Transcript

July 28, 2025, 9:00PM

Wyton, Pamela D. (Pam) (ATSDR/OCOM) (CTR) started transcription

Weems, Meghan M. (ATSDR/OAD/OCHHA) 0:07
I'm Megan Weems, an epidemiologist with the Agency for Toxic Substances and Disease Registry and the manager of the PFAS multi Site study.
I will be moderating this evening's open house before we get started.
There are a few housekeeping items I would like to draw your attention to.
This meeting is being recorded, as I said earlier.
The chat and the Q&A are disabled due to the large.
I'm sorry, I think we may be having a few audio problems.
Wanna verify with anyone who's able to.
Let me know if you can hear me now.

Wyton, Pamela D. (Pam) (ATSDR/OCOM) (CTR) 1:35
Yes, we can.

Weems, Meghan M. (ATSDR/OAD/OCHHA) 1:37
Great. Thank you very much.
I'm going to share my slides again.
OK.
Let's start again housekeeping before we begin this meeting is being recorded.
The chat and the Q&A are disabled due to the large number of participants and Microsoft Teams constraints. Despite the ability to raise your hand virtually.
Unfortunately, we are unable to take questions or comments from participants during this meeting. For questions about the PFAS multi site study.
Please e-mail.
Us at CDC, Gov.
We are unable to answer individual health or medical questions about PFS.
Us on this call, but if you have questions like that, please e-mail ATSDR, medical officer at CDC, Gov and for all media inquiries, please e-mail.
Env health media at CDC Gov.

This evening we will discuss the recently published manuscript multi site study of Communities with PFAS contaminated drinking water, methods, demographics and serum PFAS concentrations.
In addition, investigators from each of the multi site study sites will provide early findings on PFAS exposure and health effects including lipids, heart disease, blood pressure.
Diabetes, metabolic syndrome, thyroid and obesity.
For those attendees who may not be familiar with the study, I'll provide a little background information.
The overall goal of the multi site study is to provide information to communities about the health effects of exposure to PER and Poly floral alkyl substances or PFS, as are more commonly known. These are man-made chemicals which have been used in industrial and consumer products around the world.
Since about the 1940s.
Information learned from the multi site study will help all communities in the United States who have been exposed to PFAS through drinking water, even communities that were not directly involved in the study.
In 2019, CDC ATSDR established a cooperative agreement with eight study teams to carry out the multi site study in communities across the nation. These eight study teams are represented at this evening's meeting and include scientists and researchers from Portsmouth and Newington, NH.
Orange County, California.
El Paso County, Colorado Air, Massachusetts, Hyannis, MA Belmont, Rockford Area, Michigan Parchment, Cooper Township, MI.
Gloucester County, New Jersey, Montgomery and Bucks Counties, Pennsylvania.
Hoosick Falls, NY, and Newburgh, NY.
I'd like to take a brief minute to sincerely thank.
All who have been involved in this study, the participants, the Community assistance panels, involved communities and the hard working study teams. This study is is a success because of each of you.
Our topic 6 evening include an introduction to the study and results from the first MSS paper, followed by a presentation on how the MSS data are being analyzed and then onto preliminary results from researchers from study teams.
Let's begin with the presentation from Doctor.

Marion paavouk.
An epidemiologist at ATSDR and the MSS principal investigator.

Pavuk, Marian (ATSDR/OAD/OCHHA) 5:22
Good afternoon, everyone.
Can you hear me OK?
Thank you, Megan, for the kind introduction.
Good afternoon, everyone, and thank you all for being able to join us here tonight.
As Megan mentioned, I will provide a brief highlights of the recently published multi site study methods paper.
That also talks about demographics and ethos, blood concentration that were measured across the sites to step back a little bit.
On the multi site study, the ATSDR was authorized by U.S. Congress through the National Defense Authorization to conduct war contamination study that would look at multiple PFAS at sites across the nation.
The different exposure levels to provide information to communities about the health effects of Ethan exposure.
The multi site study expanded on the peace health study.
That was conducted near Portsmouth, NH. That was the first site of the multi site study that later expanded to other sites across the nation.
Study findings, as Megan mentioned, it will inform various communities in the United States with similar PF drinking exposures. Next slide, please.
Excellent.
So put our PFAS why did we study them on this chemical substances?
It's a large group of chemicals.
That was manufactured industrially large amounts, and was used by lean industry and consumer products because of their.
Properties to repel of order and oils.
During.
These production water contamination from the manufacturing facilities and the use of firefighting firms and military bases in airports resulted in contamination that is widespread and effects a large number of people.
Estimates around 80 to 100 million residents around the United States.
Most commonly studied PFAS include perfluorocenolic acid P4 and perfluorochtane sulfonic acid.

Pfos. Well, these are no longer produced in United States.
Other PFAS are being still introduced and these PFAS remain in environment and in bodies of of of people.
And there may still be manufactured in other countries.
There are other befores that are studied in MSS like PFH, excess, pfna, pfda, pfan, dafosa.
There was titles octanovic, Nona, Deca.
Just refer to number of fluorine.
In the molecule of these chemicals are usually the more fluorines, the longer they persist and the longer they stay in in human bodies. Next slide please.
So the overall goal of multi site study is to investigate the relationship between SPI fast chemicals and the health outcomes across different populations and aggregate in a study data expanding our understanding of PFAS and the risk to our health.
So we're going to look at relationship between specific health outcomes and FS exposures.
Which ones may be associated with BFA's exposure?
And also look at the information on various help points and clinical tests and biomarkers and PFS levels that we measured in blood participants. We're able to look at individual ethos, but also had a mixture of ethos through various statistical methodologies.
Part of the study is also historical reconstruction of ethos concentration that can help better understand effects of some long term exposure.
But also provide the estimates of concentration before disease that are being studies developed. Results of the study can be generalizable to number of population that are affected by FIFA through contaminated drinking water.
Next song please.
So who was able to be in this study?
Call it eligibility.
Adults age 18 and older of children aged 4 through 17 that lived or have lived in the areas of contaminated drinking water that also included people that were exposed to P Faas and utero or urine breastfeeding.
People that were exposed more than 15 years before the study began.
Are not eligible.
Also, people that could have had exposure to beef us through their workers, 50th workers in a chemical or industrial plants that use FIFA's were not eligible.

Next.
So we collected a number of data and samples in the study.
Each participants provided data on demographics, social behaviors, medical, family work, and.
In residential history, through interviewer administer questionnaires.
Then we measured the number of body measurements during the study.
Office visit we also measured resting blood pressure and collected blood, serum and urine.
From the participants.
Neurobehavioral tests were also part of the examination for some of the children on the PFAS measured in serum were analyzed at CDC laboratory at National Centre for Environmental Health. Most of the clinical tests were done by LabCorp.
Similar to the test that you would get at your at your family physician and some of the research biomarkers were done by State University of New York, Upstate Medical, University of Rochester.
Next one please.
So this is the paper that we published actually last month was published in Journal of Environment International.
I'd like to thank all the investigators.
From the cooperative cooperative partners that participated in this study, the paper I did go over some of the results that describes the methods how we selected participants who was in the study.
What methods did we use to get the information on their health outcomes?
Which tests are clinical and biomarkers?
They did, and other interesting information that will be useful as a reference for other held outcomes papers in the future. Next one please.
So.
The recruitment for the studies started in 2019. In November at Pace and continued through through the the COVID some interruptions and delays, of course, through 2020 and 2021 and continued through September 2023.
As I mentioned, the study would not be possible without our partners and the Community.
Stakeholders at different sites.
We worked with researchers at university at Albany, NY State Health Department.
Colleagues of population and public health at University of California, Irvine.

University of Colorado School of Public Health and she's Medical Campus in Denver, Michigan.
Department of Health and Human Services, RTI and Pennsylvania Department of Health.
Rodgers School of Public Health.
So in springs institute.
And Harvard School of Public Health, as well as APT Global. It was the contractor for the collection of the data and samples.
So in total we were able to enroll.
5826 adult and 710 children participants who had.
Questionnaire data.
And provided blood for pipas measurements.
Next slide please.
So the average age of the combined cohort, we aggregated all the data across the sites as well as the comparisons among sites.
Was 54 years for adult participants and 11 years for children.
60% of adult participants were female and 46 of child participants were female.
Over 77 of percent of adult participants were non Hispanic white and over 80% of adult participants had more than half.
High school education next level.
So.
On the exposure side, we have found four PFAS P4 Pfalz, BFH 6 and pfna that were detected in over 96% of all adults in the study.
The levels of these 4P files varied among the eight sides in the study as I'll be able to show you in in the graphs a few slides now and those differences over because of different in type of contamination.
On historical level of FIFA's in drinking water and we wanted to capture that by the variation in those concentrations.
The three other P files PFD API server detected it much lower in in lower in 30 to 55% of adult participants.
So overall, to summarize what we had found before, we get to the figure, we have found that the higher average level of PFA.
Which was about two times higher overall than the levels in adults in general populations.
Also, we have noted that before was about 12% higher in adults than in US general

population. Children in this study had lower P5 levels than the adults and compared to children in general populations we have seen that they had higher levels of PFH access, so the.
P valleys that are there.
Lower than 00.
One are highly statistically significant and we compared the two groups.
Also, younger women ages 12 to 49 usually had lower levels of most FIFA's than males in older adults, about 60.
Those levels are again similar between males and females. Excellent.
So here we see it in in a graphical form. So in the right top corner you see the PFA access.
In yellow you see the general US levels.
This is 1.1 nanogram per milliliter, and you can see that all sites combines there.
It's 2.1.
Also, it's almost two times higher overall.
You can see that seven of eight sites did have higher levels of PFH access.
Than the average general US population.
So to a smaller degree, we've also seen that before was higher.
1.6 for all sites combined 1.4 in general US population is considered that there are five sites that are higher than general US population.
Four, this is at the bottom left corner P4.
For P fours in in left top corner and PFNA you see that the average concentrations were lower in multi site study than in US general population.
But you still had a few sites.
That they're close or or above those levels.
So more detail on on the the demographics and the results and comparisons among the sites and the US general population can be found in our paper that is now available on the ATSDR website.
For your reference.
So thank you very much for your attention.
And I'll yield to the next presenter.

Weems, Meghan M. (ATSDR/OAD/OCHHA) 18:59
Good evening everyone.
Just to interrupt for a brief moment, it's my understanding that some folks are having

trouble seeing the slides, and I wanted to recommend if you're having trouble seeing the slides to log out and log back into the meeting, that seems to be fixing the problem for folks that.
Are having it.
Thank you very much and thank you very much, doctor Pamluk.
Doctor Pavluk next we have Tamara James Todd of the Massachusetts MSS team who is going to discuss how the MSS study data are being analyzed.

JT **James-Todd, Tamarra** 19:35
Again, so we wanted to take a moment to walk you through what our process is for analyzing data as researchers and we want to, you know, lead from where doctor Pavich left us with understanding kind of the basics of how did the study come about, how were people?
Recruited into the study and essentially what did the PFAS concentrations kind of look like overall?
To actually getting to the core piece of how do we analyze?
These data to understand how PFAS impacts health and as a step one, we have to, as researchers clean our data and prepare it for analysis.
So I will be brief in my kind of walking you through these steps for the sake of transparency.
And lead into what some of our preliminary results are, but as a first step, we really need to look and review all of the information or data that was collected across the 8 sites and we are doing that to check for consistency missing this. So if folks did.
Not answer questions and we expect that to happen from time to time.
But where there might be a lot of missingness or places where many people did not respond, we need to take a look at that.
We also need to look at potential errors.
For example, if someone or somewhere in our data, we see that someone's a 211 years old, well, that's not accurate.
That didn't happen, but it's just an example of, you know, places where we might have to make or further evaluate our data to make sure that it is indeed clean.
The next step is to really develop new variables that can be used in our analysis.
So for example, we need to really create a new variable variable, for example, that looks at total exposure to PFAS.
In my site Massachusetts, people are in and out of the study area just because of the

type of communities that people live in, so they may.
Move in and out.
Return for longer periods of time.
And so we have to add the total amount of time they lived in the exposed community in order to really get at their total exposure level.
And then finally, we have thousands of variables within this data set.
Again, there was.
There were surveys that were done.
There were biomarkers collected.
There were examinations done where we, you know, measured people's weight and height. And so we really need some sort of, you know, documentation.
Where as researchers, we can go back and kind of know exactly what a variable is called and making sure that we can use the correct variable in our data analysis. Next slide please.
Once we've made sure that our data is prepared for analysis, we then can create an analysis plan and in order to do that, we as researchers review the literature and identify what gaps may exist in the literature, and we develop plausible hypotheses.
This means hypothesis that are educated guesses and questions that are really based in the biology and the health literature, and so on.
That is, as evidenced in that is out there.
Next, we have structured a process within the MSS that allows us to write up an analysis plan.
This means that we can write up specific variables that we now have and can identify specific individuals that meet the eligibility criteria for a particular research question that we might be asking, and then applies statistical methods that are appropriate for.
The question that we are trying to address, we've set up a publications committee to review these analysis plans that ensure that there's valid methods that are being described and applied for use within a particular set of research questions and that the appropriate variables are being used and at.
Times this committee will make some recommendations that can be taken back and and integrated.
Into ensure that the best science is being done.
And that it can yield valid and reproducible research results.
Next slide please.

Once the analysis plan is approved, we as researchers can then move on to actually conducting the data analysis and there's multiple steps that are involved in being able to conduct a data analysis. You've seen a little bit of that already with the previous slides.
That doctor Pavich presented where we start with a descriptive analysis.
Or in other words, describing our data, we want to both describe the PFAS levels that are within the Community as well as the health outcomes, and also look at things like where do you know the site that individuals are recruited from or their age or other community descri.
That are relevant to whatever research question that we're asking as it relates to health outcomes.
The next thing is that we want to use the statistics or those mathematical.
Tools and resources to actually evaluate the that get to the heart of the question.
So we wanna be able to apply those methods and we work closely across our teams to be able to do that.
So some of the questions that you're gonna hear more about are the associations between tifos and diabetes or PFOS and and thyroid disease.
And so we look at that, but to also ensure that.
Our research questions are consistent.
And valid and robust. We ask additional questions oftentimes within our data analysis and these are often called sensitivity analysis.
These may allow us to look at more specific questions about certain population subgroups. For example, older individuals.
Or a particular community that may have had one type of PFAS exposure versus another.
So it allows us to get at that and kind of track and check for consistency.
Within our study results and then finally, we have to construct tables and figures to help us to best present those research findings. From that we've conducted as a part of these analysis.
Next slide please.
One important point that I just want to raise and and this came up again before, is that we did measure PFAS in blood samples.
So one of the challenges though is that in all the communities that we that this study was conducted in, the PFOS exposures have been reduced or eliminated within the study period.

With across time, which means that what we're measuring in that period.
Of 2019 to 2023, our much lower concentrations at times than what may have been concentrations or exposure levels at a time point in history or in the past where these communities may have been much more highly exposed. In other words, the past PFOS levels of.
Exposure do not equal the current PFAS exposure levels.
And that means that the blood concentrations may not be equivalent to what people were exposed to at kind of the height of of the period, because PFOS levels may be decreasing in the body over time.
So here we're looking at PFAS concentrations at a time point where they're lower as it relates to health outcomes. At the same time point.
And that's called a cross-sectional study. When we're looking at something at the same time point for both when people.
Are the exposure and the outcome that we're studying within the research study and so one way that we're trying to address this issue where we recognize that we're looking at PFAS concentrations at a much at a time point in which concentrations are much lower in the blood levels.
Is next slide please.
Is to do a reconstruction of the historical PFAS blood levels and for this we're working across our study teams with experts that really think about this question of how do we go about doing that.
So for this we can model the movement of PFAS in the environment to estimate the past PFOS levels in the drinking water.
So in other words, we know.
Where people lived within our sites.
Now, I'm not saying that.
We across all of the different sites, we know the addresses and so on, but within each site we have a sense of where individuals were located relative to where the PFAS contamination was happening.
The next step is then to estimate how much people were actually ingesting the water.
And from from, from the contaminated drinking water and we can get at that through survey data. For example, we know how much.
Three top water individuals were drinking and we can kind of estimate what that looked like next. Once we know that we have a sense based on people's age, their body size or composition as to how the PFAS moves through the body.

So this word apply. Pharmacokinetic modeling is essentially saying how does the PFAS based on some of these demographic and other characteristics that we know are relevant to PFAS concentrations in blood.
Levels. How does that impact what, you know, the exposure levels were for each individual and then once we have created these models, we can compare those to the PFAS blood concentration levels that we currently have and see as a kind of validity check that these levels are you.
Know kind of do this comparison to make sure that what we're seeing.
Is valid and then we can actually integrate this into.
To our epidemiological studies or the studies of health outcomes. So for this we can eventually have the data on the historical PFAS concentrations that each individual has and look at that at and see its association with health outcomes.
Thank you.
And we'll move on to the next presenter.

WM **Weems, Meghan M. (ATSDR/OAD/OCHHA)** 30:20
Thank you very much, Doctor James Todd.
Just a gentle reminder that if you're having trouble seeing the slides, it may help to log out and log back in again.
Next, we will discuss preliminary results for PFAS and selected health effects.
We'll begin with a discussion of lipids research by Doctor Marion Public.

PM **Pavuk, Marian (ATSDR/OAD/OCHHA)** 30:48
So I'll probably overview of results from PFAS and blood lipid levels. This work has been done or led by the group at Rutgers University of New Jersey because of personal emergency. I'm presenting for Doctor Robert Lombok.
That would have been presented this.
Results.
So.
Why are we studying blood lipids, cholesterol and triglycerides?
In general population, higher cholesterol and triglycerides levels have been strongly linked to increased risk of heart disease and stroke, and earlier studies showed that exposure to some E files might change cholesterol and triglycerides levels.
In blood.
Most of the old studies looked at before and before. As I mentioned, those are the

most studied PFAS, bfna and other PFAS are also studied.
So what are we trying to learn about PFAS and blood lipids?
We want to learn better, higher exposure to PFAS is associated with higher lipid levels.
What it means that the PFAS increase?
Or the blood lipid levels.
Increasing. So for this we used the current or blood PFS concentration that we measured in P4.
And are doing analysis that those results.
We want to know that as certain P files appear to be more strongly associated with blood lipid levels and that our findings are consistent with PFS being cause of increased blood lipid levels.
Next song please.
As I mentioned, we measured 7 Pfas in in blood samples.
And we have also measured different kind of lipids in the blood samples similar to the you go see your doctor, you would get the results for total cholesterol, low density, lipoprotein cholesterol or LDL, a high density lipoprotein cholesterol or HDL.
Non HDL or cholesterol.
And then triglycerides.
We studied the relationship of level of HPF with the level of blood lipids and in our analysis we considered number of factors that could influence those associations between PFAS and blood lipid levels such as age, sex, blood and mass indexity cigarette and alcohol use income and education.
All those factors may be independent risk factors associated with the lipid levels.
So this analysis included adults.
We do plan, analyze children, adolescents, participants, data in the future.
So what we have found so far?
We did find higher blood levels of P4RB4.
Spfna PFHS PFD PFMDA that were associated with higher total cholesterol.
LDL cholesterol, HDL cholesterol and non HDL cholesterol.
So higher levels of B fads in the blood were not linked to higher triglycerides and so either lower triglycerides or no apparent connection.
With prefiles in our mixture analysis, we found that beef has appeared to have stronger associations.
Some beef has with cholesterol than the others.

And as Doctor Todd mentioned, we may use in the future analysis possible levels.
It's been contrast to the current levels.
In the study, links between certain prefil levels and cross stor levels in children.
And and Dolson study participants. Thank you.

Weems, Meghan M. (ATSDR/OAD/OCHHA) 35:08
Thank you very much, Doctor Pavuk. A gentle reminder if you're having trouble seeing the slides logging out and logging back in May solve the problem.
Thank you very much.
Next we have Doctor Michael Bloom of the New York multi site study team, who will discuss thyroid effects research.

Michael S Bloom 35:26
Good afternoon.
I'm Professor Michael Bloom from the George Mason University College of Public Health, and I do work with the New York State study Team, University of Albany and New York State Department of Health.
I'm very happy to share some preliminary findings with you today about PFAS and thyroid disease from the MSS.
Thyroid gland is a small butterfly shaped organ.
I'm located at about the base of the neck and it produces hormones, primarily thyroxin.
We also call T4 for short and triiodothyronine.
We also call T3 for short.
Now, these hormones influence nearly every organ system in the human body. For example, controlling how fast your heartbeats or how quickly you burn calories or metabolism.
And they even orchestrate how the brain develops and controls.
Brain function. So as you can imagine thyroid function is critically important to our optimal health and function. And when the thyroid hormone production balance is altered or disrupted, this can lead to clinical if there's insufficient thyroid hormone, we call this hypothyroidism or hypothyroid disease. Some symptoms are kind of like. Chronic fatigue and weight gain. And if there's excess thyroid hormones, we call this hyper thyroidism.
And this kind of speeds everything up and can lead to anxiety and and pathologic

weight loss.
So we're studying PFAS and thyroid effects to try to determine if the level of PFAS exposure and the types of PFAS exposure among folks living in the MSS study areas were associated with thyroid disease.
And if so?
Are there any specific chemicals, PFAS chemicals that are more or less important?
To those associations, next slide please.
So we looked at 7 PFAS as individual predictors of having thyroid disease to establish if specific PFAS might be more or less important than other PFAS.
So we kind of considered each PFAS by itself at a single point in time and we also considered other important factors as doctor Pavik mentioned, like like gender and age in our statistical models to kind of remove their effects. So we could try to isolate the potential effect.
Of the pfas.
On on thyroid disease, we also took another approach where we looked at the mixture of all 7 PFAS as kind of a combined potential effect on thyroid diseases using a different statistical approach.
But we also again adjusted for these other important factors, we we call them confounders in the field that might kind of mix up the association between PFAS and thyroid disease and kind of lead the results of stray.
So we did our best to accommodate those factors.
Finally, we look to see if there were differences in the way different PFAS were related to thyroid disease among the different study sites among women and men among people with and without different thyroid autoantibodies in their blood, and also based on when thyroid disease had been diagnosed among.
Those folks with a thyroid disease diagnosis.
Our analysis included 5771 adults.
So 18 years of age and older, and who had thyroid disease data contributed to the MSS and serum P.
Fast data collected.
Next slide please.
So we found that higher blood serum PFAS levels were generally associated with less thyroid disease or were not associated with thyroid disease. And this was true for hypothyroidism.
That scenario, where there's insufficient thyroid hormone and for hyperthyroid

disease, that scenario where there's excess thyroid hormone.
The hypo.
The insufficient scenario is far more common than the hyper the excess.
Thyroid hormone scenario.
However, when we limited the analysis to only look at women.
Higher blood PFHXS was associated with a greater prevalence of hypothyroidism, so that insufficient thyroid hormone scenario.
Similarly, when we limited the analysis to only the men, we found that higher PFOS PFOS alone was associated with a greater prevalence of hyperthyroid disease, that excess thyroid.
So moving forward, we plan to focus on associations between the blood PFAS and the actual measurements of the hormones themselves that we measured in the blood that T4 and T3I mentioned earlier and also to look at PFAS and the levels of the thyroid autoantibodies in.
Folks, blood, we're going to see how those results fit in with results that we found a preliminary analysis of the thyroid disease outcomes I just discussed.
And that's our next step.
So thank you so much for your time today and thank you so much for your commitment to the MSS and and human health.

WM **Weems, Meghan M. (ATSDR/OAD/OCHHA)** 40:55
Thank you very much, Doctor Bloom.
Next, we have Doctor Anne Starling of the Colorado multi site study team who will discuss diabetes research.

SA **Starling, Anne** 41:08
Thank you.
Megan, please tap the opportunity to speak with the all tonight about our preliminary findings.
My name is Anne Starling and I'm affiliated with the Colorado site, the Colorado School of Public Health and also the University of North Carolina.
So our motivation for studying the relationship between PFAS and diabetes in the multi site study is that some previous studies have found that people with higher blood P vast levels are more likely to develop type 2 diabetes.
However, the literature is not consistent on this.

Some other studies have showed no association between PFAS and diabetes, and others have actually shown an inverse or negative relationship.
So it's notable that few of these previous studies were actually conducted in populations with high levels of PFAS in their drinking water. As with the eligibility criteria for being in the multi site study.
So for the seven PFAS that were widely detected among participants in the multi site study, we wanted to know if there is a positive relationship between their blood PFAS levels and the diagnosis of diabetes.
And furthermore, is there a relationship between PFAS levels in blood among people without diagnosed diabetes and certain blood markers that indicate pre diabetes or diabetes risks such as fasting glucose, insulin and glycated hemoglobin or hemoglobin A1?
Next slide please.
So to evaluate this question, using the data that we collected, we used common statistical method which was logistic regression to compare the frequency of a self reported diagnosis of diabetes anytime in your life. Among participants with higher levels compared to lower levels of certain PFAS, we also looked.
At the relationship between each blood PFAS level and those biomarkers that I mentioned glucose, insulin, hemoglobin A1C, using linear regression models.
And in these analysis, we accounted for these other factors.
As previous investigators have mentioned, that could influence this relationship between PFAS exposure and diabetes risk, and these include age, sex, race and ethnicity.
Socioeconomic status indicated by income and education, smoking history and body mass index.
So in the population used to examine the question of whether PFAS were related to a diabetes diagnosis, there were 5753 adults, of which 60% were women and the average age was 56 years.
There was a subset of that population who had not had diagnosed diabetes and had these blood biomarkers of diabetes risk, and that analysis was conducted in 4582 adults.
Next slide please.
So far we have found that about 12% of adults in this population had diabetes.
That's roughly similar to the percentage of adults in the US with diabetes.
But that's not adjusted for the age stratification the population.

Diabetes was not more likely to be detected or reported among participants with higher PFS levels in their blood. In fact, for some of the seven PFS that we studied, participants with higher blood P vest levels were actually less likely to report that they ever had a diabetes DI.
And the relationship between PFAS and the blood biomarkers of diabetes risk was different.
And it varied depending on which of the seven PFAS we were looking at.
For example, when we look at hemoglobin A1C, PFOA and PFOS were associated with lower hemoglobin A1C at the time of the study visit, but Pfda was actually associated with higher HVAC.
So we're continuing to examine the reasons for the differences between the PFAS and between the self reported.
Diabetes diagnosis and the blood biomarkers we're looking to see if this is related like an artifact of the cross-sectional study design and other ways that we could analyze just to be confident about our results.
Eventually we will also use the estimated historic PFAS exposure, as other investigators have mentioned, reconstructed to estimate PFAS levels historically before the diagnosis and the results I've reported today. There were not children included.
In the analysis, because there were very few cases of diabetes among children among the 710 children that were enrolled in this study, however, we will analyze the relationship between PFAS and those blood biomarkers of diabetes risk among children.
That's all. Thank you very much.

Weems, Meghan M. (ATSDR/OAD/OCHHA) 45:41
Thank you very much, Doctor Starling.
Next we have Doctor Yuting Wang of the Massachusetts multi site study team, who will discuss blood pressure research.

Yuting Wang 45:55
Hello everyone this is I'm a researcher working at Silent Spring Institute today.
I'm happy to share our preliminary findings about PFAS and blood pressure.
So why are we interested in studying PFS and blood pressure studies help on that exposure to PFS was associated with an increased risk of high blood pressure during

pregnancy, including pregnancy induced hypertension and precanceria.
You may have heard about PFS and Precan CF. From the California study.
Previous previous studies have also looked at associations between PFAS and blood pressure among non pregnant people.
But the findings have not been consistent.
Some studies found that certain PFAS were associated with increased risk of high blood pressure, but others found no association.
More importantly, PFAS are a large group of chemicals, so we're being exposed to different PFAS at the same time.
But previous studies only assess individual Ppas.
At a time without looking at the impact of P PAX mixtures, therefore, we need more studies to understand the overall effects of P fast mixture on blood pressure.
Additionally, early diagnosis and treatment of hypertension are critical for the prevention of cardiovascular disease, especially among young adults.
Therefore, it is critical to understand the impact of environmental factors like PFAS on blood pressure, which can be used to develop intervention strategies.
To prevent hypertension and future cardiovascular disease.
Yeah, we're studying.
We're trying to answer some major questions.
1st we would like to assess further high levels of PVS in blog associated with higher blood pressure.
What are the effects of HPV and the overall effect of PFAS mixture on blood pressure?
Also, do certain PFAS have stronger effects on blood pressure and whether the effects of P vas on blood pressure would be different by other factors like age, sex?
Or body.
Measures like body mass index.
Next slide please.
So in our study, we measured 75 in blood samples and measured 50 blood pressure and death stalk blood pressure.
We assessed the associations between each PFAS and blood pressure.
We also calculated the total levels of seven PFAS and assess their effects on blood pressure.
We also considered those PFAS together as a mixture and use more advanced statistical methods to assess the impacts of PFAS mixture on blood pressure.

We also assessed whether the effects of HPV's on blood pressure would be different among male versus female participants, whether the effects would be stronger among certain age groups, and whether the effects would differ by BMI.
Yeah, yeah, we're current analysis.
We included adult participants about about 80 years old for our main analysis. We focus on people who are not taking medication to lower their blood pressure.
We also did a separate analysis among people who are taking medication to treat hypertension.
We will study children and adolescents in our future analysis.
So in our study, we found that higher levels of PFAS were associated with increased blood pressure, PFOS and PFH excess were consistently associated with higher blood pressure, but we also found some positive associations for other P fast, like PFNA and PFOA and PFNA as well.
We're looking at different age groups. We found that the associations between PFAS and blood pressure were generally stronger among younger adults.
When looking at different BMI groups, we found that the associations between P fast and blood pressure were stronger among adults in the middle range of BMI.
We also found that B associations were slightly stronger among male participants compared to female participants.
Our future analysis will use historical reconstructed CRMP fast to better understand the impacts of long term P fast exposure on blood pressure. For our current analysis, although we focus on participants who are not taking anti hypertension medication, we also found some positive associations between P fast and blood.
Pressure, even among those people who are taking medication to treat hypertension.
So our future analysis will use historical reconstructive CRM PFS to study the effects of PFS.
Hypertension diagnosis we will also evaluate the associations between PFAS and blood pressure among children and adolescents.
That's all I have.
Thank you for listening.

Weems, Meghan M. (ATSDR/OAD/OCHHA) 50:46
Thank you very much, Doctor Wang.
Next we have Doctor Abbie Blin of the Massachusetts MSS team, who will discuss metabolic syndrome research.

Bline, Abigail 50:57

Thanks, Megan.

Good evening everyone, and thanks for joining today.

I'm a researcher with Harvard School of Public Health, and I've been working with Doctor Tamara James Todd to lead research on PFAS and Metabolic syndrome.

So metabolic syndrome, which I'll describe a little bit more on the next slide, increases the risk for cardiovascular diseases monocyte chemoattractant protein I, which I'll call MCP one and plasminogen activator inhibitor.

Charter one, which I'll call AI one our substance is our bodies make people with metabolic syndrome tend to have higher levels of these substances and higher levels of these substances have also been associated with increased risk of cardiovascular diseases. Some past studies have found associations between PFS and.

Metabolic syndrome, but others have not, and very few studies have looked at associations between PFAS and MCP one or Pai 1.

Through our research, we wanted to find out whether people with higher with higher levels of PFAS in their blood are more likely to have higher, are more likely to have metabolic syndrome than people with lower levels of PFAS.

We also wanted to find out if higher levels of PFAS measured in blood are associated with higher levels of MCP, one or PII one measured in blood.

Lastly, we wanted to find out if factors like age or sex affected associations between P fast and blood and any of these.

3 outcomes. Next slide, please.

Metabolic syndrome is assessed based upon meeting at least three out of five criteria that are based on waist size, HDL cholesterol, triglycerides, blood pressure and blood glucose. Based upon measurements collected during the study visit, we categorize people as either with metabolic syndrome or without metabolic syndrome. We then.

Use statistical methods to study the relationships between different PFAS measured in blood and other people had metabolic syndrome.

When considering other factors like age, we use similar statistical methods to study relationships between different PFAS measured in blood and MC one, or Pai one. Our analysis was restricted to non pregnant adults who were fasting at the time of their blood draw and did not have Type 1.

Diabetes we excluded people who didn't have blood.

P fasch measurements or measurements of the criteria we needed for assigning

metabolic syndrome status.
And for analysis of associations between P PFAS.
And MC1 or AI1.
We excluded people who didn't have these substances measured in their blood.
Next slide please.
We found that about 33% of the 5300 adults included in our analysis had metabolic syndrome.
We found that people with higher PFOA and PFOS levels in blood were slightly less likely to have metabolic syndrome than people with lower levels of these PFAS.
However, we also found that higher PFOA and PFOS blood levels were associated with higher levels of MCP, one in blood.
In addition, we found that higher levels of P, FOS and PFH excess in blood were associated with higher levels of Pai, one in blood.
Lastly, we found that some of these relationships were differed by age and sex, with stronger associations sometimes seen at older ages.
So because metabolic.
Syndrome can take a long time to develop.
It's possible we could find different relationships between PFAS and metabolic syndrome if PFAS were measured before the metabolic syndrome developed or earlier in the syndrome's progression. Therefore, a next step for this research is to study the associations between the modelled historical historical blood PFAS levels and met.
Syndrome. So thank you all for letting me share these preliminary findings and thanks so much to everyone who made this study possible.

WM **Weems, Meghan M. (ATSDR/OAD/OCHHA)** 54:58
Thank you very much, doctor Blind.
Next we have Selena Phillipson of the California Multi Site study team, who will discuss heart disease research.

CP **Celina Nicole Phillipson** 55:09
Thank you, Megan.
As Meghan mentioned, I am a part of the California site.
I actually began this project as a student volunteer in my master's, so I'm really excited to share some of this work as a part of my pH. D work, so a little bit of

background with PFAS and heart disease.
Heart disease is the leading cause of death in the United States, with over 680,000 deaths in 2023 alone.
Previous studies have linked PFAS to an increase in cardiovascular diseases.
However, other, newer, more recent studies have found no or inverse associations.
What we are trying to learn about PFAS and heart disease, we are trying to learn whether people with higher levels of PFAS in their blood are more likely to report having heart disease which was collected through.
Our questionnaires and if a certain PFAS are more strongly linked to heart disease, both individually and as mixtures.
Next slide please.
A little bit of insight into our methods, we measure PFAS.
The blood during our clinic visits, we collected heart disease diagnosis status from participants during our questionnaire and we then use logistic regression models to analyze the odds between those with and without heart disease. First, just looking at PFS and that case status that we collected and then adjust.
For or including other covariates or as other presenters have mentioned, confounders that are associated with both exposure to PFS and heart disease, and this included age.
Sex, race, ethnicity.
Education, household income, smoking history, alcohol history, waist to hip ratio, which is an obesity measure that is a better predictor for heart disease than BMI and EGFR, which stands for estimated glomalular filtration rate, which is a measure of how well your kidneys are filtering waste from your blood.
Included in our heart disease study are 5824 adult participants who answered our questionnaire and had at least one pfaser measurement.
Next slide please.
What we have found so far is that 528 participants reported having heart disease, which is a nine point, 1% prevalence rate in our cohort. Most PFAS showed no associations to heart disease except pfuma which showed an inverse association.
Our next step for this research include performing a sensitivity analysis with medically validated cases, doing mixture analysis to look at joint effects of PFAS exposure and to use historically reconstructed PFAS serum levels.
To better estimate serum concentrations prior to heart disease diagnosis.
Thank you so much everyone.

Weems, Meghan M. (ATSDR/OAD/OCHHA) 57:45

Thank you very much Celina.

And lastly, we have doctor Yaren Jung of the California Multi Site study team who will discuss obesity research.

Yerin Jung 57:54

Hi I'm yerion.

I'm a researcher with University of California Irvine and I'm happy to share our recent findings on obesity research.

Many researchers had looked at the relationship between, I'm sorry, many researchers had looked at the relationship between PFAS exposure and obesity.

Some lab studies found that PFAS can potentially increase weight. However, human studies show inconsistent findings, which means that some reported that people with higher PFAS levels are more likely or less likely to be obese, or even. Some show that they are not.

Related at all.

So in this study, we're trying to answer 3 questions.

1st is a higher blood PFAS level associated with a higher risk of obesity.

Second, are there sex differences in these associations?

3rd does using different obesity measures lead to a different conclusion?

Next please.

So here we evaluate and measure blood and obesity indicators so as obesity indicators we use body mass index or BMI, which is a famous indicator for obesity that captures body weight compared to the height and the other three indicators can capture body shape, which the body weight cannot.

Solely represent and then as compounders.

As the other presenter said, we consider.

H sex, race, ethnicity, income level, and education attainment into the analysis.

Like in the other studies, we included adults with at least one PFS measurements and with at least one obesity measurement.

And we excluded current pregnant women and every diagnosed with chronic kidney disease to avoid any biases.

Next please.

So what you have found so far is that the people with higher blood levels.

Were very, very less likely to be obese.
These results were similar across different obesity indicators.
Also, there were no significant differences in these associations between males and females.
Our next steps will be looking at joint effects of PFAS mixture into these relationship with obesity and then we're going to analyze this, the same association in the child cohort.
So that's all. And thank you for your attention.

Weems, Meghan M. (ATSDR/OAD/OCHHA) 1:00:33
Thank you very much, Doctor Jung. And to all of tonight's presenters.
The MSS researchers are continuing to analyze these and other data to understand how exposures to PFAS may affect the health of adults and children, along with the research we have discussed this evening, more research is underway on antibodies in children, pregnancy complications, and neurobehavioral outcomes in children.
OK.
Well, we received many questions about this study in advance.
We appreciate each question that you sent in.
Unfortunately, we don't have time to address all of them this evening, but we will start off with given the results of the study, what should people be doing to protect themselves and their children if they live in these affected areas?
Because PFAS are at low levels in some foods and in the environment, completely eliminating PFAS exposure is unlikely, however.
There are some actions you can take.
Avoid eating contaminated fish or game.
Check with your local or state health and Environmental Quality departments for fish or hunting advisories in your area and follow the advisories.
Follow applicable advisories or warnings about agricultural products in your area that may be contaminated with tfas, even though recent efforts to remove PFAS have reduced the likelihood of exposure, some products may still contain them.
If you have questions or concerns about products you use in your home, please contact the Consumer Product Safety Commission.
And I'm going to read a phone number and I will read it twice.
1-800-638-2772.
That's the Consumer Product Safety Commission at 1-800-638-2772.

Reach out to your local health department.
They may also be able to provide additional resources and ways to reduce your exposure.
Closure.
If you have questions about blood testing for PFAS, talk with your healthcare provider about the limitations, risks and benefits of testing.
Atsdr has created resources for healthcare providers and regional pediatric environmental health specialty units, or pesos also offer consultative services to healthcare providers about PFAS and other environmental health topics.
Our second question I'm interested in learning of further research for this and related studies is at risk due to current government cuts.
If so, what will happen to the existing data and will it ever be possible to resume the existing research effort at a future date?
Well, I have good news. The current studies are fully funded and were fully funded.
We do not anticipate the current studies to be affected by changes in personnel or funding.
Next question, if I was tested previously and found positive for PFAS substances in my blood, should I be tested again?
Test results will tell you how much of certain PFAS are in your blood.
They will not provide clear information about possible health effects, pinpoint a health problem, provide information for treatment or predict or rule out future health problems due to exposure. If you do have questions about blood testing for PFAS.
Talk with your healthcare provider about the limitations, risks and benefits of testing.
Next question I was wondering when the final cumulative results of the health analysis will be shared with the multi site study communities?
Study teams continue to finalize analysis and work on completing manuscripts.
We will share these on Atsdr's website as results become available.
And then next, are there ways to remove PFAS from the body?
Excretion is the process whereby substances like PFAS leave the body.
Some PFAS leave the body slowly over time, mostly through urine.
People who have kidney disease may not excrete as much PFAS from their body through their urine as healthy individuals.
Some PFAS routinely leave the body in blood during menstruation.
Those who menstruate may excrete more PFAS than those who don't.

Not.
Some PFAS can leave the body in breast milk.
Those who breastfeed may excrete more P fast from their bodies than those who don't.
All of these factors could affect PFAS levels measured in your blood, while PFAS blood test results can tell you the amount of certain PFAS in your blood, this test result will not provide information to pinpoint a health problem and will not predict future health outcomes. You can talk.
To your healthcare provider about the benefits and limitations of Ppas.
PFAS blood test.
And lastly, where can I learn more about the results presented today?
Study researchers will discuss further during the International Society of Exposure Science and the International Society for Environmental Epidemiology Conference starting on August 17th.
I'm going to provide a website for you to check that out. It's WWW.
Ises ISEE, 2025.
Org again.
That's WWW.
Ises.
2025.org.
This concludes the PFAS multi site study open House, CDC and ATSDR.
Thanks each of you for your participation and interest in this study.
I hope you have a great evening and take care of each other.

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