CALIFORNIA ENVIRONMENTAL CONTAMINANT BIOMONITORING PROGRAM

(BIOMONITORING CALIFORNIA)

SCIENTIFIC GUIDANCE PANEL MEETING

CONVENED VIA HYBRID FORMAT BY:

OFFICE OF ENVIRONMENTAL HEALTH HAZARD ASSESSMENT

CALIFORNIA ENVIRONMENTAL PROTECTION AGENCY

STATE OF CALIFORNIA

CALIFORNIA ENVIRONMENTAL PROTECTION AGENCY

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June-Soo Park, PhD, Chief, Biomonitoring Branch, Environmental Chemistry Lab

ALSO PRESENT:

Ken Szutu, Citizen Air Monitoring Network

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PROCEEDINGS

ACTING DIRECTOR EDWARDS: Good morning, everyone. I would like to welcome the Panel members and the audience to the July meeting of the Scientific Guidance Panel for Biomonitoring California, more formally known as the California Environmental Contaminant Biomonitoring Program. Thank you all for joining us today.

The Panel last met on March 20th, 2024. The March meeting included updates on Biomonitoring California Program activities, including initial results of a project to assess associations between per- and polyfluoroalkyl substances levels in serum in Southern California adults and in drinking water. The Panel also heard from guest speakers on challenges and opportunities for biomonitoring for oil and gas exposures.

Key discussion topics included: examining variables including drinking water sources that may be influencing the concentrations of PFASs in participants from the California Regional Exposure Study, or CARE study; using non-targeted laboratory methods to capture fluorinated compounds that are not detected by the Water Board's targeted methods to help identify PFASs in drinking water; and important considerations when planning a biomonitoring study in communities living near oil and gas facilities, including: local exposure sources and

identification of chemicals associated with oil and gas extraction activities; potential use of carboxylic's metab -- carboxylic metabolites of PAHs to identify petrogenic sources of PAHs compared to pyrogenic sources; and study of population selection and consideration of planned phase-outs of oil and gas facilities in California.

The summary and transcript of the meeting is posted on the March meeting page on the Program's website at biomonitoring.ca.gov.

Following the March meeting, we had a -- we held a reception to celebrate the 15th anniversary of the -- of Biomonitoring California. Program leaders, past and present SGP members, and friends of the Program provided brief remarks at the event, highlighting some of the key accomplishments of the Program in its first 15 years.

I would also like to announce that OEHHA Director Lauren Zeise retired at the end of June. She had been involved with Bio -- the Biomonitoring California Program since its inception, first as Chief of the Reproductive and Cancer Hazard Assessment Branch, where the Biomonitoring Section is housed, and later in 2016, as OEHHA's Director.

During her time at OEHHA, she played an integral role in guiding the Biomonitoring California Program and

supporting the mission and goals of the Program and the SGP. We want to thank Lauren for her leadership and guidance and for her service to the people of California. We wish her the very best in her future endeavors.

Following Lauren's departure, I will be serving as OEHHA's Acting Director until a new Director is appointed.

All right. So, first off, I wanted to announce that Panel Member Ulrike Luderer will be acting as the SGP Chair for this meeting. And I will now invite Panel members to introduce themselves by name and affiliation. We'll start with Jenny Quintana who is attending remotely. Jenny has been granted a reasonable accommodation to attend this meeting remotely and to not appear on camera for the whole meeting. Her remote attendance will count towards the requirement that a majority of the members shall be physically present at the same teleconference location.

Jenny.

PANEL MEMBER QUINTANA: Hi. My name is Penelope, or Jenny, Quintana. I'm a Professor of Public Health in the San Diego State University School of Public Health.

ACTING DIRECTOR EDWARDS: Great. Thanks, Jenny.

I will now call on Amy Padula who is also attending remotely.

PANEL MEMBER PADULA: Hi. My name is Amy Padula. I'm an Associate Professor in the Department of Obstetrics, Gynecology, and Reproductive Sciences at the University of California, San Francisco.

ACTING DIRECTOR EDWARDS: Great. I'll now start to my right with Carl Cranor.

PANEL MEMBER CRANOR: Carl Cranor. Until July, I was a Professor of Philosophy and faculty member in environmental toxicology. Now, I will be known as a Professor of the Graduate Division at UC Riverside, because I'm retired officially.

ACTING DIRECTOR EDWARDS: Thanks, Carl.

Next, we'll go over to Tom McKone.

PANEL MEMBER McKONE: I'm Tom McKone. I'm Professor Emeritus of Public Health at the University of California, Berkeley, School of Public Health.

ACTING DIRECTOR EDWARDS: Thanks, Tom.

Oliver Fiehn.

PANEL MEMBER FIEHN: My name is Oliver Fiehn. I am a Professor at the University of California in Davis in the Genome Center.

ACTING DIRECTOR EDWARDS: And Ulrike.

ACTING CHAIR LUDERER: My name is Ulrike Luderer. I'm a Professor in the Department of Environmental and Occupational Health in the School of Public Health at UC

Irvine.

ACTING DIRECTOR EDWARDS: Great. Now, I will hand this off to Acting Panel Chair Ulrike Luderer who will provide more details about today's meeting.

ACTING CHAIR LUDERER: Thank you, Dave.

So I want to, first of all, just remind Panel members to please comply with the usual Bagley-Keene Open Meeting requirements that all discussions and deliberations of the Panel about the subject matters at issue today need to be conducted during the meeting, not on breaks or with individual members of the Panel on- or off-line, including via phone, email, chats, or text messages.

Panel members who have not been granted a reasonable accommodation and are attending remotely must appear visible on camera during the open portion of the meeting. If you are unable to keep your camera on during the meeting because it is technologically impracticable, please make an announcement when you turn your camera off. Additionally, if someone older than 18 is in the room with Panelists who are attending remotely, you must disclose the presence of that person and their general relationship to you.

Can the remote attendees confirm whether anyone over 18 years old in the -- is in the room with them?

I see heads shaking.

PANEL MEMBER PADULA: No one -- no one is in the room with me. Thank you.

(Laughter).

ACTING CHAIR LUDERER: Okay. Thank you.

PANEL MEMBER PADULA: I'll let you know if that changes.

ACTING CHAIR LUDERER: All right. Thank you.

PANEL MEMBER QUINTANA: No one is in the room either.

ACTING CHAIR LUDERER: Thanks.

Then I'd like to next announce the Panel goals for the meeting. So first, we're going to hear an update on Program activities, including findings from the East Bay Diesel Exposure Project, or EBDEP, and laboratory method development for analysis of volatile organic compounds, VOC, metabolites in urine. And the Panel will also hear from Program staff and OEHHA on the monitoring of perfluoroalkyl and polyfluoroalkyl substances, known as PFASs, in human populations and fish species in California, and trends of PFASs and persistent organic pollutants, or POPs, among pregnant Californians later this afternoon.

There will be time for questions from the Panel and audience after every presentation. And if any of the

SGP members wish to speak or ask a question, please raise your hand. I'll call on you at the appropriate time and then you can ask your question or provide your comment.

If online webinar attendees have questions or comments during the question periods after each talk, you can submit them via the Q&A Feature of Zoom webinar or by email to biomonitoring@oehha.ca.gov. We won't be using the chat function during this meeting. Please keep your comments brief and focused on the items under discussion. Relevant comments will be read aloud and paraphrased when necessary.

If online attendees wish to speak during the public comment periods and discussion sessions, please use the "raise hand" feature in Zoom webinar and Rebecca Belloso or Stephanie Jarmul will call on you at the appropriate time. If you are attending in person and wish to comment during the public comment periods and discussion sessions, please come to the front or raise your hand and I will call on you at the appropriate moment. For the benefit of the transcriber, please clearly identify yourself before providing a comment and write your name and affiliation on the sign-in sheet at the back of the room.

So now, I'd like -- it's -- to introduce Nerissa Wu. Nerissa Wu is Chief of the Exposure Assessment

Section in the Environmental Health Investigations Branch, EHIB, at the California Department of Public Health and the overall lead for Biomonitoring California. She will provide us an update on current Program activities.

(Thereupon a slide presentation).

ACTING CHAIR LUDERER: Nerissa.

DR. NERISSA WU: Hi. Slide set up here thank you. Welcome, everyone, and congratulations, Carl, on your retirement and to Lauren as well, if you're listening in.

I thought I'd start this update with a reminder of our overall programmatic goals just as a way of structuring the Program update. So as I go through the presentation, I'll be talking both about progress we're making as a program overall, as well as on specific projects.

[SLIDE CHANGE]

DR. NERISSA WU: So our Program goals and priorities are generally derived from mandates in legislation. We regularly take input from this Panel and from stakeholders, and also from our respective departments. And the last time we talked about Program priorities, which was a couple years ago, we identified these as the primary goals.

So surveillance, or measuring exposures in a

representative sample of Californians. This is the primary part of our legislation. And surveillance would enable us to assess time trends in the context of public health programs and legislation, and also broader phenomena like climate change, which is one of the priorities that this Panel specifically identified. We also want to focus on highly exposed communities and work on mitigating inequities. We want to identify and evaluate strategies for exposure reduction. And we have the long-term goal of expanding the reach and sustainability of the Program.

So one of the constants of Biomonitoring California is that we have this very complex and challenging mission, but we are a relatively small program. So one of the other things we've talked about over time is the need to really lean into collaborations to really amplify the impact that we can have as a small program by creating meaningful partnerships.

[SLIDE CHANGE]

DR. NERISSA WU: So looking at our goals one by one. Again, as I said, one of our primary goals is to look at exposures in a representative sample of Californians and to develop representative, generalizable data that we can use to look at time trends and the effect of environmental policies and programs. So study design

and participant selection are really critical to generalizability of surveillance data. And the gold standard for surveillance is random participant selection and a sufficiently robust participation rate, so you're not introducing bias into your data by over- or underrepresenting parts of the population.

And this is really a challenge, because participation in surveillance, even for very well established surveillance programs like NHANES, the participation rate has been going down over time. This is something that biomonitoring programs everywhere are dealing with. We do ask a lot of our participants, not only to answer questions, but to provide urine and blood samples. So we need to make those participant protocols as acceptable as possible, so as not to discourage participation.

It's also really important as we develop surveillance to develop a system that's stable. We've learned something from each one of our surveillance efforts and created valuable data, but it is a challenge to compare data across studies, because the study design changes. So our goal is to create a surveillance system that gives us a stable ongoing source of data that we can use to understand population exposures and trends.

[SLIDE CHANGE]

DR. NERISSA WU: So our primary surveillance efforts to date have been the California Regional Exposure, or CARE, Study and STEPS, Studying Trends in Exposure in Prenatal Samples, which is currently in progress. And we're starting to plan for what surveillance will look like after STEPS.

We've presented here the data generated by CARE. We have STEPS summary statistics, as well as Toki Fillman's work to look at associations between drinking water and serum PFAS levels in CARE participants. And we still have this rich data set with metals and other analytes, exposure questionnaires, and other information. So we have a lot of analyses in the work, which you'll hear about over time.

[SLIDE CHANGE]

DR. NERISSA WU: And in STEPS, the laboratory analysis for STEPS has started. We focused first on Orange County samples, about half of which have been analyzed so far. And we're hoping that in the next year we'll be able to present that time trend data for Orange County.

So the plan for sample collection for STEPS is to continue through 2027 and the lab work will continue subsequently. And STEPS is going to give us really informative data on PFAS levels in Californians. We have

a number of collaborations being developed to look at STEPS to examine the exposure sources, to look at newborn outcomes, and potentially to understand the unmeasured PFAS fraction.

But using samples for prenatal samples, as STEPS does, has a number of advantages and disadvantages. The samples are easy to obtain. We can do random selection of sampling, which is all great, but there are limits on what we can measure. We only have serum and it's a fairly small volume and we don't have exposure information from participants. Although, we do have residential address and can do linkages to drinking water source and other exposure sources.

So as I said, we'll continue using STEPS for the foreseeable future.

[SLIDE CHANGE]

DR. NERISSA WU: But our goal for the next surveillance effort is that we'll have a combination of all the best attributes of our prior surveillance efforts: randomized population-based participant selection; collection of urine, blood, and serum in sufficient volume for each of those; and the ability to collect exposure data through participant questionnaires.

So our plan right now is our hope to work with an established surveillance program which would give us a

frame for random sampling. And that gives us the ability to weight the data and come up with generalizable population data. Working with an established surveillance program also enables us to link to data that they're collecting, which really amplifies our ability to do data analysis. So we still have a lot of details to work out with regard to how we'll interface with an existing surveillance project. And, of course, there's always a challenge of collecting samples in field work. So there are a lot of details to come, which is why it's not scheduled to start up before 2028. But as we develop more details, we'll bring those to this Panel and solicit input.

[SLIDE CHANGE]

DR. NERISSA WU: The second goal I wanted to highlight is identification of highly exposed communities and working towards equity. So through our AB 617 projects and the Asian/Pacific Islander Community Exposures, or ACE, Project, we've been focusing on specific demographics or geographic communities and the unique exposures that they're facing. You'll hear about ACE and the East Bay Diesel Project later today, so I'm just going to give a brief update on the other three projects.

[SLIDE CHANGE]

DR. NERISSA WU: For BiomSPHERE, biomonitoring component of the San Joaquin Valley Pollution and Health Environmental Research Study, we have data on the urinary biomarkers of response and staff are getting ready to return those results to participants. Staff are also working to evaluate air monitoring and questionnaire data. And we have just received the PAH and VOC data. Staff are just starting to look at that.

For FRESSCA-Mujeres, the Farmworker women and Respiratory Exposure to Smoke from Swamp Cooler Air, similarly staff are looking at air monitoring and questionnaire data. The urine samples are at the lab for analysis right now and there is a community meeting being planned for August 2024 to talk about air monitoring results.

[SLIDE CHANGE]

DR. NERISSA WU: Last time we met, we reported on SAPEP, the Stockton Air Pollution Exposure Project. And at the time, there was some question about the 2-NAP results. It was unclear if it might be related to naphthalene and perhaps carbaryl exposure. The lab was able to do some additional analyses to determine that the exposure is likely not carbaryl, but there still is an outstanding question of why the results were elevated.

The results have been returned to participants

and a community meeting to discuss the results was held in April, but we're continuing to work on those results and we'll report back as we have other findings.

[SLIDE CHANGE]

DR. NERISSA WU: On the goal of identifying exposure sources for all of our studies beyond trying to demonstrate a measure of chemical exposures, our primary goal is to determine how people are being exposed. And this is important, so that we can work on reducing exposures on the individual level. You know, what can I do today to reduce my exposures? We have the community level and we also have the policy level, where should we be focusing regulatory or legislative efforts.

So we've always collected questionnaire data and residential address, but the Program now has more capacity to conduct the epidemiology needed to identify exposure sources. You heard last month about the association between drinking water and serum PFHxS levels. Today, you're going to hear about consumption of fish and PFAS levels. And in all of our AB 617 work, we have air monitoring data to help us interpret and understand exposures.

[SLIDE CHANGE]

DR. NERISSA WU: Biomonitoring is really multi-disciplinary and complex. And so one of the ways

we've really been expanding our reach and impact is through our partnerships with community organizations, academic researchers, and other State programs, synergistically working together on our mutual goals. And this increased focus on reaching outward has had the effect in the last year or so that our program is getting more notice. We're getting lots of data requests, which is great. Thank you for sending us your colleagues and students. And there's really an increase in the recognition of how valuable our data is. And the hope is that over time this will translate into more support for the Program and eventually allow us to cover even more ground. The staff is super productive, but we're always -- we're always aware of the chemicals that we're not measuring, all the things that we are not able to study.

[SLIDE CHANGE]

DR. NERISSA WU: And as part of expanding the reach of our Program, the labs continue to expand the number of chemical panels that we can run. So PAH and VOC metabolite assays are now ready for use and you'll hear about the VOC metabolite method this morning and they're continuing to develop methods for PAHs and cyclosiloxanes in serum, as well as non-targeted analysis.

[SLIDE CHANGE]

DR. NERISSA WU: So we're making really good progress on all of our programmatic goals. And in addition to that overview, I want to highlight a few other program activities, including outreach and communication. We've described the CARE Study report in previous meetings. Both English and Spanish versions have been posted on our website. On April 18th, we held an open webinar. We invited CARE participants, community partners, local health jurisdictions and researchers, and other biomonitoring programs.

The webinar was presented in English with real-time Spanish translation. And we had almost 200 participants in the webinar, which was great. Lots of very active dialogue. Lots of questions back and forth, including a discussion through email which continued for some time with some participants, which really demonstrated an engagement and an interest in biomonitoring, both on an individual and a community level.

[SLIDE CHANGE]

DR. NERISSA WU: We also had a public event to discuss the results of the Stockton Air Pollution Exposure Project. Flyers went out to parents of the students at the school where the study was conducted, the All Saints Academy of Stockton. Participant families, community

partners, community members, including teachers from the school, were at the meeting. Again, it was online available in English and Spanish through real-time translation. And there was a good discussion about local exposure sources, exposures in the home and through consumer products. And we got the feedback that people really appreciated the data that was generated and the air filtration that was provided to the school.

[SLIDE CHANGE]

DR. NERISSA WU: Just finishing up with some quick lab updates. EHL is continuing to work on CARE-LA samples so that we will have population level data for speciated arsenic and phenols. We actually just got the speciated arsenic data. We should have phenols later this summer, so we'll be working on summary statistics and getting results back to participants.

The lab has also been busy measuring specific gravity and creatinine to do dilution correction for 194 BiomSPHERE samples. We just about finished up with 155 FRESSCA samples. And now they're working on PAH analyses for FRESSCA-Mujeres.

EHL has also been completing analysis of our Intra-Program Pilot study samples and results are in the queue ready to be reviewed.

And over in ECL, focusing on STEPS, we have a

queue of over a thousand samples waiting for PFAS analyses. And as I mentioned over half of the Orange County samples have been analyzed.

So lots of progress there.

[SLIDE CHANGE]

DR. NERISSA WU: And just additional news, EHLB has been able to add some new machines to the lab, which will enable some more efficient processing of our samples.

[SLIDE CHANGE]

DR. NERISSA WU: So just another way to look at the team's work over time and think about how the Program projects into the future is to look at the various activities related to different stages of study design and implementation. So for any given study, we go from planning to field work and sample collection. We have lab analysis and reporting. We create summary statistics and results return before we dive deeper into the data and look at exposure sources, and finally communicating them out to various audiences. So in order to keep the Program moving efficiently, at any given time, we have to have multiple studies at all different phases of this -- of this continuum.

So we are actively collecting samples in the field with STEPS. And at the same time, we do have samples from STEPS, BiomSPHERE, FRESSCA, and CARE at the

lab. Our epidemiologists are working on the IPP, CARE, BiomSPHERE, and FRESSCA data, getting that back to participants and having it summarized for publication. They're also doing more detailed analysis of CARE, ACE, EBDEP, MAMAS, and SAPEP data.

Our communications team is working to distribute the findings for FREES, ACE, CARE, and SAPEP, while also working on chemical fact sheets and more general public-facing material. And while all of this is going on, we're planning for the future by working on future protocols for community-based studies and for surveillance. And this is what keeps our Program moving forward and looking to the future. Of course, our labs are working on new analytical methods that we can use on all of those new studies.

[SLIDE CHANGE]

DR. NERISSA WU: So this is the team that gets all of this work done. I just have two staff changes to report. Joginder Dhaliwal who's been at ECL for some years has left the Program. So thank you Joginder for all of your work and Dr. Wenlu Song, who's the new Chief of the Inorganic Unit in EHLB. Happy to have you join us. Welcome.

And if you are interested in joining the Biomonitoring team, please visit this website and we'll

have some job openings posted during the break as well.

And with that, I will not show you extra slides. I will take questions.

ACTING CHAIR LUDERER: Thank you very much, Nerissa, for that overview. We'll start with questions -- or more clarifying questions from Panel members and then we'll have time for open discussion after that.

Tom.

PANEL MEMBER McKONE: Thank you. It's always amazing how much is going on, I mean, especially since we went through a couple of years where we couldn't do very much because of COVID, but an impressive list of accomplishments.

In that last slide about the various stages of studies, the planning phase. So early in the -- in your -- I think your second slide or one of the early slides, one of the goals that was added was to do something on tracking how climate change affects exposures or what metrics we could -- you know, we could be looking at for that. Is that in some sort of planning phase or is there a separate program to look at that or is that still like before planning in its --

DR. NERISSA WU: Well, we don't have something explicitly focused on climate change right now. I think there's recognition that in order to really do time

trends, we need to have an established surveillance system that can have an unbiased generalizable population sample. So I think getting that set up will help serve this overall goal of looking at how air pollutants, for example, are changing over time.

ACTING CHAIR LUDERER: Oliver.

PANEL MEMBER FIEHN: Yeah, thank you. I'm equally impressed, but I'm also equally interested in the future, because that's, of course, something we can change or think about. So I think I had two questions. One is if people are staying more and more indoors, what are, you know, the plans to monitor more the indoor activities? You know, as we see -- you know, just picking up on climate change. It's getting hotter and hotter. So people will stay more indoors. So that's the first question. The second question I had is deeper data dive. What are you planning there?

DR. NERISSA WU: Don't you want to wait and find out when we present it?

Well, for the indoor air pollution, that's a good point, that our environment is changing overall, whether it's with related -- related to climate change or other things. I think some of the work being done with the AB 617 communities with air filtration and what people can do to impact their indoor environment is a great step towards

that. I think we're also learning a lot about how those questionnaires should be developed in order to capture the relevant information that we need to know to interpret that, and in addition, the kinds of environmental sampling that we need to do to collect that. So maybe when we're talking about AB 617, somebody could elucidate on that.

In terms of the deeper data dive, I mean beyond the summary statistics, you've seen a lot of modeling work being done to connect, for example, drinking water with PFHxS levels. That involves a lot of geographic analyses. So having the residential address and looking at exposure sources which might be drinking water source, or industrial, or highways, there's a lot of -- I think we have a lot of hopes for the CARE data with that, as well as STEPS, since we do have the residential addresses, but also using our exposure questionnaires. And they're tricky, because you need to ask the right questions and get the right distribution of answers in order to be able to find something in your model. But there is a lot of focus on using that data going forward, as well as the environmental data, to discern what the exposure sources are.

ACTING CHAIR LUDERER: I have a question. And again, looking -- the various studies in progress is really quite impressive and all the work that's been going

on in this program and the work that -- the studies that are coming out, the results. My question was you mentioned that in the future working with established surveillance programs was one goal of the Program. And I'm wondering if you have any surveillance programs in mind that you're thinking about working with.

DR. NERISSA WU: We have talked to a couple, one being the statewide California Health Information Survey, and the other is Maternal and Infant Health Assessment. We're not at a point of agreement. We haven't negotiated a working relationship with them yet, but we are exploring the possibility of working with either of those.

ACTING CHAIR LUDERER: Great. Thank you.

And I see two raised hands. Jenny, would you like to go first and then Amy.

PANEL MEMBER QUINTANA: I think Amy was first actually, temporally.

ACTING CHAIR LUDERER: Okay. I saw your hand first. Sorry.

(Laughter).

PANEL MEMBER PADULA: I'll start, if you want. Either way is fine though. I guess, I just had a quick question about the PAHs that are being measured. It looked like from the slides that they'll all be done in urine. I was wondering if that was indeed true or if any

will be tested in blood samples in the future?

DR. NERISSA WU: Currently, the method we have for PAHs is for urinary metabolites. DTSC staff is working on a PAH parent compound in serum. I don't know if -- June-Soo is here. I don't know if you want to add to that, June-Soo?

We can't hear you up here.

DR. JUNE-SOO PARK: Yeah. Yeah. We are working on it. Dr. Amber Kramer and Eimi, the APHL fellow, working on the PAHs in blood. So we just reviewed the data yesterday. It looks good. They're making good progress. That's all I have.

ACTING CHAIR LUDERER: Jenny.

PANEL MEMBER QUINTANA: Hi. Can we go back to the slide with the vulnerable communities. I think it was one or two slides back, maybe a lot of slides back. Sorry. Yeah. Right here.

I guess one kind of really general thing to think about for the future I think is how is California different from the rest of the country? Like what monitoring would be especially important for us to do that would give information beyond what the NHANES-based CDC analysis is doing? So I think it's worth some thinking about if we're focusing on specific communities to really think about what is different here in California to really

get the most bang for our buck in a sense.

And I know we've discussed this before, but I think some things that make us different are high levels of immigrant communities, a lot of refugee communities. I know in San Diego where I live is one of the centers for refugees for various different areas. And so I think some of those -- and they come with perhaps a higher body burden already, then they're already add -- that they might be adding to, so they might be especially at risk, for example. So I just think we should maybe discuss as we move forward, you know, how best to focus on what is either unique or more prevalent in California, in terms of exposures or populations.

Thank you.

DR. NERISSA WU: Thanks for that comment. Those are really great points. I think with some of the work that Kelly is going to present this afternoon, there's definitely some -- there's definitely concern about populations including immigrant populations that are more impacted by -- because of their diet and because of their reliance on subsistence fishing potentially. Central Valley work is a concern. There's agriculture and a particular geographic structure that makes that area particularly vulnerable. But the other thing that we have in our studies that we cannot get from NHANES is the

geographic analyses, approximated sources, but also some of this behavioral work. So in addition to studying exposures that might not be studied in NHANES, I think the ability to really tease out some of those distinctions is important.

PANEL MEMBER QUINTANA: Thank you.

STEPHANIE JARMUL: Ulrike, this is Stephanie Jarmul. We have one attendee with their hand up and then a comment in the chat. So I'll let Ken unmute himself and speak.

KEN SZUTU: Good morning, everyone. This is Ken, Ken Szutu from Citizen Air Monitoring Network in Vallejo, California. I saw there is one item, which is looking for community to participate. Currently, Citizen Air Monitoring Network has a grant from CARB doing black carbon monitoring in our city. And also at the same time, we work with UC Davis Air Quality Research Center PM speciation. And I think many people in my community are interested in biomonitoring, especially including Vallejo and several surrounding city, which basically we are in a refinery community. So my question is, is there any possibility to join this program and what is the process? Thank you very much.

DR. NERISSA WU: Thanks for your question, Ken. I'm actually going to defer this to Stephanie, because

this is more of an AB 617 question.

STEPHANIE JARMUL: Hi, everyone. Yeah, thanks for that question, Ken, and I think it's kind of similar to the one we got in the chat too, but -- so there's a lot of factors that we consider when choosing to go into a community to conduct a biomonitoring study, but we are always open to having conversations with different community groups, concerned community members about potential collaboration opportunities. And so perhaps we could schedule a meeting after the SGP to talk about your particular situation and what you would hope to achieve with a biomonitoring study.

KEN SZUTU: Okay. Thank you very much.

ACTING CHAIR LUDERER: Stephanie, you said there was another question in the chat, but did you kind of just answer that?

STEPHANIE JARMUL: Well, I'll read it aloud anyways.

ACTING CHAIR LUDERER: Okay.

STEPHANIE JARMUL: How do you identify your community-based studies? Do community members reach out to you directly with concerns or does your team identify possible exposure hot spots from previously collected data?

Well, I can talk a bit about the AB 617 studies.

So it's a bit of a combination. As I was just saying with Ken, there's a lot of factors you have to take into account, such as what is the exposure of concern. You know, are there biomonitoring methods to identify -- you know, if it was metals or, you know, we often look at PAHs, VOCs. But if there's something else that perhaps we don't have the methods for, we'd have to consider that. Also, geographic location, you know, what are the exposure sources nearby, and if it's something with that we can -- we can potentially look at.

And, yeah, I think we are trying to learn from our past studies, just speaking with different communities. And we've conducted a lot of listening sessions with our communities to learn about their exposure concerns and what they hope to see from the Program. So we're trying to work off of that information and identify both community collaborators and academic collaborators that would really help build a robust study. That's it. And Nerissa, if you wanted to add.

DR. NERISSA WU: Yeah, I wanted to add on that, for example, the Asian/Pacific Islander Community Exposures Project was built because we had seen in the data from the BEST and from other studies that Asians were -- had elevated PFAS levels, but it wasn't well understood why that might be and whether there's variability due to

other factors in the Asian community. So that's how the Asian/Pacific Islander Community Study was born.

And we have learned much more about the exposure process. And also, as you'll hear this afternoon both in ACE and MAMAS, we've learned much more about subpopulations in the Asian community.

ACTING CHAIR LUDERER: All right. Thank you. We have some time now for open discussion among the Panel members. Anyone want to start?

Tom.

PANEL MEMBER McKONE: All right. It seems like I always start.

(Laughter).

PANEL MEMBER McKONE: I'd like to go back. And at least some of us have some interest in the climate, how we use biomonitoring with regard to climate change. And I'm thinking that we might want to schedule a period of discussion at a future meeting about what would -- what that might entail. I think we all have some ideas and they're probably a little different. And maybe we should see where we converge and what's possible. Like should we be looking at, I mean, the issue of do people spend more time indoors and would we see that in biomonitoring, but there's also questions about fire -- wildfire exposures going up and what would that look like? Could we find

that? Could we see the trends?

If there's a drought and it affects the availability of water, that could increase water pollutants. I mean, there's a number -- and even quite relevant to today, where I think it's going to be 107 here. I'm glad we're inside. Are there ways to see markers of heat stress? And that would be a bit advanced but maybe that's something we might want to talk about, because it's very realistic that California is going to be seeing more of these long-term heat wave events so is the rest of the country.

So again, these are just some ideas. And I don't think we can resolve -- it's not really appropriate today, but if we could carve out a short period of time about what would we do for climate in a biomonitoring context, and what could we do short-term, what could we do long-term, what would this -- you know, what would this take just so the Panel can engage with the staff on it.

DR. NERISSA WU: Thank you.

ACTING CHAIR LUDERER: Oliver, did you have a --

PANEL MEMBER FIEHN: No.

ACTING CHAIR LUDERER: No. Okay.

I just did have one question kind of going back to, you know, availability of samples that are more population representative. And I know at several meetings

we've talked about developing methods for measuring different analytes in the newborn blood spots. And I was wondering if there is any -- is that still ongoing or not?

DR. NERISSA WU: We are not working on newborn blood spots right now. APHL has actually come out with a statement against using newborn blood spots for biomonitoring, in part because of the technical difficulties and the representation of the blood spot of -- you know, compared to a venal blood sample, but also because of participant rights. Not all states have the same kind of consent process that the California Newborn Screening Program has. And that's created some issues with researchers in other states.

So at this point, we have not continued that. The other thing I guess I would say about newborn blood spots is they are -- you can probably get around this through using part of the card -- the newborn card as a blank, but they are not really stored in a way that is protective from other environmental contaminants. So, I do have concerns about what the blood spot might represent also.

ACTING CHAIR LUDERER: Thank you. I think -- is -- are there any other public comments for this part of the discussion? I just wanted to check with Stephanie and Rebecca about that.

STEPHANIE JARMUL: Nothing online. I can see Amy had her hand up though. I don't know.

ACTING CHAIR LUDERER: I was just about to say that, yes.

Amy.

PANEL MEMBER PADULA: I just wanted to follow up. I agree with the issues with the blood spots, but I was also curious with respect to available samples, whether the maternal samples that are taken for screening tests during pregnancy may be more suitable for biomonitoring and whether there's any plan to evaluate those.

DR. NERISSA WU: We are using samples from prenatal screening, second trimester, that we get from Biobank, both from the MAMAS study, which you'll hear about this afternoon from Dina Dobraca, and also STEPS. That's all based on Biobank samples. So, it's a great resource. It's amazing that we can use the prenatal screening program as a sampling frame and really get a population sample. So we will be able to do some time trend work looking at PFASs over time in several different counties. But they are only -- they are only banked for have seven different counties. And it is a very small volume and it's only serum.

So, as I said, we're going to have amazing data from STEPS. And there's a lot we can do with that data,

but there are all these other chemicals that we would like to look at. Particularly, we're missing all the whole blood and urinary samples. And it has been a challenge to think about how to use that very small volume in the best way. You know, it's precious to us, so we're thinking about pooling. We're thinking about other ways we can leverage this resource, for us much data as we can, but those challenges remain.

The other thing is that the Prenatal Screening Program is changing. It has always been -- or for the last ten years or so has been covering about 70 percent of pregnant women or pregnant Californians. And at this point, that demographic is changing. They have introduced a new screening modality for first trimester, which is outside of the State program. So the sampling frame itself is going to change. It's only a year or two old at this point, so the representativeness is something that we're really paying attention to.

ACTING CHAIR LUDERER: All right, any -- just turning to see if there are any additional comments or questions from the Panel?

And not seeing any, then thank you very much, Nerissa, for that great presentation.

And I would like to introduce our next speaker, Aalekhya Reddam, who is a Research Scientist in the Safer

Alternatives Assessment and Biomonitoring Section at OEHHA. And she's going to give a presentation on additional findings from the East Bay Diesel Exposure Project, EBDEP.

Aalekhya.

(Thereupon a slide presentation).

DR. AALEKHYA REDDAM: Thank you. Good morning, everyone. Today, I will be giving an update on the East Bay Diesel Exposure Project and all the work that's been done since you last heard about the results from the study.

Before I jump into the presentation, I would specifically like to thank Dan Sultana and Kelsey Ranjbar for all the analysis they've done for this project. Thank you guys.

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DR. AALEKHYA REDDAM: So there were a few main project goals for EBDEP. The first one was to assess exposures to diesel exhaust in impacted communities of the East Bay. We also wanted to evaluate predictors of diesel exhaust exposure using predictors, for example, such as traffic density. We wanted to compare daily measurements of exposure biomarkers in parent-child pairs to increase our understanding of exposure patterns, specifically between communities, within communities, over time, and

also within families. We also wanted to evaluate the effectiveness of diesel regulations in California and engage with community and policymakers about study results.

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DR. AALEKHYA REDDAM: Our study population consisted of 40 parent-child pairs. We collected environmental and biological samples and questionnaire and activity data between January 2018 and February 2019. And as you can see in the map over here, we -- our participants were recruited from various cities along the East Bay. We had two sampling rounds in our study to help evaluate exposures over time.

In Round 1, we collected air, dust, and urine samples and in Round 2, we collected just air and urine samples. Our air samples were collected over a span of four days and our dust samples were collected from a vacuum bag or canister. And when that wasn't available, we collected a sweeping sample. From our 40 families, 25 families gave one urine sample per participant per round and 15 families gave four urine samples per participant per round, so that we could evaluate within variability and between subject variability.

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DR. AALEKHYA REDDAM: As I mentioned before, one

of our main goals in EBDEP was to assess exposure to diesel exhaust. 1-nitropyrene, or 1-NP as I'll be referring to during the presentation is formed during the combustion by nitration of polycyclic aromatic hydrocarbons, or PAH, within the diesel engines. It is the most abundant particle associated nitro-PAH in diesel exhaust and is thought to be more specific than other PAHs when indicating exposure to diesel exhaust.

6-hydroxy-1-nitropyrene or 6-OHNP and 8-hydroxy-1-nitropyrene, or 8-OHNP, are urinary metabolites -- are two of the urinary metabolites of 1-NP and are thought to be used as exposure biomarkers to diesel exhaust.

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DR. AALEKHYA REDDAM: As I mentioned before, we collected urine samples, air samples, and dust samples. And most of these samples were analyzed by Chris Simpson's lab at the University of Washington, but the black carbon was analyzed by the Lawrence Lab at UC Berkeley. In our urine samples, we measured 1-NP metabolites, 6-OHNP and 8-OHNP, one of the pyrene metabolites, 1-hydroxypyrene, as well as metabolites of volatile organic compounds.

In our air and dust samples, we measured 1-NP, 2-nitropyrene, or 2-NP, 2-nitrofluoranthene and additionally measured black carbon in our air samples.

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DR. AALEKHYA REDDAM: The preliminary results from EBDEP were presented at the November 2019 SGP meeting and they were also finalized and published last year. This paper included a description of the EBDEP project, demographics of our participants and also correlation and summaries of 1-NP in air and dust and 1-NP metabolites.

The major conclusions were that at least one 1-NP metabolite was present in 97 participants of -- 90 percent of our urine samples. We also saw that urinary 1-NP metabolite levels were generally higher in fall and winter months compared to our spring and summer months. Urinary 6-OHNP was significantly higher in parents compared to children. And the same was true for 8-OHNP as well, but it wasn't significant.

We see that children urinary 8-OHNP were weakly correlated with 1-NP in air and dust, and air and dust 1-NP levels were higher in homes with high CalEnviroScreen diesel particulate matter score and these were our main conclusions.

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DR. AALEKHYA REDDAM: All the EBDEP demographic results were discussed in November 2019 SGP meeting and also presented in the published paper. But just as a quick recap, for the parents, 95 percent of the

participants were female and five percent were male. Whereas, in our children, our distribution was a little more even with 52.5 percent being female and 47.5 percent as male.

The average age of our parents were 36.6 years old and our children were quite young with the average age being 4.7 years old. Lastly, in our parents, five percent were American Indian/Alaskan native or native Hawaiian/other Pacific Islander, five percent were Asian, 20 percent were Black/African American, 40 percent were Hispanic/Latino, 35 percent were White, and 2.5 percent preferred not to say. And the ethnic distribution of our children were very similar to those of our parents.

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DR. AALEKHYA REDDAM: So with that, we had two main aims with our additional analysis that we completed. One was to use 1-NP in air, dust, and its metabolites in urine to examine geospatial predictors of diesel exposure and our second aim was to examine predictors of VOC metabolites.

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DR. AALEKHYA REDDAM: As we didn't have a lot of participants who are exposed to diesel occupationally, we used geospatial predictors. Previous research has shown that spatial characteristics, such as traffic volume, road

density, and population density have been useful in predicting traffic related air pollution. So we thought it could potentially be used to predict diesel exposure. ArcGIS was used to create individual spatial predictor variables for each household for multiple buffer zones. So using our Oakland OEHHA office as a surrogate for our participants residence, we can see the five different buffer zones that were evaluated in the analysis. So we had 150, 350 meters, 500 meters, 1,000 meters, and 2,000 meters.

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DR. AALEKHYA REDDAM: Within these different buffers zones, we used publicly available data, mostly from Caltrans, to generate a suite of geospatial predictors for each individual participant. For example, you can see in this map that the blue dots are bus stops and all the highlighted lines, which are blue, gray, and yellow -- I'm not sure if you can see it very clearly in the room, but those are major roads. We compiled all this data for each buffer zone for each participant -- in relation to the participant.

In addition to major roads and bus stops, we also calculated distances to different types of major roads, the length of bus routes and other types of roads, different types of traffic densities, as well as counts of

permitted sources. An example of these permitted sources are gas stations, or hospitals, or generators, or stores -- approved generators for stores.

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DR. AALEKHYA REDDAM: We then use this data to examine associations with geospatial predictors and 1-NP in air and dust and its metabolites. All the concentrations were log transformed and the urinary concentrations were adjusted for specific gravity. As most households had only one dust collection, we used linear regression models for associations between 1-NP in dust and geospatial predictors. For the few households that had more than one measure, we averaged the dust concentrations.

We used mixed effect models for associations with 1-NP in air, and 1-NP metabolites to account for their multiple measures. And the models with the metabolites were adjusted for relevant covariates. So in adults, the models with metabolites were adjusted for season, income, candle use, sweep frequency, types of food consumption, presence of gas dryer and washer in adults. And in children, the models were adjusted for season. We chose these covariates based on their association with the metabolites and selected ones that we thought were most relevant for the model.

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DR. AALEKHYA REDDAM: So all the data from the results are presented in this table. Each check mark represents a significant association and all the associations were positive. So, for example, in the first row, we see that all traffic densities at each of the five buffer zones were significantly positively associated with dust.

So there's a lot of data here, but I think our main takeaway from this table is that 1-NP in house dust had more significant associations with spatial predictor variables than air concentrations of 1-NP. Dust usually represents a more stable matrix in air and therefore may be more indicative of chronic human exposure. And moreover, geospatial predictors that are further away are thought to contribute more to ambient exposure versus closer radii are thought to contribute more to personal exposure.

We did have one participant that had very high concentrations of dust. I think they were orders of magnitude higher than the next one. And in sensitivity analysis when we've removed this participant, we lose some of the significant associations but the direction is still the same.

Our 1-NP metabolites were only significantly

associated with the length of major roads and only in our adult participants.

8-OHNP was significantly associated with length of major road at 150, 350, 500, and 1,000 meter radius. And 6-OHNP was only significantly associated in a thousand meter radius. When we ran the analyses with the metabolites summed, we see a similar direction, which is positively associated, but we do lose some of the significant associations as well.

[SLIDE CHANGE]

DR. AALEKHYA REDDAM: Now, we move on to our second aim, which was to examine predictors of VOC metabolites. These results were actually just returned to our participants earlier this year. So this is the first time that these results are being presented.

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DR. AALEKHYA REDDAM: Volatile organic compounds, or VOCs, are a class of compounds that are carbon-containing chemicals, which vaporize and can enter the body through normal breathing. And as we can see in this image, there are several indoor and outdoor sources of VOCs.

In EBDEP, 13 VOCs were specifically selected based on their associations with diesel exposure. And we wanted to examine their association with predictors of

traffic related air pollution, once again using our geospatial predictors and also with other participant characteristics and indoor sources using our information from the questionnaires.

As a quick note, the measuring of the VOC metabolites was added a little further on in our project. And therefore, while we have some questions that might be relevant to the concentrations of VOC metabolites, we weren't able to account for all potential predictors.

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DR. AALEKHYA REDDAM: So as I mentioned before, we had 13 VOC metabolites and these were analyzed in the urine by the CDC. And as these were run by the CDC, we have a few more VOC metabolites than our SAPEP and BiomSPHERE studies, which are run by UCSF.

The next presentation is actually by our Environmental Health Lab and they'll talk about their progress with their VOC method, which we hope to incorporate in our future studies, which is really exciting.

From this table, we see that other than one of the benzenes metabolite PMA, the detection frequencies for all of the metabolites were measured above 70 percent. And then other than acrylonitrile's metabolites, CYMA, all levels were higher in children compared to parents.

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DR. AALEKHYA REDDAM: We initially adjusted for metabolite concentrations with creatinine, so that we could compare our concentrations with NHANES. However, for our analyses, we adjusted for specific gravity and log transformed the concentrations. Similar to the 1-NP analysis, we used mixed effect models to account for multiple samples. We initially ran the associations with participant characteristics and relevant predictors and then used the significant associations and ones that we thought were important in the model to look in our models with the geospatial predictors.

For our adults with the geospatial predictor models, we adjusted for season, income, candle use and presence of gas dryers and children, adjusted for only season. Furthermore, when running the models with the participant cities to ensure a more even distribution, we binned the participants into Richmond, Oakland, and all other cities were binned into an Other category, an Other city. And this was again just to ensure more even distribution of participants.

[SLIDE CHANGE]

DR. AALEKHYA REDDAM: When examining the correlation of the VOCs, all of them were positively correlated with each other. However, only a few were very

strongly correlated with each other and we defined this as a Spearman correlation coefficient of more than 0.6. We see that ethylbenzene and styrene metabolites, PHGA and MADA, were strongly correlated with each other, which could be attributed to their common parent compound.

We see that isoprene, acrylonitrile, and 1,3-butadiene metabolites were strongly correlated with each other, and they're often used in conjunction with each other for consumer products to make rubber, so that could possibly be the reason for the strong correlation.

We also see that acrylonitrile metabolite and xylene metab -- one of the xylene metabolites, 2MHA, was strongly correlated with each other, which could also suggest common use patterns. And then acrolein metabolites, CEMA and HMPA, were strongly correlated with each other, which again can be potentially attributed to their common parent compound, acrolein.

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DR. AALEKHYA REDDAM: When -- we then compared our creatinine-adjusted concentrations of the VOC metabolites to levels from NHANES. We compared our parent levels to those of adults in NHANES and our children levels to those in the closest NHANEs reference group, which was between three to five years old. And just as a reminder, our children -- our mean age for our children

was 4.7 years old.

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DR. AALEKHYA REDDAM: So to focus a little bit more, the metabolites that were higher in our EBDEP population compared to NHANES are highlighted. Moreover, there were five metabolites where the 95 percent confidence intervals from the two populations don't overlap and we counted this as significant.

So in our adult populations, we saw higher levels of the metabolites of acrolein, benzene, ethylbenzene/styrene, and propylene oxide in our EBDEP population compared to NHANES. And the isoprene metabolite was significantly higher in our child populations compared to the reference NHANES population of three to five years old.

[SLIDE CHANGE]

DR. AALEKHYA REDDAM: There are potential -- there are several potential sources of the VOCs for which we saw higher urinary biomarker levels in our EBDEP population and a few of them are highlighted here in this table. Four out of the five VOCs shown here are products of combustion fuels or combustion of automobile exhaust. But several of them are also used in the creation of plastics, rubbers, and are detected in our consumer products.

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DR. AALEKHYA REDDAM: When looking at associations between VOC metabolites and demographics, we see that participants with the lowest income had a higher concentration of total VOC metabolites and higher concentration of xylene metabolites compared to participants in the higher income category. And then when looking at city of residence, we see the participants who lived in Oakland had higher concentrations of total VOC metabolites compared to Richmond. And just as a reminder, our participants were binned into Oakland, Richmond, and other.

And lastly, after adjusting for city of residence, our Black participants had higher concentrations of xylene metabolites and benzene metabolites compared to white participants. We did adjust for city in this model specifically, because all our Black participants lived in Oakland and we wanted to ensure that the association results weren't as a result of city residence.

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DR. AALEKHYA REDDAM: We also looked at associations between VOC metabolites and potential predictors based on the questionnaire data. We see that candle use in the past three days was associated with

higher total VOC metabolites in adults and children and higher benzene and isoprene metabolites in adults. We see that gas stove use in the past three days was associated with higher ethylbenzene/styrene metabolite and benzene metabolite in children. And gas washer and gas dryer in the house was associated with higher ethylbenzene metabolite in children and gas dryer only was associated with higher total VOC metabolites in adults.

[SLIDE CHANGE]

DR. AALEKHYA REDDAM: Lastly, we examined associations with the same geospatial predictors that we mentioned before with the total VOC metabolites and found that traffic density was associated with higher total VOC metabolites in adults. And then we also see a similar trend that we saw with the 1-NP metabolites, where the length of major road was associated with total VOC concentration at 150, 350, and 500 meter radius in adults, and 150 meter radius in children.

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DR. AALEKHYA REDDAM: So with all of that, our conclusions from these additional analysis is that 1-NP in air and dust was significantly associated with several geospatial predictors of traffic. And we see that 1-NP metabolites in urine was positively associated with length of major roads. For our VOC metabolites, we see that they

were significantly associated with demographic variables, with gas appliances and candle use, as well as traffic density and length of major roads.

[SLIDE CHANGE]

DR. AALEKHYA REDDAM: I'd like to thank the EBDEP parents and families for participating in this study and our collaborators at the West Oakland Environmental Indicators Project, at the University of Washington, Lawrence-Berkeley Lab, as well as the CDC and all the Biomonitoring California staff.

Thank you.

ACTING CHAIR LUDERER: Thank you very much for that really great overview and interesting presentation. We have time now to start with for clarifying questions. Any from Panel members to start with?

Oliver.

PANEL MEMBER FIEHN: Yeah. Thank you. This is a truly interesting study, but maybe underpowered. So, you know, I mean, I'm not a statistical expert, but I use statistics all the time, including all the models that you've talked about. And, you know, I'm a little concerned about the numbers here. You also sound like, you know, numbers are a little different when you remove an outlier. Well, that's the point, we don't want to remove outliers, because we want to know really the

distribution across a population. And if you then do multiple adjustments, you know, things get weaker and weaker. So in principle, there's two things, one is significance and the other one is effect size. What can you tell us about effect sizes?

DR. AALEKHYA REDDAM: Yeah. And I think something to point out also is even though our sample size were 40 people, we did have multiple measures, so I think altogether we had around 300 samples to work with. So I think we did have a relatively higher sample size. And sorry, could you repeat your question for the effect size, please?

PANEL MEMBER FIEHN: Yeah. So that, you know, it's not just the ability to be -- to say something is significant, but also in terms of meaningful -- you know, instead of meaningful statistics, people say effect size. You know, so is it -- is anything doubled? Is anything -- you know, I mean what is -- what are we talking about here?

DR. AALEKHYA REDDAM: Yeah. Yeah. Of course. So I think for the models where we use the mixed effect models, we did see maybe a 10 to 20 percent, and I'm generalizing here, increased effect size, if that be -- the beta estimate is what we saw.

ACTING CHAIR LUDERER: Tom.

PANEL MEMBER McKONE: Yeah. If you could go back to this slide on predictors of VOCs. I was curious, it says gas stove, but did you consider gas oven use?

DR. AALEKHYA REDDAM: Yeah.

PANEL MEMBER McKONE: I mean, when you say gas stove and oven?

DR. AALEKHYA REDDAM: This was just based on the questionnaires. And as I mentioned before, it was very much tagged onto the end of the study, so I think we didn't go as specific as we would have liked for the VOC metabolites. I think there wasn't a specific question about gas oven that I know of, but if anyone else knows, otherwise, please let me know.

PANEL MEMBER McKONE: Actually, I'm just -- what's a gas washer?

DR. AALEKHYA REDDAM: I think it's probably a washer that uses gas versus electric -- electricity.

PANEL MEMBER McKONE: I've never -- oh, I've never seen a gas washer, but -- I mean, well, the -- and this is a question of water heating would be a key issue, especially if they have -- the water heater is indoors.

DR. AALEKHYA REDDAM: Yes.

PANEL MEMBER McKONE: And a lot of them, especially in smaller residences in communities that aren't as wealthy, the water heater might actually be just

right in the middle of the house somewhere.

DR. AALEKHYA REDDAM: Yeah, absolutely.

PANEL MEMBER McKONE: Okay.

DR. AALEKHYA REDDAM: Thank you.

ACTING CHAIR LUDERER: Carl.

PANEL MEMBER CRANOR: This is a quick question, because I'm puzzled. Do lots of people use candles anymore?

(Laughter).

PANEL MEMBER CRANOR: You're testing for candle exhaust.

DR. AALEKHYA REDDAM: I actually was using a candle when I ran this analysis. So, I don't know. I guess -- I mean, the distribution -- there were a good amount of people that used candles. And also our question was candle and incense use. So I'm wondering if more people use incense versus candles. But there was -- there were a good number of people that used candles -- or answered yes to this question.

PANEL MEMBER CRANOR: Thank you.

ACTING CHAIR LUDERER: I have a clarifying question too, and that is I think the -- you know, the cooking -- the gas cooking was -- is very interesting and obviously an important exposure source inside. Did you ask any questions about sort of mitigation measures, like

whether they had a hood -- a vent hood and whether it vented to the outside and did that make a difference?

DR. AALEKHYA REDDAM: Yeah. No, we did have those questions. We had questions about -- and also how often the vent was cleaned and if it was cleaned based on a question -- follow-up questionnaire. And they didn't make any difference, yeah. And also, I don't think there was a great distribution of variables. I think that could have also played a role in it. And maybe if you had a larger sample size with more people that were in each of those categories, we would have seen more of an effect.

Thank you.

STEPHANIE JARMUL: I do -- this is Stephanie. I do want to maybe let Duyen also who was heavily involved in this. I don't know if you wanted to add to that, Duyen.

DUYEN KAUFFMAN: Yes. Thank you, Stephanie. Duyen Kauffman from Biomonitoring California and I just wanted to clarify some of these questions that we asked. Some of these were the questionnaire and some were based on a home walk-through that we did, but yes, we did ask about candle use and had -- the question was candle, votive, incense, and sage burning all in one question. So we did have people respond during -- affirmatively to that.

And then for gas stove, the question was specifically about stove. We didn't say oven. I think it was implied, but yes, the wording was specifically whether or not you had a gas stove. And we did ask about mitigation, like, you know, a hood range whether or not vented to the outside and how often that was used when they cooked.

And then for the gas washer and dry -- we did ask about a dryer. I don't -- I'm just flipping through the questionnaire and I don't -- we didn't -- we asked specifically for dryer and not a washer for that one. And then also about the presence of the gas water heater, whether or not it was -- it was located inside the house.

DR. AALEKHYA REDDAM: Thank you, Duyen.

ACTING CHAIR LUDERER: Thank you. I see Jenny, you have a question

PANEL MEMBER QUINTANA: Hi. Thank you for the study. Very important. I just had a quick question. I looked briefly at the article. I think you had a question about how many people smoked inside the home and maybe one person did.

DR. AALEKHYA REDDAM: Um-hmm.

PANEL MEMBER QUINTANA: And -- but I didn't see questions about exposure outside the home. And I'm just wondering not so much for 1-nitropyrene, but -- for some

of the other things like acrolein and tobacco smoke. And I'm just wondering if you measured cotinine in the urine.

DR. AALEKHYA REDDAM: I don't think we measured cotinine in the urine. Yeah, we did not. But I think moving forward, that would be a very important thing to measure. Thank you.

ACTING CHAIR LUDERER: Okay. I'd like to --

STEPHANIE JARMUL: Sorry, this is Stephanie.

ACTING CHAIR LUDERER: I was going to ask if there were any questions from the public?

STEPHANIE JARMUL: Not yet, but I did want to mention that all of our current studies SAPEP, BiomSPHERE, FRESSCA we did measure cotinine in the urine. And so we have incorporated it into our current studies.

PANEL MEMBER QUINTANA: Thank you.

ACTING CHAIR LUDERER: Okay. Great. Thank you. We have time now for general discussion about this presentation and related topics. Would anyone on the Panel like to start?

Oliver.

PANEL MEMBER FIEHN: Yes. So going back to this question on statistical power, I was a bit concerned that there were only five percent of the participants were male. And I think this sexual disparity is something that is concerning, because people might have different habits,

but possibly -- so you obviously can't go back in time, but maybe for the future, we have to really make sure that the genders are equally distributed.

DR. AALEKHYA REDDAM: Thank you.

ACTING CHAIR LUDERER: Further comments from Panel members.

Tom.

PANEL MEMBER McKONE: Yeah. I'm looking for the slide where it mentioned the first study was consistent with CalEnviroScreen. I was just curious about that. That's actually a very interesting -- well, it was just one little line there.

DR. AALEKHYA REDDAM: Um-hmm.

PANEL MEMBER McKONE: Oh, there it is. Hair and dust were higher in homes with high CalEnviro -- so that means CalEnviroScreen is useful as a starting point to find out where you're looking. And I guess the bigger question, you know, for other things we're looking at is have we missed an opportunity to use CalEnviroScreen as sort of what it's intended for, a screen to tell us where we should be looking and to sort out, so that we just aren't randomly sampling, but we have some targeted sampling, if that's a good tool.

DR. AALEKHYA REDDAM: Yeah, I think we actually did use CalEnviroScreen to target these populations. And

I think we targeted them based on their score and tried to get a distribution where they had high CalEnviroScreen and lower ones. I also want to clarify that the results -- the interpretation of this last bullet point is that participants with 90 percentile CalEnviroScreen had -- they were in the 90th percentile category of the CalEnviroScreen data had higher concentrations of air and dust. I think the range was relatively higher compared to the other participants.

STEPHANIE JARMUL: This is Stephanie. I can jump in. I'm pretty sure that with AB 617 communities, they are using the CalEnviroScreen data to help identify those AB 617 communities, which we are trying to focus on working with.

ACTING CHAIR LUDERER: I actually have a question about the fall and winter months and the 1-NP metabolites being higher. It's somewhat surprising to me if it's traffic related and you'd think people would be in their houses more in the fall and winter. Do you have any thoughts about that?

DR. AALEKHYA REDDAM: I wonder if it's related to the inversion -- the atmospheric inversion and, you know, the higher concentration of these pollutants and they just probably stayed there lower, if that's -- yeah.

ACTING CHAIR LUDERER: Any other comments,

discussion questions?

It looks we have one in -- or several I think in the chat I noticed.

STEPHANIE JARMUL: I guess I can paraphrase, unless Asa or someone wanted to get online. But so the design of EBDEP was to focus on parent-child pairs and it sounds like with -- they had a choice and it was usually the mom participated. Although, they did encourage the dads to participate, if possible, but this was the end result of the recruitment. And then the kids, of course, were evenly balanced by gender.

ACTING CHAIR LUDERER: Were there some other comments or questions in the chat? I thought I saw several. No?

STEPHANIE JARMUL: We have Ken now. I will -- an attendee. I'll allow you to talk. Please unmute yourself.

KEN SZUTU: Thank you very much. One thing which we notice in Vallejo, the difference between winter months is because the wind direction change. Normally, we have south -- wind coming from southwest. And during the winter, we have the wind direction change to from east to west and that is because we are in a refinery community and it depends on which direction the wind blow and also the location of the refinery. So that's one thing we

notice the difference.

Thank you.

DR. AALEKHYA REDDAM: Thank you, Ken.

ACTING CHAIR LUDERER: Okay. Were there any other comments online or from -- questions from the public? I don't see it right now, but.

No.

PANEL MEMBER FIEHN: Maybe one follow-up.

ACTING CHAIR LUDERER: Okay. Oliver.

PANEL MEMBER FIEHN: Yeah. Thank you for the responses to my question on sex and -- or gender inequalities. I do not find the responses acceptable. And I -- you know, there was a comment online here by a chat saying, well, that is usually the case in family studies. Well, families, you know, may consist also of male participants. And if I was to send a grant application to federal agencies, and I would say, well, I'll only take certain sexes into consideration, it would not be fundable.

So I'm just saying that if that is the case, that family, you know, studies are often imbalanced, I want to say per -- as a statistician or as a genomics expert, that is not acceptable and then it has to change. Just to stay to encourage is not an acceptable answer over -- for the future. And we did talk about what to do in the future.

You know, when we think about we want to look at all Californians, that does include men too.

ACTING CHAIR LUDERER: Thank you. We still have time for open discussion. Any -- whether any of the Panel members have additional discussion about this presentation.

Jenny.

PANEL MEMBER QUINTANA: I just wanted to comment to Oliver, I think it's a function of the study design of having a parent-child pair. And I've done various studies in low-income populations and it's very common to have a family headed by a woman and a child, and that's who participates often. I don't know the case in this particular thing. So I think it's more -- I think your comment should go to how do we design studies that will capture both genders in the future. If you're doing a parent-child pair in a low-income population and you ask people to come forward, you will probably have a biased population, unless you have made a decision ahead of time to exclude a bunch of people that want to participate and trying to oversample for other genders. But I think it's more a situation for future study designs.

ACTING CHAIR LUDERER: Thank you.

Nerissa.

DR. NERISSA WU: I just wanted to make a comment

that -- I mean, this was -- this was obviously not the design of the study to exclude men or if you are studying one particular sex, like we know we are doing in some studies, it just -- it just determines how you can generalize that data. And we are very aware of there are sex disparities in how people metabolize or are exposed to things. And so, we take that into account in our analyses. But, you know, it's never the intent going into our studies to overly generalize out of a disparity -- out of a disparate population. It is in some way the realities of doing community work. And, you know, like we talked about with surveillance, you just have to be really careful with your protocols to make them accessible to people who are going to be able to get to your study site to participate in this. They need child care. They need transportation. So it is one of the challenges of all biomonitoring studies.

ACTING CHAIR LUDERER: Thank you. Actually, related to that, did you see any indication of sex differences in the children?

DR. AALEKHYA REDDAM: No, we didn't -- we didn't look at it.

ACTING CHAIR LUDERER: Thank you.

All right. I'm looking to see if there's any additional comments or questions remotely or on the Panel

members.

I'm not seeing any. We -- let's see we have a little bit -- I do see something in the chat.

And it just disappeared.

STEPHANIE JARMUL: Did you want to unmute?

DUYEN KAUFFMAN: Yes. This is Duyen Kauffman from Biomonitoring California. And about the question of how much time was spent and possible differences in the seasons of time indoors versus the outdoors, we do have hourly time activity diaries that people kept for themselves and the kids separately over the four day period of their participation each season -- or each, you know, time -- but for both sampling periods. So we could look at how much time -- you know, the difference in time spent indoors and outdoors during the different seasons.

ACTING CHAIR LUDERER: Great. Thank you. Yeah, I think that would be interesting to look at.

Other comments, questions?

All right. Thank you very much for the great presentation. And I guess we can move on to our next presentation.

All right. So our next speaker is Paramjit Behniwal. And she is a Research Scientist in the Environmental Health Laboratory and she will give us an update today on the Program's development of the

laboratory methods for the analysis of VOC metabolites in urine.

(Thereupon a slide presentation).

ACTING CHAIR LUDERER: Paramjit

DR. PARAMJIT BEHNIWAL: Good morning. I'm -- my name is Paramjit Behniwal and I work with the Biomonitoring Program. So we developed the method for the VOC Metabolites in urine using the LC-MS/MS. And Jonathan Gallardo was our fellow so he worked mostly on it. He just finished his fellowship with us and joined the PhD program in UC Berkeley.

[SLIDE CHANGE]

DR. PARAMJIT BEHNIWAL: So what are the VOCs?

They are the compounds which can vaporize at the room temperature. And they are mostly present inside and outside air and they are very common pollutants. And they are naturally found in the environment. They can also be released by the man-made sources like paints, cleaners, cigarette smoke, wood burning, et cetera, and also from the industrial processes. And they affect our health in different ways -- in many ways, like breathing VOCs can irritate the eyes, nose, throat, and can cause difficulty in breathing, and can also damage our nervous system and other organs. And some of the VOCs if they -- we are exposed to them for a long time, they can even turn into

cancer.

[SLIDE CHANGE]

DR. PARAMJIT BEHNIWAL: So what happens once -- when we breathe VOCs or they get into our system? So once they get into our system they get -- they also do the action they -- damaging action, but also they get metabolized and get removed from the body through the urine, blood, and also breast milk and our blood. And mostly the metabolites are used as a biomarker for the VOC exposure, because they are more stable and they reflect the recent exposure to the VOCs. And mostly they are excreted as mercapturic acid in the urine. And they have longer half-life than the VOC biomarkers in the blood. And Biomonitoring California, they do the biomonitoring -- assess exposure to VOC to check the disproportionality affected to communities.

And there are some studies that have been done already and it's like East Bay Diesel Exposure Project, Stockton Air Pollution Exposure Project, FRESSCA, BiomSPHERE. And there's an Intraprogram Pilot Project, IPP7, and the CFF, California Fire Fighters Study.

[SLIDE CHANGE]

DR. PARAMJIT BEHNIWAL: So here I'm just going to talk about a little bit to the mechanism in the body, what happens once we absorb it. So it can be -- there could be

two things that go on at the same time. It can get metabolized to be more active compound, more toxic compound, and it can be directly metabolized to be excreted. So in the first one like Metabolism I, the cytochrome 450 chromosomes -- enzymes, they can oxygenate these -- especially I'm showing the example for the acrylamide. So it can do that oxidation of the acrylamide and make into glycidamide, which is more toxic to the body than the acrylamide itself.

So once it gets more active, it can react with the DNA molecules and make DNA adducts. And those DNA adducts then can damage our nerve endings and the nerves, so that's how they act as neurotoxins. And they can also make adducts with the hemoglobins and other -- and also through this process, they can also get detoxified with the glutathione conjugation and get removed as mercapturic acid.

In the second, Metabolism II, instead of getting activated to the glycidamide, it can also get just -- make conjunction with the glutathione and can get removed from the body into a different product. Like here it's AAMA.

[SLIDE CHANGE]

DR. PARAMJIT BEHNIWAL: So this is a little more chemistry of -- about chemistry in the body. So what happens, R-X is like a VOC compound. It gets attached to

the cysteine of the glutathione. Glutathione has three amino acids: glutamic acid, cysteine, and glycine. So cysteine has sulfur group where it -- VOCs get attached and then it makes a conjugation. And then that second step with the hydroxylation, glutamic acid gets removed with the help of the glutathione transferase enzymes. And then the -- in the second step within that, glycine gets removed. So what is left is just the cysteine attached to the VOC compound. And then it can get acetylated to make the mercapturic acid.

[SLIDE CHANGE]

DR. PARAMJIT BEHNIWAL: Oh. Well -- can you fix it? Okay. Maybe I can go back. Okay. Thank you.

So here, I am showing just -- all the parents compounds that we are monitoring and all their metabolites with their full names and their short names, so...

[SLIDE CHANGE]

DR. PARAMJIT BEHNIWAL: So how did we develop the method in the lab? So first we got the standards, each and every standard. And we made our solutions and then we optimized our mass spec. So for the mass spec, we get all the molecular ions and optimized all the (inaudible) that -- and choose the daughter ions.

And then once we have the mass spec method, then we make standard -- combined standard and then we

optimized our LC conditions. So we throw -- and then -- so that we get a good separation, the best possible separation that we can get, so we optimized all the solvents system and develop a gradient. So once we have that LC method optimized and the mass spec method optimized, then we go ahead and make our quality assurance/quality control samples that we do by getting -- in this case, we did synthetic urine.

So we combined -- made two pools of VOCs, one with a low level and one with a high level. And then we analyzed those QC samples over a period of some time. So we get total 20 runs. So once we get that 20 runs, then we do the statistical evaluation, we get the coefficient of variation and they we get the precision and accuracy.

And after -- also, we did -- we did the validation by getting some samples from the CDC. We get their PT samples. And we got four samples from them and we analyzed -- those four samples are were -- are for different concentrations, so we analyzed them and compared that as also -- how many we passed and what we didn't pass.

[SLIDE CHANGE]

DR. PARAMJIT BEHNIWAL: So here is our workflow -- simple workflow. In the step one, we just dilute the sample. It's a dilute and shoots method. So

we did dilute our samples and standards to 1 to 10 in our mobile phase. And then we just -- the second picture just shows how we arrange them in the tray and then put it into our instrument. And the third step is that -- shows our LC system. And the other one is the mass spec.

So what's -- we have like SCIEX mass spec. It's a triple quad and we are using an ESI as an ionization for this one in the negative mode.

[SLIDE CHANGE]

DR. PARAMJIT BEHNIWAL: Keep pushing -- sorry. I keep pushing the wrong button.

Okay. Thank you.

Okay. So here are the -- our conditions for the LC. So we have a Shimadzu Nexera LC and using the Acquity UPLC HSS T3 column, which is about 150 millimeter. And it's a 2.1 millimeter diameter. And the other mobile phase is 15 millimolar ammonium acetate as Solvent A and acetonitrile is our Solvent B. And then we have washes -- strong wash and weaker wash to wash our needle in between the injections. And then we have the gradient for our method. We start gradient from just three percent of the Solvent B. Like, it start with very aqueous conditions and then we go over the -- organic solvent goes only up to 40 percent and then it comes down. So once it gets to 40 percent, we get almost all the compounds out.

[SLIDE CHANGE]

DR. PARAMJIT BEHNIWAL: So here, I'm listing over MRM transitions for all the compounds. And there's a -- for most of the compounds, we have the different molecular ion and their daughter ions. But there are some compounds that we have -- that share the same molecular ion and/or daughter ions. So that I have highlighted here like with 2MHA and the metabolites of xylene. All three metabolites did share the same parent compounds and the daughter ion. But we are able to separate them by chromatography. So it's easy to quantitate them, even though they share the same compound.

So another example is HPM2 and CYMA. So they also share the molecular ion. So chromatography, if we can separate -- but if they're eluting very close to each other, then we can -- then they have different molecular ions. So we have like two, three different parameters to consider to separate and quantitate these ions, one is a retention time and the molecular ion of the compound and those are the two very important ones.

[SLIDE CHANGE]

DR. PARAMJIT BEHNIWAL: So here I'm showing the chromatogram for all those compounds. So some of you see them, they are very close to each other. But since we different molecular ions and daughter ions, so we can

still get a good quantitation.

[SLIDE CHANGE]

DR. PARAMJIT BEHNIWAL: So here is our quality control from our low QC and high QC. It is data from the 20 runs. Most of the compounds -- all of the compounds we get like coefficient of variation is below 20 percent. That's what we are saying. But majority of them are like below 15 percent, only few are like about 17 percent, which is on the high end. We are still trying to work out to bring it down. So see, we're -- how we can improve the method.

[SLIDE CHANGE]

DR. PARAMJIT BEHNIWAL: So like I already said that we got some reference samples from the CDC to validate our method. So there was four QC/QA samples. And 22 analytes out of the 24, they met acceptance criteria. So there was AMCA and DHBM, which didn't meet the acceptance criteria. So AMCA is a metabolite of n,n-dimethylformamide. And this didn't meet the acceptance gradient also. So this one we failed. And it's usually on the higher side for all of four samples. The results is on the higher side, so we noted something is not right with our standard. So we are trying to work out that. And DHBM failed in three samples. So we will look at -- into that also. But there are some compounds

that failed in only one sample, so that's like just a random error. So in conclusion, we kind of passed our validation from -- with the CDC sample.

[SLIDE CHANGE]

DR. PARAMJIT BEHNIWAL: So in conclusion, so we have a method for the measurement of VOC metabolites in urine. We can measure 25 samples -- compounds together, but 3MHA and 4MHA they come together. So we can say we have a method for 24 analytes. And we get good accuracy and precision, which we demonstrated through the QC pools and the CDC quality assessment samples. And we have analyzed the Intraprogram Pilot project samples, which are 39 in number. And Camp Fire Firefighter study samples, which is 66 in number. And we are reviewing the results. IPP study samples are with the QA at this time and CFF study samples are -- I'm still reviewing them.

[SLIDE CHANGE]

DR. PARAMJIT BEHNIWAL: So thank you.

ACTING CHAIR LUDERER: Thank you very much for the presentation. We have time to start with clarifying questions from the Panel members to begin with.

Carl.

PANEL MEMBER CRANOR: You -- at the beginning - it was in your second slide - indicate that there are lots of sources of VOCs. They're everywhere.

DR. PARAMJIT BEHNIWAL: Yeah, they are everywhere.

PANEL MEMBER CRANOR: I'm wondering does your data give you any indication about what major sources we ought to be paying attention to reduce them. Does that -- does this help at all in terms of the bigger public health issue?

DR. PARAMJIT BEHNIWAL: I think it should, but that's not my decision to make. I -- we just give the results to our customers.

PANEL MEMBER CRANOR: I guess I was also thinking about the slide on the parent compounds. Are there -- you know, what are the sources and which are the parent compounds that contribute to them?

DR. PARAMJIT BEHNIWAL: From the preliminary results from the IPP, I -- we found that there's like -- we are seeing the HPMA and CEMA very high. So that is the products of from the acrolein. So then we also have some high levels for the xylene. So there are some compounds which are a little -- we see in almost all the samples. And some are very low, which is like -- like only like few ppb, 3 ppb, 4 ppb. And also -- so I don't know, because I'm not in part of the planning the studies, so maybe --

DR. NERISSA WU: Yeah, I can answer that.

So it's a good question. You know, why -- you

know, what do we do with all these lab results? And I think as a Aalekhya has presented, there's a lot of profile analysis we can do to look at what the source might be, but also in conjunction with the questionnaires and the geographic analyses to try to pinpoint what are the really important sources that we can then target for reduction. But we can't do that work unless we have an available lab method. So we're really looking forward to using this analyses further.

PANEL MEMBER CRANOR: All right. That seemed to be a good deal of pride in the procedures that have been developed for analyzing them. And I was trying to take it a step further and what will this tell you about the bigger questions too that we have to worry about?

DR. NERISSA WU: Well, I think we look forward to BiomSPHERE and FRESSCA and some of these other studies where we do have questions that are really specifically looking at filtration of air, for example, and being able to come up with recommendations for what that means in terms of reducing exposures, again both on an individual level, what can you do in your own home as well as on a policy basis.

PANEL MEMBER CRANOR: Thank you.

STEPHANIE JARMUL: And I don't have my hand up, but I'll call on myself. This is Stephanie. I just

wanted to add that as Nerissa mentioned, yeah, we are excited to use these in our future studies, especially because actually EHL's panel is larger than the panel that we've had for at least SAPEP, BiomSPHERE, and FRESSCA. I think it more closely matches CDC's panel. And so we will have a few more chemicals included in our study, which will hopefully shed a bit more light on potential, you know, different exposure sources. And again to add on to what Nerissa is saying, we are trying to collect more environmental data to complement our biomonitoring data, which also might help elucidate some of those sources.

ACTING CHAIR LUDERER: Thank you.

Oliver, you had a question or comment.

PANEL MEMBER FIEHN: Yeah. Thank you. I am delighted to see analytical methods details. For those details, I saw that you dilute and shoot 10-fold dilution. But usually when people look at urine, they do some normalization on the volume either by it's based on specific gravity or creatinine. Why don't you do that?

DR. PARAMJIT BEHNIWAL: I think we will do the creatinine normalization, so --

PANEL MEMBER FIEHN: But later, like afterwards, not before.

DR. PARAMJIT BEHNIWAL: Afterwards.

PANEL MEMBER FIEHN: So often people do it before

hand to be sure that you're in the dynamic range. And I didn't see dynamic ranges here. I didn't see neat concentrations, so like without spiking. And I didn't see, you know, some other measure of two positives in a way. And that comes to my second question. So these, you know, metabolites of the volatile organic carbons of course undergo multiple steps of enzyme control. And these multiple steps of enzyme controls can be different in different people. So, you know, in one person with that exposure, you know, might go towards that route. And another person with the same exposure might go more to the other route.

So that goes to the -- you know, is it -- are those really the right compounds or, you know, you show different pathways that can be used? And so if we just look at one specific pathway, you know, then we might miss the, you know, differences. And the value we measure might not be a direct measure or, in this case, indirect measure of the true exposure. So have you thought about that?

DR. PARAMJIT BEHNIWAL: That's true, but we usually mostly follow the CDC method. And they have done a very thorough thought on it that picked out the most common -- or the most unique metabolites for that analyte -- for that VOC parent. So like benzene has

multiple metabolites. But this one, the one we are doing, is like very specific. It doesn't come from other sources. But like other metabolites they are metabolites that share many more parents. So it could -- then you cannot say that it's specific to the benzene. So these are very specific to these parents' compounds. So if you measure that, then you can -- with some certainty, you can say that, okay, this person is exposed to this particular compound.

DR. SHE: Thank you. Jianwen She. I work with Dr. Paramjit Behniwal. So I like your first question regarding to use some creatinine specificity measurement to guide in the next step to decide how much dilution we need to the protein precipitation. That's great idea. We didn't look at that, but we should look. And then also my comment on Carl's questions about the -- so how we can use this data to find out where the source come from. Basically, for example, we also -- we did some study before. Before this method, we look at BTEX exposure. I use the BTEX exposure as examples, so we can link our levels with specific process. From that process, we measure like let's say BTEX is high. We know that most of the chemical come from BTEX or the acrolein that was higher, so we can try to attribute the source and the -- where the VOC come from, we can develop an intervention or

the prevention procedures. And that's done and for what Pami said.

DR. PARAMJIT BEHNIWAL: And I also want to add that like you said about the dilution factor. It's -- if we -- our calibration range is very high for most of the compounds, the one that is more commonly found in our -- in the urine. But if it comes -- it gets out of our calibration, we are going to dilute the sample again and run it again.

ACTING CHAIR LUDERER: Question or comment.

Martha.

DR. MARTHA SANDY: Thank you. Martha Sandy with OEHHA. To speak to Oliver's point that perhaps we have to look at the metabolites that are measured of a certain VOC and see if they're truly indicative of exposure to the parent compound. That is certainly a valid concern that we would look at that more closely because of genetic polymorphisms and other exposures that people may have that may alter their metabolic pathways for a certain parent compound. So that is something we are looking at.

I believe the methods that are -- were developed here are based on the mercapturic acid metabolites.

ACTING CHAIR LUDERER: Do we have any other questions from Panel members or comments?

Oliver.

PANEL MEMBER FIEHN: So thank you again. So when you looked at the spiked examples, you used, I think, 80 ppb, if I remember correctly. You know, was this value based on literature values, like did you expect this amount as a lower measure of quantification for typical urines or where does it come from the lowest amount of spiked values?

DR. PARAMJIT BEHNIWAL: That's depends on our instrument's capability, how low it can go.

PANEL MEMBER FIEHN: So is it like --

DR. PARAMJIT BEHNIWAL: Do you mean like --

PANEL MEMBER FIEHN: What do we expect? Is the dynamic range covering what we usually expect in people?

DR. PARAMJIT BEHNIWAL: Okay. So the low QC levels, yes. So we -- based on that initial concentrations and also from the CDC, yes, from their NHANES studies, and then their publications, and from other literature. And we also usually try to keep low QC levels to the lower end, so that we can catch the lower levels and the high QC levels are closer to -- between the middle and the high end of our calibration.

ACTING CHAIR LUDERER: I just have a quick technical question. Is -- are there any specific concerns for how the samples are stored for measuring these metabolites or --

DR. PARAMJIT BEHNIWAL: Yes. They have to be kept in the cooler. If you take -- at low levels -- we store them at minus 80. So if you keep them in the refrigerator for long, then we lose some of the compounds, because they are very unstable compounds.

ACTING CHAIR LUDERER: That's a concern for collection of the samples in the field too obviously, I assume.

DR. SHE: A few years ago, we work with a collaborator, visiting scholar, and then we did a stability test. We did a long-term stability test, short-term stability test, stability post-preparation test, and also freeze and thaw sound test, but it's only with the metabolite from the BTF metabolite, and -- which is benzene, toluene, xylene and acid benzene. So we plan to do more stability test. And through that test, we identified the problems and -- when the samples store at minus 80 and for long-term test. And we didn't find freeze and thaw will affect the analytical result. That seemed to be stable. But with 22 or 25 new analytes, we plan to do more test, yeah.

DR. PARAMJIT BEHNIWAL: And to add on to that, yes, I did run into problem in the beginning with my standards, because we were keeping them in the like minus 20 and taking them out every day and put them back into

minus 20, take them -- so after 30 days, I start losing the peaks. So standards did get affected with the multiple time keeping -- taking them out and putting them back.

ACTING CHAIR LUDERER: Thank you. That's very important information.

Any other questions or comments from Panel members or from anyone online? Stephanie or Rebecca

No.

STEPHANIE JARMUL: Nothing online.

ACTING CHAIR LUDERER: Okay. Great. Thank you so much your presentation.

Let's see, I think we're running a little early. If we have no additional discussion points now, do we want to take the break now or move on to --

STEPHANIE JARMUL: Yeah, let's take the break now.

ACTING CHAIR LUDERER: -- a later presentation?

Okay. And we'll keep it at one hour as planned?

STEPHANIE JARMUL: Let's just return at the usual, so we can start back on time. So I think that's 1:15 we'll --

ACTING CHAIR LUDERER: Okay.

STEPHANIE JARMUL: -- ask everyone to return from lunch.

ACTING CHAIR LUDERER: Okay. All right. Great. So everyone has a little bit longer lunch than planned.

All right. Great. Thanks very much.

(Off record: 11:48 a.m.)

(Thereupon a lunch break was taken.)

(On record: 1:19 p.m.)

ACTING CHAIR LUDERER: All right. Welcome back everyone. We're going to start now with our afternoon session. So in the next agenda item, we're going to be hearing from two speakers. The first presenter will be Kelly Chen. She's a Research Scientist at CDPH. Today, she will be presenting on associations between serum PFAS concentrations and seafood consumption among Asian/Pacific Islanders in the San Francisco Bay Area. And the title of her talk is going to be, "Perfluoroalkyl and Polyfluoroalkyl Substances(PFASs) and Seafood in California: Monitoring of Human Populations and Fish Species."

Kelly.

(Thereupon a slide presentation).

KELLY CHEN: Hello, everyone. Thanks so much for having me. I am a Research Scientist at the Biomonitoring California Program, and, as has been mentioned, I'll be presenting on our study focusing on Asian/Pacific Islanders in the San Francisco Bay Area and how we're

using the data to examine how fish and shell fish consumption contributes to PFAS exposures and then Wes Smith over at OEHHA will be following my presentation.

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KELLY CHEN: So first, I know this Panel knows a lot of about PFASs, but I'll still give a quick overview on PFASs or per and polyfluoroalkyl substances. These are a class of chemicals widely used to make various products resistant to oil, stains, grease, and water, and commonly known as "forever chemicals", as they are very long lasting. Fortunately, they have spread extensively through the environment and have been linked to a variety of adverse health effects.

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KELLY CHEN: At a previous SGP, my colleague Toki Fillman focused on drinking water as an exposure pathway to PFASs. Today, I'll focus on fish and shell fish as other dietary sources of exposure.

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KELLY CHEN: For some context, there's been growing literature around PFAS exposures from seafood. And I'll highlight two relevant national studies. The first FDA studies that have detected PFAS in store-bought fish and shellfish, as well as a recent analysis of EPA freshwater fish samples across the U.S., which found

extensive PFAS contamination, particularly of PFOS. Other studies linking biomonitoring data in various regions of the U.S. and other parts of the world have linked seafood consumption, especially fish, as a major pathway of exposure to PFASs. And this is reflected in current fish consumption advisories and their modeling assumptions, which have applied contributions ranging from 50 percent from Washington State, 70 to 80 percent from Maine and New Hampshire, and up to 86 percent from the European Food Safety Authority.

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KELLY CHEN: And here is a closer look at the PFAS profiles in freshwater fish samples from 2013 to 2015 EPA data using targeted analyses. In the yellow, PFOS dominates as a contributor to total PFAS levels at nearly 75 percent. Several other PFASs make up the remaining quarter, noticeably, longer chain carboxylic acid PFASs. These are PFUnDA, PFDA, PFDoA, PFNA, and PFOSA. Notably, PFOA and PFHxS made very small contributions in these freshwater fish Samples, less than one percent, though they have still been detected in fish and shellfish in some other studies. And I'll also note that compared to the PFAS profiles shown here in U.S. freshwater fish, those in marine fish samples have generally been lower.

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KELLY CHEN: In the U.S., we also see that there is higher seafood consumption among Asians. According to national data from 2013 to 2016, 20 percent of all adults eat seafood at least two times per week, while nearly double, or 41 percent, of Asian adults fall into this category.

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KELLY CHEN: We're also seeing the API populations have higher PFAS levels compared to other ethnic racial groups. And in our California Regional Exposure Study in the LA region, we see that PFOS was 68 percent higher among Asians, compared to the White category.

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KELLY CHEN: And these findings were reflected in the second region as well, CARE-2, where we saw that PFOS was 147 percent higher among Asian compared to White.

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KELLY CHEN: So this context brings us to today's analysis, which had several overarching objectives. The first to characterize fish and shellfish consumption in a highly exposed population, which only a few studies have done so in the U.S. and even fewer examining the consumption of various fish parts; second, to evaluate associations between seafood consumption and serum PFAS

levels; and lastly, to share results with partners at the local, state, and federal level to aid in efforts to reduce exposures from seafood consumption.

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KELLY CHEN: So while many Panel members heard about the ACE project, as it was presented on in previous years, but I'll give some overview. This was the Asian/Pacific Islander Community Exposures Project, or ACE Project, which was a community-based study conducted in 2016 to 2017 to biomonitor Asian populations in the San Francisco Bay Area, as previous studies had found higher levels of metals and PFASs in Asian Americans. Our branch has had a long history of working on safer fish consumption messaging with local API advocacy groups. And one group, APA Family Services, had expressed interest in biomonitoring as a way demonstrate the potential impacts of dietary choices on chemical exposures. And to a specific question earlier from Jenny, we were also interested in discerning differences in specific Asian populations.

There were two phases. The first focused on Chinese American participants in the San Francisco area. And the second phase included Vietnamese Americans in the San Jose area, which is about one hour south of San Francisco.

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KELLY CHEN: In both phases of ACE, participants were biomonitored for metals, which we presented on previously and a panel of 32 PFASs. We also collected information using an exposure questionnaire, which included questions about demographics, occupation, immigration history, personal care product use, and very detailed questions about diet, particularly rice and seafood. We did not, however, collect information on drinking water, consumption or sources, parity, or breast feeding history.

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KELLY CHEN: Analyses used log transformed serum PFAS concentrations due to a skew observed in the PFAS data and evaluated several measures of seafood consumption that I will be showing in later slides. Each model was adjusted for age, sex, education, income, birth country, and portion of life in the U.S., and also adjusted for household clusters. And analyses were run combining ACE 1, and ACE 2 data, given the small sample sizes, and then if there were sufficient numbers for the exposures or covariates for each ACE group individually. And for this presentation, I'll talk through the results that were significant at a P value of less than 0.05.

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KELLY CHEN: To give you a sense of our study population, ACE 1 and ACE 2 both recruited a hundred participants, but a few were unable to provide blood samples, bringing the total for the analysis to 195. The mean age of both groups were similarly in the mid-forties and just under 50 percent male. Household income was higher among our ACE 1 population, but the median still fell well below the region's median income in both cases. Most of our participants were born outside of the U.S. and ACE 2 skewed more heavily towards recent immigrants and a non-English speaking population.

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KELLY CHEN: Now moving on to the ACE PFAS profile. Among the 32 PFASs measured in the study, we focused on six PFASs for these analyses, which were those with the highest detection frequencies and linked to seafood consumption and other studies. Here, I'm sharing geometric means broken out by each ACE group with ACE 1 in the yellow and ACE 2 in the dashed yellow. For both groups, the general trend was pretty similar, though PFHxS and PFOA were slightly higher among the Vietnamese group, or ACE 2.

I've also added in national data in blue to compare to NHANES, all adults in NHANES Asian specific PFAS data from comparable years. You can see that the

yellow bars from the ACEs tend to run a bit higher than the blue bars, the national data. Specifically, four were statistically higher in ACE than the national data marked by red asterisks, PFOS, PFUnDA, PFNA, and PFDA.

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KELLY CHEN: Looking at the PFAS data another way, you can compare the results from our ACE group to the clinical guidance issued by the National Academies, you can see how the different populations compare. As shown in the lowest row, the ACE participants disproportionately fall into the highest risk category, where the sum of seven PFASs is greater than or equal to 20 micrograms per liter, at 20 percent across both ACEs, more than double the national level at nine percent.

This further underscores why it was a priority for our program to identify exposure routes that may be contributing to these high levels of PFASs.

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KELLY CHEN: And we also saw higher seafood consumption in ACE. Adding to the graph I showed earlier on national trends in seafood consumption, ACE participants in yellow are more frequent consumers of seafood compared to NHANES Asian adults at 59 percent compared to 41 percent.

In ACE, we also asked about purchased versus

caught fish. Almost all of our participants reported eating at least some fish from stores or markets. And almost one-third reported eating fish caught by themselves, friends, or family in the past year and quite frequently with, almost a third of participants eating caught fish at least one to three times per month or more.

In the questionnaires, participants also reported a variety of speedy -- species consumed that we've shared with State and local stakeholders, but I'm not showing that on the screen just for the sake of time.

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KELLY CHEN: In ACE 2, we also introduced a question about the consumption of fish parts. And as shown, most participants reported eating at least some of these non-filet fish parts, eyes, head, organs or skin at least some of the time. This is of concern because fish advisories are often based on levels of pollutants measured in the filet and limits are often communicated in terms of servings of filet. However a number of studies have shown that there may be much higher levels of PFASs as well as other contaminants on the range of 1.5 to 5 times higher in non-fileted fish parts compared with the filet.

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KELLY CHEN: Moving on to the analyses. We next

looked at associations between serum PFAS levels and these three measures of reported seafood consumption: bought fish, caught fish, and bought shellfish. Unfortunately, we didn't have large enough numbers in the caught shellfish category to assess those associations.

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KELLY CHEN: In looking at the estimated serum increase associated with the median number of bought fish meals consumed in the past month, or five additional bought fish meals, our model showed a nine percent increase in serum PFOS levels and a 14 percent increase in PFUnDA levels among all of the ACE participants. PFDA and PFNA levels were associated with bought fish consumption in ACE 2 only. And in those models, we saw increases of 17 and 20 percent compared to participants who did not consume bought fish.

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KELLY CHEN: The increase was more pronounced for caught fish with significantly increased levels of five PFASs seen with the median consumption of caught fish.

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KELLY CHEN: The consumption of bought shellfish was also associated with PFOS, PFUnDA, and PFDA serum levels in the ACE 2 group, notably we saw an association with PFOA that I also wanted to mention of five percent

that was just approaching significance. And PFOA has been detected in clam samples and other shellfish.

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KELLY CHEN: In another analysis, we looked at the same data, but combined bought and caught fish to examine how serum PFAS levels differed by total monthly fish consumption frequency in the past month. There were four frequency levels: zero to three meals per month as the lowest category used as a comparison against one to two meals per week, two to three meals per week, which is the recommended fish consumption guidance by the EPA and FDA and adopted and put forth by the USDA dietary guidelines, and then greater than three meals per week as the highest frequency category. We examined associations with each of the six PFASs in separate models.

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KELLY CHEN: Here, we observed higher PFOS and PFUnDA levels associated with consumption above three servings per week, which can be seen in the darkest bars. Our model showed a 50 percent increase in PFOS and a 58 percent increase in PFUnDA among those who consumed greater than three meals per week compared to those who consumed only zero to three meals of fish per month.

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KELLY CHEN: We also noted several other PFASs

approaching statistical significance, PFNA, PFDA at greater than three meals per week, and for PFUnDA in all categories above zero to three meals per month.

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KELLY CHEN: We then looked at consumption of caught fish only categorized by increasing frequency of consumption within the past month.

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KELLY CHEN: And here, caught fish consumed frequently at more than three meals per week was associated with increased levels of all PFASs at even more pronounced levels ranging from 33 to 148 percent increases in serum PFAS levels.

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KELLY CHEN: Consumption of fish parts was another important factor. We compared participants who reported ever eating fish organs, heads, skin, or eyes with participants who reported not eating any of these fish parts. And we also created a variable which included consumption of any of these fish parts in the second column. Here, we found that substantially increased PFAS levels were associated with consumption of individual fish parts, fish organs, head, skin, and eyes. And increased PFAS levels were also associated with the consumption of any of these fish parts.

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KELLY CHEN: The increase was less noticeable for fish paste, cakes, or balls and shrimp sauce, all of which likely contain fish and shellfish parts, perhaps because these product are used in small quantities, or because of the types of seafood that are used for these products, or because there are many other fillers in these products.

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KELLY CHEN: As a summary across these analyses, we observed consistent associations with fish and shellfish both purchased and caught fish, purchased shellfish, and seafood products, particularly with caught fish consumed at high frequencies and non-filet fish parts. These fit -- these four PFASs in red were most commonly associated ranging from C8 to C11, which parallels other studies on PFASs associations with seafood both from biomonitoring studies and from direct sampling of seafood samples. PFOS and long chain carboxylic acids tend to accumulate in fish, while shellfish seems to exhibit slightly different patterns in the literature.

Again, we didn't have large enough numbers in the caught shellfish category to draw conclusions, but in the literature, other studies have reported shrimp, lobster, and clams associated with higher PFOA, PFDoA, PFUnDA, PFHxS levels.

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KELLY CHEN: We can see that overlap also as a flashback to the PFAS profiles in U.S. freshwater fish samples.

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KELLY CHEN: Notably, last week, the EPA released an update to their list of contaminants to monitor for fish and shellfish advisories, last updated in 2000. In this updated list, five out of the six PFASs to monitor for overlapped with PFASs associated with higher seafood consumption in the ACE study. PFUnDA, however, is included, but only as a contaminant to watch for, as there's not yet a federal measure of oral toxicity for PFUnDA. But I did want to note that we did see a strong pattern with PFUnDA in these analyses.

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KELLY CHEN: The association between frequent seafood consumption and PFAS levels and the concern that eating different parts of fish may be resulting in elevated PFAS levels remain an important topic not only for California to address but also many other states across the U.S. This was highlighted in an article by Kaiser Health News that covered the ACE Project and other State efforts around PFAS in seafood last December.

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KELLY CHEN: Over the course of working on ACE and learning more about PFASs in seafood, our program has had several helpful conversations with OEHHA's fish advisory program centered around the ACE results and how they may be helpful. And we wanted to acknowledge, especially with Wes Smith in the room, that they are the effort -- the experts in what is a complicated effort to craft fish consumption advisories across the state. And we are very glad to have Wes join today.

So I won't go too deep into this slide of where we are in California, only to provide an overview that California fish advisories are currently based on many chemicals, including mercury, PCBs, selenium, and other chemicals, but does not yet have a PFAS-based advisory.

There are, however, efforts monitoring PFASs in fish that we have learned about. And data collected in the San Francisco Bay Area does suggest that PFAS levels in fish would trigger fish consumption advisories that some other states have. The ACE data underscores the importance of better understanding PFAS exposures in seafood that Californians may eat. So we are very excited to learn more about the work that is underway in considering next steps for PFAS fish advisories. So more to come with Wes's presentation.

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KELLY CHEN: Overall, there really is a need for more PFAS data on fish, and especially shellfish. And this has been underscored while working on the ACE Project. We are learning that PFASs can vary by species, by location, and over time. So time trends and location-specific data are needed to inform site-specific advisories. And more data on consumption rate, species, and non-filet fish parts that consumers are catching themselves and buying from stores would help inform testing and advisories.

Similar to the biomonitoring world, expanding the PFASs tested beyond legacy PFASs and targeted analyses could also provide a more comprehensive understanding of the exposures.

And lastly, this study was based on Chinese and Vietnamese Americans in California, but more information on other diverse communities are needed as many other communities eat fish and shellfish in California.

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KELLY CHEN: I wanted to highlight some of the ways in which the ACE Project has fed into ongoing efforts around fish and shellfish. On the regional level in California, we have shared our data with the San Francisco Estuary Institute, as they are working on characterizing fish consumption, especially in subsistence fishers, and

sampling fish in local waters. On the State level in California, we have worked with the California Safe to Eat Workgroup and their statewide realignment and long-term planning efforts around bioaccumulation monitoring in fish. And we have also shared this data with the EPA fish group and received requests from some other states to learn about ways the ACE data can be used.

Lastly, we are working on communicating these findings, including a manuscript and accompanying two-pager in community and conference settings, and are exploring other products.

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KELLY CHEN: To summarize, fish and shellfish can be contaminated exposing consumers to chemicals such as mercury, PCBs, and PFASs. Fish, shellfish, and seafood derived products are frequently consumed by API participants in San Francisco Bay Area. Associations between seafood consumption and PFAS serum levels were observed within this highly exposed population. And more data on PFAS in seafood is needed to better connect these findings to policy and outreach.

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KELLY CHEN: And I wanted to thank the participants in ACE 1 and ACE 2, and our community partners APA Family Support Services, and VIVO, who were

instrumental in getting this project off the ground and implemented. And also, thank you to all the Biomonitoring California staff, including labs who contributed, especially to Kathleen Attfield, Nerissa Wu, Emily Beglarian, Duyen Kauffman, and Kiera Melton, who have also been working on ACE, and to OEHHA's team, San Francisco Estuary Institute, Environmental Working Group, the New Jersey Department of Health, and the EPA fish group who have all informed and taught us a lot about fish and shellfish.

ACTING CHAIR LUDERER: Thank you very much, Kelly for that interesting presentation. We have time for some clarifying questions now. I can start with Panel members. I see Tom and Oliver. Tom, you want to start.

PANEL MEMBER McKONE: Thank you. A very interesting project. I guess a clarifying question is, so we have two populations, San Francisco and San Jose. Is there any effort or method to understand how the 100 Asian/Pacific Islanders from those two locations would be representative of Asian/Pacific Islander population throughout the state or different parts of the state?

KELLY CHEN: Thank you, Tom. That's a great question. We have thought about this question and we understand there are other Pacific Islanders especially that aren't represented who may consume different amounts

of fish or different parts of fish than represented in this study. I think what we're understanding is that really anyone who eats a lot of fish or shellfish that is contaminated would be affected. And we were interested, the EPA had shared some slides on their efforts to understand fish part consumption. And they sampled across many different groups beyond API populations. And there are French -- there are -- I'm blanking right now, but many other different ethnic groups. And almost all these groups were eating different parts of the fish, not just API participants.

ACTING CHAIR LUDERER: Thank you.

Oliver.

PANEL MEMBER FIEHN: Thank you. Really interesting when we think about sources of PFAS. So first of all, you know three-quarters -- just to recap, three-quarters of the total PFOA -- something would be PFAS. And it appears that when you eat four times or more often per week fish, you double your serum levels. If I take these numbers in, can I conclude that fish is really one of the major sources of PFAS in the body? Can we do calculations of, you know, what are other sources, because people are, you know, really worried about drinking water. They are worried about, well, it's everywhere, right?

But when we now see your study and it looks like

I doubled my intake if I eat almost daily fish, especially different fish parts, but it's really doubling. So can I conclude that this is a major route of exposure?

KELLY CHEN: I wouldn't say that directly from this study, because we weren't able to account for drinking water. If we had, we might have been able to make some calculations more specifically about the source contribution. I know many other groups are interested in this question and have tried to address it. For example, the NHANES, they asked questions about fish consumption and shellfish consumption. They saw even at low levels, PFAS levels had increased with some of the same PFASs we measured in the study. I'll also pass this off to Wes in case his group has thought about this.

DR. WES SMITH: Yeah, I think it's really hard to say without -- I mean, we focused specifically on fish consumption and we don't look at the broader exposure milieu, I guess. So I don't have any data to, you know, suggest either way.

KELLY CHEN: But I would just say other studies have suggested that contaminated seafood can be a major exposure pathway to PFASs, especially in areas without contaminated drinking water, and especially in high seafood consuming populations. The Faroe Islands are a great example of some of these studies where they really

are eating a lot of seafood compared to other types of meat or dairy. And they are finding high levels of PFAS due to that consumption.

STEPHANIE JARMUL: This is Stephanie. I see Kathleen has her hand raised. She might want to contribute.

DR. NERISSA WU: Sorry. I'll put my hand up for going after Kathleen.

DR. KATHLEEN ATTFIELD: I just wanted to point out that in the CARE studies, we have a graduate student who's been looking into both the contribution from different dietary sources and some information from drinking water data from the previous EPA's UCMR 3 data. And there, it -- sort of it depends on which of the PFAS. So the smaller carbon chains that Kelly was pointing out had less of a contribution show up a little bit more in dairy, eggs, and white rice and the higher carbon chain links, PFAS show up with a greater contribution for fish.

So it kind of depends on the PFAS as far as which have the greater contributions. But even in her study, the water contributions are actually much higher than for the dietary contributions. But there's a lot of studies that still need to happen to figure this out in U.S. and different parts of the country.

ACTING CHAIR LUDERER: Thank you very much.

I think Carl had a question too.

PANEL MEMBER CRANOR: I have a complicated question and you may not know enough to answer it yet, but the PFOS has been around much longer, I think, than some of these others. And are you seeing higher concentrations of that, because it's been around longer? Does it stay in the -- given the chemistry -- biochemistry, does it stay in the body longer than these others are showing up less well? Anybody looked at that -- at the biochemistry and the interaction between these substances. We know that when companies have trouble with one compound, they say, oh, we're going to make this other compound. It will be much better. Any evidence for that?

KELLY CHEN: That's because much to the biokinetics, I've done a little bit of reading about some of the different partitioning and different affinities, different PFASs have, like PFOS compared to PFOA. Both legacy compounds have different affinities for albumin, which is highly found in the liver and in different parts of other organisms. And in part, that explains the differences in PFOS versus PFOA accumulation. I guess to your point about the temporal phasing of different PFASs, I haven't seen as many studies on that. I know legacy PFASs being phased out and other PFASs coming in probably will have an impact on what sort of PFASs appear in

different media and how that then gets absorbed into fish and shellfish in humans.

But I'll pass that off, in case Wes has an answer.

DR. WES SMITH: Yeah, and I think there's two parts. There's the sort of persistence in the environment and also the persistence in the human body. Within the human body, it seems like PFOS is five to eight years. And possibly the longer chains, I'm not particularly sure, but would assume they would be longer versus -- some studies have shown in the environment that there will be water detections that are almost undetectable, but you'll see higher accumulation in the fish. So there's that bioaccumulation component that complicates matters.

PANEL MEMBER CRANOR: (Inaudible).

DR. WES SMITH: Right. Yeah, and it's -- again, all these compounds are very different and complicated, so -- and the mixtures are different and some of the bioaccumulation pathways we see can be rela -- radically different between something like a PFHxS verse one of the long chain PFDA type compounds or so.

PANEL MEMBER CRANOR: Thank you.

ACTING CHAIR LUDERER: All right. Martha.

DR. MARTHA SANDY: Martha Sandy from OEHHA. Just to add on to what's already been said. Yes, each of the

different PFASs has different -- different half-lives is not necessarily exactly matched with or correlated to length of the chain, but PFOS and PFOA have very long half-lives as Wes has said.

There are also legacy PFASs that have been phased out, but there are hundreds -- at least a hundred for each, the PFOA and PFOS, precursors that other PFASs that are used that break down to release PFOA or PFOS. So there's a continual introduction into the environment. So it's complicated.

PANEL MEMBER CRANOR: I knew it would be.

ACTING CHAIR LUDERER: I actually had a question whether you were able -- or whether you have information about differences between saltwater and freshwater species, whether there's differences in the PFASs -- the content of the PFASs and whether you were able to look at that in this population.

KELLY CHEN: We were so interested in that question. Based on the literature we've seen, freshwater fish have higher levels of chemicals at least compared to marine and marine fish that are not found near very contaminated sites. So we tried to look at the individual fish species reported and to separate them out by marine, freshwater, or migratory fish after speaking with Wes's group and other fish groups, but we weren't able to tease

apart that question just due to low numbers. But that was an interesting question. And we also thought about trophic levels, which again we were only able to look at the highest trophic level, but not some of the other lower trophic Levels. But we thought these questions might help advisories determine which fish to focus on or shellfish.

ACTING CHAIR LUDERER: Thank you. And I see that Amy has a question and her hand up.

PANEL MEMBER PADULA: Thanks. I was wondering -- I know you didn't show the metals in this presentation, but that they were measured. And I was wondering if any of the PFAS tracked with the metals along with the fish consumption? I guess I'm just curious, especially since the guidance seems based on the metals, at this point, whether that may be protecting people to a certain extent or whether we're -- if they're mismatched, then maybe not so.

KELLY CHEN: We were interested in mercury, because that tracks pretty well with seafood consumption. And here, we saw a strong correlation with some of the PFASs, especially PFUnDA. I know some other groups have also seen that correlation in other studies. So I hope that answers your question --

PANEL MEMBER PADULA: Yeah. Thank you. And also just want -- yeah. So I just wanted to mention how, yeah,

remarkable that these data are and how important they are. And yeah, I also share the wish, I guess, of everyone to sort out all these sources, but this is a great step. So thank you.

STEPHANIE JARMUL: And Ulrike, we do have a question from Ken. I will --

ACTING CHAIR LUDERER: Great. Thank you.

STEPHANIE JARMUL: Yep. Go ahead.

KEN SZUTU: Thank you. This is just a follow-up on the mercury, because from a consumer's point of view, for example, usually we were recommended to eat smaller fish, because the accumulation. So I was wondering if you have -- you have any -- you have looked into that on the size of the fish instead of other considerations. Thank you.

KELLY CHEN: We did not in our ACE data just because we didn't have the exact fish consumed necessarily for these questions. But I think in the literature, they haven't seen a strong correlation between PFAS levels and the length of fish. That's correct, Wes?

DR. WES SMITH: Yeah. That's generally correct that the same length or age correlations that are true for mercury don't seem to hold for PFAS. So it sort of complicates matters and I'll speak to that a little bit in my presentation.

KEN SZUTU: Thank you.

ACTING CHAIR LUDERER: I have a related question. What about the -- sort of the whether the fish are predatory fish or, you know, lower on the food chain, does that not hold for PFAS either?

DR. WES SMITH: It seems to, to some extent, but not completely.

ACTING CHAIR LUDERER: Thank you.

Any other comments or questions from Panel members or from the public?

All right. I think we're actually a little past the time. So, thank you again, Kelly, for that great presentation.

And now I'd like to introduce our next speaker who we've already heard from and looking forward to hearing more. So Wes Smith joined OEHHA in 2012 as an Associate Toxicologist in the Water Toxicology Section. And now he's Chief of the Fish Ecotoxicology and Water Section. And today, he'll be presenting on considerations for PFASs in OEHHA's fish advisory development process.

Thank you, Wes.

(Thereupon a slide presentation).

DR. WES SMITH: Thank you. So I'll have a mix of describing our fish advisory development process and how we're thinking about PFAS, as well as presenting some data

of PFAS in fish in California.

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DR. WES SMITH: So starting out that our advisories apply to recreationally caught fish. So these are not commercial fish and it's all State waters, including marine waters of California or -- State waters of California, which are -- tend to be about three miles offshore that are guidelines for how one can safely consume fish from zero to seven meals per week. We currently don't go beyond seven meals per week. We use the best available science to balance the risks and benefits of eating fish. And we do a thorough data review and also use some professional judgment based on our experience collectively.

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DR. WES SMITH: So here is an image of our fish advisory webpage. There's some general information as well as I mention the two populations we offer advise for. And I'll explain more about that in the next slide. We have general information, including a short video shown to the right that we developed with UC Davis extension in how to use fish advisories. Also listed are for statewide advisories. So these apply to areas that don't have site-specific advice. And those are available for lakes and reservoirs, the California coast, rivers, streams and

creeks, and also for migratory fish like salmon. And then we also in the upper right have an advisory map that provides links to locations for sight-specific advisories.

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DR. WES SMITH: And then for the basis of the two populations we offered advise for, it's based on mercury. And that's the relative sensitivity and the increased sensitivity of the developing nervous system. So we focus on women of child-bearing age, which we define as 18 to 49 years and children 1 to 17 years as the sensitive population, and the general population as women 50 years and older and men 18 years and older.

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DR. WES SMITH: And here is a current snapshot of our advisories. And in the table in the upper left, you can see what we call our risk drivers. Those are the chemicals that restrict in the most restrictive advice. And then on the right are the percent of advisories. And you might wonder why that doesn't equal a hundred percent, because there's multiple species in advisory, so you can have fish -- one species with mercury and another species high in PCBs. So we've got these different numbers in which mercury is one of the worst offenders, with PCBs being the -- kind of a distant second being a little less than half. Selenium is a little under 10 percent. And

then DDTs, dieldrin, and PBDEs taking up the rest.

And on the right is a map of all the site-specific advisories to date with an inset of San Francisco bay just showing all of the areas that have different advisories.

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DR. WES SMITH: And then this is an image that Kelly also showed. This is the sensitive population in San Francisco Bay. And you can see a vast majority of these fish we recommend no consumption or limited consumption of one serving per week and that's due to PCBs in mercury. And I show this as a transition in describing how we develop our advisories.

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DR. WES SMITH: So there's a multi-step process starting out with collection of fish. And the Safe to Eat Workgroup that Kelly mentioned is hosted under the State Water Board. And they arrange for collection of fish samples that are used in advisory development. These fish are also collected for other water quality programs. We also receive additional samples from water utilities, dam relicensing, and then some other governmental and non-governmental organizations.

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DR. WES SMITH: And then the samples are

generally sent over to Moss Landing Marine Labs, where they are processed and analyze for contaminants. And some contaminants are analyzed by other contracted labs.

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DR. WES SMITH: And the fish tissue data are uploaded into the California Environmental Data Exchange Network, or CEDEN. And this is a publicly accessible database that has data for fish tissue as well as a range of other parameters involving water quality. We download these data for specific waterbodies when we are developing an advisory.

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DR. WES SMITH: And then we screen the data to make sure it meets our minimum fish length, which is either a legal length or what we define as an edible length, which we operationalize as a length at maturity about -- which is about 80 percent of its overall length. We're trying to get a good approximation of what the contaminant concentrations will be, so we do have protective advice.

For our sample size, we require a minimum of nine individual fish, but this changes depending on the size of the water body. And we also screen to make sure all the data meet our criteria and we confirm a final data set and take -- and develop a tissue concentration for each

chemical for each species at each water body. So generally, at least PCBs and mercury are analyzed. So we'll develop tissue concentrations for both of those for each species.

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DR. WES SMITH: And then we compare the tissue levels to our advisory tissue levels that have been developed for eight compounds to date. And they provide the recommended eight ounce servings of zero to seven per week, again encourage health benefits. And we don't view these as bright lines, but they're our starting point. And we typically look at specific characteristics of the fish species, the range of the tissue concentrations, the specific habitat, the specific water body. These data aren't as -- quite as clean as laboratory data, so there's a lot of lumping and splitting so to speak. And the ATLs can be based on cancer or noncancer endpoints.

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DR. WES SMITH: And just for a frame of reference, this is our noncancer endpoint, which we use an RfD that's chemical specific, a default body weight of 70 kilograms, a consumption rate of one to seven 8 ounce servings, which is approximately 32 grams up to 224 grams per day. And we also utilize a cooking reduction factor for organic contaminants, like PCBs and DDTs, assuming

that the fish is cooked and those juices are allowed to drain away.

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DR. WES SMITH: And for our cancer endpoints, we use a risk level of 10 to the minus 4, rather than 10 to the minus 6, because we're trying to encourage fish consumption and provide benefits. We also develop other levels, which we call fish contaminant goals, which are more of a screening level that use a 10 to the minus 6 risk level. And then beyond that, we use the same body weight of 70 kilograms per day, a cancer slope factor specific to each contaminant and then 30 years of exposure over a 70-year lifetime and we use the same consumption rate and cooking reduction factor for specific organic contaminants.

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DR. WES SMITH: So we have developed ATLs for mercury and selenium. Mercury again being a specific issue in California due to gold and mercury mining. It tends to be most pervasive in Northern California, but it's also a global pollutant due to atmospheric fallout. Selenium is an issue is seen around 10 percent of our advisories and that tends to be more problematic in Southern and Central California.

We also look at PCBs and PBDEs. And then four

pesticides, of which the last two chlordane and toxaphene don't -- aren't -- don't currently drive the risk in any of our advisories.

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DR. WES SMITH: And this the table of all the ATLs, but I won't make you strain and try to understand all of this.

[SLIDE CHANGE]

DR. WES SMITH: So we'll focus on the two populations for mercury. And one of the first numbers that we look at is the do-not-eat threshold. So for the sensitive population, it's 440 parts per billion and then 1,310 parts per billion for the general population. And this threefold difference is due to a threefold difference in the RfD used to derive these numbers.

And we also focus on these one to two meal per week frequencies, understanding that the literature suggests that these are where the benefits are gained and tend to plateau at around that two meal per week category. So they tend to be some of our important markers.

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DR. WES SMITH: And because these chemicals don't play nicely, they all tend to accumulate in fish. We do evaluate certain chemicals together, such as mercury and PCBs, which have similar adverse developmental effects.

And we use a hazard index approach assuming additivity among the compounds. And this just means that two or more chemicals may result in fewer servings per week than would be one chemical alone and this is just carried out for the sensitive population.

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DR. WES SMITH: So after we go through all that process, we develop a final report and posters that are posted on our website as well as a press release and fact sheets. And posters are produced in English, and Spanish, and occasionally other languages where merited.

[SLIDE CHANGE]

DR. WES SMITH: So transitioning over to PFAS, I'll focus San Francisco Bay. This is our best data set in California to this point. And I'd like to thank Jay Davis and Miguel Mendez of the San Francisco Estuary Institute for providing these slides to me. And I would agree that this is a great monitoring program. It's 30 years old. And it -- one of the benefits of this monitoring program is how often the sampling is repeated every three years up to 2009 and then five years since then. This is a sampling year, so data will be coming forth. There are a range of species of many hundreds of samples, many contaminants, but yet still gaps remain.

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DR. WES SMITH: And then focusing specifically on PFAS, SFEI started monitoring PFAS in 2009, and also 2014, and most recently 2019. And they used six locations. It's a little hard to see on the monitors, but the purple circles denote those locations in which they look at five species, 111 fish, and 16 samples. And it's a general practice to combine fish in what's called a composite to lower the cost of -- the analytical costs, but yet again still gaps remain. And to help close some of those gaps, SFEI has initiated evaluating some archive samples. So they're looking at samples for four species across different years of collection. And this is especially useful because some of those earlier analyses didn't have as sensitive of analytical techniques, so it -- this reanalysis -- this current analysis will bring some of those detection levels down to where the concern for human health is. And again, it is a targeted method for 40 PFAS.

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DR. WES SMITH: And this is some of the data from 2019 looking at both species and location. And you can see that that highest bar over to the left is for large mouth bass. And that is a freshwater species. And that is separate from San Francisco Bay due to a weir that separates the fresh water and the salt water. And it's

also been noted that there's a wastewater treatment plant outfall in this region, so it's potentially a worst case scenario.

And also, if you look at the next highest three bars, and if -- the common denominator there is that they're all in the south bay. And this has been postulated that it's due to a shallower bay. There's less tidal flushing and less fresh water influence. But all of the species that have been sampled there have relatively -- oops, I got a little -- I was getting away from myself -- high levels of PFAS. And to some of the questions about some of the more predatory fish accumulating more, the largemouth bass, stripe bass, and white sturgeon are higher trophic fish that are piscivorous eating other fish, so we do see higher levels. But the white croaker and shiner surfperch tend not to be as predatory, but yet they still accumulate relatively high levels.

And then just in line with some of the data that Kelly presented, focusing on PFOS and some of the PFNA, PFDA, and PFUnA, someone of the longer chain compounds making up that balance of PFAS, but also noting that PFDA and then some of the 12, 13, 14 chains also tend to be present within fish tissue.

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DR. WES SMITH: And so that's our best data set as of yet in California. But the State Water Board is really undergoing an effort to look at more samples across the State in which this 150 archived samples are slated for analysis and this is a bit of a teaser for the next STEW meeting. That is a public meeting, so people can register there and find our more about where samples will be coming from.

[SLIDE CHANGE]

DR. WES SMITH: So just some of the considerations for both bioaccumulation and exposure for PFAS. Some of the historical models that have been used for persist organic pollutants like DDT and PCBs are not necessarily predictive of PFAS accumulation. And that's just due to some of their physical chemical characteristics that can change across the different compounds and complicate matters. Again, there's no real correlation with length or age that we've seen. Some of the smallest species we've looked at have some of the highest levels. And part of that is due to the analysis, whether it's done on whole fish, fish organs, or just fish filet.

And also higher trophic species, like the striped bass, and largemouth bass tend to be high. But also bottom-feeding species in this the one study by Ye at al.,

carp were very high in PFAS levels.

And to reiterate what Kelly noted that PFAS tends to dominate the fish -- the concentrations in fish tissue.

[SLIDE CHANGE]

DR. WES SMITH: So in development of ATLs for PFOS, we're reviewing literature. The SF Bay RMP sampling is their best data set. We also have some data for Russian River. And there was some PFOS sampling down in San Diego Bay, but there are a lot more samples coming with the new statewide sampling. And also scoping our ATLs, we're planning to start with PFOS, just given that it is so abundant in fish tissue. And one thing to note is that the water program at OEHHA has developed health protective concentrations for drinking water that were used in the development of a public health goal for PFOS.

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DR. WES SMITH: And looking forward, we're also evaluating levels of PFOA in tissues. And as Kelly also stated, this tends to be more the case for shellfish, because they seem to show higher levels than do finfish. And also evaluating some of the other compounds that are detected in seafood. Again, as Kelly noted, that these four compounds acute -- account for 95 percent of the PFAS found in freshwater fish.

And just back to the point of the freshwater

versus marine fish, typically, marine fish tend to be lower from studies done by FDA. And I think that may be due to just exposure sources versus San Francisco Bay is salt water, but it's surrounded and has such high anthropogenic input.

[SLIDE CHANGE]

DR. WES SMITH: So I'd just like to thank folks at CDPH Biomonitoring. We've had some great conversations over the past year, again folks at SFEI, the OEHHA fish team, and Anna Holder is an amazing human who makes the STEW happen and makes them a very enjoyable process. So with that, I'll take any questions.

ACTING CHAIR LUDERER: Thank you very much. That was a great presentation.

Any -- we'll start with questions -- clarifying questions from the Panel as usual. Start with Oliver this time.

PANEL MEMBER FIEHN: Okay. I beat Tom. So, yeah, thank you. That is amazing.

I was looking, or specifically interested in this huge differences on location. For example, the white sturgeon south bay versus Suisun Bay sixfold differences, right, or more, maybe eightfold or so.

You know, and we see this also for, you know, like at least twofold differences for other fish like the

shiner surfperch and the white croaker. Why is that?

DR. WES SMITH: There's some -- like I mentioned, some of the tidal flushing will -- for the north bay, the water will -- at least if the PFAS are dissolved in the water, they'll move out and there will be some mixing with the marine waters. And also with the Sacramento River flowing in, there's that fresh water input. Also being that the south bay tends to be shallower, so there's less chance of PFAS -- there's less dilution and less chance for them to sediment.

I believe there has also been some suggestion that sediment levels tend to be higher in the south bay. I don't know specifically what those -- the inputs are, other than that one cited outfall by Artesian Slough.

PANEL MEMBER FIEHN: Okay. Thanks.

ACTING CHAIR LUDERER: Tom, you had a question.

PANEL MEMBER McKONE: Yeah, I actually have two, but hopefully they're short. So, I mean, the first question in the equation you had for ATL and body weight you said 70, but do you actually use a different body weight for women and children?

DR. WES SMITH: We use the same body weight for both. It's sort of an average body weight to cover -- I guess it's on the lower end to make it more health protective, but we use different RfDs for --

PANEL MEMBER McKONE: Oh, okay. So you use a child-base RfD or --

DR. WES SMITH: It's from studies based on exposure and developmental endpoints.

PANEL MEMBER McKONE: All right. Well, those studies would probably adjust the body weight, I'm guessing. But, you know, the children-specific studies and women of child-bearing age probably account for the number of differences --

DR. WES SMITH: Right, the exposure in --

PANEL MEMBER McKONE: -- number of exposure differences.

DR. WES SMITH: Right, in the -- at the specific endpoint.

PANEL MEMBER McKONE: And I guess the other question is you did bring up the problem with the bioaccumulation model, you know, the classic model that was developed for PCBs, DDD, DDT and all the other like chloro -- organochlorine compounds work great. I mean, the lipid Kow, right, was a great predictor because it's a water lipid ratio. It doesn't work for these compounds.

Is there a model that is out there that you've been able to use or has anyone proposed a model for the kind of bioaccumulation we see from PFAS compounds through protein binding and other mechanisms?

DR. WES SMITH: I haven't personally seen a model. And again, it would -- I assume it would have to be PFAS specific, because some of their characteristics change and some of the information I've read described some of the differences in sort of a -- the way they like surfaces, because they're a surfactant, so that can really skew the way the models predict what the bioaccumulation might be like. And so the short answer is no.

And also studies looking at bioaccumulation factors have shown differences depending on the water body and the bioaccumulation chain for different types of species. I think as more research is done, we'll better understand that problem. But generally for our fish advisory program, we rely on fish tissue data, so we don't have to worry about the uncertainties of the bioaccumulation, because -- to provide better public health advice.

PANEL MEMBER McKONE: That makes sense. Thank you very much.

ACTING CHAIR LUDERER: Kind of related to that question is, has anyone looked at whether there's interactions with these? You know, if you're -- because you're not just exposed to one of them at a time, right, or the fish aren't, in terms of bioaccumulation?

DR. WES SMITH: That's a really good question. I

haven't seen anyone look at the interactions. And it's interesting that we've seen some data in planted trout which usually are very low in contaminants, but that showed high levels of PFAS. And my assumption is its through insects accumulating in the PFAS, because the trout when they're young tend to eat insects, which is different than the other contaminants we look at, because mercury is generally methylated, and then it's up the chain through fish eating other fish. And the PCBs are more of a lipophilic related phenomenon. So it's -- yeah, lots of questions to be asked and answers to be found hopefully.

ACTING CHAIR LUDERER: Thank you. Very interesting.

Any other questions from the Panel members or comments, discussion?

Do we have any from the public, any questions, people who wanted to speak?

No.

STEPHANIE JARMUL: Nothing online.

ACTING CHAIR LUDERER: Okay. Thank you.

What do you think is the most --

PANEL MEMBER QUINTANA: I just -- oh, sorry, Ulrike. It's Jenny.

ACTING CHAIR LUDERER: Yes, go ahead.

PANEL MEMBER QUINTANA: I meant to -- I couldn't find the button to raise my hand. Sorry.

ACTING CHAIR LUDERER: Okay. Yeah, actually -- okay. Yeah. I was just hoping I hadn't missed you, so go ahead.

(Laughter).

PANEL MEMBER QUINTANA: Sorry about that. I just had a quick question, and I apologize if this was covered earlier, but I'm wondering how much is really known about imported freshwater fish? I've seen some stuff on marine fish, but -- like if people were thinking, oh, I can't catch my own, but I'm going to buy this other stuff that comes from some other country, like how much is known about substitutes for these fish?

DR. WES SMITH: Yeah. I mean, the U.S. FDA did a study, Young et al., that Kelly cited and I also had it in mine, and they -- there were PFAS detected in certain shellfish. I think most of those species were relatively low in the -- finfish were relatively low in PFAS. But again, it really depends on where the fish are caught. And so it's highly variable.

PANEL MEMBER QUINTANA: Yeah. I wasn't thinking so much studies around the world, but I'm just wondering literally on imports into our country if people have studied those imports.

DR. WES SMITH: Just -- I don't know. I don't remember specifics from that study, but they did look at specific imports. And one of their imports -- I think the clams from -- I don't recall if it was Asia were -- tended to be higher in PFOA.

PANEL MEMBER QUINTANA: Thank you.

ACTING CHAIR LUDERER: You know, you talked about the regional monitoring program, and that sounds like that's been going on for some time. Do we have time trend data for any of these PFASs from that program?

DR. WES SMITH: They have looked at time trend data, and they're, I think, slightly increasing unfortunately. Most of the work has been done on mercury and they're -- a lot of the statewide sampling is to look at mercury. The PFAS time trends are just for San Francisco Bay and they're either steady or a slight increase, but hopefully we'll see a decrease over time.

ACTING CHAIR LUDERER: It would be nice if we could report a decrease already, but -- any other questions from -- yes, Oliver.

PANEL MEMBER FIEHN: Yeah. Great again. I mean, fantastic discussion. And every time I think that's a good question. So I have another question on how to reach the people. So this is an OEHHA guide and then some specific on the San Francisco Bay. And, you know, you

have all the other advisories. So usually you know them like, okay, don't eat this fish, because it's endangered, and this one is very endangered. And now we have another one that says, well, this one has mercury and this one has my PFAS, and this one is from a location where you have maybe more PFASs.

So how do we convey information, and what's the strategy here, and how many shops, supermarkets, Costcos of the -- you know, let's say in the north -- in Northern California are using this?

DR. WES SMITH: Yeah, that's a really good point and that's our biggest area of where we put effort in is trying to communicate this information. And again, we're just focusing on recreationally caught fish. So we do produce those --

PANEL MEMBER FIEHN: Okay.

DR. WES SMITH: -- advisory posters, but we also try to work with different groups, such as the realignment program through the STEW and some of the work that CDPH done -- has done with the AAPI communities around the Bay. We try to do more of a train-the-trainer approach, so we provide information to community groups where possible, so that the information can be disseminated that way.

PANEL MEMBER FIEHN: Markets.

DR. WES SMITH: We haven't real -- again the

recreationally caught fish that aren't sold because it's not commercial, so these signs are posted at waterbodies. It's a State requirement that they're posted in areas where people will likely see them.

ACTING CHAIR LUDERER: Are there any comments from -- oh. Okay. Great. Thank you. I wanted to let everyone know that the -- we can open up this discussion, not only for questions on Wes's presentation, but also on Kelly's presentation. So if folks have additional questions about the -- that presentation on seafood consumption among Asian/Pacific Islanders in the San Francisco Bay.

STEPHANIE JARMUL: I do have Ken online.

ACTING CHAIR LUDERER: Great.

STEPHANIE JARMUL: Do you want to unmute yourself, Ken?

ACTING CHAIR LUDERER: Thank you.

KEN SZUTU: Thank you.

And first of all, yes, I did see this warning postcard saying -- posted on -- along our shoreline of where people can do fishing. And I think that what I learned today is actually I think that the poster will be different at different location. Originally, when I saw that, I thought that would be like one poster like everywhere you would see them, but today it looks like

it's going to be different at different location. That's the first thing.

The second thing which is I want to even make it more complicated, is I was wondering, is there any consideration regarding, for example, the nuclear wastewater discharged from Japan and will that be carried into the coastline of California?

Thank you.

DR. WES SMITH: Yeah. Thanks for the question. The advisories will be different at different waterbodies. And thank you, I'm glad to see that they are posted there and still remaining, because sometimes they get taken down.

The Fukushima disaster predated my time at OEHHA, but it was my understanding that the levels of radionuclides were low enough that there wasn't a risk to human health through the consumption of fish.

KEN SZUTU: Okay. Thank you.

ACTING CHAIR LUDERER: Thank you. Yeah. Tom.

PANEL MEMBER McKONE: If I could comment. I spent a lot of time -- I was around for Fukushima. I ended up being a resource for -- with the university. So we looked a lot with the nuclear engineering department. We spent a lot of time looking at levels airborne and what would come to the coast. I mean, the basic thing is the

Pacific ocean is really large. I mean, there's a lot of dilution there. And you couldn't find the radiation of Fukushima given the naturally occurring radionuclides that be would in the water anyway.

So it wouldn't be -- it would be kind of a futile exercise to go looking for anything. But, I mean, the advantage we have is distance, but the quantity of water in the Pacific ocean, if you want to look it up, the dilution factor you get is phenomenal fortunately.

KEN SZUTU: Thank you. But I think what I'm concerned about is just like the pollution for plastic in our ocean. I think if we just look at one source, yes, it is -- it can be diluted very quickly. But I think the concept of letting the source discharge polluted water into the ocean is a concern. Because if we look at plastic, it's like what's wrong with throwing a bottle in the -- in the river or in the ocean? But if we have everybody around the world is doing that, I think that is going to be a cumulative impact.

Thank you.

PANEL MEMBER McKONE: Yeah, just to comment. I wasn't discounting any other -- I mean, the problem with plastics and microplastics is the quantity is huge and they don't sink and they don't dilute well. They tend to, I think, stay more toward the surface. So no, it was not

an excuse. It's just don't worry about Fukushima specifically, but I was not trying to discount that we shouldn't -- that we should be able to dump things into the ocean, because it's so big. It's really not --

KEN SZUTU: Thank you.

PANEL MEMBER McKONE: -- in the context of other things.

KEN SZUTU: Thank you for that clarification.

DR. WES SMITH: And microplastics are a whole nother conference.

ACTING CHAIR LUDERER: Oliver.

PANEL MEMBER FIEHN: So since we are in a general discussion and it's always fun to have a general discussion, and looking at all the speakers today, looking at surveillance and monitoring, and then slowly looking into risk assessments. Okay. That's what you try to do here, you know. When you do these warnings, and risk assessment is, of course, very hard, because you have to know a little bit about long-term effects and we don't necessarily.

At the same time, you know, we understand now that the EPA has given specific limits for PFOS and different types of categories in drinking water. So they have some data that limits some exposures there. But I wonder at the same time that when I went to the ASMS,

American Society for Mass Spec, there were lots of talks about, you know, untargeted, non-targeted analyses of PFAS to understand how many there are. And there were numbers that reach in the thousands of individual species.

Okay. So that raises the question, are we looking at the right targets? And what do we know about individual risks for specific diseases, specific long-term consequences? And we didn't talk about it today here obviously. But from those three experts of panel -- people of speakers who, you know, may be -- or anyone else, what do we know today about different classes and relative risks for long-term consequences and illnesses?

So that's a very generic question, a very general discussion section.

DR. WES SMITH: Yeah. I haven't focused on the broader work -- the health effects of all the PFAS. I mean, because of the complexity of the bioaccumulation and the other contaminants we're dealing with, we've been focusing primarily on those that occur in fish. But there's also all the other contributions and if there's additive or synergistic type of effects, it's -- I think it presents a very complex problem to think about.

I mean, and just looking at the potential for exposure and damage from half-life I think is one potential approach to know how long these compounds stay

in the human body and fact -- as one index of risk assessment.

PANEL MEMBER FIEHN: So like short-chain sulfonated, because the turnover might be higher, would get a lower risk score?

DR. WES SMITH: Possibly. It would have to be looked at, but it's a way possibly to tier how you think about the different compounds and then maybe look at relative toxicities over time. I'm just throwing -- I'm hypothesizing different potential approaches.

PANEL MEMBER FIEHN: So, in mouse models?

DR. WES SMITH: Yes.

PANEL MEMBER McKONE: Can I follow up with --

ACTING CHAIR LUDERER: Yes, Tom.

PANEL MEMBER McKONE: So are there -- often we get toxicity data for aquatic species before we get it for humans, right, just because it's easier to -- are there guidelines for eco -- aquatic ecosystems or fish species out there that have been published by, you know, academic or --

DR. WES SMITH: I -- to be honest, I haven't seen a whole lot. I've seen -- there are clearly studies on the envir -- the ecotoxicology of PFAS, but I haven't seen where there's been a huge -- and it may be out there, just because I haven't looked at. I've seen more in the

microplastic realm than I have in the PFAS realm, but I'm not that familiar with that literature.

PANEL MEMBER McKONE: I mean, sometimes they tend to do relative ranking. I mean, that would -- to Oliver's question, that might give us some insight about which ones are more or less toxic, because most of toxicology is rats and mice anyway. And so why not -- why not start with fish.

DR. WES SMITH: Kind of like species sensitivity distribution.

PANEL MEMBER McKONE: Yeah, right, species sensitivity would be -- I guess -- and then another question is, is OEHHA working on, you know, a guideline for these compounds, like a unit risk factor or slope factor, something like a REL?

DR. WES SMITH: Like a -- are you talking the other compounds beyond PFOS, PFOA?

PANEL MEMBER McKONE: Or just the -- in the PFAS family, are there any listings on -- I was going to look, but --

DR. WES SMITH: There is a public health goal for both PFOA and PFOS. So we have derived cancer, noncancer health protective concentrations as they're termed. And there are other programs within OEHHA working on that, but I'm definitely not the best person to speak to that.

PANEL MEMBER McKONE: Yeah, I'm just -- I mean, if somebody else needed to. I was just curious whether the -- because for most chemicals, you just go to the website, type in the name, and then you get -- you get the RELs, you get the public health goals, you get Prop 65 numbers. It all pops right up on one website. So that's a --

DR. WES SMITH: And I think we have the right person for that.

PANEL MEMBER McKONE: Yes.

ACTING CHAIR LUDERER: Martha

DR. MARTHA SANDY: Martha Sandy. Yeah, so there's a whole group of people in OEHHA and various programs looking at PFASs. The water program, besides having the public health goals that Wes mentioned for PFOA and PFOS has notification levels for other PFASs. And I don't want to name them, because I'll probably miss them -- some of them, but there are several. And then there's active work ongoing.

Under Proposition 65, we've listed a few PFASs, either as carcinogens or reproductive toxicants, so PFOA and PFOS for cancer. PFOA, PFOS, PFNA are all listed for reproductive toxicity, but it's -- there's ongoing research and we're actively monitoring what other groups, states, and the federal level are doing as well, and the

research. And some of these numbers I'll just say are based on human studies, PFOS, the cancer slope factor is based on epidemiology, not toxicology data.

PANEL MEMBER McKONE: Thank you.

ACTING CHAIR LUDERER: Yeah, I think there's also human data on hypertensive disorders of pregnancy, if I'm remembering right with PFOS.

Any other comments, questions?

Public input.

STEPHANIE JARMUL: I can ask a -- this is Stephanie, but --

ACTING CHAIR LUDERER: Okay. Great.

STEPHANIE JARMUL: Can you hear me?

ACTING CHAIR LUDERER: Yes.

STEPHANIE JARMUL: Jenny was curious, are signs posted at stores that sell fishing gear or bait? This is for Wes.

DR. WES SMITH: Occasionally, as long as a -- the owner is willing to post them. They're -- yeah, for the -- for OEHHA's fish advisories, some of those are posted either near or at fishing locations. And that's a relatively new program that has been implemented over the past couple years and is still ongoing to -- where there's the requirement for those signs to be posted.

ACTING CHAIR LUDERER: All right. Any additional

questions, discussion, Panel members, members of the public?

All right. Then I think -- let's see, I think we have coming up after this a 15-minute break. Shall we just take that now and do a little -- yep.

STEPHANIE JARMUL: Yeah.

ACTING CHAIR LUDERER: Okay. So we'll do that. And than you so much, Wes, that was a great talk.

DR. WES SMITH: Yeah. Thank you very much for the questions.

ACTING DIRECTOR EDWARDS: We'll come back at 2:52.

(Off record: 2:37 p.m.)

(Thereupon a recess was taken.)

(On record: 2:58 p.m.)

ACTING CHAIR LUDERER: Welcome back, everyone. In the next agenda item, we're going to be hearing from Dina Dobraca. Dina is a Research Scientist at CDPH and today she's going to be presenting on trends of PFASs and persist organic pollutants, or POPs, in pregnant Californians. So, welcome, Dina.

(Thereupon a slide presentation).

DINA DOBRACA: Good afternoon. Thank you.

I will be presenting on all of the descriptive statistics from Biomonitoring California's studies

measuring environmental chemicals in prenatal samples.

[SLIDE CHANGE]

DINA DOBRACA: As background, the aim of our work is to have population level estimates to help assess exposures of potentially harmful chemicals throughout the state of California. And with the set of studies that I'll be presenting on today, Biomonitoring California has examined the feasibility of using prenatal samples from the Genetic Disease Screening Program. And this collaboration with the Genetic Disease Screening Program has allowed the Program to obtain regional or county-wide samples without the logistics and cost of going into the field.

[SLIDE CHANGE]

DINA DOBRACA: The Genetic Disease Screening Program contains multiple programs. The one that we are collaborating with is the Prenatal Screening Program. Prenatal screening is offered to all pregnant individuals statewide to test for birth defects during the second trimester from 15 to 20 weeks gestational age.

Historically, about 70 percent of California's pregnant population participates. And there are a number of benefits to utilizing these samples, including the standardization of their sample collection materials, in particular the narrow sample collection time frame.

[SLIDE CHANGE]

DINA DOBRACA: The California Biobank Program houses the Genetic Disease Screening Program's biospecimens. And prenatal samples from seven counties total are archived, the ones shown on the map, the Central Valley counties as well as Orange and San Diego. Prenatal samples from all other counties in the state are typically discarded after one month, but can be saved upon request.

[SLIDE CHANGE]

DINA DOBRACA: So MAMAS, or Measuring Analytes in Maternal Archived Samples, was a project that Biomonitoring California undertook that leveraged the Genetic Disease Screening Program to collect information on exposures throughout the state. This map is the sampling regions included in the MAMAS study. MAMAS 1 we utilized the archived samples in California Biobank, and they're from Orange and San Diego counties and it's shown in orange on the map.

For our second collaboration with MAMAS, we collected samples throughout the state, and thus we required non-biobanked counties. The regions we selected from are shown in blue. The northern counties, the Alameda/Contra Costa, LA County alone, and then San Bernardino/Riverside.

And then for MAMAS 3, the three regions are shown

in green. It was the north bay counties, San Francisco and central coastal counties, and then southern counties going down to Imperial.

[SLIDE CHANGE]

DINA DOBRACA: I want to highlight the study design differences between the three MAMAS studies. MAMAS 1 used archived samples. Since the sampling frame is complete at the beginning of the study, there's two major benefits to that. First is that you can sample across the entire year, because you have all the samples accessible to you at the same time. And the second is that we were able to do a random stratified sampling design and have an equal number of participants from the selected race/ethnic categories of Asian, Black, Hispanic, and White. We obtained detailed Asian ethnicity information from prenatal screening participants in the region. And the most populous Asian ethnicities were Chinese, Vietnamese, and Filipina. So we restricted our Asian sampling to those three Asian ethnicities.

[SLIDE CHANGE]

DINA DOBRACA: For MAMAS 2 and 3, we were using non-biobank samples, which again are typically discarded one month after prenatal screening. So the MAMAS 2 samples had to be collected concurrent to implementing the study. Sample collection began in September of 2015 and

completed in April of 2016. Within each region, we had a region-stratified sampling design, we had an equal number of samples by race/ethnicity using a quota-based sampling, and with again the same Asian ethnicities we had selected for MAMAS 1.

And then MAMAS 3 samples, the same as MAMAS 2 samples, were from non-biobanked counties sample collection collected prospectively. Sample collection began in July of 2016 and completed in January of 2017.

[SLIDE CHANGE]

DINA DOBRACA: This table summarizes the key differences between the MAMAS studies in terms of sample size, collection dates and geography. Of note is the final column. Due to available sample volume, participants were in two distinct subsamples, they were PFAS only or they were POPs only. And so we -- you can kind of think of these as two distinct subcohorts, as I present all the MAMAS data for this presentation.

And then due to MAMAS 1 sampling counts, we had a limited number of Asian samples. And so all of the Asian samples were put in the PFAS-only cohort and none were put in the POPs subsample.

[SLIDE CHANGE]

DINA DOBRACA: Okay. So next, I will focus on the PFAS subsample and the findings across the MAMAS

studies. As measured -- mentioned by prior presenters today, PFASs are persistent organic -- or, sorry, persistent environmental chemicals with known and suspected health effects and human exposure can occur through several pathways.

[SLIDE CHANGE]

DINA DOBRACA: Our questions were, does PFAS exposure vary by demographic characteristics and what are the trends in California residents' exposures to PFASs?

[SLIDE CHANGE]

DINA DOBRACA: So here are the demographic characteristics of the PFAS sample. By design, race ethnicity was balanced across the studies, which allowed us to calculate race/ethnic-specific estimates. Due to the smaller PFAS subsample for MAMAS 3, there are smaller numbers of Chinese, Filipina, and Vietnamese participants in that study. So when we present race/ethnic-specific estimates, we will combine MAMAS 2 and 3 numbers to have sufficient numbers to create an estimate.

When we compare our data to national data, NHANES only presents an overall Asian category and does not provide Asian subgroup information in their publicly accessible data sets. So when we compare MAMAS data to national data, within each MAMAS study, we have collapsed our Asian ethnicities into an overall Asian category. So

you'll see that as well in the presentation.

We would have liked to have a measure of socioeconomic status from the pregnant individuals, such as their income or their education. Those variables are not collected with a test request form with prenatal screening. What they do have is insurance information. So we use Medi-Cal yes/no as an approximate for that variable. Again, socioeconomic status was not part of our stratified sampling design. There is a very consistent distribution of Medi-Cal insurance across the MAMAS studies.

Age was not part of our stratified sampling design. There is a consistent distribution of age across the studies. As expected, nearly all pregnancies are between ages 15 and 45 years old with most occurring in their early 30s. And next, I'll be focusing on the PFAS results from this subsample.

[SLIDE CHANGE]

DINA DOBRACA: So PFASs are ubiquitous. Of the 11 PFASs measured in MAMAS 1, seven were detected in more than 65 percent of samples. And of those same 11, sometimes they're referred to as legacy PFASs measured in MAMAS 2 and 3, six were detected in more than 65 percent of samples.

And the highlighted PFASs on this slide were

detected in every sample. This means that every child born to these pregnant Californians will have at least four measurable PFASs in their bodies. And due to the high detection frequencies of the other PFASs, most children resulting from these pregnancies will have seven PFASs in their bodies at birth.

For the next section, I'll be focusing on the PFASs listed in the middle of the slide, those six, in that order, PFOS, PFOA, PFHxS, PFNA, PFDA, PFUnDA. Those are listed in the mean concentration measured.

[SLIDE CHANGE]

DINA DOBRACA: So across the PFAS panel, there was no consistent relationship between age and exposure. And this is likely due to our narrow age range in this study and that we have exclusively pre-menopausal female population.

In MAMAS 1, we saw lower PFAS levels among those with Medi-Cal indicated on their prenatal screening request. And in MAMAS 2 and 3, we see that same lower level of PFASs for most of the panel except for the PFASs that have the highest mean levels, PFOS, PFOA, PFHxS where there was no association.

[SLIDE CHANGE]

DINA DOBRACA: A unique feature of this study design was that we had sufficient sample size to estimate

PFAS levels by race/ethnicity. So let me orient you to this graphic. The PFAS will be listed on the left. The race/ethnic groups that we sampled will be listed across the top. The first row corresponds to the geometric means measured in MAMAs 1. The second row is MAMAS 2 and 3. A single arrow indicates a higher or a lower geometric mean and a double arrow indicates statistical significance.

So for PFOS, MAMAS 1 and MAMAS 2/3, the geometric means were the highest in Vietnamese participants and then Chinese participants. And the geometric means for both Black and Hispanic participants were lower than the other groups with Hispanic participants having the lowest levels. So these are descriptive statistics. So these are not adjusted for other variables, but by the nature of our study design, these -- this sample is restricted on both age and sex.

Next row that we have here is PFOA. In MAMAS 1, the highest geometric mean was in the White participants. For MAMAS 2/3, the geometric means among Chinese, Vietnamese, and White participants were significantly higher than other groups. And then levels among both Black and Hispanic participants were significantly lower than other groups.

For PFHxS, the mean levels are highest among White participants. In MAMAS 1, levels for Chinese,

Black, and Hispanic participants were lower than other groups with Chinese participants being the lowest. In MAMAS 2/3, the levels for both Chinese and Hispanic participants were significantly lower than other groups.

PFNA was the highest among Vietnamese participants. In MAMAS 2/3, Chinese participants also had levels higher than other groups. Black and Hispanic participants had levels lower than other groups, significantly so for MAMAS 2/3.

For PFDA, the Asian ethnicities had higher mean levels with Vietnamese participants being the highest. In MAMAS 1, among Hispanic participants, they were significantly lower than other groups. In MAMAS 2/3, Black and Hispanic participants were lower than other groups.

PFUnDA differences by race/ethnicity follow the same pattern observed for PFDA with Asian ethnicities having high levels, Vietnamese being the highest and lower levels among Black and Hispanic participants.

From this slide, that summarizes kind of the legacy PFASs. There's three summary findings. First, each PFAS has different relative levels by race/ethnicity. Second is that our Hispanic participants have the lowest exposures, usually significantly lower across the PFAS analytes. And the third is that when we collapse our

Asian subgroups into an overall Asian category, we are masking that we have a highly exposed Vietnamese population across California.

So, I just want to highlight, because we heard about the ACE studies earlier today. So these -- ACE was exclusively in the San Francisco Bay Area, but these findings are consistent with what we found in ACE. With ACE 2, which was the Vietnamese, Vietnamese/American study having higher levels in ACE 1, which was the Chinese, Chinese/American study and significantly lower levels of PFHxS in ACE 1 compared to ACE 2. And so these are statewide instead of just in the Bay Area.

[SLIDE CHANGE]

DINA DOBRACA: And then this is a written summary of the three kind of overall takeaways of the previous of graphic. So next, I'm going to compare or look at PFAS trends.

[SLIDE CHANGE]

DINA DOBRACA: So nationally, PFOA, PFOS, PFHxS, PFNA have levels that are decreasing. This national data is from females 18 to 49 years old, because in the national surveys, they have not oversampled pregnant individuals during this time period, so we can't get a stable estimate as a comparison group. So this is our best nearest comparison for MAMAS data.

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DINA DOBRACA: So MAMAS PFAS levels are consistent with the national data, both in rate of decrease and relative levels by race/ethnic group. MAMAS PFOA levels again are consistent with the national data, except for Asian participants. Nationally, Asian and White mean levels are very similar. And in MAMAS 1 and in MAMAS 3, Asian participant have lower exposures than national Asian levels. MAMAS 1 participants had higher levels compared to national data. And then either due to the fact that we are changing regions with each MAMAS study or due to time, our rate of decrease is steeper in MAMAS 2/3 compared to what's seen nationally.

And then in PFNA, while levels are decreasing across MAMAS studies, the rate of decrease is not as steep as seen nationally, which again we have a combined effect of time and geography that could be affecting the kind of trend of the MAMAS studies.

[SLIDE CHANGE]

DINA DOBRACA: For PFDA and PFUnDA nationally, among Asians, levels are decreasing, while there's some evidence that levels are flat for other race/ethnic groups.

[SLIDE CHANGE]

DINA DOBRACA: For MAMAS, PFDA levels are

consistent with national data, both the kind of rate of change and relative levels of the groups. For PFUnDA, MAMAS participants have steady levels across all race/ethnic groups, so we don't see a decreasing level among our Asian participants.

[SLIDE CHANGE]

DINA DOBRACA: Okay. So I have just focused on the PFASs measured across all the MAMAS studies, the kind of legacy PFASs. Biomonitoring California's DTS[sic] laboratory measured an additional 18 PFASs in MAMAS 2 and 19 in MAMAS 3 to try to capture emerging PFASs. So to compare across studies, we've applied the same -- the highest method detection limit for comparison's sake. And a lot of the data on this plot is stacked up at zero percent detect.

And the -- just as a reminder, the MAMAS 2/3 sampling regions differ by region and time. So I wouldn't draw a -- like a line -- a trend line between the two, because it could be a geography difference that you're seeing in the different levels of detection frequency or it could be a time scale.

[SLIDE CHANGE]

DINA DOBRACA: So we see a high detection frequency of PFBA. This is a shorter four carbon chain. It's been detected in a majority of MAMAS 2 and 3 samples.

And I'll focus on this PFAS more in the next slide. I just wanted to mention that the additional PFASs that the laboratory panel add -- or that were added to the laboratory panel that were detected in low proportion in MAMAS 2 and 3 will continue to be studied by Biomonitoring California, because we're interested in emerging PFASs.

[SLIDE CHANGE]

DINA DOBRACA: So there is -- NHANES has never measured PFBA in serum, so we are comparing PFBA to other studies to understand if this shorter four carbon PFAS is being used as a substitute for kind of longer PFOS, PFOA, eight-carbon PFASs. MAMAS had one of the higher method detection limits that we found in the literature. Yet, our percent detected for PFBA was among the highest that we'd seen. When we compared the geometric means and medians for studies that had a sufficient detection frequency to calculate a median or geometric mean, MAMAS 3 had the highest mean and median values that we found in the literature.

When comparing the 95th percentile or maximum percentile reported by these other studies, there are other parts of the country and world where the range of values is higher. But population-wide, it's been most ubiquitous here.

[SLIDE CHANGE]

DINA DOBRACA: Okay. So next I'm going to be focusing on the persistent organic pollutant findings. So POPs are a class of synthetic chemicals resistant to degradation. The Environmental Chemistry Laboratory measured three panels in the MAMAS samples.

First, polychlorinated biphenyls, PCBs. These compounds were banned in the 1970s and widely used in building materials produced before then.

Second, we have PBDEs, polybrominated diphenyl ethers commonly added to foam furniture, infant products, electronics, and upholstery. A number of the people in this room contributed to the science that led to the phase out of these products in 2006 and 2013.

And third, we have organochlorine pesticides, which were once widely used for agriculture and home pest control. And all of the organochlorine pesticides measured by Biomonitoring California are no longer in use in the United States.

So Californians can be exposed directly to these products, if they were -- that were manufactured before the phaseout or if they were produced elsewhere in the world and/or because these chemicals are so persistent, they have spread throughout the environment. And so human exposure can occur through soil, dust, consuming high fat animal products, whether dairy or meat.

[SLIDE CHANGE]

DINA DOBRACA: And one of our California legislative mandates of Biomonitoring California is to assess the effectiveness of public health efforts and regulatory programs at decreasing exposures of Californians to specific environmental contaminants. So our question for the next few slides is did California's residents to these specific POPs decrease across the MAMAS studies?

[SLIDE CHANGE]

DINA DOBRACA: So first, a caveat. Again, the POPs subsample can be thought of as a separate cohort of participants. And here are the demographics of that POPs subsample. As a reminder, all MAMAS 1 samples from Asian pregnant individuals were in the PFAS subsample and not in the POPs subsample. Within each study, the race/ethnic groups that were sampled were balanced.

[SLIDE CHANGE]

DINA DOBRACA: Here, additional demographic information. Medi-Cal insurance is not balanced across the studies and neither is age with a higher proportion of younger pregnancies in MAMAS 1.

[SLIDE CHANGE]

DINA DOBRACA: So here are -- so knowing that we have these demographic differences in the POPs sample, we

still ask is the detection of these chemicals changing, so 15 PCBs were measured in MAMAS 1 through 3. And again, to compare across studies, we used a consistent method detection limit. And even though MAMAS' samples are changing by demographics, time, and region, I have highlighted a few of the PCBs, so it's easier to sort of track the percent detect across the studies. And this is an example of a public health and regulatory success story. In 2013 -- 20 -- or, sorry, 2012-2013, most of the children born to MAMAS 1 San Diego, Orange County residents would have had at least one PCB in their body. And in 2016-2017 less than half the children born to these Bay Area coastal southern county residents would have had at least one PCB in their body.

[SLIDE CHANGE]

DINA DOBRACA: And then we did the same thing for PBDEs. There were nine PBDEs measured in MAMAs 1, 2, and 3. We applied the consistent method detection limit. And I have labeled some of PBDEs, so it's easy to track them over the course of the slide. And you can see a decrease in detection frequency, where three-fourths of children have at least 1 PBDE in their body, born to the Orange/San Diego MAMAS 1 participants. And less than one-third of children in 2016-2017, born to the MAMAS 3 participants, would have at least one PBDE in their body.

[SLIDE CHANGE]

DINA DOBRACA: So there are seven organochlorines that were in the panel and two of them were detected in nearly all samples, meaning every child born to a MAMAS participant had at least one of these two banned pesticides in their bodies. So the continued detection of para,para-DDE, which is a metabolite of DDT, and HCB is of concern to California. These pesticides haven't been used for decades in the United States, though they are still used abroad.

[SLIDE CHANGE]

DINA DOBRACA: And graphed here are the geometric mean levels measured in MAMAS 1 -- or sorry, measured in MAMAS by race/ethnicity. The levels have decreased for all race/ethnicities with the highest exposure among Hispanic and Asian participants and these trends are consistent with national data.

[SLIDE CHANGE]

DINA DOBRACA: And then here are the geographic means of HCB levels measured in MAMAS by race/ethnicity. The levels are higher in MAMAS 2 and 3 compared to MAMAS 1. As a reminder, this chemical has not been in use in the United States for 40 years. And the exposure pattern appears to be fairly similar across the race/ethnic groups. And these trends are inconsistent with national

data. Nationally, levels are flat among females of reproductive age.

[SLIDE CHANGE]

DINA DOBRACA: So next steps.

[SLIDE CHANGE]

DINA DOBRACA: The MAMAS 1 study has provided Biomonitoring California an excellent opportunity to confirm the feasibility of leveraging the Genetic Disease Screening Program's collaboration to help us understand environmental chemicals across the state. And as a reminder, our goal is always surveillance work to get a representative sample and to establish trends.

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DINA DOBRACA: So with the success of MAMAS, we were able to launch STEPS. STEPS has a different study design and sampling frame that allows us to collect a random sampling design from the counties selected, which will give us a representative sample and has -- we're going back to the same counties year over year to collect trends.

[SLIDE CHANGE]

DINA DOBRACA: Additional, next steps for MAMAS is a few academic researchers have reached out to Biomonitoring California to collaborate with us. They want to understand sources of exposures in the MAMAS

sample using geospatial mapping of PFAS exposure sources. A different group wants to work with us to understand additional chemicals in these samples, so potential add-on analyses include short chain PFASs or like a total PFAS, total fluorine method. And we have collaborators working with us to look at the relationship between exposures and health, a potential add-on analysis of biomarkers of immune response as like a health outcome, as well as looking at the associations between PFASs and birth outcomes as the Genetic Disease Screening Program staff have the ability to link the sample to the birth record.

So one of the limitations of MAMAS is that per our approvals for the study, we don't have the identity of these participants, we can't return results to these participants, but we do look forward to working with our collaborators so that we're able to create materials and tools that would be a resource for pregnant Californians. And that is one of our next steps.

[SLIDE CHANGE]

DINA DOBRACA: So I just want to say thank you to our Genetic Disease Screening Program's colleagues for collaborating with us on this work. That was a summary of all the MAMAS findings. If the Panel or the public has any questions about of the analytes, let me and I can pull up more of the data. So thank you.

ACTING CHAIR LUDERER: Thank you very much. That's a very interesting presentation.

Do we have -- we can start with clarifying questions maybe from the Panel members either online or in person.

Tom.

PANEL MEMBER McKONE: Just a question about analytes. Was there any consideration of dioxin-like compounds? It would be interesting to -- I would expect they would have some sort of a downward trend since there's been efforts to reduce the sources of those. I know analytically they're harder to deal with, but I just wondered if they were considered.

DINA DOBRACA: I'm just confirming -- June-Soo, that some of the PCBs are dioxin-like PCBs, correct?

PANEL MEMBER McKONE: Yes. Yes, that's true. Some of the P -- some of the PCBs would be dioxin like.

DINA DOBRACA: Is there -- I'm not missing any other panel, correct? Those are --

(Laughter).

DR. JUNE-SOO PARK: We do measure some dioxin-like PCB. Also, the -- some PBDE. Also, the -- you know, in terms of the structure dioxin-like, but those are the very low levels, even though we measure, you know -- also the -- our lab has the capability of

measuring dioxin itself, you know, the -- Jianwen is the kind of expert measuring dioxin. But when we measure such low levels, we need a lot of volumes of blood, which is -- we don't have so -- but always look forward to, you know, those kind of -- the compound is always on our radar, because they are very toxic, more toxic than other homologs -- the isomers -- their isomers, so -- but we do have some dioxin-like PCBs or some PBDEs too.

PANEL MEMBER McKONE: Thank you.

DINA DOBRACA: And just to address the sample volume, the samples have about 1 ml of serum. So that's --

PANEL MEMBER McKONE: Yeah. And so just to follow up, if at that level, yeah, you probably couldn't find dioxins unless you had lipid samples. And I know you're not in the business of taking lipid samples.

ACTING CHAIR LUDERER: Oliver.

PANEL MEMBER FIEHN: All right. I have two questions, I guess. The first one is easy. I think it's easy. So you normalized the level of POPs I think it was on the gram per lipid. That's interesting. When people have -- this is all postprandial, is it fasted, is it -- what is the lipid here in this case? Is it total triglycerides? Is it...

DINA DOBRACA: Oh. It is a total lipid measure,

but you would know.

DR. JUNE-SOO PARK: We only measured two major lipid, cholesterol and triglyceride.

PANEL MEMBER FIEHN: Total cholesterol?

DR. JUNE-SOO PARK: Yeah, the -- that's why we use the Phillips formula to -- you know, the -- extrapolate, calculate the total lipid. We don't measure whole panel of lipid unfortunately. We do only --

PANEL MEMBER FIEHN: No, I understand that.

DR. JUNE-SOO PARK: I'm just thinking about the next question. Okay. So next question is if I look at MAMAS 2 and MAMAS 3 --

DINA DOBRACA: Um-hmm.

DR. JUNE-SOO PARK: -- you know, you said like, hey, this, and good news, and we all love good news. Things that have -- detection frequency decreased. All right. But did it decrease for things that have been banned 20 years ago, 30 years, 40, 50, long time ago, but now they decrease in frequency between 2015 and 2016 by -- sometime by half. I don't understand that.

DINA DOBRACA: So we are in different regions. This is the limit of MAMAS.

PANEL MEMBER FIEHN: Yeah. Okay. So you said that repeatedly, but --

DINA DOBRACA: Yeah.

PANEL MEMBER FIEHN: -- you know, so I think like do we -- do we sit here on a statistical fluke or is it -- how do we look at this data?

DINA DOBRACA: So this data is in line with NHANES data. Let me just --

PANEL MEMBER FIEHN: Yeah. Yeah. Yeah. All right.

DINA DOBRACA: -- see if I have the -- so one of the limitations of the -- okay. I don't have the NHANES data in -- as back-up slide. So I have -- if you would like to see, I can pull up any of the PBDEs, PCBs, by race/ethnicity to confirm that like within race/ethnic group across these regions levels are decreasing. One of the limitations of the study as it was designed, because I kind of think of MAMAS as an extended pilot, is we were testing can we use archived samples? Is there sufficient volume? And that was MAMAS 1. And then MAMAS 2/3 was can we use non-biobanked samples? Can we go outside of the geographic region and collect throughout the state to try to understand something about exposures throughout the state?

And so that was the purpose of MAMAS 2 and 3 separately. And so it makes it -- it makes it a little difficult, because I have changing demography, changing time, and changing region, when I'm looking across the

studies. The benefit to STEPS, which have samples collected already, is that we're going back to the same county using a random sampling scheme. And so then we know that the only thing that's changing is time.

PANEL MEMBER FIEHN: Yeah, I think that --

DINA DOBRACA: This one -- this one is a bit trickier. So the best I could do is I can pull up the race/ethnic specified data and just say like here.

PANEL MEMBER FIEHN: Okay. No, I understand. So, you know, because you started with like here is some good news.

DINA DOBRACA: Yeah.

PANEL MEMBER FIEHN: You know like yay.

DINA DOBRACA: I mean, it's basically the PBDE levels get so low that in our data and NHANES data we can't even calculate medians --

PANEL MEMBER FIEHN: Okay.

DINA DOBRACA: -- by the time they we get to the more recent time frames.

PANEL MEMBER FIEHN: Thank you.

ACTING CHAIR LUDERER: All right. Any other questions from the Panel members, from the public?

Okay.

DINA DOBRACA: Can I ask the Panel a question?

ACTING CHAIR LUDERER: Please.

DINA DOBRACA: So our four-carbon PFBA I'm very intrigued by. I tried to find a lot of comparison studies. It seems like another four-carbon PFBS is more likely to be measured in other studies. I think I tried to look across the like ECHO cohort, and I think they -- in three other cohorts, they measured PFBS, that I couldn't find that they had measured PFBA.

So if the Panel has any insight? Let me pull up the slide. I think it's like -- no. No -- as to if they've -- if they've heard of PFBA or PFBS being used as a substitute kind of in this time frame. Here's the data compared to the other studies. What I found really interesting is that Italy study is a comparison of contaminated drinking water and adults living outside of the contaminated drinking water zone and they had higher detects outside the drinking water zone. That Minnesota -- that's what MDH is Minnesota East Metro Study. That pilot in 2008, known contaminated drinking water site, they want back to that same population twice. And in 2014, they sent me an email with the data, so that's why it's not on this slide.

In 2014, they included both long time residents and people who had moved into the neighborhood after the water system had filtration. And the detection frequency was higher among new residents versus old residents. So

my working theory is it's not water.

And then that last study I was very intrigued by. They went back to the same policemen six months later and had very similar levels. And PFBA has a half-life in the human body of like days, and so that's six months later. And so if anyone has -- on the Panel has any insight as to this one or another four-carbon PFAS, I'd be very interested to know.

ACTING CHAIR LUDERER: Amy, I see you have your hand raised.

PANEL MEMBER PADULA: Yes. So I did -- I have worked on the ECHO data, the PFOS data and ECHO, and also hadn't come across that one. I just went back to look and I can look deeper, because sometimes if it's not detected at a certain level across all the studies, they would drop it. But I can go back, because I have the raw data, and I'll dig deeper for you to look for that.

I -- we also did a study in Hamburg, Germany and we also didn't find that that was highly detected, even though we found nine of the 18 PFAS that we had looked for were detected in almost everyone. So kind of a -- and it's also I think -- a relatively high seafood eating population. So, yeah, I'm also curious about that one that's stuck out in your data, because I haven't run across it so far.

I had another kind of minor point. It seems like in some cases you were -- when you were looking at the race -- by race/ethnicity, it was compared to kind of everyone -- each race/ethnic group compared to everyone else and sometimes it was compared to Whites. I do think that it is important to do both, because I think depending on kind of who is high and who is low, it can sometimes be somewhat misleading. So depending on how -- what the distribution is of race/ethnicity in the study population, so I think it is kind of good practice to also compare to Whites as kind of the most privileged group in many cases, although even though, in this case, didn't have the lowest levels. Just to have a -- or sometimes maybe to always compare to the group that has the highest numbers just to be able to have an equal comparison as opposed to everyone else, because it could -- I don't know. It sometimes can be misleading. Although, it seems like the findings you had were very consistent. But I just wanted to make that comment.

ACTING CHAIR LUDERER: I had a question about, you know, the national trends that you were showing and where it was, you know, notable that for PFHxS, it was -- the pattern was quite different where White -- you know, the White population had the higher levels. And do we have any information kind of about, you know, uses that

would explain the difference for that compared to the other ones.

DINA DOBRACA: So at the prior SGP meeting, Toki, my co-worker, presented that PFHxS was the analyte where she found the significant relationship between water levels and serum levels. PFHxS we assume is exclusively from industrial sources. So whether you're living near an airport, you're living near another industrial use site. But yeah, both in national data and in our data, PFHxS highest among white individuals.

ACTING CHAIR LUDERER: Thank you. Let's see. Any -- do we have anything -- any public comments?

STEPHANIE JARMUL: Yes. We have Ken. Go ahead an unmute yourself, Ken.

KEN SZUTU: Thank you. Several things. One is, for example, the comments made earlier is about like if it was in an industrial area or close to an airport, then the impact is higher. But from the region which you are doing your experiment is -- can we detect on that level? That's the first question.

The second one is I know, for example, in the major agricultural area, like Central Valley and things like that, we use a lot of pesticide and things like that. So on top of like racial difference, have you looked into, for example, regional difference? Because I think some of

the region which you are doing your study is very wide, so I'm thinking about more like agricultural and things like that. So these are the two questions I have. And also the third one is sometime I notice especially, for example, in Vallejo, we have a very -- we have like 25 percent Black, 25 percent White, 25 percent Asian, and 25 percent Hispanic. So a lot of time is the impact is socioeconomic level -- I mean, it depends on who live in certain area, because of the cost of living and all that, or within a region, is there difference between ethnic background?

Thank you.

DINA DOBRACA: So the first question was about the geospatial understanding of sources for our data. So we -- that's one of our future next steps. We have a potential collaboration with individuals out of UC Berkeley who have been working on creating maps to understand sources of exposure. We have to independently go through human subjects approval, because this is address-based data, so it's identifiable in a way, so that we are able to collaborate and so they can overlay their sources of exposure data with the addresses of the pregnant individuals. So that's a future next step. We hope to be able to do that kind of analysis.

And then the second question was about regional

differences in our data. So this data, the sampling frame is the prenatal screening data. It's the prenatal screening test form. And by the way that MAMAS 1, 2, and 3 sampled, the kind of regions as defined are not known ways to cut up the state of California. Like you typically don't sample Alameda and Contra Costa as like a region alone. You maybe sample the Bay Area as a region. So I would love to recut the data to sort of match either the CARE -- the region that were defined for the CARE study for the CARE report. There's a couple limitations. One is that's a re-ask that I have put in to our collaborators with the Genetic Disease Screening Program, because it's a finer geographic zone than was in the original cut of the data.

The data is able to be linked to the birth record. However, not everybody who goes through prenatal screening ends up with a life birth in California. So there is a loss and there is a loss due to pregnancy loss. There's a loss due to they left the state of California between when they were pregnant and when they gave birth, so they're on a birth record of a different state or there's just a bad matching algorithm. And so due to poor data quality, they can't confirm that that sample was meant for that birth record. And unfortunately, our match rate is 75 percent. And so that would be a huge loss of

data if I had to go all the way to the address for these participants. I would lose 25 percent of my data.

So I'm hoping that we are able to get a finer geography cut from our collaborators so we could get county level data on each of these participants and sort of redraw these regional lines to do the regional analysis that you speak of. And then after I have kind of the redrawn regional lines, yes, it would be very interesting to look at it by SES. There's a couple different ways we could look at it.

Again, the only variable we have from the test request form is Medi-Cal insurance, yes/no, which is pretty imperfect variable. I would prefer not to use that one. We could use the address data to get a measure of neighborhood socioeconomic status and maybe that would be our best approximate. If we linked to the birth record to be able to get information on the education and income level of the person giving birth, then we have to lose 25 percent of our data, which I would prefer not to do. So those are the three ways we could look at SES within region.

KEN SZUTU: Thank you very much.

ACTING CHAIR LUDERER: Thank you.

Do we have any other questions or comments from Panel members or public?

No, not seeing any.

All right. Well, thank you very much. That was a really interesting presentation, as were all the presentations today.

And so we have an open public comment period now. So, do we have any requests for that?

STEPHANIE JARMUL: Nothing online at the moment, but we could give it another minute.

ACTING CHAIR LUDERER: Okay.

So I could just remind everyone that webinar attendees can submit written comments and questions via the Q&A function of the Zoom webinar or by email at biomonitoring@oehha.ca.gov, and we can read them aloud here. And if you wish to speak, you can alert us using the raise hand feature on Zoom webinar and I can call on you at the appropriate time.

If you're attending in person, you can please come to the front or raise your hand and I will call on you also. For the ben -- and for the benefit of the transcriber, please identify yourself if you wish to make a comment.

Anything coming in?

No.

Okay. So I know we're supposed to have 15 minutes. Is that -- do we have to wait 15 minutes?

No.

All right. Then we can move on to our -- did you want to say something, Oliver?

PANEL MEMBER FIEHN: No. There was a call in from Amy. It just disappeared. Amy, do you want to show it again or repeat or just say what you wrote -- who you had put in the comment.

PANEL MEMBER PADULA: Oh, I just -- I just wanted to update that I just had looked it up and only two out of 150 people that we had tested for PFBA had detectable levels. So not much information there, so, but we'll keep looking.

ACTING CHAIR LUDERER: Which -- what was the year of that, do you know?

PANEL MEMBER PADULA: Actually, yes. It was a range of years. They were pregnant people between, let's see, maybe 2011 through 2018.

ACTING CHAIR LUDERER: Thanks. So any other comments, questions, anyone thought of at the last minute?

All right. Then I'll go ahead to our wrap-up. So I just wanted to announce that there will be a transcript of this meeting posted on the Biomonitoring California website when it's available and also announce that the next SGP meeting will take place on November 7th, 2024 from 1 pm to 4 pm and that one will be in Oakland.

Information regarding options for attending the meeting will be made available closer to that November meeting date.

And I wanted to thank the Panel, all the presenters, the audience, and adjourn the meeting. It was a great meeting.

Thank you, everyone.

(Thereupon the California Environmental Contaminant Biomonitoring Program, Scientific Guidance Panel meeting adjourned at 3:44 p.m.)

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 Contaminant 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 30th day of July, 2024.

JAMES F. PETERS, CSR
Certified Shorthand Reporter
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