10 (PCM s/cc-yrs)			Lung Cancer Deaths		
Min	Max	Mid	Obs	Exp	RR
0	20	10.0	6	5.75	1.04
20	100	60.0	12	2.82	4.25
100	450	275.0	17	1.57	10.82
450	1097	773.5	21	1.23	17.07
			56	11.37	4.92

Alpha Fixed at 1.00



Value	Alpha	KL	AIC
MLE	1.00	2.72E-02	23.62
UB	1.00	3.51E-02	--

Alpha = Fitted



Value	Alpha	KL	AIC
MLE	1.21	2.16E-02	24.44
UB	0.48	6.47E-02	--

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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 1 st exposure (years)	Duration of exposure (years)										
	1	5	10	15	20	25	30	35	40	62	78
0	4.14E-03	3.34E-02	6.34E-02	8.71E-02	1.06E-01	1.20E-01	1.31E-01	1.38E-01	1.44E-01	1.55E-01	1.56E-01
2	3.82E-03	3.06E-02	5.80E-02	7.96E-02	9.63E-02	1.09E-01	1.18E-01	1.25E-01	1.30E-01	1.40E-01	1.41E-01
4	3.52E-03	2.81E-02	5.30E-02	7.25E-02	8.75E-02	9.88E-02	1.07E-01	1.13E-01	1.18E-01	1.26E-01	1.26E-01
6	3.23E-03	2.56E-02	4.83E-02	6.59E-02	7.93E-02	8.94E-02	9.68E-02	1.02E-01	1.06E-01	1.13E-01	1.13E-01
8	2.97E-03	2.34E-02	4.39E-02	5.97E-02	7.17E-02	8.07E-02	8.73E-02	9.20E-02	9.54E-02	1.01E-01	
10	2.72E-03	2.13E-02	3.98E-02	5.40E-02	6.47E-02	7.26E-02	7.84E-02	8.26E-02	8.56E-02	9.08E-02	
12	2.48E-03	1.93E-02	3.60E-02	4.87E-02	5.82E-02	6.53E-02	7.04E-02	7.41E-02	7.67E-02	8.10E-02	
14	2.27E-03	1.75E-02	3.25E-02	4.38E-02	5.23E-02	5.85E-02	6.30E-02	6.63E-02	6.86E-02	7.21E-02	
16	2.07E-03	1.58E-02	2.92E-02	3.93E-02	4.68E-02	5.23E-02	5.63E-02	5.92E-02	6.12E-02	6.41E-02	
18	1.88E-03	1.42E-02	2.62E-02	3.52E-02	4.19E-02	4.67E-02	5.02E-02	5.28E-02	5.46E-02	5.69E-02	
20	1.71E-03	1.28E-02	2.35E-02	3.15E-02	3.74E-02	4.17E-02	4.48E-02	4.70E-02	4.86E-02	5.04E-02	
22	1.56E-03	1.14E-02	2.10E-02	2.81E-02	3.33E-02	3.71E-02	3.99E-02	4.19E-02	4.33E-02	4.46E-02	
24	1.42E-03	1.02E-02	1.87E-02	2.50E-02	2.96E-02	3.30E-02	3.55E-02	3.73E-02	3.85E-02		
26	1.29E-03	9.15E-03	1.67E-02	2.23E-02	2.64E-02	2.94E-02	3.16E-02	3.32E-02	3.42E-02		
28	1.17E-03	8.16E-03	1.49E-02	1.98E-02	2.34E-02	2.62E-02	2.81E-02	2.95E-02	3.04E-02		
30	1.07E-03	7.27E-03	1.32E-02	1.76E-02	2.09E-02	2.33E-02	2.51E-02	2.63E-02	2.69E-02		
32	9.76E-04	6.48E-03	1.18E-02	1.57E-02	1.86E-02	2.08E-02	2.24E-02	2.34E-02	2.38E-02		
34	8.95E-04	5.78E-03	1.05E-02	1.40E-02	1.66E-02	1.86E-02	2.00E-02	2.07E-02	2.10E-02		
36	8.24E-04	5.17E-03	9.37E-03	1.25E-02	1.49E-02	1.66E-02	1.78E-02	1.84E-02	1.85E-02		
38	7.62E-04	4.63E-03	8.39E-03	1.12E-02	1.34E-02	1.49E-02	1.58E-02	1.62E-02	1.62E-02		
40	7.08E-04	4.16E-03	7.54E-03	1.01E-02	1.20E-02	1.33E-02	1.40E-02	1.42E-02	1.42E-02		

42	6.58E-04	3.75E-03	6.79E-03	9.09E-03	1.08E-02	1.18E-02	1.23E-02	1.24E-02	1.24E-02		
44	6.16E-04	3.39E-03	6.12E-03	8.18E-03	9.63E-03	1.05E-02	1.08E-02	1.08E-02	1.08E-02		
46	5.74E-04	3.07E-03	5.53E-03	7.35E-03	8.56E-03	9.17E-03	9.31E-03	9.31E-03			
48	5.35E-04	2.78E-03	4.98E-03	6.57E-03	7.54E-03	7.95E-03	7.98E-03	7.98E-03			
50	4.99E-04	2.52E-03	4.48E-03	5.82E-03	6.56E-03	6.77E-03	6.77E-03	6.77E-03			
52	4.61E-04	2.28E-03	3.99E-03	5.09E-03	5.60E-03	5.68E-03	5.68E-03				
54	4.26E-04	2.04E-03	3.51E-03	4.37E-03	4.68E-03	4.69E-03	4.69E-03				
56	3.87E-04	1.81E-03	3.03E-03	3.66E-03	3.80E-03	3.80E-03					
58	3.47E-04	1.57E-03	2.56E-03	2.97E-03	3.00E-03	3.00E-03					
60	3.07E-04	1.34E-03	2.08E-03	2.30E-03	2.30E-03	2.30E-03					
62	2.61E-04	1.10E-03	1.61E-03	1.69E-03	1.69E-03						
64	2.17E-04	8.58E-04	1.17E-03	1.18E-03	1.18E-03						
66	1.69E-04	6.25E-04	7.61E-04	7.61E-04							
68	1.23E-04	4.10E-04	4.43E-04	4.43E-04							
70	8.08E-05	2.17E-04	2.17E-04	2.17E-04							
72	4.22E-05	7.47E-05	7.47E-05								
74	7.97E-06	7.97E-06	7.97E-06								

2751 For calculation of Table _ApX K-1, the following procedure was used. For each cell of the table,
2752 the lung cancer and mesothelioma partial lifetime risk corresponding to the age at first exposure
2753 and duration of exposure was calculated using selected models for lung cancer and mesothelioma
2754 and potency factors from Table 3-10 and 3-11, Then lung cancer and mesothelioma risks were
2755 statistically combined using the same procedure as described in [Section 3.2.3.8.2](#).

2756
2757

Appendix L Sensitivity Analysis of Exposures for DIY/Bystander Episodic Exposure Scenarios

2758 As presented in Section 4.3.5, there are some uncertainties pertaining to the assumptions made
2759 for exposure durations for both DIY users and bystanders for the brake repair/replacement
2760 scenarios. This Appendix provides a more detailed analyses using various combinations of age
2761 at start of first exposure and duration of exposure for both the DIYers and the bystanders for both
2762 the brake repair/replacement and the UTV gasket repair/replacement scenarios.

2763 In Table L-1, the assumption is that DIY brake/repair replacement with compressed air begins at
2764 age 16 years and continues for 20 years instead of for 62 years.

2765
2766 Here, the unit risk for Users is: $IUR_{LTL}(DIY\ Brakes) = IUR(16,20) = 0.0468$ per f/cc
2767 The unit risk for Bystanders is: $IUR_{LTL}(DIY\ Bystanders) = IUR(0,20) = 0.1057$ per f/cc

2768 **Table_Apx L-1. Excess Lifetime Cancer Risk for Indoor DIY Brake/Repair Replacement**
2769 **with Compressed Air Use for Consumers for 20 year duration (exposures from Table 2-27**
2770 **without a reduction factor) (Consumers 1 hour/day spent in garage).**

Consumer Exposure Scenario	Exposure Levels (f/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	2.6 E-5	2.6 E-4	1.7 E-5	3.9 E-5

2771 $TWF_{Concomitant\ Exposures} (1\ hour\ per\ day\ every\ day) = (1/24) * (365/365) = 0.04167$
2772 DIY User: $ELCR_{(Central\ Tendency)} = 0.0445\ f/cc \cdot 0.0001142 \cdot 0.0468\ per\ f/cc + 0.0445 \cdot 0.3 \cdot 0.04167 \cdot 0.0468$
2773 DIY User: $ELCR_{(High-end)} = 0.4368\ f/cc \cdot 0.0001142 \cdot 0.0468\ per\ f/cc + 0.4368 \cdot 0.3 \cdot 0.04167 \cdot 0.0468$
2774 DIY Bystander: $ELCR_{(Central\ Tendency)} = 0.013\ f/cc \cdot 0.0001142 \cdot 0.1057\ per\ f/cc + 0.013 \cdot 0.3 \cdot 0.04167 \cdot 0.1057$
2775 DIY Bystander: $ELCR_{(High-end)} = 0.0296\ f/cc \cdot 0.0001142 \cdot 0.1057\ per\ f/cc + 0.0296 \cdot 0.3 \cdot 0.04167 \cdot 0.1057$

2776 Exposure values from Table 2-27 were used to represent indoor brake work (with compressed
2777 air) and are the basis for the exposure levels used in Table_Apx L-1. EPA then assumed that the
2778 concentration of chrysotile asbestos in the interval between brake work (every 3 years) is 30% of
2779 that during measured active use. Consumers were assumed to spend one hour per day in their
2780 garages based on the 50th percentile estimate in the EPA Exposure Factors Handbook. Based on
2781 these assumptions, the consumer risk estimates were exceeded for central tendency and high-end
2782 exposures (L-1). Estimates exceeding the benchmark are shaded in pink and bolded. Comparing
2783 these results with those of Table 4-39, we see that the ratio of the risks for the DIY User based
2784 on 20 years exposure compared to 40 years of exposures is equal to the ratio of the less than
2785 lifetime inhalation unit risks:

2786
2787 DIY Users: $[IUR(16,20) = 0.0468\ per\ f/cc] / [IUR(16,62) = 0.0641\ per\ f/cc] = \mathbf{0.73}$
2788 DIY Users: $[20\ yr\ risk\ (Central) = 2.63\ E-5] / [62\ yr\ risk\ (Central) = 3.60\ E-5] = \mathbf{0.73}$

2789 DIY Users: [20 yr risk (High) = 2.58 E-4] / [62 yr risk (High) = 3.50 E-4] = **0.73**

2790 Similarly for bystanders, the ratio of the risk based on 20 years exposure compared to 62 years
 2791 exposure is equal to the ratio of the 20-year less than lifetime risk to the lifetime unit risk:

2792 DIY Bystanders: [IUR(0,20) = 0.1057 per f/cc] / [IUR(Lifetime) = 0.16 per f/cc] = **0.66**

2793 DIY Bystanders: [20 yr risk (Central) = 1.73 E-5] / [78 yr risk (Central) = 2.62 E-5] = **0.66**

2794 DIY Bystanders: [20 yr risk (High) = 3.95 E-5] / [78 yr risk (High) = 5.97 E-5] = **0.66**

2795

2796 Using this approach, and relying on the ratios presented in Table 4-49, Table_Apx L-2 provides
 2797 and ratios for five different sensitivity pairings.
 2798

2799 **Table_Apx L-2. Ratios of risk for alternative exposure scenarios compared to DIY User**
 2800 **and Bystander exposure scenario assuming DIY User is first exposed at age 16 years for 62**
 2801 **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	0.0641	0.0641	1
	Bystander	0	78	0.16	0.16	1
Sensitivity #1	DIY User	16	20	0.0641	0.0468	0.73
	Bystander	0	20	0.16	0.1057	0.66
Sensitivity #2	DIY User	20	40	0.0641	0.0486	0.76
	Bystander	0	40	0.16	0.144	0.90
Sensitivity #3	DIY User	20	20	0.0641	0.0374	0.58
	Bystander	0	20	0.16	0.1057	0.63
Sensitivity #4	DIY User	30	40	0.0641	0.0269	0.42
	Bystander	0	40	0.16	0.144	0.90
Sensitivity #5	DIY User	30	20	0.0641	0.0209	0.33
	Bystander	0	20	0.16	0.1057	0.63

2802

2803 Table_Apx L-3 through Table_Apx L-7 below show the results of applying these ratios to all of
 2804 the possible scenarios presented in Table 4-48 using the five sensitivity analyses pairings in
 2805 Table_Apx L-2. Table_Apx L-8 at the end summarizes the results to show how only one of 24
 2806 scenarios changes from an exceedance to no exceedance for all five sensitivity analyses (DIY
 2807 user, Brakes Repair/ replacement, Outdoor, once every 3 years, 30 min/d in driveway, high-end
 2808 only).

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2810
2811
2812

Table_Apx L-3. Sensitivity Analysis #1: Summary of Risk Estimates for Inhalation Exposures to Consumers and Bystanders by COU (Cancer benchmark is 10⁻⁶) Comparing the Baseline Exposure Scenario from Table 4-48 with Risks Assuming DIY Users Are Exposed From Age 16-36 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 4-48)	Cancer Risk Estimates Users age 16-36 (*0.73) and Bystanders 0-20 (*0.66)
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	3.6 E-5	2.6 E-5
				High-end	3.5 E-4	2.6 E-4
			Bystander	Central Tendency	2.6 E-5	1.7 E-5
				High-end	6.0 E-5	4.0 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	2.6 E-4	1.9 E-4
				High-end	2.6 E-3	1.9 E-3
			Bystander	Central Tendency	1.7 E-5	1.1 E-5
				High-end	3.9 E-5	2.6 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	Central Tendency	5.4 E-6	3.9 E-6
				High End	5.3 E-5	3.9 E-5
			Bystander	Central Tendency	3.4 E-6	2.2 E-6
				High-end	7.8 E-6	5.1 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	8.2 E-8	6.0 E-8	
			High-end	4.4 E-7	3.2 E-7	
		Bystander	Central Tendency	2.1 E-8	1.4 E-8	
			High-end	1.1 E-7	7.3 E-8	

Life Cycle Stage/Category	Subcategory	Consumer Exposure Scenario	Population	Exposure Duration and Level	Cancer Risk Estimates (from Table 4-48)	Cancer Risk Estimates Users age 16-36 (*0.73) and Bystanders 0-20 (*0.66)
	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	2.4 E-7	1.8 E-7
				High-end	1.3 E-6	9.5 E-7
			Bystander	Central Tendency	5.9 E-8	3.9 E-8
				High-end	3.2 E-7	2.1 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	1.9 E-5	1.4 E-5
				High-end	5.3 E-5	3.9 E-5
			Bystander	Central Tendency	2.4 E-5	1.6 E-5
				High-end	6.1 E-5	4.0 E-5
	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	Section 4.2.3.2	DIY	Central Tendency	1.5 E-4	1.1 E-4
				High-end	4.2 E-4	3.1 E-4
			Bystander	Central Tendency	2.4 E-5	1.6 E-5
				High-end	6.1 E-5	4.0 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	2.9 E-6	2.1 E-6
				High end	8.0 E-6	5.8 E-6
			Bystander	Central Tendency	3.2 E-6	2.1 E-6
				High-end	7.9 E-6	5.2 E-6

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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 10⁻⁶) Comparing the Baseline Exposure Scenario from Table 4-48 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 4-48)	Cancer Risk Estimates Users age 20-40 (*0.76) and Bystanders 0-40 (*0.90)
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	3.6 E-5	2.7 E-5
				High-end	3.5 E-4	2.7 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 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	2.6 E-4	2.0 E-4
				High-end	2.6 E-3	2.0 E-3
			Bystander	Central Tendency	1.7 E-5	1.5 E-5
				High-end	3.9 E-5	3.5 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	Central Tendency	5.4 E-6	4.1 E-6
				High End	5.3 E-5	4.0 E-5
			Bystander	Central Tendency	3.4 E-6	3.1 E-6
				High-end	7.8 E-6	7.0 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	8.2 E-8	6.2 E-8	
			High-end	4.4 E-7	3.3 E-7	
		Bystander	Central Tendency	2.1 E-8	1.9 E-8	
			High-end	1.1 E-7	9.9 E-8	

Life Cycle Stage/Category	Subcategory	Consumer Exposure Scenario	Population	Exposure Duration and Level	Cancer Risk Estimates (from Table 4-48)	Cancer Risk Estimates Users age 20-40 (*0.76) and Bystanders 0-40 (*0.90)
	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	2.4 E-7	1.8 E-7
				High-end	1.3 E-6	9.9 E-7
			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 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	1.9 E-5	1.4 E-5
				High-end	5.3 E-5	4.0 E-5
			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 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	1.5 E-4	1.1 E-4
				High-end	4.2 E-4	3.2 E-4
			Bystander	Central Tendency	2.4 E-5	2.2 E-5
				High-end	6.1 E-5	5.5 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	2.9 E-6	2.2 E-6
				High end	8.0 E-6	6.1 E-6
			Bystander	Central Tendency	3.2 E-6	2.9 E-6
				High-end	7.9 E-6	7.1 E-6

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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 10⁻⁶) Comparing the Baseline Exposure Scenario from Table 4-48 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 4-48)	Cancer Risk Estimates Users age 20-40 (*0.58) and Bystanders 0-20 (*0.63)
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	3.6 E-5	2.1 E-5
				High-end	3.5 E-4	2.0 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 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	2.6 E-4	1.5 E-4
				High-end	2.6 E-3	1.5 E-3
			Bystander	Central Tendency	1.7 E-5	1.1 E-5
				High-end	3.9 E-5	2.5 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	Central Tendency	5.4 E-6	3.1 E-6
				High End	5.3 E-5	3.1 E-5
			Bystander	Central Tendency	3.4 E-6	2.1 E-6
				High-end	7.8 E-6	4.9 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	8.2 E-8	4.8 E-8	
			High-end	4.4 E-7	2.6 E-7	
		Bystander	Central Tendency	2.1 E-8	1.3 E-8	
			High-end	1.1 E-7	6.9 E-8	

Life Cycle Stage/Category	Subcategory	Consumer Exposure Scenario	Population	Exposure Duration and Level	Cancer Risk Estimates (from Table 4-48)	Cancer Risk Estimates Users age 20-40 (*0.58) and Bystanders 0-20 (*0.63)
	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	2.4 E-7	1.4 E-7
				High-end	1.3 E-6	7.5 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 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	1.9 E-5	1.1 E-5
				High-end	5.3 E-5	3.1 E-5
			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 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	1.5 E-4	8.7 E-5
				High-end	4.2 E-4	2.4 E-4
			Bystander	Central Tendency	2.4 E-5	1.5 E-5
				High-end	6.1 E-5	3.8 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	2.9 E-6	1.7 E-6
				High end	8.0 E-6	4.6 E-6
			Bystander	Central Tendency	3.2 E-6	2.0 E-6
				High-end	7.9 E-6	5.0 E-6

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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 10⁻⁶) Comparing the Baseline Exposure Scenario from Table 4-48 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 4-48)	Cancer Risk Estimates Users age 30-70 (*0.42) and Bystanders 0-40 (*0.90)
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	3.6 E-5	1.5 E-5
				High-end	3.5 E-4	1.5 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 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	2.6 E-4	1.1 E-4
				High-end	2.6 E-3	1.1 E-3
			Bystander	Central Tendency	1.7 E-5	1.5 E-5
				High-end	3.9 E-5	3.5 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	Central Tendency	5.4 E-6	2.3 E-6
				High End	5.3 E-5	2.2 E-5
			Bystander	Central Tendency	3.4 E-6	3.1 E-6
				High-end	7.8 E-6	7.0 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	8.2 E-8	3.4 E-8	
			High-end	4.4 E-7	1.8 E-7	
		Bystander	Central Tendency	2.1 E-8	1.9 E-8	
			High-end	1.1 E-7	9.9 E-8	

Life Cycle Stage/Category	Subcategory	Consumer Exposure Scenario	Population	Exposure Duration and Level	Cancer Risk Estimates (from Table 4-48)	Cancer Risk Estimates Users age 30-70 (*0.42) and Bystanders 0-40 (*0.90)
	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	2.4 E-7	1.0 E-7
				High-end	1.3 E-6	5.5 E-7
			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 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	1.9 E-5	8.0 E-6
				High-end	5.3 E-5	2.2 E-5
			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 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	1.5 E-4	6.3 E-5
				High-end	4.2 E-4	1.8 E-4
			Bystander	Central Tendency	2.4 E-5	2.2 E-5
				High-end	6.1 E-5	5.5 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	2.9 E-6	1.2 E-6
				High end	8.0 E-6	3.4 E-6
			Bystander	Central Tendency	3.2 E-6	2.9 E-6
				High-end	7.9 E-6	7.1 E-6

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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 10⁻⁶) Comparing the Baseline Exposure Scenario from Table 4-48 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 4-48)	Cancer Risk Estimates Users age 16-36 (*0.33) and Bystanders 0-20 (*0.63)
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	3.6 E-5	1.2 E-5
				High-end	3.5 E-4	1.2 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 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	2.6 E-4	8.6 E-5
				High-end	2.6 E-3	8.6 E-4
			Bystander	Central Tendency	1.7 E-5	1.1 E-5
				High-end	3.9 E-5	2.5 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	Central Tendency	5.4 E-6	1.8 E-6
				High End	5.3 E-5	1.7 E-5
			Bystander	Central Tendency	3.4 E-6	2.1 E-6
				High-end	7.8 E-6	4.9 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	8.2 E-8	2.7 E-8	
			High-end	4.4 E-7	1.5 E-7	
		Bystander	Central Tendency	2.1 E-8	1.3 E-8	
			High-end	1.1 E-7	6.9 E-8	

Life Cycle Stage/Category	Subcategory	Consumer Exposure Scenario	Population	Exposure Duration and Level	Cancer Risk Estimates (from Table 4-48)	Cancer Risk Estimates Users age 16-36 (*0.33) and Bystanders 0-20 (*0.63)
	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	2.4 E-7	7.9 E-8
				High-end	1.3 E-6	4.3 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 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	1.9 E-5	6.3 E-6
				High-end	5.3 E-5	1.7 E-5
			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 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	1.5 E-4	5.0 E-5
				High-end	4.2 E-4	1.4 E-4
			Bystander	Central Tendency	2.4 E-5	1.5 E-5
				High-end	6.1 E-5	3.8 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	2.9 E-6	9.6 E-7
				High end	8.0 E-6	2.6 E-6
			Bystander	Central Tendency	3.2 E-6	2.0 E-6
				High-end	7.9 E-6	5.0 E-6

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Table_Apx L-4: 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 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	1/24	DIY user, Brake repair, 30 min/day, high-end
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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Appendix M Adjustment Factors to Correct for Bias in Cancer Risk Estimation

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2839 This appendix presents estimates for an “adjustment factor” used to correct for bias in the
2840 estimation of the risk of cancer due to the lack of inclusion of cancers other than lung cancer and
2841 mesothelioma in the estimation of the inhalation risk.

2842 Biases in the Cancer Risk Values

2843 The initial analysis did not include the risk from other cancers that have been associated with
2844 exposure to chrysotile asbestos. The reason for these shortcomings in the analysis is simply that
2845 there were no studies that could be used to model the exposure-response relationship for
2846 incidence or for other causes of cancer. However, EPA has developed “adjustment factors”
2847 which are used to correct for the negative bias in the risk values derived from lung cancer and
2848 mesothelioma, which are described below.

2849 *Biases Related to Not Including Other Cancer Sites*

2850 The inhalation cancer risk estimates originally derived by EPA in the DRE were only based upon
2851 lung cancer and mesothelioma mortality. Other cancers that have been recognized as being
2852 causally associated with asbestos sites by the International Agency for Research on Cancer
2853 (IARC) include laryngeal and ovarian cancer ([IARC, 2009](#)). The IARC also noted that ‘positive
2854 associations have been observed between exposure to all forms of asbestos and cancer of the
2855 pharynx, stomach, and colorectum’. However, the evidence for an association between these
2856 cancers and asbestos exposure is mixed and IARC did not view it as sufficient for a
2857 determination of causality. The EPA concurs with the IARC’s evaluation and has limited its
2858 effort to estimating the additional risk of ovarian and laryngeal cancer from inhalation exposure
2859 to chrysotile asbestos.

2860 A direct estimate of the risk of ovarian and laryngeal cancer cannot be made since none of the
2861 published studies have reported exposure-response results for these sites. An indirect estimate of
2862 additional risk can be determined by developing an adjustment factor by comparing the excess
2863 deaths from lung cancer with the number of excess deaths from the other cancer sites using the
2864 following formula:

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$$\text{Adjustment factor} = 1 + (\text{excess other cancer})/(\text{excess lung cancer})$$

2866 This approach has been applied to estimate adjustment factors for ovarian and laryngeal cancers
2867 using data from studies of chrysotile asbestos-only exposed workers or had minimal exposures to
2868 other forms of asbestos and reported findings for these cancer sites. The results from these
2869 analyses are presented in Tables 1 and 2. Based on this analysis, the adjustment factors are 1.04
2870 and 1.02 for ovarian and laryngeal cancer, respectively. A combined adjustment factor can be
2871 estimated by summing the individual adjustment factors and subtracting one, which results in an
2872 overall adjustment factor of 1.06.

2873 The studies included in Tables 1 and 2 were the only published studies that were of workers who
2874 were only exposed to chrysotile asbestos or had only minor exposures to other forms of asbestos,
2875 and that reported results for laryngeal or ovarian cancer. They were identified by reviewing the

2876 IARC report, other reviews ([Berman and Crump, 2008a](#); [Hodgson and Darnton, 2000](#)) and
2877 Stayner et al. ([1996](#)), and published meta-analyses for ovarian ([Camargo et al., 2011](#); [Reid et al.,](#)
2878 [2011](#)) and laryngeal cancer ([Peng et al., 2015](#)).

2879 Following is a brief description of the studies that were included in the estimation of the
2880 adjustment factors with the exception of the studies by Hein et al. ([2007](#)), Loomis et al. ([2009](#))
2881 and Wang et al. ([2013a](#)), which were previously described in Section 3.2.4. It is noteworthy that
2882 the number of cases of laryngeal and ovarian cancer observed in these studies are small, and the
2883 results for these sites are generally statistically unstable (*i.e.*, wide confidence intervals). For this
2884 reason, we have pooled the results from these studies in order to estimate the adjustment factors
2885 rather than relying on the results from individual studies alone.

2886 Acheson et al. ([1982](#)) conducted a cohort mortality study of women who were exposed to
2887 asbestos in manufacturing gas masks in 1939. One group of women manufactured masks
2888 containing chrysotile asbestos at a facility in Blackburn, England (n=570), and the other
2889 containing crocidolite at a facility in Leyland, England (n=757). Follow-up of these cohorts for
2890 vital status ascertainment was from 1951 to 1980. Mortality rates from England and Wales were
2891 used in a life-table analysis to compute expected numbers of death and standardized mortality
2892 ratios (SMRs). A statistically non-significant increase in ovarian cancer (SMR=1.48,
2893 95%CI=0.48-3.44) was observed among the women who manufactured masks using only
2894 chrysotile asbestos. Lung cancer mortality was also not significantly elevated in the chrysotile
2895 asbestos group (SMR=1.25, 95%CI=0.46-2.72⁴⁸). This study did not report results for laryngeal
2896 cancer.

2897 Gardner et al. ([1986](#)) conducted a cohort study of 2167 workers (1510 men and 657 women) who
2898 were employed sometime between 1941 and 1983 in a chrysotile asbestos cement products
2899 factory in England. The factory only used chrysotile asbestos except for a “small” amount of
2900 amosite during 4 months in 1976. Follow-up for ascertainment of vital status was through
2901 December 31, 1984. SMRs were estimated using a life-table with rates for England and Wales
2902 for the referent. There was little evidence of an increased risk of ovarian (SMR=1.11, 95%CI=
2903 0.23-3.25⁴⁹), laryngeal (SMR=0.91, 95%CI=0.02-5.07⁵⁰) or lung cancer mortality (SMR =0.97,
2904 95%CI=0.69-1.31) in this study.

2905 Newhouse and Sullivan ([1989](#)) conducted a cohort mortality study of workers employed between
2906 1941 and 1979 at a factory that produced friction products (*i.e.*, brake blocks, and brake and
2907 clutch linings). A total of 13,450 workers (9104 men and 4346 women) were followed for vital
2908 status ascertainment until 1986. The factory only used chrysotile asbestos except during two
2909 short periods when crocidolite was used. A slight excess of ovarian cancer (SMR=1.08, 90%CI=
2910 61-179), and a deficit of laryngeal cancer mortality (SMR=0.64, 90%CI=0.28-1.26) was
2911 observed in this study neither of which were statistically significant. Lung cancer mortality was
2912 not elevated in this cohort (SMR= 0.99, 95%CI=0.87-1.13).

2913 Tarchi et al. ([1994](#)) conducted a cohort mortality study of rock salt workers in Italy who were
2914 exposed to chrysotile asbestos. The study included 487 workers (367 men and 120 women) who

⁴⁸ Confidence interval was estimated using Fisher Exact method

⁴⁹ Confidence interval was estimated using Fisher Exact method

⁵⁰ Confidence interval was estimated using Fisher Exact method

2915 were employed in the mine sometime between 1965 and 1989 and followed for vital status
2916 ascertainment until the end of 1989. SMRs were estimated using lifetable methods with rates
2917 from the Tuscany region as the referent. An increase in ovarian cancer (SMR=4.76, 95%
2918 CI=0.57-15.73⁵¹) and laryngeal cancer mortality (SMR=1.35, 95%CI=0.03-7.53⁵²) but these
2919 findings were based on small numbers (2 cases of ovary and 1 case of laryngeal cancer) and were
2920 statistically non-significant. Lung cancer mortality was also increased but not statistically
2921 significant (SMR=1.46, 90%CI=0.79-2.48).

2922 Germani et al. (1999) conducted a cohort mortality study of 631 Italian women who were
2923 compensated for asbestosis and alive on December 31, 1979. The women were followed up for
2924 the ascertainment of vital status until October 30, 1997. SMRs were estimated using lifetable
2925 methods and national rates as the referent population. A statistically significant increase in
2926 ovarian cancer mortality was observed (SMR=4.77, 95%CI=2.18-9.06). Only one case of
2927 laryngeal cancer was observed in this study, which was an excess, but this was a highly unstable
2928 (SMR=8.09, 95%CI=0.21-45.08⁵³). A large and statistically significant excess of lung cancer
2929 mortality (SMR=4.83, 95%CI=2.76-4.84) was observed.

2930 [Liddell et al. \(1997\)](#) conducted a retrospective cohort mortality study of 11788 men who were
2931 born between 1891-1920 and had worked for at least one month in the chrysotile asbestos mines
2932 and mills in Quebec. Follow-up of the cohort for vital status ascertainment was through 1992. A
2933 small excess of laryngeal (SMR=1.11, 95%CI=0.78-1.54⁵⁴), and a modest but statistically
2934 significant increase in lung cancer mortality (SMR=1.37, 95%CI=1.27-1.48⁵⁵) was observed in
2935 this study. The study only included men and thus did not provide any results for ovarian cancer.

2936 [Malmo and Costa \(2004\)](#) conducted a cohort mortality study of 1653 former workers who were
2937 hired before 1971 in an Italian textile plant that only used chrysotile asbestos. The cohort was
2938 followed for vital status ascertainment through January 1, 1981. SMRs were estimated using life-
2939 table analyses and rates from residents of Turin who listed manual employment during a census
2940 in 1981 were used as the referent. Only one case of ovarian cancer was observed in this study
2941 which represented a small and statistically non-significant excess (SMR=1.28, 95%CI=.02-7.12).
2942 A statistically non-significant excess of laryngeal cancer was observed among males
2943 (SMR=4.44, 95%CI=0.90-12.97), but no cases were observed among females (the expected
2944 number was not reported). A statistically significant and relatively large increase in lung cancer
2945 mortality was observed in both males (SMR=3.02, 95%CI=1.89-4.57) and females (SMR=5.23,
2946 95%CI=2.10-10.79).

2947 [Pira et al. \(2017\)](#) conducted a retrospective cohort mortality study of 1056 men who were
2948 employed for at least one year in an Italian chrysotile asbestos mine between 1930 and 1990.
2949 Follow-up of the cohort for vital status ascertainment was through 2014. SMRs were estimated
2950 using lifetable methods and national rates for before 1981 and rates from the Piedmont region
2951 where the mine was located from 1981 onward were used as the referent. A statistically non-
2952 significant excess of laryngeal (SMR=1.58, 95%CI=0.68-3.11) and of lung cancer (SMR=1.16,

⁵¹ Confidence interval was estimated using Fisher Exact method.

⁵² Confidence interval was estimated using Fisher Exact method.

⁵³ Confidence interval was estimated using Fisher Exact method.

⁵⁴ Confidence interval was estimated using Fisher Exact method.

⁵⁵ Confidence interval was estimated using Fisher Exact method.

2953 95%CI=0.87-1.52) was observed. The study only included men and thus did not provide any
2954 results for ovarian cancer.

2955 **Uncertainty in Approach**

2956 An uncertainty related to this approach is that these adjustment factors are treated as a constant
2957 when in fact the ratio varies substantially between the studies. This variation may be just random
2958 but may be likely due to differences in study design or to levels of exposure to chrysotile
2959 asbestos.

2960 *Conclusions*

2961 The adjustment factor may be used to upwardly adjust the unit risk estimates for lung cancer to
2962 take into account the biases resulting from not including cancer sites other than lung cancer and
2963 mesothelioma.

Table_Apx M-1: Estimate of adjustment factor for ovarian cancer

1st Author (Year)	Lung Cancer ⁵⁶				Ovarian Cancer			
	SMR	Observed	Expected	Obs-Exp	SMR	Observed	Expected	Obs-Exp
Acheson et al. (1982)	1.25	6	4.8	1.2	1.48	5	3.4	1.6
Gardner and Powell (1986)	1.42	6	4.2	1.8	1.11	3	2.7	0.3
Newhouse and Sullivan (1989)	0.57	12	21.1	-9.1	1.08	11	10.1	0.9
Tarchi et al. (1994)	4.14	2	0.48	1.52	4.76	2	0.42	1.58
Germani et al. (1999)	4.83	16	3.31	12.69	5.26	4	0.76	3.24
Malmö and Costa (2004)	5.23	7	1.34	5.66	1.28	1	0.78	0.22
Hein et al. (2007)	2.22	61	27.48	33.52	0.62	6	9.68	-3.68
Loomis et al. (2009)	1.96	277	141.66	135.34	1.23	9	7.34	1.66
Wang et al. (2013a)	1.23	2	1.62	0.38	7.69	1	0.13	0.87
	Sum=	389	205.99	183.01	Sum=	42	35.31	6.69
			Adjustment Factor =1 .04					

⁵⁶ Lung cancer results are for women

Table_Apx M-2: Adjustment factor for laryngeal cancer⁵⁷

1st Author (Year)	Lung Cancer ⁵⁸				Laryngeal Cancer			
	SMR	Observed	Expected	Obs-Exp	SMR	Observed	Expected	Obs-Exp
Gardner and Powell (1986)	0.97	41	42.4	-1.4	0.91	1	1.1	-0.1
Newhouse and Sullivan (1989)	0.99	241	242.5	-1.5	0.64	6	9.4	-3.4
Tarchi et al. (1994)	1.46	10	6.84	3.16	1.35	1	0.74	0.26
Liddell et al. (1997)	1.37	646	471.5	174.5	1.11	36	32.43	3.57
Germani et al. (1999)	4.83	16	3.31	12.69	8.09	1	0.12	0.88
Malmo and Costa (2004)	3.36	29	8.62	20.38	4.44	3	0.68	2.32
Hein et al. (2007)	1.95	198	101.7	96.3	1.68	6	3.6	2.4
Loomis et al. (2009)	1.96	277	141.66	135.44	1.15	6	5.21	0.79
Wang et al. (2013a)	3.76	55	14.62	40.38	4.08	2	0.49	1.51
Pira et al. (2017)	1.16	53	45.5	7.5	1.58	8	5.1	2.9
	Sum=	1653	1101.07	552.03	Sum=	71	59.86	11.14
			Adjustment Factor =1.02					

⁵⁷ Foreign language study by Sun et al. (2003) that was not fully translated and was included as a sensitivity analysis, but the adjustment factor did not change. Only one case of laryngeal cancer was reported which was close to the expected value (SMR=1.01, 95%CI=0.14-7.17). Results for ovarian cancer were not reported. A statistically significant excess of lung cancer was observed in this study (SMR=3.88, 95%CI=3.14-4.79).

⁵⁸ Lung cancer rates are for men and women combined