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FIFRA SCIENTIFIC ADVISORY PANEL (SAP)

OPEN MEETING

DRAFT PRELIMINARY PROBABILISTIC EXPOSURE AND

RISK ASSESSMENT FOR CHILDREN WHO CONTACT

CCA-TREATED WOOD ON PLAYSETS AND DECKS AND

CCA-CONTAINING SOIL AROUND THESE STRUCTURES

December 4, 2003

[8:34 a.m.]

Sheraton Crystal City Hotel
1800 Jefferson Davis Highway
Arlington, Virginia 22202

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Designated Federal Official

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**P R O C E D I N G S**

DR. HEERINGA: Good morning. And Welcome to the second day of our three-day meeting of the FIFRA Science Advisory Panel on the topic of Preliminary Probabilistic Exposure and Risk Assessment for Children Who Contact CCA-treated Wood on Playsets and Decks and CCA-containing Soil Around These Structures.

I'm Steve Heeringa. I am the session chair for this meeting of FIFRA SAP. I'm a member of the permanent SAP. I'm a biostatistician affiliated with the University of Michigan's Institute for Social Research. My individual specialty and contribution here is in the area of population research and study design.

We have a very large and very highly qualified panel joining us to provide expertise in a wide variety of other areas. And I'd like have them, beginning with Dr. Matsumura on my left here, introduce themselves.

DR. MATSUMURA: I'm Fumio Matsumura. I work for the University of California Davis in the Department of Environmental Toxicology. My area of expertise is pesticide toxicology, dioxin and molecular toxicology.

DR. THRALL: Good morning. Mary Anna Thrall. I'm a professor of veterinary pathology at Colorado State University.

DR. KISSEL: John Kissel, University of Washington, Department of Environmental and Occupational Health Sciences. Human exposure assessment.

DR. RIVIERE: Jim Riviere, professor of pharmacology at North Carolina State University. And expertise in dermal absorption and pharmacokinetics.

DR. ADGATE: John Adgate, University of Minnesota School of Public Health. Expertise in exposure analysis and risk assessment.

DR. FREEMAN: Natalie Freeman, adjunct faculty, Robert Wood Johnson Medical School and the School of Public Health. Children's activity patterns and exposures to metal and pesticides.

DR. BATES: Michael Bates. I'm an adjunct professor of epidemiology at the School of Public Health of the University of California, Berkeley.

DR. STEINBERG: J.J. Steinberg, professor Albert Einstein College of Medicine, director autopsy service and working in environmental toxicology.

DR. STYBLO: I'm Miroslav Styblo, associate professor of pediatrics and nutrition, University of North Carolina at Chapel Hill. And my expertise is in the metabolism and molecule effects of arsenic.

DR. CHOU: Selene Chou at ATSDCR, Agency for Toxic Substance and Disease Registry.

DR. WAUCHOPE: Don Wauchope, USDA Agriculture Research Service; pesticide behavior in the environment and risk assessment.

DR. LEBOW: Stan Lebow, USDA Forest Service, research scientist at the Forest Products Lab in Madison. I work in environmental effects of wood preservatives and evaluation of wood preservatives and evaluation of wood preservatives.

DR. STILWELL: David Stilwell, at the Connecticut Agricultural Experiment Station. And I do work on dislodgeable arsenic and arsenic in soils.

DR. REED: Nu-May Ruby Reed. California Environmental Protection Agency. I'm a toxicologist, and I do pesticide risk assessment. And I also teach a class at the University of California, Davis.

DR. RYAN: Barry Ryan, Emory University. I'm a professor in the Department of Environmental and Occupational Health. And my expertise is in multimedia exposure assessment.

DR. MACINTOSH: David MacIntosh. I'm a senior scientist with Environmental Health and Engineering. And I work in the area of human exposure assessment for chemicals and microbes.

DR. FRANCIS: I'm Marcie Francis. I'm a senior research scientist at Battelle, specializing in human exposure assessment and exposure modeling.

DR. HATTIS: I'm Dale Hattis. I'm a research professor at Clark University.

DR. PORTIER: Ken Portier, I'm associate professor of statistics at the University of Florida. My expertise is in environmental sampling and statistical issues in PRA.

DR. MACDONALD: Peter Macdonald, professor of mathematics and statistics at McMaster University in Canada. I have general expertise in applied statistics.

DR. HEERINGA: Thank you, members of the panel. At this point in time, I'd like to turn it over to our designated federal official for this meeting of the FIFRA Science Advisory Panel, Paul Lewis.

MR. LEWIS: Thank you, Dr. Heeringa. I'd like to welcome our panel members back for the second of three days of this important and challenging meeting. And again welcome to the public for being here and becoming actively involved in listening to deliberations that will be occurring later on today and the public comments beginning this morning.

As I mentioned yesterday, the FIFRA SAP operates under the guidance of the Federal Advisory Committee Act. As this is an open meeting, all materials for this meeting will be available in our public docket and major substantive background documents are also available on our web site.

At the conclusion of the meeting, we will publish a report that serves as meeting minutes that summarizes the Panel's deliberations that will be occurring over these past three days. And the report will be available in approximately six weeks, made available both in our docket and also on our web site.

Thank you, Dr. Heeringa.

DR. HEERINGA: The first item on our meeting agenda this morning is an opportunity for the staff, Mr. William Jordan, Bill Jordan, of the Office of Pesticide Programs to respond with clarifications and reactions to the proceedings of yesterday's meeting. Bill.

DR. JORDAN: Thank you, Dr. Heeringa.

There are two topics that came up in the conversations yesterday that we thought would be helpful for the EPA to try clarify. They are the approach that the Agency is using to the arsenic cancer slope factor and relative bioavailability.

Before I turn the microphone over to Jonathan

Chen to speak about that, I'd like to say a little bit about each of those topics to frame his comments.

With regard to the arsenic cancer slope factor, we have purposely chosen not to ask this panel questions about the methodology that we used to derive the cancer slope factor because, as we've indicated in the documents and in the presentation yesterday, we are at the Agency actively studying the report from the National Research Council on that subject and have not made a decision with regard to whether to make any changes in the cancer slope factor that we're using to estimate the risks of exposure to arsenic.

We do think, however, it would be useful to explain a little bit more clearly in this public forum the methodology that we used to derive the cancer slope factor. It is the methodology that was used in the Office of Water Risk Assessment. And we have, as we've indicated, simply used that same number. And because we're using that number, we're describing the methodology used by the Office of Water.

The second topic is the questions related to the relative bioavailability of arsenic and the calculation of our dose or exposure matrix on the toxicity side as compared to the dose or exposure matrix on the exposure side for purposes of the risk assessment. And it is our position that the calculations that are derived from SHEDS have been properly adjusted to be comparable to the values with which they are being compared, derived from the toxicity data that serve as the hazard benchmark for purposes of the risk assessment. And we'll explain a little bit, again go over, and we hope this time clarify for everyone why we believe no further adjustments in the LADDs or the ADDs are necessary to achieve comparability in those different values.

So with that introduction, let me ask Dr. Chen to go ahead with the slides that we've prepared on the arsenic cancer slope factor.

DR. CHEN: Good morning. My name is Jonathan Chen. And I'm a toxicologists with the risk assessment in the science support branch in the microbial division in the Office of Pesticide Programs.

First, I'd like to point out that the cancer risk assessment is derived from the Office of Water's drinking water risk assessment. It will be use the CCA risk assessment. The risk estimate in the drinking water risk assessment is taken from a published paper by Morales, et al., in year 2000. Morales, et al., fit a variety of dose response models to lung and bladder cancer data from analysis endemic region of Southwestern Taiwan.

In the models of Morales, et al., EPA used estimates from a poison regression model fit with no comparison population. And based on the risk derived from this model, risk was assumed to increase linearly with dose from zero to effective dose central estimate at which 1 percent of the population is affected by the CAMCO. It's called ED01. The slope of the line (inaudible) from ED01 to origin was calculated and the use of the cancer slope factor for the cancer risk assessment.

In year 2000, EPA drinking water risk assessment has two sets of the risk estimate. For the higher set of risks, assumes drinking water consumption in Taiwanese population is 3.5 liter per day from male and 2.0 liter per day for female. For the lower set of risk, EPA assumed that population in Taiwan consumed an addition of 1 liter per day in cooking due to rehydration of rice and sweet potato. And a further 50 microgram per day of arsenic directly from their food.

For this risk assessment, an oral cancer slope factor of 3.67 per milligram per kilogram per day was used. This is a mean slope factor derived from the higher risk approach for both lung and bladder cancers.

In April 2001, EPA charged the NRC to review the risk analysis used to support the revised drinking water regulation in light of the studies published since the 1999 NRC report. The NRC released its updated report in September 2001. In the report, NRC has many different comments about the drinking water risk assessment. In addition, based on the same data set, NRC also included a risk calculation in the report based on the same data set, that is Southwestern Taiwan data set, but with different model with comparison group and different drinking water rate, different dietary content of arsenic, et cetera.

The cancer slope factor number is different from the Agency's drinking water risk assessment. We can say the difference is due to the different ways to interpret the same data set. The Agency is currently considering the best way to address all of the NRC's 2001 recommendations. Based on the Agency's considerations of these recommendations, the current proposed cancer potency number may change in the final version of this risk assessment.

And this is the end of my presentation. Well, the detail of the how the number is derived can be seen in Appendix A of the Risk Assessment.

DR. HEERINGA: Dr. Chen, is it a fact that the EPA's decision, if and when is it made and if it's applied to the risk assessment, that there will be a clear determination of that rate made public?

DR. CHEN: Yes.

DR. HEERINGA: Are there any questions at this point from the panel? I'd like to keep it fairly succinct. But if there's any points of clarification. Thank you very much, Dr. Chen, that's very helpful in terms of clarification.

DR. BATES: I have comments, but I don't have clarifications.

DR. HEERINGA: Can we keep those in the context of the questions.

DR. BATES: Thank you. Just to repeat something I asked yesterday about. In the materials supplied by the industry, they suggest that there's a extra factor of 2 in the EPA's calculation. Do you have any comment on that?

DR. CHEN: Well, it's a point that because the Taiwan data set is basically based on the mortality from the different cancers from both lung and bladder cancer. And then the most difficult part is how to interpret data. If we assume the Taiwanese drink more water, than the cancer risk would drop. So if the Taiwanese -- no, it would rise or something.

So this kind of thing becomes very, very complicated. So this is the reason that the working group is working on that. So if we change, if you go to the NRC report, the drinking water rate is lower than the ones used in the office water risk assessment. And then what would be the best way to interpret the data set is very important.

MR. JORDAN: I guess I would say here as we understood the comments from the industry, it had to do with a different set of assumptions rather than a mathematical error as some folks had tried to characterize it. And we've tried to explain in the appendix material, that Dr. Chen cited, set by set how we derived the cancer slope factor. And we've double-checked that and don't believe we've made an error. But that is certainly up to the Panel to comment on if they choose. Thank you.

DR. HEERINGA: Thank you very much. And I think we'll probably have more information upcoming this morning in terms of other positions. Dr. Hattis.

DR. HATTIS: Yes. The NRC document, as you indicated, is over two years old. And they considered specifically all of those issues. In addition I believe they projected, based on transporting the relative risks to U.S. background rather than using Taiwanese --

DR. CHEN: Yes.

DR. HATTIS: -- background where it seems less than perfectly accurate for the U.S. application.

DR. CHEN: Yeah.

DR. HATTIS: Do you have any specific objections to the NRC methodology that caused you not to at least disclose the magnitude and direction of the change in your risk assessment that will result from those numbers?

DR. JORDAN: Dr. Hattis, we're, I think, fairly candid in our documentation of the risk assessment here that we are reviewing the cancer slope factor. It is an issue that is not confined just to the pesticide risks, the risk for using CCA and treated wood, but really affects a number of different programs across EPA. Because the matter is still under active discussion within the Agency, we didn't think that it was appropriate to put that in front of this panel at this time and the reason why we have tried to steer clear of that part of the risk assessment.

DR. HEERINGA: Thank you, Mr. Jordan.

At this point, you were going to do a little bit on the bioavailability?

DR. CHEN: To clarify some issues being discussed here today, I'm going to present the slide. It's about relative bioavailability of chemicals of concern.

This risk assessment, I would like to make sure some of the terms that we're using are clearly defined. The absolute bioavailability is a ratio of the amount of chemical absorbed compared to the amount of chemical ingested. For example, if 100 microgram of chemical X dissolved in drinking water were ingested and a total of 90 microgram entered the body, the absolute bioavailability would be 90 percent.

The relative bioavailability is a ratio of the absolute bioavailability of a chemical in the test material compared to the absolute bioavailability of the same chemical in the reference material. For example, if the absolute bioavailability of chemical X dissolved in drinking water is 90 percent and the absolute bioavailability of X contained in soil is 30 percent, then the relative bioavailability of X in the soil versus water would be 33 percent.

Therefore, if we are talking about the relative bioavailability of soil versus water, it would be the percentage of the chemical of concern. For example, in organic arsenic absorbed into the body of a soil-dosed animal compared to that of an animal receiving a single dose of arsenic in aqueous solution.

Because all the hazard endpoints selected for the oral exposure in the risk assessment is based on the exposed concentration not absorbed dose, in the risk assessment it is assumed the absolute bioavailability of 100 percent for the oral exposure route.

Now, why is relative bioavailability soil versus water and or wood residue versus water need to be discussed. The reason is that all of toxicity endpoints selected in the hazard assessment are based on the chemical of concern is in aqueous phase. To adjust exposure of the chemical in soil, the relative bioavailability is soil versus water and or wood residue versus water is required to define the chemical bioavailability in the media of concern relative to water.

This is the end of my presentation for relative bioavailability.

DR. HEERINGA: Any questions of clarification or fact from the Panel? Are we comfortable with the incorporation of these factors into the exposure and risk assessment equations?

DR. HATTIS: I am. I'm satisfied that they've done it correctly.

DR. HEERINGA: Dr. Styblo.

DR. STYBLO: The critical terminology here is the same arsenic species --

DR. HEERINGA: Right.

DR. STYBLO: -- which appeared in the first slide but didn't appear in the fourth or fifth slide.

DR. HEERINGA: And I think that --

DR. STYBLO: That's an important issue. Another thing is I think we are all comfortable with the definition of relative and absolute bioavailability; however, the critical part comes when we need to decide how to actually monitor or how to determine. In other words, what would be the appropriate methodology for determination of bioavailability. And that's another question we will be talking about later.

DR. HEERINGA: We're comfortable with the terminology. We're comfortable with incorporation into the exposure and risk assessment formally. But we do have this issue of the complex or elemental form.

Yes, Dr. Wauchope.

DR. WAUCHOPE: I guess our group will be addressing this to some extent in the comments. But we have been doing a lot of discussing about this. And I guess one point we'd like to make maybe now is that the Casteel experiments used these terms, absolute and relative. They make a measurement of a comparison of two uptakes based on urinary excretion and assume that that is the measure of the two absolute bioavailabilities. That's simply not true.

DR. HEERINGA: Thank you very much. And we will have plenty of opportunity, I think it's Issue 8, with regard to the complexes. Dr. Chen.

DR. CHEN: Well, I just want to make it clear that in the SHEDS model, the ADD already adjusted with the relative bioavailability. So the hazard part, we are not going to do any change.

DR. HEERINGA: Very good. Thank you. Thank you very much. I think that's a very useful beginning to our day.

At this point in time, we're going to end our presentations by the EPA staff. I thank them very much. I've been and a number of panel members have been a part of these discussions on probabilistic risk assessments for several years, and the qualities of the presentations, the organization of the material has improved tremendously as we all learn on this process. And I thank the staff of the EPA and the presenters. I think they are very clear and organized presentations.

And now we can get on to public commentary and on to discussion of your specific questions over the next two days. At this points in time, I would like to open the period for public comment. We have scheduled for today -- we actually had two public commentors yesterday, and we spent a little bit of extra discussion time on their presentation because nobody else was quite ready to carry on at that point. Today we have scheduled -- let me do a quick count. I nine public commentors and presenters, and we're scheduled now noon or slightly after noon to complete this work.

We'd like to try to keep things on schedule. I don't want to cut things short in terms of useful and productive discussion or fact-finding discussion that's needed on the part of the Panel. I guess would encourage everybody to do two things. When you come to the mike, be sure to state your name and affiliation and for panel members also. And then with regard to comments and discussion, if we could limit it to questions of fact or scientific exploration and then comments we'll have plenty of time in the next day and a half to incorporate specific comment and points of view in our response to the questions from the EPA and general response to the exposure and the risk assessment.

So at this point in time, I'd like to begin the morning's presentations, public comments. And I believe that Dr. Barbara Beck is the first public presenter, public commentor, from Gradient on behalf of the Wood Preservative Science Council. Dr. Beck. And indicate that you have about 30 minutes scheduled.

Members of the Panel, Mr. Lewis and I are trying to locate copies of these slides for you.

DR. BECK: You do have copies of my presentation. And I got a little creative and didn't change it procedurally so that the printout you have is going to be difficult to read. We will be providing the panel of all the presentations from industry which will be more legible than the version you have now.

DR. HEERINGA: Thank you. That will be very helpful.

DR. BECK: This is an overview of my comments today. I'm going to talk broadly in a number of areas. I have comments on the hand-to-mouth specific input parameters and specifically the hand-to-mouth pathway with respect to a number of issues including a potential for over estimates in the selected dates as well as the applicability of the data sets that were used.

I should note that I do think that EPA really did do a very good job with the limited data set for the situation that we're trying to model. And I think that this really is a very innovative effort on EPA's part. But a lot of my questions relate to concerns with some of the underlying inputs and some model structure in terms of being really able to replicate the activities of kids on the playsets.

I'll have comments on the CHAD diaries. We'll hear additional comments from my colleague, Dr. Barbara Peterson, who will discuss a bit of how uncertainty was characterized. And while I know that the mission here is not to discuss the slope factor, I do have some very brief comments.

There's also additional comments that we have provided to the Panel. And I should note, we also have two publications that have been accepted. One relates to issues having to do with arsenic nonlinearity and dose response. And I believe that that has been provided to you. That will be coming in "Toxicology and Applied Pharmacology" in January. And our deterministic risk assessment has been accepted in "Human and Ecological Risk Assessment." You don't have a copy of that. We just heard recently that it was accepted with revisions.

However, once we've made those revisions, we'll certainly be willing to provide Panel members with copies of the manuscript. If you don't have copies of the other manuscript, please tell me and we'll provide that.

I would also say that we've got some overall comments regarding the risk assessment, places that we find that the analysis is not always transparent. It's been difficult for us in some cases to understand exactly what was done or to duplicate calculations.

To get to the issue of dislodgeable residue, and this is in response to a number of the issues that have been proposed to the Panel regarding specific inputs, regarding characterization of uncertainty. This is clearly one of the key issues in understanding and using the model. It is the most important pathway in terms of exposure and risk. And of all the pathways, it's probably the most complicated. There are more than 10 parameters involved.

We go from a loading on a deck to on the hand, to a certain fraction on the hand, to a certain number of hand-to-mouth contacts, and then some removal from that hand-to-mouth contact, then potential for reloading. This is all linked to differences in activities pattern.

So it's a very complicated pathway. And we briefly believe that a number of parameters, in terms of the underlying data, that there's really -- the data that we would like to have to really model this pathway is either not available or there are only a few numbers of individuals, a few number of children, for example, who are engaged in the activities we'd like to model.

I'll comment specifically on hand-to-mouth frequency, the dermal transfer fraction which is also know as "salivary removal efficiency." How much of the hand actually contacts residues on the surface? How much of the hand? Is it the fingers? Is it the whole hand? What's the intensity of the contact when it is inserted into the mouth?

And one important feature is the potential for reloading and unloading. Obviously, reloading can only occur outside when the child is on the deck. There's a potential for unloading inside, of course, unloading from bathing or from sucking or licking. There's also potential for unloading even in outside activities that we don't believe the model adequately addresses.

For example, children at playgrounds will not be on playsets the whole time as was discussed yesterday. They'll be at playsets. They'll be in sand boxes. Maybe they'll be playing soccer. There's a potential for unloading activities that occur even when outside in activity near playsets.

We believe that there is also a need for a benchmarking analysis or analyses such as biomonitoring studies. Or one can look at individual pathways to determine whether the output is realistic. And we have made comments to EPA about the hand-to-mouth transfer pathway. And if one were to apply this to soil ingestion, would one get a soil ingestion rate that is consistent with what is measured because that is one parameter that, although there's still ongoing debate about appropriate distribution for soil ingestion rates, we do have some data from real kids from different locations in the U.S.

We understand EPA has done such an analysis. It's a bit difficult for us to evaluate it because we haven't seen all the specifics of that analysis.

I'd like to talk about the hand-to-mouth contact frequency. It's clearly an important parameter. EPA does have a number of original researchers who are now working at EPA in this area who have really advanced the field.

Now, when you look at, and I'll give you some information comparing some of the actual studies, the hand-to-mouth frequency varies for a number of reasons. Active outdoor play results in less hand-to-mouth activity. This is the work of Dr. Freeman. I should say it's an area that we don't have a lot of data and particularly we have a very limited data with kids on playsets. Age of child has an impact with younger children, of course, having more mouthing activity peaking probably around 18 to 24 months. Interestingly, that's probably not as significant overall in impact as the actual activity that the children are engaged in.

The intensity of the contact is important. This is really more how one categorizes the hand-to-mouth frequency. For example, contacts may be very casual, just touching a finger near the mouth. Or they may be intense in which a finger or hand is inserted into the mouth. That is really, of course, what we care about in terms of looking at CCA-treated wood exposures.

Some studies include both casual and intense contact. Some studies include only intense contacts. It's important when one develops these input distributions, we believe that the hand-to-mouth contact frequencies is most appropriately based on intense contacts because those are the ones that are going to result in the transfer to the mouth.

It's difficult for us to understand the exact logic that was used in developing the distribution that EPA provided for hand-to-mouth contact frequency. We believe there's more explanation that's required in order to better evaluate that.

As I mentioned earlier, there's a potential for reloading which, of course, is going to be dependent on activity. And for unloading. And it's not clear that unloading in particular is adequately addressed in the model.

As I mentioned earlier, we believe it's important that when you think about hand-to-mouth activity that it be matched appropriately with the intensity of the contact. If it's a study that used casual contacts and the casual contacts aren't going to contribute significantly to intake, of course, as the intense contacts.

Right now the model does not allow for separate distributions for hand-to-mouth contact frequency as a function of indoor versus outdoor activity. That could be accomplished by the recoding of the model. We think that that could be a significant improvement in the model to allow for activity specific distributions for hand-to-mouth contact frequency.

And again as I said earlier, how the data sets were combined to yield a final distribution is unclear. The data sets do appear to have different relevance to the situation we're modeling. This adds uncertainty to the analysis.

The next two slides summarize very briefly the hand-to-mouth studies. EPA's estimate was 8.45 contacts per hour as a mean based on combining results from the studies noted. It appears that the studies include both indoor and -- well, the studies do include indoor and outdoor activities. And we believe that was part of EPA's logic was to include indoor and outdoor activities in that there is a potential for continued exposure indoors from a residue that is on skin surfaces.

On the other hand, it really would be more appropriate to consider that there is an indoor specific hand-to-mouth distribution that does not allow for the potential for reloading.

Looking at the studies -- if you could just go back to the previous slide -- I call your attention in particular to the Freeman study. It's a very limited number of children. But I think it is interesting to note that of that we learned that four children were actually engaged in activity on playsets, the hand-to-mouth contact is lower by a factor of about 3 compared to a value used by EPA. And given that the outdoor activities where most of the exposure would occur, we believe that it's important that the hand-to-mouth frequency be matched appropriately to the activities that children are engaged in.

One other study that was used in the EPA's analysis is the Leckie study which does include outdoor, I note that recently tables regarding the Leckie study which does include outdoor children. And I note that recently tables regarding the Leckie study are now available on the web. Unfortunately, we have very limited information about the actual study itself and what kinds of environments the children were in where the videotaped studies were conducted and what types of activities they were engaged in.

The hand-to-mouth dermal transfer fraction is also known as salivary removal efficiency. And this relates to once a child has finger or hand in their mouth, how much residue do they remove. And it's derived from a study in which pesticide residue on skin surfaces were removed with moistened gauze. So it's a fairly intense removal process. And while it may be applicable to a thumb sucker, it's clear thumb suckers are very efficient at cleaning their thumbs compared to the rest of their hands, it's unlikely to be realistic for the more casual and less intense contact. And interestingly, this removal efficiency is greater than what it would be accomplished by hand washing where the intent is really to remove material on the hand and comparable to what's removable by bathing. So we believe there's a potential for an over estimate here. It's a bit difficult to quantify.

I also did want to just note that as an aside the fact that the bathing removal efficiency is not 100 percent. It would be useful to have some better understanding as to what this means as far as accrual of residue on skin surfaces, and does this mean at some point there's residue accrual so it almost reaches a steady state situation. So I think that it might be useful to look at what significance of a bathing removal less than 1 in terms of the overall material deposited on skin surfaces.

The fraction of the skin contacting the residue on hard surfaces is based on soil studies and involved kids playing in soil. And there are questions, again, I think overall there's a theme of not having data that is really as applicable to the situation we're modeling as we think is necessary. In some cases, it's the consequence of limitations of the underlying data. We just don't have much information of kids on playsets. In other cases, we believe that there are alternate selections that could have been considered that might have had an impact on the risks.

In this case, the transfer was relatively high, about 70 to 80 percent on average. Now, this is for soil which is a malleable material and kids were intensely playing in the soil. And there are other studies. And I call your attention in particular to the study by Brouwer where the loading of a white fluorescent powder onto hands was measured by pressing onto, multiple presses, onto glass surface. And in that case, the typical contact frequency was more on the order of -- that study, I believe it was 20 percent or so. And it reached a semisaturation after a certain point.

And interesting, when the hand was pressed on a uncontaminated surface, there was unloading of material. Again, this supports the need for the model to consider unloading. In this case the material loaded on the palm of the hand was about a fourth of what is used in EPA's model. Now I recognize while much of children's contact on playsets will be with the palmar surface, some of that will be with the other side of the hand. However, that's likely to be much less of a lower magnitude than what would accrue on the palm surface. So this is another parameter that we believe needs to be reevaluated and the range of intensity of contacts and using data on contacts with hard surfaces needs to be considered in the model.

Well, just looking at the hand-to-mouth pathway here, and we've just looked at four parameters, we haven't looked at the diary studies, looking at contact time although I have some comments on that in a few minutes. We believe overall there's opportunities for over estimates for three of these parameters and possibly for the fourth one. A Hand-to-mouth contact frequency considering activity specific contact, salivary removal efficiency, considering the fact that the contact is not always going to be a sucking type contact. The efficiency is not likely to be comparable to bathing for every contact. Fraction of hand contacting residue needs to consider differences in contact with flat surfaces with children engaged in the kind of activities that are common on playsets. And then the fraction of the hand-to-mouth, we can't evaluate very readily. We do have the data from the Leckie study which does specify fingers, hands, how much is inserted into the mouth. But we don't have the documentation as to what the activities were that the children were engaged in at the time, so it's difficult for us to evaluate.

We think it would be useful to look at not only modifying the single parameter in time, but what would be the implication of considering three changes here, for example, at once. Or what would be the implications of considering modifications to the model structure to allow for difference in contact frequency as a function of indoor versus outdoor activities. And in particular, outdoor activities on playsets.

The CHAD diaries will be commented on in more detail by Barbara Peterson of Exponent. But when one looks at the CHAD diaries and looks at what is potential playset contact, there are two broad questions. One is a number of the activities listed there are not activities which we believe would be likely to bring children into much contact with playsets such as medical care and travel.

Now, even activities at parks includes both activities on a playset as well as activities off the playset, in sand boxes, engaged in other activities, where there is a potential for a reduction in the contact time as well as a potential for unloading of material on the hands.

The CHAD diaries are based mostly on single-day diaries from children. There are a number of children in the data set who did have two-day diaries or three-day diaries. As you look at more diaries per child, the mean number of hours engaged in outdoor time is reduced. So considering this, plus we have the question of eight diaries from different subjects being used to mimic a single child's longitudinal activity profile, it's not clear that this uncertainty in this parameter has been adequately characterized and is there also a potential here for an over estimate because of the nature of the activities. Maybe there's some inappropriate activities included as well as the impact of multiple diaries.

We heard yesterday that there is a study in Southern California looking at a number of children and looking at their diaries. And the conclusion of that was that eight diaries are sufficient. I think it would be very important to review that underlying -- to review that analysis to really assess confidence which we can conclude that eight diaries are sufficient.

Now, I know we're not going to go into detail on the cancer slope factor. And you'll see that I have -- we have a much detailed analysis in the publication that will be coming out in January in "Toxicology and Applied Pharmacology." But I did have a few brief comments. The first set here relating to general arsenic carcinogenicity. I think that the evidence, when one considers possible mechanisms of action, is fairly strong that it's likely to be a nonlinear dose response model.

Now, we don't know the precise mechanism of action. And it is clear that arsenic does cause tumors at different sites. And it's likely that the plausible mechanisms may vary depending on the tumor site. And that they're not exclusive. There was probably interaction of the mechanisms. Possibilities include changes in DNA methylation patterns, the work of Walkie (ph.) showing that hypomethylation with longer term exposure can cause changes in gene transcription, inhibition of DNA repair, chromosomal damage, change in transcription factors. These are all some of the plausible mechanisms, some of the changes that we see in response to arsenic exposure.

It is clear that arsenic does not interact directly with DNA. It is not a point mutagen. And there's also continuing research on arsenic. Dr. Styblo, I'm sure, is very familiar. Every week there's two or three new articles out on methylated arsenic species or arsenic mechanisms of action. It's somewhat overwhelming at times. But we do have evidence that the metabolism of arsenic yields methylated trivalent species. Their role in chronic toxicity is still undergoing discussions because there are important kinetic issues that need to be addressed. But it's clear that they are very potent cytotoxins, very potent sources of oxidative damage.

Again all of these are likely to result in nonlinear dose response models. And particularly with oxidative damage, cells having potential -- cells having antioxidant mechanisms becomes another important element in terms of nonlinearity.

Another consideration is that in overall, and Dr. Frost will discuss this in more detail, the U.S. studies do not provide evidence of arsenic carcinogenicity in the U.S. Particularly, we will hear about the study at the Tacoma smelter of children exposed. But we also have studies from Lewis. We have other studies that, taken as a whole, do not provide evidence that the U.S. exposures are associated with carcinogenicity.

We think that this is an important perspective that needs to be discussed in terms of providing perspective to the public what a few micrograms of arsenic intake means in terms of overall public health.

There is very strong evidence that nutritional and dietary factors affect susceptibility to arsenic. People who were more poorly nourished, people who have deficiencies in beta carotene clearly did demonstrate increased susceptibility to arsenic in a number of studies outside the U.S.

NRC acknowledged this. They said it was difficult to include quantitatively in risk assessment. Again, I think it's an important aspect that needs to be discussed in terms of providing a perspective on what these exposures mean for the U.S. population.

As far as the calculation, we are -- we've had discussions with Dr. Chen, and we'll probably be talking with him more next week. We're still trying to reproduce the 3.67 number. And it's been just all the activity involved in getting ready for this meeting, we've been unable to sit down and have a long conversation. But he's been very accommodating. And we will continue that discussion because we are unable to duplicate that 3.67 value.

However, I think whether it's 3.67 or 1.8, and I'm sure we'll resolve this discussion. The important question is that there's a .3 value and a 3.67 or a 1.8 value. These are derived, as Dr. Chen discussed, the lower value from assuming that people in Taiwan were exposed to arsenic in drinking water, in water used in cooking in rice and yams. And to the extent that you have more arsenic exposure, that of course reduces the potency.

And Dr. Hattis yesterday discussed this. This really isn't like a Q1STAR or a 95 percent upper bound estimate on the slope factor. That's correct. It's not like -- almost all of the human carcinogens do not use a 95 upper confidence limit on the slope. That is seen with the animal carcinogens. There's only one human carcinogen where an upper confidence was used.

However, one could still consider this is the .367 or 1.78 is more of an upper bound estimate. We believe the .3 is really a more accurate estimate in that it more accurately reflects what the exposures were in the Taiwan population. I think that this needs to be looked at. And I think also considering arsenic nonlinearity we strongly recommend that a margin of exposure analysis be included in the analysis to provide a fuller representation of our arsenic carcinogenicity.

My last two slides relate to the characterization of uncertainty and how the model is evaluated. The uncertainty was characterized focusing particularly on quantitative matrix, looking at the applications of a single parameter, increasing it by a factor of two or decreasing it by a factor of two. But we think there are other important sources of uncertainty which were discussed yesterday but I think need to be highlighted more because, otherwise, one is left with the factor of 3 to 4 statement which I think really understates the uncertainty, particularly when considering the applicability of the underlying data for the situations we are trying to model, the relevance of studies that were selected to the scenarios being modeled.

And as I said, in some cases, it's just a function of the data not being available. We believe there are ways to fill those data gaps. That will be described more by Dr. Frost. In other cases we believe that there were either alternate studies that could have been used or studies could have been used in different manner.

The final point is another way the model was evaluated was by comparison with deterministic risk assessments. One question here or comment is that not all of these risk assessments are comparable in terms of the quality or in terms of the underlying data sets that were used. And a comparison was made to the Gradient 2001 risk assessment which is on the web site. However, since that risk assessment was published or placed on the web, we have revised it using the more recent data that has been used in EPA's risk assessment. We revised it using RTI data for surface loadings and hand loadings. We revised it using the Casteel studies for bioavailability.

And if you go to the next slide, in fact, if we examine the comparison of our RME risk assessment numbers using the new data versus the 95th percentile of the SHEDS estimate, the results are more discrepant disrepresent than were presented in EPA's analysis. The manuscript has just been submitted, so we were unable to provide it to EPA until now. But we believe that this needs to be considered if one way the model would be evaluated would be comparison of a model that used the same underlying data set.

However, I think ultimately we want to consider alternate ways to assess and validate the model such as the use the urine biomonitoring studies or the use of videotaped studies that more accurately reflect children's activities where they're on playsets.

Thank you very much.

DR. HEERINGA: Thank you, Dr. Beck. Very interesting. A couple of points, you mentioned two papers, one on the nonlinear dose response paper for arsenic cancer slope factors; and the second paper which you mentioned was, I think, under review or accepted for publication.

DR. BECK: It's accepted with revisions.

DR. HEERINGA: Okay. I guess when it's ready, instead of sending it directly to the Panel, would you be willing to provide it to Mr. Lewis for distribution to us?

DR. BECK: Oh, absolutely. We will provide the nonlinearity one right away because that has been accepted. And as soon as the other one is accepted with the revisions, as soon as we make the revisions, we'll submit that to Mr. Lewis right away.

DR. HEERINGA: And I know that you and Dr. Chen are working on the clarification of the computation of this sort of high end 3.67 value. I think if that computation is clarified, it would be beneficial to everyone potentially to have that, a short write-up and posted on the web.

DR. BECK: Yes. We'll certainly provide that.

And I say, actually, you do have this. We submitted comments to the CPS -- no, to EPA regarding methodologies for calculating the cancer risk factor, comparing the NRC versus the EPA methodology. Do you have that?

DR. HEERINGA: Mr. Paul Lewis.

MR. LEWIS: I think, were those comments provided directly to the SAP docket, or were they provided to another docket? If not, if you can give me the comments, I'll distribute them to the Panel for this meeting and also make them available to the SAP docket.

DR. BECK: I think we meant to submit it to you. And I'm not -- did we submit that?

DR. SHARMA: This is Raj representing the industry. Those comments were part of the file that was sent to you, Paul. You remember the big batch of files.

DR. LEWIS: For the Panel's interest, the large binder that was given about a month or so ago as part of those comments.

DR. SHARMA: And I think Dr. Bates has seen them because he's referred to them a number of times.

DR. BECK: Thank you, Raj.

DR. HEERINGA: Thank you very much. We're right on time with this presentation. Are there any questions from the Panel? Yes, Dr. Freeman.

DR. FREEMAN: In your last slide where you point to a 10 percent or 10 times and 15 times difference between your revised assessment and EPA's, was that for the 95th percentile?

DR. BECK: Yes. We compared EPA's 95th percentile. And ours, we used a reasonable maximum exposure approach because it was a deterministic risk assessment. And so we felt that we were comparing like versus like by doing it that way.

DR. FREEMAN: Did you do something to compare the mean or median values between the two?

DR. BECK: Our publication focuses just on the 95th percentile. We haven't looked at the mean. We can certainly go back and calculate a mean value with the new parameter.

DR. FREEMAN: That might be interesting.

DR. HEERINGA: Dr. Hattis.

DR. HATTIS: You've undoubtedly read the NRC 2001 document by this time. I'm going to ask you the same questions as I asked the EPA folks. Do you have any specific criticisms, objections to the way they analyzed the Taiwan data and also the newer data from Chile?

DR. BECK: Yes. And that's actually -- in that document that I just referred to, we go into that. So some of the concerns we have were using a relative risk versus an absolute risk model. Another was using the control population which forced a supralinear dose response model. And I think we may have had comments about how intake was calculated. So that's detailed in that set of comments.

The other general comment that we had was that that slope factor -- well, it's not a slope factor. But you can translate it to a slope factor, and it gives a value of about 23 which is what CPSC calculated. That is not consistent from the Lewis study from Utah that the cancer rate you would expect using a slope factor of 23 is not what you see in Utah.

Now more recently, and Dr. Lamm will be presenting this later, there's other evidence that would indicate that slope factor of 23 is too high for U.S. populations looking at bladder cancer.

DR. HEERINGA: Dr. Reed.

DR. REED: Coming back to the your conclusion about the 10 to 15 fold difference from the final estimation of exposure, can you identify some key parameters that are different from what was used in USEPA's analysis that could contribute to the differences?

DR. BECK: I think that one important difference was that the way we looked at hand-to-mouth frequency was what we call an empirical model. EPA uses a mechanistic model based on how the transfer is believed to occur from hand-to-mouth activity. We used an empirical model which is a model that was used by CPSC which is based on soil ingestion studies in which we know how much soil ingestion children -- how much soil children eat a day. We know how much soil is on their hands. And then you can infer from that if they eat, for example, 50 milligrams a day and they've got 100 milligrams on their hand on average that means half a hand load is transferred to the body over the course of a day. So I think that that is one of the main differences is the use of an empirical approach versus a more mechanistic approach. I believe that the hours out doors are probably not that significant in terms of the overall. We use, I think, a fairly high end number of time outdoors. We can look at that in more detail. I know at the very least it's the consequence of the mechanistic versus the empirical mode.

Some of the other factors, bioavailability, we use the same parameters. The soil ingestion rates that EPA used are higher than ours; although I think that, given that soil ingestion is not such an important contributor to risk. And I guess, finally, the fraction of the body surface that is contacted by residue was another important difference. I'd say hand-to-mouth transfer and fraction of body contacted by residue.

DR. HEERINGA: Thank you, Dr. Beck. And thank you to you and your colleagues for the materials you submitted.

Yes, Dr. Ozkaynak.

DR. OZKAYNAK: I just wanted to remind the panelists that we have received a number of these comments during the review process from the representatives of registrants including those mentioned today by Dr. Beck. And the staff went through that and responded to a number of those issues in the addendum document especially with regards to the frequency of hand-to-mouth contact. The Comments No. 31 and 40 summarizes that comment and also the Agency's response.

In addition to that, actually in the probabilistic exposure report, Table 28, page 106, looks at the sensitivity of the results to reducing the hand-to-mouth frequency by a factor of two and as well as increase being by a factor of 2. That analysis that's shown in that table in the report, as well as the additional analysis that staff performed, showed that the results do not really change that much, less than 6 or 10 percent at most.

So the results are fairly robust with regards to those assumptions. With regards to the new exposure and risk assessment that Dr. Beck referred to here, there was a difference of 10 to 15 fold was mentioned. And I guess our question or comment may be on that issue will be are there differences in the definition of the target population with regards to the population of children that's been quantified in terms of their exposure. For example, the Agency looked at the CCA-exposed population only, not the general population. For example if a general population exposure have been simulated, then it's understandable those estimates will be lower than the estimate that we're presenting at this hearing.

DR. HEERINGA: Dr. Styblo.

DR. STYBLO: Just a short technical correction for the record.

Dr. Beck said that arsenic is known not to interact with DNA. However, the study Mark Moss's lab two years ago showed clearly, at least indicates clearly, that methylated metabolites of arsenic that contain trivalent arsenic do interact with the DNA. As a matter of fact, at micromole concentrations, dimetholarsenose makes DNA in vitro and damages quite heavily DNA in human leukocytes.

DR. BECK: I believe, wasn't that with naked DNA?

DR. STYBLO: Naked DNA in plasma, but also experiments in intact leukocytes.

DR. BECK: I did want to just note for the record that our risk assessment we looked at just at CCA-exposed children. We did not look at the general population of kids.

DR. HEERINGA: Thank you very much, Dr. Beck. At this point in time, I'd like to move on to the next in the sequence of presentation, public comments. And this is a comment by Dr. Barbara Peterson of Exponent.

DR. PETERSON: Good morning. I'm Barbara Peterson, practice director for Exponents food and chemical practice. I'm speaking today on behalf of Georgia Pacific and primarily addressing your Charge

Question 12.

Dr. Lella Barraj who has worked extensively with the SHEDS-Wood model is also here and would be happy to address the more technical statistical questions.

As it's clear from today's discussion, we have many suggestions for improving the risk assessment, including suggestions that we think should be done before the results are presented to risk managers or used. Without taking away from the enormous amount of work and accomplishments that have already been done on the model, I would like to shift the focus of our discussions to next steps, continuous improvement if you will.

As you know, I've been before you many times in the past 20 years to discuss individual risk assessments, new tools for conducting risk assessments, and new types of data. And each time we have to decide what data to use and how to do the risk assessment and especially how to interpret our findings.

Today we're reviewing really a brand new tool that allows us to easily conduct complex exposure analyses and to do so in a way that can better simulate actual exposure. It's usefulness, though, is going to depend largely on the data used and the assumptions that we make. And in order to evaluate the current assessment, I think it's critical that we understand the context of how these risk assessments were derived. And in particular, I'd like to talk about CHAD diaries.

Some of the other uncertainty and the exposure estimates and again to comment different on the cancer slope factor.

There are over estimates, I think, in both exposure and in the cancer slope factor. And although we've tried some uncertainty analysis, I think a number of the assumptions are resulting in an understatement of the uncertainty in this model.

This is the first time OPP has conducted such a complex analysis and necessarily many assumptions have had to be made. If you look at the analogy with the work we've done in dietary exposure assessment, I think we've seen that as we've moved from worse case assumptions to more refined and more realistic assessments, we've found that our worst case assumptions turned out not to be terribly helpful in guiding us forward, that the pathways and sources of our exposure that we were most worried about once we had real data to replace our assumptions often turned our conclusions upside down.

And I think that's likely to happen here. Because there are so many parameters going into the model, that even when you look at a single parameter such as we did yesterday with the dermal absorption where that was contributing 50 percent of the exposure and a new study you look at that data and you would conclude that this is contributing a negligible source to the pathway.

Quite a difference. And there's so many different parameters with missing data I think you would see the same sort of differences.

Let me turn now and talk about the CHAD diaries a little bit. It's clear that as you look at the CHAD diaries that these, although they represent a particularly very useful source of data for this, they were not collected with playsets and decks in mind. They're a compilation of multiple behavior activity, and the questions were asked in a different format. There's repeat sampling for a few kids but in general what we're doing to create a longitudinal diary is taking eight single days to create a year.

I don't believe these are necessarily representative of a population of children playing on playsets in the U.S. And I also think the fact that we're compiling days for different kids into a single longitudinal diary and we don't have that many kids to draw those eight days from, we're ending up using the same data over and over and over. Whereas I think if you actually had data for single children over those multiple days, that you would see the extreme tails of these distributions regressing towards the mean.

Finally, the time-use categories as I mentioned are really not consistent with playground activities. If I can have the next slide. I won't drag you through all of these. It's pretty clear that, if you use the estimates of outdoor time as a surrogate for playground time, you would be over estimating the time on playgrounds. And there's a lot of categories that were possible to be answered under this outdoor potential playset time which clearly are not playset time as you look at indoor chores, cleanup, outdoor chores.

Now, obviously, not all of these apply to children and may not have been used. But somebody and somehow you have to get from these categories to playsets, running errands, personal care, and so forth. Even sleeping or napping, watching movies, going to museums.

What's actually missing on this is playsets in the process. It's not to take away from the usefulness of the data, and there have been a decision by EPA on how to go forward with this data and not assuming that all of this time was, in fact, on playgrounds. But there wasn't really much data in order to make the leap to the model that's used. And I think it's likely to have quite a big impact on the resulting exposure.

Again, just some more of the categories that are there.

In addition to more work on actual behavior, I think there are some other studies that really should be done and added to this model, frequency of contact with playsets and decks outdoors, the CCA residues on children's hands while playing, and as we've already discussed, the mouthing of children during active outdoor play on playsets and decks is likely to be quite different from indoor studies.

And having done a lot of these things or even perhaps before, it's useful to have a benchmark to try to say are we in the right ballpark.  And I believe that the biomonitoring study offers us that opportunity and that it should be done relatively quickly.

Which leads me to my conclusion that there is high uncertainty in the whole model but in particularly the pathway that at this point appears to be the driving force which is the residue ingestion exposure has many parameters and each of those parameters do have a high degree of uncertainty and that we're in each case biasing that exposure upwards so that when we're through, I think we're really measuring more our uncertainty than we are our actual exposures.

That corresponds because this is combined to a calculated risk estimate, we have, I think, high-end exposures that are highly uncertain and we've combined that with the high-end toxicity value that is likely biased very high in relation to U.S. populations as we already discussed. And that leads to straight forward math of an unrealistic high estimate of risk.

So in conclusion, a couple of recommendations. I think we need to provide further context regarding the uncertainty inherent within this risk assessment. These are complex parameters and they should be the assumptions that go into each one need to be explored and impact. Second, simply, we need to promote additional research to fill these data gaps.

Nonetheless, I believe that the average exposure once we get the model refined should be compared to the cancer slope factor as in all over OPP evaluations of long-term effects and that we should make sure the calculated exposures really do represent reality and basically on two sides. First, in that improved simulation of the activities of a child on the playsets on a given day and then how we translate that information into long-term exposures and then from those long-term exposures into estimates of risk.

Thank you.

DR. HEERINGA: Thank you, Dr. Peterson. At this point, do we have any questions or clarifications from the Panel? Dr. Zartarian.

DR. ZARTARIAN: Good morning. I just wanted to try to address the concern that CHAD diaries with reported outdoor time but unrealistic activities with respect to contact with treated wood were used. And the main point to clarify that is that the activities in CHAD were not relevant in this assessment. What we were really trying to do was simulate realistic patterns for outdoor time for children for the population that we defined. And the use of CHAD to do this is justified because of the similarity in the distribution for reported time outdoors for all children in CHAD versus the reported outdoor time of children who reported time at playgrounds in CHAD. And that's discussed in the report. And the comparison of those distributions is in Figure 2. So that was the basis for doing that regardless of the activity.

DR. HEERINGA: Thank you very much. With regard to the time-use issues, there is some research sort of optimal measurement of daily diaries. A colleague of mine, Graham Culton, in a book entitled, "Time Use," edited by Tom Juster, does this analysis in terms of looking at optimal numbers of sample days. And clearly we cannot get accurate measure over long longitudinal periods of time. There's a little bit of evidence to support that within the measurement environment that we're constrained to that this sort 3- to 5-day type environment is the best.

I'll comment more on that in response. Dr. Reed.

DR. PETERSON: If I could --

DR. HEERINGA: Yes, Dr. Peterson.

DR. PETERSON: I think my concern there is also it's not three to five days. It's days from different individuals that have been combined to look like three to five days.

DR. HEERINGA: Right. And that is another issue and that is to what extent the clustering of single individual's daily time-use activities, which is really what we're using in this modeling effort, is leading to added variance in the simulation. And I think it's an empirical question to some extent, but it's one that I think could be explored. Dr. Reed.

DR. REED: Dr. Peterson, could you comment on sort of the comparison between this particular scenario about the time-use and the deficiencies of database versus, say, in dietary exposure that you have also lack of a longitudinal base? How do you deal with that versus how do you deal with that information here?

DR. PETERSON: I think you see quite a similar parallel. There's been a recent European study on the dietary side whereas they went from 1 day to 7 days to 14 days to 21, what you had was not an extension of the tails in your estimates of exposure but a regression towards the mean.

And I think you would see that here. We have a limited amount of information where we had 1, 2, and 3 days. And we certainly saw the children's time outside going from three hours down to almost two. So I think the parallels look quite similar to me in fact and stress the importance of needing that data because it's the fundamental starting point for the whole risk assessment.

DR. REED: Could I have another? I have another question.

DR. HEERINGA: Dr. Reed, certainly.

DR. REED: Yesterday I was interested in what is the sum total, what's going on with the use of database the way it is. If we come up with two or three hours per day of daily outdoors and then the fraction of that going into the playing with a playset, in general do you think that that was an over estimation of two to three hours a day and then a fraction of that on the playset?

DR. PETERSON: I think we don't know what fraction of that was on the playset is my concern.

DR. REED: So it was not the number of hours per day but the fraction of time outdoors.

DR. PETERSON: Right.

DR. REED: You were referring to --

DR. PETERSON: If you've watched children on a playground, there's a multitude of activities that go on there. And I think we just simply haven't -- it's not a function of the model that's the problem. It's the function of the underlying data.

DR. REED: Right. The database, yes. Because I was a little bit confused. You were referring to the different codes, and those are different activities. And I thought you were referring to the sum total number of hours.

DR. PETERSON: No.

DR. REED: No. Okay. Thank you..

DR. HEERINGA: Well, thank you very much, Dr.

Peterson. I appreciate your comments.

At this point in time, we'll move to our next public commentor. And my intent is that -- this is Dr. Joyce Tsuji --

DR. TSUJI: Yes, thank you.

DR. HEERINGA: -- on behalf of Exponent. And we're scheduled for 30 minutes according to my records. What I would propose is that after Dr. Tsuji's comments, we will take our break just for those of you who are planning ahead. Dr. Tsuji.

DR. TSUJI: Thank you. Thank you for allowing me to address the Panel. I've been asked by ACC to present some results of work that my colleges and I have been involved with to characterize background exposures to inorganic arsenic. And my presentation probably most relates to Issue 6, which is the evaluation of the SHEDS-Wood model results.

Unlike most pesticides, arsenic occurs naturally in the environment. And we all have some exposure to arsenic and most of this comes from our diet and water.

Consequently, understanding background exposures to inorganic arsenic is important for placing risk assessment results in context. And this is especially important for arsenic because background risk of arsenic using EPA methodology are typically higher and much higher than one in a million cancer risk.

And what this means is a one in a million cancer risk doesn't tell us anything about whether the exposures that are calculated are out of the ordinary or not. Therefore, I'll be presenting information that we have been involved with to characterize background sources from diet and water.

Dietary arsenic levels have been reported in the past by the FDA, although much of this has been on total arsenic. And I just wanted to right in the beginning distinguish that much of this arsenic in our diet, the total arsenic, is organic. And that a fraction of it is inorganic. And that's what we spent a lot of time and a lot of effort has gone on in the last several years by other researchers as well to characterize how much this is inorganic.

Dietary inorganic was examined most recently for children by Yost, et al., using survey data and published analytical data for food. And what was done was a Monte Carlo probabilistic analysis of distributions for the U.S. populations of children.

This study -- and I'm sorry. I didn't bring copies with me. But if the Panel is interested, I can provide those. Basically probabilistic modeling was done using a software analysis system that incorporates the dietary patterns of individuals survey respondents. Then the program translates this food consumption pattern data into ingredients which then you could apply published results on inorganic arsenic in different types of foods to develop your arsenic intake distributions.

Inputs to this dietary model were the continuing surveys of food intake by the USDA and inorganic arsenic data on over 40 foods that were analyzed by Battelle Sequim Laboratory, published by Schoof, et al, 1999.

Now I should mention that the water used in the

Yost, et al., paper is very low. It was .8 microgram per liter. So we have more recently expanded this evaluation to include drinking water which was not included in Yost, et al., dietary analysis. And also water used in food preparation, basically, we used U.S. water data.

Drinking water arsenic levels in the U.S. are fairly low. They average 1 to 2 micrograms per liter. However, there's some variation out there. And while most are below 3 micrograms per liter depending on the sources or the region of the country, some supplies can have levels exceeding 5 or even the 10 microgram per liter new MCL level for arsenic in water.

This just gives you an example summarizing some of the differences in water system data or water source data. This is for different systems groundwater, community water systems, service water, community water systems, and then groundwater of nontransient, noncommunity water systems.

And I'm showing the percent exceedences of different levels of arsenic in water, 3, 5, 10, 20, and 5 microgram per liter. And you can see that groundwater definitely has more arsenic or has more percent exceendence than the surface water source. The actual data we used was more finely divided and more detailed then this.

So our model took into account both differences in source around the U.S. and as well as regional occurrence data. And this just summarizes percent exceedences and much more updated information. The use of the this shows that certain low arsenic in water (inaudible) very low and they tend to be high in the West and in New England as you can see.

So what we did was we did a combined probabilistic analysis of both diet and water together. We didn't do two separate probabilistic analyses and then add them together. That's inappropriate. What we did is a combined probabilistic analysis. And we used distribution information for regional water data as well as food sources. And -- I'm sorry -- and water source information. And we used this for both drinking water and water used in foot preparation.

We also used the continuing food survey information for drinking water intake rates. So we have distributional data for intake rates and we incorporate, of course, all the distributional regional information on food intake rates. So this was done for children aged 1 to 6 as well as for the whole total U.S. population.

We ran a Monte Carlo run using the existing water data, and we also ran a separate run. As you know, the new MCL is now 10 parts per billion. And we expect in the future that water levels above 10 will be addressed and brought below 10 in these water suppliers. Therefore, we ran a separate analysis in which we truncated the water data to 10 parts per billion and below. And basically what we did is we took all the data that were above 10 parts per billion for the various supplies and we assumed that they occurred in the distribution below 10 parts where billion.

This shows the results of that combined probabilistic analysis for diet and water, the blue bars.

And then I also show you the Yost, et al, diet information which is a separate diet only probabilistic analysis.

Now, I could have also put another bar on there for water only, but I thought it would get too busy. What I wanted to show you on this also, point out about this, this is different probability percentile estimates for inorganic arsenic intake for ages 1 to 6, is that you can't take diet and water and do separate Monte Carlo analyses on them and take a 95th percentile and add them together and say that is the diet water combined 95th percentile because that actually is a number that is above the 95th percentile. And as you can see there, it looks like diet is a big part of your water combined. And although it is a significant part, it's not as high as you would think when you do a combined analysis.

I'll show you what happens when you truncate water. It mainly affects the upper percentile estimates. It doesn't change the mean hardly or the middle percentiles.

Now, we look at what's contributing to inorganic intake, this shows you different components of the 95th percentile intake and the mean intake for children. And we see that water has a big influence at the 95th percentile and the mean and then other components of the diet, such as rice, fruit, and grains.

Next slide shows you the truncated data, and water has come down. But it's at the 95th percentile and even the mean, it's still a major component of inorganic arsenic intake.

So as you can see, those who had had higher end inorganic arsenic intake have higher water arsenic levels and eat more rice and fruit. Inorganic arsenic intake, as you would appreciate, would probably then vary by region and according to your food preferences. So at the high end about 3 micrograms per day of inorganic arsenic comes from rice and one cup of rice has about 4 micrograms of inorganic arsenic.

So this kind of begs the questions. I showed you a distribution that was the total U.S. population of children at 1 to 6. What about subpopulations? And the answer is, if you were to construct separate distributions for subpopulations of concern, much like EPA constructed a separate distribution of CCA children specifically exposed to CCA-treated wood, you would get a different distribution. And, of course, it would be higher.

Now, we didn't actually run that. But I was just going to show you some regional differences in water and potential difference in diet. Here's some of the regional differences you would see. I'm just showing you a summary, mean, 95th percentile, and 99th percentile rather than complete distributions for each.

Here you see for the different regions, the west again is the highest and northeast is fairly high as well. The total is on the far right. And that's kind of intermediate. And, of course, the south is fairly low.

This shows the truncated water data. It looks a little more squished because I scaled it the same as the other data. But you can see the northeast and west are still probably the highest, and the south is lower. 95th percentile for the west is still fairly high. It's the 99th that really got chopped.

Now with respect to rice, we don't have any U.S. data that could readily point to rice consuming populations. But I could have used Japanese data. And what I was able to find was mean. I couldn't find upper percentiles or distributions. But if you just look at mean comparisons between the Japan and the U.S., and this shows for different ages, from age categories from young to old, and the amount of rice consumed per day. We see that the Japanese eat quite a bit more rice than in the U.S.

And I should also point out, these are current data. And that if you looked historically, the Japanese rice consumption would be even higher. Their diet has changed somewhat.

Another thing the Japanese tend to eat a lot of, and Asians in general, I guess is seafood. And although in the U.S. seafood really didn't make the list of contributing foods items for children, most kids in the

U.S. don't each much seafood. My son is a typical example. But if you eat enough fish, you can get a fair amount of inorganic arsenic. Although most of the arsenic in fish is organic, seafood does have a high amount of total arsenic. And about 1 to 10 percent of this is inorganic. And that combined with a high seafood intake can give you measurable amounts of dietary inorganic arsenic.

For example, data by Morrie, et al., which looked at food consumed by Japanese adults and their exposure to inorganic arsenic over three days. And he looked at 12 individuals. They averaged 14 microgram per day of inorganic arsenic. Whereas the 98th percentile in the adults in the U.S. is about 13 microgram per day. So some of the more higher percentiles for diet in the U.S. for adults at least is similar to the mean in Japan for inorganic arsenic.

So I guess that kind of begs the question: Do high rice and seafood diets really increase your arsenic cancer risk? And I have to say that there's no definite study about that. There's no obvious increases in risk based on cancer incidence rates. If you wanted to compare Japan versus the U.S. for bladder cancer, which is an arsenical-type cancer, the bladder cancer incidence in women is Japan is one third the incidence for Caucasian women in the U.S.

And I actually should mention, you could look as men as well, although higher smoking rates in men which is a risk factor for bladder cancer, then do result in higher rates overall. But the same sort of relationship holds true. So there's nothing obvious with this crude comparison that I could see.

And the same is true for drinking water. And others will talk more about that or have talked more about that than I will. But overall the U.S. studies do not confirm an association between arsenic and cancer risk. And I think the Lewis study was noted. It is a large study that has been much reviewed. And then Dr. Lamm has conducted a recent investigation of bladder cancer mortality rates in comparison to arsenic levels by U.S.

county. And I understood he'll be speaking later today and probably could address any specific questions you had on that issue.

In this slide, I just wanted to compare the dietary intake rates that we have calculated versus the intake rates of arsenic by the EPA SHEDS model. And what I have here are mean 50th percentile and 95th and 99th percentile for playsets only, children exposed to playsets and decks, and then playsets and decks with a .01 percent dermal absorption using the latest research.

And I should point out that this is the worse case because it's the warm season climate scenario. And I chose immediate term intake. You could also use the short term intakes scenario, but some of the upper percentiles are actually lower than when you use intermediate term.

When you look at this, you see that at the mean the diet and water are actually going to be higher than the CCA-playset exposure or they are somewhat similar. And the same is true at the upper percentile. I think EPA's conclusions in their risk assessment that they don't expect any noncancer risk to children from short-term exposure is consistent with these data here, that the exposures on the decks and playsets, even in these extreme situations, are fairly comparable to dietary distributions and water.

Well, the important comparison for cancer risk, however, is not on a child daily intake basis. It's really an average lifetime weighted dose. And food and water intake continue throughout your live whereas exposure to CCA residue in soil is primarily during the young childhood ages when the hand-to-mouth frequency is high.

So on the next slide, I compare the lifetime intakes. This is the LADD from EPA's risk assessment to U.S. population results from our modeling of diet and water. And we find that the CCA playsets and exposure for a lifetime is far less than from diet and water.

The other thing I'd like to point out is the that upper percentiles of diet and water have a tendency to be biased upward because they're based on two consecutive day surveys -- I'm sorry. Two nonconsecutive day survey. So the average of these two nonconsecutive day surveys from individuals. Therefore, they're not averages over much longer time periods. So there's a tendency for it to be biases upwards. But the same sort of procedure was used in using the CHAD diaries where you have only one-day diaries from individual children.

But I think it's more accurate to look at diet and water at the 50th and mean percentiles which are less affected by that bias. And you can see there that the mean and 50th percentile for diet and water are similar to the 99th percentile for the CCA exposure.

So in conclusion, I think we have seen that background exposure to inorganic arsenic intake are mainly from diet and water and they vary within the population. Subpopulations probably have higher intakes. We don't have any evidence that there's a big risk from diet and water at the typical levels in the U.S. or the U.S. distribution.

And this kind of brings up the last point, that is, these higher background exposures for arsenic intakes from diet and water than from the calculated CCA exposures suggest that you wouldn't see much health benefit from reduction of CCA exposure.

Thank you.

DR. HEERINGA: Thank you very much, Dr. Tsuji.

Any questions from the Panel to Dr. Tsuji? Yes, Dr. Francis.

DR. FRANCIS: I just have a question about food because I don't know much about it. And you call it inorganic arsenic. What's the proportion of arsenic 3 to arsenic 5? Do you have any idea for different foods?

DR. TSUJI: I think it varies, and that's been characterized as well. But in terms of chronic exposure to low level arsenic levels, it doesn't really matter if it's 3 or 5 in the food.

DR. FRANCIS: I was just curious.

DR. TSUJI: And when I say inorganic arsenic, we sum it. We use the total inorganic.

DR. HEERINGA: Dr. Bates.

DR. BATES: I was just wondering why you say that the relevant dose comparison for assessing cancer risk is a time-weighted average dose over a lifetime. That suggests that an exposure earlier in life is sort of equally relevant 75 years on. I raised this yesterday in regard to the averaging time.

But do you actually believe that it's appropriate to average doses received in early life out of your whole life time even though --

DR. TSUJI: You know, that's a -- yeah, I would totally agree with you. That's always bothered me. But this is a standard EPA procedure that's done for cancer risk assessment. And this is what was done in the CCA risk assessment according to their guidelines.

And I would agree that if you had a carcinogen that, for example, had a great risk early in life versus later in live. Now you have got to take into account that earlier in life, there's better DNA repair going on than later in life. And then that would be an inaccuracy. But I can also say that there's evidence that arsenic appears to probably be more like a late-stage carcinogen than an early stage. So I think it's probably okay for arsenic.

DR. HEERINGA: Any other comments? Dr. Bates.

DR. BATES: I have one more. I'm not sure whether this is the best time to raise it. But I did notice that in the industry document that we were given, a lot was made of the Lewis study, which, of course, takes place in Utah. And the study population there was a fairly devout Mormon population, which had very low rates of smoking, we can assume anyway. And of course, smoking is the known determinant, known risk factor, main risk factor, for bladder cancer. So even though the comparison population was the whole of Utah which probably has a smoking rate, so what you ended up with was a standardized mortality rate which was quite a bit below expected.

So it raises the question whether that was even an appropriate population in which to examine rates of cancer associated with arsenic in the U.S. population because it may be that the study population actually had a higher rate of cancer, but it was obscured by the fact that they had such a low smoking rate. And there's no way to tell that.

So I'm not convinced that you can actually use the Lewis study to draw any conclusions about arsenic exposure in the U.S. population.

DR. TSUJI: Well, I think you're correct in that the comparison population is not a good way. You always want a more similar comparison population. But what I took away from that is you look at the dose response within the actual population. And there was no dose response for increased cancer risk with increased water concentration even for bladder cancer or any of the cancers that are associated with arsenic.

And I think Dr. Frost, who's an epidemiologist and has really looked at this, can better address that.

DR. HEERINGA: Dr. Frost.

DR. FROST: Yes. My name is Floyd Frost with Lovelace. I'll be speaking shortly.

We did look at the -- we've done an ecological study in the U.S., and we did look at risk factors for lung and bladder cancer. Nationally, the urban areas have higher risks of both diseases, both incidents and death from both diseases with the exception of Utah. For some reason, Utah there does not seen seem to an urban rural difference in the rate of the disease.

So that criticism, although it's true nationally, does not seem to apply to Utah as readily as you might think. So it was a reasonable thing to assume that it might. But in reality, when you look at the data, it just doesn't.

DR. HEERINGA: Thank you very much. Dr. MacIntosh.

DR. MACINTOSH: I enjoyed your presentation.

DR. TSUJI: Thank you.

DR. MACINTOSH: I enjoyed your presentation. And I'm wondering if you could comment on the relevance of your findings to the proposed biomonitoring study; and also did you or anyone in your group contribute to that design.

DR. TSUJI: I was not directly involved in the design of that study, but I have been talking with the investigators. I suggested they put the children on lower arsenic diets, that they avoid certain foods that have higher arsenic. And that would be one way to increase the sensitivity of detection.

But I think it would also be interesting to see, again, if what we're finding, based on our studies is correct, and the biomonitoring study would show that. And I think -- I predict it will be just because I've done biomonitoring study for arsenic in populations in other countries exposed to arsenic and in the U.S. And the dietary information we have indicates that the exposures are very low that are being calculated here and comparable to diet or below diet.

So I think the study is, although, I think you can increase the sensitivity based on what we know about what foods contain inorganic arsenic. And also by doing what they're doing is a repeated measure design, so you're controlling for some of those individual variation in inorganic arsenic that would increase the sensitivity. So

I think it's still of value.

DR. MACINTOSH: Thank you.

DR. HEERINGA: Dr. Wauchope.

DR. WAUCHOPE: Why is rice so hot for arsenic?

DR. TSUJI: Well, again, I don't want to say that rice is, you know, let's run out of the room and never eat rice again.

DR. WAUCHOPE: No, no, no, of course not.

DR. TSUJI: We're talking about very low microgram levels of arsenic. And rice is something that when you compare it to seafood it doesn't have a lot of arsenic. But the arsenic it does have is inorganic. And the other thing is rice is something you eat a lot of. Grapes also have inorganic arsenic, but you don't eat big bowls of grapes daily as a staple food.

DR. WAUCHOPE: I was just trying to figure out why it's higher than, say, other crops, other plants.

DR. TSUJI: I don't know. It could be because of the way it's grown or the way it incorporates arsenic. I'm not sure if that's been well studied.

I should also distinguish that the arsenic levels we used rice were measured in the U.S. And were actually fairly low compared to arsenic levels that are measured in rice in Asia and especially the arsenic levels that were measured in rice in Taiwan. So I wanted to distinguish that point as well.

DR. HEERINGA: Yes, Dr. Styblo.

DR. STYBLO: Just a curious question. Do we know anything about the chemical microenvironment of arsenic in products like rice? Is it bound to organic structures? Is it free? That's the first question.

The second question: How do you think this kind chemical environment is comparable exposure to CCA arsenic from CCA-treated debris where, in my opinion or my impression is it's mostly inorganic background.

DR. TSUJI: Well, we certainly can measure the urine of individuals that have these high rice and seafood diets. And we find inorganic arsenic and the metabolites of inorganic arsenic in their urine. So it's definitely bioavailable.

Also this is kind of a related issue, but it's been discussed about different extraction methods used to get the arsenic out of the rice, are you actually degrading the compounds that normally are not bioavailable. And so I would say that earlier studies on inorganic arsenic from the food from the 70s and 80s are not very reliable.

And with the 90s, then there was a large comparison that was done among labs. So there was better techniques and extraction methods that were developed to look at that. And it was found that there is comparable results among labs even using very mild extraction techniques like using water. So I think we're fairly confident now that the inorganic arsenic that we're measuring in rice, although I don't know if we're completely characterizing what compound or form that it exists in, that it is bioavailable and it is important for exposure.

DR. HEERINGA: Thank you very much. Well, at this point in time, I would like to adjourn briefly for a break. It's 10:32. Let's reconvene here at 10:47, in 15 minutes. And we'll continue with our presentations and public comments session. Thank you.

(Break taken at 10:32 a.m.; meeting reconvened at 10:55 a.m.)

DR. HEERINGA: Good morning again and welcome back to the continuation of our public comment session for the FIFRA SAP meeting on childrens exposure to CCA on playsets and decks. I want to continue with our public commentors. But before we do that, I want to ask Dr. Raj Sharma from the Arch Chemical to introduce himself. He has a coordinating role in these presentations. Dr. Sharma.

DR. SHARMA: Thank you, Mr. Chairman. My name is Dr. Sharma. I do represent the industry. And I'd like to introduce Dr. Frost who will provide more detail on an area which I know that several of you have commented on and finding interesting which is the area of doing biomonitoring studies as way of validating models.

In addition, I'd just like to point out we did send in the earlier packet which was bound a scope of work which outlined the details of the study; and a subsequent submission was made which included details, the full protocol for the pilot study as well as additional materials.

So with that, I think I'll hand over to Dr. Frost who will actually talk about the study in more detail.

DR. HEERINGA: Dr. Frost.

DR. FROST: My name is Floyd Frost. I worked 14 years with the Washington State Department of Health as an epidemiologist doing both chronic and infectious disease work. And since 1992, I've worked for the Lovelace Respiratory Research Institute doing a variety of externally funded studies, usually federally funded.

I'm going to talk about two issues. One is the proposed biomonitoring study, and the other is a study we did of kids who grew up around the Tacoma smelter.

The proposed biomonitoring study would address a number of issues that have been raised here. The SAP requested the study earlier. We can examine some of the exposures to see if they are appropriate, if the levels are appropriate. It contains some information on how much variation, heterogeneity there is between people in the study.

One of the concerns I had early on is is it feasible. As Joyce mentioned, the background levels of arsenic in the population are fairly small. But still the levels of that we're looking for are also very small here. And there's, unfortunately, very little background data on arsenic exposures because most of the people who've measured arsenic have gone after populations with substantial exposures.

And even the control populations, say in the case of the Tacoma smelter, the control populations had much higher exposure levels than a general population. So we have relatively little effort has been put into understanding the variation, person-to-person variations, in background arsenic exposure levels and especially in children.

So the pilot study, we need that information to estimate, first of all, the feasibility. Can we determine the sample size needed? Can we actually change the arsenic levels in people? We don't propose to actually add arsenic to anybody's diet.

What we proposed to do is instead go to areas in Albuquerque in particular where we have high background levels -- relatively high. It used to be called low -- background levels of arsenic in the drinking water. And then for these people, provide bottled water for a period of time. Measure it before; measure it after to determine for sure that we can actually measure the difference between the existing exposure and the new lower drink water exposure. There's been quite a bit of evidence that drinking at the levels we're looking at here is the primary component to the study or to the arsenic levels.

Now, the pilot study will have various components. Too bad we can't actually see this. But the idea is, as I mentioned, these people are on elevated arsenic in their tap water, usually, 10 to 13 parts per billion. We're trying to reduce as much dietary arsenic exposure as possible, so we try to restrict seafood. We're going to restrict other foods, grapes and rice and other things to the extent possible. Rice is not a big issue in children, I don't think. But we're going to try to restrict it nonetheless then measure the levels without a food component except that we can do that. And then put the kids on bottled water.

The bottled water should have no arsenic in it. We can hope that we're going to get a hundred percent of their water. We'll probably get close to that. But we probably will not achieve a hundred percent of anybody's water. And then determine whether or not we can actually see a reduction in the urinary arsenic levels in these children.

And then the variance from one kid to the next is essential to calculate the sample size for the full study. So it's both a matter of determining the feasibility, can we even do this, and measure the levels that we're interested in seeing. And, secondly, if we can what kind of sample size do we need to do the study.

So the issue here then is to identify populations in the full study, assuming that the feasibility study says you can do the full study because I don't want to move on to the full study unless it's feasible. Identify population of children exposed to CCA-treated wood decks and playground equipment, both at home and/or at day care center, using the pilot study again to determine sample size. And then do this in the summer.

The pilot we're hoping to do this winter where we can minimize other exposures for these kids. And have that early spring so we can actually be ready to do the study in the summer.

The full study is going to be similar design to the pilot in that we will sample urine two consecutive first morning voids while playing on the structure. After a period of time they'd be playing on the structure, we'll measure their arsenic two consecutive times and then restrict access to the structure. That's basically a wash-out period. And then resample them again two first morning voids and test for the urinary arsenic.

The testing, by the way, will be done at the University of Washington, Dave Coleman's lab.

In addition, we're looking at possibilities of doing surface wipes of dislodgeable arsenic. We want to make sure that, in fact, these decks have arsenic on them. We want to make sure that there's a true exposure here. Do some handwipe sampling, videotape, possibly the X-ray florescence examination of the structure. Although this turns out to be much harder than we thought it might be. And then we hope to have an external advisory board to review both the study protocol as well as the findings.

We think that the biomonitoring study can improve, assuming we can do the study and it's feasible, the SHEDS model because I think as it has been pointed out here, there needs to be a reality check. We've gone a long way here into the process without actually having any firm data that there is any exposure let alone what the magnitude of what the exposure might be. So having some real data and being able to verify the types of exposure that these kids were having will help us and will help EPA evaluate the reliability of their modeling technique.

This will be talked about a little bit later. But the main thing is we want to look at the various activities that the children are engaged in and ultimately relate those to the urinary arsenic results. So we want for each child see if we can see what are the components that might be contributing. This may be a bit optimistic, again, depending on whether it's feasible, what kind of background variance there is from child to child. If we can get that down low enough, we may be able to do all of these things. If not. We may not.

Again, it can be used to compare the SHEDS model to actual measured values. I think that's what's been brought up here on multiple occasions. This is assuming it is feasible. And then if not, can we actually look at the parameters that are being used in the SHEDS model, compare them to what we observe in the children and see why, if they do disagree, why they might disagree and how the SHEDS model could perhaps more accurately predict the measured urinary arsenic values.

As I say the time line, we've already submitted the pilot for institutional review. And we hope to start that earlier next year and complete it by the spring. We hope to conduct the main study in the summer. There's not much point doing a play structure study in the middle of the wintertime because, with few exceptions, most children aren't out in the wintertime very much.

Now, this has been pointed out in the past and I think we've commented on it. But the U.S. studies are uniformly negative in terms of low-dose arsenic exposure and adverse at least cancer risks and actually even the cardiovascular risks tended to be negative with maybe one or two exceptions. So right now we're dealing with a lot of generally negative low-dose studies in both of the U.S. and Europe.

I want to talk about a study we did when I was with the Washington State Department of Health. This was not initiated because of any concern over arsenic-treated wood. This was actually funded by ATSDR to the Washington State Department of Health. And what we wanted to look at are the kids that grew up around the Tacoma smelter.

The Tacoma smelter was built in 1890. It was initially a lead smelter, but converted to a copper smelter in 1905. And by 1922, it actually had an arsenic refinery. So not only did they refine high arsenic copper. They called it a custom smelter because it actually took high arsenic ores that other smelters didn't want and refined those. But it also took the flue dust from other smelters to recover the arsenic from the flue ducts. So they were processing huge amounts of arsenic in that smelter from the 1920s onward. It was actually one of the two main sources of arsenic in the world at the time.

So high levels. About 600 tons. We don't know how much was admitted and released in the early days because nobody measured these things at the turn of the century. But in 1951, they estimated 600 tons were lost per year. I think this was primarily done as a economic analysis here because that's a lot of money going up the stack. But it was a lot of arsenic going up the stack as well. In addition to the 600 tons, there were a lot more arsenic being emitted at what they called fugitive emissions at ground level, ground level in the smelting process.

This study, we looked at the children exposed to arsenic in the early days of the smelter, 1895 to 1925. The town of Ruston is right next to the smelter. In fact Ruston elementary school is right underneath the stack right next to the school. There's never been a bee sting in Ruston elementary. It's a very effective insecticide and was available abundantly in that whole community.

We were able to actually identify the cohort using census information that the schools did. In the early part of the century, they actually did a yearly census of all the kids going to school. They would actually, unlike today, plan for how many teachers they needed and to know exactly what grades these kids would be in and who they were and who to expect. So for each address, they went door to door and identified the number of kids. This information was stored in the Tacoma Public Library.

We identified 1,800 boys and 1,300 girls. We geocoded each address so that we could identify the distance and the location of that address relative to the stack, which was essentially the same as the fugitive emissions as well. And then we computed exposure as a function of both distance from of smelter and the duration of time in the residence.

As I say, we don't have urinary arsenic measures in the early part of the century. But in 1979, they ranged from 60 to 150 parts per billion. The background level of kids residing further from the smelter, quite far from the smelter, was 10 to 50 parts per billion. This is probably higher than it would be in a totally unexposed population since the stack emissions spread out through the city of Tacoma.

We tracked the children to identify death certificates, obtain the cause of death. We went through the states of Washington, Oregon, California. The national death index, we used that tool as well. We went to marriage records, military records, newspapers, old newspaper accounts, cemetery records, church marriage records, anything we could do to find these people.

The findings, we were able to track I think it was about a little over around 70 percent of the boys. Girls have a bad habit of changing their names when they get married, and it makes very hard to track these. So we actually were able to track fewer than 50 percent of the girls.

What we saw for the only high exposure, high elevated survival hazard ratios were for the highest exposure living 10-plus years in that area. And that was for heart disease, ischemic heart disease, and for external causes. External causes would be things like suicide, homicide, and most importantly automobile accidents.

We found no elevated lung or bladder cancer risk in this population. Again, it was not complete follow-up.

We lost somewhere in the range of about 30, a little more than that, percent of the males and about 50 percent of the females. And also we did not follow these people for bladder cancer. It occurs quite late in life, and so we may not have picked up the bladder cancers simply because we didn't track these people.

But in terms of the issue that Dr. Bates raised that these exposures may be different then exposures that would occur later in life and may be causing cancer at an earlier age, we might have been able to pick up these elevated risks in this cohort. We hope soon to be able to follow-up the cohort. It's been about 15 years since we did the last follow-up on the cohorts, so we hope to do is this again and increase our follow-up of the kids as well as we'll be able to get one more U.S. census data set because of the release of the 1920, 1930 by now, U.S. Census data.

Here's the mean urinary arsenic levels from distance from the smelters. Again, this was in the 70s after tremendous efforts were made to reduce the emissions. But there was still quite a bit of arsenic exposure in this population, and it was related to the distance from the smelter, strongly related to distance from the smelter.

So as I said, data from U.S. studies and European studies provide little or no evidence of elevated cancer risks in arsenic-exposed populations. These populations receive a lot more arsenic than the CCA-treated wood would give you. But in addition, the ASARCO children's cohort would be orders of magnitude than anything than you could imagine for CCA-treated wood. And possibly, the EPA might want to consider using some of these other lower dose exposures to adjust or consider in terms of their estimation of risks in this population in the CCA-treated wood exposure.

And I think that these points have been brought up earlier. The biomonitoring study is an opportunity to validate the estimates from the SHEDS model, assuming, of course, that we can do it. We can prove to ourselves that this study is feasible.

I think that's it.

DR. HEERINGA: Thank you very much, Dr. Frost. Are there any questions from the Panel?

DR. BATES: I just wanted to say that I think it's excellent a biomonitoring study is being planned. I have some concerns about this particular study. But my first question will be: Are you planning to carry out the full study in Albuquerque?

DR. FROST: No.

DR. BATES: You're not. Okay.

DR. FROST: No. The pilot study is being done in Albuquerque simply because we have a population that is exposed to naturally occurring arsenic. So it's a convenient place to find a population with almost any level of arsenic from 5 to 15 parts per billion, this naturally occurring. So we needed somebody who's already on elevated drinking water arsenic. So then we can actually give these kids bottled water to try to bring them down, to see if we can actually observe a reduction. So, no, we will not be doing it in Albuquerque. But pilot study will not be done in Albuquerque.

DR. BATES: I guess that raises the question: Why are you doing the pilot study Albuquerque? Pilot studies are usually meant to be sort of a small-scale version of the final study which this doesn't seem to -- it seems to be a preliminary study rather than a pilot study.

DR. FROST: Okay. You can call it that if you like.

DR. BATES: And one of the objectives of whatever you like to call it, pilot or preliminary study, was to actually look at the success of your recruitment methods. But I do wonder if you're not going to carry your full study in Albuquerque -- and I should say parenthetically I think Albuquerque would be bad place to carry out the full study because --

DR. FROST: I agree.

DR. BATES: -- the background exposures are too high. And even if you did reduce it with bottled water, you would still have difficulty detecting these incremental exposures.

DR. FROST: No. It would be a horrible place to do the full study. But it's a very good place to do the pilot study or the preliminary study as you call it.

DR. BATES: Yeah. I think one of your objectives was actually to look at the success of recruitment. But success of recruitment is probably going to vary in different places. If you carry out -- if you try to assess it based on Albuquerque where their is some awareness, I think, that the water supply is a little bit elevated in terms of arsenic, it may be different in some other place.

DR. FROST: I think they're aware that it's elevated. I'm not sure there's a lot of concern. But people are aware of it.

DR. BATES: Anyway, I think it's not strictly a pilot study. And you might consider doing a true pilot study in some other place particularly where you intend to do your final study.

DR. FROST: Well, I think in the final study, we probably need to replicate in several locations anyway. And so that the first actual implementation may be the true pilot study as you would call it. But before we even get there, I wanted to make sure that we can measure what we can say we can measure. If we think we can measure this stuff, we have to be able to prove to ourselves we can do what we think we can do.

DR. HEERINGA: Dr. Steinberg.

DR. STEINBERG: Dr. Frost, this is very responsive and front and center on our Question 11. And, therefore, there has been some discussion as it relates to this pilot study.

I think the study, if you tell me or if you can reassure me at this study at this point -- and obviously as an investigator, you have the right to carry out the study as you please with the funding that you please. If this study is a work in progress and, of course, not yet final, this would be a study that I would almost have preferred to seeing something like an RFP in a sense where there are clearly goals and stakeholders from industry and you and the EPA would have a chance of sitting around the table and devising the better study if that's the case. Obviously, again, you have the right to go on and do as you wish.

So I guess maybe that's my first comment and question. And based on that, I may have a number of others.

DR. SHARMA: Can I respond to that?

DR. HEERINGA: Yes, Dr. Sharma.

DR. SHARMA: This is a study that is being done by industry and the registrants of CCA. It is also a study that we've received input from. We've worked with staff in ORD. We've worked with staff within OPP. So there has been a joint effort between EPA and industry and the principal investigators are employed for us to make sure that everybody's point of view has been considered. And we plan to do that for the main study, too.

DR. STEINBERG: Yeah. I'm going to have to say that I'm not completely convinced of that. In my personal poll of a large number of people involved in this area, this study, of course, only came to the Agency, my best guess, was a few weeks ago. Indeed the study is undergoing change as we see it. So for example, in the PowerPoint presentation, there are a half of dozen items that were not included in the study. So to me there is -- you know, again, you certainly have the right to go out and do the study. But if it's going to be a meaningful, applicable study and it's going to have some impact and satisfy all the partners, again, it would be my suggestion that this study be discussed more broadly before it's fielded.

DR. FROST: The changes have not occurred for the pilot. The pilot has stayed pretty much the same. We have had discussions, as Raj has mentioned, with EPA and with other researchers to try to gain input. And part of the process is to modify the approach as we gather more input. We're looking for still more. The pilot I think we want to do pretty much as we have it. Or what Dr. Bates would not call it a pilot but a preliminary study. But we're open to listening to comments and critiques and would offer this panel the opportunity comment on it.

DR. HEERINGA: Dr. Steinberg.

DR. STEINBERG: I think we'll probably have a large number of comments as we respond to Question 11. And I'm not quite sure where the forum of how we should discuss this. There are many questions that people have in the way that you have put this forward. I will tell you that in any type of -- I would view this as almost a clinical trial of some type where you have an intervention and you go out and do this.

I will tell you, based on dealing with IRBs and based on dealing with large numbers of consents and large numbers of instructions, I would say it already has many, many issues. I mean simply as it relates to the IRB, I will tell you that in your biomonitoring proposal the first issue that you mentioned relates to assessing CCA. Of course, in the study, it seems to be more of a water-based study.

And, of course, in the consent -- and, of course, I would like to see that consent at least in

English and in Spanish. I will tell you that this consent would be very difficult to explain or get approval on. The whole relation to CCA is simply put in about seven words at the bottom of the first page. And, of course, I would not consider that fully informed consent.

And then you're in a little bit of a conundrum because if you go through more informed consent, which you're obligated to do, you're looking at certain behavior changes that can indeed occur in the subjects involved. Again, I'd be interested. Has this been submitted? Apparently it has been submitted mid-month. Was this submitted to the University of New Mexico IRB?

DR. FROST: No, we have an IRB with Lovelace, our own IRB.

DR. STEINBERG: So it is a separate and apart --

DR. FROST: The Pilot has been submitted. We obviously are in no position to submit the full study because we don't have sample size, we don't have variance. There's a lot of issues that we are depending on the pilot study to address, or as Dr. Bates would say, preliminary study to address, in order to actually even come up with, first of all, whether we can do the full study and, secondly, what kinds of sample size and what are the considerations.

DR. STEINBERG: And I will even tell you that even in the way of instructions and what you're looking at, if you were to try to delineate these things to a family, unless you have technicians and trained personnel and nurses aids almost on the spot, coming on a daily basis, this study cannot be reliably carried out in its present form. I think anyone who's involved in a clinical research center would have great hesitation in seeing that this thing could be carried out. And that's why I think we have some pause about what type of data you will obtain if this is indeed the pilot.

And as I say, I can go on for 30 or 40 more bullets on where we think there are issues that can be optimized in this pilot which, of course, we would like to see in its best available fashion.

DR. FROST: Well, we look forward to seeing those bullets from you.

DR. HEERINGA: I think this is a discussion that is constructive in terms of your own research design. Dr. Frost, any more comments?

DR. FROST: No.

DR. HEERINGA: Anyone else? Dr. Bates.

DR. BATES: I'd just like to refer to your specific Aim 4 in your proposal. And that is assess whether a 5- to 10-day wash out period is sufficient to allow for the substantial elimination of the body burden of arsenic resulting from chronic low-level exposures.

My concern here is that the washing out period may be very much related to the amount to be washed out. And in Albuquerque, we're talking about a relatively high exposure. Where as in the actual final study, we may be talking about a much lower exposure. And it could well be that the wash-out period is exposure-related.

In my experience, sort of higher exposures tend to decrease more rapidly. And because there will be a smaller difference you will be looking for in the final study, the data you get from this pilot or preliminary study may not be particularly applicable.

DR. FROST: You're right. It may be more conservative than it needs to be. But if it shows us we can do the study nonetheless, that's what I want to be able to tell. Because I don't want to actually do a study that turns out negative and then not have any validation that we could feasibly have detected the differences that we expected to see.

DR. HEERINGA: Yes, Dr. Hattis.

DR. HATTIS: Have you done a calculation of the cancer slope factor equivalent that you could have detected in your Tacoma study with 80 percent confidence?

DR. FROST: No, we did not.

DR. HEERINGA: Dr. MacIntosh.

DR. MACINTOSH: I have a question about this pilot or preliminary study. As I understand it, you want to go to the pilot study population and put them on a low arsenic water diet, if you will, and look to see if you can see a change in the excreted urinary arsenic levels.

DR. FROST: Right.

DR. MACINTOSH: And if you see that change, it seems to me you're going to conclude that it demonstrates that it would be feasible to see a difference between urinary arsenic levels for children who play on playsets some amount of time versus those who play on playsets a smaller amount. Is that right?

DR. FROST: If we can detect small differences in urinary arsenic from reducing their drink water exposure and that these differences are in same range as you would expect to see from the modeling done by EPA, then we feel we can probably do that.

DR. MACINTOSH: That's my question. Do you think the difference is in the same range as suggested by the modeling?

DR. FROST: Well, I think that the initial -- the first thing that we may need to actually do, to replicate this pilot on different times to make sure we that can detect. If we can detect the first level, we may want to go down too. Since Albuquerque has a fairly wide range of drinking water exposures, we could actually go to 8 parts per billion and then do the same thing and see if we can a detect a reduction of 8 parts per billion. We could probably even go down lower than that if we'd like.

DR. MACINTOSH: Yeah. It would seem to me it would be prudent to design this study you could detect a difference that was substantially lower than those predicted by the SHEDS-Wood model or through some other model. By substantial, I mean maybe 10 fold lower, 20 fold lower, because you don't know that the model is accurate. And that's what you want to evaluate -- right? -- is the model's accuracy.

DR. FROST: Right.

DR. MACINTOSH: So depending on it in your design is inherently circular.

DR. FROST: Yeah. The problem occurs that the EPA estimates of exposure, as Joyce has pointed out, are really just slightly above background. So once we start going 10 fold lower than those, we're really right at background. So we really need to be careful as how far down can we go. We can detect the high-end exposures that the model is predicting. I think we can be able to do that. But I'm not very optimistic that the low-end exposures are even detectable because they are really right slightly above background.

DR. HEERINGA: Thank you, Dr. Frost. At this point, I'd like to move on to the next public commentor. But before we, just a question of inquiry. Do you anticipate being here tomorrow, tomorrow afternoon, during the discussion?

DR. FROST: No, but Raj will be here and Barbara Beck will be here. And they've been involved in the study.

DR. HEERINGA: Very good. Thank you.

At this point in time, Raj, Dr. Sharma, if you'd like to introduce the next presenter from your group.

DR. SHARMA: Yes. I'd like to introduce Dr. Chris Chaisson from the Lifeline Group. And really what Dr. Chaisson is going to do is try and summarize for us all of the previous presentations we've seen and put them into context with respect to where we think this model is with respect to evaluation and validation and use.

So with that, I'm going to hand it over to Dr. Chaisson.

DR. CHAISSON: Good morning. My name is Dr. Chris Chaisson. I'm a senior scientist and director at LINEA, a consulting firm. And I'm also director at the Lifeline Group. Through both of these companies and through the past 20 years of my career, I have developed mathematical models to be used by risk assessors for regulatory purposes. My review of the EPA documentation and preparations of today's comments have been done independently by me and the LINEA staff and have been supported by the American Chemistry Council.

My comments to the SAP are focused on the process of taking a model from conceptual development to prime time, its use in risk management and policy making decisions. I hope these comments will provide a helpful perspective to the SAP.

The Panel's decision is important for two reasons. First, of course is the public policy about to be made regarding CCA. And, secondly, for a larger purpose, dealing with the credibility of models to be used by risk managers and policy makers in the future in the process of developing those models.

Mr. William Jordan introduced this in his opening remarks yesterday. The Agency intention is to use the analysis to go beyond mere regulatory decision because that's a fait accompli. Mr. Jordan reminds us that they will use this to set public policy, use the model for future assessments on other chemicals, and hope to use the larger SHEDS model in many other pesticide use scenarios.

EPA's Office of Research and Development has advanced the concepts of the risk assessment models and incorporated many good features and presented some unique approaches. This is the concept development, Step No. 1. The next step is called "model validation" just for convenience. But it's my opinion that a model cannot about completely validated per se except under very limited and specific circumstances.

For a given scenario, like maybe the case at hand, we can get close to a validation with some work. We can examine the individual parts, the data, the distribution, the functions, et cetera, as has been done here. Then we can explore the consequences of the issues raised about these parts.

It appears to me that there remain important issues in the debate. Issues key to the credibility of public policy decisions streaming from the analysis. Experience with a model the hands of multiple users will flush out problems. We can also compare the models to other models, and we compare computed answers to real-world measurements in carefully designed studies.

There's no real end to Step No. 2. And the transition to use in a policy context is a serious undertaking. So how do we know when a model is ready for primetime? That question is a contest between when we have tested and compared and validated enough to have confidence in the answers versus a real need for the model in a quest for public health policy. It's a balance between technical confidence and urgency of the needs for the policy.

This is a serious decision, going from validation to regulatory and public policy use. It carries some promise and peril regarding real public health consequences or real economic stress placed on, not just the producers, but on schools and communities and individuals. It relates to the credibility of the use of the models in this decision making and sets precedence for how the Agency will bring new concepts and new models. It impacts technical confidence in model and data assumptions in key components, and it either engenders trust or distrust of scientists in regulated community and the public.

The bottom line is that this step can't be taken until the Agency scientists and all user communities understand the peccadilloes and vices and representativeness, strengths and weaknesses of a model. And all models have these, and how that plays into the regulatory decision and the public policy issue of the day.

The way in which the EPA questions are presented to you created a bit of a dilemma. These are important questions for sure, but they're also finely parsed issues to which you can make focused affirmations. The questions are narrow. And when accrued, do not necessarily sum to a overarching whole question.

I fear that the Panel's affirmations to the focused questions of today's meeting will provide a general impression of a broad SAP approval. I foresee a future EPA reference that says something like this, quote, "The SHEDS model was peer-reviewed by the SAP in December of 2003 and endorsed for regulatory use in making a public policy."

EPA's Office of Research and Development obviously thinks that the model, the SHEDS model, and analysis performed with it are ready for immediate use in risk management and policy making without further work or validation. Table 1 in the report that you have, the Probabilistic Exposure Assessment for Children Who

Contacted CCA-treated Playsets and Decks, suggests that the August 2002 SAP review comments have been addressed and all necessary adjustments have been made.

Dr. Ozkaynak referred to the appendix of 20 pages of response to the public comments, but it seems to me that there are many points which were dismissed or incompletely addressed. These include bootstrapping techniques, use of alternative data, et cetera. You've heard a lot of this discussed today. Alternative runs with different assumptions which are hard-wired into the SHEDS architecture or with different user specified values, of course that just means the analysts takes a guess, were largely absent.

The EPA Office of Research and Development have indeed made significant progress in the development of a new model for the specific chemical use profile and these exposure scenarios. However, they've also introduced new concepts and applications. We're back to Step 1. And the crucial second step has certainly not yet been completed.

This SAP meeting is a very important event in that process. The question is whether or not this is the last step. Let's take a closer look at the second step.

The peer review is really limited to the SAP members, that's you, and a small group of interested parties who could afford the significant license fee of SAS and who methodically explored the code or tested the functions of the model. To my knowledge, there's been no broad use or discussion of these analyses in the academic community or, for that matter, any other group.

There has been no comparison of results to other probabilistic models only to deterministic models. That comparison is interesting, but it yields only a viewing of the differences in answers, not in any way revealing key issues about how the model functions.

A model comparison workshop conducted by EPA in 2001 compared the functions of SHEDS, Lifeline, CARES, and Calendex, four different probabilistic models. When identical data bases and assumptions were utilized by each of the models toward a defined exposure scenario, which of course was not CCA-treated wood, the results were very interesting. Differences of an order of magnitude in some cases all because of the way the models dealt with the data or the influences of approaches and assumptions in the model architecture.

Those case studies were less complex than the CCA analysis before you today. Such comparisons allowed the consideration of the reasons for the different answers and the relevance to a regulatory decision. They gave insight to OPP risk assessors on model peccadilloes and the biases or the operational underpinnings. Such enlightenment is vital to the deployment of the model for regulatory decision-making.

There had been no direct validations. If a single model is to be used without benefit of extensive peer review, wide circulation, or model comparison exercises, validation techniques should be explored. In this case, we have a unique opportunity to have a validation exercise completed within the very near future with the biomonitoring study described by Dr. Frost and perhaps amendment with more suggestions.

This is the study requested by the SAP. As I understand the situation, the SAP suggested EPA conduct the study. EPA declined, but industry stepped up to the table. There are been few alternative runs or evidence of the sensitivity analysis by the SHEDS model to ascertain the impact using the SHEDS model, of course, of the different assumptions, alternative value distributions, or uses of other databases.

Even if one does not agree with the details on these as presented by industry, we think we should know how much of a change there could be in the answers. Likewise, the assumptions suggested by other stakeholders should be considered.

The parameters discussed by Dr. Barbara Beck are examples of those analysis. What would the differences be in the exposure assessment? Where are those analyses? I was an observer at a recent meeting with EPA where ORD said that such analyses were done. And they, quote, "Didn't make much difference."

Well, that work is valuable and should be publicly available. It is certainly germane to the conversation here and would advance the process through the second step. I was pleased to see the Panel requested copies of some such analyses. Perhaps these could be shared with the public as well.

We also seem to have concern about how the CHAD diaries were used in the SHEDS model. Let me add my concerns, and a suggestion for improvement for predicting the frequency and duration of contact with CCA-treated playsets and home decks. The CHAD diaries have their limitations but offer some interesting opportunities which are not well explored in the SHEDS application.

One lesson we should all have learned by now, the worse case scenario is not always intuitive. If the model is stocked with representative data or has used data in the most representative way, the model will describe the worse case. We need not assume it, and model toward it. So often we have been dead wrong on our assumptions, including me, and skew the answers by applying the data incorrectly. The markers of this are evident in the SHEDS analysis.

Dr. Zartarian stated explicitly yesterday that these activity constructs are meant to be representative of all children in the U.S.  How do we know that children spend the most time in contact with treated decks or playsets during the summer season throughout the country, and therefore, this is the worst case.

Would that be true for all regions of the country?  How do alternative activities compete for the kids time?  Are the playsets too hot to play on?  Will the young children be directed to other activities away from direct exposures during hot seasons in the South?  So what is the best way to use the CHAD diaries?  Are there other databases to direct us here?

The assumption is that these children experience only treated decks and treated playsets.  Let me make an analogy to the dietary exposure assessments, a topic with which EPA has more experience.  If we have a suspected cancer causing chemical on some but not all apples, we assess exposure assuming that all of the population may encounter some of the apples at some times in their lifetime diet. We do not assume that all of the tainted apples will be consumed exclusively by a fraction of our population. And for those poor souls, all of their apples are treated with the chemical.

Assuming that a subpopulation of children will be the ones at risk, then assuming these kids always interact with treated surfaces is the nondietary analogy and the scenario to which the SHEDS analysis calculates cancer risk. Since we are calculating accrued exposure for a cancer assessment, the assumption that all playsets and decks are treated creates a gross over estimation.

At the other end of the stick, do the rest of the kids in the population have absolute zero risk? No matter what statistics accompany such an analysis, it's just not a realistic scenario of exposure.

One approach to improve this is to use the CHAD diaries, all of the CHAD diaries, but link all the diaries to geographical location dates and then to the weather conditions and school calendars, the probability of outdoor play and time available to be on the playground or home deck can be fashioned from such relationships. This will not correct the inherent difficulties of the data elements included in the activity profiles as reviewed by Dr. Peterson, but such problems can be offset using other information.

For example, associations between temperature and protection from the sun can also be made. When is it too hot to play on these structures or play in direct sunlight? What percentage of playsets at home or school are expected to be treated? Let the model construct many simulated children who represent realistic populations of kids who sometimes spend their time on treated playsets and sometimes on other structures. The exposure profiles presented by Dr. Zartarian would then look quite different indeed.

This is a concept development issue, we're back to Step 1 again, that speaks to the very heart of how the architecture of the model, not just the selection of the data set. This kind of architecture and data application will drive the answers. Already we see controversy on this issue which questions whether the exposure assessment is representative of the population of interest in terms of how they interact with their environment.

At issue is not so much the virtue of CHAD diaries but on how SHEDS uses the data. We have encountered exactly these kinds of problems before with data sets such as National Home Activity Pattern Surveys. SHEDS does not utilize the data well in my opinion.

Now, this slide was borrowed from Dr. Frost's presentation that you just heard. I wanted to emphasize how much information we can glean from this study, not just for CCA, but to resolve many of issues we keep discussing relevant to the representativeness and bias and architecture of data applications in model such as SHEDS but even in Lifeline and others. Having biomonitoring study will provide an opportunity to sort ought some of these issues. The alternative is more haggling on these points for the cases in the future. I think it's time to resolve some of these issues so we can continue haggling over other deserving points.

The previous presentations have highlighted some of the difficulties we have had with parameters like transfer of residues to hands as you'll see there, the hand-to-mouth activities during the indoor versus outdoor play, or fraction of the hands going into the mouth, et cetera. The biomonitoring study will address many of these points.

In my role as a consultant to many institutions and government groups, I've learned to tell the difference between a plan to get results quickly and a plan to whose primary purpose is to stall. This is a plan to get relevant, technically sound data to replace or augment contentious assumptions underlying an important regulatory risk assessment.

In one short year, the registrants have identified researchers capable of conducting the type of studies in technically sound ways, developed some protocols, and completed the business exercises necessary to enter into contractual obligations so the work can proceed promptly. Protocols are being developed as we speak and discussed with EPA. They do seek candid, constructive comment from EPA and this SAP.

To me this looks like a sincere registrant response to EPA's newest phase in exposure and risk assessment. Industry has already demonstrated a history of commitment to getting these studies done. We spend precious time arguing over the expansive interpretations and extrapolations from snippets of data. It's time to get more data to augment the little information we have.

This SAP should lend its advice to the study designers to assure that the key parameters are incorporated into the protocols as we just discussed. This is a scientific not a political approach to resolve the debate, some of which is centered on data developed by members of this very panel. What is learned will be valuable for the assessment of so many other chemicals as well.

The individual questions posed to the Panel by the EPA are slices of perspective. When your answers are taken together, will they infer an endorsement of the SHEDS-Wood model and the overarching SHEDS model? Will your answers infer approval of the process take here to develop policy with this level of validation of a model being used exclusive for policy making decision?

I hope the panel will step back and consider if the model is representative of the population. Is it representative of real-world exposure scenarios in terms of occurrence, frequency, duration, periodicity, and magnitude. In my opinion, the model may not be presenting representative analyses, and we just don't know how great the consequences may be if other approaches were used.

So when is a model ready for primetime? Part of the answer, as I said, is in the need for the Agency to make a final regulatory decision. Are there any benefits to the public health in making a decision now? Are there any benefits to the public and to the Agency to getting further validation before taking the steps? Are there any penalties to the public health from delaying the final decisions? Are there any penalties to the public or the

Agency for making the decisions final now?

Let's consider need. The top two, this two rows of this slide show the risk calculated by EPA using the SHEDS-Woods model and the EPA's cancer slope. The bottom row displays dietary and water consumption risks calculated using the background arsenic intakes modeled by Exponent multiplied by EPA's 3.67 slope factor.

No matter what percentile of the population one considers, the point here is that the contribution of risk, be it playsets or decks, is a small fraction of the overall risk. This does not argue for a compelling public health advantage to get a regulatory decision and public policy set now in lieu of the validation of the model that yielded these answers.

The case for urgency is weak. Using the SHEDS model, CCA is not the primary pathway for children's exposures. Unlike the organophosphates, the exposures scenarios under discussion are not one of acute toxicity nor is there an avenue for immediate cessation of exposure. And lastly, is decision does not delay any other agenda such as a regulation of a whole family of compounds.

The Agency is setting a dangerous precedent by using only the SHEDS probabilistic model for this policy making.  Their stated objectives are to consider risk mitigation options, and then inform the public. Yesterday, Dr. MacIntosh asked about the evaluation of the SHEDS-Woods to date.  I hope the Panel will consider if the limited steps outlined in Dr. Ozkaynak's reply are sufficient, quote, "Evaluation and validation towards such bold use of the model at this time."

The Agency has recently recognized the important of making models widely available to the public especially for serious public policy and risk management decisions. SHEDS-Woods is available only in theory.  Dr. Ozkaynak the complexity of the model, the expertise needed to load and run it, not to mention the expense and difficulties inherent with the SAS platform.  So the public has not had a spin with the model; has this panel?

The Agency used multiple models in its

consideration of the organophosphates and understood the reasons for the differences in the answers projected by those models. By comparison, CCA has not been evaluated in any other probabilistic model. I did not think the comparison of answers from the probabilistic model to answers from deterministic approach counts as model comparison. Even that comparison was selective. There was no comparison between the 95th percentile versus the reasonable maximum exposure as Dr. Beck showed you. There were no comparisons when the same underlying data sets and assumptions were used.

Even the SAP have not run the model, I think, or played with the alternative assumptions. They haven't kicked the tires, so to speak. This case deserves the same level of model evaluation and comparison and inspection as was given any other models during the 2001 model comparison workshop.

EPA justifications of their assumptions and approaches are not clinchers. In fact, we do not know if alternative assumptions would make a difference, and the

EPA discussion does not change that fact.

Dr. Ozkaynak said, We need to assure ourselves that the assumptions are reasonable and the analyses make sense. I couldn't agree more. So let's do a biomonitoring study and test the model approaches because it matters.

The use of any probabilistic model is opposed by some because there have been little validations of such approaches. The EPA has some pride of ownership bias to overcome in its application of this model to its policy making. In its rush to demonstrate its policy utility, we may miss a unique opportunity here to get a model validation study and use it to meet the critics of model use.

All chemicals pose some risk. And we'll be in a situation soon where EPA will evaluate the risk of the next chemical. That analysis could be steeped in the same debate over assumptions and biases and representativeness, et cetera. That regulatory decision will inherit the same problems unless we get it right now.

Lessons learned in the studies will aid in the development of all models and may guide future assumptions and extrapolations when such need to be made for other situations. Obviously, we think the dynamics between need for an answer versus needs for validation pull strongly towards needs for validation. This model also needs more time on the desk of all stakeholders who can explore the consequences of alternative assumptions or data usage.

Also, let me put into perspective the time needed for harvesting key information from the biomonitoring study. The improvements in the model have taken about 14 months. That's in addition to the years for the initial version development. The biomonitoring studies can begin to yield important data in one year at which time the EPA can see if the results support their modeling approaches or if the model is failing them in any critical way. Rather than validating by edict, let's evaluate with data. Do the study. Let the chips fall where they may.

Together with the industry and other interest groups, I hope the SAP joins in instructing and requesting the Agency to, one, evaluate all of the reasonable differences in data distributions, assumptions, and model operations, and make those analyses public. Two, require a biomonitoring study and the parameters of that study and impose a strict schedule for delivery. And, three, consider the impact of the validation results in relationship to this model and others before making public policy.

Thank you for the opportunity to present this perspective.

DR. HEERINGA: Thank you, Dr. Chaisson.

Are there any questions? Dr. Steinberg.

DR. STEINBERG: I think this again comes back to the biomonitoring model or the biomonitoring pilot or preliminary study. And again this is done quite routinely in science on a daily basis. This is called "peer review." And the way that one would get a study done like this is one would put out their request for proposal and agree on a committee where all the stakeholders can be represented and get a good, competitive open review and get good studies that come in which would deal all the parameters.

I think it's easy. I think it's straight forward. It is a gorgeous model. It works in science. And I think that would be, of course, the optimum recommendation that any group of scientists would seek to do.

And, obviously, if your remarks are open to a suggestion like that, I would urge you to look at some type of solution like that. I think that is the most optimum scientific solution that has everyone's concerns and rights involved. And I think would work tremendously.

DR. CHAISSON: Let me make it clear to you. I have no more influence on this group than you do. I'm speaking as a modeler. And I'm very interested in throwing in my two cents to what I want to see in this model, too. So I guess I would join you in laying out some gee-whiz-bang, I really want to see this kind of thing.

Because, obviously, my purpose here is going to be to take a look at how the data were applied in this model versus the study. So, obviously, I want to see the right parameters in there to learn what kind the assumptions work and which ones don't. Now, I'm going to apply that not to the SHEDS model. I'm going to take it home one of these days and hopefully apply lessons learned to a different model.

But your point's well taken. And I would strongly encourage this committee and maybe anyone else to lend the advice. I think there's some people here who could give them great advice as to how to proceed. Or if not to procedure, the kinds of things. I'm also want to tell you that I'm not an epidemiologist. So these studies are mysterious to me in many ways.

But the point is that I think you're right that we think we need to get that done now. It would be a real shame to get this study done, come back here, and have everybody shooting at it.

DR. STEINBERG: There's no question that as the study stands, I think people have significant qualms. I think what people would like to do is resolve this. And I have no doubt that we have the intelligence and the ability and the people to sit down and do this. And I think that's our plaint. And I think again --

DR. CHAISSON: That would be great. That would be great. I would like to see that. As I understand the situation, they are openly seeking advice.

DR. STEINBERG: I think that advice has to be translated --

DR. CHAISSON: I don't want to speak for them.

DR. HEERINGA: Dr. Sharma.

DR. SHARMA: I think those are reasonable suggestions. And I do urge the Panel to give us the comments. And what we need to do is to look at what a realistic time line is then to deliver such data as long as the Panel will consider such data for inclusion into the model.

DR. HEERINGA: There will be a discussion in response to Issue 11. And, of course, our response is directed to the question posed by the EPA. But it's a general response. And I think can be taken in a general context when it's made.

Yes, Dr. Reed.

DR. REED: This is a curious question. In your opinion, if a CCA risk assessment somehow has to be done today, would you recommend that the point estimate approach instead of a distributional approach be done or a scaled-down distributional approach be done instead of a large model.

DR. CHAISSON: I'm going to answer that question two ways. And one is from a public policy point of view.

If I was EPA, I wouldn't put in peril a regulatory public policy and the model I've invested in so heavily by risk using it before its time, like opening a fine wine before its time. Because if you're wrong, you're going to lose a lot of credibility on the model. And I think we've brought up enough issues here that, if I needed to have an answer today on CCA, I would preserve -- I would go ahead and explore these issues with the model.

But I think I would revert back to using a deterministic approach. And that's from -- I'm looking at this from sort of a policy point of view.

The second way to answer the question is I suppose you're requesting asking is what do I think of the model in predicting the right answer. I don't know what right answer is here. I think there is an egregious error in one step that they've taken. I've looked at the CHADS diaries a lot, and I've looked at the things like the INHAPS data and other data bases like this. This is not the first time we've encountered this dilemma.

The approach as you well know in Lifeline is that we've set up multiple databases and found a way to work with one database and augment it with other pieces of information rather than let that database stand alone with its strengths and weaknesses. I think that the minute they set up the risk assessment so -- I think the minute they set up the parameters such that you've got eight simulated kids which drive this analysis which then forced it to be always -- it's not a real-life situation.

I mean any kid has an opportunity to be playing on a deck, the neighbors, your own, the one at the vacation house, the one down the street, the one at school, or whatever that's CCA-treated. And they may never see another deck like that again or another playset. Or some poor kid may always encounter for every day of his life treated woods. That's fine. But you do that by multiple population simulations.

I think that setting it up, forcing the model to use this been-there-on-the-deck-all-the-time versus never-on-the-deck-never-on-the-thing makes everything that flows thereafter wrong. And there's no way to fix that. That's such a fundamental problem in my way of thinking, happening so early in the process in the model that it just defeats anything else that comes after it.

So really what they've is 18 percent of the kids who play on treated decks, 95th percentile slice of that. And it's worse than that 18 percent, they're the risking for all of us because the rest of the kids don't see it at all. And that's just not realistic at all.

So the way they've used the CHADS diaries and the way they've not applied other information sources like frequency of playing out of doors by temperature, never mind who the kid was or whatever, by age and by temperature, a week day, a weekend, school, in school, not in school, et cetera. I know in Arizona, for example, I'm not an educator, but in the summertimes, the preschools down in Florida don't let the kids go out and play on some of the decks because of the -- I don't think they're worried about the deck decks. It's the activity in the sun. And there, the spring and fall may be the time where there's the most time. But they may not be dressed the same way, but maybe they are.

But those kinds of parameters shouldn't be assumed. You should work the data and see what falls out of it. And so rather than assuming something, forcing the data to fit it and then developing the model from there, I think is such a egregious error that I don't know what the value is of the answer that comes out of it.

DR. HEERINGA: Thank you, Dr. Chaisson. Dr.

Sharma --

DR. CHAISSON: Thank you very much.

DR. HEERINGA: -- do you have another presentation, public comment.

DR. SHARMA: Yes. Thank you. The last of this series of presentations, I'd like to introduce Dr. Leonard Smith who is a wood technologist and will be talking on the topic of coatings. The EPA risk assessment does include the topic of risk mitigation and they have presented hypothetical coating scenarios. And I think it's important for us to realize the reality of the situation with respect to coatings. And I think that Dr. Smith can adequately address some of issues that are prevalent in this situation.

DR. HEERINGA: While Dr. Smith is preparing here, just an administrative note to everyone here. I think that what we will do in terms of our agenda is have Dr. Smith's presentation and discussion. And then we will adjourn for lunch and return to complete the public comment after the lunch.

Dr. Smith.

DR. SMITH: Good afternoon. I'm Leonard Smith. I'm an associate professor at the State University of New York in Syracuse, College of Environmental Science and Forestry. I've been teaching and doing research in wood and wood coatings for the past 40 years. I would like to give you a view from wood technologist's point of view of coatings for wood.

DR. HERRINGA: Panel members, you should have copies of these slides.

DR. SMITH: Ladies and gentlemen, you've heard about coatings to be used as a sealant for CCA-treated wood. EPA and CPSC are currently conducting coating performance evaluations for some sealants for a 99 or 95 percent reduction in residuals on CCA-treated wood.

However, the primary goal of all current coatings available on the marketplace for wood is to protect the wood from the degrading effects of weather. There are hundreds of these products available in the marketplace. And the coatings are not sealants, that is, they are not designed for this purpose.

Let's look at some weathering effects on wood. Horizontal surfaces of decks receive the harshest weather. Surface erosion rates for these horizontal surfaces are two to three times those for vertical surfaces. Seasonal variations, spring and summer present the most severe weather for wood outdoors. The sunlight breaks down the wood structure at the surface. Wetting and drying of the wood caused by rain the frequency of rain swells the wood which in turn stresses that wood and the coating that might be applied to the wood. On the other hand, when the wood shrinks as it dries, it again places a new set of stresses on both the wood and coating.

In geographical variations, we've heard about cold climates versus hot climates. This also pertains to coated wood. In fact, in one study the specimens in Mississippi failed much more rapidly than those in Wisconsin. Wood species variations are another aspect, and, basically, the ability of the coating to remain adhered to the wood. Some species apply a lot more stress on the coatings because of their greater expansion and contraction than other species and their greater strength so that they can exert more force on the coating.

Weathering of wood in the sense of these decks are already weathered. So it's a challenge to apply a coating to already weathered surfaces and consider the fact that it is usually only on the top surface that the coating as been applied as opposed to the other six surfaces of each board. A good sound substrate is important for the adhesion of the coating. Weathering weakens this wood surface and the adhesion of the coating has been determined to be reduced in as little as two to three weeks to exposure to the weather.

Secondly, weathered wood is known to be a poorer surface than new wood for any given coating. Finally, southern pine is especially susceptible to weathering. The southern pine does not allow coatings to adhere well to the summer wood cells especially.

Horizontal surfaces of these decks and also playsets receive this harsher weather resulting in an erosion of two to three times greater than the vertical surface. This erosion also affects the coating.

On a macroscopic scale, the weathering process to the human eye is, in the beginning there is the natural color of wood. This color gradually changes to a gray color and a rougher surface. In this case, in five years as an example of uncoated new wood.

On the microscopic scale in Figure A, we have the unweathered wood in a microscopic view. In Figure B, we have the sunlight degrading principally the spring wood cells, those that have grown during the spring of the season. In Figure C, the rain washes away some of the cells at the surface. More of them being early in the season or spring wood compared to the later season summer wood cells.

In Figure D, we're left with the eroded surface; namely, that more early wood cells have been removed compared to the later summer wood cells. The summer wood cells form the ridges and the valleys between them, give the erosion that has been named in terms of these weathering of wood surface.

Now let's look at the coatings. And, basically, I'm going to consider the choices of coatings and the performance of the coatings on wood for the purpose of reducing the effects of weather.

Coatings are divided into categories. The first being film-forming coatings. Those are the ones that form a measurable thickness of coating material on top of the wood surface versus the penetrating coatings that form an imperceptibly thin film on top, the main part of the coating penetrates into the wood surface. Both types have an opaque, which means that it covers the entire surface, a semitransparent, which means that part of the wood grain shows, or a clear, which means that all of the wood is visible beneath the coating.

They're formulated into water base or oil base. And they may or may not contain a mildewcide for the resistance of mildew growth on the surface. Within each type, there are hundreds of coatings that are available commercially on the market. So how do we choose based on performance criteria? What coating should be used?

Coating performance depends on the type, for example, film-forming versus penetrating. It depends on the specific ingredients and the formulation of the coating. And this varies widely within any single coating type.

For example, a varnish, there are many different varnishes on the market or there are many different formulations of semitransparent stains. It also is affected by the substrate, as I've mentioned, southern pine, and the local environment. Hot, cold, sun, partial sun, or total shade. It is also affected by the high-use areas, traffic on desks or hands on play surfaces.

The examples of performance studies are shown in the next slide. This is the rating by "Consumer Reports." They took 36 coatings on new wood and weathered them for three years. The results of their study are that the coating performance varies widely. It varies between groups and within groups. In this particular case, they rated from very good to unacceptable. Another important aspect of their study is that 11 out of 36 coatings in their study are already no longer manufactured. They've either been discontinued, or they've been reformulated.

In this particular case, oil-based semitransparent stains are representative of one category of coating. They were applied to CCA-treated wood surface on two-year weathering study. At the end of two years, one of these semitransparent stains was classified as good because it had 60 percent of its original coating remaining on the wood. And the variation goes all the way down to 20 percent, which was classified as poor because most of the semitransparent stain was removed within the two-year period. This illustrates that any one coating is not representative of its coating type.

The U.S. Forest Products Laboratory has done much research and published it's findings over many years in excess of 50 years. It discourages the use of film-forming coatings which would include your paints, solid color stains polyurethanes, and varnishes on wood decks. Let's examine some of the reasons for this.

Coatings are designed to protect the wood surfaces against weathering, and they are normally designed to be applied to all the exterior surfaces of the wood that are exposed to the weather. The service life is relatively short especially in horizontal exposures. Proper preparation and recoating can be very difficult. The failure of some coatings in these film-forming varieties are by cracking, blistering, and peeling.

Finally, once chosen, changing the coating type is difficult. For example, if the film form is applied to the wood first, it is not possible to apply a semitransparent stain and expect any penetration of that stain thus negating the principle for which the stain was designed.

This is an example of a clear varnish and the peeling nature of a clear varnish. In addition, coating wear and mar in high-use areas such as in traffic areas or in playsets.

This is a weathered wood surface. You can see the gray. There are cracks in the wood. And these cracks are of such a nature that a film-forming coating applied to this could not possibly form a continuous film. When the wood expands and contracts, even if you have initially a continuous film form over the crack, the coating will crack and allow water.

Secondly, you see the nail fasteners in this slide, they restrain the wood movement. Therefore, when the wood becomes wet, they introduce additional stress by restraining this movement and leads to additional cracks as a result of that higher stress.

This is an example of a high use area where wearing away of the coating has taken place. This will give you an indication of what refinishing requires in terms of the next stage after this coating has failed.

Another source of failure especially in wood decks that are existing is at butt joints. You cannot coat the end grain of these boards because it's already formed into a deck. This leaves pathways for water to enter the wood, become trapped under the coating, and lead to early failure due to expansion and contraction and the stresses that the expansion and contraction placed on the wood and coating.

Recoat preparation. This is what the owner faces when film-forming coatings fail.

I would now like to turn our attention to the penetrating coatings. I'd like to discuss the types of penetrating coatings, the life, and the failure mechanism of penetrating coatings.

Colored penetrating coatings have a small amount of pigment to add color primarily and some mildewcide present to retard mildew growth on the surface. Semitransparent stains have more pigment both for color and for some protection against ultraviolet light because the pigment reflects some of that ultraviolet light away from the wood. However, it still allows wood grain to show. There may or may not be a mildewcide present depending upon the individual semitransparent stain.

There are clear penetrating finishes called "water repellents." And in this case, they have no pigment. They have no mildewcide. Secondly, there is the water-repellant preservative.  In this case, the preservative refers to the mildewcide present in the coating.  And this mildewcide is to retard the growth of mildew.

A clear penetrating finish on new wood is illustrated by the fact that water beads on the surface of the wood.  Note that the presence of cracks in the new wood would also hinder a film-forming finish in this regard because the cracks are already present.

Semitransparent stains are intended to try to avoid the problem of cracks that film-forming finishes encounter with cracks in the wood.  The semitransparent stains have some color, and they allow the material to penetrate into the wood to some degree; but you will note both cracks in the wood and cracks associated, in this case, with another mechanical fastener, screws.

Penetrating coatings form a very thin film on the surface of the wood unperceptively so.  Their service life, therefore, is short because there isn't much there. Six months for water repellents and water-repellant preservatives are normally reported as the life of a coating. On the other hand, because of the added pigment, two to three years in semitransparent stains have been reported in studies for the life the coating.

The failure mechanism is different from the film-forming coatings. I'd like to look at how the penetrating coatings fail in the next slide please.

A wearing away of the coating is reflected by the loss of water repellency. This is not always noticed by the owner in that the owner, therefore, does not realize that the coating needs to be refinished at this stage. Another sign is mildew growth. Mildew will increase in growth rate because the mildewcide that is on the surface is also lost with the coating. Color fading in high use areas is reflected in the loss of UV protection that the semitransparent stain would offer to the wood. And, therefore, the wood will begin to experience that greater UV weathering.

This is an example of the nonuniform wearing away of a semitransparent stain on various wood decking boards.

This is an example of the water repellency, the water beading in the foreground but the water penetrating and being absorbed by the wood in the background. This absorption of water by the wood leads to an increase in moisture content of the wood. The wood will dry out at the surface first, but the water that has penetrated deep into the wood will still be there. This results in the tendency for the wood to cup and is one of the stresses that coatings are trying to reduce such that the wood will not cup and eventually crack as a result of high stress.

This is an example of the mildew growth on the deck to illustrate that it is a nonuniform, modeled, black appearance. And mildew does not generally grow uniformly across the surface.

Conclusions. Coating a deck is not a simple task nor is it a one-time occurrence. Recoating is going to be required. It depends on the life of the coating. And this case, the life is defined as the protection the coating is offering to the wood.

Film forming coatings simply don't work. And the effective lifetime of a nonfilm-forming penetrating coating is limited, usual usually two to three years. Failure of penetrating coatings, as I've mentioned, is not always easily recognized by the owner.

Short-term studies will not model the life of a coated deck. Some coatings, their lives will extend beyond the life of the study. It's unrealistic to expect the commercially available coatings will deliver the performance assumed in the EPA risk assessment, namely a 99 or a 95 percent reduction in the amount of chemicals on the surface of the CCA-treated wood.

Therefore, the recommendations. My recommendations would be that the existing coating performance data are not adequate to support any national policy recommendation. Any national policy decision needs to be based on scientific data demonstrating the effectiveness of coatings. And sound science requires the completion of the current EPA-sponsored field weathering studies until the coatings fail.

And, finally, additional studies to address the practicality of recoating and performance variability in different geographical regions of the country are equally important to the life of the coating and to the effectiveness if they are considered to be a sealant.

Thank you.

DR. HEERINGA: Thank you very much, Dr. Smith.

Are there any questions on Dr. Smith's comments and presentation on the part of the Panel? Yes, Dr. Lebow.

DR. LEBOW: Thanks for the interesting presentation. A lot of that data came out of our lab. And I think that in general most of what you've said is a pretty accurate representation. I think that in some cases I think it's not always so cut and dried. If you have a sound surface and a vertical surface, a film-forming finish may provide protection for many, many years.

But you're right on the horizontal surfaces, these film-forming finishes, because of their problem with refinishing, may not be a real great choice. And the problem here is that these are the finishes that are most effective, appears to be the most effective, at prevented the release of the arsenic.

And I also agree with your assessment that in order to really judge the long-term efficacy of these finishes, the evaluations need to be long term because I think you do need to consider the implications of refinishing. As you pointed out, some of these systems need to be sanded or scraped to reapply. And that is going to be probably something to be avoided.

On the other hand, I don't think it's all bad because I do think, as you mentioned, semitransparent stains because they can be applied with very light surface preparation. And because they can be applied effectively to weathered wood, do have some potential in this area. That is something I wanted to point out.

I think you mentioned that weathered wood does not work with any of these finishes. Actually, I think that the studies at the lab show that it actually absorbs more finish when its weathered. And I think it's important to also say that the pigment present in those finishes has the added benefit of preventing the UV damage which, I think based on these residue studies, may actually help prevent the formation of the residue.

So although in general I agree with you, I don't think it's completely hopeless as far as the finishes.

DR. HEERINGA: Thank you, Dr. Lebow. Yes, Dr. Stilwell.

DR. STILWELL: I'd like to agree with Stan and you and a lot of the problems here. Next week, I'm going to get a phone call from somebody and they're going to say, I have a two-year-old crawling around on my deck and I want to paint it. And so I would ask if you have any recommendations to them right now.

DR. SMITH: I believe that there is insufficient data available to make such a recommendation based on the fact that the purpose of the coating has been changed from protection of the wood to elimination or reduction of chemicals on the surface of the wood. And you have to take into account these preparation techniques, although semitransparent stains can be refinished, you have the variability of the type of chemicals in washing the surface.

There are many deck brighteners and cleaners available on the marketplace. Then you have power washing of the deck and the variability of whether the power washing is a very high intensity, 3,000 PSI. I've seen as high as that pressure versus 1,000.

You have the difficulty of mildew growth, and the fact that mildew growth is generally more difficult to remove than dirt accumulated on the surface. So you have the possibility that someone will use the power washer more intensively, either by frequently going back and forth across the surface or the mildewed area, as opposed to a light intensity on the nonmildewed area which can cause differences in the weathered surface as prepared.

So you have to take all of these things into consideration. And then look at the film-forming or the penetrating finishes, and what they may do after this.

So the studies I've seen have just gone for one or two years. Most of them have not been on weathered wood to begin with. And none of them that I know of are considering the refinishing, and then the performance of the refinished deck as a total process to be able to recommend.

And, finally, with respect to the individual performance of a coating, I showed that one study done at the lab, the semitransparent stain varied from good to poor depending on the commercial stain. So just saying one category of semitransparent stains, you're not indicating how good the individually manufactured coating within that category will perform.

DR. SHARMA: Can I just add to that? We're still debating whether or not coating or no coating that there's even a risk out there. So I think we shouldn't forget that before we say what should we recommend to the public when we haven't yet determined this risk assessment represents what's truly being seen out there.

DR. HEERINGA: Dr. Stilwell.

DR. STILWELL: Regardless of what the EPA says, I mean people will want to do something about it. So I don't think that's -- people actually call up, and they want an answer.

DR. HEERINGA: Dr. Styblo.

DR. STYBLO: I would like to touch another aspect of mildew growth. In addition to mildew, there are other classes of microorganism that are known to populate the surface of CCA-treated wood including bacteria and algae. And among all these three classes, fungi, bacteria, and algae, there are no types or varieties that are able to methylate arsenic, convert trivalent arsenic which may be behind some of these surprising data on arsenic 3 leakage. They're also to methylate arsenic to arsine, volatile toxic gasses.

I was wondering if you would know if a simple coating would limit, at least for some time, growths of bacteria and algae on the surface of wood.

DR. SMITH: No, I don't have a background in that.

DR. HEERINGA: Thank you very much. Dr. Macdonald.

DR. MACDONALD: Some of the coatings seem to make the decks extremely slippery. Do you think we need to be in a competing risks model now when looking at the risk of serious injury?

DR. SMITH: That is an aspect that the deck coatings as opposed to other coatings. There is a special series for deck coatings in horizontal surfaces and that is one of the primary considerations in addition to wear, namely the coefficient of friction on the deck.

DR. HEERINGA: Sounds like a risk management question, an entirely different genre. Yes, Dr. MacIntosh.

DR. MACINTOSH: I was hoping you could educate me and perhaps some other members on the panel of the CCA-treatment manufacturing process if you can. Would you describe to us physically how this wood is treated and it has a life history at least of the beginning of this type of wood?

DR. SMITH: In this particular regard, I am not an expert in and have not studied the CCA process in developing the material. I have spent time working with wood material but not developing and actually treating the wood prior.

DR. HEERINGA: Dr. Macintosh, in the interest of time, I think in the previous SAPs there has been a full discussion and presentation by industry on the treatment process. And I believe that the docket for that would contain that information so we can provide that to you. Dr. Sharma.

DR. SHARMA: Can I just make one final remark?

We've heard from Dr. Smith, and I think also from Dr. Lebow that, you know, if we are going to do coating studies, it's important to take them out to the full term of two years and wait until the coating fails. I think the Panel should look at that time line. That time line then goes out to spring of 2005. That also gives us the opportunity to generate what we've talked about in previous presentations, which is really the exposure data through a type of biomonitoring study.

So I think we do need to look at those two time lines together. And I think it does offer an opportunity to provide data on both fronts and not jump the guns to finalize any risk assessment at this point. Thank you very much.

DR. HEERINGA: Thank you very much, Dr. Sharma and Dr. Smith, for your comments. At this point in time, I would like to adjourn for a lunch hour. And before we do that, we have a comment, some announcements from Paul Lewis, designated federal official.

MR. LEWIS: Just for the members of the public and the Panel, this room will be open during the lunch break during the next hour. So I would advise you to take any personal belongings with you. And for members of the public that have not preregistered with myself, please contact me during lunch break or members of the SAP staff here to register. Thank you.

DR. HEERINGA: Thank you very much. And we'll see everyone back here at 1:40.

[Lunch recess at 12:40 a.m.;
meeting reconvened at 1:45 p.m.]

DR. HEERINGA: Let's reconvene for the continuation of the public comment session of this meeting of the Science Advisory Panel.

Before we begin, just a few announcements for Panel members and also of interest because these materials will be in the docket. We have mentioned this morning the paper on the nonlinearity of the slope factor. That paper is contained in the white binder, the supplemental references that you received. If you don't have a copy of it, see Paul Lewis. The question on the slope factor itself, the paper that's under review, we're working on getting that released. But we do not have a release on that to distribute that at this point.

In addition, there are several other things that have been put at your places over the noon hour break. The first is a series of distributional charts actually prepared by Dr. Lelia Barraj of Exponent. They just show the distributions as the occur within the SHEDS system.

Again, caveat emptor, these are Dr. Lelia Barraj's analyses and provided for your information by Exponent. But again, they would certainly need to be independently verified in your own work reporting.

And, finally, there is a handout that relates to a future presentation that's part of the response to questions by Dr. Hattis showing some distribution fitting plots. And so those added materials just to explain the nature of what's showing up.

At this point in time, I'd like to move on to continue our public comments. I'd like to invite Ms. Jane Houlihan of the Environment Working Group, if she's present, to come to the mike and provide us her presentation.

MS. HOULIHAN: Thank you. I'm Jane Houlihan, Vice President for Research at Environment Working Group. And we're a public interest research organization based here in D.C. And I've spoken to this panel before so I recognize many faces.

First of all, I'd like to thank EPA and their contractors for constructing an exposure risk assessment that I think really does significantly advance the understanding of the cancer risks faced by children who contact arsenic from decks and playsets. So thanks for all the hard work that's gone into such a sound assessment.

In October of 2001, Environmental Working Group recommended that the Panel move forward with recommending to EPA that a probabilistic assessment be the way to go. And the Panel recommended that. And we're pleased to see it and hope that EPA adopts this kind of methodology agency-wide for its exposure and risk assessments.

As you all know who've read this document, EPA's assessments show that a substantial fraction of children face a fairly high cancer risk from their contact to these structures. For instance, in warm climates, in 1 in 10 children face a cancer risk of at 1 in 10,000 according to EPA estimates. That's a substantial number of children. And given that risk, we would hope the Panel would recommend that EPA move forward rather quickly with developing advice to consumers, schools and communities who are looking for sound advice on mitigation measures.

With that said, I think there are a number of key areas we find in which we believe EPA may have underestimated risk in this model. And I would just like to outline those three areas briefly.

First of all, and I hope that the Panel can make recommendations in each of these areas. First of all, I think EPA should incorporate into this assessment its own latest guidance on increased cancer potency for early life exposures. In March of 2003, EPA released new cancer guidelines. And in these guidelines, EPA put forth its assessment of 23 peer-reviewed studies of earlier life exposure to carcinogens including a study of arsenic. EPA found increased cancer risks in early life resulting from early life exposures and in their guidelines recommended that their risk assessors use an extra potency factor of 10 for exposure of infants up to age 2 and an increased potency factor of 3 for exposures from age 2 to 15.

In particular, I want to point out -- I have detailed comments that I'll leave with Paul afterwards. But I'm just pull one point out from this. And that is one of the studies that EPA reviewed of the 23 studies was a study of arsenic from the National Cancer Institute of early life exposures to arsenic that resulted in increased incidents in later life of lung, liver, adrenal gland, and ovary tumors. So I hope that the Panel can consider making recommendations to EPA in that area to incorporate its guidance on additional potency of carcinogens in early life exposures.

The second point I'd like to make is I know this is not the charge of the Panel. But I would like to discuss just briefly the latest National Academy of Science's recommendations on the potency on the arsenic as a carcinogen.

As you know by now, in 2001, the NRC released its latest review of the potency of arsenic. And in particular in this review, the NRC found that the most recent evidence strengthens the evidence of a line between bladder and lung cancer in arsenic and that even very low concentrations of arsenic appear to be associated with a higher incidence of cancer. And the pesticide office has, as you know, adopted the drinking water office's assumptions for cancer potency. And I'd just like to point out that the NRC specifically said that they think that this cancer potency is low. And I hope that the Panel can recommend that EPA move forward quickly in looking at the NRC recommendations and at a minimum incorporate these recommendations into an assessment of the plausible range of risks in the particular risk assessment that's the subject of these three days for you.

The third point I'd like to make is that we believe EPA should incorporate direct mouthing of surfaces into this risk assessment. There are multiple studies in the peer review literature that quantify the frequency of direct mouthing of surfaces. Most recently EPA's National Exposure Research Laboratory released a study, a videotaping study, of 186 children that showed that children directly mouth surfaces four times an hour. These behaviors are real. They're quantified. They're in the peer-reviewed literature, and they could make a big difference in the risk assessment because you could have higher transfer with direct mouthing than you could with even hand-to-mouth transfer. We recommend the Panel consider making recommendations to EPA in that area as well.

I would also just like to briefly point out that there are some high-risk populations that are not included in this model because the data aren't available. EPA says specifically, for instance, in its assessment, it's not able to incorporate the high hand-to-mouth activity for autistic children for instance because the data just aren't available for that yet. A lot of work has been done recently on identifying genetic polymorphisms that might account for some of the differences in arsenic metabolism that's been observed in populations. I know Dr. Potion at the University of the Arizona recently identified a bimodal distribution for arsenic metabolism. And he believes that that may account for real differences in arsenic toxicity among individuals.

Children get arsenic on their clothes. That's not accounted for in this model. They track arsenic into the house. That's not accounted for in this model. So there are lots of behaviors and particularly high-risk behavior that are not included in this model. And I would just ask you to remember that, as you make recommendations on ways to shift and change parameters in this that some of the kids who are most likely to develop cancer later in life from these exposures are not included in this model because of data limitation and other reasons.

And, lastly, I would just encourage you to recommend that EPA move forward in finalizing this risk assessment. I don't think we need to delay for further studies. We have solid evidence that arsenic is on the surface of the wood, that it adheres to human skin, that kids do put their hands in their mouths. So there are exposures to arsenic.

I think EPA should modify your models based on your findings. I don't think they should hold up on finalizing the models because these are real risks.

Children who are exposed to arsenic are developing cancers later in life as a result of the exposures. Quantifying that is the issue. But the finalization shouldn't be held up for further studies.

And so I would just urge the Panel to make that one of their recommendations.

DR. HEERINGA: Thank you very much, Ms. Houlihan, from the members of the Panel thank you, very much.

Our next public commentor speaker is Helena Solo-Gabriele from the University of Miami. And she's presenting on behalf of University of Miami, University of Florida, Florida International University Collaborative CCA-treated Wood Research Project.

DR. SOLO-GABRIELE: I have a PowerPoint presentation. And I'd like to begin by thanking the EPA and the SAP for this opportunity to present the following information.

DR. HEERINGA: Dr. Gabriele's presentation should be available to each of the Panel members in a handout.

DR. SOLO-GABRIELE: I'd like begin by stating that I've been working on environmental issues associated with CCA-treated wood for the past seven years or so. And this work has focused primarily on disposal issues associated with treated wood. But more recently over the past three years, we've been focusing more and more on in-service issues associated with CCA-treated wood.

Throughout this time period, I've had the opportunity to work with many researchers on this issue. Tim Townsend and myself have worked over the seven-year period on disposal issues. He's from University of Florida. Yong Chai has provided expertise on arsenic speciation. He's a chemist from Florida International University. Laura Flemming from University of Miami Medical school, and Stuart Schlat of Rutgers University, are leading the biomonitoring work that's currently work in progress. And David Hahn has provided us with expertise on identifying treated wood in the field.

I'd like also to acknowledge our funding sources which include Florida Center for Solid and Hazardous Waste Management, the Florida Department of Environmental Protection, and the National Institutes of Environmental Health Sciences.

The information that I'm presenting here is essentially hodgepodge of information that comes from several different studies that I consider to be relevant to the SAP. And these topics include disposal issues in arsenic quantities associated with CCA to provide a more holistic approach or review of the overall CCA impacts.

Speciation of arsenic and leachates impacted by CCA-treated wood. In particular, when I was going through some of the comments, I notice a lot of speciation questions were coming up so I thought I'd present on that.

Also I'd like to discuss briefly our mulch ongoing study evaluating mulch. Mulch is a very common buffer material that is used to line playgrounds. I'd also like to briefly describe dislodgeable arsenic and then close with a brief status on our biomonitoring study.

As we all know, arsenic is an element. It remains in the environment indefinitely. Once it's imported into the country, it's here to stay. And within the disposal sector, right now the strategy is essentially a dilution strategy. CCA when it's in the wood on occasion will be lost during in-service leaching or through dislodgeable processes. Some of the CCA-treated wood inadvertently is recycled. For example, in Florida CCA-treated wood inadvertently gets into the mulch that is produced from recycled dimensional wood. Also it gets into the wood that is used for energy production, recycled wood for energy production. There are also losses in that point as well.

And then once the wood makes its way to its ultimate disposition, typically within a landfill, the chemical from the CCA-treated wood will leach over time and result in chemical contributions to the leachate. Leachates from landfills are typically sent to waste water treatment plants. The ultimate fate of the chemicals at waste water treatment plants I would assume that a lot of it would end up in the bottom sludges. These sludges are then typically land-applied resulting in a dilution, a continual dilution, of the chemicals upon disposal.

I also wanted to mention that the rate of loss of CCA during in-service use is faster typically on the order of several percentages per year, whereas during landfill disposal, the rate of leaching or loss tends to be a little slower. So this has impacts on the ability and of the environment to assimilate the chemicals.

To date the overall quantities of arsenic that have been imported into the United States are on the order of 380,000 metric tons for the United States as a whole. This quantity is a very, very large quantity. There are questions about whether or not our environment can assimilate this large quantity through dilution processes. Just to give you an analogy for how big this quantity is, if you take the 390,000 metric tons and you apply it to the upper one inch of soil throughout United States that will result in the increase in the background arsenic concentration of 1 milligram per kilogram to give you as sense for the size of that.

I also wanted to emphasize that we are continuing to import arsenic into the country. During the year 2001, 24,000 metric tons of arsenic have been imported for CCA production. After 2003, due to the phase down of CCA-treated wood for residential uses, there still will be an importation of arsenic at an estimated rate of 26,000 metric tons per year for the products that are exempted from the phrase down and for industrial uses.

That brings to us the next topic that I'd like to discuss which is speciation of arsenic releases.

As far as the speciation is concerned, there are three items that I'd like to cover. The first being the variation of releases speciation with respect to pH for both new and weathered wood. I'd like to describe the results of a synthetic precipitation leaching procedure, SPLP, on both new and weathered wood as well. The SPLP is the solvent that is used for the SPLP is a synthetic rain fall. The pH tests and the SPLP tests are very similar in the sense that both require that you size reduce the treated wood. You put it in contact with your solvent for an 18-hour period, and then you extract the leachate and analyze the metals within that particular solvent.

The solution used in the pH test was deionized water and with very small strong arsenic acid or strong base added to change the pH.

The last part of this speciation I'd like to describe is a field deck study, the results of our field deck study.

As far as pH is concerned, the arsenic from CCA-treated wood tends to leach greatest at the pH extremes as shown here.  These are the results for a new wood sample with a rated retention level of 0.32 PCF.  And for this particular sample, and the only species that was observed in the leachate as indicated by the green bars was arsenic 5.  There was an independent total arsenic analysis also conducted which is indicated by the yellow bars.

Also within the general pH range of the environment in the near neutral pH range, the amount that was leached or found in the leaching solution was about 5 milligrams per liter or a little bit under 5 milligrams per liter.

Same test but conducted on weathered wood. In this case the weathered wood had a rated retention level of 0.41 PCF. These results are similar in the sense that you see the highest concentrations of arsenic leaching at the pH extremes. In this case, however, as you can see from the red bars, we are observing arsenic 3 in the leachates. Arsenic 3 is observed up a pH 9.5 or so.

Also in this case, we are seeing that the amount that is leached is higher than the 5 milligrams per liter. The higher amount of leaching may be due to the higher retention level but also perhaps due to the presence of arsenic 3 in the weathered wood samples as opposed to the new wood samples.

Here is a comparison of our SPLP tests. On the left-hand side we have the results for our new wood, and on the right-hand side have the results for the weathered wood. And, again, the coloring scheme is the same as before with red bars for arsenic 3, green for arsenic 5, and yellow for the total arsenic.

Also on the horizontal axis you'll see we have different letters. And then in parentheses, there are numbers that correspond to the rated or the measured retention levels for each of those samples in units of kilograms per meter cubed.

What jumps out just by looking at the data in this fashion is that in weathered wood samples, we see more arsenic 3 within the leachates than we do with it coming off of the new wood samples. There is a little bit in some of the new samples small quantities coming off of the new wood samples; but the fraction of arsenic 3 in the weathered wood samples is larger in the faction that is observed in the new wood samples.

Also if you compare wood samples of similar retention levels, for example, we have the Sample H, which is the third one on the new side, which is a rated retention level of 24, and then Sample M, which is also 24, if I can see that correctly, which is the first sample on the weathered side. If you start comparing the averages, you'll note that for a similar retention level, it appears as though weathered wood on average is leaching more arsenic than new wood.

The results of our field scale study, our methods are depicted in this slide. It consists of a 6 foot by 6 foot deck subject to rainfall. There are two infiltration or two leachate-collection systems fitted to the deck. One includes a gutter system that collects runoff water. And then the second infiltration system or the second leachate collection system is at the bottom below 2 feet of sand. And that is routed out to a drainage port which we then collect our samples.

In this presentation, what I'd like to do is first discuss the result, go vertically, discuss first the results of the runoff water, discuss the sandy soil, and then move into the infiltrated water.

For our rain water runoff, what we have been observing as far as the concentration of the leachates is at the very beginning of the study, this was over a period of one year, we see spikes in arsenic releases up to a value of 8 milligrams per liter. The overall average, however, for all the data we've collected, weighted average based on the volume of rainfall, was .73 milligrams per liter. The spikes appeared to be somewhat random; however, when we do observe a spike, there appears to be a release of arsenic 3 associated with it that we don't see when there is not a spike.

These spikes are not correlated with rainfall depth. They're not correlated with temperature. They appear to be more random. They may perhaps be associated with some checking or cracking of the wood. We still don't understand the nature of those particular spikes.

The .73 average for the CCA-treated deck is contrasted with the ppb levels that were observed from our control deck which was primarily untreated wood.

As far as the soil is concerned, the runoff water then impacts the soil. And these are results of arsenic concentration observed with depth. And we collected sample from a 6 month period and a 1-3 month period. Concentrations of arsenic in the soil from our untreated deck was consistently less than 1 milligram per kilogram.

This data indicates that the majority of the arsenic is observed at the surface layer of the soils. And that it appears from the 6-month to the 12-month period that the concentration of arsenic within the surface soil layer tends to increase.

And speciation was conducted on the soils collected from the 13-month sample. And the primary arsenic species observed in the soil were 95 percent arsenic 5 and low levels 5 percent or so arsenic 3 was observed in the soil sample.

That brings us to the water below the soil. And this slide has different units than our runoff slide. The runoff slide was in units of milligrams per liter. This is in parts per billion or micrograms per liter. And what we can see is at the beginning of the monitoring period, the concentration of arsenic in the infiltrated water below the soil was on the order of about 2 to 3 micrograms per liter and was predominately arsenic 3. But as time has continued, what we're seeing is that arsenic 5 predominating more and more, representing the larger fraction of the arsenic observed in the infiltrated water underneath 2 feet of soil.

The general trend appears. There are some valleys. But the overall general trend appears to be increasing in time. This is important especially for the State of Florida, since our water resource, and in particular in South Florida we get our water from the Biscane aquifer. It's a very shallow aquifer. You dig a few feet, and you hit the aquifer. And what this indicates is that the impacts from CCA-treated wood are very likely or possible; and it will depend on how much dilution we have from the groundwater once the metal leachates reach that particular level.

That brings us to mulch or the buffer materials. Our early work which Tim Townsend spear-headed, in which I believe a paper was also distributed to the SAP on it, found that CCA contaminates mulches throughout State the Florida. And within that study, Tim also purchased some mulches from stores. There were three mulches that were purchased from retails stores. And he found that two of those three were contaminated with CCA.

That was of very high concern to us. And we, therefore, decided to conduct a follow-up study to look at how extensive is the contamination of commercially available CCA. These are mulches that we've purchased from stores, at retail stores, or from nurseries. So far we've collected 90 samples, and we've analyzed 20 to date.

This is an ongoing study. Of those 20, 7 were noncolored and 13 were colored. The reason we were interested in colored versus noncolored is because colored samples of the red mulches have a tendency to be made from recycled dimensional wood, because once the wood is weathered and old and it's disposed, it has a dusty color to it. So typically the red dye is added to make it more attractive.

Amongst the noncolored mulches, one contained CCA. And amongst the 13 colored mulches, 6 contained CCA in concentrations between 7 to 200 milligrams per kilogram. So what this implies from this small data set that we have analyzed to date implies that if you go to a store you have a 50-50 chance of your purchasing red colored mulches that, or close to 50-50, that you may be buying a sample that's contaminated with CCA.

All of those six samples, we've looked at those six samples very closely. They all contain evidence of plywood, so they were not made from virgin wood which we call -- virgin wood is essentially tree trunks and branches. But it was made from recycled wood, engineered wood. So it indicates that these six samples came from recycling of dimensional wood.

This is just the data that corresponds to our positive samples. In addition to being positive for arsenic, they were also positive for copper and chromium.

That brings us to mulch in the children's playgrounds issues. This is where all the disposal problems come back to the playground issues that we are discussing here. This particular playground is in Florida. And the main structure is made of CCA. There's also a see-saw made of CCA and a little playhouse in the background, which is hard to see, is also made of CCA.

This particular playground we term a "double playground" because not only is the playground made of CCA, but the mulch at the playground also has CCA in it. At this particular playground, the arsenic concentrations in the mulch were measured at 150 milligrams per kilogram total arsenic. We also, in addition to doing total arsenic analysis, we did SPLP tests on it. And we found it also leaches arsenic at 170 micrograms per liter.

And, again, if you take a close look at this mulch, there is evidence of plywood within that mulch as you can from the grains of wood going in different directions, indicating that this wood ultimately came from recycled dimensional wood.

This playground belongs to a friend of mine. I was invited to -- my daughter as well. We were invited to a birthday party at this playground. And during this birthday party, they took the swings out and they put in a pinata. And all the kids gathered around the pinata. And this pinata had the strings on it. And the children were all excited. They grabbed strings, and all the candy fell on the mulch and all the kids starting digging into the mulch and had bags full of candy and mulch.

And that particular incident happened so fast. But after the party, I had asked my friend if I could go sample her mulch. And she said, okay.

And for this particular playground, the arsenic concentration, the total arsenic concentration, was at 110 milligrams per kilogram. And similarly, the SPLP was elevated at 90 micrograms per liter.

And again looking closely at the mulch, you see evidence of plywood again indicating that the ultimate source of this wood was recycled dimensional wood.

This is another playground that we've sampled in Florida. And as you can see, where the children are playing, they're playing underneath the structure and essentially playing in the mulch. Unfortunately, we have not analyzed the total arsenic on this one yet. It's in the works. But we do have the SPLP results. And the SPLP values are elevated.

So all these playgrounds are in Florida. But I wanted to emphasize that this issue is not only a Florida issue.

We were sent mulch called "Play Safe" several years ago. This mulch came from Arizona. The father, from Arizona, found a end tag within his mulch saying that there was arsenic and poison inside the mulch. And he started doing some research on the internet and sent us an e-mail and we started communicating. And he sent us the mulch sample.

And that mulch sample, if you look at it closely, it's hard to see. The coloring doesn't come out very well. We use a pan indicator stain which when you spray it on the mulch, whatever turns a magenta red color, a strong red color, indicates the presence of CCA or at least copper in that wood sample. And you can see little bits and pieces in there that are staining a strong red color.

And also what this indicates is that you don't need that much CCA to contaminate a mulch. Relatively small amounts on the order of 5, 10 percent. You can see very significant levels of arsenic within the mulches.

For this particular mulch, again, we have not done the total arsenic analysis on it. But given the SPLP results, it also shows elevated levels of arsenic in the leachate solutions.

That brings us to dislodgeable arsenic. I just wanted to discuss some of the parameters that were provided for the warm climate scenario. In the EPA documentation, there are two values that are provided, one for cold climates and one for warm climates. And when I looked at them, I was just surprised that the warm-climate value was lower than the cool-climate value.

And, intuitively, I would think that in warmer climates the wood deteriorates faster; and, therefore, you would have more loses of chemical from the wood over time. The losses can occur as both dislodgeable and leachable arsenic.

And I went back, and I started looking at some of the raw date. Again, the cold climate number came from the Consumer Products Safety Commission and the American Chemistry Council. And the warm climate number came from the American Chemistry Council. I had the Consumer Products Safety Commission report. And I believe, it's my understanding. I wasn't able to track all these numbers all the way back to the .26 micrograms per centimeter squared of hands. But I believe the Consumer Products Safety Commission number is based upon a wipe test of 39 micrograms per 50 square centimeter of wipe. And then there's a factor applied to that to estimate hands.

Also in the literature, Dave Stilwell also did a study on dislodgeable arsenic. He found a very similar number of 37 micrograms per 50 square centimeter of wipe. I could not find the ACC report, so I can't provide a value for that.

So I plotted these on a map and superimposed on that map is wood deterioration zone indication which shows that areas in the southeast have very high severe wood deterioration potential and also the State of Hawaii.

Intuitively, one would expect that there would be more releases of chemical as you proceed towards the more severe wood deterioration zones.

We at the University of Miami and at FIU have been conducting wipe tests evaluating dislodgeable arsenic at the university. There are two stations or two sets of areas that provide us with our samples. One is what I call the "wiping station," which are the boards in the front. Our wiping station includes our untreated wood, the .25 and 2.5. And then we did wipes from our deck, the same decks that we use for our arsenic speciation study. The wipe method is consistent with the Consumer Products Safety Commission which involves the 10 strokes back and forth on weight.

And these are the results that we have so far. We've got the data for the 6 months and the 12 months illustrated here. At 6 months -- and there's three different repetitions. What we do is we do the 10 strokes then change the wipe; do another 10 strokes, that gives us the second repetition; change the wipe; and do another 10 repetitions, and that gives a third repetition.

And what we see is that the amount of arsenic dislodged in the wipes is anywhere from 6 to 100 micrograms per wipe. It appears that the average is decreased from 6 to 12 months. But this decrease is not statistically significant. I think only over time will we be able to see if there is or is not a decrease. But if you look at the second and the third repetition, the averages are essentially the same.

So superimposing the preliminary numbers that we've obtained to date, if you look at the gradient of dislodged arsenic, it appears as though possibly the amounts of dislodged arsenic it seems logical that it should increase as you go to more severe wood deterioration zone. I just throw this out as something to think about and to consider.

Other issues as far as dislodgeable arsenic is that there are many factors that influence dislodgeable arsenic. First and foremost is the retention level. Data has shown -- we've collected data at two different retention levels, a .25 and a 2.5. And we get very different amounts of arsenic dislodging on the wipes.

Also we find that there is a lower retention level on wood surfaces that are exposed. So if you have horizontal boards, the boards that are facing the top, facing the sun and the rain, you have a lower retention on those boards than you have on the boards on the side, underneath, or not exposed directly to the weather.

Also there is the issue of sap wood versus hardwood where more arsenic tends to be released from the sap wood side because the sap wood portion absorbs more of the CCA chemical versus the hardwood.

Also I wanted to emphasize that retention is variable throughout playgrounds. It can vary, according to the data that we've collected here, it can vary by a factor of 2 depending on where you obtain your retention value from. This data was collected with a hand-held XRF which provides data very, very quickly. And what we see is this particular playground has different levels in it. The very lowest level has the highest retention. There's a little picnic area, a little picnic table just above that which, according to that number, is at .24, a little bit lower. And then in the second level, it's a .25. And there's a third level, .28. But then the vertical boards are higher at .5 and .48 as illustrated at the very top numbers.

So another issue to keep in mind is that, depending on where you get your wipe sample, you can get different readings depending on the retention level of that particular part of the playground.

That brings us to the biomonitoring study which the leaders are Stuart Schalot of Rutgers University and Laura Flemming of University of Miami.

My participation on the biomonitoring study is to provide support from the environmental end to characterize the playgrounds environmentally and to collect samples, the environmental samples. And the specific aims of the biomonitoring study are to determine the levels of arsenic present in playgrounds made from CCA-treated wood, determine if dislodgeable arsenic from

CCA-treated playground structures is present on children's hands by administering hand rinses; and to determine if the levels of arsenic on the children's hands are associated with levels of arsenic measurable in the children's urine.

As far as our methods are concerned, we've gotten -- we've completed our IRB approval through both universities. We've developed a questionnaire to inquire about other possible arsenic exposures. And the questionnaires and also the educational materials developed have been written in both Spanish and English.

As far as the environmental sampling is concerned, we do confirm CCA-treatment of the playgrounds. We confirm the retention levels. We have a four-tiered approach on confirming the CCA treatment of the playgrounds. We also collect soil samples and polyester wipes. There's also information on the hands, the tracings to get the surface areas, the rinses; and also the urine samples are obtained through either a diaper insert or by use of a cup.

Our goal is to collect data on 10 subjects, 10 children from 15 to 36 months of age. A lot of materials have been developed for educational purposes both at the beginning and during follow up to explain to the parents, give them a basis for understanding the results that are obtained concerning the levels that were observed.

As far as the status, what we've done so far, we have had two children participate in the study. Within these two children, arsenic was found in the wood on their playground, on the soil, on the wipes, on the hand rinses, and in the urine. However, these arsenic samples were not speciated. And we're currently evaluating the data as far as understanding what it means.

So at this point, I'd like to ask if there are any questions?

DR. HEERINGA: Thank you, Dr. Solo-Gabriele. Yes, Dr. Freeman.

DR. FREEMAN: I understand that the first two children were from residential playsets. Do you intend to do any community play areas?

DR. SOLO-GABRIELE: Our original plan was to sample any public playground. And we got permission to -- our original IRB was to do a public playground. We got permission to do all the environmental sampling. And we had been keeping the Dade County Public Parks and Recreation informed of what we were doing. But then when it came time to -- we informed them of the epidemiologic study, or the collecting of the human samples; and at that point, they decided that they did not want the parks involved. So that's why we went to the residential parks -- residential playgrounds.

DR. HEERINGA: Yes Dr. Francis.

DR. FRANCIS: Given that there was a previous discussion about dietary arsenic, how is your study dealing with the dietary arsenic issue?

DR. SOLO-GABRIELE: Unfortunately, the only way it's being evaluated is through use of the questionnaires. And, again, this is a pilot study. We don't have a lot of funding for it. So basically the purpose is to see if there is arsenic in the urine. That's going to be one of the limitations of this pilot study. I don't know if we could even do that even if we had a good estimate of the dietary intake with 10 children. We wouldn't be able to come up with very strong conclusions on that either. So this would definitely require -- in order to come up with some definitive answers, it would require a much larger study.

DR. HEERINGA: Dr. Stilwell.

DR. STILWELL: Do you know what fraction a child would say spend on a fishing pier as opposed to a playground because a fishing pier has got a lot more arsenic on it. And in a state like Florida, people are going to hang around the water.

DR. SOLO-GABRIELE: That's a very important point. And definitely playgrounds are not the only exposure pathway, or children are not only exposed to playgrounds. And I can imagine a scenario where a child is sitting in a bathing suit, fishing for a few hours anyway in one particular day. And, again, the issue of the higher retention level on a pier would make the exposures that much more.

DR. STILWELL: Right. So you had one data there that was 1,200 micrograms.

DR. SOLO-GABRIELE: Yes. That was for the 2.5 PCF.

DR. STILWELL: That was what you would find at a pier; right?

DR. SOLO-GABRIELE: Yes.

DR. STILWELL: Or less.

DR. SOLO-GABRIELE: Yes.

DR. LEBOW: Thank you, Helena. Excellent presentation as usual.

I just wanted to mention a couple of things not so much for Helena's benefit because she already knows these but for the Panel's benefit.

First, I wanted to point out that the SPLP and the TCLP tests do require grinding of the wood and extraction of these small particles in solution for approximately 18 hours. It's a comparative method. I just wanted to make sure that nobody tried to interpret that as being relevant to a solid piece of wood or wood in service.

The other thing I wanted to point out is that she mentioned in the TCLP and SPLP, in those ground wood studies, she compared unweathered to weathered and indicated that the weathered wood was leaching slightly more. I think in studies like this, we need to be so careful because, as she mentions later, there is so much variability in the product to determine something like that, it's a fairly small difference, you would have to analyze many, many replicates. And I suspect what she saw was just a different between two pieces of wood.

The other thing I wanted to point out on the mulch issue, and this is an interesting issue that cropped up in the Midwest at one point. I think this is probably a supplier issue. And I don't know how widespread it is. The point I wanted to make on it, though, is, as she mentioned, when you get one or two pieces of treated wood in there, it raises the average concentration of arsenic. But that arsenic is not evenly distributed. It's in one chip here, one chip there. So don't be confused that all of the mulch in that playground has that high concentration of arsenic in it. One or two pieces of wood are present, and they have a much higher concentration of arsenic.

And then I wanted to also agree with her and mention about the wipe numbers. She was comparing the ACC study to some of the other studies as far as the geographical location. Again, to me looking at those wipe numbers, it's just variability. I think -- and this was something I was going mention later. As far as the warm and cold scenarios, I don't think those wipe numbers are a function of that. I think it just happened to be that set of boards probably. Now, it's possible you could differentiate that, but I think that would be incredibly difficult to do.

Finally, I wanted to mention that as far as the 2.5 PCF value. Yeah. Wood is treated with CCA to a different concentration depending on the end use. For above ground, the target concentration is usually 0.25 PCF because the exposure is less severe. For in-ground contact, the retention is 0.4 PCF because it requires a little higher concentration to protect against wood and soil. It's more of a ideal climate for the decay fungi.

For marine piles in southern waters, 2.5 PCF was used for many years. This is, in most cases, limited to the piles. But it could also be other members that are emersed in the water. The wood that is above the water in the splash zone only is treated to a lower retention. And I don't right offhand remember what it is. But it's less than 2.5.

And I just want to provide that little bit of clarification on these different clarification numbers.

DR. HEERINGA: Thank you very much, Dr. Lebow. Dr. Solo-Gabriele, do you want to comment?

DR. SOLO-GABRIELE: Can I?

DR. HEERINGA: Certainly.

DR. SOLO-GABRIELE: Yes. I agree with most of what Stan said. As far as the grinding issue on SPLP and TCLP, the purpose of the TCLP and SPLP in my opinion is to evaluate the long-term effects of a material or a waste once it's disposed in a landfill or land-applied. And the purpose of the size reduction is to accelerate the leaching process. So by no means would you expect the values SPLP to represent the runoff from a deck board. But I believe we've addressed that through the deck study.

As far as the statistics on the new versus weathered wood, I agree. Especially once we start trying to cluster the boards based on their equivalent retention levels, we start running into very small numbers. The averages are different. But statistically, we can't show it. I agree. But we can definitely see between new and weathered wood is that there's more arsenic 3 coming off of weathered wood versus the new wood.

As far as the chipping, the small piece, yes, that's correct. Not all the mulch is CCA-treated. A fraction of it is. Same is true. I agree with the splash zone CCA-treated pilings. But it's not uncommon in Florida for example, especially in the Florida Keys to find a home with a dock, a playground, a CCA-treated playground, a CCA-treated deck, and a cutting station made of CCA, to find all of that in one particular household which may be a consideration when you're looking at overall impacts to a child outside.

DR. LEBOW: Right. I found that trivalent arsenic finding very interesting because we haven't seen much data along that line. Most of it's indicated pentavalent.

Do you feel confident that there is nothing in the extraction procedure itself that could alter the valent state, or have you an opportunity to use any of the instrumentation that would allow you to do the analysis in situ, the actual wood residue or the wood itself, as we saw in some of the presentations earlier?

DR. SOLO-GABRIELE: Well, what we're finding is that the untreated wood does not show arsenic -- sometimes it will show little small amounts of untreated --

DR. LEBOW: You mean the newly treated.

DR. SOLO-GABRIELE: Yeah, I'm sorry. The new CCA-treated wood shows much lower values of arsenic 3 than the weathered wood. So as far as the solvent extraction procedure, I would have a tendency -- the new wood tends to serve as a control on the solvent itself.

The second question you had or comment?

DR. LEBOW: I've already forgotten it, but I have another one now.

Was the weathered wood that you reported on here, was that the wood that was in the Dumpster?

DR. SOLO-GABRIELE: No. These were structures that were demolished, and the research team collected those samples when they were demolished.

DR. LEBOW: They were removed directly from the site.

DR. SOLO-GABRIELE: Yes.

DR. LEBOW: I was a little concerned. In one of the papers it said something about the wood was stored in a Dumpster for a number of years which could have been more of a reducing environment.

DR. SOLO-GABRIELE: One of the samples came from a playground that was demolished. And that particular playground, a lot of that wood was put into a Dumpster. It was not mixed with anything else. It was just by itself, and it had a cover over it. The playground, I believe, at the point of being demolished was 18 years or. And I believe it sat in the Dumpster for a year or two.

DR. LEBOW: Very interesting presentation. I enjoyed your talk very much.

DR. SOLO-GABRIELE: Thank you.

DR. HEERINGA: Thank you very much. Dr. Freeman.

DR. FREEMAN: I know that this is just a pilot study and you don't have enough funding to do everything. But Dr. Schalot shared some of the data with me, and there was about a three fold difference in handloadings for the two little kids that might be within the noise once you have gathered more data. But it may actually have something to do with the behaviors since the children were of two very different ages. If there's any way that you could do some videotaping so that you could actually see what structures they were handling, that might be useful.

DR. SOLO-GABRIELE: Okay. In a full-scale study, that would definitely be a consideration.

DR. HEERINGA: Dr. Matsumura.

DR. MATSUMURA: Yes. My question was very similar to Dr. Freeman's. So you did find some amount in the hand wash.

DR. SOLO-GABRIELE: Yes.

DR. MATSUMURA: Did you compare before and after or just to make sure?

DR. SOLO-GABRIELE: The hands were washed beforehand.

DR. MATSUMURA: Yeah.

DR. SOLO-GABRIELE: Before the children went on the playground.

DR. MATSUMURA: Yeah. So you could see some difference in that case before and after.

DR. SOLO-GABRIELE: I don't recall differences in the pre and post numbers.

DR. MATSUMURA: Just to know.

DR. HEERINGA: Dr. Styblo.

DR. STYBLO: I was looking at your other study you submitted to the Panel that models wasteland or situation with arsenic treated wood in wastelands. And in one of your arrangements using lyosometers (ph.).

DR. SOLO-GABRIELE: Lyosometers.

DR. STYBLO: You found pronounced amounts of methylated compounds. I believe it was in combination with household waste.

DR. SOLO-GABRIELE: Yes.

DR. STYBLO: I was wondering, since you do speciation beyond arsenic 5 and 3, I was wondering if you saw any indication of methylated arsenicals on your structures or in the leachates? And that is the first part of the question. The second part of the question: Do you plan to do speciation beyond the arsenic 5 and 3, inorganic arsenic 5 and 3, in urines? If you do, do you have a capacity for looking at trivalent methylated species?

DR. SOLO-GABRIELE: As far as the methylated forms, all of the analysis we conducted had at least the capability to do arsenic 3, arsenic 5, monomethylated arsenic and the dimethylated arsenic.  For the SPLP and the pH tests, we did not see any methylated forms.  The only time we saw the methylated forms was in waste lyosometers and also when we collected samples of ground water in the vicinity of construction demolition landfills.

As far as being able to speciate beyond, we have access to an ICP, an HPLC-ICPMS.  I'm not aware -- I don't know if it's capable of doing the methylated trivalent, methylated forms.  I know it could do methylated forms. But I don't know if it can speciate the oxidation state on the methylated form.

DR. HEERINGA:  I have one question out of interest related to potential exposure through these mulches.  You're finding a lot of plywood in this mulch. Where is that originating in the waste stream?  Is it a Florida thing?  Is it used for basement foundations?  Are these knifings off the end of a production line?  What are they? Or do you have any indication where this is coming in?

DR. SOLO-GABRIELE: We have a very good handle on what is happening in Florida. And I wouldn't necessarily limit it only to Florida. But at least in Florida we know how it's being disposed. In Florida a lot of the wastes, this CCA-treated wood waste, ends up going to construction demolition facilities. And at these construction demolition facilities, one of two things can happen. It can either go to a construction demolition landfill, or they recycle it. And especially highly populated areas, there are many incentives for recycling because we have very limited landfill space. So it gets recycled. And so you have these recycling facilities that separate out the different components of C&D, which include the roofing material, the concrete, and then there's a pile of wood. And the assumption is that that wood is essentially untreated and clean. And then it gets recycled as mulch or as fuel at that point.

DR. HEERINGA: Thank you. No specific

indication of the differential between, say, plywood and linear lumber.

DR. SOLO-GABRIELE: The plywood -- there is some plywood that is CCA-treated. But the plywood is more of an indicator of recycled dimensional wood because mulch can be made from the dimension wood, the engineered wood, and it can be made from virgin woods, the tree trunks and the barks.

DR. HEERINGA: It's an identification issue.

DR. SOLO-GABRIELE: It's more of an indicator of C&D wood. Not necessarily of CCA itself, but that it was made from dimensional wood.

DR. HEERINGA: Well, thank you very, very much. Any other questions from the Panel? Oh, yes, Dr. Wauchope.

DR. WAUCHOPE: Just two. Can the panel get a copy of this presentation, the PowerPoint?

DR. HEERINGA: We do have it.

DR. WAUCHOPE: Do we have it?

DR. SOLO-GABRIELE: You have it.

DR. HEERINGA: It was distributed early this morning.

DR. SOLO-GABRIELE: I'll leave the C.D. with Paul.

DR. WAUCHOPE: It's interesting work. And the arsenic 3, of course, I think is the real news here because it's just generally not expected to be found. But the other place you find arsenic 3 in the environment is microbial activities. And those same microbes are obviously on the deck, most likely on the deck. I think this is a microbial. It's methodologies are also probably there.

DR. SOLO-GABRIELE: Very interesting.

DR. HEERINGA: Thank you very much.

At this point in time, I'd like to move to our final scheduled speaker. It's Dr. Steven Lamm who is a consultant in epidemiology and occupational health. Dr. Lamm. And for members of the panel, a copy Dr. Lamm's presentation with his written comments was available first thing this morning to you.

DR. LAMM: Good afternoon. I would first like to thank the FIFRA Science Advisory Panel, Chairman Dr. Heeringa, and Paul Lewis for allowing me to contribute to the discussion this morning.

My name is Dr. Steven H. Lamm. I'm a physician epidemiologist, boarded in pediatrics and in occupational and environmental medicine. I'm on faculty at the Johns Hopkins University Blumeberg School of Public Health, the Uniformed Services University to the Health Sciences, and Georgetown University School of Medicine.

I have been in the private practice for medical epidemiology for over 25 years. And I'm president and founder of Consultants in Epidemiology and Occupational Health, Incorporated.

I have been conducting epidemiologic studies on the health effects, of the human effects, particularly cancer, from arsenic exposure since 1977. This includes both field studies and systematic reviews with both occupational and the environmental arsenic exposures. All of my funded arsenic research work in the past 10 years has been funded by the U.S. Government.

The Panel will be confronted today and tomorrow with a series of 11 questions relating to the development of a reasonable CCA exposure estimate primarily focused on the SHEDS model. And in the 12th question is asked whether application of that result to the upper bound of the EPA cancer slope factor will lead to an overestimation of the cancer risk for the more highly exposed percentiles in the population. I'm here to address that question.

Cancer risk estimates depends on two components as shown in the overhead. Estimates of exposure and estimates of the relationship between exposure and outcome, the cancer slope factor. It is my presumption that the Panel is expected to be using the cancer slope factor developed last year by the EPA, although my comments would apply to those developed by NRC last year, and CPSC this year.

My comments today are directed solely at the methods used to develop estimates of the cancer slope factor from the Southwest Taiwan study that underlies all of these quantitative risk assessments. These comments are derived primarily on the article which I've submitted to you which was published in "Biomedical and Environmental Sciences" and was co-authored by my colleagues at CEOH and Johns Hopkins that's been distributed to the Panel. I wish to take you through that now.

The study that primarily underlies all three of the above mentioned risk analysis is Wu, et al., 1989 Cancer Mortality Study, for the years 1973 to '86 of 42 villages in the Blackfoot disease endemic region of Southwest Taiwan. This is one in a series of studies conducted by Professor C. J. Chen of the National Taiwan University College of Medicine Institute of Public Health and his colleagues.

These data form the empirical basis for Morales, et al., 2000, analysis of internal cancers and arsenic ingestion, which were important parts for the NRC, EPA, and CPA risk assessment. Dr. Chen this morning focused in on this particular study.

I present here a display of the village-specific information for the 42 villages. The village-specific information on arsenic levels in the well waters, the adult population, and adult bladder-cancer death counts were published by the NRC, Table A10-1 in their 1999 report. This figure shows for each village it's crude bladder cancer mortality rate plotted against the median arsenic concentration of the wells located in that village. I point out the difference between the Morales data set and the Wu data set is that Wu included all ages; Morales limited it to age of observation greater than 20 years so all childhood issues are removed from her data set. And the number of person years in the two data sets has been cut in half. Nonetheless, this is a data set upon which all the risk analysis had been built.

Used as a simplifying assumption that the bladder cancer mortality is proportional to arsenic exposure level and only dependent on the arsenic exposure level produces the statistically significant exposure response relationship shown here that is qualitatively similar to the result found in the far more sophisticated statistical of Morales, et al., NRC, EPA, and CPSC. This analysis seems to present a fairly good fit to the data for a straight line continuous risk model.

A second analytic presentation was made in the Morales, et al., 2000, paper in their Table 5, where they presented the standardized mortality rates in an exposure stratified analysis. We present here in graphic form their data on bladder cancer mortality. In contrast to the previous figure, this figure of the same underlying data does not present a good fit to the data for a straight line continuous risk model but demonstrates a discontinuity at an arsenic level of 400 micrograms per liter.

The choices of these stratea were not ours. They are calculations by the Morales, et al., authors. It is clear that the linear risk model that fits the data for the 60 percent of the population that comes from medium well arsenic levels below 400 micrograms, does not fit the data for the 40 percent of the population that comes from villages with medium well arsenic levels of 400 micrograms per liter or greater.

This discontinuity in the relationship between arsenic exposure and cancer mortality suggested to us that there was more to the story than just arsenic exposure. The contrast of the findings of these two analyses from the same data set and the quite contrary result led us to seek clarification.

Our review of the previously published papers by Professor Chen and his colleagues revealed this most interesting analysis from a 1975 publication. In this figure Professor Chen has demonstrated that the source of the village water supply is a marked determinant of the cancer mortality risk, particularly for bladder cancer mortality.

Looking at this slide, the top three bars refer to bladder cancer. Going out to the right is the strength of the SMR. And the colors indicated in the yellow, those are villages that are solely dependent on Artesian wells. The ones in white are villages that are solely dependent and only have shallow wells. And the one in the orange is villages that have both Artesian and shallow wells.

They demonstrated that the risk was six fold greater for those villages dependent upon water from Artesian wells compared to those villages having no Artesian wells. We chose to reexamine the village bladder cancer mortality rates by arsenic exposure levels but stratify by well type.

Using the statements from both Chen 1985 and Wu 1989 that Artesian wells had arsenic levels of 350 to 1,100 parts per billion or micrograms per liter arsenic, and that shallow wells had arsenic levels 0 to 300 ppb arsenic. We classified each well in the NIC table as either Artesian, i.e., as a concentration greater than 325 ppb, or shallow if the concentration was less than 300 ppb.

We then defined as Artesian well dependent village each village where all of it's wells met the Artesian classification and distinguished them from the other villages. The villages were thus separated into two groups that differed both by which water aquifer was tapped as a water source and the type of well used. The water from the Artesian well aquifer was obtained from open surface tanks with standing water that was noted to be heavy with algae. The water from the shallow aquifer was either from closed-pump systems or from wells.

The issue of the algae is important because one of the major hypotheses going on on the difference in the nature of the water is that the waters taken from the Artesian well aquifers were high in umic (ph.) substances. We also separated the other villages into those with some Artesian wells and those that had no Artesian wells.

This slide shows the exposure response relationship found first among residents of the villages that have only Artesian wells, the diagonal line going off to the right. And then among residents of villages that have at least one non-Artesian well, the horizontal line. The paper we submitted also contains graphs making the further discrimination between the 14 villages dependant only on Artesian wells, the 19 villages with only shallow wells, and the 9 villages with both shallow and Artesian wells.

The important observation we wish to make from this figure is that the villages can be separated into two groups of population, Artesian well dependent and non-Artesian well dependent.  And the dose response relationship for these two groups are decidedly different.  Our paper suggests two toxicological hypotheses that might explain these differences, but this isn't the time or place for that.

We concluded from the above where previous risk analysis based on the Wu, et al., 1989 study, and conclude that they have been in error in limiting their description of the exposure variables solely to the level of arsenic in the water.  We find that two different dose response relationships are found in the underlying data and suggest that it is critical to determine which is relevant to the arsenic cancer risk assessments from scenarios in the United States.

We suggest to the Panel that they consider what part of the Southwest Taiwanese data set is relevant to the exposure estimations that they have made, probably calculated on an ingested dose in milligram per kilogram per day.

Could you go back to the last slide? Thank you.

Our own sense is that the data from the non-Artesian-well-dependent villages is the most relevant to the U.S. population. At least that's what we found in the study of bladder cancer mortality and groundwater arsenic levels in the United States that is currently under review at the Journal of Occupational and Environmental Medicine. The editor of that journal has kindly given me permission to submit of copy of the manuscript to you. And I have done so already.

I would recommend to the Panel that they ask EPA to reassess their previous cancer risk assessment to incorporate the well-type distinction and to determine which of the cancer slopes is most relevant to the exposure scenarios in the United States and before this panel. I urge the Panel not to accept the application of

EPA's current arsenic slope factor to whatever exposure estimate the Panel accepts before EPA has included the well-type distinction into its risk analysis. Thank you very much.

DR. HEERINGA: Thank you very much for the presentation.

DR. LAMM: You're welcome.

DR. HEERINGA: Are there specific questions to Dr. Lamm at this point regarding his presentation of the Taiwan data and his analysis and assessment?

DR. LAMM: One point will I make is that all the data here is publicly available and in the hands of EPA already. So there is no difficulty in terms of being able to replicate our work or extend it.

DR. HEERINGA: Dr. Matsumura.

DR. MATSUMURA: I'm not familiar with the types of wells. Could you describe what the Artesian wells are, and were why you think that they are different from others?

DR. LAMM: Yes. First of all, the history of the place is that prior to about 1920, the water that was that used there, these are basically fishing and farming villages at the shore.  Water sources were brackish from the contamination from the salt water.  The government came in in the 1920s and built a bamboo, using bamboo poles, dug wells 100 meters down to tap an aquifer there which is where the water is under high pressure.  So that the water from that aquifer would then come up the pipe and sort of bubble over.  For that reason, they built a basin around the base, a square basin, about two-feet tall that the water would be caught into and that would then be the water that would be used by the villagers.

This is open to the sun, open to the air, and everything that's in it.  And, historically, from the 1960s is readily described as being high blue-green algae and green algae, high in iron, fluoride differences, a number of difficult characteristics of the wells that were well characterized back in the 60s.

The next series of water sources, in the 50s, it was discovered that this area had the strange disease called "Blackfoot Disease" in which you get a terminal gangrene of the hands and the feet. This has never been found anywhere else in the world. No other arsenic study has come up with evidence of Blackfoot Disease in their exposed population.

The Blackfoot Disease was recognized as occurring in people who had arsenicosis. That was recognized to be coming from the Artesian wells. And so the government built shallow wells into the shallow aquifer that then became the wells of preferential use for those that had them. In the 1960s and later, the government came in and brought piped water from other areas.

Just a last statement with respect to the shallow wells. Either the well would be with a pump handle, and therefore be a closed system. Or it would be a well where the water level was well below the surface and where the sun wouldn't get into and was basically protected in the way most water wells are handled. Thank you.

DR. HEERINGA: Thank you very much, Dr. Lamm. Dr. Styblo.

DR. STYBLO: I'm not sure the way you put the association together. Do you expect that the higher risk associated with the Artesian water has anything to do with speciation of arsenic?

DR. LAMM: I don't know enough about that. There has been all sorts of stuff back and forth on it.

DR. STYBLO: Let me just comment on it briefly because I think it may clear up some things here.

Artesian water is known to contain more arsenide and arsenade, arsenic 3 and arsenic 5 in general. However, Artesian wells are used in Bangladesh. We don't have Blackfoot Disease. There is no clear association between Blackfoot Disease and Artesian water, first thing.

Second thing, if speciation is the issue here, arsenic 3-5 status does not depend only on the character of well. It is true that Artesian wells do have more arsenic 3. However, the geology, geochemistry of surface of wells can as well affect the speciation of arsenic in drinking water. There has been a large study done by the British Geological Survey and other people who did an amazing study in Bangladesh showing how chemistry or geochemistry of sediments in water affect the arsenic 3, arsenic plus status in water. And it had been clearly shown that in some places around the world arsenic 3 could be found in great amounts also in surface water. So it's linked to the geochemistry to the composition of the water. So making a link between your suggestion if I understood correctly to apply the surface water risk criteria associated with surface water in Taiwan to U.S. just because of this association doesn't make much sense to me because even surface water will differ in terms of species greatly depends on geochemistry.

DR. LAMM: I understand that. My sense is the first major difference between the two bodies of water is that most likely the rock in which the Artesian well is located has a higher arsenic content than the rock in which the shallow wells are located.

Number two, with respect to speciation, I would ask that one ought to look at what the speciation is in the water at the well head in the tank rather than looking at what the speciation was of the water when it came out of the Artesian well because you may find that being an open oxidizable environment. You may find that what describes speciation underground does not describe the speciation at the surface level.

DR. HEERINGA: Yes. Dr. Chou.

DR. CHOU: I just, for the record, want to make a correction if you will. Actually Sunder, et al., had reported, I believe, in the 1998 paper that actually there were cases of Blackfoot Disease detected in India.

DR. LAMM: I thank you. I would be happy to have you send that reference to me.

DR. HEERINGA: Dr. Bates.

DR. LAMM: Excuse me. You made another point that I wanted to respond to. And that was why is it necessarily that it's the shallow well that is the best risk slope for the U.S. I'm not saying that. I'm saying from my further work, I've reached that. What I'm saying is that when one looks at the underlying data, there are two different populations of villages; and that the slope factor for the two villages differ.

I'm asking that people make recognition of this and then go back and reassess which of those two are more likely to represent the risk in the United States. If you found that it was the Artesian well slope, then you would be estimating that the risk that EPA came up with is far too shallow. So while I have my own expectation what's going to be fine. I'm not mandating any particular finding that way.

The other thing which I wanted to point out is that 60 percent of the study population is in that less than 400. So that's a large population. And whatever you do, you've got to have -- if there's one explanation for the bladder cancer risk, then it has to apply equally to the lower half of the data as it does to the other half of the data.

There was another question.

DR. HEERINGA: In the interest to keep us on track with our questions, I want to wrap this session up. But, please, we'll finish with the questions that Dr. Bates.

DR. BATES: I was just wondering, did you take into account in your analysis -- unfortunately, I haven't had time to read the paper, but I will -- changes in the well type over time.

DR. LAMM: Pardon?

DR. BATES: In your analysis, did you take into account changes in the well type used in the villages over time given that arsenic has a long latency and it may be --

DR. LAMM: What I took into consideration were the concentrations that were given by the National Research Council as being the concentrations for the wells. I was not able to go behind that. I've asked for data from Taiwan. It hasn't been forthcoming yet. But I'd be happy to see such. I recognize the potential for misclassification, but I've tried my best.

Thank you very much.

DR. HEERINGA: Thank you, Dr. Lamm; and thank you for providing us with the papers and the prepublication version. It was very helpful.

At this time, I'd like to ask if there are any other public commentors who would like to make a short statement to the panel.

Seeing none, I want to do one thing before we move on to the next item in our agenda. We have often private citizens and others who don't have the wherewithal to make it here for these meetings send in public comments. There are four in specific. I'm not going to read them. But they will be part of the docket.

We have received one from Joe Prager from banka.org; one from Michele Lafantaine of Ottowa, Canada; one from Andrew Wegmann of Beacher, Illinois; and another from the New Zealand Wood Preservation Council. These will be part of the docket.

Paul assures me that that is correct with what we've received at this point. So again as part of full disclose on what has been submitted to the Panel, I encourage you to take a look at those documents in the EPA docket.

At this point in time, I think I'd like to bring our period of public comment to a close. And I would like to thank everybody who participated. Obviously, a tremendous amount of information and insight on the part of different individuals and parties brought to play. And on the part of the Panel, I think quite a bit of learning and advice as well.

At this point, I want to move on to the specific questions that have been posed to the EPA Panel. And I think that we have scheduled on the agenda to continue with this part of our meeting through 4 p.m. tomorrow. But I'm also wise enough to know that there's a survival function in people's staying power on these things. And I want to make sure that we get proper attention to each question. I think that we will be going a little longer today. I think we are scheduled to 5:30. We may go just a little while longer. I have to explain that I'm going to excuse myself at 4 o'clock. I have to get to College

Park to teach a course.  It's a commitment I've had since the beginning of the semester.  I'll be back first thing tomorrow morning and Dr. Matsumura will be serving as acting chair during my absence.

At this point before we turn to the specific questions, I would like to ask Mr. Jordan if he or the EPA would have general response to any of the comments or introduction to the actual formal questions that have been posed to the panel.

MR. JORDAN:  Thank you, Dr. Heeringa.  The public comments raised a number of points far top numerous for us to try to answer.  There are certainly some statements that were made that we think may have reflected a misunderstanding or an incomplete understanding of the information that EPA has made available.  And rather than delay the discussion on the issues, what we'd like to do is to encourage the Panel to go ahead with its deliberations and discussions of these issues.  And if in the course of the discussion on a particular issue you have a question that arises from the public comments that you would like us to try to answer, please feel free to direct that question to us. And we'll to do our best.

And in the course of the discussions, if it looks like you touch on everything that we thought might be worth noting, that's great. If not, then perhaps tomorrow morning we might add a few fairly brief and focused comments that would tend to round out the record.

DR. HEERINGA: I certainly see that as appropriate. Just before we turn to the actual questions themselves, just give an overview of what I think our role as a panel is. We are assembled here to review the Preliminary Draft Exposure Assessment and Preliminary Risk Assessment for Children's Exposure to CCA-Treated Wood and Playsets and Decks.

There are a series of 11 issues, 12 issues, that have been brought to us. Some of them with multipart questions. Many of them relating initially to the exposure assessment. And then the final questions to the risk assessment.

Strictly, we are asked by the EPA as an advisory panel to provide answers to these questions using our broadest base of knowledge and expertise. And we will do that. In addition, I think it is also the case that we will be able to comment in the general venue of each of the issues and items. And we will try to make sure that we clearly distinguish those two areas in our final report.

And I think in speaking with Mr. Jordan, too, he's encouraged us and I think he has just done this again. That if there are specific areas related to these questions, that once we've answered the question as it's formally presented, there's the ability to provide comment related to that specific item.

And also I guess finally I want to say that we all recognize over the past day and a half that we are essentially taking a snapshot here in three days in a longer movie that's sort of rolling out. And as we see results coming in 10 days before the meeting, two weeks, two months, and there will be some that will come three weeks after we're done here. So we're clearly taking a slice in time. So certainly we're looking back, looking at the fixed state of these two reports but also considering the new information that's been brought and looking ahead too in terms continuous sort of quality improvement and advancement here.

So with that, I'd like to ask that Mr. Jordan if you would please read the first question to the Pane;.

MR. JORDAN: Thank you, Dr. Heeringa. We have worked on these questions. And I'm going to ask Luke to handle the ones on exposure and Winston Dang to handle the ones with regard to the risk assessment part.

DR. OZKAYNAK: First issue is on documentation, completeness, and clarity of the model source codes and the exposure assessment report.

Both the SHEDS-Wood source code and the probabilistic exposure assessment report have been significantly revised since the August 2002 SAP.

Question A, the first question states: The Source Code Directory on the CD provided to the SAP includes annotated code for the exposure and dose algorithms used in the SHEDS-Wood model. Are these algorithms consistent with the descriptions in the SHEDS-Wood CCA exposure assessment report? Does the revised SHEDS-Wood version 2 code (i.e., the code submitted for the December 2003 SAP) accurately reflect changes to the version 1 methodology (i.e., the code and methodology presented to the August 2002 SAP) described in the report?

DR. HEERINGA: Dr. Macdonald is the primary discussant, Question A, Issue 1.

DR. MACDONALD: Well, this is a very focused question. I hope we can deal with this question quickly as I think the important issues are with the assumptions and the design of the model and the data used to set up scenarios for the examples.

The model, of course, has to be correctly programmed, and we expect that the wise advice of previous SAP meetings has been accurately incorporated. And that's the focus of this question.

I have some experience with SAS. And with the time and computer facilities available to me, I was able to inspect the code and make a limited number of trial runs. A lot's gained by the choice of SAS for this model. The model is coded in relatively few lines and the speed and file-handling capability of SAS are available. The code is simple, easily inspected, and easily modified. Most of the assumptions are in separate tables easily edited by the user rather than hard-coded. Most calculations are simple products of factors.

I don't know that the scripts required to produce the graphical and tabular reports were included so it was difficult to interpret the trial runs I made. Also we were not given enough information to permit a thorough code audit. We were missing a table defining all variables and cross references linking the description of the algorithm to the various scripts. As far as I can tell, however, the code is okay and all of the modifications since the 2002 meeting have been fairly documented in it.

My biggest concern is with the assumptions that are hard-coded into the macros and, therefore, less likely to be questioned by users particularly the calculation of the height of a child who has no height from the previous year seems inconsistent in that one random monthly gain in height is generated and multiplied by the number of months of age. This makes for much greater variability in height compared to generating an increment for each month as is normally done in the model. If the child is over age six, growth parameters for a child over age six are used for this increment which applies then to all months of life.

Another odd feature which I asked about yesterday is the way the last time period of the day is forced to have a contact event if there has to be one but hasn't occurred earlier in the day. This may introduce a bias which could be avoided by selecting all times of contact at once at the start of the day.

These are just two small details. It isn't clear how often these situations arise or how important the handling of details like this is in the overall performance of the model. But I can't see any other issues with the coding.

DR. HEERINGA: Dr. Hattis.

DR. HATTIS: I don't read SAS. I wouldn't presume to try to do this.

DR. RYAN: Essentially, I went through and I did a few runs to make sure that the system worked. But I have not gone through the code line by line to identify if there were any errors. I expect that the quality of the program is knowing then that they did what they were supposed to do. And I know somewhat more about SAS than Dr. Hattis does, but I really have nothing to add.

DR. HEERINGA: Yes. Dr. Portier.

DR. PORTIER: I know a lot about SAS. And I looked at it. And I really appreciate the fact that the code is highly commented which allows us to read it really easy. One of the things that is not provided, though, is the output files. We really need a description file that defines what all the variables are that are output.

Because the way the code's run right now, you can run the procedure, look at the output file very easily within SAS, and then go into something like PROC Insight and look at histograms and box plots, and answer a lot of the questions that we've had about distributions, what do the things look like. It's all in there. You just have to know SAS to be able to use it. And that's an issue we're going to have to talk about probably in Issue 2.

DR. HEERINGA: Any other members of the Panel who would like to comment on the code?

I would just add my comment to Ken. Everything I learned about, SAS I learned from him. Not really.

In any case, I looked at the code as well. I appreciated the comment, the statements that are in there. Peter and I looked at a few of the examples together in which assumptions are sort of programmed in. There's a complex set of situations for handling missing data problems or partial data problems. Some of those are hard-coded into the program. And that's fine. As long as they're commented, I do agree with Ken that I think clear description of variable inputs and outputs to facilitate sort of post-processing would be useful.

And I think that in general what I saw in the code looks satisfactory and reflected the changes that were presumably made in response to the past meeting.

Any other questions on this fairly technical comments question from the Panel?

Okay. With that, Luk, I think we'll move on to Part B.

DR. OZKAYNAK: Can we provide a clarification?

DR. XUE: First, I think that for in term output label, I think this is important. We should provide that. We have it somewhere. But when we change so much, we just forget to include this. Definitely this very important; otherwise, it is very difficult to understand what the output is.

Second one I want to make comments about are the height and the weight. Can we show some slides about the comparison. We use real data, and there's a simulation. Do we have enough time to do that?

DR. HEERINGA: Yes, you may.

DR. XUE: Basically, when we think about how to submit as height, weight, and this is a little more complicated than we thought about how intraperson variability and interperson variability. Some have high and some the changes. So what we do is that we use the end past data to do two things. One thing that we try to say that all submission data is that corresponds really or not. This is the first that we do. Look at this as in the match quite well. This is for male. And the next slide, this is for female.

The second, we look at is the change. Because we know they have change of the weight, so we look at standard deviation, look at the population change. We cannot change of the interperson variability because we don't have data to compare. But we look at overall each person as one person, then compare overall variability of change. Look at their height and the weight.

So we do find our submission be a bit bigger, but not a very over estimate of this variability.

Next slide. And this is for height. And the next slide is for weight. We're a little bit overestimate of this variable.

Then we also do some change for the given person to change. We see it as I think for one year, we change of one year change of 2 kilogram is very, very, very small. And for all the changes that -- I think that almost 99 percent of people just gain weight. And very small people for a lifetime. Very, very small people that lost weight. So this is we do something because one problem we know, we have problem. We cannot compare interperson variability to see that compare with the real data. But we geared it to more analyses how to simulate of this height and the weight.

DR. HEERINGA: Thank you very much. Peter, in response?

DR. MACDONALD: Well, code I was referring to was to compute a height for someone who for some reason doesn't have a height from the previous year. Now, how is that going to come about in the simulation?

DR. XUE: What we do is that we split these two parts. One part is if they don't have height, then we just direct the use that count relation for data for age gender. What height it is. And then when you go to next year, we say, okay, because this given person, so we can separate two parts. One part is something because you're a person. You have intrapersonal variability. So you were given your height.

We're not -- once assumption, your height would be lower. So this way you will gain some height. From the INCAS, data we calculate how much height you will gain. If you're 140, you cannot 139 because -- so we calculate gain height.

And then we process the gained height to this person the second year, the gained height of this person. Then we use this gained height as some from the NSIDE regression to calculate weight. We did it this way. You don't have height in everything which is okay. Run them choice given age and gender. We get data INHAPS data. So given your age and gender, what's the height and what's the weight. But when you do next year, you already person there. Your heights will not be reduced. We use this height according to the next year what average height you would gain. Then use this height as a base to calculate the weight.

DR. HEERINGA: Dr. Macdonald.

DR. MACDONALD: My question there was it seemed inconsistent that once you've got somebody's height established, you're doing their height in random increments each a month at time. But for the first time you do it, you generate just one random increment and then multiply it by the months they've been alive which isn't the same thing. That's what I see in the code.

DR. XUE: We use the average height not just the random draw. Because from data, from the INCAS data, we have one person each month. When you do month, what average is the height gain for given person. We add this another random, give us a number. But if they have some -- because they have -- one run is okay to run them because we have -- when we do the analysis INHAPS data, so how the mean and they standard deviation. So we run and we use the mean. But that doesn't change the standard deviation of this one. But usually standard deviation is very, very small. So this is not absolutely random.

DR. HEERINGA: Thank you very much. I think, Peter, that at this point, you can point out the specific lines in the code; and we can focus on that. And I think just as a notice, I think in Dr. Macdonald and the group with the report, we may have sort of a list of things that are identified. I think that's the nature of the question if we find them.

Any other comments or statements in regard to Question 1A, Issue 1A? Issue 1B.

DR. OZKAYNAK: Question B: the SHEDS-Wood CCA exposure assessment report presents the model construct, selected model inputs, model results, and comparison to other CCA model estimates. Please comment on the clarity, completeness, and usefulness of this document.

DR. HEERINGA: Dr. Macdonald again, please.

DR. MACDONALD: Well, I think that the exposure assessment documentation is clear enough. The tables of user-specified assumptions are extensive. But the assumption hard-coded in the script could be highlighted and explained better.

The report assumes that a user interface will be available for setting up scenarios and analyzing results. Without that, the report is not enough and SAS expertise is necessary to use the model.

There are so many user-specified assumptions in the model that I can't see how the sample results can be compared with the results from any other model. To be a bit cynical about this, I expect you could easily tweak a few assumptions to make SHEDS-Wood agree with any other model. And that wouldn't prove that either model was correct.

DR. HEERINGA: Dr. Hattis.

DR. HATTIS: I also think the report is generally clear enough. However, I think some more documentation of the raw data that was used for the derivation for the different distributions and the goodness of fit to the underlying data would be desirable. Ideally, an interested outside analyst should be able to reproduce the derivation of the distributions or fits from publicly available information or develop their own distribution if they think some improvement is possible.

DR. HEERINGA: Dr. Ryan.

DR. RYAN: I felt the presentation of the descriptions of each one of the data elements was really quite good in the document. As far as some of the other things that have been brought up, I have some similar opinions. And I won't waste the committee's time.

DR. HEERINGA: Any other comments from the Panel with regard to the exposure assessment report and the clarity and completeness and usefulness of this document. Dr. Reed.

DR. REED: For a person who doesn't speak SAS, I find the document very useful. And I felt I've never been so intense with a document because there's things I don't understand. And I'm so visual, I need to have something that I can run and play with. And since I couldn't have that.

And I was wondering to the extent possible. I know it's not always possible. I was wondering for some places especially when the results are presented, if you know the whys of the result, sensitivity variability analysis ask so forth, to give a little bit of explanation in terms of why do you think it comes out as two fold differences when you change one parameter and so forth would be very useful for me. As I said, in some places it's not possible because there are so many factors coming into play. But just to give me a feel of what's going on would be great.

DR. HEERINGA: Yes. Dr. Portier.

DR. PORTIER: I was thinking back to the first version of this and comparing it to this one. This is much clearer because it lays out a lot more of the technical information in tables. And you've kind of removed the user interface. So my question, though, is what happened to the user interface, and will that come back in version 3?

DR. XUE: I think because of time limits, there's some much time to work on the input and all the documentation and the program and checking for accuracy, we really don't have enough personnel to do the finish interface. Definitely we'll put an interface back on. But right now, I think that we're focusing on the most important part right now to get the model right.

DR. HEERINGA: Any other comments?

I want to say that I appreciated in the risk assessment report itself the addendum or appendix in which there are specific responses to the historic comments made by the Science Advisory Panel. I think that this, even though it reflects on this document, preserving this type of the history and in this fashion gives us a good track on decision-making. It also reminds the Science Advisory Panel of what they said in 2001.

I think it's a very, very important thing to do. And also it gives concrete statements to us as a panel to how you've responded or when you've chosen or have been unable to respond to a specific recommendation or suggestion. So that is particularly appropriate.

I won't to go through specific examples, but we did see one yesterday on the screen. And I think they do exist in the report, too, where there are things such as determine hand loadings that are missing units. And that may not be so critical for people who deal with these on a day in and day out because it may be inferred. But for a lot of us going into it fairly blind, can't deal with the units at the same level. So I think being able -- just checking the tables and making sure that units are present.

Also with regard to some of the displays, I'm looking at the bootstrap results, and maybe it's a nonparametric type bootstrap. To the extent that the parameters we're boot strapping to obtain bootstrap samples for two different parameters and distributions we should label the anticipated distribution, too, if that's applicable. And, again, I'm just looking at this here as one example. I'm not sure that that's just not a nonparametric.

Just a couple of comments. I think the check on units and that's natural in any edit process to have missed some of those.

Dr. Portier.

DR. PORTIER: Something Dr. Heeringa said reminded me that you needed to check the units on all the graphics. Right? But also some of the output parameters, it would be very useful to have those distributions displayed in the exposure document. We talk the about that yesterday. I know those distributions are able to be generated because I've been able to actually look at it. But I think it helps the believability of the scenarios if you can show, not specifically the components distributions, but the combined components distributions for some of the things that are really critical.

DR. HEERINGA: Thank you, Dr. Portier. That's a follow-on, a comment there, too. I think for clarity because we have so many contributing distributions. And as we noted from your presentations yesterday, that many of them have differential affect on final outcomes and sort of uncertainty in the final simulated distributions of exposures that it probably would be good to look at a few of what I would call sort of product distributions, that are products of independent or conditional stochastic draws from three or four distributions. And one that I mentioned yesterday would be the total expected time of exposure per annum for children on playsets. I think that would be one. May be cross-tabulated or scattered against the number of actual exposure events just as a test of realism.

I think we have the dermal hand loading. And I think the -- I don't know if any other panelist can think any of these other sort of aggregated distributions. But I agree very much with Dr. Portier. Just for presentation and sort of for people who can't quite do all of the algebra in their head and multiple simulations, it's nice to see these intermediate distributions as they come out. It makes a good point of checking against some of the deterministic modeling comparison where you could look at the distribution implied stochastic or probabilistic modeling and seeing if its central tendencies match up fairly well with other deterministic modeling attempts.

Yes, Dr. Francis.

DR. FRANCIS: The other one that I thought of that I had mentioned previously is the residue ingestion which is made up of so many different components.

DR. HEERINGA: Would that be on an annual basis then, I assume, because otherwise we get a whole series of lines over time.

Any other comments on Issue No. 1.

DR. OZKAYNAK: Dr. Heeringa, I guess Dr. Xue has taken up your suggestion and ran the code to tabulate the total expected time of children on the playset. I'm not sure if you want us to present that now or if you want to defer that.

DR. HEERINGA: I'd prefer to defer it. But if copies could be distributed to the panel members. And if that could be done and if we have had any questions about it, it could be brought up in the context of the commentary tomorrow.

DR. OZKAYNAK: Yes, that sounds fine.

DR. HEERINGA: And a copy also to --

DR. OZKAYNAK: We'll arrange for getting copies to you.

DR. HEERINGA: -- Paul Lewis, too. Thank you very much. I'm not seeing any more comment on issue No. 1. Let's move on to issue No. 2.

DR. OZKAYNAK: Issue No. 2, Modifications to SHEDS-Wood model code and the exposure scenarios selected.

A number of modifications to the model code and scenario-specific changes have been made to the SHEDS-Wood model since the August 2002 SAP.

Question A: Considering the limitations of available information and state-of-the-art modeling methods required for the assessment of children's exposures from contacting CCA-treated wood residues and CCA-containing soil, are the revisions made to the SHEDS-Wood code or algorithms scientifically sound and acceptable?

DR. HEERINGA: Our lead discussant on this is Dr. Portier.

DR. PORTIER: Thank you.

I'm not sure how to answer this particular question, but I'll make an attempt. And then let the others on the Panel correct me.

We're asked to discuss the scientific soundness of the code and algorithms. But you haven't really defined "scientific soundness." So I'm going to make an attempt to do this by stating that I think scientific soundness suggests that the code and the algorithms must have three criteria.

One, it must express the logic of what I'll call the microsimulation model that underlies this assessment that's proposed for exposure. It must be transparent enough that it can be repeatable by other researchers wishing to replicate the model possibly in another format than the one that you presented. And it must be based on generally accepted data processes and parameters. And I'll return to the issue of what I mean by "generally accepted" at the end.

No one has suggested that the SHEDS-Wood code does not faithfully express the underlying microsimulation, it was designed to mimic. There are components of the simulation model that are conscientious and some components will change or probably be added in the future. The structure of SHEDS-Wood is of sufficient flexibility to facilitate these changes and additions. The speed with which the SHEDS-Wood team was able to implement the many changes proposed by the August 2000 SAP demonstrates this.

SHEDS-Wood is implemented in SAS and for the most part is transparent to anyone familiar with SAS scripts. This is a point in favor and a detriment. You must have SAS to be able to run the simulations. And, of course, SAS is not free. It's a proprietary environment. But SAS provides a flexible environment for model modifications and enhancements. So the development in SAS represents a compromise between flexible and model implementation, time available to develop the model, it provides a model that's transparent for potential users, and can be implemented and maintained with existing personnel. And you've already mentioned the fact that there's just a limited number of people available to develop these models.

I have a side comment. Actually, the version of SHEDS-Wood presented to this SAP is more transparent than the previous version because most of the complex up-front menu structure is gone and the user need only modify a macro call or a couple of data sets to change the run. So for me, a good SAS user, I find this code very transparent.

Because of this, I would suggest that SHEDS-Wood model is sufficiently transparent as to be repeatable. Note that this is not to imply that it would be a simple matter to repeat this structure in another programming environment. A simulation of temporal activity patterns implemented in the code is quite complex and is not something that could be easily implemented in, say, a spreadsheet environment. So just because the SAS code can be followed doesn't mean that this implementation is a simple implementation.

Industry representatives were able to follow the model sufficiently to understand and critically evaluate it. These external reviewers were also able to suggest how the microsimulation model should be changed and EPA was able to quickly implement these changes and assess the impact of these changes on the final estimated exposure distribution.

Other proposed changes such as indoor versus outdoor hand-to-mouth contact components distributions or intensity of contact modifications or even adding an unloading process as suggested in the industry comments and other behavioral changes as suggested in the other public comments seem to be something that could be easily implemented and evaluated quickly and responsively. So I think that adds to the argument that this is a repeatable and clear code.

Finally, we come to the issue of generally accepted data processes and parameters. This is where the limitations of available information clause in the question comes into play. In implementing the microsimulation model, the developers have had to use vast professional judgment in choosing which processes, that is, routes of exposure to be included and which to exclude. They have referenced the literature and engaged researchers to get the best data. But in some cases, the data are inadequate or unavailable, and, hence, best professional judgement must be invoked again.

Finally, because this is a probabilistic risk assessment, many of the model components have been conceptualized as random variables; and as such distributions for these random variables must be specified. This is a relatively easy task when supporting data are available, and a daunting task when little or no data are available.

There are in the SHEDS-Wood model implementation a number of components whose distributions are based more on professional judgment than on data. Some of this is unavoidable in as ambitious an undertaking as this model. Since this is also a topic that the Panel will be returning to in the rest of the questions, I'm not going to comment any further on this. I'll save those comments more for later.

Finally, as mentioned in the public comments and scientific inquiry we often gain insight through the examination of competing models. I believe this is true. And, actually, this is a foundation concept in statistics. When choosing among competing sample models, the use of a validation data set is critical to determining the best among the candidate models. As the model under consideration gets a little more complex, even something as simple as multiple regression model, the number of possible competing models can be large. And the choice of the best model, even using a validation data set, is very difficult and the task is -- even with the validation data set, the task increases proportionally in difficulty.

It is a fact of life that as the model gets more complex, our ability to fully validate the model decreases. Validation of a complex model actually involves what we're doing here, that is, examining each model component and determining the validity of the parts and how logical the components are put together. If we are lucky, we can develop one or two experiments that challenge that model. And I think many of the studies proposed by industry seem to be focusing in that direction, trying to come up with an experiment, a study that will actually challenge the outputs of the model.

In conclusion, I feel that SHEDS model code and algorithms are scientifically sound and acceptable within the limit of current data and within the framework of the exposure model that underlies the codes. Sorry for the length of that.

DR. HEERINGA: That's just fine. Thank you very much for that very comprehensive comment. Dr. MacIntosh, what do you have?

DR. MACINTOSH: I have discussed this particular subquestion with Dr. Portier and agree with the points he had made and contributed somewhat to them. And I have nothing further to add.

DR. HEERINGA: Dr. Freeman.

DR. FREEMAN: Yeah, I agree with what Dr.

Portier said. There are a couple of details I would like to discuss. One of the code changes that you have done is to use new probabilities based on Los Angeles data, longitudinal data, for switching between high, medium, and low potential exposure scenarios. This is going to make me sound like an easterner. But there should be some way to test whether what goes on in Southern California would work elsewhere.

I know that the problem is the lack of longitudinal data. You're doing the best you can with what you've got. But somehow there needs to be some verification that that truly represents the real world.

In a similar vein, you talk about using outdoor children as opposed to playground children in part because you have too small a sample in CHAD for modeling. There's an even more important reason to do that. If you only looked at the outdoor -- if you looked at the playground children in CHAD, almost all of them come from California. Very few of them come from INHAPS data.

And I'm not even sure if you've looked at the

INHAPS data to see what proportion of those playground children were also from California. So that if you had used it, you would have had a very biased data set to work with. So it's not just that it's too small, but it may be unrepresentative of the larger population that you're really interested in. I don't think we're all Californians or Los Angelinos.

The third thing that you changed that I will talk about are changes in the approach for bathing events by allowing a variable number of days between baths. I didn't really see where you got that data and how you were using it. Dr. Adgate, through the Nexus Minnesota Children Pesticide Exposure Study, does have longitudinal bathing data on -- was it 109 children? Something like that. I doubt if you have it here, but it exists. That might help.

DR. ADGATE: Not only does it exit, but ORD has it already although they might not know it.

DR. OZKAYNAK: This is the recently made available information, Dr. Adgate?

DR. ADGATE: It's from the 1997 study. I don't know if you can characterize it as longitudinal in --

DR. FREEMAN: It's a week long.

DR. ADGATE: It's a week long. And it's for 102 kids. I actually brought the diary with me. We can -- I can show it to you. I was going to address it in the next issue and talk about the questions that might be relevant. I don't think they'll change your distribution so much. But it's one way to sort of ground-truth the choices that you've made. Thanks.

DR. HEERINGA: This is Minnesota data.

DR. ADGATE: Minnesota in the summer. That helps. I don't know if that makes them Angelinos or not.

DR. OZKAYNAK: That's Boston in winter.

DR. HEERINGA: Once a week whether you need it or not. Sorry, I had to get that in.

Any other comments with regard to this particular question? So you have some additional bathing data. Members of the Panel?

I think Dr. Portier made an important point.

We've heard fairly intensive presentations from industry and other interest groups with regard to the interpretation of this exposure model. And I think, while some of it is probably a little hard to listen to after all your hard work, it is exactly the types of criticisms as I listened to it myself I heard them hitting on precisely the areas of uncertainty that I felt and that we probably all feel within the model.

So though I didn't hear many large structural criticisms of the model, there were some suggestions about returning at least to comparisons to deterministic model runs. But I think the SAP has long gone down the path of proposing probabilistic risk assessments. And as we indicated earlier, one way of looking at comparability of the probabilistic is to look at distributions of intermediate outputs against deterministic inputs that would be put into the models at some point. So I think overall I certainly agree with the comments that Dr. Portier and the others have made.

Yes, Dr. Hattis.

DR. HATTIS: I think it clearly represents a good faith effort to utilize the limited information that the EPA folks had. This doesn't mean that it's necessarily the truth. But it represents a reasonable effort that has some standing as a reasonable input to people's decision-making.

DR. HEERINGA: And I'm quite confident as we move through the responses to the other questions, some of the areas of uncertainty over input distributions or values of input parameters will be addressed specifically.

Any other comments in response to Question 2A? Should we turn then to 2B, Dr. Ozkaynak?

DR. OZKAYNAK: Yes. Question 2B.

The SHEDS-Wood model has been modified using feedback from the August 2002 SAP. In particular, the recent assessment includes: assessment of exposures of children contacting only CCA-treated public playsets; sensitivity of results to changing the age group of exposed children to 1 to 13 year's; and a separate analysis for children exhibiting pica soil ingestion behavior. The Panel is requested to comment on the appropriateness of the new exposure scenarios in the revised probabilistic exposure and dose assessment.

DR. HEERINGA: Dr. Portier

DR. PORTIER: Regarding the appropriateness of the new exposure scenarios, I'm going to have to defer to the other members of the panel. I'm convinced that the SHEDS-Wood code could implement any reasonable new scenario we could devise given that we could provide any associated parameters estimates as a random variable component of the scenario model. I'm satisfied that the SHEDS-Wood team has faithfully implemented the scenario suggested by the August 2000 SAP at least within the limits of available data.

DR. HEERINGA: Dr. MacIntosh.

DR. MACINTOSH: I agree with the comments by Dr. Portier here and would like to add just a few specific comments on top of those.

I think with respect to the first particular modification that's listed in this question, the assessment of exposures of children contacting only CCA-treated public playsets that it would be useful, I think, in the document to just put a little more emphasis on the study population or the model population here because I think that a casual or even a moderately interested reader of the report could come away thinking that public playsets exposures according to this modeling implementation are far and away the most important source of CCA exposure.

But instead it's a very special population that you've model. Right? There's really no -- you're comparing kids on playsets always to kids on playsets with decks. Right? It's not really a population-based exposure assessment or risk assessment. And I think that greater clarity on that or emphasis on that point would be useful.

The second particular point, the sensitivity of results to changing the age group of exposed children to 1 to 13 years. Maybe this goes back a little to Question 1. But I actually had some questions about how that was done. It appeared to me that you took the results from the 1 to 6 year olds and then assumed some fraction of that, various fractions, represented the 6 to 13 year olds, overall 1 to 13, I guess.

And I'm not sure that adds any really useful information. Because it's kind of like saying, if it was zero, this is what it would be. If it was half of what we thought it was for this other group, this is what it would be. If it was equal to what it was for the other group, this is what it would be. But there's no characterization of which of those scenarios you think is most likely.

And for that reason, I'm not sure it offers any useful information. That concludes my comment.

DR. HEERINGA: Dr. Freeman.

DR. FREEMAN: I agree with the statements of both of my colleagues. The approach to the 7 to 13 year olds, essentially brute forcing at 25, 50, 75, and 100 percent of the other children, doesn't actually even take advantage of the data that you do have from CHAD as far as I can tell.

It seems like it's first you generate the data for the younger kids and then you did this adjustment as opposed to taking the data from CHAD that you do have on older children and fiddling with it in some way. That may be a little bit more time consuming.

Again, you know, there -- I really wasn't clear where the adjustments were made. It sounded like they were made at the end of the analysis rather than at intermediate steps with different variables.

DR. MACINTOSH: If I could just --

DR. HEERINGA: Dr. Zartarian, if you would like to respond.

DR. ZARTARIAN: To both Dr. MacIntosh and Dr. Freeman, it sounds as if you're wondering why did we do it that way. And we did actually start doing the 7 to 13 year olds the same way that we did the 1 to 6 year olds. And we quickly ran into issues. We did have some information on hand-to-mouth behavior for the 7 to 13 year age group. But we didn't have information on soil ingestion rates or days, all these other ones. And we thought that rather than going through the same exercise of assuming distributions and putting uncertainties that this would give more of a bounding-type picture of what that additional age group would do.

DR. FREEMAN: I have a couple more comments.

DR. HEERINGA: Yes, definitely.

DR. FREEMAN: One of the scenarios had to do with pica. Pica was an interesting exercise in that it is a group of children who are soil eaters. The assumption could be that, if you were a soil eater, you could use that as the child who would maximize ingestion at a playset even though a true pica child wouldn't be eating that type of material. They're after nutrients typically. That only addresses the soil consumption.

An alternate scenario that you soon will have data for would be for autistic children. Dr. Schallit and Cathy Black are doing a study of childhood autism now that will be able to give you that data for that particular scenario.

DR. HEERINGA: Dr. MacIntosh, did you have additional comment?

DR. MACINTOSH: I just wanted to say that in recognition of your comment and what I had presumed while reading the report was that there were either very serious data limitations or time limitations on some of these modifications including the expansion of the age group. And I also understand that one of the previous SAPs suggested that you make this expansion of the age group.

But I'll offer this suggestion and welcome comment from other SAP panel members. But just because it's suggested, doesn't mean that it must be done at all costs -- right? -- despite information available to you. I think it's perfectly okay to say we're not able to do this because of data limitations.

DR. OZKAYNAK: I appreciate that advice. We may use that.

DR. HEERINGA: Let me just solicit the Panel on this. The issue of extending the age range of the exposure assessment, I think that was raised in the 2002 meeting because of adolescents and older children playing on play structures. We've talked about fishing docks, but other play structures, collection points. How do we feel as a group about the importance of this relative to some of the other aspects of some exposure assessment?

Dr. Hattis.

DR. HATTIS: I think doing it the way they did has the advantage that they aren't forced to treat those other years where we expect there to be some exposure is effectively zero. But I think it's an improvement over that. By implicitly taking a number of possibilities they considered reasonable, they communicate to the decision-makers or the audience some range of what they think is reasonable without having to do a tremendous amount of analysis or invent things that they don't really know about.

DR. HEERINGA: Dr. Freeman, are you satisfied with that? I know that you were --

DR. FREEMAN: Yes and no. I think about the data that exists. And, yes, you've got two things. You've got kids who as they get older in elementary school becoming very independent and going about doing things on their own without parental or elder supervision. And at the same time, there is an assumption that mouthing goes down.

There's really no data on that. So that on the one hand, it would really be interesting to understand the dynamics and if there is any increased or additional exposure. But on the other hand, without the data it may be sort of a fruitless exercise. And maybe what they did is as good as you can get for now.

DR. HATTIS: Yeah. I think as one gets to older age groups, I think certainly mouthing goes down. But it may well be that thinking about other pathways like eating food with dirty hands and transferring residue that way.

DR. FREEMAN: And eating at the play site, too.

DR. HATTIS: Yeah. All these different things can, you know...

DR. HEERINGA: There's a consensus of the Panel or a partial consensus that it's an important thing to keep in mind that in fact exposures don't drop to zero following this. But that the treatment that you provided in the exposure report is probably satisfactory, particularly given that data because you'd be driven into the same sort of data quandary as you face in the 1 to 6 assessment only in a power of two.

So I think that it's fair to say that that treatment in terms of recognizing that added exposure is probably legitimate; but at this point in time, it's probably not worth intensive investment.

Dr. Francis.

DR. FRANCIS: I guess I kind of agree. On the other hand, the question is: How useful is this to EPA and the public given that these essentially guesses. What's the utility of that information? And maybe that's not part of our charge. But it seems like if you're going to be putting that information in the report, people are going to want to use it to some extent. And there's clearly no guidance. Well, let's do 25, 50, and 75. I don't see any value in it personally. But if somebody can tell me what value there is, I'd like to hear.

DR. HEERINGA: I think the recommendation was a recommendation on the part of the Panel in 2002. And your point is well taken there.

Dr. Reed.

DR. REED: I wasn't at the 2002 meeting, but I do appreciate the work. Part of the reason as risk assessor and especially when you look at some oncotanic risk and the default being that oncotanic risk is proportional to the life time of exposure. It's to me very satisfying to know what would it look like if you don't zero those years when the kids are still playing in the playground. Given that there's not enough data, I think the Agency stated it very clearly. It's 25 percent, 50, 75 and 100. So if somebody comes along and says, well, you know, perfectly well that kids don't just stop playing in playgrounds; then you could say, well, I didn't have data. But if that's what you want, this is what it looks like. Take it or not taking it is a different issue.

DR. HEERINGA: Dr. Hattis.

DR. HATTIS: My comment goes to the word "appropriateness" in the question. And appropriateness depends on the decision-making use that's made of the information, of course. A limitation of the warm versus cold scenarios and the focus on frequent users is that, although one obtains reasonably high and low to moderate exposure cases, one does not obtain information on population aggregate doses. And it would be nice to have that if what you would be doing is trying judge the priority that this should have as a public health problem and also juxtapose the potential costs and benefits of different mitigation measures.

Now that doesn't lessen the usefulness of this particular set of studies for giving some not clearly incorrect range of values for foreseeable exposures. But it doesn't serve the other needs that folks in OMB perhaps would be interested in.

Also under FIFRA, there is some balancing considerations and CPSC.

DR. HEERINGA: Any other comments for the Panel?

At this point in time, what I would like to do is to recommend that we take a 10-minute break and reconvene at 4:20. At that point, Dr. Matsumura will be acting as chair for balance of the afternoon. And we will turn to Issue No. 3. Thank you.

[Afternoon break taken at 4:07;
session resumed at 4:25 p.m.]

DR. MATSUMURA: I have a question. I would like to complete today's agenda, that means Question 3 and Question 4. If it goes over 5:30, can we extend slightly just to, let's say, 6? At 6, I can stop the whole thing.

With that, we can start with Question 3, please.

DR. OZKAYNAK: Okay. I'll proceed.

Issue No. 3. Key input variable and specification of associated variability distributions.

Sensitivity and uncertainty analyses of the SHEDS-Wood model results identified the following key input variables influencing the model results: Wood surface residue-to-skin transfer efficiency; wood surface residue levels; fraction of hand surface area mouthed per mouthing event; and GI absorption fraction for residues. In addition to the above variables, sensitivity, and uncertainty analyses also indicated the importance of following additional variables:  Average number of days per year a child plays around CCA-treated playsets; frequency of hand washing; daily soil ingestion rate; and average fraction of non-residential time a child plays on or around CCA-treated playsets.

Question A:  Has the Agency used the best available information for developing input distributions for these variables?  If not, are there any other data that EPA should be aware of?  Considering the limitations and uncertainties with available information, are the choices made in developing distributions for each of these key variables using the available information reasonable and scientifically sound?

DR. MATSUMURA:  Dr. Adgate.

DR. ADGATE:  Thank you.  There's a lot to this question or this series of questions.  And I'll start out by making some general comments.  But I was also going to suggest that in the interest of time we also sort of go through it variable by variable at some point. I've sort of made a table on my computer, and we'll go through them more or less in the order that you specified them here.

So I thought I'd sort of name the variable off, and I'll comment if I feel qualified on that. But will depend on the expertise of the Panel to address specific variables if people have concerns.

I wanted to start out by noting in your block at the beginning here, block of text, you've got four variables. There are sets of four variables; ones that are related to residue ingestion and the others that are related to activity patterns. And those are two very different issues. And we'll try to keep that straight as we work through this.

I wanted to start out by saying that I was on the 2000 panel that originally suggested that really the only way to deal with this problem is sort of a two-dimensional Monte Carlo model, and I wasn't on the last one. So it's been interesting for me to come and see this. I think it's really a major step forward, and I commend you on what you've done.

I think the major issue that at least I have and from talking to a number of people, I don't think the problem is so much with the models as with the inputs which is why I think we're going to have a fairly lengthy discussion here. But, hopefully, events will prove me wrong.

That said, I'd like to sort of talk a little bit about -- you know, the first question says: Have you used the best available information? And I would say, yes, given your time constraints. One of the things I think that this meeting has demonstrated is there's always little things you can tweak at the margins and new studies will come in. But I think you've done a good job organizing the available information, even if it's not published or not quite published.

That said there, there are, as Natalie alluded to, some data sets that you could access at least for ground-truthing. The one that I'm familiar is with the one from the Minnesota Children's Pesticide Study. There are sort of four questions on this kind of activity diary related to this. And what they address are the following issues: They're related to whether or not the kids had soil contact; did they bathe or take a shower; and how many times did the kid wash their hands in that day. So this is 102 kids, aged 3 to 12, 8 days of data. It's something that's in your possession.

I know this because I sent it to Chris Saint myself, burned the CD and sent him the documentation materials. So if you want to get a hold of that, talk to him.

To back up and get back to the question itself, the thing that I think I found most troubling or difficult is when I was reading the document was exactly -- I think you good did job. I spent a lot of time looking at Table 12, which, I think, is good, and then the text that is after that that describes it. The problem that you still have, I think, is sort of a naive reader coming into this is exactly how and where professional judgment gets incorporated in sort of a systematic way.

I think you did a pretty good job most of the time. But there's always points where you scratch your head a little bit, and say, it isn't entirely clear, the clarity could use some improvement. And I think as we go around and talk about specific variables, that will become clear exactly where they are.

I've been thinking a lot about sort of formalizing a process to sort of incorporate professional judgement in this. And I know that this is an active area of research. I don't have sort of a thunder bolt from the blue sort of process that I could suggest that you use, but other people may. And I'll defer to the rest of the Panel on that. But it's a hard issue, and I think everyone recognizes that, how you incorporate professional judgment and display it in your choices that work into a complex model like this.

Those are sort of my introductory comments. I thought sort of the useful thing to do at this point would be to start working through the variables. One of the things that I did when I started looking at this was, there's Table 12 specifically. There are 41 variables which involves some double counts because you have them, specific ones, listed twice. Of the 41 variables, 13 are listed as having point -- if you look under the column, it says, "distribution has the word point," which I found just a little disconcerting because I don't see how you can have a distribution on a point. But that's a minor issue.

In working through your list of variables, we can start out with the residue ingestion variables. And the first one is wood surface reside to skin transfer efficiency. I don't have any specific comments on that one, but we have people who have more expertise on that, and I will defer to other Panel members. Marcie.

DR. FRANCIS: Yes. My comment is that it isn't clear how the ACC data and the CPSC data were combined to come up with your estimates of the transfer efficiency. And also the fact that pretty much for all of these that are distributions, it really would be helpful to see a goodness of fit for all the variables. I think it was very helpful the document that Leila Barraj provided for us that actually showed the distributions for those variables.

But for those of us that want to see if they kind of make sense, that would be particularly helpful. And this is one example. That's also true for the wood surface residues for the case, I forget, warm or cold climate; but the cold climate scenario where both are used.

That's my main comment on the transfer efficiency.

DR. ADGATE: Dr. Kissel.

DR. KISSEL: I would reiterate that it would be nice to be able to see more explicitly how those two things link together, the two data sets you're using. Other than that, I don't have any other comments.

DR ADGATE: Any one else? Dale.

DR. HATTIS: I've done some analysis based on data Dr. Whey (ph.) gave me on the ACC wipe. Is that the next one?

DR. FRANCIS: That's the residue levels.

DR. HATTIS: Okay. The residue levels. All right. Then I'll defer that until we get to that one.

DR. ADGATE: We'll get there.

DR. XUE: Can I respond?

DR. MATSUMURA: So --

DR. OZKAYNAK: Can we provide a clarification to a point?

DR. MATSUMURA: Yes. Sure. Who would like to speak first on this clarification?

DR. XUE: First, I respond to the how to combine ACC data and CPSC data. The first point is very important is at what time the professional judgment is getting to. This is the one part of professional judgment is getting to. So we did look at CDF file, look at here and look in the media and look at the study, does it use the same protocol or different protocol. And we see that it is reasonable to combine the two studies. Basically, that we just combined the two. But we do look at the CDF and look at the mean value and there's a standard deviation is relative close. So this is -- we decided to combine the two.

In terms of the second one, we look at the results of how we present the results of goodness of fit. We put all these fitness of goodness of fit into the Appendix 3. All our fitted results we just put in Appendix 3. And this is how all the results of how were fitted. Some does not fit well. Some fits well. We only have, I think, 32 percent is can pass statistic test.

For others, we see the fit good enough, fit good enough so we choose that this kind of distribution. But we did do some analysis how robust it is when we change from one distribution into another distribution. And, in fact, I have some results to show you. Basically is that it is very robust to the distribution you set up for the important variable. We said always with changes of three important key input. Then the very robust to this what the distribution you said that.

DR. MATSUMURA: Dr. Francis.

DR. FRANCIS: That's good, and that's very helpful. I think maybe if you actually said that in the report and also then referred people to Appendix 3. Because you're right. I forgot that you had at least some graphs as opposed to goodness-of-fit statistics in the body of the report or referred people to where they could find them in the appendix, that would be helpful.

Also as it's true for all the comments where you have more than one source of data, if you could just say for each of those variables how you combined them that would you helpful.

DR. MATSUMURA: Good point. Dr. Reed.

DR. REED: I'm a little bit confused in terms of which one we're at right now. We're still on Issue 3A.

DR. MATSUMURA: Yeah. 3A.

DR. REED: Correct? Just the new data itself.

DR. MATSUMURA: Yeah, 3A. Yes.

DR. REED: Or are we looking at the primaries more than A, B, C, D?

DR. MATSUMURA: We're still on 3A.

DR. REED: Thank you.

DR. MATSUMURA: But the larger question of Question 3 includes special items which mention. You could address one by one. So it's up to you. You are the discussant.

DR. REED: No. Actually, my comment is slightly different than this.

Yes, I wonder about how, you know, two data sets are combined together. But I'm also wondering about how comparable data sets coming from separate studies are used for the general simulation not particularly about the key parameters. But what I did was to just line up all the values from comparable data sets together. And I was just curious about how data from different studies might differ. And that brings into my mind about the representativeness of these data.

For example, with arsenic playset soil or soil around the playset, it looks like the warm climate has much higher value, almost 10 fold, higher than the cold climate in this case; but the soil around the deck had a different direction. So the concentration in the warm climate is actually lower than cold climate. Then for chromium, it's sort of crisscrossed.

And I was wondering if you have any mechanism to sort of double check on the comparability of these data sets because they eventually go into the same simulation and their comparable variables and also the representativeness of that. I would appreciate some -- if that had been done, some description of that.

DR. MATSUMURA: Dr. Xue.

DR. XUE: That is right. The data -- we cannot because of the limited data. We do have some problem of how representative it is just as I say that some deck is high but the soil for one was, soil was high. But for cold weather, deck was high. Because this is all the data. We try very hard to get it. The data is very -- for soil, I think that for the playset for the soil, we only have 8 data points. But for the data for cold weather, we have more because 85 data points because, basically, that data is limited. That's why it is not very representative.

DR. OZKAYNAK: But, consequently, when the data sets are limited or the sample size are small, then during the bootstrap uncertainty fitting process, the uncertainty will capture that inherent limitation of the information. So it will be larger uncertainty with those fewer observations.

DR. MATSUMURA: Is that okay, Dr. Reed? Any question? Dr. Francis.

DR. FRANCIS: I just have one quick suggestion. And maybe Dr. Kissel can actually comment on it. And that's whether or not there's a Brower, et al., 1999, reference on dermal transfer. It was done for workers based on a fluorescent study. And whether or not those data could be used either to compare or to look at the transfer the efficiency issue. And like I said, maybe Dr. Kissel can comment on that.

DR. KISSEL: The EPA responded to that in the appendix. That's one of the specific questions.

DR. MATSUMURA: Use the microphone.

DR. KISSEL: EPA responded to that. And when you take the timing kind of questions into account of the apparently low transfer efficiency is for a very short or a single contact. And the context here is longer increments of time, and so there really isn't as much discrepancy between the Brouwer numbers and what EPA has done anyway. So I was satisfied with EPA's answer on that issue.

DR. MATSUMURA: Okay. Any other questions or comment? If not, could we go on to Question B?

DR. OZKAYNAK: Yes, Question B --

GROUP: Wait a minute.

DR. ADGATE: We haven't got to the different distributions. I thought we were to go through the different distributions.

DR. MATSUMURA: Oh, you were going to go one by one?

DR. ADGATE: Well, that's sort of implied in the question. It says that each of the key variables in that.

DR. MATSUMURA: All right.

DR. ADGATE: So we've only discussed one so far and there's eight.

DR. MATSUMURA: Okay. Yes. I was going too fast.

DR. ADGATE: The second key variable is wood surface residue levels which, if you're looking at Table 12 or items -- I've sort of numbered them from top to bottom to make it easier to keep track. For me, they're No. 17 and 18.

So it's wood surface arsenic residue levels on CCA-treated decks and wood surface chromium residues on treated decks. Dr. Hattis, you have?

DR. HATTIS: Yes. Could you put up the first slide there?

If you recall the discussion yesterday, we looked at some of these summary data from Table 10. And this particular distribution seemed not only to be influential; but the distribution looked odd in the sense that the highest values quoted in Table 10 looked like they were too high. The distribution might be asymmetric.

So I thought it might be a good idea to investigate this particular distribution particularly because there's huge amounts of data here.

In particular, I looked at the ACC study where as the CPSC data were also used for one of the two cold climate scenario. But I didn't look at the CPSC data.

This is the distribution kindly supplied to me by Dr. Schway (ph.) for the cold climate. And what you see is, basically, this is a frequency histogram. And this is a lot of samples. This is over 300 samples each in each for the warm and the cold climate states. Though the cold climate comes from one state, Pennsylvania, the warm climate data come from two states, Georgia and Florida.

But what you see here is well, you know, maybe a little blip that is suggestive of some second node; but one wouldn't be completely confident of it from this direct information.

The next slide will show the comparable data for the warm climate. Here it looks like things are a bit more spread out. But it doesn't look like it's classically fully log normal either. But you can be fooled on this kind of a plot for that thing unless you have the predicted data from a fitted log normal distribution.

So what I do for this kind of analysis usually is to do what's called "probability plot" where the Z-score which is sort of a position on a cumulative normal, or in this case log normal, distribution is plotted against the log of the values. And that's what's contained on the next slide.

And here the cold climate data are the squares. And in this kind of plot, essentially what you look for is many points in a row on one side or the other of the line. The intercept is an estimate of the geometric mean, and the slope is an estimate of the geometric standard deviation. So the steeper the slope, the more variability.

The correspondence of the points to the line is a rough qualitative indicator of how well a log normal distribution is describing the data. With this number of data points, this kind of test becomes relatively sensitive. So although I don't have goodness-of fit tests, the suggestion is there is a appreciable suggestion by modality particularly in the warm climate. This is a lot more variability. It departs, systematic departure of the points from the line particularly for the warm climate than you often see.

And to some extent, the two curves are reinforcing the same conclusion because they have qualitatively the same kind of departure. So it is as if there is a high percentile, you know, second mode to these distributions. And I would speculate that these data would be reasonably well described by a mixture of two log normals. There may well be other distributions that would also be used to describe these, but I would go with the mixture of two log normals because of the idea mechanistically that there could be two populations of decks or places on decks, maybe some involving microbial action and some not as one wild speculation.

The next thing I wanted to look at was, well, how serious is this potentially for the analysis. So what I did was I tried to at least look first at how the predicted mean from the fitted log normal distribution corresponds to the actual mean of the data, the arithmetic mean now. And that's on the next slide.

And what you see is the arithmetic mean of the data -- this is the simplest kind of analysis -- is the left hand and this is in units of micrograms per square centimeters, I believe, standard deviation, standard error. And you can compare that first set of numbers with the numbers in the fourth column. And what you see is that for the warm climate where we have the largest apparent departure, it looks like the fitted log normal to that would -- it would understate the real mean by about 12 percent. The correspondence is better, about a 4 percent departure, for the mean for the cold climate scenario.

Now, that's not too serious, essentially, at least in estimating what would stand for population mean exposure for these groups that were quantified. Likely the effects would be more serious at higher percentiles. I don't have a good way of working through what that would entail. As an alternative of -- I mean you can fit the two log normal to the mixed distribution or some other distribution. Or with this amount of data, maybe you aren't doling something really terrible by just using the empirical data themselves. I mean, that's not terrible when you have so many data points.

Now, there is a glitch, however. These 700-odd data points in the two groups together don't come from 700 different decks. They come from 25 decks. So there's an issue about which distribution should I be using. Should I be using -- first of all, is there, you know, some -- how much of the variance is explained by within deck versus across deck. I don't know the answer to that. I haven't had time to analyze that. But I'm sure many other people on the Panel and the EPA would be more competent than I am in answering that question.

But it also becomes a modeling question it seems to me, because while children maybe well be considered to visit the same deck repeatedly and that they would also -- they would not -- essentially it's clear that it may well be that it's the within-deck variance that should be incorporated as stochastic variance along the children for each child. And then it's a cross-deck variance that should be done once every year or every period from child to child.

So I think there's more to be thought about here in using these data to model this. But it's not all clear that each residue concentration that the child encounters should be a random draw from this either the warm or the cold. I'm not sure exactly if that's the way it's been implemented at this stage. But it seems to me that you could do a bit more in separately representing the within-deck versus among-deck variances.

DR. MATSUMURA: Dr. Xue.

DR. XUE: Let me respond to that intradeck variability and the interdeck variability. Basically, we know that if people go to a playset and deck, there would be not much change in terms of relative concentration. That's why the SHEDS model right now we just draw once. We draw once then assume this concentration will not change because we don't have information what's the variability it is. So that's why from, when you use concentration, we draw one. The concentration is still there. Always this concentration. This concentration will not change.

DR. HATTIS: So you're essentially assuming that the same child in any given period continuously encounters this same concentration. But if there is appreciable within-deck variability, you may well imagine that the child will go to different parts of the same deck and be effectively exposed to a random variable described by the within-deck variability.

DR. MATSUMURA: Yes, Dr. Francis.

DR. FRANCIS: Well, that raises another question. And I apologize. I mean I have been put on this committee about two weeks ago. I haven't had a chance to even play with the model and look at some of these things.

Are you saying then that a child is assigned a surface residue number for what period? For a day? For a year? For their whole six years?

DR. XUE: For one year.

DR. FRANCIS: For one year. Okay. Thank you.

DR. MATSUMURA: Everybody satisfied for that?

I had one question. If you're in that distribution, if there is analytical limit, detection limit, how would you do that?

DR. HATTIS: Oh, yeah. Log normal probability plots do very well at analyzing truncated data. Essentially, what you do is calculate the Z-scores and plot the data only for those points in the detected region. But the Z-score calculation, sort of reflects the whole number order of all of the data in the points.

DR. MATSUMURA: Truncated data, okay.

DR. HATTIS: So you can judge the fit to the data even when there's some significant amount of truncation.

In this case, that doesn't seem to be a problem.

DR. MATSUMURA: Yeah, yeah, it looks like. Yes.

DR. FRANCIS: I just have one more question. It's a short question.

The greater median for the arsenic cold climate scenario wood residue levels, is that possibly a function of combining the CPSC data with the ACC data or not?

DR. XUE: CPSC don't have warm data residue. So this not combined; only cold.

DR. FRANCIS: I'm sorry. If I said warm, I didn't mean that. I meant cold. But the median value for the cold climate residue is higher than it is for the warm. And I was just wondering if that was because of combining the two data sets.

DR. XUE: No. Because CPCS data set is a very, very small contribution. In fact it's very small overall because of all ACC data more than 300. And CPSC data is very, very small. And I think that it is 30-something. I don't remember exactly.

DR. HATTIS: The geometric mean within the ACC data set for the cold climate is .28. And it's .23 for the warm climate. So it's a little bit more.

DR. MATSUMURA: Any other comments? So in that case, the next variable.

DR. ADGATE: Moving down the variable list, apropos of my earlier comment about professional judgment, I've been sitting here thinking about it as we've been having this discussion. I think it would actually be useful to have, for at least you guys to make -- by "you guys," I mean EPA -- a table of professional judgment -- variables where professional judgement was a strong component. It would be nice to see there's a rank-order thing.

And one of the things I'd like to see in it, I'm looking at Table 12. I'm thinking about this. You don't have a sense of ends when you look at Table 12, like how big are these; is this particular variable -- the underlying data set that this particular variable is based on. And that's helpful as you cross reference as you move through the report. And this is one of the problems that

I've had as I worked on this over time.

The third variable in the list is fraction of hand mouth per event, which is the 28th variable in Table 12. It's fitted with a beta distribution. 3.7 is the central tendency.

Dr. Freeman, I suspect you have some comments.

DR. FREEMAN: Yes. And it's not necessarily with the fraction. In the text, you describe the 3- to five-year-old child's hand as being 200 square centimeters. That would probably be accurate for either the total skin surface top and bottom for a two-year-old for two hands or perhaps the total skin for a older person.

In the American Chemical Council-RTI Hand Wipe Study, their measurements for adult hand surface were from approximately 112 to 180 some-odd square centimeter which would suggest that this is an over estimate for a very small child. This number would influence the area that then has the residue loading on it. And that's why that is an important issue.

The fraction that is in the mouth I think is probably adequate even though, like everything else here, the database from which it was obtained is fairly small. Children don't do whole finger or whole hand mouthings typically. I have some data for you to help on hand surface areas which you can then adjust by your proportionality which I can give you.

It's from two different databases for kids 13 to 16 months old, broken down into four one-year periods so that you can actually see the incremental changes that take place. And it's not adjusted to the data that you're using which is taking the height of the kid and the weight of the kid and then doing an extrapolation. This is real hand data.

One of the things that you do say about the proportion is one finger is 10 percent of the surface area. Fingers are different sizes. And while I think for this exercise it's probably a good rough estimate, what you find with childrens hand are, not only are the fingers getting longer with age, but the ratio between the finger length and the surface of the palm changes so that you can actually see -- it's a dynamic thing on.

On the one hand, I say 10 percent is fine. But on the other hand, I say, if you really want to be careful about it, that you have to take into account these other things.

DR. MATSUMURA: Any other comments? Additions? Yes, Dr. Francis.

DR. FRANCIS: I guess I just want to reiterate, again. Because what you've done in the table is list a number of references and it's unclear how those references were combined to come up with your information, that a little more description may be in the section later on where you do talk a little bit more about each variable. I found it hard to follow.

DR. ZARTARIAN: I'm happy to describe more about the study that we used to get the fraction of hand surface area mouthed if people are interested at this time.

DR. FRANCIS: Is it one study. Or as you have listed in here, you have a number of references. So it was unclear whether or not you combined --

DR. ZARTARIAN: For fraction of hand surface area mouthed, we used the Leckie, et al., 2000 study. You may be thinking of the frequency of hand-to-mouth.

DR. FRANCIS: Yeah, maybe I'm thinking of the frequent. Sorry.

DR. MATSUMURA: Dr. Hattis.

DR. HATTIS: I remember you saying you have no mouthing events at night. But some kids, very young kids, suck there thumbs. And I think that happens more often at night than anything else. But do you have any -- is there any quantitative information available about that, or do you think that's too rare to bother with.

DR. MATSUMURA: Dr. Zartarian.

DR. ZARTARIAN: The only study that we're aware of that has information on fraction of skin surface area mouthed is this Leckie, et al., 2000 study. And I can tell you a little bit more about it.

It's a study that was conducted Stanford University in 2000 for the Office of Research and

Development in EPA which looked at 20 suburban children ages 1 to 6 years in the Bay Area of California. The intent of the study was to look at children's behaviors in an outdoor residential setting. They looked at frequency and duration of hand-to-object contacts including hand-to-mouth as well as surface areas of fingers and objects mouthed.

The children spent 78 to 100 percent of their time outdoors. So we did have some indoor data as well as outdoor. And 36 hours of tape were collected. A total 33 to 34 of those hours were in view. And they looked at the frequency of immersions into the mouth for different hand configurations: partial finger, full finger, partial palm with finger, and the full hands. And that's the data set that we used.

DR. MATSUMURA: Dr. Freeman, do you have any comment to add?

DR. FREEMAN: Nope.

DR. MATSUMURA: All right. Any additional comments? If not we will move to the next item, GI absorption.

DR. ADGATE: That's actually four variables by my count. It's the last four in the table. Two are point estimates, the ones for chromium. Both are one. And the arsenic residue either in -- chromium residues in soil and, I think, in CCA residues, though the table doesn't say that. And the arsenic residues both from dislodgeable and soil dislodgeable is 4.7 is the central tendency. And soil is 11.4. They were fitted with beta distributions. They are based on the ACC data. I have no additional comments on this. I don't know if anyone else does.

DR. MATSUMURA: Anyone else? Dr. Francis.

DR. FRANCIS: Again, it would be nice to see how the ACC data fit to your distribution. And maybe it is in Appendix 3, but I haven't looked at it.

The other thing is people who have been on the panel before for the FIFRA SAP, obviously, you probably came up with a rationale for the point source -- for the point value being 1. But if you're putting in a point value, clearly that's going to affect at least for chromium the uncertainty in this overall residue absorption number.

DR. MATSUMURA: Dr. Hattis.

DR. HATTIS: This was one of the variables where earlier the Panel had suggested consideration of a 1-hit transformation of a log normal intrinsic absorption rate. EPA's response in this case was that they were having technical difficulties independently getting information about the absorption rate and time. And I just want to point out that you don't have to have independent information. The time factor should not be inferred from the XML protocol more or less as you've done in this 12-hour assumption.

DR. MATSUMURA: This is an important item. Is everybody satisfied? Okay. In that case, we'll move on to the next item, the average number of days per year a child plays, other than California, of course.

DR. ADGATE: All right. I think I'm with at least a fair number of people who view this number with some caution. It is probably the most diplomatic thing I could say about it. I don't particularly have any problems with your description on page 66. But it's clear that, I think, from the public commentors earlier today, that everybody would like to see some longitudinal data at least and see how that would influence the derivation of this number.

I don't have any further comments. Anyone else?

DR. MATSUMURA: Anybody else? Dr. Francis.

DR. FRANCIS: This may or may not relevant. But as I understand it, the way you came up with the 126 and the 54 was to take the diary information and look at outdoor time and then adjust it for rain days and other things. Is that not correct?

DR. OZKAYNAK: Correct.

DR. FRANCIS: Is that correct or not correct?

DR. ZARTARIAN: We have a couple of supplemental slides we'll clarify how that was derived.

DR. FRANCIS: Okay. But while you're putting it up then, my question is: It looks like the categories that you took for measuring outdoor time took a number of outdoor times that were completely irrelevant to children playing on playsets. There is one that, I think, it's called "outdoor travel." Because you list some --

DR. ZARTARIAN: We'll need to clarify our approach.

DR. FRANCIS: You'll put it up?

DR. OZKAYNAK: There might have been some confusion with this issue about 126, 186, and 54, and what are the real numbers. They're not point estimates across the population, so I think it would be helpful to go over that.

DR. GLEN: This is Graham Glen.

There's really two questions here. The amount of outdoor time is determined by the relevant mapping of the CHAD codes to SHEDS-Woods outdoor categories. And actually there were slides on that that went by. It's Numbers 3 and 4. However, those categories don't directly determine the 126 or the 54 which are a fraction of the possible number of days with outdoor time.

DR. FRANCIS: While you're looking for it.

Yeah, I understand that. On page 24, Table 4, could you just tell me then how these CHAD locations assumed locations of potential playset contact were used?

DR. GLEN: Yes. There are three categories for outdoor time. There's outdoor residence, outdoor other, and outdoor travel. And roughly speaking, the outdoor residence and outdoor other were nearly 50 percent of the outdoor time each. And outdoor travel was about 2 or 3 percent of the total.

Diaries that have any outdoor time were allowed to be used in the assembly of the year-long diary. However, the existence of outdoor time does not imply playset contact necessarily because there's a multiplication by randomly drawn probability check. That's where this 126 and this 54 come into play.

Those numbers are used to derive the probability in that random check. The method for selecting 126 and 54 are heuristically derived from an argument given in the report about the number of rain days and so on. But it's clear that different values could be selected. These are just bounding scenarios which it's unclear exactly to how many children they would represent in the population.

DR. OZKAYNAK: Actually, those are the rough daily averages across different children. So when you go through the 1,500 simulations when you draw from the diaries and assign potential contact with playsets and decks, it can range from a given child, hypothetical child as low a number as 15 contact days across a year to all the way up to 275. The average might be around 126 for the warm scenario. For the cold scenario, it could be as low as only three days or four days at the low end to something about 120 days or something like that. So it's not that it's just one fixed number per person. So it varies from diary to diary and assignment to each child that's simulated.

DR. FRANCIS: And that seems to make a certain amount of sense. And I do understand how you've done that. So let me see if I can say this correctly. You looked for diaries that had any outdoor time, and any outdoor time was defined by, say, for the public playset contact, was defined by all those codes that you have down there irrespective of whether or not they might in reality represent a potential playset contact.

DR. ZARTARIAN: That's correct. And, again, the basis for that is that the distribution of outdoor time for the playground children was the same as for the others to give us a larger sample size. And I also wanted to clarify. I think what's confusing people is that the 126 and the 54 are point estimates. And I think a better way to explain it is, that if you remember back to the methodology talk yesterday, what we start off with we construct the year long diary using the eight-diary method. And then we assign -- we determine the number of days with possible contact with -- sorry. We determine the number of days with suitable outdoor locations. And then the next step is to figure out the contact days for that child.

And to do that, we need to figure out a probability that a given day where there's a diary with a suitable outdoor location, what is the probability that

that day is an actual contact day where they contact the treated wood. So we needed to come up with a probability.

And what we assumed was that in the warm climate scenario the child contacted the playset seven days a week minus 32 percent rained out days. That gave us the 68 percent probability. And in the cold climate scenario, we assumed that they played three days a week minus 32 percent rained out days for 29 percent probability. And I think that's an easier way to understand it.

Now the reason that we came up with the 126 and 54 was that other models and the way people had been thinking about this assessment was in terms of days per year that a child contacts treated wood.

So really all we were doing was converting those assumed probabilities 68 and 29 percent into a days-per-year for people to be able to relate to. And, therefore, 68 percent translates into 126 because the average one-year CHAD diary has 185 days with possible public playset contact time. That's where the 126 comes from.

But what we're really dealing with is a probability. And from one child to the other, the range of days with possible public playset contact time ranges from 20 to 36. So even though there's a point estimate for the average time, the possible contact time, we're actually using that to get a probability to apply to get a range from child to child. I hope that clears it up.

DR. MATSUMURA: Dr. Portier.

DR. PORTIER: What would it be clear to say then that for a given child the number of days is a binomial random variable with that probability .6, whatever it was, .67. You've worked it back. But in reality if we looked at 1,500 kids and we looked at the number of exposure days a year per kid, made a distribution --

AUDIENCE MEMBER: It's in fact on the screen there.

DR. Portier: -- it's a binomial with a mean of .6 something.

DR. ZARTARIAN: That makes more sense.

DR. OZKAYNAK: I think you're right which is supported by the graph on the screen.

DR. FRANCIS: That makes more sense.

DR. ZARTARIAN: We probably would have been better off just leaving it at the probabilities. But people had been about days per year, so we tried to work it backwards.

DR. MATSUMURA: Good explanation. Any other comments on this?

DR. ADGATE: Thank you for that explanation. I was kind of in the slow-learner group in school. When I read the points I read the points earlier today. And that happens quite a bit in thinking about that. And the one additional item relates to one of the commentors points earlier today.

And my question to you is: My sense is, given this explanation, that even if you change your activity patterns maybe to reflect, for example, bimodal, bimodal being spring and fall, say, in a really hot climate where the activity on a deck, let's say, went up in the spring, went down in the summer, went up in the fall, and it went back down, if this is a warm climate would not if you looked at the SHEDS output over a year, you would see something like a bimodal distribution. Your probability would go up in the time that they were spending on the deck.

If you incorporated something like that, I'm guessing that wouldn't change the LADD over a year.

Thank you. Any other comments?

DR. MATSUMURA: You can move on to the next.

DR. ADGATE: Frequency hand washing as a Weiboll distribution. It's 29 on my list here. I have no comments about this particular variable other than what I said before about having some data on it which I will provide to you or how to find it. Dr. Freeman.

DR. FREEMAN: Yeah. Even John's data is going to have the same flaw that most of the other data has which is you're getting it from parents. And parents basically say, three to five times a day other than for a few that give other answers. But the majority say three to five. Is that Weiboll or is that log normal? Whatever it was.

Anyway, it's a shifted curve. I think it's even more shifted than the 3 to 5 would suggest based on full day observations of the kids that hand washing is even less frequent than that. But as a rough preliminary distribution, that's probably fine.

DR. MATSUMURA: Dr. Portier.

DR. PORTIER: The Weiboll is a continuous distribution. This is a count variable; right? So how are you converting a continuous distribution to count? Are you grouping it by -- if it's 8.25, is it 8?

DR. GLEN: It's not actually translated into a count. It's translated into a probability per hour of the day. There's assumed to be 16 waking hours. And if you draw a value of, say, 3.5 from the Weiboll, then you take 3.5 over 16 as your hourly hand washing probability. And then you randomly decide. You see, because the activity diaries are not, in fact, continuous in time but broken into diary events, each of even either has or has not a hand washing event. And you decide once per hour on a random basis.

DR. MATSUMURA: Dr. Francis.

DR. FRANCIS: Just a point of clarification. The hand --

DR. GLEN: That also -- excuse me. That also means that hand washings are not at the same time each day because the random draws are redone.

DR. FRANCIS: The hand washing is listed as log normal not a Weiboll, but the same comment applies putting a --

DR. PORTIER: Hand-to-mouth activity.

DR. FRANCIS: Right. But the hand washing right below it is.

DR. OZKAYNAK: Excuse me. Can you clarify that? When you said "same comment" applies, what did you mean by that?

DR. FRANCIS: His comment about, you know, what are you doing with what's, in fact, a discrete event using a continuous distribution.

DR. OZKAYNAK: It's log normal and also

continuous.

DR. FRANCIS: Right. And perhaps you should explain then how it is used as probability because that went right by me, too.

DR. MATSUMURA: Any other questions. If not, now soil ingestion. Do you have any comment?

DR. ADGATE: I'm somewhat familiar with this data set. And this looks quite reasonable to me from what I've seen and the several analyses that have been done. Dr. Francis.

DR. FRANCIS: Yeah. I don't have any problems with the data set. I think it's probably a pretty good data set. It's just unclear to me what you did, exactly how you dealt with values greater than 500 milligrams per day. And if you came up with a value in a certain distribution that was greater than 500, did you go back to the distribution and resample, or did you set it at 500?

DR. XUE: Basically, that we don't do any redraw of the distribution. We round to 300, then keep the data. But we put label variable, say, that this is more than

500. This is less than the 500. For 500, more than 500, we assume that this is a pica child. For others, we used other for the analyses of table we include is of excluded of these children who soil ingestion larger than 400.

DR. FRANCIS: So for that day, for the whole year, that child is excluded from --

DR. XUE: Because we only draw once a year for the soil ingestion because we don't have intrapersonal variability. We only draw once a year not draw every day.

DR. FRANCIS: Thank you.

DR. MATSUMURA: Did you get that? Are you satisfied? All right. Try to address the next one, average fraction of nonresidential time a child plays on or around CCA-treated playsets.

DR. ADGATE: For warm and cold, these are beta distributions. Central tendency is 1.1 for warm and 1.3 for cold. I think we had some discussion earlier about why cold was bigger than warm. And I think I understand that now. So I have no further comments. Anyone else?

DR. MATSUMURA: Any other comments? I wonder why. It's the reverse of what I thought.

DR. FRANCIS: I think it's reversed because we sort of beat it to death with the previous discussion.

DR. MATSUMURA: Dr. Xue.

DR. XUE: I think that is the -- we did not find any the change basically that this sample size is small when we gathered the data. Therefore, it's reversed.

DR. MATSUMURA: All right. Any questions about or comments? If not, we finally can move to B. Is that agreeable? Oh, Dr. Francis.

DR. FRANCIS: Just before we go to B. Clearly this question asks specifically about those variables. And there may be people on the Panel who have similar type questions on any other of other variables in Table 12.

DR. MATSUMURA: Yes, yes, yes.

DR. FRANCIS: I don't want to make more work than we have. But this might be the best time to look at that.

DR. MATSUMURA: How specific would you like to get your answer? The question's asked.

DR. OZKAYNAK: I think so far the discussion has been quite informative.

DR. MATSUMURA: Okay. All right. Okay. In that case, we'll try to go on to the next question.

DR. OZKAYNAK: The next question I believe it's B.

In some of these instances (see Table 12, page 58), because of data limitations, the Agency has made simplifying assumptions to represent them as point estimates based on professional judgement. Are the simplifying assumptions presented in the draft exposure assessment for making these decisions adequately supported by relevant scientific data? Are the choices made to quantify these variables, i.e., selected distributions or point estimates, reasonable and sound?

DR. MATSUMURA: All right. Dr. Adgate.

DR. ADGATE: One of the reasons I wanted to do what we did in Section A is basically I thought we would capture most of the answer to this. And I think we have.

The only thing that I sort of have written. And most of what I've written before we got to this was having to do with professional judgement and it not being as explicit as, I think, the Panel and I would like to see it. And we sort of have beat that horse enough already.

One thing that I'd like to see that would have helped me as I was reading this table, which has to do with professional judgment issues, is -- we're going to get more into the uncertainty analysis in one of the future questions. But is some indication of whether or not a variable is uncertain. And one of the things that took me a long time to realize as I was reading this was there were several different types of uncertainty. While you do a formal uncertainty analysis, how you identify which variables to subject to an uncertainty analysis is not very well identified in the document at least not to me as a naive reader of it.

That's getting a little far afield. But I have no further comments in respect to Question B.

DR. MATSUMURA: Yes, Dr. Reed.

DR. REED: Sort of a simplified answer to that question.  I would agree that when you have sufficient uncertainty, then I think it's desirable not to do a distribution or not to use a distribution if you're so uncertainty.  Sort of as a tentative, I think point estimate is reasonable.

Actually, what I was going to propose is something that's not going to be very popular.  But I will throw this out.  By looking at -- and this is really a credit to the team that has done such a great thorough job in your variability analysis, and uncertainty analysis, sensitivity analysis, that what I did was I -- well, first of all, I am one of those people who are naturally cautious about large models with many parameters and so many parameters inputs are in distributional form.  Is scares me.  And so when you see something that is giant and scary, I kind of stand back and take another look.

What I saw was that certain scenarios did not really make such a great difference in the outcome.  For example, I think it's -- let me see if I still have the table.  Let me find it, so I can be specific about it --

about the exposure from a playset. It does not matter whether you have the deck or having the deck in the component of it. It doesn't matter whether it's residential playset or public playset. The playset component actually has very much identical exposure levels in terms of milligram per kilogram day.

So what I was thinking is now that you have done such a thorough analysis, what I think -- let me rephrase this.

What I think is that it's somewhat desirable in my mind to be as simple as possible and not to go for the whole probabilistic if it's not going to have value-added as you're going to increase the complexity. And so now you see certain scenarios do not change.

I would like to see the team consider this approach. To step back and take a look at which component really is changing based on the distributional type of analysis and actually set more parameters to point in order to get a clearer picture of what is the variability from the outcome of the probabilistic. And I think there are several advantages.

One, it's easier for a reader to understand. The other thing is that when you get into the next step of making comparisons with other analyses, many of them were point estimates and not probabilistic at all, that it's easier to identify what is it that's making the difference because now the model becomes simpler. And that's sort of my way of looking at it.

The sum total is you would not want to go too complex to sort of compromising your visibility for people to understand and only become complex because you need to, meaning that there is a value-addedness in it. And if there isn't, step back and try it without.

DR. MATSUMURA: Any feedback from the Agency. To simplify, eliminate some of those?

DR. OZKAYNAK: We'll think about it.

DR. MATSUMURA: Yes, Dr. Kissel.

DR. KISSEL: I have to take the contrarian tack on that one. I am bothered by any point estimates in ostensibly a stochastic analysis. If it's really a point estimate, it's a conversion factor and it shouldn't be listed as a variable. Also, I think you can't count on knowing what are the important variables indifferently as the model may change. And you've already seen that, which variables are important has shifted. And I think what you want to do is make your best shot at every available in stochastic form.

Because one of the things that happens is that the naive reader will look at the output, whether it's the variability or the uncertainty output, and assume that you've accounted for everything. The kind of gut-reaction is to look at a plot and say, well, that's the spread and that's all that can happen when, if fact, you may have understated. And certainly in this case -- we haven't gotten there yet -- the uncertainty is certainly understated in this case. And it can be very misleading to have an ostensibly stochastic analysis that is incomplete.

So I would encourage you to try to fill in some type of probabilistic estimate everywhere. For instance, some of those things that it lists, we pick this based on expert judgment and consultation and it will be there sources listed. Presumably those three sources didn't all tell you the same number. So you could take the expert judgment type of approach and put in each of those three numbers with equal probability and that could be your distribution.

DR. MATSUMURA: Dr. Francis.

DR. FRANCIS: I agree completely with Dr. Kissel with a couple of caveats. One is once you've put a distribution on something, you've kind of legitimized that distribution. And I think as long as you realize that with more data or with more information that can change. That may be important.

The second thing is that even if you do keep something as a point, it would be very helpful for the sensitivity analysis to at least vary that point by some amount because clearly not everything that's listed as a point source will not come out in your sensitivity analysis. And we don't know how important those variables are for exactly the reasons because they don't have a distribution.

DR. GLEN: Most of the point values were, in fact, changed by a factor of two in Table 28.

DR. MATSUMURA: Dr. MacIntosh.

DR. MACINTOSH: I just want to make sure we're clear about this distinction between variability and uncertainty here. I can identify five factors that are expressed as point estimates in the model which are representations of population level parameters. They are, for example, the fraction of children with a CCA-treated home playset. Now that is the fraction presumably of the modeled population. There's a single true value for that number. Right? There's one fraction. We just don't know what it is. But that fraction doesn't vary among children.

DR. GLEN: Yes, it shouldn't have variables.

DR. MACINTOSH: As such, it should be in a variability sense as a point value. However, it's an uncertain value. And so it should be incorporated into the uncertainty analysis. And that's the subject of Question 4 or Issue 4. I just wanted to make it clear of that distinction here.

DR. KISSEL: That's fine. If you're defining a population for purposes of the simulation, obviously, that isn't a variable any longer.

But I wanted to respond to the previous point about these things were varied by a factor of two. If you don't know enough about them to do anything but a points estimate, then a factor of two probably is an inadequate representation of possible range of what that value might be.

DR. MATSUMURA: Dr. Zartarian.

DR. ZARTARIAN: I just wanted to point out. I was looking through the table to see which ones were point estimates. And two of them are, the fraction of children with the treated playset and the fraction of children with the treated deck as Dr. MacIntosh pointed out. So for the variability runs, we wouldn't change those.

The average numbers of days per year that the child plays on or around treated playsets, those are point estimates, the infamous 126 and 54 as we've talked about. And they have to be point estimates because they're divided by the average number of days a year in CHAD to get that probability. So that's why those are point estimates.

And the only other ones are the chromium related absorptions rates. I just wanted to point out that those are the ones.

DR. FRANCIS: There is one more. The fraction of total body, nonhand, skin surface area that is unclothed for the cold scenario.

DR. MATSUMURA: Yes.

DR. OZKAYNAK: I just wanted to add my personal thoughts into the discussion here about whether to go with point estimates or full stochastic.

My personal preference is along the lines of what Dr. Kissel expressed, to the extent possible, to quantify the extent of the knowledge or the extent of the variability. However, we tried very hard. We really started this process by really trying to indeed go fully stochastic in all variables. In talking to experts, it hasn't been that easy to get numbers from them that were numbers that they were willing to assign any kind of reliability or support behind it. And it became very clear that it was going to be very difficult to even generate some defensible distributions from the information that we will gather from either personal contacts or reviewing the literature on these limited types of information.

If we go ahead and really spend more effort on trying do that, what I fear is that next year when we come, we'll have much more of a debate like we just heard from Dr. Kissel; why did we take a range or factor of two or a factor of four. How uncertain it is? That's a whole different arena in terms of how do you do the expert solicitation and how do you quantify those ranges.

It is not that hard to be able for us, the Panel, among ourselves, to assign certain bounds perhaps with consultation with experts in the field. But trying to sort of make those decisions hold in terms of a risk assessment application that's being considered right now where a number of stakeholders and a number of different scientific views are going to play into it.

So those are sort of some of my sort of concerns how to sort of incorporate this advice that we've received on two extremes here. I see the pros and cons of either suggestion. But perhaps we can visit this issue under the uncertainty component as we go along today and tomorrow.

DR. MATSUMURA: Thank you very much for that explanation.

Any additional comments from the Panel. Dr. Adgate? No additional comments. Dr. Reed, are you satisfied? All right. I would like to move on to Question C.

DR. OZKAYNAK: Question C: Are the methods used for fitting variability distributions that are assigned to model input variables for the CCA assessment appropriate?

DR. MATSUMURA: Dr. Adgate.

DR. ADGATE: I think I'm going to defer to a card-carrying statistician on this particular issue. I didn't have a problem with what you did. And we'll let Ken explain why. I don't have a problem.

DR. MATSUMURA: Dr. Kissel, would you like to start?

DR. PORTIER: I'll get it.

DR. MATSUMURA: Okay. Dr. Portier.

DR. PORTIER: If you look at Table 8, it describes how the data were used but not necessarily how the variability distributions were fit to the raw data. In my original reading of the document, I thought I read that you used maximum likelihood and method of moments. Or that might have been in the presentation. I couldn't find the page, chapter page. But I think you mentioned that in there. Right. Were used to fit the distribution.

With maximum likelihood, you have to have adequate data to be able to do that. But you can also use things like AKIKE information criteria, AIC indices to indicate why this particular distribution was I chosen over another one. Right? I didn't see you do that. And maybe when you have a lot of data, you don't have to do that because it fits well.

The bigger problem comes when you use the method of moments. And, typically, you're obtaining estimates by linking the data moment to a theoretical distribution moment. So you're saying I've got a few data points here. Here's the mean. Here's the variance. I think it's binomial. Here's the mean and the variance of the binomial. I equate them, two equations, to unknowns. I solve. And that's my estimate. The problem is the method of moments is not a great way of fitting a distribution. And especially for things like the beta distribution, I have problems.

And then I don't really know what happens when you match the moments of a triangular distribution to the moments of a beta distribution which you've done in a number of cases. I guess I'd feel a lot better if you could just show me a plot of how well that fit on some of these situations so I could get a feeling that it looks correct.

For example, for a number of the variables on pages 65 to 76, you say things like, We fit a triangular distribution with minimum mode maximum, and then fit the triangular distribution to a beta distribution with bounds at zero and one and got these parameters. And I understand why you did that. I just feel the need to be a little more convinced that these distributions look right because the method of moments could give you a right skewed distribution and your triangular could be left skewed. It just doesn't match that strongly.

And it might even be better sometime for you to tweak those beta parameters so it looked like the triangular a little bit more and not depend on something like the method of moments. And I'm sure my other statisticians on the committee will beat me over the head for that statement.

Also I'd say, following Dr. Adgate's comments, it would be nice to identify distributions that were the result of this kind of personal judgment or team evaluation. And I'm assuming right now that any time you specify the triangular distribution with a min, max and a mode, that was probably something you came to as a team and said, you know, best judgment among us, talking to everything else; here's the range; here's our best guess; we have a triangular. The SAP told us they don't like triangulars, so fit a beta; so here's what we've got.

I think you just need to be able to kind of describe that a little better.

DR. XUE: First of all, let me clarify how we fit from triangle to beta distribution. Because I think the SAP on 2002 raised question, triangle is not good distribution. And, in fact, we agree. We would agree -- distribution is -- than one, you have truncated and less than one, less than zero, you have truncated. So this is not fittable.

But what problem is that sometimes we don't have data. We don't know the mean 5 percentile, 95 percentile. How we do that. So we think about that. We try what about if we can fit, use these three data points, we can fit a triangle distribution. Then use the triangle distribution try to use this data because it's the three data points that you cannot fit the distribution at all.

So now we borrow data from the triangle distribution. Because we use this triangle distribution, we fit a triangle distribution just like this slide shows, this triangle distribution. Then we get this triangle distribution then because of the fit of the triangle distribution, now we have much more data. There's enough of fittability of distribution.

Then we use this triangle distribution data to fit the beta distribution. Then we test to see that the whole fit between the triangle distribution and the beta distribution based on -- because we have no idea triangle distribution is better of it beta distribution is better. But if we have one thing we know that the beta distribution is the more suitable for this distribution because of how -- they have between zero and one. (Inaudible.)

So this is why we use the triangle distribution as the foundation distribution. Use this foundation distribution to translate into the beta distribution. So this is where it comes from.

In terms of the maximal estimate and the moment of method, we based that if we have more data, we test that there are no difference at all. Because this two results is very, very compatible. But if less data, so maximum (inaudible) we found is unstable.

Because we needed some help, we asked some experts from Douglas (inaudible) because he did a lot fit distribution. He suggested that the -- in this case the method of moment is more stable. But one thing that we use this fit because we also when get a distribution, we just use the distribution. We do fit different distribution. What about Weiboll distribution, log normal distribution, normal distribution, and the beta distribution. Which distribution would be more fit.

So in Appendix 3, we have all these fit to see what is a fit; what is not a fit. I mentioned it before. We use and the (inaudible) under the -- Edison Darling test to test this. Only 32 percent will pass the test.

70 percent, 70 percent, we did cannot pass it. So one thing we use to see how fit is it, we eyeball. This is the professional judgment that go there. Eyeball to see is the fit or not a fit. Which of it is from wipe just like I point out that the deck wipe does not fit well.

Because look at the data, in Appendix 3, we do see that the data residue concentration is not fit well at all. You look at Appendix 3, you can see that both for warm climate and there's a cold climate, does not fit well. Because we don't have a choice. I think that we will take your suggestion very seriously. We think about the empirical distribution or mix log normal distribution is a better way to go.

DR. MATSUMURA: Are you satisfied?

DR. PORTIER: Let me just comment. With my professional judgments and I look at that graph, I'd say you have the wrong dates parameters, the wrong parameters for the beta because that distribution doesn't fit particularly well. And I would have kind of kept going at this point.

And when you showed this graph yesterday, kind of a little thing went off in my head and said, now, I need to talk to them about that because to me that wouldn't have been the right beta for that triangular distribution. I would have shifted the mode over a little bit more and tried to capture more of the distribution.

So we maybe need to talk about kind of formalizing that, especially in that situation. I'm not worried when you have a lot of data. I'm sure you're doing that right. It's that situation where you're converting professional judgment into a distribution. And I'm not even sure how much of a little change that I would want to make is going to affect everything else that goes on either. That's the kind of concern that comes out here.

But I guess in light of the validation issue, you need to be certain that every one of these kind of stands and is defendable.

I'll go back and look at the appendix tonight.

DR. MATSUMURA: Okay. Would you like to

comment?

DR. SMITH: This is Luther Smith.

Just as a quick point of clarification to what Dr. Xue mentioned. The foundational triangle was not really fit. It was established based on the means and standard deviations that usually is what we were limited to in those cases.

DR. MATSUMURA: Any other comments?

DR. MACDONALD: It just seems to confuse things to put the triangle in there at all. It would be much simpler if you just went right to a beta and then we wouldn't be arguing about how you got between the triangle and the beta.

DR. SMITH: The problem with doing that is that generally speaking we were limited only two reported means. The standards deviations, we did not have the data to fit it to. Obviously, if you got the raw data, you're in better shape to do fitting. We tried to cover most of the data with the triangle to begin with.

DR. MATSUMURA: All right. Yes.

DR. ZARTARIAN: There were only four of the variables where we fit triangulars and then betas where we had some data to use the standard deviations. The other ones, such as this one, were just what you said, where we had no information and we just got together and used our best judgment. So it's probably not as critical for this one that the beta doesn't exactly.

DR. MATSUMURA: Thank you. Dr. Hattis.

DR. HATTIS: I had a slightly different bone to pick. This is a very important issue. My response was that it appears that where more than one study was available to estimate variability in part log normal distributive parameters like the daily soil ingestion rate, the study team has taken an arithmetic average of geometric standard deviations.

And in another case, the soil skin adherence factor on page 73, it seems that the study team chose to compute a simple average of variances. What I would suggest what in general what you want to do is to combine within study variances you should generally be combined by computing weighted averages of the variances. And I'll give you a formula that I will pass by the real statistician here. So I will reproduce that.

But essentially what you're doing is a weighted, and N minus 1 weighted average of the variances.

And I also comment on the foundational triangle a little uncomfortable with that. Where you have data, you should try to do it directly. And maybe where you have subjective estimates, you could either just do it with a group of people sitting there and saying, does that look right. Maybe that's just as well.

I mean the eyeball is, in fact, I think a pretty decent integrator of information.

DR. MATSUMURA: Any other comments? If not, I would like to finish at least Question D.

DR. OZKAYNAK: Question D: The Panel is requested to comment on whether any other model inputs are either key drivers of results or sources of large model uncertainty. Do these model input variables and the distributions assigned to them appropriately reflect available scientific data? Did EPA appropriately integrate the available data to derive the distributions for these input variables?

DR. MATSUMURA: Dr. Adgate.

DR. ADGATE: In my opinion, we've pretty much covered this already; so I have no further comment. I feel like I've beat this particular horse with a stick of CCA-treated wood long enough.

DR. MATSUMURA: It's getting late. Yes, Dr. Kissel.

DR. KISSEL: Sorry to do this at five to six or whatever it is. Because this is slightly off point in that the question is key variables. And I would just say that I think that some of the inputs that are not necessarily key variables have credibility problems that some of the industry people pointed out today. And I think that they should be adjusted for sake of overall credibility of the exercise regardless of whether they dramatically alter the outcome. And one, for instance, that I'm thinking of is the fact that the finger-licking efficiency is greater than bathing or hand washing which raises issues. And I think you might want to look at some of those things.

DR. ZARTARIAN: We have. And Dr. Xue will show that if that's okay to show some supplemental slides to look at those other variables.

DR. XUE: In fact, industry make comments we look at this -- look at what's impact it is. So let's go to slide X4-46.

So we look at we it -- we did not for the hand-to-mouth activity, we did not -- we put indoor and outdoor together. Then we changed the model, and we spread it outdoor and indoor. Because we use more data, we fit the data a little different because that data is very limited. We used some data from the mean and the standard deviation and assumed the log normal distribution.

And then we generated the median and compared the median is compatible or not. And it seems like they're compatible. So we use this data, we gather more data. So we spread this for the hand-to-mouth for indoor and outdoor. We look at this, there would be changes very, very small for if we spread indoor and outdoor.

DR. KISSEL: I don't think he responded to the point I was making. Maybe I wasn't clear. But I was specifically saying that I don't care whether it changes the model output or not. It's a public relations sort of thing. If an industry guy can stand up at a meeting and say you made this assumption to the general public and the general public says that doesn't make any sense, it doesn't matter what impact it has on the numerical results. So I think you want to look to those sorts of things and make sure they're covered regardless of whether the changed the overall answer.

DR. OZKAYNAK: Sure. I think you're both right. You're absolutely correct in what you said. And Jim was saying, I think, was that we did not have sufficient information when we first generated the model results. And that's why we had to come back do a supplemental analysis.

But at this point, let me go back and revisit it again. And, obviously, we will definitely consider any appropriate changes to the inputs or even the rest of the model code for matter to address reasonable and justifiable comments that are raised by SAP and the rest of the public.

DR. MATSUMURA: Good points. Any comments you would like to add? I guess it's getting late. So if not, please, make sure to write down whatever your key points and give to the chair, that chair there, your discussant leader, so that we have good records because we don't want to lose any of those important points.

With that, I really would like to thank everybody. That was a good discussion. I enjoyed it.

MR. LEWIS: And I want to thank my colleagues on the Panel for being so engaged during the course of today's discussion.

Just to give you some guidance for tomorrow. We'll be beginning tomorrow at 8:30. The Agency will have an opportunity to have any follow-up, clarification, from points presented today.  And then we'll continue on with Issue No. 4 and complete the other questions by the close of business tomorrow.

For Panel members, you might want to use this evening as opportunity to collect your thoughts for tomorrow's and meet individually with people you're assigned to per question and share you're thoughts with them as we get ready for tomorrow's meeting.

Thank you.  Have a pleasant evening.

CERTIFICATE OF STENOTYPE REPORTER

I, Jane F. Hoffman Stenotype Reporter, do hereby certify that the foregoing proceedings were reported by me in stenotypy, transcribed under my direction and are a verbatim record of the proceedings had.

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JANE F. HOFFMAN

**I-N-V-O-I-C-E****    ****I-N-V-O-I-C-E****

JANE F. HOFFMAN

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