

CALIFORNIA ENVIRONMENTAL CONTAMINANT BIOMONITORING PROGRAM
(BIOMONITORING CALIFORNIA)
SCIENTIFIC GUIDANCE PANEL MEETING
CONVENED VIA WEBINAR BY: OFFICE OF ENVIRONMENTAL HEALTH
HAZARD ASSESSMENT
CALIFORNIA ENVIRONMENTAL PROTECTION AGENCY
STATE OF CALIFORNIA

MONDAY, NOVEMBER 8, 2021
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JAMES F. PETERS, CSR
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APPEARANCES

PANEL MEMBERS:

Megan R. Schwarzman, MD, MPH, Chair

Carl Cranor, PhD, MSL

Oliver Fiehn, PhD

Ulrike Luderer, MD, PhD

Thomas McKone, PhD

Penelope (Jenny) Quintana, PhD, MPH

Veena Singla, PhD

José R. Suárez, MD, PhD, MPH

OFFICE OF ENVIRONMENTAL HEALTH HAZARD ASSESSMENT:

Vince Cogliano, PhD, Deputy Director, Scientific Programs

Dave Edwards, PhD, Chief Deputy Director

Cheryl Holzmeyer, PhD, Health Program Specialist, Safer Alternatives Assessment and Biomonitoring Section, Reproductive and Cancer Hazard Assessment Branch

Sara Hoover, MS, Chief, Safer Alternatives Assessment and Biomonitoring Section, Reproductive and Cancer Hazard Assessment Branch

Shoba Iyer, PhD, Staff Toxicologist, Safer Alternatives Assessment and Biomonitoring Section, Reproductive and Cancer Hazard Assessment Branch

M. Elizabeth Marder, PhD, Senior Environmental Scientist, Cancer Toxicology and Epidemiology Section, Reproductive and Cancer Hazard Assessment Branch

Kristi Morioka, JD, Senior Staff Counsel

APPEARANCES CONTINUED

CALIFORNIA DEPARTMENT OF PUBLIC HEALTH:

Kathleen Attfield, ScD, Chief, Biomonitoring
Investigations and Outreach Unit, Exposure Assessment
Section, Environmental Health Investigations Branch

Jianwen She, PhD, Chief, Biochemistry Section,
Environmental Health Laboratory Branch

Nerissa Wu, MPH, PhD, Chief, Exposure Assessment Section,
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DEPARTMENT OF TOXIC SUBSTANCES CONTROL

June-Soo Park, PhD, Chief, Environmental Chemistry
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PRESENTERS:

Kate Hoffman, PhD, School of Environmental Sciences and
Policy, Nicholas School of the Environment, Duke
University

Anna Kärrman, PhD, Deputy Head, School of Science and
Technology, Örebro University

Karl Palmer, Deputy Director, Safer Consumer Products
Program, California Department of Toxic Substances Control

Tom Webster, DSc, Boston University School of Public
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INDEX

	<u>PAGE</u>
Welcome Vince Cogliano, PhD, Deputy Director, Office of Environmental Health Hazard Assessment (OEHHA)	1
Overview of the Meeting Meg Schwarzman, MD, MPH, Chair, Scientific Guidance Panel (SGP)	5
Program Update and Overview of California Activities on Perfluoroalkyl and Polyfluoroalkyl Substances	
Presentation: Nerissa Wu, PhD, MPH, California Department of Public Health (CDPH)	9
Presentation: Karl Palmer, Department of Toxic Substances Control	17
Presentation: Kathleen Attfield, ScD, CDPH	31
Panel Questions	46
Public Comment	56
Panel Discussion and Input	59
Methods for PFAS Analysis: Possibilities and Challenges	
Presentation: Anna Kärrman, PhD, Örebro University	75
Panel and Audience Questions	89
Afternoon Session	98
Relative Importance of PFAS Exposure Sources for the General U.S. Population	
Presentation: Tom Webster, DSc, Boston University School of Public Health	98
Panel and Audience Questions	116
PFAS in Indoor Environments and Drinking Water: Relevance for Human Exposure	
Presentation: Kate Hoffman, PhD, Duke University	126
Panel and Audience Questions	148
Afternoon Discussion Session	
PFAS Biomonitoring to Support Exposure Reduction Efforts: Next Steps	
Introduction: Meg Schwarzman, MD, MPH, SGP Chair	165
Panel, Guest Speaker, and Audience Discussion	168, 209

INDEX CONTINUED

	<u>PAGE</u>
Plan for 2022 SGP Meetings	
Presentation: OEHHA	202
Panel and Audience Comments	205
Open Public Comment Period	208
Wrap-up and Adjournment	215
Reporter's Certificate	217

1 children, and second, challenges in conducting air
2 filtration intervention studies, including study design
3 issues.

4 Panel members, guest speakers, and the audience
5 participated in an open discussion section about air
6 pollution biomarkers of effect to delve further into study
7 design considerations, aspects of measurement and results
8 interpretation. Discussion topics included: Optimal
9 timing for urine sample collection; recommendations for
10 exposure questionnaire content, such as cooking practices
11 and consumption of barbecued, grilled, or fried food, time
12 spent outdoors, and mask wearing; designing a reminder
13 such as a refrigerator magnet for parents about study
14 activities, and; possible options for including a control
15 group.

16 A summary of input for the July meeting and
17 complete transcript will be posted on the July Scientific
18 Guidance Panel meeting page at biomonitoring.ca.gov.

19 Because we're meeting virtually today, I would
20 like to have the Scientific Guidance Panel members
21 introduce themselves. I'll call on each member
22 alphabetically by last name. First up is Carl Cranor.

23 PANEL MEMBER CRANOR: Thank you, Vince. Carl
24 Cranor, Distinguished Professor of Philosophy at the
25 University of California, Riverside, and member -- faculty

1 member of Environmental Toxicology on the same campus.

2 DR. COGLIANO: Thank you.

3 Ulrike Luderer.

4 PANEL MEMBER LUDERER: Good morning. Ulrike
5 Luderer, Professor of Environmental and Occupational
6 Health in the Program of Public Health at the University
7 of California, Irvine.

8 DR. COGLIANO: Thank you.

9 Tom McKone.

10 PANEL MEMBER MCKONE: Good morning. I'm Tom
11 McKone. I'm Professor Emeritus of Environmental Health
12 Sciences at the University of California, Berkeley, School
13 of Public Health.

14 DR. COGLIANO: Thank you.

15 Jenny Quintana.

16 PANEL MEMBER QUINTANA: Hi. I'm Penelope or
17 Jenny Quintana. I'm a Professor of Environmental Health
18 at the School of Public Health at San Diego State
19 University.

20 DR. COGLIANO: Thank you.

21 Veena Singla.

22 PANEL MEMBER SINGLA: Good morning, Veena Singla.
23 I'm a Senior Scientist with the Natural Resources Defense
24 Council in the Healthy People and Thriving Communities
25 Program.

1 DR. COGLIANO: Thank you. I should announce that
2 this will be Veena Singla's last meeting as a Scientific
3 Guidance Panel member.

4 MS. HOOVER: I'm sorry, Vince. I need to chime
5 in real quickly. Elizabeth, José Suárez is actually
6 attending and he doesn't have a link. Could you send him
7 a panelist link right now.

8 DR. MARDER: I'll send him a link immediately.

9 MS. HOOVER: Thank you so much. I'm going to
10 text him.

11 Back to you Vince, and when he's on, you can
12 introduce José as well.

13 DR. COGLIANO: Okay. I'll do.

14 DR. MARDER: If he's also -- Sara, if he has
15 joined. I can promote him. I didn't see him.

16 MS. HOOVER: No, he said he doesn't have a link.
17 He can't find a link, so I just want to make sure we sent
18 the link.

19 DR. MARDER: Sending one right now.

20 MS. HOOVER: Thank you.

21 DR. COGLIANO: Okay. I'll be watching for his
22 name to pop up. Okay. Anyway, I would like to announce
23 that this is going to be Veena Singla's last meeting as a
24 Scientific Guidance Panel member. Veena was appointed by
25 the Senate Rules Committee in 2018, and prior to that,

1 provided input at SGP meetings as a program stakeholder.
2 She has decided not to seek reappointment to give more
3 attention to her many other commitments, which include
4 work as a Senior Scientist at the Natural Resources
5 Defense Council, serving on the U.S. EPA's Children's
6 Health Protection Advisory Committee, the National
7 Toxicology Program's Board of Scientific Counselors, and
8 the Board for the Clean Air -- Clean Production Action.
9 She did not come to this decision lightly, but is
10 confident that she is leaving behind both a strong program
11 and a supportive and involved SGP. We would all like to
12 thank her for her outstanding service to the people of
13 California and wish her the best -- the very best in
14 future endeavors.

15 And now I think I will turn the microphone over
16 to Meg Schwarzman, the Chair of the SGP who will provide
17 more details about today's meeting.

18 CHAIRPERSON SCHWARZMAN: Thank you. I'm Dr. Meg
19 Schwarzman, a Physician and --

20 MS. HOOVER: I'm sorry, Meg. Can you just hold?
21 For some reason, although we have these invitations, José
22 didn't get his and Oliver didn't get his, so can you just
23 hold here.

24 CHAIRPERSON SCHWARZMAN: Should we just wait?

25 MS. HOOVER: Yeah.

1 CHAIRPERSON SCHWARZMAN: Okay.

2 MS. HOOVER: Let's just hold for a moment.
3 Elizabeth, if you could follow up. We have it listed that
4 we sent it out, so you might want -- might be able to
5 just --

6 DR. MARDER: I have sent -- I have sent José's.
7 If either wants to use the public link, I can promote them
8 instantaneously on the website.

9 MS. HOOVER: Okay. Does Oliver have his --
10 Oliver have his? I thought that that was sent out, so you
11 should just be able to forward it.

12 DR. MARDER: Yes.

13 PANEL MEMBER SUÁREZ: This is José Suárez. Good
14 morning, everybody. I'm in now.

15 MS. HOOVER: Thank you so much, José, and sorry
16 about that slight glitch.

17 PANEL MEMBER SUÁREZ: Thank you.

18 PANEL MEMBER FIEHN: Hello. Now, I'm in.

19 MS. HOOVER: Thank you so much, Oliver. Sorry
20 for that slight glitch. Okay. Everybody is on. Welcome.
21 Over to you Meg.

22 PANEL MEMBER SCHWARZMAN: I guess we should
23 return back and have José and Oliver introduce themselves.

24 PANEL MEMBER FIEHN: Oliver Fiehn, UC Davis, mass
25 spectrometry analysis of chemicals.

1 CHAIRPERSON SCHWARZMAN: Thank you.

2 José.

3 PANEL MEMBER SUÁREZ: I'm José Suárez, Associate
4 Professor in the Herbert Wertheim School of Public Health
5 at UC San Diego.

6 CHAIRPERSON SCHWARZMAN: Thank you. And I'm Meg
7 Schwarzman on the faculty at UC Berkeley School of Public
8 Health, Environmental Health Sciences Division.

9 And with that, thank you for getting everybody in
10 who needed to be in and introduced and we'll start the
11 rest of the meeting. I want to give an overview of the
12 meeting by starting with the Panel goals for today. We
13 will, as usual, first receive a program update with the
14 remainder of the meeting focusing on discussion of
15 perfluoroalkyl and polyfluoroalkyl substances, which we
16 refer to collectively as PFASs. State staff will discuss
17 California's activities on PFAS, including PFAS
18 biomonitoring in surveillance studies and
19 community-focused studies, and CalEPA's efforts to address
20 these compounds also.

21 We will have guest speakers from Örebro
22 University in Sweden, Boston University School of Public
23 Health, and Duke University. And they will present --
24 present, excuse me, on PFAS laboratory methods and also
25 sources of human exposure.

1 After the presentations, we'll hold an open
2 discussion with guest speakers and the audience, and that
3 will be to address questions on how Biomonitoring
4 California can support efforts to reduce exposure to PFAS,
5 including possible next step for the Program.

6 After each presentation, as we usually do, there
7 will be time for questions from Panel members and from the
8 audience. So let me take just a moment to explain how we
9 do these comment periods and discussions on the remote --
10 in the remote format. So during the question periods that
11 come after each talk, speakers please remain unmuted with
12 your webcam showing, so that you can respond to questions.
13 If SGP members want to speak or ask a question, just raise
14 your hand. You'll have your webcam on and I can see you.
15 And then you'll unmute yourself after I call on you and
16 comment or ask your question.

17 If webinar attendees have questions or comments,
18 please submit them via either the Q&A feature of the Zoom
19 webinar or by email to biomonitoring@oehha.ca.gov. And
20 please just keep your comments focused on the items under
21 discussion and brief. We'll read aloud any relevant
22 comments paraphrasing them if they're long.

23 During both the morning and afternoon public
24 comment periods and in the afternoon discussion session,
25 webinar attendees can also speak. If you don't want to

1 submit a written comment, you can speak. Then please use
2 the raise hand feature in Zoom and I will call on you.

3 So with that, I want to introduce our first
4 speaker. Nerissa Wu is the overall lead for Biomonitoring
5 California and Chief of the Exposure Assessment Section in
6 the Environmental Health Investigations Branch, or EHIB,
7 at California Department of Public Health. She'll provide
8 an update on current Program activities.

9 (Thereupon a slide presentation.)

10 DR. WU: All right. Can you hear me?

11 CHAIRPERSON SCHWARZMAN: (Thumbs up.)

12 DR. WU: Allow me to get my screen. And do you
13 now see my slides?

14 Everything appear okay?

15 DR. MARDER: We do.

16 DR. WU: Okay. Great. Well, welcome everybody.
17 Thanks for joining us, especially those of you who are
18 calling in from different time zones. I just want to
19 start by adding my thanks to Veena as well for your
20 participation on the Scientific Guidance Panel and just
21 your ongoing support for the Program. We'll miss having
22 on you the Panel.

23 So I only have you for 10 minutes today. So I'm
24 going to be brief. I'm covering some administrative
25 updates, where we are as a Program. And then I will turn

1 to updates on two of our projects. So last time we met,
2 we had just gotten news of our newly signed budget, which
3 includes an additional \$2 million annually from general
4 fund. This is super welcome news for the Program. So we
5 are going through all the administrative tasks. To make
6 sure the budget -- the funding gets to the right place and
7 is used to support the Program in the key areas we've been
8 highlighting over the years.

9 --o0o--

10 DR. WU: We've talked about the need for
11 sustainable funding to help maintain lab staff and to keep
12 our expertise both the labs have developed. And on the
13 epi side, we need to be able to analyze and release data
14 more quickly. We've talked about the need to support
15 field work and to be able to reestablish our surveillance
16 efforts.

17 --o0o--

18 DR. WU: So towards those goals we are recruiting
19 for a number of different positions, epidemiologists so
20 the Research Scientists I, III, and IV levels. We're
21 looking for Health Program Specialists and we have
22 laboratory and chemists for the lab posted. All of these
23 positions are available on CalCareers. They're also --
24 for the EHIB positions, they're also listed on our EHIB
25 website. And I think there will be a notice going out to

1 our listserv as well through the Biomonitoring membership.
2 So please pass this information along to anyone who might
3 be interested in joining our team or if you yourself are
4 interested in coming to be part of Biomonitoring
5 California.

6 We do have a workshop planned for November 22nd
7 to talk to people about what it's like to work in the
8 public sector and particularly for Biomonitoring
9 California and how to go about applying for jobs in the
10 California State sector -- State system. So I'm happy to
11 share links and registration information on that after my
12 talk. Bringing in staff into the State system is a long
13 slow process, but we hope to have some progress to report
14 back to you at our next meeting.

15 --o0o--

16 DR. WU: We do have two new staff people to
17 introduce to you. We have Faye Andrews our new
18 epidemiologist, also a new doctor at EHIB, and Cheryl
19 Holzmeyer, who has recently joined OEHHA as a Health
20 Program Specialist. And she's helping to run this
21 meeting. They're both making contributions already and
22 welcome to the two of you. And I also wanted to mention
23 that this is Jed Waldman's last meeting as a part of
24 Biomonitoring California. Although he's welcome to join
25 as a member of the general public next year. Jed is

1 retiring at the end of this year and our Program won't be
2 quite the same without him. So thank you to Jed for
3 everything you've done for the Program as well.

4 --o0o--

5 DR. WU: So on to project updates. The Stockton
6 Air Pollution Exposure Project, or SAPEP, has made a lot
7 of progress in the last month finalizing study tools on
8 things like consent forms, questionnaires, recruitment
9 materials. They've gotten their full approval from the
10 IRB and have confirmed a school site, the All Saints
11 Academy of Stockton, which is a small school of about 90
12 kindergarten to 8th graders with a very supportive
13 principal. So they've done a site visit and they are
14 actually starting recruitment this week.

15 Field work is scheduled to begin in early
16 December. You remember this project, it involves two
17 sample collection points, one week apart. And we'll have
18 a much more thorough update at the March SGP meeting.

19 --o0o--

20 DR. WU: We also have some progress to report for
21 the California Regional Exposure, or CARE, Study.

22 --o0o--

23 DR. WU: We had just returned results for our
24 participants the last time we met. And we should have our
25 summary results posted to the web in the next few weeks.

1 Noting that while we recruited participants following the
2 same CARE protocol as our first two regions, the early
3 closure of CARE-3 means that we only had 90 participants.
4 And so there are limits to how we can interpret that data.
5 We're also working on the CARE report, which will include
6 detailed study methods and results.

7 --o0o--

8 DR. WU: And I want to say just a word about the
9 choice to do a report, because this is something a little
10 bit new for the Program. The report gives us an
11 opportunity to talk about the study in the context of our
12 larger Program and to also get really into the details of
13 the method and choices we made as part of the study
14 design. And I think that will help the reader understand
15 what the data represents and how it can be used and
16 interpreted.

17 The report will have both unweighted and weighted
18 data, which will provide better exposure estimates for the
19 region and that will be a better comparison both for us
20 but also for other researchers to use when we have
21 comparative data. We'll also have data by demographics
22 strata, which will be very useful.

23 So releasing a report like this does not mean we
24 won't be publishing in scientific journals as well. As we
25 delve more into statistical analyses, there will be other

1 opportunities for us to publish via that route. In any
2 case, we hope to be finishing up this report in the next
3 month or so and releasing it in early 2022.

4 Five minutes already. Oh, goodness.

5 As we work on the CARE report, it's also an
6 opportunity for us to learn from our previous experiences
7 and think about our next steps in conducting surveillance.
8 We're continuing to meet with other collaborators and
9 defining Program's priorities.

10 --o0o--

11 DR. WU: We're also taking input from different
12 stakeholders and recommendations from experts, like this
13 Panel, into consideration. So these are the
14 recommendations that you provided at our last meeting. It
15 was after that discussion. So thanks to Meg and Jenny for
16 summarizing these for inclusion in the Seventh Report to
17 the Legislature.

18 --o0o--

19 DR. WU: One of the prioritizations we also have
20 to keep in mind is which chemical panel should we be
21 focusing on? And this is not just an issue for the lab
22 with respect to what methods they should be prioritizing,
23 but our focus also has bearing on the design of a study,
24 where we might try the study, whom we want to include in
25 the study, and what questions to include in the

1 questionnaire.

2 So the topic for today's discussion is a chemical
3 class that has been a priority for this Program as well as
4 for State and nationwide concerns. And likely it does not
5 need any introduction for this audience. But in case you
6 are joining us for the first time or new to biomonitoring,
7 these are the per- and polyfluoroalkyl substances, the
8 PFASs. There are several different definitions in play.
9 The definition presented here is from Buck et al., and
10 it's the definition that this Program uses for the
11 purposes of designation.

12 Tom Webster will talk a little bit more about the
13 different definitions and the implications thereof in this
14 afternoon's session. But again, for our Program, the
15 definition is relevant because of what the designated list
16 means, in terms of what we are able to measure as a
17 Program.

18 --o0o--

19 DR. WU: PFASs are primarily used to make
20 products resistant to stains, water, and grease. And many
21 of the products we use in our everyday lives, things like
22 stain-resistant carpets or stain-resistant furniture,
23 takeout containers that we want to hold soupy or greasy
24 foods are often treated with PFASs or used in industry
25 added to metal plating and finishing processes to reduce

1 toxic air emissions. And, of course, they are part of the
2 AFFF fire suppressant foams used to fight fires.

3 Manufacturing of the long-chain PFASs have been out --
4 phased out of the U.S. Many of those -- but thousands of
5 PFASs are continued to be used and manufactured worldwide.

6 --o0o--

7 DR. WU: And why do we care? Why are we
8 concerned about PFASs? Well, different PFASs have been
9 found to be associated with a wide range of health
10 impacts, including thyroid disease, and some cancers,
11 increased cholesterol, infertility, and adverse birth
12 outcomes, altered child development, impacts on liver
13 enzyme activity, and a weakened immune system.

14 As I said, many have been phased out, but as some
15 of the legacy PFASs are persistent and bioaccumulative,
16 we're still finding them in our bodies. And the shorter
17 chain PFASs are still widely used. As we have seen for
18 many chemicals, there is this opportunity for regrettable
19 substitutions. As we move away from one set of PFASs, we
20 introduce the use of another. This trend and use over
21 time, the decreases in some PFASs and increases in others
22 is the kind a scenario for which biomonitoring can be very
23 useful to monitor how our body burdens follow
24 manufacturing trends.

25 --o0o--

1 DR. WU: So during the day, you'll hear about
2 different lab methods. Currently, our lab has several
3 methods available for looking at PFASs, including the
4 method to measure the 12 legacy PFASs. There's the
5 expanded 40-PFAS replacement for -- 40-PFAS panel, which
6 includes some of the replacement PFASs. And then there is
7 the non-targeted analysis for PFASs and other chemicals of
8 concern. These are currently available in serum. And the
9 lab is working to further automate and make these more
10 sensitive, faster, and greener, and also validate the
11 methods in plasma.

12 So with that overview, I want to conclude my
13 portion of the talk and turn things over to our PFAS
14 experts, but I will be open to questions after our next
15 couple of speakers.

16 CHAIRPERSON SCHWARZMAN: Thank you so much,
17 Nerissa. Yeah, and just to repeat that that we'll have a
18 question session once we've heard from some of the other
19 staff scientists about PFAS. So I want to introduce Karl
20 Palmer our next speaker.

21 Karl is Deputy Director for the Safer Consumer
22 Products Program in the Department of Toxic Substances
23 Control and he will provide an overview of CalEPA
24 activities on PFAS.

25 (Thereupon a slide presentation.)

1 MR. PALMER: Thank you, Meg. I'm just going to
2 share my screen here. Can you see my screen?

3 CHAIRPERSON SCHWARZMAN: Yes, that's perfect.

4 MR. PALMER: Okay. Great. Well, thank you, Meg.
5 And I also want to thank Veena for your Service on the
6 SGP. We'll look forward to engaging with you in your
7 other endeavors and capacities. So thank you very much.
8 And thanks to Nerissa for the good summary.

9 I'm going to move ahead here, I think.

10 --o0o--

11 MR. PALMER: There we go. My disclosure is I
12 have no financial conflicts of interest as I'm the Deputy
13 Director of the Safer Consumer Products at DTSC of CalEPA.

14 --o0o--

15 MR. PALMER: So as you're probably familiar at
16 the highest level CalEPA's mission is to really restore,
17 protect, and enhance the environment, and ensure public
18 health, environmental quality, and economic vitality. We
19 do this by developing and implementing and enforcing
20 environmental laws that regulate air, water, soil quality,
21 pesticide use, hazardous and solid waste, recycling and
22 reduction, and the development of safer consumer products.

23 I always like to say that chemicals don't adhere
24 to the laws of man but to the laws of nature. So while we
25 tend to regulate chemicals within these frameworks and

1 silos of bureaucracies, they don't pay much attention.
2 They do what they do, which creates challenges for all of
3 us. Our collaborative effort at CalEPA is manifested
4 in -- one way in establishing this PFAS working group, and
5 we've invited our partners at the Department of Public
6 Health to join us as well. And the role of our workgroup
7 is really to share information about what we're all doing,
8 so that we can learn, coordinate and collaborate, and move
9 forward in our mission.

10 --o0o--

11 MR. PALMER: So I'm going to start with what's
12 going on at the Water Board. They're doing a lot of
13 things, so bear with me. The State Water Board statewide
14 PFAS investigations have targeted airports in both fuel
15 terminals and refineries, because they use aqueous film
16 foaming flame retardants. They use -- and they look at
17 chrome plating facilities -- the Water Board looks at
18 chrome plating facilities, because of their use of mist
19 suppressants which contain PFASs. And they are looking at
20 municipal solid waste landfills and waste water treatment
21 plants, because they receive waste that contains PFAS.

22 In coordination with the issuance of orders to
23 the public water systems, they ask to sample their wells
24 adjacent to airports and landfills, and those wells with
25 PFAS detections from EPA's third unregulated contaminant

1 monitoring role sampling events and in the vicinity of
2 those wells.

3 Since the issuance of those -- excuse me --
4 initial screening sampling events, additional orders have
5 been issued to public water systems to expand outward from
6 the previous detections and in the vicinity of DOD sites.
7 Future sampling will be performed as data comes in and
8 they determine the extent of source areas and additional
9 sampling needs.

10 --o0o--

11 MR. PALMER: To give you some look at what
12 they've done, the primary investigatory objectives of the
13 statewide orders are to gather information on the
14 occurrence of PFAS in California's drinking water sources
15 and watersheds. The data will be evaluated to identify
16 impacted drinking water wells and identify areas where
17 additional work is needed to ensure that communities
18 reliant on those drinking water wells are provided safe
19 drinking water and where additional public water supply
20 well sampling would be appropriate. The data will also be
21 used to inform additional areas where watershed specific
22 source identification efforts are needed and to inform
23 future investigation requirements.

24 Finally, the data collected will also continue to
25 inform the consideration of public health goals developed

1 by OEHHA and eventually lead to the maximum contaminant
2 levels adopted by the State Water Board.

3 --o0o--

4 MR. PALMER: To give you some idea of what the
5 data has shown since 2019, the results of the sampling at
6 the public water systems are indicating that only 13
7 percent of those wells have an exceedance of the response
8 level. The response levels for PFOA is 10 nanograms per
9 liter and for PFOS is 40 gram -- nanograms per liter. And
10 if there's an exceedance of the response level, the public
11 water system must either take the well offline, treat the
12 well usually through blending, or notify the public. The
13 Division of Drinking Water will continue to require public
14 water systems to sample for PFAS in wells outward of any
15 of these exceedances. Additionally, sampling for PFAS
16 will continue in these wells until further notice by the
17 Drinking Water Division.

18 --o0o--

19 MR. PALMER: This next slide is a little
20 complicated, but essentially what it does is it reports on
21 the results from the public water systems - those are the
22 bars in orange - and the results from airports and
23 landfill investigations, which are the bars in gray. And
24 you can see the percentage of PFASs detected in those
25 efforts. There were two different methods used for these

1 events. And so you can see that the data from the
2 investigations that the airports and landfills show many
3 more compounds specifically the shorter chain PFAAs that
4 are being detected at high frequency.

5 Because of these results, the Division of
6 Drinking Water is considering shifting from the EPA method
7 used, what you see in the orange bars, to the DOD
8 developed method, which shows a greater broader array of
9 analytes captured and particularly the shorter chain
10 PFAAs.

11 --o0o--

12 MR. PALMER: Now, the importance of the
13 information is because the Water Board is tasked with
14 developing MCLs for drinking water standards. And so this
15 is a multi-stage part process, the Office of Environmental
16 Health Hazard Assessment is a key part of this process.
17 And you can see here that notification levels have been
18 established for PFOA and PFOS and there's recommendations
19 for the public health goals for both those compounds.
20 There's a -- for PFBS, there's a notification level
21 proposed. And the hope is that for PFOA and PFOS that
22 we'll have MCLs in place in 2025.

23 So also it's important to note that the Water
24 Board has requested additional work by OEHHA to look at
25 five additional compounds.

1 --o0o--

2 MR. PALMER: This is a pretty busy slide, but it
3 has a lot of links to really good information. The Water
4 Board is acting for CalEPA to kind of collect a lot of the
5 information that the different boards, and offices, and
6 agencies are collecting. They have some really good
7 information there. I encourage everyone to look at that.

8 --o0o--

9 MR. PALMER: Moving on, I'll talk a little bit
10 about what OEHHA is doing. And there are many people in
11 this meeting who know better than I. But OEHHA's mission
12 really is to protect and enhance the health of
13 Californians and the state's environment through
14 scientific evaluations that inform, support, and guide
15 regulatory and other actions. They're the lead State
16 agency for conducting health risk -- for evaluating health
17 risks posed by environmental contaminants. They also
18 implement Prop 65.

19 And so you can see here that OEHHA has completed
20 notification levels for PFOA and PFOS. I'm not going to
21 go into the details. You can read those there and as well
22 as for PFBS. And then there are proposed health goals for
23 PFOA and PFOS that are also established. These are an
24 important part of establishing the drinking water
25 standards and they are working closely with the Water

1 Board in that process.

2 Note that the notification levels are
3 health-based advisory levels. They're not regulatory.
4 And that OEHHA conducts risk assessment of a chemical and
5 provides recommendations to the Water Board who then sets
6 the notification levels.

7 --o0o--

8 MR. PALMER: Additionally, as I mentioned
9 earlier, the Water Board has requested that notification
10 levels be set -- or be provided from OEHHA in the journey
11 towards health-based drinking water standards for these
12 additional six PFASs.

13 Now note -- well, I'll move on.

14 --o0o--

15 MR. PALMER: Also I just want to mention that in
16 the responsibilities to implement Prop 65, OEHHA listed
17 PFOA and PFOS on Prop 65 for reproductive -- as
18 reproductive toxicants, and then in March of this year,
19 they issued a Notice of Intent to list PFOA as a
20 carcinogen.

21 Additionally, there's two important meetings
22 coming up in December, one of the Carcinogen
23 Identification Committee that will be considering listing
24 PFOS as a carcinogen. And December 14th, there will be a
25 meeting of the Developmental and Reproductive Toxicant

1 Identification Committee to consider PFNa and its salts
2 and PFDA and its salts as reproductive toxicants.

3 --o0o--

4 MR. PALMER: Moving on to our colleagues at the
5 Air Resources Board. They're in the process of updating
6 their Airborne Toxic Control Measures. And what their
7 focus is right now is looking at PFASs that are used as
8 chemical fume suppressants in plating baths. And this is
9 at -- particularly at chrome plating facilities. They've
10 also funded research by UC Berkeley looking at
11 environmental assessment methods to collect and analyze
12 PFAS in air, dust, and soil. And this is a general
13 challenge across the agency is -- and I know you're going
14 to be talking more about this later is how do you assess
15 where PFAS is in the environment and potential exposures
16 that ultimately end up in people and other media.

17 --o0o--

18 MR. PALMER: Moving on to CalRecycle.
19 CalRecycle's primary mission is to promote the reduction
20 of solid waste and to promote recycling as well as
21 composting. And they're working with UC Davis to look at
22 composting and what happens to PFAS in that environment.
23 And so there's a lot of interesting work going on there.

24 They also this year adopted regulations pursuant
25 to the Sustainable Packaging for the -- Act that was

1 passed in 2018. That Act required that CalRecycle put
2 forth regulations that require food service facilities
3 used on State properties to use reusable, recyclable, and
4 compostable food packaging. And they've done that. And
5 interestingly they've put in there some provisions that
6 address PFAS and limit PFAS in those products. And you'll
7 note that again, it's important for us to be able to
8 how -- to assess where PFAS is, not only in the
9 environment, but also in the products that we use, and
10 what methods we use to do that. So they've been working
11 on that.

12 --o0o--

13 MR. PALMER: Our colleagues at the Department of
14 Pesticide Regulation found out earlier this year that
15 while they did an initial search to look at all of the
16 registered pesticides to see if PFASs were used and they
17 didn't find that any PFASs were used in the pesticides
18 themselves however, they did come in to information that
19 some containers contained PFAS. And that those containers
20 had certain PFASs that had leached into the product. And
21 so they've been working with those manufacturers and with
22 U.S. EPA to change out and use non-fluorinated containers
23 for pesticides.

24 --o0o--

25 MR. PALMER: The area for which I'm most familiar

1 with and responsible for is at DTSC. And I wanted to note
2 that at DTSC, we have three core programs. We have our
3 Cleanup Program, our Hazardous Waste Program, and the
4 Safer Consumer Products Program.

5 In the Cleanup's program, much like the Water
6 Board's challenges, we're looking at dealing with PFAS in
7 groundwater and remediating PFAS in groundwater to
8 particularly protect drinking water wells.

9 In our Hazardous Waste Program, we're considering
10 looking at whether we should regulate PFAS-containing
11 wastes as hazardous wastes in California. Note that
12 others have petitioned U.S. EPA to make hazardous waste
13 out of -- excuse me, to include PFAS waste as hazardous
14 waste under the Resource Conservation and Recovery Act.

15 And then my program, which I'm going to talk
16 about a little bit more, but I also wanted to also note
17 that our Environmental Chemistry Lab, which is a partner
18 in the Biomonitoring California Program, helps us in our
19 program and our other programs to both evaluate different
20 media that contain PFAS, as well as consumer products that
21 contain PFAS.

22 --o0o--

23 MR. PALMER: So I'm going to talk a little bit
24 about my program, because I know it best and because I
25 think it's also relevant. First and foremost, I wanted to

1 thank the SGP for listing PFAS as a class on your priority
2 chemicals list in 2015. That opened the door for us to
3 look at these chemicals in consumer products, because our
4 regulations require that we look at 23 other authoritative
5 body lists, one of which is the SGP priority list, for
6 chemicals that are on our menu that we can consider when
7 we regulate these chemicals and products.

8 It's important to note that we are viewing this
9 as a class approach, because one of our missions is to
10 ensure that we don't move from one hazardous or
11 problematic chemical to another one in the chemical
12 whack-a-mole process that we've experienced -- all
13 experienced. And so by treating PFAS as a class, we can,
14 through our regulations, ensure that when we ask people to
15 look at a safer alternative to that PFAS, they don't just
16 move from one PFAS to another PFAS, but they have to
17 consider the entire class and look for alternatives
18 outside that class, which is a very efficient way to
19 regulate when you've got thousands of chemicals that you
20 might be considering in that class.

21 So we published a paper on this in Environmental
22 Health Perspectives, documenting our approach. And we
23 wouldn't have been able to do that without the SGP.

24 --o0o--

25 MR. PALMER: What that looked like in practice

1 then, is earlier this year, we adopted, as a priority
2 product in our rulemaking framework, carpets and rugs that
3 contain PFAS. And what that meant is that anyone who
4 sells a carpet or rug into California that contains PFAS
5 is now subject to Safer Consumer Products Regulations,
6 they're required to notice that -- notice us if they're
7 selling those products and then go through a robust
8 alternatives assessment process to hopefully find a safer
9 alternative.

10 We're going to be -- we're in the process right
11 now of adopting regulations that will capture treatment
12 products, things like Scotchgard and other treatments that
13 are sprayed onto textiles and leathers. And then we will
14 be looking potentially at children's products and
15 cosmetics that contain PFAS as well. I note that PFAS
16 food packaging is something we spent a lot of time looking
17 at PFAS on. We had several workshops. We put together a
18 technical document.

19 --o0o--

20 MR. PALMER: And I'm going to talk briefly about
21 what the outcome of that was in that the California
22 Legislature looked at that work we did on food packaging
23 and passed a law, AB 1200, which banned plant fiber-based
24 food packing with PFAS starting in 2023 and some other
25 aspects of it as well. The important thing there is it

1 was looking at PFAS as a class using the good work that we
2 did to support that action to accelerate regulation of
3 those products.

4 Other bills that were passed, which were also
5 related to PFAS Friedman Bill, AB 652, was for juvenile --
6 a variety of juvenile products banning PFAS in their use,
7 and then note that AFFF foams containing PFAS we're also
8 restricting from sale via SB 1044 effective this coming
9 January.

10 Now, I'll also note that many other states across
11 the country from Maine, to Washington, to New Mexico are
12 passing states related to PFAS in a variety of consumer
13 products, because of concerns of potential exposure and
14 harm.

15 --o0o--

16 MR. PALMER: Lastly, I'll just wrap-up by saying
17 that in -- last month, the U.S. EPA put out their
18 strategic roadmap for PFAS. It's an ambitious look at how
19 they can use a variety of authorities under U.S. EPA's
20 umbrella to look at PFAS throughout its lifecycle in all
21 media over time. And this is -- there's a lot of depth to
22 this. I encourage people to look at it. It's very
23 ambitious, but we certainly need to move forward with this
24 class on so many different fronts.

25 And hopefully what you see in my brief overview

1 of what's going on at CalEPA, that PFAS touches each one
2 of our departments. It doesn't pay attention to our
3 political or regulatory bureaucratic barriers and there's
4 a lot of work to do.

5 So with that, that summarizes just a brief look
6 at what we're doing at CalEPA.

7 --o0o--

8 MR. PALMER: This is my contact information. I'm
9 happy to answer any questions.

10 Thank you.

11 CHAIRPERSON SCHWARZMAN: Thank you so much, Karl.

12 Again, we'll have time for questions after our
13 third panelist -- or presenter right now, who is
14 Katherine -- Kathleen Attfield. She's Chief of the
15 Biomonitoring Investigations and Outreach Unit, which is
16 part of the exposure assessment section in EHIB at the
17 California Department of Public Health, DPH. Kathleen
18 will discuss Biomonitoring California's findings on PFAS
19 from the CARE study and some earlier work.

20 (Thereupon a slide presentation.)

21 DR. ATTFIELD: Good morning. Can you hear me
22 properly?

23 CHAIRPERSON SCHWARZMAN: Yep, that's good.

24 DR. ATTFIELD: Wonderful. And you can see my
25 slides?

1 Okay. So good morning. Again, my name is
2 Kathleen Attfield. I'm with the California Department of
3 Public Health in our Biomonitoring California Program.
4 And I want to provide some updates on our activities as
5 related to PFAS.

6 CHAIRPERSON SCHWARZMAN: Kathleen, I'm not seeing
7 your slides, but that might be a problem with mine not
8 with others.

9 PANEL MEMBER CRANOR: No, they're not available.

10 DR. ATTFIELD: Okay. Excuse me. Sorry.

11 CHAIRPERSON SCHWARZMAN: Perfect.

12 DR. ATTFIELD: Okay.

13 --o0o--

14 DR. ATTFIELD: So before I launch into my talk,
15 I'd like to quickly revisit that our studies of PFAS are
16 situated within the Biomonitoring California's mandate to
17 determine biological levels of environmental chemicals in
18 Californians, to establish trends in these levels of
19 chemicals in Californian's bodies over time, to help to
20 assess the effectiveness of public health efforts and
21 regulatory programs to decrease exposures to specific
22 chemicals.

23 --o0o--

24 DR. ATTFIELD: Biomonitoring California's general
25 approach to understanding pollutant biomarker trends has

1 been to conduct surveillance activities and look for
2 indicators of concern where we may then characterize
3 specific populations using community-based approaches.
4 And these might be in specific geographic areas and
5 specific racial or ethnic communities, or occupational
6 groups, or in sensitive subpopulations, such as with
7 pregnant women.

8 --o0o--

9 DR. ATTFIELD: In today's talk, I will visit some
10 of the different populations we have assessed with PFAS
11 within and dive into demographic trends we have observed,
12 including ethnic and racial disparities. And since the
13 program has conducted a number of studies to date with a
14 lot of valuable information waiting to be explored, I will
15 end with a discussion of opportunities for further data
16 analyses and asking for the Panel's suggestions for
17 prioritizing these in terms of their best impacts on
18 public health and regulatory efforts, and learning more on
19 exposure sources.

20 --o0o--

21 DR. ATTFIELD: So here's a list of the
22 Biomonitoring California studies that have measured PFASs
23 from 2010 to 2020. In most of our studies, we've been
24 measuring the 12 common legacy PFAS, but we have a couple
25 studies where we have measured up to 30 PFAS.

1 --o0o--

2 DR. ATTFIELD: So the ones I'm going to spend the
3 most time today in speaking with -- speaking about are the
4 CARE regional exposure studies, the California Regional
5 Exposure studies primarily on our first two regions in LA
6 and CARE-2, eastern and southeastern counties. I will
7 also talk about opportunities that we have with the ACE
8 studies of Asian Americans in the San Francisco-San Jose
9 area, the MAMAS studies of pregnant women, and our back --
10 harkening back to our very first population-based study
11 with Kaiser members that is called the BEST study.

12 --o0o--

13 DR. ATTFIELD: So from these various studies in
14 the past, we are look -- we have been learning, as -- from
15 these in order to look at our data from our CARE studies.
16 We have seen a number of trends from these prior studies,
17 including very high detection frequencies, PFNA PFOA,
18 PFOS, PFHxS when it's over 95 percent detections in those
19 three studies, and also very frequent detections of
20 others.

21 We've seen levels that increase with age,
22 differences by sex and gender in which males often have
23 higher levels, and also differences by race and ethnicity
24 where Asian populations tend to have higher levels of many
25 of the PFAS.

1 --o0o--

2 DR. ATTFIELD: So for CARE, the California
3 Regional Exposure studies, we have presented periodic
4 updates to this Panel. And for these, we have recruited
5 across each region to represent the demographics of that
6 particular area using a quota sampling approach. In
7 CARE-LA, we visited the entire county of Los Angeles in
8 the spring of 2018 and garnered 430 participants. At our
9 second region, CARE-2, from Mono all the way down to
10 Imperial counties, we recruited 359 participants over the
11 spring of 2019.

12 --o0o--

13 DR. ATTFIELD: Our participants who completed the
14 studies ended up skewing slightly female with a median age
15 of 51 and race percentages generally reflected the
16 population of the region.

17 --o0o--

18 DR. ATTFIELD: However, to improve our ability to
19 use our central estimates as population estimates and to
20 better enable comparisons across regions, we are currently
21 undergoing a calculation of weights that Nerissa alluded
22 to and we'll be using those in the future. For the rest
23 of this presentation today, however, I'll be referring to
24 interim analyses performed with unweighted data.

25 --o0o--

1 DR. ATTFIELD: So among these 12 PFAS that were
2 measured in these two populations, we found PFAS in almost
3 all or all participants in CARE-LA and CARE-2 of just one
4 person not having a detect -- any detections in CARE-2,
5 and on average, six or seven of them per participant.

6 So the red box here is drawn around the PFAS for
7 which we have detection frequencies over 65 percent. And
8 that's the threshold we use for diving in deeper to look
9 at particular trends in those analytes.

10 --o0o--

11 DR. ATTFIELD: So our first step would be to say
12 how did these regions differ or are similar to national
13 levels? So to compare here with NHANES from the most
14 recent cycle for which there is available data, 2017 to
15 2018, I first have to make a slight caveat about the
16 methods used here, in that NHANES has higher levels of
17 detection than our DTSC lab. So to make the comparisons,
18 we do have to re-censor the data to the NHANES LOD. And
19 that meant that three PFAS there listed on the bottom
20 PFDeA, PFUA, methyl PFOSA, then we wouldn't be comparing
21 as they drop below that 65 percent detection threshold.

22 --o0o--

23 DR. ATTFIELD: But for the four remaining, they
24 do seem to be lower than national levels. I'll give you a
25 moment to eyeball that.

1 We do have to keep in mind though that there's
2 still one to two years difference in these comparisons.
3 So there still could be a small remaining role for
4 temporal effects. As we know, many of these are declining
5 over time.

6 --o0o--

7 DR. ATTFIELD: These two CARE studies are focused
8 on the general population, so it's not too surprising that
9 our 95 percentiles are way below those of highly impacted
10 communities, such as these in the examples from West
11 Virginia, Alabama, and New Hampshire.

12 --o0o--

13 DR. ATTFIELD: Analysis of demographic trends
14 displayed the known impact of gender and sex with the
15 largest impacts seen in PFHxS with 87 percent higher in
16 males for CARE-LA, 80 percent in CARE-2. And we actually
17 didn't see a statistical difference for methyl PFOSA and
18 PFDeA, but we do see it in those others.

19 --o0o--

20 DR. ATTFIELD: You see the impact of increasing
21 age for all six of these PFAS. And this is by decade of
22 age of participant. And the most substantial effect is
23 seen with PFOS with 20 to 22 percent increase by decade of
24 age of the participant.

25 I'm sorry, my slides are not advancing.

1 --o0o--

2 DR. ATTFIELD: There it goes.

3 Patterns in race and ethnicity followed the
4 general trend of Asian participants having the highest
5 levels, followed by White participants, Hispanic, and
6 Black participants. In these tables, I've ordered the
7 PFAS from left to right to indicate the largest effects on
8 the right-hand side and in darker shades of blue.

9 Here, we see the PFDeA has the greatest
10 differences between Asians and all other groups, ranging
11 from 84 percent there at the bottom compared to White
12 participants in CARE-LA up to 144 percent higher than
13 black participants in CARE-LA.

14 PFOS was the next largest in differences up to
15 132 percent greater than blacks. And I should note that
16 because of the fewer number of Black and Asian
17 participants in CARE-2, some comparisons did not reach
18 statistical significance here.

19 --o0o--

20 DR. ATTFIELD: So extending to other com -- other
21 group comparisons, these are not as great, but still we
22 see levels higher in White participants than Black and
23 Hispanics primarily in PFOA with the largest difference
24 compared to Black participants for CARE-LA with PFHxS.

25 --o0o--

1 DR. ATTFIELD: An interesting little side note is
2 that the PFOS precursor, methyl PFOSA, uniquely had a
3 different racial pattern than the others, though often
4 this did not reach statistical significance. For the one
5 in which it did, levels compared to Hispanic participants
6 in CARE-LA were significantly different at 38 percent
7 greater concentrations.

8 --o0o--

9 DR. ATTFIELD: From our exposure questionnaire,
10 we had begun to look into the contribution of fish and
11 shellfish consumption to these demographic patterns we're
12 observing. In these regions, there are not known large
13 local PFAS contamination sites, those similar to many
14 parts of the rest of the country. As Karl just talked us
15 through, PFAS have been measured in some drinking water
16 systems and groundwater.

17 Fish and shellfish contributions have been linked
18 to studies of recent PFAS biomarkers, including in our own
19 BEST study in California within NHANES data from 2003 to
20 2014, and in San Francisco pregnant women in 2014 to 2016
21 data. These are usually most often seen with PFOS, PFNA,
22 PFDeA, PFUdA, so the longer chain PFAS there, the decanoic
23 and the undecanoic versions, the 10 and 11 carbon chains.

24 These studies also had looked at other dietary
25 contributors. But for what I'm going to talk you through

1 today, we're going to mostly focus on fish and shellfish.

2 --o0o--

3 DR. ATTFIELD: So I'll start with CARE-2, where
4 we have been able to look across many different exposure
5 variables. PFDeA was the only PFAS positively associated
6 with fish and shellfish after multi-variable analyses.
7 Just for your information, we had asked about fish and
8 shellfish in two ways, those that you buy in the store and
9 those that may be caught by someone known to the
10 participant. So this is primarily for our metals analyses
11 for looking at local fish versus fish that might be more
12 wide -- sourced from a wider area of the world, but also
13 could be useful for PFAS analyses.

14 --o0o--

15 DR. ATTFIELD: I have combined them here, so when
16 bought and caught fish are looked at collectively, eating
17 fish one to three times per week increased concentrations
18 by 22.4 percent. And if you look at it in the next
19 exposure category, up over three times per week of each,
20 we reached 60.6 percent higher levels.

21 Shellfish was knocked out of the final model and
22 attempts to make a combination variable with the two did
23 not increase our explanatory power.

24 --o0o--

25 DR. ATTFIELD: So fish consumption seems to have

1 impacted the estimates for differences by race, looking
2 here at the Asian participant breakdown. So the adjusted
3 change moved from 73 percent to 62 percent. And this may
4 be showing a potential current or historic exposure source
5 among this region's population.

6 --o0o--

7 DR. ATTFIELD: Now, moving on to CARE-LA, for
8 this, we've only managed so far to look at single exposure
9 sources in tandem with demographics. And here, we still
10 see an association of PFDeA with fish consumption, so up
11 to 43.5 percent higher in the group eating over three
12 times per week of each of those bought and caught, but
13 less of a modification of the estimates tied to race and
14 ethnicity.

15 --o0o--

16 DR. ATTFIELD: However, in PFUdA, the undecanoic
17 PFAS, we see a large impact with fish consumption, 181
18 percent increase over those eating less than once per week
19 and those that eat over three times per week. And we see
20 a fair decrease in the estimates of the contribution for
21 Asian identification.

22 --o0o--

23 DR. ATTFIELD: The benefits of having a study
24 that looks at multiple panels is that some of the panels
25 do end up being correlated based on exposure source. So

1 we had the opportunity here to look at the blood mercury
2 levels, which are also an indicator of fish and shellfish
3 consumption. And for CARE-LA, six of these -- all six of
4 these had a correlation with blood mercury, and three for
5 CARE-2. And our strongest correlations are with the two
6 that I was just showing you previously, so with PFUDa and
7 PFDeA.

8 --o0o--

9 DR. ATTFIELD: So marching on with the current
10 work that is happening with CARE data. We do have those
11 90 people from San Diego and Orange County, for which we
12 are readying data for CARE-3 to be placed on the web
13 repository. As mentioned, we are working on weighting our
14 participant data for better population estimates. And as
15 Nerissa detailed, we have a report in progress on CARE-LA
16 and CARE-2 data. We also have a new effort on
17 population-based pharmacokinetic modeling with --

18 --o0o--

19 DR. ATTFIELD: -- Matt MacLeod out of the
20 University of Stockholm, where his team will be simulating
21 lifetime intakes -- excuse me -- body burdens, and
22 elimination kinetics at the population level.

23 --o0o--

24 DR. ATTFIELD: Looking forward to other
25 opportunities with CARE data. We can follow the work

1 further on fish consumption and PFAS relationships to be
2 able to understand where there may be links to
3 intervention efforts. We can extend data analyses to
4 address other exposure sources where we have suitable
5 information in our survey data to link with the potential
6 for evaluating ongoing policy efforts.

7 We have address information, so we may be able to
8 look into links to drinking water. And as Tom Webster
9 will talk -- discuss in his talk briefly, we had the
10 opportunity to investigate profiles of PFAS, which some
11 researchers are beginning to use to be able to tease out
12 different relative sources of PFAS.

13 --o0o--

14 DR. ATTFIELD: Before moving on to our other
15 studies, I did want to contextualize our work within other
16 biomonitoring investigations of PFAS in California. So
17 other populations under study are middle aged women in the
18 California Teachers Study, female firefighters and office
19 workers, pregnant women and children, and, as Kate Hoffman
20 will later describe, Orange County residents are being
21 recruited for the multi-site ATSDR PFAS studies. And
22 lastly, firefighters at military sites across the U.S.
23 including California will have started having PFAS
24 biomonitoring as part of their physical exams, which began
25 this past fiscal year.

1 --o0o--

2 DR. ATTFIELD: From these cohorts, recent
3 publications described descriptive distribution or
4 detection data, such as developing non-targeted suspect
5 screening workflow on blood samples in concentrations in
6 those female firefighters and office workers. They also
7 addressed dietary predictors of PFAS and links to the
8 health endpoints of birth outcomes, offspring, and
9 telomere length.

10 Now to revisit some of our prior studies, for
11 which we have on -- some ongoing work, but also many
12 opportunities. And we hope you will help us with thinking
13 about prioritization and collaborations that could expand
14 the reach of our work. We are currently also working on
15 weighting this BEST -- this data, because it can give us
16 an ability to better describe population estimates.
17 Opportunities here exist with prior analyses on
18 demographics and diet that have not been finalized or
19 published, and potential, of course, to work with other
20 data sources, such as looking at links to drinking water.

21 --o0o--

22 DR. ATTFIELD: In the ACE projects, which were
23 with Asian-American populations in the San Francisco and
24 San Jose areas, we have seen interesting demographic
25 trends within these, but we have the wonderful opportunity

1 that we had very detailed dietary questionnaire for these
2 studies that are not as much in the same depth in our
3 other studies, and it seemed so far some interesting
4 associations with organ meat consumption.

5 We also have the potential to learn more about
6 the impacts of California -- of immigration in California
7 and whether the associations we have seen with birth
8 country and time in the U.S. are truly indicative of
9 transported body burdens.

10 As a targeted study, they can also help inform us
11 in strategizing around designs for future targeted
12 studies, because of the strengths and limitations
13 involved, one possibly being the limits due to homogeneity
14 of exposures within a targeted group. We also have the
15 interesting opportunity of the -- what may be revealed
16 with PFAS profiles here and how they may be illustrative
17 of different exposure patterns.

18 --o0o--

19 DR. ATTFIELD: And lastly for our work with the
20 MAMAS studies, and these were with obtained maternal
21 samples from different areas of California through the
22 Genetic Disease Screening Program, we have newly finished
23 laboratory data from 2015, 2016 - thank you, labs - that
24 we are readying to place into our web repository. We also
25 have a number of interesting opportunities in that we can

1 use weights more cleanly here and into the future in order
2 to really examine time trends as we go forward. And then
3 also, we have received the information from GDSP in an
4 anonymized fashion. So this would enable us to be able to
5 do non-targeted screening approaches, because of this not
6 incurring our report-back requirements.

7 --o0o--

8 DR. ATTFIELD: So with that, here's my list of
9 references and I'll be interested in our discussion.

10 --o0o--

11 DR. ATTFIELD: I want to thank our participants
12 across all of our studies for their time and their
13 willingness to give us their biological samples, and our
14 supporting organizations as well as Biomonitoring
15 California staff, and our State and federal funding.

16 So with that, I will wrap-up.

17 CHAIRPERSON SCHWARZMAN: Thank you so much.
18 Kathleen and also to Karl and Nerissa. So we have our
19 time now for questions for each of these three presenters.
20 Just as a reminder, we'll do questions from the Panel
21 first, and then we'll have public comment, and then we'll
22 have a Panel discussion on -- you know, specifically
23 addressing some of these questions that Kathleen has
24 invited input on.

25 So if the presenters could have their cameras

1 back on and we'll be able to -- maybe if I adjust my view,
2 I'll be able to see our panelists better. There you are.

3 And we have ten minutes now for questions from
4 the panelists on any of these three.

5 Tom.

6 PANEL MEMBER MCKONE: Sorry. I'll get my mute
7 off. And if you hear -- I apologize. There's some
8 construction going on nearby. It tends to come in.

9 First of all, I want to thank the presenters.
10 This was really, really interesting. Just a lot to
11 digest. I'm actually trying to digest it. But I do have
12 a question and I think -- I mean, it's kind of directed at
13 Karl, but at all three talks. But Karl Palmer, who did a
14 really nice job about how this has to be integrated across
15 so many different organizations.

16 And I was sort of looking at numbers and
17 pathways, and one of the things that comes up is, you
18 know, the level of communication about health levels
19 and -- for example, in looking at the effort at OEHHA to
20 develop notification levels and MCLs mainly for drinking
21 water. You know, and I was wondering, well, when we see
22 the later presentations -- or Kathleen's presentation
23 about where it seems to be coming from in the
24 biomonitoring level, it's coming from a lot of food
25 pathways. And so is there some effort to say, you know,

1 we have to -- like when we set a drinking water standard,
2 we're going to have to realize that that's only going to
3 control a small part of it. We have to be aware of the --
4 either the relationship of water to food, but also, you
5 know, food operates independently. Food comes from all
6 over the place. It's not just a California food source.

7 So I guess what I'm getting at is how do we stand
8 back and look at like the cumulative exposure and really
9 understand that better, and then how do we ultimately
10 think of the health effects in terms of biomonitored
11 levels, so we'll know how to give guidance -- not we. I
12 mean the State will know how to give guidance about what
13 levels -- what biomonitored levels should be -- require
14 notification or concern?

15 So it's kind of a long-winded question, but I
16 guess I'm just focusing more on understanding a little bit
17 better on how the exposure pathway analysis and
18 biomonitoring really worked together to help us really
19 understand the cumulative different pathways of exposure
20 and then what actions -- what action is needed and what
21 actions can be taken? So I'll leave it at that.

22 MR. PALMER: Well, I'll go first and others can
23 chime in. Thanks, Tom. Good question.

24 I think part of -- I look at this as there's
25 different buckets of issues here. One as I kind of

1 highlighted, each of the agencies has our own perspective
2 that it's -- that's provided to us by our authority, and
3 our mandates, and our resources. And so we do the best we
4 can to collect information from others who are looking at
5 things that intersect in the real world. But it's very
6 challenging, because we don't have the science for
7 cumulative impacts really well defined. It's not in the
8 regulatory language, let alone practice, like risk
9 assessment has been over the years.

10 And so I would say at the big buckets what we
11 need is we need to have good up-front information about
12 where we can find these chemicals. So that's a huge
13 benefit from biomonitoring, but we need it over time, so
14 that we can see that when we do take action, that we can
15 measure our success hopefully or at least gauge it. And
16 that -- so we need to be in it for the long run and we
17 also need to keep looking under different lamp posts, if
18 you will, for where the information is, because it comes
19 from many different sources. Food obviously is one
20 exposure pathway, but as we see in products, when we were
21 looking at carpet, you know, dust, air, dermal, all of
22 these things are factors and we don't have all the
23 information.

24 So I guess what the long-winded answer is we just
25 need to keep more of what we're doing. We need to

1 coordinate and we need to be strategic as best we can to
2 go to those kind of critical path areas that will help us
3 all meet our mandates. It's a lot of work.

4 DR. WU: I think that's a really good question,
5 and Karl, I really appreciate your answer as well. And I
6 think you've sort of summarized why biomonitoring is so
7 hard for us to figure out our priorities, because all of
8 these things are so important. I mean, do we want to look
9 at legacies or the new ones? Is it more important to
10 figure out the percentage of exposure source for -- you
11 know, is it the bought exposures or every little exposure.
12 Are the highly exposed individuals or the general
13 population more important? And then is it -- you know,
14 how do we get this information? How do we actually make
15 an impact? How do we work with our partners to message
16 out how people can be healthier and make more safe
17 choices?

18 So all of these things are important and you
19 would need a much bigger program to address all these
20 things. So it is why we often have these questions, like,
21 how do we make the biggest impact? What -- which one of
22 these parameters would be -- would be key for us to follow
23 through?

24 And I think it's great that we've had a much more
25 robust interagency collaboration on PFAS. I think it's

1 one of the things that really feeds our work in PFAS and
2 helps us kind of address all of those things. But it is a
3 giant machine for us to be addressing with a very small
4 Program, and so I appreciate the difficulty of it.

5 CHAIRPERSON SCHWARZMAN: Sara, you have something
6 to answer. And then I just want to say, it sounds like
7 Kathleen has something to add to this question, and then
8 we'll move on to Ulrike's question, and I see Carl next.

9 MS. HOOVER: I just had a quick logistics matter.
10 Just for the benefit of the transcriber, particularly if
11 your camera is not showing, make sure to identify
12 yourself. So that was Nerissa, which I'm sure Jim will
13 figure out. But for those of you who are visible, it's
14 pretty easy for him to figure out who is speaking, but
15 make sure you identify yourself again when you speak.

16 Thanks.

17 CHAIRPERSON SCHWARZMAN: Thank you.

18 Kathleen.

19 DR. ATTFIELD: Thank you. And apologies, my
20 video does not seem to be working. So I will try to speak
21 clearly. Kathleen Attfield.

22 I also wanted to point, Tom, this -- you know,
23 this is the huge question and point Tom to our later
24 speakers, who are going to help us with thinking about
25 other ways that we are looking into being able to

1 understand exposure sources. So we do have -- of course,
2 biomonitoring is cumulative across many different types of
3 exposure sources and we have questionnaires, but, you
4 know, that is only going to inform us so far. We do not
5 have, so far, information on people's general dust levels
6 or, of course, drinking water, and, of course, PFAS have
7 bioaccumulated in our bodies for such a long time, so
8 questionnaires definitely have their limitations as far as
9 being able to reveal historic sources. But more
10 conversation on that from the presentator -- presentations
11 coming after us.

12 CHAIRPERSON SCHWARZMAN: Thank you.

13 Ulrike.

14 PANEL MEMBER LUDERER: Yeah, I wanted to also
15 thank the presenters for those really interesting and
16 thought-provoking presentations. My questions I think
17 though are maybe more for Kathleen. And they relate to
18 these associations of the racial disparities and
19 association with seafood consumption as regards some of
20 these PFAS results.

21 And so one question I had was whether -- so you
22 looked at mercury, and you saw that with blood mercury,
23 there was also -- that that seemed to -- you know, that
24 also was associated -- had the same kind of racial ethnic
25 disparities. And I was wondering if you did speciation of

1 arsenic, which is also very strongly associated with the
2 organic forms of arsenic with seafood consumption, and
3 whether you saw similar results with that, if those -- the
4 speciation was also done, because that would help to I
5 think maybe support that association even more.

6 And then another question I had was whether you
7 had information about the specific types of seafood they
8 were eating? And I think with these long-lived PFAS you
9 would expect that the -- you know, the predatory species
10 higher up in the food web would have a stronger
11 association. So those were my two questions. Thanks.

12 DR. ATTFIELD: Thank you, Ulrike. For arsenic,
13 we do speciate when they hit a certain threshold. And so
14 we don't have that information across the entire CARE
15 study, but it does mean, yes, then we can look a little
16 bit more in those folks that have been speciated.

17 And your other question was about the specific
18 types of fish that are consumed. So, no, for CARE, we
19 don't have that granularity of information and those types
20 of questions are about general consumption patterns, so
21 not tied to a particular time period. However, in the ACE
22 studies, we've got quite detailed questions about the
23 types of fish and shellfish that people have been
24 consuming, not only kind of general and over the past
25 year, but in the last 30 days.

1 So different time periods can tell you different
2 things and tie differently to the analytes of interest.

3 CHAIRPERSON SCHWARZMAN: Thank you. I had the
4 same question about the species of fish, thinking does it
5 travel the same way mercury does and can you give
6 advisories about consumption in that same way just because
7 it's a -- they're persistent and bioaccumulative
8 compounds?

9 Thanks.

10 I think Carl is next up with a question. And it
11 could be tricky to remember to put your hand down on the
12 Zoom interface. So if you can do that when you're done,
13 that will help us.

14 PANEL MEMBER CRANOR: Thank you. I thought those
15 were terrific presentations. One of them caught my eye.
16 It's always the shortage of funds for dealing with these
17 problems. I did notice that there -- one of the items I
18 believe Carl highlighted was the possibility of
19 compensation for spreading PFAS and their varieties all
20 over California, and in the food and so forth. Minnesota
21 had a very successful suit against 3M, I believe, and
22 DuPont. And I'm wondering if there has been thought given
23 to that, because they had a -- in Minnesota they have a
24 huge clean-up problem. They also have huge clean-up
25 problems in West Virginia and northern and southern Ohio.

1 And I don't know how detailed their health
2 effects had to be, but that was part of it. And I have a
3 legal document that was used in the Minnesota case as
4 evidence.

5 MR. PALMER: Can I just make a quick comment to
6 Carl's point. So one of the things that U.S. EPA is
7 proposing in their roadmap is to list PFAS as a CERCLA
8 hazardous substance, which would then bring it into the
9 domain of the clean-up authorities that many states have,
10 and they have at the federal, and the liabilities and
11 responsibilities that come with that. Similarly under the
12 Resource Conservation Act -- Recovery Act, EPA has been
13 petitioned to add PFAS containing waste as RCRA hazardous
14 waste. So many of us, whether it's in hazardous waste or
15 in water, you know, you have certain authorities, only if
16 you're captured in the regulatory framework.

17 And I think the other thing is -- that's relevant
18 is that what we're talking about is moving upstream
19 hopefully, which is rather than waiting till we see it in
20 people and the environment, what can we do to encourage
21 using safer alternatives. And that's difficult as well,
22 because we don't have the authority and we also don't have
23 the knowledge of where all these chemicals are used in the
24 supply chains.

25 And we see it in the environment. We see it in

1 fish. We can measure it in people. We need to do better
2 to coordinate on that, but we also need to move upstream
3 to find out why these chemicals are actually being used
4 and if there are safer alternatives.

5 PANEL MEMBER CRANOR: Thank you.

6 CHAIRPERSON SCHWARZMAN: Thank you.

7 So that's -- José, did you have a question.

8 We're just about out of time for Panel questions, but then
9 we'll come back after a moment of comment to Panel
10 discussion. So if it's a longer point.

11 PANEL MEMBER SUÁREZ: It can wait.

12 CHAIRPERSON SCHWARZMAN: Great. Okay. We'll
13 hold it till then. So I think we have 10 minutes for
14 public comment here and I want to start that by just
15 reading a question that was put into the Zoom chat from
16 Silent Spring Institute. And Sara, you can tell us if we
17 need a name or if that's sufficient identification?

18 MS. HOOVER: I'll just -- this is Sara answering
19 Meg. Sure, if they're willing to identify themselves,
20 that would be great for the transcript. They're not
21 required to, but yeah.

22 CHAIRPERSON SCHWARZMAN: So the question is, "Are
23 the CARE participants provided with their individual
24 results and translational resources to understand their
25 significance"? My understanding is, yes, under the

1 statutory requirements, but I'll let someone in the
2 Program explain more.

3 DR. WU: The answer is yes, as you have said. In
4 accordance with our legislation, all participants with
5 biomonitoring studies are -- their results are made
6 available to them. And about 98 percent of our
7 participants do elect to receive their results. And so
8 production of these packets, which include not only their
9 results, but comparison to NHANES and study statistics,
10 but also potential exposure sources and associations with
11 health impacts are provided to participants of all of our
12 studies. The one exception that Kathleen alluded to is
13 the MAMAS study for which we don't have the identification
14 of participants. It's an anonymous sample, for which we
15 only have some demographic guidance.

16 CHAIRPERSON SCHWARZMAN: Great. We have a
17 comment here from Nancy Buermeyer of BCPP, Breast Cancer
18 Prevention Partners. Thank you to the SGP Biomonitoring
19 California -- that is, I'm just going to read the comment.

20 "Thank you to the SGP Biomonitoring California
21 and the Safer Consumer Products Program for your work and
22 specifically for considering and prioritizing PFAS as a
23 class. Not only did it support passage of the food
24 packaging, juvenile products, and firefighting foam PFAS
25 ban bills, the class approach has also allowed us to

1 require disclosure of all PFAS in various consumer product
2 sectors, including cleaning products, fragrance and
3 flavors, and personal care and cosmetic products, feminine
4 products, and most recently cookware".

5 So let me check in with staff about whether there
6 are any comments on the emails -- submitted by email.

7 DR. HOLZMEYER: There are not.

8 CHAIRPERSON SCHWARZMAN: And I can't see
9 participant requests to speak, can you Cheryl?

10 DR. HOLZMEYER: I --

11 DR. IYER: Hi. This is Shoba Iyer. I'm
12 monitoring for any raised hands amongst attendees and I am
13 not seeing any at the moment.

14 CHAIRPERSON SCHWARZMAN: Great. I have another
15 short comment in the question and answer section from
16 Sharyle Patton. "Two pesticides containing PFAS are
17 registered for use in California. They are hexaflumuron
18 and novaluron". Just to add on to the discussion of
19 pesticides that showed up earlier.

20 I want to leave just another moment for public
21 comment, since we are not out of time for that yet and it
22 could take a minute to navigate the interface and get a
23 question posted or raise a hand and have it spotted.

24 So as long as Shoba and Cheryl don't see -- oh,
25 Sara, did you want to --

1 MS. HOOVER: I'm just -- I'm just respecting the
2 pause. But when you're done with your pause, I wanted to
3 chime in on one of the questions that was raised, so
4 whenever that's appropriate.

5 CHAIRPERSON SCHWARZMAN: Maybe I'll just check
6 with Cheryl and Shoba that there's no additional requests
7 for comment or submissions online through email. And
8 then, Sara, please go ahead.

9 MS. HOOVER: Okay. For those of you who have
10 been around for a long time, this will be of no surprise,
11 but not everyone is aware of OEHHA's very early and
12 foundational work on developing chemical groups and
13 classes for identifying for biomonitoring. So that's an
14 approach that Gail and I came up with very early in the
15 Program. We started with flame retardants and we extended
16 it. And that has been the standard approach that we've
17 used for chemical selection, including for PFASs.

18 So Gina Solomon, who is a former SGP member,
19 actually encouraged us to write it up in a paper, which we
20 did, and it was published in EHP. So I'm going to drop
21 that into the Q&A and we'll link to it on the meeting page
22 as well.

23 CHAIRPERSON SCHWARZMAN: Great. And I want to
24 second that just from somebody who wasn't involved about
25 how influential I've seen that be, the fact that

1 Biomonitoring Program scientists really went through the
2 tremendous amount of work that it requires to designate
3 and defend a class, and how then that ripples through, and
4 the way that Karl Palmer described how other groups both
5 within and outside of government can pick that up and use
6 it in other purposes. So I think it really has been a
7 tremendous contribution that the Program has made. And
8 then I was very happy to see it published and appreciate
9 that and I've given it to students and appreciate it being
10 in the literature.

11 So we have time now for Panel discussion. We
12 have 15 minutes. Actually, we're like five minutes ahead
13 of time, so we're okay. And I wanted to start with José,
14 who didn't get his question asked earlier.

15 PANEL MEMBER SUÁREZ: Thank you and thank you
16 very much for the presentations. One general question.
17 So right now, are the data made available say for
18 interested researchers in analyzing some of the CARE study
19 data and obtaining information of variables available, in
20 particular, addressing these questions that will be
21 pertinent to exposure sources of PFAS? I'm sure that the
22 questionnaires have captured a lot of information. Yet,
23 it might be, you know, interesting to have multiple people
24 starting to understand what are the main sources of these
25 exposures -- or at least exposures that are associated

1 with P -- greater PFAS concentrations within these
2 California groups.

3 DR. WU: Kathleen, are you answering the
4 question? I can also take it.

5 DR. ATTFIELD: Sure. Sure. Sure. So what is
6 available readily online is our distribution data, but we
7 do have the policy of wanting to work with outside
8 researchers. So there's a application process I think
9 detailed on our website. I was going to give a little
10 more information about what kind of questionnaire data is
11 available for CARE. So there is, as I said, some
12 information related to general dietary habits, as well as
13 occupation, drinking water source, some consumer product
14 use, such as water resistant sprays or water and stain
15 resistant clothing, and furniture. So there's a good
16 number of items that it covers.

17 And of course, for women, we have information on
18 pregnancy, because that is, of course, correlated. We do
19 not have weight and height, which is a limitation of the
20 data. I think that covers most things.

21 PANEL MEMBER SUÁREZ: And just as a follow-up.
22 So I'm actually on the website. Is it easily available to
23 obtain that information from the website? Is that
24 something you want to have available?

25 DR. ATTFIELD: I'm sorry. Are you asking the

1 types of questionnaire data that are available?

2 PANEL MEMBER SUÁREZ: Yeah, for that matter, I
3 mean, what is available for a researcher to be able to ask
4 you a more direct question about maybe you want to get
5 these variables and look at these associations. Is that
6 on the website? I'm just -- haven't spent too much time
7 with it.

8 DR. ATTFIELD: If you -- José, no, it is not
9 currently on the website and I will punt that to Nerissa,
10 as far as that has definitely been something we have
11 wanted to do.

12 DR. WU: So what is on the website are our
13 questionnaires. I believe both the ACE and maybe the CARE
14 questionnaire are available, which would give somebody
15 starting to think about this an idea of the kinds of
16 questions we ask. Of course, the next step is to talk to
17 us about, you know, how did a question work? Was there
18 homogeneity or heterogeneity in the answers? Is it a
19 question that we're really going to be able to do analyses
20 with. But I think as Kathleen has described in her talk,
21 there are lots of opportunities for research. And it's
22 beyond what we as a program can do. And one of our -- one
23 of our big challenges is to get to all of this analysis.
24 We ask all these questions. We have piles of data. And
25 for those of you in academia who may have students looking

1 for projects to work on things, we are very happy to work
2 alongside your students.

3 PANEL MEMBER SUÁREZ: Yeah, I mean, and just the
4 final piece -- and this is -- can be a little more
5 complicated, but could help very profound -- it's -- it
6 could be a profound way to also involve people from the
7 community -- is in some sites, if somebody wants to just
8 log in there and just click, click, click away, as, you
9 know, one of the exposure concentrations in certain groups
10 sometimes some sites have ways in which you can click and
11 look at that, and then you get some summary output
12 statistics. Of course, that involves some investment from
13 the other side, right, from the website generation things,
14 but it could be something to start getting the community a
15 little more engaged, so they can look at these things.

16 Any comment in that regard, the feasibility of
17 doing something like that or maybe you're doing something
18 like that already?

19 DR. WU: I think our web platform is not as
20 sophisticated as some private organizations. And so I
21 think there are limits to what we might be able to post on
22 our website. One of the reasons we are putting up the
23 CARE report is so that data will be available. And it's
24 not in a clickable easily accessible format, but it will
25 get into more detail about, you know, different cells

1 within our -- within our study population. We'll get much
2 more into exposure questions that were considered and why
3 or why not they were -- we followed them with additional
4 analyses.

5 So the report is kind of our step in that
6 direction to make more transparent and more available the
7 kinds of information we've done. And it's a learning
8 process for us if a question doesn't work, you know, why
9 or why not, and that informs our next questionnaire. But
10 we also want to have that kind of information available
11 for other researchers who might be thinking of asking a
12 similar question.

13 So I think your question -- your proposal is a
14 good one. I think it's -- IT work is very -- is fairly
15 difficult for us to accomplish in the Program, but the
16 report is one way we'll try to accomplish those goals.

17 PANEL MEMBER SUÁREZ: Thank you.

18 CHAIRPERSON SCHWARZMAN: So maybe just to flag
19 for the moment that because Kathleen was specifically sort
20 of requesting for collaboration and essentially help
21 analyzing some of the data that are available, and José is
22 asking about accessibility of the same information, it
23 sounds like some of it may not be, you know -- you can't
24 passively access it, but there's an open invitation to
25 engage with the Program and do more analysis of the data.

1 So I just wanted to flag that, because I think that's what
2 I took from that part of the discussion.

3 PANEL MEMBER SUÁREZ: Yeah. I mean, and it would
4 be nice to make it maybe perhaps a little more explicit.
5 Just by looking at the website, I have to get -- so the
6 options are to learn more about the study and then it
7 talks about the CARE study in LA County and frequently
8 asked questions. But it might be good to have a section
9 saying, well, for -- if you want to find out more how to
10 get data out of what it is that you need to do or what
11 data is available, first of all, so a researcher can first
12 see what's available, and then have a more direct question
13 to you, so you don't have to start explaining the same
14 thing over and over as to what data is available and
15 things like those.

16 CHAIRPERSON SCHWARZMAN: Great.

17 Veena.

18 PANEL MEMBER SINGLA: Thank you. Thank you so
19 much to all the presenters for really informative slides.
20 And I wanted to really express my appreciation to Vince
21 and others who -- for their kind words on my service on
22 the Panel. And, you know, I'll say this is definitely not
23 the last of me in these meetings. I'm sure I will be
24 back. So it's not goodbye, just until next time.

25 And I had a comment and a question related to the

1 discussion of sort of the priorities around PFAS
2 biomonitoring for the Program, because as usual, there is
3 no shortage of work to be done here and many, many
4 different avenues and angles that are worthy of
5 exploration. So, you know, I did want to second a comment
6 that Karl had made around gathering data and evidence
7 relevant to understanding if policies are effective.

8 So we heard about a lot of great legislation and
9 work at the agency on kind of different sources and
10 products, so, you know, drinking water, food packaging,
11 food serviceware, juvenile products. So, you know, I
12 think to the extent that data and study designs can really
13 help speak to how the -- those policies, as they're being
14 implemented, are effective, and changing or affecting PFAS
15 exposures would be extremely valuable.

16 And then my other question on kind of the
17 priorities piece is the sort of ability to kind of get
18 input from communities or partners as to their priorities
19 moving forward, because I know the Program has really good
20 relationships with some of the groups they've partnered
21 with like for the ACE study and other studies. So I think
22 that could also be a really good discussion to inform
23 priorities moving forward to kind of reflect what is most
24 important to communities and what they want to know.

25 CHAIRPERSON SCHWARZMAN: Thank you, Veena.

1 I kind of want to echo something that you just
2 said sort of with illustration from my own work. I think
3 this has come up in past -- our past discussions too. And
4 I think maybe we have echoed each other's points on this,
5 but as people who, I think, both of us work with sort of
6 finding evidence for and against various policy
7 interventions, and that in my work on it what has proved
8 the most challenging is finding data from which you --
9 that you can use to establish time trends. And I think we
10 all understand why that's hard. You know, you were -- you
11 have to come back and measure either the same or
12 comparable populations with the same or comparable methods
13 for the same chemicals over time. And so to manage to
14 have done that for a lot of different chemicals over a
15 long period of time probably requires a level of resources
16 that has never been put into biomonitoring essentially,
17 you know, somewhat, of course, through NHANES at the
18 federal level.

19 But just to -- just to kind of echo what Veena
20 said about when we're looking at the impact of policies,
21 what we really need is to be able to suss out time trends,
22 because there was one level of exposure. There were
23 things that happened in the interim and then we want to
24 know what is happening to the other exposure levels, and
25 just acknowledging how -- what a big ask that is of a

1 research study to create that data, but that that is kind
2 of the Holy Grail in terms of being able to see what's
3 happening over time and make some guesses about which
4 interventions have the greatest effect. So just to sort
5 of echo that point and how hard it has been -- how hard it
6 has been to find data on that.

7 Jenny.

8 PANEL MEMBER QUINTANA: You just said very
9 eloquently one of my points about time trends -- the
10 importance for policy and seeing that public health
11 policies work. But the other point I wanted to add on top
12 of that was -- and to get you back to your original
13 question about what should be our priority -- priorities
14 for Biomonitoring California, I think that also monitoring
15 disparities and changes in disparities over time is
16 important. I think we saw that a little bit with the
17 flame retardants that exposures change and then they
18 changed over time with, you know, increasing or continuing
19 exposures in certain populations and reductions in others.
20 So I just wanted to add that as a priority I think for the
21 Program.

22 CHAIRPERSON SCHWARZMAN: Great.

23 Tom.

24 PANEL MEMBER McKONE: So this is sort of a
25 comment and a question. And it follows the trend. I

1 mean, this is one of the hard things to do when you're
2 just looking at tissue levels or biomonitored levels is to
3 really understand what's going on. And I think -- I mean,
4 we brought this up many times about multiple pathways.

5 So in making this comment, I have to, you know,
6 first reveal my conflict, or bias, or whatever. Matt
7 MacLeod was a post-doc with me about 20 years ago for two
8 years, so I'm -- and I've collaborated with him a lot.

9 But I raise that -- so I was impressed to see
10 that you're working with his group in Stockholm. I mean
11 there are other groups who are as good and -- but I think
12 they're outstanding. And the reason they're useful for
13 trying to under -- piece this together is that Matt is a
14 modeler who sees models not for prediction, but for
15 understanding. And I think that's what we need in this
16 and that's why I say I'm biased, because I think that way
17 too. I don't -- I don't think models are tools that you
18 go out and say this is what -- you know, we're going to
19 predict what happens, we're just trying to see if
20 they're -- you know, if we can begin to connect more dots
21 and put things together.

22 And so my question is I hope that there's some
23 continuing collaboration, either with Matt MacLeod or
24 other people who do that kind of cumulative exposure,
25 multiple pathway exposure linked to pharmacokinetics to

1 try and see if we can make sense of what's happening in
2 the relationship.

3 DR. ATTFIELD: Well, this is Kathleen Attfield.
4 I'll respond to that in that we're just in the
5 beginning stages of that collaboration.

6 PANEL MEMBER MCKONE: Okay.

7 DR. ATTFIELD: So it will be continuing. And it
8 actually had started a couple years ago, but he had a
9 delay. So the upside of that being that now we have
10 CARE-2 data that he can work with as well.

11 CHAIRPERSON SCHWARZMAN: Any other responses or
12 thoughts from the Panel based on the morning's
13 presentations so far?

14 Ulrike.

15 PANEL MEMBER LUDERER: This is sort of a minor
16 and specific question, but something that I found
17 intriguing in -- I think it was in Karl's presentation
18 related to the chrome plater -- platers as a source of
19 exposure to PFAS. And I noticed on the map that the
20 location of the chrome platers was only suspected. And I
21 was wondering if you can say more about that, because
22 obviously knowing where these exposures are coming from is
23 really important. And if there's not information about
24 where chrome plating is happening, that's a potentially
25 important source of information that there may be a

1 lack -- you know, may be lacking actually.

2 MR. PALMER: Yeah. Thanks for the question. I'm
3 not sure the Air Board specificity on that. I do know,
4 having worked with chrome platers for many years, is that
5 we know where -- I know the Air Board knows where most of
6 them are. Part of the big question is what are they
7 using, because there's a variety of different bath that
8 they use in processes. And they purchase these chemicals
9 based on a spec and a function, not on content.

10 And so oftentimes, the platers don't --
11 themselves don't know what are in those chemicals. And
12 even some of the companies that provide them may not know,
13 depending on their supply chain. So it's complicated, but
14 I think Air Board does know where all the chrome platers
15 are, but I think the bigger issue is the chrome platers
16 may themselves not know what's in the materials that they
17 use.

18 PANEL MEMBER LUDERER: Thank you. That is --
19 that's a huge issue for sure.

20 CHAIRPERSON SCHWARZMAN: Yeah. I'm just going to
21 say like another illustration of the problem that has
22 plagued the use of chemicals in products and materials
23 since they were invented, at least in our system of
24 governance.

25 We're just about to move on to our next

1 presentation, but I just want to check for any final
2 comments.

3 Yes, please, José.

4 PANEL MEMBER SUÁREZ: Just one final -- just more
5 of a methods comment. Given the vast distribution of
6 PFAS, how much thought or concern was there for the
7 methods during the sample collection, sample storage
8 during aliquoting and things like those to reduce some of
9 the PFAS exposures that may be coming in say from
10 cryovials or other storage media in which you have?

11 DR. WU: Well, our lab - I think June-Soo is on -
12 could address that, other than I, but we do run blanks for
13 everything sealed and lab blanks. June-Soo, do you want
14 to weigh in on this?

15 DR. PARK: Yeah. Sure. Thanks for the question.
16 Not only the carrying -- to collect field blank, but also
17 it was wider goal before we decided to use the test to
18 collect the blood, we tested them -- we purchased and
19 tested them for PFAS background. And we confirmed the --
20 it has a background free from PFAS compound. Then we
21 decide to purchase work and send them out for the field
22 collection. That's what happened. So not only covered by
23 method blank, field blank, but also we already tested it
24 out -- test it totally before we choose that brand.
25 That's what we always do for Biomonitoring Program. I

1 hope I answered your question, José.

2 PANEL MEMBER SUÁREZ: Yeah. Yeah. And just out
3 of curiosity, so you found -- did you find a wide range of
4 PFAS concentrations just in the like vacutainer tubes
5 or -- I mean, I'm not picking on the brand, but on the
6 blood collection tubes?

7 DR. PARK: No, we didn't. Yeah, we didn't. We
8 tested, I believe, a couple of brand, so -- but we didn't.
9 Also, we stopped using the red-top tube to -- in order
10 to -- the easier implement -- you know, for field staff,
11 we choose using the serum-separation tube, but we didn't
12 find much background for the test tube with that brand we
13 tested, yeah.

14 PANEL MEMBER SUÁREZ: And then was this measured
15 in plasma or in serum for PFAS? And then the next
16 question is were there samples that were stored in other
17 cryovials and did you get a chance to look at PFAS in some
18 of those cryotubes for instance?

19 DR. PARK: Yeah. Go ahead, Sara. I can answer.

20 MS. HOOVER: I just wanted to suggest that you
21 hold this for the later discussion, because we really do
22 need to move on to stay -- to stay on our scheduled time
23 slot. And we have a whole hour later to talk about these
24 issues.

25 CHAIRPERSON SCHWARZMAN: José, will you just make

1 a note so you don't forget.

2 PANEL MEMBER SUÁREZ: Wonderful. Will do.

3 CHAIRPERSON SCHWARZMAN: Okay. Thank you.

4 DR. PARK: Thank you

5 CHAIRPERSON SCHWARZMAN: Thanks very much. And
6 thank you to the staff who updated us this morning.

7 We're going to move on and I want to introduce
8 Anna Kärrman. She's Deputy Head of the School of Science
9 and Technology and Associate Professor of Environmental
10 Chemistry at Örebro University in Sweden. Her main
11 research agenda is to unravel the drivers of toxicity by
12 seeking relevant and sensitive methods, including applying
13 non-targeted methodologies to identify and quantify
14 organic pollutants. She focuses on analytical chemistry
15 and emerging organic pollutants, their distribution in the
16 environment, sources, and human exposure. Anna has
17 conducted studies on per- and polyfluoroalkyl substances,
18 microplastics, and other contaminants of concern. And
19 here she'll be discussing novel approaches for expanding
20 the range of PFAS analyses.

21 (Thereupon a slide presentation.)

22 DR. KÄRRMAN: Thank you very much, Meg, for the
23 introduction. Can you hear me okay?

24 CHAIRPERSON SCHWARZMAN: Yep. Perfect.

25 DR. KÄRRMAN: Thank you.

1 Well, good morning and good evening, I could say
2 at the same time. I'm going to share my slides here. So
3 I'm currently in Sweden at the -- in Örebro, and I will
4 talk to you about measuring PFAS. Let's see if I can get
5 it in the right mode. Is this the --

6 CHAIRPERSON SCHWARZMAN: That's good. Perfect.

7 DR. KÄRRMAN: Okay. Excellent. Thank you very
8 much. Thank you for inviting me to talk about measuring
9 PFAS.

10 --o0o--

11 DR. KÄRRMAN: I would like to start with a short
12 disclosure that I have no conflict of interest to
13 disclose.

14 --o0o--

15 DR. KÄRRMAN: So I would like to take the
16 opportunity to focus on the analysis of PFAS as a whole
17 group and present to you some of the possibilities and
18 challenges that I have identified in the last couple of
19 years trying pursue this measuring method. And more
20 specifically, I would like to present some of the
21 experiences you've seen in combustion ion chromatography
22 analysis. So I will present a few studies on
23 environmental and human matrices using this CIC method and
24 compare it to target PFAS screening. And also, we have
25 done a little bit of quality control using this method.

1 And finally, I will present some of the conclusions from
2 my work.

3 --o0o--

4 DR. KÄRRMAN: So in the sake of discussing PFAS
5 as a group, I would like to focus on fluorine in my
6 introduction. So the most common form of fluorine found
7 in nature is fluoride. So here it exists as different
8 mineral salts in quite high abundances in some
9 environmental compartments.

10 There are only a few examples of natural
11 occurring organofluorine. So one example is the molecule
12 that you can see in this picture. And it's fluoroacetate,
13 which is being produced by some plants as a protection
14 against grazing. There are also a few known examples of
15 natural occurring organofluorine compounds from volcano
16 activities.

17 But the large proportion of organofluorine that
18 we might find in nature is anthropogenic organofluorine,
19 such as PFOS. And this belong -- these compounds then
20 belong to per- and polyfluoroalkyl substances that
21 represent the class of substances depending on the
22 definition, but I have chosen to use the latest OECD
23 definition saying that they should contain at least one
24 perfluorocarbon moiety.

25 So, of course, it's important -- as mentioned

1 before today, it's important to acknowledge which kind of
2 definition we are choosing. So this has been the -- this
3 has been on the discussion for many years, how to define
4 this class.

5 --o0o--

6 DR. KÄRRMAN: So narrowing it down a little bit,
7 when it comes to monitoring, it tends to be around three
8 different groups of PFASs. So the first group is
9 perfluoroalkyl acids. They are the perfluorinated acids.
10 For example, the sulfonic acids or the carboxylic acids.
11 We have a large group of precursor compounds that are
12 semi-persistent and can be further transformed to the
13 perfluoroalkyl acids. And we have a group that contains
14 different kind of fluoropolymers.

15 And when it comes to usage in products and
16 production volumes, it's the two classes to the right that
17 are the most important. So they are being produced in the
18 highest volumes, and they are being used in products the
19 most, or even -- or more than the perfluoroalkyl acids.

20 --o0o--

21 DR. KÄRRMAN: So the motivation behind monitoring
22 PFAS is obviously that we want to be able to study these
23 different classes, how they affect the environment and how
24 they affect us humans. So the latest news from Europe,
25 you might say, is that the European Union decided earlier

1 this year to revise the drinking water directive and
2 include a group approach for PFAS total, meaning that the
3 totality of PFAS will have a threshold concentration of
4 0.5 micrograms per liter in drinking water. So this new
5 threshold concentration is to be served as a complement to
6 the limit that is based on 20 individual PFAS compounds.

7 However, there is no method mentioned in the
8 drinking water directive. And this new group approach
9 should be implemented as soon as the required method
10 becomes available. And this is quite good news for us
11 scientists, I would say, that the European Union has
12 adopted this group-based approach. And the motivation of
13 this is, of course, the problem with different replacement
14 products showing up and also about the regrettable
15 substitution.

16 So with this group approach, we will have some
17 more tools for PFAS control. And the basis of this group
18 approach is obviously the precautionary principle that
19 allows decision-makers to take measures, even though the
20 scientific evidence is not really showing exactly which
21 compounds are environmental or human hazards. But when
22 there is really high stakes, there is no need to show the
23 full scale of evidence. So, for example, in Sweden, we
24 have had this precautionary principle when it comes to
25 pesticides for a long time.

1 So if a substance is being used as a pesticide,
2 it cannot end up in the groundwater. Even though there is
3 no toxicity data, there is a rule saying that it should
4 not end up in the groundwater regardless. So there is a
5 limit value of all pesticides regardless their structure
6 and properties.

7 --o0o--

8 DR. KÄRRMAN: So for PFAS then, if we want to
9 look at the total PFAS, it comes quite close to mine to
10 look at fluorine as like a marker for PFAS. And this
11 picture -- I will not go into so much details, but this
12 picture tries to illustrate what we can do and how we can
13 define different types of fluorine.

14 So if we start from the very top, we have total
15 fluorine, which we might be able to measure when we take
16 food packaging material and we take some sort of fluorine
17 detection and we measure directly on the packaging
18 material, we will get the total fluorine content. But we
19 don't really know so much what the fluorine consists of.
20 And the very opposite going down in this tree, we have the
21 target organofluorine, which might be PFOS, PFOA, or 20 or
22 40 different target PFASs.

23 Total fluorine, of course, can consist of
24 inorganic fluorine and organic fluorine. And we're not
25 very -- we're not interested in the inorganic part, so we

1 want to try to isolate the organic fluorine. And doing
2 that often it involves some sort of extraction to be able
3 to take away the inorganic form.

4 And doing this, we might absorb the
5 organofluorine on the carbon material, we might extract
6 out it using different sorbents or different solvents, but
7 there's always a risk that there are organofluorines that
8 we will not be able to extract out.

9 And, of course, going from the top to the bottom
10 in this fluorine tree, we gain increasing specificity of
11 PFAS, meaning that we will be more certain that we are
12 actually looking at the CF2 chemicals that we want to
13 target.

14 --o0o--

15 DR. KÄRRMAN: So there are a number of different
16 possibilities to be able to assess the total PFAS. If we
17 want to directly measure PFAS total, that will be very,
18 very challenging to do. So in the literature today, you
19 can find two other assessments that are more commonly
20 used. So we have the extractable or the adsorbable
21 organofluorine that I mentioned before, which means that a
22 suitable extraction method is chosen for the sample matrix
23 in question together with some fluorine-specific detection
24 or total fluorine where we use direct measurement of
25 fluorine with some sort of detection that is specific for

1 fluorine.

2 --o0o--

3 DR. KÄRRMAN: And there's a number of methods
4 that are being described. So a number of
5 fluorine-specific methods are available: the combustion
6 ion chromatography, CIC, the Particle Induced Gamma-ray
7 Emission, the PIGE spectroscopy; we have inductively
8 coupled plasma mass spectrometry, ICP-MS; and continuum
9 source graphite furnace molecular absorption spectroscopy.
10 So I will not go into any of these details.

11 And at the very bottom quite interesting, we can
12 find actually specific methods for perfluorinated
13 substances. So that is exactly that I said was very
14 challenging, so how can we be very specific on CF₂ parts
15 or the molecule? So we do have methods that can be
16 specific, but unfortunately the detection limits are a bit
17 too high to be useful in all applications. But in a few
18 applications, there's definitely good methods for
19 perfluorinated substances.

20 --o0o--

21 DR. KÄRRMAN: So what are the challenges? So at
22 the moment, I would say that one challenge is that the
23 high standardization requirements might prevent data from
24 coming out on PFAS as a group. And this is data that
25 could be very useful at the moment to do initial hazard

1 assessment of the whole group of PFAS.

2 Another challenge is that there's a huge demand
3 for low quantification levels. So at the same time as
4 the -- is the requirement to measure PFAS as a group, at
5 the same time, there's also high demand for very, very low
6 quantification levels of the target PFAS. So one example
7 is that the European Food Safety Authority, EFSA, reduced
8 the tolerable weekly intake with three orders of magnitude
9 from only 2008 to 2020. So currently, there is a TWI of
10 4.4 nanogram per kilogram body weight per week for the sum
11 of PFOS, PFOA, PFHxS and PFNA.

12 And member state has reacted to this. So in
13 Sweden where I live, we have not really revised any of our
14 limit values yet. But our neighbor Denmark recently
15 launched a new limit value for the sum of the four PFAS in
16 drinking water to 0.002 microgram per liter. So there is
17 definitely a demand for very sensitive analytical methods.

18 --o0o--

19 DR. KÄRRMAN: Another challenge is, of course, to
20 obtain these PFAS total measurements to remove the
21 inorganic fluoride before using any fluorine-specific
22 detection method, which is very important, especially for
23 some matrices.

24 Another question that is heavily debated is if we
25 really want to target all organofluorines? So, for

1 example, there are pesticides and pharmaceuticals that are
2 low-fluorinated compounds that might not be all too
3 relevant when it comes to human health or even
4 environmental health. So in Europe, at the moment,
5 there's a large discussion about this trifluoroacetic
6 acid, which is a transformation product from many
7 different chemicals. And it's occurring in very, very
8 high concentrations in our natural waters. And this has
9 the CF₃ group, which makes it a PFAS compound.

10 We also have some pesticides and pharmaceuticals
11 that also contain the CF₃ groups. And I have one example
12 here of an LCM-28 substance, which is a liquid crystal
13 monomer, which is used in flat screens, cell phones, for
14 example, tablets. So is this what we want to target in
15 our PFAS total assessment or not?

16 And finally, even though we have the detection
17 methods needed, we do have a quite big challenge with the
18 extraction method to be able to capture a wide range of
19 different PFAS compound that constitute this PFAS total.
20 So probably there will be requirements for multiple
21 extraction approaches to capture PFAS total.

22 And there was a discussion about blank
23 contamination before. And I can mention that it's like
24 starting all over again when measuring PFAS total when it
25 comes to checking all the lab equipment for any kind of

1 fluorine-containing substances.

2 --o0o--

3 DR. KÄRRMAN: So I would like to continue now
4 with one of these techniques that I mentioned that could
5 be used for fluorine detection and that is the combustion
6 ion chromatography, CIC. So in this technique, we can
7 introduce a sample that can be a solid or a liquid
8 containing all different kinds of organofluorines. And we
9 have a combustion oven that we have it working on 1,050
10 degrees Celsius to be able to break the bond between
11 carbon and fluorine. And the combustion is done together
12 with water, so we have a hydrolysis forming, HF which
13 is captured in water forming fluoride. And we can measure
14 it using very conventional ion chromatography.

15 So what we also do is that we take the same
16 sample or extract and we also measure the target PFAS in
17 the same extract. Together, with the fluorine
18 concentration, we can do this fluorine mass balance, so we
19 know how much of the sample's organofluorine do we know
20 about from our target PFAS analysis and how much is
21 unknown.

22 --o0o--

23 DR. KÄRRMAN: So I would like to just go through
24 a few of our studies. So this a study where we did a
25 screening on -- of many different environmental matrices

1 from the Nordic countries. So we extracted out
2 organofluorine and also we targeted 73 known PFASs.

3 So as you can see here, we have a number of
4 different matrices that we analyzed. The blue bars are
5 the percentage of the known PFAS and the gray bar is the
6 percentage of the unknown organofluorine.

7 And here is the average target PFAS of the
8 extractable organofluorine in percentage. And as you can
9 see, the lowest percentage of known PFAS was found in
10 surface water, wastewater treatment sludge, and also
11 effluent water. And the highest proportion of known PFAS
12 was found in bird eggs.

13 So this shows -- this shows that we have quite a
14 large proportion of unknowns. However, we have no
15 information from this analysis on the identity of unknowns
16 and also measuring fluorine with CIC is less sensitive
17 than measuring the target PFAS. So we have quite a big
18 difference between detection limits for these two methods.

19 So the next step is, of course, to try to find
20 out what are the missing fraction, what is the unknown
21 fraction. And for this one method that is frequently used
22 by other labs as well and also including us is to do the
23 suspect screening, to identify unknowns.

24 --o0o--

25 DR. KÄRRMAN: So we used the database provided by

1 the Norman Network constituting of 3,236 individual PFAS.
2 And here are the same matrices. And we have a positive
3 hit on the red and the pink cells in this figure. So
4 there's two -- there's two different identification levels
5 for the red and the pink matches.

6 And by comparing different matrices like this, we
7 can conclude or we can see that there seems to be like
8 more low molecular weight PFASs in the water and effluent.
9 And then moving up to marine mammals and to bird eggs, we
10 have a higher molecular weight PFASs. However, we don't
11 have that great confidence in the identification, because
12 we don't have any standards for these compounds. We have
13 also seen that we have some transformation products, or at
14 least probable transformation products. So there could be
15 a biopic transformation going on, and we will actually be
16 able to extract out the transformation products.

17 Another thing is that one question that arises
18 if -- whether the analytical method will be able to ionize
19 all PFASs that our CIC instrument managed to analyze the
20 fluoride from. So comparing these to instruments in the
21 fluoride mass balance can be a little bit difficult
22 because the detection techniques are so different. So
23 there's definitely some challenges here.

24 --o0o--

25 DR. KÄRRMAN: This is a study of human blood

1 samples from Sweden during the same sort of fluorine mass
2 balance. And so this is Swedish whole blood from males
3 and females of different age groups. And the -- you can
4 see in this figure, the unexplained organofluorine or the
5 unidentified organofluorine as the black portion of the
6 bars. And after that, we have PFOA, PFHxS. We have a
7 branched PFOS, linear PFOS. And then we have a white
8 portion of the bar which is the sum of 60 other different
9 target PFAS.

10 So what was quite interesting in this study is
11 that looking at the fluoride -- organofluorine content of
12 blood, females had higher levels than males. Looking at
13 the target PFAS levels, that usually is the opposite. So
14 we did find large variations in groups and between groups.
15 But despite that, we could see a significant difference
16 between men and women, but also between some of the age
17 groups.

18 So this is a study that came out from our group
19 this year from our former PhD student Rudolph Aro.

20 --o0o--

21 DR. KÄRRMAN: So one can also question whether if
22 we have enough reliability in the method. And this is a
23 study that we also published this year looking at
24 groundwater effluent and sludge. And we could see that
25 between three laboratories we had quite a good coherence

1 using this CIC method.

2 --o0o--

3 DR. KÄRRMAN: So we could also demonstrate that
4 the methods were specific for organofluorine and that it
5 looked to be quite promising to be used as the drinking
6 water directives method for PFAS total in drinking water.

7 --o0o--

8 DR. KÄRRMAN: So my final -- let's see, I will
9 skip this. So my final slide here is that we do have
10 methods for assessing PFAS as a group. They are
11 available. And what we need to be looking at more
12 closely, in my opinion, is the extraction methods, which
13 are the key aspect of the PFAS total assessment.

14 Probably there's no single analytical approach
15 that will fulfill the policy goals and using the
16 extractable organofluorine CIC method shows that we do
17 have a large fraction of unknown organofluorine in
18 environmental and human samples that is probably needed to
19 look into more detail.

20 --o0o--

21 DR. KÄRRMAN: So I have a slide with references
22 at the very end.

23 --o0o--

24 DR. KÄRRMAN: And also would like to thank you
25 for listening. Thank you very much.

1 CHAIRPERSON SCHWARZMAN: Thanks so much, Anna.
2 That's wonderful to hear. We have until 12:25 for
3 questions from both the Panel and the audience. And I
4 will just check in with staff to see if there's questions
5 from the audience as we go through.

6 I have a question in the chat that -- asking
7 whether the slides will be made available. And I believe
8 everything is posted on Biomonitoring California website,
9 the page for today's meeting.

10 MS. HOOVER: Meg, this is Sara. Yes, I can
11 confirm. Oh, sorry, Cheryl. I just chimed in over you.
12 Yes, everything is posted.

13 CHAIRPERSON SCHWARZMAN: So -- and I also just
14 want to note that because Anna is joining us from Sweden,
15 she will not be with us for the afternoon discussion
16 session, which is not afternoon her time. And so we have
17 this 20-minute session now for discussion and questions
18 for her talk because she won't be available this
19 afternoon, so now is your chance.

20 I have a question in the chat here from Simona
21 Balan of DTSC, "do your conclusions or recommendations on
22 testing change in any way with regards to detecting PFAS
23 in consumer products as opposed to in drinking water"?

24 DR. KÄRRMAN: Yes. Thank you, Simona, for that
25 question. So I do think it's a little bit different when

1 considering consumer products versus drinking water,
2 because I believe that in consumer products, we might not
3 have the same problem with extracting out the relevant
4 organofluorine. So my experience with consumer products
5 is that it seems to be quite okay to analyze them directly
6 without actually even concentrate or extract out the
7 organofluorine, as opposed to drinking water where we do
8 have the need to both concentrate the organofluorines and
9 remove the inorganic fluoride in the water.

10 CHAIRPERSON SCHWARZMAN: Thank you.

11 Oliver, did you have a question or comment?

12 PANEL MEMBER FIEHN: Yeah. So thank you. That
13 was enlightening. Now, not everyone of us has these
14 methods. And these methods, as you say, are never
15 perfect, and they need to be combined, and more
16 extractions, which makes it harder for people to
17 implement. If you would compare methods, can you also
18 look for in an untargeted manner, using the mass defect of
19 fluorine in very high resolution mass specs, like
20 orbitraps and how would you rate those in comparison to
21 the methods you have just shown to us?

22 DR. KÄRRMAN: Yeah. Thank you, Oliver. It's a
23 very interesting question, because this is something that
24 we are doing at the moment comparing different methods and
25 trying to figure out where we can get the most relevant

1 information. And in addition to the ones that you
2 suggested, I might also want to mention the top assay
3 method come in -- that come out from Berkeley University.
4 That is also something that we are using quite frequently.

5 I would say that with the CIC method, we will get
6 a very comprehensive screening directing our interest to
7 samples of interest. It might be human samples from
8 cohorts that have been contaminated through the drinking
9 water. We can easily detect that with our CIC without
10 knowing which PFAS they was -- they were exposed to from
11 drinking water. But, of course, knowing which PFAS and
12 where it comes from, you kind of need more information
13 than just a fluorine signal.

14 So my experience, using high resolution MS is
15 that it's quite good to be able to sort out, which classes
16 we have, which chain length we have, but to be able to
17 distinguish immediately a contaminated cohort versus an
18 uncon -- a normal or occu -- or a background-contaminated
19 cohort. It's not that easy.

20 So I think using mass defect plots, you will be
21 able to detect new PFASs, but you might not immediately
22 see the whole proportion of the problem so to speak.

23 PANEL MEMBER FIEHN: A follow-up question. If
24 not by mass spectrometry, you could use ion mobility.
25 Aaron Baker from North Carolina University has shown that

1 fluorinated compounds, including PFAS, have a very clear,
2 and exactly what you say, a typical pattern that separates
3 out all the fluorinated compounds from non-fluorinated
4 compounds.

5 She's done it in pine needles over decades that,
6 you know, were sampled in botanical reserve -- reserves,
7 and so she could see how the PFAS in different locations,
8 for example, close to airports and so on, were, you know,
9 sampled, even historical samples. So she did it with ion
10 mobility. Have you considered that as well?

11 DR. KÄRRMAN: We don't have an ion mobility MS in
12 our lab, but I have used one in other labs. We also had a
13 cooperation with a group in Japan that uses that quite a
14 lot. I think definitely it's a good instrument, a good
15 way to go to, as you can -- as you say, compare different
16 samples from different regions to detect whether there is
17 an exposure somewhere that is new or different from
18 another group.

19 So I do think it's quite good. But honestly in
20 having a lab where I have the possibility to go both to
21 the orbitrap and to the CIC, I mean, I always go to the
22 CIC first, because it's very easy. It's fast and you get
23 a very clear quantitative result. But having the CIC
24 alone might not help, might not be able to -- I might not
25 be able to give you the research question directly, only

1 having this instrument. So, of course, if I had to
2 choose -- I had to choose between my mass spec lab and my
3 CIC lab - I couldn't keep both - of course, I would keep
4 my mass spec lab.

5 CHAIRPERSON SCHWARZMAN: Thank you. We have -- I
6 think you might have just answered this, but I just want
7 to say in the Q&A on Zoom, we have a question that says,
8 "Did you investigate Kendrick plots mass defect CF2 for
9 identifying fluorinated unknown compounds"?

10 DR. KÄRRMAN: Yes, we have done that. Also, I
11 would say that CF2, from my experience, is not the best
12 mass defect plot to make to be able to detect
13 fluorine-containing compounds. But often it's quite good
14 to include some oxygen-containing fragments as well. But,
15 yes, we have -- we have done that in our process of the
16 non-target screening, even though we usually use suspect
17 screening nowadays, because of the good libraries that are
18 available.

19 CHAIRPERSON SCHWARZMAN: And another question in
20 the Q&A is, "Have you looked into the use of XRF and LIBS,
21 laser-induced breakdown spectroscopy for total fluorine
22 testing, and if so, how do they compare with CIC and
23 PIGE"?

24 DR. KÄRRMAN: Yeah. No, unfortunately, I don't
25 have any comparison with those two methods, how they

1 compare with the CIC. I've been involved in some studies
2 comparing the CIC and the PIGE, and I am suspecting that
3 Simona knows about those already. But for the XRF and the
4 LIBS, I don't have any experience of those, no.

5 CHAIRPERSON SCHWARZMAN: Thank you.

6 We have just five or seven minutes remaining.
7 And I want to check in with staff if there are any
8 questions from the audience or attendees that we're not
9 seeing in the Q&A that you're getting by email or with a
10 raised hand.

11 DR. HOLZMEYER: There are not, no.

12 CHAIRPERSON SCHWARZMAN: Okay. Great.

13 DR. IYER: And no raised hands either.

14 CHAIRPERSON SCHWARZMAN: Thank you, Shoba and
15 Cheryl.

16 Ulrike, please.

17 PANEL MEMBER LUDERER: Yeah. Hi. Thank you Anna
18 for that very interesting presentation. I was curious
19 about the -- you know, I think one of the last things that
20 you said where you were talking about how females having
21 greater organofluorine total than males, but then the men
22 have higher levels of the targeted PFAS, whether you have
23 any information about what specific PFAS are driving that
24 higher level in the females?

25 DR. KÄRRMAN: No. That's a very good question.

1 And that is something that we want to look into in more
2 depth, and partly was in collaboration with Tom Webster
3 that's going to present later today. So there's, of
4 course, different speculations and hypotheses why women
5 would have a higher organofluorine level in their blood.
6 And some hypothesis involves higher exposure from personal
7 care products and other theories concerns more
8 pharmaceuticals that might be used more or less depending
9 on the gender. But there's just speculations at the
10 moment. So this is an observation that we made and we
11 need to look into it in more depth.

12 CHAIRPERSON SCHWARZMAN: One -- let's see there's
13 two questions. We have just five minutes, but
14 hopefully -- I think these are relatively short from the
15 Q&A. Sophia Schreckenbach asks, "Could you expand a bit
16 on what mass defect plots you prefer to use for PFAS as
17 opposed to CF2? Thank you".

18 DR. KÄRRMAN: Oh, so that I think will be very
19 quick from my point of view. I -- yeah, I think you
20 shouldn't do anything opposed to CF2. So CF2 should also
21 be included, but I think it's relevant to include other
22 mass defect plots containing fluorine as well. So there's
23 a few in my previous publications, but also in
24 publications from Mark Strynar, for example, from the U.S.
25 EPA. So I might just leave it by that.

1 CHAIRPERSON SCHWARZMAN: And Eric Gaudreau is
2 asking whether CIC is sensitive enough to detect organic
3 extractable fluorine in human serum when you only have a
4 hundred microliters available?

5 DR. KÄRRMAN: Yeah. So firstly, I would like to
6 mention that we have -- we've seen quite a lot of PFASs in
7 the red blood cells as well. So just looking at serum
8 will underestimate the internal body -- the internal
9 exposure and a hundred microliter is also a quite small
10 volume for the CIC. So unfortunately, the detection limit
11 is much higher compared to normal LC-MS analysis. So we
12 use at least 10 times higher than that at the moment,
13 yeah.

14 CHAIRPERSON SCHWARZMAN: Thank you.
15 Kathleen.

16 DR. ATTFIELD: Thank you. Thank you for the
17 presentation. And I was also very interested in the
18 differences by gender that Ulrike brought up. I was
19 wondering if you had more -- any historic samples that you
20 were able to do a comparison.

21 DR. KÄRRMAN: So my colleague Leo Yeung did a
22 time trend analysis on German blood some time ago, but
23 it's definitely something that we would like to continue
24 with and look into more, also the historical part of it.

25 We are also very interested in -- we have looked

1 at populations exposed by drinking water, the background
2 population. But it also seems to be quite different
3 depending on geographical location in Sweden, which we are
4 not very used to to see when it comes to the target PFAS.
5 So that is also something we would like to look into more
6 in detail.

7 CHAIRPERSON SCHWARZMAN: Great. Anna, thank you
8 so much for joining us and for your presentation.

9 We will break for lunch now. It's scheduled to
10 last an hour and we will restart right at 1:25. We're
11 asking that everybody rejoin the webinar no later than
12 1:20, so that we can start the afternoon session on time.

13 And before we adjourn, I'll just provide this
14 informal Bagley-Keene reminder that -- for Panel members
15 please comply as usual with Bagley-Keene requirements and
16 refrain from discussion -- discussing Panel business
17 during lunch or during the afternoon break.

18 And with that, I will adjourn the morning session
19 of the meeting and we'll reconvene here at 1:20 to start
20 again at 1:25.

21 Thank you, everyone.

22 (Off record: 12:24 p.m.)

23 (Thereupon a lunch break was taken.)

24

25

1 of you, even if it's not in person. And thanks for the
2 introduction.

3 --o0o--

4 DR. WEBSTER: So, you know, the usual thing. I
5 don't have any conflicts of interest.

6 --o0o--

7 DR. WEBSTER: All right. So what am I going to
8 be talking about today? Several different things. First
9 of all, which PFAS exactly are we talking about, something
10 about methods for investigating exposure to PFAS. I'm
11 going to touch briefly on some of the major exposure
12 routes, water, diet, and indoor exposure. Kate Hoffman
13 will say a little bit more about indoor exposure and
14 water. And then I'll finish up by really talking about
15 the relative contributions.

16 --o0o--

17 DR. WEBSTER: All right. So what PFAS are we
18 talking about?

19 So this is a variation on the slide that Anna
20 showed. And it's just again to underline what she said.
21 There's substantial amounts of unidentified organic
22 fluorine in human blood, environmental media, and consumer
23 products. And I really like this idea of using
24 extractable organic fluorine and then targeted analysis to
25 look at mass balance. And this is something that, as Anna

1 said, we're collaborating together to try to figure out
2 what some of this unexplained stuff is in human blood.

3 --o0o--

4 DR. WEBSTER: And really I think the question I'm
5 interested in is what is this unexplained stuff? Is it
6 PFAS that we're not measuring? So, for example, due to
7 lack of standards because for most PFAS we don't actually
8 have analytical standards. And there was a very
9 interesting case about this -- about a compound called
10 C604, that if someone wants to ask me about it later, I'll
11 tell you the story about that or is it something else?

12 The answer likely depends on the media. So, for
13 example, in wastewater, there's some nice work out of Rob
14 Letcher's group showing large amounts of side chain
15 fluorinated polymers in wastewater. In human serum, again
16 we don't know. Some of it is probably pharmaceuticals,
17 but there may be other things in there. And it depends on
18 the definition of PFAS, as someone mentioned earlier
19 today.

20 --o0o--

21 DR. WEBSTER: So this is actually something that
22 I got interested in a while ago and I have a student
23 writing a paper about this, that she presented at FLUOROS
24 last month. And we found at least eight different
25 definitions of PFAS that are sort of out there.

1 There's the bucket-all definition, which -- you
2 don't have to read all this. You can look at it later.
3 It's the one that California Biomonitoring uses. And it
4 essentially -- the key thing it has to be aliphatic and it
5 has to have one of these CF₃ groups on it. There's the
6 OECD definition, which was revised, let's see, this year
7 that Anna mentioned. And at the minimum, it has to have a
8 CF₂ group where the carbon has four bonds, two of them are
9 fluorine and those other two bonds are not a hydrogen or
10 some other things.

11 And just to mention, there are other definitions.
12 For example, there are several state laws that use a
13 definition of fluorinated organic chemicals containing at
14 least one fully fluorinated carbon atom. So to my mind,
15 this is actually ambiguous and potentially much broader,
16 because it depends what you mean by fully fluorinated. So
17 if you use -- if what you mean by that is the hydrogens
18 have been replaced by fluorine and at least one carbon,
19 which is what is usually meant, then that could include a
20 benzene ring that has a fluorine on it, right, because
21 you've replaced the hydrogens. So the problem is you're
22 not saying anything about the bonding of the carbon. And
23 that is a much, much broader definition.

24 --o0o--

25 DR. WEBSTER: So just an example of the

1 implications of this is this is the chemical structure of
2 Prozac, which you have heard of. A very commonly used
3 drug in the United States. And it has this fluorinated
4 methyl group up here. And it would be included under the
5 OECD definition, but not under the Buck definition, right,
6 because it has aromatic rings. It's not aliphatic.

7 And I think part of the point here is that, you
8 know, to my mind definitions can't actually be right or
9 wrong, but you can ask whether they're clear or not and
10 you can ask what is the purpose of the definition. Is it
11 for descriptive purposes? Is it for regulatory purposes?
12 Is it for surveillance?

13 And it all sort of depends. And so whether you
14 want to include drugs like Prozac in your definition or
15 not sort of depends on what the purpose of the definition
16 is. We can have a very interesting discussion about that.

17 --o0o--

18 DR. WEBSTER: All right. So the caveat about my
19 talk is that when we -- when I'm discussing exposure to
20 PFAS, I'm talking about what most people mean by this is
21 it's usually legacy PFAS, such as PFOA or PFOS, for which
22 there are actually data. Okay. I'm not talking about
23 exposure to Prozac.

24 --o0o--

25 DR. WEBSTER: All right. So how do we go about

1 figuring this out?

2 --o0o--

3 DR. WEBSTER: So again, PFAS is sort of a very
4 interesting situation, because we're mostly using serum or
5 plasma. Some have -- people have used urine for
6 biomonitoring. And we are interested in the persistent
7 compounds that we actually target, for example PFOA. And
8 that can result from either stable compounds or
9 precursors. That's the external exposure. And then they
10 are modified by pharmacokinetics to give us whatever we
11 see in serum or urine. So you all -- you all know this.

12 --o0o--

13 DR. WEBSTER: And again, biomonitoring integrates
14 different exposure routes. You can't actually tell, per
15 se, from the biomonitoring where it came from, because it
16 can come from diet, or water, or indoor environment, or
17 other things, like personal care products. And so what
18 you see say in blood is the resulting combination of
19 whatever was in those environmental media, things like
20 behavior -- human behavior, which connects us to exposure,
21 and then toxicokinetics.

22 --o0o--

23 DR. WEBSTER: So we really have two primary
24 methods, and I'm going to illustrate these for water. One
25 we'll -- I'll call it the epidemiologic approach, because

1 I actually think it is an example of epidemiology. It's
2 just that the biomonitored chemical is the outcome of
3 interest, not a disease. And so what you do is you
4 essentially regress serum, or blood, or whatever PFAS
5 concentrations against water concentrations for water,
6 okay? And you can do the same kind of thing for diet or
7 dust.

8 And the other is the exposure factor approach,
9 where essentially you take water concentrations that you
10 measure and you multiply it by a water consumption rate
11 that you get from the EPA's Exposure Factor Handbook, or
12 you ask people, or whatever.

13 And each of these approaches sort of has its
14 strengths and weaknesses, and we could -- I've done lots
15 of work with both of them and we could sort of talk about
16 those as well.

17 --o0o--

18 DR. WEBSTER: I should say there are two other
19 things that are used, maybe not quite as much,
20 chemometrics. So this was mentioned a little bit earlier.
21 There's been a handful of papers. A pretty good paper by
22 Elsie Sunderland's group using principal component
23 analysis as sort of a fingerprint idea and do the patterns
24 of the different PFAS that you measure in blood tell you
25 something about the sources? So I think that's -- this is

1 studies, in particular one with Kate Hoffman, that you'll
2 hear about later in the C8 studies in the West
3 Virginia/Ohio area. And what we found is that water
4 concentrations of PFOA in drinking water predicted serum
5 levels near the DuPont production facility there. And you
6 can actually use regression to estimate the increase in
7 serum concentrations per unit increase in water. And what
8 we found is that those were actually consistent with the
9 pharmacokinetic estimates, which is very nice.

10 And you can -- so you can -- I think you -- this
11 works pretty well and this has been done a number of
12 places now. So we know that at least in contaminated
13 areas, water can make a very significant contribution to
14 what we see -- what we biomonitor.

15 --o0o--

16 DR. WEBSTER: The next one I'd like to talk about
17 a little bit more is diet. Okay. So I think someone said
18 earlier today that diet is one of the major routes of
19 exposure. This is certainly what a lot of people say and
20 we can talk a little bit about the evidence basis for
21 that. There are several U.S. epidemiologic studies of the
22 kind I mentioned that found that diet significantly -- was
23 significantly associated with blood levels. So just two
24 of them. Just -- I picked these two, because I worked on
25 them and I know them in, you know, boring detail.

1 There was one that we did with blood samples back
2 in the late 90s and we found associations between various
3 PFASs and things like fish, so you've heard fish earlier
4 today. Shellfish, meat, poultry, these were all
5 associated with the PFAS plasma levels that we targeted.

6 Another study that we did a long time ago using
7 NHANES data, we found again in NHANES they use dietary
8 questionnaires. And red meat consumption was associated
9 with PFAS and PFOA -- PFOS and PFNA concentrations in
10 blood. And then when we looked at fast food, either total
11 calories from fast food or fast food items eaten per day,
12 that was associated with changes in PFOA level.

13 And so that actually begins to suggest that there
14 might be some differences between the different PFASs,
15 whether they're from a -- may -- possibly food packaging
16 or whether it's a bioaccumulation process. And it's
17 probably both going on with PFASs.

18 So there is actually, I would say, quite a number
19 of studies now that have used this sort of design and have
20 established that diet significant -- is significantly
21 associated with blood levels.

22 --o0o--

23 DR. WEBSTER: Now, the last one I'll mention a
24 little bit, and Kate I think is going to say a little bit
25 more about this, is sort of indoor exposure. This is much

1 less steady than diet or water, but there are now sort of
2 a small number of studies that, again using this
3 epidemiologic approach, found that serum concentrations or
4 plasma are associated with concentrations of the more
5 volatile types of PFASs found in air. So, for example,
6 fluorotelomer alcohols. Whereas, the levels in
7 concentrations in serum were not particularly associated
8 with dust concentrations from people's homes or very
9 weakly.

10 So as an example the kind of data that we
11 found -- changes in serum PFOS level in people's blood
12 compared to these FOSAs and FOSEs. These are so-called
13 pre-FOS, so they degrade into PFOS and they get in these
14 nice relationships. This is from pregnant women in
15 Vancouver.

16 And then again from the same study, we found that
17 levels of PFOA and PFNA in serum were related to tertiles
18 of one of the fluorotelomer alcohols, again another
19 precursor. I would say one of the things I was interested
20 in - I don't know if I was surprised or not - is that the
21 diPAPs, which were huge in the dust in these people's
22 homes, were not particularly related to levels in blood.
23 So that's very interesting.

24 --o0o--

25 DR. WEBSTER: And then finally, I think an even

1 less studied area has to do with personal care products.
2 And there was this very nice paper out of Stockholm
3 University a few years ago, and there's been a couple more
4 since, where they've looked at PFAS in say cosmetics. And
5 what they found was high levels of total fluorine,
6 somewhat less levels of extractable organic fluorine and
7 an even less identified PFAS.

8 And there's the potential for high dermal
9 exposure. I would say we know almost nothing about dermal
10 absorption of PFASs. There's just a handful of papers out
11 there on that. I think they're -- unless there's new
12 ones, they may all be about PFOA. So this is sort of a
13 big unknown. Potential important source, it might be
14 related to things like sex differences in some ways, but
15 we just don't know very much about it.

16 --o0o--

17 DR. WEBSTER: All right. So let me turn now to
18 relative contributions. So there was a nice paper again
19 out of Elsie Sunderland's group and they used really -- it
20 was clever. They used stored water in serum from the
21 Nurses Health Study dating back to the late 80s. And what
22 they found was that tap water PFOA and PFNA were
23 significantly predicted plasma levels among high consumers
24 of water.

25 And there's -- they were able to estimate using

1 pharmacokinetics that they got something like 12, 13
2 percent of what they measured in blood could possibly be
3 explained for by water exposure. And there's a few
4 more -- there was a few more data points in there. But
5 this is one of the -- one of the few studies I know of
6 that has used empirical data like an epidemiologic
7 approach to try to estimate this.

8 --o0o--

9 DR. WEBSTER: So, relative contribution estimates
10 other than that one I just spoke about. Almost all of
11 them use the relative -- the exposure factor approach. In
12 principle, you could do it either way, the epidemiologic
13 approach or the exposure factor approach. Most of them
14 have used the exposure factor approach. And those have
15 very substantial uncertainties, I would say particularly
16 the dietary ones, because it matters a lot where you
17 sample food and how you measure it. So the detection
18 limits are usually not very -- the levels in food are not
19 very good. It's hard to measure PFAS in food. And so it
20 starts to matter a lot how you treat non-detects.

21 You have to think about whether you're measuring
22 precursors or not, because we know precursors are very
23 important. You have to think about conversion rates of
24 precursors and to the stable things we see in blood.

25 And then if you start moving out of diet to other

1 things, if you want to look at indoor exposure, inhalation
2 rates we know pretty well, but dust ingestion rates are
3 really terrible for adults. We don't know that very well.
4 And as I mentioned before, dermal absorption really flux
5 through skin is very poorly understood for PFAS. And I
6 don't necessarily believe very many of the models that are
7 out, because PFAS are just sort of weird chemicals.

8 --o0o--

9 DR. WEBSTER: This is a table that is too busy.
10 It comes from the review paper that we put together
11 earlier this year. But there's a column here for percent
12 exposure via diet. I mean, basically the takeaway is that
13 diet is the main source. There are only two of these
14 studies that are U.S. And they're about, what, ten years
15 old now and they were from Matt Lorber --

16 --o0o--

17 DR. WEBSTER: -- if you remember him. And he
18 estimated in this -- these papers that PFOA and PFOS were
19 about, you know, 60 to 70 percent exposure was due to
20 diet. But again, I think -- I think these are very
21 uncertain. Diet is important, but I -- I'm not sure I
22 really believe these numbers. And they're old too and
23 things change over time.

24 --o0o--

25 DR. WEBSTER: Okay. So how can we sort of

1 summarize that? Well, I think we have empirical data, you
2 know, from the sort of epidemiologic approach that water,
3 diet, and indoor air all predict blood levels of some PFAS
4 in some populations. And it's going to vary by population
5 and personally within the population. Water is very
6 important in contaminated areas and there's some reason to
7 think based on old data that it accounts for something
8 like 10 to 20 percent, or something like that, in general
9 populations. That's actually important when you set water
10 quality standards that you need to have some factor to
11 account for how much exposure is coming from other stuff,
12 as I think Tom mentioned earlier.

13 Diet is generally thought to be the major route
14 of exposure in general populations, but I think that those
15 are very -- it's very uncertain and we don't really know
16 very much about diet. So this is underlined by the -- a
17 comparison of the recent studies that came out of the
18 European Food Safety Authority and just this last year a
19 study by FDA of PFAS and diet. And they kind of reach
20 very different conclusions. So I think we need a lot
21 of -- a lot more work on diet. And again, we know almost
22 nothing about personal care products at this point. So we
23 really need comprehensive exposure studies. And this is
24 going to require intensive sampling to really figure this
25 out.

1 --o0o--

2 DR. WEBSTER: A little bit more relevant now
3 towards what California Biomonitoring can do. I think
4 it's important to think about trend -- we were talking
5 about trends earlier and what they might imply. So vast
6 consumables like food packaging and cosmetics as PFAS --
7 as some PFAS get phased out of those, there should be
8 rapid changes in exposure.

9 There will be slow consumables like furniture and
10 carpet that may take a long time to work their way through
11 the system. Meanwhile, we have a -- we have global
12 distribution of persistent and mobile PFASs. So there's
13 kind of a worldwide background exposure that these signals
14 are on top of. And so I think that part of what that will
15 mean is that for diet is as a shift in food packaging, it
16 may imply that bioaccumulation routes will become more
17 important for some of the legacy PFAS.

18 And very important, and this was touched on
19 before, that when we start looking for trends in how they
20 respond to interventions, that external exposure can do --
21 may decline much faster than serum, because a lot of the
22 PFASs have long half-lives, so you have to build in the
23 right kind of lag structure.

24 So I think scientifically, we need to understand
25 a bunch of things like unidentified organofluorines. I'm

1 really interested in that. I think we really need to do
2 more work on dietary exposure, and indoor exposure, and
3 dermal exposure. I think we still don't have a very good
4 handle on real relative source contributions. I think we
5 know a lot of the important ones, but, you know, the
6 relative contributions I'm not so sure, and that we --
7 this is a rapidly changing world and we need to have
8 exposure studies addressing the changing production out
9 there.

10 --o0o--

11 DR. WEBSTER: Someone did ask me to say a little
12 bit about food, because it gets asked about a lot. It
13 seems to be, at least a combination -- or it was a
14 combination of bioaccumulation in food contact materials.
15 And so you can imagine things like in fish, the persistent
16 PFAS can accumulate in the fish or things can come from
17 farming.

18 Food processing we know almost nothing about.
19 I'm sure there's contamination of food during food
20 processing, but we -- you know, this is a huge hole. Food
21 contact materials, we do know a little bit about. There's
22 a little bit known about the effects of cooking and then
23 we have exposures.

24 So what exactly is going on with diet depends on
25 all this stuff and it probably depends on the food and the

1 type of PFAS. It's worth noting for the general audience
2 that teflon pans themselves are generally not considered
3 to be a major source. The problem with teflon is more
4 making teflon rather than using it, and again, that food
5 processing is not very well studied.

6 --o0o--

7 DR. WEBSTER: All right. I'd just like to end
8 with some thoughts on what I think a biomonitoring
9 surveillance program like that in California, which is
10 really good. What can they do regarding PFAS exposure?

11 Well, public health -- and a lot -- some of these
12 things have been touched on before. We can -- you know, I
13 think you can point out -- because you have location data,
14 you can potentially point out when exposure to water and
15 contaminated communities is connected to blood levels and
16 the science behind that is pretty well established at this
17 point.

18 You can try to monitor time trends, both up and
19 down, and evaluate interventions. Although, again, you
20 have to think about lags with the compounds with longer
21 half-lives. And you can certainly look at its
22 disparities. So Kathleen mentioned several of these.

23 In terms of research, I -- there is a -- there is
24 a good precedent for using surveillance methods like
25 NHANES to try to look at exposure. And I think with the

1 questionnaire data that you do have, you can look at some
2 of the non-water sources, such as consumption of certain
3 foods or use of carpet. I think you have questions about
4 carpet.

5 There are important limitations. I mean, you
6 know, dietary questionnaire data is notoriously hard,
7 particularly with time lags. And so you have to think
8 really hard about that, but remarkably, it does seem to
9 work sometime. And there are, of course, other problems
10 like about the precursor.

11 And then finally, I do think the chemometric
12 fingerprint approach is worth doing. It's not super
13 straightforward, but it is worth looking at.

14 --o0o--

15 DR. WEBSTER: So let me just end by I'd like to
16 acknowledge a bunch of -- a whole bunch of people I have
17 worked with, and again some of my current and former
18 students who have worked on PFAS and some in particular,
19 Kate Hoffman who you'll hear from in a minute.

20 All right. So thank you very much.

21 CHAIRPERSON SCHWARZMAN: Thank you, Tom. We
22 have -- I really appreciate that overview, and a summary
23 of the questions, and what's clear and what's not. We
24 have time now for -- we have about 15 minutes for
25 questions from both the Panel and the audience before Kate

1 Hoffman speaks next.

2 Any questions from Panelists for Tom?

3 Tom.

4 PANEL MEMBER MCKONE: Hi, Tom, very good. I
5 really enjoyed the presentation. Doing great work.

6 I guess I would -- don't -- you know, these
7 different methods for -- I mean, I think we're always
8 going to be limited on our understanding of some of the
9 complicated relationships. You know, just -- there's just
10 too many factors that come in. I mean, I think unless we
11 really can go into people's homes and observe like what
12 products they're using, what carpets they have, what they
13 spray on their carpets, what -- you know, it's just going
14 to be really hard to sort this out.

15 So I do think I'm kind of interested that you
16 suggested some methods that are sort of kind of inverse --
17 inverse modeling or principal components. And I don't
18 know if you could talk a little bit more, especially like
19 if we start getting some really good pharmacokinetic
20 models. And again, it would have to be -- I mean, the
21 class is not going to behave the same way. So we probably
22 would have to have it for individual specific chemicals.
23 But, you know, would that maybe help sort out some
24 hypothesis testing about food with some questionnaires
25 more than just sort of brute force questionnaires?

1 I mean, I think -- I think it's going to be
2 important to have a little bit better pharmacokinetics,
3 but I also would like -- would like your thoughts on that.

4 DR. WEBSTER: Yeah. I mean, I agree with you. I
5 think to really sort this out, we would have to do very
6 detailed sampling. And I don't of any way to fund that.
7 In our current world, you know, because we don't have an
8 exposure study section at NIH. So it's -- anyway. So,
9 yeah, I mean, the pharmacokinetics, you know, we could
10 have a long discussion about. I mean, what people
11 typically do is something very simple-minded, which is
12 assume first order pharmacokinetics and steady state,
13 which is wrong.

14 And then you have to estimate half-lives, which
15 we have pretty good estimates for, but we have to have
16 volume of distribution. That we don't know very well. We
17 have to usually extrapolate. And it depends a lot on
18 binding to albumin. It gets really com -- it gets
19 complicated, but they do work sort of okay for at least a
20 handful of the long-lived compounds.

21 So I think it's not a -- I mean, I think it's not
22 a bad approach. You know, I do like what Matt MacLeod and
23 company is doing, because they're actually looking at
24 nonlinearities over time and they're not assuming steady
25 state, which is really what you have to do, if you're

1 going to try to really nail those. So I appreciate what
2 he's doing there.

3 And again, I think it's -- is a first order
4 approach to trying to figure out the contribution of
5 exposures that you understand well like water. I think we
6 understand water pretty well. I think it's okay. Diet --
7 if we want to figure out diet, we're going to have to
8 invest a lot of money, because it's really -- it's really
9 hard and it's the -- seems to be the big one.

10 PANEL MEMBER MCKONE: All right. Thank you.

11 DR. WEBSTER: Yep.

12 CHAIRPERSON SCHWARZMAN: I have Jenny and then
13 José.

14 PANEL MEMBER QUINTANA: Hi. Jenny Quintana.
15 Thank you for a really great talk. I have kind of a naive
16 question, because I don't really follow nutritional
17 biomarkers very closely. But I was just kind of wondering
18 if one could look -- if you could comment or speculate
19 about if you had a -- you're looking at these compounds
20 and biological fluids, are there other compounds that
21 would indicate sources you could look in the same fluid?
22 And I'm just thinking, for example, if we suspected
23 tobacco smoke, if you looked at cotinine, you know, and if
24 other -- is there anything like fish oils you could look
25 at to indicate a fish source, but -- so I'm kind of

1 looking at it from within the sample itself.

2 DR. WEBSTER: Yeah. Hi. Yes, I think that -- I
3 think that's a really good idea. I don't know of anyone
4 who's done that. I mean, one of the differences would be
5 the sort of vast differences in time scale, right? I mean
6 PFASs have half-lives of years a lot of them and -- I
7 don't know, omega-3 fatty acids, I don't what it is, but
8 it can't be very long. So we have to think hard -- and
9 then you get into all those problems if you do one sample,
10 you know, how much do you believe one sample and short
11 half-lives.

12 But I think it's a good idea and it actually --
13 it reminds me of, you know, like the work that was done
14 that the Biomonitoring people here mentioned of looking at
15 mercury as a marker for exposure to seafood. So I
16 actually think that's a great idea. I don't really know
17 what they would be. I'm not a, you know, nutritional
18 epidemiologist, but it's a good -- it's a good idea. I
19 hope someone looks at that some more.

20 PANEL MEMBER QUINTANA: Thank you.

21 CHAIRPERSON SCHWARZMAN: José.

22 PANEL MEMBER SUÁREZ: Yeah. Hi, Tom. Thanks for
23 the presentation. Good to see you. And just a quick -- a
24 quick question. How much should we be concerned about or
25 how much is known about cross-contamination of samples, so

1 contamination of PFAS during the sample collection or
2 storage, given the ubiquitous presence of a lot of these
3 compounds? Do you know much about that. I know that
4 there are a lot of certain recommendations. Some people
5 are saying, well, avoid storing -- or contacting glass
6 containers, because PFAS -- many of them can attach to
7 glass very readily, or avoid, I don't know, low-density
8 polyethylene to store it, because there could be some PFAS
9 in there if you have not tested for that. Do you have any
10 comment on that?

11 DR. WEBSTER: Well, I'm not an analytical
12 chemist, so I let my chemistry friends worry about this.
13 But I certainly look at the blanks. I mean we always try
14 to do blanks and when we get high blanks, then you get
15 really worried, right? So I think that -- my feeling, you
16 know -- and maybe Oliver can chip in here, is my feeling
17 is that with the traditional PFASs, it's not that bad.
18 But with combustion ion chromatography, you have to work
19 really hard, because there's fluorine everywhere, right?
20 And so you have to actually work really hard and that's
21 one of the reasons they have high blanks and, you know, so
22 they need more sample in order to get detection levels and
23 all that sort of stuff, so it's a -- it's a -- it's a big
24 deal.

25 Again, my impression for the traditional PFASs is

1 that it's not as big a problem as say it was with PCBs
2 when people used to worry about, you know, PCBs coming out
3 of the -- out of the fluorescent lights and, you know, all
4 that kind stuff, but anyway.

5 PANEL MEMBER SUÁREZ: Thank you.

6 DR. WEBSTER: Our blanks always seem to come back
7 pretty good for regular PFASs?

8 CHAIRPERSON SCHWARZMAN: I want to ask one of the
9 questions that's in the Q&A function, and then Kathleen,
10 and Veena, and then I have another question in the Q&A.

11 So from Simona Balan, "Great presentation. Thank
12 you. Do you have any recommendations for how to assess
13 the impact on human exposure of phasing out PFAS from
14 carpets? How would you tease that apart from other
15 changes in PFAS use or exposure sources"? Not unlike
16 Jenny's question about, you know, how can we mark the
17 sources based on other co-exposures or speciation or what?

18 DR. WEBSTER: Yeah. No, carpet is a good one.
19 And it's com -- again, I, you know, really try to figure
20 out -- you could imagine doing intervention studies, where
21 you replace carpet and you look at people over time or
22 something like that, right?

23 I mean, I -- I'm very interested in trying to
24 trace back the compounds we find in the indoor environment
25 like in the air or in dust to their actual sources in the

1 home. And it's -- that's actually hard. There's not been
2 a lot of work done on that. And I suspect that carpets
3 have something to do with it.

4 And then there may be complicated things going
5 on. It may not just be a release of PFAS from the carpet
6 that's just attached to it, but it could be actual
7 abrasion mechanisms and all sorts of things. And it's
8 kind of not very well understood. But I actually think
9 indoor exposure is probably going to turn out to be quite
10 important for some groups of people.

11 There's a -- there's a famous exam -- God,
12 there's a famous sort of case study that came out of
13 Canada where there was a family that was using tons of
14 Scotchgard or something, directing -- like it -- I don't
15 quite maybe remember the details, but they were basically
16 treating their furniture or their carpet all the time and
17 they had sky high levels of the hexanesulfonate, as I
18 recall. So there are clearly going to be cases where this
19 turns out to be true. And if we can change that product
20 formulation, it ought to make a difference.

21 CHAIRPERSON SCHWARZMAN: We just have a few
22 minutes until our next talk, so I'm going to call on
23 Kathleen, who's been waiting, and then I'm going to just
24 for the folks who have written something in Q&A, know that
25 we'll hang on to that and bring that out in the next

1 question session -- question section.

2 So Kathleen and then Veena.

3 DR. ATTFIELD: I could defer mine, because it's
4 related to what the anonymous attendee has asked, back to
5 assessing sort of PFAS profiles. And I did want to say to
6 Jenny and to the others I really love the idea of working
7 across panels to give indicators. Of course, I presented
8 that on blood mercury, but, you know, we do have a phenols
9 panel. So we could sort of -- of course, that's a
10 possibly different exposure window and shorter half-lives,
11 but also there's cadmium for smoking and within metals.
12 So there -- it's going to be interesting to think about
13 triangulating from different panels.

14 CHAIRPERSON SCHWARZMAN: Veena.

15 PANEL MEMBER SINGLA: Thank you. Thank you for
16 that presentation Tom. It was really informative.

17 I wonder like kind of thinking about reducing or
18 preventing PFAS exposures, what do you think are some of
19 the most important data gaps or questions, if we're -- if
20 we're trying to think about policy approaches to reducing
21 or preventing exposures?

22 DR. WEBSTER: Well, I tried to lay that out --
23 them out on that one slide. I mean, I really want to know
24 what this unexplained organic fluorine is. I think that's
25 actually one of the most important questions. And, I

1 mean, I'm working on it and I know a couple other people
2 are working on it, but I really want to know. Because if
3 it turns out that its other PFASs that we just don't have
4 standards for, that's a really big deal. If it turns out
5 it's mostly drugs, that's different. So they're very,
6 very different implications of the answer to that question
7 or if it's pesticides or something.

8 I think that the understanding diet is really
9 important. If it really is one of the major reasons --
10 routes of exposure, we need to know what's going on there.
11 And I mean in the meanwhile, we can do important things,
12 like getting PFAS out of food packaging it seems like.
13 It's great that's being done, because that might actually
14 turn out to make a big deal. We just don't know yet and
15 it's something we should be looking for, but I think that
16 that's very important.

17 Now, if it's bioaccumulation, then we're in big
18 trouble, right, because this stuff is everywhere out there
19 in the world. So it will mean that we have to wait for
20 steps to come down in the environment before we can fix
21 that. And that will -- that will -- that's going to take
22 a while.

23 So we should go after -- I think we should go
24 after the ones that are easy and that should respond
25 quickly, you know.

1 CHAIRPERSON SCHWARZMAN: Thank you for that, Tom.

2 We will go on now to our next speaker. I want to
3 introduce Kate Hoffman, who is an Assistant Research
4 Professor at the School of Environmental Sciences and
5 Policy at the Nicholas School of Environment at Duke
6 University.

7 Kate holds a PhD from the Boston University
8 School of Public Health. Her research focuses on
9 assessment of human exposure to PFAS, flame retardants,
10 and other chemicals used in consumer products, as well as
11 the health impacts of exposure to those chemicals. And
12 she will discuss the relevance for human exposure of PFAS
13 in indoor environments and drinking water.

14 (Thereupon a slide presentation.)

15 DR. HOFFMAN: Wonderful. Thank you so much. I
16 am, in fact, one of those former students of Tom's. Let
17 me just get my screen shared here real quickly.

18 Okay. Great. Can everybody see?

19 CHAIRPERSON SCHWARZMAN: Yes.

20 DR. HOFFMAN: I'll assume that's a yes that you
21 can see and hear me. But if you can't, somebody please
22 let me know.

23 So again, yeah, thanks everybody for the
24 invitation to be here today. I am really excited to be
25 joining you and to talk more about these indoor

1 environmental exposures and also exposures through
2 drinking water.

3 Tom, gave a really nice introduction to that.
4 And he is a tough act to follow, but I'm going to try. So
5 to give you kind of an idea of what I'm going to talk
6 about, I'm going to first talk about the CDC and ATSDR
7 multi-site study, which is really geared towards
8 understanding PFAS exposures through drinking water and
9 their potential impacts on human health. And I'll talk a
10 little bit about the multi-site study, specifically in
11 California at UCI.

12 And then I'll also talk a little bit about some
13 work that I've been involved in looking at exposures to
14 PFAS and the indoor environment.

15 --o0o--

16 DR. HOFFMAN: But before I do that, I just want
17 to acknowledge that I have no financial disclosures or
18 relevant conflicts of interest with the materials included
19 in this presentation. I will be discussing the CDC and
20 ATSD multi-site study and the work of colleagues related
21 to that study. The views expressed are completely my own.
22 I'm not currently involved in any CDC ATSDR multi-site
23 study projects.

24 --o0o--

25 DR. HOFFMAN: Okay. So as Tom mentioned, we do

1 think that drinking water is main source of exposure to
2 PFAS. And the reason for that is just mainly that PFAS
3 are highly soluble, they're detected in many drinking
4 water supplies across the country. And much of what we
5 know about that exposure comes from the C8 health project
6 study, which is based in Parkersburg, West Virginia and
7 the surrounding communities.

8 And that study looked at exposure primarily to
9 PFOA and PFOS, but there's sort of this desire to know
10 more about other PFAS compounds. And that was a really
11 large study. It's kind of limited in the scope of
12 compounds that we know things about from that work. And
13 so to that end, the CDC and ATSDR started this multi-site
14 study project, with the goal of looking at sites kind of
15 across the United States that would have different PFAS
16 exposure profiles and comparing exposure at those sites in
17 its relation to different health outcomes.

18 The health parameters of interest include things
19 like immune response of the metabolism, kidney function,
20 thyroid disease, liver disease, glycemic parameters, and
21 diabetes. And these are all kind of non-cancer health
22 endpoints.

23 And I'll just say that I think there is some
24 interest in also trying to understand cancer health
25 endpoints, also reproductive health endpoints, but keeping

1 in mind that even though the multi-site study will be
2 large and only about 10,000 participants, it still will be
3 hard to study some of those rare health outcomes or those
4 that take a long time to develop in this population. So
5 that's sort of one kind of limitation. I think that's
6 data they hope they'll get, but we'll see how that
7 develops.

8 So it is a five-year study. And the sites in the
9 multi site-study were announced in late 2019, which as all
10 of you can imagine was a difficult time to start a large
11 nationwide kind of epidemiologic study. So there's some
12 challenges with that, that I'll talk about in a few
13 minutes.

14 But I do think it's important to note that there
15 are sort of seven multi-site study sites, but the study
16 framework really comes from the Pease study, which is in
17 Portsmouth, New Hampshire. And that study it is sort of
18 based on the Pease International Tradesport, which had
19 PFOA contamination of drinking water there. And so they
20 started this study there, which outlined sort of the
21 protocol for the other multi-site study sites.

22 That study has enrolled about 700 people. They
23 started enrolling about the same time as the other
24 multi-site studies were announced. So they've made good
25 progress over the last couple years. And I'll just note

1 here that there's a link to the multi-site study website
2 on the bottom of my slide. So if you're interested in
3 knowing more about the sites, you can look there.

4 --o0o--

5 DR. HOFFMAN: So there are sites of the
6 multi-site study all across the United States. The one
7 thing that all of these sites have in common is a
8 documented history of PFAS contamination of their drinking
9 water, but the source of that contamination is different
10 across sites. So some of them have drinking water
11 contamination from past military or AFFF firefighting foam
12 applications near those sites. Others are related to
13 industrial activities. And really importantly for your
14 discussion today in Orange County, California, you have a
15 multi-site study site right there in your home state.

16 --o0o--

17 DR. HOFFMAN: And so at each one of these sites,
18 they hope to recruit a thousand adults, and 300 children.
19 So all told, if you include Pease in that, you'll get
20 about 10,000 people enrolled in the study. As I
21 mentioned, this did start at the end of 2019, which was a
22 hard time to kind of get a bunch of in-person visits and
23 study enrollments done. So these sites I looked this week
24 at their websites to kind of see where everybody was in
25 terms of enrollment.

1 sort of highlight is, you know, while the C8 health study
2 had kind of a lot of people with one sort of high PFOA, a
3 little bit of PFOS kind of exposure profile, or at least
4 we think that's what it had, these sites all have kind of
5 different PFOS exposure fingerprints. So there's some
6 variability in exposure across sites.

7 --o0o--

8 DR. HOFFMAN: I won't go into the details here
9 too much just to save time about what exactly is being
10 measured. There's a lot of information on questionnaires,
11 including residential history and water consumption
12 information. And I'll just say that the water consumption
13 information is more detailed than when it was collected in
14 the C8 health study. There's information -- a bunch of
15 health information that's being collected that's also
16 being validated with medical records and also medication
17 lists.

18 And then really importantly for the study, there
19 are fasting blood and urine samples that are shipped
20 for -- to the CDC for analysis of PFAS, as well as
21 biomarkers for some of these health endpoints. So those
22 are all being analyzed in the same lab. If you're
23 interested in more detail about what exactly is being
24 included in that protocol, you can click on the link there
25 and go and see all of the detailed measurements and how

1 they're collecting those in the study.

2 --o0o--

3 DR. HOFFMAN: It is sort of a limited number of
4 PFAS under consideration still. Okay. So now I'll say a
5 little bit about the PFAS health study at UCI. So this is
6 headed by Dr. Scott Bartell and Dr. Russ Detwiler.
7 Probably most of you are much more familiar with
8 California geography than I am, but this study is based in
9 Orange County, which is in Southern California. And it's
10 primarily in the Orange County Water District, which is
11 outlined here in this blue color.

12 Over 500,000 people are served by water systems
13 within 10 miles of the University of California, Irvine
14 Medical Center. And in that UCMR3, which was conducted in
15 2013 and 2015, all of these water systems had at least one
16 exceedance of that 70 parts per trillion for PFOS and
17 PFOA. So there is a documented history of PFAS
18 contamination of the drinking water there.

19 And how this site compares to other multi-site
20 studies, in general, it's more diverse than other sites.
21 About 50 percent of children in this area speak a language
22 other than English at home. And then one thing I would
23 just point out is that the source of exposure is a little
24 bit different or at least the presumed source of PFAS
25 contamination in drinking water in this area is a little

1 bit different than at some of the other sites.

2 --o0o--

3 DR. HOFFMAN: So what else makes this a good
4 site? And one thing about that is that in California you
5 have a lot of information about drinking water that's
6 super valuable for a study like this. So this is a
7 picture of the -- it's a cross section of Orange County's
8 groundwater basin. And so because you have a growing
9 population and years of a current drought, there's really
10 good management by local water utilities of source water
11 and water consumption.

12 So local water utilities use seasonally varying
13 combinations of groundwater and surface water, as well as
14 imported water to meet that demand. And there's a lot of
15 really good data on that. And the groundwater supply is
16 really carefully managed to that end.

17 One important thing that I'll just point out here
18 is that the source of the PFAS in this drinking water
19 supply is thought to be through wastewater treatment
20 plants, so the reuse of that water. And I think that's an
21 important difference between some of these other
22 communities where we think that, you know, the main source
23 of PFAS in their drinking water is through AFFF or other
24 military activities.

25 --o0o--

1 DR. HOFFMAN: Okay. And so one thing about PFAS
2 in the Orange County Water District, and I think a lot of
3 other water districts particularly in California, is that,
4 you know, once PFAS were detected in that 2013, 2015 UCMR
5 report, those wells with the highest concentrations were
6 taken offline. And so since that time, there's been sort
7 of additional well monitoring and sort of changes in which
8 wells were used at different times, including 38 wells
9 being taken offline in July of 2020, in response to new
10 stricter State health guidelines to sort of reduce the
11 levels of PFAS in finished drinking water that people were
12 receiving.

13 And I'll also just note that at great cost,
14 Orange County is also currently testing advanced treatment
15 systems for their drinking water to remove PFAS.

16 So I think if you kind of take this all together,
17 and take this picture all together, it's very likely that
18 exposure to PFAS in Orange County has probably decreased
19 substantially since 2013 and 2015. And that's something
20 that the study will kind of work through and help identify
21 that.

22 And I think this is probably a story that's going
23 to be true in a lot of -- a lot of other company -- or a
24 lot of other communities as well, particularly with the
25 new EPA roadmap.

1 --o0o--

2 DR. HOFFMAN: So as we see these impacted
3 communities with decreasing PFAS in their drinking water,
4 it's possible that they'll become more and more like sort
5 of general population exposures, and that other sources of
6 exposure will become more significant contributors to
7 overall exposure.

8 --o0o--

9 DR. HOFFMAN: And one source of exposure that I'm
10 very interested in are -- is exposure in the indoor
11 environment. And so why I am exposure -- interested in
12 exposure in the indoor environment? And the reason for
13 that is sort of twofold. One, the average American spends
14 like 90 percent of their time indoors, maybe even more
15 than that over the last couple years. And so if you don't
16 have a whole lot of PFAS in your drinking water, indoor
17 exposures may be more important in your overall level of
18 exposure.

19 But we also know that PFAS are used in a bunch of
20 products in our home, right? Like they may be used in
21 textiles, or carpets, or furniture products, maybe to a
22 lesser extent in California, but because these are sort of
23 slow replaceable products in our homes, we would expect
24 them to kind of stick around for a period of time.

25 They may also be used in other applications like

1 paints, or personal care products, or clothing, So we're
2 going to expect to detect these in our homes for a long
3 period of time. And because they're used in so many
4 products, we're going to expect that they're going to be
5 found commonly in indoor dust and indoor air, presenting a
6 possible source of human exposure.

7 --o0o--

8 DR. HOFFMAN: And, you know, Tom actually talked
9 about this in his presentation as well, just highlighting
10 that this figure shows past research on different
11 environmental media and their importance as pathways of
12 exposure to PFAS. And you can see here that between dust
13 and diet, those have been considered in about 70 percent
14 of past studies, but only 11 percent of studies have
15 considered indoor exposures through indoor air or dust,
16 and what exposure through those pathways may look like.

17 So while these pathways may be really important
18 sources of exposure for the general population, we know
19 much less about them.

20 --o0o--

21 DR. HOFFMAN: And so I'm going to talk about sort
22 of one study, where we've been looking at indoor exposure.
23 There are certainly other groups that have been doing
24 this. And I'll try to highlight some examples from those
25 groups as we go through. For the sake of time, I'm going

1 to talk about this one study, even though it has some
2 limitations for some of the applications we'll talk about.

3 But I do just want to mention briefly that this
4 study is a collaboration with Heather Stapleton and Tom
5 Webster, as well as their students, and post-docs, that
6 we've been working on for several years. I want to
7 acknowledge that effort.

8 We call this study the Toddlers Exposure to
9 Semi-Volatile Organic Compounds and Indoor Environment
10 Study, TESIE. And we've been working on this since 2014.
11 But we visited kids' homes between 2014 and 2016. We did
12 200 home visits in Central North Carolina during that
13 time.

14 And we had kids in the study provide a blood
15 sample during home visits and they also provided a bunch
16 of different samples, which you can see here. The numbers
17 on these different samples are a little bit different.
18 And that's, you know, just due to different challenges
19 with getting different samples from kids. I'll say a
20 little bit more about each sample type when I talk more
21 about them.

22 We measured PFAS in indoor dust and air. And
23 when I say PFAS here, I'm really kind of, like Tom, using
24 a pretty narrow definition. So I'm talking about those
25 legacy compounds, and also, to some extent, some of the

1 precursor compounds, like the FTOHs or the pre-FAS
2 compounds. But I'm using that as a pretty narrow
3 definition here to just refer to the compounds that we
4 measured. The serum samples we sent to the CDC and they
5 were analyzed for that kind of standard NHANES type panel.

6 And one really important thing to note about this
7 cohort is it's -- it is Central North Carolina. And if
8 any of you know about our drinking water here, North
9 Carolina does have some PFAS and water concerns of our
10 own. But this is the -- primarily our participants came
11 from Durham, North Carolina and using municipal water
12 here, which has generally fairly low levels of PFAS
13 contamination. There's a little bit of PFOA detected, but
14 in general, the levels are quite low. We've sampled it
15 several times and that's been a pretty consistent finding
16 over the last few years.

17 --o0o--

18 DR. HOFFMAN: Okay. So, you know, one thing I'll
19 note is that we call this a toddler's study, but by the
20 time we made it out to do these home visits, the kids were
21 a little bit older. As you can see, this is one of our
22 participants here. The participants were about four and a
23 half years old by the time we visited them in the study.
24 They were about 40 percent non-Hispanic white, 40 percent
25 non-Hispanic Black, and about 20 percent Hispanic, and

1 than the legacy PFAS, which are shown here in blue. And
2 FTOHs and diPAPs were both detected at medians above 100
3 nanograms per gram in dust or higher.

4 And I just want to show that slightly
5 differently, because I think it's kind of hard with a log
6 scale here. You can get a little -- a little bit lost in
7 that data. But if you look on a -- and this is not like a
8 non-targeted analysis or anything like that. If you look
9 at just the percentage of the targeted analytes that we
10 measured that were each of these particular compounds, the
11 6:2 and the 8:2 FTOH made up greater than 90 percent of
12 the total mass of the targeted PFAS that we measured in
13 dust. So these two compounds contributed quite a lot to
14 the total PFAS burden of dust that we measured.

15 --o0o--

16 DR. HOFFMAN: Okay. And I'll just kind of
17 mention some good news in this story. Sam went out and
18 got -- she collected the medians from six other studies
19 who had data for PFOA, PFOS, and PFHxS in U.S. house dust
20 samples and plotted those over time. And that's what is
21 shown here. You can see that for those compounds, it
22 really seems like there's some real significant
23 increases -- or decreases over time in the medians in
24 those house dust samples, particularly just noting that
25 this is plotted on a log scale. It just seems like it's a

1 really substantial decrease from over the last maybe 20
2 years.

3 One kind of important caveat about that and one
4 thing to think about is that while these three compounds
5 are decreasing, we don't have good data on some of the
6 replacement compounds, so it's possible there are
7 concurrent increases in other PFAS compounds in house
8 dust. And similarly, we don't have a lot of information
9 on some of the precursor compounds over time. So some
10 good news here, but potentially some bad news too, if we
11 had more data to look at that.

12 --o0o--

13 DR. HOFFMAN: And now I'll talk just a little bit
14 about the air data from those same homes. This work was
15 led by Jessica Craig, who's a former doctoral student of
16 Tom's, but she defended recently. We deployed these air
17 samplers. It's a passive air sampler, one of them shown
18 here, and each one had sorbent-impregnated polyurethane
19 foam disks. There's a little -- a little foam piece on
20 here that collected PFAS in the home. These were deployed
21 in participants' homes for about three weeks.

22 And kind of the main thing to note here is that
23 like the dust, we detected these more volatile precursor
24 compounds, really frequently, in participants' homes.
25 Again, the FTOHs were the most frequently detected. We

1 also detected these pre-FOS, ethyl-FOSE and methyl-FOSE in
2 most of the homes that we sampled.

3 --o0o--

4 DR. HOFFMAN: Okay. And so now the big question,
5 right? So were any of these things that we measured in
6 air or dust related to children's serum concentrations?
7 And that's something that Jessica spent a lot of time
8 looking at in her dissertation work. And one thing I'll
9 just point out is that, you know, we didn't have huge
10 numbers of overlap between all of these sample types, as I
11 mentioned, because we had some fewer blood samples or some
12 fewer air samples.

13 But even with that limited sample size, she saw
14 really strong associations between some of these
15 pre-cursor compounds in air and PFAS in children's serum
16 samples. So just like in the paper that Tom showed with
17 the women in Vancouver, we saw this association between
18 this pre-FOS compound methyl-FOSE in air and PFOS in
19 serum, which kind of makes sense on that potential
20 breakdown pathway.

21 All I know this is three dots here, but this is
22 just the linear and branch PFOS as well as the sum total
23 of both of those. So again, this is consistent with the
24 results that Tom showed from that Vancouver cohort as
25 well.

1 Similar associations have also been reported for
2 the FTOHs. We didn't see that as strongly here with air,
3 but that was certainly reported in the previous cohort Tom
4 mentioned, but also in a cohort of office workers in
5 Boston that Tom was involved in as well.

6 Importantly, there were no strong associations
7 with dust in this cohort, so it really does look like that
8 kind of airborne pathway may be more important in terms of
9 tracking exposure.

10 --o0o--

11 DR. HOFFMAN: And then one thing I'll talk about
12 because I just can't hold myself back from talking about
13 wristbands at any opportunity. I know some of you may
14 work with silicone wristbands as well. We use these as
15 kind of an alternative sampling tool to address some of
16 the limitations with collecting samples in the home
17 environment.

18 As you can imagine, if you collect a sample in
19 someone's home, that's really helpful, but it doesn't
20 capture every indoor environment that they visit, because
21 you may go to your office, or go to school, or leave your
22 home to go some other places, so we asked kids to wear
23 these wristbands. And the idea behind their use is that
24 they kind of sorb compounds from their ambient environment
25 while they're being worn.

1 So we asked kids to wear those for seven days.
2 And we saw really similar patterns of association between
3 the compounds detected on wristbands and PFOS in serum for
4 example. So we saw kind of similar patterns. I won't go
5 into a lot detail just for the sake of time, but this
6 suggests that these wristbands may be a useful tool in
7 monitoring indoor environmental exposures.

8 --o0o--

9 DR. HOFFMAN: Okay. Now, I'm going to talk about
10 just some challenges in investigating and regulating
11 indoor PFAS exposure and I think Tom touched on some of
12 these as well.

13 One is just that sampling the indoor environment
14 is much less standardized than biomonitoring approaches.
15 So we can agree on how to collect a blood sample, I think.
16 But when it comes to how to collect a vacuum cleaner
17 sample, it's like all bets are off. You know, the -- do
18 you collect someone's vacuum cleaner dust bag or do you
19 vacuum it yourself? Do you vacuum the living room? Do
20 you vacuum the bedroom? Where do you vacuum? How big a
21 spot do you vacuum?

22 And so there's a -- there's a lot of challenges
23 with that. And I certainly don't mean to say that the
24 biomonitoring piece of that is easy, but there's just sort
25 of these different batch of challenges. We still have to

1 DR. HOFFMAN: So just to summarize sort of the
2 main points from my talk today. The multi-site study will
3 provide important information on PFAS exposure in
4 communities with varying levels of contamination across
5 the United States in sort of this veering fingerprint of
6 PFAS exposure.

7 As Tom said, and I think, you know, I hope this
8 will show as well, when water PFAS concentrations are
9 known, pharmacokinetic modeling can provide pretty good
10 information about the contribution of that exposure to
11 overall PFAS exposure. That's true for some compounds,
12 but not all. You know, there are some that we don't have
13 good pharmacokinetic assumptions for yet, and so that's
14 kind of a challenge.

15 Exposures in Orange County may become more like
16 general population exposures over time and other sources
17 may become more important, in terms of how they contribute
18 to that overall cumulative exposure.

19 And at the same time while investigations of
20 indoor exposure pathways remain limited, extensions of the
21 multi-site study could be really helpful in addressing
22 those indoor exposure pathways, or just other pathways
23 outside of drinking water in general.

24 --o0o--

25 DR. HOFFMAN: So I want to just briefly

1 acknowledge folks who have been involved in this work, but
2 in particular, I want to acknowledge Heather Stapleton and
3 Tom Webster who have both been involved in the TESIE study
4 as well as many of the other studies that I mentioned in
5 this project, and also the UCI PFAS health study team,
6 including Scott Bartell, Russ Detwiler and Veronica
7 Vieira.

8 And with that, I know I'm standing between you
9 and the break, but I'm happy to take any questions.

10 CHAIRPERSON SCHWARZMAN: Thank you so much,
11 Heather -- you just said Heather Stapleton -- Kate who's
12 been presenting. My apologies.

13 We have a couple of questions that are sort of
14 teed up in the chat. And I think these will overlap with
15 your work and I'd also invite Tom to chime in if it's --
16 if that's helpful.

17 One is, "There are several sort of"... , in
18 quotations, "'...epidemiological' studies showing much
19 higher levels of some PFAS in infant blood relative to
20 that of mothers, for example PFOA. Could you please
21 comment on early life exposures and its importance in PFAS
22 risk management"? And that wasn't something that was
23 directly in your presentation or even necessarily in
24 Tom's, but I think you folks are well aware.

25 DR. HOFFMAN: Sure. So I can chime in on that a

1 little bit, in terms of -- so we actually, in the study
2 that I talked about, had measurements of maternal serum,
3 as well from this cohort, because it's a spin-off of a
4 pregnancy study. And in that study, you know, even --
5 these kids were like four and a half years old and we
6 still found associations with maternal serum for some of
7 them.

8 And so, you know, clearly it's something that's
9 still important, in terms of predicting their overall
10 exposure. So certainly it's something to think about. I
11 don't know if -- I mean, I'm happy to have Tom weigh in on
12 that as well. I don't know.

13 CHAIRPERSON SCHWARZMAN: I'm sure that's the open
14 question about is that from prenatal exposure or because
15 of shared environment?

16 DR. HOFFMAN: Yeah. I mean, certainly it's hard
17 to know. I mean, I think that's, you know -- and I don't
18 think -- you know, this cohort that we have we're
19 obvious -- it's not an ideal source to look at that, just
20 because we have some people who moved, some people who
21 didn't, so, you know, I don't know. But, yeah, I mean, I
22 guess it could be both.

23 DR. WEBSTER: I think it's clearly both. I mean,
24 we know PFAS crosses the placenta. We know it's in breast
25 milk. Little kids are down on the floor sticking things

1 in their mouth. So, you know, I think it's going to --
2 and they're living in the same environment. So I think
3 it's going to be very hard to disentangle actually, I
4 would imagine.

5 CHAIRPERSON SCHWARZMAN: The next question is one
6 that was being -- that Kathleen was wanting to echo.
7 "From an epi perspective, how do you try to examine
8 exposure sources of one PFAS congener to another given
9 that they're so correlated"?

10 DR. HOFFMAN: Yeah. You know, and it's really
11 tough. I mean, even in the -- you know, even in our data,
12 we see some things at times that don't necessarily make
13 perfect sense, right, because we see some of these
14 associations with compounds that are like in the -- even
15 in those precursor pathways, sometimes those breakdown
16 pathways are not compounds that we know break down into
17 each other. And I think part of that is because you get
18 this pattern of people who are using similar products or
19 people who are having similar types of things in their
20 homes.

21 So I think you're going to have that problem of
22 some of those exposures tracking together, and I think
23 that's going to be true of a lot of things. So we
24 actually see correlations between a lot of these compounds
25 in dust and serum that don't necessarily follow a pattern

1 you would think. Like, some of these PFAS compounds are
2 actually a little bit correlated with things like
3 phthalates. Now, why is that? It's not because of the
4 common use, I don't think, but, you know, is it because
5 you have more consumption of goods in your home. So I
6 think there will be some things like that. And it is hard
7 to tease that apart and identify exactly what products
8 that's coming from.

9 So I don't know if, Tom, you want to weigh in on
10 that at this point.

11 DR. WEBSTER: Yeah. No. I mean, I think this is
12 one of the challenges of doing an epidemiologic approach
13 is that you actually have to think like an epidemiologist.
14 You have to think about confounding. All right. I mean,
15 the air and diet pathways are going to be, to some
16 degree -- sorry, air, and inhalation, and dust pathways
17 are going to be partly correlated with each other. And
18 then things like socioeconomic position can influence
19 purchasing of what's in your home and also diet, right?
20 So all those things you have to actually think about that
21 with that. And -- but epidemiologists, we sort of know
22 how to do that, so it's possible.

23 CHAIRPERSON SCHWARZMAN: There's a brief question
24 here about in -- with respect to Kate's note about PFAS
25 being removed from municipal water. And the question is,

1 "Is it treated in any way or is it just treated as waste"?
2 That is, what happens with PFAS removed from municipal
3 water?

4 DR. HOFFMAN: I think that's going to depend
5 totally on where you are and how it's being managed. So I
6 think one strategy that's being used in California is when
7 it's found in a well, it's stop using that particular
8 well. So we're not -- we're not pulling it from that
9 source at the time, so we're going to other source uses.

10 You know, and then I think there are varying
11 degrees of success with using other products to remove
12 PFAS from water. We did a study a couple years ago
13 looking at the -- like how successful different home-based
14 filtration methods of removing PFAS from drinking water
15 were. And it's pretty variable honestly.

16 And so, you know, obviously, if you're just
17 pulling that out with your home Brita, you're just
18 throwing that filter away or recycling that filter after
19 you're using it. So, I mean, I think that's going to be
20 super variable depending on what water district you're in,
21 how they're actually managing that, if it's just a purely
22 we're just going to dilute it with some water that we
23 think doesn't have PFAS or what kind of treatment that
24 they're using, you know, is it an activated carbon system,
25 or what exactly is being done.

1 CHAIRPERSON SCHWARZMAN: Two more questions, if
2 you don't mind continuing the rapid fire. From Aaron
3 Maruzzo, "Nice presentation, Kate. I have a couple of
4 questions. One, were U.S. territories considered when
5 selecting impacted communities for the multi-State
6 study -- multi-site study? And more generally, can you
7 talk about how sites were selected"? This second question
8 is, "What are the detection limits for dust in air samples
9 -- samplers? And the third is, "Have the concentration
10 data been disaggregated by race and ethnicity"? And I can
11 repeat those questions for you if you need them as we go
12 along.

13 DR. HOFFMAN: So we'll start at the beginning and
14 I'll say that I -- you know, I didn't participate in the
15 review panel or anything like that for the multi-site
16 study, so I don't know exactly how sites were selected for
17 that. You know, I mean, the criteria were certainly a
18 documented prior PFAS exposure. I am certain that those
19 were limited to U.S. sites. I don't know what territories
20 would have been included in that. So the first one is an
21 easy, I don't -- I don't exactly know.

22 Let's see, you asked about detection limits, was
23 that the next one?

24 CHAIRPERSON SCHWARZMAN: Detection limits for
25 dust and air samplers.

1 DR. HOFFMAN: Okay. So they're going to be
2 variable across the compounds that I mentioned. And
3 they're certainly published in the papers that are linked
4 with those. So I don't have them offhand. You know,
5 they're not -- they're not particularly unusual. They're
6 comparable with sort of other studies in the literature on
7 that. I'm happy to follow up on that or you can look to
8 those published papers on them.

9 CHAIRPERSON SCHWARZMAN: And the third is have
10 the concentration data been disaggregated by race or
11 ethnicity?

12 DR. HOFFMAN: Okay. So let's see, in this cohort
13 in general -- so I'm assuming you're asking if there are
14 sort of differences by race and ethnicity in terms of
15 these indoor concentrations?

16 CHAIRPERSON SCHWARZMAN: It's not my questions,
17 so I can't answer, but I assume so. That's how I would
18 interpret it, so...

19 DR. HOFFMAN: Okay. Yeah. So, you know, it's
20 interesting. We saw some -- so we have looked at that in
21 terms of biomarkers in this cohort. We did not look at it
22 in terms of the indoor exposures to the same extent. And
23 part of the reason that we didn't do that is because we
24 don't have as many samples. So, you know, we had 50 air
25 samples. And so, you know, when we start breaking that up

1 by group, we're just a little bit limited in our ability
2 to do that.

3 But I'll talk about the biomarkers for a second
4 with that. So we did see higher levels of those
5 biomarkers in general, PFAS biomarkers, in our
6 non-Hispanic white participants compared to the
7 non-Hispanic Black and Hispanic participants in this
8 cohort. And that's sort of an opposite trend for other
9 semi-volatile compounds that we measured in this
10 population.

11 CHAIRPERSON SCHWARZMAN: Okay. I have one more
12 here in the Q&A and then we'll have cleared our backlog.

13 DR. HOFFMAN: Okay.

14 CHAIRPERSON SCHWARZMAN: From Summer-Solstice
15 Thomas at Silent Spring Institute. "Kate, brilliant
16 presentation. Thank you so much. I know you talked about
17 the difficulty of standardizing collection of indoor dust
18 samples like the methods of vacuum collection, but from
19 your expert perspective, is there a best practice method"?

20 DR. HOFFMAN: Gosh. Well, so I think the -- like
21 it's completely out on this right now. So I think, you
22 know, one thing that I'm really interested in right now is
23 is there variability in these compounds within the home?
24 I think that's something we actually don't know and that's
25 a key thing to determine in thinking about this question.

1 So we sampled the main area that the child played in the
2 home while they were awake, because we are interested in a
3 wide range of compounds and we were thinking about this
4 idea of hand-to-mouth exposure. Knowing that air is
5 particularly important for these compounds, I'm not sure
6 that's necessarily the right environment to sample. We
7 might have wanted a sample in another location too, but,
8 you know, I think this is a really important question. Do
9 you see differences throughout the home?

10 I imagine you're going to see differences in
11 rooms with carpet, versus rooms with no carpet, versus,
12 you know, how does that -- how does that change throughout
13 the house? So I'm giving you a really unsatisfactory
14 answer there in saying that I don't know, but I think this
15 would be a really important area of research to say do
16 these compounds vary throughout the home, what areas are
17 they really high in, does ventilation matter, and some of
18 those kind of questions.

19 CHAIRPERSON SCHWARZMAN: Kate, you're going to be
20 here for the afternoon discussion session, right?

21 DR. HOFFMAN: Yes.

22 CHAIRPERSON SCHWARZMAN: Okay. I see there's a
23 hand raised in the participants and we haven't gotten to
24 Panel questions, but since we're going to discussion after
25 the break, perhaps we could just touch base and get any

1 remaining questions after the break. Rather than leaving,
2 you know, 30 seconds for the last question here, I would
3 suggest instead that we break and resume in 15 minutes.

4 So we will begin promptly again at three o'clock
5 and pick up where we left off. Thank you so much.

6 (Off record: 2:44 p.m.)

7 (Thereupon a recess was taken.)

8 (On record: 3:00 p.m.)

9 CHAIRPERSON SCHWARZMAN: Okay. I have that it's
10 three o'clock and we'll restart the meeting. And this
11 sort of launches our afternoon discussion session. I want
12 to use the Chair's prerogative to spend the last few
13 minutes just making sure we've answered all the questions
14 from the presentations before the break. We have one
15 participant with a hand raised and I would invite you to
16 unmute and ask your question.

17 It looks to me like you're still muted. I don't
18 know if that's on our end or yours.

19 DR. MARDER: We have invited the person -- the
20 attendee to speak. They need to unmute themselves.

21 CHAIRPERSON SCHWARZMAN: We can return to that
22 person, if they're not back from break yet.

23 Maybe we should go to Ulrike who has a question.

24 PANEL MEMBER LUDERER: Thanks. Yeah. Thank you,
25 Kate, for that really interesting presentation. I had a

1 question, if you could maybe tell us a little bit more
2 about whether your study provided, you know, any clues as
3 to what some of the main sources of the airborne PFAS
4 precursors were that you found?

5 DR. HOFFMAN: Yeah, it's tough. I mean, I don't
6 think we know and I honestly don't know that we have the
7 questionnaire data to be able to answer that. Although, I
8 do think there is some potential. I noticed there was a
9 question about this in the Q&A before the break about --
10 thinking about carpets. And I do think there's some
11 potential to do some of that with questionnaire data.

12 But, you know, this study was really geared
13 towards looking at all kind of semi-volatiles in general.
14 And so I don't know that we going into it necessarily had
15 all the right survey questions at the time. It was also
16 2014 when we started, so I think if we had some of that
17 data going back, it would have been really helpful.

18 One kind of interesting thing that we did see was
19 some sort of seasonal variability in some of that. It's a
20 small sample, so when you start to parse that out over
21 seasons, there's a little bit of difficulty in looking at
22 that. I do think there's some differences in ventilation
23 that could be really important to think about, so that's
24 one that's certainly interesting. It doesn't get you to
25 source at all, but I think it's an interesting

1 consideration for moving forward.

2 PANEL MEMBER LUDERER: Thank you. Were the
3 concentrations lower, just if I could follow up, in the
4 seasons when you would expect people would have more
5 ventilation, I mean, open windows, or -- you know, I
6 don't -- I don't know what types of -- what seasons
7 were -- had the lower levels I guess is what I'm asking?

8 DR. HOFFMAN: Yeah. In general, I think they
9 tended to be lower in springtime. Although, I would have
10 to confirm that for sure. I believe it was either spring
11 or fall, but I think part of the reason for that is, you
12 know, we're North Carolina. We're hot and humid. We have
13 central air conditioning everywhere. And so, you know, I
14 think -- I think the only time of year when we have our
15 windows open. Maybe it was actually fall, but it was a
16 season when we expect MORE potential window open kind of
17 weather.

18 CHAIRPERSON SCHWARZMAN: Veena, go ahead.

19 PANEL MEMBER SINGLA: Thank you. Thank you so
20 much for that presentation Kate. I've been a fan of your
21 flame retardant's work for a long time, so really nice to
22 see you.

23 I had two questions. One is you know that for
24 other contaminants that we find in indoor air and dust,
25 sometimes levels can reflect kind of infiltration or

1 migration from the outdoors to the indoors like,
2 pesticides being trapped in or brought in or air
3 pollutants infiltrating from the outdoors to the indoors.
4 So I wondered if there's any indication that those types
5 of patterns might be contributing to indoor levels of
6 PFAS. And my other question was were there any sort of
7 associations between levels of PFAS between air and dust?

8 DR. HOFFMAN: You know, so -- I mean, so one
9 thing I want to be really careful of is to not make a
10 overly broad statement about all PFAS, just because I --
11 you know, it is a really broad class and I touched on this
12 at the end, but just to say that, you know, we're looking
13 at a huge range of properties. And so for some, we're
14 going to see different things.

15 So, in general, I would say for the compounds I
16 mentioned, I think the concentrations of indoor air and
17 dust were higher than outdoors. So I think that the idea
18 of that coming from outdoors is probably not very likely.
19 So I would say that probably is true for most. You know,
20 could there be some? Potentially.

21 And let's see, can you remind me of the second
22 part of your question? I lost that one.

23 PANEL MEMBER SINGLA: Did you see any kind of
24 correlations between levels of PFAS between the air and
25 dust?

1 DR. HOFFMAN: Yeah. You know, Tom, you can chime
2 in too? If you remind me of this, because I know Jess did
3 that as part of her dissertation. I think in this
4 population they weren't super strongly correlated, I will
5 say in some previous work, particularly Tom's office study
6 I know, and I think probably Colleen's work, has
7 previously shown pretty good correlations between indoor
8 air and dust. I think in this population they were a
9 little lower than what's been reported previously.

10 DR. WEBSTER: Yeah. You know, I haven't looked
11 at these data in a while, but that's what I remember as
12 well. One would expect there to be some association just
13 from part -- from partitioning theory. I do know that
14 there's some weird stuff with PFAS. I mean, we -- I've
15 looked at some of this data before where the sort of air
16 to dust ratios are not what you would predict based on
17 octanol-air partition coefficients.

18 And I think part of it is that we don't actually
19 know some of the P chem properties very well, because
20 PFASs are -- PFASs are weird, right? But the other is I
21 think some of the stuff that we're measuring dust is
22 actually maybe bound to the carpet or whatever it is. And
23 so it's not actually fully, you know, in equilibrium with
24 the air. So there's a -- there's a lot to be understood
25 here of exactly where it's coming from and how it's

1 getting into air and all that sort of stuff. But
2 we just -- you know, there's been very little work done on
3 it.

4 CHAIRPERSON SCHWARZMAN: Thank you for that.
5 Jenny.

6 PANEL MEMBER QUINTANA: Thank you for a really
7 interesting talk. I was just thinking what a natural
8 experiment might be happening now with a lot of --
9 especially at workplaces going to increased outside air,
10 because of COVID. So really increasing the outside air
11 ventilation. And so it might be an interesting study to
12 see the effect of this, you know, increased outside air
13 ventilation on people's levels as kind of a -- of a really
14 broad scale natural experiment.

15 DR. HOFFMAN: Yeah. I mean, I think, boy, you
16 know, one thing about the last two years is it's been a
17 really interesting -- it has definitely sprung so many
18 ideas about thinking about indoor exposure for me,
19 because, you know, it's this one time where we -- where
20 suddenly everyone was spending all of their time at home,
21 right? And so you had one environment that you were
22 spending all your period of time in. And so I think that
23 was a really unique period of time.

24 And now certainly, we have this idea where we
25 have more ventilation or different ventilation, and

1 particularly in schools. I know my kids' schools right
2 now are like open all the time, and how that might impact
3 exposure is a really interesting question. I don't know
4 if anyone is doing anything on that. I'm not aware of
5 anybody. I think that's really interesting.

6 CHAIRPERSON SCHWARZMAN: One thought that occurs
7 to me kind of in concert with that is just the -- you
8 know, we've seen so many disparities or inequities kind of
9 widen in the pandemic. And of course that status of are
10 you at home or are you at work depends a lot on your
11 occupation. When there's a big chunk of people who aren't
12 able to stay home and I don't know of anybody specifically
13 looking at this, but I would be intrigued. It feels like
14 an opportunity to really understand some of the impacts of
15 particular workplaces when there are --

16 DR. HOFFMAN: Yeah.

17 CHAIRPERSON SCHWARZMAN: -- places that people
18 have had to go where everyone else who isn't a front-line
19 worker of some sort for a time didn't go into their
20 workplaces.

21 I want to check in again. I see June-Soo Park
22 with a hand raised. I want to check in and see if Kimiye
23 Touchi is back and invite you to give -- ask your
24 question. If you're back, you are permitted to talk and
25 just need to unmute your -- at your end, if you want to

1 ask a question.

2 DR. PARK: Hi. It was a great presentation
3 again. I'm just curious the -- because I see dominant
4 compound, PFAS compound, detected and your dust samples
5 were -- or even number the FTOH 6:2, 8:2. I remember two
6 years ago when publication surveyed municipal landfill
7 leachate, they didn't measure dominant congener some odd
8 number the -- you know, the FTOH. The -- have you looked
9 into the like 5:3 7:3 FTOH or in your sample -- dust
10 samples?

11 DR. HOFFMAN: Yeah. No, we didn't do that here.
12 And, in fact, in these particular dust samples, we only
13 measured the 6:2 and the 8:2. We also measured some
14 firehouse dust samples at the same time. And those we
15 were -- we measured 10:2 as well, but these were just the
16 6:2 and the 8:2.

17 DR. PARK: Got it.

18 DR. HOFFMAN: It's like a -- it's a very
19 limited -- you know, I say we measured PFAS in these.
20 It's still like a tip of the iceberg list, right, like
21 it's still really small.

22 DR. PARK: Yeah. Yeah. I totally understand.
23 Yeah. Thank you.

24 CHAIRPERSON SCHWARZMAN: Thank you. I want to
25 move on to our discussion portion and just make one last

1 call to see if the participant Kimiye Touchi wants to ask
2 a question, and if not, request that you lower your hand,
3 so we know that that moment has passed.

4 Okay. I think we will go on and start our
5 discussion now, which I just want to introduce for a
6 moment with some questions that the Biomonitoring Program
7 has posed to us. So the overarching question for this
8 discussion is how Biomonitoring California can support
9 PFAS exposure reduction efforts? Basically, how could --
10 how could data from the Program be used in that way?

11 And we can think about what we heard so far today
12 to help inform our advice to the Program on the next
13 steps, both in terms of future study design and the
14 opportunities there for data analysis that Kathleen
15 highlighted.

16 So I want to show some slides that the Program
17 has put together to just frame this conversation.

18 I'm working on it. Get the right screen shared.
19 (Thereupon a slide presentation.)

20 CHAIRPERSON SCHWARZMAN: I think that should do
21 it. So each of these has a question on it just meant to
22 focus us on guiding the Program. So referring to the
23 opportunities for further analyses of the existing data
24 sets on PFASs that were highlighted by Kathleen Attfield
25 this morning.

1 The Program's questions are, number one, which
2 are the most promising for illuminating PFAS trends in
3 California? And what type of analyses would you recommend
4 to further understand the sources of the racial
5 disparities that were reported that were observed in those
6 data sets? So what analyses would you prioritize for
7 looking at PFAS trends in the state and how should we
8 better understand what's driving the racial disparities
9 that were observed?

10 --o0o--

11 CHAIRPERSON SCHWARZMAN: The second is when
12 looking at the PFAS data for pregnant women from the MAMAs
13 project, which is based on the blood spots, as a reminder,
14 what potential limitations and confounders should we be
15 concerned with? And specifically, how should these
16 limitations and confounders inform the design of future
17 sample selection from the GDSP, which is the Genetic
18 Disease Screening Program, Biobank that is the repository
19 of these blood spots? So thinking about what are the
20 limitations and confounders of that as a sample source and
21 how to keep that in consideration?

22 --o0o--

23 CHAIRPERSON SCHWARZMAN: With regard to
24 identifying key PFAS exposure sources in biomonitoring
25 studies that are intended to inform exposure reduction

1 CHAIRPERSON SCHWARZMAN: And then the final
2 question is just a broad one. Any other input on next
3 steps for Biomonitoring California that would support PFAS
4 exposure reduction efforts. So I think I'm going to stop
5 sharing my screen, because otherwise we have to dedicate
6 it to one question or another and I also can't see
7 participants. But let me know if you want to -- me to
8 reiterate any of those questions and we can revisit them,
9 if we run out of things to say to make sure that we
10 responded to all of the Program's questions.

11 I also want to just mention that in this
12 discussion session, I will call for public comment at some
13 point, but let's start off with folks who have their hands
14 raised.

15 I have Kathleen and then Nerissa.

16 MS. HOOVER: Nerissa, maybe you could just go
17 ahead.

18 DR. WU: Yeah, I'll just -- I just wanted to
19 chime in. One correction is that the samples for the
20 MAMAs are actually a prenatal serum sample taken during
21 the second trimester of pregnancy, not a newborn blood
22 spot. And I think the timing of when that is taken -- one
23 of our questions is related to how you would design or
24 analyze MAMAs data related to things like blood volume and
25 how pregnancy might impact that and their subsequent PFAS

1 levels.

2 CHAIRPERSON SCHWARZMAN: Thank you so much,
3 Nerissa. Yes, I missed that. Thank you for clarifying.
4 Carl Cranor.

5 PANEL MEMBER CRANOR: Can you hear me?

6 CHAIRPERSON SCHWARZMAN: Yes.

7 PANEL MEMBER CRANOR: You raised -- gosh. I'm
8 echoing. You raised a question about minority
9 communities. And I'm wondering have you ruled out where
10 they live? I mean, P -- the PFOAs may or may not be part
11 of a -- living near an industrial area or something like
12 that, but they get a lot of contamination for, you know,
13 things that they live next to, their houses, their sources
14 of air pollution, some studies have been done, things like
15 that. You could rule them out or maybe rule them in.
16 Just a question.

17 CHAIRPERSON SCHWARZMAN: Kathleen might want to
18 say something about this, but -- or Nerissa. But as I
19 remember from the morning's presentation, it was Asian
20 participants had the highest exposures followed by White
21 and then Black -- Hispanic and then Black, is that right?
22 Do you all mind repeating that?

23 DR. ATTFIELD: Yeah, that's correct. I did find
24 it interesting that in the new North Carolina studies,
25 they were also saying White is greater than Hispanics. So

1 the same pattern is happening elsewhere in the country for
2 that.

3 As far as Carl's comment, it is interesting to
4 try and think about which locations might be of immediate
5 concern. And I might punt that over to Karl Palmer a
6 little bit. But we definitely don't have any large
7 manufacturers of PFAS in California for one.

8 PANEL MEMBER CRANOR: Um-hmm.

9 DR. ATTFIELD: And so, yeah, maybe the chrome
10 plating or -- well, then you're -- as Kate Hoffman was
11 talking about, then you're looking at some of the more
12 dispersed source pollutants, which is sort of somewhere in
13 between the very local and a very diffuse pollutant
14 source, such as the wastewater treatment plants feeding
15 into the Santa Ana River.

16 PANEL MEMBER CRANOR: I just thought it was worth
17 mentioning, because often these communities live in
18 buildings or areas that have been pretty contaminated.

19 DR. WU: Right. And that's certainly a kind of
20 analysis we could do, similar to what we've done with
21 1-nitropyrene where we could do some GIS work, if we knew
22 what sources we were looking at and do kind of distance
23 to -- traffic or distance to a facility in this case. It
24 is -- we do have addresses for our non-MAMAs data. And
25 so, yeah, that is -- that is something we could look at

1 for -- I mean, it's a question of if that is our priority.
2 I think it's one of the dominant exposure sources.

3 CHAIRPERSON SCHWARZMAN: Tom McKone.

4 PANEL MEMBER MCKONE: Hi. Thank you. So this is
5 a -- I'm kind of struggling with some of the conversations
6 we had earlier about how hard it is to really tease out,
7 you know, what we're seeing, because there are competing
8 pathways and similar substances with the same biomarkers.
9 So it's kind of a chicken and egg, because I think, you
10 know, the only way -- I mean, we're thinking about how to
11 improve biomonitoring to understand how to reduce
12 exposures. And in a way, the only way we're going to
13 understand that is to reduce exposures and see what
14 happens, right, which, you know, we don't -- I don't know
15 how you do that. And so maybe that's what we need to
16 think about.

17 Are there ways to look at market trends or
18 reductions, you know, things that should reduce exposures
19 and see if they're actually happening? I mean, it's sort
20 of like, you know, in an ideal world, if we could do
21 everything we wanted, we would -- we would, you know, do
22 this differential analysis. We would remove one product
23 from the market totally, and then put it back in the
24 market, right, a case crossover kind of study. And we
25 can't really do that, but maybe it's possible to think

1 about ways to watch the factors, like consumption
2 patterns, consumer products, or even some very targeted
3 questionnaires to see where there might be reductions in
4 exposure, and see how that plays out in the biomonitoring
5 data. And then we can go back and say, okay, we kind of
6 confirmed that hypothesis to some extent, so that might be
7 somewhere we want to put resources.

8 But again, it's very com -- but I struggle with
9 just absolutely at this point saying, oh, given the
10 uncertainties we heard about today, we're supposed to say
11 this is the best thing to do to understand how to reduce
12 exposures, where that's going to be very hard to do until
13 we have a better understanding of these complicated
14 relationships.

15 CHAIRPERSON SCHWARZMAN: Thank you, Tom. If it's
16 okay, I'll insert my own comment, and then Ulrike, I'll
17 call on you next.

18 One thing that's been on my mind is something
19 that comes up a lot is sort of treating PFAS as a -- as a
20 group and talking about it -- about as PFAS in general,
21 all PFAS versus the different chemicals that are used in
22 different applications and that seem to change over time.
23 And, of course, with the absence of, you know, required
24 reporting, we don't -- about chemicals that are in
25 products, but that are used in particular applications, we

1 don't have a general understanding. There's no sort of
2 publicly accessible information about what is used in
3 products and how that might be changing over time, how
4 that chemical profile might be changing over time.

5 But I'm thinking about some -- so what that says
6 to me is that as much as we can be looking across the
7 board at different types of PFAS compounds, that that
8 might tell us something about exposure sources and what's
9 happening. The example that I'm thinking of is sort of
10 what we've seen in shifting patterns of use of phthalates
11 and how that has been reflected in biomonitoring data, and
12 how you can see action taking place on some phthalates,
13 and then, you know, with a little bit of lag, the
14 concentration of replacement phthalates rises over time.
15 And so we've seen that sort of play out in that switch
16 from one chemical to another within a related class of
17 compounds. But the only way that we're going to see that
18 of course is by looking for a fairly wide range of the
19 compounds, and there's such a long list with PFAS, that
20 that's pretty daunting.

21 But people who know more about which types of
22 PFAS are used in which types of applications than I do,
23 could probably help inform some hypotheses about, well, if
24 they're being eliminated from food contact materials,
25 these are the chemicals that we might expect to see go

1 down. And whereas, you know, the legacy compounds that
2 have very long half-lives, yes, those are declining over
3 time, but maybe not as dramatically as when something is
4 pulled from the market and the chemicals have shorter
5 half-lives.

6 So I don't have the details to fill in the
7 substance of what that recommendation would be, but that's
8 the approach that I would think about is all of the
9 information that you can get on which types of PFASs are
10 used in particular applications that are under scrutiny or
11 that -- or that there's action being taken on like food
12 serviceware. And I know that's all really difficult
13 information to obtain.

14 Ulrike.

15 PANEL MEMBER LUDERER: Actually, the last thing
16 that you just said was essentially the direction that I
17 was going in also, which is that this -- you know, the
18 effort to remove PFASs from food contact materials
19 provides an opportunity to see, you know, whether that
20 intervention actually results in reductions in exposure
21 levels over time, but we need to know what are the PFASs
22 that are in food contact materials. And it sounds like
23 from some of the presentations that we heard today, we
24 don't necessarily know that. And so there may need to be
25 two research initiatives happening at the same time to

1 better understand what's in those -- what's currently
2 being used, and then as they're taken out, you know, to be
3 able to try to follow the trends over time and see if
4 there's a reduction in biomonitored concentrations of
5 those particular PFASs.

6 CHAIRPERSON SCHWARZMAN: Go ahead, Nerissa.

7 DR. WU: I was just going to say that, I mean,
8 it's important to have a comparison group as well, just
9 because we have overall downward trends. So maybe there's
10 an opportunity where there's some municipalities that are
11 being more progressive with reduction in food packaging to
12 compare to do surveillance in different communities, just
13 because otherwise you're looking at it in this overall
14 context, and you have lots of different changes happening
15 over time and it's difficult to interpret.

16 CHAIRPERSON SCHWARZMAN: Yeah, I really hear that
17 and probably the only time to do that is in that kind of
18 liminal space when there are changes being made and there
19 will actually be differences in various markets, because
20 once large areas like the State of California do something
21 to eliminate the use of PFAS in a -- in a whole product
22 category, ultimately that will trickle down to the rest of
23 the market, but at the -- at the beginning of a shift like
24 that, there's going to be differences. And the tricky
25 thing is to be responsive, you know to be able to do

1 anything either fast enough to capture a change like that
2 or, I mean, that's the point of surveillance, right, is
3 that if you're measuring consistent substances over time,
4 you're already measuring them, and so you can look back
5 and see these changes reflected or try to understand them.
6 But it's hard if you don't have the capacity for that
7 level of surveillance.

8 DR. WU: And it does take a certain nimbleness to
9 be able to go out and grab the samples, but that is one of
10 the ways MAMAs or the biobank samples are well suited,
11 where you can get retroactive samples. We could go back
12 to the San Francisco area and look over a particular time
13 period in comparison to other places. And I thought of
14 that also when Kate was talking about the Santa Ana area
15 and the interventions in Orange County with their water
16 supply in 2020, going -- we could go back in time and then
17 follow it up prospectively to see how those levels are
18 dropping compared to the other parts of the State.

19 I know I keep adding stuff to things we could do,
20 which is not the point, but it is -- there are so many
21 questions we're trying to answer.

22 CHAIRPERSON SCHWARZMAN: I want to just flag that
23 Simona Balan has mentioned in the Q&A that we know -- we
24 do know what's used in food packaging, because the
25 chemicals are listed in FDA's food contact notifications

1 database, but we don't know all the impurities and the
2 degradants of those PFAS, so to add that to the
3 conversation.

4 Let me -- I want to turn to Karl Palmer next and
5 just remind folks to lower their hand, if you would, so
6 that I can keep track of who else needs to speak.

7 MR. PALMER: Thanks, Meg. Yeah, I'll just -- to
8 riff on what Simona pointed out is that I think there are
9 potential strategic opportunities to look at partnering
10 with other regulatory bodies and know what food contact
11 notifications are required. We also know that CalRecycle
12 has implemented certain restrictions on PFAS in food
13 packaging used at State facilities. So there might be an
14 opportunity to find a cohort of people that ostensibly
15 will be having reduced exposure to certain kinds of
16 packaging.

17 And so those kinds of things -- kudos to the
18 Biomonitoring staff who worked really hard to leverage
19 their limited resources with the other agencies, but I
20 think there's opportunities to expand that and to find
21 potential opportunities there that could perhaps get good
22 data.

23 CHAIRPERSON SCHWARZMAN: Great.

24 Veena.

25 PANEL MEMBER SINGLA: Thank you. Maybe this

1 doesn't make any sense, but I wondered about looking at
2 the populations that have very low PFAS exposures and
3 seeing if there's information there that could kind of
4 speak to what might be helpful in reducing PFAS exposures.
5 So, you know, do like -- I'm just making this up right,
6 but like -- like maybe a vegan diet or people that don't
7 eat fast food at all, or, you know, use very few personal
8 care products, because I think like both types of
9 information, both, you know, trying to like really
10 understand the sources of exposures for the kind of
11 populations with the highest exposures as well as what
12 might -- what kind of behaviors or actions might prevent
13 exposures, like both kinds of information are useful, so
14 just a thought.

15 CHAIRPERSON SCHWARZMAN: Go ahead, Kathleen.

16 DR. ATTFIELD: I love that suggestion, Veena,
17 because we've partly already done it.

18 (Laughter.)

19 DR. ATTFIELD: We had -- in addition to asking
20 about people's individual food item frequencies, we try to
21 ask them about different types of diets. And in the ACE
22 study we also asked about dietary changes over time. Not
23 an initial smoking gun, I'm afraid, for sort of vegetarian
24 or vegan in the CARE population though. I had held out
25 hope for it.

1 (Laughter.)

2 CHAIRPERSON SCHWARZMAN: So maybe I'll just point
3 us back to some of the other questions. We've sort of
4 been talking about general opportunities or priorities how
5 Biomonitoring California data that exists or that could be
6 gathered would support PFAS exposure reduction efforts.
7 And so just to return to some of the other questions,
8 maybe to highlight one potential for doing additional
9 analyses on the existing data sets. And although we
10 talked for a minute about the sort of racial separation of
11 some of the results with the PFAS data that Kathleen
12 presented this morning. Another question was about
13 potential limitations and confounders with the GDSP
14 biobank data used for the pregnant women and the MAMAS
15 project and what we might recommend to keep in mind with
16 that. Another is questions about designing PFAS exposure
17 questionnaires or other ways of evaluating PFAS exposure
18 sources.

19 Kathleen, are you still wanting to say something?

20 No. Okay.

21 Any feedback from the Panelists on those
22 questions?

23 José.

24 PANEL MEMBER SUÁREZ: When it comes to the
25 race/ethnicity piece, in many ways now, that's considered

1 more of a social construct than really a truly genetic
2 one, when we're looking at differences in just about any
3 health outcome with maybe some rare exceptions, but
4 overall in that sense. But I think the findings that were
5 presented today about very substantial differences --
6 maybe somebody can remind me how much higher the
7 concentrations were for some of the PFAS for Asians
8 compared to some of the other groups. From what I recall,
9 it was something like 80 percent, is that right, for some
10 of them?

11 DR. ATTFIELD: It depends on the comparison, but
12 yeah, 144 percent was the highest, you know, when you're
13 going -- you know, comparing the highest group, Asians,
14 down to the lowest group, which were Black participants.

15 PANEL MEMBER SUÁREZ: Uh-huh. So I mean I see
16 that as a --

17 DR. ATTFIELD: It was in the slides. I'm happy
18 to pull up any slides anyone would like to see again, if
19 you would like me to.

20 PANEL MEMBER SUÁREZ: Well, thank you, Kathleen,
21 but I think the -- I think overall there was some -- there
22 were some pretty stark differences for many of the PFAS
23 being substantially higher among Asians compared to most
24 other groups. And so I think that gives this window of
25 opportunity of starting to get a little bit deeper than

1 that. It's really more of a behavioral or environmental
2 difference there.

3 And if we're talking about 140 percent difference
4 for some of these, there's something important that one
5 group is doing that the other ones are not when it comes
6 to getting exposures to a lot of these compounds. Of
7 course, PFAS are present in so many -- there's just so
8 many sources that it's hard to even be able to fathom
9 constructing a very thorough or all-encompassing survey
10 for identifying the sources, but I think this is one of
11 those particular settings in which we might be able to get
12 a little more of a straightforward answer, given these
13 stark differences across these constructs, these groups.
14 So that's something worth looking at.

15 Also, we can't really say Asians and say it's all
16 a homogeneous group, obviously. And so from there, it's
17 parsing it out, right? So do we have -- or do you have
18 any information about the subgroups within the different
19 Asian populations, in which there may be another way to
20 even start getting a little bit closer to what some
21 behavioral or environmental differences there may be.

22 PANEL MEMBER FIEHN: If I might add directly to
23 this question. You know, I also find these kinds of
24 ethnicities, asking people how they feel, it's a little
25 outdated, because many people say I'm mixed race anyway,

1 and they feel more and more comfortable to tick that box
2 and it's actually true. So I wonder about that in terms
3 of socioeconomic status, rather than, you know, their
4 cultural and ethnic backgrounds. Do you have information
5 about that?

6 So it looked to me that these people were just
7 eating more fish and that one of the reasons could be
8 because they are richer. I'm just making it up here as we
9 go, but it could be, right? And that is getting lost in
10 that, you know, adding a label of some kind of ethnic
11 backgrounds.

12 CHAIRPERSON SCHWARZMAN: I definitely support
13 that, and -- but I also remember that Kathleen showed us
14 that the -- those racial category differences persisted
15 once fish consumption was controlled for. So it may still
16 be what's driving it, not the fish, but it may be a
17 socioeconomic thing that's driving other sources of
18 exposure also. But I completely agree with you that, you
19 know, we all know that race is a social construct not a
20 biological determinant of health. And so the question is
21 what is it connected to? I mean, these are all the same
22 question, right, is like what exposure source is that
23 connected to?

24 Kathleen, did you want to respond to something
25 there and then -- and then I'll get to Tom.

1 Oh, no. Hand down.

2 Okay. Tom, go ahead, please.

3 DR. ATTFIELD: Oh.

4 DR. WEBSTER: Do you want me to go ahead?

5 CHAIRPERSON SCHWARZMAN: Sure. I think I misread
6 Kathleen putting her hand down, but please, you go ahead
7 Tom and then Kathleen.

8 DR. WEBSTER: So I -- PFASs I think is a pretty
9 interesting group of compounds, at least the legacy ones.
10 It seems to increase with socioeconomic position, contrary
11 to lots of things. And that suggests that it's -- again,
12 it's not the biology of race. This is like where
13 environmental epidemiology and social epidemiology
14 intersect, that people have more income, and so they have
15 different purchasing, and maybe it's you buy carpet, or
16 your diet is different, or you eat more fast food. I
17 don't know. There's all sorts of things going on there.

18 And, I mean, you know, NHANES does have some
19 pretty nice data on socioeconomic status that they manage
20 to collect that I think makes a pretty good case that
21 that's an important variable for PFAS. So I don't --
22 again, I don't know what California Biomonitoring has for
23 that, but I'm sure that's part of it.

24 CHAIRPERSON SCHWARZMAN: Sorry to have skipped
25 over you there, Kathleen. Please, go ahead.

1 DR. ATTFIELD: I think I hit the lower hand
2 instead of the mute -- or unmute.

3 Thank you for making the point that, yes, we
4 didn't see the contribution and that's in kind of
5 quotation marks, contribution by race disappear with the
6 addition of fish into our models. So there's still more
7 to this relationship to uncover. And I won't claim that
8 we have plumbed it completely.

9 One additional piece of information that is good
10 to know about the CARE study is that in relation to law
11 changes, we -- even though I presented these as, you know,
12 very simplistic categories of racial/ethnic
13 identifications, we did allow everybody to identify, as --
14 in as many categories as they agreed with their
15 background. So, of course, for analysis purposes, you
16 know, sometimes you do have to then take various
17 simplifications, but we do have that underlying
18 information, so that we can look at things in different
19 ways going forward.

20 And I would say our income data is we had let
21 that be an optional category, so we don't have that for
22 the entire data sets. We do have education. Of course,
23 these aren't completely correlated of course, but give you
24 extra information about socioeconomic status. And at
25 least for education, usually it drops out of the model

1 once you put age, sex, and race into it for many of the
2 compounds, not for PFNA, but for the others.

3 CHAIRPERSON SCHWARZMAN: Thanks, Kathleen. José
4 were you wanting to join back in?

5 PANEL MEMBER SUÁREZ: No. Oh, sorry, my hand
6 was -- should have been lowered, but --

7 CHAIRPERSON SCHWARZMAN: Okay. Thanks then.
8 Tom.

9 DR. WEBSTER: I was just going to say that I
10 think this is where, you know, economic theory can --
11 sorry, epidemiologic theory can actually help us, because
12 dietary exposure is going to be a poorly measured
13 variable, because it's really -- it's what you've eaten
14 over the last five to 10 years that matters for the
15 persistent PFASs, not what you ate yesterday. So it all
16 depends on, you know, if you use -- so this comes up in
17 NHANES that you use food frequency questionnaires, whether
18 you use dietary surveys, and all that sort of stuff.

19 But the point is that a poorly measured
20 confounder will not fully control for confounding, right?
21 And it can actually bias things in either direction. So
22 it could be that, you know, we control for fish, but it
23 doesn't fully remove the effect of fish, and so it's still
24 there. So I'm not saying that's not the explanation. It
25 just -- it's -- you know, it's hard. It's hard. Diet is

1 sort of notoriously hard to measure. And, in fact, I'm
2 kind of -- I'm always surprised that we see any
3 relationship with dietary questionnaires.

4 CHAIRPERSON SCHWARZMAN: Nerissa.

5 DR. WU: I just wanted to say that these are all
6 great points about race, and the questions that we still
7 have remaining. And it could be that the ACE data set is
8 one of the places we should be looking for some of these
9 analyses. ACE, of course, the impetus for that was
10 because we did want to understand why Asians were higher
11 in metals as well as PFASs. And it is an opportunity for
12 us to look at, you know, Asians is -- just is a very
13 heterogeneous group. We're able to look at Chinese and
14 Vietnamese and it would be great to get more information
15 with robust numbers to be able to look at these
16 subcategories of Asians in a way.

17 We did -- we do struggle with the homogeneity of
18 some of the answers, because everyone ate a lot of rice
19 and fish in that group, but it is -- we have sufficient
20 numbers and we have a lot of detail on diet that we just
21 are not able to include in something like the CARE study.

22 CHAIRPERSON SCHWARZMAN: Thanks.

23 Kathleen.

24 DR. ATTFIELD: I just wanted to add on to that
25 point of Nerissa's. So the ACE study looked at

1 Chinese-Americans in the San Francisco area in one year
2 and the subsequent year was in Vietnamese-Americans in the
3 San Jose area. And back to the point about sort of
4 chemometrics and PFAS profiles. They did have different
5 profiles between the two and we haven't been able to move
6 beyond sort of recognizing that the patterns were
7 different there, but I think it's a good promising arena
8 that we could explore more.

9 PANEL MEMBER SUÁREZ: Well, in that sense, if I
10 can chime in, so if there was a year difference and
11 location difference from where the two different groups
12 were located, of course, that adds a lot of new variables
13 to that, right? So we're talking about temporal changes
14 and geographical effects to it, maybe not necessarily
15 fully behavioral differences or otherwise across the
16 different groups. Of course, it would have been ideal to
17 have inter-mixed, at the same time ideally somewhere
18 around the same areas where both groups or multiple groups
19 were collected. So, you know, that's just adding
20 additional levels of complexity to disentangling, I guess,
21 the differences.

22 CHAIRPERSON SCHWARZMAN: Maybe I will take this
23 moment to do our sort of formal call for public comment.
24 I think it's been understood that participants and anyone
25 in the audience can raise a question or provide a comment,

1 but I want to remind you that you can use the raise-hand
2 feature in the Zoom webinar or send an email to
3 biomonitoring@oehha.ca.gov, or type a question into the
4 Q&A function on Zoom.

5 So I just want to check in with staff and see if
6 there's any public comment that we haven't tended to.

7 DR. HOLZMEYER: There's no emails.

8 CHAIRPERSON SCHWARZMAN: Okay.

9 DR. IYER: And no hands raised.

10 CHAIRPERSON SCHWARZMAN: Thank you, Cheryl and
11 Shoba.

12 Kathleen.

13 DR. ATTFIELD: Apologize, I keep not tending to
14 it.

15 CHAIRPERSON SCHWARZMAN: Maybe there's a sort of
16 call to folks for other kind of nominations for other
17 potential sources of exposure around which we might see
18 differential exposure that would help us understand
19 exposure sources, like we've already talked about, the
20 elimination of PFAS from food contact materials or food
21 serviceware, and to flag any other ideas like that for the
22 Program.

23 Jenny.

24 PANEL MEMBER QUINTANA: Hi. We have such experts
25 among us, I'm hoping that we could hear -- especially I'm

1 interested in occupational exposures. It seems like this
2 is an exposure that might be very prevalent, firefighters,
3 military bases or people that live on military bases, you
4 know, airports. I'm just wondering if you have any
5 comments on occupations, which would be important to
6 study?

7 CHAIRPERSON SCHWARZMAN: If I understand you,
8 right, Jenny, you're asking for comments from any of our
9 expert speakers who have contributed today?

10 PANEL MEMBER QUINTANA: Yes, I just thought what
11 a great opportunity to get advice about what we should do
12 from them.

13 CHAIRPERSON SCHWARZMAN: Yes, absolutely.

14 DR. WEBSTER: Well, I have to say my experience
15 with PFAS and occupational exposure is really chemical
16 workers, you know, chloropolymer facilities. So, I mean,
17 I would expect that there might be some difference with
18 firefighters and maybe the -- you know, I don't know,
19 these chrome plating things, I've never done any work on
20 that, but that sounds like that would be worth looking at.

21 There are a lot of them and I -- actually, off
22 the top of my head, I don't know if anyone has actually
23 looked at that.

24 DR. HOFFMAN: Maybe I'll just throw on to add on
25 to what Tom said there. We also looked at firefighter

1 dust in the same study that I referenced looking at the
2 household dust. And there, we did find higher levels of a
3 lot of those compounds in dust in fire stations,
4 indicating some occupational -- or potential for
5 occupational exposure there. I know it's dust and I
6 showed you that maybe dust isn't the most important
7 exposure, but I think you might expect a similar pattern
8 there. And certainly, you know, just given the use of
9 these compounds and AFFF were also like firefighting gear,
10 you might expect that exposure there. So I think there
11 are studies looking into that now, so you might expect
12 that as well. Like Tom, I don't know about anything with
13 the plating industry, although that's an interesting
14 question too.

15 PANEL MEMBER QUINTANA: I actually -- sorry, go
16 ahead.

17 CHAIRPERSON SCHWARZMAN: No go ahead.

18 PANEL MEMBER QUINTANA: Well, I was kind of
19 unfairly going to ask another question. Is that okay?

20 CHAIRPERSON SCHWARZMAN: That's okay. Carry on.
21 While you -- yep. Go ahead.

22 DR. WEBSTER: You know, it made me think, like I
23 don't know if anyone has looked at food workers, people
24 who work in fast food restaurants or in pat -- the food
25 processing industry. I don't know if anyone has actually

1 ever looked at that. So I don't know how much exposure
2 there would be, because I think a lot of the food contact
3 materials are actually polymer based. And so you might
4 have residuals and so it's going to be complicated and
5 there's lots of them and they're hard to measure, right,
6 so...

7 MR. PALMER: I just might add that, you know,
8 this is one of the challenges that we have when we're
9 looking at certain consumer products that contain the
10 materials. Once you know that they're there, then you can
11 start breaking down how they get there and the process
12 that they're manufacturing.

13 Food packaging is a good example. Food packaging
14 is generally a combination of a lot of different
15 materials, sometimes with multiple people in the supply
16 chain. And so, for example, some of the fiber based food
17 packaging uses mold releasers that contain PFAS. I'm not
18 sure if those are sprayed on and there's someone there
19 spraying it or if it's automated and what workers -- but
20 that's one of the challenges, not only where are these
21 chemicals in the products, but how are they actually
22 manufactured, which would speak to the role of workers,
23 but certainly platers are a good example of someone who
24 there's probably a good chance they're exposed.

25 DR. WEBSTER: Another one that might be worth

1 looking at would be, you know, people who work in places
2 that sell carpet, if the carpet is treated. Karl, do you
3 know if anyone is -- has done that. I mean, I know this
4 has been done with things like flame retardants in the
5 past, but I --

6 MR. PALMER: Well, I don't know and Simona might
7 have a better idea. She led our PFAS team. But we did,
8 when we were looking at these treatment products for
9 example --

10 DR WEBSTER: Yeah.

11 MR. PALMER: -- you can purchase a piece of
12 furniture that is not treated and then when you buy it,
13 they say would you like other treatment. And so we don't
14 know if there's some poor guy on the back loading dock
15 who's spraying it or if it's done in a factory on order
16 and things like that, so those are certainly good
17 questions.

18 PANEL MEMBER QUINTANA: My second somewhat
19 unrelated question, but you brought up the dust issue
20 again, Dr. Hoffman, and I'm just wondering that even
21 though dust might not be as correlated to the air -- the
22 body burden as air levels, I'm wondering if it could still
23 be serving as a reservoir, and that the variability in air
24 levels could be, you know, the reservoir partitioning into
25 air, plus ventilation in the home leading to air levels or

1 something like that. I was just kind of curious if
2 there's any thought that dust could be a reservoir,
3 because it certainly has a lot of stuff in it.

4 DR. HOFFMAN: Yeah, definitely. And, I mean, I
5 just -- you know, I want to make really clear too that,
6 you know, we looked at a limited set, right? And so dust
7 might be really important for some other things. And
8 that's part of the hard part about understanding these
9 compounds. We're going to get some that are going to be
10 really important in air and some that are going to be more
11 important in dust. So I think that's an important point.

12 And you're right, you're going to get this kind
13 of equilibrium and partitioning between the two and you
14 may see that sort of as a reservoir for what's coming into
15 air as well.

16 CHAIRPERSON SCHWARZMAN: Thanks.

17 Ulrike and then I have a question from the Q&A
18 from a participant.

19 PANEL MEMBER LUDERER: Thanks. Yeah. I just had
20 a couple of comments. One, apropos of firefighters. I
21 know one of the questions of the Program was asking us was
22 whether there are additional measurements that would --
23 potentially could be made in some of the archived samples
24 from prior Biomonitoring California studies. And I know
25 in the FOX study, which is the Firefighter Occupational

1 Health -- Occupational Exposures study, the smaller
2 original kind of group of 12 PFAS was measured. And I
3 think it might be worth looking at the expanded set of
4 PFAS in that -- in those samples, because dust was in a
5 subset of the fire stations in that study. I know that
6 PFAS were also measured in dust as I recall. So that
7 might be -- I mean, it would be a while ago, so you are
8 going to be talking more about historical exposures in
9 firefighters, but that might an opportunity to use
10 existing samples from the Program's archives.

11 And the other thing apropos of, I noticed -- I
12 recall that there was a study that was done by the Program
13 where people were replacing their upholstery and it was
14 looking at flame retardant biomonitoring levels, but I
15 wonder whether that study might be an opportunity to look
16 at the effects of that on PFAS. Now, I don't remember how
17 long -- you know, what the time interval was with these
18 longer lived compounds that might be too short. But that
19 it might be another thing to think about and -- you know,
20 an intervention study that was done that might be
21 informative.

22 CHAIRPERSON SCHWARZMAN: Thank you, Ulrike.

23 While we're on the topic of occupational
24 categories, I'll just add two comments from the question
25 and answers. Simona Balan says that, "There were some

1 studies of air monitoring in carpet stores, but not in
2 California".

3 And I'll also add that I just know of a doctoral
4 student who was trying to measure exposure to carpet
5 recycling workers in California, because we have a mandate
6 for carpet recycling and wasn't able to gain access to the
7 facility. So that's a potentially highly exposed
8 occupational category. I just want to say that's my own
9 comment.

10 And Anna Reade comments that, "Some other
11 occupational exposures that may be of interest include ski
12 areas..." -- I assume that's like people who are doing ski
13 waxes -- "...car washes and cleaners who are..." -- like
14 janitors, I assume here, because of floor waxes.

15 And I'll add to that maybe just that it's so
16 tricky, because all of those uses of PFAS-intensive
17 materials also involve environmental contamination with
18 those products. And so teasing out what gets into the
19 environment and what gets into the workers, it can be
20 tricky.

21 Tom, did you have something to add?

22 DR. WEBSTER: Oh, yea. There's definitely been
23 work on ski waxers in Scandinavia. There's been several
24 very good studies. They have very high exposure.

25 CHAIRPERSON SCHWARZMAN: Presumably because

1 they're applying it with heat --

2 DR. WEBSTER: Absolutely.

3 CHAIRPERSON SCHWARZMAN: -- and there's
4 volatilization.

5 DR. WEBSTER: Little tiny, not very well
6 ventilated rooms, and -- you know.

7 CHAIRPERSON SCHWARZMAN: Right.

8 DR. WEBSTER: Although, I think that stuff is --
9 they're taking it out of the wax for professional
10 competitions now, I believe.

11 CHAIRPERSON SCHWARZMAN: I have got a question
12 from Summer-Solstice Thomas from Silent Spring Institute.
13 "Has it been considered..." -- so this is sort of getting
14 to the point of questionnaires. "Has it been considered
15 the importance of asking individuals the date of their
16 most recent menstrual cycle when taking blood samples for
17 PFAS biomonitoring? Has Biomonitoring California looked
18 at PFAS levels in breast milk"?

19 So that's two questions, one specifically for the
20 Program about breast milk and another more generally about
21 the role of asking for date of last menstrual cycle.

22 DR. ATTFIELD: This is Kathleen Attfield.

23 As far as breast milk, at least four of these
24 studies that the design of the study is conducted by
25 Biomonitoring California, we haven't measured any breast

1 milk, I believe. I can't speak for our lab
2 collaborations. And no, we have not asked about most
3 recent menstrual cycle. What we have -- the relevant
4 information is mostly about -- related to age and to
5 parity that we have for various studies.

6 DR. WEBSTER: Parity is huge.

7 CHAIRPERSON SCHWARZMAN: And, Tom, you're saying
8 that because levels decline with increasing parity?

9 DR. WEBSTER: Absolutely. I mean, I think that's
10 very well established in the literature now.

11 CHAIRPERSON SCHWARZMAN: Is that independent of
12 breast feeding?

13 DR. WEBSTER: It's connected to breast feeding,
14 but I believe it's independent. Although, I can't swear
15 by that and I'd have to look.

16 DR. ATTFIELD: And I should add we have months of
17 breast feeding as well for the CARE studies at least.

18 CHAIRPERSON SCHWARZMAN: I have June-Soo and then
19 Sara.

20 DR. PARK: Yeah. Just a quick comment, because
21 you guys talked about the ski wax. Actually, Anna Kärrman
22 was the one -- her group and her former advisor did a lot
23 of work a lot of work on the ski wax, ski -- the worker
24 for the PFAS exposure.

25 I just want to comment that since I opened my

1 microphone, I also would like to make -- keep comment
2 toward José's earlier concern about the background. To
3 our experience, by far, PFAS background, you know, came
4 mainly from our instrument, when we purchased it and
5 installed -- purchased a new instrument and installed it,
6 it took us long time to get the background levels down,
7 even after we replaced all the teflon liners. That's
8 what -- what's happening to our new instrument just
9 installed.

10 We had a 6:2 fluorotelomer sulfonate background,
11 which is gradually coming down, but it just takes time,
12 like our old instrument. But, you know, the old test
13 tubes we tested has a little background was because I just
14 realized we published the paper. You know, the reserve we
15 tested the serum separation too, compared to the red-top
16 tube we historically used for the blood collection
17 analysis. So I think I can forward that publication to
18 you, that's the 2014 one.

19 Thank you.

20 MS. HOOVER: So, Meg, I just wanted to chime in
21 and say it's almost 4:05, which means we have very little
22 time left in the discussion. However, I can tell you that
23 the plan for the 2022 SGP meetings is extremely short, so
24 you could consider, you know, that we cover that and then
25 you come back and close up this discussion. So think

1 about if that feels right, because I don't think we're
2 going to have time to close up this discussion very well
3 or address some of the other questions.

4 And actually, I was raising my hand to answer the
5 other question that Simona had posed, which is, "Is
6 Biomonitoring California considering updating its PFAS
7 definition to match the revised definition from OECD"? We
8 have not at this point. And that would actually be an SGP
9 decision. Now, you all may recall that I did raise
10 potentially expanding and looking at more fluorinated
11 compounds as a past possible chemical selection item, and
12 that was not of interest to the SGP. But if the Panel is
13 interested in reconsidering the definition, that's
14 something that we could bring for your consideration at a
15 future meeting.

16 CHAIRPERSON SCHWARZMAN: Nerissa.

17 DR. WU: Thanks. I just wanted to address
18 Ulrike's comment about using archived samples to go back
19 and look historically at PFASs. A reminder that when we
20 do any analyses on old samples, we are obligated to then
21 return the results to participants. And so that triggers
22 another concern, which is that people have signed up for a
23 study maybe years ago, and so returning results to them
24 may be coming to them out of a little context. And so
25 it's something that we always consider when going back.

1 We have done that with the FOX participants going back to
2 look another class of flame retardants. And so there's
3 some precedence for it.

4 But the MAMAS also does offer the same kind of
5 benefit in terms of being able to do historical profiles
6 of PFASs. And then that way also we can -- we can get
7 more of a surveillance type of data, rather than a
8 particular cohort that we would have recruited to the
9 study.

10 And because we are coming to the end of the time,
11 I just want to put in a plug again. We've talked a little
12 bit about collaborations. We've talked about more types
13 of analyses than the Program can do on our own. And so
14 inviting all of you to think about students who might be
15 interested in doing this kind of work. We have lots of
16 data sets. And I think Cheryl or Sara will also post the
17 links to our positions available in Biomonitoring
18 California, because if there are people listening, who
19 would like to come work on some of these questions, we are
20 looking for good epis.

21 CHAIRPERSON SCHWARZMAN: I have a question about
22 that, Nerissa, of how to better matchmake between the
23 needs for data analysis that the Program has and the rich
24 data sources that are here. And, you know, the very
25 spread out, diffuse sort of placement of doctoral students

1 and working with researchers in different universities all
2 around. And I -- is -- I've been kind of mulling it over
3 all day, but I wonder if there's any -- if we could think
4 through some kind of format that is not too burdensome for
5 the Program, like putting together a short slide deck that
6 would illustrate some of the opportunities that there are
7 that could be circulated or if there could be one webinar
8 held that everybody could tune into, so it wouldn't have
9 to be outreach to individual schools of public health
10 or like that, that -- I have a sense that we could do more
11 to proactively kind of, I think, speed up that
12 matchmaking.

13 DR. WU: I think that's a great idea. We have
14 started down that road kind of coalescing all of this data
15 of like, you know, who's in the study, what panels do we
16 measure, what are the kinds of questions we've asked. And
17 we do have that in a database. I think there's another
18 step to that we have -- which we have done for CARE, where
19 we've started to just do a quick analysis of, you know,
20 who answered this question and what kind of variability
21 are we seeing? So these are the questions that will be
22 useful for some kind of analyses.

23 So it's quite an effort to go back and do that,
24 but I think we have -- it's one of those things on our
25 to-do list that we want to come out with this menu, so

1 that we can say, you know, here what's available to you,
2 researchers, and what are questions that you might want to
3 be interrogating our data for.

4 CHAIRPERSON SCHWARZMAN: Great. Thank you.

5 At this point, I want to pass it over to Sara.
6 And as she mentioned, if we move through the next part of
7 the meeting quickly and there's still -- I'll check in at
8 the end of Sara's presentation, if there are un -- if
9 there's sort of unfinished business from this discussion
10 that we can reopen before we close the meeting.

11 So I want to pass it over to Sara for the plan
12 for the 2022's Scientific Guidance Panel meetings. Sara
13 Hoover is Chief of the Safer Alternative's Assessment and
14 Biomonitoring Section in OEHHA, and she'll provide a brief
15 overview of that plan.

16 (Thereupon a slide presentation.)

17 MS. HOOVER: Thank you, Meg. And I will also add
18 that after I finish my brief presentation, we could also
19 call for open public comment. And then you could clear
20 both the items and turn back to this discussion, if that
21 seems reasonable.

22 Okay. I'm going to give this a shot. My first
23 try in -- let's see now. This is interesting. I have it
24 open. Okay. I'm just going to share my screen and
25 navigate to my PowerPoint.

1 Let's see, slide show from beginning. Okay. Can
2 everyone see this?

3 DR. MARDER: We're still seeing your I think
4 Teams screen.

5 MS. HOOVER: Okay. This is why we practice
6 ahead. Okay. Let me stop sharing, and -- so, Elizabeth,
7 when I pick the share screen, it did not give me -- okay.
8 Now it's giving me the PowerPoint option. All right.
9 Let's try that. Okay.

10 DR. MARDER: And now we see your PowerPoint.

11 MS. HOOVER: There you see it. Fantastic.

12 It wasn't -- that window was not coming up.

13 Okay. Really briefly. Normally, every November,
14 we talk about possible topics for the next year's
15 meetings. And in conferring with my team, with Nerissa's
16 team, with other Program leads, with our management, we
17 realized that we want to take a simpler approach in 2022
18 for a number of reasons. And those reasons are, number
19 one, my team and at OEHHA we're spending our time on AB
20 617 biomonitoring. We're launching the Stockton project
21 this week and we're also going to be working on another
22 project in the coming year.

23 Meanwhile, at CDPH and DTSC, they're busy working
24 on implementing the new budget augmentation and hiring
25 people. So we just realized we have to go to a simpler

1 model. So to start with, thank you to the Panel members
2 for responding to a couple surveys. We pinned down our
3 three dates for next year. They're all going to be
4 half-day meetings from one to four p.m. on March 25th,
5 July 22nd, and November 18th. And those are all Fridays.

6 Fortunately, even though the Bagley-Keene
7 exemption of not having to meet in person is going to
8 expire. At this moment, it's slated to expire in January.
9 However, given the nature of the meetings we're having,
10 we're still going to be able to join -- have attendees and
11 Panel members join via Zoom webinar. We will set up a
12 meeting room for each meeting in case the public wants to
13 come to a meeting room, where they will then watch the
14 webinar.

15 --o0o--

16 MS. HOOVER: So we're just planning to have a
17 very simple standing agenda for all three meetings, where
18 Nerissa would give her Program update. Susan Hurley would
19 give the AB 617 biomonitoring update, and then we'd really
20 just have an open discussion with Panel members, Program
21 staff, and the audience about whatever issues we're
22 confronting in our work at that time. Then we'd also make
23 sure to have some dedicated time for specific Panel input
24 as well as public comment. And that's the plan for 2022.

25 I also want to remind everybody on this call and

1 in general that it's always possible to submit public
2 comment on any topic to the Program to our Program email.

3 So I'll just stop there, and see if before I
4 close the slides, if anybody has any questions about this
5 plan for me, either from the Panel or the audience?

6 And any comments about the plan or if it seems
7 reasonable. I should add -- I'm sorry. I should add one
8 other thing which is that if there were a specific topic
9 that came up, we could always consider scheduling that.
10 So this is the standing agenda, but, you know, we're not
11 banning the possibility of talking about other things.
12 There might -- something might come up that we all feel is
13 important to address.

14 CHAIRPERSON SCHWARZMAN: Tom, you had a question
15 or a comment.

16 PANEL MEMBER MCKONE: A brief question. So it
17 sounds like these are going to be a hybrid meeting where
18 there will be a room?

19 MS. HOOVER: Exactly. It's a hybrid.

20 PANEL MEMBER MCKONE: And then so are the --

21 MS. HOOVER: I'm calling it the hybrid model.

22 PANEL MEMBER MCKONE: And the Panel members do
23 have the option? I mean, if I'm -- if it's easy to get
24 there, if it's local, like over in Richmond.

25 MS. HOOVER: Sure. Yeah. I think what we

1 probably would do is set up a room in the Oakland building
2 of OEHHA. And, yeah, absolutely, Panel members will be
3 welcome to join there.

4 PANEL MEMBER MCKONE: Okay.

5 CHAIRPERSON SCHWARZMAN: Would -- as results
6 continue to kind of come out from analysis of CARE and
7 things like that, is that what would be included in the
8 Program update? Would that be a chance to sort of see
9 some snapshots into that?

10 MS. HOOVER: Yeah. Definitely.

11 Okay. Well, I don't see any other questions.
12 Cheryl or Shoba, are there any public questions or
13 comments on this?

14 DR. IYER: I'm not seeing any attendee hands up,
15 no.

16 DR. HOLZMEYER: And I don't see any new emails.

17 MS. HOOVER: Okay. Great. So again, if anybody
18 does think of something later, feel free to email us. And
19 that is the end of that presentation. Thank you very
20 much.

21 CHAIRPERSON SCHWARZMAN: Great. Thank you, Sara.
22 And I just want to -- it's sort of like a good moment to
23 note the tremendous amount of effort that goes into
24 preparing these really rich and informative meetings
25 from -- on the part of the staff of Biomonitoring

1 California, and all of our guest presenters, and
2 discussants. And I think we've all benefited enormously
3 from that, but that it's also important to recognize how
4 much work they are, and that we'll -- it will be
5 interesting to try on for size this new format that
6 hopefully will reduce the burden a bit on staff, and --
7 and let's see how it -- what kinds of meetings it produces
8 and we can go from there.

9 Carl Cranor, did you have a question or a
10 comment?

11 Carl, were you going to make a comment or no?

12 PANEL MEMBER CRANOR: Yes, I was muted. These
13 were great presentations and efficient. Thank you.

14 CHAIRPERSON SCHWARZMAN: Great.

15 Tom. Webster, that is.

16 DR. WEBSTER: Hi. Yeah, I was -- I wanted to
17 comment a little bit on expanding the definition. So like
18 I said before, I have one of my PhD students is looking at
19 the different definitions, and in particular, with the
20 implications they have for organofluorine,
21 pharmaceuticals, and pesticides. And I'm not saying it's
22 a good or a bad thing. But one of the consequences of
23 going to the OECD def -- the new OECD definition is you
24 would pull in a large number of high-volume fluorinated
25 pharmaceuticals. And that's going to cause IRB problems,

1 so just be aware. You know, if you do it, have your eyes
2 open.

3 CHAIRPERSON SCHWARZMAN: So I want to say
4 something about the rest of the meeting. We have 15
5 minutes, if we need it, and there's a few things that we
6 need to do during that time. One is I want to open public
7 comment. We have 10 minutes allotted for the public
8 comment period and this is an opportunity to comment on
9 any topic related to Biomonitoring California. It doesn't
10 have to be constrained to the topic of today's meeting.

11 And a reminder that if you're attending via
12 webinar, you can submit written comments or questions in
13 the Q&A function or by email to
14 biomonitoring@oehha.ca.gov. You can raise your hand via
15 the Zoom function and we'll call on you to speak your
16 comment.

17 There's two comments that I want to flag that
18 were posted on -- or links to which are available on the
19 November meeting page under the open public comment
20 section, and those are both by Dr. Ahimsa Porter Sumchai
21 of the Hunters Point Community Biomonitoring Program. And
22 that commenter submitted two links, one is called
23 "Unraveling the Breast Cancer Conundrum in San Francisco",
24 and the other is, "Biomonitoring Saves a Life". And so
25 both of those public comments are available via links from

1 the website.

2 And then a third comment that was emailed to the
3 Program is just following up. It's from Sharyle Patton
4 and just following up on a comment dropped in the Q&A
5 about the two pesticides that contain PFAS. And the
6 details -- the comments includes the details on those two
7 pesticides and Biomonitoring California has information
8 now. So rather than share all the content, I just want to
9 refer to it.

10 So that's to acknowledge the three public
11 comments that have come in. And I want to pause for a
12 moment to see if there are any public comments submitted
13 by email. I don't see any attendees with hands raised or
14 anything in the Q&A.

15 DR. HOLZMEYER: There are no new emails.

16 CHAIRPERSON SCHWARZMAN: Okay.

17 So aside from -- assuming that we don't have to
18 wait. There's no lag in submitting public comments, the
19 only remaining thing that we have is if there's additional
20 comment and discussion from our -- from our discussion
21 period that we didn't get to before the time came for
22 Sara's presentation about meetings in 2022.

23 So I want to leave a moment here for any
24 Panelists or attendees to raise hands or drop a comment or
25 question in the Q&A before we adjourn the meeting?

1 Any thoughts that that discussion triggered
2 around the topic of PFAS and how Biomonitoring California
3 can contribute to understanding sources of PFAS and how to
4 reduce exposures?

5 Sara.

6 MS. HOOVER: Yeah, I'll just chime in briefly,
7 since no one else is. I am curious about the Panel's take
8 on the possibility of expanding the definition. As I
9 said, I've raised the issue of considering other
10 fluorinated compounds. I have not been an advocate for
11 expanding the definition in part, because of the cautions
12 that Tom Webster raised, but I would be interested to hear
13 the Panel's thoughts on that particular question.

14 CHAIRPERSON SCHWARZMAN: Anyone have
15 contributions to that at this moment? Jenny, did you have
16 something?

17 PANEL MEMBER QUINTANA: Just to say I agree with
18 Dr. Webster.

19 CHAIRPERSON SCHWARZMAN: And that's about the
20 complication of --

21 PANEL MEMBER QUINTANA: About the complications
22 and maybe getting too diffuse as well.

23 CHAIRPERSON SCHWARZMAN: It's reminiscent to me
24 of the difficulties that arise around non-targeted
25 screening and how to handle illicit substances and/or

1 prescription substances, pharmaceuticals, and all of that.
2 It's a little bit reminiscent of that.

3 Veena.

4 PANEL MEMBER SINGLA: I -- my thoughts are that I
5 think that we'd just want to make sure that the current
6 definition does capture all of the PFAS that could be of
7 interest related to the kind of exposure types and sources
8 we're interested in, you know, including some of the
9 components that go into fluoropolymers. And I think it
10 would be worthwhile to just take a little bit of a closer
11 look at that question in terms of the definition, and that
12 certainly the listing could be written in a way to exclude
13 pharmaceuticals, if that's not of interest, similar to how
14 halogenated organic chemicals used as flame retardants is
15 very specific to chemicals used as flame retardants.

16 CHAIRPERSON SCHWARZMAN: Yeah. That's something
17 I've appreciated about the Program's class definitions is
18 that they're not -- they haven't been like strictly --
19 they've managed to span that distance between like is it
20 strictly a sort of molecular definition or is it -- or is
21 it also a use definition. And I've appreciated how the
22 Program has kind of spanned that divide in the past.

23 Maybe it's just a vote of confidence for the
24 Program's capacity to do that.

25 Sara, did you have a comment?

1 MS. HOOVER: Yeah, if I can just chime in to say
2 that I will take note of that suggestion, and I'm actually
3 really interested in this question. I've been looking at
4 it a lot. I know Tom has been looking at it a lot. So
5 I'll plan to take a closer look and see, you know, because
6 I agree there are some things that are missed through the
7 Buck et al. definition, but there might be another way to
8 handle that rather than changing -- I've resisted changing
9 that definition, because that is the definition that
10 established the class of PFASs. So my idea would be to
11 instead, well, is there another group of fluorinated
12 compounds that we want to bring in, and how would be --
13 what would be the best way to do that. So why don't --
14 why don't I go back to that to, you know, look at that.
15 I'll confer with Tom and others about it and we'll just at
16 some point report back on what we found. Does that sound
17 good? Is that okay?

18 CHAIRPERSON SCHWARZMAN: That's great.

19 MS. HOOVER: Okay. Great.

20 CHAIRPERSON SCHWARZMAN: Tom Webster, did you
21 have a comment?

22 DR. WEBSTER: Yeah. I was going to say that I
23 actually really like what she just said, that I don't
24 think the Buck definition is really comprehensive enough
25 for what you want. But you need to think hard about what

1 is it that your organization wants to get out. That's the
2 point about a definition is what's the purpose of it? And
3 I -- you probably don't want to include fluorinated
4 pharmaceuticals. I don't think that makes a lot of sense
5 for you, but you might want to include, you know, liquid
6 crystal monomers, for example, if people might be exposed.
7 I don't know. I think it's worth sort of thinking about
8 fairly seriously.

9 CHAIRPERSON SCHWARZMAN: Thank you.

10 Jenny.

11 PANEL MEMBER QUINTANA: Was Sara ahead of me to
12 make a comment?

13 CHAIRPERSON SCHWARZMAN: I think Sara made her
14 comment.

15 PANEL MEMBER QUINTANA: Oh. Okay. I was hoping
16 I could make a really quick open public comment, and then
17 for something for maybe another session to discuss this so
18 that --

19 CHAIRPERSON SCHWARZMAN: Sure. And -- but you
20 can make a comment at any time.

21 PANEL MEMBER QUINTANA: Okay. Well, I can hold
22 it if you want to wrap-up the meeting.

23 CHAIRPERSON SCHWARZMAN: No, that's good. Now is
24 good.

25 PANEL MEMBER QUINTANA: Okay. I just want to

1 make a -- throw out for a future discussion for our
2 Guidance Panel that we think about if we want to stick
3 with pure biomarkers of exposure because of a recent study
4 in our updates where we might be looking at biomarkers of
5 early genetic damage, for example, which is kind of a
6 departure for the study for our Program to look at
7 anything but biomarkers of pure exposure. So I just want
8 to throw that out there that we might want to have a
9 discussion about it.

10 MS. HOOVER: Can I chime in on that, Meg?

11 CHAIRPERSON SCHWARZMAN: Yeah.

12 MS. HOOVER: So just to clarify, Jenny, if you're
13 referring to the AB 617 study, remember that that study
14 spans -- it goes beyond just Biomonitoring California, so
15 we're funded to support the AB 617 mandate. The
16 biomonitoring -- the exposure biomonitoring is run under
17 Biomonitoring California, but we have other funding, so I
18 wouldn't say that we're actually expanding in
19 Biomonitoring California beyond what we've traditionally
20 done. I don't know if that's helpful.

21 I will also note that technically, in terms of
22 our purview, we can choose whatever biomarkers we think
23 are reasonable as indicators of exposures to chemicals on
24 the designated list, for example, so that's another way to
25 look at it.

1 PANEL MEMBER QUINTANA: I don't want to take up
2 too much time. Just for another -- for another meeting
3 perhaps.

4 CHAIRPERSON SCHWARZMAN: Great. And Jenny, did
5 you have an additional comment?

6 PANEL MEMBER QUINTANA: Me? No. Sorry, my dog
7 is barking.

8 CHAIRPERSON SCHWARZMAN: It's okay. I thought
9 you said you had had two. No worries.

10 Okay. I don't see any other hands raised. But
11 now is the moment if anyone has any last contributions
12 before we wrap-up the meeting?

13 In that case, I will do the final announcement
14 that the transcript of this meeting will be posted as
15 usual on the Biomonitoring California website when it's
16 available. Our next meeting, as Sara mentioned, will be
17 on March 25th, 2022 from one to four p.m. and attendees
18 will be able to join via Zoom webinar or at a meeting room
19 that will be announced.

20 I want to thank Biomonitoring California staff
21 for putting together this meeting. I want to thank all
22 the presenters who brought such rich content to the
23 discussion, and all of the audience members who
24 participated, and, of course, to the Panel with a
25 especially hearty thank you to Veena for everything that

1 you have contributed to our conversations over the past
2 few years, more than that, while you've been a member of
3 the Panel. And I understand you'll still be involved, but
4 I'll just be sorry not to have you as a -- as a fellow
5 Panelist here. But thank you so much for everything you
6 brought to the Program.

7 And with that, I'll adjourn the meeting and we'll
8 see you in March.

9 (Thereupon the California Environmental
10 Contaminant Biomonitoring Program, Scientific
11 Guidance Panel meeting adjourned at 4:27 p.m.)

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CERTIFICATE OF REPORTER

I, JAMES F. PETERS, a Certified Shorthand Reporter of the State of California, do hereby certify:

That I am a disinterested person herein; that the foregoing California Environmental Contamination Biomonitoring Program Scientific Guidance Panel meeting was reported in shorthand by me, James F. Peters, a Certified Shorthand Reporter of the State of California, and thereafter transcribed under my direction, by computer-assisted transcription.

I further certify that I am not of counsel or attorney for any of the parties to said meeting nor in any way interested in the outcome of said meeting.

IN WITNESS WHEREOF, I have hereunto set my hand this 29th day of November, 2021.

JAMES F. PETERS, CSR
Certified Shorthand Reporter
License No. 10063