

U.S. ENVIRONMENTAL PROTECTION AGENCY (EPA)
TOXIC SUBSTANCES CONTROL ACT (TSCA)
SCIENTIFIC ADVISORY COMM. ON CHEMICALS (SACC)

OPEN MEETING

DRAFT RISK EVALUATION FOR ASBESTOS

TSCA SACC WEBSITE:

<http://www.epa.gov/tsca-peer-review>

DOCKET NUMBER:

EPA-HQ-OPPT-2019-0501 (ASBESTOS)

PHONE AND WEBCAST

June 8-11, 2020

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NEXT STEPS 832

OPENING OF MEETING

MS. SARA WILSON: Good morning, everyone. We will start the meeting in just a moment. We'll let a couple more people log on. Thank you. Good morning. Welcome to the meeting of the U.S. EPA peer review of the draft risk evaluation for asbestos. Battelle is an EPA contractor providing meeting support for this series. This event is being recorded. Please be aware that the host may use WebEx chat to share announcements with all attendees, but attendees will not be able to respond to the chat. I will now introduce Dr. Diana Wong, the designated federal official.

DR. DIANA WONG: Thank you. Good morning. I am Dr. Diana Wong and I will be serving as the designated federal official to the U.S. EPA Toxic Substances Control Act Science Advisory Committee on Chemicals, TSCA SACC, for this meeting. I want to thank Dr. Portier for agreeing to serve as chair.

1 I also want to thank the members of
2 the committee, the ad hoc peer reviewers, and the
3 public for attending this important meeting. We
4 appreciate the time and effort of the peer
5 reviewers in preparing for this meeting, taking
6 into account your busy schedules. In addition, I
7 want to thank EPA's Office of Pollution Prevention
8 and Toxics and my colleagues on the TSCA SACC
9 staff for their hard work in preparing for this
10 important review of EPA's draft risk evaluation
11 for asbestos.

12 As an added note, Tamue Gibson and
13 Todd Peterson, they are my colleagues and they are
14 both DFOs and they will be online this week and
15 will serve as backups for my role as DFO. In
16 addition, Don Wood, Steve Knott our Executive
17 Secretary, and also Dr. Hayley Hughes, our Office
18 Director, have been working very hard to prepare
19 for this meeting.

20 Today, through Thursday the SACC
21 peer review will focus on asbestos. This is a
22 virtual meeting, meaning that audio is provided by

1 telephone or over your computer and that graphics
2 are presented by the WebEx online internet
3 platform. If for any reason, the WebEx platform
4 or audio transmission encounters any technical
5 difficulties you will find additional information
6 to refer to at our website, [www.epa.gov/tsca-peer-](http://www.epa.gov/tsca-peer-review)
7 [review](http://www.epa.gov/tsca-peer-review).

8 The TSCA SACC is a federal advisory
9 committee that provides independent scientific
10 peer review and advice to the EPA on chemical-
11 related issues regarding the impact of proposed
12 regulatory actions on human health and
13 environment. The TSCA SACC only provides advice
14 and recommendations to EPA. Decision making and
15 implementation authority remains with the Agency.

16 For the present meeting, there are
17 10 ad hoc peer reviewers and 15 of the 19
18 established SACC members are contributing to the
19 peer review of asbestos. As the DFO or the
20 Designated Federal Officer for this meeting, I
21 serve as a liaison between the TSCA SACC and the
22 Agency. I am responsible for ensuring provisions

1 of the federal advisory committee act, FACA, are
2 met.

3 TSCA SACC meetings are subject to
4 all FACA requirements. This includes open
5 meetings, timely public notice of meetings, and
6 document availability. Note: documents for this
7 meeting are located at the public docket at
8 www.regulations.gov. The docket ID is EPA-HQ-
9 OPPT-2019-0501.

10 As the designated official for this
11 meeting, a critical responsibility is to work with
12 appropriate Agency officials to ensure that all
13 appropriate ethics regulations are satisfied. In
14 that capacity, committee members receive training
15 on provisions of the federal conflict of interest
16 laws. In addition, each participant has filed a
17 standard government financial disclosure report.
18 Our deputy ethics official for the Office of
19 Science Coordination and Policy and in
20 consultation with the Office of General Counsel
21 have reviewed these reports to ensure all ethics
22 requirements are met.

1 For the next four days, we have a
2 very full agenda and meeting times are
3 approximate. Thus, we may not keep to exact times
4 as noted due to committee discussions and public
5 comment. We strive to ensure adequate time for
6 Agency presentations, public comments, and
7 committee deliberations. We may take a little
8 extra time at various points in the meeting to
9 help with coordination and thus work step by step
10 through the agenda.

11 For presenters, committee members,
12 and public commenters, please identify yourself
13 and speak into the microphone. This meeting is
14 being webcasted, transcribed, and recorded. One
15 added note, we highly recommend use of a landline
16 for those who are speaking as a committee member,
17 oral commenter, or Agency OPPT representative.
18 Copies of all EPA presentation materials and
19 written public comments are available in the
20 public docket at [regulations.gov](https://www.regulations.gov).

21 Copies of presentation materials
22 submitted this week by public commenters will be

1 available in the public docket within the next
2 week. Members of the committee are encouraged to
3 fully consider all written and oral public
4 comments submitted for this meeting. For members
5 of the public that have not pre-registered for
6 public oral comments, please notify either myself
7 or another member of the TSCA SACC staff if you're
8 interested in making a comment.

9 At this time, the agenda is full.
10 However, as we move through the proceedings if
11 time allows, we may be able to accommodate
12 additional brief comments of five minutes or less.
13 As I mentioned previously, there is a public
14 docket for this meeting. All background
15 materials, questions posed to the committee by the
16 Agency, and other documents related to this
17 meeting are available in the docket. Some
18 documents are also available on the EPA SACC
19 website. The docket number and website are noted
20 on the meeting agenda.

21 For members of the press, EPA media
22 relations staff are available to answer your

1 questions about this meeting. Please address all
2 questions to Ken Labbe. His email is
3 labbe.ken@epa.gov.

4 At the conclusion of the meeting,
5 the TSCA SACC will prepare a report as a response
6 to questions posed by the Agency, background
7 materials, presentations, and public comments.
8 This final report also serves as meeting minutes.
9 We anticipate the final report and meeting minutes
10 will be completed in approximately 60 days after
11 the meeting.

12 And again, for anyone joining us a
13 bit late I repeat, this is a virtual meeting,
14 meaning that audio is provided by telephone or
15 over your computer and that graphics are presented
16 by the WebEx online internet platform. If for any
17 reason the WebEx platform or audio transmission
18 encounters any technical difficulties you will
19 find additional information to refer to at our
20 website, www.epa.gov/tsca-peer-review. Again, I
21 wish to thank the committee for your
22 participation. I now turn the meeting over to our

1 chair, Dr. Portier.

2
3 **INTRO AND IDENTIFICATION OF PANEL MEMBERS**
4

5 **DR. KENNETH PORTIER:** Good morning.
6 Thank you, Dr. Wong. Am I coming in clear?

7 **DR. DIANA WONG:** Yes.

8 **DR. KENNETH PORTIER:** Thank you.
9 Okay. I want to start by thanking the chartered
10 committee and the experts we've invited to this
11 conversation for the hard work they've done in
12 preparing for this meeting. At this time I'm
13 going to identify -- we're going to call the roll
14 and have each of these members of the committee
15 identify themselves and give us a little bit of
16 background on them.

17 I'll start with myself. I'm Dr. Ken
18 Portier, I'm a retired biostatistician with 40
19 years of experience in applied statistics in
20 environmental and public health. And now I'll
21 start with the chartered committee. Dr. Henry
22 Anderson.

1 **DR. HENRY ANDERSON:** Good morning.

2 I'm Dr. Henry Anderson. I am a physician. A
3 specialist in occupational environmental medicine.
4 I am a retired state health officer and state
5 epidemiologist with the Wisconsin Division of
6 Public Health. And I'm currently an adjunct
7 professor at the University of Wisconsin School of
8 Medicine and Public Health in the Population
9 Health Department.

10 **DR. KENNETH PORTIER:** Thank you.

11 Dr. Charles Barton. I know Dr. Barton's been
12 having a little trouble getting signed in. We'll
13 come back to him. Dr. Bennett, Steve Bennett.

14 **DR. STEVEN BENNETT:** Good morning.

15 I'm Steve Bennett with the Household and
16 Commercial Products Association and also an
17 adjunct professor at Johns Hopkins University. I
18 provide expertise to the panel on consumer use and
19 exposure, and a chemist by training.

20 **DR. KENNETH PORTIER:** Thank you.

21 Dr. Sheri Blystone.

22 **DR. SHERI BLYSTONE:** Good morning.

1 Sheri Blystone. I am also a chemist by training.
2 Been working in the chemical industry as a product
3 safety and compliance practitioner for over 20
4 years. Currently with SNF Holding Company.

5 **DR. KENNETH PORTIER:** Thank you.

6 Dr. Deborah Cory-Slechta. Debbie, you're still
7 muted in WebEx.

8 **DR. DEBORAH CORY-SLECHTA:** Sorry
9 about that. Hi. This is Dr. Cory-Slechta. I'm a
10 professor of environmental medicine at the
11 University of Rochester Medical Center. My areas
12 of expertise are neurotoxicology both
13 developmental and neurodegenerative. Thank you.

14 **DR. KENNETH PORTIER:** Thank you.

15 Dr. Holly Davies -- Davies.

16 **DR. HOLLY DAVIES:** Hi. This is
17 Holly Davies from the -- I'm a toxicologist at the
18 Washington State Department of Health. I'm a
19 developmental biologist by training and I also
20 have expertise in people's exposures.

21 **DR. KENNETH PORTIER:** Thank you.

22 Dr. William Doucette.

1 **DR. WILLIAM DOUCETTE:** Hi. This is
2 Bill Doucette. I'm an environmental chemist and
3 professor in civil and environmental engineering
4 at Utah State University.

5 **DR. KENNETH PORTIER:** Thank you.
6 Dr. Concepción Jiménez-Gonzalez. I don't see Dr.
7 Gonzalez logged in this morning. Dr. Mark
8 Johnson.

9 **DR. MARK JOHNSON:** Hi. This is Mark
10 Johnson. I'm a director for toxicology at the
11 U.S. Army's Public Health Center where I've been
12 doing environmental toxicology and risk assessment
13 for about 25 years. Past president of the
14 American Board of Toxicology and a Fellow at the
15 Academy of Toxicological Sciences.

16 **DR. KENNETH PORTIER:** Thank you.
17 Dr. Alan Kaufman.

18 **MR. ALAN KAUFMAN:** Hi. Good morning
19 all. This is Al Kaufman. I'm currently Senior
20 Vice President of Technical Affairs for the Toy
21 Association, a general biologist by training. And
22 expertise is in downstream uses of chemicals and

1 manufacturing processes as well as some work on
2 consumer exposure. Thank you.

3 **DR. KENNETH PORTIER:** Thank you.

4 Dr. John Kissel.

5 **DR. JOHN KISSEL:** Hi. I am John
6 Kissel, Professor Emeritus of Environmental and
7 Occupational Health Sciences at the University of
8 Washington in Seattle. I am an environmental
9 engineer by training and a human exposure
10 scientist by practice.

11 **DR. KENNETH PORTIER:** Thank you.

12 Dr. Craig Rowlands.

13 **DR. CRAIG ROWLANDS:** Good morning.
14 I'm Craig Rowlands. I'm a Senior Toxicologist at
15 Underwriters Laboratories. I'm trained in
16 molecular toxicology and focus on carcinogenesis
17 and chemical mode of action.

18 **DR. KENNETH PORTIER:** Thank you.

19 Dr. Daniel Schlenk.

20 **DR. DANIEL SCHLENK:** Good morning.
21 Dan Schlenk, Professor of Aquatic and
22 Ecotoxicology at the University of California

1 Riverside training molecular toxicology expertise
2 and latent effects of chemicals on aquatic
3 organisms.

4 **DR. KENNETH PORTIER:** Thank you.

5 Dr. Sheela Sathyanarayana. I don't see Dr. Sheela
6 logged in yet this morning. Let me go back and
7 see if Dr. Barton's gotten lined on. Okay, we'll
8 move on to the ad hoc members. Dr. Kenny Crump.

9 **DR. KENNY CRUMP:** This is Kenny

10 Crump. I'm a mathematician by training. For the
11 past 40 years I've been involved in risk
12 assessments, quantitative risk assessments
13 primarily statistical methodology for conducting
14 quantitative risk assessment including asbestos.
15 And right now I don't have any professional
16 attachment.

17 **DR. KENNETH PORTIER:** Thank you.

18 Dr. Jeffrey Everitt.

19 **DR. JEFFREY EVERITT:** This is Jeff

20 Everitt. I'm in the school of medicine at Duke
21 University. I'm a veterinary pathologist. I've
22 had extensive experience in animal pathology

1 looking at particle and fiber-induced lung
2 disease.

3 **DR. KENNETH PORTIER:** Thank you.

4 Dr. Robert Herrick.

5 **DR. ROBERT HERRICK:** Hi. This is Bob
6 Herrick. I'm retired from the Harvard School of
7 Public Health. Prior to that, I was at NIOSH for
8 about 20 years. In both places, my research focus
9 was on exposure assessment for epidemiology.

10 **DR. KENNETH PORTIER:** Thank you.

11 Dr. Michael Jayjock. Dr. Jayjock, you're muted in
12 WebEx. Oh, you unmuted then muted again.

13 **DR. MICHAEL JAYJOCK:** Okay.

14 **DR. KENNETH PORTIER:** Unmute --

15 **DR. MICHAEL JAYJOCK:** All right.

16 I'm back. I'm back. Can you hear me now?

17 **DR. KENNETH PORTIER:** Yes.

18 **DR. MICHAEL JAYJOCK:** Hello? Can
19 you hear me?

20 **DR. KENNETH PORTIER:** Yes.

21 **DR. MICHAEL JAYJOCK:** Okay. Yeah.

22 Mike Jayjock here. I've been an exposure assessor

1 for about 40 years. 35 of those years I worked
2 for Rohm and Haas Company doing exposure and risk
3 assessment for chemical products. My primary
4 expertise is the modeling of exposure.

5 **DR. KENNETH PORTIER:** Thank you.

6 Dr. Marty Kanarek.

7 **DR. MARTY KANAREK:** Marty Kanarek,

8 I'm an epidemiology professor at the University of
9 Wisconsin Madison School of Medicine and Public
10 Health.

11 **DR. KENNETH PORTIER:** Thank you.

12 Dr. Steven Markowitz.

13 **DR. STEVEN MARKOWITZ:** Yeah, hi.

14 It's Steven Markowitz. I'm an Occupational
15 Medicine Physician with training in epidemiology.
16 I'm a professor at the Barry Commoner Center at
17 the City University of New York.

18 **DR. KENNETH PORTIER:** Thank you.

19 Dr. Elizabeth Sheppard.

20 **DR. ELIZABETH SHEPPARD:** Good

21 morning. This is Lianne Sheppard and I'm a
22 professor of both Biostatistics and Environment

1 and Occupational and Health Sciences at the
2 University of Washington.

3 **DR. KENNETH PORTIER:** Thank you.
4 Dr. Arti Shukla.

5 **DR. ARTI SHUKLA:** Hi. I am Arti
6 Shukla. I'm professor of Pathology and Medicine
7 at the University of Vermont. I'm a cell
8 biologist or cancer biologist and have been
9 studying asbestos in these cancers, mostly
10 mesothelioma and mechanisms for last -- almost
11 last 19 years.

12 **DR. KENNETH PORTIER:** Thank you.
13 Dr. Emanuela Taioli.

14 **DR. EMANUELA TAIOLI:** Good morning.
15 This is Emanuela Taioli. I'm the Associate
16 Director for Population Science at the Cancer
17 Institute at Mount Sinai School of Medicine in New
18 York. I'm a physician by training and a cancer
19 epidemiologist.

20 **DR. KENNETH PORTIER:** Thank you. Dr.
21 Bradley Van Gosen.

22 **MR. BRADLEY VAN GOSEN:** Hi. Brad

1 Van Gosen with the -- a research geologist with
2 the U.S. Geological Survey and I have expertise in
3 natural occurrences of asbestos.

4 **DR. KENNETH PORTIER:** Thank you.

5 Dr. Jiménez-Gonzalez. I see she's on the web, but
6 she hasn't dialed in yet. So we're missing Dr.
7 Barton and Dr. Sheela.

8 **DR. CHARLES BARTON:** This is Dr.

9 Barton --

10 **DR. KENNETH PORTIER:** Hello?

11 **DR. CHARLES BARTON:** -- can you
12 hear? Yes. This is --

13 **DR. KENNETH PORTIER:** Yeah. Yeah.

14 **DR. CHARLES BARTON:** Right.

15 **DR. KENNETH PORTIER:** Go ahead and
16 introduce yourself, Chuck.

17 **DR. CHARLES BARTON:** Okay. My name
18 is Chuck Barton. I am trained in toxicology. I
19 have experience in toxicology risk assessment and
20 I'm currently an independent consultant in
21 toxicology.

22 **DR. SHEELA SATHYANARAYANA:** And this

1 is Sheela. Can you hear me?

2 **DR. KENNETH PORTIER:** Ah, Dr.

3 Sheela. Good morning. Yes. Please introduce
4 yourself.

5 **DR. SHEELA SATHYANARAYANA:** I am Dr.

6 Sheela Sathyanarayana, and I'm an Associate
7 Professor of Pediatrics and Environmental and
8 Occupational Health Sciences at the University of
9 Washington and Seattle Children's Research
10 Institute. I focus on pediatric environmental
11 epidemiology.

12 **DR. KENNETH PORTIER:** Thank you. I

13 think that's the whole committee. Dr. Concepción,
14 when she connects her audio, we'll be able to
15 introduce her. At this point on the agenda, we
16 have scheduled welcome and introductory comments
17 by Dr. Alex Dapolito Dunn. Is Dr. Dunn on yet?
18 Yeah. I see you're muted in WebEx Dr. Dunn.
19 There you go. We should be able to hear you now.

20 **DR. ALEX DAPOLITO DUNN:** Can you all

21 --

22 **DR. KENNETH PORTIER:** We got you

1 now. Please proceed.

2 DR. ALEX DAPOLITO DUNN: Does it
3 sound okay?

4 DR. KENNETH PORTIER: Yes.

5 DR. ALEX DAPOLITO DUNN: All right.
6 Can I get some technical assistance and if my
7 computer's supposed to on, off or my phone?

8 DR. KENNETH PORTIER: Alex, I think
9 your computer should be off and you should be
10 talking on your phone because I think that's the
11 clearer -- the clearer message.

12 DR. ALEX DAPOLITO DUNN: Okay.
13 That's what I'm doing right now.

14 DR. KENNETH PORTIER: Perfect. No
15 reverb.

16
17 **WELCOME AND INTRODUCTORY COMMENTS**
18

19 DR. ALEXANDRA DAPOLITO DUNN. All
20 right. Well, good morning everyone. I'm Alex
21 Dunn. I'm the Assistant Administrator of EPA's
22 Office of Chemical Safety and Pollution

1 Prevention. I want to thank you for accommodating
2 this meeting so soon after our meeting in May on
3 Perchloroethylene.

4 Today we mark the beginning of the
5 peer review for the draft risk evaluation on
6 asbestos. And this is the last meeting on the
7 first 10 chemicals under TSCA. I want to
8 congratulate you all for this incredible
9 accomplishment. June 2020 also marks four years
10 since Congress passed the Lautenberg Act
11 Amendments to TSCA.

12 I know for some of you, this is your
13 10th SACC draft risk evaluation review and for
14 others, this may be your first. Regardless of how
15 many reviews you have participated in, your time
16 and efforts in discussing and providing feedback
17 on our draft risk evaluations is sincerely
18 appreciated. I want to say thank you on behalf of
19 everyone at EPA for stepping up to the task, for
20 your support this year, and for your extraordinary
21 efforts during the COVID-19 pandemic.

22 We have had so many important issues

1 facing our communities and nation and you have
2 remained steadfast to the task of the SACC. We
3 know that this meeting had to be rescheduled
4 because many of you are front line responders for
5 the COVID public health emergency. Again, the
6 fact that you've made time to be with us this week
7 is so humbling and appreciated.

8 Please know that we consider and
9 incorporate your recommendations on each of your
10 draft risk evaluations. The comments of the SACC
11 have provided us detailed guidance for making
12 improvements to the risk evaluations and to our
13 processes. We are focusing going forward on how
14 to improve our overall risk evaluation process and
15 the peer review experience.

16 EPA is committed to meeting our
17 obligations under TSCA. We are working
18 thoughtfully to review all the comments from the
19 public and our peer reviewers on the nine prior
20 draft risk evaluations and we'll finalize them
21 later this year. As you gather this week, we want
22 to hear your important advice and recommendations

1 on the draft risk evaluation for asbestos. We
2 have a large panel with both the chartered
3 committee and ad hoc reviewers, and we have an
4 incredible depth of expertise and experience.

5 As you may be aware, we have
6 recently received nominations for the charter
7 committee and in the near future, we will release
8 the names and the biological -- excuse me, the
9 biographical sketches of interested and available
10 nominees for public comment. Definitely don't
11 want to just be focused on those biographical
12 sketches. We have a robust pool of experts
13 including many of the current SACC members and
14 some ad hoc members to consider as we fill SACC
15 appointments over the next year.

16 On behalf of all of us again at
17 EPA, I extend our deepest gratitude to you for the
18 time and feedback you have given us over the last
19 two years as appointed panel members and as
20 selected ad hoc reviewers. Your commitment to
21 public service and to the American public is
22 highly commendable and your dedication and the

1 efforts with our staff have allowed us to
2 accomplish the significant Lautenberg Act
3 Amendment goals and bring forward TSCA's mission.
4 I thank you all for your great work.

5 Again, as we have mentioned, peer
6 review is the cornerstone of EPA's work. Your
7 efforts ensure that EPA uses the best science for
8 our decision making while honoring innovation and
9 fostering stakeholder feedback and input. Every
10 one of you is essential to EPA accomplishing our
11 mission of promoting chemical safety in this
12 country and beyond. We look forward to your
13 feedback on the draft risk evaluation for asbestos
14 as this week unfolds. We wish you great success
15 during this meeting and if you haven't heard it
16 already, we thank you from the bottom of your
17 hearts. Dr. Portier, back to you.

18 **DR. KENNETH PORTIER:** Thank you, Dr.
19 Dunn. Now we're going to move on to the technical
20 presentation by OPPT of the summary of the DRE.
21 And the lead on this Dr. Louis Scarano. Dr.
22 Scarano, would you introduce yourself and

1 introduce the other members of your team that may
2 be on this call that will provide backup to
3 committee questions as we move forward?
4

5 **OPPT TECH PRESENTATION - OVERVIEW RISK EVAL**
6

7 **DR. LOUIS SCARANO:** Yes. Thank you,
8 Dr. Portier. Can everyone hear me okay?

9 **DR. KENNETH PORTIER:** Yes. You're
10 coming --

11 **DR. LOUIS SCARANO:** Okay.

12 **DR. KENNETH PORTIER:** -- in clear.

13 **DR. LOUIS SCARANO:** Wonderful.
14 Thank you. First I'll address your first point.
15 On this first slide are all the asbestos workgroup
16 team members. And most of them are on the call
17 and will be available. I would like to point out
18 that the starred individuals are not available and
19 pretty much everyone else listed should be. I'd
20 also like to acknowledge the external asbestos
21 epidemiology experts, two of whom are on the call.
22 Doctors Dana Loomis and Leslie Stayner.

1 So now I'll just go to my formal
2 presentation if I may. Good morning to the
3 Scientific Advisory Committee on Chemicals, the
4 public commenters, and stakeholders. My name is
5 Louis or Gino Scarano and I'm the management lead
6 and the current acting team lead for the asbestos
7 team. I'm fortunate to be a part of this talented
8 group. This list is here, and I just called out
9 the ones that will not be on the call, but the
10 rest are. And also recognized the three external
11 experts, Doctors Loomis and Stayner who are
12 available and on the call.

13 Before I begin, I would like to
14 point out that this draft risk evaluation and the
15 associated supplemental documents were publicly
16 posted on March 30th of this year. The public
17 comment period closed on June 2nd and we'll be
18 hearing other public comments this afternoon and
19 perhaps later during the course of this peer
20 review meeting.

21 We at EPA have been reviewing the
22 comments that have come in, and there are many

1 substantive, detailed, and technical comments.

2 And we'll try and address and respond to some as
3 we -- we will address and respond to them as we
4 finalize this draft risk evaluation, but we will
5 identify and acknowledge a couple of them in this
6 presentation. But we would like to note that as
7 of this morning on the official docket only 27 of
8 the 75 substantive comments have been able to be
9 posted in the docket and are available publicly.

10 As I go through the presentation,
11 I'll call out the slide numbers to help everyone
12 follow along in case there are any connection
13 issues. This is slide two and it's an overview of
14 the presentation. We'll begin with general topics
15 on the regulatory history of asbestos. And then
16 we will then briefly describe the refining of the
17 scope of the draft risk evaluation as it moved
18 from the 2017 scope document to the 2018 problem
19 formulation, and now this 2020 draft risk
20 evaluation. And we are going to include how we at
21 EPA will address the recent court decision on
22 legacy issues.

1 This will then be followed by an
2 overview of the PCHEM properties, the Physical-
3 Chemical Properties of asbestos, the draft scope.
4 And then we'll move into the overview of the
5 technical assessment beginning with environmental
6 fate and transport, and moving into the
7 environmental risk assessment, and finally the
8 human health risk assessment. The asbestos draft
9 risk evaluation had a deeper focus on the human
10 health assessment portion. However, the
11 presentation does cover the environmental
12 assessment in accordance with the layout of the
13 document and the charge questions.

14 This is slide three. The next few
15 slides will present the regulatory history of
16 asbestos in the United States and briefly at the
17 international level. EPA would like to speak to
18 three of the TSCA specific regulations first.
19 First is title two of TSCA, which is the Asbestos
20 Hazard Emergency Risk Act or AHERA. This law
21 defines asbestos as the asbestos form variety of
22 six different types and requires local regional

1 districts to inspect school buildings for asbestos
2 and submit asbestos management plans to the
3 appropriate authority.

4 The second TSCA specific regulation
5 is in 1989 EPA issued the Asbestos Ban and Phase-
6 Out Rule under section six of TSCA at that time,
7 banning most asbestos-containing products. But in
8 1991 this rule was vacated and remanded by the
9 Fifth Circuit Court of Appeals and most of the
10 original ban on the manufacture, importation,
11 processing or distribution, and commerce, the
12 majority of the asbestos-containing product
13 originally covered in the 1989 final rule was
14 overturned. The partial 1989 asbestos ban
15 prohibits the uses of asbestos in products that
16 would be initiated for the first time after August
17 25th of 1989 and prohibits five other specific
18 product types that were no longer in use in 1989,
19 and they are listed here on the slide.

20 This is slide four and this is the
21 third important regulatory authority under TSCA
22 that has to do with asbestos and it's the 2019

1 significant new use rule or SNUR. Section 5(a)(2)
2 of TSCA authorizes EPA to determine that a use of
3 a chemical use of a substance is a significant new
4 use and a SNUR can be promulgated to regulate
5 either new chemicals or existing chemicals.

6 Once EPA determines that a use of a
7 chemical substance is a significant new use, then
8 TSCA section 5(a)(1) kicks in and requires
9 submission of a significant new use notice or a
10 SNUN. And EPA -- at least 90 days before the
11 substance can be manufactured, imported, or
12 processed for such use. So TSCA prohibits the
13 manufacturing, including importing the processing
14 from commencing until EPA had conducted a review
15 of that SNUN and made an appropriate determination
16 on the notice and taken such actions as are
17 required in association with that determination.

18 Slide five, so we issued our
19 proposed SNUR in June of 2018. It was finalized
20 on April 25, 2019, and it became effective on June
21 24th. The final rule identified as significant
22 new uses all the continued uses of asbestos not

1 subject to the partial 1989 asbestos ban, and uses
2 not identified as conditions of use subject to the
3 ongoing asbestos risk evaluation, which is the
4 current topic for this SACC. So this means that
5 because of the asbestos SNUR, manufacturing
6 including importing or processing for these
7 discontinued uses is prohibited unless and until
8 EPA conducts a thorough review of a SNUN or a
9 significant new use notice and puts in place any
10 necessary restrictions or approve its use.

11 Here I'd like to say that EPA
12 acknowledges public comments that we've received,
13 especially from the Asbestos Disease Awareness
14 Organization or ADAO who have claimed in their
15 comments that EPA missed ongoing importation and
16 associated uses of asbestos. But we have no
17 evidence of other uses and the customs and border
18 protection import data that we at EPA reviewed
19 included significant misreporting based on our
20 contacting the importers directly. EPA further
21 believes that the SNUR that I just talked about is
22 an important regulatory instrument because

1 manufacturing, including importing or processing
2 for uses not subject to the draft risk evaluation,
3 would have required notice to and review by us
4 here at the EPA and it would be a violation of the
5 SNUR not to do so.

6 Slide six, this is simply a list of
7 other non-TSCA regulations that are from EPA
8 pertains asbestos at EPA. The problem
9 formulations EPA identified exposure pathways and
10 other EPA environmental statutes and regulations
11 which address exposures and for which long-
12 standing regulatory and analytical processes
13 already exist, such as the Clean Air Act, the
14 Clean Water Act, NESHAP or the Asbestos National
15 Emissions Standards for Hazardous Air Pollutants,
16 the Safe Drinking Water Act, and the Resource
17 Conservation and Recovery Act. OPPT worked
18 closely with these EPA offices and determined that
19 asbestos falls under their jurisdiction of these
20 regulatory programs. OPPT believes that TSCA risk
21 evaluations should focus on those exposure
22 pathways associated with TSCA uses that are not

1 subject to the regulatory programs presented here.

2 This is slide seven. And this last
3 slide of the EPA regulations identifies other EPA
4 laws and regulations that pertain to asbestos.
5 The Comprehensive Environmental Response
6 Compensation Liability Act or CERCLA, the Asbestos
7 School Hazard Abatement Act, the Emergency
8 Planning and Community Right to Know Act, or
9 EPCRA, to which section 313 is the Toxics Release
10 Inventory or TRI, the Asbestos Information Act,
11 and the Asbestos Worker Protection Rule.

12 And this slide 8, it simply lists
13 the non-EPA federal regulations that exist on
14 asbestos. And the details for all of these are
15 provided in Appendix A in the draft risk
16 evaluation. These include OSHA, Occupational
17 Safety and Health Administration, the Consumer
18 Product Safety Commission or CPSC, the Food and
19 Drug Administration, FDA, Mine Safety and Health
20 Administration, MSHA, and the Department of
21 Transportation.

22 This is slide nine and this is the

1 last of the regulatory slides. So 39 states have
2 EPA approved model accreditation plan programs.
3 12 states have also applied to and received a
4 waiver from EPA to oversee implementation of the
5 Asbestos Containing Materials in Schools Rule
6 pursuant to a hearing I just talked about. All of
7 these details are also in Appendix A.

8 Asbestos is also regulated
9 internationally. Nearly 60 nations have banned
10 asbestos. Brazil banned it in 2017 and Canada in
11 2018. And both in Canada they are allowing the
12 use in chlor-alkali industry up to the year 2030
13 and the European Union is allowing -- has banned
14 asbestos and is allowing use in the chlor-alkali
15 industry until 2025.

16 Finally, I'd like to mention the
17 Rotterdam Convention. They are considering adding
18 chrysotile asbestos to Annex III in 2021. Annex
19 III is a list of the banned or restricted
20 substances for which a Prior Informed Consent or
21 PIC is needed prior to importing or exporting any
22 substance that's on the list. Annex III currently

1 lists five different asbestos substances;

2 actinolite, anthophyllite, amosite, crocidolite,

3 tremolite.

4 This is slide 10 and now we'll move
5 to the specifics of the draft risk evaluation.

6 Here we show the major changes that occurred in
7 the development of this draft risk evaluation.

8 First, we published our scope document in 2017.

9 And between the publication of the scope in 2017
10 and the publication of the problem formulation in
11 2018 we narrowed some of the conditions of use or
12 COUs, and there was the decision to focus on the
13 inhalation route of exposure and cancer as the
14 endpoint of concern.

15 In moving from the problem
16 formulation in June of 2018 to the release of the
17 draft risk evaluation in March, we derived a
18 commercial chrysotile specific inhalation unit
19 risk and there was a further narrowing of the COUs
20 or conditions of use. Here we just like to point
21 out that it's important to understand that
22 commercial chrysotile is not pure in a laboratory

1 as in a laboratory-grade highly pure substance.
2 Rather, it represents raw material that is mined
3 and processed for the specific COUs identified in
4 this draft risk evaluation and so it may have
5 small amounts of other amphibole fibers.

6 This is slide 11. The other major
7 issues that changed in the last few years since
8 the publication of the problem formulation is, we
9 published the SNUR, the Significant New Use Rule
10 in April and as I said, it became effective in
11 June. And then the most -- other most important
12 thing is, there was the decision by the U.S. Court
13 of Appeals in November of 2019 in the 9th Circuit
14 that stated that EPA must consider legacy uses.
15 And we will consider the asbestos legacy uses and
16 associated disposal in a subsequent scope and risk
17 evaluation.

18 Here again, we'd like to say a few
19 words about some public comments that we received
20 last week. Some comments made statements about
21 EPA taking years to review legacy uses. But now
22 that the court decision has been made, EPA is

1 committed to performing the scoping and risk
2 evaluation for legacy uses and follow as closely
3 as possible the amended TSCA timelines for risk
4 evaluations.

5 This is slide 12. And now we're
6 going to be getting into the technical parts of
7 the draft risk evaluation. Here we describe the
8 physical-chemical properties -- chemical
9 properties of chrysotile asbestos. As discussed
10 in section 1.4 of the draft risk evaluation, we
11 focused on chrysotile asbestos given EPA's
12 knowledge of the current conditions of use of
13 asbestos. This table here is adapted from table
14 1-1 in the draft risk evaluation and provides the
15 PCHEM properties for chrysotile asbestos.

16 EPA understands the importance of
17 the diameter and length of asbestos fibers in the
18 aspect ratio described a bit more later in the
19 draft risk evaluation. The values reported here
20 are ranges of diameter and length measurements
21 associated with chrysotile asbestos fibers. I
22 would like to note here that a new charge question

1 was added, charge question 1.2, about this very
2 topic based on discussions at the virtual meeting
3 that was -- the virtual pre-SACC meeting which was
4 held on April 7th. And some of you were present.

5 This is slide 13. In this slide, we
6 show the life cycle diagram which is figure 1-1 in
7 the draft risk evaluation. The color-coded
8 schematic shows, going from left to right the
9 importing, which is the gray boxes; the
10 processing, which is green; and the uses in blue;
11 and disposal in white, which represent what EPA
12 knows about the current uses of asbestos in the
13 United States at this time.

14 It is important to recognize that
15 raw asbestos is imported only for processing and
16 use for the chlor-alkali industry and there are
17 reports of the volume coming into the U.S. as
18 noted here. 750 metric tons were reported in 2019
19 and the reference that the U.S. Geological Survey.
20 And then in 2020 100 tons -- metric tons were
21 reported. All the other uses are for imported
22 products, not raw chrysotile asbestos and we do

1 not know the volume or amounts of those materials.

2 Slide 14. In this slide, we list
3 the general descriptors for the major categories
4 of the conditions of use or COUs for chrysotile
5 asbestos in the United States. The second column
6 to the right is the commercial or occupational,
7 and the last column are the consumer do-it-
8 yourselfers column.

9 So for the occupational scenario,
10 there are eight uses of diaphragms in the chlor-
11 alkali industry. The stamping of sheet gaskets in
12 chemical production, the use of sheet gaskets in
13 chemical production, and then the use in oil field
14 brake blocks, aftermarket automotive brakes and
15 linings, other vehicle friction products, in this
16 case specifically for cars for export only, and
17 the NASA Super Guppy use which is another vehicle
18 fit -- friction product. And then finally, number
19 eight, the use of other gaskets. You'll note that
20 for the consumer or DIY uses, it's the aftermarket
21 automotive brake lining and then the other
22 gaskets.

Slide 15. In the asbestos draft risk evaluation we've performed -- EPA performed distinct assessments for occupational including occupational non-users or ONUs, and consumers including bystanders for inhalation exposure scenarios. The workers and ONUs are exposed to asbestos, are considered potentially exposed or susceptible subpopulations. In addition, the evaluation of all life stages, children to the elderly are included in the consumer evaluation. Particularly the bystander scenario.

This is slide 16. We're now going to be moving into the environmental fate and transport section which is section 2.1 of the draft risk evaluation. Slide 17. Based on physical-chemical properties and environmental fate characteristics overall commercial chrysotile asbestos is a persistent mineral fiber, is largely chemically and biologically inert in the environment, and has low bioaccumulation potential in the environment. However, once inhaled or possibly ingested by an organism it would be

1 considered to be bio-persistent.

2 To address comments at the virtual
3 pre-SACC meeting on April 7th, EPA does want to
4 acknowledge that asbestos fibers have been shown
5 to accumulate in fish tissue and clams. But this
6 is different from bioaccumulation and
7 biomagnification up the food chain.

8 This is slide 18. And now the next
9 few number of slides are going to be getting into
10 the environmental risk assessment in the draft
11 risk evaluation. First, we're going to talk about
12 environmental release and exposure.

13 This is slide 19. After the problem
14 formulation was released, EPA gathered and
15 analyzed the reasonably available information on
16 the environmental releases of asbestos for surface
17 water. Very little information was located that
18 indicated release of asbestos to surface water.

19 Assuming asbestos disposal from the
20 conditions of use in this draft risk evaluation is
21 to an approved landfill, EPA noted in the problem
22 formulation section 2.5.3 there, that asbestos is

1 a fiber and is not likely to be leached out of a
2 landfill and into groundwater.

3 The next three slides will give
4 specifics about this -- the results of our
5 investigation. Slide 20. This table is adapted
6 from the table in Appendix D, and it's Table
7 Appendix D-2. Here we show the results of the
8 toxics release inventory data on asbestos.

9 And what we showed here are the
10 results of looking at the past four years of
11 reporting. We note that the -- there's no
12 releases to water and also note that the amount of
13 air releases is between approximately 150 and 300
14 pounds per year nationwide. Clearly, the vast
15 amount of the TRI releases to the environment is
16 to land and via land disposal.

17 This is slide 21. The results of
18 the EPA Office of Water six year review cycle data
19 for the last two cycles, six year reviews two and
20 three are presented here. Note that there's --
21 and this is from the Safe Drinking Water Act
22 requirements and these are data from 1998 to 2011.

1 Note the low number of detections, between 3 and 4
2 percent of all samples, and the even lower number
3 of samples with a detectable level above the
4 maximum contaminate level or MCL of 7 million
5 fibers per liter, less than 0.2 percent.

6 Importantly, it's extremely unlikely
7 that any of the residues are from a conditions of
8 use that's identified in this draft risk
9 evaluation. In December of 2016, the Office of
10 Water announced the progress for six year review
11 three and asbestos fell into the category of no
12 new information, national primary drinking water
13 regulation remains appropriate after review. And
14 so the six year review number four which is
15 currently underway and we have no specific
16 information on that at this time.

17 This is slide 22. The Clean Water
18 Act guidelines and standards do not require that
19 industrial facilities specifically monitor
20 asbestos concentrations in discharges. These
21 guidelines cover legacy uses such as the
22 manufacture of asbestos cement pipe, asbestos

1 cement sheet, roofing paper, et cetera and may not
2 be particularly useful to the COUs for asbestos in
3 this draft risk evaluation. There are effluent
4 guidelines for the chlor-alkali industry that
5 cover chlorine, mercury, and lead but they are not
6 specific to asbestos. And the EPA Industrial
7 Wastewater Treatment Technology database does not
8 currently include any data for asbestos.

9 Slide 23. So based on the
10 reasonably available information in the published
11 literature provided by industries using asbestos
12 and reported in EPA databases, there are minimal
13 or no release of asbestos to surface water
14 associated with the COUs that EPA's evaluating in
15 this draft risk evaluation. So now we're going to
16 go to the environmental hazard portion of the
17 environmental risk assessment.

18 This is slide 25 and it presents the
19 environmental hazard data for aquatic organisms
20 that were evaluated by EPA in this draft risk
21 evaluation. Acute exposure studies were performed
22 on invertebrates and chronic exposure data were

1 available for both vertebrates and invertebrates.
2 The data indicate that the chronic exposure to
3 water-born chrysotile asbestos at concentrations
4 equivalent to 0.01 to 100 million fibers per liter
5 may result in reproductive growth and or sublethal
6 effects to fish and invertebrates or clams.

7 In addition, acute exposure of
8 water-born chrysotile asbestos to a similar
9 concentration range demonstrated reduced siphoning
10 activity in clams. So now we'd like to go to the
11 environmental risk characterization. This is
12 slide 26 moving on to 27.

13 This diagram represents the
14 environmental exposure pathways and receptors that
15 we assessed in the draft risk evaluation. So EPA
16 determined that there were minimal or no releases
17 of asbestos by COUs included in the risk
18 evaluation because water releases associated with
19 the COUs are not expected and were not identified.
20 And EPA determined that there were no further
21 quantitative analyses necessary for exposure
22 pathways to terrestrial species based on the Clean

1 Air Act regulation of asbestos for the conditions
2 of use for asbestos.

3 So on slide 28, we present the
4 terrestrial pathway risk characterization. EPA
5 made refinements to the conceptual models during
6 the problem formulation stage that resulted in the
7 elimination of the terrestrial exposure pathway.
8 EPA did not include the emission pathways to
9 ambient air from commercial and industrial
10 stationary sources, or associated inhalation
11 exposure of terrestrial species because the
12 stationary source release of asbestos to ambient
13 air are addressed under the Clean Air Act.

14 Here on slide 29 is the risk
15 characterization for the aquatic species. Having
16 found there were minimal or no releases of
17 asbestos to surface water associated with the
18 conditions of use that were evaluated in this
19 draft risk evaluation, risks were not indicated
20 for aquatic or sediment-dwelling organisms.

21 In slide 30 we gave a brief synopsis
22 of some important assumptions and uncertainties

1 that were associated with the environmental risk
2 section along with a statement of the overall
3 confidence and strength of evidence that was made.
4 All of this is in section 4.3.2. Under
5 assumptions and uncertainties, we'd like to point
6 out that the available chronic exposure studies
7 that were available with fish and invertebrates or
8 clams.

9 There were limited data were
10 available on acute exposure to aquatic organisms
11 and there was only one short-term aquatic
12 invertebrate study. However, all the data were
13 considered high quality. And although there is
14 some uncertainty regarding extrapolation across
15 aquatic -- other aquatic species EPA considers the
16 overall uncertainty for the reasonably available
17 information -- environmental hazard data as low.

18 In terms of the environmental
19 exposure data, EPA concludes that there are
20 minimal or no releases of asbestos to surface
21 water associated with the conditions of use that
22 EPA is evaluating in this draft risk evaluation.

1 While this does introduce some uncertainty, EPA
2 views it as low to medium due to the limitations
3 of the existing data.

4 In terms of confidence and strength
5 of evidence overall, the strength of the evidence
6 is medium because of minimal or no exposure data
7 regarding potential environmental releases to
8 water for the conditions of use in this draft risk
9 evaluation. And this conclusion is also based on
10 the information that is currently in section 2.2
11 and summarized in section 4.3.2 of the draft risk
12 evaluation.

13 So our environmental risk
14 conclusions are on slide 31. Based on the
15 reasonably available information in the published
16 literature, provided by industries using asbestos,
17 and reported in the EPA databases there are
18 minimal or no releases of asbestos to surface
19 water and sediments associated with the COUs in
20 this draft risk evaluation. EPA preliminarily
21 concludes that there is no risk to aquatic or
22 sediment-dwelling environmental organisms.

1 This is slide 32 and we'll be moving
2 into the human health assessment portion of the
3 draft risk evaluation. First, we're going to talk
4 about the occupational exposure section, which is
5 section 2.3.1, and charge question two.

6 This is slide 33. There are four
7 main points or objectives that we'd like to point
8 out. And the first two are not going to be
9 presented later on but just want to make sure that
10 you're aware of the four objectives of the
11 occupational exposure section.

12 First, for the occupational exposure
13 scenarios, we provide a description of the
14 condition of use in terms of the process
15 description, disposal, description of the worker
16 activity, and an assessment of the potential
17 points of worker exposure. And this is all in
18 section 2.3.1.

19 Second, we estimate the number of
20 workers and occupational non-users for each of the
21 scenarios. Again, this will not be presented in
22 the presentation but it's in section 2.3.1. And

1 we'd like to make sure you understand that the
2 workers are potentially exposed employees who
3 directly handle asbestos. And occupational non-
4 users are potentially exposed employees who do not
5 directly handle asbestos but perform work in an
6 area where asbestos may be present.

7 Items three and four have to do with
8 the occupational inhalation exposure results. EPA
9 -- for -- EPA used air monitoring information both
10 area and personal breathing zone samples that were
11 provided by industry when available to assess
12 occupational inhalation exposures. And we also
13 considered worker exposure monitoring data
14 published in the open literature.

15 And finally, we did provide an
16 estimate of the inhalation exposure for each
17 scenario. We developed central tendency and high-
18 end estimates where possible for workers and ONUs.

19 This is slide 34 and it's simply a
20 schematic identifying the main approach used in
21 the occupational exposure assessment. We were
22 looking at the worker and the occupational non-

1 user, we're looking for chronic exposures and
2 inhalation was the only route of exposure that we
3 were interested in and investigated.

4 Slide 35. This slide presents the
5 general overview of the occupational exposure
6 assessment used in the asbestos draft risk
7 evaluation. For the concentrations, EPA reported
8 central tendency and high-end estimates for both
9 workers and occupational non-users where possible.
10 Both personal breathing zone, PBZ, and area
11 monitoring data where possible eight-hour time-
12 weighted averages from directly applicable
13 scenarios were used and the key sources were for
14 industry in the open literature.

15 All values that were reported -- all
16 values that we calculated are reported as eight
17 hour time-weighted averages and where the data
18 were available, short term monitoring data were
19 included separately as part as another eight-hour
20 shift scenario for occupational or ONU exposures.
21 And we called this short-term. And this topic of
22 short term is the subject of a charge question

1 we're seeking public comments and SACC advice on.

2 So this is slide 36 and the table
3 here shows each of the eight occupational
4 conditions of use exposure scenarios and what
5 monitoring or estimation method was used for both
6 workers and occupational non-users. Note that the
7 personal breathing zone values were used in seven
8 of the eight scenarios for workers and area
9 monitoring measurements were used for one. The
10 latter is -- an area monitor was used for only one
11 of the eight worker scenarios but were used for
12 many of the ONU scenarios.

13 Also in three of the scenarios,
14 three of the seven scenarios in which ONU
15 calculations were presented, the use of a
16 reduction factor was used to represent the
17 occupation non-user. EPA is particularly
18 interested in feedback from the SACC and the
19 public on this particular issue as well.

20 We'd like to note that the chlor-
21 alkali row that's highlighted in this slide. This
22 is because part of the public comments that we

1 received last week; the American Chemistry Council
2 notified EPA the monitoring data that they'd
3 submitted is duplicative of some of the data that
4 EPA subsequently received from individual
5 companies. So we are working through this and we
6 will remove duplications and update both the
7 exposure numbers and the risk numbers as we go
8 through that.

9 This is slide 37. As is done in
10 slide 30 for the environmental risk assessment
11 here we're just describing the assumptions,
12 uncertainty, and strength of evidence for the
13 occupational exposure data. So for assumptions
14 and uncertainties, most of the studies used in the
15 draft risk evaluation reported asbestos fiber
16 counts made by phase-contrast microscopy both for
17 occupational exposures in the COUs described and
18 in the studies used to support the derivation of
19 the chrysotile inhalation unit risk value.

20 Phase-contrast microscopy detects
21 only fibers longer than 5 microns and greater than
22 0.4 microns in diameter while transmission

1 electrons microscopy, or TEM often found in
2 environmental monitoring measurements, can detect
3 much smaller fibers. The representativeness of
4 the personal breathing zone and the area
5 monitoring data that we used is not known. When
6 there are few data points available, the exposure
7 estimates may not be representative of the
8 industry. And a third assumption that's important
9 is EPA grouped personal breathing zone and area
10 data to assess worker or occupational non-user
11 estimates.

12 Overall, EPA believes the strength
13 of evidence for occupational exposure estimates
14 vary from low, the oil field brake blocks exposure
15 scenario specifically, to high, the chlor-alkali
16 where there's a fair amount of data available and
17 was reviewed. The information on the strength of
18 evidence is presented in various parts of the
19 draft risk evaluation, specifically section 2.3.1,
20 Table 2-24, and the summary and section 4.3.3.

21 So now we're going to go into the
22 second part of the human health risk assessment.

1 And this is going to be the consumer exposure
2 part, section 2.3.2, and charge question three,
3 for which there are seven sub-questions. This is
4 slide 39 and here is an overview of the consumer
5 exposure approach. Consumers or do-it-yourselfers
6 or DIY or DIY mechanics and bystanders may be
7 exposed to commercial chrysotile asbestos under
8 two scenarios. When consumer repair or replace
9 aftermarket automobile brakes and linings or when
10 they're repairing or replacing gaskets in utility
11 vehicles or UTVs.

12 While peer-reviewed literature
13 indicates that much of the asbestos -- much of the
14 asbestos brake pad or shoe use has been phased out
15 and that the majority of existing cars on the road
16 do not have asbestos brakes, asbestos-containing
17 brakes and shoes can still be purchased in the
18 United States. Systematic review of the
19 reasonably available literature on brake repair
20 and replacement and gasket repair and replacement
21 resulted in insufficient inhalation information
22 for personal breathing zone or area monitoring,

1 specifically for the DIY consumer. Therefore, the
2 DIY brake and gasket repair/replacement scenarios
3 use surrogate monitoring data from occupational
4 studies.

5 This is slide 40 and is a schematic
6 that shows, as we did for the occupational
7 exposure route -- occupational exposure
8 evaluation, we're interested in the user or the
9 DIY, any bystanders that are present there
10 watching or being near the user doing the
11 activity, and we're interested in chronic
12 exposures, and inhalation as the route of
13 exposure.

14 Slide 41. This table shows each of
15 the three major consumer bystander COU exposure
16 scenarios and what monitoring or estimation method
17 was used. For the consumer, all three exposure
18 values used were based on personal breathing zone
19 measures adapted from occupational studies. For
20 the bystanders, two of the three scenarios used
21 area monitoring studies and again adapted from
22 occupational exposures. And the outdoor brake

1 scenario used a reduction factor approach. And
2 again, there we did in the residential -- in the
3 consumer exposure, changing the brakes indoors and
4 outdoors, and changing the gaskets.

5 This is slide 42. The following
6 assumptions are used to assess the consumer
7 inhalation exposure to asbestos during the DIY
8 repair or replacement for brakes and or gaskets.
9 There are four items here, the location, duration
10 of activity, the cleaning method and the frequency
11 of the repair job.

12 In terms of location, EPA presents
13 an indoor -- that's garage with the door closed --
14 and outdoor in a driveway scenario for brake
15 repair and replacement work. We only did indoor
16 for the gaskets.

17 In terms of duration of activity, a
18 typical brake or gasket repair job for a
19 professional brake mechanic for a single vehicle
20 takes between one and two hours. EPA assumed a
21 consumer do-it-yourselfer doing either brakes or
22 gaskets could take twice as long, or about three

1 hours.

2 In terms of cleaning methods, EPA
3 assumes for the indoor scenario that a consumer
4 may use compressed air to clean the brake
5 assemblies. EPA assumes for the outdoor scenario
6 that they do not use compressed air.

7 And finally, in terms of frequency
8 of repair jobs, EPA assumes the average consumer
9 performs a single brake or gasket repair job about
10 once every three years. We would like to note
11 that we did a sensitivity analysis in Appendix L,
12 and in that we also included scenarios when only
13 one brake or gasket job is performed in a
14 lifetime.

15 This is slide 43. Here we present a
16 few more specific assumptions about the consumer
17 DIY exposure assessment. EPA used survey data
18 from the EPA Exposure Factors Handbook to estimate
19 the median or 50 percentile, and the high-end or
20 95th percentile of amount of time that people
21 spend in a garage or outdoors on driveway
22 respectively.

1 And here are the values. According
2 to the handbook, indoor the median time is one
3 hour a day and it could be as long as eight hours
4 for the high users or the people who spend a lot
5 of time in the garage. And outdoors five minutes
6 and thirty minutes. That last column on the right
7 are the values that we used to estimate the
8 residual exposure to asbestos between jobs. These
9 are not based on the Exposure Factors Handbook but
10 are assumptions that we use to estimate daily
11 exposures between jobs.

12 To account for fibers that are
13 present, resuspended in air, or re-entrained. The
14 indoor figure is based on a 50 percent loss for
15 each of the three years between jobs resulting in
16 an average of 30 percent exposure. And the
17 outdoor figure is based on a 95 percent loss for
18 each of the three years for a 2 percent average.

19 This is slide 44 and here we present
20 the last slide to show some specific assumptions
21 used for two important parameters to estimate
22 exposure and risk to consumers and bystanders.

1 The age at start of exposure and the duration of
2 exposure. So a high and low-end bounding
3 assumption is presented for both the consumer,
4 DIY, and the bystander. As you can see, the
5 parameters that change are the duration of
6 exposure and the number of brake or gasket jobs
7 performed.

8 Slide 45. In this slide we use the
9 previous slide to show the bounding assumptions
10 and in this one, we're showing the different
11 scenarios we used in the sensitivity analysis that
12 was presented in Appendix L. And I think it's
13 important to point out the age at first exposure
14 here. The assumption is that someone would start
15 changing their brakes around age 16, that would be
16 the youngest, but as a bystander, any age could be
17 exposed. So there are the ages at first exposures
18 and the different scenarios for duration of
19 exposures depending on the scenario.

20 So this is slide 46. Similar to
21 some of the others you've seen, this has to do
22 with assumptions, uncertainty, and strength of

1 evidence for the consumer exposure piece. So
2 under assumptions and uncertainties, we'd like to
3 point out, EPA used personal breathing zone and
4 area monitoring data from occupational studies as
5 a surrogate for consumer bystander exposure for
6 both the aftermarket auto -- the aftermarket brake
7 and the gasket repair scenarios.

8 And this introduces uncertainty, for
9 example in the physical setting, in the hours of
10 work, and in the equipment used. Although this
11 presents some uncertainties that could
12 overestimate exposures using the physical setting,
13 for example, and the volume of work, and large
14 workspace with multiple vehicles on the
15 occupational side, which is not present in -- for
16 the resident -- the consumer.

17 But this uncertainty was offset by
18 using data under certain environmental conditions
19 expected to be more representative of a do-it-
20 yourselfer, such as no engineering controls, no
21 personal protective equipment, and the fact that
22 it's a residential garage. And we feel that by

1 doing the sensitivity analysis, varying the age at
2 start of exposure and duration of exposure that
3 was helpful in handling uncertainties.

4 So for the strength of evidence for
5 the consumer exposure estimates for both the DIY
6 and the consumers, we figured -- we believe it's
7 low to medium for the aftermarket auto brakes and
8 lining scenario and medium for the other gasket
9 scenario. All this information is presented in
10 sections 2.3.2.1.4, 2.3.2.2.3, Table 2-32, and
11 summarized in Table 4 and section 4.3.4

12 Now we're going to get into the
13 human health hazard characterization portion of
14 the human health assessment which is section 3.2
15 and charge question 4. Moving to slide 48. This
16 is the first of two general overview slides. The
17 human health hazard was identified in the problem
18 formulation and focused on cancer by the
19 inhalation route. The existing EPA inhalation
20 unit risk, or IUR, for asbestos, was developed in
21 1988 and was based on studies that included
22 occupational exposure to chrysotile, amosite, or

1 mixed mineral exposures including chrysotile,
2 amosite, and crocidolite.

3 This draft risk evaluation indicated
4 that only chrysotile asbestos is currently being
5 imported in the raw form or imported in products
6 into the United States. So the studies of
7 population exposed only to commercial chrysotile
8 provide the most informative data to develop a
9 TSCA risk estimate for the conditions of use for
10 chrysotile asbestos in this draft risk evaluation.
11 Again, we'd like to point out that commercial
12 chrysotile is not pure chrysotile but is the raw
13 material that's mined and may have small amounts
14 of other amphibole fibers.

15 Slide 49. In this second general
16 slide, we'd like to point out that in developing
17 the draft risk evaluation there were early
18 discussions about evaluating cancer and non-cancer
19 effects following inhalation exposures. A
20 discussion and rationale to focus on cancer only
21 is presented in section 2.4.2 in the 2018 problem
22 formulation. In this slide, we paraphrase that

1 narrative because the problem formulation presents
2 this conceptually but here, we're showing it
3 quantitatively.

4 For the high-risk inhalation unit
5 risk or IUR for general asbestos that was
6 developed in 1988 is 0.23 per fiber per cc. The
7 IRIS assessment for Libby amphibole asbestos
8 derived a reference concentration or an RFC for
9 non-cancer health effects and that concentration
10 is 9×10^{-5} fibers per cc. The risk of cancer for
11 general asbestos fibers was 2×10^{-5} . Thus the
12 cancer IUR was protective of the non-cancer
13 effects for Libby and EPA extrapolated that for
14 chrysotile for this draft risk evaluation with use
15 of the benchmark of 1×10^{-6} or 1 in a million for
16 consumers and bystanders.

17 But we realize that for the
18 occupational setting EPA has a different benchmark
19 of 1 cancer per 10,000. So at this risk level if
20 the non-cancer effects for chrysotile are similar
21 to Libby amphibole asbestos, the non-cancer
22 effects of chrysotile are likely to contribute

1 additional risk to the overall risk of asbestos
2 beyond the risk of cancer.

3 This is slide 50. And this is just
4 a short synopsis of a small section in the draft
5 risk evaluation that addresses the cancer mode of
6 action. So the International Agency for Research
7 on Cancer or IARC has proposed a mechanism for the
8 carcinogenicity of asbestos fibers and it's in
9 3.2.3.1 of the draft risk evaluations. Asbestos
10 fibers may lead to oxidant production to
11 interaction with macrophages and through hydroxyl
12 radical generation. Inhaled fibers that are
13 phagocytosed by macrophages may be cleared or lead
14 to frustrated phagocytosis which results in
15 macrophage activation, release of oxidants, and
16 increased inflammatory response.

17 Research on various types of mineral
18 fibers supports a complex mechanism involving
19 multiple biological responses to harming exposure
20 to asbestos. That is genotoxicity, chronic
21 inflammation, cytotoxicity leading to the oxidant
22 release and cellular proliferation in the

1 carcinogenic response to mineral fibers.

2 Now onto slide 51. And this marks
3 the first of five slides specific to the
4 derivation of the inhalation unit risk. I would
5 like to acknowledge the three asbestos
6 epidemiology experts who helped with this effort.
7 Drs. Leslie Elliot, Dana Loomis, and Leslie
8 Stayner. Drs. Loomis and Stayner are on this
9 call. Dr. Elliot could not make it.

10 This slide briefly addresses the
11 systematic review process used on the epidemiology
12 cohorts and studies and how this is slightly
13 different than what was done with the other risk
14 evaluations' systematic review process. Using the
15 TSCA systematic review process that was adapted to
16 asbestos exposure and outcomes, EPA identified
17 several studies of occupational cohorts exposed
18 only to partial chrysotile that were of adequate
19 overall study quality.

20 With the assistance of the experts
21 in asbestos epidemiology, EPA conducted additional
22 evaluations of exposure and outcome ascertainment

1 in these cohorts with particular attention to the
2 suitability for dose-response estimation. And the
3 three things that we looked for were exposure to
4 the commercial chrysotile, the assessment of
5 exposure, and the mesothelioma ascertainment.

6 Slide 52. As described in section
7 3.2.4, five different cohorts were evaluated to
8 determine the appropriate group from which to
9 derive a chrysotile specific IUR for this draft
10 risk evaluation. Using a published method to
11 adjust for mesothelioma under ascertainment, the
12 range of candidate lung cancer and mesothelioma
13 IURs was between 0.08 and 0.32 per fiber per cc.
14 The North Carolina and South Carolina cohorts were
15 selected based on high quality for developing the
16 IUR. And the range of lung cancer and
17 mesothelioma IURs was between 0.08 and 0.16 per
18 fiber per cc.

19 There were two identified downward
20 biases that were considered. Number one was the
21 lack of available data from other cancer mortality
22 due to laryngeal and ovarian cancers that have

1 been tied to asbestos exposure, and the use of
2 cancer mortality data rather than cancer incidence
3 data. Although this may be a small uncertainty
4 due to the low survival rates especially from
5 mesothelioma.

6 Slide 53. This slide is from Table
7 3-13 in the draft risk evaluation and shows the
8 chosen IUR. Note that the value takes the lung
9 cancer results from the South Carolina cohort and
10 the mesothelioma results from the North Carolina
11 cohort to arrive at the combined lifetime IUR of
12 0.16 per fiber per cc. Now, the definition of the
13 IUR is for a lifetime of exposure.

14 For the estimation of lifetime risk
15 for each condition of use, the partial lifetime or
16 less than lifetime IUR has been calculated using
17 the life table approach and the values for
18 different combinations of age at first exposure
19 and duration of exposures. And they're presented
20 in Appendix K. The approach and calculations of
21 life tables are in Appendices H and I.

22 Slide 54. Again, the assumption,

1 uncertainty, and strength of evidence slide.

2 Under assumptions and uncertainties we'd like to
3 point out for the exposure assessment part, there
4 was low uncertainty for the North Carolina and
5 South Carolina cohorts used for the IUR
6 derivation. Second, there was an underestimation
7 of risk to all cancers because of the lack of
8 being able to use the laryngeal and ovarian cancer
9 information. And there was an underestimation of
10 risk due to being based on mortality versus
11 incidence.

12 So overall the strength of the
13 evidence for the development of the IUR is high
14 and the range of possible IURs was between 0.08
15 and 0.16. And the choice of the higher value was
16 used to offset the uncertainties that were
17 identified above. And all of this is described in
18 detail in sections 4.3.5 and 4.3.6.

19 So finally we're getting to the
20 human health risk characterization part, which is
21 section 4.2 and the charge question five, of which
22 there are five subset questions. So moving to

1 slide 56. This schematic shows the human health
2 populations for which EPA has developed
3 quantitative risk numbers from exposure to
4 chrysotile asbestos under the conditions of use
5 described previously.

6 As you can see, the left-hand side
7 of the table shows the four main groups, workers,
8 occupational non-users, consumers, and bystanders,
9 and that the route of exposure evaluated is
10 inhalation. On the right hand, part of the slide
11 are the equations used. And some important
12 caveats, for the worker and occupational non-user
13 scenarios, risk assessments were provided with and
14 without personal protective equipment or PPE,
15 risks were not aggregated across exposure sources
16 or routes, and risk for consumers and bystanders
17 did not assume the use of personal protective
18 equipment.

19 Slide 57. This slide and the next
20 three present specific information on important
21 methodological issues that we -- that was used in
22 the asbestos risk calculations. First, is about

1 size and measurements of asbestos fibers. Work
2 has been done on differential toxicity by fiber
3 length. However, there's some lack of consistency
4 in those results. Earlier research Berman and
5 Crump 2008 emphasize higher toxicity for longer
6 fibers in North Carolina cohorts. Recently, using
7 Bayesian methods for highly coordinated exposures
8 across fiber groups, Hamra et al show that for the
9 Carolina cohorts there is toxicity for chrysotile
10 asbestos across the entire range of fiber lengths.

11 The third point is that phase-
12 contrast microscopy is the only data available for
13 chrysotile IUR including both lung cancer and
14 mesothelioma. Phase-contrast microscopy counts
15 all fibers greater or equal to five microns with a
16 three to one aspect ratio and does not allow for
17 evaluation of differential toxicity. While there
18 is data using transmission electron microscopy
19 information for modeling lung cancer there is no
20 TEM data for modeling mesothelioma, at least in
21 the North Carolina and South Carolina cohorts.

22 So a derivation of a TEM based IUR

1 was not possible. But what was possible was to
2 look across different industries which may have
3 differences in fiber lengths. In Table 3-8 EPA
4 compared the lung cancer toxicity values for
5 textile versus mining industries and found they
6 substantially overlapped which did not indicate
7 major differences in toxicity.

8 This is slide 58 and here we'd like
9 to talk about the respiratory personal protective
10 equipment information that's specific for
11 asbestos. First, worker personal protective
12 equipment use assumes proper training, fitting,
13 and use during the activity. However, there are
14 reports specific to the use of PPE and asbestos
15 and assigned protection factors with asbestos, and
16 we're referencing Riala and Riipinen in here. And
17 we used the term assigned protection factors, they
18 used the term nominal.

19 Riala and Riipinen investigated
20 performance of respirators and HEPA units in 21
21 different exposure abatement scenarios. And most
22 had very high exposures that we did not consider

1 consistent with the conditions of use identified
2 in this risk evaluation. But the important point
3 in this study was, they measured air concentration
4 inside and outside the worn PPE to quantify the
5 protection factor for comparison to the assigned
6 or nominal protection factor.

7 For three of the scenarios relevant
8 to the conditions of use in this draft risk
9 evaluation, meaning it was lower exposures below 1
10 fiber per cc, the measured protection factors were
11 reported as 50, 5, and 4. The results demonstrate
12 that while some workers have protection above the
13 nominal APF, some workers have protection below
14 the nominal APF. So even with every worker wearing
15 a respirator, some would not be protected.

16 This is slide 59. And here we'd
17 like to speak for a moment on air volume
18 adjustments and the risk calculations. In the
19 derivation of the IUR, the cancer potency values
20 were obtained from occupational cohorts. These
21 values were then adjusted for differences in air
22 volumes between the workers and other populations

1 so that those values could be applied to the U.S.
2 populations as a whole in standard EPA life-table
3 analyses, they derived the IUR for lifetime
4 exposures. However, for the occupational exposure
5 scenarios, this adjustment was not made. It was
6 reversed back to the worker's air volume.

7 Slide 60. Here we'd like to
8 introduce you to the use of the life tables. So
9 the cancer potency values for lung cancer and
10 mesothelioma were used to estimate an exposure
11 concentration associated with a one percent extra
12 risk of each cancer mortality caused by chrysotile
13 asbestos. Each cancer-specific unit risk is equal
14 to one percent divided by the exposure
15 concentration that yielded that extra risk.

16 Partial lifetime unit risks were
17 also derived for a range of exposure scenarios
18 based on different ages at first exposure and
19 different durations of exposure. This life table
20 approach is based on one reported in the NRC
21 report in 1988 for evaluating lung cancer risk for
22 radon. And it's a standard methodology used in

1 multiple published EPA risk evaluations.

2 Equations are detailed in Appendix H, and the SAS
3 codes are provided in Appendix I.

4 Slide 61. In this slide, we've
5 shown a handful of rows of the results of using
6 the life tables to identify partial lifetime IUR
7 values. They show the different ages at first
8 exposure and duration of exposure all in years.
9 The highlighted IUR values are the ones that were
10 used most often in the draft risk evaluation and
11 that's why they're presented here. This again is
12 in Appendix K.

13 This is slide 62. This is an
14 example of part of the occupational risk
15 characterization table for two of the COUs. This
16 is adapted from Table 4-38. As you can see, the
17 two COUs that we point -- that we present here are
18 the use of diaphragms in the chlor-alkali industry
19 and the stamping of sheet gaskets. The results
20 show risk estimates above the benchmark of 1×10^{-4}
21 for most of the worker scenarios without the use
22 of personal protective equipment. And these were

1 all shaded in pink.

2 Wherever the benchmarks are not
3 exceeded for many -- whereas for many -- I'm
4 sorry. I have to take a drink of water. Thank
5 you. Whereas the benchmarks are not exceeded for
6 many of the occupational non-user scenarios, I
7 would like to point out that the exposure duration
8 level column denotes an eight-hour time-weighted
9 average for central tendency and high-end
10 exposures. And the designation short term refers
11 to the incorporation of short-term monitoring
12 information into a separate eight-hour time-
13 weighted average.

14 As noted in slide 36, part of the
15 public comments that we received last week from
16 the ACC, we will be removing duplicate numbers
17 from the chlor-alkali information and that might
18 change the exposure and risk numbers in the final
19 evaluation.

20 Next is slide 63 which is a similar
21 table for the consumer risk characterization. And
22 this comes from, is adapted from Table 4-48 in the

1 draft risk evaluation. Here the estimates for
2 both the DIY consumer, the do-it-yourselfer, and
3 the bystanders are presented for the brake
4 repair/replacement only. Again, the pink denotes
5 risk. And in this case the benchmark, the risk
6 above the benchmark, and in the case for consumers
7 and bystanders, the benchmark is 1×10^{-6} . And both
8 central tendency and high-end exposures are
9 evaluated. Results showed there's only one
10 scenario beneath the benchmark and that's the
11 outdoor one.

12 This is slide 64 and here again, we
13 point out the assumptions, the uncertainty, and
14 the strength of evidence for the risk
15 characterization. For the assumptions and
16 uncertainty, we have four we'd like to point out.
17 First, given the high confidence in the phase-
18 contrast microscopy data and the large number of
19 analytical measurements, the exposure uncertainty
20 is overall low in the South Carolina and North
21 Carolina cohorts.

22 The epidemiology studies are

1 observational and as such are potentially subject
2 to bias and confounding errors, but that both the
3 Carolina cohorts did not have information to
4 control for cigarette smoking, which is important
5 for lung cancer but not for mesothelioma.
6 However, this bias is believed to be small because
7 the exposure-response analyses for lung cancer
8 were based on internal comparisons.

9 And for both studies, the regression
10 models included birth cohorts thus introducing
11 some control for the changing smoking rates over
12 time. And for the purpose of combining risks, it
13 is assumed that the unit risks of mesothelioma and
14 lung cancer mortality are normally distributed.
15 EPA believes the strength of evidence for the
16 human health risk characterization is high and
17 it's presented in section 4.3.7 and Appendix L
18 presents a sensitivity analysis.

19 Slide 65. In this slide and the
20 next, we briefly describe how the draft risk
21 evaluation addresses potentially exposed or
22 susceptible subpopulations or PESS. EPA

1 identified groups that have greater exposure than
2 the general population. Workers and occupational
3 non-users are adults including women of
4 childbearing age and adolescents. And for
5 consumers and bystanders, adults and children
6 greater than 16-year-olds as users but all ages as
7 bystanders.

8 And then on the susceptibility side,
9 EPA identified groups having greater
10 susceptibility from the general population as
11 lifestyle factors such as smoking history for lung
12 cancer, variability in physiological factors
13 across life stages such as breathing rates and
14 tidal volume, and the age at first exposure which
15 is very important for the asbestos evaluation.

16 Slide 66. In accounting for PESS
17 and risk characterization, we'd like to say a few
18 words about the specific to smoking as a lifestyle
19 factor. Adverse effects -- it is theoretically
20 possible that the risk of estimated lung cancer
21 mortality reflects a positive synergy between
22 asbestos -- between smoking and asbestos.

1 Adverse effects of chrysotile
2 asbestos on lung cancer among the potentially --
3 potentially non-smoking workers may have been
4 overestimated. However, the majority of cancer
5 risk comes from mesothelioma, not lung cancer and
6 we point to Table 3-12 in the document.
7 Mesothelioma is not related to smoking, so the
8 combined overall cancer IUR would then be health-
9 protective for any population that have a lower
10 prevalence of smoking than that of the worker
11 cohorts.

12 Slide 67. This is our final human
13 health risk summary slide. So risks are indicated
14 when estimates are above the cancer risk benchmark
15 which is 1×10^{-4} for workers and occupational non-
16 users, and 1×10^{-6} for consumers and bystanders. In
17 terms of inhalation exposure for workers and
18 bystanders, all worker scenarios -- risk for all
19 worker scenarios for both central tendency and
20 high end without personal protection equipment and
21 for some even with personal protective equipment,
22 are above the benchmark.

1 Risk for all occupational non-user
2 scenarios for high-end exposures only and some
3 central tendency and high-end exposures are above
4 the benchmark. In terms of inhalation exposures
5 to consumer users and bystanders, risks for all
6 indoor scenarios for both the consumers and
7 bystanders, both central tendency and high end are
8 above the benchmark.

9 Risk for some outdoor scenarios for
10 consumers for the high-end time spent outdoors
11 only are above the benchmark. Risk for some
12 scenarios are below the benchmark. The brake
13 replacement/repair scenarios for consumers or
14 bystanders with a median time spent outdoors both
15 central tendency and high end.

16 On this last slide, slide 68, I'd
17 like to close the presentation by bringing forward
18 the risk characterization considerations --
19 considerations per the procedures for chemical
20 risk evaluation under the amended Toxic Substance
21 Control Act. So when considering risk EPA will
22 number one, integrate the hazard and exposure

1 assessments into quantitative and or qualitative
2 estimates of risk for the identified populations,
3 including the potentially exposed susceptible
4 subpopulations.

5 We'll describe whether aggregate or
6 sentinel exposures under the conditions of use
7 were considered and the basis for their
8 consideration. We would not -- we will not
9 consider cost or other non-risk factors. We'll
10 take into account, where relevant, the likely
11 duration, intensity, frequency, and number of
12 exposures under the conditions of use of the
13 chemical substance. And finally, describe the
14 weight of the scientific evidence for the
15 identified hazards and exposures. With that, I'd
16 like to close the presentation and I thank you for
17 your attention.

18 **DR. KENNETH PORTIER:** Thank you, Dr.
19 Scarano. At this point, we're going to take a 15
20 minute break to have the committee prepare any
21 questions on this presentation. I'm hoping to
22 keep the committee discussion to closer to 45

1 minutes than the hour and 15 minutes we have
2 specified. So let's take a break until 12 noon
3 eastern time at which point, we'll reconvene and
4 take questions from the panel. Thank you.

5
6 **[Break]**

7
8 **DR. KENNETH PORTIER:** Let's
9 reconvene, please. Soundcheck is -- is there
10 anybody hearing me?

11 **MR. MARTIN ALVARADO:** Yeah. We hear
12 you fine, Ken.

13 **DR. KENNETH PORTIER:** Thank you.
14 Just checking. Before we get into the questions,
15 I wanted to introduce Dr. Concepción Jiménez-
16 Gonzales who was having a little technical
17 problems when we ran the roll this morning. Dr.
18 Gonzales, please introduce yourself.

19 **DR. CONCEPCIÓN JIMÉNEZ-GONZALEZ:**
20 Dr. Portier, can you hear me?

21 **DR. KENNETH PORTIER:** Yes. We can.

22 **DR. CONCEPCIÓN JIMÉNEZ-GONZALEZ:**

1 Finally. Yeah, just for the record I have been in
2 the call all the time. Just got to disconnect and
3 reconnect a few times to make this work. So thank
4 you for the assistance. So I am Concepción
5 Jiménez-Gonzalez. I am a chemical engineer by
6 training. I work in GlaxoSmithKline leading the
7 environmental health safety and sustainability
8 support for R and D, and I'm also an adjunct
9 professor at North Carolina State University. My
10 specialty is in environmental engineering and risk
11 assessment.

12
13 **SACC DISCUSSION ON OPPT TECH PRESENTATION**

14
15 **DR. KENNETH PORTIER:** Thank you. I
16 want to point out to the committee I misspoke. We
17 have about an hour for questions to EPA on the
18 slides and the DRE. I'd just remind the committee
19 at this point it's clarifying questions, we're not
20 going to get into the discussion of the questions
21 that EPA has presented to us. This time it's just
22 comment, clarifying questions. Please remember to

1 use the hands up functionality in WebEx so I can
2 identify who wants to ask questions. And I see
3 Dr. Schlenk has his hand up. Dan.

4 **DR. DANIEL SCHLENK:** Yeah. Thanks,
5 Ken. I've actually got three questions so I'll
6 just kind of go one by one through this. The
7 first has to do with -- and all these have to do
8 with the surface water conclusions, I guess, which
9 will be addressed a little bit later in the
10 question. But, just based on the presentation and
11 the documents that I looked through, the question
12 I have -- and this is something that actually has
13 been, you know, it's been sort of proposed for the
14 other nine compounds I think that we've done, and
15 that's the addition of -- requirement for
16 monitoring data.

17 And then, with NPDES permits, I'm
18 just curious, it's my understanding that those are
19 regulated at the local and state level. And I'm
20 wondering, does OPP have the authority or can they
21 actually require industry for monitoring for
22 specific agents to be placed on that? And if so,

1 how long does it take to get that permit made, I
2 guess, or that request to me made such that they
3 can get data? What sort of timeframe are we
4 talking about?

5 **DR. KENNETH PORTIER:** Dr. Scarano or
6 anyone on the EPA team?

7 **DR. LOUIS SCARANO:** This is Gino
8 Scarano. I don't know the answer to that. I
9 don't know if any of my team members do but thank
10 you for the question. So I'll see if someone else
11 can answer.

12 **DR. KENNETH PORTIER:** Dr. Barone has
13 his hand up. Stan --

14 **DR. STANLEY BARONE:** Dr. -- Dr.
15 Portier, we'll have to follow up on Dr. Schlenk's
16 question after the break. We'll see if we can
17 find the answer to that.

18 **DR. DANIEL SCHLENK:** The reason why
19 I'm asking it is --

20 **DR. KENNETH PORTIER:** Dan --

21 **DR. DANIEL SCHLENK:** Yeah. The
22 reason why I'm asking it is, you know if we're

1 seeing a problem formulation in 2016 and 2017, and
2 to say that there's potential discharge at a
3 particular COU, is it possible to actually get
4 monitoring data before the DRE comes out? That's
5 the question. So that's the rationale for that
6 first question.

7 Second question is, so I'm just
8 curious, is it required for EPA to make a yes/no
9 decision on a -- on -- again, this is the
10 environmental risk component. Is it possible to
11 actually just conclude that we cannot make a risk
12 assessment at this time because we don't have
13 certainty in our exposure assessment? Is that
14 even a possibility? Does it have to be a yes/no?

15 **DR. KENNETH PORTIER:** Dr. Scarano?

16 **DR. LOUIS SCARANO:** Sorry. Thank
17 you. I also do not know the answer to that. We
18 can either find later or I invite some other --
19 someone who knows more the respond specifically.

20 **DR. DANIEL SCHLENK:** Okay. Third
21 question, this has to do with slide 30. You can
22 put it up if you'd like, I guess. This is with

1 regard to definition of uncertainty. Basically, I
2 -- letting you get there if you want. But the
3 slide 30 basically says, and it's interesting that
4 in this presentation it -- the uncertainty was
5 changed from low to low-to-medium based --
6 compared to the earlier slides that I got a few
7 days ago. But the Agency considers, again, the
8 exposure assessment to surface water with --
9 there's essentially low uncertainty.

10 But if I go to page 52, on the
11 bottom of page 52 it says, "Asbestos releases from
12 chlor-alkali facility treatments to surface water
13 and POTWs are not known. While the treatment
14 technologies employed would be expected to capture
15 asbestos solids, precise treatment efficiency is
16 not known. That to me would indicate uncertainty.

17 So I'm just curious, is there
18 something I'm missing in terms of a definition of
19 uncertainty that's present that's utilized in
20 slide 30? Based upon what I'm reading in the
21 document I'm -- those things are disconnected. Is
22 there -- is there, you know an Agency specific

1 definition of uncertainty that -- that I'm
2 missing? Hello? Can anybody hear me?

3 **DR. KENNETH PORTIER:** Dr. Schlenk, I
4 think they're thinking.

5 **DR. DANIEL SCHLENK:** Okay.

6 **DR. KENNETH PORTIER:** You're asking
7 tough questions today.

8 **DR. DANIEL SCHLENK:** Yeah. Well,
9 it's the last one, right? For a while anyway.

10 **DR. KENNETH PORTIER:** Dr. Scarano?

11 **DR. LOUIS SCARANO:** Yes. Thank you.
12 I'm sorry. I'm trying to unmute, and I don't have
13 a specific, quick answer. Thank you for the
14 question. I can see how you would ask that given
15 what you just read from page 52. Again, we will -
16 - I will get back to you unless, again, some of my
17 teammates have something that they can say
18 specifically right now. But --

19 **DR. DANIEL SCHLENK:** Okay. Thank
20 you very much.

21 **DR. LOUIS SCARANO:** No, thank you.

22 **DR. KENNETH PORTIER:** Thank you.

1 This is Ken Portier. I have a related question to
2 something. You mentioned that I think it's slide
3 28, deposition to natural waters. You said no
4 risk to direct deposition to surface waters. And
5 I wondered, did you look at indirect deposition?
6 In other words, asbestos that's -- that is air
7 born that then deposits in the water. Was that
8 even looked at?

9 I mean, I realize, you know, direct
10 would be from a commercial site directly input
11 into the water which would be a much higher
12 concentrations, but -- and I guess an adjunct
13 question to that would be, did indirect deposition
14 to water follow from a risk evaluation performed
15 under the Clean Air Act? Dr. Scarano?

16 **DR. LOUIS SCARANO:** Yeah. I had my
17 hand up. I was waiting for you to call. I think
18 --

19 **DR. KENNETH PORTIER:** Oh, okay. You
20 don't need for me to call you. Just answer these
21 questions.

22 **DR. LOUIS SCARANO:** I apologize. I

1 apologize. So are you talking about slide 28?

2 **DR. KENNETH PORTIER:** Yeah. I think
3 -- or it's somewhere around 28 where you were
4 talking about direct deposition to surface waters,
5 and I started thinking about the indirect
6 deposition and where would that be covered? You
7 know, I -- I mean, under Clean Air Act, Clean
8 Water Act or TSCA? I mean, that's one of the
9 issues the committee's had with almost all these
10 chemicals is where the TSCA risk evaluation
11 boundary is on some of these -- especially some of
12 these exposure issues. And I just wondered if
13 that had been looked at and thought about or
14 whether it's assumed and it's going to be covered
15 under one of the other jurisdictions.

16 **DR. LOUIS SCARANO:** I see. I can't
17 seem to find the term direct or indirect in any of
18 the slides, but maybe --

19 **DR. KENNETH PORTIER:** It was on page
20 -- it's on page 35 too where we -- where --

21 **DR. LOUIS SCARANO:** Oh.

22 **DR. KENNETH PORTIER:** -- you talked

1 about, I think, conditions of use. At least I
2 think that's where I found it.

3 **DR. LOUIS SCARANO:** Anyway, I can
4 just respond that I think in terms of air
5 deposition we are referring to the Clean Air Act,
6 and also I think the data from the Toxics Release
7 Inventory that show very low release nationwide of
8 between 100 and 300 pounds into air. And so I'll
9 just point to those two points and invite anyone
10 else on the team to say more. Or if you want
11 more, we -- if you have more specifics to that
12 question that we can research and get back to you,
13 be happy to do that.

14 **DR. KENNETH PORTIER:** No. That's
15 fine. I'm going to stick with the environmental
16 questions and go to Dr. Doucette. And Dr.
17 Anderson, I see your hand up, and Dr. Markowitz.

18 **DR. WILLIAM DOUCETTE:** Okay. This
19 is Bill Doucette and it's -- I think it's more of
20 a regulatory nexus clarifying question. But I
21 grew up in northern Wisconsin, Minnesota back in
22 the '60s and '70s and there's substantial taconite

1 mining. It was low-grade iron ore.

2 And it turned out they were mining
3 the iron ore, but they were releasing asbestos
4 during the mining operation. That wasn't what
5 they were mining but it was essentially a
6 byproduct of a natural deposit of asbestos
7 associated with the iron ore.

8 And they actually dumped that into -
9 - the waste into Lake Superior and it was
10 ultimately found in drinking water supplies in
11 Duluth, Minnesota and several other communities.
12 And what my question is, if I understand if it's
13 asbestos being mined that's one thing but if it's
14 a natural deposit that is disrupted either from
15 mining or construction and then that is released
16 into the environment, who has -- what regulations
17 cover that? And is that considered under this
18 TSCA panel?

19 **DR. STANLEY BARONE:** Dr. Doucette,
20 this is Stan Barone. I believe we'll check back
21 with you on that, but in general, mining tailings
22 are not covered under TSCA to the best of our

1 knowledge. I believe that's OLEM.

2 **DR. WILLIAM DOUCETTE:** Okay. As a
3 follow up too, just looking at a map of natural
4 asbestos deposits in the U.S. and its relationship
5 to population areas, there's certainly the
6 opportunity for construction, highways, even in
7 large housing developments to bring asbestos out
8 from the natural deposits or disturb them, and
9 enter it into the environment either through
10 fugitive air emissions or possibly into streams
11 and surface water bodies from runoff.

12 Would -- so that's not a mining
13 issue, that's just a construction issue. Same
14 question, is that covered under TSCA? I mean, I
15 don't know the answer so that's -- you know, and
16 it -- but it helps me understand, you know, what
17 jurisdiction that falls under.

18 **DR. STANLEY BARONE:** We'll confer
19 and get back to you. This is Stan Barone again.

20 **DR. WILLIAM DOUCETTE:** Okay. Thank
21 you. That's all I have.

22 **DR. KENNETH PORTIER:** Dr. Anderson.

1 **DR. HENRY ANDERSON:** Yeah. I will
2 focus on the exposure issues. And this is kind of
3 too generic here but other times we've faced it as
4 well is that's when you have a kind of unusual
5 circumstance of having exposure data, measurement
6 data from multiple facilities and manufacturing
7 concerns such as here we have multiple chlor-
8 alkali plants. And it appears that you simply
9 combine them all together and then come up with a
10 common -- or the high end or low-end modeling.

11 And I'm just wondering, it would
12 seem to me it might be worthwhile to look at each
13 of the companies individually. Just remember in
14 the PCE where we had -- data was provided by three
15 different companies, two of them had the online
16 detects and one of the companies actually had
17 methodologies of identified measurement levels.
18 If you combine them all then the median value or
19 mean, is going to be considerably lower than if
20 you looked at them individually.

21 And so, kind of, one question is, is
22 there a protocol for how you deal with those

1 multiple sites and potentially weights of the data
2 on the proportion of samples that come from one or
3 the other? That's the first question.

4 And then that could be again it
5 would carry over to, like, in the brake work here.
6 You choose a single study to say this is the one
7 we think is the best and don't particularly then
8 describe all of the potential data together and
9 say because of their -- they're not really
10 comparable we chose this one over the other one.
11 So it's how -- especially with the combining
12 multiple companies, how do you do that to have it
13 be truly representative, but not -- or to identify
14 the different conditions and different facilities,
15 which part of the COUs.

16 Do you want me to give you the
17 second question now? And that has to do with the
18 gig economy that you're -- interested in when you
19 did on the consumer use of the brake replacement
20 and gaskets, your assumption of well, it's only
21 done once every three years or only a one time, at
22 least in Wisconsin, you can go to a neighbor or

1 there's individuals who will do your brake repair
2 or replacement job, especially on your older, kind
3 of old beater vehicles that you use for a whole
4 lot less than taking it in to a certified shop.
5 And those individuals would be doing far more than
6 once every three years.

7 And the other is if you have a clan
8 or a large family group, there may well be one of
9 the -- at least in my instance it was younger
10 students who are wanting to go into the field,
11 they will service all of the vehicles in a
12 household. So it's kind of -- have you considered
13 other scenarios or, again, the gig economy where
14 some of these service activities can in fact be
15 done at home and/or by somebody who's actually
16 being paid to do that work, just not being hired
17 by or working at a specific company.

18 And one thought there would be, you
19 could do a survey of -- if you're going to be
20 doing that kind of work you've got to buy your
21 parts somewhere. And there are online as well as
22 local parts stores and they'll all keep a record

1 of who buys. They won't tell you who it is, but
2 you might be able to get a, so what proportion of
3 people are buying in kind of bulk supplies of some
4 of these things and they're not associated with a
5 specific business. Could you hear me?

6 **DR. KENNETH PORTIER:** I hear you,
7 Dr. Anderson. I think they're struggling with
8 answers.

9 **DR. HENRY ANDERSON:** Okay. That's
10 okay. I don't necessarily need -- I just think
11 there's some commonality of sorts of activities
12 when we get to consumers that is a little
13 different than just it's a single person or a
14 single household using something. And moving
15 forward, EPA may want to consider that or if you
16 do a revision of these, use an example of how many
17 of these are being done and put it in gig economy
18 rule.

19 **DR. STANLEY BARONE:** Dr. Anderson,
20 this is Stan Barone. Do you have specific
21 information of articles or conditions where a gig
22 economy type scenario is applicable? We're not

1 sure of what exactly you mean, what you're
2 referring to.

3 **DR. HENRY ANDERSON:** I mean,
4 articles I don't know that's not something that's
5 necessarily going to be -- I'm not aware of any
6 scientific publications that have evaluated that.
7 On the other hand, there are parts stores where
8 you can buy brake issues and you mentioned it
9 yourself as to -- in here. And it appears your
10 assumption is that it's all individuals who are
11 just buying for their own use and there's no
12 limitation on that.

13 I haven't done a survey, but I would
14 -- I am familiar in my own circumstance of my
15 neighbor who does brakes. Now, right now it's on
16 newer vehicles, but if you want to you could get
17 the other types of brakes repaired. So I know
18 there's an economy out there that does it.

19 I haven't tried to do a survey like
20 that, but again I think it is apparent that EPA
21 hasn't thought about that either, and that's what
22 I'm just raising. If you only rely on what might

1 be published, that's not the sort of thing that
2 necessarily is covered there. There may well be
3 data in the economic field that could get you at
4 some of that.

5 And this would also pertain to UTVs.
6 I mean, you're characterizing the removal of an
7 exhaust system from an automobile for that of a
8 relatively small UTV. And apparently, I don't
9 know if you ever went to any of the thousands of
10 facilities that sell and mostly service their own
11 things to find out how exactly how is that done
12 and what are the circumstances. In my experience
13 with UTVs is going ice fishing and having to drive
14 three miles out onto a lake is very rough ice and
15 doing that in a covered UTV. And they have to be
16 a pretty sturdy little vehicle.

17 And one of the issues is you get
18 hung up and you bend your exhaust system and it's
19 cheaper to take it apart at the -- where the
20 exhaust connects -- where you have a gasket where
21 on the automobile often as was done on the samples
22 you used, they use a torch to cut off part of this

1 because it's very difficult to get the fitting
2 apart. Where the gasket is holding it's basically
3 welded between the two parts, so it takes less
4 time to use a torch, tie it off, and then remove
5 it at the manifold rather than where it attaches
6 elsewhere. So it would be nice to have had a
7 description of how the UTV repairs replacements
8 are actually done and is it comparable. But to
9 the example that you used which was, you know
10 older automobiles.

11 **DR. KENNETH PORTIER:** Thanks, Dr.
12 Anderson. I think we'll keep that for the
13 conversation and would like to move on to get a
14 couple of other committee members. Dr. Markowitz
15 and then Dr. Davies. Dr. Markowitz.

16 **DR. STEVEN MARKOWITZ:** Sure. Steve
17 Markowitz. So I think I have two questions that
18 are probably answerable. If you could go to slide
19 49. So when you reviewed this the -- it looked
20 like for about the last few lines of that, it says
21 that I'm quoting, "that the non-cancer effects of
22 chrysotile are likely to contribute additional

1 risk." So my question is, is that representative
2 of evolution in the thinking of EPA? Does that
3 mean that EPA believes that the non-malignant
4 mortality due to asbestos, chrysotile asbestos,
5 should be included in the risk evaluation?

6 **DR. LOUIS SCARANO:** This is Gino.
7 The person who can best respond to that may be
8 online, but he may be having troubles. We will
9 get back to you on that. That's a good question.

10 **DR. STEVEN MARKOWITZ:** Okay. Okay.
11 Thanks. So could you go to slide 57? Okay. So
12 here's a discussion about the textile studies and
13 the asbestos fiber, the variation size, and
14 measurement et cetera. The -- so the potency
15 factors, the unit risks for lung cancer and
16 mesothelioma were derived from the two textile
17 cohorts. They provide, clearly, the best
18 available data and therefore the most valid
19 results that we have.

20 But none of the conditions of use,
21 of course, are in textile facilities so that
22 raises the issue of generalizability. Validity is

1 more important than generalizability it's -- if
2 it's not valid you can't generalize. But there is
3 in the table in the risk -- the draft, Table 3-28,
4 which is referred to here in the last bullet item
5 which is the range of the values of the lifetime
6 unit risks for lung cancers within mining and
7 separately within textile are broad.

8 And that there's clear overlap
9 between those two industries. And so this is a
10 question I think maybe for Dr. Loomis, Dr.
11 Stayner. What's your sense then about
12 generalizability of the results that are obtained
13 in the risk evaluation in terms of the lifetime
14 risk to other settings that use chrysotile beyond
15 textile?

16 **DR. STANLEY BARONE:** Yeah. Can you
17 hear me?

18 **DR. STEVEN MARKOWITZ:** Yeah.

19 **DR. STANLEY BARONE:** Yeah. It's a
20 very good question, Steve. So we certainly, first
21 of all, do not have data from the chlor-alkali
22 exposure-response that we could use for the risk

1 assessment. And there's long been recognized that
2 risk of vary by industry. We also have attempted
3 to look at analyses of the South and North
4 Carolina cohorts -- . They explain some of those
5 industry differences. And it's been very
6 difficult because those different fiber sizes are
7 all correlated.

8 The most recent attempt at doing
9 that, using a Bayesian model, concluded that there
10 really was no good evidence for differences in
11 fiber size. But anyway, I think it is a concern
12 that is something that we simply can't address in
13 terms of these industries. I should mention also
14 that the brake workers, there's simply not that
15 good study that would be suitable for exposure-
16 response analysis. Okay. Is that your question?

17 **DR. STEVEN MARKOWITZ:** Sure.

18 **DR. DANA LOOMIS:** This is Dana
19 Loomis. Let me try to add to that. So I think,
20 you know, number one I would reiterate that we
21 have tried to look at fiber size-specific risks.
22 And in the most recent analysis of the data from

1 North Carolina textile plants, there's good
2 evidence that the smallest fibers are also
3 carcinogenic, and they may be -- the potency of
4 those fibers may be equal to or greater than that
5 of longer fibers.

6 But I think the important part about
7 the textile studies is that fibers of all sizes
8 are present in that environment even though on
9 average the textile industry preferred longer
10 fibers because those are better for making woven
11 products. So those workers are exposed to short
12 fibers and most of the fibers that they're exposed
13 to in fact are very small ones.

14 **DR. STEVEN MARKOWITZ:** Thank you.

15 **DR. KENNETH PORTIER:** That was Dr.
16 Loomis speaking, is that correct?

17 **DR. DANA LOOMIS:** That's correct.

18 **DR. KENNETH PORTIER:** Thank you.
19 Thank you, Dr. Markowitz. Dr. Davies and then I'm
20 going to go down to Dr. Kissel and then Dr. Crump.
21 Dr. Davies.

22 **DR. HOLLY DAVIES:** Hi. I had a

1 question that goes back to the regulatory nexus.
2 So this draft risk evaluation didn't include
3 asbestos contaminants in products like talc. And
4 so my question is, where is EPA going to include
5 the risk for contaminants? And the second
6 question was more on the timeline on the -- and
7 the process. I thought I heard that the legacy
8 evaluation is going to follow the TSCA timeline.
9 And does that mean a scope in six months and the
10 risk evaluation in three years?

11 **DR. LOUIS SCARANO:** This is Gino
12 Scarano. I can address the second issue pretty
13 fast. Yes. That is the idea. In other words,
14 we're going to issue a draft scope and then that
15 will kick in the timeline that's associated with
16 the amended TSCA for three years when we complete
17 the risk evaluation for the legacy asbestos.

18 **DR. HOLLY DAVIES:** Great. Thanks.

19 **DR. LOUIS SCARANO:** Yeah. In terms
20 of the first question, I think I will, you know,
21 yield to some of my work plan members. But I do
22 want to point out that, you know, the current -- a

1 lot of the current news is around the cosmetic use
2 of talc which is under the FDA or the Food and
3 Drug Administration purview.

4 **DR. KENNETH PORTIER:** Anyone else
5 from the EPA team want to chime in on this? I'm
6 not hearing anyone. Why don't we move on to Dr.
7 Kissel?

8 **DR. JOHN KISSEL:** Hi. I have maybe
9 three questions. The first one has to do with
10 slide 12, which is the reproduction of Table 1-1.
11 And fiber length is listed there as less than a
12 millimeter, which is a thousand microns to maybe a
13 centimeter, which is 10 thousand microns. And my
14 read of the literature is all the debate is about
15 numbers that are less than or greater than five
16 microns.

17 And so I guess I have a couple of
18 questions or sub-questions. One is, is how is
19 that description of length relevant to much of
20 anything? And the second one would be, does
21 anybody know -- we're importing raw chrysotile.
22 Has anybody done a particle size distribution for

1 that material? Does anybody know what the actual
2 distribution of fiber lengths looks like in a bag
3 of raw material as imported now?

4 **DR. LOUIS SCARANO:** This is Gino.
5 Once again, I invite some team members who may
6 know more. I will definitely look and make sure
7 that that is appropriately -- if that's the right
8 number. I think it is from the reference we
9 used. And I can't answer about the other. Yes.

10 **MR. ABHILASH SASIDHARAN:** Yes.
11 Hello Gino, this is Abhilash.

12 **DR. LOUIS SCARANO:** Thank you
13 Abhilash.

14 **MR. ABHILASH SASIDHARAN:** Actually,
15 this is a physico/chemical properties of
16 commercial chrysotile asbestos. Chrysotile
17 asbestos fiber bundle samples usually contain
18 broad distribution of fiber sizes and usually
19 exhibit diameter anywhere from 0.1 microns to 100-
20 micrometer size, and the length ranging from a few
21 micrometers to millimeters.

22 This is actually that for

1 aggregates, Chrysotile asbestos aggregates. These
2 are not for individual -- individual fiber size
3 may be in the below five-micrometer range but this
4 is data for chrysotile asbestos.

5 **DR. JOHN KISSEL:** And is there any
6 distribution data for the product as imported?
7 You know, mean, median, 95th percentile?

8 **DR. ABHILASH SASIDHARAN:** I don't
9 know the answer to that question. I'll get back
10 to you later.

11 **DR. JOHN KISSEL:** Okay. So the --
12 in supplement to the question that Holly Davies
13 asked, the scoping document mentions talc and then
14 the notion is kind of dismissed without actual
15 explanation. But it appears to me from reading
16 the literature that dermal exposure is generally
17 not of interest here with asbestos but it --
18 asbestos fibers do penetrate some selective
19 tissues including vaginal tissue. And that
20 pathway is not even mentioned in passing and I
21 wondered, you know, even if you think it's not
22 your purview couldn't it at least be mentioned

1 here?

2 **DR. STANLEY BARONE:** Dr. Kissel,
3 this is Dr. Barone. I'm not sure we understood
4 the last part of your question. Mention which?
5 Are you talking about the other fiber types or
6 what exactly was the question? It's not clear.

7 **DR. JOHN KISSEL:** So the question
8 is, why is nothing other than exposure by
9 inhalation considered when there does seem to be
10 evidence that asbestos fibers will penetrate
11 vaginal tissue which is a big part of what the
12 talc issue is about?

13 **DR. STANLEY BARONE:** Okay. So we
14 didn't consider talc in the conditions of use in
15 this particular assessment.

16 **DR. JOHN KISSEL:** Right.

17 **DR. STANLEY BARONE:** And you're
18 asking about why not consider dermal/vaginal
19 uptake of asbestos fibers is what you're asking?
20 Just to clarify.

21 **DR. JOHN KISSEL:** Well, that is a
22 consumer use. You know, talc is a consumer product

1 and has been used and I think you can still buy it
2 although there's a lot of bad publicity in the
3 press and so --

4 **DR. STANLEY BARONE:** Right.

5 **DR. JOHN KISSEL:** -- people are
6 shying away from it. But it is an active consumer
7 product. It's not banned so far as I know.

8 **DR. LOUIS SCARANO:** This is Gino.
9 You are correct, Dr. Kissel. But as Stan had
10 pointed out and I had mentioned earlier, I think
11 the cosmetic use which might lead to the
12 potential, you know, vaginal exposure and dermal
13 absorption is not under the TSCA purview as far as
14 I understand. So I can appreciate how you might
15 want us to mention it but it -- there -- it's --
16 anyway, I'll just stop there and see if anyone
17 wants to say anything else.

18 **DR. MARK JOHNSON:** Hey, Ken, this is
19 Mark Johnson. If I could just get in real quick
20 because the question I was going to ask is very
21 pertinent to the current issue. I was just
22 wondering --

1 **DR. KENNETH PORTIER:** Go ahead,
2 Mark.

3 **DR. MARK JOHNSON:** I'm not going to
4 talk about talc, I was looking at do-it-yourself,
5 people who are doing brake jobs, okay? Is the
6 asbestos form that comes off of these brakes a
7 form that could be dermally absorbed? I mean,
8 right now, the DRE discounts it. But why is that
9 any different than talc, I guess is my question.

10 **DR. KENNETH PORTIER:** This is Ken
11 Portier. You mean talc absorbed or asbestos as a
12 contaminant in talc absorbed? Are you talking
13 about the absorption of asbestos dermally --

14 **DR. MARK JOHNSON:** Yes.

15 **DR. KENNETH PORTIER:** -- or the
16 absorption of talc with an asbestos contaminant?
17 I just wanted to clarify Mark.

18 **DR. MARK JOHNSON:** Yeah. Well, I --
19 quite honestly, I don't know enough. I don't know
20 if there's any compound facilitated transport.
21 Does talc facilitate the transport of asbestos
22 particles through the skin? I mean, I guess

1 that's possible. I've seen it with other
2 chemicals but not with a mechanical substance like
3 asbestos.

4 But my question is, is the form of
5 asbestos that's in brake dust any different than
6 the form that's in talc? And why is it
7 unreasonable to not think it would also go through
8 the skin if you're exposed?

9 **DR. STANLEY BARONE:** This is Stan
10 Barone. I think we're talking about very
11 different conditions of use. Again, personal care
12 products versus an occupational setting. Personal
13 care products being applied after showering to the
14 body versus actual -- the conditions of use that
15 we're assessing in occupational conditions of use
16 where clothing and PPE may be worn -- or clothing
17 would be worn and possibly PPE would be worn.

18 **DR. MARK JOHNSON:** Okay. Dr.
19 Barone, this is Mark Johnson again. I guess what
20 you're saying, and correct me if I'm wrong, is
21 that if you're doing a brake job you're not
22 applying it to the sensitive areas of your skin

1 where dermal absorption would be more likely to
2 occur. You're exposing maybe the palms of your
3 hand, very keratinized areas of the skin where
4 dermal absorption would most likely be very, very
5 low. Is that correct?

6 **DR. STANLEY BARONE:** Those are the
7 assumptions we described in the evaluation.

8 **DR. MARK JOHNSON:** Okay. Thank you.

9 **DR. KENNETH PORTIER:** Back to Dr.
10 Kissel.

11 **DR. JOHN KISSEL:** Yeah. So back to
12 my last question. Bill Doucette mentioned
13 taconite releases to the aquatic environment, but
14 that also raises the issue of what about the
15 taconite miners that's active, current, and future
16 activity and there is some epi. It's not
17 particularly scary but there is epidemiological
18 evidence of miner exposure and miner cohabitant's
19 exposure to asbestos fibers as a consequence of
20 mining. And those are also not included here even
21 though they aren't really legacy uses, they're
22 ongoing and future uses.

1 **DR. KENNETH PORTIER:** You know, John
2 -- this is Ken Portier. What -- you know,
3 language means something here. And I think EPA is
4 trying to say that if you're -- if asbestos is not
5 -- if it's not mining asbestos, you're mining iron
6 and the asbestos is a contaminant in that, part of
7 the exposure, it's covered somewhere else. And I
8 think that's why it's excluded here but maybe EPA
9 can clarify that.

10 And I understood Dr. Doucette's
11 question kind of under the same light is that
12 certainly there's exposures there. But under the
13 condition of use kind of framework that, you know,
14 these last -- these first 10 risk assessments have
15 been focused, mining iron is not a condition of
16 use of asbestos. It relates to exposure. And
17 this is, I think, one of the problems -- another
18 one of the problems we've experienced with these
19 risk assessments is the condition of use framework
20 doesn't always cover all of the occupational
21 exposures that we identify. Is that kind of what
22 you're getting at, John?

1 **DR. JOHN KISSEL:** Yeah. That's -- I
2 mean it's -- we're back to the regulatory nexus
3 thing. And what I'm arguing for is not
4 necessarily inclusion of everything I can think of
5 in this document but I would like to see, kind of
6 a master road map diagram that says, here's how
7 people get exposed to asbestos and here's what's
8 covered under this framework.

9 **DR. KENNETH PORTIER:** Yeah. I think
10 I'm going to go on to Dr. Crump and see what Kenny
11 wants to add. Dr. Crump.

12 **DR. KENNY CRUMP:** Yeah. I have a
13 question about the PESS particular exposures of
14 cigarette smokers. I think there's a pretty fair
15 amount of literature on a synergistic relationship
16 between cigarette smoke and asbestos and causing
17 lung cancer. I think that would make cigarette
18 smokers potentially a susceptible subgroup. And I
19 think TSCA mandates that you should estimate
20 exposures to susceptible subgroups whenever
21 possible.

22 And it seems to me this would be

1 easily done in this situation. You could just use
2 the rates and the life table that -- the death
3 rates that pertain to smokers. So I just wonder
4 if there was some particular reason why the
5 document doesn't contain something like that, an
6 assessment of the risks to smokers in particular.

7 **DR. LOUIS SCARANO:** Dr. Crump,
8 thanks. This is Gino. We will get back to you on
9 that. The right people will be able to address
10 that.

11 **DR. KENNY CRUMP:** Given that, that's
12 a related question I had. If they're considered a
13 susceptible subgroup and you won't estimate the
14 risk particularly for them, should your risk for
15 the general population not exclude that
16 susceptible subgroup? Should your risk for the
17 general population be -- include only non-smokers?
18 I just throw that out as a possibility.

19 **DR. LOUIS SCARANO:** Yeah. This is
20 Gino. Interesting point. So noted. Yeah.

21 **DR. KENNETH PORTIER:** Yeah. This is
22 Ken Portier. Dr. Crump, I think you pointed out

1 another issue that's come up a number of times in
2 these risk evaluations is where -- what's the
3 bright line between a potentially exposed
4 susceptible population and the population that is
5 covered in the risk assessment. And I think
6 there's kind of uncertainty and who's in and who's
7 out, who's covered by uncertainty factors and
8 who's not. I'm sure that's going to come up again
9 in the discussion. Dr. Kissel, your hand's still
10 up. Did you have a follow-up question? Hand went
11 down. Dr. Anderson, your hand is still up. Do
12 you have a follow-up question?

13 **DR. HENRY ANDERSON:** Yes. I just
14 wanted to get some clarification on a regulatory
15 nexus issue. And this assessment is labeled as
16 asbestos, although really it's focused on
17 commercial chrysotile. And what I was wondering
18 since the earlier response was, we're probably
19 looking at at least three years before there would
20 be the legacy set of asbestos exposures
21 considered, is there a specific regulatory reason
22 why -- when you call this asbestos document that

1 would seem to imply all asbestos. And why
2 couldn't you when you revise this revise it to be
3 an asbestos document? That would include the
4 legacy uses in it and that might shorten it and
5 make this more of a composite approach as was done
6 IRIS and some of the others rather than treat this
7 as a whole separate chemical?

8 **DR. LOUIS SCARANO:** This is Gino.
9 I'll actually take a stab at answering one. So,
10 Dr. Anderson, I mean, I think the important thing
11 here is, this asbestos draft risk evaluation
12 covers what EPA believes is actually actively
13 active uses of asbestos. So it is important that
14 we finalize and finish this. The asbestos -- the
15 legacy decision ended up going to the courts.
16 Decision has come down. We're going to honor
17 that. But it is going to be the next thing that
18 we do. I don't think we want to prolong the
19 finalization of this to include asbestos which
20 will be a substantial effort. So I hope that
21 helps.

22 **DR. HENRY ANDERSON:** There's going

1 to be some disagreement on that. I mean, the
2 others, you have a whole new industry that's
3 actually working with existing products and that's
4 the remediation and removal workforce. What I've
5 suggested is an industry that is using asbestos.
6 They're not manufacturing but they are using all
7 of the asbestos products. So that's why I think
8 it all fits together better in a single approach
9 to asbestos since most of those legacy uses, the
10 predominant specified mineral there is chrysotile.

11 **DR. STANLEY BARONE:** Dr. Anderson,
12 this is Stan Barone again. For clarification, are
13 you talking about disposal and remediation or are
14 you talking about including those conditions of
15 use which are already regulated into our risk
16 evaluation? Just trying to understand what you're
17 --

18 **DR. HENRY ANDERSON:** I would think
19 that would include it.

20 **DR. STANLEY BARONE:** But we've
21 already --

22 **DR. HENRY ANDERSON:** I mean, it's

1 the same as in these industries, the asbestos is
2 regulated under OSHA.

3 **DR. LOUIS SCARANO:** Well, no. In
4 the -- and this is Gino again. In the first dozen
5 slides or so we went over the EPA regulations.
6 There's a fair number of them that address that
7 particular issue and different states have
8 different regulations in place for remediation,
9 and then there are different rules. So there is a
10 lot of that that is currently being regulated.

11 **DR. HENRY ANDERSON:** And you're
12 saying that the chlor-alkali industry and the
13 brake replacement industry is not regulated? Is
14 that covered by OSHA? Are they exempted?

15 **DR. LOUIS SCARANO:** Well, what I'm
16 saying is that those are active uses that come
17 under the authority of TSCA which is the purpose
18 of this risk -- draft risk evaluation. If that
19 makes sense.

20 **DR. HENRY ANDERSON:** Okay. I was
21 just trying to see if it wouldn't make more sense
22 to approach all asbestos. Either that or you're

1 going to need to change the title on this to be
2 commercial chrysotile asbestos and just use that
3 term throughout.

4 **DR. STANLEY BARONE:** So is -- this
5 is Stan Barone, Dr. Anderson. So you're
6 suggesting a potential title change as in the
7 slides which would refer to this risk evaluation
8 focusing on chrysotile?

9 **DR. HENRY ANDERSON:** I would make it
10 commercial -- I mean, you -- in your charge
11 questions somebody must have thought about that
12 and called it commercial chrysotile. So I --

13 **DR. KENNETH PORTIER:** Well, Dr. --

14 **DR. HENRY ANDERSON:** -- appreciate
15 you either need -- you either change that title
16 and I'm -- you read through the document it seems
17 to be asbestos, chrysotile asbestos, commercial
18 chrysotile, you're using those interchangeably.

19 **DR. KENNETH PORTIER:** Dr. Anderson,
20 this is Ken Portier. I had exactly the same
21 thought and I'm sure we're not the only ones on
22 the committee that looked at the title of the risk

1 assessment and then looked at what was discussed
2 in this document and saw a disconnect. And I
3 suspect we'll make a recommendation for a title
4 change at some point in the next three days.

5 **DR. HENRY ANDERSON:** Yeah. I know.

6 **DR. KENNETH PORTIER:** Any additional
7 comments from the committee? I have about two
8 minutes to one at which point we're going to break
9 45 minutes for lunch and come back and take public
10 comments. I'm trying to figure out where in the
11 agenda we will come back to the six or seven, kind
12 of, questions that remain unanswered by the
13 committee and it may be tomorrow morning before we
14 get to those, maybe first thing in the morning
15 tomorrow morning we'll revisit some of Dr.
16 Schlenk's, Dr. Anderson's, Dr. Markowitz's, Dr.
17 Kissel's, and Dr. Davies' unanswered questions.
18 So just kind of provided EPA staff with a warning
19 that I haven't -- we're not giving up on those
20 questions before we jump into our committee
21 discussions.

22 At this point, I think I'm going to

1 call the break for lunch. We're going to
2 reconvene at 1:45 and begin with public comment
3 presentations. We have a large collection of
4 public commenters that we're going to hear this
5 afternoon. So I really want to start on time. So
6 we'll break on time. We'll reconvene at 1:45
7 eastern. Thank you very much.

8 **[Lunch Break]**

9
10 **DR. KENNETH PORTIER:** Good
11 afternoon. I'd like to reconvene the meeting. I
12 have 1:45 eastern time. At this point, I'm going
13 to turn the meeting over to DFO Dr. Wong who will
14 invite the public commenters to make their
15 presentations before the committee. Dr. Wong.

16
17 **PUBLIC COMMENTS**

18
19 **DR. DIANA WONG:** Thank you, Dr.
20 Portier. Now we come to the public comments
21 section. There are 33 registered oral commenters.
22 You have five minutes each to speak, and they may
23 answer questions if any from the committee. The

1 first speaker is Linda Reinstein. Are you on the
2 line?

3 **MS. LINDA REINSTEIN:** Yes, I am.
4 Yes. I am.

5 **DR. DIANA WONG:** Are you ready to
6 start?

7 **MS. LINDA REINSTEIN:** Yes, I am.
8 Thank you, Dr. Wong.

9 **DR. DIANA WONG:** Thank you.

10 **MS. LINDA REINSTEIN:** I'm Linda
11 Reinstein, the co-founder of the Asbestos Disease
12 Awareness Organization, ADAO, and a mesothelioma
13 widow. ADAO, an independent non-profit is the
14 only national organization dedicated to preventing
15 asbestos exposure and eliminating all asbestos-
16 caused diseases. Once again, I want to voice
17 ADAO's deep concern about the draft risk
18 evaluation for asbestos. But first, in keeping
19 with ADAO's tradition, I'd like to dedicate my
20 oral remarks to Mike Mattmuller who recently died
21 from preventable mesothelioma just days after his
22 38th birthday. And Mike leaves behind a young

1 wife and a three-year-old daughter.

2 The facts are irrefutable. Each
3 year nearly 40,000 Americans die from preventable
4 asbestos-related diseases. Leading health
5 authorities have agreed for years that asbestos is
6 a human carcinogen and there is no safe level of
7 exposure. Nearly 70 countries have banned
8 asbestos, yet our government has failed time and
9 time again to ban this known carcinogen.

10 EPA tried to ban asbestos in 1989
11 but industry won, and the Fifth Circuit Court of
12 Appeals overturned the ban. And since 1989 we
13 have consumed 400,000 metric tons of asbestos and
14 buried 1 million Americans. Asbestos became the
15 poster child for the need to reform the Toxic
16 Substances Control Act of 1976. And in 2016 we
17 had hoped that the Lautenberg Chemical Safety Act
18 would bring around the rapid elimination of
19 asbestos.

20 However, EPA's inaction has been
21 dangerously disappointing. We're not alone in our
22 concern. EPA's own scientists and civil servants

1 have expressed deep concerns of the Agency's weak
2 approach to asbestos in internal documents shared
3 with ADAO and reported in the New York Times. The
4 chlor-alkali industry is the primary importer and
5 user of this deadly carcinogen. According to the
6 EPA three corporations, Olin, Occidental Chemical,
7 and Westlake owned 15 chlor-alkali plants and use
8 asbestos diaphragms to produce industrial chlorine
9 and caustic soda. These corporations continue to
10 rely on asbestos for their production even though
11 safer alternatives exist.

12 There are numerous pathways of
13 exposure that put Americans at risk outside of the
14 plants that are not being evaluated in this draft
15 risk evaluation. The draft risk evaluation is the
16 first comprehensive assessment of asbestos in over
17 30 years. We are depending on the staff to
18 provide the honest and hard-hitting feedback that
19 the EPA needs to do its job correctly and fully
20 examine the impact of this deadly chemical.

21 As EPA evaluations shows, current
22 uses of asbestos may expose 1.5 million and up to

1 32 million consumers. But even that doesn't fully
2 reflect the threat of this chemical poses to the
3 public health. ADAO and numerous stakeholders
4 have filed extensive comments in the draft
5 evaluation including 14 Attorneys General,
6 American Public Health Associations, Safer
7 Chemicals Healthy Families, AFL-CIO, and more.

8 I want to briefly summarize our
9 major concerns in the draft risk evaluation. One,
10 it excludes legacy asbestos despite a ruling from
11 the Ninth Circuit Court of Appeals last year
12 stating that the EPA is obligated to evaluate
13 these risks. It only evaluates the risk of
14 chrysotile and fails to consider the other five
15 amphibole fibers.

16 It only evaluates the risk of lung
17 cancer and mesothelioma and excludes the other
18 asbestos-caused cancers like ovarian, laryngeal,
19 as well as pleural diseases like asbestosis. It
20 fails to examine the risk of talc-based industrial
21 consumer products contaminated with asbestos such
22 as crayons, toys, and paint that fall into the

1 EPA's jurisdiction.

2 **DR. DIANA WONG:** You have one more
3 minute.

4 **MS. LINDA REINSTEIN:** Sure. It
5 excludes the dangerous Libby amphibole which can
6 be found in 15 to 30 million homes in attic
7 insulation. As you can see there is broad
8 agreement that the draft risk evaluation contains
9 numerous exclusions that the result is dangerous
10 incomplete picture of asbestos risk. Americans
11 deserve more from the EPA's risk evaluation or
12 this will just be another attempt to regulate a
13 deadly carcinogen without success and the EPA will
14 have failed again. Thank you for listening to my
15 remarks.

16 **DR. DIANA WONG:** Thank you. Is
17 there any questions for the speaker? If not --

18 **DR. KENNETH PORTIER:** The panel --
19 Diane, the panel can raise their hand at any time
20 and we'll be able to recognize if you've got a
21 question. Thank you. Sorry.

22 **DR. DIANA WONG:** Okay. The next

1 speaker is --

2 **DR. KENNETH PORTIER:** Dr. Crump has
3 a question.

4 **DR. DIANA WONG:** Go ahead.

5 **DR. KENNY CRUMP:** Yes. I was sorry
6 to hear about your friend that died of
7 mesothelioma. I wondered if you had any
8 information to his exposure to asbestos.

9 **MS. LINDA REINSTEIN:** I prefer not
10 to speak of that individual exposure. But I can
11 speak about my own husband's that was both
12 occupational and non-occupational.

13 **DR. DIANA WONG:** Any more questions?
14 If not, the next speaker is Doug Gillepsie. Are
15 you online? Doug Gillepsie, are you online?
16 Okay. We will skip Doug. The next speaker is
17 Abdeljalil Mekkaoui. Again, the next speaker is
18 Abdeljalil Mekkaoui. Okay. The next speaker is
19 Denise Winder. Denise Winder, are you online?
20 Okay. The next speaker is David Garabrant. David
21 Garabrant.

22 **DR. DAVID GARABRANT:** Can you hear

1 me? I'm on -- I'm online.

2 **DR. DIANA WONG:** Okay. We have to
3 wait for your slides. Okay. You may start.

4 **DR. DAVID GARABRANT:** Okay. David
5 Garabrant, Emeritus Professor with the University
6 of Michigan. Next slide. I want to talk about
7 amosite and crocidolite asbestos were used at the
8 Marshville North Carolina plant that makes EPA's
9 reliance on that textile cohort study untenable.
10 Unarco operated the Marshville plant for 16 years
11 in '47 to 1963, made insubestos felt with amosite
12 and insutape tubing with amosite, wovenstone with
13 amosite, and braided packings with crocidolite.
14 Next slide.

15 This is documenting from a
16 deposition a response to interrogatories that are
17 40 years ago. Marshville, North Carolina was in
18 operation as part of Unarco from April 1, '47 to
19 October 31st '63. Next slide.

20 These are the products at issue.
21 Insubestos insulating felt was made with rovings
22 of amosite into tape, wovenstone with amosite

1 rovings, all of the products were manufactured
2 prior to '48 when Unarco bought the plant and they
3 were manufactured until '63 when Unarco sold the
4 Marshville plant. Next slide.

5 This is a deposition from a former
6 Unarco employee. Are you familiar with
7 insubestos? Yes. It was an extra thick asbestos
8 felt. Where was it made? I believe the start of
9 the process was in Marshville and it was finished
10 in Bloomington. Next slide.

11 This is a picture of Unarco
12 insubestos felt made with amosite asbestos rope.
13 Next slide. These are pictures of woven stone
14 made with woven amosite asbestos and insutape
15 which was a wrap on insulation. Next slide.

16 Wovenstone was woven on Loom number
17 five at Marshville in widths between 30 inches and
18 60 inches. Next slide. Insutape was listed in
19 inventory in Marshville, North Carolina in large
20 quantities. Next slide. Insutape tubing was
21 shipped even after Marshville was sold to
22 Manville. Manville made the insutape and shipped

1 it to Unarco in Bloomington in miles of it, 27,000
2 feet on the left here. Next slide.

3 Answers to plaintiff's
4 interrogatory. This was the braided asbestos
5 packings. They were treated with neoprene. They
6 were made at Marshville, shipped to Bloomington.
7 Number 193 was blue asbestos acid-resistant
8 packing made with blue asbestos yarn. I believe
9 that refers to crocidolite. Next slide.

10 Okay. Dr. Dement was aware of the
11 Manville documents in a deposition taken of him in
12 2012 and he never made any effort to look at them.
13 He was asked, "So bottom line, you're not saying
14 that amosite wasn't used at Marshville. All
15 you're saying is you haven't seen the records."
16 He said, "That's correct." Next slide.

17 Small amounts of tremolite were
18 found by Dr. Dement in the Marshville stuff but
19 Dr. Dement admitted that he had no fiber type by
20 transmission electron microscopy measurements
21 available for the time period during which Unarco
22 owned Marshville. At the bottom here it says,

1 "That's correct." Next slide.

2 It's my opinion EPA cannot rely on
3 the North Carolina textile studies. Your own
4 rules prohibit it because these were mixed
5 chrysotile, chrysolite, and amosite exposures.
6 Next slide. I want to point out that of the four
7 plants in the Loomis studies, plant one is the
8 only one that was pure chrysotile. It accounted
9 for 3,900 person-years out of 100,000 person-
10 years. The other two plants, three and four, were
11 mixed chrysotile and amphibole asbestos plants.
12 Next slide.

13 The --

14 **DR. DIANA WONG:** You have one more
15 minute.

16 **DR. DAVID GARABRANT:** Okay. These
17 will be the last few slides. The Quebec, Canada
18 chrysotile miners were downgraded for reasons that
19 made no sense compared to North Carolina.
20 Chrysotile -- the Canada study was superior in
21 every aspect. This is for lung cancer. Next
22 slide.

1 And this is for mesothelioma.
2 Again, chrysotile -- Quebec was downgraded. Next
3 slide. And there are numerous studies of motor
4 vehicle mechanics and brake repair workers that
5 show no increase in mesothelioma. They were not
6 considered by EPA and they must be considered.
7 It's inappropriate to base risk evaluation on long
8 fiber chrysotile in textiles but not consider
9 studies of workers exposed to short fiber
10 chrysotile in brakes. Thank you.

11 **DR. DIANA WONG:** Any questions for
12 Garabrant?

13 **DR. KENNETH PORTIER:** Dr. Crump, I
14 see your hand's still up. Do you have a question
15 or is that legacy hand up?

16 **DR. DIANA WONG:** No questions?
17 Okay. The next speaker is Elissa --

18 **DR. KENNY CRUMP:** Yes. I do have a
19 question.

20 **DR. DIANA WONG:** Okay. Go ahead.

21 **DR. KENNY CRUMP:** Excuse me. I do
22 have a question. This is Kenny Crump. I'm sorry.

1 I didn't know I had my hand up, but I do have a
2 question. I was wondering if any of the people on
3 the call that actually did work on the North
4 Carolina plants if they have any response to this
5 comment that was made.

6 **DR. DANA LOOMIS:** This is Dana
7 Loomis. I was the principal investigator of the
8 North Carolina studies and I can respond if you
9 would like. Do you have a specific question or
10 shall I just respond in general to things that the
11 last speaker said?

12 **DR. KENNY CRUMP:** I would just
13 prefer you to just -- what you -- whatever you'd
14 like to say about what the last speaker said.

15 **DR. DANA LOOMIS:** Well, okay. Well,
16 essentially, we don't disagree with the
17 information that he showed that Unarco produced
18 products that contained amphibole asbestos. And
19 that that was done during the period that they
20 owned the Marshville plant as well as before and
21 after that. However, the process -- none of the
22 information that he showed indicated that the

1 amosite components were produced at Marshville.

2 That was a standard textile spinning
3 and weaving operation and prior to Unarco's
4 ownership when Manville owned that plant, Unarco
5 produced the same products as the deposition that
6 the speaker showed indicated. Those products were
7 -- the woven textile components of those products
8 were indeed produced at Marshville then they were
9 sent on to Bloomington for finishing, which
10 including -- included adding the amosite elements.

11 There were really no process changes
12 at the Marshville plant over the period that it
13 operated, that is up until the late '80s or early
14 '90s. So it was just a standard textile spinning
15 and weaving operation much like other southern
16 textile mills. There's no documentary evidence,
17 including in the information that the speaker
18 showed, that amphibole asbestos fibers were used
19 there.

20 We made an extensive survey of all
21 of the available information at the time we did
22 the studies, including reports by North Carolina

1 dusty trades commission, various insurance
2 companies, the manufacturer themselves, the U.S.
3 Public Health Service. And none of those indicate
4 any use of amphibole asbestos or production of
5 products other than standard textile products in
6 the Marshville plant. So --

7 **DR. DIANA WONG:** Okay.

8 **DR. DANA LOOMIS:** -- we are -- I
9 would just add that, you know, we are aware of
10 various documents that have been shown purporting
11 to indicate that amphibole asbestos was used
12 there, but in fact, none of those documents
13 provide clear evidence that that was true in
14 contrast to all of the other information that we
15 have, which indicates they were not.

16 **DR. DIANA WONG:** Thank you. The
17 next speaker is Elissa Favata. Are you online?
18 Elissa Favata. If not, the next speaker is
19 Anthony Tweedale. Anthony Tweedale.

20 **DR. KENNETH PORTIER:** Diana, this is
21 Ken. We may need to give them a little bit more
22 time to come off of mute. It's a little harder

1 with WebEx to jump right in.

2 **MR. ALAN KAUFMAN:** And Ken, this is
3 Al Kaufman. I don't know if you called the roll,
4 but I got here a couple minutes late.

5 **DR. DIANA WONG:** Okay. Thank you.

6 **MR. ALAN KAUFMAN:** Thank you.

7 **DR. KENNETH PORTIER:** We forgot to
8 call the roll right after lunch. Thank you for
9 reminding me. We may do it right before break.

10 **DR. CONCEPCIÓN JIMÉNEZ-GONZALEZ:**
11 And Dr. Portier, I wonder if to help the speakers
12 -- this is Dr. Concepción Jiménez-Gonzalez by the
13 way. To help the public speakers if we have some
14 sort of running list of the speakers that we can
15 present so they get prepared before speaking
16 because it does take a little bit of time to come
17 out of mute. I wonder if that will help.

18 **DR. DIANA WONG:** I think we waited
19 long enough for Anthony Tweedale. The next
20 speaker is Christine Oliver.

21 **DR. CHRISTINE OLIVER:** I am on the
22 line. Can you hear me?

1 DR. DIANA WONG: Yes. Good.

2 DR. CHRISTINE OLIVER: Okay. Okay.

3 So my name is Christine Oliver. I'm an adjunct
4 professor at the Dalla Lana School of Public
5 Health at the University of Toronto and a
6 consultant in occupational and environmental
7 medicine in Boston where I was an Associate
8 Clinical Professor of Medicine at Harvard Medical
9 School and a practicing physician at the
10 Massachusetts General Hospital.

11 The EPA draft risk evaluation for
12 asbestos in its present form underestimates and
13 understates the true risk posed by asbestos. It
14 understates the risk with its singular focus on
15 only one of six asbestos fiber types regulated in
16 the U.S., namely chrysotile asbestos. The draft
17 risk evaluation ignores risks from amphibole
18 fibers in widespread use in the U.S. in insulation
19 and construction material, and vermiculite, and a
20 variety of other products.

21 It understates the risk by including
22 only two asbestos-related diseases, lung cancer,

1 and malignant mesothelioma. Excluded are cancers
2 of the ovary and larynx, and non-malignant
3 diseases such as asbestosis and diseases of the
4 pleura. The draft risk evaluation underestimates
5 the risks in at least two ways.

6 The statistical model chosen to
7 define the dose-response relationship between
8 asbestos and disease is less protective of the
9 public health than the model used by the EPA and
10 other federal regulatory agencies for decades,
11 namely the linear model; a validated and accepted
12 model showing no dose of asbestos below which
13 there is no risk for related diseases. In other
14 words, no threshold.

15 The inhalation unit risk is
16 calculated using data from only two epidemiologic
17 studies whereas unit risk was previously
18 calculated using data from multiple scientific
19 studies. The newly calculated IUR is considerably
20 lower and therefore less protected -- protective
21 than the one previously calculated.

22 Finally, and importantly, the draft

1 risk evaluation sets legacy asbestos aside for
2 another and an uncertain date. Legacy asbestos is
3 asbestos leftover from asbestos contained
4 materials previously used in public, private, and
5 commercial buildings, homes, and industrial
6 workplaces, and other settings as well as in a
7 variety of commonly used products.

8 In failing to consider legacy
9 asbestos in a timely way, EPA ignores asbestos
10 that presents the greatest risk to the greatest
11 member of Americans presently. These Americans
12 include students, teachers, and custodians in
13 public schools; clerical workers, administrators,
14 and maintenance personnel in commercial and
15 government office buildings; healthcare workers
16 and patients in hospitals and out-patient clinics,
17 and workers in a variety of industrial settings.

18 The EPA's singular focus on
19 mortality versus incidence underestimates the
20 health risks to the public for at least two
21 important reasons. First, incidence of asbestos-
22 related disease is far more prevalent than

1 asbestos-related mortality in developed countries,
2 particularly with regard to non-malignant diseases
3 such as asbestosis.

4 Second, the EPA uses outdated
5 survival data for lung cancer and malignant
6 mesothelioma to characterize survival for these
7 two cancers as short term. While this was the
8 case at one time, it is no longer as low dose
9 chest CT screening, LDCT screening for lung cancer
10 is reducing and has reduced mortality and improved
11 treatment for both cancers and prolongs survival.
12 This change will only increase in the future. The
13 limitations of this EPA draft risk evaluation for
14 asbestos clearly demonstrate that the only way to
15 protect the American public from asbestos is to
16 ban it.

17 **DR. DIANA WONG:** Thank you. Any
18 questions for Christina Oliver.

19 **DR. STEVEN MARKOWITZ:** Steve
20 Markowitz. I have a question. Actually, not for
21 Dr. Oliver but I was wondering whether Dr. Loomis
22 and Dr. Stayner might want to comment on Dr.

1 Oliver's point about the use of the exponential
2 versus linear model.

3 **DR. STAYNER:** This is Dr. Stayner.
4 The main reasons for us using exponential model
5 was that it fit the data better. But there's no
6 actual good reason for one model or another in
7 terms of biology, other than looking at model fit.
8 I'm not sure what you would base your choice on.

9 **DR. STEVEN MARKOWITZ:** Thank you.

10 **DR. DIANA WONG:** Any more questions?

11 **DR. KENNY CRUMP:** I have -- I have
12 one. I have a comment.

13 **DR. DIANA WONG:** All right.

14 **DR. KENNY CRUMP:** I --

15 **DR. DIANA WONG:** Go ahead.

16 **DR. KENNY CRUMP:** This is Kenny --
17 Kenny Crump. The question -- the issue about the
18 linear model versus the exponential model. Dr.
19 Elliott in her analysis of the North and South
20 Carolina data actually used both models. We can
21 look at that and see how much they differ. Both
22 models are low dose linear. But the linear model

1 produced a KL about 25 percent larger than the
2 exponential model. So that's the difference we're
3 talking about here, about 25 percent between the
4 two models.

5 **DR. DIANA WONG:** Thank you. Any
6 more questions? If no more, we go on to the next
7 speaker. The next --

8 **DR. HENRY ANDERSON:** This is Dr.
9 Anderson. I have my hand up.

10 **DR. DIANA WONG:** Okay. Go ahead.

11 **DR. HENRY ANDERSON:** Yeah. I just
12 wanted -- I noticed that there was a public
13 comment submitted by Dr. Dement. And I think he
14 raised the issue that all of his data from both
15 plants were not utilized in the Elliott paper and
16 that when you include the rest of the data, then
17 the linear model kind of better fit than the
18 exponential data. So I was wondering whether the
19 EPA consultants had seen Dr. Dement's letter.

20 **DR. DANA LOOMIS:** I haven't seen it.
21 This is Dana Loomis. I haven't seen Dement's
22 comment. I'm not sure what he would mean because

1 there were -- the only exclusions in the combined
2 analysis by Elliott were just to make the dates
3 and inclusion criteria for the two cohorts match.
4 So I'm not sure what additional data would --
5 could be included. It wouldn't be very much
6 information. So I'd be very interested to see
7 that comment from Dr. Dement.

8 **DR. HENRY ANDERSON:** Yeah. Probably
9 hasn't been posted yet so it only came to us at
10 the last moment. But I think that'd be
11 interesting to look at. Thank you.

12 **DR. DIANA WONG:** Thank you.

13 **DR. KENNETH PORTIER:** Diana, this is
14 Ken -- Ken Portier. We got a note from the
15 technical staff that you need to give them a few
16 minutes to find the commenter's name to allow them
17 to connect. So it's taking a little bit longer
18 and because we have such a long list it takes them
19 a while to find the names. So they have it under
20 control. Plus, two of the commenters that we
21 called were incorrectly connected and they're
22 trying to work to reconnect them, so we may have

1 to loop back to the beginning of the list to
2 capture some of those people that we missed.

3 **DR. DIANA WONG:** Okay. We can go
4 back.

5 **DR. KENNETH PORTIER:** Especially --

6 **DR. DIANA WONG:** We can go back.

7 **DR. KENNETH PORTIER:** Yep.

8 **DR. DIANA WONG:** Doug Gillepsie. Is
9 he online now?

10 **MR. MARTIN ALVARADO:** He is online
11 and I have just unmuted him.

12 **DR. DIANA WONG:** Okay. So you're
13 ready to speak? You may start.

14 **DR. KENNETH PORTIER:** He may have
15 his phone muted.

16 **MR. MARTIN ALVARADO:** Doug
17 Gillepsie, your phone might be muted or your
18 computer audio. He is connected and he has been
19 unmuted in WebEx but for whatever reason --

20 **DR. DIANA WONG:** He's not speaking.

21 **MR. MARTIN ALVARADO:** -- reason,
22 yeah, or his line is muted somehow on his end.

1 You may want to consider moving on and we can try
2 reaching out to him again.

3 **DR. DIANA WONG:** Do we have the next
4 speaker, Abdeljalil Mekkaoui? I'm going back now.
5 Do you have him online?

6 **MR. MARTIN ALVARADO:** He is logged
7 in but not to audio.

8 **DR. DIANA WONG:** He is not on audio.

9 **MR. MARTIN ALVARADO:** Right. And
10 he's not responding to our messages to him
11 offline.

12 **DR. DIANA WONG:** So how about going
13 back to Doug Gillepsie. Is he on mute now?

14 **MR. MARTIN ALVARADO:** I just
15 unmuted him again. Mr. Gillepsie, if you can go
16 ahead and speak. You have been unmuted in WebEx.
17 He is either not speaking or something's wrong on
18 his end with audio. And by that, I mean his
19 computer audio might be muted, he might not have a
20 microphone on his computer. He may need to call
21 in via telephone.

22 **DR. DIANA WONG:** Well, he needs to

1 try another way. We have to move on to the next
2 speaker.

3 **MR. MARTIN ALVARADO:** Okay.

4 **DR. DIANA WONG:** Denise Winder. Do
5 you have her online?

6 **MR. MARTIN ALVARADO:** We do have her
7 online. She is connected twice. I'm able to
8 unmute one of her lines and the other line I am
9 not. I've gone ahead and unmuted the one line I
10 am able to. Ms. Winder, if you could please speak
11 up. Make sure you are unmuted through your
12 computer and that you have a microphone. Again,
13 she is unmuted but we can't hear anything from her
14 so either her line is muted or she -- her
15 microphone is -- might be faulty.

16 **DR. DIANA WONG:** So who else do you
17 have online right now? Elissa Favata?

18 **MR. MARTIN ALVARADO:** We're not
19 seeing her. So, she was on earlier, but she
20 dropped off.

21 **DR. DIANA WONG:** It was a drop off?
22 Okay?

1 **MR. MARTIN ALVARADO:** Elissa was
2 next and she's not available. Alec Farquhar was
3 next and he's not available. Nicholas Chartres is
4 available.

5 **DR. DIANA WONG:** Can you slide
6 those? Do you have his slides?

7 **MR. MARTIN ALVARADO:** Yes. And he
8 will be unmuted momentarily.

9 **DR. NICHOLAS CHARTRES:** Ah, yes.
10 Can you hear me?

11 **DR. DIANA WONG:** Yes. We can hear
12 you. You may start.

13 **DR. NICHOLAS CHARTRES:** Thank you.
14 Good morning. My name is Nicholas Chartres. I'm
15 the Associate Director of Science and Policy at
16 the Program Reproductive Health and the
17 Environment. Sorry. Is that my -- is there an
18 echo there?

19 **MR. MARTIN ALVARADO:** There is an
20 echo. Are you connected through audio twice? If
21 so, can you either mute your computer or mute your
22 audio and pick up your phone?

1 DR. KENNETH PORTIER: Now he's muted
2 in WebEx.

3 DR. DIANA WONG: Should I try
4 again?

5 DR. KENNETH PORTIER: No. He's
6 available now.

7 DR. DIANA WONG: Okay. Go ahead.

8 DR. NICHOLAS CHARTRES: Okay. Thank
9 you. Good morning. My name is Nicholas Chartres
10 and I'm the Associate Director of Science and
11 Policy at the Program for Reproductive Health and
12 the Environment, University of California San
13 Francisco. My comments will focus on our concerns
14 with the application of systematic review in the
15 asbestos draft risk evaluation. Next slide,
16 please.

17 I have no conflicts to disclose.
18 Next slide, please. EPA is required by the TSCA
19 statute to use the best available science and the
20 weight of the scientific evidence to make
21 decisions about chemical risks. EPA defined the
22 weight of the scientific evidence in its 2017 risk

1 evaluation rule as a systematic review method that
2 uses a pre-established protocol to
3 comprehensively, objectively, transparently, and
4 consistently identify and evaluate any stream of
5 evidence including strengths, limitations, and
6 relevance of each study and integrate evidence as
7 necessary and appropriate.

8 However, EPA states in the draft
9 risk evaluation for asbestos that although EPA
10 will make an effort to adopt as many practices as
11 practical from the systematic review community,
12 EPA expects modifications to the process to ensure
13 timely regulatory decision making under the
14 aggressive timelines of the statute.

15 Authoritative bodies, U.S. agencies, and academic
16 scientists have developed and implemented
17 validated environmental systematic review methods
18 including the NTP's Office of Health Assessment
19 and Translation and UCSS navigation guide. If the
20 EPA uses one of these aforementioned methods the
21 Agency would not have to make an effort to adopt
22 as many practices as practical. Next slide,

1 please.

2 We heard the previous comments or
3 recommendations made by the SACC to EPA in the
4 previous draft risk evaluations across each step
5 of the systematic review process that EPA has
6 failed to address once again in the asbestos draft
7 risk evaluation, therefore leading to a
8 potentially biased evaluation of the evidence.

9 These include; describe and query the rationale
10 for the differences to the TSCA systematic review
11 relative to other peer-reviewed systematic review
12 approaches currently in use. And since large
13 percentages of studies are excluded, the number of
14 items being rejected for each criterion should be
15 summarized to enable readers to determine why
16 studies were excluded. Next slide, please.

17 Today we will highlight that EPA is
18 not systematically reviewing the studies relied on
19 in the evaluation and is inappropriately excluding
20 a significant proportion of the body of evidence.
21 Specifically, EPA has failed to one; account for
22 all references identified in the literature search

1 for human health hazards, and two; offer
2 sufficient justification for why each study has
3 been excluded the full text stage.

4 In order for EPA to adequately
5 address these issues relating to its lack of
6 transparency and mechanical references identified
7 in the literature search and applying a predefined
8 eligibility criteria to references in the
9 literature search, EPA must immediately implement
10 protocols for all future draft risk evaluations.
11 Next slide, please.

12 In the asbestos draft risk
13 evaluation, it states that the literature search
14 and screening strategy is specifically applied to
15 asbestos, it's described in the strategy for
16 conducting literature searches for asbestos,
17 supplemental document to a TSCA scope document,
18 and the results of the title and abstract
19 screening process were published in the asbestos
20 bibliography.

21 For studies determined to be on-
22 topic after title and abstract screening, EPA

1 conducted a full text screen to exclude further
2 references that were not relevant to the risk
3 evaluation. However, in asbestos bibliography
4 supplemental file, there are 344 pages of on-topic
5 studies following the title and abstract screening
6 of the human health hazard literature search
7 results with approximately 30 studies per page
8 totaling approximately 10,320 studies.

9 However, in the asbestos draft risk
10 evaluation Figure 1-8 Key Supporting Data Sources
11 for Human Health Hazards shown here, EPA has
12 excluded this step and failed to show how many on-
13 topic studies went through full text screening.
14 The Agency has also failed to provide a rationale
15 as to why these studies were excluded in this
16 step. Next slide, please.

17 The numbers shown here Figure 1-8
18 Key Supporting Data Sources for Human Health
19 Hazards, they do not accurately reflect the number
20 at each step and do not account for all references
21 that go through data quality evaluation as cited
22 in the systematic review supplemental file data

1 quality evaluation of human health hazard studies,
2 mesothelioma, and lung cancer studies. In the
3 data search results shown here --

4 **DR. DIANA WONG:** You have one more
5 minute.

6 **DR. NICHOLAS CHARTRES:** there are
7 24,050 data sources, three sources are later
8 incorporated as key supporting data in the data
9 verification step. Mathematically, then 24,047
10 data sources should move to data screening step.
11 Yet only 24,036 do so leaving 11 studies EPA is
12 not -- EPA has excluded without justification. In
13 the data screening step, EPA states there are
14 24,036 data sources with 24,012 excluded.
15 Therefore, there should be 24 data sources moved
16 to the data evaluation step, yet only 23 moved to
17 the step.

18 Finally, in the data evaluation
19 step, EPA states that it evaluated 26 data
20 sources. However, in the systematic review
21 supplemental file data quality evaluation of human
22 health hazards Table 2.1, EPA's evaluated 44 data

1 sources for asbestos exposure and lung cancer
2 incidents, which also includes the studies on
3 asbestos exposure and mesothelioma incidents.
4 Therefore, there are 18 data sources EPA has not
5 accounted for in the draft risk evaluation without
6 any explanation for their exclusion. Thank you
7 very much for your time.

8 **DR. DIANA WONG:** Any questions for
9 the speaker? If not, we'll move on to the next
10 speaker. Do you have Robyn Brooks online?

11 **MS. ROBYN BROOKS:** Hi. Are you able
12 to hear me?

13 **DR. DIANA WONG:** Yes.

14 **MS. ROBYN BROOKS:** Okay. Thank you.
15 Good afternoon. My name is Robyn Brooks and I am
16 Vice President, Health, Environment, Safety and
17 Security at the Chlorine Institute. The Chlorine
18 Institute is a 185 member not-for-profit trade
19 association of chlor-alkali producers worldwide.
20 The Institute's American producer members account
21 for 91 percent of the total chlorine production
22 capacity in the U.S. The Institute's mission

1 chemicals are used throughout the U.S. economy and
2 are paramount to the protection of public health.

3 Chlorine chemistry is used in many
4 applications including municipal drinking water
5 disinfection and as a raw material to produce
6 sodium hypochlorite, also known as household
7 bleach. It is the raw material or intermediate
8 for 88 percent of pharmaceuticals produced in the
9 U.S. Chlorine also plays a role in the production
10 of polyvinyl chloride or PVC, sterile packaging,
11 neoprene wetsuits and electronic instruments among
12 other applications.

13 Within the U.S. there are 10 large
14 chlorine production facilities that use asbestos
15 in the process which account for 38 percent of
16 installed capacity in the U.S. Asbestos is an
17 ideal substance to provide this barrier during the
18 production process because of its mechanical
19 strength, chemical resistance to both acids and
20 bases, low electrical resistance, and a physical
21 structure that minimizes backflow. It can last up
22 to a year or longer in the -- in production before

1 changing the spent asbestos if necessary.

2 The facilities that use asbestos
3 within the chlorine production process understands
4 chlorine's important role as a building block for
5 the chemical industry and have a robust
6 engineering and administrative controls to handle
7 all parts of production, including handling
8 asbestos. The majority of the time asbestos is
9 used in the process is non-friable. As you can
10 see from the infographic and description that was
11 previously submitted to the docket by our
12 colleagues at the American Chemistry Council, the
13 asbestos becomes non-friable early in the process
14 before it is inserted into a closed electrolytic
15 cell where it stays in place for a year or longer.

16 CI commends the repeated asserted by
17 EPA that the use of controls and respirators
18 mitigates the exposure risk to acceptable levels.
19 However, EPA applied the respirator protection for
20 the entire shift or not at all. This does not
21 consider the respirator use is based upon years of
22 industrial hygiene data and its use may not be

1 required for the entire shift due to other
2 controls in place and many steps during the
3 process, including the use of modifiers and wet
4 methods to render and maintain asbestos in a non-
5 friable state. The engineering administrative
6 controls are summarized in my written comments.

7 All chlorine production facilities
8 that utilize asbestos diaphragms have detailed
9 procedures and controls for the specially trained
10 workers who handle it. Facility process controls
11 and procedures include enclosed glove boxes and
12 negative pressure handling systems or remote bag
13 handling, HEPA filters, the use of modifiers, and
14 wet methods to render and maintain asbestos in a
15 non-friable state, and administrative controls
16 such as restrictive access and permitting
17 requirements. These controls in addition to the
18 PPE requirements such as respirators maintain
19 controls that comply with the Clean Air Act and
20 OSHA standards.

21 Respiratory protection is used by
22 employees engaged in asbestos work as an

1 additional protection on top of the engineering
2 and administrative controls. A comprehensive
3 respiratory protection program has been
4 implemented as required by OSHA which includes a
5 written program, training, and fit testing for all
6 employees required to use respiratory protection.

7 The asbestos procedures dictate
8 minimum PPE requirements for specific tasks such
9 as respiratory protection. The respiratory
10 protection program outlines how respirators are
11 properly selected, worn, and maintained following
12 OSHA standards.

13 I also want to draw your attention
14 to EPA's inaccurate claim that janitorial staff
15 are occupational non-users or ONUs. Only separate
16 non-process area break rooms or office space which
17 are in designated clean areas are cleaned by non-
18 operator janitorial staff. At most, once per day.
19 All janitorial, like housekeeping activities, in
20 asbestos process areas within the restricted
21 areas, are handled by workers who perform the
22 asbestos handling tasks with appropriate PPE in

1 compliance with site procedures. In addition,
2 maintenance workers should not be considered
3 occupational non-users. OSHA specifically
4 mentions --

5 **DR. DIANA WONG:** You have one more
6 minute.

7 **MS. ROBYN BROOKS:** Thank you.
8 Maintenance workers and repair technicians in the
9 asbestos standard. When maintenance assistance is
10 required, this triggers the safe work permitting
11 process which requires a review of the job scope
12 and minimum personal protective equipment as
13 required for the task. Before maintenance occurs
14 in restricted areas, process area supervisors
15 establish task base requirements by using specific
16 safe work permitting requirements including any
17 required PPE.

18 The chlor-alkali industry has a
19 proven record of the safe use of asbestos within a
20 chlorine production process. CI members believe
21 that with the effective engineering and
22 administrative controls currently in place,

1 including respiratory protection, a scientifically
2 based risk assessment of the chlor-alkali
3 industry's use of asbestos in chlorine production
4 will demonstrate that this does not pose an
5 unreasonable health risk to workers. Thank you
6 for your time and attention.

7 **DR. DIANA WONG:** Thank you. Any
8 questions for the speaker? If not, the next
9 speaker is Chacko Matthew. Do we have him online?

10 **DR. HENRY ANDERSON:** I have a
11 question for the last presenter.

12 **DR. DIANA WONG:** Go ahead.

13 **DR. HENRY ANDERSON:** Yes. I was
14 wondering, did you send your monitoring plan and
15 strategy to EPA when you sent them the test
16 results?

17 **MS. ROBYN BROOKS:** We do not -- I
18 did not include that in the written comments. I'd
19 be happy to follow up offline. We're with the
20 Chlorine Institute. In terms of the monitoring
21 results, that was actually done through our
22 colleagues at the American Chemistry Council.

1 **DR. HENRY ANDERSON:** Okay. Well, I
2 think it would be helpful to -- I mean, it's --
3 been doing so much monitoring result ought to be
4 helpful to know what their industrial hygiene plan
5 is, how often, and that sort of thing.

6 **MS. ROBYN BROOKS:** Okay. I would
7 have to confer with the members but it may vary
8 across the different facilities, but I'd be happy
9 to follow up.

10 **DR. HENRY ANDERSON:** Thank you.

11 **DR. DIANA WONG:** Okay. Still --

12 **DR. KENNETH PORTIER:** Diana --

13 **DR. DIANA WONG:** Yes, go ahead.

14 **DR. KENNETH PORTIER:** This is Ken.
15 Diana, Celeste Monforton with American Public
16 Health Association is connected now and ready to
17 speak. I don't remember if --

18 **DR. DIANA WONG:** Oh, okay.
19 Good.

20 **DR. KENNETH PORTIER:** -- you called
21 her name.

22 **DR. DIANA WONG:** Okay. So we'll go

1 back to --

2 **MS. CELESTE MONFORTON:** This is
3 Celeste, can you hear me?

4 **DR. DIANA WONG:** Yes. You may start
5 when you're ready.

6 **MS. CELESTE MONFORTON:** Terrific.
7 Thank you so much. I am Celeste Monforton. I'm a
8 lecturer in public health at Texas State
9 University and I'm speaking on behalf of the
10 American Public Health Association. APHA is a
11 diverse community of public health professionals
12 who champion the health of all people. We are the
13 only organization that combines 150 years'
14 perspective with broad-based membership who speak
15 out for public health issues and policies backed
16 by science.

17 Dr. Christine Oliver who spoke just
18 moments ago is a member of APHA and we concur with
19 her comments. We are approaching the four year
20 anniversary of the Lautenberg Chemical Safety Act,
21 a legislative accomplishment where asbestos was
22 used to illustrate the profound need for a

1 stronger law. Since that time, APHA has submitted
2 comments and met with EPA at every opportunity to
3 explain the grave health risk of exposure to
4 asbestos, particularly to workers and to
5 communities of color that already experience
6 health inequities because of income, race, and
7 ethnicity.

8 In May 2019, APHA testified before
9 Congress to express our deep concern that EPA's
10 decisions to date would fail to address the threat
11 asbestos poses to public health. Our concerns
12 were reinforced when we saw the Agency's draft
13 risk evaluation. TSCA requires EPA to look
14 holistically at all sources of exposure that
15 contribute to risk. Yet this risk evaluation
16 fails to consider the millions of metric tons of
17 asbestos already in use in buildings and
18 infrastructure. This means, for example, ignoring
19 the risk to communities where families live in
20 substandard housing and children go to school
21 where cities have disinvested in public buildings.
22 And the risk is increasing because of extreme

1 weather events brought on by climate change.

2 Failing to include the potential
3 exposures from legacy uses of asbestos understates
4 the risk to children and adults. EPA must combine
5 the potential exposure to asbestos from all
6 sources in order to evaluate the total risk. I
7 want to highlight just a few items from APHA's
8 written comments.

9 EPA indicates it reviewed asbestos
10 air concentrations from OSHA inspections. EPA
11 does not mention however whether it reviewed the
12 data from the 22 states and territories that
13 operate their own OSHA programs. Most of these
14 agencies have their own analytical laboratories
15 and maintain their own data of sampling results.

16 EPA should also examine asbestos
17 violations data from OSHA and these state OSHA
18 programs, not merely the air sampling data.
19 Violations provide evidence of worker's potential
20 exposure to asbestos and citations are not
21 contingent on violating the PEL. When reviewing
22 workplace inspection data, EPA must assume that

1 samples collected by inspectors are the best-case
2 scenario. The safest day for workers is when an
3 inspector is present. It's all the other days we
4 need to worry about.

5 The central goal of the 2016
6 amendments to TSCA is for risk evaluation to
7 examine all of the diverse pathways and modes of
8 release that may result in harm, for example, from
9 disposal. We made particular note of information
10 provided at industry comments asserting the
11 meticulous way that asbestos-containing material
12 is disposed of, like double bagging and following
13 all state and local requirements. What we don't
14 see in the risk evaluation is how EPA reconciled
15 these claims with other information in the record.
16 For example, a consultant at a gasket manufacturer
17 wrote about observing unused scrap pieces of
18 asbestos-containing material which had been placed
19 in a dumpster to be --

20 **DR. DIANA WONG:** You have one more
21 minute.

22 **MS. CELESTE MONFORTON:** Thank you.

1 Placed in a dumpster to be disposed of with normal
2 plant waste. We could not identify how EPA used
3 this information in its risk evaluation to look at
4 disposal risks at gasket plants.

5 EPA should include assumptions in
6 its risk evaluation to adjust for actual
7 practices, not the best practices offered by
8 entities with interests in the outcome of the risk
9 evaluation. The benefit of doubt must bend
10 towards public health. More than a decade ago
11 APHA called for a complete ban on asbestos. We
12 remain steadfast in that position and it's EPA's
13 responsibility to do it expeditiously. Thank you
14 so much.

15 **DR. DIANA WONG:** Thank you. Any
16 questions for the speaker? If no questions we go
17 back to Chacko Mathew. Is he online?

18 **MR. CHACKO MATHEW:** Can you hear me?

19 **DR. DIANA WONG:** Yes.

20 **MR. CHACKO MATHEW:** Oh. Okay. My
21 name is Chacko Mathew. I'm from United Industries
22 Unlimited. I am a retired industrial hygienist

1 from the city of New York. I just wanted to
2 express some of my concerns regarding the risk
3 assessment, this draft risk evaluation conducted
4 by the EPA.

5 The following concerns that I have,
6 I think EPA failed to list all the new consumer
7 products that will be coming out of this draft
8 evaluation process. And it failed to list all
9 available types of asbestos that will be used. It
10 also failed to conduct a risk assessment on all
11 available types of asbestos that will be used on
12 the new consumer products.

13 And it failed to conduct a separate
14 study on the fiber and non-fiber material that
15 will be made available. And failed to conduct a
16 study for the potential for a non-friable material
17 that has a potential to become friable. And at
18 the same time, also failed to identify the
19 consumer product that has the potential for damage
20 and potential for exposure.

21 And also the impact -- the finance -
22 - by making all this non-friable risk that --

1 plenty of non-friable material is already
2 available in the market that is costing a lot of
3 government because of the OSHA roles, and there
4 are other regulatory agencies who are local who
5 are costing municipalities and building owners a
6 lot of money. Even now to remove by introducing
7 or putting more non-friable materials available is
8 going to cost a lot more to remove out the load.
9 And it's going to cost a lot more to local
10 municipalities and the commercial owners. These
11 are my concerns.

12 The other two more things I want to
13 add is I think this study, given the latency
14 period of 10 to 30 years and the short time that
15 the draft evaluation committee is in existence --
16 I think it's only the last three years -- I think
17 it is a short time to conclude all the draft
18 evaluation given the latest latency period of the
19 asbestos. And also given the dose-response
20 relationship of asbestos, I think that this in
21 itself is concluding too short. I insist on the
22 latency period and the dose-response relationship

1 to insist that this study has gone too fast to
2 conclude. Thank you for listening.

3 **DR. DIANA WONG:** Thank you. Any
4 questions for the speaker? If not, the next
5 speaker is Nathan Borsheim. Is he online?

6 **MR. MARTIN ALVARADO:** He does not
7 appear to be online Diana.

8 **DR. DIANA WONG:** Nathan Borsheim.

9 **MR. MARTIN ALVARADO:** Nathan
10 Borsheim is not connected, Diana.

11 **DR. DIANA WONG:** Okay. Next speaker
12 will be Dennis Paustenbach and he has slides.

13 **DR. DENNIS PAUSTENBACH:** Okay.
14 Diana, do you want me to go on mute?

15 **DR. DIANA WONG:** He can start if
16 he's there. The slides are there.

17 **DR. DENNIS PAUSTENBACH:** Okay.
18 Good. My name's Dennis Paustenbach. I am an
19 independent consultant.

20 **DR. KENNETH PORTIER:** Dr.
21 Paustenbach?

22 **DR. DENNIS PAUSTENBACH:** Yes.

1 **DR. KENNETH PORTIER:** You either
2 have to hang up your phone or your computer.
3 You're getting feedback.

4 **DR. DENNIS PAUSTENBACH:** I'm happy
5 to do that.

6 **DR. KENNETH PORTIER:** Your phone is
7 better.

8 **DR. DENNIS PAUSTENBACH:** Can you
9 hear me now?

10 **DR. KENNETH PORTIER:** Perfect.

11 **DR. DENNIS PAUSTENBACH:** Oh, great.

12 **DR. KENNETH PORTIER:** Please
13 proceed. Sorry.

14 **DR. DENNIS PAUSTENBACH:** Thanks for
15 bringing that to my attention. Next slide. So as
16 I mentioned, I'm a chemical engineer and also an
17 industrial hygienist as well as an inhalation
18 toxicologist. I've been studying asbestos pretty
19 much every day for the last 20 years. Next slide.

20 I have a number of comments, as
21 you're aware. I submitted 139 pages of comments
22 and documents. The first thing I would bring to

1 your attention is I found nearly 100 relevant
2 documents that were not cited in this evaluation
3 which I think deserve to be looked at. Next.

4 The focus of the document is of
5 course gaskets and brakes. I think what's
6 probably misunderstood is this a forward-looking
7 document? Most of the comments thus far have been
8 concerned about past exposures or exposures that
9 are, I guess, hypothesized today. But this is a
10 forward-looking document.

11 The foundation of the assessment is
12 that there are many people exposed to these two
13 family of products, that I don't think that's true
14 going forward. It's predicted there's 1.5 million
15 persons per year exposed in this document, and I
16 think it's unlikely because I've looked at this
17 for 20 years and I think there's unlikely to be
18 more than 100 persons exposed to that family of
19 encapsulated materials. And I have explained why
20 in my comments.

21 Also, the doses are exceedingly low
22 for the persons going forward for chrysotile. I

1 would estimate they're 1,000 to 10,000-fold less
2 than those that might cause asbestos-related
3 disease. Next.

4 I think it's relatively well
5 accepted that if chrysotile can cause
6 mesothelioma, which I recognize is disputed every
7 day, it probably only does so at doses that are in
8 the vicinity that cause asbestosis. This is
9 different than the amphiboles which needs to be
10 emphasized throughout the current document.

11 And the other fact that seems to be
12 mischaracterized, I think, is that this is almost
13 certainly a threshold carcinogen. Many papers
14 support that view. And EPA's own panels over the
15 last 20 years seem to support that view in large
16 measure. Next.

17 So concerning brakes, we have to
18 understand that the people that wrote those
19 sections of the document are not as sensitive to
20 the difference between disc brakes and drum
21 brakes. Over the last 20 or 30 years, most cars
22 in the U.S. have gone to disc brakes. There are

1 vanishingly the few, if any, current new cars with
2 drum brakes.

3 The only ones that were very popular
4 in about 1984, 1985, that's a long time ago and
5 they're rarely worked on. When they are, they're
6 usually commercially available brakes, not
7 imports, with one percent of imports that are
8 mentioned in the document.

9 I tried to buy these brakes that
10 were discussed about being available on the
11 internet quite frequently over the last 20 years
12 and I've looked for them to see if they have
13 asbestos. And I have yet to find my first set of
14 brakes after all these years trying to find them.
15 I know there's been a rumor for years and I read
16 the one percent number that was submitted a few
17 years ago. I don't think it's right. Even if it
18 were, it doesn't make any difference because these
19 are for disc brakes. That's not recognized by the
20 authors of the document.

21 The next thing is, in the last three
22 years, Dr. Bernstein has shown, I think, pretty

1 conclusively that these fibers that are soaked in
2 phenolic resin appear to be biologically inert for
3 the asbestos-related disease mechanisms.

4 **DR. DIANA WONG:** You have one more
5 minute.

6 **DR. DENNIS PAUSTENBACH:** Thank you.
7 Next slide. I'll just go say that the gaskets are
8 no longer sold, haven't been sold for 40 years.
9 Most were destroyed except for two industries.
10 They have fully limited use and I would dare say
11 that there is less than one thousands of one
12 percent of new gaskets that might contain
13 asbestos. Again, I've looked for them for years
14 and can't find them. Any old gaskets were
15 replaced usually 20 or 30 years ago. They're
16 very, very hard to find. Next.

17 The epidemiology has already been
18 covered fairly well by Dr. Garabrant and I would
19 encourage you to look carefully at my comments. I
20 would say in closing on this slide that I agree
21 with Dr. Garabrant that vehicle mechanic studies
22 are the really most powerful ones for

1 understanding the cohorts that EPA cares about in
2 this document. Next slide.

3 **DR. DIANA WONG:** The time is up.

4 **DR. DENNIS PAUSTENBACH:** All right.
5 I'll just close by saying that I think the fiber
6 risk discussion is inappropriate. Happy to take
7 questions.

8 **DR. DIANA WONG:** Any questions for
9 the speaker? Any questions? If not, the next
10 speaker is Greg Brorby. Is he online?

11 **MR. MARTIN ALVARADO:** He is, and I
12 have unmuted him.

13 **DR. DIANA WONG:** Okay.

14 **MR. GREG BRORBY:** Hello. Can you
15 hear me?

16 **DR. DIANA WONG:** Yes. You can
17 start.

18 **MR. GREG BRORBY:** Oh. Good
19 afternoon. My name is Greg Brorby. I am a Senior
20 Consultant with ToxStrategies and I'm presenting
21 today on behalf of the American Chemistry Council.
22 We appreciate the opportunity to provide brief

1 comments related to EPA's assessment of
2 occupational exposure in the chlor-alkali
3 industry. These comments supplement written
4 comments previously submitted by ACC and oral
5 comments to be provided this afternoon by Mr.
6 Steve Risotto.

7 First, EPA did not use a consistent
8 method for assigning values to sample results
9 below the limit of detection or LOD.
10 Specifically, in worksheets for Axiall Westlake
11 and Olin EPA replaced non-detect values by the LOD
12 divided by two or the square root of two,
13 depending on the calculated geometric standard
14 deviation. However, in the worksheet for
15 Occidental, EPA used the full value of the
16 detection limit as the proxy for non-detect values
17 for this facility. This is an important issue
18 that should be remedied given that these non-
19 detect observations represent approximately 60
20 percent of the values in this worksheet.

21 Further, in the all data worksheet
22 it appears EPA also included the full value of the

1 LOD as the proxy non-detect values for the Olin
2 results, even though EPA assigned a value of LOD
3 divided by two or the square root of two to non-
4 detect data in the Olin worksheets. This
5 apparently inadvertent mistake should also be
6 corrected.

7 Second, EPA needs to redefine the
8 combination of exposure factors for the central
9 tendency and high-end scenarios such as they
10 better represent worker exposures in the chlor-
11 alkali industry. Specifically, the risk estimates
12 presented by EPA are not representative of central
13 tendency in high-end estimates. But rather
14 reflect a combination of either a central tendency
15 exposure point concentration and maximum exposure
16 frequency and duration assumptions, or an extreme
17 high-end exposure point concentration in the same
18 maximum exposure frequency and duration
19 assumptions. This is contrary to long-standing
20 EPA guidance that recommends a combination of
21 exposure assumptions from the range of probable
22 values to be used to derive central tendency and

1 high-end risk estimates.

2 Regarding the exposure point
3 concentration, the Agency should use the mean
4 concentration instead of the 95th percentile for
5 the high-end scenario consistent with EPA's 2008
6 framework for investigating asbestos-contaminated
7 superfund sites. We refer you to ACC's detailed
8 discussion of this issue in their written
9 comments. This change alone would reduce the risk
10 by approximately a factor of four for this
11 scenario.

12 Both age at first exposure and
13 exposure duration affect the less than lifetime
14 unit risk factor. While there are no specific age
15 requirements for employment in the chemical
16 industry, ACC's members generally require
17 employees to have a process technology degree or
18 relevant work experience. Realistically, an
19 individual would likely be at least 18 to 20 years
20 of age to meet the industry's employment
21 requirement.

22 Furthermore, EPA's assumption that a

1 worker's exposed for 40 years for both its central
2 tendency and high-end scenarios is far longer than
3 the information available from the Bureau of Labor
4 Statistics, which indicates median employment
5 tenure for workers in the chemical industry is 10
6 years or less taking into account all age groups.
7 As such, 10 years is a conservative estimate of
8 the central tendency exposure duration and should
9 be used for this scenario.

10 In addition, data from the Employee
11 Benefit Research Institute, or EBRI, show that the
12 median tenure with one employer of all wage and
13 salary workers ages 25 or older has remained
14 steady at approximately five years. EBRI data
15 also show that approximately 80 percent of older
16 workers ages 55 to 64 have tenures at one employer
17 of less than 25 years. And the current trend is
18 consistently toward lower tenures. As such, 25
19 years not 40 years is a conservative estimate for
20 high-end exposure duration --

21 **DR. DIANA WONG:** You have one
22 minute.

1 **MR. GREG BRORBY:** -- and should be
2 used -- thank you. And should be used for this
3 scenario. Finally, EPA should only use the full
4 shift data as the basis for estimating cancer risk
5 to chlor-alkali workers. In the draft risk
6 evaluation, EPA presents cancer risk estimates
7 based on either a full shift monitoring data or
8 calculated eight-hour time-weighted average
9 concentrations using a combination of short term
10 exposure data and full shift data. This latter
11 method incorrectly assumes that exposures
12 represented by the full shift data are exclusive
13 of exposures represented by the short-term data.
14 To the contrary, the full shift data already
15 include exposures associated with different short-
16 term activities.

17 The full shift data best reflects
18 the varied activities of individual workers across
19 an entire workday. As such, cancer risk should be
20 estimated using the full shift data only. Thank
21 you for your time.

22 **DR. DIANA WONG:** Thank you. Any

1 questions for the speaker? The next speaker is
2 Adele Abrams.

3 **MS. ADELE ABRAMS:** Yes. Hello. Can
4 you hear me all right?

5 **DR. DIANA WONG:** Yes.

6 **MS. ADELE ABRAMS:** Great. Thank you
7 for the opportunity to present brief comments on
8 your draft risk assessment for asbestos as part of
9 your TSCA review. My name is Adele Abrams and I'm
10 President of the Law Office of Adele L. Abrams PC.
11 We're a firm with offices in the D.C. area,
12 Colorado, and West Virginia, but we work
13 nationwide on occupational and also mine safety
14 and health issues. We provide safety and health
15 consultations, audits, industrial hygiene, and
16 training services, and also offer litigation
17 support with MSHA and OSHA. We do not do tort
18 litigation and I want to make that very clear.

19 I'm a safety professional, certified
20 mine safety professional specifically, and an
21 attorney. But I am not a physician, I am not an
22 epidemiologist. However, I've been involved with

1 this issue since 1989 and I was a safety
2 professional before I became an attorney. I
3 started working in the construction sector in 1986
4 and then in 1989, I went to work for the National
5 Stone Association as Director of Government
6 Affairs where literally on my first day of work I
7 learned about the issues that OSHA was looking at
8 concerning the distinction between asbestos and
9 non-asbestiform minerals such as actinolite,
10 tremolite, and anthophyllite. So I got a rapid
11 education in that area before I even became an
12 attorney.

13 Now, at this point, I do continue to
14 work with a variety of construction and also
15 mining companies, particularly those in the
16 construction aggregate sector and industrial
17 minerals sector. And I want to stress that I'm
18 testifying in my personal capacity today and not
19 on behalf of any associations that I have a
20 membership with, nor any of the companies that
21 I've been affiliated with over the years.

22 As I mentioned, I got involved with

1 this issue -- have subject matter interest because
2 of the work that I did developing comments and
3 working with OSHA back in the late '80s and early
4 '90s when they considered whether to regulate the
5 non-asbestiform minerals in the same manner as
6 asbestos. And MSHA, Mine Safety and Health
7 Administration, for its part has never really
8 considered this issue. And their air contaminants
9 standards simply incorporate by reference the
10 ACGIH threshold limit values for 1972 for coal and
11 1973 for metal and non-metal. So obviously
12 they're pretty outdated. I want to note up front
13 that I do concur with the findings in the draft
14 concerning chrysotile asbestos, the serpentine
15 variety, perhaps this docket really should be
16 termed that because it is primarily limited to
17 that.

18 I am not really clear why you are
19 not addressing the legacy uses and associated
20 disposal issues associated with this. It would
21 seem to me a comprehensive approach would make
22 sense. But if you're not going to address it now,

1 it must be addressed. And just anecdotally the
2 construction companies that I work with, very many
3 of them are small subcontractors, for example,
4 plumbing contractors, who are midway into a
5 project on an old building when they discover that
6 they are in fact dealing with asbestos-containing
7 materials. So study in that area certainly is
8 warranted.

9 Your draft notes that among the
10 other -- that other five forms of asbestos are now
11 subject to rulemaking at it does include the
12 anthophyllite, tremolite, and actinolite among
13 those five and says that they will be under
14 consideration in raw form or as part of articles
15 as part of that SNRU rulemaking.

16 So I want to focus my testimony here
17 on the terminology you're using in your report to
18 identify the regulated amphibole asbestos
19 varieties and to distinguish their non-asbestiform
20 analogs. I want to urge caution in including non-
21 asbestiform amphiboles --

22 **DR. DIANA WONG:** You have one more

1 minute.

2 **MS. ADELE ABRAMS:** Thank you.

3 Within a broad definition of asbestos or asbestos-
4 containing materials. They are clearly different
5 and OSHA's findings that they should not be
6 regulated as asbestos still remain good findings
7 today.

8 I also want to note quickly, and I
9 did submit my testimony for the record, that the
10 National Academy of Sciences was looking at this
11 issue in 2011. I testified at that to give NIOSH
12 valuable feedback on how to proceed in its health
13 effects research on asbestos and asbestos-
14 containing materials. NIOSH is also looking at
15 elongate mineral particles going beyond the focus
16 on aspect ratio. And I just want to, I guess,
17 urge you to avoid mission drift on this. And at
18 this point, there's still research going on but
19 there's no new studies to show any change in how
20 non-asbestiform minerals should be regulated. We
21 do need more precise analytical methods --

22 **DR. DIANA WONG:** Time is up.

1 **MS. ADELE ABRAMS:** Thank you. From
2 PCM to TEM and hopefully better procedures in the
3 future. Thank you very much and I ask that my
4 written comments that I submitted to Diana Wong by
5 email be included in the docket.

6 **DR. DIANA WONG:** Thank you. Any
7 questions for the speaker? Any questions for the
8 speaker? If not, the next speaker is Karen
9 Minott. Is she online?

10 **MR. MARTIN ALVARADO:** Diana, Karen
11 Minott is not online.

12 **DR. DIANA WONG:** Okay. Then the
13 next speaker is Brent Kynoch.

14 **MR. BRENT KYNOCH:** And hello. This
15 is Brent Kynoch. Can everyone hear me?

16 **DR. DIANA WONG:** Yes, we can hear
17 you. You can start.

18 **MR. BRENT KYNOCH:** That's great.
19 Hello. My name is Brent Kynoch. I'm the managing
20 director of the Environmental Information
21 Association or EIA. EIA is a membership
22 organization that was founded in 1983 as the

1 National Asbestos Council. EIA members are
2 companies, organizations, and persons involved in
3 asbestos abatement and asbestos management in
4 buildings and facilities. Our members include the
5 entire vertical spectrum of persons involved in
6 the abatement industry including contractors,
7 consultants, laboratories, training providers,
8 regulators, equipment suppliers, owners, and
9 managers.

10 I have been actively involved in
11 EPA's process of risk evaluation for asbestos
12 since it was first declared as a chemical under
13 review in December 2016. I've submitted comments
14 to the docket at every possible opportunity and
15 I've been involved in meetings with EPA officials
16 who were involved in the review so I'm intimately
17 familiar with this issue and I've been involved in
18 the process since day one.

19 To begin let me say that I and the
20 members of the EIA are delighted that EPA has
21 found that almost every condition of use of
22 asbestos have been deemed to present an

1 unreasonable risk. This is truly wonderful news.
2 What is most surprising however is that EPA did
3 everything possible in the development of the risk
4 evaluation to underestimate and downplay the risk
5 of exposure.

6 Yet even with the decisions by EPA
7 in the development of the risk evaluation process
8 that resulted in these underestimates and
9 undercounting, the numbers still don't lie.
10 Asbestos is a killer and EPA had no choice but to
11 conclude that it presents an unreasonable risk.

12 In spite of the fact that EPA has
13 concluded that asbestos presents an unreasonable
14 risk, I want to outline three specific areas where
15 EPA's decisions have systemically resulted in
16 undercounting and underestimating of the risk.

17 First, EPA has chosen not to comply with the
18 requirements imposed by the Ninth Circuit of
19 Appeals in the Safer Chemicals Healthy Families
20 matter. This is the matter that involves so
21 called legacy asbestos. It's currently in homes
22 and buildings but not in the stream of commerce.

1 EPA's plan to submit a mere
2 supplement to the risk evaluation for legacy
3 asbestos will result in two separate evaluations
4 or pictures of exposure and risk rather than a
5 risk evaluation considering all pathways of
6 exposure that currently exist in the U.S. in one
7 coordinated document. This current risk
8 evaluation without considering legacy materials
9 has narrowed the modeling and is undercounting
10 both exposure and mortality rates.

11 Second, and related to the issue
12 surrounding legacy uses is EPA's decision to use
13 only chrysotile in its risk evaluation. If legacy
14 uses of asbestos are considered, then it requires
15 the EPA considers exposure to amphibole varieties
16 of asbestos including the Libby amphiboles. It
17 has been documented in many studies that exposure
18 to amphibole forms of asbestos may in fact result
19 in higher mortality rates than exposure to only
20 chrysotile. Again, by considering exposures to
21 only chrysotile EPA has narrowed the modeling
22 resulting in an undercounting of both exposure and

1 mortality.

2 Third, EPA's decision to make a risk
3 evaluation based only on mortality rates and to
4 not include incidences of cancer that do not
5 result in death is just plain wrong. Think about
6 this colleagues, EPA is saying that asbestos
7 disease is not a problem if you don't die from it.
8 And EPA's discussion in the draft risk evaluation
9 is misleading. Even today, Dr. Scarano used the
10 term incidences of cancer in his presentation.

11 The only incidences being counted
12 are deaths. Granted, mesothelioma has a high
13 mortality rate, but other forms of cancer have
14 surprisingly good survival rates. So counting
15 only mortality clearly results in an
16 underestimation. And please colleagues, think
17 also about this, by counting only mortality rates
18 --

19 **DR. DIANA WONG:** You have one more
20 minute.

21 **MR. BRENT KYNOCH:** -- EPA is likely
22 only counting incidences of mesothelioma. And why

1 not? Because by counting only mesothelioma it
2 eliminates that hassle and the problems associated
3 with separating other causative agents in the
4 formation of lung cancer, such as smoking. EPA
5 has hinted at this, but it hasn't said it overtly.

6 Lastly, and most importantly is,
7 what will EPA do now? I do not believe the SNUR -
8 - the SNUR enacted by EPA adequately protects
9 persons from exposure to asbestos. EPA has the
10 authority to ban asbestos under TSCA. EPA's
11 determined that most uses of asbestos present an
12 unreasonable risk. EPA should move finally to ban
13 asbestos in the United States. Thank you.

14 **DR. DIANA WONG:** Thank you. Any
15 questions for the speaker? Any questions? The
16 next speaker is Steve Risotto. Is he online?

17 **MR. STEVE RISOTTO:** Yes. Good
18 afternoon. My name is Steve Risotto. I am a
19 senior director at the American Chemistry Council
20 and serve as a technical advisor to ACC's chlorine
21 chemistry division. The division represents major
22 producers and users of chlorine in North America

1 and works to promote and protect the
2 sustainability of chlorine chemistry processes,
3 products, and applications.

4 Chlorine is a chemistry that
5 continues to prove its public health benefits from
6 protecting the nation's drinking water supply for
7 over 100 years to fighting against the current
8 COVID-19 pandemic. Make no mistake, chlorine
9 chemistry is essential to life. Today, chrysotile
10 asbestos diaphragm technology is used to produce
11 about one-third of the nation's supply of
12 chlorine.

13 Consequently, CCD has actively
14 participated in EPA's evaluation of asbestos use
15 in the chlor-alkali industry including
16 facilitating site visits to three production
17 facilities and submitting a significant amount of
18 information on the diaphragm process including
19 nearly 800 worker exposure measurements. I'd like
20 to focus my comments this afternoon on those
21 measurements and to review operating practices at
22 chlor-alkali facilities.

1 Greg Brorby who conducted an
2 extensive technical analysis of EPA's evaluation
3 has already provided additional comments on our
4 behalf. Unfortunately, and in our zeal to provide
5 worker exposure information we submitted data to
6 the Agency that duplicated information submitted
7 by one of our members. The duplicate data include
8 293 full shift measurements and about 300
9 measurements of shorter duration. Since the data
10 provided by the member company included additional
11 details, we have encouraged EPA to exclude the
12 duplicate data provided by ACC from its analysis.
13 We apologize to EPA and to the SACC for the
14 confusion related to these duplicated data.

15 While Greg Brorby has addressed the
16 Agency's exposure analysis, I wanted to briefly
17 touch on the use of personal protective equipment
18 or PPE at chlor-alkali plants. The OSHA workplace
19 standard for asbestos requires the use of PPE,
20 including respirators, during certain operations
21 where potential asbestos exposures may be
22 relatively higher including during unloading and

1 transport, weighing and handling, and hydro
2 blasting. The exposure measurements provided to
3 the Agency both the short term and full shift data
4 are taken from their personal breathing zone and
5 do not reflect actual exposure to the worker
6 during all the time that a respirator is required
7 to be worn.

8 While EPA considers respirator use
9 as part of its risk characterization, it does not
10 include respirators in its base-case assumptions
11 which is not consistent with standard operating
12 practice at the facilities. Mandated respirator
13 use should be included as part of EPA's base-case
14 analysis.

15 The OSHA asbestos standard also
16 requires the creation of administrative controls
17 that limit who may enter designated restricted
18 areas and that specified minimum PPE to be
19 utilized. Given the OSHA requirements and as
20 instructed through asbestos awareness training for
21 all site personnel it is not likely for
22 occupational non-users or ONUs to pass through or

1 work near restricted areas as suggested in the
2 draft risk evaluation.

3 Consequently, EPA's assumption that
4 the 15 area sample measurements taken from
5 restricted areas can be used to estimate exposures
6 for ONUs not trained and assigned to work in the
7 area is incorrect. These area samples should not
8 be used to estimate ONU exposure.

9 EPA's reference to maintenance and
10 janitorial staff also needs to be clarified.
11 Maintenance workers should not be considered ONUs
12 as they are included in the OSHA asbestos standard
13 and maintenance is conducted with designated PPE
14 for specific tasks. Moreover, restricted areas
15 are not attended by non-operator janitors.

16 The confusion over who is an ONU and
17 who has access to restricted areas may explain the
18 discrepancy in the estimates for the numbers of
19 ONUs in the draft.

20 **DR. DIANA WONG:** You have one more
21 minute.

22 **MR. STEVE RISOTTO:** While chapter

1 four -- thank you. While chapter four suggests
2 that there may be as many as 3,000 ONUs at chlor-
3 alkali facilities, chapter two estimates the
4 number to be about 100. Even this lower number is
5 likely an overestimate of individuals in or around
6 restricted areas due to the access limitations and
7 training and procedures in place at the
8 facilities.

9 Given the restrictions on access to
10 those areas where asbestos exposure may occur to
11 minimize potential ONU exposures, and the
12 significant steps taken to protect asbestos
13 workers while in these restricted areas, the risk
14 at chlor-alkali facilities are at or below the 1
15 in 10,000 risk threshold EPA has employed to
16 determine unacceptable risk under TSCA. Thank
17 you. I would be happy to answer any questions
18 from committee members.

19 **DR. DIANA WONG:** Thank you. Any
20 questions for the speaker? Any questions? If
21 not, the next speaker is Penelope Fenner-Crisp.
22 Is she online?

1 DR. PENELOPE FENNER-CRISP: Yes, I
2 am.

3 DR. DIANA WONG: Great. You may
4 start.

5 DR. PENELOPE FENNER-CRISP: Go
6 ahead?

7 DR. DIANA WONG: You ready to start?

8 DR. PENELOPE FENNER-CRISP: I am.

9 DR. DIANA WONG: Go ahead.

10 DR. PENELOPE FENNER-CRISP: Thank
11 you. Good afternoon. My name is Penelope Fenner-
12 Crisp. Today I will be presenting comments on
13 behalf of the Environmental Protection Network or
14 EPN. It's an organization comprised of more than
15 500 EPA alumni volunteering their time to protect
16 the integrity of the EPA, human health and
17 environment. I will focus on three of the many
18 topics that we've addressed in our written
19 comments submitted to the docket.

20 We've known for many decades that
21 asbestos is a human carcinogen so not surprisingly
22 that endpoint's the primary focus of the human

1 health risk assessment. A clear causal
2 relationship between asbestos exposure and lung
3 cancer and mesothelioma has been established.
4 However, both the National Academies and IARC have
5 concluded that asbestos also causes laryngeal
6 cancer, both noting that there was evidence of a
7 dose-response relationship seen in some studies.

8 IARC also concluded that asbestos
9 causes ovarian cancer noting positive exposure-
10 response relationships in some studies. EPA has
11 articulated rationale for narrowing the focus to
12 lung cancer and mesothelioma excluding all other
13 tumor sites can be summed up in one dismissive
14 quote in the DRE, "There's inadequate data for
15 exposure-response analyses." EPN questions this
16 blanket conclusion and believes that the cancer
17 assessment should be expanded to include a more
18 transparent analysis of the data on laryngeal and
19 ovarian cancer, with an emphasis on their adequacy
20 to support dose-response assessment and potential
21 for inclusion in the calculation of the combined
22 IUR.

1 EPN also questions whether it was
2 appropriate to exclude from the assessment those
3 studies of scenarios in which exposure occurring
4 to both chrysotile and amphibole fiber forms could
5 not be separated out given that trace amounts of
6 other forms can be found in commercial chrysotile.
7 This decision seems to be somewhat duplicitous.
8 This should be resolved when the chrysotile
9 assessment is integrated to the assessment of all
10 forms of asbestos which EPA must now conduct.

11 EPN is also concerned that the
12 draft evaluation does not assess and quantify the
13 non-cancer risks of asbestos exposure. When one
14 compares the 2014 IRIS reference concentration for
15 Libby amphibole for non-cancer effects with the
16 1988 generic IUR for cancer risk, cancer is the
17 clear risk driver. However, one cannot assume the
18 same relationship would play out in the current
19 situation for several reasons. The Libby
20 amphibole RFC was compared against the one in a
21 million benchmark that applies to the general
22 population, and in this case, consumer uses.

1 However, the risk benchmark for the
2 occupational setting is 1 in 10,000 or 10 -- 100
3 fold higher. Secondly, EPA is proposing a
4 combined IUR that is about a third lower than the
5 1988 IRIS value, but it could change if and when
6 the IURs for the other tumor types are
7 incorporated.

8 And thirdly, an RFC calculated for
9 chrysotile may be lower than that for Libby
10 amphibole. The combination of these three factors
11 could result in a flipping of the risk driver from
12 cancer to a non-cancer effect, but in any case,
13 ostensibly leads to an underestimation of the
14 overall risks of asbestos exposure based on cancer
15 alone as EPA acknowledges.

16 EPA is obligated to determine if
17 underestimation is the case and to what degree.
18 But we won't know until and unless an analysis of
19 the non-cancer effects is conducted. One final
20 point on a topic that may not be within the
21 committee's scope but it is within EPN's, in spite
22 of employing a very narrow and exclusionary

1 approach in this risk evaluation, the Agency has
2 concluded that most of the conditions of use
3 examined pose an unreasonable risk. That makes
4 the risk management options narrow too when
5 relatively simple in our view. Just two steps --

6 **DR. DIANA WONG:** You have one more
7 minute.

8 **DR. PENELOPE FENNER-CRISP:** --
9 vacate the SNUR and issue a comprehensive ban on
10 all forms of asbestos. 30 years is more than
11 enough time to develop alternatives for the uses
12 that were not previously phased out. Thank you
13 for your attention.

14 **DR. DIANA WONG:** Thank you. Any
15 questions for the speaker? Any questions? If
16 not, the next speaker is Arthur Frank. Is he
17 online?

18 **DR. ARTHUR FRANK:** I am here. Can
19 you hear me?

20 **DR. DIANA WONG:** Yes.

21 **DR. ARTHUR FRANK:** My name is Arthur
22 Frank. I'm a Professor of Public Health and a

1 Professor of Medicine at Drexel University. I've
2 submitted written comments and my disclosures are
3 there plus a lot of material to supplement my
4 comments today.

5 I've spent more than 50 years
6 working in the area of asbestos and asbestos-
7 related research having trained at Mt. Sinai under
8 Dr. Irving Selikoff. Much of what I wanted to say
9 has been mentioned by other speakers, but I will
10 reiterate some of those points briefly.

11 It is inappropriate, I feel, to
12 study only mesothelioma and lung cancer.
13 Laryngeal and ovarian cancer should be studied.
14 And consideration with the growing literature on
15 these subjects should be looking at even other
16 cancers such as a variety of gastrointestinal
17 cancers and kidney cancers.

18 It's inappropriate to study only
19 chrysotile. Amphiboles are still very much part
20 of the work and environmental world that we live
21 in. While cosmetic talc may be controlled by the
22 FDA, industrial talc which is heavily contaminated

1 clearly has amphibole in it and many people are
2 exposed. Textiles containing asbestos are still
3 out there in terms of gloves, welding blankets,
4 and generator blankets. Legacy issues shouldn't
5 wait for another assessment. We had mentioned
6 this morning of the taconite issues. That has
7 been shown to cause significant disease, is
8 ongoing and contains amphibole.

9 There are a number of people who
10 argue that chrysotile, especially from brakes,
11 does not cause mesothelioma. They've cited
12 literature and want other literature looked at.
13 Some positive literature with regard to the
14 hazards have been neglected I feel and again
15 mentioned in the written materials that I've
16 posted. But if one wants to believe that
17 mesotheliomas are not caused by chrysotile, that
18 really begs the question of why aren't amphiboles
19 also being studied because many auto mechanics and
20 even family members of auto mechanics have come
21 down with lung cancer and mesotheliomas.

22 The chlor-alkali industry is asking

1 for continued use yet much of the rest of the
2 world are phasing it out and there are non-
3 hazardous materials available to replace asbestos
4 in the chlor-alkali industry. They should be
5 looked at.

6 Lastly, when one considers exposures
7 by product or by utilization as Dr. Selikoff wrote
8 as far back as 1964 in his seminal paper in the
9 Journal of the American Medical Association, "It's
10 not just the job that matters. It's the exposure
11 to asbestos that is important." And there are
12 still many current and obviously past exposures to
13 asbestos including to vermiculite in over 10
14 million homes in this country.

15 With over 60 other countries in the
16 world banning asbestos, it really is appropriate
17 that the United States joins other countries in
18 the banning of this toxic and carcinogenic
19 material. Thank you for your consideration of my
20 comments.

21 **DR. DIANA WONG:** Thank you. Any
22 questions for the speaker? Any questions? If

1 not, at this time we are scheduled for a 10-minute
2 break. So we will have our 10-minute break and we
3 will resume at 3:30.

4 **MR. MARTIN ALVARADO:** Diana.

5 **DR. DIANA WONG:** Yes.

6 **MR. MARTIN ALVARADO:** I just wanted
7 to remind you and Dr. Portier that you mentioned
8 taking roll call before the break.

9 **DR. DIANA WONG:** Yes. Dr. Portier -
10 -

11 **DR. KENNETH PORTIER:** We'll --

12 **DR. DIANA WONG:** Do you want to do
13 it now?

14 **DR. KENNETH PORTIER:** We'll take it
15 right after the break. Let's take it right after
16 the break.

17 **DR. DIANA WONG:** Okay.

18 **DR. KENNETH PORTIER:** Thank you for
19 reminding me. We'll do it right after the break.
20

21 **[Break]**
22

1 **DR. KENNETH PORTIER:** Let's
2 reconvene. Very quickly, let's call the roll of
3 the committee to establish who's here this
4 afternoon. I should have done that right after
5 lunch. I apologize. Dr. Anderson. Dr. Barton.
6 **DR. HENRY ANDERSON:** I'm here.
7 **DR. CHARLES BARTON:** Here.
8 **DR. HENRY ANDERSON:** I'm here.
9 Sorry.
10 **DR. CHARLES BARTON:** And I'm here
11 too.
12 **DR. KENNETH PORTIER:** Thank you.
13 Dr. Bennett.
14 **DR. STEVEN BENNETT:** I am here.
15 **DR. KENNETH PORTIER:** Dr. Blystone.
16 **DR. SHERI BLYSTONE:** I am here.
17 **DR. KENNETH PORTIER:** Dr. Cory-
18 Slechta. Dr. Cory-Slechta?
19 **MR. MARTIN ALVARADO:** She's on here
20 twice and --
21 **DR. KENNETH PORTIER:** Dr. --
22 **MR. MARTIN ALVARADO:** -- one of them

1 is muted.

2 DR. HOLLY DAVIES: Ken, did you call
3 Dr. Davies? I'm here.

4 DR. KENNETH PORTIER: Yeah. Thank
5 you. Dr. Doucette.

6 DR. WILLIAM DOUCETTE: Here.

7 DR. KENNETH PORTIER: Dr. Jiménez-
8 Gonzalez. Dr. Johnson.

9 DR. MARK JOHNSON: I'm here.

10 DR. KENNETH PORTIER: Dr. Kaufman.

11 MR. ALAN KAUFMAN: I am here.

12 DR. CONCEPCIÓN JIMÉNEZ-GONZALEZ:
13 Dr. Jiménez-Gonzalez is here as well. Took me a
14 while to --

15 DR. KENNETH PORTIER: Thank you.

16 DR. CONCEPCIÓN JIMÉNEZ-GONZALEZ: --
17 unmute.

18 DR. KENNETH PORTIER: Dr. Kissel.

19 DR. JOHN KISSEL: Here.

20 DR. KENNETH PORTIER: Dr. Rowlands.

21 DR. CRAIG ROWLANDS: I'm here.

22 DR. KENNETH PORTIER: Dr. Schlenk.

1 DR. DANIEL SCHLENK: Here.

2 DR. KENNETH PORTIER: Dr. Sheela.

3 DR. SATHYANARAYANA: Here.

4 DR. KENNETH PORTIER: Dr. Crump.

5 DR. KENNY CRUMP: Present.

6 DR. KENNETH PORTIER: Dr. Everitt.

7 Dr. Herrick. Dr. Herrick, your phone might be
8 muted.

9 DR. ROBERT HERRICK: Oh. I'm here.

10 DR. JEFFREY EVERITT: Everitt is
11 here ---

12 DR. KENNETH PORTIER: Dr --

13 DR. JEFFREY EVERITT: -- on mute.

14 DR. KENNETH PORTIER: Yeah. Dr.
15 Jayjock.

16 DR. MICHAEL JAYJOCK: Yeah. I'm
17 here.

18 DR. KENNETH PORTIER: Dr. Kanarek.

19 DR. MARTY KANAREK: Here.

20 DR. KENNETH PORTIER: Dr. Markowitz.

21 DR. STEVEN MARKOWITZ: Here.

22 DR. KENNETH PORTIER: Dr. Sheppard.

1 DR. ELIZABETH SHEPPARD: Here.

2 DR. KENNETH PORTIER: Dr. Shukla.

3 DR. ARTY SHUKLA: I am here.

4 DR. KENNETH PORTIER: Thank you, Dr.
5 Sheppard. Thank you, Dr. Shukla. Dr. Taioli.
6 Dr. Van Gosen. Dr. Taioli, your phone might be on
7 mute.

8 DR. EMANUELA TAIOLI: Sorry.
9 Everything was on mute. I hope I'm on now. Yes.

10 DR. KENNETH PORTIER: Yep. I can
11 hear you.

12 DR. EMANUELA TAIOLI: Okay. Good.
13 Thank you.

14 DR. KENNETH PORTIER: Dr. Cory-
15 Slechta. And Dr. Van Gosen.

16 MR. MARTIN ALVARADO: Cory-Slechta
17 is showing up as online. She's muted in WebEx. I
18 don't know why.

19 DR. KENNETH PORTIER: Yeah. I see
20 that. Okay. Let's move on.

21 DR. DIANA WONG: All right. Thank
22 you. So we'll continue with the public comments

1 section. Our next speaker is David Bernstein. Is
2 he online?

3 **MR. MARTIN ALVARADO:** No. We've
4 never -- we've not seen him online.

5 **DR. DIANA WONG:** Okay. So we'll go
6 back. And the next speaker is Victor Roggli.

7 **DR. VICTOR ROGGLI:** Hello. Can you
8 hear me?

9 **DR. DIANA WONG:** Are you online?
10 Are you ready?

11 **DR. VICTOR ROGGLI:** Yes. Yes.

12 **DR. DIANA WONG:** Okay. Thank you.
13 You may start.

14 **DR. VICTOR ROGGLI:** Thank you. My
15 name is Victor Roggli. I'm a professor of
16 pathology at Duke University Medical Center in
17 Durham, North Carolina. I've been studying
18 asbestos-related diseases in 44 years,
19 specializing in the analysis of lung asbestos
20 fiber content. I've published 89 articles in the
21 peer-reviewed literature, 23 chapters in books,
22 and five textbooks that have dealt with fiber

1 analysis issues. I've testified at more than 300
2 asbestos trials including 176 times for plaintiffs
3 and 136 times for defendants.

4 In the EPA draft analysis, there is
5 heavy reliance on the North and South Carolina
6 textile cohorts for determining the risk of lung
7 cancer, mesothelioma, and exposure to chrysotile
8 asbestos. In particular, the Marshville, North
9 Carolina plant is touted as representative of
10 chrysotile only cohort. In 1983 I analyzed lung
11 tissue from a Marshville mesothelioma victim as a
12 consultant for plaintiff attorney. As expected,
13 there were increased levels of chrysotile and
14 tremolite asbestos. But the patient also had
15 elevated levels of amosite. Her husband worked
16 for HK Porter and she laundered his dirty work
17 clothes.

18 Furthermore, she worked for HK
19 Porter prior to working at the Marshville plant.
20 The point is that in claiming exposure only to
21 chrysotile, Loomis and Dement may have had no
22 information about prior exposure to asbestos or

1 household contact exposures. A recent study of
2 354 mesothelioma in women that we published, we
3 found that 197 had histories of exposures as
4 household contacts. I'm not aware of any follow-
5 up information published by Loomis and Dement
6 regarding asbestos exposure as a result of prior
7 occupations or household contact exposures from
8 any of the Marshville cases.

9 Similar criticisms apply to other
10 textile cohorts where claims have been published
11 in peer-reviewed literature of exposure
12 exclusively to chrysotile asbestos.

13 Representative cases from the South Carolina
14 textile cohorts, and the Rochdale cohort in the
15 United Kingdom, have been analyzed by others who
16 found excess levels of commercial amphibole
17 fibers.

18 These findings are crucial in any of
19 the analyses relying on textile cohorts for
20 determining chrysotile potency and mesothelioma
21 causation, considering that amosite and
22 crocidolite have potencies that are hundreds of

1 times greater than those for chrysotile.

2 Another important issue to be
3 considered is fiber dimensions. In one of the
4 studies referenced in the EPA draft document, Dr.
5 Berman reported a substantial percentage of long
6 chrysotile fibers in the textile cohorts,
7 particularly for fibers greater than 20 or even 40
8 microns in length.

9 This is an important distinction
10 when one considers chrysotile product exposures
11 involving an overwhelming percentage of shorter
12 fibers, since studies have indicated a lack of
13 pathogenicity for fibers that are less than five
14 microns in length. This is important in my
15 opinion that such information be taken into
16 account in a detailed analysis such as the one
17 presented here by the EPA.

18 Finally, I want to close with some
19 comments regarding asbestos exposures among brake
20 mechanics. To date, I have analyzed lung tissue
21 samples from 21 patients with mesothelioma and
22 allegations of asbestos exposure confined to

1 automotive friction products, 20 of which were
2 auto or brake mechanics and one a do-it-
3 yourselfer. Fifteen cases had asbestos contents
4 within the range of our reference population, and
5 6 had elevated levels of amosite or crocidolite.
6 None had elevated concentrations restricted to
7 chrysotile or its contaminant, tremolite.

8 According to Helsinki criteria for
9 determining causation, there is no evidence in
10 case with fiber burdens within the range of
11 control population are asbestos-related. Our
12 published findings do not support an asbestos
13 etiology --

14 **DR. DIANA WONG:** You have one more
15 minute.

16 **DR. VICTOR ROGGLI:** Thank you. Of
17 mesothelioma as a result of friction product
18 exposures and are entirely consistent with
19 epidemiological observations which I will leave to
20 others to address.

21 In summary, fiber analysis provides
22 important information relevant to the present

1 investigation by the EPA. Although no method of
2 investigation is without limitations, fiber
3 analysis provides a powerful tool to assist in the
4 exploration of the very questions addressed in the
5 current EPA document. You should be wary of any
6 comments to the contrary. I hope that the
7 scientific advisory board will carefully consider
8 the information I provided and thank you for the
9 opportunity to share it publicly.

10 **DR. DIANA WONG:** Thank you. Any
11 questions for the speaker?

12 **DR. KENNY CRUMP:** I have a question.
13 Maybe a follow-up. Kenny Crump.

14 **DR. DIANA WONG:** Go ahead.

15 **DR. KENNY CRUMP:** So when you say
16 you have elevated levels of fibers in lung tissue,
17 how do you -- what's your definition of elevated
18 levels? How do you determine levels are elevated?

19 **DR. VICTOR ROGGLI:** Yeah. These are
20 compared to 20 control cases which we've
21 identified that had no asbestos-related disease,
22 had normal lungs at autopsy, that had asbestos

1 body counts within our previously determine normal
2 range. So those 20 cases were our controls and
3 the elevated level is one that's greater than all
4 of those 20 controls.

5 **DR. KENNY CRUMP:** When you say
6 normal levels, you have found asbestos fibers in
7 all the subjects then, right? Both chrysotile and
8 amphibole even in people not exposed to asbestos?

9 **DR. VICTOR ROGGLI:** No. what we have
10 found is substantial number I think that for
11 tremolite, it's contaminants, it's 11 out of our
12 20 controls. For actinolite, there was several
13 cases in the controls identified. For chrysotile,
14 I think it was less than half of our controls when
15 we found it. We're counting only fibers that are
16 five microns or greater in length. So we are not
17 counting the short fibers.

18 **DR. KENNY CRUMP:** Thank you.

19 **MR. ALAN KAUFMAN:** This is Al
20 Kaufman. I had one question. I didn't catch it.
21 You said the -- you mentioned a cohort that was
22 largely automobile mechanics and then there was

1 one person who was something else that I didn't
2 catch.

3 **DR. VICTOR ROGGLI:** Oh. A do-it-
4 yourselfer. That's what the EPA's defined it for
5 shade-tree mechanics.

6 **MR. ALAN KAUFMAN:** Okay. Got it.
7 Thank you.

8 **DR. DIANA WONG:** Any more questions?
9 If not, the next speaker is Robert Sussman. Is he
10 online?

11 **MR. ROBERT SUSSMAN:** Yeah. Can you
12 hear me?

13 **DR. DIANA WONG:** Yes.

14 **MR. ROBERT SUSSMAN:** Okay. Good
15 afternoon. My name is Bob Sussman. I'm here
16 today as counsel to the Asbestos Disease Awareness
17 Organization. I'm an attorney and I served two
18 tours of duty at EPA in senior positions under
19 President's Clinton and Obama.

20 This is the first comprehensive
21 evaluation of asbestos in 35 years. The EPA IRIS
22 program issued a definitive peer-reviewed

1 assessment in 1988, which with some refinements is
2 the foundation for the EPA, OSHA, and state
3 asbestos regulations that are in place today.

4 EPA's draft evaluation of asbestos
5 under the TSCA departs from this long-standing
6 framework and calculates a considerably lower
7 cancer risk than EPA's 1988 IRIS assessment.
8 Because of this lower risk, the EPA approach if
9 fully analyzed would require rethinking of all
10 existing asbestos protection programs. As you
11 heard today, EPA's approach focuses on a single
12 fiber type, chrysotile, bases risk estimates on
13 two studies that purportedly reflect exposure to
14 this one fiber, and uses a non-linear exponential
15 model that is claimed to, "best fit the data in
16 those two studies."

17 This approach is very similar to an
18 unsuccessful effort in 2008 to replace the IRIS
19 assessment with fiber specific potency values.
20 There was considerable opposition at the time from
21 asbestos experts and EPA pulled the plugged after
22 a highly critical report from the EPA Science

1 Advisory Board. The thrust of much of the
2 criticism was that the data did not support
3 differential risk estimates for individual fibers
4 and that the most defensible approach was to
5 derive a single IUR for all fibers based on a
6 comprehensive review of all the studies for all
7 fiber types.

8 The rationale was that there are
9 significant uncertainties in looking at individual
10 epidemiology studies and also anomalies in all of
11 those studies with complicated interpretation.
12 And so, therefore, it's best to start with the
13 most comprehensive database and narrow it down to
14 the best studies which reflect a cross-section of
15 fibers.

16 Now the discredited fiber specific
17 approach is being revived on the draft TSCA
18 evaluation. And I think the many presentations
19 we've heard today about whether the cohorts in the
20 North Carolina and South Carolina studies were
21 exposed only to chrysotile, or instead to other
22 fibers as well, is a very compelling illustration

1 of the dangers of relying on two studies on the
2 assumption that they represent chrysotile
3 exposures.

4 **DR. DIANA WONG:** You have one
5 minute.

6 **MR. ROBERT SUSSMAN:** The premise of
7 the chrysotile only approach is that all active
8 asbestos uses involve chrysotile. But in fact,
9 talc-based industrial and consumer products
10 contaminated with asbestos contain amphiboles as
11 well as chrysotiles -- and I want to emphasize
12 here that the consumer products are not cosmetics.
13 They are crayons and children's toys subject to
14 EPA's jurisdiction under TSCA which have been
15 tested and found to contain asbestos. In
16 addition, many legacy products contain multiple
17 fiber types, and as a result real world exposure
18 is to different fibers in combination.

19 Let me emphasize here that --

20 **DR. DIANA WONG:** The time is up.

21 **MR. ROBERT SUSSMAN:** Okay. Thank
22 you very much.

1 **DR. DIANA WONG:** Thank you. Any
2 questions for the speaker?

3 **DR. KENNY CRUMP:** Kenny Crump. I
4 have a question.

5 **DR. DIANA WONG:** Go head.

6 **DR. KENNY CRUMP:** The decision by
7 the Science Advisory Board not to adopt that other
8 approach that five different potencies, or
9 different fibers, or different types, or different
10 dimensions -- my understanding was that was due
11 because of the lack of data not because of the
12 lack of any belief that the idea that they have
13 got different potencies was invalid. Do you have
14 the same understanding of that?

15 **MR. ROBERT SUSSMAN:** Yeah. I guess
16 I have a somewhat different understanding which is
17 that the lack of data involved the persuasiveness
18 of the available studies to zero in on differences
19 in potency and fiber size. So I think the
20 conclusion was in part based on the limitations of
21 the database to support very much of a fine grain
22 binning approach.

1 **DR. DIANA WONG:** Any more questions?

2 **DR. SATHYANARAYANA:** Yeah. This is
3 Sheela. I had a question. You just mentioned the
4 amphibole in crayons and children's toys. Is
5 there any exposure assessment that you know of
6 that explores that in more detail? And maybe Alan
7 Kaufman or someone else on the committee knows the
8 answer to that.

9 **MR. ROBERT SUSSMAN:** Yeah. Well, I
10 can't speak to the exposure side of it but there's
11 really quite a bit of data that confirms the
12 presence of asbestos in these products and
13 quantifies the levels. And all that information
14 has actually been submitted to EPA so it's a
15 little surprising that EPA is assuming that talc
16 exposure only occurs to cosmetics. On the issue
17 of what the risk might be and what the exposure
18 might be, I don't know that that's been analyzed
19 but certainly, that's the sort of thing that might
20 be done in the TSCA risk evaluation.

21 **DR. DIANA WONG:** Any more questions?
22 If not, the next speaker is Suresh Moolgavkar and

1 he has slides.

2 **DR. SURESH MOOLGAVKAR:** I'm here.

3 Yes. I am --

4 **DR. DIANA WONG:** Okay.

5 **DR. SURESH MOOLGAVKAR:** Thank you.

6 I am a principal scientist at Exponent and a
7 former member of the Fred Hutchinson Cancer
8 Research Center and a Professor of Epidemiology,
9 Applied Mathematics, and Biostatistics at the
10 University of Washington. Next slide, please.

11 I have three fundamental problems
12 with this current draft risk assessment. First,
13 in the face of I think incontrovertible and
14 mounting evidence the Agency assumes that most
15 cases of mesothelioma are attributable to asbestos
16 exposure. In fact, this is not true. There are
17 multiple well-designed epidemiology studies
18 estimating the population attributable fractions
19 from mesothelioma in Europe and in the United
20 States and these studies have not been funded by
21 industry. Next slide, please. Next slide.

22 What these studies show is that a

1 substantial fraction of cases of mesothelioma
2 cannot be attributed to asbestos exposure. So
3 for example, in the United States, if you look at
4 the pleura, the strongest association with
5 asbestos exposure is with the pleura. No more
6 than 80 percent of cases among men and no more
7 than 20 percent among women in the U.S. are
8 attributable to exposure.

9 If you look at peritoneal
10 mesothelioma, on the other hand, no more than 10
11 percent among men and less than one percent among
12 women in the U.S. attributable to asbestos
13 exposure. So this is a fundamentally erroneous
14 assumption made by the Agency. Next slide,
15 please.

16 The next fundamental problem that I
17 have is that the robust body of epidemiologic data
18 that demonstrates no increased risk of disease
19 among vehicle mechanics or users of AABL has been
20 completely ignored by the Agency. So these are
21 epidemiology studies and there are toxicologic
22 studies as well to support the results of the

1 epidemiologic studies. Next slide, please.

2 In terms of a hierarchy of
3 epidemiologic study designs, at the very bottom,
4 you have case reports and case series and at the
5 very top, you have systematic reviews and
6 metanalyses. Obviously, randomized control trials
7 have no role in asbestos-related disease. In the
8 middle, you have the analytical epidemiology
9 studies that is a case-control and cohort studies,
10 and these multiple studies have been completely by
11 the Agency. Next slide, please.

12 This is a list of the multiple case-
13 control and cohort studies of vehicle mechanics
14 showing not even a statistical association between
15 exposure to AABL as a vehicle mechanic and an
16 increased risk of mesothelioma. And let me state
17 right here that most of these studies have not
18 been funded by industry and have been conducted in
19 various countries in Europe and in the United
20 States. Next slide, please.

21 Same sort of situation for vehicle
22 mechanics and increased risk of lung cancer. No

1 evidence of even a statistical association let
2 alone a causal association. Next slide.

3 **DR. DIANA WONG:** One more minute.

4 **DR. SURESH MOOLGAVKAR:** Yes. So
5 obviously epidemiologic studies have their
6 strengths and limitations. And the strengths and
7 limitations of the analytical studies of brake
8 mechanics, I mean vehicle mechanics and
9 mesothelioma have been carefully looked at in a
10 comprehensive meta-analysis by Professor Garabrant
11 and his colleagues in 2016. And after this
12 evaluation, they conclude that actually there is
13 no increased risk of mesothelioma associated with
14 work as a vehicle mechanic. Next slide, please.

15 Some investigators have reached a
16 different conclusion by using basically case
17 reports and case series which cannot even show a
18 statistical association --

19 **DR. DIANA WONG:** Time is up.

20 **DR. SURESH MOOLGAVKAR:** Time is up?

21 **DR. DIANA WONG:** Yes.

22 **DR. SURESH MOOLGAVKAR:** You didn't -

1 - oh, okay. Thank you.

2 **DR. DIANA WONG:** Any questions for
3 the speaker?

4 **DR. KENNY CRUMP:** Yes. Yes. I have
5 -- this is Kenny Crump. I have a question.

6 **DR. DIANA WONG:** Go ahead.

7 **DR. KENNY CRUMP:** Hi, Suresh.
8 You're pointing out that some of the mesotheliomas
9 are of no known asbestos exposure may be
10 attributed to asbestos but a previous speaker
11 pointed out that for several people in his control
12 group with no exposure they still had asbestos in
13 their lungs. So how do you rule out the fact that
14 even people with no known particular asbestos
15 exposure could have had asbestos exposure that
16 might have caused their mesothelioma?

17 **DR. SURESH MOOLGAVKAR:** Okay. Let
18 me answer that question by giving you an example.
19 The quintessential linear non-threshold carcinogen
20 for most agencies is ionizing radiation. We know
21 that ionizing radiation now, by epidemiological
22 studies, is an important risk factor for the

1 development of mesothelioma. And we know that we
2 are all exposed to ionizing radiation in the
3 background. Every time we go on a skiing vacation
4 our exposure to ionizing radiation increases.

5 Every time we have a medical
6 procedure, we get ionizing radiation. Every time
7 we fly across the country, we increase our
8 exposure to ionizing radiation. So even if
9 background levels of -- if assume that background
10 levels of exposure to asbestos can increase the
11 risk of mesothelioma then why not background
12 levels of ionizing radiation that we all receive?

13 That's one answer. The second
14 answer is that a study has been done looking at
15 the differences in rates of mesothelioma for women
16 in the urban areas versus women in rural areas
17 where the difference in background levels of
18 exposure is about ten-fold. So the background
19 exposure to asbestos, there is about a ten-fold
20 difference, but there is absolutely no difference
21 in the incidents of mesothelioma in the SEER
22 registry since 1973.

1 If you do the same comparison for
2 males, of course, you see a difference because
3 males are occupationally exposed to asbestos in
4 the urban areas. But in the rural areas there is
5 no difference between female rates, urban and
6 rural. So, I mean, if you're going to take
7 background exposure into account, why don't you
8 take background exposure of ionizing radiation?

9 **DR. DIANA WONG:** Any more questions?

10 **DR. HENRY ANDERSON:** Yes. I do.

11 It's just a question about the radiation exposure.
12 I mean, I'm familiar with the literature and it
13 seems that cases that have that attributed are all
14 people who received not airline flight type
15 radiation but actually x-ray radiation for tumors.
16 Could you comment on whether the dose estimates
17 from the mesotheliomas where radiation has been
18 found to be causal without any asbestos?

19 **DR. SURESH MOOLGAVKAR:** Yeah.

20 That's a good question and let me again draw the
21 comparison there. Yes, epidemiology studies both
22 of ionizing radiation, I might point out to you,

1 also of asbestos exposure have been done in very
2 highly exposed cohorts. So the radiation
3 epidemiology has been done among patients who have
4 received radiation therapy for a previous cancer.
5 That is where most of the radiation studies come
6 from.

7 But when you talk about the asbestos
8 exposure and the link to mesothelioma, we have
9 that information also from only highly exposed
10 cohorts. This is precisely what I'm trying to
11 point out. When you look at occupations in which
12 there are very low levels of exposure like the
13 vehicle mechanics, there is absolutely no
14 indication of an increase in risk. So I think
15 there is a complete parallel. There is no
16 difference. It's a good analogy, radiation, and
17 asbestos exposure.

18 **DR. HENRY ANDERSON:** I guess there'd
19 be people that disagree with that.

20 **DR. SURESH MOOLGAVKAR:** Well, show
21 me an epidemiology study that shows these risk --

22 **DR. HENRY ANDERSON:** I ask you -- I

1 asked you what are the dose differences? I would
2 assume you are assuming radiation is a dose-
3 related effect so have you done the assessment of
4 what would -- have many cases would expect given
5 the background radiation exposure versus the doses
6 that radiation treatment is given to directly kill
7 tumor tissues.

8 **DR. SURESH MOOLGAVKAR:** Excuse me, I
9 would still answer in the same way. We have no
10 information on increased risk at low levels of
11 radiation exposure. Similarly, we have no
12 information on increased risk at low levels of
13 asbestos exposure. In fact, for asbestos
14 exposure, we have epidemiological studies of low
15 levels of exposure that show clearly that there is
16 no increased risk. For asbestos, we have those
17 studies showing no increased risk.

18 **DR. HENRY ANDERSON:** You have
19 household, you have family members, and you have
20 communities living in the neighborhood of a plant.
21 Do you consider that low-level exposure?

22 **DR. SURESH MOOLGAVKAR:** Well, it

1 depends upon the environment in which you live.

2 If you live in northern Italy around one of the
3 crocidolite plants or mines your risk is increased
4 and we actually do have information on the levels
5 of exposure to crocidolite around those plants.

6 If you look at Manville in New Jersey, there is an
7 increase in that town of Manville in New Jersey
8 because of the emission from the Johns Manville
9 plant and there was exposure to amphibole
10 asbestos.

11 And there are measurements,
12 environmental measurements around facilities and
13 factories that show there are increased levels of
14 fibers. So it is -- I would consider that a
15 medium exposure to amphibole asbestos and we know
16 that amphibole is at least two orders of magnitude
17 more potent than chrysotile in causing
18 mesothelioma.

19 **DR. DIANA WONG:** Okay.

20 **DR. KENNETH PORTIER:** Dr. Kissel --
21 Dr. Kissel, did you have a question?

22 **DR. JOHN KISSEL:** Yeah. I did. I'm

1 not immediately familiar with the brake mechanic
2 literature. Could you tell us something about how
3 large those studies were and whether they actually
4 had the power to detect risks, which in this case
5 the do-it-yourselfer brake mechanic risk estimates
6 are in the 10^{-5} , 10^{-6} level? So could those epi
7 studies, could it be possibly true that those epi
8 studies are negative, and the risks are good also?

9 **DR. SURESH MOOLGAVKAR:** Okay. So
10 let me ask you also a question in return. Would
11 you be able to design an epi study of exposure to
12 environmental cigarette smoke in which you could
13 measure the risk of the level of 10^{-5} or 10^{-4} ? How
14 do we decide that tobacco smoke does not cause
15 mesothelioma? We have multiple epidemiology
16 studies and even though tobacco smoke is a multi-
17 site carcinogen, epidemiologists decided that the
18 epidemiology does not support an association
19 between tobacco smoke and mesothelioma.

20 Now you apply the same principle to
21 the brake mechanic's epidemiology, the vehicle
22 mechanics, there are many more of those studies

1 than for tobacco smoke and mesothelioma and there
2 is no association with work as a vehicle mechanic.
3 Now you're asking for -- you'd require a study
4 involving millions of individuals to find the risk
5 of the level of 10^{-5} , but that's not reasonable.
6 When you find multiple epidemiology studies
7 showing no increase in the risk, you have to
8 conclude that that exposure does not increase the
9 risk. That is what we do in epidemiology. That
10 is how we decided that tobacco smoking does not
11 cause mesothelioma. I don't see the difference.

12 **DR. JOHN KISSEL:** Well, I think I
13 was just asking you what's the power of detection
14 of those studies and I don't think you really
15 answered the question.

16 **DR. SURESH MOOLGAVKAR:** Okay. I'll
17 answer that question too. Individually, the
18 studies, some of them have low power, some of them
19 are very large studies like the one done by Julian
20 Peto's group in the United Kingdom in 2009
21 published as Rake et al, R-A-K-E et al. And also
22 a larger report by the HESE in the United Kingdom

1 describing all the occupations in which risks are
2 -- and that is probably one of the largest case-
3 control studies involving several hundred cases of
4 mesothelioma that has been done.

5 And if you look collectively at all
6 the studies there are 15, 14, or 15 case-control
7 studies and then there are a few more cohort
8 studies, studies that I would call analytical
9 epidemiology studies. If you look collectively at
10 all those studies, those studies have more than
11 adequate power to find a relative risk of around
12 1.2. At least, I mean, maybe even lower than
13 that. You can't expect to find a relative risk of
14 1.0005 from an epidemiology study. That is not a
15 reasonable requirement.

16 **DR. DIANA WONG:** Okay. We move on
17 to the next speaker. The next speaker is
18 Jacqueline Moline. Are you online?

19 **DR. JACQUELINE MOLINE:** Can you hear
20 me?

21 **DR. DIANA WONG:** Yes.

22 **DR. JACQUELINE MOLINE:** Okay. Good

1 afternoon. My name is Jacqueline Moline. I'm a
2 board certified occupational and environmental
3 medicine specialist. I'm currently the
4 chairperson of the Department of Occupational
5 Medicine, Epidemiology, and Prevention at the
6 Zucker School of Medicine at Hofstra University
7 Northwell Health. Thank you for the opportunity
8 to speak today.

9 Throughout my career, I've dedicated
10 myself to studying the prevention and treatment of
11 exposure-related illnesses. I had the honor of
12 testifying in Congress in December regarding the
13 adverse health effects of asbestos-containing
14 cosmetic talc. In 2019 I published a paper
15 regarding mesotheliomas associated with cosmetic
16 talc. I'm concerned that the EPA's draft risk
17 assessment has serious omissions and does not
18 reflect the full magnitude of the dangers of
19 asbestos exposure.

20 Today I want to focus my comments on
21 one significant source of asbestos exposure the
22 draft fails to address and has been discussed to

1 some degree earlier today, the presence of
2 asbestos contamination in talc in industrial and
3 consumer products. There is extensive test data
4 documenting the presence of asbestos in different
5 grades of talc.

6 Now, while the FDA regulates
7 cosmetic talcum powder, other talc products in
8 industrial operations fall within the purview of
9 TSCA which is the basis for this evaluation. To
10 state the EPA does not regulate non-cosmetic talc
11 is misleading. The omission of talc from the
12 current draft also highlights two other serious
13 gaps in the evaluation, some of which have been
14 covered earlier today.

15 But this includes the fact that
16 asbestos causes ovarian cancer including asbestos
17 found in talc, yet only lung cancer and
18 mesothelioma are included in the risk evaluation.
19 In addition, asbestos found in talc was a mixture
20 of fiber types, but the EPA is only addressing
21 risks from chrysotile in the mistaken belief that
22 this is the only fiber in current products.

1 If asbestos in talc is added to the
2 draft evaluation, EPA will need to address both
3 these limitations so the talc related risks can be
4 fully assessed. Talc is the softest mineral known
5 to man. Its properties afford advantages in a
6 wide range of consumer and industrial products.
7 In the U.S. talc is found in the eastern
8 Appalachian and Piedmont regions from New England
9 to Alabama. It's also found in California,
10 Montana, Nevada, Texas, and Washington.

11 While not every talc deposit
12 contains asbestos, there are talc deposits that
13 include or are located near asbestos deposits.
14 This includes tremolite, anthophyllite, both forms
15 of asbestos, amphibole asbestos as well as
16 chrysotile. For example, talc from Death Valley,
17 California has tremolite and actinolite. Talc
18 contaminated by asbestos is regulated under TSCA.

19 As the health officials long ago
20 noted in New York industrial talc miners that they
21 were dying from lung scarring, lung cancer, and
22 pleural cancer. Their lung pathology was similar

1 to that reported in asbestosis. There have been
2 several published reports of mesothelioma among
3 these talc miners.

4 Talc based consumer products, not
5 just cosmetic talcum powder, contain asbestos.
6 Several groups found asbestos amphibole present in
7 crayons due to the addition of talc to the crayon
8 mixture. This is particularly concerning because
9 children are the primary users of crayons.

10 Industrial talc use is widespread. In 2018
11 894,000 metric tons of industrial talc were used
12 in the United States from domestic and imported
13 sources.

14 The top four areas for use are
15 plastics manufacturing, ceramics, paint, and
16 paper. It's also used in fertilizer and
17 insecticides on seeds, and in gloves to assist in
18 donning. It's used in the plastics as a filler,
19 ceramic products such as bathroom fixtures,
20 ceramic tile, pottery, and dinnerware often
21 contain asbestos. When it's used as a filler the
22 talc in ceramics, talc can improve the firing

1 characteristics of the greenware and its strength.
2 Mix in the talc ceramic risk poses risks for
3 potters, manufacturers, and hobbyists who work
4 with ceramics.

5 It's used in paints as an extender
6 and filler. It can improve the opacity --

7 **DR. DIANA WONG:** You have one
8 minute.

9 **DR. JACQUELINE MOLINE:** --
10 brightness and whiteness of paper to improve
11 paper's ability to absorb ink. It's used in
12 certain construction products as a filler. These
13 industrial uses likely expose thousands of workers
14 and consumers to cosmetic -- to talc products
15 through inhalation. The extent to which this talc
16 contains asbestos isn't known. Based on my
17 investigations the link between talc-based talcum
18 powder and mesothelioma and ovarian cancer of
19 women, I'm concerned industrial talc exposure also
20 causes asbestos-related death and disease.

21 In conclusion, the omission of talc-
22 based consumer and industrial applications in the

1 draft evaluation is a significant gap because of
2 the likely some grades of talc used in these
3 products are contaminated by asbestos putting
4 consumers and workers at risk. These -- EPA
5 should include these conditions of use in its risk
6 evaluation to ensure that these risks are fully
7 assessed it should also expand the evaluation to
8 include the other known health impacts of asbestos
9 --

10 **DR. DIANA WONG:** Time's up.

11 **DR. JACQUELINE MOLINE:** -- cancer
12 and address all fiber types, not simply
13 chrysotile. Thank you.

14 **DR. DIANA WONG:** Any questions for
15 the speaker? Any questions? If not, the next
16 speaker is Jessica Ryman-Rasmussen.

17 **DR. JESSICA RYMAN-RASMUSSEN:** Yes.
18 I am present on the line. Can you hear me?

19 **DR. DIANA WONG:** Yes. Go ahead.

20 **DR. JESSICA RYMAN-RASMUSSEN:** Okay.
21 Great. Good afternoon. My name is Jessica Ryman-
22 Rasmussen, I am a senior policy advisor at the

1 American Petroleum Institute, API. API is a
2 national trade association that represents all
3 facets of the natural gas and oil industry. As a
4 core component of our business model, we
5 prioritize the promotion of public health and
6 environmental safety while ensuring a strong,
7 viable, and sustainable U.S. natural gas and oil
8 industry. API's written comments on the draft
9 risk evaluation for asbestos address both the
10 hazard and exposure components of the risk
11 evaluation.

12 Today, we introduce and summarize
13 these written comments which have been submitted
14 to the docket where they are available in greater
15 detail. First, we note that for the systematic
16 review, the scope of the literature search appears
17 to go beyond chrysotile asbestos. This is
18 important because it suggests that the inhalation
19 unit risk factor for chrysotile asbestos is
20 confounded by other asbestos forms. Moreover, we
21 note in our written comments additional variables
22 that impact the magnitude of the inhalation unit

1 risk that were not well described.

2 The conditions of use for chrysotile
3 asbestos relevant to the natural gas and oil
4 industry are in gaskets and in oilfield brake
5 blocks. For both of these conditions of use, EPA
6 has made exposure assumptions that result in
7 overestimates of risk. First, API's written
8 comments provide information indicating that EPA's
9 asbestos fiber and exposure characterization
10 methodology overestimate exposure.

11 Second, our written comments provide
12 information indicating that EPA's calculations for
13 time-weighted averages over an eight-hour shift
14 overestimate exposure. Finally, EPA does not seem
15 to recognize that occupational non-user exposures
16 to asbestos are restricted by OSHA regulations.

17 Our written comments, therefore,
18 provide regulatory information indicating that
19 requirements to protect occupational non-users are
20 already in place. Finally, we note that asbestos
21 is highly and broadly regulated. EPA regulates
22 asbestos in air and soil as well as when disposed

1 as hazardous waste. Occupational exposures to
2 asbestos are regulated by both the Occupational
3 Safety and Health Administration and in the mining
4 industry by the Mine Safety and Health
5 Administration. Consumer exposures are regulated
6 by the Consumer Products Safety Commission.

7 EPA should consider the protections
8 put in place by these and other entities when
9 considering exposures and workers and in the
10 general population. We appreciate the opportunity
11 to provide oral comments today and we look forward
12 to the revised assessment.

13 **DR. DIANA WONG:** Thank you. Any
14 questions? Any questions for the speaker? If
15 not, our next speaker is Gabor Mezei.

16 **DR. GABOR MEZEI:** Yes. Good
17 afternoon. I'm here. Can you hear me?

18 **DR. DIANA WONG:** Yes. You have to
19 wait for your slides. You have to wait for your
20 slides.

21 **DR. GABOR MEZEI:** Yes. Thank you.

22 **DR. DIANA WONG:** Go ahead. You can

1 start.

2 **DR. GABOR MEZEI:** Okay. So thank
3 you very much. Good afternoon. My name is Gabor
4 Mezei. I'm a medical doctor and an epidemiologist
5 and I'm currently a principal scientist with
6 Exponent. Next slide, please.

7 Disclosures are included in my
8 written submission which I submitted with my
9 colleagues. And of these three main comments that
10 are included in our written submissions, I would
11 like to focus my oral comments on the second
12 bullet here, namely that a large body of
13 epidemiologic evidence was ignored by the Agency.
14 Next slide.

15 The large body of epidemiologic
16 evidence that I referred to is specific to motor
17 vehicle mechanics, an occupational group that are
18 exposed to -- that is exposed to lower levels of
19 chrysotiles. And a number of studies, dozens of
20 epidemiologic studies, analytic epidemiologic
21 studies were conducted to investigate the
22 increased risk of mesothelioma and lung cancer.

1 And many of these studies were not funded by the
2 industry. These studies were conducted in various
3 time periods using various study designs, data
4 collection methods, and various research groups
5 across Europe and North America mostly. Next
6 slide.

7 Dr. Moolgavkar showed the hierarchy
8 of evidence that we epidemiologists look at and
9 obviously, the top of the evidence are the
10 systematic reviews and meta-analyses and you see
11 the randomized trials are not applicable to this
12 area. The highest level of analytical
13 epidemiologic evidence are the case-control
14 studies and cohort studies.

15 The bottom of the hierarchy are the
16 case reports and the case series which are not
17 epidemiologic studies in themselves because they
18 lack an appropriate comparison group and
19 therefore, they are not able to demonstrate any
20 statistical association. They purely just
21 register the co-occurrence of an exposure and a
22 disease. They are not able to look at a

1 particular association, therefore we cannot draw
2 causal inference based on these studies. Next.

3 This slide and the next slide shows
4 a couple of dozens of epidemiologic studies that
5 look at the incidents -- whether the risk of
6 mesothelioma is increased among motor vehicle
7 mechanics who are exposed to lower level of
8 chrysotile due to their work with brakes and other
9 products. And then we can see from this slide
10 that a number of case control studies -- more than
11 a dozen case control studies -- and then also a
12 number of cohort studies that looked at this area
13 and then none of them report a statistically-
14 significant increased risk of mesothelioma among
15 motor vehicle mechanics. Next slide, please.

16 The same is true for lung cancer.
17 So there are approximately a dozen case-control
18 epidemiologic studies and then also two cohort
19 studies that looked at the risk of lung cancer
20 among motor vehicle mechanics. And I would like
21 to point out that these studies were all
22 controlling for the potential confounding effect

1 of smoking. And none of these studies indicated a
2 statistically-significant increased risk of lung
3 cancer among motor vehicle mechanics. Next slide,
4 please.

5 And as I mentioned, the highest
6 level of epidemiologic evidence are the systematic
7 reviews and meta-analysis that rely on the
8 available body of evidence and summarize the
9 results. And Professor Garabrant conducted the
10 most recent meta-analysis of mesothelioma risk
11 among motor vehicle mechanics. And he carefully
12 evaluated the strength and limitations of each of
13 the included --

14 **DR. DIANA WONG:** You have one
15 minute.

16 **DR. GABOR MEZEI:** --
17 epidemiologists. Thank you. And then arrived to
18 the conclusion that there is no increased risk of
19 mesothelioma among motor vehicle mechanics. Next
20 slide, please.

21 This is my final slide. So some
22 authors arrived to a different conclusion. But I

1 would like to point out that they basically
2 dismiss the analytical epidemiology studies and
3 then rely on case series and case reports, which
4 are not epidemiology studies.

5 And I would like to point out again
6 that the Hill criteria was designed to distinguish
7 between statistical association and causal
8 association. And so, when there is no statistical
9 association the application of Hill criteria is
10 not appropriate. And in this case, low-level
11 chrysotile exposure there was no statistically
12 significant association. Thank you.

13 **DR. DIANA WONG:** Okay. Thank you.
14 Any questions for the speaker?

15 **DR. ELIZABETH SHEPPARD:** This is
16 Lianne Sheppard I actually have a comment more
17 than a question just to note that pretty much all
18 of the studies that were shown on those four spots
19 showed a wide range of effect and were consistent
20 with elevated risk and I think focusing on
21 statistical significance is misleading. And so I
22 just wanted to make that comment.

1 **DR. DIANA WONG:** Any questions? Any
2 questions for the speaker? If not, the next
3 speaker is Barry Castleman. Is he online?

4 **DR. BARRY CASTELMAN:** Hello. Do you
5 hear me?

6 **DR. DIANA WONG:** Yes.

7 **DR. BARRY CASTELMAN:** Yes. I have
8 worked with environmental groups, government
9 organizations, and international organizations on
10 asbestos and public health for over 40 years. I
11 also testify about the public health and corporate
12 history of asbestos in personal injury trials,
13 usually at the request of plaintiffs. I've
14 published widely on asbestos in the scientific
15 literature.

16 The EPA draft repeatedly
17 underestimates asbestos exposures from asbestos
18 products use. In particular, it assumes that
19 750,000 mechanics in this country don't use
20 compressed air hoses to clean brake assemblies
21 without presenting any evidence that this is the
22 case, other than the publication of regulations in

1 the -- on the federal -- in the federal register
2 and the availability of an EPA guidance document
3 on the internet, which I think very few mechanics
4 have probably seen.

5 The EPA draft neglects the Longo
6 study on wire brushing of asbestos-containing
7 gasketing from pipe flanges. During the life
8 cycle of the gaskets, the adhering gasketing on
9 surfacing and its removal is the dustiest
10 operation. And if this isn't counted the dangers
11 of these products are underestimated
12 substantially.

13 The EPA draft also ignores legacy
14 exposures including flooring felt -- there are
15 130,000 metric tons a year of asbestos was used in
16 flooring felt in the 1970s. This stuff's out
17 there all over the place. The sanding technique
18 widely used in the field to remove patches of
19 cemented asbestos felt adhering to floors and --
20 is runs from 37 to 56 fibers per cc according to
21 an internal document of one of the companies in
22 the field who -- who's memo I've submitted in my

1 statement. And even after the sanding, 13 fibers
2 per cc were in the air.

3 Hundreds of thousands of miles of
4 asbestos cement pipes supply drinking water and
5 carry wastewater in this country creating a hazard
6 for thousands of utility workers and the public.
7 This hazard isn't noted at all in the EPA draft.

8 With respect to asbestos product
9 imports, these are not fully investigated by the
10 EPA. The one that concerns me most is 85 metric
11 tons a year in 2019 continuing through the decade
12 -- and we've called attention to EPA about this
13 before, of asbestos yarn and thread from Mexico.

14 The EPA has not gone to the importer
15 of record to find out how this stuff is used, what
16 it's used in, and what are the occupational and
17 environmental exposures. And the EPA has also not
18 gone to the recipients of asbestos cement and
19 asbestos building materials that are available to
20 them through U.S. Customs records.

21 The EPA tracking of imported gaskets
22 was limited to one importer and one of the firm's

1 customers. The other importers and their
2 customers should have been questioned. With
3 respect to chlor-alkali plants, the EPA is relying
4 on selected data provided by the industry trade
5 association and does not have complete information
6 for the 15 plants where asbestos diaphragms are
7 used.

8 EPA did only two site visits and
9 admits that the high exposure activities are
10 probably not included in the air monitoring data
11 that was selected for submission to the EPA. EPA
12 should have gotten all of the OSHA monitoring data
13 that these plants created since that was required
14 in 1972 under the OSHA regs.

15 Chemical industry trade has said
16 that heavy fines were levied by EPA against chlor-
17 alkali producers over asbestos and cell renewal
18 areas. And we've repeatedly provided the trade
19 article to the EPA and asked them to show us
20 what's their enforcement record and this has been
21 withheld so far despite our repeated requests.

22 The EPA draft says nothing about the

1 health dangers of contaminant asbestos in talc,
2 vermiculite, taconite, and crushed stone.

3 Vermiculite from Libby, Montana is referred to as
4 Libby amphibole asbestos in EPA's own reports.

5 **DR. DIANA WONG:** You have one minute
6 left.

7 **DR. BARRY CASTELMAN:** Surely the
8 presence of this in millions of home attics,
9 especially as people are cooped up inside now is
10 creating an extraordinary danger and the EPA ought
11 to deal with that with some urgency. These
12 hazards to the public need to be fully
13 investigated.

14 Enough is already known however to
15 justify banning imported asbestos and asbestos
16 products as over 60 countries have already done.
17 Last, I wish to pay tribute to the career civil
18 servants who are hanging in there trying to
19 protect the environment during the remainder of
20 this administration. Thank you.

21 **DR. DIANA WONG:** Thank you. Any
22 questions for the speaker? Any questions? The

1 next speaker is Steven Compton. Is he online?

2 **DR. STEVEN COMPTON:** Yes. I'm here.
3 Can you hear me?

4 **DR. DIANA WONG:** Yes. We have your
5 slides so you may start.

6 **DR. STEVEN COMPTON:** Okay. Good
7 afternoon. Thank you for the opportunity to
8 speak. My name is Steven Compton. I have a Ph.D.
9 in condensed metaphysics and a background in
10 microscopy. So my opinions will focus on the
11 asbestos content of different materials and
12 mechanisms by which those materials may release
13 air born fibers. The company I work with MVA
14 Scientific Consultants has been analyzing samples
15 and performing activity-based sampling simulations
16 for several decades for clients in industry and in
17 litigation, mostly for attorneys representing
18 plaintiffs, but also for attorneys representing
19 defendants.

20 Because of this, I am in a unique
21 position to have exposure data, some published and
22 some unpublished which may be helpful to the

1 panel. I've also had an opportunity to review the
2 data from other groups. I'm encouraged that the
3 risk assessment has determined that there is
4 unreasonable risk when working with asbestos, but
5 I do have serious concerns that the assessment is
6 delaying comment regarding legacy uses. As long
7 as there are still asbestos-containing materials
8 hiding in plain sight there is no way to de-couple
9 those legacy products from current potential
10 exposures to building maintenance crews,
11 firefighters, mechanics, workers from other
12 professional trades, as well as individuals
13 performing DIY projects.

14 Because of this, the review appears
15 to ignore the wealth of knowledge regarding
16 construction materials and other products that are
17 presumably no longer on the market if they're out
18 there and they contain chrysotile. The review
19 also appears to belittle the importance of
20 materials that are currently available on the
21 market, like talc and other minerals, which could
22 potentially contain a host of hazardous mineral

1 fibers not limited to chrysotile.

2 But I also have concerns related to
3 the exposures that are covered under the current
4 draft risk evaluation. Most importantly, I've
5 noticed that a number of the published studies
6 that serve as the source for exposure monitoring
7 data were conducted by a few specific companies
8 who are performing studies at the request of
9 defendants in litigation. These companies have
10 been shrouded in controversy regarding industry
11 involvement, and how those studies and the
12 resulting data should be interpreted, and how they
13 have been criticized for censoring data that
14 proves to be unfavorable to their clients. Next
15 slide, please.

16 If we take gasket work as an
17 example, on slides two and three I've summarized
18 some exposure monitoring data from different
19 studies looking at the formation/installation and
20 removal of gaskets, respectively. Some of the
21 studies have been reviewed by this panel and some
22 have not. Next slide, please.

1 If we look at this slide, in
2 particular, I'd like to point out that some of
3 these data points are three to four orders of
4 magnitude lower than the rest. These data points
5 come from a 2002 paper published by Boelter.
6 There are two reasons why these numbers are lower
7 than the rest of the studies shown on slide three.
8 First, the study included work on gaskets that
9 didn't even contain asbestos. This is a common
10 theme that I've seen in several industry-sponsored
11 studies.

12 Second, a diluted eight hour time-
13 weighted exposure is estimated without any regard
14 to residual exposure or secondary exposure from
15 settled dust on floors, equipment, and the workers
16 themselves. Something that this panel
17 acknowledges is important based on the
18 presentation that was given this morning. Next
19 slide, please.

20 I've also included references to
21 that data. Again, next slide. And next slide.
22 Similarly, next slide. I've assembled a

1 collection of exposure monitoring data -- next
2 slide, on the topic of brake linings. Next slide.
3 Next slide. And next slide. This data should be
4 considered by the panel. Next slide. Several of
5 these have been included by the panel's review.
6 Next slide.

7 However, I have serious concerns the
8 industry-sponsored data that was included is
9 artificially reducing any reasonable estimate for
10 potential exposure and therefore diminishing the
11 implications of risk of consumer risk that has
12 been evaluated by this panel. Next slide.

13 Finally, on the last slide, I've
14 summarized some of the exposure monitoring data --
15 next slide, that I've published in a 2018 paper on
16 fiber release from asbestos cement - board. Next
17 slide.

18 **DR. DIANA WONG:** You have one more
19 minute.

20 **DR. STEVEN COMPTON:** Thank you.
21 This source of potential exposure has relevance to
22 workers working on previously installed materials.

1 Next slide. But also to improperly discarded
2 materials that may be discovered and handled by
3 children or other unsuspecting individuals. Next
4 slide. So what Agency should cover that potential
5 exposure? Since the panel has deemed previously
6 installed products to be part of the subsequent
7 legacy use that will be analyzed, perhaps this
8 data can be utilized when those potential
9 exposures are eventually evaluated. Thank you
10 very much for your time.

11 **DR. DIANA WONG:** Thank you. Any
12 questions for the speaker?

13 **DR. HENRY ANDERSON:** Yes. I have a
14 question. Given your experience in work of the
15 field, on one of the issues is estimating the
16 occupational non-user's exposure as well as
17 bystanders. And EPA has used a reduction factor
18 of, I think 8.7 or something like that, to
19 estimate the bystander exposure from a worker
20 exposure without about a five-foot or so area.
21 What's your experience on the fall off of
22 exposures at close to somebody's who's disturbing

1 brakes or doing some of this work?

2 **DR. STEVEN COMPTON:** That's a great
3 question. I've looked at and incorporated area
4 samples in chamber studies that we've conducted in
5 the past so I have that kind of data for specific
6 activities, but I've never tried to take that
7 actual data and come up with some kind of general
8 rule of thumb. I know there's a 2011 Donovan
9 paper that attempts to do that. I would consider
10 that a conservative approach because it does
11 depend on -- I wouldn't necessarily consider it to
12 be a linear relationship. And it also depends on
13 whether you're talking about OSHA fibers, things
14 that are readily resolvable by PCM or fibers that
15 can be identified by TEM.

16 **DR. HENRY ANDERSON:** Thank you.

17 **DR. STEVEN COMPTON:** You're welcome.

18 **DR. DIANA WONG:** Any more questions?

19 **DR. JOHN KISSEL:** Yeah. This is
20 John Kissel. Can we assume that all of the
21 numbers on your slides are fibers per cc?

22 **DR. STEVEN COMPTON:** They are fibers

1 per cc, and they are for the duration that the
2 activity itself takes place. So for with the
3 exception of the Boelter study, they're not eight
4 hour time-weighted averages. There aren't any
5 assumptions based on what the worker may or may
6 not have done for the rest of his or her time
7 working with that material. It's just evaluating
8 the activity and what the average airborne fiber
9 concentration was in the personal breathing zone
10 of that worker during the length of that activity.

11 **DR. JOHN KISSEL:** Okay. I also note
12 that many of your -- the footnotes are to things
13 that are not in the conventional scientific
14 literature. What's the likelihood if we Googled
15 one of those things, we would actually be able to
16 find such a study?

17 **DR. STEVEN COMPTON:** If any --

18 **DR. JOHN KISSEL:** You said --

19 **DR. STEVEN COMPTON:** -- that's been
20 published -- oh, go ahead.

21 **DR. JOHN KISSEL:** You've said that
22 we should use this data but it's not clear to me

1 that the data are actually accessible.

2 **DR. STEVEN COMPTON:** I don't know
3 whether it would be generally accessible without
4 knowledge of the data. Obviously, if you know
5 about it then anyone who's -- who can contact me I
6 would be willing to provide it and it's available
7 through Google search on our website by request.
8 But in terms of journal publications, if it's not
9 listed as being published in the journal it would
10 either need to be something that you would have to
11 obtain either through me or by looking at the
12 public record in any kind of lawsuit where it's
13 been referenced in the past.

14 **DR. JOHN KISSEL:** Okay. Thank you.

15 **DR. STEVEN COMPTON:** You're welcome.

16 **DR. DIANA WONG:** Okay. The next
17 speaker is Richard Lemen. Is he online?

18 **DR. RICHARD LEMEN:** Hello?

19 **DR. DIANA WONG:** Good.

20 **DR. RICHARD LEMEN:** Hello.

21 **DR. DIANA WONG:** You're ready?

22 **DR. RICHARD LEMEN:** Yes. I'm ready.

1 **DR. DIANA WONG:** Go ahead. You can
2 start.

3 **DR. RICHARD LEMEN:** My name is
4 Richard Lemen. I'm a former Assistant Surgeon
5 General of the United States. I'm a former -- I'm
6 Deputy Director and Acting Director of the
7 National Institute for Occupational Safety and
8 Health, and I have testified in litigation
9 principally on behalf of plaintiffs and I've been
10 studying asbestos for a little over 50 years.

11 The danger in relying on only
12 selected epidemiology studies is revealed by the
13 growth of our historical understanding of asbestos
14 hazards. In the early 1970s studies estimated
15 that exposure to asbestos below 200 to 300 fiber
16 per cc years were not associated with increased
17 cancer deaths. By the 1980s studies found no
18 increased risk of lung cancer deaths below 20
19 fibers per cc years, a level 10 times lower. By
20 1998 another study found exposure of 0.5 to 0.99
21 fibers per cc years produced a four-fold increased
22 risk of cancer. And in the early 2000s yet

1 another study found roughly an eight-fold
2 increased risk at exposures above 0.15 fiber per
3 cc years.

4 These historical lessons repeatedly
5 show we are incapable of identifying a threshold
6 level of exposure. The draft excludes legacy
7 asbestos exposures which make up by far the
8 greatest potential exposures to asbestos in the
9 U.S. today. Additionally, limiting its assessment
10 to just lung cancer and mesothelioma where there
11 are other recognized cancers, and non-malignant
12 lung disorders still occurs in the United States
13 today is a flaw in the proposal as cited by Drs.
14 Frank and others.

15 In limiting the draft risk
16 evaluation to do a chrysotile specific risk
17 analysis is not realistic since by doing so it
18 screened out all studies in which exposures were
19 not solely to chrysotile, ending up with only two
20 studies to calculate the IUR. This makes no
21 sense. Unnecessarily limiting studies using the
22 IUR increases uncertainties where expressing their

1 relevance to other exposure situations, and
2 because all asbestos studies have limitations such
3 as low exposure is made -- such as how low
4 exposure is measured, or death and disease was
5 tracked. Not to mention the smaller the database
6 the greater likelihood the peculiarities of the
7 individual studies will drive risk calculations.

8 Relying -- and I would like to put
9 in here that relying on lung cancer tissue alone
10 is not appropriate for analysis for chrysotile has
11 been shown in many studies. The draft risk
12 evaluation abandons the customary linear approach
13 to dose-response analysis and applies exponential
14 model that agencies have never used before and has
15 never -- and has been disfavored by the consensus
16 groups of asbestos scientists.

17 The draft risk evaluation approach
18 calculates a risk which is considerably lower than
19 the IUR used by EPA in the previous 1988 peer
20 review IRIS assessment and by other agencies, like
21 OSHA, using a similar framework. This in itself
22 is of significant concern.

1 The draft risk evaluation suffers
2 from the same underlying error that many now
3 debunked risk assessments have suffered in
4 comparing one set of exposure studies with
5 entirely separate epidemiological studies. The
6 universal consensus remains that there has not
7 been shown a level of exposure below which
8 increased risk of cancer has been identified. In
9 the United States today, recent estimates estimate
10 that there are still nearly 40,000 deaths
11 occurring each year from exposure to asbestos.
12 And much of those deaths are a result of legacy
13 asbestos. I'd like to thank you for your time and
14 my written comments in full have been submitted to
15 the register -- to the docket. Thank you very
16 much.

17 **DR. DIANA WONG:** Thank you. Is
18 there question for the speaker? Any questions?
19 The next speaker is Liz Hitchcock. Is she online?

20 **MS. LIZ HITCHCOCK:** I am on the
21 line.

22 **DR. DIANA WONG:** Okay. Thank you.

1 **MS. LIZ HITCHCOCK:** Are you ready
2 for me?

3 **DR. DIANA WONG:** Yes. Please.

4 **MS. LIZ HITCHCOCK:** Okay. Good
5 afternoon and thank you for the opportunity to
6 provide comments to the committee about the EPA's
7 draft risk evaluation for asbestos. We are
8 grateful to the members of the SACC for your
9 service at a challenging time to participate in
10 these meetings. My name is Liz Hitchcock and I
11 direct Safer Chemicals Healthy Families.

12 Throughout the legislative campaign
13 to reform TSCA, we led a coalition of hundreds of
14 health, consumer, environment, and business groups
15 to make a case for a reformed law that could truly
16 protect public health from hazardous chemicals and
17 substances in our home and workplaces. My
18 comments today are amplified in the letter we
19 submitted to the docket cosigned by 27
20 organizations.

21 TSCA is a tough law to explain to
22 the general public and to most members of Congress

1 and their staff. Explaining what was wrong with
2 it got really complicated really fast. One of the
3 best ways to explain the problem we faced with
4 TSCA was to tell the story of how EPA tried but
5 couldn't even ban asbestos under the '76 law. The
6 public understands the dangers of asbestos.
7 Thousands of Americans die from illnesses
8 connected to asbestos exposures every year. We
9 all know someone who has lost someone they love.

10 The failure to protect us from
11 asbestos became a symbol of TSCA's failure to
12 protect us from other toxic chemicals. So when
13 the Lautenberg Act was finally passed it gave EPA
14 the tools, they needed to do the job of protecting
15 us from one of the most dangerous substances we
16 know. We fear that the failure of this draft
17 evaluation to incorporate comprehensive use and
18 exposure information will mean continued failure
19 to protect workers and consumers and will signal a
20 failure of our reformed TSCA. For decades
21 asbestos has been all around us, in your homes,
22 schools, public buildings, and workplaces across

1 America.

2 Asbestos remains in the built
3 environment where it continues to pose a threat to
4 public health to this day. As states get back to
5 work in the wake of the Coronavirus pandemic,
6 among the first to open up is construction work.
7 I'm in Washington D.C. where changes are
8 constantly being made to your built environment
9 with buildings torn down or gutted to make way for
10 new construction all over the city. As that
11 happens across the country, asbestos waste is
12 generated and disposed of in the U.S. in
13 significant quantities. That asbestos waste in
14 commerce, at landfills, and construction sites
15 puts workers and the public in significant danger.

16 Demolition and construction are not
17 the only on the job exposures from legacy
18 asbestos. Firefighters face greater risks from
19 asbestos exposures and related diseases as they do
20 their jobs protecting the public. School
21 teachers, especially in elementary and middle
22 schools are at higher risks for mesothelioma than

1 the general population because of widespread
2 presence of asbestos in schools built in the '60s
3 and '70s.

4 The asbestos risk evaluation is an
5 important tool to update our understanding of the
6 prevalence of legacy asbestos and its risk.
7 Without this important information, how can we
8 protect those of us at risk of exposure? Our
9 concern about early indications that EPA would not
10 meet its obligations under TSCA lead us to file
11 our first ever lawsuit to compel EPA to address
12 the use and disposal of legacy asbestos in its
13 risk evaluation.

14 While the court agreed with us and
15 our co-plaintiffs this draft risk evaluation fails
16 to address the risks from legacy asbestos. The
17 American public expects and deserves better from
18 new TSCA. The science demands better of this risk
19 evaluation. EPA is obliged to include all uses in
20 its risk evaluation including legacy uses. It has
21 failed to do so in this evaluation, thus failing
22 to provide the complete and accurate accounting of

1 how asbestos affects public health that the public
2 is entitled to and that the EPA needs for the best
3 possible understanding of asbestos risk an --

4 **DR. DIANA WONG:** You have one more
5 minute.

6 **MS. LIZ HITCHCOCK:** -- to inform its
7 management of risks for this deadly substance. We
8 urge the SACC to call on EPA to strengthen this
9 current draft to incorporate comprehensive use and
10 exposure information and the best available
11 science and to remove the many exclusions and
12 limitations that result in this understatement of
13 risk. These improvements would both reinforce
14 EPA's determinations of the unreasonable risk and
15 provide additional support for the complete
16 asbestos ban that EPA must impose under TSCA.
17 Thank you for your time and thank you for your
18 service on this important committee.

19 **DR. DIANA WONG:** Thank you. Any
20 questions for the speaker? Any questions? If
21 not, we can go back to see if some of the speakers
22 are here now. Do we have David Bernstein?

1 **MR. MARTIN ALVARADO:** That's a no
2 for Berstein.

3 **DR. DIANA WONG:** Okay. How about
4 Karen Minott?

5 **MR. MARTIN ALVARADO:** She is also
6 not connected.

7 **DR. DIANA WONG:** She's not --

8 **MR. MARTIN ALVARADO:** Yeah. And
9 Elissa Favata is ready.

10 **DR. DIANA WONG:** Oh, okay. She
11 doesn't have slides, right? Okay.

12 **MR. MARTIN ALVARADO:** Correct. She
13 was --

14 **DR. DIANA WONG:** All right. Go
15 ahead. Go ahead. Ready?

16 **MR. MARTIN ALVARADO:** Ms. Favata are
17 you ready? You are unmuted in WebEx.

18 **DR. DIANA WONG:** Are you ready to
19 speak?

20 **MR. MARTIN ALVARADO:** Yes.

21 **DR. DIANA WONG:** If you're ready,
22 please talk.

1 **MR. MARTIN ALVARADO:** Ms. Favata,
2 you might be muted on your line. She may be
3 experiencing technical problems.

4 **DR. KENNETH PORTIER:** Do you want to
5 try Denise Winder?

6 **DR. DIANA WONG:** Yeah. Sure.
7 Denise?

8 **DR. KENNETH PORTIER:** She's in here
9 twice and Martin --

10 **DR. DIANA WONG:** I know. Go ahead.
11 If she's ready, go ahead. We don't hear her
12 though.

13 **MR. MARTIN ALVARADO:** Yes. I've
14 gone ahead and unmuted the one line that I'm able
15 to. The other line is still muted. I don't know
16 if she can hear us. Ms. Winder, we can't hear
17 you. I've gone ahead and muted her. I don't
18 think she's available.

19 **DR. DIANA WONG:** How about going
20 back to Elissa Favata.

21 **MR. MARTIN ALVARADO:** I've unmuted
22 her line. She's ready to go if she can hear us

1 and is ready to speak.

2 DR. DIANA WONG: Are you ready to
3 speak? We don't hear her.

4 MR. MARTIN ALVARADO: Yeah. Isn't
5 clear that she is available.

6 DR. DIANA WONG: Okay. Who else is
7 online because we were missing a few?

8 MR. MARTIN ALVARADO: I think that's
9 everybody.

10 DR. DIANA WONG: How about Doug
11 Gillepsie?

12 MR. MARTIN ALVARADO: He dropped
13 off.

14 DR. DIANA WONG: Oh. He dropped
15 off? Okay.

16 DR. KENNETH PORTIER: This is Ken
17 Portier. There was a Dr. Castleman or Mr.
18 Castleman.

19 MR. MARTIN ALVARADO: He spoke. He
20 spoke.

21 DR. DIANA WONG: Yeah. He did.

22 DR. KENNETH PORTIER: He did. Very

1 good.

2 DR. DIANA WONG: Barry Castleman, he
3 spoke.

4 DR. KENNETH PORTIER: Yeah. I
5 missed that.

6 MR. MARTIN ALVARADO: He was number
7 31.

8 DR. KENNETH PORTIER: So we're still
9 missing Favata.

10 DR. DIANA WONG: Yeah. She was
11 trying to get on but for some reason did not go
12 through.

13 MR. MARTIN ALVARADO: The other
14 names besides Favata or Winder, we have not seen
15 them log in, so they evidently were not able to
16 get onto WebEx or withdrew for some reason.

17 DR. DIANA WONG: Okay. So they are
18 not online anymore.

19 MR. MARTIN ALVARADO: That's
20 correct.

21 DR. DIANA WONG: Okay. How about
22 Alec Farquhar?

1 **MR. MARTIN ALVARADO:** He sent an
2 email saying he was caught up in COVID and could
3 not participate today. It just arrived as the
4 public comment period was starting.

5 **DR. DIANA WONG:** Okay. So I think
6 we covered all the speakers if nobody else is
7 going to be online.

8 **MR. MARTIN ALVARADO:** Can those who
9 are not able to submit still submit to the public
10 docket?

11 **DR. DIANA WONG:** Yes. Yes. They
12 can also send me the comment because it's oral
13 comments so they can just send it to me directly.

14 **DR. KENNETH PORTIER:** Thank you,
15 Diana, this is Ken.

16 **DR. DIANA WONG:** Yes.

17 **DR. KENNETH PORTIER:** This is Ken.
18 Why don't we go ahead and move on with the agenda
19 then? We recognize the fact that there are a
20 couple of people that may want to comment to the
21 committee. We strongly recommend that they write
22 their comments and submit them to the docket, and

1 I will ensure that through the DFO, that the
2 committee gets to see those.

3 I'm just reminding that those
4 comments need to arrive probably at the latest
5 Wednesday afternoon to give the committee a chance
6 to look at it and provide any comments because we
7 plan to complete our discussions by Thursday. At
8 this point I think we're going to close the public
9 comment section for today.

10 I wanted to point out that I've been
11 in the background capturing the unanswered
12 questions from this morning's discussion of the
13 EPA presentation. And I'm very shortly going to
14 submit that document to Dr. Wong, who then can
15 submit it back to the EPA staff so they'll have
16 something in writing as to what questions we felt
17 were unanswered in this morning's session.

18 The meeting tomorrow morning will
19 begin again at 10 a.m. eastern time and we'll do
20 some follow up on today's discussion and then
21 hopefully jump into the committee discussion on
22 the EPA questions starting fairly early. I don't

1 think there are any open issues at this point.

2 Dr. Wong, I think I'll turn it over to you for any
3 final comments as we close the meeting --

4 **DR. DIANA WONG:** Yes --

5 **DR. KENNETH PORTIER:** -- we ask that
6 the committee stay on for --

7 **DR. DIANA WONG:** So, we're going to
8 close the public comment section and adjourn for
9 the day and then we will reconvene tomorrow
10 morning at 10 a.m. However, the panel members can
11 stay for the administrative meeting.

12 **DR. KENNETH PORTIER:** So with that,
13 we will close the public meeting and reconvene
14 tomorrow morning at 10. Thank you.

15
16 **[MEETING ADJOURNED FOR THE DAY]**
17

OPENING OF MEETING - DAY 2

MS. SARA WILSON: Good morning, everyone. We have about two minutes until the meeting begins. We'll let people continue to log in, and then we will begin. Thank you. Good morning, everyone. Welcome to this meeting on the U.S. EPA Peer Review of the Draft Risk Evaluation for Asbestos. Battelle is an EPA contractor providing meeting support for this series.

This event is being recorded. Please be aware that the host may use Webex chat to share announcements with all attendees, but attendees will not be able to respond to the chat. I will now introduce Dr. Diana Wong, the designated federal official.

DR. DIANA WONG: Good morning. I am Dr. Diana Wong and the designated federal officer. It is my pleasure to open the second day of the four-day meeting for the Science Advisory Committee on Chemicals, TSCA SACC, Peer Review of EPA's of Draft Risk Evaluation for Asbestos.

1 Yesterday's Webex-hosted meeting went well.

2 However, if you encounter any technical
3 difficulties, any problems with audio or video
4 transmission today please go to [www.epa.gov/tsca-](http://www.epa.gov/tsca-peer-review)
5 [peer-review](http://www.epa.gov/tsca-peer-review).

6 A reminder to the peer reviewers, if
7 you must step away from the meeting for a time,
8 please send a note to myself and the chair, and
9 please let us know when you're back. In a minute
10 we will do our check-in roll call and then start
11 today's meeting. I now turn the meeting over to
12 our chair, Dr. Ken Portier.

13 **DR. KENNETH PORTIER:** Good morning
14 and welcome. We'll start with the roll call to
15 establish who on the Committee is attending this
16 morning. Dr. Anderson.

17 **DR. HENRY ANDERSON:** I'm present.

18 **DR. KENNETH PORTIER:** Dr. Barton.
19 Dr. Bennett.

20 **DR. STEVEN BENNETT:** Good morning.
21 I am present.

22 **DR. KENNETH PORTIER:** Dr. Blystone.

1 DR. SHERI BLYSTONE: Good morning.

2 DR. KENNETH PORTIER: Dr. Cory-

3 Slechta?

4 DR. DEBORAH CORY-SLECHTA: I'm here.

5 DR. KENNETH PORTIER: Dr. Davies.

6 DR. HOLLY DAVIES: I'm here.

7 DR. KENNETH PORTIER: Dr. Doucette.

8 DR. WILLIAM DOUCETTE: Virtually

9 present.

10 DR. KENNETH PORTIER: Dr. Jiménez-

11 Gonzalez.

12 DR. CONCEPCIÓN JIMÉNEZ-GONZALEZ:

13 Here.

14 DR. KENNETH PORTIER: Dr. Johnson.

15 DR. MARK JOHNSON: Good morning.

16 DR. KENNETH PORTIER: Dr. Kaufman.

17 MR. ALAN KAUFMAN: Good morning.

18 I'm here.

19 DR. KENNETH PORTIER: Dr. Kissel.

20 DR. JOHN KISSEL: Here.

21 DR. KENNETH PORTIER: Dr. Rowlands.

1 DR. CRAIG ROWLANDS: I'm here. Good
2 morning.

3 DR. KENNETH PORTIER: Good morning.
4 Dr. Schlenk.

5 DR. DANIEL SCHLENK: Here.

6 DR. KENNETH PORTIER: And Dr.
7 Sheela, sorry.

8 DR. SHEELA SATHYANARAYANA: Dr.
9 Sheela's great. I'm here.

10 DR. KENNETH PORTIER: Dr. Barton? I
11 see you logged in, and you're muted in Webex. He
12 may have stepped away. I'll come back. Dr.
13 Crump.

14 DR. KENNY CRUMP: I'm here.

15 DR. KENNETH PORTIER: Dr. Everitt.

16 DR. JEFFREY EVERITT: Here.

17 DR. KENNETH PORTIER: Dr. Herrick?

18 DR. ROBERT HERRICK: Here.

19 DR. KENNETH PORTIER: Dr. Jayjock?

20 DR. MICHAEL JAYJOCK: Here.

21 DR. KENNETH PORTIER: Dr. Kanarek?

22 DR. MARTY KANAREK: Here.

1 DR. KENNETH PORTIER: Dr. Markowitz?

2 DR. STEVEN MARKOWITZ: Here.

3 DR. KENNETH PORTIER: Dr. Sheppard?

4 DR. ELIZABETH SHEPPARD: Here.

5 DR. KENNETH PORTIER: Dr. Shukla?

6 DR. ARTI SHUKLA: I'm here.

7 DR. KENNETH PORTIER: Dr. Taioli?

8 DR. EMANUELA TAIOLI: I'm here.

9 DR. KENNETH PORTIER: Dr. Van Gosen?

10 MR. BRADLEY VAN GOSEN: Good

11 morning.

12 DR. KENNETH PORTIER: Good morning.

13 And Dr. Barton, again.

14 DR. CHARLES BARTON: I'm here. Can
15 you hear me now?

16

17 FOLLOW-UP ON PREVIOUS DAY DISCUSSIONS

18

19 DR. KENNETH PORTIER: Yep, got you.

20 So everyone is -- everyone's present this morning.

21 That's great. Yesterday we had a good discussion.

22 And at the session right before lunch we had a

23 discussion of the OPPT technical presentation.

1 And there were a number of questions that were
2 left unanswered. And the EPA staff said that they
3 would go and look for the answers. So I thought
4 we'd spend maybe 25 minutes, at least until 10:30
5 Eastern, kind of discussing some of these if EPA
6 has had an opportunity to look at these questions
7 and come up with some answers.

8 We realize that many of these
9 questions relate to issues around the regulatory
10 nexus, and this is something that the panel has
11 mentioned in almost every one of previous risk
12 evaluations that we've reviewed. But we continue
13 to try to understand how EPA has focused these
14 TSCA risk evaluations in light of all the other
15 environmental regulations that EPA -- other
16 federal agencies and international agencies work
17 with. So at this point I'll ask Dr. Scarano, the
18 EPA lead for this evaluation, if he wants to
19 comment or point to someone else on his team to
20 address some of these questions. Dr. Scarano?

21 **DR. LOUIS SCARANO:** Thank you, Dr.
22 Portier. Can everyone here me okay?

1 DR. KENNETH PORTIER: Yes.

2 DR. LOUIS SCARANO: Okay. Thanks.

3 I'm not sure if you wanted to put the questions
4 up? Or I'll be happy to read them as they were
5 sent to us, and I will ask the appropriate party
6 to respond. Is that okay?

7 DR. KENNETH PORTIER: Yeah. Why
8 don't you just read it because I don't think we've
9 transmitted anything to the session host to
10 display. So just go ahead and read the question.

11 DR. LOUIS SCARANO: Okay. Thank
12 you. So there were 12 questions. So this is the
13 first question. And I will ask our Kevin
14 Vuilleumier to answer it. The question -- and
15 it's multiple parts -- can the EPA request or
16 mandate the collection of exposure data -- for
17 example, require monitoring -- when risk scoping
18 and problem formulation indicate risk for
19 potential exposures under a specific condition of
20 use? And a related question, can OPPT require
21 manufacturers and importers/processors to collect
22 and provide monitoring data through the NPDES --

1 the NPDES permit process or any other means for
2 conditions of use scoped to have potential hazard,
3 and, if so, how long would this take to get these
4 data? Kevin --

5 **MR. KEVIN VUILLEUMIER:** This is
6 Kevin Vuilleumier. Can everyone hear me okay?

7 **DR. LOUIS SCARANO:** Thank you,
8 Kevin. Go ahead.

9 **MR. KEVIN VUILLEUMIER:** Okay. Yeah.
10 In response to those questions, we have -- EPA
11 does have authority under TSCA to request certain
12 testing or monitoring. That's under TSCA Section
13 4. We can also request data, which is obtained,
14 as an example, through, say, NPDES permits, and
15 that would be through our information collection
16 rule. And that's Section 8 in TSCA.

17 One thing we couldn't really do
18 through TSCA is require specific monitoring be
19 incorporated into NPDES permits, for example,
20 permit terms and conditions. That's done through
21 the NPDES permitting program which is often
22 delegated down to the states by our Office of

1 Water. Along those similar lines regarding the
2 time it would take to gather data, the fact that
3 the NPDES permit monitoring data is often
4 submitted under the permit to the state -- and
5 certain data may get submitted to U.S. EPA.
6 Otherwise it's maintained at the state level --
7 the information like that would be very disparate
8 and difficult to capture. Some states may have an
9 electronic data collection system. Others may
10 not. It may be in reports.

11 So regarding the timing of how long
12 it would take to collect that data from every
13 state -- and it even goes further to every local
14 agencies sometimes who maintain the data -- would
15 be considerably long timeframe. I hope that
16 answers the questions in generally.

17 **DR. KENNETH PORTIER:** Thank you.

18 Does anyone -- Dr. Anderson, do you want to follow
19 up?

20 **DR. HENRY ANDERSON:** Yeah. A follow
21 up would be it's one thing to -- it could take
22 quite a bit of time to do additional sampling, but

1 can you require them to send in any monitoring
2 data they have? It seems that you request them to
3 voluntarily send in data, and in this case a
4 number did. But it's unclear whether you actually
5 can require them to submit the data that they
6 have, which would potentially carry some kind of a
7 penalty if they did not do that. So do you get
8 information back saying "We don't have any
9 monitoring data," or do you just not get a
10 response?

11 **MR. KEVIN VUILLEUMIER:** I think that
12 defers to is anything wrong that you try to move
13 into there? With regards to requesting the data,
14 we request voluntarily. We'll also seek the data
15 from our companion offices, like the Office of
16 Water, when it does get submitted. We also can
17 reach out to states and local agencies to see if
18 they can submit that data to us. Sometimes they
19 do. Other times they would not because they may
20 not be able to collect it or capture it in a
21 timely manner.

1 With regards to going to the actual
2 facilities, that is where the information
3 collection rule in TSCA Section 8 comes into play.
4 So I don't know if you're referring to the seeking
5 the information from the facility or from the
6 states who might get that data reported to them
7 under the NPDES permitting program, as an example.
8 Could you clarify who we're looking at getting the
9 information from?

10 **DR. HENRY ANDERSON:** Well, I was
11 specifically looking at the conditions of use
12 companies that are using the data, if you can
13 identify them. Then a question is have they
14 submitted all the data they have, or could it be
15 just a selected portion of it?

16 **MR. KEVIN VUILLEUMIER:** When we ask
17 for the data voluntarily, they would submit what
18 they submit. So it may not be all of the data.
19 When we put together -- and if we went down this
20 path to get an information collection rulemaking
21 under Section 8(a), that's actually a regulatory

1 rule-making process, which can take quite a bit of
2 time.

3 When you develop something like
4 that, however, we include very specific
5 information that we are looking for, and we would
6 require that be submitted. Then under the rule,
7 they would have to submit that data. But again,
8 the information collection rule to develop a
9 rulemaking asking for that information can take
10 many, many months or even years, depending on how
11 detailed it has to get.

12 **DR. HENRY ANDERSON:** Okay. Thank
13 you.

14 **DR. KENNETH PORTIER:** Dr. Schlenk,
15 you were the one who asked this question. Does
16 this answer your question?

17 **DR. DANIEL SCHLENK:** Yeah. It
18 answers it. It's a little bit sad. But there's
19 no -- I guess to follow up there's no way that you
20 can make a request to Office of Water to mandate
21 three -- I think there was maybe ten industries
22 that were mentioned yesterday that have chlor-

1 alkali processing. There's no way to go into
2 those ten industries and say, "Hey, can you get us
3 some wastewater discharge data?" That's basically
4 the simplistic way of asking that. So that
5 question alone would take many months or years is
6 what you're saying?

7 **MR. KEVIN VUILLEUMIER:** Well, I
8 wouldn't say it would go through our Office of
9 Water after they submit it voluntarily. And if
10 it's required of NPDES to maintain these records,
11 then you'd have it and they may be able to submit
12 it to the rule. But that would be also sought
13 through the Office of Water. They also have
14 independent information request authorities, where
15 they can collect information that's not otherwise
16 immediately available to them.

17 So there's a few different processes
18 we can go about getting that information. Most of
19 the statutes have their own information collection
20 authorities, where we can request specific
21 information. And then that requires -- as you

1 guys are alluding to, that requires they submit
2 the information that we asked for.

3 **DR. DANIEL SCHLENK:** So I guess the
4 bottom line is if we make a recommendation to
5 collect more data, is that even a valid
6 recommendation? I mean, can that be followed up
7 on, or are we just kind of wasting our breath? I
8 guess that's what I'm after.

9 **MR. KEVIN VUILLEUMIER:** No. That's
10 absolutely a good recommendation to do. And if
11 you have some insight into particular information
12 that those that are out in the field may know are
13 out there, then you can certainly request that.
14 And we will and actually are coordinating with our
15 other offices to try to gather additional
16 information where we are able. But that's
17 certainly a valid recommendation, and there are
18 various authorities we can use under TSCA and
19 other offices to get such information.

20 **DR. KENNETH PORTIER:** Thank you,
21 Dan. I think we got it.

22 **DR. STANLEY BARONE:** Dr. Portier.

1 **DR. STANLEY BARONE:** Dr. Portier,
2 this is Stan Barone. Just for clarification on
3 that last point that Kevin made, if the Committee
4 has specific questions that they think -- or
5 they're recommending that we gather additional
6 information for the context for those questions in
7 your report would be very, very helpful.

8 **DR. KENNETH PORTIER:** Good. Let's
9 go ahead and take Question 2, Dr. Scarano.

10 **DR. LOUIS SCARANO:** Thank you.
11 Question 2. Does TSCA require the conclusions of
12 a risk assessment be reported as binary? That is
13 the risk conclusion is stated as either yes or
14 no. Can the risk evaluation end in a conclusion
15 that there is not enough information? For
16 example, there is no exposure information --
17 "There is no exposure data, hazard is unknown" to
18 characterize risk, for example, "lacks sufficient
19 information to make a reasoned evaluation" for
20 other than new chemicals. And we don't have
21 anyone to answer this one specifically.

1 This is a big policy question that
2 has -- we do have a lot of internal discussions
3 upon. So we're not prepared to answer that one
4 directly right now.

5 **DR. KENNETH PORTIER:** That's a big
6 one that's come up a number of times, and we keep
7 asking. So just -- it's good to know if you can
8 find someone who could answer that question. That
9 last part, where it says "lack sufficient
10 information to make a reasoned evaluation," the
11 reason I put "for other than new chemicals" is
12 because in the TSCA law there seems to be an
13 ability there for new chemical assessments to
14 conclude lack sufficient information to make a
15 reasoned evaluation. And then EPA pushes it back
16 on the manufacturer, processor, importer to
17 provide that additional information needed to make
18 a reasoned evaluation.

19 But I didn't see anything that
20 allowed you to do that for legacy chemicals, which
21 is what we've been discussing for the last year.
22 So it would just be nice if we could get an

1 assessment of that. Why don't we go on to
2 Question 3, then? We've got a little time.

3 **DR. LOUIS SCARANO:** That's fine.

4 Sure. Although if I may just address the new
5 chemical issue, we have opportunity there because
6 it's coming in the door and going into commerce.
7 So it's much better from a timing standpoint in
8 trying to understand a chemical where we can ask
9 for information. But on to Question 3. So Slide
10 number 30 indicates "low to medium uncertainty"
11 regarding minimal to low surface water discharge.

12 However, on the bottom of page 52
13 and other locations, the draft risk evaluation
14 states, "Asbestos releases from chlor-alkali
15 facility treatment systems to surface water and
16 publicly-owned treatment works are not known.
17 While the treatment technologies employed would be
18 expected to capture asbestos solids, the precise
19 treatment efficiency is not known. Chlor-alkali
20 facilities are not required to monitor effluents
21 for asbestos releases, and EPA's broader research
22 into this condition of use did not find asbestos

1 water release data." These statements indicate
2 significant uncertainty generated by the lack of
3 measured data -- total lack of measured data. Is
4 there a unique definition of "uncertainty" by the
5 Agency that ignores lack of data for a conclusion?

6 So here we'd simply like to say that
7 we acknowledge that Slide number 30 said something
8 different from what is in the current draft on
9 page 52 and elsewhere in the draft risk
10 evaluation, and we will make them consistent. And
11 we understand the need to be more clear about what
12 we consider in determining uncertainty for the
13 TSCA risk evaluation. Uncertainty is defined in
14 many different parts of EPA and elsewhere.

15 We are going to reference the risk
16 characterization handbook, and we have it. And
17 that's in the rule -- the TSCA rule for risk
18 evaluation. And so we can be more explicit and
19 will be more explicit and be consistent in the
20 draft risk evaluation. I hope that answers the
21 question.

1 **DR. KENNETH PORTIER:** Dr. Schlenk,
2 did you want to follow up?

3 **DR. DANIEL SCHLENK:** No. That's
4 what I figured the answer would be. That's fine.

5 **DR. KENNETH PORTIER:** Well, we have
6 a little more time. Let's go on to Question 4.
7 Maybe we can get half of them done this morning.
8 That'd be great.

9 **DR. LOUIS SCARANO:** Thank you.
10 Okay. Number 4. EPA should consider the gig
11 economy when developing the consumer examples of
12 brake examples where EPA assumed only one brake
13 job every three years and one job in a lifetime.
14 Did EPA consider the gig economy? The suggestion
15 is that we add at least one example to cover that
16 use. Or EPA could indicate the minimum number of
17 jobs in a lifetime or five-year period that would
18 result in reaching the "unreasonable risk" level.
19 And if I may, I'd like to ask Kevin to respond to
20 this. Kevin?

21 **MR. KEVIN VUILLEUMIER:** Thank you,
22 Gino. This is Kevin Vuilleumier again. And we

1 appreciate the question, and we will look into the
2 gig economy in more detail. We did include in our
3 sensitivity analysis in the risk evaluation
4 Appendix L, where we estimated the risk to a
5 single brake job in a lifetime, and it showed risk
6 above the benchmark.

7 We also briefly touched on the point
8 that was brought up yesterday about an individual
9 having maybe more than one car, But we will in the
10 uncertainties section of consumer -- and that was
11 Section 2.3.2.1. But we can expand the
12 uncertainties to bring in the gig economy point
13 that you brought up. We appreciate you bringing
14 up that point.

15 **DR. HENRY ANDERSON:** Great. Thank
16 you.

17 **DR. KENNETH PORTIER:** Dr. Anderson,
18 that was your question. Okay. Number 5. Dr.
19 Scarano, you might be muted if you're talking.
20 Yes.

1 **DR. LOUIS SCARANO:** Oh. I just read
2 the whole question. You mean no one heard it?
3 All right.

4 **DR. KENNETH PORTIER:** No one heard
5 it. Start over.

6 **DR. LOUIS SCARANO:** Nobody? That's
7 fine. That's fine. Thank you and I apologize.
8 Question 5, when there are multiple companies that
9 provide monitoring data, EPA appears to combine
10 all the data and then calculates "central
11 tendency" and "high-end" summary statistics. Does
12 EPA first look to see if the individual company
13 data have similar central tendencies and, if
14 dissimilar, consider weighting the data from each
15 facility so that one company does not dominate and
16 skew the summary statistic for the industry?

17 I'll ask Jay Jon to respond to this.
18 He has been having connection problems. If he's
19 not available, I'll be happy to. Jay, are you
20 available? I'll assume he is having problems.

21 **DR. KENNETH PORTIER:** He is still
22 muted under Webex. He needs to unmute under

1 Webex. Let's try that first. I see him still
2 muted there. And maybe Martin can check to see if
3 he is -- oh, now he's unmuted.

4 **MR. JAY JON:** Can you hear me?
5 Hello.

6 **DR. LOUIS SCARANO:** Yes. Thank you,
7 Jay.

8 **DR. KENNETH PORTIER:** Yes. We can
9 hear you. Thank you.

10 **MR. JAY JON:** Okay. This is Jay Jon
11 and thank you for the comment. And the short
12 answer to your question is yes. EPA plans on
13 doing this by industrial sector, and most of the
14 industry information we received was from the
15 chlor-alkali sector. But the principle will be
16 applied and used in others where appropriate.

17 Previously, this was not possible
18 because some of the data that we received lacked
19 the facility identification, and they are all from
20 ACC. But as mentioned at the presentation
21 yesterday, EPA recently learned that all the data
22 from ACC are duplicative. So we can now remove

1 them and proceed with our recalculation. That's
2 it.

3 **DR. KENNETH PORTIER:** Thank you.

4 Does anyone on the Committee want to follow up
5 with that? That's pretty clear. So what I heard
6 is that you haven't done it in the past, but you
7 will do it and attempt to do it for this one.

8 **MR. JAY JON:** That is correct.

9 **DR. KENNETH PORTIER:** Got it.

10 Question 6.

11 **DR. LOUIS SCARANO:** Thank you.

12 Question 6. And this is going to be Jay again
13 soon too. Does EPA request the monitoring plan
14 strategies from the data submitters? Jay?

15 **MR. JAY JON:** Yeah. This is Jay
16 again, and the short answer is yes. We do. We
17 ask. And if it's not submitted, we ask for it.
18 As part of the data collection, we ask information
19 about their process and specifics and as well as
20 any monitoring data. And as part of that, we ask
21 for the background information. And that's my
22 response.

1 DR. KENNETH PORTIER: Dr. Anderson.

2 DR. HENRY ANDERSON: Yeah. Thank
3 you. That's helpful. I think it would be useful
4 if that could be -- unless it's considered
5 proprietary -- to summarize or put that into the
6 risk assessment or into the document. One of the
7 issues is if you have what the plan is, then you
8 can look at the data you get coming in to see if
9 it appears to be consistent with the overall data
10 collection plan so data hasn't been lost somewhere
11 in the transmission process. Some years -- I mean
12 there -- some data will be in some years and some
13 in not years. And it would be good to know was
14 that -- why is there missing data in a data set.

15 DR. KENNETH PORTIER: Thank you, Dr.
16 Anderson. Dr. Kissel?

17 DR. JOHN KISSEL: Yeah. The
18 question or the response was that, yes, we do ask
19 for the metadata, but I didn't hear anyone say
20 whether it was actually provided on a routine
21 basis. So you can ask for it, but, if you only
22 get it a third of the time, that would still leave

1 you at a disadvantage with respect to Dr.
2 Anderson's concerns.

3 **DR. KENNETH PORTIER:** Jay?

4 **MR. JAY JON:** And yes. This is Jay
5 again. Yeah. That's absolutely right. And a lot
6 of times these monitorings were done as a routine
7 monitoring, and sometimes they don't have it. And
8 that type of information is easy to obtain if we
9 ask for a new set of data -- have them develop
10 plans for monitoring. But for the legacy
11 monitored data, a lot of times they just don't
12 have them.

13 **DR. KENNETH PORTIER:** So it sounds
14 like, "Well, we just go out and collect the data.
15 We've always collected the data. We don't have a
16 plan. We just do it." I think I'd like to take
17 on Question 8, which has to do with p-chem
18 properties. And then we'll leave 7, 9, 10, 11,
19 and 12, which maybe deal more with human health,
20 to talk about tomorrow morning if that's okay?
21 Dr. Scarano?

1 **DR. LOUIS SCARANO:** That's fine.

2 Okay. So you'd like to go right to 8?

3 **DR. KENNETH PORTIER:** Yes.

4 **DR. LOUIS SCARANO:** All right. Then
5 question number 8, in Table 1-1, which is Slide 12
6 from our presentation yesterday, EPA describes
7 chrysotile physical-chemical properties. Can the
8 stated fiber lengths in Table 1-1 -- less than
9 1,000 to about 10,000 micrometers -- be easily
10 reconciled with the long fiber/short fiber
11 arguments in the human health literature that
12 emphasize lengths of less than -- less than versus
13 greater than five micrometers?

14 EPA answered that the lengths in
15 Table 1-1 pertained to agglomerates in commercial
16 products. This produced a follow-up question as
17 to whether the distribution of particle sizes in
18 commercial chrysotile is known. And I'd like to
19 ask Abhilash to answer this if he's there.

20 **MR. ABHILASH SASIDHARAN:** Thank you,
21 Gino. This is Abhilash. To the best of our
22 knowledge, the size distribution that there is not

1 available for air borne chrysotile. And I also
2 heard a question like if anyone on the panel has
3 information on this, please provide with
4 citations. Thank you.

5 **DR. KENNETH PORTIER:** So what about
6 the first part of the question having to do with
7 long fiber/short fiber and the health literature?
8 There was a lot of discussion on this yesterday in
9 the public comments and trying to -- I think a lot
10 of us were trying to figure how to take those
11 comments.

12 **DR. LOUIS SCARANO:** This is Gino.
13 And I think -- and I understand that. And I think
14 that'll be a part of our discussion as we get into
15 some of the charge questions as we talk about
16 fibers and toxicity. Let me just say that. I
17 mean, clearly that's an important thing, and we
18 thought long and hard about it. And to answer
19 specifically, there's no -- to the best of our
20 knowledge -- no size distribution for the raw
21 chrysotile. And there's a lot of information on
22 size, fiber, and length, which is why we did spend

1 time on it later on in the hazard characterization
2 -- the dose response part.

3 So we understand that this might not
4 satisfy you for now, but I think it addresses the
5 one part of the question. And the whole fiber
6 length and toxicity will come up over the course
7 of the deliberations if that is okay.

8 **DR. KENNETH PORTIER:** It's fine.
9 We'll reserve it. I know it will come back up
10 again. We thought we'd get the easy answer up
11 front. Obviously, we're not. I think with that,
12 I'd like to stop this. We'll come back to some
13 questions tomorrow morning if you don't mind.

14 But right now I think I'd like to go
15 ahead and move forward with the Committee starting
16 to answer some of the charge questions. So if we
17 can have the first charge question put up? And,
18 Dr. Scarano, you're going to read the questions?
19
20
21

CHARGE QUESTION 1 (1.1)

DR. LOUIS SCARANO: Thank you, yes.

So this is Question 1, and it has to do with environmental exposure and releases. Based on the reasonably available information in the published literature, provided by industries using asbestos and reported in EPA databases, there are minimal or no releases of asbestos associated with the conditions of use that EPA is evaluating in this draft risk evaluation. So there are two parts to this charge question. Question 1 -- would you like me to read both, Dr. Portier, or one at a time?

DR. KENNETH PORTIER: Go ahead and read both.

DR. LOUIS SCARANO: Okay. Thank you. Question 1.1, please comment on whether the information presented supports the analysis and conclusion in the draft environmental exposure section, which are in Section 2.2 and Appendix D. Question 1.2, please comment on whether EPA

1 adequately, clearly, and appropriately presented
2 the physical-chemical properties/characteristics
3 of chrysotile asbestos.

4 **DR. KENNETH PORTIER:** Thank you.

5 And I think, Dr. Schlenk, you have the lead on
6 these two questions. Do you want to lead off?
7 Dan, you're muted in Webex.

8 **DR. DANIEL SCHLENK:** How's that?

9 Can you hear me?

10 **DR. KENNETH PORTIER:** We got you

11 now. Yes, I can hear you.

12 **DR. DANIEL SCHLENK:** Okay. Strange.

13 Okay. Just wanted to say that, yeah, most of the
14 associates provided information. So I've tried to
15 incorporate that into sort of a general response
16 that's present now.

17 Overall, the Agency concludes that
18 there is little, if any, discharge of chrysotile
19 fibers to surface water in facilities associated
20 with COUs. The Agency bases its conclusion on
21 several evaluations. The evaluation of TRI Data
22 estimated that the majority of asbestos is

1 disposed in landfill and is under different
2 regulatory oversight because of this. Evaluations
3 of TRI Data indicated zero discharge into POTW and
4 non-POTW facilities from COUs targeted in the DRE.

5 It is unclear how minimal to no
6 releases are consistent with statements from the
7 problem formulation that the chlor-alkali industry
8 in years leading up to the problem formulation --
9 this was before 2017 -- stated it does discharge
10 asbestos to wastewater. Following visits to these
11 COUs in late 2017, the DRE stated that follow-up
12 evaluations were conducted. But since the COU was
13 not required to monitor asbestos, through NPDES,
14 it claims in 2018 water discharges were zero. It
15 is unclear how a conclusion of zero discharge
16 without measurement can be made with this COU. It
17 is also unclear why the Agency did not ask the
18 facility to provide data after its initial scoping
19 exercise in 2016.

20 Several Committee members pointed
21 out that EPA describes the filter press treatment
22 used in the chlor-alkali industry prior to being

1 discharged to wastewater treatment plants, but it
2 also adds that the efficiency of separation is not
3 known. One Committee member indicated that filter
4 presses normally operate with an efficiency
5 greater than 90 percent. But any residual
6 asbestos in the effluent will depend on the
7 initial concentration, pH of the mixture, and
8 other factors.

9 The Agency concluded that the
10 efficiency of removal or filtration was unknown,
11 nor the concentrations in wastewater after this
12 treatment. This appears to be contrary to the
13 conclusion of minimal to low uncertainty from this
14 COU. Regarding NPDES data, the Agency also
15 evaluated DMR databases, which include NPDES
16 monitoring data. In addition, a more thorough
17 assessment of NPDES monitoring was conducted.

18 After these evaluations, only a
19 surface water sample from one historical mining
20 site reported concentration of chrysotile fibers.
21 Since mining operations are no longer present in
22 the U.S. since the activities are covered under

1 other regulatory processes, the Agency concluded
2 chrysotile fibers resulting from this activity do
3 not warrant a risk quotient evaluation for
4 environmental receptors. The available
5 information in the problem formulation indicated
6 that there were surface water releases of
7 asbestos. However, not all releases were subject
8 to reporting, i.e., effluent guidelines, or were
9 applicable, i.e., friability.

10 EPA does provide data in Table 2.1,
11 "Six Year Review Cycle Data for Asbestos in
12 Drinking Water," that shows systems which report
13 asbestos levels in drinking water. EPA mentions
14 that this most likely represents the measures of
15 finished, i.e., tap water, which leads several
16 Committee members to suspect that surface water
17 concentrations may indeed be higher. One
18 Committee member wondered why these data were even
19 considered as surface water targets for sampling.

20 Although it is difficult to
21 attribute asbestos to specific COUs, EPA could
22 consider doing a system-wide assessment of

1 asbestos in surface water or drinking water as a
2 total. This would not allow to differentiate the
3 contributions from each COU, but it could provide
4 an overall assessment of the environmental risk
5 for asbestos in waterways. This may also account
6 for air releases that may be transported into
7 waterways. If that occurs, see Appendix D, Table
8 APX D-2. By the way, the Table 2.1 could use a
9 footnote describing abbreviations for the benefit
10 of readers not familiar with the environmental
11 quality shorthand.

12 Based in part on these and other
13 evaluations, i.e., the WQX, the Agency conclude a
14 minimal to no discharge from the COUs. However,
15 there were some concerns noted from the Committee.
16 Since chrysotile asbestos is a natural component
17 of many freshwater streams where serpentine is
18 found, one Committee member thought that would
19 likely require large concentrations to elicit
20 adverse effects that would result in unreasonable
21 risk. While the conclusions are reasonable to

1 this member, precise information and logic to
2 support them is lacking.

3 Overall, the Agency based their
4 conclusion minimal to no exposure because they
5 could not find relevant data; thus, a stronger
6 statement of uncertainty is necessary in the
7 executive summary. Discrepancies between the
8 problem formulation and the DRE need to be
9 highlighted in any further documentation. In the
10 problem formulation, it was stated repetitively
11 that surface water concentrations were within the
12 same order of magnitude as concentrations that
13 cause adverse effects to aquatic biota. However,
14 that conclusion was abandoned in the DRE because
15 monitoring data could not be obtained.

16 Given the relatively high
17 concentration of asbestos found in biosolids, 10
18 percent of dry ash weight, and surface waters in
19 general -- this comes from the ATSDR document --
20 to state minimal to no discharge of asbestos
21 occurs from targeted COUs based on no measured
22 data is highly uncertain. It may be the

1 concentrations of chrysotile fibers may be derived
2 from sources other than COUs, but only monitoring
3 data will provide the evidence needed to conduct a
4 robust risk evaluation and assessment.

5 Contrasting opinions were provided from another
6 Committee member who thought the Agency did a
7 decent job of looking for potential releases to
8 water.

9 The Committee also had some concerns
10 with Section 2.2.2.5. They wondered why it was
11 assumed that water isn't used in the cleanup of
12 aftermarket automotive parts or by consumers
13 conducting brake replacements in automobiles at
14 residential locations. At a minimum, consumer
15 clothing used in the process would be laundered,
16 and people may shower. The document does not
17 appear to address take-home exposure associated
18 with the transport of asbestos-contaminated
19 clothing and other items from the workplace to
20 places of residence. This has been a well-
21 documented source of asbestos with a number of

1 articles in the literature. There's a couple of
2 references here.

3 One Committee member raised this
4 here because several articles quantify the
5 airborne asbestos fiber levels associated with
6 handling contaminated clothing in the home. The
7 handling can include laundering the clothing at
8 home, which could release asbestos to domestic
9 wastewater. Although, no studies have addressed
10 this. Similarly, brake dust or other materials
11 derived from washing down areas where chrysotile
12 asbestos products were used would also likely be
13 transported to wastewater storm water. Even
14 though it may be a small overall contribution,
15 several Committee members suggest this be
16 addressed as a potential source of asbestos
17 released to surface water.

18 Several Committee members did not
19 agree with the logic the Agency used to exclude
20 terrestrial pathways. EPA states that "other
21 Agency regulations adequately assess and
22 effectively manage exposures from asbestos

1 releases to terrestrial waterways including
2 biosolids for terrestrial organisms." That's a
3 quotation by the way. But the same conclusion
4 seems to occur with drinking water exposure
5 pathways. The EPA writes that the drinking water
6 pathway, quote, is currently addressed in the Safe
7 Drinking Water Act's regulatory, analytical
8 process for public water systems, end quote.

9 Several Committee members disagree
10 with EPA's decision to exclude environmental
11 pathways based strictly on statutory
12 considerations and recommend that EPA consider the
13 inclusion of terrestrial and drinking water
14 pathways. Also regarding aquatic receptors, one
15 Committee member indicated that a cursory review
16 of the ECOTOX database found 226 results for
17 chrysotile asbestos, and only a few studies were
18 selected. As in previous DREs, the selection of
19 single studies to represent toxicity for classes
20 and phyla in organisms is not well supported.
21 This member recommended that EPA use a species
22 sensitivity approach in calculating an LC5 or EC5

1 for acute and chronic, respectively. If the data
2 are not sufficiently robust for this approach, it
3 is suggested the EPA show the variation in the
4 effects using a scatter diagram to support the use
5 of key studies for COC derivation. And there's
6 some references that have been provided here, as
7 in earlier DREs for other compounds.

8 In summary, EPA did not evaluate the
9 risk to aquatic species from exposures to surface
10 water. It was unclear how the Agency could come
11 to this conclusion of no risk without measured or
12 predicted concentrations that could be compared to
13 hazard values. Our recommendation here is the
14 Agency should require monitoring data either from
15 NPDES mandates or specific requests to COUs where
16 asbestos can be released in the wastewater.

17 A couple of specific comments --
18 since asbestos is a natural mineral, the DRE
19 should include a map showing the locations of
20 naturally occurring asbestos deposits such as
21 provided by the ATSDR document. To better
22 understand the potential impact of these natural

1 deposits, it would be appropriate to determine if
2 there's correlation between the location of
3 asbestos deposits and prevalence in drinking water
4 and/or air measurement detects. Is asbestos found
5 more often in higher concentrations in areas
6 located near natural deposits, for example?

7 And then there's a question. What
8 regulations deal with fugitive emissions
9 associated with disturbing naturally occurring
10 asbestos deposits through activities like
11 construction? Are impacts of non-asbestos mining
12 operations addressed when asbestos is released
13 along with other generated waste? And that's all
14 I got.

15 **DR. KENNETH PORTIER:** Sounds like a
16 lot, and I could certainly hear the voice of many
17 of the associates in that response. But I'll kind
18 of go through a list and call on the associates to
19 see if they want to add anything. Dr. Blystone?

20 **DR. SHERI BLYSTONE:** No. I think
21 Dan did a great job of capturing everything. I am
22 in the camp of tending to agree with EPA's

1 conclusions of minimal releases to water from the
2 conditions of use that they're examining. But
3 there's some gaps in there that I think could
4 bolster their argument.

5 **DR. KENNETH PORTIER:** Dr. Jiménez-
6 Gonzalez?

7 **DR. CONCEPCIÓN JIMÉNEZ-GONZALEZ:**
8 Nothing that I could add. He did a really good
9 job summarizing the input, and we also got to see
10 the summary beforehand to provide additional
11 comments. So I'm happy with it.

12 **DR. KENNETH PORTIER:** Thank you.
13 Dr. Johnson?

14 **DR. MARK JOHNSON:** Yeah. I also
15 agree Dr. Schlenk did a great job. I just
16 expected to see a table with derived COCs compared
17 to exposure estimates. But they talk that we just
18 don't have those kind of data or the
19 concentrations we do have are so below where the
20 toxicity occurs that we're not going to do that.

21 But given the mechanism that's cited
22 from asbestos -- this frustrated phagocytosis of

1 macrophages -- that seems like a phylogenetically
2 conserved system or pathway. I wonder about
3 longer-lived aquatic species. Some Ambystoma
4 salamanders can live 30 years, at least in
5 captivity. I'm not sure about what their lifespan
6 is in the wild.

7 So I wonder about longer lived
8 aquatic species and what the effects could be in
9 them? We really don't have data on those
10 organism, and so I think that's an uncertainty
11 that needs to be discussed somewhere in the
12 document. And that's all I have.

13 **DR. KENNETH PORTIER:** Thank you.

14 Dr. Taioli?

15 **DR. EMANUELA TAIOLI:** Sorry. I was
16 muted. No, I have nothing to add. Thanks.

17 **DR. KENNETH PORTIER:** Thank you.

18 We'll now open it up to the panel. Dr. Kissel?
19 John, you're muted in Webex.

20 **DR. JOHN KISSEL:** Nothing more to
21 add.

1 **DR. KENNETH PORTIER:** Okay. I just
2 saw your hand up. That's all.

3 **DR. JOHN KISSEL:** Oh. That's left
4 over.

5 **DR. KENNETH PORTIER:** A legacy hand
6 up. Okay. Anyone else want to comment? Dr.
7 Doucette.

8 **DR. WILLIAM DOUCETTE:** Yeah. I
9 think Dan did a great job of summarizing the
10 comments, and my comments were included. I just
11 had a thought mainly because the previous reviews
12 that I was involved with, most of them were
13 volatile organics, and I was very familiar with
14 the analytical techniques and had actually
15 performed a lot of them myself. But in this
16 particular case, I was not as familiar with the
17 analytical techniques, which go into pretty much
18 everything here.

19 And I was going to suggest that
20 maybe EPA -- and maybe this is relevant for all of
21 the DREs -- have a short section on analytical
22 methods to help kind of put things in perspective.

1 And I think that would certainly help me as a
2 reader of this. Even though I'm a chemist, I'm
3 not familiar with the analytical techniques used
4 to distinguish between the different particle
5 sizes and things like that in this particular
6 case, and that's crucial in evaluating the health
7 risks. So it's just a suggestion that maybe a
8 short section on analytical methodology be
9 included in the DREs. That's all I have. Thank
10 you. Dan did a great job.

11 **DR. KENNETH PORTIER:** Anyone else?

12 Dr. Kanarek?

13 **DR. MARTY KANAREK:** I don't know if
14 this is the time to bring it up but might as well
15 start. It was a great report, but Dr. Schlenk
16 went back and forth saying asbestos and then
17 occasionally chrysotile asbestos. And to a reader
18 that doesn't understand the nexus -- the why we're
19 not considering all types of asbestos -- that
20 would be very confusing. I don't know if this is
21 the time to bring it up or we're going to have to
22 talk about this eventually. But this is not a

1 risk assessment for asbestos. It's a risk
2 assessment for chrysotile asbestos.

3 **DR. KENNETH PORTIER:** Yeah. Dr.
4 Schlenk, I kind of had the same thought as I was
5 listening and wondering if you're going to be able
6 to kind of focus down on when the comments are
7 kind of specific to chrysotile and when the
8 comments are more generic. Is that what you were
9 trying to get at, Dr. Kanarek?

10 **DR. MARTY KANAREK:** Exactly.

11 **DR. DANIEL SCHLENK:** Well, I will
12 hopefully get some input from people that know the
13 literature of asbestos better than I do. So I
14 know at least my comments dealt with chrysotile.
15 But with the other associates perhaps in the
16 overview of the document, someone that's more
17 familiar with general aspects of asbestos and when
18 we should use chrysotile would help in the editing
19 of those minutes.

20 **DR. KENNETH PORTIER:** Thank you.
21 Dr. Anderson?

1 **DR. HENRY ANDERSON:** Yeah. I think
2 it would -- kind of back to the first thing of
3 having laboratory methodologies, the question
4 would be how would you look at water and these
5 other things? My understanding is they look for
6 fibers, and they are not differentiating between
7 chrysotile or all the other fibers that could be
8 there. So what would be helpful is, is there
9 actual attempts to identify what type of fiber it
10 is in discharge like this? So it would be helpful
11 to know the -- I mean, I think I would tend to
12 agree that there's a minimal releases that seem to
13 be, but there's not a lot of data on it. And I
14 suspect there's no data that will specifically say
15 chrysotile in the lab report.

16 So we're kind of generic asbestos
17 especially for minerals are part of that
18 measurement technique but are not differentiated
19 by fiber type, which kind of gets back to the
20 issue of can we separate out effects either in the
21 environment or in house without more extensive
22 fiber type identification data?

1 **DR. KENNETH PORTIER:** Thank you.

2 Dr. Doucette, your hand's still up. Dr. Van
3 Gosen?

4 **MR. BRADLEY VAN GOSEN:** Yes. I'll

5 add that if you're looking specifically at
6 chrysotile and water and water supply, just as an
7 example, I can provide references where on Staten
8 Island the drinking water reservoir has been
9 studied for its chrysotile content, which was
10 rather high. So it's not something that's been
11 completely ignored, but, getting down to risk
12 factors, it's not been studied very well from my
13 experience. And you can distinguish between the
14 types of fibers. Any good lab will.

15 And there is one basic question that
16 we'll go back to over and over is do you want to
17 focus? Because you've decided chrysotile is the
18 only asbestos type of concern right now in this
19 assessment, which I disagree, then you should
20 focus the discussion on chrysotile because there's
21 a humongous difference between the toxicology,
22 epidemiology of chrysotile versus most of the

1 amphibole asbestos. I'll leave it at that for
2 now.

3 **DR. KENNETH PORTIER:** Thank you.
4 Now, let's go on and discuss the second question
5 because I think it will continue when we start
6 talking about the p-chem properties. Dan?

7 **DR. DANIEL SCHLENK:** Okay. Can you
8 hear me?

9 **DR. KENNETH PORTIER:** Yes. It takes
10 me a second because I mute and then I have to
11 unmute to say yes. But yes. We can hear you.

12
13 **CHARGE QUESTION 1 (1.2)**

14 **DR. DANIEL SCHLENK:** All right.
15
16 Yeah. Maybe we should just stick your little hand
17 up thing. That would actually do it, I guess.

18 Okay. Question 1.2, with the
19 exception of size characterization, the Committee
20 thought the physical and chemical properties of
21 chrysotile are generally well catalogued in the
22 table. But for those not familiar with asbestos
23 characteristics, the written description is rather

1 dry, non-visual, and uninformative. In fact if
2 allowed, a photograph or two of examples would be
3 very useful to the reader, and the Committee
4 member provided a couple examples there.

5 So this Committee member also
6 provided a text edit. For example, one of the
7 paragraphs that begin with "As with all silicate
8 materials," the Committee member thought that only
9 mineralogists would care to read that paragraph.
10 For everyone else it would be lost and find that
11 uninformative. Even though accurate, that
12 description does appear to set apart chrysotile
13 from the great number of other silicates and adds
14 nothing. Most readers will have no idea what they
15 are meant to visualize by this description.

16 And this Committee member provided a
17 much clearer example of text there that basically
18 -- and I can read that if you like. "It's
19 asbestos is not a mineralogical term but rather a
20 commercial and industrial term used to describe a
21 group of specific silicate minerals that form
22 bundles of long, very thin mineral fibers often

1 described as asbestiform. When crushed or
2 handled, asbestos bundles readily disaggregate and
3 release microscopic mineral fibers."

4 "Asbestos fibers are typically less
5 than a micrometer -- one thousandths of a
6 millimeter -- in diameter and range from several
7 micrometers to hundreds of micrometers in length.
8 Commercial-grade asbestos is composed of long,
9 thin, durable mineral fibers in fiber bundles that
10 exhibit high tensile strength, flexibility, and
11 resistance to heat, chemicals, and electricity.
12 These properties, especially its exceptional
13 insulation and fire-resistant abilities have made
14 asbestos widely used in numerous products and
15 industrial applications." And again, just more
16 text that basically make the description a little
17 bit more easier to understand.

18 Another point that was highlighted
19 by several Committee members was the issue of
20 purity of the chrysotile used in the products that
21 are being evaluated by this review. Some of the
22 largest and most productive chrysotile mines from

1 Monic, Quebec, Russia have claimed pure deposits
2 of chrysotile, which means that they lack
3 amphiboles. In most chrysotile deposits, the
4 fibers or asbestiform amphiboles can coexist with
5 the chrysotile ore body and are anthophyllite,
6 tremolite, and/or actinolite.

7 One member indicated no reason to
8 question the purity of the Russia chrysotile as
9 being used in the chlor-alkali industry. The few
10 references that exist about this enormous
11 chrysotile deposit indicate that it is amphibole
12 free. However, this member doubts that the
13 aftermarket brake pads and linings are
14 consistently amphibole free. This Committee
15 member indicates aftermarket products coming from
16 Asian sources may have issues with purity.

17 One Committee member suspects that
18 without extensive testing to indicate otherwise,
19 the aftermarket brake pads, linings, gaskets, and
20 other vehicle friction products could contain some
21 amount of amphiboles that were naturally
22 intermixed with the chrysotile. This member

1 acknowledges that the amphiboles may exist in
2 small amounts, and if they exist, their impact and
3 exposure will be difficult to model. The
4 recommendation here would be the potential for
5 coexisting fibrous amphiboles should be mentioned
6 in the document.

7 Another Committee member thought one
8 of the most important physical properties
9 associated with asbestos fiber is the aerodynamic
10 aspects of the fiber that allow penetration into
11 pulmonary areas of the lung. This did not seem to
12 be discussed in the document. If available,
13 metrics of aerodynamics for each fiber type Could
14 be provided. At a minimum, discussion regarding
15 this characteristic should be provided in the
16 text.

17 Single values for size and length
18 were provided in Table 1.1 and provided as median
19 values without variance. However, during the
20 presentation, the Agency did address size but only
21 with regard to human health. Fiber lengths less
22 than five microns termed -- and this is termed

1 five microfibers in the plastic literature -- have
2 been shown to be readily absorbed by aquatic
3 biota. And some references will be provided for
4 that.

5 Aggregation is also another very
6 important endpoint with microfibers, and there
7 doesn't appear to be a metric characterizing this
8 feature. Size and aggregation are becoming
9 significant issues in the characterization of
10 microfibers derived from plastics. Although
11 chrysotile is inorganic, several methods of
12 characterization with plastic microfibers in the
13 environment may allow a better estimate of
14 exposure.

15 One Committee member suggest -- and
16 references will be provided for that. One
17 Committee member suggested that the Agency be
18 careful when using the term "biologically inert"
19 as many micro and nano size materials have been
20 shown to have significant biological effects
21 following absorption, even though they may be
22 chemically inert. This, again, highlights the

1 importance of size and length characterization.

2 Two Committee members thought a consideration of
3 the changes to the table suggested in the public
4 comments by Mark Ellis representing the Industrial
5 Minerals Association - North America may be
6 warranted. Another Committee member would agree
7 with public commenters' recommendations to include
8 specific CAS numbers for the other asbestiform
9 fiber types to reduce confusion with non-
10 asbestiform materials -- or sorry -- minerals.

11 This is in Table 1.1. This commenter also
12 suggested other changes to optical properties in
13 1.1. Again, see comments from IMA-NA.

14 Two Committee members thought the
15 DRE doesn't have enough discussion concerning how
16 chrysotile structure differs from other forms of
17 amphibole asbestos with respect to the issues
18 important to biological outcomes. The members
19 indicated that discussion is warranted concerning
20 how chrysotile differs from amphiboles in its
21 chemical composition, durability, and morphology,
22 and how these parameters relate to dissolution and

1 clearance of chrysotile fibers in the lung and
2 pleura. The Committee believes that the entire
3 DRE could use some additional discussion in places
4 with what we have learned about asbestos from
5 animal studies, mainly that durability and
6 dimension and certain physical-chemical properties
7 are critical to the outcomes noted following
8 inhalation exposure.

9 The Committee recognizes that this
10 document isn't a summary of toxicological effects
11 but thinks a complete risk assessment should
12 incorporate some of the learnings from what are
13 thought to be translational, experimental models.
14 In the same light, the Committee thinks that some
15 discussion of the physical-chemical properties of
16 friction products is warranted with respect to
17 fiber dimension and surface changes as these are
18 known from animal studies to be important in study
19 outcome. As discussed above, the Committee
20 suggests a discussion on properties related to the
21 suspension of fibers, i.e., agglomeration and
22 settling rates. And that's it.

1 **DR. KENNETH PORTIER:** Thank you,
2 Dan. Dr. Blystone, anything to add?

3 **DR. SHERI BLYSTONE:** No. Nothing to
4 add.

5 **DR. KENNETH PORTIER:** Dr. Doucette?

6 **DR. WILLIAM DOUCETTE:** No. I think
7 Dan adequately captured all the comments.

8 **DR. KENNETH PORTIER:** Thank you.
9 Dr. Jiménez-Gonzalez?

10 **DR. CONCEPCIÓN JIMÉNEZ-GONZALEZ:**
11 Nothing to add. He did a great job again.

12 **DR. KENNETH PORTIER:** Dr. Johnson?

13 **DR. MARK JOHNSON:** I also have
14 nothing to add. Thank you.

15 **DR. KENNETH PORTIER:** Dr. Taioli?
16 We're not hearing her. Dr. Van Gosen?

17 **MR. BRADLEY VAN GOSEN:** It's a very
18 nice summary by Dr. Schlenk.

19 **DR. KENNETH PORTIER:** Dr. Everitt?

20 **DR. JEFFREY EVERITT:** Nothing to
21 add. I think Dr. Schlenk captured it.

1 DR. KENNETH PORTIER: And Dr.

2 Kanarek?

3 DR. MARTY KANAREK: Can you hear me?

4 DR. KENNETH PORTIER: Yes.

5 DR. MARTY KANAREK: I wonder if
6 there should be a section on microscopy that the
7 historical database is a prisoner to PCM. They
8 didn't measure below five and just -- that would
9 be -- I think that's an important addition. And
10 I'm sure Dr. Van Gosen knows much more about that
11 than I do, about how we measure chrysotile
12 asbestos.

13 DR. KENNETH PORTIER: Yeah. That's
14 a good point. This is Ken Portier. Thinking back
15 to one of the discussions, the last question we
16 talked about earlier this morning about -- that
17 related to size, both length and diameter, it
18 would be interesting to know a little bit more
19 about the distribution rather than the midpoint
20 median values. Dan mentioned a variance estimate
21 would be good or in a quartile range, something
22 like that.

1 But also, it'd be interesting to
2 know in natural samples or in some of these, in
3 this case, commercial chrysotile asbestos, what
4 fraction of the samples would be in the
5 biologically active size range, both length and
6 width. Is it 5 percent? Something a little bit
7 more than just the median size in Table 1.1. Any
8 additional comments from the panel on this
9 question? Dr. Van Gosen, I see your hand's still
10 up.

11 **MR. BRADLEY VAN GOSEN:** Yeah. It
12 is. It never got erased, but that's fine. You
13 have parallel efforts going on again here, where
14 NIOSH has developed a roadmap to research on
15 elongate mineral particles, which is a reference
16 that -- in the text. And everything we're talking
17 about has been -- and the FDA is --

18 **MR. MARTIN ALVARADO:** Having some
19 trouble hearing.

20 **MR. BRADLEY VAN GOSEN:** Oh, sorry.
21 Can you hear me now?

22 **DR. KENNETH PORTIER:** Yes.

1 **MR. MARTIN ALVARADO:** Yeah.

2 **MR. BRADLEY VAN GOSEN:** There's a
3 parallel effort now going on with the FDA to
4 address everything that has been discussed and
5 queried about this and this issue of size and what
6 biologically is important. And I think it's very
7 debatable right now. Many consider the short
8 fibers biologically important, too. So I do agree
9 with the comment that's saying biologically inert
10 is not technically true. Even if it's chemically
11 inert, it still causes irritation.

12 And then there's a document by
13 NIOSH, "A Roadmap to Research on Elongate Mineral
14 Particles." And it definitely should be cited and
15 read by the panel because it addresses all the
16 issues that everyone's having confusion with. And
17 it's a very complex issue, but it shouldn't be
18 understated. But what I am not clear on is the
19 character of the chrysotile size and length that
20 is used in the commercial products, and I don't
21 know if that information can be found. Thank you.

1 **DR. KENNETH PORTIER:** Dr. Van Gosen,
2 if you will send that reference or the document to
3 the DFO, it probably would be good to both
4 distribute it to the Committee but also put it in
5 the public docket for the public to find as well.
6 It may be not so easy to find on the FDA website.

7 **MR. BRADLEY VAN GOSEN:** Yeah. I'll
8 send it to Diana, and you can distribute it.

9 **DR. KENNETH PORTIER:** Thank you.
10 Dan?

11 **DR. DANIEL SCHLENK:** Yeah. I'll
12 address this a little bit later when we get to the
13 uncertainty sections on this but just to
14 highlight, size is becoming a very important
15 feature, again, in the plastics industry that
16 we're seeing over and over again that -- and,
17 again, I recognize plastics are not asbestos, but
18 they are fibers. And fibers are one of the most
19 commonly observed plastics that are being found in
20 effluents and in waterways right now.

21 And so there's a huge push by other
22 parts of the Agency to come up with a size-based

1 classification for these materials because we're
2 finding that, in most cases, the smaller the size,
3 the more adverse effects you see as opposed to the
4 large the size, which seems to be more important
5 at least in the human health side of things that
6 in that five -- up to the five to ten micron
7 category. Obviously, if it gets too big, then
8 obviously the lung filters that out. But there
9 seems to be this optimum size range. It seems to
10 be a bit larger than what we see as important in
11 the ecological side of things. So that's
12 basically my point.

13 **DR. KENNETH PORTIER:** Thank you.

14 Dr. Everitt?

15 **DR. JEFFREY EVERITT:** Yes. I'd just
16 like to make a comment. There seems to be a
17 disparity between epidemiology and animal studies
18 a little bit on the size issue. Clearly in animal
19 studies, short chrysotile and long chrysotile have
20 very different kinds of effects.

21 And I don't want to get into the
22 debate of is chrysotile toxic. But the fact that

1 we can't separate it out on epidemiology studies -
2 - if we accept that some of our animal models are
3 truly translational models, then I think we really
4 need to look carefully at, at least with friction
5 products, how toxic is it because short chrysotile
6 fiber that's non-amphibole contaminated has not
7 been shown in animal studies to be extremely
8 toxic.

9 **DR. KENNETH PORTIER:** Sounds like a
10 topic we'll come back to. Dr. Van Gosen, I see
11 your hand's still up. I didn't know if you wanted
12 to add any comments?

13 **MR. BRADLEY VAN GOSEN:** No. We
14 don't seem to have the ability to un-raise our
15 hand. It has to be turned off.

16 **DR. KENNETH PORTIER:** It's at the
17 very bottom of the participant page on the right.

18 **MR. BRADLEY VAN GOSEN:** Got it. Got
19 it.

20 **DR. KENNETH PORTIER:** You see the
21 little -- there you go.

1 **MR. BRADLEY VAN GOSEN:** Yeah. My
2 hand is down. But here --

3 **DR. KENNETH PORTIER:** Does anyone
4 else on the committee has a question? Yes. You
5 were saying, Dr. Gosen?

6 **MR. BRADLEY VAN GOSEN:** I was just
7 saying that a member of the NIH would have a lot
8 to say about this discussion right now, but he's
9 not part of this panel.

10 **DR. KENNETH PORTIER:** I don't see
11 any additional comments. At this point, I'll turn
12 back to EPA. Dr. Scarano, do you have any
13 clarifying questions?

14 **DR. LOUIS SCARANO:** I do not. I can
15 ask my fellow team members if they do. We
16 appreciate the comments that were made, and then
17 I'm anxious to read them more carefully. So thank
18 you for the panel's thoughts.

19 **DR. KENNETH PORTIER:** I guess it's
20 open to anyone else on the EPA team to ask a
21 question if it's not there. I'm not seeing any
22 questions. So I think we'll go ahead and move on.

1 For the EPA team there's always the
2 opportunity in the morning to bring back up a
3 question. Or any time during the day, if it comes
4 up to you, just let me know or let the DFO know
5 that you'd like to revisit a topic, and we'll be
6 happy to go back and discuss it a little bit.
7 Let's move on to Question 2, occupational
8 exposure.

9
10 **CHARGE QUESTION 2 (2.1)**

11
12 **DR. LOUIS SCARANO:** Okay. Thank
13 you. So this is Charge Question 2 on occupational
14 exposure. Workers and occupational non-users may
15 be exposed to commercial chrysotile asbestos when
16 workers perform activities associated with several
17 of the conditions of use or COUs: use of asbestos
18 diaphragms in the chlor-alkali industry, asbestos-
19 containing sheet gaskets, both stamping them and
20 using them, oil field brake blocks, aftermarket
21 automotive brakes and linings, other vehicle

1 friction products, and other gaskets. Next slide.

2 Do I have slide control? No. Okay. Thank you.

3 So EPA evaluated what is known about
4 chronic exposures to workers and occupational
5 nonusers (ONUs) for the COUs listed above via the
6 inhalation pathway only. The principle approach
7 EPA used to estimate occupational exposures -- for
8 both workers and ONUs -- was reviewing and
9 interpreting monitoring data, whether provided by
10 industry or documented in the peer-reviewed
11 literature. EPA assumed that workers and
12 occupational non-users would be adolescents and
13 adults of both sexes greater than or equal to 16
14 years of age.

15 Question 2.1, please comment on the
16 estimation methods and assumptions used for
17 occupational exposure assessment, including ONUs,
18 in terms of concentration, frequency, and duration
19 of exposures and their use in the risk evaluation.
20 Below are two specific issues in which EPA is
21 particularly interested in feedback from the SACC.
22 Next slide, please. Thank you.

1 This is the first of the two topics
2 under this sub-question. Incorporation of Short-
3 Term Occupational Monitoring Results, EPA received
4 from industry, or obtained from the published
5 literature, short-term -- that is less than a full
6 8-hour work shift -- monitoring data for several
7 of the COUs (chlor-alkali, sheet gaskets/stamping,
8 aftermarket automotive parts, and other vehicle
9 friction products). For these COUs, EPA
10 calculated a separate "full-shift" asbestos
11 exposure estimate, as well as a short-term
12 exposure estimate to account for these occasional,
13 short, high-exposure scenarios. Please comment on
14 the method used. Next slide, please. Thank you.

15 And this is the second part, and it
16 has a lot of parts to it. This has to do with ONU
17 exposure estimates. Based on the readily
18 available information, EPA used different methods
19 to estimate ONU exposures. ONU estimates were
20 made for each COU; however, the limited
21 information did not allow the development of ONU
22 exposures for short-term exposure scenarios for

1 chlor-alkali, sheet gasket use, oil field brake
2 blocks, or other gaskets/utility vehicles. Please
3 comment on the methods used which are identified
4 below.

5 So for the chlor-alkali portion,
6 which is Section 2.3.1.3.5, for the ONU exposure
7 estimates, area samples were used. Two chlor-
8 alkali facilities provided a total of 15 area
9 samples, which were all below the limit of
10 detection. There were two different detection
11 limits in the two submissions. Although true
12 exposure values below any limit of detection may
13 be unevenly distributed from zero to the limit of
14 detection, we assumed that the central tendency
15 exposure concentration estimate is based on one-
16 half of the detection limit for individual samples
17 and the high-end concentration is based on the
18 highest detection limit across the samples. Next
19 slide, please.

20 For the sheet gasket stamping and
21 use sections, which are 2.3.1.4.5 and 2.3.1.5.5,
22 respectively, EPA did not identify any ONU

1 exposure measurements for these conditions of use.
2 However, the literature includes "bystander"
3 exposure studies. Specifically, in one
4 publication -- Mangold, 2006 -- they measured
5 "bystander" exposure during asbestos-containing
6 gasket removal. The "bystander" locations in this
7 study were between five and ten feet from the
8 gasket removal activity, and asbestos
9 concentrations were between 2.5 and nine times
10 lower than those measured for the worker. Based
11 on these observations, EPA assumes that ONU
12 exposures for these COUs are a factor of 5.75,
13 that is, the midpoint between 2.5 and nine, lower
14 than the directly exposed workers. Next slide,
15 please.

16 This is for the oilfield brake
17 blocks COU, which is Section 2.3.1.6.5. EPA has
18 not identified specific data on potential ONU
19 inhalation exposures from brake block use. It is
20 assumed that ONUs do not directly handle brake
21 blocks and draw works machineries and that this
22 equipment is always used and serviced outdoors

1 close to the oil wells. Given the limited
2 information identified in Section 2.3.1.6.4 --
3 that is the worker monitoring values -- the lower
4 of the two reported values was used to represent
5 ONU exposures for this COU. Next slide, please.

6 This is for the aftermarket
7 automotive brakes COU, which is in Section
8 2.3.1.7.5. EPA has not identified data on
9 potential ONU inhalation exposures from
10 aftermarket auto brake scenarios. ONUs do not
11 directly handle brakes, and the ONU exposure
12 estimates in Table 2-15 were generated by assuming
13 that asbestos concentrations decreased by a factor
14 of 8.4 between the worker location and the ONU
15 location.

16 EPA derived this reduction factor
17 from a publication -- Madl, 2008 -- that had
18 concurrent worker and bystander exposure
19 measurements where the bystander was approximately
20 five feet from the worker. The value of 8.4 is
21 the average concentration reduction across four
22 concurrent sampling events. Next slide, please.

1 Finally, in the other gaskets or UTV
2 COU, which is Section 2.3.1.9.4, Paustenbach in
3 2006 reports have included area sampling results
4 that EPA thought was appropriate for ONU
5 exposures. These samples were collected at
6 breathing zone height at locations near the ends
7 of the muffler shop bays where the exhaust system
8 work was performed. The area sample durations
9 ranged from 25 to 80 minutes, and these samples
10 were collected during exhaust system work.

11 Overall, 21 area samples from these
12 locations were analyzed by phase contrast
13 microscopy or PCM, and 16 of these samples were
14 non-detectable for asbestos. Among the PCM data
15 from this subset of area samples, the authors
16 report that the average asbestos concentration was
17 0.005 fibers per cc, and the maximum asbestos
18 concentration was 0.015 fibers per cc. The study
19 authors did not report eight-hour time-weighted
20 average concentrations for the area sample
21 locations. EPA used these average and maximum

1 asbestos concentrations to estimate potential ONU
2 exposures. Next slide, please.

3 That's the first question. This is
4 Question 2.2 under occupational exposure. Please
5 comment on EPA's reasonableness of these
6 assumptions, the uncertainties they introduce, and
7 the resulting confidence in the occupational
8 exposure estimates which is summarized in Section
9 4.3.3. Question 2.3, please provide specific
10 suggestions or recommendations for alternative
11 approaches, estimation methods, or information
12 sources that EPA should consider for improving the
13 occupational exposure assessment. Next slide,
14 please.

15 Okay. No -- back. I thought that -
16 - those are the occupational exposure sections
17 listed under Charge Question 2. Thank you.

18 **DR. KENNETH PORTIER:** Thank you.
19 Let's scroll the slide back to where Question 2.1,
20 the main question is displayed. Thank you. And
21 the lead on this Question 2.1 is Dr. Kissel.

1 **DR. JOHN KISSEL:** Yes. So I got a
2 fair amount of responses. They aren't necessarily
3 directed at all of those sub-questions. Most of
4 the responses fall under the general rubric of 2.1
5 and a little bit under 2.2 and 2.3. So I need to
6 start, because I got a fair amount of text on
7 these issues, with some kind of preparatory
8 comments and general carryover from prior
9 meetings.

10 So the preparatory comment, which
11 has already come up, is that the document says
12 asbestos, and we're talking about commercial
13 chrysotile. And the other forms of asbestos are
14 not well represented. And other materials that
15 contain asbestos are not covered in this document.
16 So the generic issue here is, is the systematic
17 review actually systematic? I do have a list of
18 citations that members of this subgroup of the
19 Committee put forward, and I will provide those to
20 EPA.

21 But we also note that a very much
22 larger number of references were suggested by

1 public commenters. We have not reviewed all of
2 the papers since a lot of that dumped on us just
3 yesterday. We haven't reviewed all of those
4 suggestions and don't know their merit, but we do
5 believe that EPA should investigate whether those
6 multiple citations that they are not considering
7 in this document do, in fact, have merit and
8 relevance and should have somehow made it through
9 their document filtering system.

10 A second generic point is that the
11 exposures are not aggregated. Now, in this
12 particular case, only inhalation exposures were
13 calculated, so there was nothing to add together.
14 But it's an ongoing question with the Committee as
15 to why total exposures are not calculated rather
16 than route-specific exposures.

17 There are PPE -- a third generic
18 point, there are PPE assumptions built into this
19 risk evaluation. Again, EPA takes standard
20 numbers and applies them. In this particular
21 case, the paper by Riala and Riipinen would appear
22 to contradict some of the standard assumptions.

1 And a further discussion of why more weight wasn't
2 put on that experimental outcome would be
3 valuable.

4 And then the last part is legacy
5 exposures are left out and of which there are many
6 different types and involve talc and vermiculite
7 and EMP (elongate mineral particles). And one
8 particular thing that needs to be mentioned in the
9 context of asbestos is that there is a very
10 significant abatement industry which involves, at
11 least potentially, very significant occupational
12 exposures, which is not considered here because
13 legacy uses are not considered here. Oh, and one
14 final one, for asbestos, generically, there is
15 also a pretty significant literature on household
16 cohabit and exposures. And those are not
17 occupational non-users. Those are occupational
18 bystanders, and we don't have occupational
19 bystanders in this DRE or the previous one.

20 Okay. So Section 2.1, multiple
21 members mentioned that the frequent use of
22 graphics, pictures, and other graphics to display

1 the scenario development is very helpful and
2 appreciated by the Committee, as are the trip
3 reports from the visits to the chlor-alkali
4 plants. There are multiple data issues brought up
5 by members of the Committee and by public
6 commenters. One that was mentioned by many people
7 is the duplication of data. EPA has already
8 announced that they are attempting to fix that.
9 So I think everybody's on the same page with the
10 view that that duplicate data should not be used.

11 The metadata associated with
12 sampling activities, that actually came up in the
13 earlier discussion in the response to questions
14 raised from the presentations. So I will just
15 note that we are basically saying that, again,
16 that interpretation of data depends upon knowing
17 things about how it was actually collected. And
18 if you have multiple data sets, it might be that
19 if you read the sampling plans, you would not
20 weight them equally. Whereas if you don't -- if
21 all you have is the numbers, then the inclination
22 is to weight them equally. And that's potentially

1 a shortcoming and could lead to inadequate use or
2 inefficient use of better data and overemphasis of
3 weaker data.

4 There is mention in some places in
5 the write up for occupational exposure that older
6 data was available and wasn't used, but then that
7 older data is not described. At least one member
8 suggested that it would be useful to say something
9 at least qualitatively about the older data, not
10 incorporate it in the analysis but provide it so
11 that trends or other differences, gross
12 disparities might be noted which might cause
13 people to start thinking about the data that is
14 being used. With respect -- and this is one of
15 the specific questions -- so the Committee was
16 appreciative of the tabulated short-term data,
17 thought that it was well presented and that it was
18 helpful.

19 The issues, however, that were
20 raised by seeing the short-term data were how
21 often do "off normal" events occur, and to what
22 extent are they captured in the time-weighted

1 average data? And to what extent might they be
2 scrubbed from the time-weighted average data,
3 which might give a false picture of overall
4 workplace safety? And a second comment was that
5 the presence or the availability of short-term
6 data would show relatively high short-term events,
7 highlight the shortcomings of -- or potential
8 shortcomings of area data because the area data
9 might fail to pick up a local spike in air
10 concentration due to some type of an event.

11 Overall, the members are supportive
12 of a hierarchy of estimation which says that data
13 is superior to models and models are superior to
14 just using assumptions based upon occupational
15 exposure limits in the absence of data. And the
16 members do acknowledge that data scarcity is very
17 much a problem. There are mixed evaluations with
18 respect to a general thumbs up/thumbs down kind of
19 reviews of how well EPA has adapted to data
20 scarcity. But among the mentions were that the
21 treatment or -- the strategies for estimating
22 exposures to ONUs seem to be somewhat ad hoc. And

1 it might be to EPA's advantage, going forward, to
2 develop some type of a decision tree which says
3 something like "If you have this information, then
4 do this, and, if you don't, then the next step is
5 this and then the next step is that" so that the
6 process is more systematic than ad hoc.

7 Other comments that were
8 particularly noted that, if the ONU strategy for
9 the brake block scenario used the lower of two
10 available data points, two available data points
11 is obviously not an adequate data set, but at
12 least one member questions why if you have two
13 numbers, you choose the lower one. And with
14 respect to the worker scenario for the brake
15 blocks, the question is raised as to the scenario
16 is built around the replacement activity of the
17 brake blocks. But it would seem logical that
18 there is some continuous release associated with
19 the actual use of the brake blocks. And some
20 characterization of that would be of interest
21 because the members basically don't have any idea

1 how those numbers would compare to the replacement
2 numbers.

3 Some other specific points, a 16-
4 year starting age is not appropriate for all of
5 the COUs. 16 years starting age is fine for some
6 of the consumer activities under Question 3, but
7 there is a question as to why that young of an age
8 was chosen as a starting point. One of the public
9 commenters mentioned fiber behavior. And one of
10 the members of the Committee supports that
11 suggestion that perhaps EPA is underestimating the
12 settling of these particles. They are relatively
13 dense. The specific gravity is 2.5 for nominally
14 -- for chrysotile asbestos.

15 As lead, I will add a kibitz that
16 these -- most of the estimates are based upon
17 measured data. And measured data has embedded in
18 it a balance between ventilation, removal,
19 settling, and resuspension. And if you do a mass
20 balance on the system and jack up the settling
21 rates to a five-minute half-life, that requires --
22 in order to explain the observed measurements,

1 then you have to also jack up the emission rate
2 from the activity or the resuspension rate from
3 foot traffic. And one of the things that a more
4 rapid settling rate would imply is a much greater
5 dust load on the ground, which people are walking
6 around in.

7 So I think that comment has a nuance
8 to it that was lost in the way it was presented.
9 And I say fine. If you want the stuff to settle
10 fast, then explain the air measurements by greater
11 emissions and greater resuspension or inadequate
12 ventilation -- ventilation that's actually lower
13 than assumed.

14 Let's see. Another specific
15 comment, the definition of friable should be
16 included where it's first used. As -- well,
17 that's common for people that are immersed in this
18 thing. For a public document, that's a term of
19 art that is perhaps not widely recognized.

20 And on page 59, there is a
21 discussion of asbestos being described as "vital,"
22 citing a USGS document. One member notes that the

1 vital designation is from the chlor-alkali
2 industry and not from USGS. And I think -- oh, I
3 should have predicated my comment to say that I
4 got a lot of written comment, which I'm trying to
5 assemble into a logical text because it did not
6 follow the outline and the charge questions. And
7 so what I've given here are what I assessed to be
8 the bullet-point version.

9 And any member of the Committee who
10 feels like their specific points were not
11 adequately addressed is now invited to speak up so
12 that at least a tag for their issue would be
13 entered into the record. And I anticipate that we
14 will be expanding on these points as we actually
15 kind of produce the final write up. Okay. That's
16 it for 2.1.

17 **DR. KENNETH PORTIER:** Thank you, Dr.
18 Kissel. Dr. Anderson, do you want to add?

19 **DR. HENRY ANDERSON:** Yeah. I would
20 add just to cover it was difficult because of the
21 way the questions and the sub-questions were
22 organized as to where you'd put what comments. I

1 think some of my points about the knowing more
2 about the data collection and the methodologies
3 and the strategies that it's coming from it and
4 then how it's handled has already been covered.

5 I would say a question to EPA would
6 be, in the previous nine documents for ONU is when
7 there really wasn't specific data related to the
8 type of activity that we're seeing --, I would say
9 the default seemed to be to use the occupational
10 exposure central tendency -- the worker central
11 tendency as the exposure for the ONU. And that's
12 sort of incorporates some of the comments that
13 were made here. And you need to know all of the
14 things that are going on and what the sources in
15 this -- or the amount at the source and the
16 airflow and other things.

17 And then a number of the ONU
18 databases that were used were really not the same
19 work that was being done. I mean, the one for one
20 of the gaskets -- or one of them used -- actually
21 they were unloading boxes that contained brakes,
22 or it wasn't really the brake work being done

1 itself. Now, that does show the closeness of
2 somebody, but I think the issue of what actually
3 is the drop off, I don't think there was very good
4 data on that.

5 And so the confidence in actually
6 what the ONU exposures that were generated from
7 simulated studies, mostly on automobiles -- how
8 relevant that is -- it's more it was what was
9 available, And EPA used it, which is why I raised
10 the issue of it would be useful to have this
11 outline of how it's done. That if you use area
12 samples, you need to know how close the area
13 sample is to where these individual activities are
14 being done that really were documented by the
15 short-term samples.

16 So is the ONU people who are within
17 five feet? Is it within 15 feet? Or there needs
18 to be some kind of a systematic approach to
19 understanding what it is. And previous COUs in
20 other chemicals, kind of the question came up,
21 well, how is that ONU data? If those are away
22 aren't they just included with the workforce?

1 So there's quite a bit there on,
2 again, as I say, be useful to know why they feel
3 the central tendency, which certainly might
4 overestimate the ONU exposure -- but, again, the
5 data that was actually used could well
6 underestimate exposures as well. So there's quite
7 a bit of uncertainty there that sampling
8 strategies certainly would be helpful to add to
9 that. So I think we'll continue to work as a
10 group on some of this, though.

11 The other one, I thought there are a
12 lot of these have been addressed internationally
13 as well, and it wasn't clear that EPA had queried
14 -- other than trying to look at readily available
15 databases -- the international agencies that have
16 addressed some of these issues as well, especially
17 for the chlor-alkali industry since it was
18 mentioned in some of the public comments that the
19 EU has approached this and has a deadline for
20 internationally -- for the removal of asbestos
21 from it. So one would assume they must have done
22 some type of an exposure assessment that might

1 help inform the data that was generated here
2 that's now going to be reanalyzed. So that's
3 about all I would have addition to that.

4 **DR. KENNETH PORTIER:** Thank you, Dr.
5 Anderson. Dr. Blystone?

6 **DR. SHERI BLYSTONE:** Actually, I
7 think Dr. Kissel did a great job of summarizing
8 the varied inputs that he's received so far.
9 Might have something more to say later, but for
10 right now, I'm okay. Thank you.

11 **DR. KENNETH PORTIER:** Thank you.
12 Dr. Herrick?

13 **DR. ROBERT HERRICK:** Yeah. I
14 thought John's approach was really sound overall,
15 given that the way the questions were framed, it
16 was a little difficult to know exactly what
17 response to place where. Just a couple things
18 that I would throw in for consideration. One is
19 in Figure 1-5 where EPA talks about their
20 hierarchy and their preference for data over
21 modeling over information just from limits. It
22 comes up later in the confidence discussions, but

1 I actually think it's worth a little bit of
2 thought somewhere here about that all data isn't
3 really created equal and that some data --
4 particularly I'm thinking about data that was
5 generated from simulation studies -- might be a
6 little bit questionable in terms of how relevant
7 it actually is to the real world.

8 I think that's something that's
9 implicit. We acknowledged in the way the
10 confidence discussion plays out that it might be
11 worth raising that earlier in this section.
12 That's really the main thing I would add. I think
13 John put this all together nicely, and I agree
14 with his synopsis.

15 **DR. KENNETH PORTIER:** Thank you.

16 Dr. Jayjock?

17 **DR. MICHAEL JAYJOCK:** Yeah. Can you
18 hear me?

19 **DR. KENNETH PORTIER:** Yes.

20 **DR. MICHAEL JAYJOCK:** Hello. Can
21 you hear me? You can?

1 **DR. KENNETH PORTIER:** Yeah, just
2 fine. Yeah.

3 **DR. MICHAEL JAYJOCK:** Great, great.
4 Yeah. I don't think I sent this comment to Dr.
5 Kissel, and I'm going to make it again for the
6 non-occupational. But I think modeling can help
7 us here with regards to the ONUs or in the case of
8 the consumers, the bystanders, in that we did some
9 modeling some time ago that looked at dispersion
10 coefficients as a function of air speed outdoors
11 and dispersion coefficients indoors as a function
12 of just natural eddy diffusivity. And I think
13 those models could definitely inform what's been
14 going on with regards to any of the measured ONUs
15 or bystander exposures. So I'm going to forward
16 that information to the Agency with regards to
17 those references.

18 **DR. KENNETH PORTIER:** Thank you.
19 Send that, definitely, to Diana Wong so that she
20 can put it in the docket.

21 **DR. MICHAEL JAYJOCK:** Okay. And
22 what sort of deadline do we have on that?

1 **DR. KENNETH PORTIER:** Well, it'll be
2 referred to in the minutes so before we publish
3 the minutes. But the sooner you get it to her,
4 the sooner she can put it in the docket. I think
5 she'd probably prefer to have it before the end of
6 the meeting.

7 **DR. MICHAEL JAYJOCK:** Okay.

8 **DR. KENNETH PORTIER:** Those were the
9 associates. Any additional comments from the
10 Committee? Dr. Kanarek?

11 **DR. MARTY KANAREK:** I don't think in
12 the document there's a clear enough -- it was
13 mentioned by the discussant -- a clear enough
14 definition between worker, bystander, and the
15 people at home -- the take home exposures.
16 There's a whole literature -- I think Dr. Anderson
17 started it 50 years ago -- on the asbestos coming
18 home on the clothes and the body of the worker and
19 exposing the children and the spouses at home --
20 people at home, and that's not even mentioned in
21 the document.

1 **DR. KENNETH PORTIER:** Yeah. And I
2 think Dr. Kissel was referring to that because you
3 have the occupational worker, the occupational
4 non-worker, and then what we've, the Committee has
5 been referring to is the occupational bystander,
6 which is family and friends who are exposed
7 through being exposed to the worker bringing the
8 material home or to them. Is that what you're
9 referring to?

10 **DR. MARTY KANAREK:** Exactly.

11 **DR. KENNETH PORTIER:** Yeah. That's
12 --

13 **DR. MARTY KANAREK:** So there's
14 bystanders, so that's a person in the workplace,
15 but there's the household exposures. That's
16 different.

17 **DR. KENNETH PORTIER:** Well, under
18 the TSCA formulation what you're calling -- what
19 you're thinking of as bystander, they're calling
20 the occupational non-user. And what we're calling
21 the bystander is the person who is at home.
22 They're not standing by the worker. They're

1 residing with the worker and sharing exposures
2 through that, but their exposure is not in the
3 occupational setting. I guess that's the way to
4 put it. And anyone else here, Dr. Kissel, you can
5 correct me if I've got that wrong. Dr. Markowitz?
6 Oh, Dr. Kissel, yeah.

7 **DR. JOHN KISSEL:** Yeah. I was going
8 to say, yeah, that's correct. And occupational
9 bystander is probably not -- for people that are
10 starting of -- haven't been in this process all
11 along, we needed to get around the ONU setting,
12 which is a term of art which I hadn't heard before
13 I started working on this Committee. And in the
14 consumer side, we have bystanders who are the
15 child who's sitting next to mom while she does a
16 craft which causes some type of exposure to the
17 child. And so that's why I moved the -- why I
18 took the bystander label, which was not being used
19 for occupational, and inserted it in or suggested
20 it would be inserted into occupational, just so we
21 can kind of keep our terminology somewhat
22 consistent in the SACC TSCA world.

1 **DR. KENNETH PORTIER:** Dr. Markowitz?

2 Dr. Markowitz, you're muted in Webex.

3 **DR. STEVEN MARKOWITZ:** How's that?

4 Is that good?

5 **DR. KENNETH PORTIER:** Yep. We can
6 hear you now.

7 **DR. STEVEN MARKOWITZ:** Okay.

8 Thanks. So there are couple of issues I think
9 that maybe deserve additional highlighting. One
10 is that the data that were obtained from industry
11 via sampling data had some limitations and some
12 gaps, and perhaps EPA can help resolve some of
13 those gaps. I note EPA said in the address that
14 all of the chlor-alkali sites provided monitoring
15 information. Actually, the American Chemistry
16 Council, I think, in their public comments said
17 the same. But in fact, it appeared as only 10 or
18 11 out of the 15 of facilities actually provided
19 air monitoring. So I think EPA should be
20 encouraged to obtain all these data that are
21 available or at least to rest aside that
22 assertion.

1 The second aspect of the chlor-
2 alkali data is that it was -- EPA stated that it
3 was uncertain if certain high-exposure activities
4 were captured. Again, that's a gap that should be
5 noted and resolved if possible. I would point out
6 on the sheet gasket stamping air monitoring data
7 that that was -- as we see in the draft risk
8 evaluation, that was limited to a single day and
9 perhaps not representative of actually what goes
10 on.

11 Finally, on the issue of gaps and
12 data, the QR sampling data was obtained from
13 industry but lacked some important documentation
14 that should be obtained if it could. And the
15 second point I just want to emphasize -- and I
16 think this was reviewed by Dr. Kissel, but I'm not
17 entirely certain. In the write up of certain of
18 the conditions of use, in terms of what -- not the
19 original industry air monitoring but that obtained
20 from the peer-review studies, it actually quoted -
21 - the draft risk evaluation actually quotes very

1 few studies, particularly for gasket use and for
2 the brake literature.

3 In the gasket write up, for
4 instance, there are just two studies that were
5 identified, Spence and Mangold. There are many
6 more in the published literature, which show
7 varying results in both directions, and I think
8 that -- as I include in the write up -- it should
9 include some of those studies so we can have
10 confidence that their estimates of typical
11 exposures actually apply. And we know that EPA
12 reviewed at least many of these from the quality
13 assessment and from Figure 1.5 , which reviewed
14 the data sources. But they're not included in the
15 write up here, and I think it ends up being a very
16 narrow kind of recitation of relevant literature.
17 And I can provide some of those studies, but,
18 actually, I think many of them are already
19 included in the data review.

20 The same comment applies to the
21 brake literature, which is that really just a few
22 studies are cited, a couple of which are not so

1 relevant or were actually rejected by EPA. But
2 there is a much larger brake literature, again,
3 with a whole variety of results that should be
4 included in the development of the proper
5 estimates to use for brake exposure. Otherwise,
6 frankly, the credibility of this review or of the
7 estimates used is subject to question.

8 **DR. KENNETH PORTIER:** So Dr.
9 Markowitz, this is Ken Portier. So you reference
10 the quality evaluation. Are there papers that
11 were -- I wanted to make sure I heard you right.
12 There may be references to studies that were not
13 covered in the quality review, and there's other
14 studies that were covered in the quality review
15 which weren't carried forward and discussed in the
16 DRE that you felt should be discussed? Is that
17 correct?

18 **DR. STEVEN MARKOWITZ:** The latter is
19 true, certainly, for the gaskets.

20 **DR. KENNETH PORTIER:** Yeah. So it
21 would be nice to be able to kind of pull that out
22 and discuss the reasons for why they should have

1 been carried forward because it sounds like you're
2 not necessarily agreeing with EPA's assessment of
3 the quality of that study. There was something
4 about the study that you felt should be brought
5 forward. And I think they're always interested to
6 hear why that -- because I think it helps them
7 fine tune their quality assessment.

8 **DR. STEVEN MARKOWITZ:** I think what
9 I'm saying is that, in the write up, the basis for
10 their selection of typical exposures should be
11 based on a broader literature, which they are
12 aware of and which they haven't carried forward to
13 the draft risk evaluation.

14 **DR. KENNETH PORTIER:** Yeah. Dr.
15 Jayjock.

16 **DR. MICHAEL JAYJOCK:** Yeah. Just
17 along those lines, I wanted to kind of chime in to
18 say that most of the data, essentially all the
19 data from the chlor-alkali, I think, is from non-
20 published work, and so it's industry sponsored or
21 industry done work. And so I think the criteria
22 on the other side for people that were doing work

1 for NGOs or other areas should be included in this
2 as well. I mean, the fact it was industry work I
3 think has its own bias, and then I think the NGOs
4 and perhaps universities might have their own bias
5 from the left. So I certainly echo that it should
6 be broader and include that. And some of the
7 commenters yesterday said they had data of that
8 type that really needs to be included.

9 **DR. KENNETH PORTIER:** Thank you.
10 Dr. Blystone.

11 **DR. SHERI BLYSTONE:** Yeah. Just
12 reacting to the monitoring data from the chlor-
13 alkali business, I mean, remember that this is
14 under an OSHA program. It's not industry deciding
15 to do it. It's under OSHA regulations.

16 **DR. KENNETH PORTIER:** Thank you.
17 Dr. Markowitz, I see your hand's still up. There
18 you go. I don't see any additional hands. Before
19 we go on to Question 2.1, I'd like to scroll down
20 to Slides 5 through 8 and just touch base with Dr.
21 Kissel to see -- you feel like you have
22 information to be able to answer kind of -- or to

1 comment on this short-term occupational monitoring
2 result? I mean, just kind of yes or no or are we
3 going to need to work on this? I wasn't quite
4 sure where all these things are. John?

5 **DR. JOHN KISSEL:** I don't have
6 specific answers to those -- I don't know -- the
7 sub-questions under 2.1, generally speaking. Some
8 of the things that I mentioned actually were on
9 point, but I didn't get reviews that addressed
10 those specifically. I don't think all those
11 subpoints were in the original version of the
12 charge questions.

13 **DR. KENNETH PORTIER:** Yeah. I tend
14 to agree with you, John. That's kind of why I was
15 asking. I mean, these are questions that we've
16 addressed before, like the eight-hour work shift
17 estimation from shorter term monitoring data,
18 these kind of things. Let's go onto Slide 6. We
19 talked a little bit about the ONU exposures. And
20 it sounded we need -- we've talked a little bit
21 about the chlor-alkali. The next slide, we've

1 talked a little bit about the sheet gasket
2 stamping.

3 Next -- Slide 7, please. We just
4 had a little bit of talk about this. The
5 questions here are specifically about distance and
6 then using that to estimate the ONU exposure. And
7 anyone who has any comment on that it would be
8 nice to be able to -- I just didn't quite hear
9 the answer to some of these fairly specific
10 questions. Next, Slide 8.

11 **DR. HENRY ANDERSON:** Ken, let me --
12 I can make a --

13 **DR. KENNETH PORTIER:** Yeah. Dr.
14 Anderson.

15 **DR. HENRY ANDERSON:** Yeah. Again,
16 this kind of gets into the general what's
17 appropriate data to use or do you just look for
18 any data that you might be able to use and say,
19 "Well, that's the best we can do"? In a number of
20 these that are -- it says that in the write up.
21 It's surrogate data, so it isn't actual data
22 that's related to the kind of work that's be -- or

1 that is COU. But they go to another COU and then
2 say, "Well, this mentions the terminology and,
3 therefore, that's surrogate data that we can use
4 for the ONU."

5 And that was kind of why most of my
6 comments here is I would say it's often a stretch
7 as to -- because it was said to be an occupational
8 bystander of a certain distance or whatever, but
9 it really wasn't totally applicable. Or it's hard
10 to say that this would be -- a good one-time
11 characteristic that's going to characterize all of
12 the ONU types of exposures. So it's when you go
13 back to -- and I think Dr. Jayjock mentioned using
14 some of the other characteristics of fiber
15 movement or particle movement to augment or come
16 up with these estimates.

17 So there isn't a good overall
18 estimate. As I said in the previous chemicals,
19 when we faced this, EPA just said, "Well, in these
20 instances we use the central tendency data from
21 the worker to estimate the ONU exposure," and then
22 you don't have, necessarily, a high-end exposure

1 for the ONUs. So it would be helpful to have a
2 closer look by EPA at you always have to come up
3 with data or a mechanism to estimate an ONU. And
4 when do you use a default type of assumption to
5 give that exposure?

6 **DR. KENNETH PORTIER:** Thank you, Dr.
7 Anderson. I want to make one more comment, and
8 then we're going to break. We're going to take a
9 15-minute break. On Slide 10, it mentions the 21
10 area samples. One of the things I was looking for
11 was you mentioned that 16 were non-detects, but it
12 doesn't mention what the detection level was.

13 So it's hard for me to know whether
14 this average asbestos concentration of 0.005
15 fibers per cc is a reasonable estimate. I suspect
16 because 16 of 21 were non-detects, this 0.005 is
17 probably one half the detection limit and 0.01 was
18 the detection limit. But I don't know. And when
19 I went to Section 2.3.1.9.4, I don't see the
20 detection limit mentioned there either. So I'm
21 going to leave that question open.

1 Let's go ahead. I have 12:05 p.m.
2 Eastern Time. Let's take a 15-minute break and
3 return at 12:20 Eastern. Thank you.
4

5 **[BREAK]**
6

7 **DR. KENNETH PORTIER:** Okay. I have
8 12:20. Can we reconvene? Dr. Anderson, I still
9 see your hand up. I wonder if you have any
10 additional comments on Question 2.1? Otherwise,
11 I'm ready to move onto Questions 2.2 and 2.3.

12 **DR. HENRY ANDERSON:** Yeah. I did.
13 I actually had written under each of the ONU
14 issues. So let me just read the one, and this is
15 just -- I've already talked about this. It's
16 maybe a little more specific, though. Related to
17 the sheet gasket stamping and use, I state here in
18 the instance of the sheet gaskets were used, EPA
19 went to a totally different COU where the work
20 environment was not a factory setting and used
21 database on automobile gasket removal rather than
22 a sheet gasket making process. So using that data

1 for the sheet gasket stamping activity seemed to
2 be a bit of a reach to me.

3 And then further on, the Mangold
4 study is used for underlying exposure, and that
5 really was looking at, again, a simulated
6 environment in a marine vessel. One has to
7 question it. They don't say how that type of that
8 work in that environment is very similar to what
9 was being done at the titanium dioxide factory
10 circumstance. So I say the use of the surrogate
11 data hasn't been, I think, adequately defended by
12 EPA. It's just what they found but why it's truly
13 relevant.

14 And then on the UTVs, they spoke to
15 that a little bit yesterday, but, again, surrogate
16 data is selected to estimate ONU exposure. And
17 it's a research simulation study on automobiles --
18 older automobiles rather than actual UTVs. And I
19 didn't see any write up really describing the UTV
20 process other than to say that most of the UTVs
21 are serviced in the UTV sales programs rather than
22 in automobile service facility. So it would have

1 been helpful if there would be some description of
2 how difficult it is to get the gasket out of the
3 UTVs when those exhaust systems are replaced.

4 And, again, it's surrogate data from
5 a simulation study that is being used. And that
6 seems to be really pushing out the envelope on
7 what data is reasonable to use to accurately
8 estimate what ONUs exposures in these
9 circumstances are. So those are just two of the
10 write ups that I have, and I had others in here as
11 well.

12 **DR. KENNETH PORTIER:** Thank you, and
13 make sure those get copied over Dr. Kissel. Does
14 anyone else on the Committee want to comment on
15 2.1? I'm not sure. Maybe I'll turn to EPA right
16 now and see whether they had any clarifying
17 questions on 2.1?

18 **DR. LOUIS SCARANO:** Thank you, Dr.
19 Portier. This is Gino Scarano. I do not but,
20 once again, I invite my team members if they have
21 anything like that they'd like to ask.

1 **DR. STAN BARONE:** This is Stan
2 Barone, Dr. Portier. There were a number of
3 comments about data from different sources. And I
4 want to remind the Committee and the public that
5 any data we receive will have to go through the
6 same data evaluation criteria that we have for
7 peer-reviewed publications as we have for non-
8 peer-reviewed publications.

9
10 **CHARGE QUESTION 2 (2.2)**

11 **DR. KENNETH PORTIER:** It's a good
12 point. Dr. Kissel, let's go ahead and take on
13 2.2, the reasonableness of assumption and
14 uncertainties.
15

16 **DR. JOHN KISSEL:** Okay. I got very
17 little comment on 2.2. One member found the Table
18 2.24, which is on page 105, was well presented and
19 easily followed. But another member said that
20 generally that -- and I think this kind of mirrors
21 what Dr. Anderson just said -- that the discussion
22 of the assumptions that EPA made was inadequately
23 defended in Section 4.3.3, which is the

1 uncertainty part on occupational, which is on page
2 193.

3 So generally, EPA looked at
4 inadequate data and made decisions, and now
5 they're asking us how do we feel about it. And I
6 think most of the Committee members looked at it
7 and said, "Well, there isn't enough data and you
8 have to do something, so you have." But it's not
9 -- I think many of those assumptions are not
10 greeted with great enthusiasm. It's just the
11 general resignation that there isn't adequate data
12 in many of the cases.

13 **DR. KENNETH PORTIER:** Thank you.
14 Let's see, this is -- Dr. Anderson, anything to
15 add? I know a lot of what you just said discussed
16 reasonableness.

17 **DR. HENRY ANDERSON:** Yeah. I think
18 it would be very helpful -- because this has come
19 up in all of the other chemicals as well. There
20 needs to be almost a white paper on the ONU
21 approach and when does the data reach what would
22 be reasonable to use or give you a great deal of

1 confidence. Frequently, EPA makes a confidence
2 statement, and it tends to be either it's a
3 moderate confidence, but, given the amount of data
4 that's there, it's hard to say that it's really
5 representative. I mean that I think is really the
6 overall problem. It's describing it and then
7 moving on with it, but the degree of confidence --
8 and not carrying that necessarily forward into the
9 overall discussion later on in the document on the
10 hazard assessment thing. You're basing your
11 hazard assessment on these determinations that are
12 on pretty shaky grounds.

13 **DR. KENNETH PORTIER:** Thank you.

14 Dr. Blystone.

15 **DR. SHERI BLYSTONE:** Nothing to add
16 at this point.

17 **DR. KENNETH PORTIER:** Dr. Herrick?

18 **DR. ROBERT HERRICK:** Oh, there we
19 go. Yeah. I would agree with Dr. Anderson's
20 point. I actually kind of thought that Table 2-24
21 was a good presentation of the information, and I
22 especially thought including the confidence

1 ratings was really valuable. But like him, I'm
2 not sure how that sense of confidence is carried
3 through into the final synthesis of the risk
4 estimate. So I would just encourage the EPA to
5 try to make sure that that information is actually
6 used.

7 **DR. KENNETH PORTIER:** This is Ken
8 Portier. I was kind of listening to you and Dr.
9 Anderson. And I thought to myself do you agree
10 with the confidence rating, especially on these
11 ONUs given the discussion we've just had on these
12 approaches to ONU estimation that were just
13 discussed? I mean, they have these confidence
14 levels as medium, and I thought I was hearing much
15 lower confidence.

16 And I think, in answer to your
17 question, I think the confidence rating moves
18 forward when we characterize risk and uncertainty
19 around risk and that it gets discussed at that
20 point. And so it does have an impact whether the
21 confidence rating is low, medium, or high at that
22 point. So Dr. Herrick, anything else?

1 **DR. ROBERT HERRICK:** Yeah. Well,
2 just on that point, I mean -- and it may be in
3 here and I just missed it. But it might be nice
4 to have some text in here from EPA to sort of help
5 us calibrate what they really mean by the
6 confidence high, confidence medium, confidence
7 low. Is there some way to expand on those
8 concepts a little bit? Because I think everyone
9 who deals with data may have their own sort of
10 working definitions of high, medium, and low. But
11 it would be interesting to see that discussed in a
12 little more detail here.

13 **DR. KENNETH PORTIER:** You'll
14 probably find it in Section 2.3.1.9.5 under data
15 assumptions, uncertainties and level of
16 confidence. Typically, they try to lay out the
17 criteria that they use to assess confidence. I'm
18 not seeing it in this, whatever, five paragraphs
19 in this section. So it may actually be something
20 that's missing here. That would be a good thing
21 to point out. Dr. Jayjock?

1 **DR. MICHAEL JAYJOCK:** Yeah. I think
2 the -- I just did some -- I actually did some
3 research --

4 **DR. KENNETH PORTIER:** I hear you
5 now.

6 **DR. MICHAEL JAYJOCK:** Can you hear
7 me? Can you hear me? Hello? Hello. Can you
8 hear me?

9 **DR. KENNETH PORTIER:** Dr. Jayjock?

10 **DR. MICHAEL JAYJOCK:** Hello. Yeah.
11 Oh, hold on. Sorry. I had the phone on mute and
12 then not the computer. Yeah. The issue with
13 uncertainty for outdoors -- and I just did some
14 research during the break, actually, where I found
15 an outstanding paper on indoor bystander exposure
16 and, as I mentioned before, some work that we did
17 on outdoor bystander exposure. And my sense is
18 that the outdoor bystander exposure was
19 dramatically overexposed in the DRE. So yeah. I
20 think the confidence is low for outdoors.

21 **DR. KENNETH PORTIER:** Dr. Anderson,
22 I see your hand's still up.

1 **DR. HENRY ANDERSON:** I was just
2 going to add in there that I think some of this
3 begins when we talked earlier about the systematic
4 review and the scoring table that's used to grade
5 the various studies. And then that gets carried
6 forward into these characterizations of confidence
7 and things like that. So all of these kind of fit
8 together but if you change the criteria or looked
9 at it differently in the systematic review and the
10 way the studies are scored, that might change,
11 then, which you use. So it's kind of problematic
12 how some of the issues we would have with a
13 systematic review then carry forward and lead to
14 what I think are problematic choices,
15 subsequently, in the document.

16 **DR. KENNETH PORTIER:** Dr. Kissel, I
17 kind of thought you were ready to jump in on
18 something.

19 **DR. JOHN KISSEL:** No. I'm ready to
20 go to 2.3 if --

21 **DR. KENNETH PORTIER:** Let's go to
22 2.3.

CHARGE QUESTION 2 (2.3)

DR. JOHN KISSEL: Okay. So 2.3 is the question about alternative approaches and methods, and most of what I had to say actually got brought up already. I had moved the question of the prior ONU standard, which was to just use the central tendency of workers. That showed up in many of the prior DREs, and so now it becomes - - because it wasn't used here, then it becomes an alternative for in this DRE. And I also already mentioned that both members and public commenters have come up with a bunch of potential articles, which would have to be reviewed by EPA, but that are potentially relevant and that I will be assembling and providing a list of at least the ones that are provided by the associate members. EPA already has suggestions from the public commenters.

And then the last point, which kind of overlaps with the consumer exposure world a bit, is that a couple of us have gone and searched

1 the Internet because this issue of what can be
2 purchased and what's out here has come up. And
3 there are effectively chatrooms where people talk
4 about how you grind brake pads. And there are
5 YouTube videos on how to grind brake pads, which
6 suggests that there's people still doing it either
7 in a professional setting or on their own. And
8 that's where the overlap comes from.

9 But some of the discussion groups
10 say things of the type, "Well, my local mechanic
11 will still do that for me." And this is non-
12 quantitative. This is just qualitative
13 information, but just the observation of the
14 Internet world is that there are people doing
15 things that are ostensibly not done anymore.

16 **DR. KENNETH PORTIER:** Is that it,
17 John?

18 **DR. JOHN KISSEL:** Yeah. That's what
19 I have for 2.3.

20 **DR. KENNETH PORTIER:** Well, we'll
21 move down through the associates. Dr. Anderson,
22 anything else on suggestions?

1 **DR. HENRY ANDERSON:** Sure. It's
2 never been done on the exposure side, but there's
3 lots that hasn't been done in what EPA has
4 proposed over the time. So one thought here is
5 when we do risk assessment on animal data, we have
6 an uncertainty factor for data quality. So one
7 kind of subjective issue could be to when you have
8 such poor or unreliable data to have a data
9 uncertainty factor that would be added into the
10 exposure assessment. I wouldn't advocate for
11 that.

12 But again, it's coming up with a
13 fallback position of default if in fact you can't
14 have the option of saying "We just don't have
15 enough data in order to come up with these
16 assessments." So if you have to have it, then
17 some kind of a default needs to be used, as I said
18 earlier in the other, it was to use the median
19 occupational worker exposure. Another would be to
20 have some kind of an uncertainty factor that would
21 take into account your confidence level.

1 DR. KENNETH PORTIER: Thank you.

2 Dr. Blystone?

3 DR. SHERI BLYSTONE: Nothing to add.

4 DR. KENNETH PORTIER: Dr. Herrick?

5 DR. ROBERT HERRICK: Well, in a
6 field like this where it's so clear that the data
7 is extremely sparse -- and I'm trying to keep in
8 mind the comment someone made yesterday to remind
9 us that this office of EPA is not a research
10 entity -- it's not really fair for us to be
11 suggesting a major research agenda to generate new
12 information, at least to these folks. But I
13 wonder if there is some place in a report like
14 this to identify research needs to help fill in
15 what's very obviously a great big set of gaps in
16 the information that EPA has to make this kind of
17 a decision?

18 DR. KENNETH PORTIER: Thank you.

19 Dr. Jayjock?

20 DR. MICHAEL JAYJOCK: Yeah. Can you
21 hear me now?

1 DR. KENNETH PORTIER: Yeah. Just
2 fine.

3 DR. MICHAEL JAYJOCK: Thank you.
4 Good. I think the idea of trading conservatism
5 for data is kind of a time-honored piece in risk
6 assessment. I've been kind of doing it for 30
7 years. When the data are very sparse, you default
8 to reasonable worst case, and I think the Agency
9 has done a creditable job of doing that.

10 DR. KENNETH PORTIER: I thought I
11 heard a "but" there. But --

12 DR. MICHAEL JAYJOCK: No. No "but"
13 from me.

14 DR. KENNETH PORTIER: Okay. Just
15 wondering. Anyone else on the committee want to
16 comment either one of these questions? I don't
17 remember who mentioned it, but the idea of a white
18 paper on ONU approach is something that I've
19 thought about in the past and plan to include in a
20 series of recommendations to the OPPT
21 administrator for a future task for the Committee
22 -- or future task for EPA to do and the Committee

1 to review because I think we've seen that need
2 there -- kind of guidance on how this should be
3 done within the TSCA framework.

4 Okay. I'm not seeing any additional
5 comments, hands going up. I'll turn to EPA. Dr.
6 Scarano and your team, any questions on this,
7 clarifying comments?

8 **DR. LOUIS SCARANO:** Thank you, Dr.
9 Portier. This is Gino. I have none. Once again,
10 I invite my team members. But by the way, thank
11 you for all the comments.

12 **DR. KENNETH PORTIER:** So while the
13 EPA team's thinking about this, I'm going to turn
14 to Dr., I guess, Bennett, who's the lead on Charge
15 Question 3, about whether you want us to jump into
16 Question 3.1 or go ahead and take our lunch break
17 at this time? Dr. Bennett?

18 **DR. STEVEN BENNETT:** Yeah. Good
19 afternoon. This is Dr. Bennett. I'm -- I think,
20 looking over responses to 3.1 through 3.7, there
21 is a lot of overlap between a lot of our
22 responses. And I think the conversation would go

1 a lot better if we kept it together as we went
2 through that. But looking through the responses,
3 there's quite a bit of similar comments because of
4 the way the questions are structured and the way
5 we're evaluating those. So I think that would be
6 the better approach.

7 **DR. KENNETH PORTIER:** Okay. That's
8 kind of my preference, too. I had low blood sugar
9 at the break, and I really need some food at this
10 point. Anyone else on EPA team, comments? I'm
11 not seeing any hands going up. Dr. Wong, do you
12 have any objection to us going ahead and taking
13 our lunch break 15 minutes early here? That would
14 bring us back from lunch at, what, 1:30? I guess
15 we're a half hour ahead. Come back from lunch at
16 1:30 Eastern?

17 **DR. DIANA WONG:** No objection from
18 me. So we have an earlier lunch.

19 **DR. KENNETH PORTIER:** Okay. Let's
20 go ahead then and break. I have 12:45. We're a
21 little bit ahead of the schedule. So maybe we'll
22 return at 1:30, 1:35. Let's say 1:35. Give us a

1 little five minutes' extra lunch. Is that okay?

2 Let's make it happen, then.

3 **DR. DIANA WONG:** Okay with me.

4 **DR. KENNETH PORTIER:** I'll see you
5 at 1:35 Eastern. Thank you.

6
7 **[LUNCH BREAK]**

8
9 **DR. KENNETH PORTIER:** Good
10 afternoon. I'd like to reconvene at this point.
11 We're going to start the afternoon session by
12 calling the roll. Let's see who's made it back
13 from lunch. Dr. Anderson.

14 **DR. HENRY ANDERSON:** Yes. I'm here.

15 **DR. KENNETH PORTIER:** Dr. Barton.

16 **DR. CHARLES BARTON:** Here.

17 **DR. KENNETH PORTIER:** Dr. Bennett.
18 Dr. Bennett? Dr. Blystone.

19 **DR. SHERI BLYSTONE:** I am here.

20 **DR. KENNETH PORTIER:** Dr. Cory-
21 Slechta?

22 **DR. DEBORAH CORY-SLECHTA:** I'm here.

1 DR. KENNETH PORTIER: Dr. Davies.

2 DR. HOLLY DAVIES: I'm here.

3 DR. KENNETH PORTIER: Dr. Doucette.

4 DR. WILLIAM DOUCETTE: Good

5 afternoon.

6 DR. KENNETH PORTIER: Dr. Jiménez-

7 Gonzalez.

8 DR. CONCEPCIÓN JIMÉNEZ-GONZALEZ:

9 I'm here.

10 DR. KENNETH PORTIER: Dr. Johnson.

11 DR. MARK JOHNSON: I'm here.

12 DR. KENNETH PORTIER: Dr. Kaufman.

13 MR. ALAN KAUFMAN: Still chewing,

14 but I'm here.

15 DR. KENNETH PORTIER: Dr. Kissel.

16 DR. JOHN KISSEL: Here.

17 DR. KENNETH PORTIER: Dr. Rowlands.

18 Dr. Rowlands.

19 DR. CRAIG ROWLANDS: Sorry. I was

20 on mute. Yeah. I'm here.

21 DR. KENNETH PORTIER: Thank you.

22 Dr. Schlenk.

1 DR. DANIEL SCHLENK: Good morning.

2 DR. KENNETH PORTIER: Dr. Sheela.

3 DR. SHEELA SATHYANARAYANA: Here.

4 DR. KENNETH PORTIER: Dr. Bennett?

5 DR. STEVEN BENNETT: Sorry. This is
6 Dr. Bennett. I'm here. I couldn't figure out how
7 to turn off the mute.

8 DR. KENNETH PORTIER: Okay. Dr.
9 Crump. Dr. Crump? Dr. Everitt.

10 DR. JEFFREY EVERITT: Here.

11 DR. KENNETH PORTIER: Dr. Herrick?

12 DR. ROBERT HERRICK: Here.

13 DR. KENNETH PORTIER: Dr. Jayjock?

14 DR. MICHAEL JAYJOCK: Here.

15 DR. KENNETH PORTIER: Dr. Kanarek?

16 DR. MARTY KANAREK: Here, I'm here.

17 DR. KENNETH PORTIER: Dr. Markowitz?

18 DR. STEVEN MARKOWITZ: Present.

19 DR. KENNETH PORTIER: Dr. Sheppard?

20 DR. ELIZABETH SHEPPARD: Here. It's
21 still morning here.

1 **DR. KENNETH PORTIER:** Yeah. I was
2 going to say you guys just had breakfast. I
3 understand, late breakfast. We're having late
4 lunch. Dr. Shukla.

5 **DR. ARTI SHUKLA:** I'm here.

6 **DR. KENNETH PORTIER:** Dr. Taioli.
7 Dr. Taioli? Dr. Van Gosen.

8 **MR. BRADLEY VAN GOSEN:** Brunch here.

9 **DR. KENNETH PORTIER:** Let's see if
10 Dr. Taioli is on. I don't see Dr. Taioli. Dr.
11 Crump.

12 **DR. KENNY CRUMP:** All right. I'm
13 here now. I've been having some technical
14 difficulty. We lost power for a while, but then
15 my internet wasn't working, so I finally got it
16 cobbled together. So I'm back.

17 **DR. KENNETH PORTIER:** You know if
18 you guys do lose power, your phone may still --
19 your cell phone may still work, and you can dial
20 in the number if you have that number written down
21 somewhere. Everyone's here.

1 **DR. KENNY CRUMP:** But how do you --
2 how do you unmute your line?

3 **DR. KENNETH PORTIER:** Well, we'll
4 unmute you on this side, and then you use your
5 phone to mute or unmute. I don't know. The host
6 may be able to tell you how to do that. You won't
7 be able to see the slides obviously. Let me make
8 sure the DFO is here. Dr. Wong?

9 **DR. DIANA WONG:** I'm here. I just
10 unmute myself.

11 **DR. KENNETH PORTIER:** Okay. Good.
12 Everyone's here, and I guess we're ready to move
13 on with Question 3 around consumer exposure. Dr.
14 Scarano, would you read in Question 3.1?

15

16 **CHARGE QUESTION 3 (3.1)**

17

18 **DR. LOUIS SCARANO:** Yeah. Thank
19 you. And I understand now I can control the
20 slides, so I have it. Question 3 on consumer
21 exposure, there are seven sub-questions.
22 Consumers or do-it-yourselfers, or DIY or DIY

1 mechanics, and bystanders may be exposed to
2 commercial chrysotile asbestos when consumers
3 perform activities associated with these COUs:
4 aftermarket automotive brakes and linings, and
5 other gaskets or utility vehicles, or UTVs. Sub-
6 question 3.1, please comment on the estimation
7 methods and assumptions used for consumer/DIY
8 exposure assessment, including bystanders, in
9 terms of concentration, frequency and duration of
10 exposures and their use in the draft risk
11 evaluation. Please include your thoughts on the
12 reasonableness of the estimated age at start of
13 exposure and duration and frequency of exposure
14 for the consumer, DIY and bystander, Section 4.2.3
15 in the draft risk evaluation. Sub-question 3.2 --

16 **DR. KENNETH PORTIER:** Let's just go
17 ahead and take 3.1 to start with because we have
18 one lead for that, and then we have a lead for 3.2
19 to 3.4. So let's start with Dr. Bennett on
20 Question 3.1.

21 **DR. STEVEN BENNETT:** All right.
22 Good afternoon. Yeah. As I indicated -- this is

1 Steven Bennett. As I indicated before lunch, I
2 think there'll be quite a bit of overlap in some
3 of these questions, so I think it's a good
4 approach of kind of taking these in groups. But I
5 think in general with the question to the
6 estimation methods in the assumptions, I think the
7 Agency did a good job with the model assumptions
8 for the DIY consumers and bystanders. They both
9 appear reasonable for both inside and outside.
10 The mileage estimates, et cetera, for 35,000 and
11 three-year replacement seemed to be consistent.

12 I think one concern with the age
13 distribution from going from age 16 up to 78
14 seemed mostly reasonable, but the upper end of
15 that probably needs to be described better. The
16 model made an assumption of an average age -- or
17 excuse me -- an average mileage over a lifetime.
18 And I think that these same Department of
19 Transportation references that the Agency used has
20 a distribution by both sex and age. And I think
21 it'd be prudent for the Agency to look at that to
22 see if by making an adjustment for an age/sex

1 distribution, if that will better describe the
2 populations than anything particular because a
3 younger user -- a younger DIY consumer is much
4 more likely to be doing that type of activity than
5 the older population. It certainly would be
6 worthwhile to explore that to make sure that that
7 is or is not the case.

8 The one area where there's a lot of
9 question and uncertainty around this -- and this
10 came up during the Question 2 piece -- was the --
11 having a good understanding of how many
12 aftermarket brakes and the UTV gaskets are
13 available. And I think where the Agency could
14 make their arguments stronger or give them more
15 confidence in the piece is giving a better
16 understanding of where -- what recent sources they
17 utilize to try to answer. In this case, I look at
18 this as they're trying to answer a negative.

19 There is certainly not widespread usage of these
20 products, but it's not clear that the imported
21 products -- how many are coming in that are
22 legitimately -- contain asbestos or whether they

1 are non-asbestos-containing products that are
2 being marketed as containing asbestos. Certainly,
3 the Agency makes that assertion, and a number of
4 the public commenters have made that assertion
5 within the public comments.

6 But then we also had the one public
7 commenter, Paustenbach, who made quite a
8 presentation on his efforts to obtain aftermarket
9 brake shoes containing asbestos over a significant
10 amount of time. And he indicated as of yet, he's
11 been unsuccessful. So there certainly appears to
12 be a disconnect one way or the other there.

13 I think what leads some credence
14 that there's very -- relatively few of these
15 products in the marketplace is because they've not
16 been used in vehicles -- automobiles for 25 to 30
17 years, in some cases even approaching 40 to 50 to
18 60 years in some vehicle lines. So that's
19 certainly something that I think the Agency could
20 do a better job of identifying what types of
21 vehicles may be using those and the relative
22 numbers that this could possibly be in there.

1 Members of the panel, certainly we've done our due
2 diligence in trying to identify whether or not
3 there are asbestos-containing brake parts or brake
4 drums that are out there. And there certainly are
5 a few websites out there that market -- that claim
6 to have these types of products.

7 And I think earlier, going back to
8 Dr. Kissel's discussion about the forms that he
9 identified, there's clearly websites. There's
10 clearly description of those products that are out
11 there. So there is either a market for these
12 products, or there's at least a perceived market
13 for those products. So again, I think it would be
14 very useful to have a little bit of definition
15 around there to assist the Agency in their piece.

16 Another piece feeding on this,
17 there's relatively few of these out there. I
18 think, one, there's certainly from a retailer,
19 either in person or an online retailer -- they
20 would have some degree of liability if they were
21 to sell these products. So that's something that
22 a company would consider. Another piece from a

1 regulatory perspective is California's Proposition
2 65 has banned asbestos -- or excuse me -- has
3 required labeling for products that contain
4 asbestos. And I did a search of the notice of
5 violation or the 60-day notification to OEHHA.
6 And the in the lifetime of Proposition 65, there
7 has been one asbestos-related NOV for brake parts
8 potentially containing asbestos. So I think that
9 from a broad perspective, the panel is comfortable
10 if the Agency takes this conservative approach.
11 But I think they should expand upon references
12 here that they have a good understanding of what
13 is actually out there or at least identify what
14 methods they've gone through.

15 Going further a little bit, a couple
16 things, obviously, I think there's a couple things
17 that needs to be updated. The USGS maintains a --
18 they do a yearly report with respect to asbestos,
19 the mineral commodity sheets. And the Agency's
20 sources or the risk evaluation cites the 2019
21 reference. But that has been updated, and I think

1 the Agency would update that and make sure that
2 that information is fully up to date.

3 And I do -- in the summary for this
4 year's report, they notice that there is a small,
5 unknown quantity of asbestos that's imported
6 within manufactured products, including brake
7 blocks for the oil industry, rubber sheets for the
8 gaskets used to create a chemical containment
9 field in the production of titanium dioxide,
10 certain other types of preformed gaskets, and some
11 vehicle friction products, which those latter two
12 would incorporate the two that could possibly make
13 it into those consumer markets. The other piece I
14 think that the Agency did a little bit better is
15 an understanding of consumer use and exposures --
16 or excuse me -- consumer patterns in the DIY
17 market with respect to utilization of compressed
18 air if that's used for cleaning of brake drums and
19 whether or not consumers are continuing to use the
20 compressed air or whether they shifted to brake
21 cleaning products of other nature -- just again, a

1 way to assist in better understanding what's out
2 there.

3 One area that gave me a little -- I
4 was a little confused about the, not lack of
5 specificity, is there's quite a discussion about
6 the UTVs and the utility vehicles. And at one
7 point, the Agency refers to a specific type of
8 utility vehicle that uses a gasket. But no
9 details are provided on what that specific type
10 would be. So I don't know whether it's a specific
11 manufacturer, specific size classification. I
12 think an expanded description would help greatly
13 and also give a better understanding of the size
14 and the scale of vehicles that would potentially
15 be utilizing those UTV gaskets.

16 And then one last piece, again, with
17 the data quality evaluation, there was some
18 question as to some of the data -- some of the
19 references were identified in the data quality
20 evaluation that were rated medium or high but were
21 not included in the draft risk evaluation. And
22 it's unclear what the connection is between the

1 data quality evaluation and going to the risk
2 evaluation. There seems to be no clear what --
3 some studies were selected, some were not. So
4 there's some ambiguity around there. And I think
5 when it gets her place, I'll let Dr. Davies talk
6 about some of her things she had identified. So I
7 believe that's all the points I have, so I'll turn
8 it over to the other members.

9 **DR. KENNETH PORTIER:** So why don't
10 we go to Dr. Davies?

11 **DR. HOLLY DAVIES:** Hi. I'm pulling
12 up my notes. I wasn't expecting Dr. Bennett to
13 throw it over to me. But looking at the studies
14 that were in the DQE, there was a lot of studies
15 in the DQE. And then there was five of them that
16 were in Table 2.25 on brakes. But they were -- in
17 the data quality evaluation, there were, I think,
18 29 studies that said they had data extracted.

19 And then 27 of those studies were in
20 the data extraction file where there was more
21 information on whether they were for brakes or the
22 age of brakes, whether they had engineering

1 controls, which gave some information for why they
2 might not have been carried forward. But it just
3 wasn't really clear why more of the studies -- we
4 had talked about one -- in your estimation of
5 personal exposure to asbestos of brake repair
6 workers from Cely-Garcia in 2016 or Cely-Garcia,
7 sorry, in 2016 why that wasn't carried forward, so
8 just looking for more explanation on which studies
9 were used.

10 **DR. KENNETH PORTIER:** Holly, are you
11 done?

12 **DR. HOLLY DAVIES:** Yes. I was --
13 you're waiting for me? Yeah. So I was done. But
14 generally, Steve did a good job of bringing up all
15 of our concerns.

16 **DR. KENNETH PORTIER:** Thank you.
17 Dr. Kaufman, anything to add?

18 **MR. ALAN KAUFMAN:** Yeah. And I
19 would echo two things. One is Steve did an
20 excellent job of collating all of our concerns
21 here. But also there is significant overlap among
22 these various questions, and so some of what I say

1 will get repeated later. I'm the lead on to 3.2
2 to 3.4, and a lot of these same issues are going
3 to be repeated there as well.

4 The one thing I would add -- Dr.
5 Bennett is absolutely correct that, as a consumer
6 gets older, they are less likely to do their own
7 work on their car. I think also there is a factor
8 -- and I didn't mention this to Dr. Bennett
9 because it only occurred to me in the last half
10 hour or so. There's also going to be a
11 socioeconomic aspect to this that generally people
12 who are not only younger but also are in
13 socioeconomic classes where their income is lower
14 would be more likely to do DIY work.

15 And I think also there is some
16 question on frequency of how often that'll be
17 done. But there's a kind of an overlay here. In
18 other words, if drum brakes are being used on new
19 cars, they're only going to be used on the least
20 expensive cars. And so that might play into the
21 fact that you're selling those to people with
22 limited income and, therefore, are more likely to

1 be doing the repair work on their own cars. But
2 again, we get back to the fact that we believe
3 that some of these parts are just not available as
4 aftermarket parts any longer.

5 In terms of frequency, I think there
6 are going to be some outliers. I mean, using the
7 average is probably a good place to start, but I
8 think there are going to be some people who maybe
9 put 50- or 100,000 miles on their car and,
10 therefore, might be changing their brake shoes
11 more often. That's really all I've got on this
12 question.

13 **DR. KENNETH PORTIER:** Thank you.
14 You mentioned DIY by age. Do you have reference -
15 - a source where we could look at that? I mean,
16 it seems to have a -- what do you call it -- face
17 value. But I wondered if we could actually have
18 some real data on that? Dr. Davies, you raised
19 your hand as well.

20 **DR. HOLLY DAVIES:** Yeah. This is on
21 a different topic with brakes, though. I just
22 wanted to expand a little bit on what Dr. Bennett

1 said about California's labeling and brakes
2 because Washington also has a labeling law for
3 asbestos products and a ban on asbestos in brakes.
4 And we have a new ban on asbestos in products,
5 generally. It just passed this session, so it's
6 not -- it's too recent for the DQE to mention it.
7 So just more support on not seeing evidence of
8 asbestos in products in Washington.

9 **MR. ALAN KAUFMAN:** Yeah. And
10 thanks, Holly. And to answer your question, Dr.
11 Portier, I think it's more -- I don't know if
12 you'd call it anecdotal. It seems to make sense
13 from a prima facie standpoint that as someone gets
14 older and is -- maybe as their income goes up,
15 they're more likely to say "Let me just have
16 somebody do that -- do a brake job for me rather
17 than spending a Sunday afternoon doing it." But I
18 don't have a reference in answer to your question.
19 It's one of those things that you sort of assume
20 is true, but I don't know that anybody's ever done
21 any particular work it. But we can certainly
22 look.

1 **DR. KENNETH PORTIER:** Thank you.

2 Dr. Kissel, anything to add?

3 **DR. JOHN KISSEL:** I just wanted to
4 say on the air compressor question, air
5 compressors are really common hardware store items
6 now, and lots of people have them. And they
7 typically come on wheels. And I don't -- so
8 they're not stationary units. And I think
9 somebody that's got one is just as likely to use
10 it in his driveway as in his garage, so I don't
11 agree with the indoor-outdoor distinction.

12 **DR. KENNETH PORTIER:** Yeah. I know.
13 Mine is in the garage, but I carry it outside to
14 do any work like this. Dr. Herrick.

15 **DR. ROBERT HERRICK:** Yeah. I
16 totally agree with John's point. That was just
17 about the only thing I really had to add here is I
18 don't think the idea that compressed air is not
19 used outside is -- it was supported by anything
20 more than just a speculation. Just one thought on
21 this discussion we just had, though, about who
22 works on their own cars. One thing I've noticed

1 as I've advanced in years, there's a lot more
2 people in my cohort who are interested in
3 collecting and preserving and showing cars that
4 were maybe more associated with their youth.

5 And they tend to be centered around
6 the brand of car, and so you see Corvette clubs
7 and Thunderbird clubs and clubs where these people
8 cherish these older cars and frequently work on
9 them themselves. So I just wonder if that isn't
10 an avenue to somehow explore to see what the
11 demographic is of people who actually work on
12 these older cars themselves.

13 **DR. KENNETH PORTIER:** All right.

14 Dr. Jayjock?

15 **DR. MICHAEL JAYJOCK:** Yes. You can
16 hear me?

17 **DR. KENNETH PORTIER:** Yep.

18 **DR. MICHAEL JAYJOCK:** Good. Okay.

19 Yeah. Just to go further with Bob's point is that
20 I think there's some of the shade tree mechanic
21 that do it more often than is assumed here, or
22 they do it with friends because of their skill set

1 and their propensity that allows them to work on
2 antique cars because it's by definition -- at
3 least in this state I think an antique of 20 years
4 old or older. So almost all of these cars with
5 drum brake will be antiques. And it's the gig
6 economy, and it's this cohort that Bob was just
7 talking about. That's it for me.

8 **DR. KENNETH PORTIER:** Thank you.

9 Dr. Markowitz?

10 **DR. STEVEN MARKOWITZ:** A couple of
11 comments really just to amplify previous comments.
12 One is, since it's unclear how many asbestos-
13 containing brakes there actually are in the U.S.,
14 I wonder whether EPA might consider purchasing a
15 sample of brakes and having them analyzed to see
16 whether and to what extent that actually occurs.
17 Even if they don't find any asbestos-containing
18 brakes, if the sample is big enough, it would give
19 them better information to try to estimate the
20 number of people at risk, which seems to be quite
21 sizeable in the draft risk evaluation. So I don't

1 know whether EPA does that kind of thing, but it
2 would probably provide some useful information.

3 And the other comment I have is
4 about a shade tree mechanic, which is it's
5 probably fairly often enough that in the draft
6 risk evaluation it might be considered as one of
7 the scenarios that EPA includes in their
8 description -- someone who does several brake jobs
9 per year and to look at the risk associated with
10 that. Thank you.

11 **DR. KENNETH PORTIER:** Yeah. And
12 didn't someone mention yesterday somewhere in
13 between the DIYer and the commercial mechanic is
14 the shade tree mechanic who does it for the family
15 and the friends and the neighborhood, and it's
16 just a little sideline source of income. But it's
17 not the DIYer who may do one every 3 years or 4
18 years. Dr. Anderson.

19 **MR. ALAN KAUFMAN:** I think -- Dr.
20 Portier, I think side hustle was the term you're
21 looking for.

22 **DR. KENNETH PORTIER:** What is it?

1 **MR. ALAN KAUFMAN:** Side hustle.

2 **DR. KENNETH PORTIER:** A side hustle.

3 Well, yeah, which for many has become the gig
4 economy, right?

5 **MR. ALAN KAUFMAN:** Yes. Exactly.

6 **DR. KENNETH PORTIER:** Dr. Anderson.

7 **MR. ALAN KAUFMAN:** I also had --
8 this is Al again. I also had my hand up. Just in
9 terms of the issue on antique cars, it would be
10 interesting -- and I don't know whether any data
11 exists on this -- but I would think that the
12 people who collect and work on those cars and
13 restore them fall into two categories: the people
14 who are buying them as an investment, in which
15 case they want to keep them as close to original
16 as possible; and those who are -- I don't want to
17 assume anything here but maybe trying to recapture
18 their youth and so might buy a car that maybe they
19 lusted after in high school. But if they're going
20 to drive it around, in many cases, conversion kits
21 are available to convert drum brakes to disk
22 brakes because they are so much better. And in

1 that cohort, you probably don't have the same
2 concern for keeping the car 100 percent original
3 or as close as you can get it.

4 **DR. KENNETH PORTIER:** Thank you.

5 Dr. Anderson, I see your hand's still up, and you
6 are muted in Webex.

7 **DR. HENRY ANDERSON:** Yeah. I just
8 have one brief thought -- is there's also what
9 used to be the old junkyard, which now is an
10 online inventory that most of the ones -- at least
11 near me here in Wisconsin, that's not a whole
12 bunch of old cars in a farm field somewhere. They
13 take them apart, inventory the parts, and you can
14 go on. And so there's got to be somebody there
15 who's taking them apart to get the wheels off and
16 do other things. And there may be a group of
17 workers there that are working for a car parts
18 place that could have some exposure by taking them
19 apart and then inventorying the parts. And the
20 last would be you can go online and look for
21 classic car clubs, and they love to talk so just
22 ask them.

1 **DR. KENNETH PORTIER:** Good point.
2 Anyone else on the Committee want to comment on
3 Question 3.1? I'm not seeing any hands go up.
4 Dr. Scarano, do you want to address the questions
5 one by one or just kind of wait until the end on
6 this?

7 **DR. LOUIS SCARANO:** Thank you, Dr.
8 Portier. I think it's best to do them one by one
9 the way you're handling it. So if that's okay, I
10 don't have any questions. I appreciate the
11 comments so far. I invite any team member to ask
12 anything.

13 **DR. KENNETH PORTIER:** Yeah. And
14 I'll ask for comments at the end of each question
15 --

16 **DR. LOUIS SCARANO:** I apologize.

17 **DR. KENNETH PORTIER:** -- and then at
18 the end of the whole section. That's fine. Let's
19 go on to Question 3.2, please. Let's go ahead and
20 read all three of these in.

21

CHARGE QUESTION 3 (3.2, 3.3, 3.4)

DR. LOUIS SCARANO: Okay. Thank you. So Question 3.2, please comment on EPA's approach to developing consumer/DIY exposure estimates for aftermarket automotive brakes and linings, which is in Section 2.3.2.1. Please include your thoughts on the reasonableness of the estimated age at start of exposure and duration and frequency of exposure for the consumer, DIY, and bystander, Section 4.2.3. Question 3.3, please comment on EPA's approach to develop bystander exposure estimates, specifically the use of reduction factors, or RFs, as described in Sections 2.3.2.1 and 2.3.2.2. And Question 3.4, please comment on EPA's approach to develop consumer/DIY exposure estimates for other gaskets and utility vehicles, UTVs, which is Section 2.3.2.2. Thank you.

DR. KENNETH PORTIER: Now, Dr. Kaufman, you kind of indicated these could have

1 all run together with your comments. Is that
2 right?

3 **MR. ALAN KAUFMAN:** That's correct.
4 It was most of the comments that I had and that I
5 received sort of overlapped the three questions.
6 So I'll go ahead and do this. And then I'm going
7 to -- I know Dr. Davies, for at least one, had
8 some additional comments that I didn't get to
9 incorporate in here, so I will try to do that.
10 But she may want to jump in after I'm done.

11 Anyway, overall, the reviewers
12 thought the methods and the explanation for
13 estimating the consumer or do-it-yourself
14 exposures in aftermarket auto brakes and linings
15 and UTV gaskets were straightforward. EPA relied
16 on peer-reviewed literature to the extent that it
17 exists, which is to some extent a little
18 surprising. I mean I think the dangers of
19 asbestos have been recognized for 80 plus years,
20 and yet in some cases there just isn't a lot of
21 data or at least a lot of peer-reviewed
22 literature.

1 As the strategies depicted in Figure
2 1-5, the accompanying text states that EPA prefers
3 using information in accordance with a hierarchy
4 of data and then modeling and then occupational
5 exposure limits or release limits, which seems
6 like a reasonable approach. One reviewer
7 suggested within the category of data that studies
8 that were done in real-world settings for purposes
9 such as establishing compliance with regulatory
10 limits be prioritized over simulations that were
11 conducted in support of litigation. There is,
12 however, considerable skepticism among the group
13 regarding the continued availability of asbestos-
14 containing automobile repair parts, particularly,
15 I said -- it's written as brake drums, but it
16 really should be brake shoes.

17 This is based on public comments as
18 well as the knowledge that asbestos-containing
19 parts would require California Proposition 65
20 warnings and also warnings in Washington. They
21 don't seem to be present -- and the fact that drum
22 brakes have become increasingly rare in new cars

1 over the past several decades. The assumptions
2 EPA makes about the age at starting and the
3 duration of exposures seem reasonable, as is the
4 approach to estimating bystander exposures, but
5 there's not complete agreement on the frequency.
6 Some felt EPA's estimates are reasonable. Others
7 felt that those underestimated exposure at younger
8 ages and overestimated them at younger ages. And
9 also, there's the distribution of mileage driven
10 per year.

11 Line 3355 states, "EPA also
12 considered the published literature on asbestos
13 exposures associated with automobile brake
14 repair." The link's to HERO ID 6322140, a
15 technical report "Draft Risk Evaluation for
16 Asbestos Systematic Review Supplemental File Data
17 Quality Evaluation of Environmental Releases and
18 Occupational Exposure." The document's identified
19 as pending, doesn't seem to be available as of
20 6/3. I didn't go back and check to see if it's
21 now available, but it'd be interesting to see how
22 the data quality evaluations turned out.

1 Specific comments on 3.2, the
2 reasonableness of the estimated age at the start
3 of exposure along with duration of exposure
4 appears to be appropriate and reasonable, while
5 possibly first -- worst case inputs per one
6 reviewer. The reduction factors used for -- on
7 3.3, the approach to developing bystander exposure
8 estimates, the reduction factors used for indoor
9 bystander exposure were databased and appear to be
10 reasonable. But it's highly variable, which would
11 be dependent on such factors as strength and
12 directionality of the indoor air movement and
13 ventilation rate. Given this variability, the
14 reduction factors chosen by the Agency for indoor
15 use would appear to be reasonable.

16 The outdoor factor of 10 would most
17 likely provide a substantial overestimation of
18 bystander exposure. It could be used as an upper-
19 bound estimate. However, a more accurate estimate
20 could come from previous work that provides a
21 means to estimate airborne concentration fall off

1 outdoors at typical air speeds. And there is a
2 reference, Shade and Jayjock, 1997.

3 And then for 3.4, "please comment on
4 EPA's approach to develop consumer do-it-yourself
5 exposure estimates for other gaskets." The
6 exposures used by the Agency -- early approaches
7 by the Agency to develop this exposure estimates
8 for UTV gaskets, it's detailed. It's well
9 explained and, again, appears to be reasonable
10 given the fact that there's not a lot of data, and
11 there is considerable uncertainty associated with
12 it.

13 Let me see if I can find real
14 quickly Dr. Davies' comments. Not finding it.
15 Rather than hold everybody up, I think I'm just
16 going to turn it over to the associates and have
17 them add what they feel is appropriate. Thanks.

18 **DR. KENNETH PORTIER:** Well, let's go
19 ahead and go to Dr. Davies first then. Holly?

20 **DR. HOLLY DAVIES:** Hi. Yes. So I
21 think my comment was just on not understanding the
22 write up quite so much on the RF. Again, as we

1 mentioned, the user RF is 10 for bystanders
2 outdoors for brake repair, rounding that up from
3 6.5 in a reference, which seems like a lot of
4 uncertainty. But that seemed like a reasonable
5 thing to do. But then on the -- later on page
6 113, they mention using RFs based on a gasket
7 scenario of 5.75.

8 And then paragraph then on page 119
9 on gasket bystanders, they don't use an RF because
10 they have sampling data in another paper. And I
11 think what confused me was that the 5.75 is
12 actually referring to the occupational exposure
13 and not the consumer exposure. So I think it's
14 just the way it's written up. I wasn't figuring
15 out what was going on for a while. So I think the
16 recommendation would be to clarify that.

17 **DR. KENNETH PORTIER:** Good.

18 **DR. HOLLY DAVIES:** And that's it.

19 I'm done.

20 **DR. KENNETH PORTIER:** Dr. Bennett,
21 did you want to add anything to these three

1 questions? If you are speaking, we are not
2 hearing you. Where did you go?

3 **DR. STEVEN BENNETT:** All right.
4 Sorry about that. I muted myself twice. Am I
5 here? Are you hearing me now? Steve Bennett.

6 **DR. KENNETH PORTIER:** Yes. Now I
7 can hear you. Yeah. Now I can hear you.

8 **MR. MARTIN ALVARADO:** Okay. Now, we
9 can hear you. Yes.

10 **DR. STEVEN BENNETT:** Sorry about
11 that. I double muted myself inadvertently. So I
12 don't have anything to add to Dr. Kaufman, so all
13 that to say I don't have anything else to say.

14 **DR. KENNETH PORTIER:** All that
15 trouble. Dr. Kissel.

16 **DR. JOHN KISSEL:** Yeah. I would
17 just like to point out that despite all of the
18 bans and whatnot, those are in the United States
19 not necessarily in the rest of the world. And
20 there are countries where asbestos-containing
21 parts are still quite normal and unremarked and
22 unlabeled. And as far as the internet goes, you

1 can buy -- there are internet sellers in China
2 that will sell you fentanyl. And the notion that
3 they would be scared of selling asbestos because
4 of liability just I think doesn't really pass the
5 laugh test. So we don't really know, but
6 certainly there's a lot of stuff being advertised
7 online, and there's certainly reason to believe
8 that people that do not have physical locations,
9 that do not have shops in the United States are
10 not impressed by Prop 65 or by liability issues.

11 **DR. KENNETH PORTIER:** Point taken.

12 Dr. Herrick?

13 **DR. ROBERT HERRICK:** Yeah. I agree
14 with the comments that have been made so far, and
15 I don't have anything to add.

16 **DR. KENNETH PORTIER:** Dr. Jayjock.

17 **DR. MICHAEL JAYJOCK:** Yeah. My
18 comments were already given. I'm fine.

19 **DR. KENNETH PORTIER:** Anyone else on
20 the Committee wish to comment on these three
21 questions? Dr. Markowitz.

1 **DR. STEVEN MARKOWITZ:** Sure. Just a
2 couple of things. I want to point out Table 2-26,
3 which is the summary table for the studies used to
4 estimate the exposure concentrations for DIY
5 activity. And it's limited to two studies, Blake
6 and Sheehy. And this goes to the comment I made
7 before, which is the narrowness of the set of
8 references -- the literature used in making some
9 of the estimates about exposure.

10 This is about DIY exposure. The
11 Blake study is of a professional auto mechanic
12 simulating in a retired auto repair facility
13 various scenarios. And Sheehy is a compilation of
14 NIOSH work to figure out how best to control
15 exposures in the brake repair setting. It's
16 unclear to what extent those control measures are
17 actually undertaken by DIY mechanics. So I think
18 that's just about all I wanted to say about that.

19 **DR. KENNETH PORTIER:** Thank you.
20 Anyone else which to comment?

21 **MR. ALAN KAUFMAN:** Hi. This is Al
22 Kaufman again. Yeah. Just one comment to follow

1 up on Dr. Kissel's comment that he was able to
2 find at least one example of allegedly asbestos-
3 containing brake shoes on the internet and agreed
4 that there are certainly a lot of direct-to-
5 consumer sellers in other countries that do not
6 really care about what the regulatory restrictions
7 are here in the U.S. On the other hand, given the
8 fact that some people believe that asbestos-
9 containing brake shoes are somehow superior to
10 non-asbestos brake shoes, it may well be that we
11 have some non-asbestos brake shoes that are being
12 advertised or being marketed as containing
13 asbestos when they, in fact, don't. So I think
14 it's a really murky area, so I think some work is
15 maybe necessary in that area.

16 **DR. KENNETH PORTIER:** Any additional
17 comments? Not seeing any I'll turn to EPA. Any
18 clarifying questions, comments?

19 **DR. LOUIS SCARANO:** Not from me.
20 Thank you Dr. Portier.

1 **DR. KENNETH PORTIER:** Okey-doke.

2 Let's move on to 3.5, 6, 7, is that the next
3 slide?

4
5 **CHARGE QUESTION 3 (3.5, 3.6, 3.7)**

6
7 **DR. LOUIS SCARANO:** Yes. Okay. So

8 continuing and Question 3.5, please comment on
9 EPA's reasonableness of the assumptions used, the
10 uncertainties they introduce, and the resulting
11 confidence in the consumer exposure estimates,
12 Section 4.3.4. Question 3.6, please comment on
13 the methods and assumptions used in approaches for
14 the sensitivity analysis for the consumer, DIY and
15 bystander, risk estimates for both aftermarket
16 automotive brakes and UTV gaskets, Appendix L.
17 And the last question, 3.7, please provide any
18 specific suggestions or recommendations for
19 alternative approaches, estimation methods,
20 assumptions, or information that should be
21 considered by the Agency for improving the
22 consumer exposure assessment.

1 **DR. KENNETH PORTIER:** Thank you.

2 Dr. Jayjock, you have the lead.

3 **DR. MICHAEL JAYJOCK:** Yes. I'm

4 basically going to be going over a lot of old
5 ground that has been mentioned before. There's a
6 lot of inter-lap here. But just to get it on the
7 record, I'm going to essentially read the
8 responses that I've gotten and integrate it into
9 this Question 3.5.

10 Overall, we found the assumptions to
11 be sound. The methods were straightforward as EPA
12 relied on peer-reviewed literature for
13 occupational exposure as a surrogate to the extent
14 that it exists for COUs. As the strategies
15 depicted in Figure 1-5 and the accompanying text
16 state that EPA preferred using information in
17 accordance with the hierarchy of data modeling
18 occupational exposure limits or release limits --
19 this is a reasonable approach.

20 We suggest that within the category
21 of data the studies that were done in real-world
22 settings for the purpose, such as establishing

1 compliance with regulatory limits, be prioritized
2 over simulations that were conducted for support
3 of litigation. The confidence ratings assigned to
4 the exposure estimates are medium or medium low,
5 and we think that's appropriate given the consumer
6 exposure estimates that were derived from
7 occupational exposure data, which is generally
8 assigned a medium confidence rating itself.

9 I'm going to be saying "we" a lot
10 here, and it's because I've integrated the
11 comments from other associate members. But I
12 haven't gotten comments from all associate
13 members. So this may change somewhat in the file.

14 It would appear that the critical
15 consumer exposure scenarios involving DIY or
16 hobbyist mechanics working with asbestos-
17 containing materials, especially brake shoes --
18 it's really kind of dominating the exposure
19 assessment. A critical point of contention is the
20 reasonableness of the assumption that any asbestos
21 is even in these automotive products in 2020.
22 Some public commenters have argued intensely --

1 and some would say compellingly -- for various
2 reasons that mostly legal liability that it has
3 actually disappeared from these products in the
4 last few decades. This may indeed be true.
5 However, the panel members in general could not
6 find any direct evidence or compelling evidence
7 that asbestos is not in these imported aftermarket
8 products.

9 One panel member, Dr. Kissel,
10 reported he was able to locate asbestos-containing
11 brake shoes from China available for sale on the
12 internet. Some evidence was found that DIY arc
13 grinding of asbestos-containing brake shoes is
14 still being done today. A 2017 YouTube video is
15 available demonstrating arc grinding of drum
16 brakes which shows the user using a belt sander
17 because they didn't have an arc grinder. And
18 there's a reference for that.

19 During the video, the narrator
20 advises not grinding them too much "especially if
21 you're working with asbestos shoes." Furthermore
22 this video has had almost 3,000 views in the three

1 years that it's been posted, which indicates a
2 fairly substantial level of internet savvy people
3 with an interest in DIY arc grinding of brake
4 shoes. It's difficult to estimate how many
5 additional DIY mechanics in the U.S. might also
6 engage in arc grinding but are not interested or
7 capable of accessing this fairly obscure video.
8 So perhaps some of the older population is not
9 internet savvy but still does this is a cohort
10 that's not represented.

11 A 2017 piece from a market research
12 group was located on the internet that asserts
13 that "Most auto manufacturers haven't installed
14 asbestos-containing brake components since the
15 1990s due to health concerns for those that
16 perform brake-related automotive repair or
17 maintenance. And yet asbestos-containing products
18 continue to present a health risk in the
19 automotive aftermarket industry in North America
20 primarily due to high sales of low-cost asbestos-
21 containing brake shoes from countries such as
22 China and India. In fact, between 1996 and 2006

1 the number of asbestos-containing imported brakes
2 have increased by 83 percent. And the low-cost
3 advantage of such imports has continued to promote
4 their sales to the current day putting automotive
5 mechanics at an increased risk of asbestos-related
6 disease." And there's a webpage that goes to that
7 reference.

8 The above 1996 to 2006 numbers for
9 86 percent increase comes from a 2006 *Baltimore*
10 *Sun* piece which further quotes the following. Bob
11 Virta of the U.S. Geological Survey, who tallies
12 data on imported minerals, said that there has
13 been a 83 percent increase in imports of asbestos
14 brakes and brake materials over the past 10 years.
15 That's 1996 to 2006, and there's a reference for
16 that. The U.S. Geological Survey and Mr. Virta
17 would appear to be reasonably credible sources.
18 However, this lead should definitely be followed
19 relative to the actual data and current situation
20 with regard to asbestos in imported automotive
21 parts. The incentive of lower cost, perhaps
22 perceived better performance, and lack of specific

1 regulation may be driving this critical potential
2 exposure to asbestos.

3 The consensus among panel members
4 responding to this issue is that at best the
5 situation is unclear and the default to reasonable
6 worst-case taken by the Agency would appear to be
7 warranted. Indeed these could represent
8 potentially many thousands of exposed consumers
9 which should be ignored or written off without
10 definitive evidence. Overall, the other
11 assumptions and uncertainties within this document
12 also appear to be quite reasonable. Some public
13 commenters advised that even if it isn't asbestos
14 brake shoes, the asbestos fibers that were
15 released during braking or from machining --
16 that's grinding, drilling, or sanding -- during
17 brake installation would be different or less or
18 even non-biologically active compared to asbestos
19 that has not been manufactured in the brake
20 material.

21 One relatively detailed and complete
22 account of this assertion is presented in a public

1 document, and this was Dr. Williams. And I give
2 the reference for that here. The full citation of
3 the reference in that public comment document is,
4 of course, available and all the references. The
5 salient excerpt below is below.

6 "Numerous studies have shown that
7 the original chrysotile asbestos in brake shoes
8 undergoes thermal degradation and dihydroxylation
9 due to heat and friction during normal use, which
10 results in even shorter chrysotile fibers that
11 have been altered -- physical and chemical
12 properties." And she presents, oh, half a dozen
13 references to that. "The drilling and grinding of
14 chrysotile have also been shown to alter the
15 surface properties and structural characteristics
16 of these fibers." And again, a number of
17 references given.

18 "Indeed, several NIOSH studies have
19 demonstrated that only a fraction, approximately
20 30 to 55 percent, of the asbestos concentrations
21 measured during brake repair work using phase
22 contrast optical microscopy methods were

1 subsequently identified as unaltered chrysotile
2 fiber. When selected area electron diffraction or
3 energy dispersion X-ray analysis methods were
4 used, these methods can differentiate between the
5 different types of -- between the different types
6 of fibers on the basis of morphological and
7 structural characteristics." And again, a number
8 of references.

9 "Noted by the EPA, transmission
10 electron microscopy methods also show that PCM
11 measurements may overestimate asbestos exposures
12 in automotive repair facilities because the
13 machining operations can release non-asbestos
14 fibers. With respect to respirable fibers Weir
15 and Meraz found that the grinding and drilling of
16 chrysotile-containing brakes release airborne
17 fibers that are bound to a resin matrix. These
18 authors conclude that -- these authors concluded
19 that their findings offer 'offer support for a
20 consideration that the process of grinding and
21 drilling do not pose an important source of this
22 exposure to respirable asbestos fibers.'"

1 And so here is my response to that
2 quote. Assuming that 30 to 55 percent of the
3 asbestos concentrations measured during brake
4 repair is "unaltered chrysotile fibers" does not
5 appear to be particularly exculpatory. The
6 current assessment is not switched by a factor of
7 two or three less exposure. This is a significant
8 amount of long, unaltered fiber.

9 Furthermore, even altered asbestos
10 chrysotile could still be biologically active.
11 Also, this fact does not appear to be in line with
12 the second highlighted text above that the
13 grinding and drilling do not pose an important
14 source of exposure to respirable asbestos fibers.
15 If they are unaltered airborne asbestos fibers as
16 identified by standard methods for machining, then
17 it would appear that almost certainly they could
18 be an important source of inhalation exposure.

19 This work does not mention the
20 sanding or arcing of brakes, which we have found -
21 - which we have seen firsthand in the above
22 YouTube done by DIY folks doing arc grinding.

1 During that clip, a considerable amount of dust
2 was visible in the back of the vertically mounted
3 belt sander. And as mentioned above, the sander
4 was wearing what appeared to be an N95 mask. All
5 of this points to the possibility that significant
6 amounts of chrysotile asbestos could -- underline
7 could -- be in the breathing zone to people
8 sanding drum brakes.

9 In summary, it may be entirely
10 possible that asbestos is all but gone in the U.S.
11 aftermarket brakes purchased and replaced by DIY
12 and small automotive shops working on antique cars
13 older than 30 to 50 years old. It may also even
14 be that, if they contain asbestos, that the
15 aerosols emitted from these operations may not
16 contain any significant -- relative to hazard
17 levels -- biologically active chrysotile.
18 However, the absence of proof of these facts is
19 not proof that they can be ignored or that we can
20 ignore the potential exposure and risk that could
21 to thousands of U.S. consumers and occupationally

1 engaged folks. In that regard, the current DRE
2 seems to do a credible job.

3 Most risk assessments need more or
4 would benefit greatly from more data. This
5 assessment seems to beg for it. The toxic potency
6 of asbestos and its airborne exposure during
7 handling has caused unacceptable levels of human
8 disease and suffering. The proper assessment and
9 the management of this substance requires
10 definitive information regarding the exposure
11 potential in order to conclude relative safety.
12 As such, one cannot simply dismiss the possibility
13 of exposure and risk based on presumptive,
14 undocumented, or incomplete information.

15 The Agency's evaluation has, like
16 all prudent assessments, traded conservatism for
17 data. That is, when the information is not
18 definitive, it has defaulted to reasonable worst
19 case. Absent conclusive state of the art data on
20 the absence of biologically active chrysotile
21 asbestos in the aftermarket brake drums, our sense

1 is the Agency's general approach and evaluation is
2 valid.

3 Recommendations will be made in
4 answer to 3.7 to provide these data. Relative to
5 the possibility of dermal exposure as a
6 significant route of exposure, one panel member,
7 Johnson, has written "On page 107 lines 3859 to
8 3860, EPA discounts the dermal exposure pathways
9 citing it will not absorb into the body through
10 protective outer skin layers. However, is this
11 not precisely the route of exposure for those who
12 have been cited using body powder containing
13 asbestos, where fibers have been reported in
14 internal tissue, ovaries?" -- and then a reference
15 to hierarch.

16 Inhalation and dermal contact
17 through peritoneal application of talc powders are
18 primary routes of exposure. That was on the page
19 234 of that reference. More logic or further
20 information is needed to support the decision to
21 discount the dermal pathway. That's all I had on
22 3.5. Shall I go on to --

1 **DR. KENNETH PORTIER:** Go on to,
2 what, 5, 6, and -- well, you have 6 and 7?

3 **DR. MICHAEL JAYJOCK:** Oh, yeah.
4 Yes.

5 **DR. KENNETH PORTIER:** Yeah. Go
6 ahead and -- go ahead and do all three of them.

7 **DR. MICHAEL JAYJOCK:** Okay. Thank
8 you. Okay. Methods and assumptions used in the
9 sensitivity analysis for consumer and bystander
10 risks appear to be well thought out and complete.
11 Assumptions used for the analysis are reasonable.
12 The effect of changing the duration of exposure
13 for users and the ages at first exposure for
14 bystanders is a useful addition to the risk
15 estimates from a sensitivity analysis.

16 To further evaluate sensitivity but
17 at the risk of being even more complicated, the
18 Agency could try using a Monte Carlo methodology
19 in which known or assumed distributions for the
20 critical drivers of exposure determinants, such as
21 age at start, age at end, airborne exposure
22 concentration, hours of exposure per incident,

1 number of incidences per year, are used and
2 combined with cancer benchmarks as distributions
3 to provide an output distribution of cancer risk
4 in which the percentage of exposed population that
5 occurs above and below the benchmark are
6 displayed. A real advantage of such a methodology
7 is the built-in sensitivity analysis that
8 indicates which variables are controlling the --
9 or contributing the most to the probabilistic
10 estimate of risk.

11 That is it can provide valuable
12 insight as to whether any specific determinant is
13 or are driving the variability or width of the
14 output distribution from either the determinant's
15 natural variation or the uncertainty born of a
16 pure lack of knowledge about it. If the latter
17 factor dominates, then research into the variable
18 can refine its input distribution and thus
19 significantly lower its contribution to the width
20 of the final distribution of risk. That is it
21 could substantially raise confidence and lower
22 uncertainty in the final analysis.

1 Okay. 3.7, during public comment on
2 June 8, a number of speakers indicated that they
3 had significant amount of monitored exposure data
4 that they or their associates had taken that was
5 not published but did show a relatively high level
6 of inhalation exposure potential. These data
7 should be considered by the Agency if the
8 methodologies appear to be valid and should be
9 considered valid as unpublished data -- it should
10 be considered as valid as unpublished data from
11 industry. Presumably, each source presents its
12 own bias, from the left done in support of
13 plaintiffs and NGOs and from the right for work
14 sponsored or done by industry.

15 As mentioned above, our response to
16 Question 3.5, this assessment requires better data
17 that is currently available in order to even
18 possibly conclude a lack of significant risk from
19 asbestos, especially in the aftermarket brake
20 replacement scenario for DIY consumers and small
21 automotive repair shops working on antique cars.
22 In this regard, definitive data could come from

1 market research and sample analysis. A primary
2 recommendation would be for the Agency, or
3 concerned and motivated stakeholders, to hire
4 independent market research groups -- group or
5 groups -- to specifically study the current state
6 of asbestos in the automotive brake pads, brake
7 shoes, and gaskets coming into the U.S.
8 aftermarket from overseas. This would require a
9 comprehensive representative and statically
10 significant sample acquisition and testing from
11 the universe of aftermarket brakes sold in the
12 U.S. consumer and workers -- to U.S. consumers and
13 workers.

14 If asbestos is found in any of the
15 samples, a representative sample should be tested
16 for asbestos content and type. Samples of brake
17 shoes in which asbestos are found should be
18 subject to machining by a belt sander or with
19 emission sampling and analysis specifically for
20 biologically active asbestos. See the above
21 recommendations for Monte Carlo analysis in lieu
22 of deterministic sensitivity.

1 Overall, this evaluation is a
2 classic case of having to provide an analysis
3 giving relatively meager and imperfect
4 information. Research has not occurred that
5 renders source rates for asbestos emissions --
6 airborne emissions of asbestos from articles
7 during exposure scenarios, thus physical modeling
8 of these scenarios is not possible. One must
9 depend on the relatively meager concentration and
10 exposure data available to render a reasonable
11 worst-case analysis. This data set should be
12 expanded, indicated above, to include data from
13 credible sources.

14 In conclusion, suppositions of a
15 lack of exposure potential, even subjective data
16 are not conclusive data. And without definitive
17 data one must choose the prudent approach of
18 reasonable worst case. The panel senses that the
19 Agency has done this relative to the exposure
20 assessment of U.S. consumers to asbestos. That's
21 it.

1 **DR. KENNETH PORTIER:** Thank you.

2 That was pretty comprehensive, but let's go
3 through the associates. Dr. Bennett, do you have
4 anything to add?

5 **DR. STEVEN BENNET:** I do not have
6 anything to add at this point.

7 **DR. KENNETH PORTIER:** Thank you.
8 Dr. Davies?

9 **DR. HOLLY DAVIES:** I also don't have
10 anything to add.

11 **DR. KENNETH PORTIER:** Dr. Kaufman?

12 **MR. ALAN KAUFMAN:** Just an
13 additional comment on one thing that was mentioned
14 regarding dermal exposure. The statement was made
15 that the -- to start out, I guess I would say
16 let's leave aside the equivocal data on the link
17 between asbestos exposure and ovarian cancer. But
18 the primary route of exposure in those cases is
19 not through the skin. It's through a mucous
20 membrane. And so I think EPA is on the right
21 track in discounting the dermal exposure, but I

1 think it just needs to be explained better than it
2 is currently.

3 **DR. KENNETH PORTIER:** Thank you.

4 Dr. Kissel?

5 **DR. JOHN KISSEL:** Yeah. I would
6 just point out we -- everything -- all of the
7 discussion is automotive. And I'm not sure how
8 people are interpreting that word, but brakes are
9 also used in motorcycles. And so people that are
10 advertising online for asbestos automobile brakes
11 are also advertising asbestos motorcycle brakes.
12 And there are -- that's another population with
13 probably a larger fraction of do-it-yourselfers
14 and probably a little more of the rebellious
15 nature and "I don't care what the rules are, I'm
16 going to do it my way" kind of attitude. So
17 motorcycle people should be included in this
18 overall analysis.

19 **DR. MICHAEL JAYJOCK:** I'll make that
20 point.

21 **DR. KENNETH PORTIER:** Let's see.

22 Who else --

1 **MR. ALAN KAUFMAN:** Yeah. I was
2 going to say -- this is Al Kaufman. That's a
3 really good comment. Also to add to that, it may
4 be likely -- and I'm not positive if this is still
5 the case because I haven't ridden a motorcycle in
6 many years -- but it may be possible that there
7 are more motorcycles with drum brakes than is true
8 in the automotive world.

9 **DR. KENNETH PORTIER:** Thank you.
10 Dr. Herrick?

11 **DR. ROBERT HERRICK:** I don't have
12 anything to add to what's been said already.

13 **DR. KENNETH PORTIER:** So on the
14 brakes issue, as Dr. Kissel mentioned
15 motorcyclists, I was thinking of farmers and
16 brakes on tractors. I don't know how often those
17 get -- but that's a very do-it-yourself
18 population. And I know kind of the average age of
19 a tractor exceeds the average age of an
20 automobile. That's for certain.

21 Dr. Jayjock, I was thinking of
22 something when you mentioned on Question 3.6 about

1 the sensitivity analysis. And first, I looked at
2 what EPA did, and I asked myself is this really a
3 sensitivity analysis? Or was it really more a
4 thread of scenarios that they looked at just to
5 get a feel for how the risk estimates vary for
6 these -- it looked like four factors -- how often
7 it's done, age at starting, whether it's done in
8 the garage or not, use of compressed air? There
9 may be one other factor there. And I thought to
10 myself it's not really a sensitivity analysis.
11 But then I thought, like you did, of Monte Carlo.

12 But, actually, I was thinking more
13 in terms of using uniform or triangular
14 distributions for a lot of these factors just to
15 get a feel for the space and a feel for which of
16 these factors is really the most sensitive. And I
17 don't get that from this analysis. I don't get
18 the feeling -- is it how often they do it or when
19 they start that has the main factor? So if I had
20 a criticism about Appendix L, it would be that
21 it's not really a sensitivity analysis.

1 And the calculations are not that
2 complicated I don't think so that a real
3 sensitivity analysis could actually have been done
4 using some of the spreadsheet add-on packages to
5 really be able to address some of this. So I
6 would encourage you to add something like that in
7 there that this maybe should be done, and the
8 focus should be about addressing true sensitivity
9 with these factors.

10 **DR. MICHAEL JAYJOCK:** Right. Right.
11 And one of the things it really forces you to do
12 is lay plain what your assumptions were. And I
13 think that's a good thing to do, and then, in
14 doing this, you can see whether you're driven by
15 just, like I say, pure uncertainty about that
16 particular variable or ignorance, if you will, or
17 whether it's natural variability of that variable.
18 So yeah. I'll beef this up a bit.

19 **DR. KENNETH PORTIER:** Yeah. I think
20 you know what I'm talking about and then --

21 **DR. MICHAEL JAYJOCK:** I do. I do.

1 DR. KENNETH PORTIER: Yeah. Any
2 additional comments? Does anyone else have a --

3 DR. HOLLY DAVIES: Dr. Portier?

4 DR. KENNETH PORTIER: Yeah. Hi.
5 Yes.

6 DR. HOLLY DAVIES: Hi. This is
7 Holly Davies. I wanted to add to the comments on
8 other vehicles and refer to the -- there's a
9 comment letter from the National Tribal Toxics
10 Council. And they talk about other vehicles,
11 snowmobiles and other kind of rural based tribes
12 and different situations there and just recommend
13 that EPA acknowledge that in some fashion within
14 the DRE. That's it.

15 DR. KENNETH PORTIER: Thank you.
16 Another good point. I'm not seeing any additional
17 comments. I'll turn it to EPA. Any clarifying
18 questions?

19 DR. LOUIS SCARANO: Thank you, Dr.
20 Portier and the Committee. I don't have any.
21 This is Gino. Do any of my team members have
22 questions?

1 **MR. KEVIN VUILLEUMIER:** Hey, Gino,
2 this is Kevin. And I don't know if we'll be able
3 to get an answer for this, but a general thought
4 is I really appreciate the comments of looking at
5 other types of vehicles. Does the panel have some
6 ideas or directions on how we might look at that?
7 Given the limited data on, for example,
8 automobile, is it even more limited data or no
9 data on motorcycles? So it's very good to
10 consider the other vehicles. Can it be done with
11 the limited data that's out there, if they have
12 any thoughts for how we might look into those.

13 **DR. KENNETH PORTIER:** Dr. Anderson's
14 hand flew up. Dr. Anderson.

15 **DR. HENRY ANDERSON:** Being the home
16 state of Harley-Davidson and being a proud owner
17 of a classic Harley, I would suggest -- I would
18 think that Harley-Davidson, who's got a great
19 program for their occupational folks, would be a
20 resource on the use of asbestos brakes. I have a
21 '59 Harley that they were kind enough to find some
22 of their squirreled away parts to share. So there

1 is a market and a resource there, and I would just
2 contact some of the U.S. based manufacturers to
3 learn more about it. They tended to be high end,
4 and the early motorcycles have been around a long
5 time.

6 **DR. KENNETH PORTIER:** Yeah. I would
7 think it doesn't even have to be U.S.
8 manufacturers because a lot of the foreign have
9 distribution centers where there should be a
10 person there who would be able to at least answer
11 the question of whether asbestos brakes have been
12 used in their vehicle and maybe when they stopped
13 using that. That's a good point. And I was
14 thinking on the farm equipment stuff, USDA keeps
15 track of those things. I seem to remember seeing
16 a distribution of age of tractors and, again,
17 people like -- companies like John Deere can
18 certainly tell you whether they even ever have
19 bothered to use that in their brakes and clutches.
20 So it just seems to me I don't think we're
21 recommending you go heavy duty into it but at

1 least kind of feel out the universe of potential
2 brake and clutch uses.

3 **DR. HENRY ANDERSON:** There's also
4 some of the equipment -- it's not unlike the oil
5 industry. In some of the foundries for lifting
6 large things, they have cranes, and I think they
7 all at one point had asbestos brakes as well.

8 **DR. KENNETH PORTIER:** Dr. Jayjock?

9 **DR. MICHAEL JAYJOCK:** Yeah. Just a
10 -- and I'm not quite sure about the resources that
11 the Agency has, but all of these seem to become
12 under the umbrella of market research, looking at
13 the Moline or the various other people who make
14 farm equipment or looking at the market vis a vis
15 the internet for brake plates. But the
16 recommendation of doing market research either by
17 the Agency or hiring a good third party to do it
18 sounds like it would just strike at the heart of
19 what's really a critical issue for this risk
20 assessment.

21 **DR. KENNETH PORTIER:** Thank you. At
22 this point we have -- I have a quarter to 3:00. I

1 want to thank Dr. Sheppard for reminding me that
2 people on the West Coast are just getting ready
3 for their lunch, and a 10-minute break is really
4 not enough for them to get their lunch. And we
5 should be aware of that.

6 So I'm going to go ahead and call a
7 break until -- I have 2:50. Let's go ahead and
8 take a break until 3:15. We're a little ahead of
9 our schedule anyway, so I think we have plenty of
10 time to complete Question 3 and begin Question 4
11 this afternoon. So we'll -- 3:15. We'll break
12 until 3:15. Thank you. Dr. Jayjock, you need to
13 mute your line.

14
15 **[BREAK]**

16
17 **DR. KENNETH PORTIER:** Okay. I have
18 3:15 if we could reconvene. It looks like we've
19 completed our discussion on Question 3. And we're
20 ready to move into the discussion on human health
21 hazard. Dr. Scarano, do you want to go ahead and
22 read in at least Question 4.1? These are some

1 major questions, so I expect we are going to need
2 quite a bit of time to discuss them.

3
4 **CHARGE QUESTION 4 (4.1)**

5
6 **DR. LOUIS SCARANO:** Okay. Thank you
7 and I will do just 4.1. Human Health Hazard
8 Question 4, EPA derived the chrysotile-based
9 inhalation unit risk based on a review of the
10 epidemiology literature describing occupational
11 cohorts exposed to commercial chrysotile that
12 provided adequate data for assessment of lung
13 cancer and mesothelioma risks. Cancer potency
14 values were either extracted from published
15 epidemiology studies or derived from the data
16 within those studies. Once the cancer potency
17 values were obtained, they were adjusted for
18 differences in air volumes between workers and
19 other populations so that those values can be
20 applied to the U.S. population as a whole in the
21 standard EPA life-table analyses. Next slide,
22 please.

1 The life-table methodology allows
2 the estimation of an exposure concentration
3 association associated with a specific extra risk
4 of cancer mortality caused by commercial
5 chrysotile asbestos. According to standard
6 practice, the lifetime unit risks for lung cancer
7 and mesothelioma were estimated separately and
8 then statistically combined to yield the cancer
9 inhalation unit risk. Less-than-lifetime or
10 partial lifetime unit risks were also derived for
11 a range of exposure scenarios based on different
12 ages of first exposure and durations of exposure.
13 Next slide, please. Question 4.1, please comment
14 on EPA's choice of focusing on only lung cancer
15 and mesothelioma.

16 **DR. KENNETH PORTIER:** Okay. Dr.
17 Taioli, you're the lead on this one.

18 **DR. EMANUELA TAIOLI:** Yes. I'm
19 here.

20 **DR. KENNETH PORTIER:** I can hear
21 you.

1 **DR. EMANUELA TAIOLI:** Okay. You
2 can? Okay. Good. So I summarized the comments
3 and then if I miss something, the others can add.
4 So there was a sense among the members of the
5 group that the choice of lung and mesothelioma was
6 too limited. And there were two issues that were
7 discussed.

8 One were the non-cancer endpoint and
9 the other cancer endpoint. For the non-cancer
10 endpoint, there were several comments. One member
11 noted that asbestosis and pleural thickening were
12 other examples of serious illness caused by
13 asbestos exposure. And there is a reference that
14 I now added to report on that.

15 And the same member noticed that the
16 primary reason for not considering non-cancer
17 endpoints was lack of a reference concentration
18 for chrysotile for this disease. But the test and
19 knowledge that if the non-cancer effects are not
20 considered, then the health risk is
21 underestimated. And the member felt like it
22 needed a little bit more discussion on the

1 reasoning why asbestosis and other non-cancer
2 endpoints were excluded and still reports a
3 reference to a study that is in the text that
4 reports non-cancer endpoints.

5 Another member noticed that there
6 are existing studies mentioned in the text of EPA
7 document that allows to add asbestosis mortality
8 to the risk evaluation and are the same studies
9 that were reviewed for lung cancer and
10 mesothelioma. There are, for reference, Hein,
11 Deng, Wang, and Loomis. They're all in the text.

12 These studies show that asbestosis
13 is a substantial contributor to mortality among
14 the chrysotile-exposed workers and should be used
15 by EPA in the review process. There are details
16 on this publication, as well as the figures, where
17 the data is available. So that will be added.

18 And the same member notes that
19 asbestosis mortality rate per fiber-year is
20 approximately one-third that of lung cancer
21 mortality rate. In counts, there were 36 excess
22 deaths due to asbestosis versus 96 excess deaths

1 due to lung cancer and two deaths ascribed to
2 mesothelioma. And the same data can be extract by
3 this other study from Wang that reports non-
4 malignant respiratory disease.

5 Now, in terms of other cancer
6 endpoints, there is discussion here on how even if
7 we consider inhalation alone, there is a systemic
8 inflammatory immune response that could elicit
9 tumor progression in other organs. And there are
10 references here and a large component related to
11 ingesting during the respiratory process, which
12 hasn't been commented at all and should be at
13 least considered in addition to lung cancer and
14 mesothelioma. Another member reported a
15 reference, again, in lung cancer has effects in
16 addition to lung and mesothelioma.

17 In general, I commented on the fact
18 that asbestos and chrysotile are used as
19 synonymous all over the document. And I feel it's
20 not appropriate because this is about -- it's not
21 about asbestos in general, and it may create

1 confusion. But this was already mentioned this
2 morning.

3 There are additional comments and
4 details here. It's not clear what the lack of
5 sufficient number of workers means at page 21, and
6 so maybe we want to discuss that, Dr. Markowitz,
7 because those were your comments. I agree that
8 data do not exist in current chrysolite asbestos
9 studies to include cancer of the ovary and larynx
10 in this EPA asbestos evaluation. This should be
11 revisited when the legacy asbestos use evaluation
12 is complete.

13 And then there is a comment about
14 the fact that the diagnosis -- about the diagnosis
15 of mesothelioma, where the member says that it's
16 almost always based on pathology review of dermal
17 tissue. And there is a reference here. So this
18 is a main summary of our comment.

19 **DR. KENNETH PORTIER:** Thank you.
20 Let's see what the associates have to say. Dr.
21 Davies.

1 **DR. HOLLY DAVIES:** I don't have
2 anything additional right now.

3 **DR. KENNETH PORTIER:** Okay. Dr.
4 Johnson. Dr. Johnson, you are muted in Webex.

5 **DR. MARK JOHNSON:** Sorry. I have
6 nothing additional either.

7 **DR. KENNETH PORTIER:** Oh, okay. Dr.
8 Kaufman. Dr. Kaufman? You are also muted in
9 Webex. Let's move on to Dr. Rowlands.

10 **DR. CRAIG ROWLANDS:** No. Dr. Taioli
11 covered everything nicely. I have nothing to add.

12 **DR. KENNETH PORTIER:** Dr. Everitt.

13 **DR. JEFFREY EVERITT:** I don't have
14 anything to add either. I'd just like to
15 reiterate I really strongly agree with the mixing
16 of asbestos and chrysotile asbestos as terms. I
17 think we have to make sure they really stay
18 separate.

19 **DR. KENNETH PORTIER:** Dr. Markowitz?
20 Dr. Markowitz, I see you as still muted in Webex.
21 There you go.

1 **DR. STEVEN MARKOWITZ:** Yeah. Yes.
2 Sorry. I don't have anything to add per se. I do
3 have a comment about the under-ascertainment
4 adjustment for mesothelioma, but I'm not sure this
5 is the right place to make that comment, so I
6 accept the advice on that issue.

7 **DR. KENNETH PORTIER:** Why don't you
8 just go ahead and make the comment now and then,
9 when we write up the reports, if this was not the
10 best place, we'll move it to the best question.

11 **DR. STEVEN MARKOWITZ:** Sure. Can
12 you hear me now?

13 **DR. KENNETH PORTIER:** Yes.

14 **DR. STEVEN MARKOWITZ:** Yeah. Okay.
15 So in the risk evaluation there was an adjustment
16 in the unit risk mortality risk it made for the
17 under-ascertainment of mesothelioma. And it was
18 based on a couple of studies. Actually, the
19 adjustment was 1.39.

20 Chief among those studies was a
21 study by Kopylev, which relied on a couple of
22 studies by Camidge and one by Pinheiro. Anyway, I

1 looked at that literature and actually most of the
2 deaths in the relevant mortality studies occurred
3 before ICD-10 and before 1998-1999. And so it's
4 really that period of time which you need to look
5 at in terms of under-ascertainment.

6 And the underlying studies,
7 Pinheiro, Camidge, and Selikoff and Seidman show,
8 really, the death certificates only before 1999
9 before ICD-10 only detected about 40 or 50 percent
10 of mesotheliomas or, that is to say, listed it as
11 a relevant ICD code, which means that the
12 adjustment factors should be higher than 1.39. It
13 should be closer to two to make up for that 40 to
14 50 percent sensitivity. There were some deaths
15 that occurred after 1999 when ICD-9 was applied,
16 and the sensitivity for mesothelioma is closer 80
17 percent. But there were relatively few deaths in
18 the mortality studies from that later time period.
19 Thank you.

20 **DR. KENNETH PORTIER:** Thank you. I
21 noticed that Dr. Kaufman sent me a note that he
22 was going to be away until 3:30. So does anyone

1 else want to comment on -- I think that's everyone
2 on the list. Right? No. Dr. Van Gosen.

3 **MR. BRADLEY VAN GOSEN:** My concerns
4 were already addressed.

5 **DR. KENNETH PORTIER:** Thank you.
6 Dr. Anderson.

7 **DR. HENRY ANDERSON:** Yeah. I just
8 want to underscore that it's not clear to me why
9 they only chose lung cancer and mesothelioma. It
10 certainly is not consistent with the approach for
11 all the other nine chemicals. And it seems the
12 argument for not including the non-asbestos -- or
13 non-carcinogenic or cancer endpoint is because,
14 well, they may have -- they may not be the driver
15 of the risk.

16 In all the other chemicals we
17 reviewed, went through immunology, went through
18 reproductive, all of those and did the assessments
19 rather than just saying, "Well, we think is going
20 to be the driver, so we'll cut to the chase and
21 just do that one." So if this is really going to
22 be a risk evaluation of whatever you want to call

1 it -- whether it's going to commercial chrysotile
2 -- it ought to be not the cancer risk assessment
3 but cover all of the others that if you want to
4 add -- these don't get done that often, so it
5 ought to be as comprehensive an assessment of what
6 are the health risks from the chrysotile, not just
7 narrow in on the cancers.

8 So I think it needs to be very clear
9 in the document the -- we're going to have to go
10 to the effort to look for all the other endpoints.
11 They just focused on inhalation earlier today. It
12 was mentioned that there may be other routes of
13 exposure that, if you're inhaling, a lot of fibers
14 are drawn up in your -- if you swallow your
15 sputum, and there's an ingestion of asbestos.

16 All of that ought to be discussed.
17 And if there is information that can build the
18 health risk on that, it really needs to be put in
19 here. Even if it doesn't end up being a driver, I
20 just think for comprehensiveness in a single-
21 source document on the risks of chrysotile or

1 commercial chrysotile asbestos really ought to all
2 be referenced and discussed in the document.

3 **DR. KENNETH PORTIER:** Thank you,
4 Henry. Dr. Barton.

5 **DR. CHARLES BARTON:** Yes. I agree
6 with Dr. Anderson. The incidences of other
7 cancers should be at least addressed in the body
8 of the document. But this has probably already
9 been pointed out, but I just wanted to emphasize
10 that what was looked at in the document was
11 mortality from lung cancer and mesothelioma. But
12 the incidences of cancer wasn't looked at. So
13 this could be grossly underestimating the real
14 incidences of tumor formation.

15 I just said if there's -- this
16 should at least be addressed as to why mortality
17 was chosen. And I imagine that was due to
18 limitation of the studies. You can't conduct
19 autopsies on people until they die. But I think
20 this should be addressed, and it would be a smooth
21 place to connect the animal data with human data.
22 That's all.

1 **DR. KENNETH PORTIER:** Yeah. This is
2 Ken Portier. I was thinking about the incidence
3 of cancers this morning and was reviewing it
4 because someone mentioned it yesterday, one of the
5 public commenters, and the DRE says or argues that
6 they don't look at incidence because the survival
7 rate is pretty -- the five-year survival rate for
8 lung cancer mesothelioma is pretty low. Although,
9 it's not as low as you would think. Lung cancer
10 male survival rate is approaching five-year, 20
11 percent, which is pretty high, one in five --

12 **DR. CHARLES BARTON:** Right.

13 **DR. KENNETH PORTIER:** --
14 mesothelioma -- hello?

15 **DR. CHARLES BARTON:** Yes.

16 **DR. KENNETH PORTIER:** Who's
17 speaking?

18 **DR. CHARLES BARTON:** Chuck.

19 **DR. KENNETH PORTIER:** Oh, oh, Chuck.
20 Yeah. I was just looking at the statistics. I
21 think they could do a better job than just saying
22 it. They really need to look at it. I don't

1 think the survival rates have quite gotten as high
2 as some people think. Some people felt that the
3 survival rate has really improved dramatically.
4 It's been improving over the last 20 years. I
5 wouldn't say it's improved dramatically for lung
6 cancer.

7 What we don't know is lung cancer
8 caused by mesothelioma? And I couldn't find any
9 good data on that. You can find SEER data on lung
10 cancer, but you can't find SEER data on lung
11 cancer that has a mesothelioma association. But
12 that may be something that the SEER program would
13 be able to look at, especially for more recent
14 data with the new ICD codes. So I'm not quite
15 sure that the DRE's justification for not looking
16 at incidence data is quite as strong as they make
17 it out to be.

18 The other thing -- the other reason
19 I think they looked at cancer hazard is they have
20 these -- what is it -- one, two, three, four,
21 five, six, seven previous hazard assessments based
22 on epidemiology data that all have ended up

1 concluding that it's lung cancer and mesothelioma
2 that are the critical endpoints that these studies
3 have chosen to base the hazard assessment on. But
4 again, it would have been a little nicer to pull
5 some of those arguments into this document to
6 strengthen that overall conclusion a little bit
7 better.

8 I think Dr. Markowitz was next and
9 then Dr. Sheela. Dr. Markowitz?

10 **DR. STEVEN MARKOWITZ:** Sure. Just a
11 couple points. One is on nomenclature. Lung
12 cancer and mesothelioma are entirely distinct
13 conditions. But the real point I wanted to make
14 was about the exclusion of cancer of the larynx
15 and ovary. I think part of the problem is that
16 the relatively narrow literature that ended up
17 being used to calculate the IUR focusing on the
18 textile studies, it wasn't sufficient to actually
19 to do a similar analysis exposure response for
20 those two outcomes. It's really, I think, the
21 broader literature -- published literature that

1 better demonstrates laryngeal cancer and ovary as
2 being caused by asbestos.

3 And so it's one of the
4 disadvantages, I think, of carving out chrysotile
5 asbestos as a separate DRE -- separate from the
6 legacy analysis -- legacy asbestos use analysis,
7 which will have to be done. In which case, a
8 broader set of studies are going to be used, and
9 the issue of cancer of the larynx and ovary can be
10 better addressed.

11 **DR. KENNETH PORTIER:** One of the
12 questions that kind of came up is where are -- you
13 get away from the two Carolina studies if you
14 broaden the -- to all cancers from chrysotile
15 cancer, do you get to include some of the mining
16 studies then begin to offer more information?
17 Just trying to understand what the benefit might
18 be of broadening that over then looking at two
19 additional cancers. Anyone? Dr. Markowitz?

20 **DR. STEVEN MARKOWITZ:** Well, there
21 are a range of other environments where there have
22 been cohort study including mining -- including

1 non-chrysotile mining where exposure response data
2 are available, perhaps not of the quality of the
3 textile studies. And those would presumably be
4 brought to bear on the legacy analysis because
5 legacy exposures clearly are going to include the
6 whole variety of -- a whole portfolio of asbestos
7 fibers.

8 **DR. KENNETH PORTIER:** Dr. Sheela.

9 **DR. SHEELA SATHYANARAYANA:** I just
10 wanted to reiterate a couple of the comments that
11 were made earlier in a little different way. I
12 was surprised at how narrowly focused the human
13 health hazard section was on lung cancer and
14 mesothelioma. And I think it needs to be expanded
15 according to all the comments that have already
16 been said.

17 But there's an implication in the
18 writing that by using mortality and these very
19 limited studies that the endpoint that they have
20 come to is protective from other health effects.
21 But there's really no evidence of that anywhere,
22 and so I really agree with Henry's point that they

1 need to discuss other health effects and even if
2 it is to say "We don't have data, or we're relying
3 on limited data." I think that there's been kind
4 of a pattern in these risk evaluations where EPA
5 cites past risk assessments and relies on those
6 risk assessments. But I don't think that that's
7 the purpose of TSCA and this process. The purpose
8 is to uniquely look at health hazards related to
9 the specific chemical at hand. And I don't feel
10 like that was really done here. I feel like it is
11 way too narrowly focused.

12 **DR. KENNETH PORTIER:** Thank you.

13 Dr. Anderson?

14 **DR. HENRY ANDERSON:** Yeah. I just
15 wanted to add my -- I agree with those statements.
16 I think what would be helpful is to also -- look,
17 I mean, they started with a number of other
18 studies in total, and then they focused on the two
19 studies. And then they focused on the health
20 outcomes that could be combined between the two
21 studies.

1 It would seem if you can't determine
2 the mortality for asbestosis or the clinical
3 occurrence of pleural plaques or things like that,
4 that could be looked at -- which studies allow you
5 to do that rather than have to combine them and
6 only have it all come from the same combination
7 databases which then have, on the exposure side,
8 this commonality of problems with them. There's
9 some Chinese studies that have been looked at on
10 chrysotile exposures that I think could provide a
11 greater richness to the information that's
12 provided in just these two specific studies. And
13 again, looking at each of those studies separately
14 for some endpoints might be a useful tool.

15 And then the other is there really
16 is no data suggesting that the asbestosis or
17 pleural plaquing is different amongst the
18 chrysotile workers or the miners than the other
19 groups, so again, to use as I mentioned here. But
20 don't use if they could just use the Libby,
21 Montana, non-cancer endpoint outcome data. But
22 there's other prevalence information that could be

1 looked at for the occurrence of disease. And if
2 they do spread out to look at other outcomes,
3 there's the studies -- at least two of them that
4 analyze the prevalence of pleural plaques in the
5 general population using the NHANES chest X-ray
6 data. And that was done at two points in time,
7 which gives you an idea of how prevalent pleural
8 plaques are.

9 You could argue, oh, "Well, they're
10 not" -- it's like not all mesotheliomas are due to
11 asbestos. But, again, these are descriptive
12 health things where certainly a dominant
13 contribution would be from asbestos or chrysotile
14 exposure in some of these cohorts. So at least
15 looking over that clinical database, I think,
16 would be a helpful tool.

17 Again, I'm just more bothered by I
18 don't get the feeling that this really was a
19 comprehensive review. It has, because of the
20 narrowness of it, sort of a rushed feel, which
21 does disservice, I think, to both EPA and to the
22 TSCA process.

1 **DR. KENNETH PORTIER:** Dr. Crump?

2 **DR. KENNY CRUMP:** I pretty much
3 agree with that everything's been said about this
4 issue. I'd like to add that, for the other types
5 of cancer, I think you could probably go to other
6 asbestos cohorts where they have looked at those
7 cancers that have data on them and just look at
8 the ratio of the cancers in those types of cancers
9 to lung cancer and mesothelioma. You could make
10 some judgements about the relative amount of risk
11 they would add. So I would suggest that, to take
12 those hazards into account, you would look at some
13 other studies of asbestos where they have
14 tabulated the data from those cancers. That's
15 all.

16 **DR. KENNETH PORTIER:** You know,
17 Kenny, I was kind of thinking the same thing. The
18 question uses the word "focus," but some of our
19 concerns are more like exclusion. So while they
20 focused on lung cancer and mesothelioma and
21 chrysotile asbestos, we know that there's a lot of
22 information on lung cancer, mesothelioma in other

1 populations that they could at least have compared
2 it to and given us a feel for is this very
3 different than what we've been seeing before?

4 I have no feeling for that because I
5 haven't gone and read the other seven hazard
6 assessments that have been done. And maybe
7 they're done very well in those other seven, but
8 it would help us to be able to bring some of that
9 forward as part of the justification for keeping
10 the focus on just these two cancers. In my
11 statistical way of thinking, this is kind of like
12 Bayesian shared information.

13 If I don't have a lot of data from
14 my sample, can I bring in information from kind of
15 associated things to help convince you that I'm at
16 least in the ballpark? And I don't think they've
17 done that here. Or they've put all the onus on
18 the reader to go and read these other reports and
19 then read their report to come to their own
20 conclusion that this is or is not appropriate.
21 Dr. Crump, your hand's still up.

1 **DR. KENNY CRUMP:** Oh. No. I'll put
2 it down.

3 **DR. KENNETH PORTIER:** Dr. Blystone.

4 **DR. KENNY CRUMP:** Figured out how.

5 **DR. SHERI BLYSTONE:** Yeah. I just -
6 - hearing the comments from several of the folks
7 here, I want to weigh in also in that EPA needs to
8 be approaching these assessments consistently
9 according to their guidance and not kind of alter
10 it every time based on what seems to be unclear.
11 At least it isn't clear to the audience. So if
12 they're going to say, "We're going to use previous
13 hazard assessments," then there should be some
14 guidance around when you can do that or when it's
15 appropriate to do that. Or otherwise I would
16 support the comments of Dr. Sheela and others that
17 you need to at least address the same way you did
18 with all the other chemicals -- other endpoints.
19 That's it.

20 **DR. KENNETH PORTIER:** Yeah. If
21 nothing else a paragraph for each one. Dr.
22 Kaufman?

1 **MR. ALAN KAUFMAN:** Thanks. I had to
2 step away for a few minutes. I sent you a note,
3 Dr. Portier. So I'm assuming we're on 4.1 still?

4 **DR. KENNETH PORTIER:** Yes. We are.

5 **MR. ALAN KAUFMAN:** Okay. Yeah. I
6 don't have anything to add. I think everybody
7 else has pretty well covered it. I think the
8 issue is when you look at other health effects
9 like asbestosis, I think if you could make a good
10 case that, if you are protecting against lung
11 cancer and mesothelioma, you are also adequately
12 protective against asbestosis, I'd be fine. But
13 that really wasn't addressed in any -- I don't
14 want to say systematic -- but with any rigor as
15 far as --

16 **DR. KENNETH PORTIER:** How about
17 strong?

18 **MR. ALAN KAUFMAN:** -- I'm sorry?

19 **DR. KENNETH PORTIER:** How about
20 strong? Any strong argument or rigorous argument.
21 That's good.

1 **MR. ALAN KAUFMAN:** Yeah. There you
2 go or robust. Yeah. Exactly. There's no robust
3 argument for why you would exclude those
4 endpoints, and so that I find troublesome. Thank
5 you. Or troubling.

6 **DR. KENNETH PORTIER:** Dr. Anderson,
7 your hand's still up.

8 **DR. HENRY ANDERSON:** Yeah. I will
9 just add to that that EPA has gone the route of
10 adding mesothelioma and lung cancer risk. You
11 could add the -- I mean, the asbestosis fatalities
12 or just the illness or pleural disease is not
13 specifically linked to lung cancer or mesothelioma
14 particularly, so it's an added risk -- that, if
15 you're going to add two cancers, why not add into
16 that the added risk of asbestosis? So I think
17 that would be the additivity I would see to it.

18 And then -- I don't know -- I just
19 tried to look through the questions here. I was
20 quite impressed with the literature that's being
21 developed on the gene and environment interaction
22 and special risk groups. And I don't see that we

1 have any charge questions on that. Am I correct?
2 Previous, we've been asked to comment on the past
3 groups.

4 **DR. KENNETH PORTIER:** I think you
5 are correct.

6 **DR. HENRY ANDERSON:** And that would
7 be the same for the cigarette smokers. I think
8 that's mentioned in the write up. There's some
9 mention of these, but it doesn't really go into
10 it. So how important are those, and are they
11 covered? Again, we've gone over a little bit the
12 factor of ten that's been used in some of the
13 other studies to account for human variability.
14 And I think both of these are probably outside
15 that factor.

16 **DR. KENNETH PORTIER:** I think this
17 is going to come up in the next question, Henry.

18 **DR. HENRY ANDERSON:** Okay. That's
19 good. I just want to be sure it gets discussed.

20 **DR. KENNETH PORTIER:** Because I
21 think in Question 4.2, we start to get into mode

1 of action and issues like that. So any additional

2 --

3 **DR. HENRY ANDERSON:** Okay.

4 **DR. KENNETH PORTIER:** Yeah. Any
5 additional comments on Question 4.1? Dr. Kaufman,
6 I see your hand's still up.

7 **MR. ALAN KAUFMAN:** Sorry. I'll take
8 it down.

9 **DR. KENNETH PORTIER:** Thank you.
10 I'll turn to EPA, Dr. Scarano?

11 **DR. LOUIS SCARANO:** Yes. Dr.
12 Portier, would you like me to read 4.2, or should
13 we comment on the discussion so far? How would
14 you like it to be?

15 **DR. KENNETH PORTIER:** Well, I just
16 wondered if you wanted to comment on the
17 discussion related to focusing. I think 4.2 is
18 going to be a more robust discussion but --

19 **DR. LOUIS SCARANO:** Yes. Okay. I
20 think some of my team members would like to weigh
21 in. So I'll leave it open and ask them to
22 identify themselves and speak up. Before they do,

1 I do want to thank the panel for the comments,
2 especially the references that you're finding and
3 will be sending us. So anyway, now I'll open it
4 up.

5 **DR. LEONID KOPYLEV:** Hi. This is
6 Leonid Kopylev. Can you hear me?

7 **DR. KENNETH PORTIER:** Yes. What was
8 your last name again?

9 **DR. LEONID KOPYLEV:** My last name is
10 Kopylev, K-O-P-Y-L-E-V.

11 **DR. KENNETH PORTIER:** Got it. Thank
12 you.

13 **DR. LEONID KOPYLEV:** Okay. So I
14 just want to clarify something on lung cancer for
15 chrysotile asbestos. I mean, in general, EPA
16 based its lung cancer number on the earliest and
17 least adverse lung cancer effect to protect for
18 the other known cancer effects. A good example of
19 that already articulated was non-cancer number for
20 Libby asbestos that was developed and was based on
21 pleural plaques incidence. So when we have the
22 data on pleural plaques, we use it.

1 However, for chrysotile there is no
2 such data available, nor on pleural changes, nor
3 on spirometry, nor on any other early effect. And
4 what that means that there is no such data, there
5 is no such dose response modeling that we could
6 use to derive, then, any non-cancer number. Only
7 dose response monitoring, that data available for
8 chrysotile is mortality, either for the sources of
9 non-malignant respiratory disease, which is way
10 different from the dose response information on
11 pleural changes for the Libby amphibole lung
12 cancer number.

13 As you probably know, mortality in
14 general is not preferred for lung cancer dose
15 response derivation. But as we explain -- and it
16 was in Slide 49 yesterday -- the assumption stated
17 there for consumers cancer risks would be
18 protective from non-cancer risks. So that's
19 there. And for the case of occupational exposure,
20 we couldn't use quantitative information, but we
21 use qualitative information to come up with

1 unreasonable risk in risk determination. You
2 could see it in the document.

3 And so I just want to ask the panel
4 to provide not -- if they're aware of non-
5 mortality dose response modeling data for things
6 like pleural plaques or diffuse pleural thickening
7 or spirometry or any such thing for chrysotile
8 asbestos, could they provide this with appropriate
9 citations? Thank you.

10 **DR. KENNETH PORTIER:** So Leonid, let
11 me ask kind of a related question. Is there any
12 indication or any information that says that those
13 conditions do not occur for chrysotile asbestos?
14 In other words, you're saying you don't have the
15 data to model it. But do we have information that
16 says that kind of exposure doesn't dissolve in
17 those endpoints?

18 **DR. LEONID KOPYLEV:** Well, I mean, I
19 don't know. All the chrysotile cohorts we
20 identify have data on mortality. We are not aware
21 of the modeling. We haven't investigated if
22 chrysotile asbestos causes spirometry. I mean,

1 there is ample data that all asbestos that people
2 studied results looking at the kinds of asbestos
3 causes decreases of spirometry similarly with
4 pleural plaques. But I don't believe any such
5 data was investigated for chrysotile asbestos
6 cohorts.

7 **DR. KENNETH PORTIER:** Yeah.

8 **DR. LEONID KOPYLEV:** So --

9 **DR. DANA LOOMIS:** Can I comment on
10 that?

11 **DR. LEONID KOPYLEV:** -- if we are
12 wrong and panel is aware of that, we would be
13 very eager to see this data.

14 **DR. KENNETH PORTIER:** So who's that?
15 Who's speaking?

16 **DR. DANA LOOMIS:** This is Dana
17 Loomis.

18 **DR. LEONID KOPYLEV:** This is Leonid
19 Kopylev.

20 **DR. KENNETH PORTIER:** Yeah. Dana.

21 **DR. DANA LOOMIS:** I'm trying to
22 comment on what was just said. Leonid is correct

1 except that there were studies done of pleural
2 plaques and monitoring of pulmonary function in
3 the North Carolina plants very early on. And
4 those were, in fact, the basis of the first TLVs
5 for asbestos. So those were based on chrysotile
6 exposures. Those data were reported in a number
7 of government reports and other documents at the
8 time. And I think the EPA has seen some of those.

9 **DR. KENNETH PORTIER:** Thank you.
10 That's Brad, right?

11 **DR. DANA LOOMIS:** Dana Loomis.

12 **DR. BRADLEY VAN GOSEN:** No.

13 **DR. KENNETH PORTIER:** Oh, Dana.
14 Okay. Dana. Yeah. Dr. Loomis, thank you. Dr.
15 Van Gosen and then Dr. Shukla. Dr. Van Gosen?

16 **MR. BRADLEY VAN GOSEN:** Yes. I'm
17 staring at a -- I made a long list that I was
18 going to bring up in 4.2, but it's a long list of
19 Chinese chrysotile factory studies that weren't
20 cited. And one of them is a study on the dose
21 response relationship between asbestos exposure
22 level and asbestosis among workers in the Chinese

1 chrysotile product factory. These are journal
2 articles too.

3 There's another one, "Cancer
4 mortality and Asbestosis Among Workers in an
5 Asbestos Plant in China." So I was rather --
6 beyond the original concern that you're only
7 talking about mortality and you're only talking
8 about cancers, I was rather surprised that it
9 seems like there is some information on chrysotile
10 and mesothelioma -- oh, yeah. Sorry -- and
11 asbestosis.

12 **DR. KENNETH PORTIER:** Thank you.
13 That's good. That means we have something to
14 begin to address that. You see, what I was trying
15 to get to the point -- we always get to this
16 point. Lack of data doesn't mean lack of effect.
17 It just means somebody didn't look at it.

18 And in this case, you're pointing
19 out to the fact that somebody has looked at it.
20 EPA hasn't looked at that data or looked at those
21 studies and judged them to be of low quality or of
22 not relevant. And we need to look and see whether

1 that's the case. Dr. Shukla, you wanted to jump
2 in on this? Dr. Shukla, you're muted in Webex.

3 **DR. ARTI SHUKLA:** Am I muted?

4 **DR. KENNETH PORTIER:** No. Now
5 you're unmuted.

6 **DR. ARTI SHUKLA:** Yeah. Okay.
7 Yeah. Sorry about that. So I was talking about
8 relationship between asbestosis and lung cancer.
9 So there are two references which come from a
10 book, chapter 1 from Lynn et al, 1983, and then
11 another one is Englert et al, 2014. In that, they
12 have discussed chrysotile-induced asbestosis.
13 It's possibly responsible for development of lung
14 neoplasm. So that's what I just wanted to mention
15 that not pleural, but at least for lung cancer it
16 seems like that fibrosis or asbestosis may be the
17 beginning point.

18 **DR. KENNETH PORTIER:** Thank you.
19 Dr. Van Gosen, your hand's still up. You have
20 more comments?

21 **MR. BRADLEY VAN GOSEN:** Sorry. I
22 keep forgetting to put my hand down.

1 **DR. KENNETH PORTIER:** That's okay.

2 Dr. Anderson?

3 **DR. HENRY ANDERSON:** Yeah. I was
4 going -- I just was trying to find it. But going
5 way back to doing the literature searches and all,
6 was "pleural plaques" one of the search phrases or
7 that it could be that there's -- I mean we've
8 mentioned some literature? I had mentioned the
9 Chinese studies. But there may be some out there
10 if they'd done their systematic review and
11 included asbestosis and pleural plaques. And
12 again, I would suggest you may search on those
13 terms.

14 When you require there also be
15 mentioned chrysotile, it may not be until you
16 actually get into the document that you find out
17 that it's a chrysotile facility. Most of these
18 will often not mention the fiber type, and you
19 have to discern that from the type of work and
20 type of product that was being looked at. But if
21 it wasn't part of the systematic review, then I
22 think EPA needs to go back and search for that and

1 not just rely on some of us on the Committee here
2 to remember a few documents out of thousands that
3 we may have read.

4 **DR. KENNETH PORTIER:** So Henry,
5 looking at the asbestos lit search and doing a
6 quick find on "pleural," there's a lot of
7 discussion, and it seems like they did look at
8 pleural plaques, pleural thickening, localized
9 pleural thickening, and -- gosh -- pleural and --
10 yeah. So pleural got -- it's in there.

11 **DR. HENRY ANDERSON:** Okay.

12 **DR. KENNETH PORTIER:** So they did
13 look at it. It may be that the Chinese --

14 **DR. HENRY ANDERSON:** My point is
15 that I couldn't. Thank you. Right. Never mind.

16 **DR. KENNETH PORTIER:** It may be that
17 the Chinese articles weren't translated at the
18 time they looked at the literature, too. Dr.
19 Crump.

20 **DR. KENNY CRUMP:** Just looking
21 ahead, supposedly, EPA decided they want to
22 incorporate these non-cancer effects. It's not

1 real clear to me how we would do that. They're
2 very different from cancer. And there are
3 different degrees of those effects and different
4 degrees of plaques or thickening. And it's not
5 that cancer is usually yes or no. And so would we
6 use some -- change the IUR?

7 I'm not sure how we would do that.
8 I'm not sure EPA has any experience doing that,
9 combining those kinds of risks -- those kinds of
10 different risks. I'm not saying not do it. I'm
11 just saying it's going to take a lot of thought
12 and a lot of planning, I think, before we figure
13 out how to do that.

14 **DR. KENNETH PORTIER:** Dr. Stayner, I
15 guess you're with EPA.

16 **DR. LESLIE STAYNER:** Yeah. I agree
17 with Kenny. I think maybe some outcomes of
18 pleural plaque is not only --

19 **DR. KENNETH PORTIER:** You're hard to
20 hear, Dr. Stayner. You need to speak up.

21 **DR. LESLIE STAYNER:** So can you hear
22 me now?

1 **DR. KENNETH PORTIER:** A little bit
2 better.

3 **DR. LESLIE STAYNER:** Okay. So I
4 agree with what Kenny just said that it's going to
5 be hard to combine some things like pleural
6 plaques. And in addition to a zero/one outcome
7 like cancer, I think most of the studies are
8 probably prevalence studies rather than long-term
9 follow up studies.

10 But one thing I just wanted to point
11 out is that, for asbestosis, we do have a good
12 dose response information from the South Carolina
13 cohort, which was in the paper that we published
14 in 1997. And that is for mortality. So I think
15 that's quite conceivable that you could combine
16 that risk with the cancer risk to get an overall
17 risk for asbestosis and for the cancer endpoints.

18 I also -- I like the other idea that
19 Kenny presented of using the proportion of
20 laryngeal and ovarian cancers as a way of coming
21 up with an estimate of dose response for those
22 outcomes -- the proportion of them to lung cancer

1 and mesothelioma. We had -- we had explored that.
2 But it does run into some small-number problems
3 because there are, in these studies, a very small
4 number of predicted ovarian cancers. That's all I
5 have, so thank you.

6 **DR. KENNETH PORTIER:** Good thing I
7 can -- something like an inflation factor?

8 **DR. LESLIE STAYNER:** Right. So if
9 there were, let's say, one-quarter of the number
10 of ovarian cancers to lung cancer -- so you could
11 take those out for lung cancer and reduce it by
12 three-quarters. It's probably the only thing we
13 can do, though, because there really is no data on
14 dose response data for ovarian or laryngeal in
15 chrysotile-exposed workers.

16 **DR. KENNETH PORTIER:** Yeah. Kenny,
17 your hand's still up.

18 **DR. KENNY CRUMP:** I'll just add one
19 thing to what -- just said. It may be -- I mean,
20 it would seem that it very well may be that
21 accounting for these other cancers might have very
22 little effect on the IUR. I think that still

1 might be worth doing just because of the
2 impression that people are getting that we're
3 underestimating risk. We should try to include
4 that in order to show how much we were or were not
5 underestimating it. But I'm sort of expecting
6 that it's not going to have a big effect on the
7 overall IUR. But that remains to be seen.

8 **DR. KENNETH PORTIER:** Exactly.

9 **DR. LESLIE STAYNER:** I think you're
10 correct. We should try it.

11 **DR. KENNETH PORTIER:** Thank you, Dr.
12 Stayner. Kenny, put your hand down. Dr. Stayner,
13 put your hand down. I think we discussed -- oh,
14 who else? What? I think we've discussed this one
15 pretty good. Turn back to Dr. Scarano, are there
16 any residual parts there that we haven't talked?
17 That was a good dialog here.

18 **DR. LOUIS SCARANO:** No. I think
19 it's fine. Thanks to the team members and our
20 epi experts, Drs. Stayner and Loomis, for chiming
21 in. So I'm ready to proceed to Question 4.2 if
22 you'd like.

1 **DR. KENNETH PORTIER:** Well, let me
2 check with the lead. I know the group with 4.2
3 have been working very hard to develop their
4 answer and focus it. I'm just not sure they're
5 ready because we're now going into tomorrow's
6 agenda. And, usually, I'm a little reluctant to
7 jump too far ahead.

8 But I do know that Dr., I guess,
9 Kanarek has been looking hard to kind of focus
10 that discussion. And we may just begin the
11 discussion in the next hour -- 50 minutes, and
12 then we can complete it in the morning if we feel
13 like we haven't covered everything. Dr. Kanarek,
14 are you ready to proceed?

15 **DR. MARTY KANAREK:** This is Dr.
16 Kanarek. I think how about we take a 10-minute
17 break, and then we start because it might -- Yeah.
18 We can start today and finish tomorrow.

19 **DR. KENNETH PORTIER:** Yeah. That's
20 what I was thinking. Dr. Markowitz, your hand's
21 up.

1 **DR. STEVEN MARKOWITZ:** Oh, I was
2 just going to suggest that there's some four or
3 five questions pending with EPA, whether they
4 wanted to answer those questions this afternoon
5 instead of postponing it until tomorrow just as an
6 alternative. That's all.

7 **DR. KENNETH PORTIER:** I'm glad you
8 reminded me of that. I had thought about that at
9 the break, and then it slipped through my mind.
10 What is the -- kind of what is the desire of the
11 panel? We can certainly take a 10-minute break
12 and come back and maybe have EPA answer a few more
13 of those questions.

14 I apologize that we didn't get the
15 questions out to the whole panel first thing this
16 morning. But I did see that Diana mailed them out
17 right after the lunch break, so you should have
18 that in your mailbox. Maybe we should spend --
19 that's a good point, Steven. Maybe we'll ask EPA
20 to answer a couple of those questions. Let's go
21 ahead and take a 10-minute break. We'll come back
22 at 4:20 Eastern Time, or 4:21, I guess.

1
2 **[BREAK]**

3
4 **DR. KENNETH PORTIER:** Okay. Let's
5 reconvene. Please check to make sure you're
6 muted. I heard some shuffling in the background
7 during the break, so somebody's line is still
8 active. I think the suggestion to go back to the
9 unanswered questions and maybe take those because
10 they really do relate to human health hazards and
11 risks -- so Dr. Scarano, Question 7 related to
12 Slide 49 would have been the next question.

13
14 **FOLLOW UP ON PREVIOUS DAY DISCUSSION-Con't**

15
16 **DR. LOUIS SCARANO:** Thank you, Dr.
17 Portier. And I believe the discussion that just
18 occurred addressed that. And Dr. Kopylev --
19 Leonid Kopylev from EPA essentially said much of
20 what we were going to say in response to that
21 question. It was all about the non-cancer effect
22 and the need to address it in what we said in

1 Slide 49. So I think we're okay there. But I
2 would also see if the panel is happy with that.

3 **DR. KENNETH PORTIER:** Well, I was
4 wondering if you could summarize kind of what came
5 out of that discussion. I mean, he was talking
6 about lack of data, lack of models. And I'm just
7 -- I think it would help us if you kind of at
8 least answered the last question about whether
9 EPA's thinking on this is changing or not. You
10 are interested -- I guess what I'm hearing is you
11 are interested in including non-malignant lung
12 disease but in situations where you have some
13 data. Is that right?

14 **DR. LOUIS SCARANO:** I think that's
15 accurate. I'll let Leonid expound if he'd like.
16 But I am comfortable with that. Leonid, I don't
17 know if you're on?

18 **DR. LEONID KOPYLEV:** Yes. I'm on.
19 I think we -- yes, we need to come up with a
20 number -- we need data. So just saying that there
21 are those hazards are not really helpful. I mean,
22 yes, they could be discussed. But what is

1 important for the assessment are numbers. And for
2 the numbers, we need dose response data. And by
3 the way, to Dr. Van Gosen, we're familiar with
4 this Chinese study, but it's asbestosis and
5 mortality from asbestosis. I mean, mortality
6 isn't preferred for deriving reference
7 concentration. So -- yeah. That's all.

8 **DR. KENNETH PORTIER:** Does anyone on
9 the panel want to follow up? Dr. Taioli. You're
10 muted in Webex.

11 **DR. EMANUELA TAIOLI:** Sorry. Yes.
12 So I suggest to use two steps. One is to
13 acknowledge whether there could be a risk or not
14 for those other endpoints and then to discuss
15 whether there are results or not and then if the
16 results are lacking. The way it's written now, it
17 really looks like there is only a possible
18 association with lung cancer and mesothelioma, and
19 everything else is fine. And that's not really a
20 reflection of reality.

21 **DR. LEONID KOPYLEV:** That's actually
22 not how the document treats. For example, other

1 cancers we acknowledge them, ovarian cancer and
2 laryngeal cancer, and we talk about this as
3 uncertainty that we accounted for by taking the
4 largest IUR from the range 0.08 to 0.16. And we
5 chose 0.16 -- and not saying we doubled this range
6 to account for them for these two cancers. So to
7 say that we don't consider them is not exactly
8 correct if we look what we have done in the
9 document.

10 **DR. EMANUELA TAIOLI:** I'm talking
11 about the non-cancer endpoints. I was not talking
12 about the cancers. I think the cancers should be
13 discussed more in general as a systemic problem of
14 chronic inflammation in this population of the
15 immunity and other areas where the encounter
16 happens -- ingestion, dermal contact. But for the
17 non-cancer endpoint, there should be some
18 discussion about those.

19 **DR. LEONID KOPYLEV:** Agreed. Thank
20 you.

21 **DR. KENNETH PORTIER:** Yeah. I'm
22 kind of looking. It's almost like we need a

1 3.2.3.2 section talking about non-cancer in the
2 hazard discussion. Dr. Anderson.

3 **DR. HENRY ANDERSON:** Yeah. I just
4 wanted to say I thought Dr. Stayner said there was
5 asbestosis death information in at least one of
6 the cohorts that are included? So that seems to
7 be low-hanging fruit to take a look at for non-
8 cancer endpoints.

9 **DR. KENNETH PORTIER:** Dr. Bateson,
10 you have your hand up. Your phone might be muted.

11 **DR. THOMAS BATESON:** Can you hear me
12 now?

13 **DR. KENNETH PORTIER:** Yes.

14 **DR. THOMAS BATESON:** Great. So hi,
15 this is Tom Bateson, EPA. So we definitely take
16 the panel's point that we need to increase the
17 transparency talking about the other cancers and
18 non-cancer health effects. And I think we've
19 heard a few ideas on how we might estimate some
20 risks for those or methods where we could say,
21 "Well, what if the ratio of these other cancers to
22 the lung and mesothelioma could be used? What

1 number would we get so that we could feel more
2 comfortable that we're not missing those risks?"

3 But as Leonid said, we did
4 specifically acknowledge those other cancers and
5 that the risk that we derived didn't naturally
6 include those. And so by selecting the value of
7 0.16 to be the chrysotile inhalation unit risk, we
8 attempted to cover for the risks of laryngeal and
9 ovarian cancers as well as the issue about
10 mortality and the incidence. That's all.

11 **DR. KENNETH PORTIER:** Yeah. Thank
12 you. Good point. Dr. Markowitz?

13 **DR. STEVEN MARKOWITZ:** I just wanted
14 to clarify something because I may have misheard.
15 The issue of pleural disease caused by asbestos is
16 not really an issue of mortality, except the Libby
17 amphibole. So when we discuss mortality from non-
18 malignant asbestos-related disease, we're really
19 referring to asbestosis, which is scarring of the
20 lung tissue itself -- the lung parenchyma and not
21 to pleural disease. The issue of pleural disease,
22 outside of Libby, it may rarely cause death, but

1 it's really a cause of morbidity or a health
2 impact while a person's alive but not really an
3 issue for mortality. That's all.

4 **DR. KENNETH PORTIER:** Thank you.

5 Dr. Taioli, your hand's still up. Your phone may
6 be muted. No. You're muted now on Webex.

7 **DR. EMANUELA TAIOLI:** Sorry. I'm
8 just trying to figure out how to get my hands
9 down.

10 **DR. KENNETH PORTIER:** Oh. It's in
11 the bottom right-hand corner of the participant's
12 box.

13 **DR. EMANUELA TAIOLI:** Got it.

14 **DR. KENNETH PORTIER:** You see the
15 little hand there?

16 **DR. EMANUELA TAIOLI:** Yeah.

17 **DR. KENNETH PORTIER:** Got it.

18 **DR. EMANUELA TAIOLI:** Got it, thank
19 you.

20 **DR. KENNETH PORTIER:** Okay. Why
21 don't we go on to -- what is it -- Question 9, the
22 comment by Dr. Davies?

1 **DR. LOUIS SCARANO:** Thank you. Yes.
2 Do you want me to read the question or --

3 **DR. KENNETH PORTIER:** Yeah. Why
4 don't we read the question? Thank you.

5 **DR. LOUIS SCARANO:** Okay. I'm sorry
6 we didn't read the other one, but it was what we
7 were just talking about. So Number 9, 10, and 12
8 are very similar, actually. So I'll read Number 9
9 first from Dr. Davies.

10 "The exposure to asbestos as a
11 contaminant of talc raises the question of whether
12 it is the association of asbestos with talc, the
13 size of the asbestos, or the area exposed --
14 vaginal tissue -- that allows the asbestos to
15 enter the body. While some consumer uses -- brake
16 grinding, taconite mining, living close to or
17 living with taconite mining -- whole body
18 exposures to dust containing asbestos might
19 represent a scenario similar to dusting the body
20 with talc in which case this would impact how
21 dermal exposures are addressed." And Number 10

1 and Number 12 -- Number 11 we had already
2 addressed -- also bring up the issue of legacy.

3 We were going to have an internal
4 discussion tomorrow morning. But I think it's
5 important for -- at least I'd like to say one
6 thing that for all these questions that have to do
7 with mining, talc, legacy, intentional versus non-
8 intentional inclusion of asbestos, they're the
9 subject of the scoping exercise for the legacy
10 asbestos assessment. We understand that with the
11 Ninth Court's decision, I think, and the panel is
12 interested in talking about its legacy in this
13 particular risk evaluation. But this is going to
14 be the focus of the next one. So I just wanted to
15 say that.

16 And I do want to address the dermal
17 exposure a little bit simply because we did
18 evaluate it in the problem formulation, and it
19 wasn't the talc. And I guess that the exposure --
20 the site and exposure scenario is very different.
21 And I think it was Dr. Kaufman that mentioned the

1 mucous membrane versus dermal, and I agree with
2 that.

3 But in terms of dermal exposure to
4 the skin, which is what we're looking at for the
5 conditions of use here, we did find some data,
6 early data, and the only effects that we were
7 aware of were warts and corn formation on the
8 hands of workers that were installing amosite
9 installation in ships. Couldn't find any
10 chrysotile-specific ones. And we did talk about
11 that in the problem formulations. So in terms of
12 skin as the barrier in dermal exposure that way
13 and chrysotile exposure information, we made the
14 decision that it was the dermal exposure pathway
15 was not a concern and didn't need to be included.

16 **DR. KENNETH PORTIER:** So Question 10
17 is a little bit different, though. You should go
18 ahead and read 10, then, and see if there's
19 something --

20 **DR. LOUIS SCARANO:** Sure. "Where
21 will contaminated products, as talc, be considered
22 if they are not in this draft risk evaluation?"

1 Asbestos has been found in products like crayons,
2 so it's not just cosmetics regulated by the Food
3 and Drug Administration. Linda Reinstein from the
4 Asbestos Disease Awareness Organization mentioned
5 crayons, toys, and paints."

6 And I did refer to it in my generic
7 statement, but I think this is an example, which
8 is a big issue. And we understand that it's the
9 concept of intentional versus non-intentional and
10 the level of asbestos that might be found in the
11 crayons, toys, and the paints -- and the fact that
12 it's not intentionally added, and it may be a
13 small amount and as an impurity. And so these
14 things were addressed in some of our -- in the
15 scope and problem formulation. And we understand
16 that's at the center of these discussions about
17 legacy and what that might mean. But now that the
18 decision has been made to go in the direction of
19 developing a separate legacy risk evaluation, it
20 is going to be an important part of scoping for
21 that particular and separate scope and risk
22 evaluation.

1 **DR. KENNETH PORTIER:** And this is
2 Ken Portier --

3 **DR. LOUIS SCARANO:** And I can read
4 Number 12, too, if you'd like, or we can just --

5 **DR. KENNETH PORTIER:** Yeah. Go
6 ahead and read it in because I think there's
7 another aspect of that that I'd like you to
8 discuss.

9 **DR. LOUIS SCARANO:** Thank you.
10 Okay. So in Number 12, which was I think the last
11 of the questions that were sent around, why is EPA
12 not considering legacy exposures and risks in this
13 draft risk evaluation? Consideration of legacy
14 uses and exposures and risks seems to be required
15 given the recent lawsuit. Saying that legacy uses
16 will be considered in a later supplement is not
17 the reason for putting off these issues.

18 **DR. KENNETH PORTIER:** This is Ken.
19 I think one of the issues that's come up in some
20 of the preparatory discussion that the Committee
21 has had is this whole idea that TSCA looks for
22 aggregate exposure assessments. And it's hard to

1 think about -- even focused on chrysotile asbestos
2 exposures moving forward, which seems to be the
3 focus of this DRE -- the legacy exposures are part
4 of the lifetime exposure that's going to drive
5 what we see moving forward.

6 And we're having a hard time seeing
7 how the legacy exposure assessment is going to
8 integrate with this assessment to address those
9 kinds of aggregate exposure or cumulative
10 exposure, whatever it is -- aggregate and
11 cumulative exposures to asbestos. And I think
12 part of that was part of the justification here
13 for the question.

14 **DR. LOUIS SCARANO:** This is Gino.
15 And I'll invite other people to chime in, but I
16 think that's a good comment. And I understand. I
17 don't --

18 **DR. KENNETH PORTIER:** Usually, this
19 is when Dr. Barone comes in, and says we
20 understand what you're talking about. We'll take
21 it under consideration.

1 **DR. LOUIS SCARANO:** You need another
2 Italian? You need another Italian?

3 **DR. KENNETH PORTIER:** Yeah. Dr.
4 Crump, your hand's up.

5 **DR. KENNY CRUMP:** I think my
6 question has been answered. But I may just say --
7 let me just say what I think I heard. Somebody
8 can correct me if I'm wrong. Talc is not being --
9 asbestos in talc is not being assessed in this
10 report because it's going to be assessed later in
11 the legacy when they consider legacy exposures.
12 Is my understanding correct?

13 **DR. KENNETH PORTIER:** I didn't quite
14 hear that, Kenny, because again, there's the issue
15 that these are non-uses of asbestos. They're
16 exposures, but they're not -- they're low-level
17 contaminant exposures. And I don't think that's
18 going to be in a legacy exposure assessment
19 either. It's something else entirely. Let me let
20 Dr. Barone jump in here.

21 **DR. STAN BARONE:** So this is Stan --

1 **DR. KENNY CRUMP:** Okay. Well, that
2 -- so --

3 **DR. STAN BARONE:** Yeah. I do want
4 to -- I do want to reinforce and amplify what Gino
5 was saying. We haven't decided. We'll be looking
6 at this. We'll be looking at trying to determine
7 what are the conditions of use that are legacy
8 uses, regardless of intentional or unintentional
9 inclusion in products.

10 So those are some difficult
11 conversations post the Ninth Circuit's decision.
12 Those are important conversations that we have to
13 have internally, and we'll put out a scope for
14 public comment. And they include looking at what
15 the levels of exposure are and what the
16 consequences of those exposures are.

17 **DR. KENNETH PORTIER:** Dr. Sheela,
18 this was your question. Do you want to follow up?

19 **DR. SHEELA SATHYANARAYANA:** Yeah. I
20 guess from yesterday I still didn't hear a clear
21 explanation of why EPA felt like they needed to
22 focus on commercial chrysotile, specifically, for

1 this evaluation and not incorporate the legacy
2 uses. I just didn't hear a good enough answer to
3 that. That's one piece. The other is that if EPA
4 -- I mean, I think that the biggest concern here
5 is that the risk evaluation could be used and
6 interpreted incorrectly by end-user stakeholders
7 given the language that's in it right now. If EPA
8 was really focused on commercial chrysotile only,
9 then I don't think the language would be so loose
10 on asbestos. There's a lot of reference suggest
11 asbestos and not specifically these fibers.

12 And we've talked a lot about
13 changing that language, but I'm just confused why
14 it wasn't discussed previously or what the
15 thinking behind doing just the chrysotile was.
16 And I think that the legacy use is something that
17 we're all very concerned about because of the --

18 **DR. KENNETH PORTIER:** Dr. Scarano.

19 **DR. STAN BARONE:** This is Stan
20 Barone again. I'm going to try to assess and
21 clarify. So again, there have been developments
22 over the course of the last nine months with the

1 Ninth Circuit decision which affects the actual
2 risk evaluation that was already in the draft
3 stage. And the draft in our approach in the first
4 ten risk evaluations were to focus on ongoing
5 uses, not legacy uses.

6 And that was a distinction that was
7 made in the post-problem formulation phase as we
8 looked at -- particularly looked at asbestos and
9 the uses, what the ongoing conditions of use were.
10 So it's part and parcel of how we're going to
11 develop a new scope, a revised scope. But we've
12 already done the risk evaluation for the
13 conditions of use that focus on the ongoing uses.
14 And that's what we're presenting to you for peer
15 review. We intend to follow the other uses with
16 an additional scoping exercise and public comment.

17 **DR. KENNETH PORTIER:** Thank you.

18 Dr. Blystone.

19 **DR. SHERI BLYSTONE:** Yeah. A couple
20 of things here. First, I think that while I
21 understand what Dr. Barone and Dr. Scarano are
22 saying about legacy uses and talc and

1 unintentional, there's not a mention of talc in
2 the DRE. I think that needs to be added
3 explicitly, given the focus on that right now.
4 Similar to how you talk about legacy uses going to
5 be analyzed later, you need to say something about
6 that about talc as well.

7 I also want to point out that from
8 the impurity perspective or asbestos unintentional
9 low-level impurities in a broad sense is related
10 to our discussion on 1,4-dioxane where EPA already
11 made a decision to not include those types of uses
12 in the risk evaluation. So again, going back to
13 trying to make sure EPA is consistent in how
14 they're doing that, I would just recommend that
15 you think about that. That's it.

16 **DR. KENNETH PORTIER:** Thank you.

17 Dr. Markowitz?

18 **DR. STEVEN MARKOWITZ:** So I think
19 some new nomenclature might be needed because
20 current use of industrial talc is significant, and
21 it's not under legacy use. It's not a current
22 intentional use. But it is a real use, and so if

1 it is going to be included in the next risk
2 evaluation, there has to be some clarity around
3 that. And that same comment would apply to
4 consumer products containing asbestos even if the
5 exposure in relation to that is less clear. The
6 legacy doesn't really capture what the current DRE
7 has missed.

8 **DR. KENNETH PORTIER:** Thank you.

9 Dr. Everitt?

10 **DR. JEFFREY EVERITT:** Yes. I had a
11 question on the talc. My understanding has always
12 been that it's amphibole contamination of the talc
13 and not chrysotile contamination in the talc
14 deposits going forward. Is that not the case?
15 Because aren't we restricting ourselves here to
16 commercial chrysotile to go forward?

17 **DR. KENNETH PORTIER:** I think this
18 question came up in the broader issue of a focus,
19 like Question 4.1, how this has been focused down.
20 And so I'm not sure. I think you are right that
21 the majority is the amphibole --

1 **DR. JEFFREY EVERITT:** I'm not a
2 mineralogist. But I was under the understanding
3 that it's mainly tremolite and anthophyllite in
4 talc deposits not chrysotile.

5 **MR. BRADLEY VAN GOSEN:** This is Brad
6 Van Gosen. We're in my wheelhouse now. First of
7 all when you get to talc, regardless if it's
8 sooner or later, you need to coordinate with the
9 FDA who has already spent a few years focusing on
10 talc and not just cosmetics. But it's become the
11 cosmetics talc issue. But the litigation and some
12 of the arguments about talc in cosmetics,
13 including baby powder, is chrysotile is being
14 found also in addition with tremolite or
15 anthophyllite. So there is chrysotile that's been
16 confirmed in some of the cosmetics talc.

17 **DR. KENNETH PORTIER:** Thank you, Dr.
18 Van Gosen. That's helpful.

19 **MR. BRADLEY VAN GOSEN:** And if I
20 could add, the more I look at the title I think
21 part of what's bothering people is it's a draft
22 risk evaluation of asbestos. And it gives the

1 impression that all asbestos in all forms is being
2 addressed. And it's really the current uses of
3 asbestos in products that you're addressing right
4 now. That's just a thought.

5 **DR. KENNETH PORTIER:** Well, that's a
6 recommendation I expect to make in the last
7 question when we look at the general -- because I
8 have the same feeling that the title doesn't match
9 the evaluation and it confuses you because it sets
10 up an expectation that's not there. Dr. Anderson.
11 Henry, you're muted in Webex.

12 **DR. HENRY ANDERSON:** Okay. One of
13 the issues I see here is, again, when I ask the
14 question about groups that are at high risk, one
15 way to address the legacy issue would be to say
16 are individuals who had legacy exposures -- and
17 this would be especially true for the do-it-
18 yourselfers -- if you were a long-time insulator,
19 you'd have pretty substantial risk that you bring
20 to when you go into doing your own brakes.
21 Somehow, that group of people we have to
22 accommodate the -- or we could accommodate the

1 legacy issues as part of the high-risk --
2 especially high-risk group for adding in the
3 additional chrysotile exposure. Because a lot of
4 those legacy uses, those people were exposed to
5 not just the amphibole. They also had a lot of
6 chrysotile exposure as well.

7 So it's hard to deal with just kind
8 of it's the dog -- the tail wagging the dog here.
9 That most of the exposure is not being covered.
10 But that exposure is out there, and there are some
11 groups in the population that have considerable
12 exposure in addition to what they're getting from
13 this chrysotile issue.

14 **DR. KENNETH PORTIER:** So Henry,
15 you're seeing this as part of the PESS discussion,
16 the potentially exposed susceptible subpopulation?

17 **DR. HENRY ANDERSON:** Oh, well, I'm -
18 - that's one place they could fit. And I'm just
19 trying to -- it's clear to me that we're not going
20 to get a unified human health hazard to asbestos
21 document, which is really what I think is the best
22 fit is to put it all into one document that brings

1 up the previous IRIS assessment and all those and
2 kind of unifies the approach. Sounds to me like
3 that's not going to happen.

4 So it's somehow we have to recognize
5 the legacy exposures as being significant,
6 important. And we're going to have to wait three
7 to five years to get that addressed. So one way
8 to address it would be through this particular
9 document referencing it and trying to
10 characterize, at least in a general sense, the
11 significance of that exposure to a certain segment
12 of the current population. That may be totally
13 impossible to do, but that's one way I think would
14 begin to address some of the concerns that we're
15 really -- this document ignores a whole lot of at-
16 risk people and ongoing exposures in addition to
17 what is a relatively small number of COUs here.

18 **DR. KENNETH PORTIER:** It's an
19 interesting thought. I think we're going to have
20 to sleep on that one. Dr. Markowitz, I see your
21 hand's still up.

1 **DR. STEVEN MARKOWITZ:** Yeah. Just
2 briefly, a friendly amendment to renaming the
3 current effort which, really, it's not just the
4 current uses of chrysotile. It's the current
5 intentional uses of chrysotile. That's what this
6 document's about.

7 **DR. KENNETH PORTIER:** I think that
8 answered the unanswered questions from Day 1.
9 We've covered all the material we expected to
10 cover on Day 2 here. And I think it's a little
11 late in the day to start a very important
12 question, which is Question 4.2, which I know is
13 going to take some time to discuss. But it also
14 might be best for us to take this up first thing
15 in the morning when we're relatively fresh.

16 I want to thank EPA for taking the
17 time to answer our questions. This is the first
18 time that the Committee has kind of written up its
19 questions on the presentation and presented it
20 back to EPA and said, "Would you please take the
21 time and answer these for us?" And Dr. Scarano,
22 thank you and thank you for your team for getting

1 that information back to us. It may not have been
2 the answer we were hoping for, but at least now we
3 have an answer. And we can move forward with the
4 discussion.

5 So Dr. Kanarek, you're going to have
6 to wait until tomorrow to begin the discussion.
7 And we'll start that first thing tomorrow at,
8 whatever it is, 10:15, once we run through the
9 Committee attendance. Unless there is additional
10 comments or questions by the Committee, it's my
11 intention to end today's discussion and turn it
12 back over to Diana Wong for any final comments.
13 Any additional comments from the panel, the
14 Committee? Dr. Wong, any final comments?

15 **DR. DIANA WONG:** Yes. Yeah. This
16 is Dr. Diana Wong. As the DFO, I would like to
17 thank you to the SACC peer reviewers and the
18 public listening online. And this concludes the
19 peer review activities for the agenda for today.
20 And we will reconvene tomorrow morning for Day 3
21 at 10:00 a.m. Eastern Time. So this day's session
22 is now adjourned.

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DR. KENNETH PORTIER: Thank you,
everyone. Goodbye.

[MEETING ADJOURNED FOR THE DAY]

OPENING OF MEETING - DAY 3

MS. SARA WILSON: Hello, everyone.

We have about two minutes until the meeting will begin. We'll let a few more people log in and then start the third day of our -- of this risk assessment meeting. Thank you.

Good morning. Welcome to this EPA peer review of the draft risk evaluation for asbestos. Battelle is an EPA contractor providing meeting support for this hearing. This event is being recorded. Please be aware that the host may use WebEx chat to share announcements with all attendees, but all attendees will not be able to respond to the chat. I will now introduce Dr. Diana Wong, the designated federal official.

DR. DIANA WONG: Thank you. Good morning. I am Dr. Diana Wong and the designated federal officer. It is my pleasure to open the third day of the four-day meeting for the Science Advisory Committee on Chemicals, TSCA SACC, peer review of EPA's draft risk evaluation for asbestos. Monday and yesterday's Webex went well.

1 However, if you encounter any problems with audio
2 or video transmission today, please go to
3 www.epa.gov/tsca-peer-review.

4 A reminder to the peer reviewers, if
5 you have to step out from the meeting for a time,
6 please send a note to myself and the chair and
7 send another note when you're back. In a minute,
8 we will do our check-in roll call and then start
9 today's meeting. I now turn the meeting over to
10 our chair, Dr. Ken Portier.

11 **DR. KENNETH PORTIER:** Thank you, Dr.
12 Wong. Good morning, everyone. We're going to
13 begin our meeting this morning with the roll call
14 and try to get through that pretty quick. Dr.
15 Anderson?

16 **DR. HENRY ANDERSON:** Present.

17 **DR. KENNETH PORTIER:** Dr. Barton?

18 **DR. CHARLES BARTON:** Here.

19 **DR. KENNETH PORTIER:** Dr. Bennett?

20 **DR. STEVEN BENNETT:** I am here.

21 **DR. KENNETH PORTIER:** Dr. Blystone?

22 **DR. SHERI BLYSTONE:** I am here.

1 DR. KENNETH PORTIER: Dr. Cory-
2 Slechta?

3 DR. DEBORAH CORY-SLECHTA: Hello,
4 I'm here.

5 DR. KENNETH PORTIER: There you are.

6 DR. DEBORAH CORY-SLECHTA: I'm here.
7 Can you -- okay.

8 DR. KENNETH PORTIER: Yes, I can
9 hear you. Thank you, Deborah.

10 DR. DEBORAH CORY-SLECHTA: Thanks.

11 DR. KENNETH PORTIER: Holly Davies,
12 Dr. Davies.

13 DR. HOLLY DAVIES: Good morning.
14 I'm here.

15 DR. KENNETH PORTIER: Dr. Doucette?

16 DR. WILLIAM DOUCETTE: Good morning.
17 Present.

18 DR. KENNETH PORTIER: Dr. Jimenez-
19 Gonzalez sends her regrets. She hopes to join us
20 at 3:00 this afternoon or 3:30. Dr. Johnson?

21 DR. MARK JOHNSON: Good morning.

22 DR. KENNETH PORTIER: Dr. Kaufman?

1 **MR. ALAN KAUFMAN:** I am here, and
2 somebody's typing.

3 **DR. KENNETH PORTIER:** I can hear
4 that. Dr. Kissel?

5 **DR. JOHN KISSEL:** Here.

6 **DR. KENNETH PORTIER:** Dr. Rowlands?

7 **DR. CRAIG ROWLANDS:** I'm here. Good
8 morning.

9 **DR. KENNETH PORTIER:** Good morning.
10 Dr. Schlenk?

11 **DR. DANIEL SCHLENK:** Here.

12 **DR. KENNETH PORTIER:** Dr. Sheela?

13 **DR. SHEELA SATHYANARAYANA:** I'm
14 here. Did you hear me?

15 **DR. KENNETH PORTIER:** Yup. Now I
16 hear you. Good morning.

17 **DR. SHEELA SATHYANARAYANA:** Okay.
18 I'm here.

19 **DR. KENNETH PORTIER:** Dr. Crump?

20 **DR. KENNY CRUMP:** Here.

21 **DR. KENNETH PORTIER:** Dr. Everitt?

22 **DR. JEFFREY EVERITT:** Here. Here.

1 DR. KENNETH PORTIER: Thank you.

2 Dr. Herrick?

3 DR. ROBERT HERRICK: Here.

4 DR. KENNETH PORTIER: Thank you.

5 Dr. Jayjock?

6 DR. MICHAEL JAYJOCK: Here.

7 DR. KENNETH PORTIER: Dr. Kanarek?

8 DR. MARTY KANAREK: Here.

9 DR. KENNETH PORTIER: Dr. Markowitz?

10 DR. STEVEN MARKOWITZ: I'm here.

11 DR. KENNETH PORTIER: Thank you.

12 Dr. Sheppard?

13 DR. ELIZABETH SHEPPARD: I'm here.

14 Good morning.

15 DR. KENNETH PORTIER: Good early
16 morning. Dr. Shukla?

17 DR. ARTI SHUKLA: I'm here.

18 DR. KENNETH PORTIER: Dr. Taioli?

19 DR. EMANUELA TAIOLI: I'm here.

20 Present.

21 DR. KENNETH PORTIER: Thank you.

22 Dr. Van Gosen?

1 **MR. BRADLEY VAN GOSEN:** Good
2 morning.

3
4 **FOLLOW-UP ON PREVIOUS DAY DISCUSSIONS**

5 **DR. KENNETH PORTIER:** Good morning.
6
7 Everyone's here. Today, we're going to continue
8 the discussion on human health hazard and human
9 health risk. And I wanted to start off the
10 morning -- I know that a couple of emails back and
11 forth yesterday evening related to some
12 information we wanted to make sure was stated at
13 the meeting related to Question 2.1 and 2.3. And
14 I think, Dr. Jayjock, you wanted to read some
15 information into the record.

16 **DR. MICHAEL JAYJOCK:** Yes, please.
17 This shouldn't take more than two minutes. We've
18 recently -- we have recently been made aware of
19 extensive contemporary data from Colombia -- this
20 was mentioned yesterday, but I really wanted to
21 get it on the record, perhaps, even a little
22 stronger -- which shows a dominance of asbestos in
23 brakes being serviced in that country. These

1 values should be used in the current evaluation as
2 representative of the potential exposure to U.S.
3 workers who may be servicing asbestos-containing
4 brakes in this country. And there's a reference.

5 There appears to be an excellent
6 reference done specifically for bystander
7 exposure, ONU, and ONU exposure to asbestos. And
8 the study was done by ChemRisk, ostensibly in the
9 defense of asbestos litigation. It appears to be
10 an excellent -- it appears to provide excellent
11 data, and the study may have cost tens of
12 thousands of dollars to complete. It only covers
13 indoor exposure. This reference is listed by the
14 EPA but unused in the DRE. We believe that this
15 is an excellent work to look at quantitative data
16 based on a solid first principle modeling effort
17 that will inform the reduction factor.

18 The second paper I, Jayjock, did
19 with a colleague some time ago and is for outdoors
20 only. It would require someone familiar with
21 modeling to use it for ONU or bystander exposure
22 outdoors, but all the elements are there in the

1 paper. Last year, I, Jayjock, recommended that
2 the EPA get deeper into modeling expertise in my
3 comments on 1,4-Dioxane. If they did this, they
4 can handle it easily. If they did not, they
5 should do so. And I have the references there.
6 That's all I wanted to include.

7 **DR. KENNETH PORTIER:** So Dr.
8 Jayjock, your written comment had something on
9 2.3, as well. But is that something that we said?

10 **DR. MICHAEL JAYJOCK:** No. It was
11 only 2 point -- the only thing I wanted to get
12 into the record was 2.1 stuff.

13 **DR. KENNETH PORTIER:** Yeah. Because
14 it looks like this reference in 2.3 also
15 references the Colombian study, so it's discussed.

16 **DR. MICHAEL JAYJOCK:** Yes.

17 **DR. KENNETH PORTIER:** Okay. Thank
18 you for that. I appreciate you guys following up
19 and making sure all of this information is put
20 back into the record. So any additional comments
21 from anyone? Dr. Kissel, I see your hand up.

1 **DR. JOHN KISSEL:** Yeah. I wanted to
2 add that I also -- well, I received by email from
3 someone who is in the listening audience some data
4 that has to do with asbestos fiber counts in air
5 associated with various activities, including
6 servicing of some farm vehicles and other things
7 that we talked about yesterday. And the numbers
8 have been sent to EPA, and I will include them in
9 the record also.

10 **DR. KENNETH PORTIER:** Okay. Thank
11 you. Anyone else? Not seeing any hands go up.
12 Good. So we had a good conversation on 4.1 --
13 Question 4.1 yesterday. And I think we're ready
14 to pick up Question 4.2. But before I do that,
15 let me turn to EPA and see if there are any legacy
16 issues that EPA would like us to revisit at this
17 time. Dr. Scarano?

18 **DR. LOUIS SCARANO:** Thank you, Dr.
19 Portier. No, I don't think so, unless Stan Barone
20 -- Dr. Barone has something he'd like to add. I
21 think we're ready to proceed, but I'll let Stan
22 see if he wants to say anything.

1 **DR. STANLEY BARONE:** No, Dr.
2 Portier. This is Dr. Barone. I think we had a
3 very good discussion yesterday. The Agency is
4 looking forward to the report and the comments
5 provided and the public comments. A lot of good
6 information was provided in our dialogue.

7 **DR. KENNETH PORTIER:** As is always
8 the case. So if the host will scroll up the
9 questions and scroll to Question 4.2. Back one.
10 There we go. Dr. Scarano, would you read 4.2 into
11 the record?

12
13 **CHARGE QUESTION 4 (4.2)**

14 **DR. LOUIS SCARANO:** Thank you. Yes.
15
16 Question 4.2, please comment on the
17 appropriateness of the approach to derive the
18 commercial chrysotile based IURs, including the
19 underlying assumptions, strengths, and weaknesses
20 of the choice of study cohorts used, the key
21 calculation decisions, and the modeling used to
22 derive the IUR, all in Section 3.2.4 of the draft
23 risk evaluation.

1 **DR. KENNETH PORTIER:** Thank you.

2 And I know Dr. Kanarek has been working really
3 hard to organize the discussion around this topic.
4 Dr. Kanarek, I turn it over to you.

5 **DR. MARTY KANAREK:** I just want
6 everybody to understand the magnitude of this
7 question on the inhalation unit risk. The
8 assumptions, the strengths, the weaknesses, the
9 choice of the study cohorts, the key calculation
10 decisions and the modeling, we got the biggest
11 question. We started out with 18 different
12 issues, and with the help of Dr. Portier, we
13 narrowed it down to, I think, five that we'll
14 discuss today, unless people want to bring up
15 other things. So what I propose to do is to read
16 the -- is just mention the five that we want to
17 discuss and then circle back and discuss them one
18 by one if that's okay.

19 Before I discuss the -- name the
20 issues, I want to say that there was unanimity --
21 or near unanimity among our group that it would
22 have been better to calculate an inhalation unit

1 risk for all types of asbestos instead of just
2 chrysotile. However, that's not in our charge,
3 but I want to read that in -- I want that read
4 into the record. So as part of our charge, the
5 five we settled on that we should discuss were
6 that some commenters thought it was inappropriate
7 to accept potency factors from the literature
8 without checking them. So we'll come back to
9 that.

10 Many public commenters thought --
11 the second issues -- that other study populations
12 besides the North Carolina and South Carolina
13 textile plants should be used in the analysis.
14 And they brought up the Italian and Canadian
15 miners and others and Chinese and others. They
16 also brought up the idea of comparing textile to
17 the data from North Carolina and South Carolina to
18 non-textile uses of -- so we can come back to
19 that.

20 Some commenters thought that the
21 lack of cigarette smoking data in the North
22 Carolina and South Carolina studies would make it

1 difficult to model lung cancer. And we'll come
2 back to that. Many commenters went back -- had
3 different views on the model that should be used.
4 Should it be the linear risk model or the
5 exponential model? EPA used the exponential
6 model, but many people disagreed. We'll come back
7 to that. Some -- we brought up this next one
8 somewhat yesterday, but I don't think we finished
9 it. Some commenters thought the model should have
10 been done on incidence and not just mortality
11 data.

12 So if it's all right with everybody,
13 I think maybe we should start with the selection
14 of just the North Carolina and South Carolina
15 textile data, and I do understand -- I think
16 everybody might agree that they were picked
17 because of their superior exposure data as
18 compared to any other cohort. I think that's been
19 well known for years that they have the best
20 exposure data. But since so many of the public
21 commenters, at least, thought we should add in

1 China or Italy or Canada, I'd like discussion on
2 that issue.

3 **DR. KENNETH PORTIER:** So at this
4 point, any of the associates want to jump in or
5 any of the Committee members? Let's see if anyone
6 wants to comment on this. You can raise your
7 hand. I'll call on you. There we go. Dr.
8 Stayner, EPA, did you wish to comment?

9 **DR. LESLIE STAYNER:** Yeah. Sorry to
10 take the time to unmute both the phone and the
11 website. Yeah. Well --

12 **DR. KENNETH PORTIER:** Dr. Stayner,
13 speak up -- really speak up loudly because we have
14 a hard time hearing you.

15 **DR. LESLIE STAYNER:** Okay. We did
16 explore doing analyses on the other cohorts that
17 were mentioned, but we also did feel that there
18 were significant disadvantages in using those
19 cohorts, particularly related to the quality of
20 the exposure data, underlying exposure response
21 analyses. And this is particularly true with the

1 Canadian cohort. I think it's been fairly well
2 established now.

3 There's papers by Berman looking at
4 what was measured -- when you measure the Canadian
5 mine dust versus textile dust and found that a
6 large portion of what they're measuring were not
7 fibers. The problem really boils down to in the
8 Canadian is that the measurements they had were
9 not -- were all in impinger-based measurements.
10 And converting that to parts -- fibers per CC has
11 never been well done in that.

12 And if you look at different papers,
13 the conversion factor is varied considerably -- to
14 where in the North Carolina/South Carolina we had
15 the benefit of side-by-side sampling the filtering
16 and the impinger methods to convert that typically
17 count differences. And they ranged quite a bit --
18 conversion for (audio skip) operations within the
19 industry. So anyway, that's the primary reason
20 that we felt that the best available information -
21 - as pointed out, previous reviewers have also
22 suggested this, including the review by Dr. Crump

1 some years ago, that the best exposure response of
2 data available for chrysotile comes from the
3 Carolina studies.

4 **DR. KENNETH PORTIER:** Dr. Loomis?

5 **DR. DANA LOOMIS:** Thanks. I would
6 just add to that we also considered the studies of
7 Italian chrysotile miners. There are several of
8 those over a period of years -- and again,
9 determined that the exposure data were of much
10 lower quality in those studies. The available
11 data are actually based on reconstructions of
12 historical exposures.

13 And in addition to that, at the time
14 we did the review the dose response data that were
15 available from those studies were really not
16 suitable. The Chinese studies are also difficult
17 for similar reasons. There have been a number of
18 papers published, but we determined after careful
19 review that the exposure data were of much lower
20 quality and so decided not to rely on those papers
21 for exposure response information.

1 **DR. KENNETH PORTIER:** And Bateson,
2 Bateson Thomas, I guess. I don't know if it's
3 Thomas Bateson or Bateson Thomas. Go ahead. If
4 you're speaking, we're not hearing you.

5 **DR. THOMAS BATESON:** Hi, this is Tom
6 Bateson, EPA. Can you hear me now?

7 **DR. KENNETH PORTIER:** Yes. Now we
8 can hear you.

9 **DR. THOMAS BATESON:** Okay. Great.
10 I think Dr. Loomis and Dr. Stayner really covered
11 each of the major points. We really thought the
12 North and South Carolina had the highest quality
13 exposures going back several decades for South
14 Carolina specifically. And in doing a dose
15 response, one of the key components is the quality
16 of the exposure data, so that's why we really
17 focused on those. That's it.

18 **DR. KENNETH PORTIER:** Thank you.
19 Dr. Crump?

20 **DR. KENNY CRUMP:** I think the main
21 issue is not really the quality of the exposure
22 data in the Carolina studies. It's the question

1 of do those data match the kind of data that we're
2 trying to estimate the risk for. I think that's
3 the issue that we need to be debating,
4 particularly for example the do-it-yourselfers.
5 The kind of asbestos they're exposed to, it's very
6 different from exposures in the textile mills.

7 **DR. KENNETH PORTIER:** Thank you.

8 Dr. Sheppard?

9 **DR. ELIZABETH SHEPPARD:** Yeah. I
10 wanted to comment on this as well. I agree with
11 Dr. Crump and I agree with the speakers, Dr.
12 Bateson and others from EPA. In order to draw
13 conclusions from an epidemiologic study, it's
14 really important to have high quality exposure
15 data.

16 And the reality is it's really
17 difficult to get good quality exposure data in the
18 kinds of levels that we're actually more
19 interested in from a risk assessment point of
20 view. It's extremely difficult to measure that
21 adequately and to quantify it. So we end up
22 needing to rely on higher exposures. But with

1 asbestos, just it's qualitatively potentially very
2 different, at least that's my understanding
3 because of the fiber size and dynamics are very
4 different in the textiles than they are in the
5 kinds of population exposures that we're worried
6 about.

7 So I can understand EPA's decision
8 to rely on the cohorts they did, and I think the
9 arguments that they presented in the document were
10 actually quite convincing, given the textile
11 worker cohorts. But the document doesn't really
12 cover at all why not these other cohorts. And
13 particularly, we heard from the representatives
14 from Exponent about all these other studies that I
15 haven't had the time to look at in any detail.
16 But why are we not even talking about those.
17 There's probably very good reasons, but I think
18 the document should at least address that.

19 **DR. KENNETH PORTIER:** Thank you.

20 Dr. Anderson?

21 **DR. HENRY ANDERSON:** I would not
22 argue with the decisions that were made by EPA and

1 their consultants. My issue would be -- and I
2 think maybe that was a previous speaker's as well.
3 If there are these other studies, they may not
4 have as good quality data. So the choice to use
5 the best quality data studies, I think, as they
6 did is a good one.

7 On the other hand, it would be
8 helpful, if it can be done, to look at doing the
9 risk assessment on some of these other cohorts, if
10 it can be done, and see, even though the data is
11 of lesser quality, does the result come into about
12 the same ballpark? Or is the risks from the South
13 Carolina a whole lot higher than the risks in the
14 others? And that just gives a sense of the
15 available data sources. Is there some ballpark
16 consistency between them which would give,
17 perhaps, a greater confidence in the choice and
18 the results?

19 I think having all the other data
20 that's available in the two cohorts that were
21 chosen and the TEM data and you also have some
22 asbestosis data -- I think it's a richer dataset.

1 But I think it would be helpful to not just say
2 there's these others but to at least describe
3 them, if there is any way to do that. If they're
4 just simply not enough there and there's too many
5 caveats, then it might be not helpful. But I
6 think it's be useful to say that that was looked
7 at and here's kind of the qualitative assessment
8 of the dose response that might be seen there.

9 **DR. KENNETH PORTIER:** Henry, this is
10 Ken Portier. You know, as I was reading Section
11 3.2.4.5, which describes these studies, I kind of
12 had the same feeling you did that it would have
13 been nice if they could have -- despite the data
14 quality issues, and I understand all the
15 qualifiers that are there -- in some qualitative
16 way to be able to use this to support or weaken
17 the results that we got from the North and South
18 Carolina textile plant data. I guess I have a
19 feeling that the textile data from China probably
20 supports it, and the mining data may not be
21 aligned the same way.

1 But it would be interesting to at
2 least see those results, if, like you said -- if
3 they can carry it that far. It's unclear from the
4 description to what extent any modeling can be
5 done with the data. I realize there's major data
6 quality issues, but if those could be put aside
7 and with the realization that there's going to be
8 a lot of uncertainty in these estimates, but do
9 they support or not support?

10 I see a lot of EPA hands, but I'm
11 going to concentrate on the Committee first. And
12 then I'll come back to the EPA speakers, if you
13 don't mind. Dr. Crump, your hand's still up.

14 **DR. KENNY CRUMP:** Yes. Well, I
15 think we're really looking at this sort of the
16 wrong way. We're never going to find a cohort
17 that has exposures, for example, like the do-it-
18 yourselfers. But I think what we should have been
19 looking at is maybe getting a very good analysis
20 of the North and South Carolina data using TEM.
21 And we could determine the potencies of various
22 lengths of fibers and types of fibers for causing

1 these diseases. And we could apply those if we
2 have TEM data on the exposures we're concerned
3 about. We could actually apply the good North and
4 South Carolina data to that. I think that's what
5 we should have been doing all along, and maybe
6 there's still time to approach the probably from
7 that point of view.

8 **DR. KENNETH PORTIER:** Dr. Sheppard?

9 **DR. ELIZABETH SHEPPARD:** Yeah. I
10 wanted to add a little bit more to the
11 conversation in that I think Dr. Crump's points
12 are valid. I hadn't thought as deeply -- well, I
13 thought about a couple of aspects of additional
14 analyses of those North Carolina and South
15 Carolina cohorts. But I think there's also this
16 fundamental question with asbestos. Should we be
17 focusing on textiles and even is mining good
18 enough? I think that at least addressing
19 mechanics in the document and why we're not
20 considering that whole group of studies that we
21 saw is important just to understand that and give
22 that perspective.

1 Frankly, I think that another thing
2 that actually wasn't mentioned much in the
3 document is the challenge of exposure measurement
4 error, and that's a challenge in absolutely all of
5 these cohort studies. But it's going to be
6 magnified in, for instance, the mechanic studies
7 where there're going to be a lot lower exposures
8 and a lot more difficult to quantify than it
9 presumably was in the North and South Carolina
10 studies. I felt like the arguments for relying on
11 those were well supported. I just didn't see
12 enough other perspectives about other kinds of
13 cohorts.

14 **DR. KENNETH PORTIER:** Thank you.

15 Dr. Anderson, your hand's still up.

16 **DR. HENRY ANDERSON:** No, I was just
17 going to add that, again, in the qualitative sense
18 -- especially on the mesothelioma side -- now with
19 the number of the registries -- and I'm just
20 reminded of a couple of the papers from Australia
21 where they looked at you can't do a kind of fiber
22 count type assessment, but the length of

1 employment information is really quite useful
2 qualitatively. They have quite a number of cases
3 where the workers had only three months of
4 exposure. There's one of the papers where they
5 did the fiber analysis from the lungs and looked
6 at those that had only --

7 **DR. KENNETH PORTIER:** Dr. Anderson,
8 you're breaking up very bad. We're having a hard
9 time hearing you. I don't know -- I don't know if
10 your computer connection is going to be good
11 enough today. You may have to call in so we can
12 hear you, at least for a little while. Sorry. Go
13 ahead and continue.

14 **DR. HENRY ANDERSON:** Okay. So I was
15 just saying looking at some of those -- the
16 studies out of registries by the length of
17 employment gives you some information, even if you
18 don't have fiber counts. And again -- are you
19 hearing me better now?

20 **DR. KENNETH PORTIER:** Yes, it's
21 better now.

1 **DR. HENRY ANDERSON:** I might have
2 been too close to my microphone. Sorry. And they
3 also did this lone fiber burden, which of course
4 has its own problems. But they did have a group
5 that had only chrysotile in their lungs, and they
6 saw a dose response relationship to that. Yet it
7 can't be used really for -- as traditional dose
8 response, but it does get some information on the
9 length of employment that is necessary to develop
10 a mesothelioma. And there's many of them that are
11 well short of the 40 year that's being used here.

12 **DR. KENNETH PORTIER:** Can anyone
13 hear Dr. Anderson?

14 **MR. MARTIN ALVARADO:** Yeah. I can
15 hear him just fine. Yeah. He's fine.

16 **DR. STANLEY BARONE:** I can hear him.

17 **DR. STEVEN BENNETT:** I can hear him
18 just fine, as well.

19 **DR. HENRY ANDERSON:** Ken, it's your
20 machine, not mine. That's it.

21 **DR. KENNETH PORTIER:** Dr. Anderson,
22 I can kind of --

1 **DR. HENRY ANDERSON:** That's it for
2 me.

3 **DR. MARTY KANAREK:** Well, that was a
4 good discussion, unless other people want to talk.
5 We brought up, I think, all the issues that the
6 group had. I'm not sure we came up with magic
7 solutions. Dr. Portier?

8 **DR. STEVEN MARKOWITZ:** This is
9 Steven Markowitz. I'd like to make a comment.

10 **DR. MARTY KANAREK:** Go ahead.

11 **DR. KENNETH PORTIER:** Hello, Ken
12 Portier back again. So it wasn't Dr. Anderson who
13 got shut off. I got hung up on. Sorry. Took me
14 a second to come back. Dr. Crump?

15 **DR. KENNY CRUMP:** Yeah. I want to
16 kind of say something historical. Back in 2008,
17 Wayne Berman and I worked on this project to
18 estimate potencies of fibers of different lengths
19 and different types. And we had limited data, and
20 we had to use -- we needed TEM data. We didn't
21 have the kind of data that we needed, but we used
22 what we had in a kind of proof of concept. And it

1 was called a binning project by some people, and I
2 didn't even realize what had happened to it.

3 But I've learned since I've been on
4 this Committee that the Science Advisory Board,
5 the EPA, looked at it and rejected it. Which you
6 know, what it was designed to do was get us out of
7 the conundrum we're in right now. If we had
8 developed that and perfected it getting better
9 data and applying it, we could use the South
10 Carolina data to estimate exposures.

11 And if we have good TEM data for the
12 studies that we have -- to the exposures that we
13 have, we could use this approach to estimate risk
14 from all of these activities. I think that was
15 very short sighted of the Science Advisory Board
16 for rejecting that at that time. And I think
17 we're reaping the consequences of it now.

18 Let me say something positive. I
19 think that we might could still use some of the
20 ideas in that project to try to estimate the risk
21 in these exposure situations. And I would

1 encourage EPA to pursue that and see if that is
2 possible.

3 **DR. KENNETH PORTIER:** Thank you.

4 Dr. Rowlands?

5 **DR. CRAIG ROWLANDS:** Yes. So I have
6 some questions actually about the criteria that
7 was used by the Agency to define when you had a,
8 quote/unquote, pure chrysotile fiber exposure
9 versus a chrysotile with amphibole fiber mixture.
10 I didn't find anywhere where that was clearly
11 described as when the Agency would determine based
12 on what criteria that this was a chrysotile only
13 exposure versus some mixtures. And that's clearly
14 obviously very important because that's the basis
15 for the IUR is chrysotile only.

16 And the other thing I had -- so I
17 think it's important to put that in there and very
18 clearly define that. The second thing is we heard
19 a lot of comments, especially during the public
20 comment discussion, about some of the data now
21 that's come into play around these North
22 Carolina/South Carolina textile worker cohorts

1 that some of those may not in fact have been a
2 pure chrysotile -- however that's defined --
3 exposure, that there actually may have been mixed
4 of chrysotile and amphibole fibers as well. And I
5 think the Agency needs to provide some discussion
6 around that -- consider that information again,
7 given the importance to the IUR calculation. And
8 also there was the other miners cohorts that seem
9 to have a more pure exposure, so maybe they now
10 become more relevant as we bring in this other
11 information.

12 The other thing is there's some data
13 out there -- several studies that have actually
14 shown that there isn't an actual background level
15 of mesothelioma that can't be attributed to
16 asbestos. So I think it's really more relevant to
17 use a relevant risk model rather than absolute
18 risk model if that's true. And the Agency, I
19 think, needs to go in and do a clear assessment of
20 those studies to determine whether or not that
21 actually does change the approach to how the risk

1 model is calculated. So I just wanted to say
2 those things.

3 **DR. KENNETH PORTIER:** Thank you.
4 Dr. Scarano, I see Dr. Loomis, Dr. Kopylev, Dr.
5 Stayner, and Dr. Bateson all have their hands up,
6 but I'm not sure which to call on next. Do you
7 have a suggestion?

8 **DR. LOUIS SCARANO:** I would suggest
9 you start with Bateson, Kopylev, and the subs, in
10 that order, if I may.

11 **DR. KENNETH PORTIER:** Okay. Dr.
12 Bateson.

13 **DR. THOMAS BATESON:** Hi, this is Tom
14 Bateson, EPA. First off, I wanted to call the
15 panel's attention to Tables 3-3 to 3-7 where we
16 look at all five of the cohorts, and we show what
17 the lifetime unit risks for chrysotile -- or for
18 lung cancer and mesothelioma are. So those are
19 the data that you can use to do the ballpark
20 estimation that was requested. So it's the
21 righthand most columns where it says lifetime unit
22 risk based on 97th percentile -- or 95th

1 percentile confidence interval. So that data is
2 there. We computed it, so please take that into
3 account.

4 Regarding the background rate of
5 mesothelioma, EPA has long used a model that's
6 developed by Peto 30-some years ago. That model
7 has served well. It is the basis of the current
8 general asbestos inhalation unit risk with EPA for
9 all six fiber types. We also use that again in
10 the 2014 Libby amphibole analysis, and that model
11 had Dr. Peto on the panel.

12 So this model has a long history.
13 It does assume that there is not background rates
14 of mesothelioma. We did hear in the public
15 comments that there is a background rate from Dr.
16 Moolgavkar. But we also heard from one of the
17 public commenters that in their control series
18 they found chrysotile fibers in about half of the
19 people's lungs. So those people didn't think they
20 were exposed to asbestos, but they were.

21 So we can't know that there's no
22 background rate when people are getting exposed

1 without their knowledge. So to say that there are
2 no known exposures, therefore that's just the
3 background rate -- that's not clear to me. So
4 that's why we relied on the standard Peto model.
5 So that's what I have to say now and let Leonid
6 weigh in as well.

7 **DR. KENNETH PORTIER:** Thank you.

8 Dr. Kopylev? Dr. Kopylev, we're not hearing you.
9 You're unmuted in WebEx. Your phone may be muted.

10 **DR. LEONID KOPYLEV:** Oh, thank you
11 very much. Leonid Kopylev, EPA. I want to call
12 the panel's attention to the Table 3.8 where we
13 used the data we had to compare lung cancer risks
14 -- ranges of lung cancer risk from textile studies
15 to mining studies. And you could see that this
16 range is quite intercepting. It's impossible to
17 say they're different. Of course, we didn't have
18 the data to compare from mesothelioma, but if it's
19 for lung cancer, these numbers are very, very --
20 rather similar.

21 I want to add on background risk of
22 mesothelioma. Yes, there are evidence that

1 Thorotrast which is agent which was used in some
2 scanning -- human scanning medical work -- did --
3 indeed, cause, mesothelioma. But this is one
4 agent that is not used anymore. And then studies
5 of radiation mostly look at the cohorts that also
6 have exposure to asbestos. So it's not attributed
7 to ionizing radiation and not asbestos is not
8 really well supported. So the evidence cited
9 isn't by any means definitive to reject
10 longstanding EPA model. That's all.

11 **DR. KENNETH PORTIER:** Thank you.

12 Dr. Stayner?

13 **DR. CRAIG ROWLANDS:** Can I just say
14 one thing that I have?

15 **DR. KENNETH PORTIER:** Yeah. Sure.

16 **DR. CRAIG ROWLANDS:** This is Craig
17 Rowlands. Yeah. I think those are very
18 interesting arguments you're putting forth. They
19 cite 10 publications, and I think the Agency
20 should go ahead and evaluate that in the context
21 of the DRE itself for -- write all those comments

1 down. I think that's important for us to hear.

2 That's all.

3 **DR. KENNETH PORTIER:** Thank you.

4 That was Dr. Rowlands. Dr. Stayner? Dr. Stayner,
5 you're unmuted in WebEx, but we still can't hear
6 you. Your phone may be muted.

7 **DR. LESLIE STAYNER:** Oh, okay.

8 Sorry about that. Yeah. I wanted to add -- so we
9 -- I was part of the review seven years back of
10 the risk analysis by Crump and Berman. And the
11 problem then and the problem now is we really have
12 a lack of good quality data for other industries
13 on this fiber size distributions. But the other
14 problem is the analyses that we did in South
15 Carolina and North Carolina using TEM -- what we
16 found is all the different fiber sizes which would
17 be used for the extrapolations to other industries
18 -- the results are they're very highly correlated.

19 So there's some more recent papers
20 that are -- I don't know, Kenny, if you've had a
21 chance to look at papers by Hamra that have these
22 Bayesian methods to try to overcome these

1 correlations. But still, some of the fiber sizes
2 were nearly perfectly correlated, so it's very
3 hard to come up with separate estimates of potency
4 based on fiber size from our work.

5 **DR. KENNETH PORTIER:** Thank you.

6 Dr. Loomis?

7 **DR. DANA LOOMIS:** Thanks. I wanted
8 to go back to some earlier comments, beginning
9 with the statement by Dr. Crump, which I think is
10 really important. And that is that, ideally, we
11 would have data on the risks we're trying to
12 assess. So if you accept that, then including the
13 miner studies doesn't really help us very much.
14 That is most of the other cohorts that were
15 mentioned have not been -- we determined not to
16 include are studies of chrysotile miners.

17 So the mining environment is really
18 different from not only the textile environment
19 but the ones that concerns have been raised about,
20 like brake repair. For one thing, it's outdoors.
21 For another thing, it involves processing rock,
22 basically, to extract the asbestos ore. So a lot

1 of the material that's generated in mining and
2 processing the raw asbestos is not, in fact,
3 asbestos fibers. There are data that are
4 available show that the majority of it is actually
5 dust. So I don't think that including those
6 mining cohorts really contribute very much to
7 understanding the situations that exist today as
8 there is no asbestos mining in the United States
9 anymore.

10 Another point is that one speaker
11 said that chrysotile mining involves a more pure
12 exposure, but I don't think that's true either in
13 the sense that serpentine and amphibole asbestos
14 forms can co-occur and do co-occur in many of the
15 natural deposits. And in addition to that,
16 there's also silica dust in the raw asbestos
17 that's processed in mining situations. So it's
18 not a more pure exposure either.

19 **DR. KENNETH PORTIER:** Thank you.

20 Dr. Kanarek, I think we've had a good discussion
21 on this issue. Why don't we move on to the next
22 issue?

1 **DR. MARTY KANAREK:** I agree.

2 **DR. KENNETH PORTIER:** I don't want
3 to spend all day on one, and you've got a lot of
4 things to talk about. So let's go ahead and move
5 on. I thank you, EPA guys, but you can put your
6 hands down. We'll move on to the next discussion.

7 **DR. MARTY KANAREK:** This is not an
8 easy one either. It has to do specifically with
9 the lung cancer modeling and cigarette smoking.
10 There's been a huge literature, starting with
11 Selikoff, the synergism -- he calls it -- between
12 cigarette smoking and lung cancer. And I think
13 over the years it's evolved into many research
14 papers, and I think now you would call it super-
15 additive. But we're a prisoner here of having
16 chrysotile only because the data on cigarette
17 smoking is in cohorts like Dr. Markowitz' and
18 others that's all kinds of asbestos -- asbestos
19 insulators. So to be provocative, I want to say I
20 don't think we can model lung cancer in chrysotile
21 asbestos without having data on cigarette smoking.
22 And I'd like to hear from others on that.

1 **DR. KENNETH PORTIER:** Put your hands
2 up and I'll call on you. Dr. Shukla, I know you
3 wrote quite a bit of data in your initial
4 comments. Please proceed.

5 **DR. ARTI SHUKLA:** So yes, I agree
6 with Dr. Kanarek that it is very important. And
7 as he mentioned, there are multiple publications
8 using animal study as well as epidemiologic
9 studies that it could be additive. It could be
10 only the cigarette smoke and asbestos co-exposure
11 could be additive, synergistic, or even
12 multiplicative of the lung cancer, nothing to do
13 with mesothelioma. So and mechanism could be
14 very, very different also.

15 But that is the second thing. So
16 not considering cigarette smoke exposure in
17 modeling for asbestos induced lung cancer I think
18 is not adequate. And I'm not a modeler or I
19 cannot do. But I think this is an important
20 aspect to be taken care of in modeling.

1 **DR. KENNETH PORTIER:** Dr. Shukla,
2 the DRE basically says -- or claims that
3 mesothelioma's not related to smoking.

4 **DR. ARTI SHUKLA:** Yeah. No,
5 mesothelioma, there is no --

6 **DR. KENNETH PORTIER:** So is
7 mesothelioma out of the --

8 **DR. ARTI SHUKLA:** No. Yeah.
9 Mesothelioma is out of there. As per my
10 knowledge, I haven't seen anything cigarette smoke
11 and mesothelioma. So mesothelioma is out of
12 there, definitely. But lung cancer, at least I
13 cannot think it could be out of that. So...

14 **DR. KENNETH PORTIER:** So the other
15 thing it says is the bias related to this failure
16 to control for smoking is believed to be small.
17 And I think, to me, that's kind of the main issue
18 here. If we can't control for smoking -- tobacco
19 use in these cohorts, what's the expected impact?
20 Is it small, or do you think it's large? I heard
21 you basically say it could be a promoter on the

1 one hand, and on the other hand it could actually
2 work in an opposite direction.

3 **DR. ARTI SHUKLA:** So working in
4 opposite direction are very few, but promotor in
5 the way of synergism, in way of addition, or even
6 multiplication of the lung cancer. There are
7 many, many references, and I consider that as very
8 important. I mean, sometimes, way back --
9 sometimes I thought that maybe asbestos itself
10 cannot cause lung cancer, but if cigarette smoke
11 is there, then there are like 97 percent chances
12 of causing cancer because cigarette -- asbestos
13 can absorb carcinogens from cigarette smoke and
14 take them deeper into. So that's how it can
15 enhance the cigarette smoke induced lung cancer.

16 Also, there are reports that it can
17 -- cigarette smoke -- I don't know how, but it can
18 help and create asbestos deeper into lung. I
19 don't know how, what is the mechanism? But then
20 another report which I read just said -- it's like
21 2015, and it said that both -- we know that
22 asbestos is inflammatory reagent. It can cause

1 inflammation. And there are so many constituents
2 of cigarette smoke. They are also inflammatory in
3 nature. So two agents causing inflammation in
4 parallel, that could be -- so I think cigarette
5 smoke is promoter. And it should be taken into
6 account. Otherwise, what we are calculating may
7 not be appropriate or may be estimate we are
8 getting -- it could be not the correct one.

9 **DR. KENNETH PORTIER:** I'm looking to
10 see if any other panelists want to comment on
11 this. While they're thinking about it, Dr.
12 Bateson, you wanted to comment.

13 **DR. THOMAS BATESON:** I'll take a
14 shot. Hi, Tom Bateson. Yes. So we are well
15 aware of the relationship between smoking and
16 asbestos. The cohorts that we had to analyze did
17 not have smoking data. So we had to think what
18 was the impact of that? What kind of bias would
19 that create?

20 Now, the association between --
21 there's a correlation between smoking and the dose
22 of chrysotile that you're getting is likely to be

1 small. I don't think that the people who are
2 smoking more were having higher exposures to
3 chrysotile. At the time of this cohort in the
4 Carolinas, the overall prevalence of smoking was
5 probably quite high. Let's say it was 60, 70
6 percent. The prevalence of smoking changes very
7 slowly. It has been going down, and that's been a
8 great thing. But at that time, year after year,
9 the prevalence of smoking is pretty constant.

10 Meanwhile, these people are
11 accumulating more accumulative chrysotile
12 exposures. So those two variables should be
13 rather independent from each other. So we
14 wouldn't expect there to be very much confounding
15 bias, and that's why we said we thought the bias
16 would be small.

17 We recognize that smoking can cause
18 a synergistic effect. And what would that mean?
19 That would mean that the effect that we observed
20 in those cohorts with 60 or 70 percent smoking
21 might be higher than in a nonsmoking population.
22 But it also means that the unit risks derived from

1 those populations will be health protective of
2 populations that have lower prevalence of smoking,
3 like we do now. So that's the best estimate that
4 we could get from the best data that was
5 available. We did not have an opportunity to
6 control for confounding, but we did think hard
7 about what the impacts were.

8 I want to raise one other thing
9 about smoking and asbestos. In the Libby
10 amphibole asbestos assessment, which Dr. Kopylev
11 and I did the dose response, we had a similar
12 situation. The cohort in Libby did not have
13 smoking data. And there was a similar concern
14 that there would be potential confounding.

15 So we relied on a rather novel and
16 clever idea by David Richardson, who's published
17 on this. And his idea was to take the asbestos
18 exposures and regress those on another disease,
19 like COPD. And if there's association between
20 asbestos and COPD, that probably indicates that
21 there was a correlation between smoking and
22 asbestos. And when we did that, we actually got a

1 negative data coefficient showing that there
2 wasn't an association.

3 It wasn't significant. It was
4 actually somewhat downwards. And from that, we
5 felt much more relieved that there wasn't
6 confounding by smoking in that population. So
7 those are the rationales by which we thought the
8 bias would be small and why the unit risk derived
9 from these populations was still solid. Thanks.

10 **DR. KENNETH PORTIER:** Thank you, Dr.
11 Bateson. Dr. Sheppard.

12 **DR. ELIZABETH SHEPPARD:** Yeah. I
13 wanted to echo some of what Dr. Bateson said. The
14 key thing to be paying attention to here in terms
15 of the concern about unadjusted smoking is how
16 correlated it is with asbestos exposure. So I
17 think that's the important thing. And it would be
18 good to know whether there's any data at all from
19 any kind of external source or anything that would
20 suggest that there might be relatively low
21 correlation, which I think is what EPA has
22 concluded.

1 I hadn't thought about the changing
2 prevalence being associated with the cumulative
3 lifetime exposure, but I think in other industries
4 there's suggestions that people in different jobs
5 with higher or lower exposure would have
6 different. And I'd be interested in hearing
7 whether EPA or anybody else who knows these
8 cohorts well has any insight into the potential of
9 the different, say, more highly exposed jobs
10 having more smoking exposure or less.

11 **DR. KENNETH PORTIER:** Dr. Stayner?
12 Dr. Stayner, your phone may be muted.

13 **DR. LESLIE STAYNER:** Oh, okay.
14 Unfortunately, we don't have that information, but
15 there is some literature on this, a paper by Aaron
16 Blair a while ago looking at different cohorts
17 where he has the smoking information and looking
18 at analyses with or without. And in general, what
19 most epidemiologists believe is it's based on past
20 experience that within the factory (audio skip)
21 varies between different jobs on the shop floor
22 that smoking would be different in different areas

1 of the plant. So the idea that there'd be a
2 correlation between smoking and exposure -- the
3 probability is pretty low. And that's what we've
4 gone on.

5 But I wanted to make another point.
6 So it's easy to get confused here between two
7 issues. One is there's bias of compounding by
8 smoking, which we think is unlikely. And the
9 other's whether there's effect modification or
10 sometimes it's called interaction between smoking
11 and asbestos. There's a huge literature on that
12 issue. So we weren't able to address the issue in
13 our analysis of effect modification because we
14 don't have smoking data.

15 But somebody suggested yesterday --
16 I think it's a useful suggestion -- that what we
17 could do is we could in our life table analysis
18 use rates for smokers rather than rates for the
19 general population and that way come up with
20 estimates of risk for smokers as well as for
21 nonsmokers. That could be done in assumption that
22 the relationship between smoking and asbestos is

1 multiplicative. There's a lot of debate about
2 whether it's additive or multiplicative or
3 something in between.

4 But anyway, so that's an issue that
5 we might dig a little further on. But as far as
6 confounding bias, unfortunately we don't have any
7 information. But we do believe that it's unlikely
8 to be large. Thank you.

9 **DR. KENNETH PORTIER:** This is Ken
10 Portier. I notice in the DRE there's a discussion
11 of a Chinese study by Deng 2012, in which you fit
12 some log linear and relative rate models with
13 adjustment for age, smoking, and calendar period.
14 I just wondered if smoking was significant in that
15 analysis or not. It sounds like they had some
16 smoking data. Again, it was a weak study for many
17 reasons. But I just wondered if any of you
18 remembered that study and whether that association
19 was strong, positive, or negative. I see Dr.
20 Loomis still has his hand up. Dr. Loomis?

21 **DR. DANA LOOMIS:** Thanks. Well, I'm
22 looking at that paper by Deng et al right now.

1 And unfortunately, I can't answer your question.
2 They did have some smoking data, but their only
3 table that shows the model results doesn't have
4 the coefficient for smoking. So I don't know. It
5 is a very small cohort though. It's less than 600
6 people. So as you said, it's not a very reliable
7 study for our purposes.

8 I wanted to go back to an earlier
9 comment though. The reason I put my hand up was
10 to add something to the discussion of distribution
11 of smoking within these cohorts. I think it's
12 generally known that smoking prevalence was quite
13 high in the Carolinas during the follow up period.
14 We don't have any smoking data for the cohorts.

15 But in North Carolina, at least, one
16 of the things that we know is that the dirtiest
17 jobs were typically done by African American
18 workers. It's one of the only places in the mills
19 that they were found. So they did the dusty work
20 of opening the bags and removing the raw asbestos
21 and putting it into the carting machines, which
22 were some of the dustiest jobs. And at that time,

1 African Americans were less likely to be smokers
2 and typically smoked less. So this is all
3 speculative. I don't think it's as strong as the
4 evidence that Dr. Bateson presented from the EPA's
5 analysis. But it is something to consider.

6 **DR. KENNETH PORTIER:** Thank you.

7 Dr. Markowitz?

8 **DR. STEVEN MARKOWITZ:** So it's
9 pretty clear that there's not much headway that
10 can be made with chrysotile specific studies and
11 smoking. There's no reason to believe the smoking
12 effect for chrysotile exposed workers would be any
13 different than for the other fiber types. But I
14 just wanted to point out that this is -- if
15 smoking is an important issue, this is one of the
16 disadvantages of doing a chrysotile only risk
17 evaluation.

18 There are a lot more data available
19 in the much broader asbestos literature, including
20 a lot of case control studies, which necessarily
21 aren't fiber specific. So when EPA gets to the
22 much broader risk evaluation, maybe this issue can

1 be looked at more seriously. But it does
2 illustrate a real limitation in confining the
3 evaluation of risk just to chrysotile or, in fact,
4 just to intentional uses of chrysotile at this
5 point.

6 **DR. KENNETH PORTIER:** Thank you.

7 Dr. Crump and then Dr. Anderson. Dr. Crump?

8 **DR. KENNY CRUMP:** Yeah. I would
9 like to just say that I agree totally with what
10 Dr. Stayner said about using the life table
11 analysis. And the smokers are a susceptible
12 subgroup, and we should quantify their risk, if we
13 can. And it would be very easy to do with the
14 life table analysis. All we have to do is replace
15 the smoking rates and all the rates with rates
16 that pertain to smokers. And you'd have the risk
17 for whatever rate of smoking you assumed. You'd
18 have the risk for those people that smoke that
19 way.

20 But what we have now, we use just
21 the general rates in the population, which
22 includes smokers and non-smokers. So the risk

1 that we have doesn't really apply to anyone. It
2 doesn't apply to nonsmokers. It doesn't apply to
3 smokers. It's something in between.

4 So I would think it will be very
5 reasonable to make two sets of analyses, make a
6 risk estimated for nonsmokers and risk estimated
7 for smokers. And it can be very straightforward
8 to do using the life table analysis. Just put in
9 the right background rates for smokers and
10 nonsmokers. Even seeing that might encourage some
11 smokers to maybe they want to quit smoking.

12 **DR. KENNETH PORTIER:** Thank you,
13 Kenny. Dr. Anderson, and then I think we'll move
14 on to a new topic. Dr. Anderson?

15 **DR. HENRY ANDERSON:** Yes. I was
16 just going to underscore that smokers are a highly
17 susceptible subgroup. I think a major challenge
18 would be not so much as we've been talking here.
19 That certainly is challenging. But should there
20 be some focus on are we protecting the smokers to
21 the same degree as the others, and how could EPA
22 include them as a susceptible special population?

1 And the good news is the proportion of smokers is
2 going down, but I do think -- and I think EPA does
3 recognize in the document that they are a
4 susceptible population. But the question is how -
5 - having recognized that, what are the
6 consequences or what steps need to be taken to be
7 sure that cigarette smokers are adequately
8 protected?

9 **DR. KENNETH PORTIER:** Thank you, Dr.
10 Anderson. Dr. Kanarek, I think I heard at least
11 one, if not two, recommendations there. So that's
12 good. Why don't we move on to the next issue?

13 **DR. MARY KANAREK:** Yeah. Can I just
14 say that the document -- the draft document
15 doesn't have any of the great comments we heard
16 today from Dr. Bateson and the EPA thinking and
17 Dr. Stayner and Dr. Loomis. So the document is
18 very deficient in why they -- we need to include
19 what was said today in that document because it
20 just says, "Well, there's no bias." But
21 explaining how they arrived at that thinking is
22 very important.

1 **DR. KENNETH PORTIER:** I think if you
2 do a search on smoking in the document you see
3 that there's actually quite a bit of discussion of
4 smoking in a lot of different sections. Even in
5 the PESS section, they discuss smoking history as
6 a source of variability and susceptibility between
7 people. But I agree that at least one of the
8 recommendations having to do with using the
9 smoking life tables rather than the general
10 population life table is not done and not
11 included. And that's a good recommendation. And
12 you can certainly point out these other things in
13 the write up where you think they need to add that
14 discussion back in. So Dr. Kanarek, why don't we
15 move on to the third issue.

16 **DR. MARTY KANAREK:** It started with
17 the public commenters and that the EPA used the
18 exponential risk model. And a lot of people
19 thought the linear risk model should have been
20 carried through and maybe compared. And if it's
21 okay, I would like Dr. Crump to lead the
22 discussion on this, the linear risk model versus

1 the exponential risk model. Is that all right,
2 Kenny?

3 **DR. KENNY CRUMP:** Sure. I'd like to
4 start off by talking about the 19 -- 2005 cancer
5 guidelines. This will not only just cover the
6 linear versus the exponential, but I think it'll
7 shed some light on some other decisions as well.
8 This is what the 2005 EPA cancer guidelines say:
9 "When the weight of evidence evaluation of all
10 available data are insufficient to establish the
11 mode of action for a tumor site and when
12 scientifically plausible based on the available
13 data, linear extrapolation is used as a default
14 approach because linear extrapolation generally is
15 considered to be a health protective approach.
16 Nonlinear approaches generally should not be used
17 in cases where the mode of action has not been
18 ascertained." That's what the EPA guidelines say.

19 Now, you need to understand what
20 they mean by linear and nonlinear, and they say
21 that very uncarefully in the guidelines. By
22 linear, that's a shorthand way of saying low dose

1 linear. Low dose linear dose response they simply
2 mean a dose response that has a positive slope and
3 zero dose. That's all it means. So a perfectly
4 linear dose response would be a low dose linear
5 dose response. And the exponential model is also
6 low dose linear. So either one of these models
7 would be acceptable as far as the cancer
8 guidelines go for doing linear dose response as a
9 default approach.

10 The exponential model was selected
11 on the basis of goodness of fit. I think the
12 entire dataset had fit our dataset a little bit
13 better than the linear model. The linear model
14 has been used historically for asbestos. I'm not
15 really sure that the extent of all the data is
16 necessarily the best way to decide on the basis of
17 the models. You could -- I think the fit to the
18 low dose data are more important than the fit to
19 all the data because you're trying to estimate low
20 dose response. So EPA might try fitting these two
21 models to a low dose subset of the data. And that

1 might be a better way for making the decision.

2 But I don't think this is a critical decision.

3 One of the South Carolina studies,
4 Eliot, applied both the linear model and the
5 exponential model. And the KLs they found from
6 those are only different about fivefold one and a
7 quarter. So and that's not a lot of uncertainty
8 compared to all the other sources of uncertainty
9 in this issue.

10 So I think -- my feeling is it's not
11 a huge issue. They both satisfy the EPA
12 guidelines -- so for doing a linear dose response
13 when the mode of action is not known for sure. So
14 I think probably either one would be acceptable.
15 Interestingly, if you start reducing the data
16 lower and lower doses, the two models will
17 converge. Eventually, they'll approach the same
18 value.

19 **DR. KENNETH PORTIER:** Does anyone
20 else on the Committee wish to comment? Dr.
21 Bateson, you want to clarify something?

1 **DR. THOMAS BATESON:** Yes, thank you.
2 Tom Bateson, EPA. I just want to make sure it was
3 crystal clear to everybody that the Eliot model,
4 when we're talking about linear versus
5 exponential, that's the fit within the range of
6 the data, so two different models. But once we
7 find the POD, then it is a linear low dose
8 extrapolation. It's not an exponential low dose
9 extrapolation. It was linear. So I just want to
10 make sure that was clear for everyone.

11 And the reason that we chose the
12 exponential model over the linear model for the
13 range of the data was based on the fit. Looking
14 at the AIC, which is Akaike's Information
15 Criterion, they were several hundred points better
16 for the exponential than the linear. So that was
17 the basis of that selection. So we appreciate Dr.
18 Crump's comments.

19 **DR. KENNETH PORTIER:** Yeah. Thank
20 you. Dr. Sheppard, I think you wanted to --

21 **DR. KENNY CRUMP:** I didn't think you
22 wanted to -- so then your extrapolation from the

1 exponential into account. So the difference
2 between the two models would probably be even less
3 than I said.

4 **DR. KENNETH PORTIER:** Dr. Sheppard,
5 you wanted to talk about model fit?

6 **DR. ELIZABETH SHEPPARD:** Yeah. I
7 actually was pretty concerned with this choice or
8 putting all the weight on relying on the
9 exponential model over the linear model on the AIC
10 because that's a measure of fit in all the data.
11 And what we care about is down at the low end.
12 And the exposure data are highly right skewed, and
13 there's a couple of very, very high exposures that
14 are not really representative of the population
15 but are probably dominating the model fit.

16 So if the standard model -- and I
17 don't know the asbestos literature as well as
18 other people who are participating, so I would
19 appreciate more comments on this. If the standard
20 model is the linear rate model, then why are we
21 relying on the exponential model based on an
22 overall fit criterion when what we really care

1 about is how well the data fit at the low end of
2 the dose distribution, which we don't have that
3 information.

4 **DR. KENNETH PORTIER:** Dr. Anderson
5 and Dr. Shukla, I see your hands up. But I want
6 to ask Dr. Kopylev -- I think he wants to comment
7 on this -- clarify.

8 **DR. LEONID KOPYLEV:** Yes. I agree
9 with everything Dr. Crump said, and I want maybe
10 to explain what people's possible confusion here.
11 When people say linear, they talk about what was
12 done in 1988 or '86 assessment where the linear
13 model was fit for grouped data. And this is the
14 linear model they talk about. Here we have
15 epidemiological data, and here we chose between
16 exponential and linear fit to the individual data,
17 not the group data. I don't know if it helps.
18 But I thought that clarification would be maybe
19 helpful.

20 **DR. KENNETH PORTIER:** Thank you.
21 Dr. Loomis, do you have something to add on this?

1 I think your phone might be muted. You're unmuted
2 in WebEx, but we don't hear you.

3 **DR. DANA LOOMIS:** Right. Gotcha.
4 Yes, with respect to Dr. Sheppard's comments,
5 we're primarily discussing the model fit for the
6 South Carolina cohort. And for that cohort, the
7 exposure data are not quite as extensively skewed
8 as those for North Carolina. If you look at
9 Eliot's paper, there are graphical depictions of
10 the distribution of exposures. So South Carolina
11 didn't have quite the same level of extremely high
12 exposures that are likely to drive the models. So
13 I think the question about model fit is probably
14 less pertinent for that particular cohort.

15 **DR. KENNETH PORTIER:** Dr. Anderson
16 and then Dr. Shukla.

17 **DR. HENRY ANDERSON:** I just wanted
18 to -- I raised it before -- is the notes sent in
19 by Dr. Dement, I think, address this issue
20 somewhat. But the additional question or issue I
21 have is so if we use the exponential here, have
22 any other modelers also looks at the broader

1 asbestos cohorts or used for the IRIS document
2 that we could easily end up with two different
3 IURs when EPA now goes on to the legacy issue?
4 And many of the legacy issue, the predominate
5 exposure is going to be to chrysotile, but they
6 have the other exposure to amphibole as well. And
7 we could be left with arguing as to, for an
8 individual or others, which of the IURs are
9 appropriate to use in estimating exposures and
10 risks.

11 So that's why I kind of lean towards
12 having consistency between when the differences in
13 the choice is not all that great. So that would
14 be the only thing for EPA to consider that there
15 is some confusion. And then the question would be
16 what should the occupational component, which is
17 using the linear model and where all measurements
18 in the field now that OSHA's doing do not
19 differentiate which fibers. So I think we have a
20 potential here for TSCA in this limited
21 circumstance to have a broader impact on the whole
22 regulatory community.

1 DR. KENNETH PORTIER: Thank you.

2 Dr. Shukla?

3 DR. ARTI SHUKLA: Yes. So I'm a
4 little confused, or maybe I don't know much about
5 it. But I was reading about from -- ranging from
6 1993 Dr. Churg's study up to Bernstein's study
7 2019. And these studies are pure chrysotile
8 exposure. And what they came up with a conclusion
9 that chrysotile -- it is like a -- if it causes
10 cancer, it has to accumulate in greater number,
11 and it takes very long. So suggesting that
12 they're -- and all these reports suggest that
13 there has to be a greater accumulation of this.
14 So that means there has to be a threshold for pure
15 chrysotile, too.

16 I know what we are talking about is
17 not chrysotile, but our title is still commercial
18 chrysotile. So I'm just a little confused about,
19 okay, so like is it -- do we meet -- as reported
20 in the paper, it seems like there you would have
21 to have certain number of fibers. If not, low
22 exposure will not cause anything. So I was

1 thinking that there is likely a threshold for
2 chrysotile fibers to cause lung cancer or
3 mesothelioma.

4 **DR. KENNETH PORTIER:** Thank you.

5 Dr. Loomis?

6 **DR. DANA LOOMIS:** Thanks. I just
7 wanted to make one other point about the choice of
8 models. Dr. Sheppard asked whether the linear
9 relative rate model is considered a standard in
10 the asbestos literature. And I don't think you
11 could say it is. Various investigators have used
12 a whole range of models, including that one, the
13 normal relative risk model, power models and a
14 number of other things. And none of them seems to
15 be clearly preferable in terms of either
16 biological justification or fit to the data.

17 **DR. KENNETH PORTIER:** Dr. Bateson?

18 **DR. THOMAS BATESON:** Hi, can you
19 hear me?

20 **DR. KENNETH PORTIER:** Yes.

21 **DR. THOMAS BATESON:** I just wanted
22 to respond to Dr. Shukla. You're the expert in

1 mode of action and not me. But what I understand
2 is that it's a complex mechanism involving
3 multiple biologic processes, which include
4 genotoxicity, chronic inflammation, cellular
5 proliferation. And in situations like that, EPA
6 has relied on linear low dose extrapolation below
7 the POD.

8 And I wanted to recall the public
9 comments from Dr. Lemen. I believe he said -- I
10 believe it was him that -- and he's been following
11 asbestos for five decades. And every decade, the
12 levels at which cancer is caused by asbestos go
13 down as the measurements get better and the
14 datasets get bigger and that each and every decade
15 they find effects at lower and lower and lower
16 cumulative exposures. So that has been the data,
17 and I wanted to just be clear about that. Thank
18 you.

19 **DR. KENNETH PORTIER:** Dr. Loomis,
20 and then I think we'll go on to another topic.
21 Dr. Loomis?

1 **DR. DANA LOOMIS:** Oh, I forgot to
2 put my hand down. Sorry. I don't have anything
3 else.

4 **DR. KENNETH PORTIER:** Dr. Bateson?
5 Good. His hand is down. Okay. Dr. Kanarek, why
6 don't we move to another issue.

7 **DR. MARTY KANAREK:** Many commenters,
8 starting with the public, said that the modeling
9 was done on mortality. And they pointed out that
10 especially for lung cancer survival time has
11 increased over the years and that maybe EPA should
12 do a revision that includes modeling for
13 incidence. So that's the topic.

14 **DR. KENNETH PORTIER:** Okay. And I
15 know this -- in the DRE it says the endpoints for
16 both mesothelioma and lung cancer was mortality,
17 not incidence. Incidence data are not available
18 for any of the cohorts. So I just wondered -- in
19 theory, it's nice to have that. But if there's no
20 data, what can you do? Anyone on the panel wish
21 to comment? Dr. Stayner, you want to add to this?

1 **DR. LESLIE STAYNER:** Yes. One thing
2 to keep in mind is, during the time period of the
3 studies that we're talking about -- which were
4 more than 20 years ago -- there was really no
5 difference in incidence and mortality for cancer
6 or mesothelioma. So as far as the data that we're
7 using, if we had incidence, it probably would have
8 turned out to be the same as mortality, the dose
9 response. One option, though, of addressing this
10 would be to, in the life table analysis, using for
11 background rates -- we could use lung cancer
12 incidence rather than mortality in order to get
13 estimates for today that reflect the increase in
14 risk for incidence.

15 **DR. KENNETH PORTIER:** I had noticed
16 the quote about mortality and incidence rates
17 possibly being close for lung cancer 20 years ago.
18 And I went back and looked at the statistics, and
19 you're pretty correct on that. The real spreading
20 is more in the last decade since we've gotten many
21 more effective therapies for many of the lung

1 cancers. But 20 years ago, that was not
2 available. Dr. Anderson?

3 **DR. HENRY ANDERSON:** He took my
4 suggestion of maybe taking a look at the life
5 table using incidence there. The other option --
6 and I think Kenny talked about that and some of
7 the other relationships -- is you could look at
8 over the years using the tumor registries that go
9 back quite a ways to look at the incident data.
10 And they will also have what the cause of death
11 was listed on the death certificate for mortality.
12 So you could look at the ratio between incidence
13 and mortality through the years and use that as an
14 adjustment factor. But I think the life table is
15 a little easier to do and just see if that makes
16 any difference.

17 **DR. KENNETH PORTIER:** Dr. Bateson,
18 you want to add something?

19 **DR. THOMAS BATESON:** Yeah. Thank
20 you. Tom Bateson, EPA. I recall that when we did
21 the Libby amphibole assessment, we did do this
22 exact suggestion of replacing mortality rates for

1 lung cancer with the incidence rates. And my
2 recollection is that it made about a 10 percent
3 difference in the estimated unit risk for lung
4 cancer. So I think that that's a good suggestion,
5 and that's definitely something that we could do.
6 So we could look at that.

7 **DR. KENNETH PORTIER:** Dr. Crump?

8 **DR. KENNY CRUMP:** Well, I keep
9 following Dr. Stayner, and I have to say again I
10 think he's got a good suggestion just to use the
11 incidence data in the life table analysis. But I
12 would like to add that the way it's done in the
13 current draft report, they adjust it for this by
14 just taking a larger -- what the highest lung
15 cancer potency factor that they've got from any
16 study. And I basically think that's not a good
17 idea.

18 If you're going to try to adjust
19 something like this, I think you ought to need to
20 use the data that speaks to that thing you're
21 trying to adjust, not just adjust something else
22 and think you've made an adequate adjustment. I

1 think the idea of maybe using the incidence data -
2 - or maybe it's another approach -- but use
3 something that uses the data on incidence versus
4 mortality to adjust for this.

5 **DR. KENNETH PORTIER:** Thank you.

6 Dr. Markowitz, your hand went up and then went
7 back down again. And it's back up again.

8 **DR. STEVEN MARKOWITZ:** Sure. So
9 just to put a number on this, I looked back at the
10 CR data from approximately 1980 -- excuse me. And
11 there was about a 15 percent three-year survival
12 for lung cancer. So it wasn't -- whereas now,
13 it's closer to 25 percent. So it wasn't trivial
14 back then, nor was it very large, unfortunately.
15 But that gives some sense of the magnitude.

16 **DR. KENNETH PORTIER:** Thank you.

17 One of the things that I want to mention right now
18 that kind of ties in with the model fitting that I
19 thought of and I've been looking for -- I'm going
20 to provide a reference to a model fitting
21 technique that allows you to look at the impact of
22 some of these influential points. It's something

1 I've thought about a couple of times as we've come
2 across model fitting and we have this discussion
3 about how we'd like to see more emphasis on better
4 fits in the lower doses and less emphasis on the
5 fits in the higher doses. And that was an
6 approach proposed about a decade ago that allowed
7 you to at least do a series of sequential fits
8 that allowed you to gauge the impact of that. And
9 I'm going to find that and include that and send
10 that to Dr. Kanarek so we can kind of include that
11 as just a reference and a suggestion for an
12 approach that might allow you to -- allow EPA to
13 kind of at least gauge the impact of those higher
14 doses on the estimation of the point of departure.
15 Dr. Kanarek, any additional issues?

16 **DR. MARTY KANAREK:** Unless any of
17 the associates think we left something important
18 out. I don't want to step on the toes of the --
19 all the following groups. I think we've done
20 enough. Any of the associates want to chime in
21 that we haven't?

1 **DR. KENNETH PORTIER:** I'm looking
2 through your list. Did we talk about the
3 acceptability of potency factors from older
4 literature?

5 **DR. MARTY KANAREK:** We didn't.

6 **DR. KENNETH PORTIER:** Some
7 commenters thought it was inappropriate to accept
8 potency factors from the literature without
9 checking them. I just wanted to make sure we had
10 that discussion.

11 **DR. MARTY KANAREK:** Yeah. We
12 haven't. Some of the commenters thought they
13 should be checked because EPA took the potency
14 factors directly from the literature. That would
15 be our last issue if people want to comment on
16 that.

17 **DR. KENNETH PORTIER:** Dr. Sheppard?

18 **DR. ELIZABETH SHEPPARD:** So I
19 actually wanted to return to something else, so
20 maybe I should wait?

21 **DR. KENNETH PORTIER:** We'll allow
22 the Committee to be thinking about the potency

1 factor issue. It was just a one liner, so maybe
2 it's not that big. Or maybe it'll come up in the
3 remaining discussion today. Why don't you go
4 ahead and move on with your issue?

5 **DR. ELIZABETH SHEPPARD:** So I just
6 wanted to clarify why there was not review and
7 discussion of mechanics cohorts in this document.

8 **DR. KENNETH PORTIER:** So you're just
9 saying that you see that as a hole -- as an unmet
10 need that this should have been included. Can you
11 suggest a section where that might have needed to
12 be included, Dr. Sheppard?

13 **DR. ELIZABETH SHEPPARD:** Well, I'd
14 have to go back and look at the organization of
15 the document directly, but we ended up focusing on
16 textile cohorts. And I think that from all the
17 discussion we've had the exposure to mechanics is
18 more relevant to the exposure that we care about
19 in terms of this risk assessment. So I can see
20 lots of reasons to not talk about the mechanics
21 cohorts, but I think it's a gap in the document
22 that we just focus on the textile and mining

1 cohorts and don't really talk about the mechanics
2 cohorts, unless I missed something. I think that
3 needs to be addressed.

4 **DR. KENNETH PORTIER:** So I'm looking
5 at the table of contents in Section 3 on hazard
6 effects -- Section 3.2, human health hazards. And
7 what I don't see is a section discussing
8 assumptions and uncertainties and kind of
9 summarizing that. That might be one place where
10 there could have been additional discussion about
11 how the inhalation unit risk for chrysotile
12 asbestos is, in this case, only available -- the
13 risk estimate is only available for that cohort.
14 And what does that imply? It may be part of the
15 risk characterization. I'd have to double check
16 that. Maybe Section 4.3.5? But we'll look into
17 that.

18 Dr. Kanarek, please make a note that
19 we need to think about first research that when we
20 get a break. And if we can't find it, we'll need
21 to go ahead and point that out. I'm not hearing
22 any takers on the potency factors discussion.

1 We'll kind of hold that in the reserve. And I
2 think at this point I'd like to move on to
3 Question 4.3 if we can -- yes. Dr. Scarano, would
4 you read that in? And thank you, Dr. Kanarek.
5 Excellent job of organizing and helping us get
6 through what I think is one of the most important
7 questions that we've had to address so far. Dr.
8 Scarano?

9
10 **CHARGE QUESTION 4 (4.3)**
11

12 **DR. LOUIS SCARANO:** Thank you, Dr.
13 Portier. Question 4.3, please comment on EPA's
14 approach to characterizing the implications of the
15 assumptions and uncertainties for the confidence
16 associated with the derivation of the IURs, which
17 is Section 4.3.5 in the draft risk evaluation.

18 **DR. KENNETH PORTIER:** Thank you.
19 Dr. Sheppard, you have the lead on this.

20 **DR. ELIZABETH SHEPPARD:** Okay. So
21 let's see. I'll cover our comments in a very high
22 level, and then I can go into more details in the

1 write up that I shared with the associates
2 yesterday. So EPA has arguments about four
3 different uncertainties, one exposure, and three
4 endpoint related exposure measurement omitting
5 other cancers, mortality versus incidence, and the
6 IUR only characterizing cancer risk.

7 And then we thought there were some
8 other uncertainties that weren't addressed by EPA.
9 So one of them was this impact of using AIC as the
10 model fit criterion for choosing the exponential
11 model and then the choice of the exponential
12 rather than the linear rate model and the
13 mesothelioma potency adjustment approach and
14 addressing under ascertainment of mesothelioma.
15 And then the last major topic that we talk about
16 was addressing biases and the direction of biases.

17 So one major point is that we didn't
18 think it was appropriate for EPA to use one bias -
19 - the presence of one bias to address or
20 compensate for other sources of biases, which was
21 done in the document. And then we talked about
22 direction of biases. And there are some that are

1 likely downward biases, and there are some that
2 are possible upward biases.

3 And so for the likely downward
4 biases, we identified only focusing on
5 mesothelioma and lung cancer and omitting other
6 cancers using mortality instead of incidence, the
7 IUR only characterizing cancer risk, the form of
8 the risk model suggesting that it would be slower
9 with the exponential risk model, not considering
10 dermal exposures, and also exposure measurement
11 error in the cohort studies, which is something
12 that I added this morning. As far as possible
13 upward biases, there's the contamination of
14 asbestos by more toxic forms than chrysotile.
15 Fiber potency is a function of length and width
16 and fiber length is a function of activity,
17 specifically changing of brakes. So that's the
18 high-level summary. I can go into a little bit
19 more detail on some of these.

20 I wanted to say, with respect to the
21 arguments that EPA presented with respect to the
22 exposure measurement, I would say we had multiple

1 perspectives there, as this might be worth
2 discussing a little bit. Some of us seemed
3 reassured by their discussion about the comparison
4 of PCM versus the TEM. And others of us were not
5 convinced by that argument and were concerned that
6 the thin, short chrysotile fibers are commonly
7 missed by PCM. But they're visible by TEM. And
8 the importance of the thinness of asbestos fibers
9 for lung diseases, and the treating in PCM of all
10 fibers longer than five microns as the same
11 regardless of length. And then the factor of
12 textile productions.

13 So I can elaborate on several of
14 these points that I just gave the high-level
15 summary of, or we can open it up for discussion
16 and come back to some of it.

17 **DR. KENNETH PORTIER:** So right now,
18 all I see is Dr. Crump's hand up. I'll call on
19 Kenny. And anyone else on the Committee, please
20 raise your hand if you want to comment on any of
21 these. Dr. Crump?

22 **DR. KENNY CRUMP:** Can you hear me?

1 DR. KENNETH PORTIER: Yes.

2 DR. KENNY CRUMP: Oh, okay. I
3 actually thought that our chairman did a really
4 good job laying that out just a few moments ago.
5 I had a process question, actually. I have a list
6 of things that I think are mistakes in the draft
7 report. And I understand that I have to say those
8 publicly in order to get them in the -- have
9 considered. Is that right? If not, I can --

10 DR. KENNETH PORTIER: So Kenny --

11 DR. KENNY CRUMP: If I don't need to
12 do that, I wouldn't want to take the time.

13 DR. KENNETH PORTIER: Kenny, this is
14 Ken Portier. If they're primarily editorial, you
15 can just say, "I've got some editorial comments
16 that we'll include at the end of the minutes."
17 And we'll just capture those from all of the
18 Committee members and put them on. We typically
19 have decided not to get into primarily editorial
20 comments. If the statement changes the -- if the
21 correction changes the intent of the statement or
22 you feel like the statement is wrongly stated,

1 then it might be more important to actually state
2 that at this point. Are these primarily editorial
3 comments, misspellings, commas in the wrong place?

4 **DR. KENNY CRUMP:** No. I think
5 they're a little bit more important than that, but
6 I don't particularly desire to take the group's
7 time to point them out. But I do want them in the
8 record, though.

9 **DR. KENNETH PORTIER:** Give me an
10 example of one. Go ahead and say one, and let's
11 get a feeling for that.

12 **DR. KENNY CRUMP:** Okay. Most of
13 these deal with the -- excuse me -- the DRE
14 misstated several things about the Berman and
15 Crump 2008 analysis. There's a whole list of
16 those, actually, and I just wanted to get those
17 corrected.

18 For example, here's one. Line 4993
19 on modeling and mesothelioma data, it states
20 "Crump and Berman provided estimates for Quebec
21 miner from analyses of original, individual level
22 data," which is correct. But it also says,

1 "Berman and Crump provided estimates for South
2 Carolina using group data." That's not correct.
3 We provided estimates also for Hein et al, using
4 the individual data. So it's just things like
5 that.

6 **DR. KENNETH PORTIER:** I'll let the
7 DFO override me, but I would say that that's still
8 kind of editorial comments and that we'll include
9 them. We typically -- after we've had the minutes
10 written, we have editorials and corrections. And
11 we just kind of list page and line number and say,
12 "This is wrong. It should say this," or "You said
13 no, and you said have said yes." Those kind of
14 things we typically include at the end. So I can
15 ask -- I'll talk with Diana during the break and
16 we'll figure out whether we need to read those in.
17 The other thing is, Kenny, they can be put in at
18 the last question. Okay?

19 **DR. KENNY CRUMP:** If you have time,
20 you mean?

21 **DR. KENNETH PORTIER:** Well, we'll
22 make time. We'll make time.

1 **DR. KENNY CRUMP:** Whatever you
2 decide is fine with me. I just wanted to get
3 those corrections in.

4 **DR. KENNETH PORTIER:** Okay. Back to
5 Dr. Sheppard. I don't see any hands up, so I
6 think it would be good if you read in a little bit
7 more of the comments. One suggestion, I know
8 you're probably on a conference phone. I think if
9 you could pick up the phone and talk at least for
10 this part it would be a little clearer for us.

11 **DR. ELIZABETH SHEPPARD:** Okay. Is
12 that better?

13 **DR. KENNETH PORTIER:** Oh, much
14 better. Thank you.

15 **DR. ELIZABETH SHEPPARD:** Yeah.
16 Okay. No problem. Let's see. So the EPA's
17 arguments with respect to exposure measurement, I
18 went into some depth about that and that there
19 were different perspectives among the associates.
20 With respect to omitting other cancers, EPA argued
21 that the numbers are insufficient to add other
22 cancers, such as laryngeal and ovarian. But we

1 felt like that was of concern when it was coupled
2 with the undercounting of mesothelioma risks and a
3 reliance on mortality rather than incidence data,
4 suggesting together there's a source of
5 undercounting that operates only in the direction
6 of the IUR being too low.

7 So -- mortality rather than
8 incidence, I think given the discussion we just
9 had, maybe this should be updated because the
10 survival of lung cancer was not as impacted as it
11 has been in recent years. The document also made
12 the argument underestimation for mesothelioma has
13 been directly accounted for. And we come back to
14 that, and I may ask Dr. Crump to elaborate on that
15 when I get to that or afterwards because this
16 hasn't gotten a chance to pull that into the
17 summary that I wrote up.

18 So the IUR only characterizes cancer
19 risk, and EPA makes the argument that cancer is
20 the risk driver. But that ignores that noncancer
21 risks are also important. And together they would
22 produce a more comprehensive and informative IUR.

1 And also we note that the text in this section's
2 really difficult to follow.

3 We had discussion about model --
4 with respect to the other uncertainties not
5 addressed by EPA, we had discussion about the role
6 of using AIC to choose the exponential over the
7 linear model and concerns about that already. And
8 the choice of the exponential model, it says here
9 that it has major implications. But as Dr. Crump
10 pointed out, it's maybe only a factor of, what,
11 1.4 if I remember that number correctly. But
12 still, that is -- would be a bigger IUR estimate
13 if we use linear rate model.

14 And there were some arguments from
15 Berman and Crump that Dr. Crump wrote about that
16 suggest that the assumption that mesothelioma risk
17 varies linearly with exposure was firmly rejected
18 by some other datasets, and Australia and Quebec
19 and the best fitting models had exposure exponents
20 of less than one. And this nonlinearity, if it
21 continues throughout the low dose region, it would
22 mean that mesothelioma risks from very small

1 exposures is underestimated in the risk
2 assessment. And that should certainly be
3 discussed, and this could have a larger impact on
4 risk estimates for mesothelioma. I'm going to ask
5 Dr. Crump to weigh in about mesothelioma potency
6 adjustment and addressing the under ascertainment.

7 Do you want to -- maybe I'll go on
8 with talking about the biases, and then we can
9 come back to that? So using one bias to address
10 another, we thought that was problematic. EPA
11 compensated for two sources of bias by selecting
12 the largest IUR from among four candidates, Table
13 3-12, stating that this largest estimate was most
14 likely cover the total risk of incident cancers.
15 But there's no connection between the likely
16 effect of the two biases identified in the DRE and
17 the largest IUR. So it would be more appropriate
18 to use data related directly to the two biases to
19 estimate their effect.

20 The distribution of times from
21 diagnosis until death from mesothelioma could be
22 used to estimate bias in the use of mortality

1 statistics, and information on the risk of other
2 cancers compared to lung cancer and mesothelioma
3 in studied cohorts could be used to modify the IUR
4 accordingly. We recommend that the biases be
5 compensated for using data that informed the
6 amount of biases to the extent possible. And
7 then, with respect to direction of biases, I don't
8 have any elaboration here on the likely downward
9 biases.

10 But on the possible upward biases,
11 with respect to asbestos contamination,
12 chrysotile's typically contaminated with small
13 amounts of amphibole. There's a long history of
14 debate about whether mesothelioma or cases
15 associated with chrysotile exposure are due to
16 chrysotile itself or caused by contamination from
17 amphibole. With respect to fiber potency as a
18 function of length and width, potency of fibers
19 depends on their length and possibly their width,
20 with longer fibers being more potent. And the RE
21 should discuss the likely difference in length of
22 fibers in the North and South Carolina textile

1 mills compared to fibers to which workers are
2 exposed in the various jobs being evaluated and
3 the implications of these to the accuracy of the
4 estimates of risk in the RE.

5 And then finally, fiber length as a
6 function of activity, specifically changing
7 brakes, the relationship of the fibers in asbestos
8 used in making brakes to the fibers in the
9 airborne dust produced when replacing brakes
10 should be addressed. The degradation from using
11 brakes will result in shorter fibers overall than
12 those in the original asbestos material. So
13 that's -- those are my main points, but I don't
14 have elaboration compiled yet for the mesothelioma
15 comments. Maybe Dr. Crump wants to weigh in on
16 that.

17 **DR. KENNETH PORTIER:** Dr. Crump?

18 **DR. KENNY CRUMP:** Am I not muted?

19 You can hear me?

20 **DR. KENNETH PORTIER:** No, we can
21 hear you. Go ahead.

1 **DR. KENNY CRUMP:** Okay. Yes, in the
2 Berman and Crump analysis in 2008 we looked at
3 several predictions of these models, and we ran
4 tests to see if they are borne out by the data.
5 We checked to see if the linear model was --
6 excuse me, dose response model for both cancer and
7 mesothelioma. There's a power of three in the
8 Peto mesothelioma model.

9 We checked to see if another factor
10 might be better than three. And we checked to see
11 -- both of these models, the mesothelioma and the
12 lung cancer assume that risk continues to increase
13 long after exposure ends. And that was
14 particularly entertaining to me. So we tested
15 that to see if the data bore that out.

16 And what we found was, yes, even for
17 50 years, past the end of exposure, the risk tends
18 to increase both with mesothelioma and lung cancer
19 in those data. The only effect that we found to
20 be significant was the linearity of the
21 mesothelioma. We add a parameter to the Peto
22 model to check for the linearity with exposure.

1 And we tested that using the Wittenoom data at 222
2 mesotheliomas. And we also checked it with the
3 Quebec data. We had 35 mesotheliomas there.

4 In both of those analyses, there was
5 a highly significant effect that the power on
6 those was significantly less than one, which would
7 make a super-linear dose response at low dose.
8 And if that continued into the low dose region, it
9 would mean that we're underestimating the risk
10 from mesothelioma. I don't know if anyone else
11 has ever looked at that, but that's what we found.

12 **DR. KENNETH PORTIER:** Thank you, Dr.
13 Crump. I noticed in Dr. Sheppard's written
14 document that there were comments by other
15 Committee members that she may or may not have
16 included. Does anyone want to add their comments
17 to this discussion? Any Committee member?

18 I tell you what. I see 12:00 noon
19 Eastern Time on my clock, and our schedule says
20 we're scheduled to take a break. Why don't we
21 kind of break at this point, and we'll come back
22 to finish up this question, 4.3 and then 4.4? So

1 let's see. So it's 9:00 Pacific Time. I guess we
2 probably should go ahead and take a 15-minute
3 break and let our colleagues on the West Coast get
4 a decent breakfast or second breakfast. So let's
5 return at 12:15, please.

6 **[BREAK]**

7 **DR. KENNETH PORTIER:** Okay. I have
8 12:15. Let's reconvene, please. Go back to Dr.
9 Sheppard and any of the Committee want to add
10 anything to the discussion on 4.3 related to the
11 characterizing and implications of the assumptions
12 and uncertainties for the confidence associated
13 with the IURs? Any additional comments? Dr.
14 Sheppard, is there anything you wish to add before
15 we end this discussion and move to Question 4.4?

16 **DR. ELIZABETH SHEPPARD:** No, I think
17 I covered everything in my notes. So I think
18 we're okay. The other Committee members, I tried
19 to capture everything that the other Committee
20 members sent me. So hopefully they have been well
21 represented.

1 DR. KENNETH PORTIER: Thank you.

2 Dr. Markowitz?

3 DR. STEVEN MARKOWITZ: A couple of
4 comments that I think relate to Dr. Sheppard's
5 summary. One was there was some discussion about
6 an upward bias in the estimate due to
7 contamination of chrysotile by tremolite. And my
8 reading of this entire document -- and I think it
9 says it somewhere, but I'm not sure where -- is
10 that the issue they're looking at is commercial
11 chrysotile, which very frequently is contaminated
12 by tremolite. So that distinction isn't made
13 throughout the document.

14 The other issues is what was
15 mentioned was upper bias relating to fiber potency
16 by size of fiber. And in fact, the study by Hamra
17 in 2017 -- Dr. Loomis is a co-author, so he may
18 want to comment. I think although Dr. Loomis
19 addressed this a couple of days ago -- which is
20 the result of that study, which looked at TEM
21 versus PCM for potency, and they concluded that
22 they could not find substantial differences in the

1 effect on lung cancer by size of fiber. So there
2 is some specific results in the literature that
3 address this issue that was raised.

4 **DR. KENNETH PORTIER:** Good. Any
5 additional comments? Dr. Kaufman?

6 **MR. ALAN KAUFMAN:** Yes, can you hear
7 me?

8 **DR. KENNETH PORTIER:** Yes, I can.

9 **MR. ALAN KAUFMAN:** Okay. Yeah. I
10 don't know whether this is 4.3 or 4.4. But I'll
11 throw it out there at this point, and we can defer
12 the conversation. The other source of bias that
13 occurred as we were looking through this was,
14 aside from the fact that we've used chrysotile as
15 a proxy for all asbestos types, even though we
16 agree it's probably not the most potent initiator
17 of the health effects -- but we understand why
18 that was done. The data cohort was most readily
19 available and was probably of the best quality,
20 even though there are some obvious gaps there.

21 But the other piece -- and I had to
22 step away for a few, so I'm not sure if somebody

1 else mentioned this -- was the fact that there was
2 an assumption that the background level or
3 background incidence of mesothelioma was zero.
4 And just a quick look at the American Cancer
5 Society's website indicates that approximately 20
6 percent of mesothelioma cases are in individuals
7 that have no known exposure to asbestos. So I
8 think that's a source of bias we need to think
9 about as well. And that was it.

10 **DR. KENNETH PORTIER:** Thank you,
11 Alan. I think Dr. Sheppard mentioned that and has
12 some of that written up. Is that right, Dr.
13 Sheppard?

14 **DR. ELIZABETH SHEPPARD:** Let's see,
15 I guess -- I'm sorry. Can you hear me?

16 **DR. KENNETH PORTIER:** And maybe we
17 talked about that in 4.2, not 4.3.

18 **DR. ELIZABETH SHEPPARD:** I think we
19 didn't talk as much about the zero background risk
20 in our response, so we can add that to the
21 possible biases.

1 **MR. ALAN KAUFMAN:** Yeah. And I
2 think Dr. Crump also has it in 4.4 when we get
3 there as well.

4 **DR. KENNETH PORTIER:** Dr. Loomis,
5 you have a clarifying comment?

6 **DR. DANA LOOMIS:** I do, only a brief
7 comment on tremolite. It is, as we've said, true
8 that commercial chrysotile contains some amphibole
9 asbestos, and that is typically tremolite. But in
10 most of the cohorts, the tremolite content of the
11 material used is quite small. So unless you're
12 willing to assume that the unit risk from
13 tremolite is much larger than that from
14 chrysotile, which is not at all clear for lung
15 cancer, at least, the potential for upward bias
16 from the small amount of tremolite in the
17 commercial chrysotile is probably negligible.

18 **DR. KENNETH PORTIER:** Thank you.
19 Dr. Kaufman, your hand's still up. Did you want
20 to follow up?

21 **MR. ALAN KAUFMAN:** No, sorry. I
22 forgot to take it down.

1 **DR. KENNETH PORTIER:** Dr. Everitt?

2 **DR. JEFFREY EVERITT:** Yeah. I have
3 a comment. I was curious about the bias that
4 could occur from what Dr. Roggli brought up that
5 some of that North Carolina and South Carolina
6 cohort may have been exposed to amphibole, and it
7 wasn't adequately discussed in the background for
8 that study. And that could be potentially much
9 higher than small amounts of tremolite in
10 chrysotile in the lungs that he's looked at fiber
11 counting on that he discussed in the public
12 comments.

13 **DR. KENNETH PORTIER:** I see Dr.
14 Stayner has his hand up. Maybe he wants to
15 provide a clarification. Dr. Stayner?

16 **DR. LESLIE STAYNER:** Yeah. First of
17 all, on the tremolite issue, I don't really think
18 it's proper to think of that as an upward bias
19 because our model's based on exposure to
20 commercial chrysotile that has this small
21 percentage of tremolite. So we are estimating
22 exposure for commercial asbestos -- chrysotile

1 asbestos. So I don't really see that as a
2 problem.

3 I think you're referring though to
4 the comments by Dr. Garabrant on -- and I'll just
5 respond to the South Carolina. I'll leave it to
6 Dana to talk about the North Carolina allegations
7 of exposures to amphiboles. So he refers to a
8 paper by Sebastian where they found amphibole
9 fibers in the lungs of workers from the textile
10 plant. So there's no control group there, so it's
11 not really possible to say whether that's high or
12 low for -- but there is results in that paper for
13 the miners of Quebec. And actually, the miners of
14 Quebec seem to have higher exposures of amphiboles
15 in their lungs.

16 The other thing is -- I'm not sure
17 if we can introduce this or not. But Dr. Dement
18 shared with me he did an analysis looking at
19 workers who were first employed after 1940. Now,
20 in the paper by Sebastian, he found no evidence of
21 amphiboles in the lungs of workers who were
22 employed after 1940. And what Dr. Dement found in

1 the analysis is that the exposure response for
2 those employed after 1940 was maybe even a little
3 stronger than the entire cohort but certainly not
4 lower. So it's really strong empirical evidence
5 that the confounding by amphiboles was not an
6 issue in the South Carolina cohort. So anyway, I
7 think, Dana, maybe you would like to address the
8 North Carolina comments.

9 **DR. KENNETH PORTIER:** Dr. Crump?
10 Kenny, you're muted in WebEx.

11 **DR. KENNY CRUMP:** Okay.

12 **DR. KENNETH PORTIER:** Yes, I can
13 hear you now.

14 **DR. KENNY CRUMP:** Okay. This is a -
15 - well, let me get my stuff back. Yeah. Loomis
16 in 2010 used paired PCM and TEM samples to
17 estimate lung cancer potency in North and South
18 Carolina cohorts. I guess it's both North and
19 South -- anyway, in terms of concentration of
20 different lengths and widths measured by TEM and
21 compared the fit for TEM versus the fit with PCM.
22 And he found best fitting TEM model had an AIC 4.8

1 units higher than the PCM model. But the risk
2 evaluation does not report this difference, but
3 they say only that the PCM versus TEM was
4 generally equivalent about two AC units. And if I
5 read all that correctly, it just doesn't sound
6 quite right. So I wish that EPA would take a look
7 at that and see if they have that rest
8 appropriately.

9 In general, I think that this DRE
10 could profit by having a discussion of the
11 evidence in the literature. There's a lot of
12 evidence about the different potencies of fibers
13 of different lengths, and there's a lot of animal
14 data, a lot of human data. And I think that data
15 is important to what we're talking about. And I
16 think the DRE would profit by having a review of
17 those data.

18 **DR. KENNETH PORTIER:** Thank you.

19 Dr. Loomis?

20 **DR. DANA LOOMIS:** My comment was
21 about the potential exposure to amphibole fibers
22 among workers in the cohorts. I don't have very

1 much to add to what Dr. Stayner said except that
2 it is true that for these cohorts we don't have
3 information about exposure of the workers, except
4 in the industries we'd studied. But that is
5 typical of occupational cohort studies. So none
6 of the available cohorts will have that
7 information if workers were exposed to amphiboles
8 or some other carcinogens in another job outside
9 of the chrysotile using industries. They wouldn't
10 know about it, and it wouldn't be recorded in any
11 of the available studies.

12 The other thing I would say is that
13 Dr. Roggli's comment mentioned a case who worked
14 in one of the North Carolina plants who had
15 mesothelioma and had been exposed to amphibole
16 asbestos apparently by washing her husband's work
17 clothes. At least that was his interpretation.
18 But that individual is not counted as a
19 mesothelioma case in our analysis.

20 **DR. KENNETH PORTIER:** Dr. Crump,
21 your hand is up.

1 **DR. KENNY CRUMP:** I'm having trouble
2 putting it down, actually. I'm getting a little
3 bit of confusion of what we've talked about and
4 haven't. I had something on this section about
5 risk from replacing brakes. I think it presents a
6 unique issue for risk assessment because of the
7 unique fibers that people are exposed to. Have we
8 talked about the completely yet, or am I still
9 okay mentioning this?

10 Anyway, the DRE says in line 3295
11 that "Due to high friction environment and vehicle
12 braking, asbestos fibers in the brake material
13 degrade both chemically and physically." So I
14 think those make the fiber in the brake
15 replacement activity very different from those in
16 the textile mills of North and South Carolina.
17 And it will result in shorter fibers overall and
18 more -- very short fibers that aren't counted by
19 PCM.

20 So my guess would be using the
21 Carolina data to estimate risk in these groups,
22 the risk from fibers longer than five microns

1 would be overestimated because the fibers are much
2 longer in the Carolina cohorts than in these
3 situations. But the risks from fibers shorter
4 than five microns, if there is any -- I don't
5 think we have to do that but if it's the risk risk
6 -- but probably some risk, that would be
7 underestimated. So it's hard to say if we're
8 overestimating or underestimating the risks. I
9 expect we're overestimating it, but I can't be
10 sure about that. But anyway, I think that
11 discussion needs to be in the DRE.

12 And there needs to be, I think, a
13 review of the epidemiological studies on brake --
14 people working with brakes and these exposure
15 occupational people that are working in these
16 garages that work with brakes. There's a lot of
17 epi studies, apparently. I don't know much about
18 them. But it seemed to me that they need to be
19 reviewed at least in the draft risk analysis. And
20 if possible, we might should use those studies to
21 evaluate the risk from exposures to brake dust.
22 So I would make that as a recommendation.

1 **DR. KENNETH PORTIER:** Dr. Sheppard?

2 **DR. ELIZABETH SHEPPARD:** So just to
3 say that we did capture that. I've taken some
4 notes on those recent comments to try to maybe
5 expand that a little bit because we put it under
6 the possible upward bias. And from what I heard,
7 it could go in either direction, actually. And
8 the recommendation with respect to exposures and
9 risks from these activities I think is consistent
10 with what I was saying earlier about why is there
11 no discussion of studies and mechanics. There may
12 not be enough good data to use in this. And I
13 just think if that's the case then that just needs
14 to be addressed head on.

15 **DR. KENNETH PORTIER:** And Dr.
16 Kanarek?

17 **DR. MARTY KANAREK:** I just want to
18 point out in brake replacement there's two types
19 of exposure to asbestos. There's taking out the
20 old brake -- and I agree with Kenny that's a
21 different type of asbestos. It's undergone
22 heating and all that. But when you put in the

1 replacement brake, that asbestos has not been
2 heated, and often they grind it or sand it. So
3 they're exposed there to longer fibers.

4 And as to the epidemiology brake --
5 the brake literature -- the epidemiology brake
6 literature, there's been a lot written about it.
7 And in my opinion, it's very weak. The cohort
8 studies that could have been done in large brake
9 manufacturing facilities like Ford or others were
10 not done. It's not like -- we don't have very
11 good studies. We have case control studies that
12 really weren't designed to do anything about
13 brakes. But the question might have been asked.
14 So it's quite a discussion we could put in about
15 those studies, but it would require a lot of
16 critical thinking.

17 **DR. KENNETH PORTIER:** So just so the
18 Committee knows I'm looking for the opportunity to
19 move to Question 4.4, so I'd like to kind of wrap
20 this discussion up. Dr. Crump?

21 **DR. KENNY CRUMP:** Yeah. Like the
22 previous speaker just said, I'm not familiar with

1 those studies at all. But it just seems to me
2 that, on the surface, they look like they apply.
3 And I think they should at least be mentioned and
4 told why they're not used if that's what the
5 solution -- what the decision would be.

6 **DR. KENNETH PORTIER:** Thank you.

7 Dr. Kissel?

8 **DR. JOHN KISSEL:** Yeah. I asked a
9 question about the brake mechanic or auto mechanic
10 epidemiology on Monday because it was presented --
11 actually the same figures were shown to us by
12 three different people with clearly the intention
13 -- I don't know that anybody actually said it --
14 but clearly the intention was to declare that
15 brake dust doesn't cause mesothelioma. I haven't
16 had time to go -- it's a big list of studies. I
17 looked at one on the recommendation of one of the
18 commenters and also on the basis of the Garabrant
19 metanalysis.

20 One study is listed as most -- as
21 the highest quality and therefore most
22 authoritative. And it's the Rake et al, which is

1 data from the United Kingdom, so Peto is a co-
2 author on Rake. Rake is the journal submission of
3 the health and safety executive data on this
4 that's also available in report form, and it's the
5 same underlying data. Peto's the first author on
6 the HSE report.

7 That report finds no significant
8 elevation among auto mechanics against a
9 background -- which in the United Kingdom the
10 background mesothelioma risk is on the order of
11 ten to the minus three. That's explained in the
12 HSE report as a consequence of widespread use of
13 amosite in construction materials. But basically
14 what that finding is, is that auto mechanics
15 cannot be shown to be elevated above a ten to the
16 minus three level, whereas EPA is trying to
17 establish risk targets here of ten to the minus
18 four in the occupational sector and ten to the
19 minus six in the nonoccupational sector. And so
20 basically, that HSE study, which is touted as the
21 best of all the auto mechanic studies has no power
22 to be informative in the current debate.

1 DR. KENNETH PORTIER: Thank you.

2 Dr. Anderson?

3 DR. HENRY ANDERSON: Yeah. I would
4 just add to that the other problem that's occurred
5 -- and it goes way back to the very first reports
6 from Mount Sinai where I worked -- is that auto
7 mechanics -- 25 percent of them never reported
8 working with brakes. So you have an occupational
9 group that may or may not have had much brake
10 exposure. So that's part of the problem is the
11 terminology that's used in the large databases is
12 not specific in a lot of those studies to brake
13 workers.

14 There's really no brake worker good
15 definition. So you're including a lot of people
16 that didn't have exposure, didn't have very much
17 exposure. So that's part of the problem with a
18 lot of the case control studies. We just didn't
19 do a good job when these definitions were defined
20 in focusing on their asbestos exposure for
21 mechanics who work on all parts of the cars.

1 DR. KENNETH PORTIER: Thank you.

2 Dr. Kissel, I see your hand's still up. Dr.

3 Crump, final word?

4 DR. KENNY CRUMP: No, my hand

5 shouldn't be up.

6 DR. KENNETH PORTIER: You're just

7 having trouble taking your hand up and down. I

8 see.

9 DR. KENNY CRUMP: Here we go. Here

10 we go.

11 DR. KENNETH PORTIER: Got it. Okay.

12 I think at this point it's a good opportunity to

13 move on to Question 4.4. Dr. Scarano, would you

14 read it into the record, please?

15

16

17 CHARGE QUESTION 4 (4.4)

18

19 DR. LOUIS SCARANO: Sure. Thank

20 you, Dr. Portier. Question 4.4, please provide

21 any specific suggestions or recommendations for

22 alternative approaches that should be considered

1 by the Agency in deriving the commercial
2 chrysotile based IUR.

3 **DR. KENNETH PORTIER:** Thank you.

4 Dr. Crump, you have the lead on this question.

5 **DR. KENNY CRUMP:** Yes, it's getting
6 kind of late, and some of these issues I want to
7 mention have already been discussed. So I'm a
8 little bit confused about what I should talk
9 about. Let me just start out.

10 My first point is about the mode of
11 action, which that discussion kind of stands
12 alone. It's not mentioned anywhere else in the
13 DRE. And I think, typically, that information is
14 used in determining which dose response models and
15 decision about estimating low dose risk. So it
16 just seems to me there should be some tying of the
17 mode of action discussion to the rest of the
18 report. That's one comment.

19 Also, in the review of the DRE, I
20 think that the way it now stands the data was
21 selected -- the final data was selected based on
22 overcoming biases. And I think several people

1 have mentioned that's probably not a good idea.
2 So I think -- what should we use? So I would
3 suggest that maybe we should use the data on both
4 the North and South Carolina as appropriate basis
5 driving risk factors, at least for estimating risk
6 from fibers exposed -- the same kind of fibers. I
7 think that would give you a bigger database. I
8 think they have roughly equal support. And in
9 Eliot 2012, there was a combined analysis of the
10 two databases. Let's see.

11 There was a derive -- okay. EPA
12 derived an analysis on mesothelioma from the North
13 Carolina cohort. Berman and Crump did one from
14 the South Carolina cohort. Both of these are on
15 the individual data, so those could be combined
16 and come up with a combined risk of mesothelioma.
17 So I think that might be a better basis for the
18 final risk assessment than just using North
19 Carolina for one and South Carolina for the other
20 kind of cancer.

21 But I do have some questions about
22 the EPA's estimate of KM based on Loomis 2019. I

1 couldn't find that analysis. Maybe I missed it.
2 But I looked for it, and I couldn't find the basis
3 of that KM that was used for -- the analysis that
4 was used for the KM. And they said they based it
5 on Loomis et al, but I couldn't even find any data
6 in Loomis that looked like it would be a good
7 basis for doing the estimate because he doesn't
8 report the data in the way you would need to apply
9 the model to. So unless I missed something, I was
10 confused about what model that EPA finally used to
11 estimate the KM for mesothelioma. So maybe the
12 Agency could clear that up.

13 **DR. KENNETH PORTIER:** Dr. Bateson,
14 do you want to provide some clarity?

15 **DR. THOMAS BATESON:** Can you hear
16 me?

17 **DR. KENNETH PORTIER:** Yes.

18 **DR. THOMAS BATESON:** Yeah. I
19 believe the modeling results are in Appendix J.

20 **DR. KENNY CRUMP:** I looked there,
21 but I may have -- like I say, I may have
22 overlooked it.

1 **DR. THOMAS BATESON:** I will check
2 again at the next break.

3 **DR. KENNY CRUMP:** Okay. It doesn't
4 have to be that quick. Just when you're revising
5 the document, just keep that in mind.

6 **DR. THOMAS BATESON:** Thank you.

7 **DR. KENNETH PORTIER:** So Dr. Crump,
8 any additional -- Dr. Crump, any additional
9 alternate approaches?

10 **DR. KENNY CRUMP:** I still have a
11 bunch of points here. I'm still looking for --
12 but I don't see anything we haven't already
13 discussed. The charge question asks for alternate
14 approaches to the chrysotile based IUR. But the
15 differences among the chrysotile asbestos
16 encountered makes it very questionable as to
17 whether a single IUR is appropriate. That's
18 something we've already discussed.

19 And particular problems in the
20 friction -- those people -- I think that's already
21 been discussed. At least looking at the --
22 mentioning the epidemiologic studies of garage

1 workers -- I think even if they're not good they
2 need to be mentioned and tell why they're not
3 used.

4 **DR. KENNETH PORTIER:** So while
5 you're looking, Dr. Sheppard, do you want to jump
6 in at this point?

7 **DR. ELIZABETH SHEPPARD:** Yeah. I
8 wanted to take this opportunity since it came up
9 to talk a little bit about Appendix J, which is
10 page -- starts on page 293 of the document if
11 anybody wants to look at it. And if the web
12 people wanted to put that page up so that people
13 could see it easily, that would be helpful. So
14 it's hard to understand because there's like no
15 text. There's just tables and figures for each
16 study.

17 And I thought it was valuable to
18 show this group linear analysis of the published
19 data in Hine, Loomis, and Wang. But I was
20 profoundly concerned by the use of the midpoint of
21 the category for all categories. I don't really
22 think it's that much of a problem for anything but

1 the highest category. But the highest category is
2 a huge problem because it's highly influential on
3 the results. And the midpoint is probably
4 representing the 99.9th percentile of the
5 distribution because the data are so highly
6 skewed.

7 So it would make much more sense to
8 use the lower end of the highest category or maybe
9 a 10 percent inflation on that. Or if you could
10 get the median for the person years in that
11 category, that would be a useful alternative
12 number. But the midpoint of the lowest -- of the
13 bottom of the range and the maximum in the dataset
14 is just unrepresentative of the exposure in that
15 category and highly influential on those results.
16 So I thought that was a useful exercise.

17 As far as I could tell it didn't do
18 anything but enter the tables as another set of
19 numbers and wasn't carried forward, so maybe it's
20 not that important. But it is relevant to this
21 discussion about alternative approaches. And if
22 this is going to be paid attention to in any

1 important way, it should be redone so that that
2 highest category doesn't have the kind of
3 influence in the analysis that it does.

4 **DR. KENNY CRUMP:** Yeah. I had
5 exactly the same point to make. In fact, if you
6 look at -- that's the analysis of Loomis, I
7 believe. So that particular estimate that they
8 got from that analysis is lower than the other
9 estimates, and it's probably -- maybe at least
10 partially due to the fact they use a very large
11 value for the exposure in the highest category.
12 And of course, the highest category is most
13 important probably than any of the other
14 categories. So that is an important point when
15 you're trying to use grouped data.

16 The problem is that you have these
17 data grouped by exposures, and each group we need
18 an estimate of a single exposure for the group.
19 So you can just take the midpoint. That's
20 probably good enough. But the highest exposure
21 group you can't do that because it's greater than
22 500. How much greater than 500?

1 And the analyses done in the DRE
2 took the largest value in the study, and that
3 probably really overestimates the exposures in
4 that group. And if you looked at, I believe,
5 Loomis -- I believe it's 2010 Loomis, one of the
6 Loomis studies -- he has a graph of the exposures.
7 And you can tell that the one that was used in
8 their analysis is much too high for that group.
9 Thank you, Lianne.

10 **DR. ELIZABETH SHEPPARD:** Yeah. I
11 just want to follow up briefly. I did some work
12 on this yesterday. And from the data that's
13 reported in Loomis 2009 it looks like the 90th
14 percentile of the distribution is down around --
15 it's a little bit higher than the low end of that
16 category range. So I would say something like on
17 that figure -- for that group linear analysis that
18 exposure midpoint should probably be somewhere
19 down around 160 and not up at 1700, which is what
20 was used. So that's a huge difference.

21 **DR. KENNY CRUMP:** Yeah. When we did
22 that analysis, we used -- I did an analysis of

1 that data -- I think I used 180 for the upper
2 bound. People have looked at this and given -- I
3 think they've given some advice for how to set
4 that. I think something says in my mind five-
5 thirds of the lower bound might be a reasonable
6 value for the upper bound -- upper exposure
7 category. And if you want to assume the data log
8 norm is distributed, you could probably make an
9 estimate from that assumption also or any other
10 particular assumption you wanted to make about the
11 distribution of the exposures.

12 **DR. KENNETH PORTIER:** So I think the
13 point's been made, and I think -- there we go.
14 And I think the idea is that in all three cases
15 that the choice of the upper bound tends to lower
16 the estimate, right? It pulls the estimate down.
17 So shifting this closer to 150, 180 will slightly
18 raise the result. And I'm assuming the slope here
19 is the KL estimate. So the KL estimate will go
20 up. But I think the point's been made.

21 **DR. KENNY CRUMP:** If you lower the
22 upper bound. Right.

1 **DR. KENNETH PORTIER:** Yeah. So does
2 anyone else want to -- I see Dr. Loomis. Your
3 hand is up. Did you want to clarify?

4 **DR. DANA LOOMIS:** I would. So I
5 think this is an interesting discussion about the
6 slope in North Carolina. One thing that we've
7 observed consistently in those cohorts is that,
8 although nobody's mentioned it, we also fit spine
9 curves based on a continuous exposure variable
10 instead of group data. And we see that the slope
11 flattens quite a bit at the highest exposure
12 levels.

13 Interestingly enough, we don't see
14 that in South Carolina, but the thing to remember
15 is that the North Carolina plants were much
16 dustier than South Carolina. South Carolina was
17 kind of the test case for exposure-controlled
18 technology at the time, and that wasn't applied in
19 North Carolina until much later. So the mean
20 exposures and the exposure distributions are
21 considerably higher in North Carolina.

1 And as a result -- well, I don't
2 know if it's a result or not. But in parallel
3 with that, we see this flattening of the slope at
4 the highest exposure levels. And this probably
5 also has some bearing on the discussion right now
6 about which cut point to use for the upper group
7 with group data. I would add that this kind of
8 flattening of the slope is not really unusual in
9 occupational cohort studies. We see it with a lot
10 of different agents. So it's different from the
11 situation for the South Carolina cohort but not
12 really remarkable otherwise.

13 **DR. KENNETH PORTIER:** This is Ken
14 Portier. But it's a little bit difficult to
15 really explore that when you have group data.
16 That's always the most difficult thing. Now, am I
17 to understand, though, you had individual data for
18 some of this, right? But that wasn't used to fit
19 this -- to come up with these KL estimates?

20 **DR. DANA LOOMIS:** I'd have to let
21 EPA answer that question. I have individual data,

1 but EPA would have to answer the question about
2 model on the figures shown here.

3 **DR. KENNY CRUMP:** Let me just add
4 that it wasn't clear to me whether they -- which
5 was used for the mesothelioma data. It didn't
6 seem like there's enough information in the study
7 to do the group data, so maybe they did use the
8 individual data. But I couldn't figure out which.

9 **DR. KENNETH PORTIER:** Yeah. The
10 Appendix J data -- Appendix J is only mentioned
11 three times in the DRE, and there's really not --
12 there's no text in the Appendix. And there's very
13 little discussion. And it seems to me that it was
14 put in there to illustrate certain things. But I
15 still think a paragraph or two in Appendix J to
16 kind of describe what is done here and what's the
17 purpose of this analysis would help if you're
18 going to keep Appendix J in the DRE. So that's
19 just a point. Dr. Kopylev? Your phone may be
20 muted, Dr. Kopylev.

21 **DR. LEONID KOPYLEV:** I apologize.
22 Dr. Crump did pick up on something that we should

1 indeed correct. I think that J at some point
2 contained results of the modeling for North
3 Carolina. It's oversight that it doesn't in the
4 final -- in the draft report. It should have been
5 there.

6 **DR. KENNETH PORTIER:** Okay. So I
7 think the point's been made here. Dr. Crump,
8 anything else on Question 4.4?

9 **DR. KENNY CRUMP:** Well, I have one
10 other thing. I have found one other thing that we
11 talked about some but maybe not completely, and
12 that's whether the mesothelioma has background or
13 not. The Peto model does not provide for
14 background, but I'm kind of agnostic as to whether
15 there's background or not. But maybe EPA should
16 modify the Peto model to include background and
17 see what they get. It seems to me it shouldn't
18 make much difference, but that might be worth
19 doing just to satisfy the public.

20 Also, the Peto model really assumes
21 you have constant exposures, and Berman and Crump
22 2008A introduce and extension of the model which

1 allows exposures to vary with time. But it
2 becomes the Peto model when exposure is constant.
3 So I think EPA should at least consider putting
4 out that extension and even consider extending it
5 to include a background. I think this is about
6 all I can see that hasn't been discussed elsewhere
7 -- other times, so I'd like to invite the other
8 people that provided input to make comments. I
9 know I received some comments this morning, but I
10 haven't had time to digest and haven't mentioned
11 at all. So please speak up.

12 **DR. KENNETH PORTIER:** Dr. Kopylev, I
13 see your hand still up. So Dr. Crump, I'm not
14 seeing any hands fly up at this point. I think
15 you've covered a lot of the topics, at least that
16 I saw.

17 **DR. KENNY CRUMP:** Okay. I think
18 I've said all I need to say.

19 **DR. KENNETH PORTIER:** I'm just
20 checking to see. Okay. Okey-doke. I think at
21 this point we've kind of run out everything we can
22 on Question 4. Dr. Scarano, I think we've had

1 some good discussion on this, and I really
2 appreciate the four EPA staff that have kind of
3 helped in this discussion.

4 It's not typical in these Committee
5 reviews that we have this kind of dialogue with
6 EPA scientists. But in this case, I appreciated
7 it because the issues are very complex, and not
8 all of the panel are as expert with the theory or
9 the data. So I think that's helped clarify some
10 things. I think the Committee has identified a
11 couple of places where we can recommend
12 improvements to the risk assessment, and certainly
13 we're going to include that in the write up.

14 Do you have any final comments
15 before we break for lunch here? And after lunch
16 we'll start on Charge Question 5. Dr. Scarano?

17 **DR. LOUIS SCARANO:** Thank you, Dr.
18 Portier. No, I just want to echo the fact that
19 we're lucky and proud to have the staff we have
20 and the epi experts, Drs. Loomis and Stayner, to
21 help us with this very difficult assessment. And
22 I also agree that it was a robust discussion. And

1 I do want to apologize because there does seem to
2 be some material missing from Appendix J, so we
3 will take care of that. But thank you. I was
4 writing furiously during the whole time. I
5 thought it was a great discussion. Thank you.

6 **DR. KENNETH PORTIER:** Good. So our
7 agenda says we're breaking for lunch at 1:15 and
8 it's 1:05. But I think we'll go ahead and break
9 at this point. We're behind our agenda, but that
10 was kind of to be expected because I figured that
11 the discussion on Question 4 would be pretty
12 intense. And discussion on Question 5 will
13 probably continue. At this point, let's break for
14 lunch and return in an hour at 2:05. Is that
15 right? Oh, no. Return in 45 minutes. So that
16 should make us returning at 10 minutes to 2:00,
17 1:50. So we will return at 1:50 and begin with
18 Charge Question 1 -- 5.1. Thank you.

19 **[BREAK FOR LUNCH]**

20 **DR. KENNETH PORTIER:** Good
21 afternoon. I have ten minutes to 2:00. Let's
22 reconvene. I'm going to start by calling the

1 afternoon roll to establish who's here on the
2 Committee. Dr. Anderson? Dr. Bennett, I think
3 that's you. You need to mute your line. Thank
4 you. Okay. Dr. Anderson?

5 **DR. HENRY ANDERSON:** I am here.

6 **DR. KENNETH PORTIER:** Dr. Barton?
7 Dr. Bennett?

8 **DR. STEVEN BENNETT:** I'm here, and
9 hopefully that wasn't me making those noises. But
10 if it was, I'm off.

11 **DR. KENNETH PORTIER:** Okay. Thank
12 you. Dr. Blystone?

13 **DR. SHERI BLYSTONE:** I am here.

14 **DR. KENNETH PORTIER:** Dr. Cory-
15 Slechta?

16 **DR. DEBORAH CORY-SLECHTA:** I'm here.

17 **DR. KENNETH PORTIER:** Dr. Davies?

18 **DR. HOLLY DAVIES:** I'm here.

19 **DR. KENNETH PORTIER:** Dr. Doucette?

20 **DR. WILLIAM DOUCETTE:** I'm here
21 also.

1 **DR. KENNETH PORTIER:** I know Dr.
2 Jimenez-Gonzalez is going to join us a little late
3 and Dr. Johnson sends his regrets. He's out for
4 an hour and half this afternoon. Dr. Kaufman?

5 **MR. ALAN KAUFMAN:** I'm here.

6 **DR. KENNETH PORTIER:** Dr. Kissel?

7 **DR. JOHN KISSEL:** Here.

8 **DR. KENNETH PORTIER:** Dr. Rowlands?

9 **DR. CRAIG ROWLANDS:** I'm here. Good
10 afternoon.

11 **DR. KENNETH PORTIER:** Good
12 afternoon. Dr. Schlenk?

13 **DR. DANIEL SCHLENK:** Good morning.

14 **DR. KENNETH PORTIER:** I was going to
15 say continued good morning. Dr. Sheela?

16 **DR. SHEELA SATHYANARAYANA:** Also
17 good morning.

18 **DR. KENNETH PORTIER:** Thank you.
19 Dr. Crump?

20 **DR. KENNY CRUMP:** Here.

21 **DR. KENNETH PORTIER:** Dr. Everitt?

22 **DR. JEFFREY EVERITT:** Here.

1 DR. KENNETH PORTIER: Dr. Herrick?

2 DR. ROBERT HERRICK: Here.

3 DR. KENNETH PORTIER: Dr. Jayjock?

4 DR. MICHAEL JAYJOCK: Here.

5 DR. KENNETH PORTIER: Dr. Kanarek?

6 DR. MARTY KANAREK: Here.

7 DR. KENNETH PORTIER: Dr. Markowitz?

8 Dr. Markowitz? He may not have rejoined us yet.

9 Dr. Sheppard?

10 DR. ELIZABETH SHEPPARD: Yes, good

11 morning.

12 DR. STEVEN MARKOWITZ: I'm here.

13 I'm here. Steve Markowitz is here.

14 DR. KENNETH PORTIER: Thank you.

15 Dr. Shukla?

16 DR. ARTI SHUKLA: I'm here.

17 DR. KENNETH PORTIER: Dr. Taioli?

18 DR. EMANUELA TAIOLI: I'm here.

19 DR. KENNETH PORTIER: Dr. Van Gosen?

20 MR. BRADLEY VAN GOSEN: Here.

21 DR. KENNETH PORTIER: Thank you.

22 Let's see. Dr. Barton? Dr. Barton hasn't called

1 back in yet. Okay. We're going to begin now the
2 discussion on risk characterization, Question 5.
3 Dr. Scarano, would you go ahead and read the
4 question in, please?

6 **CHARGE QUESTION 5 (5.1)**

8 **DR. LOUIS SCARANO:** Yes. Thank you,
9 Dr. Portier. Question 5, EPA posited that there
10 were minimal or no releases of asbestos to surface
11 water associated with the conditions of use
12 evaluated in this risk evaluation and thus
13 concluded there is no risk to aquatic or sediment
14 dwelling organism in Section 4.1. As discussed
15 above, EPA calculated the potential for extra
16 cancer risk via inhalation exposures for
17 occupational -- workers and ONUs -- and consumers
18 -- DIYers and bystanders -- for cancer effect.
19 The risk characterization provides a discussion of
20 the uncertainties surrounding the risk
21 calculations. Next slide, please.

1 On the basis of the estimated
2 exposure and risks, EPA concluded that inhalation
3 of chrysotile asbestos presents an unreasonable
4 risk of injury to workers and ONUs and consumers
5 and bystanders in Section 4.2. EPA also concludes
6 that asbestos does not present an unreasonable
7 risk to environmental receptors exposed via
8 surface water in Section 4.1. EPA makes this
9 determination considering risk to potentially
10 exposed and susceptible subpopulations identified
11 as relevant under the conditions of use without
12 considering costs or other non-risk factors. Next
13 slide, please. Dr. Portier, do you want me to
14 read all five or one at a time?

15 **DR. KENNETH PORTIER:** I'm sorry.
16 Let's just read 5.1 to start with. Thank you.

17 **DR. LOUIS SCARANO:** Thank you. 5.1,
18 EPA presented overall human health risk
19 conclusions in Sections 4.5.2 and 4.5.3 based on
20 risk estimates for cancer. Please comment on
21 EPA's approach, including any alternative
22 considerations for determining and presenting risk

1 conclusions including the risk summary tables,
2 which are Tables 4-55 and 4-56.

3 **DR. KENNETH PORTIER:** Thank you.

4 And we're going to return to our normal Committee
5 protocol. I'm going to call on the lead, and then
6 I'll just check in with each of the associates to
7 see if the lead's comments contain everything that
8 they wanted to have included in this question. So
9 Dr. Cory-Slechta, you have the lead.

10 **DR. DEBORAH CORY-SLECHTA:** Yeah.

11 Let me just preface this by saying that some of
12 the comments that came in for Charge Question 5.1
13 are things that we have already discussed, for
14 example, in great detail. One of the concerns
15 raised by Committee members or respondents was
16 that there was a lack of inclusion of other cancer
17 types and disease, that these health risk
18 conclusions are based only on lung cancer and
19 mesothelioma mortality, and that ovarian, larynx,
20 digestive cancer, as well as asbestosis, are left
21 out. So there were a couple of comments to that
22 effect.

1 There were also comments about
2 leaving out the other routes or sources of
3 exposure and that, first of all, this was limited
4 to chrysotile and that the tables leave out
5 exposures to amphibole asbestos and to mixed
6 fibers from other uses: industrial talc, drinking
7 water, pipe, et cetera. Another one -- again,
8 something that has come up multiple times before -
9 - was that there was no consideration of legacy
10 uses and the feeling being that you could only
11 ascertain human health risks if EPA considers
12 legacy asbestos uses and that incorporation of
13 these is essential to understand how humans might
14 be affected by multiple sources or pathways of
15 exposure. There were numerous comments then
16 related to the assumptions about PPE use. The
17 inclusion of respiratory protection and the
18 associated APFs for consideration in risk
19 estimation for workers -- and this relates to
20 Table 4.5-5 -- were considered to be unjustified.
21 And there were several pieces of evidence given to
22 support that.

1 First of all was the citation -- the
2 note that citation by OSHA for inadequacy of
3 respiratory protection programs has routinely been
4 among the most five common OSHA violations that
5 are reported. So the notion that everybody's
6 using them clearly is not the case. There were
7 also comments about what was being used. So for
8 example in the sheet gasket stamping operation
9 that's included in this risk evaluation, EPA found
10 that workers were using N95 respirators. But
11 according to EPA and OSHA, these are not the
12 appropriate respirators to use when working with
13 asbestos.

14 There was concern as well that most
15 of the airborne concentrations that were used to
16 estimate worker exposures in the risk evaluation
17 did not exceed the permissible exposure levels or
18 the action levels that have been established by
19 OSHA and, therefore, they would not trigger the
20 requirements of the employer to enforce a proper
21 respiratory protection program. So this would be
22 another issue, as well. There were concerns that

1 small businesses were unlikely to have a
2 respiratory protection program, either because
3 they believe they're not covered by OSHA or
4 because they don't have the knowledge or resources
5 to establish such a program. So included in this
6 would be replacement of brakes, clutches, and UTV
7 gaskets. Small businesses are likely to be the
8 dominate user of the asbestos product.

9 Another one with regard to PPE was
10 the businesses that use asbestos containing
11 friction products when asbestos free alternatives
12 are readily available may not even know that the
13 products they're using contain asbestos and may,
14 therefore, be insufficiently impressed by the
15 hazards of asbestos to even think about
16 implementing the appropriate use of respirators.
17 And again, this goes back to another statement.
18 In trying to digest the information provided of
19 particular concern is the note -- and this is line
20 7391 of the text -- that nominal APF may not be
21 achieved for all respirator users. So the reader
22 is left to decide what this means without any

1 additional context. Does this mean that no PPE
2 scenarios are the most appropriate to consider?

3 Another thing that was brought up
4 was the plausibility of the bystander scenarios
5 that were presented. Table 4.56 summarized risk
6 estimates for inhalation exposures to consumers
7 and bystanders in relation to repairing and/or
8 replacing brakes and UTV gaskets. But it was felt
9 by -- in the comments that the presentation of
10 these exposure scenarios would be more plausible
11 if it highlighted the more likely scenarios in
12 Table 4.56.

13 So EPA cites on page 203 that two-
14 thirds of U.S. households have two or more
15 vehicles, and one-quarter have three or more
16 vehicles. People who replace their own brakes of
17 their own vehicles often also replace brakes of
18 cars of family members, friends, and neighbors --
19 the shade tree mechanics, as they've been called.
20 So replacing five or six sets of brakes per year
21 would be a reasonable scenario for people who do
22 this work. It was also felt that they were

1 unlikely -- this has also come up before --
2 unlikely to do this work until age 78. Stopping
3 at age 50 or 60 would seem more reasonable and
4 starting in the later teenage years more
5 reasonable.

6 Others wanted more information on
7 some of the scenarios -- a greater diversity of
8 scenarios. So for instance, what is the risk for
9 a worker who started as a sheet gasket stamper at
10 age 20 and worked for 10 years before moving to an
11 occupation without asbestos exposure. The
12 scenarios could be developed to describe people
13 who could -- who would characterize individual
14 table cells as well as alternatives that aren't
15 captured in the table. And that was felt that it
16 would make this information really available to a
17 broader group of individuals.

18 There was also indication that there
19 needed to be more information on assumptions that
20 were being made in the derivations. For instance,
21 what is the risk for a worker who start -- I'm
22 sorry. I already said that. Some of the other

1 comments were about presentation and readability.
2 And as we said before, we can include these in the
3 comments without going into them in detail.

4 I would point out, however, one
5 suggestion -- and it goes back to the readability
6 and providing a document with understandability to
7 a broader group of people -- was that
8 consideration should be given to providing an app
9 with the risk estimations either imbedded in the
10 RE or with a link to the app in the RE that would
11 allow a user to calculate risk from a particular
12 scenario that he or she is interested in. The app
13 would have KL and KM hardwired in and have options
14 for selecting the industry of interest -- nine
15 different selection -- full shift or ONU workers,
16 level of exposure, central tendency or high end,
17 APF value, with the user able to select the age at
18 which exposure begins and the duration of that
19 exposure. And they noted that other options that
20 allow the user to compute the risks from scenarios
21 not considered in the RE could also be included.
22 And I think that was pretty much what I received.

1 **DR. KENNETH PORTIER:** Thank you, Dr.
2 Cory-Slechta. Excuse me. I'm always surprised
3 when I see something I haven't seen before, but
4 the suggestion of an app as an adjunct to the DRE
5 document is interesting.

6 **DR. DEBORAH CORY-SLECHTA:** I think
7 the commenter should have kept it quiet and gone
8 for a patent.

9 **DR. KENNETH PORTIER:** Let me go
10 through the associates. Dr. Barton, anything to
11 add? Let's see. Dr. Barton hasn't rejoined us.
12 Dr. Johnson told me that he had sent all of his
13 comments to Dr. Cory-Slechta and apologized for
14 not being here. Dr. Kanarek?

15 **DR. MARTY KANAREK:** She did it well.

16 **DR. KENNETH PORTIER:** Dr. Markowitz?

17 **DR. STEVEN MARKOWITZ:** Only briefly
18 just to reiterate something. Table 4-55 would
19 suggest that if you put on respiratory protection
20 except for some high-end short-term exposures or
21 high-end exposures in general you could solve the
22 problem of exceeding the IUR. But in fact,

1 respiratory protection is not a realistic -- in
2 the real world is not a realistic solution for the
3 reasons that Dr. Cory-Slechta mentioned. That's
4 all.

5 **DR. KENNETH PORTIER:** Thank you.

6 Dr. Sheela?

7 **DR. SHEELA SATHYANARAYANA:** I don't
8 have anything else to add. Thanks.

9 **DR. KENNETH PORTIER:** Thank you.

10 Dr. Sheppard?

11 **DR. ELIZABETH SHEPPARD:** I think
12 that generally that was summarized quite well. I
13 thought the -- and I think Dr. Cory-Slechta said
14 this -- that the presentation was just really
15 difficult to digest, and I would like to see more
16 graphics, too, in addition to the app.

17 **DR. KENNETH PORTIER:** Thank you.

18 And those are suggestions we can bring up again in
19 Question 7 where we talk about kind of enhancing
20 the presentation. Dr. Crump?

21 **DR. KENNY CRUMP:** I agree with
22 what's been said. I really think there are too

1 many tables. It's just confusing, and I would
2 suggest that we try to combine them and eliminate
3 some of them. They can make it a little bit
4 easier to follow.

5 **DR. KENNETH PORTIER:** Thank you.

6 Dr. Taioli?

7 **DR. EMANUELA TAIOLI:** No, nothing to
8 add.

9 **DR. KENNETH PORTIER:** Dr. Blystone,
10 your hand's up.

11 **DR. SHERI BLYSTONE:** Yes, it is. As
12 always, I like to weigh in on the PPE discussion.
13 And since there was quite a bit of that here, I
14 want to go on record again as saying that I think
15 it's appropriate for the Agency to present risk
16 both with and without PPE because there are
17 certainly industries and individual facilities
18 that do comply with respiratory protection
19 protocols. So they should be represented, as
20 well, as a condition of use.

21 I'm not sure that everyone on the
22 panel has seen the evolution of this overtime. I

1 have to recognize that this presentation has
2 improved from what we originally saw. I do -- I
3 think from the last time to this time the use of
4 the color coding in the table is helpful. So I
5 just wanted to go on record as saying that as
6 well.

7 **DR. KENNETH PORTIER:** Thank you,
8 Sheri. Anyone else have any questions? I'm not
9 seeing any hands go up. Dr. Cory-Slechta,
10 anything else?

11 **DR. DEBORAH CORY-SLECHTA:** No, I've
12 captured the comments from -- can you hear me?

13 **DR. KENNETH PORTIER:** Yes. I can
14 hear you. Thank you.

15 **DR. DEBORAH CORY-SLECHTA:** I capture
16 the comments in my draft response about the
17 difficulties of reading the document and the need
18 for better summary tables and color coding. So
19 those are already in there as well.

20 **DR. KENNETH PORTIER:** Dr. Anderson?

21 **DR. HENRY ANDERSON:** Just an
22 interesting aside, in the Paustenbach 2006, I

1 think it is, study that EPA used, I saw a comment
2 by the authors that the workers that were doing
3 the simulations did not wear respirators, even
4 thought this would have been OSHA covered. And
5 that was largely because the researchers didn't
6 think there would be excessive exposures. It also
7 said that the workers reported typically in their
8 work they did not wear respirators. So that's
9 just one of the issues. I think it's unusual to
10 have any of the research papers talk about what
11 protection or not protection was provided to
12 workers, so I thought that was just an interesting
13 aside that they did include some information like
14 that.

15 **DR. KENNETH PORTIER:** Thank you.

16 Anyone else? Let's move on to 5.2. Dr. Scarano?

17
18 **CHARGE QUESTION 5 (5.2)**

19 **DR. LOUIS SCARANO:** Thank you, Dr.

20
21 Portier. Question 5.2, please comment on the
22 clarity and validity of specific confidence
23 summaries presented in Section 4.3.

1 DR. KENNETH PORTIER: Thank you.

2 Dr. Taioli, you have the lead.

3 DR. EMANUELA TAIOLI: Yes. I'm
4 here. So I have organized the answers into areas
5 so that hopefully we will follow a scheme, and
6 some of the things have been already discussed.
7 But I will mention them.

8 There is a section about everybody
9 touched by the asbestos legacy, so I'll have to
10 repeat that members felt it was difficult to
11 understand the source of exposure when we were
12 ignoring a large source of such exposure. And
13 there was a suggestion to have at least some
14 discussion of the prevalence of legacy users and
15 how that changed over time so that there is no
16 easy conclusion that the legacy use does not
17 contribute to population risk. Another aspect
18 about asbestos, the text mentioned voluntary
19 report of importing asbestos, and that seems like
20 a low bar of information. And we wonder if there
21 are other public records that can be mined by EPA

1 and make this a little bit of a more precise
2 estimate with less uncertainty.

3 Then I have a whole section on the
4 uncertainty of occupational exposure. So the EPA
5 acknowledge it has limited data on occupational
6 exposure, and they may have not captured some
7 workers' exposures or variation between
8 manufacturing facilities. They acknowledge that
9 data they received from various companies may not
10 be representative -- that they are uncertain about
11 the number of exposed workers.

12 It's important to note that
13 underestimation of the range of exposures implies
14 that the high-end exposure risk are most likely
15 underestimates. The direction and magnitude of
16 some of the uncertainties were not described. So
17 this may be an important recommendation. We also
18 wonder whether EPA had some other ways to request
19 this information and whether there's a discussion
20 on how this information can be generated even for
21 the future. Okay.

1 There are some mortality studies
2 from other parts of the world that came out, I
3 think, probably after this document was prepared.
4 So there are a couple of references here that EPA
5 may want to look through to maybe have an idea of
6 how to increase the precision of the estimates.
7 Now, consumer exposures, there are uncertainties
8 in the duration of exposure activities, the size
9 and typical ventilation of the areas where brake
10 maintenance occurs. I think we touched this point
11 in other answers, but I have them all here.

12 The age of start of exposure and
13 exposure duration for individual is uncertain.
14 Only for the latter was there an attempt to
15 quantify this uncertainty through a set of
16 sensitivity analysis. For many of the
17 uncertainties, EPA did not quantify the direction
18 and magnitude. The uncertainties could be better
19 documented, and judgements made about the
20 direction and magnitude of the bias that may
21 result from the assumption applied.

1 There is a comment on the
2 sensitivity analysis for the consumers and
3 bystanders, which the associate suggests it should
4 be retained. However, the notion that the average
5 consumer DIY does one brake repair or replacement
6 every three years may not be representative. I
7 think this is something else we have discussed
8 that this happens more frequently because people
9 replace their own brakes and brakes from other
10 family members or friends. So we already talked
11 about this. Okay. Assuming that an average of 30
12 percent of the concentration of asbestos that is
13 generating during active COU for consumer DIY
14 brake repair is present in average residential
15 garage for the three years between brake repair
16 episodes seems to be high, so that's another
17 comment.

18 I have a section on confounders. So
19 there is comment on smoking, many of which we have
20 talked about this morning. So I think the same
21 thing we discussed about being important to
22 understand if smoking is correlated with

1 occupational exposure. We have a couple of
2 studies that were published probably very recently
3 on frequency of smoking where asbestos workers
4 maybe at least can give some ideas of the
5 frequency of smoking and may be helpful for making
6 a more precise estimate.

7 Now, in terms of exposure, Section
8 4.3.7 appears to indicate that estimates of cancer
9 risk depends on age at first exposure. The cancer
10 risk models do not include the age of first
11 exposure. Mesothelioma risk depends on time since
12 first exposure but does not include age at first
13 exposure as a variable. These two variables are
14 not equivalent. This distinction requires
15 clarification and revisions of Table 4-49 and
16 Appendix K.

17 Now, there are some other more
18 limited comments. I don't know if I should go
19 through, or maybe we can have a discussion so the
20 other associates can bring up these other
21 comments, many of which are about the direction of
22 the bias and estimate of the direction.

1 **DR. KENNETH PORTIER:** So Dr. Taioli,
2 that's a good -- let's just scroll through the
3 associates and see if they bring them up, and then
4 you check them off. If they're not there, we'll
5 come back to them. I wanted to make one point
6 that you mentioned in the discussion on smoking
7 that I hadn't heard before, and that was this idea
8 that age at starting smoking may be more relevant
9 for lung cancer than age at start working in an
10 asbestos using facility.

11 And I thought that was an
12 interesting concept. We haven't really talked
13 about that. We've talked about smoking/not
14 smoking. But if you start working in the facility
15 at 30 but you've been smoking already for 15
16 years, that may represent much more of a shorter
17 risk.

18 **DR. EMANUELA TAIOLI:** Or at least
19 maybe a different risk. And this should be
20 considered as two different variables.

1 **DR. KENNETH PORTIER:** I notice you
2 have a reference there to -- what is it -- Olson?
3 Is that where that thought came up?

4 **DR. EMANUELA TAIOLI:** Let me look
5 back. So these are the frequency -- no, these are
6 the studies that have frequency of smoking among
7 the exposed workers. I don't think we have a
8 reference here on age of start smoking but let me
9 see. I think there's a general Peto reference. I
10 think this one came from Girardi, but we can
11 certainly -- there is a lot of reference about age
12 of start smoking and lung cancer that we can
13 provide.

14 **DR. KENNETH PORTIER:** I'm sure of
15 that literature. It's just that that concept of
16 linking that age of start smoking with age at
17 working that I just hadn't heard that one before.
18 And it struck me. Let's go through the associates
19 here and see what they have to say. Let's see.
20 Dr. Barton. Has he joined us? I see Dr. Barton's
21 on the line now. Dr. Barton, do you have anything
22 to add? You're muted in WebEx. He may only be

1 virtual, like virtually not there. Dr. Johnson's
2 not here. Dr. Markowitz?

3 **DR. STEVEN MARKOWITZ:** Yeah. I have
4 nothing to add. Thank you.

5 **DR. KENNETH PORTIER:** I'm sorry.
6 You said nothing to add. Got it. Dr. Crump?

7 **DR. STEVEN MARKOWITZ:** Yeah. That's
8 correct. I have nothing in addition to Dr.
9 Taioli's presentation.

10 **DR. KENNETH PORTIER:** Thank you.
11 Dr. Crump?

12 **DR. KENNY CRUMP:** I have a couple of
13 editorial comments. The EPA uses the word
14 benchmark in a different and very specific way in
15 low dose risk assessment. I think to avoid
16 confusion a different descriptor would be better.
17 I suggested maybe the word "goal" or "target" be
18 used instead of benchmark.

19 And the other comment is that
20 there're cases in the draft risk assessment where
21 the wording suggests that risk below ten to the
22 minus four or ten to the minus six are the same as

1 no risk, where the draft risk assessment says
2 "risk still persisted," which implies the
3 exposures below the target entail no risk. So I
4 think to avoid maybe misinterpretation by the
5 public at large, these statements should be
6 revised to say something like "risk above the
7 target risk still persisted." That's all.

8 **DR. KENNETH PORTIER:** Thank you.

9 Dr. Sheela?

10 **DR. SHEELA SATHYANARAYANA:** I don't
11 have anything else to add. Thanks.

12 **DR. KENNETH PORTIER:** Thank you.

13 Dr. Sheppard?

14 **DR. ELIZABETH SHEPPARD:** Same for
15 me. Nothing more.

16 **DR. KENNETH PORTIER:** Anyone else on
17 the Committee wish to comment? Dr. Anderson?

18 **DR. HENRY ANDERSON:** Yeah. Maybe it
19 was presented by EPA earlier, but in the document
20 it talks about at the time that the document was
21 being developed they were still researching for
22 information on water releases. And I was

1 wondering if I didn't catch it or maybe I just
2 didn't write it down. Has that been completed?
3 That's kind of the first -- kind of a yes or no
4 question.

5 And then the second issue is it
6 would seem to me that we've identified a lot of
7 data needs and a lot of issues in this limited
8 approach to chrysotile asbestos. And now EPA's
9 going to be look at a three- to five-year process
10 for the legacy uses. It would seem to me an early
11 recommendation to them would be to take a look at
12 what we recommended here, what were the weaknesses
13 and the data gaps that we identified. And there
14 will be enough time for them to develop a strategy
15 to prioritize those and then try to develop that
16 information so we're not -- or not us, but
17 whoever's going to review the legacy document
18 doesn't end up just taking this and pulling out
19 our recommendations and say nothing has changed.

20 So I think there's a good
21 opportunity here to use the time and effort that
22 we put into this for EPA to come up with what are

1 the key things that they need to try to improve,
2 if it's possible. That may be for tomorrow in
3 Section 7.

4 **DR. KENNETH PORTIER:** Yeah. It's
5 something to think about, Dr. Anderson. Thank
6 you. Anyone else want to add? Dr. Taioli, were
7 any of the bulleted things that were left out not
8 discussed that you want to bring up at this time?

9 **DR. EMANUELA TAIOLI:** Hold on. I
10 was mute. No, I think that I had the comments
11 from Dr. Crump, and he mentioned the things that I
12 have not reported. But they are in the text that
13 I compiled. So I think we're good.

14 **DR. KENNETH PORTIER:** I do see a
15 bullet about potentially exposed susceptible
16 subpopulations, something about needing to
17 characterize ballpark estimates of how many people
18 might be fully exposed. We talked a little bit
19 about the market share of asbestos containing
20 products.

21 **DR. EMANUELA TAIOLI:** I didn't see
22 that. Let me look through. Oh, when there is a

1 section on the uncertainty related to the
2 sensitivity of various subpopulation and how the
3 number of potential impact on ONUs is unknown?
4 Okay. Yeah. Specifically, so there is a comment
5 about how many times the EPA declares that value
6 was unknown, and there was no attempt to
7 characterize an estimate, even if it's a general
8 estimate. This matters because in some cases,
9 such as workers stamping sheet gaskets, the number
10 exposed are so small as to suggest that this is a
11 category where less attention is needed.

12 However, other categories there is
13 sufficient information to even put bounds on
14 potential numbers of exposed individuals. It
15 seems that some effort to characterize the market
16 share of asbestos containing products is
17 warranted, at least to determine a broad
18 characterization. As another example, while it
19 may not be known how many DIYer service asbestos
20 containing UTVs, it should be reasonable to
21 estimate this from the fraction of UTVs with

1 asbestos containing parts, a value which is not
2 provided.

3 Another concern is that the
4 representativeness of the auto parts warehouse
5 online survey. There are at least three places in
6 this document where we ask to mine existing
7 sources of data, sometimes economic data, to get
8 better estimates of import, consumption, and usage
9 as a recommendation.

10 **DR. KENNETH PORTIER:** Thank you.

11 This is Ken Portier. The reason I brought it up
12 is if we think of this document as really a risk
13 assessment of chrysotile asbestos conditions of
14 use, currently and moving forward, it's really, I
15 guess, upsetting not to have some estimate of how
16 many people are at risk now and moving forward.
17 Putting aside the legacy discussion, which may
18 indeed be a larger population, if this risk
19 assessment is forward looking, we need in here
20 some better estimate of the population at risk.

21 And I think that was -- to me,
22 that's what this bullet is kind of getting at,

1 that on the exposure side I think the Committee
2 would like EPA to push a little harder to come up
3 with some numbers, especially in the categories of
4 conditions of use that are expected to have large
5 numbers of people. That's kind of my point.

6 Okay. I don't see any additional hands going up.

7 Dr. Scarano, let's move to 5.3, please.

8
9 **CHARGE QUESTION 5 (5.3)**

10
11 **DR. LOUIS SCARANO:** Thank you, Dr.

12 Portier. Throughout this charge we have asked
13 reviewers to comment on the uncertainties and data
14 limitations associated with the methodologies used
15 to assess the environmental and human health
16 risks. Please comment on whether that information
17 has been carried forward to the characterization
18 of the risk evaluation such that the strength of
19 the unreasonable risk conclusions is characterized
20 in a clear and transparent manner. And this
21 refers to Section 4.3.

22 **DR. KENNETH PORTIER:** Dr. Taioli,
23 back to you.

1 DR. EMANUELA TAIOLI: Yes. So I
2 have several comments here, but I have not
3 reorganized them yet. So I will go through the
4 way they are.

5 So the first comment is about -- I
6 think we partly talk about this before with Dr.
7 Sheppard -- but we don't understand this concept
8 of compensation bias. So the lack of -- the way
9 it's reported it's the lack of sufficient number
10 of workers to estimate risk of ovarian and
11 laryngeal cancer is a downward bias leading to
12 lower IUR estimates and in overall cancer
13 assessment. However, the selected IUR was chosen
14 to compensate for this bias. So we think that
15 this has to be described better and why there is
16 this attempt of compensating the information.

17 There is another section about using
18 lung cancer mortality as a proxy of incidence and
19 how that inserts a low level of uncertainty. It's
20 again something we talked about this morning. But
21 currently, lung cancer screening detects a larger
22 proportion of stage one lung cancer, which are 80

1 percent curable. These workers are a candidate
2 for lung cancer screening because of their
3 exposure and work history. Therefore, we expect a
4 large proportion of early stage lung cancer in
5 this population. So there should be a discussion
6 about how mortality's not a proxy of incidence, at
7 least of lung cancer for sure.

8 Another comment is about the fact
9 that the risk could be underestimated because of
10 legacy exposures. It's an understatement. Almost
11 all the existing sources of exposure come from
12 legacy exposure. The so-called bystander exposure
13 is, again, very limited in scope and focused on
14 few cases of population, and it's hard to believe
15 that it can be generalizable.

16 Now, again, there is a point about
17 why information on market share is not available.
18 We're going back to what we wrote for 5.2. By
19 omitting legacy exposure, reported estimates are
20 almost certainly underestimated. An important
21 feature is that legacy exposure could impact some
22 exposure more than others and thus differentially

1 impact the risk estimate. Some effort to quantify
2 this, at least to characterize differential
3 impacts of legacy exposure across category should
4 be considered. This, I think, is something we
5 have discussed, but it's an important point.

6 Although it's difficult to weight
7 the importance of the uncertainties and assumption
8 in the context of the values reported and thus
9 either trust the values reported or not, it would
10 be helpful to have a better sense of the
11 uncertainty of the reported estimates. So the
12 suggestion to do a tabular summary of the
13 uncertainties and the judgement about the
14 direction of the bias, and there is a suggestion
15 here to -- one second -- carrying some of the
16 uncertainties through to provide risk estimate in
17 sensitivity analysis by making ultimate
18 assumption. Okay.

19 There is a positive comment about
20 the section on explaining methods and data source
21 risk estimate. There is lack of information about
22 the actual availability and quantity of imported

1 asbestos containing products, and this should have
2 been addressed by the purchase of samples. And
3 these items have varying locations in the U.S. and
4 testing the products for their asbestos content.
5 Even if asbestos containing products were not
6 found, an estimate of the upper bound of frequency
7 of asbestos containing products and a more
8 realistic estimate of the population at risk of
9 brake and UTV gasket exposure may have been
10 obtained.

11 Then while EPA has described some of
12 the limitation of the available exposure data and
13 estimated associated with the six target COUs, the
14 reliance on industry generated data and their
15 limited documentation for the chlor-alkali
16 facilities and the gasket stamping operation is
17 problematic. The lack of details provided by
18 companies on the sampling methods undermines
19 confidence in the sampling result.

20 EPA notes that there is unclear --
21 it's unclear if certain high exposure activity in
22 the chlor-alkali industry were associated with air

1 monitoring results. EPA should have used its
2 authority to obtain all sampling data from these
3 facilities. EPA used industry supplied data to
4 estimate exposure, even though those data did not
5 include sample duration or how long a gasket
6 removal was performed.

7 Then, there is a comment about the
8 fact that very few studies in peer review
9 published literature were used to estimate
10 asbestos exposure in some COUs, especially in the
11 area of repair and replacement of brakes and
12 gaskets where one or two studies and references
13 are used. EPA states that it's highly certain
14 that import of ACM beyond the six product
15 categories does not occur. If these have been
16 investigated, they should be listed in Appendix C,
17 so under references of the various other asbestos
18 products with numbers here in parentheses. And
19 that's all I have for now.

20 **DR. KENNETH PORTIER:** Thank you.

21 And the associates. Let's see. Has Dr. Barton
22 returned? Dr. Barton? Dr. Barton, do you have

1 anything to add? I don't hear Dr. Barton. Dr.
2 Johnson hasn't returned. Dr. Markowitz?

3 **DR. STEVEN MARKOWITZ:** Just to add
4 to Dr. Taioli's final point about the import of
5 asbestos containing products, so there's certain
6 HTS codes in the U.S. geological survey data that
7 are just not addressed in the document, including
8 Appendix C. And I included those codes. It
9 includes yarn and thread crocidolite products,
10 clothing and building materials. And they just
11 need to be accounted for somewhere, either in the
12 text or in the appendix to the extent that EPA
13 actually has knowledge about them. Thank you.

14 **DR. KENNETH PORTIER:** Thank you, Dr.
15 Markowitz. Dr. Crump?

16 **DR. KENNY CRUMP:** I think most of
17 this has been discussed earlier, but I do think
18 that, just in general, the discussion of bias
19 needs to be expanded. And we've made a lot of
20 suggestions about doing that. So I think that's
21 been pretty well covered.

22 **DR. KENNETH PORTIER:** Dr. Sheela?

1 **DR. SHEELA SATHYANARAYANA:** I don't
2 have anything else to add.

3 **DR. KENNETH PORTIER:** Dr. Sheppard?

4 **DR. ELIZABETH SHEPPARD:** Same for
5 me. Nothing else.

6 **DR. KENNETH PORTIER:** Anyone else on
7 the panel? This is Ken Portier. You know
8 something you said here, Dr. Taioli, got me
9 thinking about the legacy uses and this whole
10 concept of this document looking at current COUs
11 and looking forward versus the legacy exposures
12 that are in the past to today. And to me, in
13 characterizing the risk, that risk needs to say
14 this is the risk moving forward from these limited
15 COUs. There will be excess risk from the legacy
16 exposures that are going to show up in depth in
17 the future.

18 But I don't know how to kind of
19 phrase that and capture that because I think
20 that's been part of the concern with not covering
21 legacy exposures in this document is being able to
22 characterize risk from these exposures and these

1 expected conditions of use and additional risk
2 from legacy exposures and uses that maybe no
3 longer be -- no longer are operable but have
4 expressed exposure in the past. Does that make
5 sense? Does anybody want to add to that and
6 clarify my thinking? I'm thinking maybe one of
7 you epidemiologists. Dr. Herrick? Your phone may
8 still be muted, Dr. Herrick. We're still not
9 hearing you.

10 **DR. ROBERT HERRICK:** Oh, I'm sorry.
11 I didn't have it unmuted. I can't promise
12 complete clarity, but we did address that in
13 Question 6.

14 **DR. KENNETH PORTIER:** Okay. Good.
15 Good. Then maybe we don't need to talk about it
16 here. Dr. Davies?

17 **DR. HOLLY DAVIES:** Hi, I just wanted
18 to mention that while legacy uses aren't included
19 in this document, because the legacy uses are
20 still in existence, the exposures are also current
21 and moving forward. People are still being
22 exposed to the legacy asbestos that was put in

1 building materials in the past, for instance, and
2 the disposal of those materials.

3 **DR. KENNETH PORTIER:** Point taken,
4 Dr. Anderson. No, I was thinking about -- I'm
5 still thinking about mortality. And you're right.
6 It's not just past exposures. It's not just
7 legacy exposures. It's exposures from legacy COUs
8 that are in the past and continue into the future.
9 I understand that. Dr. Anderson?

10 **DR. HENRY ANDERSON:** I was going to
11 say the concept here is one of cumulative risk.
12 And this particular document assumes that the only
13 exposure to asbestos that these individuals will
14 have isn't COUs, which is not a very practical
15 approach. So clearly the asbestos risk that these
16 workers will have is only partially characterized
17 here. But their cumulative risk will continue.

18 **DR. KENNETH PORTIER:** Dr. Blystone?

19 **DR. SHERI BLYSTONE:** I don't know
20 that I have anything great to add here, but in my
21 mind, it's sort of like a special PESS -- that
22 perhaps you have individuals who have worked in

1 legacy conditions of use that now work in a
2 current condition of use where their exposure,
3 therefore, might be greater or different than just
4 a regular worker in that condition of use. But
5 the intent is really -- in my mind, under these
6 evaluations, is to determine for current
7 commercial uses is there an unreasonable risk or
8 not. And the concepts of background exposures and
9 old uses and I don't know if someone who is -- the
10 likelihood of someone working in one of these
11 current conditions of use that also then is
12 actively working in asbestos remediation or
13 disposal, that could be a consideration. It's not
14 an easy question is, I guess, my brilliant
15 comment, or not so brilliant.

16 **DR. KENNETH PORTIER:** Thank you.

17 Dr. Herrick, your hand is still up. You wish to
18 add? Okay. Dr. Herrick's hand went down. Thank
19 you. Dr. Taioli, any final comments?

20 **DR. EMANUELA TAIOLI:** No, I think I
21 covered everything. There was one -- I may have
22 forgotten one thing which is about other known

1 cancer condition. And I forgot to read this. So
2 the EPA considered that other known cancer related
3 endpoints are relevant when discussing cancer risk
4 as many of their precursors of mesothelioma and
5 old lung cancer. So the associate didn't agree
6 that the uncertainty is low when leaving lung
7 cancer endpoint out of the equation. So I forgot
8 to -- I skipped this one, but it's an important
9 point.

10 **DR. KENNETH PORTIER:** Okay. Dr.
11 Scarano, do you have any comments, or are you
12 ready to move forward?

13 **DR. LOUIS SCARANO:** I think I'm
14 ready to move forward. I don't know if some of my
15 team members would like to say anything. It was a
16 good and robust discussion.

17 **DR. KENNETH PORTIER:** I don't see
18 any hands going up, so why don't we at least
19 scroll up 5.4 while they're thinking. I'll keep
20 looking.

21 **DR. LOUIS SCARANO:** Okay.

1 **DR. KENNETH PORTIER:** Okay. Nobody
2 jumped in, so let's go ahead and read Question
3 5.4.

4
5 **CHARGE QUESTION 5 (5.4)**

6
7 **DR. LOUIS SCARANO:** Thank you. This
8
9 is the last of the risk characterization
10 questions. Please comment on whether the analysis
11 presented in Section 4 supports the conclusions
12 for both the environment, Section 4.5.1, and human
13 health, Section 4.5.2 and 4.5.3, in the draft risk
14 characterization section concerning asbestos. If
15 not, please explain the limitations of these
16 conclusions and whether there are alternative
17 approaches or information that could be used to
18 further develop the risk estimates within the
19 context of the requirements stated in EPA's Final
20 Rule, *Procedures for Chemical Risk Evaluation*
21 *Under the Amended Toxic Substances Control Act.*
22 Thank you.

1 **DR. KENNETH PORTIER:** I will point
2 out there is one more question on Question 5 that
3 has to do with other aspects. We'll get to that.
4 So Question 5.4, we're back to Dr. Cory-Slechta.

5 **DR. DEBORAH CORY-SLECHTA:** Hi, let
6 me start with comments that came in about
7 conclusions on the environment. One of them is
8 that on page 52 EPA states that the treatment
9 efficiency of the chlor-alkali treatment regimen
10 for asbestos is unknown. How, then, do we know
11 whether asbestos fibers survive the filtration
12 process when, quote, asbestos releases from chlor-
13 alkali facility treatment systems to surface water
14 and POTW are not known? In view of this, how can
15 EPA make a determination of no exposure regarding
16 potential releases to water for the COUs in this
17 evaluation and no unreasonable risk to aquatic
18 organisms? So an uncertainty there.

19 Another one related to the
20 environment came up before. And this was that
21 they expected to see a table with derived COCs
22 compared with exposure estimates. Instead, the

1 EPA rationalizes that available information
2 suggests levels in freshwater sources are well
3 below those where toxicity is described. But
4 given the mechanism that's cited for asbestos --
5 and this is the frustrated phagocytosis of
6 macrophages -- and that macrophage-like cells
7 exist in many other aquatic organisms, it raises
8 questions about effects to longer lived species,
9 for example, Ambystoma salamanders, turtles, et
10 cetera. And the suggestion here is that the EPA
11 explicitly discuss this possibility in uncertainty
12 sections.

13 There was another case of
14 uncertainty in which Section 4.5.1 -- it was that
15 of basing a risk determination of no environmental
16 risk on a lack of, quote, reported exposure data.
17 Determinations of risk should be based on measured
18 data, rather than expectation and/or lack of
19 identification. There were numerous comments
20 about potential sources of bias in the human
21 health determinations.

1 The first of these relates to
2 mortality risk. And again, this goes back to the
3 fact that we're not considering risk from cancers
4 other than lung and mesothelioma. And one of the
5 concerns here is that EPA compensated for these
6 two sources of negative bias by selecting the
7 largest IUR from among the four candidates, even
8 though there's no direct relationship between the
9 two sources of bias and the largest IUR.

10 A recommendation here was that data
11 related specifically to the magnitude of the two
12 sources of bias addressed in the RE should be used
13 to adjust for them. Data on the time between when
14 lung cancer is identified and subsequent death
15 from lung cancer could be used to adjust the risk
16 estimates based on mortality data to pertain to
17 incidence. Data from studies of human populations
18 on cancers other than lung and mesothelioma
19 possibly caused by asbestos could be used to
20 adjust risk estimates to account for those
21 cancers.

1 There are also uncertainties related
2 to the use of the North and South Carolina
3 datasets, which are of roughly equal quality. But
4 there was a recommendation that the data for
5 calculating an IUR for chrysotile data from the
6 Carolina textile mills actually should be the
7 combined data from the North and South Carolina
8 mills rather than simply selecting data as a means
9 of overcoming biases. So the study by Eliot et
10 al. contains a pooled analysis of data from North
11 and South Carolina for lung cancer. Berman and
12 Crump and Loomis et al contain analyses of
13 mesothelioma data from South Carolina and North
14 Carolina respectively, both of which are based on
15 individual data. And these analyses could then be
16 combined to evaluate the risk of mesothelioma.

17 Then we go back to the issue of
18 which of the fibers is the issue or whether some
19 of the problem is caused by contamination from
20 amphibole. The potency of chrysotile for causing
21 mesothelioma may depend largely upon the amphibole
22 contamination, and it's stated that both the North

1 Carolina and South Carolina textile mills used
2 asbestos from Quebec, which is known to be
3 contaminated by tremolite asbestos. Also, both
4 the South Carolina mill and some of the North
5 Carolina mills processed some amounts of
6 amphibole. So the risk evaluation should discuss
7 this potential bias and risk of mesothelioma
8 stemming from contamination of chrysotile by
9 amphiboles and consider whether it's feasible to
10 adjust the mesothelioma risk estimates for the
11 amount of amphiboles to which workers in the North
12 and South Carolina mills were exposed compared to
13 the amounts of amphiboles in the asbestos products
14 for which risk is being assessed in the risk
15 evaluation.

16 We also come back to fiber length
17 associated risk. Potency of fibers is dependent
18 upon their lengths, and longer fibers are
19 considered more potent based on human and animal
20 data. Thus, the same fiber concentrations
21 measured by PCM can pose different cancer risks
22 because PCM counts all fibers longer than five

1 microns as equally potent, regardless of their
2 length, and fibers shorter than five microns as
3 non-potent.

4 Asbestos textile manufacture require
5 asbestos that has been milled to contain a greater
6 percentage of longer fibers compared to asbestos
7 used in products for which risk is assessed in the
8 risk evaluation, which will produce a positive
9 bias in the risk estimates in the RE. And a
10 recommendation here is that this could be resolved
11 if the exposure data used in risk assessment had
12 been adequately analyzed by TEM rather than PCM --
13 something we've talked about before -- and that
14 clearly it's unfortunate that despite the huge
15 amount of research that's been conducted on the
16 health effects of asbestos, most of this
17 quantitative exposure data is still PCM based.
18 But the risk evaluation should acknowledge and
19 discuss all these potential sources of bias that
20 are not now mentioned. And they should consider
21 adjusting risk estimates pertaining to specific
22 work environments for differences in fiber

1 dimensions using data that was analyzed by TEM if
2 it's possible.

3 And then finally, there was one on
4 the degradation of fibers and uncertainties
5 associated with that. The high friction
6 environment in vehicle braking causes the asbestos
7 fibers to degrade, both chemically and physically.
8 That shortens the length of the fibers released
9 during brake replacement compared to their
10 original length and compared to the length of
11 those encountered in the textile mills in North
12 and South Carolina. Concomitantly, the braking
13 action will likely increase the proportion of
14 fibers shorter than five microns, which are not
15 counted by PCM. Thus, if you base the risk on PCM
16 counting rules, the bias in the estimated risk
17 from fibers longer than five microns will be
18 positive, whereas the bias in the risk estimated
19 from fibers shorter than five microns, assuming
20 they pose a risk at all, is negative.

21 So the recommendation here is that
22 the differences in the fiber size is between those

1 in North and South Carolina studies and those
2 encountered in brake replacement are so profound
3 that it's highly questionable as to whether North
4 and South Carolina data should be used at all to
5 quantify the risk from exposure during brake
6 replacement. On the other hand, there have been a
7 number of epi studies of workers involved in brake
8 replacement, and these should be reviewed in the
9 RE and considered as a basis for conclusions
10 regarding the risk from brake replacement
11 activities. And that's all I have.

12 **DR. KENNETH PORTIER:** So Dr. Cory-
13 Slechta, I was listening to this, and the
14 environmental ones kind of address the question
15 whether the characterization section supports the
16 decision. And I think I heard there they didn't
17 feel like the environmental risk characterization
18 truly supported the conclusion of no risk in the
19 environment. But I didn't hear kind of the
20 support for the risk conclusion for the human
21 health stuff.

1 A lot of what you mentioned still
2 seemed to be uncertainties and data quality
3 issues. I was looking at your write up and
4 there's -- just right before the second to last
5 paragraph, you have human health "Given the
6 information presented and the aspects that have
7 been quantified, the reported conclusions seem to
8 be reasonable supported. However, it would be
9 useful --" Is that your conclusion?

10 **DR. DEBORAH CORY-SLECHTA:** You know,
11 based on what came in, that was the only comment
12 that actually explicitly referred to that. I
13 think most of the others were, as you said,
14 related to the uncertainties and the need to
15 really define those. So at least reading over
16 these, I would say you're right about the
17 environmental conclusions -- that there are two
18 big issues there -- but that those did not seem to
19 come up in the human health comments as much. But
20 certainly the uncertainties will lend to those
21 conclusions.

1 **DR. KENNETH PORTIER:** Yeah. I was
2 just trying to think of -- when we write this up
3 and we try to address this question on the human
4 health side, what does the first paragraph say?
5 And I was trying to figure out how we're going to
6 put that together. But you can think about it.
7 Let me run through the associates and see what
8 they think. Dr. Barton? Let's see. Where am
9 I? Dr. Markowitz?

10 **DR. STEVEN MARKOWITZ:** So you had a
11 replication of the overly simplistic discussion of
12 the contrast between the textile environment and
13 the brake mechanic environment. I would just
14 point to an article by John Dement and others,
15 *Occupational and Environmental Medicine* 2011,
16 looking at the North Carolina and South Carolina
17 textile plants and looking at TEM and PCM. So I
18 guess that's the key to looking at the whole
19 spectrum of fiber size.

20 And they note that the vast majority
21 of asbestos fibers in the textile plants were less
22 than five microns in length. So the idea that

1 textile facility only include long fibers is not
2 true. And in fact, the vast majority of fibers at
3 those sites were less than five microns. And
4 likewise, in the brake mechanic environment
5 there's some studies that show that not all fibers
6 are short, that when TEM is used in addition the
7 PCM that longer fibers have been identified.
8 References include the NIOSH Roberts study 1982
9 and also the Rohl study 1976. And there are other
10 references as well.

11 So my only point is that it doesn't
12 break down by fiber size as simplistically in the
13 two environments. And then finally, a point I've
14 made already a couple times, the studies by Hamra
15 2014 and 2017, that when you use TEM and PCM, the
16 fiber size -- the risk of lung cancer at least in
17 fiber size, there's not clear distinction between
18 the shorter and the longer fibers. That's all.

19 **DR. KENNETH PORTIER:** Thank you.

20 Dr. Crump?

21 **DR. KENNY CRUMP:** No further
22 comments. Thank you.

1 DR. KENNETH PORTIER: Dr. Sheppard?

2 DR. ELIZABETH SHEPPARD: No further
3 comments.

4 DR. KENNETH PORTIER: Dr. Taioli?

5 DR. EMANUELA TAIOLI: No further
6 comments. Thank you.

7 DR. KENNETH PORTIER: Dr. Schlenk, I
8 see your hand up.

9 DR. DANIEL SCHLENK: Can you hear me
10 now?

11 DR. KENNETH PORTIER: Yes.

12 DR. DANIEL SCHLENK: Actually, I
13 think you missed Mark Johnson. He was also an
14 associated first. I can wait until he goes if you
15 want.

16 DR. KENNETH PORTIER: Well, Mark's
17 not back until 3:30.

18 DR. DANIEL SCHLENK: Ah, okay.
19 Gotcha. Yeah. Based upon the questions that we
20 had asked on the first day, I waited to actually
21 put some recommendations in here, which apparently
22 is this particular number. So based on the

1 responses that EPA provided in those questions, I
2 would strongly recommend monitoring of wastewater
3 by COUs that actually have an aqueous component in
4 their processing, particularly the chlor-alkali
5 industry. I think that is necessary.

6 The second recommendation I will do
7 is until you can actually get monitored data I
8 think uncertainty drives the assessment to a worst
9 case scenario, which, again, we seem to keep
10 missing in most of our uncertainty analysis
11 sections of risk characterization. For the
12 environmental aspects, the recommendation I would
13 say is, until you can prove otherwise, use the
14 highest value of chrysotile fibers that have been
15 measured in the environment and then work
16 backwards. Again, that's just a recommendation
17 for overcoming the uncertainty there -- or at
18 least provide that. It doesn't necessarily need
19 to use it for a risk -- a binary decision of one
20 versus the other, but at least it shows -- if that
21 binary decision is necessary, then it at least
22 gives you that option to set a scenario where

1 there is an accurate risk quotient evaluation to
2 take place.

3 And the third thing would be -- and
4 this is -- I think I sent this for Question 5.5,
5 but since we're supposed to make recommendations
6 here and size keeps coming up -- and again, this
7 is mostly on the human health side in terms of the
8 fiber size and length and those issues. But from
9 the environmental side, I think I would highly
10 recommend the TEM be used to provide distributions
11 of size that are present, particularly in waste
12 effluent because, again, based upon the literature
13 that's coming out with microplastics, anything
14 less than five microns is generally considered
15 potential for absorption in aquatic organisms.
16 That's been seen all over the world now. So those
17 are my recommendations based upon the uncertainty
18 and risk characterization sections.

19 **DR. KENNETH PORTIER:** So Dan, don't
20 go away. I want to ask you a question. You've
21 participated in these things. And if you look at
22 this question, it really asks do you think that

1 the draft risk characterization section supports
2 the conclusion. And on the environmental side, on
3 line 5747 and 48, basically says, "EPA concludes
4 there is low or no risk to aquatic or sediment
5 dwelling organisms." That's their risk
6 characterization conclusion. What's your feeling
7 on this?

8 **DR. DANIEL SCHLENK:** Well, I already
9 sent that. Deborah already read that. That was
10 my comment that the environment -- you can't say
11 there's environmental risk since you don't have
12 the numerator of the risk quotient equation. So I
13 don't agree that they can conclude that you do not
14 have environmental risk because you really have no
15 monitoring data or any data in the numerator for
16 exposure to make those risk quotient analyses. So
17 the only other way, as I just recommended, would
18 be to use a worst-case scenario where you actually
19 either get water based -- surface water-based
20 numbers and make that assessment. Otherwise, to
21 make an assessment on no exposure data, I don't
22 think you can do that. But again, I may be alone

1 in that sort of recommendation. But in my mind,
2 the environmental side is not there.

3 **DR. KENNETH PORTIER:** And this is
4 Ken Portier. I tend to agree with you. I wasn't
5 picking on you, Dan. I just wanted you to make
6 that statement because now I'm going to turn back
7 to the health effect side people and try to get
8 kind of a similar assessment from them. Dr.
9 Doucette, your hand flew up here. Dr. Anderson, I
10 know you're hand's up. I'll get to you. Dr.
11 Doucette?

12 **DR. WILLIAM DOUCETTE:** I just wanted
13 to support Dan's argument. I totally agree that
14 without any monitoring data at all we can't really
15 make a decision like that. We can't really
16 support that conclusion that EPA made. That was
17 also addressed when Dan gave the summary to
18 Question 1.1 and 1.2. That same issue came up,
19 and this is just reiterated now in this question.

20 **DR. KENNETH PORTIER:** Thank you.
21 Keeping with environmental --

22 **DR. SHERI BLYSTONE:** Dr. Portier?

1 **DR. KENNETH PORTIER:** -- I see Dr.
2 Johnson has joined us. Yeah. Is that Sheri?

3 **DR. SHERI BLYSTONE:** Yeah. Because
4 this was also on the environmental side. This is
5 Sheri Blystone.

6 **DR. KENNETH PORTIER:** I'm not
7 leaving it yet. Go ahead.

8 **DR. SHERI BLYSTONE:** Okay. We can
9 go to Dr. Johnson first. That's fine.

10 **DR. MARK JOHNSON:** Okay. Thanks,
11 Sheri.

12 **DR. KENNETH PORTIER:** He just
13 joined.

14 **DR. MARK JOHNSON:** I was going to
15 say quickly I like to come to the same conclusion
16 but from looking at the toxicity data. I don't
17 think EPA's wrong. I think they're probably
18 right. It's just not very well substantiated from
19 the toxicity side.

20 We have four orders of magnitude
21 variation in some of the data that's displayed in
22 a table earlier on. And as Deb mentioned earlier,

1 there's some aquatic organism we just don't know.
2 So there's never a situation where there's no
3 risk. I think words are important here. I think
4 there's a high probability there's no unreasonable
5 risk, but we still have to be specific on the
6 uncertainty that we're willing to accept to make a
7 statement like that. Over.

8 **DR. KENNETH PORTIER:** Thank you, Dr.
9 Johnson. Dr. Blystone?

10 **DR. SHERI BLYSTONE:** So I more or
11 less agree with what Dr. Johnson just said. I
12 think the conclusion is probably correct. We
13 pointed out in the Day 1 discussion some of
14 inadequacies there to bolster that. But I didn't
15 want it to come across as if we thought there was
16 no support. I again think that EPA did a fairly
17 decent job in trying to find any evidence that
18 there are water releases related to these
19 conditions of use and was unable to find that.
20 That's all.

21 **DR. KENNETH PORTIER:** Thank you.
22 Dr. Anderson, I see your hand. Just one second.

1 Where was I? Dan, your hand's still up and Bill
2 Doucette.

3 **DR. DANIEL SCHLENK:** Yeah. Just to
4 respond to Sheri. I totally agree that the Agency
5 tried their best with what they had. I'm not
6 discounting that a bit. I'm just saying the logic
7 that is used for risk, you need exposure and you
8 need toxicity. And there's always going to be
9 uncertainty.

10 I totally agree with what Mark said,
11 as well. There is uncertainty that's present
12 there. You just have to describe it or to say,
13 "We just don't have the data." So we can't
14 conclude there's no risk, and you can't conclude
15 in this case that there is risk either because you
16 don't have the data. So that's what I say. I
17 totally agree with the efforts put into the Agency
18 in trying to find that that's there. But the
19 data's just not there.

20 **DR. KENNETH PORTIER:** Okay. Dr.
21 Bateson, I didn't know if you wanted to comment on

1 the environmental side or on the human health
2 side.

3 **DR. THOMAS BATESON:** I wanted to
4 come in on the side of we did the best with what
5 we had. The last commenter phrased that nicely.
6 It was a lot of discussion about TEM and how it
7 would be helpful to have TEM measurements of the
8 fibers in the textile plants and the fibers that
9 are in the brakes and in the different
10 environments. But what we have is fibers counted
11 with PCM in all of the conditions of use.

12 All of those exposures are in PCM
13 units. And all of the health data are also in PCM
14 units. So we're applying what we have, fibers
15 longer than five microns, to risks -- for
16 exposures which are longer than five microns. And
17 we're doing the best with what we have.
18 Recommendations to move to TEM, while good in the
19 long term, doesn't help us with the current
20 problem. So I just wanted to lay that out and
21 make that plain to remind everybody. Thank you.

1 **DR. KENNETH PORTIER:** Yeah. And I
2 think the point Dr. Schlenk was making is that to
3 be able to draw the environmental risk
4 characterization conclusions that you want to draw
5 you're going to need new data. Absence of data
6 isn't the same thing as absence of exposure. So
7 he wants you to go out and collect data. And when
8 you do collect and measure that data, use TEM so
9 that you have the maximum amount of data. Dr.
10 Schlenk?

11 **DR. DANIEL SCHLENK:** You got it
12 right on the nose, Ken. This is a recommendation
13 for -- again, this is why I asked the question on
14 the first day, "Should we be making
15 recommendations for more monitoring data?" And
16 this is a recommendation for monitoring data and a
17 methodology for that monitoring data because I
18 think you're going to need a size distribution
19 data eventually to get to the environmental
20 aspects of this. I know what you have, and I know
21 the problem with the data that you have currently.
22 This is for -- again, as the question states, this

1 is what we need to do in the future. So that's
2 why I make that recommendation.

3 **DR. KENNETH PORTIER:** Thank you.

4 Dr. Anderson?

5 **DR. HENRY ANDERSON:** I didn't want
6 to comment on health specifically. I just want to
7 support Dan on the environmental side. But also,
8 one of the pieces of evidence that EPA used was
9 the very low proportion of public drinking water
10 well data that had fibers in it. And I was going
11 to suggest rather than lump all of the water
12 supplies together you might want to look at the
13 difference between surface water and groundwater.
14 So I think the data there, you might be able --
15 even though it's a small proportion of the --
16 whatever it was -- 44,000 public water supplies,
17 you might get a sense of whether there are some of
18 the surface waters, which would be a discharge
19 from air, also discharge from surface into street
20 runoff and things like that. That wouldn't be
21 specific to the COUs but would give it under --
22 some understanding of the contamination of the

1 overall environment that then could make its way
2 into the wildlife in those waterbodies, which
3 really was the way it was done with taconite mine
4 discharges that showed up in the public water
5 supplies of those using Lake Superior.

6 **DR. KENNETH PORTIER:** Thank you,
7 Henry. And now turning back to Dr. Cory-Slechta
8 and the associates, especially on the human health
9 side. Again, I'm trying to think in my head what
10 that first paragraph on how we're going to answer
11 this particular question -- what does it sound
12 like? Clearly, I think we mostly agree EPA did
13 the most it could do with what they had.

14 Now, there's a lot more data on the
15 human health side than there is on the
16 environmental side, and they are able to compute
17 central tendency and high-end risk estimates for
18 these five COUs. And they draw risk conclusions
19 in their risk characterization section on each of
20 these. I'm trying to get a sense of how the
21 Committee -- I realize there's caveats on all of
22 this.

1 There are -- on warts, on all of the
2 analysis, right? We keep pointing out the
3 uncertainties, the data quality issues. But at
4 the end of the day, how do we answer this
5 particular question on does the draft risk
6 characterization section support the conclusions
7 that there is or is not risk? How do we answer
8 that question? Dr. Cory-Slechta.

9 **DR. DEBORAH CORY-SLECHTA:** Yeah.
10 Just the feeling I get from, as you mentioned, all
11 of the warts and issues that have arisen, it's by
12 no means a slam dunk. So if we assigned
13 confidence levels to it or quality levels to it or
14 believability levels, if you will, I'm somewhere
15 in the range of, you know, maybe. Yeah. Probably
16 better than nothing. But I'm not overwhelmingly
17 convinced that this is the be all and end all risk
18 assessment because of all the uncertainties and
19 the other issues that have been brought up.

20 **DR. KENNETH PORTIER:** Does anyone
21 else on the panel want to chime in on this? I'm
22 sorry if I'm pushing you on it, but I think it's

1 important for this substance that we kind of at
2 least discuss that. I am looking at the report.
3 I'm trying to find the table usually where they
4 assign their confidence in the estimate, and I'm
5 not seeing that table. Is that in Chapter 5? Dr.
6 Jayjock?

7 **DR. MICHAEL JAYJOCK:** Okay. Yeah.
8 I just wanted to chime in and say that, as far as
9 the human health piece is concerned, you basically
10 take your best estimate of exposure and your best
11 estimate of hazard and you compare them. And then
12 you wind up with a putative risk. And in this
13 particular case, you said the conclusion was that
14 it's unacceptable risk. I think, from my
15 perspective, they did as good a job as they could.
16 I'd be very surprised if the risk wasn't
17 unacceptable, although you always have the
18 opportunity -- well, you should have the
19 opportunity to get more data to refine the
20 assessment that might turn the risk assessment
21 from an unhappy face to a happy face. But I don't

1 think that would be happening in this particular
2 case.

3 Now, on the side of the
4 environmental exposure assessment, that simply
5 isn't my area. But I think the idea that there is
6 no data, vis a vis surface water, would indicate
7 to me that perhaps it doesn't support the
8 conclusion of no unacceptable risk. That's my
9 comment.

10 **DR. KENNETH PORTIER:** Dr. Taioli?

11 **DR. EMANUELA TAIOLI:** Yes. So I
12 think that a simplification and the limitations of
13 all the approaches for the human health risk makes
14 it very difficult to generalize because it doesn't
15 really reflect the reality of what's going on. So
16 although I respect the conclusion, but it may be a
17 very limited conclusion that doesn't have a lot of
18 meaning for real life. So that's what I feel.

19 **DR. KENNETH PORTIER:** Thank you.
20 Anyone else? I'm looking something up. Just one
21 second. Actually, why don't we go ahead -- I
22 think we're due for a break and lunch for the West

1 Coast people. We're scheduled to have a break at
2 3:30. Why don't we break at this point and return
3 at 3:40? That gives us a 20-minute break. That
4 gives me a chance to look up some things and get
5 my mind straight on this. And then right after
6 the break, we'll finish up with Question 5.5 and
7 move on to Question 6. So 20-minute break. Thank
8 you.

9 **[BREAK]**

10 **DR. KENNETH PORTIER:** Okay. This is
11 Ken Portier. I have 3:40. Let's reconvene. Dr.
12 Cory-Slechta, during the break I finally had my
13 ah-ha moment that what I was trying to get at just
14 before the break with regards to, again, continued
15 discussion on 5.4. And I'll read kind of the
16 response that I've started to put together on
17 this. "So comparing the discussion in Section
18 4.3.3 where EPA summarizes key assumptions and
19 uncertainties --" Can everybody hear me? I
20 should check.

21 **DR. DEBORAH CORY-SLECHTA:** Yes, I
22 can hear you.

1 **DR. KENNETH PORTIER:** Good. I
2 should have checked. “So comparing the discussion
3 in Section 4.3.3 where EPA summarizes the key
4 assumptions and uncertainties to similar
5 discussions in DREs for previous TSCA chemicals
6 shows that what is missed in this chrysotile
7 asbestos DRE are statements expressing the
8 confidence EPA has in the IUR values and the
9 exposure estimates used to assess risk. In
10 previous assessments, an overall assessment of
11 confidence rated high, medium, or low was assigned
12 to risk estimates for each condition of use. This
13 was not done here, and the Committee recommends it
14 be done to provide users with this important
15 rating.”

16 And then I kind of want to talk
17 about -- because to me, this is what this Question
18 5.4 is really asking, but EPA didn't provide us
19 with their assessment. And just to give you an
20 idea, in the perchloroethylene DRE, Section
21 4.3.2.1, there's a statement that reads “There's
22 medium-high confidence in the acute, noncancer

1 POD; high confidence in the chronic noncancer POD
2 selected to represent each health domain; and
3 medium confidence in the cancer POD. Confidence
4 is reduced for dermal PODs due to the use of route
5 to route extrapolation and the absence of dermal
6 compartments in the PBPK model.” And it goes on
7 like this and at the end concludes that we have
8 high, medium, or low confidence.

9 None of that is in this asbestos
10 DRE. I looked at various sections in both the
11 hazard assessment, the exposure assessment and in
12 the overall key assumption sections. And I don’t
13 see those confidence statements. And I think it
14 makes it hard for the Committee to come back and
15 answer Question 5.4 because we don’t have EPA’s
16 statement of confidence.

17 My reading of our discussion is
18 that, for most of these COUs, we have low to
19 medium confidence. If we had to put an
20 assessment, I’d say it was low to medium
21 confidence. And I’d kind of like for a few

1 minutes to hear some of the Committee maybe
2 express whether I'm getting this wrong or not.

3 That when we take into account all
4 of these data quality issues, all of these
5 uncertainties, do we still feel that risk is maybe
6 medium to high or high -- not risk, confidence is
7 medium high to high? Or what's your kind of
8 confidence reading on that? And Dr. Cory-Slechta,
9 that's kind of what was in the back of my head,
10 but it took me the 20 minutes of the break to
11 actually research enough to realize that's what
12 was kind of bothering me.

13 **DR. DEBORAH CORY-SLECHTA:** Yeah.

14 And I tried to get to that a little bit right
15 before the break saying, you know, if we had to
16 assign confidence levels to it. If they had
17 assigned confidence levels, then we could weigh in
18 on, well, we agree with that confidence or we
19 don't agree with that confidence. But what comes
20 through is more of the, gee, I'm not sure where
21 things are with this assessment. Clearly, they
22 did the best they could. But there's so many gaps

1 and there's so many uncertainties that I would
2 agree with you. I would say low to medium
3 confidence in the whole human health side of it.

4 **DR. KENNETH PORTIER:** Thank you.

5 Dr. Taioli? I'm sorry. Dr. Taioli?

6 **DR. EMANUELA TAIOLI:** Sorry. I
7 couldn't unmute. Nothing to say.

8 **DR. KENNETH PORTIER:** Dr. Anderson?

9 **DR. HENRY ANDERSON:** Yeah. I would
10 agree with the low to medium. And I think it
11 would be another recommendation could be that EPA
12 needs to have a discussion somewhere about how do
13 you combine these levels of confidence. So if you
14 have low confidence on the exposure assessment,
15 can you have a higher confidence than low as you
16 move it through the system? So you have kind of
17 cumulative issues of having kind of compounding
18 the lack of confidence.

19 So it'd be helpful to have a
20 discussion or a written thing about how they go
21 about that. It seems sometimes you end up with
22 they're highly confident in their assessment of

1 the risk but have low confidence in the data that
2 goes into the assessment. So that, I think, leads
3 to some confusion.

4 **DR. KENNETH PORTIER:** Yeah. It's
5 kind of like the concept of propagation of error,
6 propagation of confidence.

7 **DR. HENRY ANDERSON:** Exactly. Well,
8 it is an error. That's probably a better way to
9 raise it, I think.

10 **DR. KENNETH PORTIER:** And we now
11 that if you propagate uncertainty sometimes
12 uncertainty is propagated when uncertainties don't
13 necessarily lead to more uncertainty because the
14 things may be highly correlated. But I find it --
15 as you say, it's kind of hard to say if we have
16 low confidence in the inhalation risk estimate but
17 medium confidence exposure, do we then have medium
18 confidence in the final risk estimate? Or is it
19 still low confidence? And we haven't seen that,
20 and I think that recommendation is also in the
21 perchloroethylene minutes that are going to come
22 out in a couple of weeks.

1 **DR. HENRY ANDERSON:** Yeah. We had
2 that.

3 **DR. KENNETH PORTIER:** Anyone else
4 want to chime in on this? And Dr. Cory-Slechta, I
5 will send you a paragraph on this with a
6 recommendation. Dr. Van Gosen?

7 **DR. DEBORAH CORY-SLECHTA:** Thank
8 you.

9 **MR. BRADLEY VAN GOSEN:** Hello?

10 **DR. KENNETH PORTIER:** Yeah. Go
11 ahead, Brad.

12 **MR. BRADLEY VAN GOSEN:** You can --
13 back just briefly to the environmental assessment
14 which is a similar thought is, just briefly,
15 obviously, there are many locations where asbestos
16 can join the environment. That's mainly due to
17 the deterioration of asbestos cement pipes,
18 demolition of old buildings, rotting of old
19 asbestos containing materials, landfills, even
20 natural occurrences of asbestos where I live. And
21 these can all add fibers to the streams, the
22 rivers, the lakes, the ground water in some cases,

1 reservoirs that are drinking supplies. And the
2 effects of these fiber loads on the biome from
3 what I've seen are not well studied or often. And
4 some studies of fiber content in drinking supplies
5 are published, but they generally don't reach a
6 conclusion. They just point out that the fibers
7 exist.

8 So the debate over the effects or
9 non-effects of drinking water containing asbestos
10 fibers is not totally resolved even 40 years later
11 where it started, actually, with taconite issue in
12 Minnesota. But I assume that this document, in
13 particular, was focusing on the discussion of
14 environmental effects only on the situations being
15 evaluated by this review, such as the chlor-alkali
16 plants and the brake mechanic shops. And because
17 this data doesn't exist -- we don't know what the
18 runoff from the brake mechanic shops are -- a
19 confidence risk couldn't even exist. The data
20 doesn't exist, so how could you have an
21 assessment. And that's my two cents.

1 **DR. KENNETH PORTIER:** And I think
2 that was Dr. Schlenk's conclusion as well. Dr.
3 Crump? Kenny, your phone may be muted. Oh, Dr.
4 Crump's hands up, but his phone's not connected.
5 So I think we've lost him. We may need to look
6 into that. Dr. Markowitz?

7 **DR. STEVEN MARKOWITZ:** So I have a
8 procedural question. Is it within the realm of
9 options of this Committee to conclude that,
10 because of all the problems that have been
11 discussed and in view of the fact that EPA is
12 committed to a much larger look at the risk of
13 asbestos in terms of legacy and other uses -- is
14 it within the realm of our options to say that
15 this risk evaluation of this narrow slice of
16 chrysotile exposure should not be completed but
17 rather should be subsumed in the larger effort of
18 a more complete risk evaluation of asbestos?

19 **DR. KENNETH PORTIER:** So Steven,
20 this is Ken Portier. We're a science advisory
21 committee. We look at the science, and typically
22 the science we look at is what's focused in the

1 document. This borders on policy. EPA's made a
2 policy decision to address asbestos in two parts.
3 I think what we can conclude is that this may be
4 an incomplete assessment, which I think most of us
5 will agree. And we've made major suggestions on
6 how it can be improved.

7 But without inclusion of the other
8 aspect, we know we're not going to get the
9 complete picture of asbestos risk. That's a
10 given, and we can state that. But I think to tell
11 EPA, "No, don't come out with this," is not --
12 it's not a scientific decision on our part.
13 That's a policy decision. But we can make it
14 clear that our preference would be to see a more
15 complete picture of risk in a true asbestos risk
16 assessment. Does that answer your question?

17 **DR. STEVEN MARKOWITZ:** In part. I
18 appreciate that policy is not within our purview.
19 But I have some concern that this risk evaluation
20 will be finalized at some point. And then when
21 the broader risk evaluation is done, which is
22 going to include a lot more science and a lot

1 broader set of data and studies, that they may
2 have to go back and modify this risk assessment or
3 this risk evaluation because of the ability to
4 look at that broader set of knowledge.

5 Either impacts on the issue of the
6 choice of models, maybe that would be looked at
7 differentially if you're just looking at the
8 textile and mining studies versus the broader set
9 of studies. Impacts on the much more available
10 information about the cancers beyond lung cancer
11 and mesothelioma from the broader literature --
12 there's a much broader set of studies on exposure
13 response. And when all that's looked at -- and
14 then the question is, well, is EPA then going to
15 have to retrospectively, retroactively -- no,
16 retrospectively is the right word -- go back and
17 modify this or change certain aspects of this in
18 order to accommodate the bigger picture. So in
19 that sense, I think it is a scientific issue. But
20 I accept your advice on this.

21 **DR. KENNETH PORTIER:** Well, I think
22 that's a good statement, and that's one we should

1 make -- that we do have that concern. I have that
2 concern, but I don't know if that statement or
3 even a statement like your suggesting -- that we
4 tell EPA outright, "Just stop work on this and do
5 the whole thing" -- is going to have any impact.
6 I think EPA needs, under TSCA, to provide some
7 assessment of risk. And they're going to do that,
8 whether we accept it or not. But I like that
9 statement. I think we should include that
10 somewhere in this document because I tend to agree
11 with you. Dr. Blystone?

12 **DR. SHERI BLYSTONE:** So given that
13 this assessment is for commercial chrysotile
14 current conditions of use -- and it is very narrow
15 -- while I appreciate the idea that data from a
16 broader assessment of all forms of asbestos and
17 legacy uses would have different data, if that
18 data were applicable here, I would assume that EPA
19 has already identified that and used it. So I'm
20 not entirely convinced that the risk of having to
21 adapt this portion of an assessment is as high as
22 it might -- as some members of the Committee might

1 think it is if and when the rest of the assessment
2 is completed.

3 **DR. KENNETH PORTIER:** And Sheri and
4 I have discussed about the issue that we're going
5 to recommend that they retitle this thing to
6 basically show that it's a much more focused risk
7 assessment than an asbestos risk assessment. Dr.
8 Taioli?

9 **DR. EMANUELA TAIOLI:** Yeah. So
10 following up on what Steve just suggested, we are
11 not giving policy advice. We're just saying that
12 the science is incomplete because of the narrow
13 scope. So I'm not sure that it's a policy advice.
14 It's more of a science advice. So it falls in our
15 roles, and I like the idea, actually, that Steve
16 had. I think it's a good one.

17 **DR. KENNETH PORTIER:** Dr. Anderson?

18 **DR. HENRY ANDERSON:** Yeah. I think
19 if you phrase it in the context of does the data
20 support the conclusions in the document. And I
21 think we're saying on the one side -- on the
22 environmental side, there really isn't any data to

1 support the conclusion that they've come to. Now,
2 as you move on to the health side, we have some of
3 the uses that have some data. Others there's no
4 really measured data, and you're using surrogate
5 data. And it adds additional uncertainty to it.

6 So it's kind of then the science
7 judgment call of, as Steve was saying, is it
8 sufficient to support the conclusions of the
9 document. And if it isn't, then we need to say
10 that. And then the follow up to that would be,
11 "And here's what we think you need to do in order
12 to make that happen." If we are confident that we
13 just aren't comfortable that it isn't very
14 accurate and there's a great deal of unsettled
15 data gaps in it but you've done the best you can,
16 then there'll be lawsuits that whatever their
17 ultimate decision is will challenge it one way or
18 the other, unfortunately.

19 **DR. KENNETH PORTIER:** Dr. Everitt?

20 **DR. JEFFREY EVERITT:** Yeah. I'm
21 just struck -- I was a member of the panel that,
22 in 2008, was looking at the binning exercise and

1 the issue of can you do potency by fiber type.
2 And that panel struggled with the exact same
3 questions. And I'm just wondering what are the
4 new data that's come out in the new datasets that
5 really, fundamentally changed how the EPA's
6 looking at could we do this for such a narrow
7 strip of chrysotile.

8 **DR. KENNETH PORTIER:** We don't have
9 an answer to that question. Yeah. I think at
10 this point let's go ahead and go on to Question
11 5.5 and kind of complete Question 5. And then
12 we'll see if EPA has some comments on this overall
13 that they want to add. Dr. Scarano, please read
14 in Question 5.5.

15
16 **CHARGE QUESTION 5 (5.5)**

17 **DR. LOUIS SCARANO:** Thank you, Dr.
18 Portier. Question 5.5, please comment on any
19 other aspects of the environmental or human health
20 risk characterization that has not been mentioned
21 above. Thank you.
22

1 DR. KENNETH PORTIER: Thank you.

2 Dr. Cory-Slechta?

3 DR. DEBORAH CORY-SLECHTA: So

4 comments that I got were largely, again, things
5 that we have already talked about, particularly
6 the studies that people felt hadn't been included.
7 And we have a whole list of those and the
8 accompanying references. In addition to that,
9 there were some indications of some comments
10 regarding studies that weren't felt to be very
11 strong and maybe shouldn't be retained, for
12 example, Cowan 2015, and other places where there
13 were studies that could be included there and were
14 not yet included. So there is kind of a listing
15 of those, and the references are here already.
16 Thank you very much. So we won't be going out and
17 trying desperately to get those from people.

18 The other thing was what Dan brought
19 up before, which were the uncertainties related to
20 water discharge. But again, those are now --
21 those are also in my response to Charge Question

1 5.4. But there were no other listings of things
2 that were not mentioned above.

3 **DR. KENNETH PORTIER:** Yeah. I was
4 looking at your list, and I see that it's stuff
5 that we've already talked about. And there's
6 probably no additional -- no other aspects that
7 have been mentioned. Anyone else on the Committee
8 wish to add an additional aspect? Okay. Not
9 seeing any questions -- not seeing any hands, I'll
10 turn to EPA and see, do you want to have any final
11 comments on the discussion on Question 5? It's
12 been far-ranging and pretty interesting. Dr.
13 Scarano?

14 **DR. LOUIS SCARANO:** Thank you, Dr.
15 Portier and the Committee. I can turn it to
16 others to say something, but to be quite honest,
17 many of the issues that have been raised are
18 things that we've been wrestling with for several
19 years as we've been trying to pull this together.
20 So we appreciate the complications that we've had
21 to deal with and what I think is some good advice

1 that you're providing with new references, new and
2 different ways to think about it.

3 And I also do want to say, and
4 appreciate, that this is a science advisory
5 committee, and this is a true interesting nexus of
6 legal, technical, and policy issues and, as I
7 think most people know, one of the drivers to the
8 whole amendments -- the Lautenberg amendments for
9 TSCA. So it's fitting to me that this is the last
10 of the first ten SACC reviews, and I'm glad that
11 we have the same SACC that has been looking at all
12 ten. So I'll just say that because I've really
13 been writing furiously and have been enjoying the
14 back and forth among the Committee members and my
15 team members and our epi experts. So I'll stop
16 there and see if Dr. Barone or anyone else would
17 like to say anything.

18 **DR. KENNETH PORTIER:** Yeah. I'd be
19 very interested if you or Dr. Barone could address
20 the issue of why we don't see the quality
21 assessments in the conclusions here. The high,

1 medium, low, confidence statements is what I
2 meant.

3 **DR. LOUIS SCARANO:** This is Gino.
4 I'll say it so Stan doesn't have to. And as the
5 management lead, I'll take the responsibility for
6 it. We did spend some time developing confidence
7 tables, especially on the exposure side, and
8 developing a narrative as we ended up doing on the
9 hazard side as well. But we did not get a chance
10 to pull together at the end the kind of
11 culmination table that you're looking for and have
12 seen in some of the other draft risk evaluations.
13 So we will -- I accept that we will be doing that
14 and generating that.

15 I do want to point out that in the
16 slides that we presented on Monday we did make
17 some confidence statements or strength of evidence
18 statements in the risk characterization portions
19 of both the health and the environment. But those
20 are the facts.

21 **DR. KENNETH PORTIER:** Yeah. And
22 unfortunately, the presentation is usually a

1 little late for the Committee to be able to
2 integrate into their discussions. I think at this
3 point, unless Dr. Barone wants to jump in, I think
4 we're ready to move on to Question 6. Is that
5 right? We're at Question 6, right? Yes. I don't
6 know if we'll get through Question 6 today -- it
7 has three parts -- but we will try. We're about
8 25 minutes behind our agenda. So we'll see. Dr.
9 Scarano, please read in Question 6.1.

10
11 **CHARGE QUESTION 6 (6.1)**

12
13 **DR. LOUIS SCARANO:** Thank you.

14 Question 6, additional questions, the Frank R.
15 Lautenberg Chemical Safety for the 21st Century
16 Act, amended TSCA in 2016, states that
17 "potentially exposed or susceptible
18 subpopulations," or PESS, be considered in the
19 risk evaluation process. PESS is defined in the
20 Lautenberg Act to include populations with greater
21 exposure or greater response, including due to
22 lifestyle, dietary, and biological susceptibility
23 factors, than the general population.

1 Question 6.1, has a thorough and
2 transparent review of the available information
3 been conducted that has led to the identification
4 and characterization of all PESS, Sections 2.3.3,
5 3.2.5, and 4.4.1 in the draft risk evaluation? Do
6 you know of additional information about PESS that
7 EPA needs to consider? Additionally, has the
8 uncertainty around PESS been adequately
9 characterized? Next slide for 6.2, or do you want
10 to do it one by one, Dr. Portier?

11 **DR. KENNETH PORTIER:** Well, let's
12 just do 6.1.

13 **DR. LOUIS SCARANO:** Okay. Thanks.

14 **DR. KENNETH PORTIER:** I'll turn to
15 the lead, Dr. Herrick.

16 **DR. ROBERT HERRICK:** Yes. Hi.
17 Thanks. Can everybody hear me alright? Hello?

18 **DR. KENNETH PORTIER:** Yes. We can
19 hear you.

20 **DR. ROBERT HERRICK:** Oh, good.
21 Okay. Just wanted to make sure I had all the
22 mutes off. Let me just start by thanking everyone

1 who contributed to the responses around Question
2 6. There were about five or six main themes that
3 I sort of distilled out from people's responses.
4 A lot of them focused around additional
5 information that EPA needs to consider.

6 So what I'd like to do is start off
7 and just say that, for the most part, there was
8 agreement, almost consensus I think, among the
9 comments that people provided. Having said that,
10 though, I'll start with the first one where there
11 wasn't exactly a strict consensus. There was
12 general agreement, however, that there should be
13 separate risk estimates for smokers. It was
14 recommended that the DRE be expanded to include
15 quantitative estimates of the carcinogenic risk
16 from exposure to asbestos among smokers, also
17 among individuals who have chronic lung disease,
18 including COPD, and pulmonary fibrosis who have an
19 elevated risk of lung cancer and form a
20 susceptible population.

21 There was another comment offered,
22 however, that the person felt there was really

1 insufficient smoking data in the epi mortality
2 studies to allow for this separate analysis by
3 smoking status. So perhaps our recommendation to
4 EPA should be that efforts should be made to
5 address the added risk of lung cancer for smokers.
6 So Dr. Portier, I guess given that there wasn't
7 strict consensus around this pretty important
8 point, do you think it's worth stopping now and
9 asking for discussion?

10 **DR. KENNETH PORTIER:** It's up to you
11 as the lead, if you think there's going to be
12 additional comments. I've noticed in your written
13 material that you go on to have some discussion
14 about that, which is good. We can open it up at
15 this point. I can go through the associates. Dr.
16 Kanarek?

17 **DR. MARTY KANAREK:** Given that
18 smokers are definitely a susceptible population
19 when it comes to lung cancer, they should not be
20 allowed to work with asbestos, plain and simple.

21 **DR. KENNETH PORTIER:** Thank you.
22 Dr. Sheela?

1 **DR. SHEELA SATHYANARAYANA:** I don't
2 have anything else to add.

3 **DR. KENNETH PORTIER:** Thank you.
4 Dr. Taioli? Dr. Taioli, we don't hear you. She's
5 still around.

6 **DR. EMANUELA TAIOLI:** Sorry. I had
7 the computer mute. Sorry. So the only thing, we
8 have a couple of studies on smoking in the section
9 I reviewed, so maybe I'll share it with Question
10 6, just to be added as a possible reference.

11 **DR. KENNETH PORTIER:** Good. Dr.
12 Markowitz?

13 **DR. STEVEN MARKOWITZ:** Yeah. I was
14 the one who made the comment about trying to
15 address smoking, though not by developing separate
16 risk estimates, in part because we heard from some
17 of the authors of the textile studies that
18 actually they don't have the smoking data that
19 would exactly allow them to calculate separate
20 estimates of risk. There's a lot of information
21 on smoking risks in relation to asbestos exposure,
22 but that's from a broader literature, broader than

1 the chrysotile only literature. So that gets back
2 to my previous point that it would be helpful to
3 use that broader literature in a broader analysis
4 of risk.

5 **DR. KENNETH PORTIER:** Dr. Anderson?

6 **DR. HENRY ANDERSON:** Yeah. I would
7 add a special risk group that hasn't been
8 discussed -- there would be the percent of the
9 population that already has asbestosis or fibrotic
10 lung disease. Those are individuals that are at
11 increased risk, both of malignancy as well as
12 further damage to their lungs. So usually you
13 have a person with asbestosis, you would not put
14 them back into an asbestos exposed circumstance.
15 And that, I don't think is -- that preexisting
16 disease is not discussed anywhere. And then I
17 think the genetics was talked about some.

18 There's some good -- public
19 commenter presentations were very interesting.
20 And I think it's covered in the document, but it's
21 what to do about it or how to incorporate it is
22 the challenge. Clearly those individuals who have

1 the wrong genes should not be exposed to asbestos
2 or work with it anyway.

3 **DR. KENNETH PORTIER:** Thank you.

4 Dr. Crump?

5 **DR. KENNY CRUMP:** Yes. I think I've
6 made this point before. But assuming that there's
7 no bias in the North and South Carolina datasets
8 that would bias the data because of differential
9 smoking, to quantify the risk of smoking in the
10 population as a susceptible subgroup, that's the
11 easiest thing we can do. All you have to do is to
12 use -- in the life table analysis is use the data
13 for smoking, and that does it. That incorporates
14 the effect -- the multiplicative effect of smoking
15 and cancer. It does that properly and gives you
16 the risk in smokers.

17 And also as I said before, we might
18 want to consider -- I mean, the risk we have now
19 doesn't apply really to anyone, smokers or
20 nonsmokers because it does a combination of the
21 two. We might also want to recommend that the
22 document present risk for nonsmokers and then use

1 the smoking as a susceptible population and the
2 nonsmokers as the general population. And to do
3 that, you would just use the lung cancer risks and
4 the total cancer risk -- total death risks for
5 nonsmokers. And I think that's very straight
6 forward and very doable, and I think it should be
7 done.

8 **DR. KENNETH PORTIER:** Thank you, Dr.
9 Crump. Dr. Kopylev, you want to comment on this?
10 Dr. Kopylev, we're not hearing you.

11 **DR. LEONID KOPYLEV:** Can you hear me
12 now?

13 **DR. KENNETH PORTIER:** Yes, we can
14 hear you.

15 **DR. LEONID KOPYLEV:** I don't want to
16 minimize the risk of smoking and cancer, but I
17 want to suggest that people look at the combined
18 risk of mesothelioma smoking, which is inhalation
19 unit risk. And you could see the two-thirds of
20 risk comes from mesothelioma and one set of risk -
21 - and I'm approximating -- comes from lung cancer.
22 So while the discussion is important and the

1 analysis approach certainly we can do, but the
2 risk wouldn't change dramatically because
3 mesothelioma doesn't depend on smoking. I just
4 wanted to make this clarification.

5 **DR. KENNETH PORTIER:** Thank you.

6 Dr. Crump?

7 **DR. KENNY CRUMP:** There is an effect
8 on smoking on mesothelioma by virtue of the fact
9 that cigarette smokers don't live long enough to
10 get mesothelioma. So their inhalation risk would
11 go down with mesothelioma in smokers. So it might
12 make more of a difference than we think.

13 **DR. LEONID KOPYLEV:** Thanks.

14 **DR. KENNETH PORTIER:** Yep. Thank
15 you. Dr. Herrick, I think it might be best if you
16 read in your red text so we don't -- I think it'll
17 be more efficient if you kind of lay out all the
18 issues. Otherwise, we're going to be discovering
19 them as we go along. It's going to take us a lot
20 longer.

21 **DR. ROBERT HERRICK:** Sure.

22 Definitely. Let me pick up the pace a little bit

1 here, too. The next topic had to do with the DRE
2 assessment being focused on chrysotile and that we
3 encourage EPA to incorporate in the assessment
4 other asbestos and asbestos-like fibers in
5 addition to chrysotile. We noted that EPA intends
6 to address legacy asbestos use in another risk
7 evaluation.

8 Asbestos exposure encourages a
9 result of engaging in one or more of the six COUs
10 occurs in addition to the asbestos exposure that
11 some workers and consumers have as a result of
12 other sources of exposure to asbestos, including
13 the legacy uses. So the DRE doesn't appropriately
14 account for these legacy uses, which are required
15 by the current regulatory framework. How the
16 increment in exposure associated with the COUs in
17 the current DRE may cause some people with
18 antecedent asbestos exposure to exceed the
19 designated thresholds of unreasonable risk is not
20 addressed.

21 It needs to be evaluated as part of
22 the current risk evaluation. Since asbestos

1 exposure risk is cumulative, those workers who
2 have been exposed to asbestos, whether it be
3 chrysotile or amphibole, are hypersusceptible
4 population. EPA needs to consider other asbestos
5 exposures in addition to the exposures described
6 for the six COUs.

7 Next point is that because of the
8 long latent period for mesothelioma, younger
9 workers should be considered an especially
10 susceptible population. Another comment was that
11 tribes should be considered susceptible groups.
12 The health disparities are relevant to the current
13 manufactured chrysotile asbestos in this DRE, and
14 some points were made that there's 17 percent
15 higher mortality from lung cancer compared to non-
16 Hispanic whites. Alaskan natives have a 53
17 percent higher lung cancer incidence compared to
18 non-Hispanic whites, higher rates of stomach
19 cancer and higher mortality rates compared to non-
20 Hispanic whites, and that the native population
21 has a higher rate of smoking.

1 Another point was made about family
2 members as a susceptible population. Family
3 members of workers exposed to asbestos can be
4 exposed by the clothes or the bodies of the
5 workers when they come home, especially
6 problematic if the exposure is doing the laundry
7 of asbestos exposed workers. The document does
8 not appear to address take-home exposures
9 associated with the transport of asbestos
10 contaminated clothing and other items from the
11 workplace to places of residence.

12 Another point around susceptible
13 populations was a concern that the document may
14 not really have a complete focus on asbestos
15 containing construction materials that may still
16 be in commerce. In the scoping document -- that's
17 EPA 2016-0736 -- it indicates that some of these
18 building materials may contain asbestos. And the
19 sample was from Table 2-3 where, if you follow the
20 link for the Fields Coatings and Mastics
21 Corporation, it takes you to an MSDS for a
22 product, C200 group bond, that contains 4 to 12

1 percent asbestos by weight. Another company,
2 Denver Industrial supplies and Coatings, includes
3 an MSDS for a product that is between 5 and 20
4 percent asbestos. So the question really then is
5 whether EPA was able to determine whether these
6 materials are still in commerce, as they're listed
7 on the companies' websites.

8 Another comment was that EPA should
9 add that there's no evidence that dermal exposure
10 poses a cancer risk, that is unless the asbestos
11 is re-entrained from the skin. Another comment
12 around the risk evaluation of the BAP1 mutations.
13 It mentions that these are associated with an
14 increased risk of malignant mesothelioma and some
15 other mutations less commonly. However, for both
16 scientific and practical reasons, until additional
17 research progress is made, there is at present
18 insufficient justification or opportunity for EPA
19 to make a policy or regulatory decision factoring
20 in BAP1 or other mutation status with regard to
21 asbestos exposure.

1 Those were the response to the first
2 question about the susceptible populations. And
3 maybe this would be a time to see if there were
4 general discussion about that.

5 **DR. KENNETH PORTIER:** Sure. And Dr.
6 Herrick, I was reading your dark text, and I think
7 what you said here in the meeting allows you to
8 add in that dark text. You don't really have to
9 say it because it just essentially explains the
10 phrases that you said. So that's great the way
11 you wrote it up. Does anybody else want to
12 comment on additional information EPA needs to
13 consider around PESS? Dr. Crump?

14 **DR. KENNY CRUMP:** Needs to consider
15 around what? I'm sorry. I didn't quite
16 understand that question.

17 **DR. KENNETH PORTIER:** PESS. This is
18 Question 6.1. P-E-S-S. Your hand is still up, so
19 I don't know if you just couldn't put it down or
20 you specifically wanted to comment.

21 **DR. KENNY CRUMP:** Okay. I did not.
22 I'll move it down. Sorry about that.

1 **DR. KENNETH PORTIER:** Okay. You
2 leave your hand up I'll call on you. Anyone else?

3 **DR. KENNY CRUMP:** Thank you.

4 **DR. KENNETH PORTIER:** I'll invite
5 any of the associates that want to chime in. I'm
6 assuming Dr. Herrick really incorporated your
7 concepts because his writeup is very complete.
8 I'm not seeing any hands go up, Dr. Herrick. Why
9 don't we move on to the discussion of the
10 uncertainty?

11 **DR. ROBERT HERRICK:** Okay.
12 Definitely. The second question had to do with
13 whether the uncertainty around the PESS had been
14 adequately characterized. And I think the general
15 sense of the group was that it has. In general,
16 the assumptions, data gaps, and limitations and
17 the rationale for the risk characterization for the
18 workers were clear and easy to follow.

19 The process descriptions, including
20 the pictures and flow diagrams, were very helpful
21 for reading and understanding the context. There
22 were several sections where EPA indicated the data

1 assumptions, uncertainties and level of
2 confidence. There was a sensitivity analysis done
3 in 4.3.7 and Appendix L, although someone did make
4 the point earlier in the meeting that that
5 actually wasn't perhaps as formal a sensitivity
6 analysis as one might have seen.

7 There's another comment that seems
8 to have been the only time that a sensitivity
9 assessment was done. So the recommendation was
10 that additional sensitivity analyses be considered
11 for those areas so they can be used concurrently
12 with the estimation of confidence to aid in
13 decision making. Another comment was one of the
14 reviewers considered that EPA's practice of going
15 to facilities and making direct observations was
16 considered good practice and that it allowed them
17 to identify areas in the processing where
18 exposures may be present but may not be directly
19 reported in the reported data. So the
20 recommendation was that, resources permitting,
21 that these direct visits be conducted in future

1 reviews. And I think that may be -- yeah. That's
2 the sum of the comments that we had on 6.1.

3 **DR. KENNETH PORTIER:** Does anyone on
4 the Committee wish to comment? Dr. Kanarek? Dr.
5 Sheela? Dr. Taioli? Dr. Markowitz?

6 **DR. SHEELA SATHYANARAYANA:** No, I
7 think Dr. Herrick did a great job of summarizing.
8 I don't have any other comments.

9 **DR. CONCEPCION JIMENEZ-GONZALEZ:**
10 Yeah. This is Dr. Jimenez-Gonzalez, as well. He
11 captured the comments really well.

12 **DR. KENNETH PORTIER:** Thank you.
13 This is where, when we'd be sitting in a room, I
14 could look around and people would shake their
15 heads no, no more. And we could move on.
16 Unfortunately, we don't have a shaking head icon
17 here that would be "No, I'm not interested."

18 I do want to go back and just query
19 one more thing. Way back at the beginning, you
20 also mentioned in addition to lung cancer
21 sufferers COPD and pulmonary fibrosis, and Dr.
22 Crump mentioned asbestosis. I was wondering I

1 don't know how they would -- we have very little
2 data on lung cancer, and I don't even think, Dr.
3 Crump, that there's life tables for some of these
4 other conditions that would help EPA even make a
5 first order attempt at looking at the effect on
6 these populations.

7 I guess we can make the
8 recommendation, but I'm usually a little leery of
9 making a strong recommendation when I can't even
10 see how I would start that analysis. I'll just
11 kind of leave that comment there. Let's go on to
12 Question 6.2. Dr. Scarano?

13
14 **CHARGE QUESTION 6 (6.2)**

15 **DR. LOUIS SCARANO:** Thank you, Dr.
16 Portier. Yes. So Question 6.2, please comment on
17 whether EPA has adequately, clearly, and
18 appropriately presented the reasoning, approach,
19 assumptions, and uncertainties for characterizing
20 risk to workers using PPE, or personal protection
21 equipment? This is in the exposure section
22

1 2.3.1.2, risk section 4.2.1, and Tables 4-3 and 4-
2 38. Thank you.

3 **DR. KENNETH PORTIER:** Thank you.

4 Dr. Herrick?

5 **DR. ROBERT HERRICK:** This is one
6 where we actually had pretty much unanimity among
7 the comments from the reviewers, and it won't
8 surprise you. It's very similar to some of the
9 points that have been made earlier today. EPA
10 cited this study by Riala and Riipinen, looking at
11 the performance of respirators and HEPA units in
12 some abatement scenarios. And they found that
13 actual APFs, the protection factors, were reported
14 as 5, 55, and 4. And EPA concludes that, even
15 with every worker wearing a respirator, some of
16 these workers would not be protected.

17 Comments came into the effect that,
18 well, when you're presenting risks based on use of
19 PPE, the assumption in the DRE is that the level
20 of protection of the PPE is exactly the same as
21 the assigned protection factor of the respirator.
22 But if you look at the way OSHA sets those APFs

1 for respirators based on whether 95 percent of the
2 samples in the study of the respirator where a
3 good PPE training program is in place would have
4 protection at least equal to the assigned APF.
5 Therefore, a worker whose respirator does not fit
6 well, perhaps due to facial geometry or facial
7 hair, or works in an establishment without a good
8 PPE training plan in effect may receive far less
9 protection from the respirator than indicated by
10 the APF.

11 The DRE should discuss these issues,
12 take them into account to the extent possible in
13 calculating the risks to workers wearing
14 respirators with the specific assigned APFs.
15 There's quite a substantial literature around this
16 going back to a NIOSH publication. It's NIOSH
17 number 87-16. And I can just read briefly from
18 that. NIOSH said, "Many of the assigned
19 protection factors that appear in this decision
20 logic are based on laboratory studies and should
21 be regarded as approximate."

1 And they go on and say, "For the
2 present, the APFs should not be considered
3 reliable predictors of performance levels that
4 will be achieved during actual use since APFs are
5 not based on a sufficient amount of workplace
6 testing." The way the calculation of risks were
7 finally done it turns out the assumptions made
8 about the PPE use have a large effect on those
9 risks. If you look at Table 4-38, which is the
10 summary of risk estimates for workers, it clearly
11 shows the effect of assuming different levels of
12 PPE use on the risk determination.

13 So there's a number of places in the
14 text where EPA states that, well, some workers
15 have protection above the nominal APF. Some have
16 it below. Despite acknowledging these
17 limitations, they then go ahead and use the APF
18 values as though they were hard numbers and so
19 calculate risk estimates that are reduced by
20 factors of five or ten or even 25.

21 In one case, in Table 4-3, for sheet
22 gasket stamping, workers were wearing N95s, which

1 EPA did note are not approved for protection
2 against asbestos. But then, inexplicably, they
3 assigned a hypothetical APF of 10 to 25. I think
4 this whole concept of the hypothetical APF should
5 be revisited.

6 And just one last thing, the whole
7 topic around personal protection has been an area
8 of active research at NIOSH almost from the
9 beginning. And NIOSH has a national personal
10 protective technology laboratory that might be
11 able to help on estimating realistic protection
12 factors. And I included in the text here a link
13 to the website. That might be a useful resource
14 to consult. I think that pretty much covers our
15 comments around the APFs and the protection
16 factors.

17 **DR. KENNETH PORTIER:** Thank you.

18 Let's turn to the associates and see if they have
19 anything to add. Dr. Blystone?

20 **DR. SHERI BLYSTONE:** Nothing to add.

21 **DR. KENNETH PORTIER:** Dr. Davies?

1 **DR. HOLLY DAVIES:** I also don't have
2 anything to add right now.

3 **DR. KENNETH PORTIER:** Dr. Taioli?

4 **DR. EMANUELA TAIOLI:** No, nothing to
5 add.

6 **DR. KENNETH PORTIER:** And I don't
7 know if Dr. Jimenez-Gonzalez has rejoined us. She
8 was supposed to. She said she would. There you
9 are. Hey.

10 **DR. CONCEPCION JIMENEZ-GONZALEZ:**
11 I'm here. Yeah. No, I have been here since 3:40.
12 But yeah. Nothing to add other than this is a
13 topic and a piece of feedback that has gone
14 through the entire set, so something to consider
15 for the next set of risk assessments.

16 **DR. KENNETH PORTIER:** Yes. Dr.
17 Anderson?

18 **DR. HENRY ANDERSON:** Just to add
19 again, in the past we've gone over all sorts of
20 issues with respirators and respirator programs.
21 And one could adjust this as assign of proportion
22 of the people that would use it and the other

1 proportion that wouldn't. The other thing is, if
2 you're going to have your workforce wearing a
3 respirator and you're following the NIOSH or OSHA
4 rule, they have to have medical certification to
5 be able to wear a respirator.

6 So there's going to be a proportion
7 of the workforce that are not going to be wearing
8 respirators. Then you have to determine are they
9 going to be put into jobs where they don't have
10 exposure, or are they going to just not be certain
11 of a job? So the proportion of the activity that
12 requires a respirator would be another factor to
13 perhaps have an estimate, and that would be the
14 various companies -- I think, especially on the
15 chlor-alkali talked about when their workers would
16 wear respirators and when they would not.

17 So assuming that the whole period of
18 time when you use a respirator is -- again, you'd
19 have to come up with some kind of an estimate. I
20 don't think there's good data for that. I think
21 it's unrealistic to assume that the workers would
22 all wear respirators, all have the compliance that

1 you need. And therefore, there should probably be
2 a range -- a confidence interval about what factor
3 to use. And you can put into that various
4 assumptions on who uses respirators and who
5 doesn't. And NIOSH could probably have that.

6 And as I said on the brake issues,
7 some of the larger companies have programs. But
8 by and large, the smaller facilities do not. So I
9 think the assumption there that respirators,
10 especially for the ONUs are not used, is a good
11 one. The only question is what proportion of the
12 workers will have an effective respirator on for
13 what period of time. And I think you could do
14 some kind of a simulation to work on changing the
15 -- or coming up with a factor that would be
16 different than just assuming the whole factor is
17 used.

18 **DR. KENNETH PORTIER:** Dr. Blystone,
19 I saw your hand go up and then go back down again.

20 **DR. SHERI BLYSTONE:** Yeah. I was
21 just -- I thought I heard an implication that if
22 you were working somewhere where they had a

1 qualified respiratory protection program and you
2 were not medically qualified to wear a respirator
3 that somehow you would still be doing a job that
4 requires you to use a respirator. And I just was
5 -- I don't believe that that would be the case.
6 If you cannot wear a respirator, you would not be
7 doing jobs that require the use of a respirator,
8 assuming the facility has a quality OSHA
9 compliance respirator program.

10 **DR. HENRY ANDERSON:** I would just
11 say that also depends on the quality of how you do
12 your respirator certification program. Really, I
13 did a paper on that that obviously would not be
14 here where we worked with one of the clinics that
15 did respirator certification and then went to the
16 workers to see did they wear respirators and how -
17 - were they able to for what period of time. So
18 you have people with asthma, COPD that can be
19 certified to wear a respirator for limited periods
20 of time, but for a longer period of time, it's
21 spent around their neck. Or you've got to go to
22 air supply, and most of the companies don't want

1 to do that. I'm just giving my personal
2 experience.

3 **DR. KENNETH PORTIER:** So Sheri and
4 Henry, in the past, we've also talked about the
5 engineering controls concept. And I'm thinking
6 more in terms of the auto mechanic brake and
7 gasket repair people. They're unlikely to be
8 wearing PPE when they do those jobs.

9 **DR. SHERI BLYSTONE:** I would agree
10 with that.

11 **DR. KENNETH PORTIER:** Should we have
12 some recommendation in here about thinking -- the
13 thing about engineering controls. I don't think
14 EPA's really looked at are there engineering
15 controls. Again, thinking of this as a forward-
16 looking document -- and I see this PPE discussion
17 as more supporting the ultimate use of this
18 document in risk management.

19 It helps the industrial hygienists
20 think about the impact of PPE versus some other
21 modifications to reduce risk and engineering
22 controls. I was just trying to think about what

1 the means for the other big COU where we don't see
2 PPE as a factor. And I'm not quite sure whether
3 we put anything here except maybe we'll put our
4 standard statement about -- what is it they call
5 it, Henry? The hierarchy of controls? Dr. Davies?

6 **DR. HENRY ANDERSON:** Yeah. That's
7 what we had. And I would say, if you look at what
8 the chlor-alkali industry's done, they follow
9 that. One of the reasons for doing the short-term
10 sampling is to evaluate the effectiveness of your
11 controls. And the database that was there showed
12 that the application of their control technologies
13 largely worked. But in other circumstances,
14 that's very difficult to change.

15 **DR. SHERI BLYSTONE:** This is Dr.
16 Blystone again. I thought the hierarchy
17 discussion was in the document somewhere, but
18 maybe I missed it. It's important to always
19 reference that.

20 **DR. HOLLY DAVIES:** This is Dr.
21 Davies. I was agreeing with Dr. Blystone because
22 Section 2.3.1.2 explains the hierarchy of controls

1 pretty well, I think better than what we've seen
2 in the past. It also clearly explains the
3 specific OSHA regulations.

4 **DR. SHERI BLYSTONE:** Mark the day
5 when Holly and I agree.

6 **DR. KENNETH PORTIER:** That explains
7 why it wasn't in the discussion. Okay. I stand
8 correct.

9 **DR. HENRY ANDERSON:** I went over the
10 (inaudible) pages before.

11 **DR. KENNETH PORTIER:** For the rest
12 of the listeners, you should know that the charter
13 committee has PPE fatigue. It's been a topic of
14 strong discussion in each of the nine previous
15 risk assessments. And the fact that we don't
16 spend two hours on it here may be a sign that the
17 EPA staff -- most of them who've listened to our
18 discussions in the past -- already know what we're
19 going to say on this. But we're saying it anyway.
20 Let's go on to Question 6.3. Dr. Scarano?

21

CHARGE QUESTION 6 (6.3)

DR. LOUIS SCARANO: Thank you, Dr. Portier. 6.3, please comment on whether EPA has adequately, clearly, and appropriately presented the reasoning, approach, assumptions, and uncertainties for characterizing risk to occupational nonusers, or ONUs, who would not be expected to use personal protective equipment, Sections 4.2.1 and 4.3.7.

DR. KENNETH PORTIER: Dr. Herrick?

DR. ROBERT HERRICK: Okay. Let me summarize the comments that we have on this point. Before I do that, though, I will just flash back to the previous discussion for one minute. If EPA wanted to talk about engineering controls in a really detailed assessment in the brake and clutch area, that article by John Sheehy that's included in the discussion, actually, that's exactly what he looked at was the various engineering controls as they were applied to brake work. So that is already out there in this case.

1 But back to this one, 6.3, our
2 general conclusion was that it's appropriate for
3 EPA to not assume that PPE is used by the ONUs.
4 In general, we thought that the approach EPA took
5 to looking at the risk of the ONUs was well
6 supported. The data gaps, limitations, and
7 rational for the risk characterization were clear
8 and easy to follow and that the process
9 descriptions and the pictures and flow diagrams
10 were useful to help the readers understand the
11 context. One particular thing on that question,
12 though, about the use of PPE.

13 In addition to the protection
14 factors that we just talked about, there is this
15 issue concerning the frequency of actual
16 respirator use. And EPA acknowledges in Table 4-3
17 the use in repair with aftermarket auto brakes and
18 clutches, there's an unknown amount of respirator
19 use occurring among these workers. But there may
20 be some.

21 Actually, there is some information
22 on this point. NIOSH and the Bureau of Labor

1 Statistics did a survey of respirator use across
2 industry groups. The title of the document is
3 "Respirator Usage in Private Sector Firms," 2001.
4 This could be a useful source of information to
5 try to approach this whole topic about respirator
6 use a little more quantitatively. So that's going
7 to be a recommendation that EPA consult that
8 resource. And I think that pretty much captures
9 what we had to say on 6.3.

10 **DR. KENNETH PORTIER:** I was just
11 looking through your writeup. Let's go ahead and
12 turn to the associates. Dr. Davies, anything to
13 add?

14 **DR. HOLLY DAVIES:** I don't think so
15 right now.

16 **DR. KENNETH PORTIER:** Dr. Taioli?

17 **DR. EMANUELA TAIOLI:** No, nothing to
18 add.

19 **DR. KENNETH PORTIER:** Dr. Jimenez-
20 Gonzalez?

21 **DR. CONCEPCION JIMENEZ-GONZALEZ:**
22 Nothing to add. Thanks.

1 **DR. KENNETH PORTIER:** I think those
2 are the three associates on that question. Yes.
3 Anyone else want to comment on characterizing risk
4 to ONUs? I do want to emphasize in the writeup
5 you do have a statement that says, "It's
6 appropriate for EPA to not assume PPE for ONUs."
7 That's your clear conclusion there.

8 **DR. ROBERT HERRICK:** Yeah. I think
9 that -- I don't recall that anyone disagreed with
10 that.

11 **DR. HENRY ANDERSON:** I would only
12 raise the confidence levels. Again, if we go back
13 to the exposure assessment, there's very limited
14 data for ONUs, and the methodology used there has
15 a great deal of variability in it and uncertainty.
16 And I don't know if that's been adequately carried
17 forward.

18 I certainly agree on the PPE issue.
19 I think that's very cut and dry. But the
20 confidence in the ONU risk assessments, I don't
21 know if that carries with it the uncertainties and
22 confidence in the exposure assessment components.

1 So I think we talked about that previously in
2 other areas, but that would just be one thing to
3 underscore here as well.

4 **DR. KENNETH PORTIER:** And I guess
5 this section might be a good place to mention the
6 -- whatever we call the occupational bystander,
7 who's a type of ONU or not an ONU, the person
8 who's being exposed by their contact with the
9 worker, not with the workplace. So ONUs are
10 exposed through their contact with the workplace
11 and workers in the workplace. But this
12 occupational bystander concept that we talked
13 about previously is also maybe not acknowledged in
14 this document and should be.

15 Any additional comments? I'll turn
16 to Dr. Scarano and the EPA team. Any questions,
17 clarifying comments?

18 **DR. LOUIS SCARANO:** This Gino. None
19 from me. Anyone from the team?

20 **DR. STANLEY BARONE:** This is Stan
21 Barone. With regard to that last comment about
22 occupational bystanders, it would be really

1 helpful to get clarification on what is meant by
2 an occupational bystander. We currently consider
3 occupational nonusers as workers that are in the
4 vicinity of the user. But I'm not really sure
5 what is meant by an occupational bystander.

6 **DR. KENNETH PORTIER:** Dr. Barone, I
7 think that was the term created to address people
8 like the family at home who come in contact with
9 the exposed clothes of a worker, the person who
10 washes their clothes and who shakes it out and
11 then gets exposed. I think that was the general
12 concept, but we had that conversation earlier.
13 And I think there is a note for us to do just that
14 to kind of clarify that term.

15 **DR. STANLEY BARONE:** Thank you.

16 **DR. KENNETH PORTIER:** I'm trying to
17 remember who coached that term, but we did have a
18 conversation on that earlier. I'm just looking
19 forward to the degree of preparation that we have
20 for Questions 7.1 and 7.2 and whether we want to
21 move forward in the next 40 minutes to try to
22 complete those questions, in which case we

1 wouldn't reconvene tomorrow, or whether we want to
2 just stop at this point. Maybe I'll turn to Dr.
3 Johnson, who's the lead on these two questions and
4 ask him where we stand. Except maybe Dr. Johnson
5 had to step out again, in which case that solves
6 the problem. Yeah.

7 I think he did have to step out. He
8 sent me a message. Yeah. Okay. I think at this
9 point we've completed what we had planned to
10 discuss in today's agenda. And at this point I'm
11 going to turn the meeting back over to DFO Dr.
12 Wong for any final comments. We'll reconvene
13 tomorrow morning and finish off the last two
14 questions. I don't expect that to be a long
15 meeting, but it will give us an opportunity to
16 think overnight about any additional topics that
17 we haven't discussed. Dr. Wong?

18 **DR. DIANA WONG:** Thank you. This is
19 Dr. Diana Wong. As the DFO, I would like to thank
20 the SACC peer reviewers and the public listening
21 online. This concludes the peer review activities
22 for the agenda for today, and we will reconvene

1 tomorrow morning for Day 3 at 10:00 a.m. Eastern
2 Time. Today's session is now adjourned.

3 **[MEETING ADJOURNED FOR THE DAY]**
4

OPENING OF MEETING - DAY 4

MS. SARAH WILSON: -- two minutes to give the last few people time to connect. Good morning. Welcome to the fourth day of the meeting on the U.S. EPA Peer Review of the Draft Risk Evaluation for Asbestos. Battelle is an EPA contractor providing meeting support for this series. This event is being recorded. Please be aware that the host may use Webex chat to share announcements with all attendees, but attendees will not be able to respond. I will now introduce Dr. Diana Wong, the designated federal official.

DR. DIANA WONG: Thank you. Good morning. I am Dr. Diana Wong, and as designated federal officer, it is my pleasure to open the fourth and final day of the four-day online meeting for the Science Advisory Committee on Chemicals, TSCA SACC, Peer Review of EPA's Draft Risk Evaluation for Asbestos. This week's Webex-hosted meetings have gone well. However, if you encounter any problems with audio or video

1 transmissions today, please go to

2 www.epa.gov/tsca-peer-review.

3 For members of the press, EPA media
4 relations staff are available to answer your
5 questions about this meeting. Please address all
6 questions to Ken Labbe. His email address is L-A-
7 B-B-E-dot-K-E-N-@-E-P-A-dot-G-O-V. Overall, the
8 Committee sessions are going well, and I thank all
9 our committee members for your contributions to
10 this meeting. I now turn the meeting over to the
11 Chair, Dr. Portier.

12 **DR. KENNETH PORTIER:** Good morning.
13 Thank you, Dr. Wong. We're going to start the
14 meeting by doing our Committee roll call so that
15 the public knows who's attending. Dr. Anderson.

16 **DR. HENRY ANDERSON:** I'm here.

17 **DR. KENNETH PORTIER:** Dr. Barton.

18 **DR. CHARLES BARTON:** I'm here.

19 **DR. KENNETH PORTIER:** Dr. Bennett.

20 **DR. STEVEN BENNETT:** I am here.

21 **DR. KENNETH PORTIER:** Dr. Blystone.

22 **DR. SHERI BLYSTONE:** Good morning.

1 DR. KENNETH PORTIER: Dr. Cory-
2 Slechta?

3 DR. DEBORAH CORY-SLECHTA: Yes. I'm
4 here.

5 DR. KENNETH PORTIER: Dr. Davies.

6 DR. HOLLY DAVIES: I'm here.

7 DR. KENNETH PORTIER: Dr. Doucette.
8 I think we were having some issues with Dr.
9 Doucette's connection. He was on. Now he's off.
10 Dr. Jiménez-Gonzalez.

11 DR. KENNETH PORTIER: Dr. Jiménez-
12 Gonzalez.

13 DR. DIANA WONG: I don't think she's
14 here this morning.

15 DR. KENNETH PORTIER: Oh, that's
16 right. Dr. Johnson.

17 DR. MARK JOHNSON: I'm here.

18 DR. KENNETH PORTIER: Dr. Kaufman.

19 MR. ALAN KAUFMAN: I'm here. Good
20 morning.

21 DR. KENNETH PORTIER: Dr. Kissel.

22 DR. JOHN KISSEL: Here.

1 **DR. KENNETH PORTIER:** Dr. Rowlands
2 emailed me that he's going to join the Committee
3 at 10:30. Dr. Schlenk?

4 **DR. DANIEL SCHLENK:** Good morning.

5 **DR. KENNETH PORTIER:** Dr. Sheela

6 **DR. SHEELA SATHYANARAYANA:** I'm
7 here.

8 **DR. KENNETH PORTIER:** Thank you.
9 Dr. Crump.

10 **DR. KENNY CRUMP:** Here.

11 **DR. KENNETH PORTIER:** Dr. Everitt.

12 **DR. JEFFREY EVERITT:** Good morning.

13 **DR. KENNETH PORTIER:** Dr. Herrick?

14 **DR. ROBERT HERRICK:** Good morning.

15 **DR. KENNETH PORTIER:** Dr. Jayjock?

16 **DR. MICHAEL JAYJOCK:** I'm here.

17 **DR. KENNETH PORTIER:** Dr. Kanarek?

18 **DR. MARTY KANAREK:** I'm here. Good
19 morning.

20 **DR. KENNETH PORTIER:** Good morning.
21 Dr. Markowitz?

22 **DR. STEVEN MARKOWITZ:** Present.

1 DR. KENNETH PORTIER: Dr. Sheppard?

2 DR. ELIZABETH SHEPPARD: Good

3 morning.

4 DR. KENNETH PORTIER: Good morning.

5 Dr. Shukla?

6 DR. ARTI SHUKLA: I'm here.

7 DR. KENNETH PORTIER: Dr. Taioli?

8 DR. EMANUELA TAIOLI: Present. I'm

9 here.

10 DR. KENNETH PORTIER: Dr. Van Gosen?

11 MR. BRADLEY VAN GOSEN: Here.

12 DR. KENNETH PORTIER: Did Dr.

13 Doucette join us? Dr. Doucette, you're muted in

14 Webex. I think we'll have to reach out. I can

15 see him as having logged in and connected, but we

16 can't hear him.

17 DR. WILLIAM DOUCETTE: Ken, this is

18 Bill Doucette.

19 DR. KENNETH PORTIER: Yeah. Oh, hey

20 Bill, good morning.

1 **DR. BILL DOUCETTE:** Sorry, I was
2 having some technical difficulties with the
3 computer.
4

5 **FOLLOW UP ON PREVIOUS DAY DISCUSSIONS**
6

7 **DR. KENNETH PORTIER:** Yeah. Thank
8 you. So we have a quorum, and I thank all of you
9 for showing up on Day 4. We have only one more
10 question left to address. But before we go into
11 Question 7, I wanted to open it up to any follow-
12 up discussions to questions we've discussed
13 previously. Dr. Jayjock, you seemed to indicate
14 that you wanted -- you have something you wanted
15 to discuss?

16 **DR. MICHAEL JAYJOCK:** Yes. That's
17 right. Is now a good time?

18 **DR. KENNETH PORTIER:** Yes. If we
19 could bring up the slide you wanted?

20 **DR. MICHAEL JAYJOCK:** Yes. Thank
21 you. Yeah. Actually, the discussion yesterday
22 about the quality of the data and what we should

1 be doing about it, it brought me back to this
2 particular slide if we can get it up. And I guess
3 while that slide's coming up, I just wanted to say
4 that every risk assessment -- or virtually every
5 risk assessment I've done in the last four decades
6 has presented me with the dilemma that we have in
7 this particular risk assessment. That is that
8 there's uncertainty around the exposure assessment
9 and there's uncertainty around the hazard
10 assessment. I'm seeing a white screen. Is
11 everybody else just seeing a white screen?

12 **DR. KENNETH PORTIER:** I think that
13 was -- I think your first slide was blank. There
14 it is.

15 **DR. MICHAEL JAYJOCK:** Oh. Okay.
16 There it is. Thank you. Okay. So given that the
17 relatively high uncertainty here in both what I
18 call dose response, or exposure limit, and
19 exposure, you can see that when you have high
20 uncertainty in both and you draw a line across,
21 you wind up concluding that the exposure is above

1 the exposure limit, which is the case here with
2 regards to brake linings.

3 So this is the putative risk, and
4 the conclusion of the DRE is that it's an
5 unacceptable risk, and I certainly support that.
6 Almost every assessment that I've done could have
7 been flipped by getting better data -- flipped
8 meaning going from an unhappy face conclusion of
9 unacceptable risk to a conclusion of acceptable
10 risk or not unacceptable risk. So if you lower
11 the uncertainty around the exposure by actually
12 getting information about what's going on with
13 brakes -- and the true situation is, as Dr.
14 Paustenbach and others have said, that it's not
15 there -- then you have lower uncertainty and lower
16 putative levels of exposure, and you conclude a
17 lack of significant risk.

18 I think the threshold question is do
19 we have enough information? Is there a threshold
20 of information and data to allow us to make an
21 assessment of risk? And there are some situations
22 where you don't have that threshold of

1 information, and I've told clients that. But in
2 this case, I believe we do, and I think the Agency
3 has done so appropriately. That's all I wanted to
4 say.

5 **DR. KENNETH PORTIER:** This is Ken
6 Portier. Looking at this graph, though, your dose
7 response kind of goes up, right? You're saying if
8 you had better information, the actual dose might
9 go up. The actual dose might go down as well,
10 right?

11 **DR. MICHAEL JAYJOCK:** Yeah. Now,
12 I'm sorry. Yeah. I'm sorry for the confusion
13 there. I'm putting it in as the exposure limit,
14 or EL, which is the reciprocal of the dose
15 response. So in this particular case, the
16 exposure limit would go up, or the benchmark dose
17 would go up.

18 **DR. KENNETH PORTIER:** Well, what I
19 was saying, though, it's quite likely it could go
20 down as well with better information. You don't
21 know. That's the issue of uncertainty, right?

1 **DR. MICHAEL JAYJOCK:** Right. The
2 other idea is that we intentionally -- you'll
3 notice that I only have half an error band here.
4 The meatball is the point estimate for most
5 likely. And the error bands are -- the upper
6 error band for exposure limit and the lower band
7 for exposure, that's because of the natural
8 tendency. And I think it's been kind of a time-
9 honored way of doing risk assessments that you
10 bias for reasonable overestimation or reasonable
11 worst case.

12 **DR. KENNETH PORTIER:** Okay. Any
13 questions? Dr. Barone, you wanted to comment on
14 this.

15 **DR. STAN BARONE:** Yes. I like the
16 way Dr. Jayjock has tried to frame this. I wanted
17 to make sure that folks understand what he's
18 talking about in the left panel is about the
19 hazard and what the dose response -- how you
20 characterize the dose response and its
21 distribution. And on the right side of this graph
22 is really about the exposure. And taking the --

1 basically looking at the uncertainty and the
2 distribution of those estimates together is giving
3 you some idea of what the degree of overlap may
4 be. I think it's a good pictorial way to describe
5 what we have.

6 **DR. KENNETH PORTIER:** Thank you.

7 And Dr. Jayjock --

8 **DR. MICHAEL JAYJOCK:** And I think

9 that --

10 **DR. KENNETH PORTIER:** Oh. Go ahead.

11 **DR. MICHAEL JAYJOCK:** Yeah. I was
12 just going to say the reality is -- and I think I
13 tried to state this in my comments. The reality
14 is that there may not be any exposure or any
15 significant exposure in brakes. But given the
16 state of knowledge that we have, we have these
17 error bands that are raising that number -- that
18 exposure number above the benchmark dose. And
19 that's what I would call -- that's what I call
20 putative risk or the risk that we're dealing with.

21 **DR. KENNETH PORTIER:** Thank you.

22 I'm not quite sure if you want to include this in

1 our meeting minutes, which I recommend, but I
2 think you'll need to provide a couple of
3 paragraphs of what you just said to go along with
4 that. And in one of the previous questions that
5 we've discussed, probably you're talking about --
6 whatever it is -- Question 6.3 or 6.2, something
7 like that?

8 **DR. MICHAEL JAYJOCK:** Okay. Yeah.
9 I'll see where it belongs and send that to the
10 lead.

11 **DR. KENNETH PORTIER:** Thank you.
12 Any additional follow-up questions for the
13 previous days? So I'm not seeing any. I guess
14 we'll just kind of move on to Charge Question 7.
15 I need to -- there's Dr. Scarano. So Dr. Scarano,
16 good morning, and would you read in Question 7?

17
18 **CHARGE QUESTION 7 (7.1, 7.2)**
19

20 **DR. LOUIS SCARANO:** Yes. Thank you,
21 Dr. Portier. Question 7, Content and
22 Organization, EPA's Final Rule, *Procedures for*

1 *Chemical Risk Evaluation Under the Amended Toxic*
2 *Substances Control Act* stipulates the process by
3 which EPA is to complete risk evaluations under
4 the Frank R. Lautenberg Chemical Safety Act for
5 the 21st Century. As part of this draft risk
6 evaluation for asbestos, EPA evaluated potential
7 environmental, occupational, and consumer
8 exposures. The evaluation considered reasonably
9 available information, including manufacture, use,
10 and release information, and physical-chemical
11 characteristics. It is important that the
12 information presented in the risk evaluation and
13 accompanying documents is clear and concise and
14 describes the process in a scientifically credible
15 manner. Next slide, please.

16 To increase the quality and
17 credibility of scientific information disseminated
18 by the EPA, EPA uses the peer review process
19 specifically as a tool for determining fitness of
20 scientific information for the intended purpose.
21 The questions below are intended to guide the peer
22 reviewers toward determining if EPA collected,

1 used, and disseminated information that is "fit
2 for purpose" based on utility (the data's utility
3 for its intended users and for its intended
4 purpose), integrity (the data's security), and
5 objectivity (whether the disseminated information
6 is accurate, reliable, and unbiased as a matter of
7 presentation and substance). The peer reviewers'
8 critical focus should pertain to recommendations
9 of the technical information's usefulness for
10 intended users and the public. Next slide,
11 please.

12 Question 7.1, please comment on the
13 overall content, organization, and presentation of
14 the asbestos draft risk evaluation. Please
15 provide suggestions for improving the clarity of
16 the information presented. Question 7.- -- would
17 you like me to do 7.2, Dr. Portier or --

18 **DR. KENNETH PORTIER:** Yeah. It's
19 the same team that's looking at it, so go ahead
20 and read 7.2.

21 **DR. LOUIS SCARANO:** Thank you.
22 Please comment on the objectivity of the

1 information used to support the risk
2 characterization and the sensitivity of the
3 Agency's conclusions to analytic decisions made.
4 Thank you.

5 **DR. KENNETH PORTIER:** Thank you.

6 Dr. Johnson? We've asked Dr. Johnson to be the
7 lead on these two questions.

8 **DR. MARK JOHNSON:** Okay. Generally
9 the Committee wanted to commend the EPA for doing
10 an overall good level of organization and clarity.
11 I think it's particularly informative that we had
12 a couple Committee members who are new to the
13 process and thought it was fairly well organized,
14 and they could follow things fairly clearly. But
15 there are some suggestions. One was to add more
16 explanation to Appendix J for an explanation of
17 modeling for IUR derivation -- potentially color
18 coding maybe part of the tables to highlight key
19 data.

20 One is a big issue with the
21 consistency of terms, using commercial chrysotile
22 versus asbestos. And I think that was brought up

1 before if you could be consistent with terms
2 there. There was also comments from one Committee
3 member regarding debating things like talc and how
4 it was covered regarding children's exposure,
5 ovarian cancer. Some more explanation in how that
6 may apply or may not apply is probably needed. A
7 clear layman's approach to what's covered is
8 needed up front in the introduction.

9 A single area of the DRE that one
10 Committee member thought was most lacking was
11 Section 3 in the Mode of Action. They felt the
12 concepts are important here, particularly issues
13 associated with fiber size, physio-chemical
14 properties, bio-persistence should be mentioned
15 and put into a framework of why low dose
16 chrysotile exposure for friction products may
17 differ substantially from other forms of low-dose
18 chrysotile exposure. For the environmental
19 sections, significant detail was provided showing
20 evaluation of the number of databases and site
21 visits in discussions with COUs showing
22 significant degree of uncertainty for

1 environmental exposures due to a general lack of
2 monitoring data. Hazard sections for
3 environmental effects were relatively well
4 presented with the Weight of Evidence section for
5 risk characterization. The environmental
6 assessments, once again, is limited in providing
7 uncertainty discussions, and worst-case scenarios
8 were not provided. It's generally unclear how the
9 Agency is defining uncertainty in its relationship
10 to the precautionary principle, which the Agency
11 should drive towards worst-case assessments where
12 necessary data is necessary.

13 Also, a problem formulation
14 assessment also generates data gaps that need to
15 be discussed in the uncertainty section. In this
16 particular case, it appears the Agency ignored the
17 gap identified in the problem formulation and did
18 not mandate monitoring data specifically for
19 chlor-alkali use to assess the efficacy of
20 asbestos removal by filtration. And so an overall
21 recommendation is data gaps should be identified
22 in the risk characterization as uncertainties, and

1 EPA should decide up front how much uncertainty is
2 acceptable to even determine what an unacceptable
3 risk may be to make that sort of decision and what
4 kind of information is specifically needed to
5 close those information gaps. I bring up the
6 example with the IRIS program on the hazard ID
7 side where I believe they decide that any value
8 they derive that would require an uncertainty
9 factor greater than 3,000 is a number not worth
10 deriving.

11 The same sort of logic may go into
12 these sorts of risk assessments where the Agency
13 may want to think about what is the minimum amount
14 of data we need up front to be able to make a
15 decision? It goes on. The risk determination
16 section for environmental risk, again, was
17 unclear. It's not logical to state low or no
18 potential for environmental risk to aquatic
19 receptors because water release is associated with
20 COUs and not expected were not identified. I
21 think this was brought up before. A more
22 appropriate determination would be environmental

1 risk could not be ascertained because we just
2 don't know because water releases associated with
3 COUs was not expected and were not identified.

4 Several commenters noted that
5 several varieties of amphiboles are present in
6 both asbestiform and non-asbestiform habits, like
7 tremolite, anthophyllite, and actinolite. Whether
8 asbestiform varieties of these substances are
9 referenced, asbestos should be attached to their
10 name to be more specific. One commenter noted
11 that the CAS Registry Numbers referring to the
12 non-asbestiform varieties for these amphiboles are
13 used when the CAS Registry Numbers for asbestiform
14 varieties should have been used. These errors
15 should be corrected both in the RE and scope
16 document insofar as the current version of the RE
17 contains them. And that's what I have for 7.1.

18 I'll go on to 7.2. They say
19 objectivity information used to make and support
20 risk was somewhat unclear for the Environmental
21 Risk Assessment sections, again. To make an
22 analytic decision of risk based on a lack of

1 analytical data is non-objective. Further, the
2 selection of some key studies to be used to
3 address environmental hazards seems to be
4 arbitrary and of limited value. Some ranges of
5 reported effects -- I think we brought this up
6 before -- spans four orders of magnitude with no
7 explanation for this variation. I think more is
8 needed here.

9 In some cases, reviewers thought
10 that logic could help support this decision when
11 all of it is presented together. But that logic
12 is not there complete. So a recommendation would
13 be to provide a more complete review of the
14 environmental tox data providing a range of data
15 for species concentration endpoint for other the
16 data. As mentioned before, these data could be
17 presented as a SSD or scatter diagram table
18 listing study relevance for hazard ID and quality
19 for COC derivation. Either provide some exposure
20 data from release points to be clear that risks to
21 aquatic organisms could not be determined. And
22 that's what I have received so far.

1 **DR. KENNETH PORTIER:** Thank you, Dr.
2 Johnson. Let me query the associates. I heard a
3 lot of Dr. Schlenk's comments there, but let's see
4 if he has any additional ones. Dan?

5 **DR. DANIEL SCHLENK:** No. Mark got
6 everything -- did a great job.

7 **DR. KENNETH PORTIER:** Thank you.
8 Dr. Everitt?

9 **DR. JEFFREY EVERITT:** No. He
10 covered it.

11 **DR. KENNETH PORTIER:** Dr. Crump?

12 **DR. KENNY CRUMP:** Just a couple of
13 minor points. So it seems to me that the idea of
14 exposure lag is never discussed in the document,
15 but it's used in all the dose response models --
16 exposure lag of 10 years. I think that issue
17 somehow needs to be discussed and pointed out.
18 And I believe I've said this before, but I'll say
19 it again. I really think the overall readability
20 would be improved if tables listing risk to
21 workers and ONUs and various exposure scenarios
22 would be reduced to only one table for each use

1 investigated. I think that too many tables would
2 confuse the general public. That's all I have.

3 Thank you.

4 **DR. KENNETH PORTIER:** Thank you, Dr.
5 Crump. Dr. Sheppard?

6 **DR. ELIZABETH SHEPPARD:** Yeah. Can
7 you hear me?

8 **DR. KENNETH PORTIER:** Yes.

9 **DR. ELIZABETH SHEPPARD:** Well, so
10 something that I don't think we've discussed much
11 and I've been reflecting on because I think
12 there's -- it's a lot of work, but it's something
13 to consider -- is the need to do some independent
14 reanalysis of the evidence that is used in the
15 document. And the reason I say that is because
16 sometimes the results that are reported are
17 misleading, and EPA could potentially discover
18 that. I hesitate to say that because I think it
19 opens a can of worms. But I think that it's
20 something to consider with respect to the
21 objectivity of the information.

1 With respect to -- and this is all
2 in response to 7.2 in response to sensitivity. I
3 think that what we discussed yesterday about
4 choosing the risk model based on the overall AIC
5 fit criterion is not focusing on the range of
6 exposures of interest in the data, and I think
7 that that's a particular concern. There were a
8 lot of assumptions that weren't quantified with
9 respect to direction and magnitude, and I think
10 that doing a better job on that would also be
11 helpful.

12 **DR. KENNETH PORTIER:** Thank you, Dr.
13 Sheppard. Dr. Johnson, I wanted to make a comment
14 on Question 7.1 relating to overall content. And
15 I think we've mentioned this a couple of times,
16 but I think this is the place we need a
17 recommendation about how the title of the document
18 relates to the content. In the executive summary
19 in the paragraph starting on line 695, it
20 specifically limits the risk evaluation to the
21 chrysotile form and excludes the other five forms
22 because they are the subject of a SNUR.

1 So I think the title basically
2 should reflect what the risk assessment has done.
3 So either we leave the title like it is, and EPA
4 then needs to do a much better job of discussing
5 all forms of asbestos, which I know some on the
6 panel have pushed for in our emails back and forth
7 as we have discussed the different questions. And
8 I think it's come up in a couple of places in the
9 questions that there would be a desire to actually
10 do an asbestos risk assessment. Or EPA needs to
11 focus this title back on what they've really
12 focused which is COUs moving forward -- current
13 and future COUs of chrysotile asbestos. And that
14 would be my recommendation on that. Does anyone
15 else wish to comment on either of these two
16 questions? Dr. Blystone.

17 **DR. SHERI BLYSTONE:** I just would go
18 on record as supporting your recommendation, Dr.
19 Portier.

20 **DR. KENNETH PORTIER:** Thank you.
21 Dr. Markowitz.

1 **DR. STEVEN MARKOWITZ:** Yeah. I also
2 agree with that recommendation. I'd like to make
3 a separate comment about clarity and organization.
4 The section that describes the epidemiology
5 studies that were used to obtain the potency
6 estimates in the IUR is very clear in terms of the
7 universe of studies considered, the strategy for
8 selecting studies that were ultimately used, and
9 the basis for decision making. And whether
10 there's universal agreement on that strategy or
11 the results is sort of a separate issue.

12 I think by contrast, in the
13 occupational exposure section, how EPA got from
14 the data quality review of the relevant studies to
15 the actual selection of the studies they used and
16 the fact that very few studies are actually cited
17 in the text of the DRE about occupation exposures
18 -- specifically about gasket use and brake repair
19 -- that requires both clarity and I also think a
20 broader use of the relevant literature. Thank
21 you.

1 **DR. KENNETH PORTIER:** Thank you, Dr.
2 Markowitz. Dr. Anderson.

3 **DR. HENRY ANDERSON:** Yeah. I would
4 support changing the title. Like we've made that
5 recommendation in our very opening remarks on
6 Question 2. So I would go further and say it
7 ought to be "commercial chrysotile" because that's
8 really the product that's being incorporated into
9 other materials. And that sort of gets into
10 there's trace contamination in it, and I think by
11 calling it "commercial" that covers that. We
12 don't have to go into great depth about all the
13 trace contaminants, other than to recognize they
14 exist.

15 **DR. KENNETH PORTIER:** Thank you,
16 Henry, for that modification to the
17 recommendation. Are there any other
18 recommendations? This is always a good time when
19 we also look at the executive summary and kind of
20 consider whether it's adequate. And I thought it
21 was actually a pretty good executive summary.
22 There's a lot of language that EPA puts in at the

1 very beginning that's legalistic, I guess, would
2 be the right term. But the rest of it is a pretty
3 good summary of what they've done. Any additional
4 comments? Dr. Taioli. We're not hear- --

5 **DR. EMANUELA TAIOLI:** I just wanted
6 to say I support the -- I support the change of
7 the title as well. I think it's an important
8 matter.

9 **DR. KENNETH PORTIER:** Thank you.
10 Dr. Anderson, I see your hand's still up.

11 **DR. HENRY ANDERSON:** Oh, sorry about
12 that.

13 **DR. KENNETH PORTIER:** Dr. Doucette.

14 **DR. WILLIAM DOUCETTE:** Ken, I
15 mentioned this before, and I think maybe it's more
16 appropriate for this question. But I think it
17 would be helpful for the EPA to provide a short
18 summary section on the analytical methods used to
19 quantify the various asbestos particle types and
20 sizes and maybe discuss how the methodology has
21 evolved over time and how that might impact the
22 study quality assessments.

1 **DR. KENNETH PORTIER:** Good
2 suggestion. Dr. Anderson, I still see your hand
3 up.

4 **DR. HENRY ANDERSON:** Yeah. No. I
5 have one more thing here. I think it might be
6 helpful -- it was helpful at least to me to
7 understand what the timeline would be for legacy.
8 But it just mentions that legacy issues will be
9 considered at a later time. I think it would be
10 helpful to put a timeline for that so, when the
11 public looks at this, they're not expecting this
12 to occur within the next few months.

13 **DR. KENNETH PORTIER:** Thank you.
14 That's a good point. Dr. Davies, you didn't make
15 the recommendation that we restructure the whole
16 document as you've done, I think, for nine of the
17 previous reports. I just wondered is this one --
18 have you given up on that, or is this just better
19 than the previous ones?

20 **DR. HOLLY DAVIES:** I guess I just
21 got tired of making the recommendation. I still
22 find it -- skipping around the way it is, it's

1 harder for me to go through than the way they
2 present it in the slides on the first day, where
3 you kind of go through all the environmental and
4 then you go through the exposure and the human
5 assessment. I also like the idea -- since I'm
6 talking, I'll mention I like the idea of changing
7 the title to the commercial chrysotile.

8 **DR. KENNETH PORTIER:** Yeah. For
9 those on the Committee who haven't been there
10 before, one of the issues that we've discussed a
11 number of times is reorganizing this so that
12 environmental exposures, hazard, and risk are
13 discussed as a unit followed by human health
14 exposure, hazard, and risk. And that would be an
15 alternative structure to the document that many on
16 the charter Committee have kind of preferred EPA
17 consider, moving forward. I'm kind of --

18 **DR. HOLLY DAVIES:** Dr. Portier?

19 **DR. KENNETH PORTIER:** Yeah.

20 **DR. HOLLY DAVIES:** This is Dr.
21 Davies again. I would also add to Dr. Markowitz's
22 comment on the occupational exposure the same

1 thing for the consumer exposure -- making it more
2 clear how they got from the studies and reviewed
3 and in the data quality assessment to the study
4 selected.

5 **DR. KENNETH PORTIER:** Thank you.

6 Dr. Blystone.

7 **DR. SHERI BLYSTONE:** I just wanted
8 to thank you, Dr. Portier, for making sure we put
9 in the record yet again that we prefer all
10 environment in once and then the human health --
11 and Dr. Davies for making that recommendation
12 again. And I would also support -- it's always
13 been a bit of a black box to try to figure out how
14 the studies get selected and used, so I think that
15 those recommendations from Holly and from Dr.
16 Markowitz to clarify that and make that more
17 explicit is a good one.

18 **DR. KENNETH PORTIER:** Dr. Markowitz?

19 **DR. STEVEN MARKOWITZ:** Yeah. Just I
20 had a further thought about the title. Most
21 exposure to legacy asbestos is going to be
22 commercial chrysotile because commercial

1 chrysotile represented 95 percent of all the
2 asbestos used in the U.S. So this document is
3 really about the current intentional uses of
4 commercial chrysotile. So it's quite narrow and
5 limited, and I think that the new titling should
6 reflect that.

7 **DR. KENNETH PORTIER:** That's a good
8 point. Dr. Kaufman?

9 **MR. ALAN KAUFMAN:** Yeah. I just
10 wanted to add my support to the recommendations
11 that were made by Dr. Davies and Dr. Markowitz.

12 **DR. KENNETH PORTIER:** Kind of last
13 call. Dr. Taioli, your hand's up.

14 **DR. EMANUELA TAIOLI:** Yeah. So
15 following up on the last comment about the
16 intentional use, I wonder if we could recommend a
17 paragraph or an introduction where it is explained
18 how this focus has limited a lot of the evaluation
19 of the subsequent evaluations that were done
20 throughout the document because that's what we had
21 to deal with for four days -- that a lot of the
22 uncertainty were related to the narrow focus of

1 the scopers. So maybe there could be an
2 introduction or a section describing that.

3 **DR. KENNETH PORTIER:** Thank you. I
4 think that was my intent when I listed line 695
5 paragraph 2 of the approach in the executive
6 summary because I think that's where EPA kind of
7 lays out that focus and the approach. And the
8 next paragraph talks about the scoping document
9 and the problem formulation. So at least in the
10 executive summary, that's where that defining of
11 focus tends to occur. Dr. Johnson.

12 **DR. MARK JOHNSON:** Yes, sir. One
13 thing -- and I just propose this for discussion
14 with the group as well and consideration by the
15 EPA. We have sections titled "Environmental
16 Hazards," and, in my world, environmental hazards
17 are hazards to not only humans but non-humans as
18 well. I mean, if you have an environmental
19 release, that could potentially result into a
20 human health hazard. It could also result into a
21 hazard to aquatic organisms or even terrestrial
22 organisms and wildlife. And so I'm wondering if

1 it would be better to rename that -- are "Hazards
2 to Environmental Receptors?" That would actually
3 include humans. Has the EPA ever given this any
4 thought? It doesn't apply here, but it may apply
5 to other chemicals in the future.

6 **DR. KENNETH PORTIER:** So you're
7 talking about like Section 3.1 right now that's
8 labeled "Environmental Hazards" probably should be
9 labeled "Hazards to Environmental Receptors," and
10 then "Human Health Hazards" could be "Hazards to
11 Human Health" or something like that?

12 **DR. MARK JOHNSON:** Yes.

13 **DR. KENNETH PORTIER:** Dr. Crump?

14 **DR. KENNY CRUMP:** Just a brief
15 comment about the title, I want to be sure that we
16 include the word "asbestos" in the title, not just
17 leave it with "chrysotile." I think "chrysotile
18 asbestos" would be much more definitive, easy to
19 understand with the general public than just
20 "chrysotile." That's all.

1 **DR. KENNETH PORTIER:** Yeah. Thank
2 you. Dr. Taioli, your hand's still up. Dr.
3 Johnson?

4 **DR. EMANUELA TAIOLI:** Sorry, a
5 mistake.

6 **DR. MARK JOHNSON:** Yes. So just one
7 request from Dr. Sheppard. You mentioned earlier
8 -- I just didn't get the correct wording on, I'm
9 afraid -- when you made a remark about choosing
10 the best BMDL that we should focus on a range of
11 exposure data available? If you could send me the
12 precise language, I would appreciate it for the
13 text. Thank you.

14 **DR. KENNETH PORTIER:** Dr. Crump,
15 your hand's still up. Dr. Kopylev, you wanted to
16 add some clarifying comments? Dr. Kopylev, we're
17 not hearing you. Your phone may be muted.

18 **DR. LEONID KOPYLEV:** I would like to
19 clarify maybe what she means by general asbestos
20 could address uncertainties in this assessment?
21 It doesn't seem that general asbestos would
22 address both exposures for brakes because this is

1 chrysotile asbestos. It doesn't seem that general
2 asbestos would clarify the dose response issues.
3 So it would be helpful for EPA to point out how
4 general asbestos could reduce actual uncertainties
5 to chrysotile asbestos because I don't think this
6 has been articulated. Thank you.

7 **DR. KENNETH PORTIER:** Dr. Anderson,
8 your hand flew up.

9 **DR. HENRY ANDERSON:** Yep. I'm still
10 looking at points I wrote down on here. One thing
11 I think that would be good for them to expand on,
12 I noticed early on in the introductory part of the
13 document they say -- they had a goal of updating
14 or reaffirming the 1988 EPA inhalation unit risk
15 for general asbestos. And they made no comment
16 about whether they reaffirm that or any -- I mean
17 there really isn't any reference to how does this
18 one link to that. Is this totally separate or --
19 I think that it would be nice to have some
20 statement in there -- did they review the EPA
21 inhalation unit risk and find that to be

1 inappropriate here or -- there really isn't any
2 mention about that.

3 **DR. KENNETH PORTIER:** So, Dr.
4 Anderson, kind of which section are you pointing
5 to?

6 **DR. HENRY ANDERSON:** I don't have
7 the document open right. It's fairly early on.

8 **DR. KENNETH PORTIER:** So it would be
9 nice if you could kind of point to chapter, verse
10 so that Dr. Johnson can kind of get that comment
11 in context. Or if you could type that up and send
12 that to Dr. Johnson, I think that would be
13 helpful.

14 **DR. HENRY ANDERSON:** Yeah. I'll try
15 to find that.

16 **DR. KENNETH PORTIER:** Oh, I
17 remember. I was looking at the table of contents
18 and all of the sudden something else clicked. One
19 of the recommendations we've made in the past,
20 which I haven't heard -- which was really what we
21 discussed prior to getting into the questions with
22 this regulatory nexus issue. And I think we have

1 a Section 1.3 where EPA summarizes regulatory and
2 assessment history. But it doesn't really kind of
3 lay out this regulatory nexus for the substance
4 under review.

5 And our discussion prior to the
6 questions, there was a lot of -- how do I say this
7 -- a lot of interest in the Committee on having
8 that regulatory nexus better summarized in the
9 introductory portion of the report to help the
10 reader put this whole thing into context. And
11 we've seen in the past the Clean Water Act, Clean
12 Air Act, and other regulations have kind of prior
13 -- have done prior studies and kind of owned
14 certain aspects of the risk from those chemicals.
15 But asbestos is even more complicated, I think,
16 than some we've seen in the past. So I think it's
17 more -- it's even more important in this case to
18 spend some time on that regulatory nexus
19 discussion in Section 1.3 beyond just summarizing.
20 Dr. Anderson?

21 **DR. HENRY ANDERSON:** Ken?

1 **DR. KENNETH PORTIER:** Yeah. Your
2 hand was up.

3 **DR. HENRY ANDERSON:** Yeah. I found
4 the reference I had. It's on page 18 of the
5 document, line 744-745.

6 **DR. KENNETH PORTIER:** Oh, 744-745.
7 Oh, wow. Okay. Good. That should help Dr.
8 Johnson. Anything else? Dr. Kopylev, I see your
9 hand's still up. I don't know if you've got
10 another comment to make? I'm not sure we've
11 answered your question, but I think we'll kind of
12 keep that in mind as we write up the report. Any
13 additional comments? These have all been good.
14 Dr. Johnson, you capture all this?

15 **DR. MARK JOHNSON:** Largely, yeah. I
16 just could get more clarity from the comment I
17 mentioned before from Dr. Sheppard. And Dr.
18 Anderson, I got your citation, but I didn't quite
19 get the nexus of your comment. So if you could
20 just email that to me, I would appreciate it.

21 **DR. KENNETH PORTIER:** Okay. Well,
22 these are the last two questions that EPA has

1 posed for us so -- and -- oh, I should turn to EPA
2 and say has this discussion on Question 7 -- do
3 you have any additional comments, questions, Dr.
4 Scarano?

5 **DR. LOUIS SCARANO:** Thank you, Dr.
6 Portier. Well, I actually would like to point out
7 the paragraph that Dr. Anderson points to in the
8 executive summary. I think he accurately portrays
9 what's in the first half, but the second half of
10 that same paragraph says we change direction
11 because we focused on commercial chrysotile. But
12 I just wanted to point that out. I'd like to
13 think that that was clear.

14 But other than that, I know I
15 appreciate all the recommendations that we've
16 heard today on Questions 7.1 and 7.2. And then
17 the only other thing is if someone can answer Dr.
18 Kopylev's question, that would be great.

19 **DR. KENNETH PORTIER:** And Dr.
20 Kopylev's question was how does discussing
21 asbestos in general support the risk discussion

1 for commercial chrysotile asbestos? Is that kind
2 of the gist of the question?

3 **DR. LOUIS SCARANO:** I think so, but
4 I think he also referred to brakes. But I'll
5 refer to Leonid if he wants to --

6 **DR. LEONID KOPYLEV:** Right. To
7 issues -- to uncertainties identified in the
8 Committee discussion, there are definitely
9 uncertainties the Committee identified. But the
10 changes that involving general asbestos except
11 maybe informing non-cancer hazard -- which is
12 important but wouldn't change any conclusions in
13 the document. So outside of that, there seems to
14 be no information that's really for brakes or to
15 dose response in the Carolina cohorts. Thank you.

16 **DR. KENNETH PORTIER:** Thank you.
17 Dr. Markowitz?

18 **DR. STEVEN MARKOWITZ:** Sure. Well,
19 what this document does for the first time is --
20 and this is in response to Dr. Kopylev's question.
21 What this document does for the first time is try
22 to establish a fiber-specific risk assessment.

1 That's never been done. That approach was
2 specifically rejected multiple times in the past,
3 including the 2008 review by the Scientific
4 Advisory Board of EPA.

5 In the latest IARC review of
6 asbestos 2012, it specifically said that there was
7 a lot of uncertainty around exposure, so much so
8 that you really couldn't differentiate lung cancer
9 risk by fiber type, which the current risk
10 evaluation in a sense tries to amend by specifying
11 a risk by fiber type, specifically commercial
12 chrysotile, albeit under limited circumstances.
13 So the issue is -- and this is part of the reason
14 why I raised yesterday whether this risk
15 evaluation should be subsumed in a broader risk
16 evaluation of asbestos -- is whether that larger
17 literature -- which is a lot about mixed
18 exposures, but much of that mixed exposure is
19 commercial chrysotile -- whether that brings a
20 broader context and wealth of additional studies
21 that would be relevant to this issue and to the

1 broader issue of asbestos-related risk for lung
2 cancer.

3 These comments are a little less
4 true for mesotheliomas since there appears to be
5 some hierarchy of potency. But on the lung cancer
6 side, this has not been established. So in that
7 sense, the distinction between commercial
8 chrysotile and the broader asbestos exposure is
9 artificial. And there should just be an
10 acknowledgement that this is a fiber-specific risk
11 assessment, which, frankly, has never been done
12 before and never been accepted before.

13 **DR. KENNETH PORTIER:** Dr. Kopylev?

14 **DR. LEONID KOPYLEV:** Well,
15 technically, that first -- this is not the first.
16 I think Libby amphibole asbestos was for the
17 fiber, so that's a small technical point but --
18 thank you.

19 **DR. KENNETH PORTIER:** Thank you.
20 Dr. Blystone.

21 **DR. SHERI BLYSTONE:** Yeah. This
22 discussion sort of revolved around the continuing

1 evolution of understanding of what a TSCA risk
2 assessment is all about, right? Because it does
3 bring in this element of conditions of use as
4 determined by the administrator. So when we talk
5 about this narrow focus, that's exactly right.
6 That's what they were trying to do.

7 **DR. KENNETH PORTIER:** Yeah. In
8 thinking about this discussion, to me this is a
9 paragraph that goes at the beginning of our
10 minutes report that kind of sets the -- I don't
11 know -- the tone for how the Committee tended to
12 look at this. I think what Dr. Markowitz just
13 said is very useful. And I don't think it changes
14 anything, but it kind of helps the reader of our
15 report kind of see where the Committee was coming
16 from on this.

17 And, Dr. Blystone, I understand also
18 what you've added to that. And you said while
19 it's a fiber-specific report, it's done to address
20 these ongoing and future conditions of use that
21 seem to be focused on one fiber type because the
22 other five fiber types have basically covered

1 under a "we're not going to use those anymore"
2 rule. Right? I think we need to kind of say that
3 up at the beginning. Dr. Markowitz, if you could
4 type up that -- what we just said -- a short
5 paragraph and send that to Diana and myself, I'd
6 like to include that kind of -- that verbiage
7 somehow at the beginning of our -- as a general
8 comment.

9 **DR. STEVEN MARKOWITZ:** Sure.

10 **DR. KENNETH PORTIER:** Thank you.

11 Dr. Anderson.

12 **DR. HENRY ANDERSON:** Yeah. I just
13 wanted to support what Dr. Markowitz said that
14 it's -- since so much of the asbestos was
15 chrysotile, I think narrowing it to current uses
16 is probably the direction to go. Again, back to
17 my first question about whether the IRIS IUR,
18 which included all of the chrysotile studies,
19 included the cohorts that have mixed exposures.
20 I think they need to say why that isn't used or
21 why you have to be solely focused on the
22 individual fiber.

1 That I think is kind of the problem
2 that I think throughout the last days we've talked
3 about how there's so much -- the whole literature
4 base is so much richer when you include all of the
5 groups that have been exposed to mixtures of
6 asbestos, as it were. And so keeping it focused
7 here only on the current uses when there's all
8 these other exposures to chrysotile that they're
9 still ongoing in workers who are exposed -- that
10 is this legacy issue -- if legacy is included, I
11 would think it needs to be included here as well,
12 so I would agree with what Steve was saying.

13 I don't know how we put that
14 together in this document or not. But I think
15 that is an important issue, and I don't want it
16 to, when this comes out -- to imply that somehow
17 the IRIS assessment and that IUR, which really is
18 more relevant to the current worker exposures --
19 somehow that isn't appropriate to this group of
20 worker exposures as well who may have prior
21 exposures to all the other fibers as well as
22 additional chrysotile.

1 **DR. KENNETH PORTIER:** Dr. Davies?

2 **DR. HOLLY DAVIES:** Since we're
3 talking about the title, I just wanted to bring up
4 that EPA is still deciding on the scope for the
5 legacy asbestos supplemental assessment. And so
6 whatever title this has should fit with what their
7 thinking of for a title for the next one to
8 distinguish them and make it clear what assessment
9 is in which document.

10 **DR. KENNETH PORTIER:** Thank you. We
11 would hope that together those two reports will be
12 the risk assessment for asbestos. So somehow the
13 two titles have to merge. I understand.

14 **DR. HOLLY DAVIES:** Yeah. And I was
15 thinking we're kind of saying we recommend you
16 name it this. We don't know what the other name
17 is going to be, so just kind of leaving that open.

18 **DR. KENNETH PORTIER:** Yeah. Dr.
19 Kanarek?

20 **DR. MARTY KANAREK:** I support Dr.
21 Markowitz in, by leaving out the mixed exposure
22 cohorts, we leave out a whole literature on lung

1 cancer and cigarettes. It's a gigantic
2 literature. And it's really -- there's a hole in
3 this document because we're concentrating on
4 commercial chrysotile. And I think anybody out of
5 this country who sees that will say "They left
6 that whole thing out because they focused in on
7 this one fiber type." And I think we're going to
8 regret that.

9 **DR. KENNETH PORTIER:** Thank you.

10 Dr. Kopylev, I see your hand's still up. I can't
11 tell if it's a legacy hand up or you have
12 additional comment? Thank you. His hand went
13 down. Dr. Kanarek, your hand's still up. I want
14 to point out to listeners that Dr. Rowlands joined
15 us at 10:30, as he said he would, so that he's
16 included on the attendance.

17 Any additional comments because I'm
18 thinking we've just about wrung everything out of
19 this? So I'm not seeing any comments, and I think
20 we've had a good discussion here. At this point,
21 I'm going to turn the meeting back over to the DFO
22 Diana Wong, and with, basically, the

1 recommendation that the Committee has done its
2 deliberation, and we're ready to close. Dr. Wong.

3
4 **NEXT STEPS**

5
6 **DR. DIANA WONG:** Thank you. At this
7 point, I will go over the next steps. The panel
8 chair has requested the lead discussants to
9 prepare summary slides for their charge questions
10 and circulate to your team members for input. So
11 these summary slides should be completed by
12 tomorrow and sent to me and the Chair. I will
13 then compile all the summary slides and circulate
14 to the entire panel next week.

15 So, basically, this compilation of
16 summary slides will be the basis of the panel
17 report, which is based on the deliberations during
18 this four days' meeting. The lead discussants
19 will then start writing up their sections for the
20 report, and they will send me -- they will
21 circulate to their team members for feedback

1 first. And then they will send it to me in about
2 one week.

3 I recognize that the SACC members
4 have been very busy because they're still writing
5 up the reports for the perchloroethylene meeting
6 just a week ago. So I really appreciate your hard
7 work working on two reports at the same time. But
8 nevertheless, our goal is to complete the draft
9 report in 60 days, so I really appreciate your
10 hard work. So is there any questions about the
11 timeline? If not, as the DFO, I would like to
12 thank the SACC peer reviewers and the public
13 listening online.

14 This week's TSCA SACC meeting is a
15 virtual meeting. And from what I can hear, we
16 have many members of the public listening, and I
17 thank all for your virtual attendance online. I
18 want to make a special recognition of the OPPT
19 team for being online in an efficient and
20 responsive way and for providing your interactions
21 with the SACC peer reviewers.

1 In addition, I will say over and
2 over again that the peer reviewers really deserve
3 our continual thank you for your work and robust
4 deliberations and contributions that will lead to
5 the recommendations and advice to the Agency on
6 how the draft evaluation for asbestos can be
7 refined. I would also like to point out that
8 today's meeting marks an important milestone. The
9 Science Advisory Committee on Chemicals, or SACC,
10 has completed its peer review on the first ten
11 chemicals under the TSCA Lautenberg Act.

12 A special thanks to the Chair, Dr.
13 Ken Portier, who has led the SACC for the review
14 of ten chemicals in one year. On behalf of the
15 staff of the Office of Science Coordination and
16 Chemicals, OSCP, I would like to express our
17 appreciation for support and Dr. Portier's
18 dedication on his role in leading the Committee
19 through the peer reviews. At this point, I would
20 like to introduce our office director, Dr. Hayley
21 Hughes to make a remark. Dr. Hughes?

1 **DR. HAYLEY HUGHES:** Good morning.

2 Can you hear me okay?

3 **DR. DIANA WONG:** Yes.

4 **DR. HAYLEY HUGHES:** Great. Well
5 Thank you, Diana. I appreciate that. Again, as
6 she said, this marks an important milestone, and
7 our thanks go out to all of the hard work that the
8 TSCA SACC has done over the last year on these
9 first ten chemicals. And, again, we'd like to
10 extend our appreciation for Dr. Portier's
11 dedication in his role and really spearheading the
12 discussions and making sure that we get
13 recommendations from the SACC in a timely manner.
14 So, again, thank you, again, for all of your hard
15 work and effort. And I'll turn the time back over
16 to Diana Wong to wrap up this meeting.

17 **DR. DIANA WONG:** Thank you. So this
18 concludes the peer review activities for today,
19 and this concludes the SACC meeting for the Peer
20 Review of EPA's Draft Risk Evaluation for
21 Asbestos. This day's session is now adjourned.

1 **DR. KENNETH PORTIER:** Thank you,
2 everyone. Goodbye.

3
4 **[MEETING ADJOURNED]**