CALIFORNIA ENVIRONMENTAL CONTAMINANT BIOMONITORING PROGRAM

(BIOMONITORING CALIFORNIA)

SCIENTIFIC GUIDANCE PANEL MEETING

CONVENED VIA WEBINAR BY:

OFFICE OF ENVIRONMENTAL HEALTH HAZARD ASSESSMENT

CALIFORNIA ENVIRONMENTAL PROTECTION AGENCY

STATE OF CALIFORNIA

TUESDAY, MARCH 7, 2023

10:00 A.M.

JAMES F. PETERS, CSR
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APPEARANCES

PANEL MEMBERS:

Megan R. Schwarzman, MD, MPH, Chair

Carl Cranor, PhD, MSL

Lara Cushing, PhD, MPH

Oliver Fiehn, PhD

Ulrike Luderer, MD, PhD

Thomas McKone, PhD

Penelope (Jenny) Quintana, PhD, MPH

OFFICE OF ENVIRONMENTAL HEALTH HAZARD ASSESSMENT:

Vince Cogliano, PhD, Deputy Director, Scientific Programs

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

Susan Hurley, MPH, Research Scientist, Safer Alternatives Assessment and Biomonitoring Section, Reproductive and Cancer Hazard Assessment Branch

Stephanie Jarmul, MPH, Senior Environmental Scientist, Safer Alternatives and Biomonitoring Section, Reproductive and Cancer Hazard Assessment Branch

CALIFORNIA DEPARTMENT OF PUBLIC HEALTH:

Kathleen Attfield, ScD, Chief, Exposure Surveillance and Epidemiology Unit, Environmental Health Investigations Branch

APPEARANCES CONTINUED

CALIFORNIA DEPARTMENT OF PUBLIC HEALTH:

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

Nerissa Wu, PhD, MPH, Chief, Exposure Assessment Section, Environmental Health Investigations Branch

CALIFORNIA DEPARTMENT OF TOXIC SUBSTANCES CONTROL:

Kelly Chen, MS, Research Scientist, Exposure Surveillance and Epidemiology Unit, Environmental Health Investigations Branch

Sabrina Crispo Smith, PhD, Chief, Biomonitoring Section, Environmental Chemistry and Biomonitoring Branch, Environmental Chemistry Laboratory

June-Soo Park, PhD, Chief, Environmental Chemistry and Biomonitoring Branch, Environmental Chemistry Laboratory

GUEST SPEAKERS:

Matt MacLeod, PhD, Department of Environmental Science, Stockholm University

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PROCEEDINGS

DR. COGLIANO: Good morning, everyone. I'd like to welcome Panel members and the audience to this meeting of the Scientific Guidance Panel for the California Environmental Contaminant Biomonitoring Program, more popularly known as Biomonitoring California. Thank you all for participating and for sharing your expertise and experiences.

The Panel last met on November 18th, 2022. The meeting included updates on the Biomonitoring California Program activities including community biomonitoring studies.

We also heard from guest speakers Nayamin Martinez, Director of the Central California Environmental Justice Network and Gina Solomon, a principal investigator at the Public Health Institute and also a clinical professor of medicine at the University of California, San Francisco. Together, they presented a study called a Filtration for Respiratory Exposure to wildfire Smoke from Swamp Cooler Air, or FRESSCA-Mujeres, which the Program is adding an exposure biomonitoring component.

The Panel, staff members, and audience members delved into planning for future Program activities. The Panel also provided feedback on current activities. Key discussion topics included:

First, planning and designing the Studying Trends in Exposures in Prenatal Samples, or STEPS project. This discussion included input on considerations for selecting counties in California for retrospective and prospective sampling.

Second, options for timing of urine collection for the FRESSCA-Mujeres project and suggestions for the types of information to be collected through the study.

Third, potential topics to consider for potent -- for 2023 SGP meetings.

A summary of input from the November meeting and the complete transcript are posted on the November meeting page at biomonitoring.ca.gov.

I'll now invite Panel members to introduce themselves. I'll call on each member and ask you to state your name and affiliation.

First, Lara Cushing.

PANEL MEMBER CUSHING: Hi. Good morning. This is Lara Cushing. I'm at the University of California, Los Angeles in the Department of Environmental Health Sciences. And just one note, I will have to step out early at about 11:50 today. Nice to be here.

DR. COGLIANO: Okay. Thank you.

Ulrike Luderer.

PANEL MEMBER LUDERER: Hi. I'm Ulrike Luderer.

I am a Professor of Environmental and Occupational Health at the University of California, Irvine.

DR. COGLIANO: Thank you.

Jenny Quintana.

PANEL MEMBER QUINTANA: Hi. I'm Penelope, or Jenny, Quintana. I'm at the School of Public Health at San Diego State University.

DR. COGLIANO: Okay. Thank you. We also have Oliver Fiehn from UC Davis, Tom McKone from UC Berkeley and Lawrence-Berkeley Lab, and Carl Cranor from UC Riverside who will be joining a little bit later in this meeting.

And now, I'd like to turn the meeting over to Meg Schwarzman from UC Berkeley who is our Panel Chair.

CHAIRPERSON SCHWARZMAN: Thanks so much. I appreciate it, Vince.

Let's see, my -- I think my first task is to provide a reminder to Panel members that -- to please comply as usual with the Bagley-Keene requirements. So that's a requirement that all discussions and deliberations of the Panel need to be conducted during the meeting and not on breaks or with individual members of the Panel on- or off-line, including via phone, email, chats, or text messages.

So our goals for the meeting today are, first,

we're going to hear a presentation from our guest speaker on the use of a population-based pharmacokinetic model to help interpret the PFAS data from the CARE study. We'll also, after that, be hearing an update on Program activities, including community biomonitoring studies, so that's a two-part update. There will be time for questions from the Panel and the audience after each presentation.

So here's logistics for how to ask and answer questions and comments, provide questions and comments. So during the question periods after each talk, it's great if speakers could remain unmuted with your webcam showing, so that you can respond to questions from the Panel and from the audience. For SGP members, if you want to speak or ask a question, please just raise your hand like physically. I'll watch you and call on you at the appropriate time. You can unmute yourself and ask your question or provide your comment. I think we're all mostly used to this by now.

For webinar attendees, if you have questions or comments for the question periods after each talk, submit them via the Q&A feature of Zoom webinar or by email. And that address is biomonitoring@oehha.ca.gov. And I will be checking in with staff about questions from webinar attendees during the process. We won't be using the chat

function during the meeting, so if you put something in that way, we won't see it. Please keep your comments brief and focused on the items that are under discussion and we'll read aloud relevant comments, paraphrasing them, if necessary, for length.

If webinar attendees want to speak during the public comment periods and discussion sessions, use the raise-hand feature on the Zoom webinar and I'll call on you.

So our first agenda item, as I mentioned, is a presentation by Matt MacLeod. I'll introduce him and then we'll go ahead. So Matt MacLeod is Professor of Environmental Chemistry in the Department of Environmental Science at Stockholm University in Sweden. We really appreciate your willingness to stay up late to attend our meeting in this time zone. He is a Fellow of the Royal Society of Chemistry and Associate Editor of the RSC journal *Environmental Science: Processes & Impacts*. He studies the factors that control human and environmental exposure to pollutants using mathematical models to quantify exposure and design and interpret laboratory experiments and field studies. The goal of his research is to build a quantitative and process-level understanding of factors that determine exposure to environmental pollutants and microplastics, and to develop practical

tools and guidance that support rational management strategies.

Today, Matt will be presenting on the application of a population-based pharmacokinetic model for interpreting PFAS data from the California Regional Exposure Study, or CARES. I'll turn it over to you, Matt. Thanks for being here.

(Thereupon a slide presentation).

DR. HOLZMEYER: You need to unmute, Matt.

DR. MACLEOD: There we are. I was just saying let me know if you have trouble hearing me. And now that worked perfectly. So I hope you can hear me now. And you see my slides, is that right, Meg?

CHAIRPERSON SCHWARZMAN: All good.

DR. MACLEOD: Perfect. Good. Okay.

Yeah, I appreciate the opportunity to talk to you a bit about -- I'm really going to talk about 13 years I think of research that I've been involved in in developing and applying what we call population-based pharmacokinetic models to describe biomonitoring data. And at the end of this talk, I will show you a few slides where we have applied this modeling approach to some of the California Biomonitoring data. And this I've done in collaboration with Kathleen Attfield.

All of this work, in collaboration with -- or

with the California Biomonitoring data, was actually possible because I came to visit in Berkeley four or five years ago before the pandemic on a Marie Curie funded secondment, which was money from the European Union that I was awarded to find and make new collaborations. So that was a nice opportunity and you'll see why as I go through the talk. Quite a bit of the talk deals with biomonitoring data from the United States. I'll only get to the California data at the end, but you'll see lots of data from NHANES as I get into the -- into the talk.

On my title slide here, I have myself as the presenter. Malicka Laroussi did a lot of the work that you'll see at the end of the talk on the California data. She's a student who worked with me until recently here in Stockholm. Kathleen, of course, is our collaborator there in California. All of these people at the bottom have been involved in developing this modeling approach over the last decade or so. Very notable in this list is Roland Ritter. And you'll see that he was the original developer of this population-based pharmacokinetic modeling as part of his PhD work about ten years ago now.

So I want to start -- let's see if this works.

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DR. MACLEOD: Yeah. I think on this -- in this Panel and in this group, I don't have to tell you that we

all have chemicals in our bodies. How much chemical you have in your body is determined by the balance between exposure and elimination. And if you want to estimate the concentration or the body burden of chemical in somebody's body, in your own body, or in an individual's body, a simple way to do this is with a one-box pharmacokinetic model. That's a model that just balances exposure with elimination to calculate concentration. So you might, for this individual, estimate intake of a chemical as a function of time, and maybe if this is time in years and this is the intake of a persistent organic pollutant, there is some increasing phase of exposure, a near peak of exposure, and then a decreasing phase of exposure.

And -- so this is your exposure function. The elimination in a simple one-box pharmacokinetic model, you could parameterize as a first-order process. Just assume that this elimination rate constant is -- that this elimination is characterized by a first-order rate constant that's independent of concentration. And this works very well for lots of different kinds of persistent organic pollutants and pollutants that we have in our bodies, especially when they're at low concentrations, such that you're not having a physiological response that's causing a concentration-dependent elimination. So this one-box pharmacokinetic model is very useful for

individuals.

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DR. MACLEOD: And it look likes this if you write it down mathematically. And here, if you don't want to get too far into the mathematics, I just drew some arrows here, so that you could see the concentration is in these two terms. On the left side of the equation it's what we're solving for, the rate of change of concentration with time in this case in a differential equation. That's actually dependent on the concentration itself. The rate of change is just these first-order elimination rate constants multiplied by that concentration.

Here now, I just told you that we would characterize elimination with a first-order elimination rate constant. There's actually two here, one for the elimination rate of the chemical, which could be by excretion into urine, for example, or into feces, or sloughing off of skin, all these different mechanisms. This other term is a rate constant for growth dilution, especial -- this is especially important for children who grow very quickly over the course of a certain period of their life. It can be important also when you speak about demographic -- in a demographic sense for older populations where people tend to lose weight as they get older and you get actually negative growth, which can

cause a concentration of the chemicals that you're carrying within in your body.

And then the exposure part of the equation is here at the end. This is, in this case, an intake function for the chemical through diet I've assumed as the -- as the dominant exposure pathway in this case. Again, this is a function of time. And if this particular one-box pharmacokinetic model equation was set up for a lipophilic chemical that tends to accumulate in lipids, so I've included here an *f* factor for absorption efficiency and then the massive lipid within the body sort of assuming that we're measuring this concentration on a lipid-normalized basis. You'll see in a second that we take away this assumption when we work with PFAS, which are not lipophilic chemicals. But that's a one-box pharmacokinetic model. Probably many of you in this group have seen this kind of model before.

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DR. MACLEOD: We turn that one-box pharmacokinetic model into a population-based pharmacokinetic model just by running it a bunch of times for different representative individuals born in different years. So that's what I've illustrated here. Each of the lines in this plot of concentration now of a lipophilic chemical in nanograms per gram lipid normalized

concentration within the bodies of people over time. Here are nine individuals, one born every 10 years starting in 1931.

So the first individual I guess is this blue line. They are born in 1931. They start to accumulate this chemical. I believe this chemical is PCB 155. You'll see it in a second on the next slide. Lots of accumulation early in life from transfers from breast feeding. Then there's a period of growth dilution perhaps, where concentration goes down a little bit. This is all superimposed upon an assumed intake function, which is increasing between the 1930s and the 1970s for PCBs. So you see all of these different individuals born in 1931, 1941, 1951, and 1961 with rising concentrations in their bodies over time up to about 1973, 1975, when you have peaks of exposures.

And then all of these individuals who were born before the peak in exposure from PCBs in this case, they all start to fall. They have declining concentrations with the same rate constant. This is determined by this intrinsic elimination half-life. People born after the peak of exposures have much lower body burdens over the course of their lifetimes, because they're not experiencing this high exposure -- this high peak of exposure.

And this is -- this is our population-based pharmacokinetic model. This is what it does. We put together a whole bunch of individual single-box pharmacokinetic models. We don't model an individual born every 10 years, but an individual born every year for about 100 or 120 years. And we use this to build a picture of the population composed of these representative individuals.

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DR. MACLEOD: And with that, we can then look at the population in a couple of different dimensions. So you can then look at across the whole population, the band of the range of concentrations in -- of, in this case, PCB 153 and PCB 52 now the range in concentrations in the whole population at different times and you can look at cross sections of the population in term -- as concentration within the bodies of the people as a function of age at different times.

So here, I took two times -- or four time slices out of this -- these population distributions, one in 1983 shortly after the peak of exposures to PCBs. At the top here are graphs of the average daily intake or the adult reference daily intake for PCBs. All of this data actually is, in this case, parameterized for the UK population, because we've used monitoring data or

measurement data from -- of body burdens of PCBs within the UK population from these two studies as a case study for the model.

This is also a nice case study, because there are many whole diet intake studies for PCBs from the UK. So we can simultaneously then fit the model to exposure levels and trends, which come from total diet surveys, and whole body or body burden estimates that come from analytical chemistry studies of concentrations of PCBs in the bodies of the people. And then we can fit the model to both of these things simultaneously to get the best possible picture of how intake and elimination conspire with each other to determine the levels that we see within the population.

And so what we see is a changing in the shape of the concentrations with age within the population. Shortly after that peak of exposure, you see almost everyone in the population over the age of about 20 has the same concentration of PCB 153 in this case. Then, in 1990, and in 2003, and in 2015, you start to see this plateau effect, where it's only the older members of the population who have this level or flat level of concentrations. That's that memory of the peak concentration. All of those members of the population have declining body burdens along that same curve. There

are fewer and fewer of them as time goes on, of course, because we don't model people above the age of about 90. We're assuming that they've died.

This is for PCB 153, which is -- which has long residence time in the body as a -- as a persistent PCB congener. PCB 52 is metabolized and excreted much more rapidly. And in that case, you don't see this sort of memory effect of the peak exposure, but instead everybody in the population is stepping down at sort of the same rate. This is determined by the rate of change of exposure actually as where -- because exposure is falling more slowly than the rate of elimination of the chemical.

So this is the kind of information you can get from this population-based pharmacokinetic modeling approach. You get explanations -- mechanistic explanations for these age concentration shapes that you see in biomonitoring data. You get explanations -- or you get a mechanistic explanation that -- of why persistent substances have different age concentration profiles than less persistent substances or less biopersistent substances, and you get a quantif -- and you get a quantification of this relationship between intake and elimination in determining concentration.

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DR. MACLEOD: So this is where we started. I

mentioned at the beginning about Roland Ritter. These are the two papers that Roland published as part of his PhD thesis. This is from 2009, 2010, so about 13 years ago now.

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DR. MACLEOD: With that as background, we became, or I especially became interested in applying this framework to perfluorinated substances, especially substances like PFOS. And the reason here, the motivation here is clear because PFOS is currently the most abundant persistent organic pollutant measured in humans. And this is just a summary of data from NHANES from a few years ago, which takes averages of this age concentration profile for PFOS. And one of the interesting things about PFOS, you see a few things that are common with the PCBs. If you look just at men, you see this increase and then a sort of a plateau, so it looks a bit like PCB 153, a persistent pollutant with perhaps long residence times in the body.

But you also see something interesting, this interesting difference between men and women within the population, where women have much lower - outside of the range of variability within the data - much lower body burdens than men, especially up to the age of about 55 or 60 when then they sort of come back together.

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DR. MACLEOD: So one of the things that we wanted to investigate starting around 2014 was this research question, could loss of PFOS by menstruation explain the different body burdens between women and men? And this was a research question that was kind of in the air around 2014.

On the first slide you might have seen the name Jochen Mueller. He's a professor in Australia who I've collaborated with on this work. And he was already looking at this question of elimination of PFOS, especially through blood loss. He was looking at cohorts of patients who have hemochromatosis, which is a particular disease which is treated by frequent blood removal to prevent the build-up of heavy metals in blood among people who don't eliminate these naturally. And he had noticed this difference between women and men and had posed this as a hypothesis in at least one research paper before we came along to try to investigate this question.

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DR. MACLEOD: So what we had to do to address that research question was modify our population-based pharmacokinetic model. Remember it's built on this framework of individual pharmacokinetic models for individuals. What we needed to do was add another

elimination rate constant to this K-elim as an extra elimination pathway from blood loss and then parameterize this to represent menstrual blood loss elimination by women as a way of addressing that research question that I talked about before.

So we did this by introducing a new term to describe losses of perfluorinated chemicals, or PFOS, with menstrual blood. This new term is just a flow rate -- a volumetric flow rate of blood loss divided by something called the volume of distribution. Volume of distribution is, if you're an environmental scientist or an environmental chemist, it's just a partition coefficient, but it's a partition coefficient with funny units that measures the distribution of perfluorinated chemicals or any chemicals between the whole body of a person or an organism and the blood.

So keep in mind as we go through the next slides, it has these funny units then of milliliters per kilogram, because you use by convention a measure of the whole body concentrations of chemicals in nanograms per kilogram and the concentration in blood in nanogram per milliliter. So you get these funny units of milliliters per kilogram. To keep in mind here is a low volume of -- a low volume of distribution means that the chemical prefers blood. You have a high fraction of the total amount of chemical in

your body in your blood, a high volume of distribution. You have a high fraction of the chemical in other tissues, other organs of your body. So PCBs for example have very high volumes of distribution, up in the millions, probably unmeasurable.

You'll see for PFOS and perfluorohexane sulfonate, we can get values here that are more like 100 nanogram per milliliter. This is a low volume of distribution for chemicals that are distributed appreciably into blood when you look at the whole body.

So with this new process description, we needed to parameterize it. Men, of course, don't have menstrual blood loss as a loss pathway for PFOS. Their flow rate of menstrual blood throughout their life is zero. Women have menstrual blood loss, so they have a non-zero loss of blood, especially in these years between puberty and menopause, between about the ages of 15 and 50, I believe, in our modeling framework.

And just for modeling purposes, we kept a set of imaginary women within our modeling framework. These were -- this is the way that we modeled women in the beginning when we were thinking about PCBs, which do not distribute appreciably to blood. We didn't include menstrual blood loss originally, so implicitly we were modeling women as not having this as a loss pathway

either. We kept them in here for comparison sake when we started to investigate this hypothesis, but these are obviously imaginary women.

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DR. MACLEOD: So this is a -- for this case study, we worked with the NHANES data. This was in 2014 or 2015. At that time, from NHANES, we had five years of cross-sectional biomonitoring data of PFOS in men and women within the U.S. population. We set up an initial estimate of the intake function or the rate of intake as a function of time of PFOS for the U.S. general population based on product use data. We used that as an input to our pharmacokinetic model. We used that then -- we used then as fitting parameters the whole body elimination rate constant and a refined intake fraction where we just used two fitable parameters to describe this intake function, so we could then go in a kind of loop here and iteratively fit the model to the data until we got the best possible fits.

And the outputs of interest then are this intrinsic elimination rate constant for men, women, and for men, for menstruating women, and for non-menstruating women, and a refined intake function estimate for PFOS over time.

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DR. MACLEOD: So what this looks like, here is the five years of biomonitoring data that we had to work with in 2014 from the NHANES study. Men are at the top in blue, women underneath in red. This looks a lot like that first slide that I showed you, men in general having higher whole body -- or higher concentrations of PFOS in their blood than women over time. Everything is falling, because by the -- by 1999 already, there was a phaseout of PFOS underway and concentrations within the population are falling throughout this period.

When we fit our population-based pharmacokinetic model to this data for men, we inferred an elimination half-life of five and a half years for PFOS. And this is the model fits in this blue line. For these imaginary women who do not have menstruation as a loss process, you can see the model fits are quite a bit worse than they were for the men. The shape of the curve is not correct. The half-life that we calculate is 4.3 years. It's faster than it is for men, which represents this sort of faster losses by women, but we're not fitting the data -- we're not fitting the cross-sectional data in a reasonable way. And only when we include this menstruation as a loss process do we get more qualitatively the right shape in this age concentration relationship for women for the PFOS in the general U.S. population.

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DR. MACLEOD: So a little bit of a frustrating thing in this was the intrinsic elimination rate constant that we calculated for men and women -- for men and menstruating women did not quite overlap our confidence intervals. Intrinsic elimination rate constant for men - this is elimination by all processes that are available to men - was about five and half year -- or was five and a half years. The intrinsic elimination rate constant for women - now this is for loss processes that are not including menstruation - is 4.9 years.

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DR. MACLEOD: These two do not overlap. If menstruation was the only explanation for the difference in body burdens between men and women, then these two should overlap, because we would have included in the model the thing -- the key thing that was different for women from men. So this was a little bit frustrating for our hypothesis. If you remember at the beginning, the hypothesis we were investigating was whether menstruation explained the difference between men and women. And from this study in 2014, we concluded that it did not quite explain the difference. It explains quite a lot of the difference, but not all of it.

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DR. MACLEOD: So here's that research question. The answer is a qualified yes. Assuming the same intake, the model fits for data were just -- or the model fits to data for women were just as good as they were for men.

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DR. MACLEOD: That's what I've shown here. Here's the root mean squared error of the model for fits to men. This is the root mean squared error for -- sorry, I'm bouncing around. This is the root mean squared error for fits for women when you include menstruation. If you don't include menstruation, there's much higher model error. There's something missing in the model. So this is part of our explanation for saying that menstruation is an important loss process.

But these elimination rate constants that should have overlapped if menstruation could have accounted for all of the differences between men and women did not overlap and this was a little bit disappointing actually at that point in our research.

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DR. MACLEOD: This study was published in these -- in this paper in *ES&T*. And there's actually a very nice comment that came afterwards from a couple of doctors -- medical doctors who helped us to -- who pointed out actually that we had parameterized menstrual blood in

not the most optimal way. And when we reparameterized with the recommendations from this comment, we actually got improved data fits with the model.

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DR. MACLEOD: So that was a nice case of getting some outside. It would have been nice to have this I guess before we published the study, but it was nice to get it corrected as well.

So this brings us up to Malicka's work and together with Kathleen. When we went to -- when I went to California and visited with Kathleen and I learned about the California Biomonitoring data, one of the things that we wanted to do was as preparation for trying to compare the California Biomonitoring data to the NHANES, to compare the California populations to the general U.S. population, was to update our work on NHANES, because by 2022 when Malicka started to do her work, the NHANES data had been expanded in the first case. There were several new years of biomonitoring data available. I don't -- did I get the -- yeah, I got these correct. 2011, 2013, and 2015 were now available.

And in addition, the 1999 data had been retracted, which was actually quite interesting for me, because if you go back in the slides and look, the 1999 data was a little bit funny looking even in our -- in some

of our population-based pharmacokinetic model fits. So we went back and revisited these assumptions.

We -- I'll show you in a second. Based on our results, we actually applied the population-based pharmacokinetic model to many more PFAS, not just PFOS, but several others using this assumption that menstrual blood loss does account for the difference between men and women. A barrier to doing this -- to doing this kind of analysis for perfluoroalkyl substances where you don't have any independent estimate of volume of distribution is that you can't apply the model without that volume of distribution information. So I'll show you in a second with these new data.

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DR. MACLEOD: Here is an example. So here's the update for PFOS, again men in blue and women in red. Now, what we had previously was 1999, that data has been rescinded or taken back by the NHANES people. So the same -- we have the same data from 2003, 2005, 2007, and 2009. And then for PFOS there, so it's just two more years of data from 2011 and 2015. Here are the model fits for these data. Here is the intake function for PFOS from the optimized model. It shows basic -- you know, this is negligible intake -- intake rising exponentially between about 1950 and 1990, then a peak of intake between 1990

and 1998, and exponentially falling exposures after 1998.

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DR. MACLEOD: The -- these model fits for men and women, now I'm only showing menstruating women. I'm not showing these imaginary women who don't, have intrinsic elimination half-lives of 4.3 years for men and 4 years for women. These do overlap when we use a volume of distribution for PFOS of 250 milliliters per kilogram. This is a value that's in very good agreement with other independent studies and in good agreement with what we used before in the 2014 study.

So now, we were in a case where menstrual blood loss does account for most -- or for enough of the difference between men and women that it's -- it seems like a reasonable assumption for other PFAS to use the model fits to estimate volume of distribution. Here's the root mean squared error plots for PFOS. And actually, we fit the women in these new NHANES data with a little bit higher -- a little bit lower root mean squared error and a little bit higher coefficient of determination than we do the men.

There's a few sort of technical reasons why that is the case. But if any of you are really interested in models and data analysis, we can talk about it in the questions I guess.

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DR. MACLEOD: Here's PFOA, perfluorooctanoic acid. A similar story to PFOS. Again, volume of distribution of about 250 or 260 milliliters per kilogram. So you see quite a difference between women and men, women between the ages of about 15 and 50 and men. Again, the inferred intake function.

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DR. MACLEOD: Now, the elimination half-life for PFOA is a bit shorter than it is for PFOS. Remember, for PFOS, this was a little more than four years. And now for PFOA, it's more like three and a half or three years. But again, there's not such a large discrepancy between the women and men in this analysis. And again, the model fits for women and men are both quite good and comparable to each other.

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DR. MACLEOD: Now for PFNA, perfluorononanoic acid, already in these -- in the biomonitoring data, you can see that the difference between men and women for perfluorononanoic acid is less dramatic than it was for PFOA and for PFOS. And if you remember what I was saying before, a low volume of distribution implies a high affinity of the chemical for blood. This smaller difference between men and women implies that

perfluorononanoic acid is less -- you know, has a higher volume of distribution, meaning higher affinity for other organs relative to blood. You do see that. This is 300 compared to about 250 for PFOS and PFOA in our model fits. The intrinsic elimination half-life is quite similar to those for PFOS, something around four years.

Again, we get an inferred intake function. An interesting thing now is even in this pharmacokinetic modeling, we're seeing now for perfluorononanoic acid a later start of the decline -- start of the decline in exposures. This is now in the year 2007. You can see this in the biomonitoring data - 2003, 2005, 2007. All of these biomonitoring years look quite similar. If anything, there's a little bit of an increase in concentrations during this time. And then you don't see a decline starting until 2011, 2013, 2015. This is in contrast if you look back to PFOA or PFOS where we had declining concentrations right through the biomonitoring series.

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DR. MACLEOD: Excuse me. Yeah. Sorry, this plot is missing. I realized when I made up the slides actually that I had the wrong plot here for the men. And rather than show the wrong data, I just deleted the plot. But you'll have to trust me that the fits for men and women

are comparable for perfluorononanoic acid as well.

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DR. MACLEOD: Here's perfluorodecanoic acid. Once again, a later start of the decline in exposures. Now you see an even smaller difference between men and women. It's getting hard to even detect visually in the plots. This volume of distribution of 600 milliliters per kilogram much higher.

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DR. MACLEOD: And again, the model fits in -- for men and women are comparable.

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DR. MACLEOD: Perfluoroundecanoic acid similar to the perfluorodecanoic. Again quite a high volume of distribution compared to PFOS and PFOA.

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DR. MACLEOD: And then finally an interesting one, perfluorohexanesulfonate. Now you see a very dramatic difference between men and women. This corresponds to the lowest volume of distribution that we've seen for any of the substances that we've looked at so far. Again, quite a -- quite a high biopersistence here, but very strong affinity for blood relative to other organs for perfluorohexanesulfonate.

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MS. JARMUL: Hey, Matt, this is Stephanie. Sorry.

DR. MACLEOD: Yeah.

MS. JARMUL: We're a little bit over. I was wondering if maybe we can talk about the CARE data and --

DR. MACLEOD: I'm very -- yeah.

MS. JARMUL: Okay.

DR. MACLEOD: I will jump there.

MS. JARMUL: Thank you.

DR. MACLEOD: I'll jump over the -- I'll jump over the inferred volumes of distribution. The summary of this is our volumes of distribution are consistent with many of those reported in the literature but not all. And then -- so finally then, here's the CARE biomonitoring data to put this in context.

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DR. MACLEOD: I think you guys in this group will be familiar with these data from 2018 from LA County and from 2019 from Southern California population.

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DR. MACLEOD: What I've done is just added them to the bottom here. So across the top here is the PFOS NHANES data that we saw before. And then using the same exposure parameters and elimination parameters, but fitting the intake functions to these data.

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DR. MACLEOD: We can get very good fits to the 2018 and 2019 CAREs -- CARE data. For PFOS, for perfluorohexanesulfonate again showing this big difference between men and women attributable to low volumes of distribution. And I only showed those two examples, but we've done all of the PFAS that I talked about before in comparing the California data to the NHANES data.

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DR. MACLEOD: So under the assumption that volume of distribution and whole body elimination rate constant are the same in the California populations and in the U.S. general population, we test the hypothesis that there is different exposures in California and we don't see obvious evidence of that. There's a few cases where there's some differences, but the CARE data is these are much smaller data sets than the NHANES data, so it's a bit difficult to say where just variability from small sample size or smaller sample sizes is causing a bit of a discrepancy. But there isn't an obvious difference in the intakes between the California populations and the general U.S. population at least in this first application of the model.

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DR. MACLEOD: So with that, I could come to

conclusions. Sorry, Stephanie, for going a bit long. But what I wanted to illustrate here was this population-based pharmacokinetic modeling as a tool for interpreting biomonitoring data. Using this, we can, for perfluoroalkyl substances, get estimates of intake levels and trends from biomonitoring data and estimates of intakes where they're available. And the model delivers estimates of intrinsic elimination half-lives and volumes of distribution for these substances.

And I'll end there.

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DR. MACLEOD: I have -- I have an acknowledgment slide, but these are all European funding agencies that funded my travel. So I don't think that they're familiar to many of you or European projects that funded my travel actually.

CHAIRPERSON SCHWARZMAN: Great. Thank you so much, Matt. We have time now for questions from Panel members and from the audience and then we will have a longer open discussion period. So for the moment, let's do clarifying questions from webinar attendees and from Panel members. And Panel members can just raise their hands. I see Ulrike and Jenny. And so we'll start. Go ahead, Ulrike.

PANEL MEMBER LUDERER: Yeah. Thank you. That

was a really very interesting talk. I have one question or kind of -- it's kind of two questions. The first part is I may have missed this, but so when you modeled menstruating women, did you assume that after a certain age, like 50, that was no longer a source of loss?

DR. MACLEOD: Yes. Exactly.

PANEL MEMBER LUDERER: Okay.

DR. MACLEOD: There is a -- there is a dynamic function in the model where the -- there is no menstrual blood loss before the age of 15 and then it stops after the age of 50, I believe.

PANEL MEMBER LUDERER: Okay. And then the second question is what about loss via lactation in women as another possible source?

DR. MACLEOD: Yeah, these -- this is very -- these are great questions, because there is also lactation and there is also child birth and blood loss associated with child birth and just birth -- and just the child itself. And Kathleen has actually opened my eyes and pointed me to a few studies where there are statistical correlations at least where women with higher parity have lower PFAS concentrations.

And it is all -- it is significant enough that I think we should be able to see it in the population-based pharmacokinetic model. We have not -- so far, I have not

included that in any of our model scenarios. It's comparable. The lactation and depuration due to breast feeding is probably -- based on my best guess on this at this point, the lactation and depuration due to breast feeding is probably smaller than the blood loss associated with childbirth and the -- and the birth of the kid itself.

But I think that this is something that I want to investigate a bit more. I think there is a chance that we could even further -- now, we're talking about explaining variability within the cohort of women in each age. And what we might need is instead of just one representative individual born each year, for women, we might need three or four who have different parity over their lifetime to get a -- to see if we can explain that range. And I think that would be the first step toward answering this question, at least in our model framework. I think there's other independent studies that say that this is an important depuration process for individuals. And then the question is how important is that at the population level that we would be interested in getting at.

CHAIRPERSON SCHWARZMAN: It sounds like a complex balancing, because, of course, there's amenorrhea during pregnancy and amenorrhea during breast feeding. And so, you know, it's a complex --

DR. MACLEOD: Exactly.

CHAIRPERSON SCHWARZMAN: -- give and take and --

DR. MACLEOD: Yeah, yeah, yeah.

CHAIRPERSON SCHWARZMAN: -- some women experience significant blood loss during delivery and some don't.

DR. MACLEOD: Not. This is part of the reason that I've --

CHAIRPERSON SCHWARZMAN: So I respect the complexity of modeling that process.

DR. MACLEOD: This is part of the reason that I've not tried to take it on quite yet, because the model is useful for looking at things at a population level. And I've been sort of hoping that at the population level all of this would -- you know, but what we do see is this difference between men and women. This is the first thing we were interested in. Trying to explain the difference between women is another level of complexity lower, which is -- yeah, which we haven't tried to tackle yet.

CHAIRPERSON SCHWARZMAN: Jenny had a question.

PANEL MEMBER QUINTANA: Hi. One of my questions was the same as Ulrike, which was regarding lactation, which you already answered, but I also was thinking at a population level that women, at least in San Diego County where I live, are tending to have children at an older age. And it's not only is lactation an issue, another

complexity would be age at lactation, which is changing at a population level, I think. And so I was just thinking through those complexities, but -- so that was one question I had.

Another question I had was what was the effect of increased body mass or obesity on population changes on the volume of distribution as well and can you comment on that?

DR. MACLEOD: Yeah. This is a super interesting question actually. We have parameterized the model with age, body weight, data from the exposure factors handbook. And this is fairly -- you know, it represents a snapshot in time from whenever the -- now, I'm not a hundred percent sure which version of the exposure factors handbook we used. But certainly, there are changes in obesity which -- or obesity rates, which are -- where were -- which are affecting this.

I mentioned it at the end -- actually, if you look back in the slides, you see for the very oldest age group of men, especially you see a rise in concentration at the end. And this is because men in their 80s and 90s tend to shrink quite a bit in this exposure factors handbook. So there, you see an increase in concentration.

Across the whole population, I think it's a more difficult question to say what this whole shift towards

higher obesity is causing. I'm not sure that it's causing a change in volume of distribution, but it does have implications I think for -- at an individual level. At a population level, I think it's harder to say. I wonder -- I don't -- I don't have a great answer, I guess. It's interesting, but it's not something that we've looked at yet in model scenarios.

PANEL MEMBER QUINTANA: Thank you.

CHAIRPERSON SCHWARZMAN: We have a question from Jianwen.

Go ahead.

DR. SHE: Thank you very much Matthew and then this modeling work, as everyone noted -- noticed, the very complicated work is very useful.

So my question is to this first older chemical reaction modeling of the pharmacokinetic modeling. You know, I believe the major purpose is to interpret the data what we already found in the laboratory bimonitoring data we collect. Is that a predictor of future levels? And so my basic question is how to use it? When is a good time to use modeling? When is a good time to use real laboratory monitoring? How do these two factions, two measurements help each other?

DR. MACLEOD: Great question. I think we need both actually. A really interesting thing in the modeling

is you might have seen all of my exposure curves just had two phases and -- well three phases, an increasing exposure phase, a plateau, and then a phase of exponentially declining exposure. At some point, that exponentially declining exposure is going to stop and we're going to reach a point where even though we've phased out all of the obvious sources that were causing contaminations of the food supply or drinking water, and we're going to reach, especially for these very persistent substances like PFOS, and PFOA, perfluorohexanesulfonate, we're going to reach some plateau of exposure that we won't go below. And the modeling cannot predict where that plateau is. You need to continue to do biomonitoring to find where -- what -- you know, where that -- where there is no longer possible to reduce exposures just from the actions that we've taken to restrict or ban PFOS and perfluorohexanesulfonate.

So the model can tell you what this will look like. It will look like a flattening again in the -- in the -- in the decline rate, but we don't know when it will happen. We have to continue to monitor to find out when and at what level.

DR. SHE: Thank you.

CHAIRPERSON SCHWARZMAN: Any other Panel clarifying questions?

I have one question in the Q&A.

DR. MACLEOD: I see it. Should I -- can everyone see it --

CHAIRPERSON SCHWARZMAN: Yes.

DR. MACLEOD: -- and read it or should I read it?

CHAIRPERSON SCHWARZMAN: I think so. I think everybody should be able to read it. Go ahead.

DR. MACLEOD: Okay. Well, I could just summarize. It's a question.

MS. JARMUL: Meg, I'm actually -- I'm not sure that the attendees can see it --

CHAIRPERSON SCHWARZMAN: Oh, okay.

MS. JARMUL: -- so maybe just read it out loud first.

DR. MACLEOD: Okay. I'll just summarize.

CHAIRPERSON SCHWARZMAN: That's a good point. And also for the transcription. That's a great point.

MS. JARMUL: Yes, thank you.

DR. MACLEOD: Yeah. The question is asking about whether there is an opportunity to compare the model, especially for this hypothetical group of non-menstruating women to a population of women who really don't menstruate, because of contraceptive use, or there are women with very low body fat, for example, who don't menstruate. I think this is a -- this is a -- this would

be useful if we were really interested in that subpopulation of women.

I think the other way to frame this question is how well does the model work for men who lose -- who have blood loss -- regular blood loss? And there are men like this who are these hemochromatosis patients for example. And there, you do see that they have body burdens of PFAS that are lower than the general population. And there's even been some studies with highly exposed populations of firefighters. There's a nice paper that came out a year ago that looked at a population of firefighters from Australia who gave blood regularly and reduced their body burdens of perfluorinated -- perfluoroalkyl substances at a rate that was much faster than the general population by giving blood regularly.

So I think like from a validation point of -- like depending on how you want to interpret this question, if you're very interested in this particular subpopulation, of course, you could do modeling and do measurements of these non-menstruating women. But from a model validation point of view, it's equally interesting to look at men who have regular blood loss for other reasons and see that they do actually have lower body burdens or enhanced elimination of the perfluoroalkyl substances.

CHAIRPERSON SCHWARZMAN: Okay. I think we can move on to the section where we just have an open discussion period. And a reminder that both Panelists and the audience members can ask questions or provide comments. And webinar attendees can do that through the Q&A or through the Biomonitoring California email. And we'll do this until we have a break at 11:20.

Comments or reflections?

Yes, please. Let's see, Jenny.

PANEL MEMBER QUINTANA: Hi. Thank you again for the talk. I'm just thinking about what you mentioned about the importance of biomonitoring and modeling, and how they can intersect or inform each other. And I was also thinking about, if I could hear your thoughts about if we had deviations from your model, are there times you think this could indicate a previously unknown exposure pathway, for example, or could it inform hypotheses we should be investigating and -- or something like that? I'm just curious about what it could tell us.

DR. MACLEOD: I think so. And I wonder, Kathleen, if you want to weigh in a little bit also, because Kathleen and I have discussed a little bit about, especially in the California population, about whether we should look, for example, at Asian subpopulations or subpopulations with a high number of immigrants who might

have had a different exposure history than the general U.S. population.

You know, there's reason to believe that exposures in China are much higher than they would be in the United States in the last decade or two. And so recent immigrants who've come from China, for example, could -- an interesting hypothesis to test would be to look at that subpopulation and see if there's evidence of higher exposures. I think this was one of your hypotheses, Kathleen, that you thought about, but I wonder if you want to comment on a couple others.

DR. ATTFIELD: Oh, sure. I was just going to confirm what you're saying that we've seen that with our Asian Pacific Islander community exposure studies, which is actually a nice little seed, because I'm going to bring it up later in the later talk.

So we've seen higher levels in PFAS in the Chinese Americans and Vietnamese Americans that were part of that study. But we've also seen it in both CARE-LA and CARE-2 in California. So it's interesting, but we'll have to figure out how to work it into what you're -- what you're modeling, Matt.

DR. MACLEOD: Yeah. I mean, I think I --

DR. ATTFIELD: I'm sorry. Just to add a little bit more to that. We were seeing difference in time spent

in the -- in the country and for those born outside the U.S. versus inside the U.S. So there is this concern about differing body burdens that people bring to, you know, a state that has such a high immigration population -- immigrant population.

DR. MACLEOD: I think for some of these -- for many of these kind of questions, the more powerful modeling tools are just going to be the purely statistical modeling of -- that an epidemiologist like Kathleen would apply, because with this mechanistic modeling, you need quite high -- you know when we're looking at the whole -- when we're looking at population averages, we need quite big populations to iron out the interindividual variability. And actually the statistical analysis that epidemiologists do will get at those kind of questions -- we'll get answers to those kind of questions at P less than 0.05 quite a bit quicker -- or on much smaller sample sizes than we will with our mechanistic model. So I think there's a role there for different types of modeling for investigating different types of hypotheses.

CHAIRPERSON SCHWARZMAN: Matt, do you want to look at the two points that are in the Q&A and restate them and respond to those?

DR. MACLEOD: Okay.

MS. JARMUL: Or I can go ahead and just say them

out loud just so it's easier for the transcriber.

DR. MACLEOD: Yeah. Thanks, Stephanie.

MS. JARMUL: So we have one question. Wouldn't looking at women that don't menstruate versus women who do help to prove this point better than looking at men who bleed?

DR. MACLEOD: Oh, I don't know about better, but in addition. The problem with this is I don't have access to any data from non-menstruating women, where I do have access to data from men who give blood regularly. So I think this is correct that this would be another line of evidence. I don't know that it's better. But the practical barrier is that I don't have access to those data, so maybe it's a back end of my question.

Yeah, go ahead.

CHAIRPERSON SCHWARZMAN: Oh, a quick clarifying point about that. When you say you don't have access, is it that like within NHANES, you don't know who is menstruating and who isn't, so you can't separate the populations?

DR. MACLEOD: Exactly. Yeah, exactly. I mean, beyond over 50 and under 15, but there we just assume. But within the population of women of child-bearing age, I don't -- I don't have access to the information, yeah.

CHAIRPERSON SCHWARZMAN: And do you also not

have --

DR. MACLEOD: And for the men, it's a case of -- it's not population studies. It's, you know, campaigns where they're looking specifically at those groups. So it's not actually population biomonitoring, but exposed groups.

CHAIRPERSON SCHWARZMAN: Understand. And in general, NHANES does also not include information on parity?

DR. MACLEOD: Now, maybe there's better -- there's people who are more expert on NHANES than I am, but I don't believe it does. I don't believe that you can link parity to it. But maybe, Kathleen, do you know if that's correct, if it's possible in NHANES to link parity to the individual measurements?

DR. ATTFIELD: I actually don't have that information on hand, but I would believe they would collect it.

DR. MACLEOD: And is a -- and it can be associated with the individual measurements. Maybe there's someone else who knows more about NHANES than I do.

DR. ATTFIELD: Well, I would say we definitely have that information for CARE. So that is something that we could add to the CARE component.

CHAIRPERSON SCHWARZMAN: Well, and I'm thinking about some of the sort of subanalyses that have been done on NHANES data looking at chemicals that occur in pregnant women. So there must be NHANES data that identifies -- that connects individual data to pregnancy status at the time, at least.

DR. MACLEOD: Yeah.

CHAIRPERSON SCHWARZMAN: So I think that would be a really interesting point to follow up on.

DR. MACLEOD: That would be a good extension then as a way of -- yeah. Yeah.

CHAIRPERSON SCHWARZMAN: Stephanie, do you want to do the next Q&A.

MS. JARMUL: Yep. And this was just a comment from Dr. Ahimsa Porter Sumchai, which says that, "Research conducted on elite athletes exposed to air pollution and heavy metals found exercising muscle aids in excretion."

DR. MACLEOD: That's interesting. I mean, on the one hand because of what we were talking about about the elite athletes who maybe don't menstruate but this corresponds with my own experience where if I drink too much coffee and then I go out for a run, I feel much less hyperactive from caffeine overdose. So I believe that from a personal point of view as well. I'm not an elite athlete though, I would say.

CHAIRPERSON SCHWARZMAN: Maybe, Stephanie, I could use this pause just to ask if there's comments via the email that we should check in with.

MS. JARMUL: No, nothing from the email as of yet.

CHAIRPERSON SCHWARZMAN: So other questions, or comments, or discussion points from what we've seen?

One thing I'm curious about is just thinking about this -- the metabolic pathways question for different substances. This is sort of extending it to other categories of pollutants and synthetic chemicals that we find in people. Like the example that was just given in that point is for metals. And I -- I don't know detailed information about how metals are eliminated, but certainly, you know, they're not carried in blood the way PFAS are. And I just wonder if you have reflections, from your experience with working on these models and varying them for different contaminants, what some of those different elements are? There's, you know, whether a substance is lipophilic, whether it's, you know, excreted through kidney, or metabolized in the liver, or does it go to bone the way metals tend to, does it go to blood like PFAS, et cetera, and just if you have any kind of reflections on that?

DR. MACLEOD: I think this is a great question,

because this is where I think the mechanistic modeling is most powerful. Like, we talked earlier in -- about some of these questions about different exposures in subpopulations, where it's probably just straight statistical modeling that's going to be the most efficient way to answer the question about whether Asian and Pacific Islanders, for example, have higher exposures than the general population.

But these kind of questions about different elimination pathways that might be relevant for different types of chemicals, you can only answer with a mechanistic model. And that's where this like menstrual blood loss is a pathway for loss of PFAS is a nice example, because this is different than the PCBs, which are more traditional lipophilic compounds that have very high volumes of distribution, so blood loss is immaterial to your rate of elimination of PCBs. And instead, what's important is -- or what's -- back when we did that work on PCBs in the 2010s actually, we were very interested to see whether we would see a dose-dependent elimination rate constant for PCBs, because it's, you know, quite well documented that for PCBs and dioxins if you get a very high dose, you can have chloracne and -- you know, where the body is -- you activate detoxification mechanisms within the body that I assume are evolutionarily designed to help you to

eliminate these kind of lipophilic toxins.

We didn't see any evidence of dose-dependent elimination in the general population in that -- in those studies for PCBs, which kind of makes sense, because this was general population. It wasn't anybody who was having these activations. But those kind of questions you can only get at with a mechanistic model. So now I'm out of my depth a little bit, because I've never looked at metals myself, but if you have hypotheses about, you know, different elimination pathways for metals that you could build into the model, you can test those hypotheses with the model to see whether you improve model fits for subpopulations where these are important. So I think that this is a good use of modeling and a good use of this mechanistic modeling.

The other thing that came in my mind when you started to ask your question was for the PFAS specifically, there are actually other possible explanations for the differences between men and women. And they could be accounting for -- if you look back in the slides, even in the newest -- with the newest NHANES data in the new model fits for PFOS and PFOA, especially the intrinsic elimination half-lives for women are still a bit faster than they are for men. And we haven't done our complete uncertainty and error propagation analysis on

this yet. I don't think that they'll be statistically significantly faster for women, given our uncertainties in other parts, but there could be something still in there.

And in animal studies, like in rats, rats -- female rats don't menstruate is something I learned when I started to do this work, but yet you still see faster elimination of PFOS and PFOA by female rats than males. And this is attributed to differences in hormone balance that determine differences in efficiency of reabsorption of PFOS and PFOA in the kidneys, which is very interesting.

And I think it's possible that there is some version of that type of mechanism also operating in humans. It's against a background of this bigger difference between males and females associated with persistent blood loss, but it -- but it could still be there and is still something that could be teased out, I think, in this modeling. There's still room in our modeling for that kind of mechanism to be active in humans, I guess, is what I want to say.

CHAIRPERSON SCHWARZMAN: Thank you for that. It's interesting to think about how to apply mechanistic models to other classes of substances.

We have about 10 more minutes if others have discussion points or questions, comments.

Stephanie, I'll do one last check about email questions or comments and then is your preference to break -- take our break 10 minutes early or take a longer break to stay on our published schedule?

MS. JARMUL: I think we would just take a longer break so we can stay on our published schedule.

CHAIRPERSON SCHWARZMAN: Okay.

MR. JARMUL: Yeah, we'll see if there's -- there's nothing in the email right now, but if anyone has a question or comment in the next minute or two, we can.

CHAIRPERSON SCHWARZMAN: Yeah, we have one that just came in.

MS. JARMUL: Oh, great.

DR. MACLEOD: This is a helpful -- a helpful comment about the availability of parity information only for a subset of women.

MS. JARMUL: And I'll just read it out loud. It's a comment from Gina that says, "Comment on pregnancy in NHANES. Parity is available for a subset of women who completed the reproductive health questionnaire." And then also, "Pregnancy status at time of exam is suppressed for women under 20 and over 44 years old."

CHAIRPERSON SCHWARZMAN: Jenny.

PANEL MEMBER QUINTANA: Hi. Just was thinking maybe to recommend a more inclusive language in talking

about subjects. There are people that identify as men, people that identify as men who menstruate. And so I just was thinking perhaps going forward to frame it perhaps a little bit differently. Thank you.

CHAIRPERSON SCHWARZMAN: Do you have any reflection on that, Matt, given that it's another point of complexity, because there what you're referencing is physiology and some of that is connected to gender assigned at birth, unless there's, you know, gender affirming care in process that changes hormonal functions and associated physiologic functions. I mean, hormonal levels and associated physiologic functions.

So I appreciate Jenny that it raises kind of points of like being clear about that. Maybe that -- in addition to inclusivity of the language, there's also sort of specificity of the designations in a way they point to physiologic processes. So gender assigned at birth is more specific maybe.

DR. MACLEOD: Yeah, I don't know. I don't have any specific thoughts about how to do this. I'm open to suggestions on how to do better in describing this certainly. So I'm open for suggestions. I would say mostly we are talking at the population level here, so -- but I'm open for opinions or suggestions on how -- on better terminology certainly and how to be more precise

about this. And, Kathleen, do you have an idea maybe?

CHAIRPERSON SCHWARZMAN: Kathleen.

DR. ATTFIELD: I was going to more address Meg's second point just to say what information we have available that's pertinent to this for the CARE studies. So for CARE-LA, we only asked about gender, but for CARE-2 and CARE-3 we asked both about gender and sex assigned at birth. So for CARE-2, there was actually a hundred percent correlation between the two, so we would not be able to look at any distinction between the two types of identification. And CARE-3, of course, was a very small number of participants with -- what with the beginning of the pandemic.

CHAIRPERSON SCHWARZMAN: Jenny.

PANEL MEMBER QUINTANA: To add briefly that I was not -- I know what data you have is kind of how you're characterizing your analysis. I'm just saying when I hear you talk about is women who menstruate and men who don't, given our discussions in our classes with our students at the School of Public Health, it just seems a little jarring to me and I think it would be to them.

That's all I meant, not that you have necessarily control over what data you're analyzing, so just to put it in context.

DR. MACLEOD: Yeah, I wouldn't want to -- wooh,

okay, I have to think about how to say this in a more precise way, because definitely there are men who menstruate.

CHAIRPERSON SCHWARZMAN: Nerissa.

DR. WU: Just to add to this conversation. Thank you, Jenny, for your comment. I think just there's a whole world of realities of health and identity. And I think describing women as imaginary who don't menstruate, there is an entire world of women who don't menstruate for varying reasons or -- but you know who are at different phases of their lives and you acknowledge some of that through the framing of 15 through 50 and post-menopause. But I think just -- it just feels a little dismissive to consider them imaginary, and so just be cautious in your language. And this is kind of going afield from the study design, but just because we're very careful to acknowledge that different people exist and have health consequences and we want to just be precise in our language about how we talk about them.

DR. MACLEOD: I have had another comment on this related to this actually, because -- and even in our paper we have this thing which is called the intrinsic elimination rate constant. And we have defined this intrinsic elimination rate constant to be elimination due to elimination processes that are common between men and

women. And I have had women tell me that this is poor framing, because menstruation is intrinsic to being a woman. This was some years ago maybe before there was more of this discussion about -- so I don't know. I find this -- it gets a little difficult to do it in a way where you make everybody feel included all the time simultaneously, I would say. But as I said, I'm open for suggestions on how to do better.

CHAIRPERSON SCHWARZMAN: Any final questions, or comments, or additions to the discussion before we take a break?

Seeing none, I want to thank you, Matt, for your time and what is your evening and for bringing us your study results and explaining it and how you've applied it to the CARE data. It gives us a lot to think about.

So we will have a break now until 11:30. Just a reminder to return promptly so that we can -- because we'll start right at 11:30. And with that, we'll start our break. Thanks.

DR. MACLEOD: Thank you, everybody.

(Off record: 11:18 a.m.)

(Thereupon a recess was taken.)

(On record: 11:30 a.m.)

CHAIRPERSON SCHWARZMAN: I have that it's 11:30 so I want to call the meeting back together. We will have

two presentations now that are Program updates, but we'll -- they're separate presentations and we will have separate question and answer after each and then followed by a larger open discussion -- a longer open discussion period. So I want to start by introducing Kathleen Attfield. Kathleen is Chief of the Exposure, Surveillance, and Epidemiology Unit, which is part of the Exposure Assessment Section in the Environmental Health Investigations Branch, EHIB, at the California Department of Public Health, CDPH.

She will give an update on current Program activities and planning for future studies. And after Kathleen's presentation, we'll have five minutes for clarifying questions and then we'll have a presentation from Susan Hurley of OEHHA. And then we'll have the larger discussion on Program activities after both presentations.

So turning it over to you, Kathleen.

(Thereupon a slide presentation).

DR. ATTFIELD: Wonderful. Thank you, Meg. And let me just confirm that you can see my slides, yes?

MS. JARMUL: Yes.

DR. ATTFIELD: Thank you.

CHAIRPERSON SCHWARZMAN: Yes. Sorry. That took me a moment to find them, but I've got them.

DR. ATTFIELD: Okay. So good morning. Thank you, everyone, for attending today.

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DR. ATTFIELD: For today's Program update, I will talk through some administrative updates as well as project updates for STEPS, a project and collaboration with the Water Board, a renewal of work with the Asian Pacific Islander Community Exposures Project that was mentioned earlier, and some updates from our communications team and from our laboratories.

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DR. ATTFIELD: We'd like to welcome new staff to the Environmental Chemistry Laboratory at DTSC, Julian Edmonds, Ilaria Lentrichia, and Bisha Neupane, and also acknowledge the contributions of Lily Wu, who is currently serving as Acting Chief of the Safer Alternatives Assessment and Biomonitoring Section at OEHHA.

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DR. ATTFIELD: Last time we met, we described our developing surveillance project. And so this time, we're going to offer just a short update and we'll spend more time talking through additional projects that are underway. To update you on the progress with the STEPS study or the Studying Trends in Exposures in Prenatal Samples. We are in the process of requesting chosen

samples from the Biobank at the Genetic Disease Screening Program for the years of 2015, 2018, and 2021. We are also working with staff from the Genetic Disease Screening Program on planning our prospective sampling in a non-Biobank county.

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DR. ATTFIELD: In our work on our California Regional Exposures Study, or the CARE Study, we've been collaborating with the California Water Boards to understand data coverage and overlaps between our serum PFAS data and their drinking water PFAS testing data. We have identified initial goals of identifying data gaps that the Water Board -- where the Water Board could take action with investigative orders to cover these gaps. So an example of this would be if there are public water systems where CARE participants had high blood levels, but there is no existing drinking water testing data for PFAS.

We're also looking at the feasibility of different investigative questions with the different data sets, so looking at the relationships between drinking water and biomarker data to see if we can predict values of biomarker concentrations, as well as whether it may be possible to estimate the relative source contribution of drinking water to PFAS exposure to lend a hand in risk assessments at the State.

So for this effort, we are using CARE data from all three iterations, the 2018, 2019, and 2020 CAREs from eastern and south -- Southern California.

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DR. ATTFIELD: And for drinking water data, there are currently two main sources of PFAS drinking water data that we are employing. So those from the EPA's Unregulated Contaminant Monitoring Rule of UCMR 3, where during -- which took place during 2013-2015, and from investigative orders issued by the California Water Board in 2019 to 2021. The UCMR 3 data covers mainly public water systems serving over 10,000 people and samples from points of entry to the distribution system. So the data from the investigative orders is a little different, in that it mostly covers source wells with some finished water and focuses mainly on areas near prior detections of PFAS or possible contamination sources, such as landfills and airports. And there is data subsequent to this in 2022 and into 2023, so there will be continuing to be a rich source of data that can be used.

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DR. ATTFIELD: Our first steps in looking at the data was to geocode our CARE participants, the ones that have serum PFAS levels and we've been matching them to water system boundaries and are actually achieving a

pretty good coverage. So 848 participants matched to a system out of the 872 that were geocoded.

While a greater number of participants lived in water systems that had testing in UCMR 3, so 96 percent, Water Board -- then Water Board testing - sorry - a greater number are seeing detects of PFAS in the Water Board data, so 53 percent as compared to 8 percent. And, you know, there are a good number of reasons for the difference in detection frequency. As you can see, the method detection limit is rather different between the two phases and, of course, there were different sampling points that were used between the two.

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DR. ATTFIELD: When we look at the data by water system, our participants match to 150 water systems with an average of 7 or 10 participants per water system with a maximum of 184 and that's in the Los Angeles area.

More systems were tested in UCMR 3, 79 percent versus 50 percent, but a greater percentage had detects in the Water Board testing. There's 64 percent.

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DR. ATTFIELD: So we just want to present some initial looks geographically. You can see the geographic extent of our participants. They are jittered. So this is not their exact home location, but approximate. The

dots in green are below the top 10 percent, so the bottom 90 percent, and in blue are the top 10 percent. And so for this, our initial aim with the Water Board was to look at folks with the higher level of PFAS, and PFOA, and other PFAS detections. So here we're showing the top 10 percent and in the gray are the polygons of the water system boundaries.

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DR. ATTFIELD: And a zoomed in version here of Southern California. And now the systems have been colored with their different quantiles of PFOS concentrations. So a visual correlation is not immediately evident here, but what we've learned about the overlap of these data so far is that --

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DR. ATTFIELD: -- we have identified four participants with serum PFOA or PFOS levels that are in the top 10 percent of participants, but who have no water testing data so far, and 11 people who are in the top 25 percent. So we've shared this data with the Water Board. They are active participants in this project, of course, and they're planning to use this information in their next phase of testing requirements.

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DR. ATTFIELD: Some challenges we're working with

include assigning participants to a single water system. So the Water Board is in the process of validating some of the water system boundaries. So temporarily there are situations where water boundaries may overlap. But in the process of this project, we've been able to reduce the number of participants with an overlapping situation from 274 to 91.

We are also contemplating how to create different summary statistics for the end drinking water user, since systems have many different sampling points and have been collecting the data for regulatory purposes often to evaluate the raw sources, so not the finished water.

We know there may be other uses for the overlaps between these two data sets, so that is a question that we do have for the Panel. So we'd be interested in some commentary on further uses of the overlap of this data.

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DR. ATTFIELD: So moving on to another project in progress. Due to our increased staffing, we're able to revisit the data analyses within the Asian Pacific Islander Community Exposures Project. This was an extension of collaborations with community groups on health education and outreach related to safer fish consumption. That led to a community-based study to biomonitor Asian populations for metals and PFASs, which,

as I had mentioned, had been observed in higher levels in Asians within a prior Biomonitoring California study.

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DR. ATTFIELD: There were two phases for ACE. First in 2016, where we worked with APA Family Support Services to recruit 100 Chinese Americans, and then -- in the San Francisco area. And then in 2017, we worked with the Vietnamese Voluntary Association to recruit 100 Vietnamese Americans in the San Jose area.

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DR. ATTFIELD: In ACE, we found a fair number of participants with levels of metals above our levels of concern that the Program has for following up with participants with elevated levels to help them consider different ways of reducing the potential exposures. So this slide is here as a reference of which -- what levels we do use for our levels of concern cutoff for arsenic and for mercury.

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DR. ATTFIELD: And within ACE, we had seen these levels that are a fair amount higher than -- or more frequently occurring in the ACE population as versus CARE. So CARE-LA, as an example here, two to six percent of our participants had levels above the LOCs, while in ACE 26 participants had elevated inorganic arsenic in both phases

of ACE, and up to 16 percent of women of reproductive age there in this first line with elevated blood mercury.

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DR. ATTFIELD: As mentioned earlier, we also observed higher levels of PFAS, so five PFAS in comparison to national data, the NHANES iteration of 2016-2017. And we even saw higher levels than Asians within that same cohort of NHANES for PFOS and PFNA. As with metals, we had seen often acculturation factors were associated with higher levels. So, for example, birth country, time spent in the U.S. and interview language. And so this will be instructive for outreach and educational programming with our partners as well as our further investigations into the data.

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DR. ATTFIELD: Our recent efforts on this project include reconnecting with existing stakeholders and exploring how the initial findings from the project are consistent with the group's current concerns. We're also following up on educational efforts and exploring the utility of particular additional analyses.

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DR. ATTFIELD: So the additional analyses that we're circling around at the moment are looking into PFAS concentrations and fish consumption, because we have a

fair number of fish questions within our questionnaire so we can address different types of fish and different parts of the fish; for metals, and herbal remedies, and personal care products; and in a collaboration with Silent Spring Institute, they are looking into occupational exposures within the ACE cohort and the differentiation between those with recent immigration history versus not.

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DR. ATTFIELD: We are interested in learning from the Panel your suggestions for other questionnaire analyte investigations that could be informative for educational and outreach efforts at the community level, as well as for enriching the general field related to PFAS in metals. We're also interested in hearing about other outreach panel -- excuse me, outreach partners the Panel may have suggestions for. So I can return to this slide later.

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DR. ATTFIELD: Next, were updates from our Outreach and Communications Team. They have been hard at work finalizing the beautiful version of our CARE report that you've heard us mention before. So a couple teasers of images that are part of the CARE report.

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DR. ATTFIELD: And there's going to be an accompanying dashboard two-page summary.

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DR. ATTFIELD: Additionally, they are focusing on visual fact sheets and other accessible and engaging materials for the general public as it relates to information around arsenic and rice, and a brief -- briefer about our Foam Replacement Environmental Exposure Study paper that is underway.

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DR. ATTFIELD: So next, updates from the Environmental Health Lab. They are initiating additional environmental phenols analysis for the CARE Study. So if you may remember that we had done phenols analysis on a subset for CARE-LA and CARE-2. So 370 for CARE-LA are remaining and 190 are remaining for CARE-2. So this will be fantastic to have it for the entire cohort of both studies.

Oh, there's a typo there. The second bullet is meant to be talking about bisphenol A metabolite method. That is in progress where the metabolites that will be able to be detected are sought for glucuronide and sulfate conjugates.

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DR. ATTFIELD: They are also validating the speciated urinary mercury method looking into inorganic and monomethyl mercury. They're developing the total

nickel analysis by ICP-MS for use in pollution community studies -- air pollution community studies, excuse me, and continued work on the VOC urinary metabolite method.

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DR. ATTFIELD: As for our Environmental Chemistry Lab, they are finishing their instrument analysis of serum and plasma comparison for their extended PFAS method with the final data analysis in progress, and have updated the persistent organic pollutants method for PCBs, OC -- organochlorine pesticides, and PBDEs, where they've reduced the sample preparation time from 48 hours to 7 hours by the use of an upgraded automated SPE system. And finally, they have new methods under development for siloxane and PAHs in serum.

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DR. ATTFIELD: So with that, that finishes our update portion of the presentation and I'll pass it back to you, Meg.

CHAIRPERSON SCHWARZMAN: Thank you, Kathleen.

We have five minutes now for questions -- clarifying questions before we move to our next update.

So a reminder that Panelists just raise your hand -- turn on your camera, raise your hand and I'll call on you if you have a question. And webinar attendees, you can use the Q&A function, or email, or raise hand

function.

Thank you.

Lara, yes, please.

PANEL MEMBER CUSHING: Hi. Sorry that I -- I'll have to jump off after this and leave early, but it's really great to see this exciting work on PFAS and the ACE Project. I had one quick question about the PFAS. I know the Water Board has been doing -- has, through a different analytical method, some evidence that there are perhaps many PFAS that are not in the typical panel that are analyzed for.

DR. ATTFIELD: Um-hmm.

PANEL MEMBER CUSHING: UCMR 3. So I don't know if you're planning -- I was curious if -- what PFAS specifically are -- were tested for in CARE and if there's any opportunity to kind of look at not just the usual suspects but some of the more obscure, less common, or more recently put into production PFOS with that project.

DR. ATTFIELD: That's an interesting question and really of the moment. For CARE, both iterate -- well, all three iterations used a method that just looks at the 12 legacy compounds -- sorry. I'm making sure the cat doesn't enter the screen. And for ACE, that was an extended method, which used a sort of manual preparation method in the laboratory, so that actually has a larger

number of PFAS. I don't remember the number off the top of my head, but about 30 or so.

For our study with STEPS we're going to use that extended PFAS method, which I mentioned, which will have about 40 PFAS for it. It's an interesting question of whether we would have capacity to go back and look further at our samples for CARE. We do sometimes have volume restrictions and restrictions around what people have given permission for for additional analyses that we'd have to consider, but that's an interesting question that we -- that we will consider.

It looks like Nerissa has a comment as well.

DR. WU: Yeah. Thanks for your question, Lara.

One of the issues with going back to participants even if they have given permission for additional analyses is that we are obligated to return results to people if we measure them. So if we are doing a method where we're sort of exploring what new PFASs may be showing up in people biologically, we have to think about what the messaging would be like. But as Kathleen said, the STEPS samples are a very good match for this, if we have enough volume, because there is not a results return component, but also because we are getting a real time trend with that sampling. So we might be able to see the emergence of newer PFASs coming in and hopefully match that with

some of the new Water Board data.

DR. ATTFIELD: Thank you, Nerissa.

CHAIRPERSON SCHWARZMAN: Great. Yes, Jenny.

PANEL MEMBER QUINTANA: Hi. I wanted to thank you for that update. I think it's really exciting to see California Biomonitoring interface with other State agencies to really extend the reach of what we're doing, so California Air Resources Board, and now the Water Board. And I just think that's a really great approach to take.

And I'm also interested in your community kind of translation of your materials. And I'm always interested what people can actually do. You know, so for example for the rice, you're talking about giving some outreach about arsenic in rice. I mean, is there -- I haven't really looked at my rice packages. Do they tell you where it's grown, for example. Like, don't buy it from the south where they used to use arsenic, or, you know, how much information people easily get to reduce -- to buy rice that's cleaner as opposed to reducing rice consumption. I'm just kind of curious again from a naive point of view, like how do you translate these findings into, you know, what people can use? So thank you for that.

DR. ATTFIELD: Thank you. Yes, it's not been an easy project. And I'll pass that to Nerissa or Emilie to

speak more about it.

DR. WU: Sure. You've identified some of the key issues we're wrestling with with the Biomonitoring Outreach and Communications Group. And rice is complicated because it's really -- I mean, obviously the uptake is going to depend on things like soil conditions, is it flooded or not, is there arsenic in the soil? So there's so much variability that it becomes a very difficult message to convey to folks in a simple communication.

So we do have some sort of broad indicators that California rice tends to be lower than some other areas, but we want to be careful not to -- you know, just to be careful in our language that it's not a guarantee that eating California rice is safe. But like I said, the science behind it is quite complicated and we are sort of picking our way through the messages and identifying what we can say and backup. We want to be really careful not to recommend things that might lower the nutrient value of rice, like washing it until supplemental nutrients come out or recommending that you eat a different food that then might have elevated levels of something else.

So it is -- it is quite a complicated process, but we have -- we've -- we're getting closer to what our messaging will look like and hope to have something to

share with this group, but also some of our community partners to what is an effective message soon.

PANEL MEMBER QUINTANA: I just wanted to say that how often California Biomonitoring has been a leader in these kind of efforts. And I really appreciate it, you know, just for the results return. You know, I use that -- I hand that out to people I know as an example of how to do it. And this is another example of leading on these kind of difficult issues and walking the tightrope. So I appreciate what you're doing. Thank you.

DR. WU: Thanks, Jenny.

CHAIRPERSON SCHWARZMAN: We have time for one more brief question, if anyone has for Kathleen.

Yes, Ulrike.

PANEL MEMBER LUDERER: Thank you, Kathleen, for the update on all the amazing things that Biomonitoring California is doing. I just had a quick question about you mentioned the new methods under development included siloxane in serum. I remember years ago when those were designated by the Panel. And I'm just wondering which -- are you -- you know, do you know yet which ones are going to be, you know, included, which siloxanes? Is that -- or is that still under discussion?

DR. ATTFIELD: This is one that I will pass to June-Soo for the particulars.

June-Soo, are you able to join us?

DR. PARK: Yeah. Yeah. No. I think I understand your concern about the siloxane. We feel very bad about this long due method of development. You know, the siloxane and/or some other, you know, the fragrance chemicals like musk. Unfortunately, you know, the -- we've been running the Biomonitoring California Program at least our side only by you -- with only two people, two staff. Recently, we were able to increase two more staff. So that's the kind of where we were. We are getting better.

Also, the -- we had -- only few month ago, we had a right instrument it's called GC-MS with a special sampling system or auto injector system that can minimize background contamination. So our designated Biomonitoring staff, Judy Wang, she is now devoted to work on this method. So I know it's still slow, but at least we're on it. So that's the only thing I can say for now.

DR. CRISPO SMITH: Hi. This is Sabrina --

DR. PARK: Oh, Sabrina. Yeah.

DR. CRISPO SMITH: -- from ECL. I was just going to quickly say, we're just looking at the cyclosiloxanes, so the D3, D4, D5, and D6. We may try to do some linear ones later, but we were having a bit of trouble Sourcing certified standards for those. Does that answer your

question?

PANEL MEMBER LUDERER: Yeah. Yeah. Great.

DR. CRISPO SMITH: Okay.

PANEL MEMBER LUDERER: Thank you. No, it's very exciting that you're moving forward on that. I think that's wonderful.

CHAIRPERSON SCHWARZMAN: Great. Okay. I'd like to move along and introduce Susan Hurley. Susan is a Research Scientist in the Safer Alternatives Assessment and Biomonitoring Section, SAABS, of OEHHA. And Susan will present an update on some of the Program's community biomonitoring studies and planning for future biomonitoring studies.

(Thereupon a slide presentation).

MS. HURLEY: Okay. Let me -- thank you, Meg. Let me just get my slides up. Can everybody see those okay?

CHAIRPERSON SCHWARZMAN: It's not yet on presenter view.

MS. HURLEY: Okay. How does that look?

CHAIRPERSON SCHWARZMAN: That's good.

MS. HURLEY: Okay. Thanks.

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MS. HURLEY: Okay. So today, I'm going to start with just a really brief update on our Bios -- BiomSPHERE

and the FRESSCA-Mujeres projects and then be spending most of my time talking about some of the initial biomonitoring results we got for our Stockton Air Pollution Exposure Project.

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MS. HURLEY: So for BiomSPHERE, recruitment and urine sample collection is currently under way and will continue through the end of the summer. And if you would like any more information about that project, we've got more information posted on Biomonitoring California's webpage. You can check out some of these links on the slide here.

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MS. HURLEY: And then for our FRESSCA project, we are just in the very initial stages of getting recruitment launched and are planning to be out in the field in May to start the urine collection. And that will continue through early fall. And again, there's more information at these links on the slides, so I'm not going to say anything more about those two projects today.

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MS. HURLEY: And I just want to move on to our Stockton Air Pollution Exposure Project, otherwise known as SAPEP.

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MS. HURLEY: And these are the two primary objectives of the project. So one is to learn more about air pollution exposures to schoolchildren in Stockton and to evaluate the effectiveness of school air filtration at reducing those exposures. And today, the initial results I'll be sharing are really focused on characterizing the air pollution exposures to the kids in our study. So it's really focused on this box here. I won't be talking at all about the evaluation of the effectiveness of the school air filtration, because we haven't completed those analyses yet.

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MS. HURLEY: So many of you have seen this slide before, but just to go over quickly what the design of the study was. It was conducted at one school in Stockton, the All Saints Academy, where we measured air pollutant levels both inside and outside of the school and then installed air filtration units or portable air cleaners in about half of the classrooms of participating students. Parents completed online questionnaires to get some more information about potential exposure sources. And then we collected children's urine before and after school. And then in those urine samples measured chemicals that could indicate exposures to air pollutants.

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MS. HURLEY: So our goal was to enroll 50 children. We actually ended up with 18 and that's primarily a reflection of trying to launch a study in the middle of a global pandemic. It left us with very little time for recruitment and limited access to the campus. But the samples were collected on two days of consecutive weeks in early December of 2021, where we -- for each child, we collected one sample before school and one sample after school on each of those two days. So about four samples per child. So ultimately, we ended up with 69 urine samples.

And then those samples were sent to the Clinical Pharmacology Lab at UCSF, where under the direction of Dr. Peyton Jacob, they were analyzed for hydroxy metabolites of these four PAHs as well as stable metabolites of VOCs for these six VOCs.

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MS. HURLEY: So last month, we sent to all the SAPEP participants their biomonitoring results for the VOCs, the PAHs, and the nicotine metabolites that were measured in their urine. And then later this year, we will send out the individual urine results for the markers of oxidative stress and inflammation that were also measured in the urine.

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MS. HURLEY: So this is just a quick picture of who was in our study. Most of the kids were male, about three-quarters were male. They ranged in age from five to 13 years old with most of them in the five to seven year old category, and most of the kids were Hispanic.

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MS. HURLEY: So for the initial analyses that I'll be showing the results for today, they're really just focused on comparing the metabolite levels in our study for the VOCs and PAHs to those in a nationally representative data from children in NHANES.

So we did this for -- so -- for all the samples, so regardless of time of day or the filtration status of their classroom. And we used random effects models to calculate the geometric means and 95 percent confidence intervals. And then we compared those metabolite levels to those found in the most recent data we could find for these analytes in kids. So for most of them, it was the NHANES 2015-16 cycle. For a couple, we had to go back to 2011 and '12.

And then to make our methods analogous to the methods CDC uses in reporting NHANES data, we -- for the non-detects, we imputed the value -- imputed values equal to the level of detection divided by the square root of two. We also did not calculate geometric means for any

analytes where the detection frequency was 65 percent or less.

And then just before launching into the results, I just want to call attention to some of the considerations to be thinking about as we're looking at these results. One is just a reminder - we have a small number of samples. It's, you know, among 18 kids. In some cases for some of the analytes, there were some differences in the levels of detection between our lab at UCSF and CDC's lab for NHANES, which should just be kept in mind in interpreting some of the -- some of the results.

And then just calling attention to the fact that our data was collected in 2021. We're use -- we're comparing to NHANES data that is mostly about five years prior, and in some cases 10 years prior, so it's not a perfect comparison.

My slide thing --

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MS. HURLEY: Oh, there we go. Sorry. Cursor was misbehaving. Okay. So these are -- for the VOCs for acrolein, acrylonitrile, crotonaldehyde, propylene oxide, you can see the metabolites were found in nearly all of the SAPEP participants as well as the NHANES participants.

For benzene and 1,3-butadiene, the metabolites

were found less frequently. Especially in SAPEP, we're seeing them found in less than half of the participants.

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MS. HURLEY: For the PAHs, most of the SAPEP and NHANES participants showed evidence of exposures to the PAHs -- the four PAHs that we looked at. You can see detection frequencies are pretty high in both cases, even though in some -- for some analytes the levels of detection are quite different. And I guess that's all I wanted to say there.

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MS. HURLEY: Okay. So these are the geometric -- this is the comparison of the VOC metabolites in SAPEP versus NHANES. And the blue bars represent the geometric means for NHANES. The white bar is for SAPEP. The little whiskers are the 95 percent confidence intervals. And you can see overall that the geometric means look quite similar across SAPEP and NHANES participants. And, in fact, none of these geometric means were statistically different. And note that we don't -- we're not trying benzene or 1,3-butadiene here, because their detection frequencies were less than 65 percent.

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MS. HURLEY: Okay. So for the PAHs, it's a little bit of a different story. Here again, the blue

bars are NHANES, white bars are SAPEP. And here we see the geometric means are generally lower in SAPEP for fluorene, for phenanthrene, and for pyrene. And in contrast for naphthalene, this metabolite is quite a bit higher in SAPEP compared to NHANES. The geometric mean here is about four times what is seen in the NHANES kids.

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MS. HURLEY: So just to briefly summarize. Nearly all SAPEP participants showed indications of exposure to acrolein, acrylonitrile, crotonaldehyde, and propylene oxide. Exposures to benzene and 1,3-butadiene were comparatively less common. And overall the metabolite levels did not differ in our study from those reported in NHANES.

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MS. HURLEY: For the PAHs, most SAPEP participants were exposed to fluorene, naphthalene, phenanthrene, and pyrene. And metabolite levels here were generally lower in SAPEP participants compared to NHANES, with the exception of naphthalene for which the metabolite 2-naphthol was significantly higher in our study compared to NHANES.

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MS. HURLEY: So just to talk a little bit more about the naphthalene results, which are intriguing, we

haven't really had a chance to really dig deep into these findings. They're sort of hot off the presses so to speak, so -- but what we can tell you is that it doesn't appear that the higher geometric means in our study are being driven just by a few high outliers. All the SAPEP participants had at least one urine sample that had a level above the median seen in NHANES.

And we also have a lot -- quite a bit of information about tobacco and vaping related exposures, both from the questionnaire and then also from cotinine analyses. It doesn't appear that the high levels are being driven by those exposures. We haven't really done a formal analysis yet to evaluate the association between the 2-naphthol in urine and the naphthalene air concentrations at the school. But just overall the air concentrations of naphthalene in and around the school during the study period, didn't seem to be especially high. And it should be noted that there may have been some interference in the 2-naphthol measurements by coelution with 1-naphthol.

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MS. HURLEY: And just a few more additional considerations to think about as we're trying to interpret these findings, you know, reminding you all again that the NHANES data were collected five to six years before our

data. And there does seem to be some indication from U.S. and European biomonitoring surveillance data that shows urinary 2-naphthol levels seem to be increasing in recent years. And then sort of relatedly, we don't have much biomonitoring data yet in populations from sort of the post-COVID or during COVID years. And so, you know, what we know about primary sources of naphthalene exposures, which include air emissions from fossil fuel combustion, tobacco smoke, use of mothballs, that those all come from information gathered before the pandemic. And we all know the world has changed a lot in the last few years, so perhaps there could be newer unrecognized sources of naphthalene that might be emerging as important. And then also it's important to note that other chemicals besides naphthalene might contribute to urinary 2-naphthol levels.

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MS. HURLEY: So as I said, we haven't really had a chance to really drill down into the data and sort of figure out what's going on. We are intending certainly to do more detailed analysis of the SAPEP data that will include not just looking at the urinary levels in 2-naphthol, but also considering the naphthalene air concentration values that we have, some of the questionnaire data to see if they point to any particular predictors of exposure. We can also look beyond the SAPEP

data and look at other data that may be available that could indicate sources in the San Joaquin Valley or in Stockton that could be driving these results.

And then we certainly are planning to do a literature review to get a better understanding of the pharmacokinetics of naphthalene and the specificity of 2-naphthol as a biomarker of exposure.

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MS. HURLEY: So beyond, you know, just digging deeper into our naphthalene results and some of the initial results of the other metabolites, we now are ready to really look at all the data in its totality, so conduct an integrated analysis of the biomonitoring, the air quality and the questionnaire data to really address the primary aims of the study. So to further characterize air pollutant exposures and potential predictors of the exposures, to explore associations of the PAH and VOC metabolites with the biomarkers of oxidative stress and inflammation, which may provide some insights towards potential health effects and to evaluate the effectiveness of the school air filtration.

So, you know, we may have a small -- we do have a small sample size. It's unlikely the biomonitoring data is going to be able to answer all these questions, you know, all by themselves, but we do have a wealth of data

that we've collected in this small study. And hopefully by -- you know, each piece will provide a little clue when we put it all together -- could help give us some good insights into answering, you know, some of our study questions.

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MS. HURLEY: So that -- with that, I will finish up and thank you for your attention and happy to take any questions.

CHAIRPERSON SCHWARZMAN: Thanks so much, Susan. I appreciate the presentation. We have -- let's take five minutes for Panel questions, clarifying questions about the presentation, and then we have a longer stretch for open discussion and input on both update presentations.

So questions for Susan. I want to acknowledge there's a comment or question in the chat, but that's more directed toward the previous speaker and so we'll return to that in our open discussion section -- session.

Jenny, you had a question.

PANEL MEMBER QUINTANA: Hi. Thank you for that presentation and it's exciting to again measure these metabolites and look into community solutions like you're doing. I think that's great. I did have a question about the NHANES data, especially the finding that the PAH levels were higher in the NHANES population for many of

the PAH metabolites. And, you know, I'm always struck when I look at NHANES data about how much more exposure to tobacco smoke that children have across the United States than they do here in California. And I'm just wondering if you got the NHANES data and really screened out anybody with cotinine indicating any kind of environmental tobacco smoke exposure, if that would be a more fair comparison between the two?

I'm not talking about just really high levels, but even fairly low levels of cotinine are indicating tobacco smoke exposure and are so much more common in other states to be honest. So I was just kind of curious whether that would go away or even show an opposite effect if that was removed.

And then my other comment was, and this is just from my very -- not very good memory, but I seem to remember in the Central Valley and in the Imperial Valley that naphthalene is thought to be a marker of agricultural burns. And I know you said the air at the school was not particularly high, but I'm not sure exactly where these children live. And I also remember it was triggered from your picture that I think you had one of your sampling events was raining a lot. And so perhaps that would not be an issue anyway, they can't really burn in the rain, but, you know, I was just kind of curious. That is a

fairly unique exposure to Central Valley residents. So that was something that I was interested in. Thank you.

MS. HURLEY: Yeah. Thanks for those questions.

So on the smoking related issue, I can't remember now and I -- I'm not sure I -- if -- I think Dan is on the line, but we did -- so these are the comparison we used for NHANES was kids in the same age range. And I can't remember - and, Dan, if you're on, if you could jump in - if we were able to screen out the non-smokers. I think we were, but, of course, that wouldn't screen out passive smoking exposures.

PANEL MEMBER QUINTANA: Yeah, and that passive smoking.

MS. HURLEY: Yeah. Okay.

PANEL MEMBER QUINTANA: Even if the parents were non-smoking, it doesn't mean they don't go see grandma that smokes and so it's --

MS. HURLEY: Right.

PANEL MEMBER QUINTANA: You'd have to get the data set and eliminate those people with -- or those children with higher cotinine values -- higher passive smoke cotinine values, I guess.

MS. HURLEY: Yeah, it's -- I mean, you know, it's curious, because I think all of these PAHs have significant sources from tobacco -- you know, tobacco is a

significant source, so it's kind of weird that we see a flip with naphthalene being higher in our population and the others lower, so it might be a complicated story.

And then in terms of the agricultural burning, yeah, I -- that certainly is a recognized source of naphthalene in, you know, general populations. I don't know -- I don't, at my finger tips, have information about, you know, what it specifically looks like in the Silicon Valley. Although, we did come across an interesting like doctoral thesis or unpublished data from UC Davis that a student did that showed that naphthalene concentrations in wheat were higher in the Silicon Valley than they were in the Sacramento Valley, which was just kind of interesting. I don't know how much agricultural burning of wheat happens in the -- in the valley, but, yeah, very good thoughts.

And then in terms of the rain -- oh, did I just -- I just got a text message. I think I said Silicon Valley again. I meant San Joaquin Valley.

(Laughter).

MS. HURLEY: Sorry. What was I going to say? Oh, about the rain. So, yeah, we haven't -- we haven't had a chance at -- like literally we were just churning out these results a couple weeks before this meeting, so we haven't really had a chance to look to see

separately -- looking separately at week one versus week two. And it could be that the rain is, you know, going to cause us some problems in interpreting some of the week two data.

PANEL MEMBER QUINTANA: Thank you.

MS. HURLEY: Yeah.

CHAIRPERSON SCHWARZMAN: Any other clarifying questions?

In that case, we'll move on to the open discussion section about both of these two previous presentations. With regarding Susan's presentation, the Program is interested in feedback on these results, the initial VOC and PAH results from SAPEP, including any insights or resources you might be aware of to further explore and interpret the 2-naphthol findings.

And then for Kathleen's presentation, we have sort of a series of follow-up questions. And I wonder if it would be best for Kathleen to reshare that slide.

DR. ATTFIELD: Certainly. Would you like to start with comments on Susan's first while I bring that up?

CHAIRPERSON SCHWARZMAN: Sure. Since -- yeah, that's fine, since it's the information that's just been presented. That would be helpful.

Any guidance from Panelists on how to think about

these results or resources?

If there is nothing further to add there, we can go to Kathleen's slide with questions -- sort of follow-up questions for the Panel and discussion points.

DR. ATTFIELD: Sure, just a moment.

MS. JARMUL: And while she's doing that, I could always read the public question and comment.

CHAIRPERSON SCHWARZMAN: We have a hand from Ulrike. Hang on one second.

PANEL MEMBER LUDERER: I just had a quick question related to the naphthalene. Susan, do you -- did you ask about mothballs in the questionnaire?

MS. HURLEY: We did not.

PANEL MEMBER LUDERER: Oh, that's too bad.

(Laughter).

MS. HURLEY: Yeah.

PANEL MEMBER LUDERER: Thanks. Yeah, that would be helpful.

CHAIRPERSON SCHWARZMAN: Stephanie, do you want to read the question that was a follow-up question for Kathleen?

MS. JARMUL: Yeah. This is from Jen, one of our attendees. She says, "Thanks for the great presentations. Can anyone speak to the Water Board's implementation of the new requirement for all water systems regardless of

size being tested for PFOS? I live in a very small water district serving 815 homes and our watershed includes a ski resort. There are papers showing higher PFOS levels in ski resort impacted watersheds and I'd like us to test. However, there's concern about cost since our rates are already double the area water rates and about to increase."

PANEL MEMBER McKONE: Can I -- I can address some of that, if that's okay?

DR. ATTFIELD: Please, go ahead.

PANEL MEMBER McKONE: Hi. Sorry, I was late coming to the meeting. I'm Tom McKone. So this is an interesting question, because I think it -- I can't really address the question of cost, but the -- there's been a lot written lately and a lot of discussion about recreational equipment in general, and ski equipment, and ski waxes, and specifically. So in the recreation field, there's a lot of water resistant, water repellant boots, clothing, rain gear. And in skiing, the same thing applies -- specifically applies. People want clothing that is water resistant, water repellant.

And in cross-country skiing and I think somewhat in downhill skiing, the waxes that they use to coat the surfaces are -- have fairly high concen -- there's been some work showing the high concentrations of PFAS in these

substances. So it does raise this concern that particularly ski areas where there's so many people concentrated with this kind of equipment, you know, and falling into the snow or skiing across the surface that there is a concern about the watershed. So it's a legitimate concern.

And I think, for me, it raises getting to Kathleen's bigger points about how do we use some of the water data? I think it would be useful to sort of do some cross comparisons of hot spots and address this question. You know, I don't really know. I mean, there's concern, because people measure -- mainly measure the PFAS compounds in ski waxes, ski equipment, boots -- waterproof boots, coatings for things. And I don't know if there's been a lot of corresponding focus on watersheds that are specifically linked. And again, maybe ski areas again, because they're so concentrated, but other recreational areas.

And then the other comment would be should we be thinking more broadly about how to use water system data to look for hot spots, areas where there's like occupational or production facilities that would be producing these and we might expect to find a hot spot in a water supply.

So these are just some thoughts, but thank you

for the -- for the question, because I do think it gets to the core of some of the things we're trying to answer.

DR. ATTFIELD: I just want to give a moment for -- we had -- I know we had a couple members of the staff from the Water Board attending today. I did get a message that one had to drop off, but I just wanted to give a chance in case there's a member who would like to speak to this point, otherwise I can give an approximate answer.

So what I know of the 2023 required testing that they're asking of all public water systems in the state is that they're -- that they are working on contracts for funding for smaller community systems, so the ones that are defined as disadvantaged communities and severely disadvantaged communities with, I believe the definition is, disadvantaged communities being at 80 percent of the state median income, and severely being at 50 percent of the state's median income. So I know the contracts are in process now, but have not been released.

CHAIRPERSON SCHWARZMAN: Thank you for that.

Kathleen, do you mind showing the slide that had your sort of follow-up questions.

DR. ATTFIELD: Oh. Well, there were two sets. There was sort one set of follow-up questions wasn't actually on a slide and it was related to this overlap of

the EPA UCMR 3 data as well as the Water Board data as far as what other uses the Panel might suggest that we put the overlap of data with. So this is just a reminder of the data that we have mostly available to us so far. We don't have all the 2022 data, of course.

CHAIRPERSON SCHWARZMAN: So this question is sort of getting at what other projects or organizations might you contact in terms of thinking about PFAS in drinking water that can complement or inform this work, is that the point, Kathleen?

DR. ATTFIELD: Yeah, we were also interested in the sort of different investigative questions that could be -- like could be looked at with the serum data as well as the drinking water data. These are sort of the two that are top on our list, but we want to make sure all this State-collected information is used to the best of its extent.

CHAIRPERSON SCHWARZMAN: Go ahead, Ulrike. Sorry, I was muted.

PANEL MEMBER LUDERER: No, that's okay. You know, kind of apropos that I -- so do you have data -- did you ask all these participants about, you know, their source of drinking water like whether they drank mainly bottled water versus tap water or if they filtered it, those kinds of questions?

DR. ATTFIELD: Yeah, so we asked two questions in CARE. One, what is the main source of water in your home? And the possible answers were public water system, private well, and then other and missing, obviously. And the other question was what kind of water do you drink most of the time? And that got at if people were drinking tap water, filtered, store bought, bottled, or other water source. So for the main source of water to the home, about 92 percent across the three CARES said public water system. Yeah. Only, yeah, 1.5 percent said that they were on private wells. And then what kind of water do you drink most of the time? There's a fair split actually, tap water being about 14 percent across the studies, filtered, 41 percent, store bought, bottled water, water coolers, 40 percent, so...

PANEL MEMBER LUDERER: And have you had a chance to look at any of those and how they were associated with the PFAS in the participants or not yet?

DR. ATTFIELD: We haven't looked at that yet.

PANEL MEMBER LUDERER: Thanks.

DR. ATTFIELD: We thought that question might come, so I had the data available for you. Thank you to Toki Fillman for that.

And I -- the other slide you were alluding to, Meg, I can fast forward to. That was for our ACE work.

Sorry for the flashing of slides. So these are sort of our prioritized investigations. The fish consumption in PFAS, and herbal remedies and personal care products, and metals. But we had a very rich survey. So if the Panel wanted to highlight other sorts of investigations for both community education as well as enlightening the scientific community, it would be great.

MS. JARMUL: And also, if it's helpful, Meg, I could at least show a slide with the discussion questions that we had.

DR. ATTFIELD: Oh, great. I didn't know you had that.

CHAIRPERSON SCHWARZMAN: Okay. Let's sit with these for a minute that shows the other information available from the questionnaire results, so folks can think for a minute about potential other research questions, either for the Program or for outside collaborators, and then we can bring up that slide with the other questions. That would be great, Stephanie.

Ulrike, please.

PANEL MEMBER LUDERER: Yeah. Related to a point that Jenny brought up earlier, did -- in the rice -- were among the 18 questions about rice products or rice included like where the rice was grown, you know, or if they, you know, tried to buy rice that was from specific

regions because they knew -- you know, because they -- the levels of, you know, arsenic, for example, were found to be lower?

DR. ATTFIELD: I do believe we do have some information about rice origin in these questionnaires, if -- is Kelly Chen able to comment?

I know she has a competing --

MS. JARMUL: Yes. I will allow her to speak now.

And Kelly, just unmute.

MS. CHEN: We do have the country of origin of the rice eaten most frequently. That is one of the questions we ask participants.

PANEL MEMBER LUDERER: But not like region within the U.S.

MS. CHEN: Just country.

PANEL MEMBER LUDERER: Okay. Is there -- is it -- there's, you know, some data out there about the southeast, like Louisiana area, having higher levels due to the use of pesticides previously on cotton fields where they now grow rice versus California having lower concentrations of arsenic in this -- in the rice.

DR. WU: I guess we've talked about before, the bags are not always labeled and sometimes they'll say product of the USA. So it's -- it is difficult to know, you know, how good that data is that we've collected from

folks. But also, I think there's -- there is something about access of data. I know when I go to Berkeley Bowl or Whole Foods it talks about what farm your rice came from and a lot about the agricultural source. But that's not true of a lot of rice products. And so we'll have to see how that bears out in Kelly's analyses.

PANEL MEMBER LUDERER: Thanks.

CHAIRPERSON SCHWARZMAN: Other thoughts about directions of inquiry or research programs based on research questions -- sorry -- based on the information that's available to the Program through the exposure questionnaire?

If no more specific thoughts about that, maybe Stephanie you could bring up the slide that proposes some general discussion questions for the remainder of our session here.

MS. JARMUL: Will do. Can you see that okay?

Can everyone see that?

CHAIRPERSON SCHWARZMAN: That's great.

MS. JARMUL: Great.

CHAIRPERSON SCHWARZMAN: So the question that -- Kathleen's slide that we were looking at is that first bullet point. And then the third bullet point is -- gets to the issue of the overlap between PFAS monitoring and water testing. Thoughts on any of this or other

reflections from the two Program update presentations are welcome at this point.

Jenny.

PANEL MEMBER QUINTANA: Are we -- am I correct in thinking we're supposed to be answering these questions that you're showing right now? Is that what you just said?

CHAIRPERSON SCHWARZMAN: Yeah. I think if you have any comments --

PANEL MEMBER QUINTANA: Okay.

CHAIRPERSON SCHWARZMAN: -- or suggestions for the Program, it's all welcome.

PANEL MEMBER QUINTANA: Oh, okay. No, I was just thinking about tribal communities and ski areas, I guess, following up on the participant who made the comment that -- and also thinking about California and the large tribal communities. And I'm just thinking -- curious how much we've reached out to tribal communities for issues that California Biomonitoring might be assisting with. So that just came up to my mind.

And the other thing that came to mind was occupational exposures, again using the power of this analysis to look at occupational exposures, and especially -- and this is just a question I was just thinking of. We had this great textbook I used to use,

Case Studies on Occupational Health. I'm sure Ulrike remembers that one. It's just really nice case studies from I think it was NIOSH, and, for example, Vietnamese Americans and solvent exposures. And I'm just curious if there's -- if there's exposures that track with occupations that we should -- in California, that we should be -- and especially specific communities that we could be -- could be really helping to investigate. So that kind of very general question or comment. Thank you.

DR. ATTFIELD: Thank you. For the occupational question, we do have a fair amount of occupational information both for ACE and for the CARE studies. And as I mentioned earlier, our collaborators at Silent Spring Institute are looking into the occupational exposures and doing all the hard work of classifying the open-ended answers that people provide into categories that can be -- that are associated with the exposure analyte levels.

And for CARE, that's something we haven't had a lot of time to explore yet, but we both have open-ended questions on it and categorical questions like, you know, military service or firefighting. Some of those are going to be quite low in numbers, so we may not be able to have the power to analyze them.

PANEL MEMBER QUINTANA: Yeah, I'm just thinking, if I remember correctly, we oversampled older non-Hispanic

white women. And so it would be nice to kind of really focus on some of -- in the future, I just feel like this is such a powerful tool for bringing attention to exposures and occupational exposures are often so very much higher than the general population, the effect on families too, like with take-home exposures. So I was just hoping we can keep thinking of that as we move forward.

DR. ATTFIELD: Thank you.

CHAIRPERSON SCHWARZMAN: Tom.

PANEL MEMBER McKONE: Yeah, I have -- I have some thoughts about -- or have always been concerned about water testing and matching the exposures. I mean, this goes back to when we were doing exposure tracking. And, you know, it's one thing to do -- to match people to their air, because pretty much people breathe the air where they live, but that's not true of water. And I don't know how much this can be used to really try to understand a little bit more about, you know, the -- the chemicals in your water are related to not where you live, but where the water you drink comes from. Now, that's somewhat related to where you live, but not always. It's gets -- water distribution systems are a bit complicated.

So I don't know. Just a suggestion about it might enhance the ability to understand the biomonitoring

if there's a little more effort to do some mapping of water supply to water consumption. Again, it's not -- it's actually -- we -- I mean, we've tried to do this before. It's a bit difficult and involves a lot of records with water companies, because they actually switch sources at certain -- some of them use surface water for some of the year or they'll go to local groundwater for another part of the year. So again, it's just a thought about how to enhance or better understand that, to match people to their water supply, and particularly the variations in where that water supply comes from, so what's coming out of their tap.

And I guess -- I mean an additional thought in that area is, you know, I forgot whether you put this in the questionnaire or -- I came in late. I might have missed this is you asked people about how much they have home gardens or consume food they produce, you know, in their own backyards.

DR. ATTFIELD: (Shakes head).

PANEL MEMBER McKONE: Oh, you don't do that. Okay.

DR. ATTFIELD: No. No.

PANEL MEMBER McKONE: Because that's actually a way for some of these more persistent chemicals that bind to vegetation. And it's actually an issue of, you know,

if somebody has a home garden and consumes any significant amount of food from it. I know it was -- it was an issue with other chemicals. But anyway, it's just some thought.

DR. ATTFIELD: And chicken eggs internationally.

(Laughter).

DR. ATTFIELD: To respond to the first part of your comment, yes, water distribution systems are quite complex and we -- as I said, we have sort of a diversity of data around different sampling points and different sampling time points from the different water systems. Our partners at the Water Board do have access to schematics of how the -- how the different point sources funnel into different treatment situations and then into the distribution point, and may be able to access information on blending and when certain wells are turned off and when different sources are employed. But as I'm stating it, it is all very complex. So we're still at the point of assessing like exactly how much we'll be able to incorporate into the work.

CHAIRPERSON SCHWARZMAN: Nerissa.

DR. WU: Hi. I just wanted to respond both to Tom and also to Jenny's question that we sort of went by about tribes. And we did -- we have reached out to tribal organizations as part of our EJ listening sessions that we did now quite a few years ago, when we were trying to

identify what were concerns about environmental health across the state. So we do have some data on that. And, of course, it is a group when we do our demographic analyses for any of our -- any of our results. It is a group for which there are very, very small numbers and so we haven't been able to produce stable statistics for that group.

But I'm thinking that for both occupation and for tribes, the STEPS data, which will eventually be a larger data set, we have -- we do have parental occupation. We will have racial identity by, you know, non-exclusive categories. So we will be able to look any -- you know, anyone who checks off Native American as part of their identity and maybe accumulate numbers over time. That might be able to be -- help us summarize any of those statistics.

And with regard to Tom's question, I mean, this is a little bit of what we're talking about with Matt's presentation. There are so many variables. There's so many different sources. There are lots of things we want to capture for any study we're doing. And it's difficult. It's one of the things for which -- which explains why we need such large numbers for any one of these studies. But every questionnaire, if we're not focusing on like the 20 questions you can ask about rice, if you're going to do

that for every exposure source, you end with a very, very large questionnaire and you start bumping up against what participants are willing to answer and also how much time they're willing to spend, which is why the power of something like NHANES which collects so much information, you know, why they have so much more power than we do.

But it's something we think about. And some of these smaller studies, which can really delve down like ACE into very particular exposure sources, are a good complement for the surveillance work we do, which we can't ask about everything that we're interested in.

CHAIRPERSON SCHWARZMAN: Before we go to public comment -- open public comment, any more points for this discussion session. Or Stephanie, if there's anything that has come across the email?

DR. HOLZMEYER: This is Cheryl. There's no new emails. Thank you.

CHAIRPERSON SCHWARZMAN: In that case, thank you to both Kathleen and Susan for your presentations.

And our last agenda item is an open public comment period. So we have 10 minutes allotted for this, if necessary, and commenters can provide opinions on any topic related to Biomonitoring California. And a reminder that webinar attendees can submit written comments and questions either via the Q&A function of Zoom webinar or

by email to biomonitoring@oehha.ca.gov. We'll read them out loud. If you want to speak, please use the raise hand feature in Zoom and I can call on you.

So maybe we'll just leave a few minutes here, even if no one is raising hands, to let folks submit via the various mechanisms.

Stephanie, does that sound good?

MS. JARMUL: Yeah. And we do actually have a hand raised.

Nancy.

MS. BUERMEYER: My name is Nancy Buermeyer, Breast Cancer Prevention Partners. As always, thank you for all of the presentations and all of the great work. And it's great to see the additional resources allowing you to do more of those analysis that is so useful to the work that all of the advocates do.

I had a question. It's -- I wasn't able to attend all of the meeting, so I don't know if this is possible, but I thought I heard something about looking at bisphenols in, I don't know if that was CARE data or some of the other things. But there is some legislation this year in the Legislature in California to ban the use of BPA and BPS on thermal paper. And so having any data on sort of particularly occupational exposures for cashiers or retail workers who have to handle those on a daily

basis, and their exposure to BPA or BPS. I don't even know if you work on BPS or not. But anyway, I was just curious if you could talk a little bit about whether that data might be coming.

DR. ATTFIELD: So for CARE -- and thank you, Nancy. It's great that this is so topical to what is happening in the Legislature right now. So for CARE-LA and CARE-2, we had a subset where we were measuring environmental phenols. And so we're actually working on expanding that to the rest of the CARE population. So hopefully in a year or two we'll be able to deliver information on that.

What I had mentioned about bisphenol A was a method development work from the Environmental Health Laboratory at CDPH, where they're developing the ability to track the specific metabolites of BPA in addition to the free form. So that's BPA. But the regular method does BPA and BPS and I believe BPF. And that -- the data that is available for the subsets is already up on our website and can be viewable. Happy to send you a link as well or post a link.

MS. BUERMEYER: Great. Thank you.

CHAIRPERSON SCHWARZMAN: Cheryl, anything by email that we should tend to?

DR. HOLZMEYER: I believe Stephanie is the only

person who can see that at the moment, as I'm screen sharing.

MS. JARMUL: No further emails.

CHAIRPERSON SCHWARZMAN: In that case Stephanie, is there any -- do we -- do we need to keep it open for the full 10 minutes or is it okay to adjourn a little bit early?

MS. JARMUL: We can adjourn a little bit early if there are no further public comments.

CHAIRPERSON SCHWARZMAN: Okay. In that case, we will move toward adjournment. There will be a transcript of the meeting posted on the Biomonitoring California website when it's available. And the next SGP meeting will be on August 21st, 2023 from 1 to 4 p.m. And the information regarding options for attending that meeting will be available closer to the August meeting date.

So thank you to the staff who put together the meeting, and to the Panel for being here, and the audience also, and our speakers. And I'll adjourn the meeting.

Thanks.

(Thereupon the California Environmental Contaminant Biomonitoring Program, Scientific Guidance Panel meeting adjourned at 12:51 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 16th day of March, 2023.

JAMES F. PETERS, CSR
Certified Shorthand Reporter
License No. 10063