FIFRA SCIENTIFIC ADVISORY PANEL (SAP)

OPEN MEETING

SELECTED ISSUES ASSOCIATED WITH THE RISK ASSESSMENT PROCESS FOR PESTICIDES WITH PERSISTENT, BIOACCUMULATIVE AND TOXIC CHARACTERISTICS

U.S. ENVIRONMENTAL PROTECTION AGENCY

CONFERENCE CENTER- LOBBY LEVEL

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OCTOBER 29, 2008

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FIFRA SCIENTIFIC ADVISORY PANEL

MEETING

OCTOBER 29, 2008

MS. CHRISTIAN: Good morning. My name is Myrta Christian; I am the designated Federal Official for this meeting; and I would like to welcome everyone today to this meeting to review Selected Issues Associated with the Risk Assessment Process for Pesticides with Persistent Bioaccumulative and Toxic Characteristics.

Again, I would like to thank the panel, presenters and the public for participating in this meeting. Also, I would like to remind everyone that all the documents related to this SAP meeting are available at the EPA docket, in addition to our EPA website.

For presenters, panel members and the public, please identify yourself and speak into the microphone provided, since this meeting has been recorded. I look forward to another day filled with lively discussion and great panel participation.

At this point, I would like to introduce Dr. Steve Heeringa, Chair of the FIFRA Scientific Advisory Panel.

DR. HEERINGA: Good morning, everyone;

and hello again. As Myrta indicated, I'm Steve Heeringa; I'm with the University of Michigan. I am a biostatistician, applied statistician, with a specific expertise in research design for population-based studies.

I would like this morning, again -- I don't know if we'll do it each morning this week, but certainly on the second morning, to have each of the members of the panel introduce themselves and provide a little bit of background for the EPA participants and the public.

DR. SCHLENK: Good morning. My name is Daniel Schlenk; I am a professor of aquatic ecotoxicology at the University of California Riverside. My area of expertise is mode of action of pesticides in aquatic organisms, and I'm a member of the permanent panel.

DR. POPE: Hi, my name is Carey Pope; I'm a professor of toxicology at Oklahoma State University Center for Veterinary Health Sciences. My area of interest is mammalian toxicity and organophosphorus neurotoxicity.

DR. PORTIER: Good morning. I'm Ken Portier, director of statistics at the American Cancer Society national home office in Atlanta. I'm a

biostatistician and a member of the permanent panel.

DR. CHAMBERS: I'm Jan Chambers; I'm a professor in the College of Veterinary Medicine at Mississippi State University. My area is pesticide toxicology, and I am a member of the permanent panel.

DR. BUCHER: I'm John Bucher; I'm a toxicologist at NIEHS. I'm the Associate Director of the National Toxicology Program, and I am a member of the permanent panel.

DR. DONNELLY: Good morning. I'm Casey Donnelly; I am a professor in toxicology at Texas A&M University School of Public Health, and my expertise is exposure assessment in complex chemical mixtures.

DR. ORIS: I'm Jim Oris; I am a professor of zoology at Miami University in Ohio, and my expertise is in aquatic toxicology.

DR. SIMONICH: My name is Staci Simonich; I'm an associate professor at Oregon State University in the Departments of Chemistry and Departments of Toxicology. I'm an environmental chemist, and my specialty is in long-range and regional transport.

DR. STEENHUIS: I'm Tammo Steenhuis; I'm a hydrologist and I come from Cornell University.

DR. THIBODEAUX: I'm Louis Thibodeaux,

Professor of Chemical Engineering at Louisiana State University. My field of expertise is chemical transport across natural environmental interfaces.

DR. MEHTA: Ashish Mehta; University of Florida; coastal hydraulics and sediment transport.

DR. MEADOR: James Meador, environmental toxicologist at the National Oceanic and Atmospheric Administration in Seattle, Washington.

DR. NORSTROM: Ross Norstrom. I am an adjunct research professor at Carlton University in Ottawa, Canada, and a private contractor, formerly a research scientist with the Canadian Wildlife Service. My expertise is, I'm an environmental chemist with a focus on food led biomagnification to wildlife.

DR. MADDALENA: Hi, I'm Randy Maddalena; I'm at Lawrence Berkeley National Lab; environmental chemist, analytical chemist, and do a little bit of fate and transport modeling.

DR. LICK: Willy Lick; University of California at Santa Barbara; sediment contaminant transport in surface waters.

DR. HICKIE: Brendan Hickie; I'm a professor in Environmental Science at Trent University in Ontario, Canada. My specialty is bioaccumulation in the aquatic food web.

DR. GAN: My name is Jay Gan; I'm from the University of California Riverside; a professor in Environmental Chemistry. My specialty is fate and transport of para -- of pesticides.

DR. DOUCETTE: Bill Doucette; Utah State University; environmental chemistry.

DR. DELORME: Peter Delorme; I'm with Health Canada, Pest Management Regulatory Agency, with expertise in the area of environmental risk assessment of pesticides.

DR. BIDLEMAN: Terry Bidleman with Environment Canada; an adjunct at University of Toronto; environmental chemist.

DR. ABBOTT: Linda Abbott; USDA's Office of Risk Assessment and Cost-Benefit Analysis; I'm a regulatory risk analyst; and my area of expertise is ecological modeling.

DR. HEERINGA: Thank you very much, again, members of the panel. Before we begin today's session, just a note of appreciation to John Bucher for filling in for me yesterday afternoon while I had a teaching obligation. John, I understand that you kept everything very well on track, so we are starting out exactly on our agenda this morning.

At this point in the process, we have

heard the first set in the sequence of presentations in which we expand on the topics covered in the White Paper, the presentations from the scientific staff of the EPA.

Before we open and turn to our first presentation of the morning, I'd like to turn to Steve Bradbury and Don Brady to see if they had any opening comments, any follow-up from yesterday. Don.

DR. BRADY: Thanks, Dr. Heeringa. I think we're ready to proceed --

DR. HEERINGA: Okay.

DR. BRADY: -- unless there's anything from--

DR. HEERINGA: No. Very good. Thank you, Dr. Brady.

At this point in time, then, I'd like to introduce Kristina Garber, who is with the Environmental Fate and Effects Division, and she's going to cover the topic of the Assessment of Terrestrial Bioaccumulation.

Good morning, Kristina.

MS. GARBER: Good morning. Thank you. Good morning, members of the panel.

This presentation will start out with a brief review of relevant definitions for this topic.

I'll go into a conceptual model depicting bioaccumulation in the terrestrial habitat, and then go into the current OPP approaches for assessing exposures to terrestrial organisms.

I'll discuss some future directions that OPP could potentially pursue for assessing terrestrial bioaccumulation, and then discuss some of the models that are available in the literature that can be used to assess bioaccumulation in terrestrial habitats.

As we've already discussed yesterday, bioaccumulation is defined as the net uptake of the chemicals on all the exposure routes that would be relevant to that organism.

Biomagnification is an increase in the chemical concentration in the tissues of the higher-trophic-level organisms as compared to the lower-trophic-level organisms that that higher-trophic level would be feeding upon.

This is a conceptual model depicting bioaccumulation in a terrestrial system. Spatially speaking, this habitat could be the treatment site where a pesticide would be applied; it could be an area adjacent to a treatment site. And thus, this area would be receiving pesticide from direct deposition from an application or from spray drift from an

application.

Alternatively, this terrestrial habitat could represent an area that is far away from a treatment site; and thus, it would be receiving pesticide mass from long-range transport and subsequent deposition.

And in this figure, the arrows represent movement of pesticide mass from different -- between different compartments. The compartments are represented by air, soil, plants and animals.

Pesticide mass can move into organisms from the air through gas exchange or from deposition, whether that be wet or dry deposition. Pesticide mass can move into organisms from the soil, through uptake, either by direct ingestion or through dermal contact, or through -- no. Sorry.

Once into them, pesticide mass can move from one organism -- one trophic level to another through trophic transfer.

So plants could be exposed to a pesticide through uptake from the soil, from air exchange, and then, through wet or dry deposition.

Animals could be exposed through dietary uptake, whether that be ingestion of soil or ingestion of organisms that have accumulated a chemical in their

tissue. They could also be exposed through inhalation, through dermal contact or through ingestion of contaminated drinking water.

Organisms can eliminate a chemical through several different routes, including respiration, through incretion -- excretion of different fluids, including urine, feces or milk. They could also eliminate a pesticide or chemical through biotransformation into a less-toxic form.

As with the aquatic exposure assessment, OPP used a tiered approach for assessing exposures to terrestrial organisms. The first tier is intended to be conservative, and it's assumed that the non-target organisms are located either directly on a treatment site or adjacent to the treatment site.

And with this, it's assumed that a pesticide concentration will be highest at the treat -- at the treatment site, with decreasing concentrations as the distance from the field increases.

The Tier I approaches for assessing exposures to plants and animals do not explicitly account for bioaccumulation.

There are some refinements available to OPP risk assessors to account for the routes of exposure that aren't accounted for in the Tier I

approach; and some of those approaches can be used to account for bioaccumulation to some degree. And those refinements include models as well as empirical data.

The Tier I approach for assessing exposures to terrestrial plants involves use of the Terra Plant Model.

In this model, it's assumed that non-target plants are located directly adjacent to a field. They are receiving spray drift and runoff that would contain a pesticide directly from the treatment site.

And in this model, it's -- exposures are assessed from single pesticide applications only, multiple applications that may be made over a season or a year, are not assessed.

This pesti -- this model does not assess bioaccumulation in plants, and so it cannot be used to estimate concentrations of a pesticide in plant tissue.

This is the conceptional model that Keith Sappington introduced yesterday. The blue-highlighted portions of the model depict the parts of the model that are represented in the Terra Plant Model. In this conceptional model, you can see that pesticides are applied to a field, and then they're moved offsite through spray drift and runoff to

terrestrial plants.

For terrestrial animals, the Tier I approach involved use of the T-REX Model. This model was used to assess the site exposure to herbivore and insectivore, and the animals in them, and birds.

Exposures assessed using T-REX involve dietary uptake only for pesticide residues that are on plants and insects. The T-REX model does not involve pesticide uptake through dermal contact, inhalation or drinking water. It also does not account for bioaccumulation in a terrestrial habitat.

Each bioaccumulation would be something that -- of interest to PBC chemicals; there is concern that the comparability of the EECs that are generated using the T-REX Model to excessive exposures to terrestrial animals that would be expected from bioaccumulation is an unknown.

This is the conceptional model again, depicting the portions of the model that are accounted for in the T-REX exposure portion of the model. This -- T-REX basically assumes that pesticide mass is directly deposited onto terrestrial food residues that would be ingested by terrestrial animals.

As you can see by the model, there are several other potential exposure routes that are

depicted as dotted lines. It could be -- that could represent pesticide exposure routes for terrestrial animals. These are dotted because they're not part of the Tier I approach. In cases where a particular route of exposure may be of concern for a pesticide, a risk assessor may utilize other tools that are available to assess exposures through those routes.

For assessing potential bioaccumulations, there are some models available to OPP risk assessors. One of these models is the Earthworm Fugacity Model. This basically can be used to estimate pesticide concentrations in earthworm tissues, and then assess an exposure through dietary uptake through -- of those earthworms. Exposures can be assessed to mammals or birds that would consume the earthworms.

This model is typically used for pesticides that have a granular formulation that would be incorporated into the soil, but it could potentially be used with pesticide concentrations that are generated using the PRZM Model. And that was something that -- that was an approach that was discussed yesterday in the discrete sections.

KODAM is a bioaccumulation model that incorporates the Arnot and Gobas bio, aquatic

bioaccumulation models. It's an -- that model is used to estimate pesticide concentrations in aquatic organisms; and then, dietary in some species of birds, mammals and birds that would consume those aquatic organisms. This model is currently undergoing QA/QC within EFED.

The Tin Model is a probabilistic model used to assess acute exposures to birds. This model includes multiple exposure routes that are relevant to birds, including dietary uptake, as well as inhalation, dermal and drinking water.

It has the ability to assess bioaccumulation in the bird that's being assessed; however, in order to do that, it would be necessary for the model user to input elimination rate constants that are derived externally.

So, getting back to that conceptual model one more time, highlighted in dark blue is the portion of the model that's incorporated into the T-REX of Tier I approach. The blue portions of the model that are -- that are dotted represent exposure pathways that EFED risk assessors have the -- have some tools available to characterize.

As you can see from these -- as you can see on the bottom in red, there is a -- right there --

trophic transfer is an exposure pathway for terrestrial animals that OPP risk assessors have a limited ability to assess. That can be done using, assessing trophic transfer from earthworms to birds or mammals, as well as trophic transfer from aquatic animals to birds and mammals.

However, there are no tools available for assessing trophic transfer from plants to animals, as well as from some smaller, lower-trophic-level animals to higher-trophic-level animals, such as from herbivores and insectivores to carnivores.

In addition to models, OPP risk assessors utilize empirical data that may be available to characterize the bioaccumulation potential of a chemical. Risk assessors can utilize available metabolism data for plants and for livestock, and perhaps for, for that.

If available, risk assessors will also use monitoring data from within field studies or from the scientific literature. Generally, studies that are available in the scientific literature are of limited utility for risk-assessment purposes, because the studies are generally not targeted to -- and the bioaccumulation that's observed in the study could potentially be from multiple sources. In addition,

these studies cannot be used to link a specific application of a pesticide to a field through the bioaccumulation of the earth.

Because trophic transfer potentially represents the exposure pathway of an animal, of a terrestrial animal, due to a pesticide, there is a need to have proof of this though for risk assessors to assess test site exposures resulting from trophic transfer. Now, in order to do this, this would involve modeling bioaccumulation and this would also require the ability to determine which pesticide would be expected to accumulate in tissues of organisms and thus move through trophic transfer into higher-level organisms. So that would involve having the ability to identify which characteristics of a pesticide would be expected to accumulate.

If OPP were to develop a tool to assess terrestrial bioaccumulation, this would involve representing the -- that conceptual model that I initially introduced in this presentation; and that would include representing the routes of exposure to terrestrial plants and animals, as well as accounting for the routes of elimination.

It would be necessary for risk-assessment purposes to have the ability to connect

a specific pesticide application to the observed bioaccumulation and the resulting trophic transfer. It would also be necessary for risk-assessment purposes to have the ability to represent high-end concentrations in the environment. So basically, for this model to be conservative.

Tool development would involve defining the non-target terrestrial habitat of concern, whether that be a habitat that's overlapping the treatment site or an area that's far from the treatment site. And this may involve consideration of specific sensitive species, such as endangered species. And finally, tool development would involve identification of specific mathematical models to represent bioaccumulation.

There are several bioaccumulation models present in the literature. This table represents some of the models that are in the literature. It's not meant to be comprehensive, but just to give an idea of the different processes that can be represented.

Generally speaking, for plants, there are models available to represent movement of a pesticide from air into plant tissues, as well as from soil into plant tissues. And all of the models depicted can be used in some manner to estimate concentrations of the chemical in plants.

If you look at the chemical-specific parameter in this table, in one way or another, all of these models rely upon the KOW of a chemical to predict the transport of the chemical from the media, and the model by Cho et al uses Koc as a determinant.

These models -- the models that would account for movement of a pesticide from air into plant tissues rely upon the vapor pressure of the, of the constant of a chemical.

Two of these models that are depicted rely upon partitioning coefficients that are specific to the plant being modeled. The model by Reader requires a partitioning coefficient between the cuticle of a plant and water, and the model by Cho relies upon a partitioning coefficient between the plant organic matter of a chemical and water. I mean, the plant organic matter and water.

And so, this brings up an interesting point when considering plant bioaccumulation models, in that the model itself may rely or be dependent upon the characteristics of the model plant that's being depicted.

There are some terrestrial bioaccumulation models in the literature that account for bioaccumulation in plants and animals. The model

published by Kelly and Gobas in 2003 tracked the transport of a chemical between air and lichen, lichen to caribou, caribou to wolf, as well as between the air and caribou and the air and wolf. This model uses the KOW and the KOA, the alcohol/air partition coefficient of a chemical to estimate chemical concentrations in the tissues of lichen, caribou and wolves.

The model published by Armistead and Gobas in 2007 tracked the bio -- the accumulation of a pesticide from soil into earthworm tissue, and then from earthworm into shrew, and then also from soil into the shrew tissue. This model also uses KOW and KOA to estimate chemical concentrations in earthworm tissues and shrew, as well as bio to soil-accumulation factor.

Both Kelly and Gobas and Armistead and Gobas concluded in their publications that biomagnification in terrestrial food webs may be predicted using the KOA and the KOW of a chemical. Specifically, they concluded that a chemical with a KOA greater than 10-to-the-5th, and a KOW greater than 10-to-the-2 has the potential to biomagnify in a terrestrial food web. Now, this is assuming that there is no metabolism of the chemical in the organisms.

This is of relevance to OPP because this indicates that a chemical that may not be expected to

bioaccumulate in an aquatic system may have the potential to bioaccumulate in a terrestrial system.

These conclusions have relevance to the example pesticides that are discussed in the White Paper and at this -- at the meeting yesterday. If you look at this table, the estimated log KOA values of all four example pesticides exceeds 5; and the log KOW values of all four example pesticides exceeds 2.

And so, based on the conclusions of Kelly and Gobas and Armistead and Gobas, all four of these example pesticides may bioaccumulate or bio -- I'm sorry, may biomagnify in terrestrial habitats.

To summarize, organisms can receive pesticides through several different outputs -- or, several different sources in the terrestrial habitat, and they can also eliminate a pesticide through several different mechanisms.

OPP's Tier I approaches for estimating exposure to terrestrial organisms do not exclusively account for bioaccumulation; and this is of concern for PBC chemicals because the comparability of the EEC as generated using the Tier I approach to exposures due to bioaccumulation is unknown.

OPP has some refined approaches available for characterizing exposures due to trophic

transfer, but these approaches are limited in that they're incomplete. There is a need for OPP to have some tools available to assess exposures of terrestrial organisms to pesticides resulting from bioaccumulation.

In order to effectively implement such a tool, it would be necessary for OPP to have an understanding of the chemical's characteristics that would indicate the potential of a chemical to move through trophic transfer in the terrestrial food web.

With that, if anyone has any questions?

DR. HEERINGA: Thank you very much, Kristina, for that presentation.

Are there any questions as to clarification? Yes, Dr. Norstrom.

DR. NORSTROM: Thanks for your presentation. I have two questions here, if I can find them.

The first one is: In your T-REX Model, it wasn't clear to me whether your insects are eating plants, or they're just kind of being exposed by direct contact.

MS. GARBER: Both plants and animals are -- it is assumed in the T-REX Model that they're exposed to the chemical through direct deposition. So those EECs that are generated using T-REX are a result

of -- they're a reflection of deposition just to the surface of --

DR. NORSTROM: Okay.

MS. GARBER: -- the organisms.

DR. NORSTROM: So there's no food-chain kind of thing at all in there.

MS. GARBER: That's right.

DR. NORSTROM: The other one was: I'm not clear what types of terrestrial habitats are assumed in your model. In slide 8, you had the plants -- it was about plants adjacent to the treatment site. And if that's the case, what kind of plants? And is it characterized? And it would seem necessary that you would have to do that in order to make any sense out of it.

In slide 18, you addressed some of this concern that seemed more focused on ten to the species, if I recollect, than it was, sort of, characterizing the actual type of vegetations in that area. It's just unclear to me how you would apply this model in real life, somehow.

MS. GARBER: Okay. So. . .

Okay. The first question related to slide 8; is that right?

DR. NORSTROM: It just -- well, I mean,

basically, my question is: How -- how do you apply this model actually? As an -- in an example ecosystem, do you define the kind of vegetation and that kind of thing? And then, what do you do with it?

MS. GARBER: In reference to the Terra Plant model, which is our Tier I approach that we're using now, we actually differentiate the plants between -- we defined them as either terrestrial or as semi-aquatic.

And the terrestrial plants don't have -- really, it's more related to where they're located, and that is -- it's assumed that they're located directly adjacent to the -- to the field where a pesticide would be applied. So they're not differentiated as broadleafs or monocots or anything, in terms of how we assess exposure.

DR. NORSTROM: But surely you must have to say something like total leaf surface or whatever per hector or something like that; right?

MS. GARBER: No, we don't do that in our Tier I approach.

Currently, our effects data for terrestrial plants are received in terms of an application rate. So the report -- the results are reported as of pounds a.i. per acre application rate.

And so, essentially, what happens in these effects tests is: There are two tests that we are concerned with. And I know it's a little backwards that I'm talking about effects first, but that's important to understand in how we do the exposures.

So, we have two terrestrial plant tests that are received as data to understand the toxicity of the chemical to the plant. One is the seedling emergence test, where pesticides -- I'm sorry, where seedlings of a plant would be present in the soil of -- of the pods, basically. And then the pesticide is applied to the surface of the plant -- or, of the soil. And then we look at the effects that would be observed in the treatment plants as compared to the controls. That's the seedling emergence test.

The second test is vegetative vigor, and where you would have a plant that's grown up to a certain height or a certain -- based on a certain time. And then the pesticide would be applied directly to the foliage.

And so, the way that those tests are reported is in terms of the application rate to those plants. And so our exposure is assessed in terms of an application rate that would be representative, that would be comparable to those effects data. So we'd

come up with a pounds a.i. per acre that would be either -- well, that would be deposited onto a non-target site as a result of spray drift and runoff.

I hope that answers your question. Does that--

DR. NORSTROM: That answered it.

MS. GARBER: Okay.

DR. NORSTROM: Thank you.

DR. HEERINGA: Dr. Maddalena.

DR. MADDALENA: I'm curious, why hasn't the OPP looked at this TRIM FaTE model that could be adopted very easy?

MS. GARBER: I can't answer that.

DR. MADDALENA: Has -- are you aware of the model?

MR. SAPPINGTON: Yes, it's a multimedia-type model that evaluates exposure coming down from air. And -- but that's one of the models, I suppose, that we would look into and consider. I think we just provided just the representations of several of them here.

DR. MADDALENA: Actually, it -- full disclosure. I helped on that model, as -- in part, for a number of years. And it has -- it's a fugacity-based fate and transport model, not just air; it's

multimedia.

It's -- the conceptual model, I mean, you almost can't tell the difference between your conceptual model and the one they started with. And so, obviously, it's a very complex model, but it incorporates a lot of these pathways. So it should be, I don't know, just considered before you reinvent one; they put a lot of effort into that one.

DR. HEERINGA: Randy, they should definitely get that into comments in our report too obviously.

DR. MADDALENA: Mm-hmm.

DR. HEERINGA: Other questions. Dr. Delorme?

DR. DELORME: Just a clarification: You indicated that you had some tools available for looking at the dotted blue lines and limited tools for the red. I'm assuming that's for quantitative risk assessment, not for qualitative risk assessment? So if we're actually generating EECs, that you can then compare with the test data?

MS. GARBER: Yes.

DR. HEERINGA: Well, that took a long time.

Dr. Hickie.

DR. HICKIE: Couple of quick questions: In the models you have used, are the plants treated as a single compartment, or is there any consideration that these different parts of plants, seeds, fruits, leaves, things of that sort?

MS. GARBER: As far as the T-REX model is concerned, that model does compartmentalize plants out into -- it generates different EECs for different parts of the plant. One of them is seeds; one of them is leaves; and I think another part is fruit. And then, it also differentiates fru -- the leaves between grasses and broadleaves.

DR. HEERINGA: Dr. Gan.

DR. GAN: On your slide 21, the accumulation that's happening is that, do you mean plants or animals?

MS. GARBER: That would mean in the entire food web. So the chemical is not biotransformed in any part of that food chain. So that would be in the lichen, the caribou, the wolves, the shrews and the earthworms. In those two models.

DR. GAN: Okay. You know, from what I have seen, like in plants, clearly 90 percent or more of the residue is just, like, in the concentrated form; and I think bioavailability would be a very important

factor here.

MS. GARBER: Mm-hmm.

DR. GAN: Is that considered somehow?

MS. GARBER: In those two models, let's see, they -- they both have factors that would account for efficiency of uptake. The -- both of the models break out the tissues of the organisms into three phases, basically, into lipid, non-lipid or organic matter, which would encompass carbohydrates and proteins, essentially, and then water.

And so, there are different efficiencies of digestibility of those different tissues, which would be related to the uptake of the chemicals. So that would indirectly account for that.

DR. GAN: Thank you.

DR. HEERINGA: Dr. Abbot.

DR. ABBOTT: Hi. Perhaps I missed this, but the T-REX Model doesn't take into account any kind of dietary exposure. Is this true?

MS. GARBER: T-REX is a dietary-based model, so it's --

DR. ABBOTT: Oh. Go ahead.

MS. GARBER: Oh. So, T-REX assesses exposures through dietary dose base exposures, actually. And so, it accounts for -- the EECs

represent pesticide residues that are on either insects or on the surfaces of the plants.

But it doesn't account for bioaccumulation that would be expected in those organisms, or those food items.

DR. ABBOTT: I knew I had missed that.

MS. GARBER: Okay.

DR. ABBOTT: Thank you.

DR. HEERINGA: Dr. Maddalena and the rest of the panel.

DR. MADDALENA: Is the ultimate goal to consider the whole universe of chemicals, or are you really trying to focus on boxes?

MS. GARBER: This would be relevant to the chemicals with PBT characteristics. And, you know, one of the issues is that -- actually, as I pointed out at this slide, is that we need to be able to define which -- which chemicals would be expected to bioaccumulate in terrestrial food webs, and that, you know, using the KOW as we do for aquatic systems may not capture the chemicals that would be expected to bioaccumulate in terrestrial food webs.

DR. MADDALENA: Okay, because two of -- some of the models or most of the models you showed had KOA as the driver, and that indicates that it's a --

it's an air pathway to get into the system.

MS. GARBER: Mm-hmm.

DR. MADDALENA: And PRZM doesn't provide that air pathway; all you have is spray drift. And for persistent chemicals, the idea is you're going to have this cycle going over and over, and you don't seem to capture that in the current draft.

MS. GARBER: In the Kelly and Gobas model and Armistead and Gobas model, the KOA actually factors in, in terms of the elimination-rate constant. And so, basically, as your KOA increases, you have less elimination through that route. And so that could be one of the parts that would be affecting your accumulation in those -- or, your biomagnification in that food web.

And so -- although, that's not generated using PRZM, but this would be another -- a separate issue from that.

I think it -- it might be relevant to point out here that, you know, we're -- we're not as far along in this -- in terrestrial bioaccumulation as we are -- in terms of our method, of characterizing and of quantifying the -- we're not as far along in terrestrial bioaccumulation as we are in -- in aquatic bioaccumulation. This is more -- we're a lot more

conceptual here, and just trying to -- trying to get some feedback at the beginning of the process.

DR. HEERINGA: That's why we're here, I think --

MS. GARBER: Yes.

DR. HEERINGA: -- Kristina, so you get that.

MS. GARBER: And that's appreciated.

DR. HEERINGA: Dr. Doucette.

DR. DOUCETTE: This is a follow-up to Randy's question, although, instead of looking at the PBT chemicals that are going to be primarily taken up into plants from an aerial route, the other end is also interesting. I've looked at the -- your two models that you've highlighted here for the plant bioaccumulation from root uptake, and I think there's several others that are -- that should be considered; they're a little bit more up-to-date.

Don Kiseman has published a model, Seppen Trapp is I think on his fourth generation --

MS. GARBER: Yep.

DR. DOUCETTE: -- was that one. And just make sure that the models that you do choose look like, I guess, the state-of-the-art.

MS. GARBER: Thank you.

DR. HEERINGA: Thank you. And again, we'll make sure we get this worked into the minutes of our report, too, so references are there.

Other questions or clarifications? I'm not seeing any at this time. I'd like to thank you very much for your presentation, and I think if any other questions occur to the panel, we'll hopefully be able to call you back to help us out. Thanks again.

At this point in time, we'll move on to the second of this morning's presentations, which is going to be a presentation by Faruque Khan, Dr. Faruque Khan, who is the -- going to introduce the topic of the assessment of long-range transport.

Dr. Khan.

DR. KHAN: Thank you, Dr. Heeringa, and the panel members.

I'd just like to introduce the topic of the long-range transport. It has been issue throughout the -- yesterday, so let me start with: That the persistent bioaccumulative and the toxic, which is the PBT, chemicals are of particular concern up in North America, because they're found in one or more sensitive areas, such as arctic, Great Lakes, and national parks of the western states.

And transport of PBT chemicals to the

polar of the U.S region are well-documented. In this presentation, I would like to introduce to you the issues that are likely important consideration in the characterizing of long-range transport.

My presentation outlines the reproductions, and then the factors affecting long-range transport, and some of the global efforts such as treaties designed to impact and how do we address this long-range transport and the limits and challenges we are facing, and the references I use for this presentation.

This is a conceptual diagram describing the movement of applied pesticides. As you can see in the diagram, that the applied pesticides can be potentially partitioned into various media, such as air, water or soil. The intermedia mass exchange and the transformation process that fuels the long-range transport processes.

Once airborne, there's the volatilization or spray drift or wind duration from the application site, but, but move into upper atmospheres for a widespread regional and global distribution to the redistribution. And persistence in air will play an important role in the redistribution.

Although the atmosphere pathways lead to

the dominant pathway for the long-range transport of volatile, and the semi-volatile chemicals. Non-volatile pesticides transferred by ocean current is a governing transport mode for long-range transport.

In addition, certain migrating species and drifting ions can also play a role in the long-range transport of chemicals. However, that contribution from migratory species and drifting ions are relatively small, as compared to the media such as air and water.

And as the -- several speakers yesterday and today, we talked about our risk-assessment process is actually a very narrow field, but we are -- the scale is very limited scale. We don't go from the regional or remote for our risk-assessment purposes. We have a limited tool, as you can see, to this presentation.

Let's recap the previous slide. Define the long-range transport again, when a chemical enters into the transport medium, such as air, water, it can potentially travel long distances, from the point of release to the limit region. And then the following factors influence long-range transport of chemicals, the properties related to the parts to and transport, properties related to persistence, and the

environmental factor. And in a few slides, I will address each of the factors affecting long-range transport.

Okay. Getting to know your chemical and how it likely behave at various given organisms. This interest intrinsic peak in properties such as water solubility, vapor pressure, partition coefficient, as well as absorption and desorption of the soil and sediment will dictate the mobility of a chemical.

For example, a chemical with a very high vapor pressure and Henry's Law Constant will have a tendency to emit from the first to an aquatic environment to the atmosphere. Also, a chemical with a very high Koc and low velocity can transport as a particulate in the air or suspended material by the ocean current.

In general, there is very strong surviving product chemistry data to the Agency except octanal air coefficient, which can be estimated from octanal water and Henry's Law Constant.

Any number of things can happen when the pesticides get applied to the field. A biodegradation process such as hydrolysis in soils and water, as well as biodegradation in the soil, water and sediment can provide persistent behavior of a chemical in various

media.

This then provides us internal fate data of the substance along the entire length it perform. However, agents that rarely get studied, we have the colloidals in air. Which, the critical information for volatile and semi-volatile chemicals to evaluate long-range transport.

Any number of things can happen to pesticides into these environmental factors. And one factor, such as climate, geology, hydrology, vegetation, many other factors, can influence the fate of the transport behavior.

For example, high temperature in the tropics will increase the volatilization and transport by -- via air currents, than deposits to the cool -- cooler regions such as the polar region. As the cooler temperatures slow down the degradation rate in the polar regions, that transport chemical become more persistent.

Another example is the general ecology, you can shape our landscape and consequently the distribution of the polar. The map, lower and right-hand corner is the global distribution of the polar region, shows the distribution of various toxic particles. And each particle has unique

characteristics, such as texture, organic matter content, moisture, that can influence the fate properties of the chemicals, and will contribute to the transport properties.

Understanding how a chemical migrates through the global environment is very important to address in the long-range transport. And when Nehr and Mackay presented a conceptual model, how chemicals move around the globe.

I'm sure those who are familiar or have worked with long-range transport get this concept of our guidance documents or guidance picture of all of this -- looking into it and try to you know, evaluate how things are actually conceptualized.

On a global, various hypotheses have been conceptualized, to both the net transport of chemicals from lower latitudes to the higher latitudes. Many transfer processes are depicted in this figure. In this figure you can see that in the lower latitudes is a more of an upper breaking than the deposition. As you move to the mid-latitude is encountered very similar deposition to what I am saying. But when we go to the higher latitude, the more deposition compared to the air transport, evaporation of the chemical.

High volatile compounds tend to remain

airborne and move, migrate faster. Semi-volatile compounds have a tendency to pursue into soil, water, ice, or using this type of chemical to fractionate and migrate at different velocity and that's on the right hand side of the picture as you see, that, that better mobility of the chemical, which is pictured here.

Also the semi-volatile chemicals also migrate at a higher latitude when it's feasible to do a short jump, known as the grasshopper effect, which is here. And this is the, how a simple temperature variation, you know, greatly affect under the grasshopper of the cannonball.

And deposition rates will vary within latitude and potentially deposition in the temperate regions, deposition will differ in the temperate regions and polar regions because of the condensation.

Since this is a global problem, international efforts were taken to address long range transfer. Since long range transfer is a global problem, the formal treaties like LRTAP or long range transboundary air pollution were signed by many countries to reduce or eliminate the use of persistent pesticides.

Although LRTAP convention was initially to respond to the acid rain, a total of 8 protocols now

negotiated. And how do you find specific measures to be taken by the parties for the range of air pollutants?

This regimen include the European countries, the United States, and Canada. And in 1998, they negotiated a protocol for persistent air pollutants with a permission to consider other substances for the future. Since then nations throughout the world began negotiations under the United Nations and MR program on a global regimen to prohibit or arrest or reduce or eliminate the use of persistent chemicals. And in 2001 the Stockholm protocol negotiated a similar protocol to the 1998 LRTAP protocol.

The Stockholm protocol is much broader if you look at the list of limitations than the LRTAP protocol. It particularly is the protocol to address PBT and long range transport potential of chemical, and there were lots of nomination criteria for the PBT and long range transport potential. I guess this is the, because you've heard about the PBT, I'm more interested in the long range transport and this is the criteria that the Stockholm had been proposed and I just summarized a little part of it.

Now the measured level of the active substance and the location distance from the source

offers release and are of potential concern. And one figure is showing that the longest transport of an active substance is the potential for the transport of the receiving environment, may offer via air, water or migratory species.

Or in long fate property and the model result illustrating that active substance has potential for long range transport and environmental transport through the air, water, and migratory species. For the active chemical that migrates significantly through air, its half-life in air should be greater than two days.

This criteria is useful in identifying the environmental hazard for the chemical, but it has limited utility for the risk assessment process. As Dr. Steve Bradbury mentioned yesterday and the FIFRA also report addressed risks and benefits given the risk assessment process.

Now I move to the methods how we can actually address this long range transport. And the next few slides I would like to introduce that important method to characterize long range transport of this chemical. At top we see, understanding that transport and persistence property and monitoring and the modeling.

As I mentioned earlier, that how physical, chemical and environmental fate properties provide a lineup and formation that can be used to characterize whether a compound is persistent and had a long range transport potential.

Moving from that, let's move to the next topic, which is the monitoring. Monitoring data in the model efficient distance from the use side can be unambiguously satisfy the long range criterion. And the detection of example pesticides 1 and 2 in the regions such as Arctic are well documented. Many nations and international programs are reflecting monitoring information on a toxic substance in the various media.

For example, USEPA and Environmental Canada operate an integrated atmospheric deposition network that measures level of toxic substance in the air of the Great Lakes region. Council of Air and the substance have been selected since 1998 and the substance monitored of the network including the PCB, organo-chlorine pesticide and many other substances, like a metal.

In addition, the Great Lakes National Program Office also funded selected monitoring program that samples water, aquatic life, sediment, in order to

assess the health of the Great Lakes ecosystem.

And there are so many others, like there is a program for monitoring the Arctic. Also there is a recent publication about the Park Service working on monitoring in our, in our western states. So this actually should be able to provide us some sort of a indication of the pesticide distributions and the long range transport.

And I'll also explain that the monitoring data has limitations. The monitoring data can show presence and in what location but not the route of transport.

It's a non-targeted area linked to the specific pesticide application site. Also monitoring takes time to validate the data. So that's reactive, not proactive.

The monitoring data is only available for probably residual pesticides. Monitoring data is not likely to exist for the newer chemistry which is, we are facing, you know, when you're trying to characterize some of these chemicals in the modeling.

The model, the behavior of chemicals in the environment is very complex, and we have seen it from time to time that nothing in the models can characterize

this partitioning behavior in the various media. Multimedia models are based on the application of the fugacity input versus environmental problems.

This model serves appropriate in treating transfer and transformation of the chemical that's coming out from the non-point sources over literally a long time, and the model can also sustain new chemicals. Multimedia models are valuable tools for providing screening assessments for a long term, suspended long range transport. And in recent years researchers have developed several multimedia models that compute numerous indicators for overall persistence and the long range transport potential.

And as I mentioned in my previous slide, these two indicators overall persistence and characteristic distance are important components of the multimedia models dealing with the long range transport.

These are the two terms I will use, I'll be using during rest of my presentation and tend to define as the overall persistence is derived from the degradation rate constant in soil, water and air, weighted by the chemical mass fraction present in two media. The overall persistence is different from the single media half life or the soil half life, and the

characteristic travel distance is the distance at which the chemical concentration at the point of release has decreased to 37 percent, assuring that the chemical is transported by the constant flow of air or water.

There are many mod -- multimedia models that are available in a plotted model assessment. The most widely used multimedia model are mass conservative mechanic type of compartmental model. And the full levels of the complexity presents it in the bioassay and summarized in the table. And the key is on the second column, and based on that assumption, what you'd like to get from that model. And I'm not going to go very far with this one because you already have this from Mackay, he can answer all your questions for everything you wanted to know about the model, the new model.

As one progress from the level one to the level four estimation, the reliability of the estimation to the actual environment increases, but requires additional data and effort.

This slide has a few examples of the multimedia model, and most of these models are level three except the CEMC level 2, which is a level 2 and global pop which is a level four and dynamic model.

In 2001 the Organization of Economic

Cooperation and Development and the United Nations Environmental Program organized a workshop to define the role of multimedia models in chemical exchange and assessment for the persistence and long range transport. This literature is publicly available about models and transport calculating over persistence and long range transport.

Several comparative, comparative studies by Theron 2005 and Bismarck in 2006 evaluated these models and concluded that the most models predict similar rankings of overall persistence and long range transport potential values for a set of chemicals encompassing a wide range of physicochemical and in all fate properties.

Based on this model evaluation that OECD experts then developed a screening tool to estimate, you know, overall persistence and long range transport. EFED used this screening, OECD screening tool to evaluate the overall persistence and long range transport of this whole example chemical or pesticide.

In the next few slides I will describe the OECD model features and the results obtained by the tool. With the tool the consensus multimedia model developed by the OECD work group, it calculates the multimedia indicators for overall persistence and the

potential for long range transport from the chemical properties. It uses a benchmark approach to provide the context of the model results, which can allow a comparison PBT parameters against acknowledged PBT or pops.

Also has the capability to perform Monte Carlo uncertainty analysis and analyze this. This model based on actual worksheet that includes embedded visual basic and application approach. The model is also publicly available and easy to use.

This is a model features the level three and steady state fugacity model, and the unit is the whole mode. Has a compartment like the air, the height is about 600 meters, the land is 21 percent, and water is 79 percent. And it should have a constant velocity in air is a full meter per second in air and two centimeters per second in water. And this is a screen shot of the first of the fugacity 2 model.

The environmental attributes, percent of air to particles, water content to suspended particles, includes the actual long range transport to the stratosphere in a deep ocean, includes small burial, leaching to the deep soil and soil changes in deep ocean water.

Chemical properties such as molecular

weight, log KOW and log KOA and the degradation half-life in air, water, and soil are needed to enter in the chemical column, which is on the right hand side of the screen.

As I mentioned earlier, the registrants provide most of the required values for the two, except the half-life and the air, which can be obtained from epi-3, another model. OECD too had built in the warning system, there's two types of warnings that are available, if values entered are suspect or invalid, a color code next to the input parameters and input status indicate whether this entry is possible. It's just like your street light, if it's a red, it's a no go.

Here is the results from the tool and the stimulus has a persistent, overall persistent and characteristic travel distance as well as the transfer efficiency for this example. As well as some reference chemicals. In column two, the left, over here, has over-persistence ranging from 81 days to over 599 days, for example. And this third column has a characteristic KOW range from 153 miles to about 221,000 miles. And also the transfer efficiency as seen in the last column, as a percentage of transfer efficiency.

And transfer efficiency is a ratio between the deposition flag in the network region and the emission flag from the source area. Key examples that transfer efficiency for the pesticides exceeded 100%. And it is possible to obtain more than 100% transfer efficiency. In this case, there is multiple cycles of air and surface media and that is through several cycles of deposition and re-fertilization and again deposition in that case.

So let's summarize the OECD tool results. There's chemicals under consideration except pesticide one and isomer one, have a comparable higher overall persistence than the odd ring, but lower than the PBT and end ring.

Pesticides one and three have comparable or higher number for estimate than the end ring. Two and four are comparable, are higher for long distance estimates for all three reference chemicals. All pesticides under consideration for PBT have a higher transfer efficiency estimate than those of the odd ring or end ring.

The models also have a capability to differentiate the contributions from the vapors or aerosols for the long range transport. But some are going to hunt pesticides, 99% vapor from the pesticides

one and two. And for the high Q.O.C. pesticides, which is slide four, 99% is aerosol for pesticide four.

So the estimated maximum travel distance has been estimated from air transport for the vapor phase of pesticides one and two. And for air transport of aerosols for pesticide four and the transport of pesticide three. The OECD model results of the overload persistence and the characteristic travel distance suggests that the example chemicals have a potential for long range transport.

Also, we have limitations and challenges. Again, to characterize the long range transport- monitoring data can provide definitive evidence of long range transport of pesticides, but this data has limitations for providing quantitative estimates of chemical loading from the various environmental media from specific use sites.

Multi-media models have limitations in estimating quantitative loading in the various compartments of the environment. And lastly, a number of multi-media models have emerged to provide screening attachments for environmental persistence and long range transport.

Application of screening models is critical in determining the long range transport of

pesticides, specifically for the new chemicals. This is the references I used for this presentation. Also, before I conclude my presentation, I would like to take this opportunity to thank Professor Don Mackay for his contribution in the development and evolution of these multi-media models. We are honored to have him here as our guest and thanks for your attention. If you have any questions, comments....

DR. HEERINGA: Thank you very much, Dr. Khan. Dr. Simonich has the first question.

DR. SIMONICH: Thank you for a very excellent presentation. I appreciate it. So one thing we're struggling a bit with is the definition of long range transport. How would you define long range transport?

DR. KHAN: In my sense, in my work, anything from the application side is a long range transport. But you know, sometimes you have to go with the flow, you know. And we do, especially when we did the field, we looked beyond our field application of the field side. Beyond that. Even in some other cases, we did our assessment beyond our, like on the regional level, not the regional level, but a few miles from the application site.

DR. SIMONICH: So, in your mind,

transport to the great lakes, transport to the arctic, maybe transport to remote mountain ranges all fall under that?

DR. KHAN: Right, they fall under that.

DR. SIMONICH: Okay, another question. In kind of the more routine assessment of pesticides for registration, how do you currently assess the potential for long range transport?

DR. KHAN: We don't. Case by case, we do. For example, Lindane, we have looked at because we know it is problematic. Our main source was this monitoring data.

DR. SIMONICH: But for new-to-the-world pesticides?

DR. KHAN: Yes, new-to-the-world, that's where, we are struggling with that one. Because in some of the characteristics of the chemicals suggest to you, in a way, like it has the potential. Just to characterize the hazard side of that, at this point.

DR. HEERINGA: Dr. Bidleman?

DR. BIDLEMAN: I also want to say thank you. I really enjoyed that overview of long range transport and I was going to ask similar questions to what Stacy asked and that's what EPA is using now and I guess....you're not using anything right now, you're

sort of keeping an open mind.

DR. KHAN: Right. Absolutely. That's why you are here.

DR. BIDLEMAN: The OECD screening tool, well, we'll talk more about this in our panel tomorrow. But I think the prediction that pesticide #4 has a characteristic travel distance of about 2500 kilometers really needs to be viewed with caution. Because one of the features of most of these models, including the screening tool, is that you have a constant precipitation. It must be a very, very dreary world, because it's always drizzling. And of course, this removes particles quite efficiently.

If you run the models with intermittent precipitation, you get very different results than if you run them with continuous precipitation and that increases the transport capability if you have intermittent precipitation. But then you also have to take into account the forest filter effect, the fact that when the air passes over vegetative forests, you remove a lot of things, including particles, quite efficiently. And this decreases the characteristic transport distance.

So I would say when you get into compounds of partition, appreciably to particles, which

could be a lot of currently used pesticides, then you may want to look a bit beyond the OECD screening tool.

DR. KHAN: No, I agree with you. Even when we do a prism exam or our tool we use, we go into deep actuaries where we see this uncertainty and we describe in our risk characterization that they may be showing us these results but it may have some effect for this region.

DR. BIDLEMAN: But for an initial tool, it's pretty good. It's a whole lot better than just two days.

DR. HEERINGA: Dr. Thibodeaux?

DR. THIBODEAUX: Yes, I agree, very nice presentation. You answered Dr. Simonich's question by saying that your definition of long range transport is everything from the field to the north pole.

DR. KHAN: You can't stretch it however you want to.

DR. THIBODEAUX: But my question is I think maybe your priorities are sort of reversed. It seems to me your slide #2, if you could bring it up, for your purposes, particularly with pesticide and their impact, it seems to me the local, that endpoint, particularly with the previous speaker having to do with uptake of terrestrial, is of much more concern and

should be higher on your radar than what happens to the polar bears.

DR. KHAN: Okay. This one you're talking about?

DR. THIBODEAUX: Yes. And looking back, both air and water, you've got local, regional and remote. And that's a lot on your plate. So, I guess my question is, shouldn't you be focusing more on the local?

DR. KHAN: Well, local would do, like the near field do local. But there are some chemicals we just talked about like in the eliminated data. It moves around. And that's where we, we're concerned about that.

DR. THIBODEAUX: Do you have any, you might say, legislative mandate that you're following on long range transport? Is there a law?

DR. HEERINGA: Dr. Bradbury, think that's in your court.

DR. BRADBURY: Before I answer that question, let me go back to the earlier question about near field and near the site of application. One of the tools we've only alluded to but haven't gone into great detail includes modeling approaches, includes a spray-drift model that allows us to make estimates of

what the flux can do, what the concentrations of the pesticide could be at the ground application or the air application to get a sense of pesticide movement through spray-drift.

Then Faruque also discussed some models that we've used with a fumigant registration decision we completed a few weeks ago in which we used different models to get a sense of flux, of fumigants off field. Not to say there isn't room for improvement on those near fields, we've got an SAP coming up later next calendar year, we were looking at volatilization in both human health and ecological risk, near field, if you will.

And I realize we were switching definitions, but, so while we can always improve on that, what we're sort of struggling with right now is beyond sort of what a spray-drift model could do a few thousand feet off the overhead fumigant, the flux model is giving us, in terms of a mile or so off.

We're serious about perhaps use of pesticide A and the potential it could end up at 13,000 feet in Boulder or further, sort of gap that we're trying to get some insights on how to approach that. I'm going to turn to some of the treaties that Faruque mentioned and I think I'm about 90% right.

The country more or less has ratified, or since ratified the treaty at the Stockholm convention. But TOSK and FIFRA haven't been amended to fully implement the protocols from Stockholm.

So the U.S. goes to the meetings and dialogues at the meetings where we sort of sit at the table, in terms of the parties making the actual decisions. But having said that, our perspective here is that we should take a look at the properties of the chemicals, look at exposure properties, look at effects properties and try to do the best ecological or human health risk assessment we can with the information we have and take that into account with the risk management decision.

And what we're trying to work through here is, in this specific talk, is if there are attributes of a compound that suggest it may move beyond what our spray-drift models would suggest or our fumigant flux models would suggest, how do we deal with that. What's the best available science that can then inform us in terms of FIFRA as to what the proper risk management decision would be. So that's a challenge for risk analysis.

DR. HEERINGA: Dr. Simonich? And then Dr. Delorme.

DR. SIMONICH: I just wanted to mention that the U.S. EPA, NOAA, and NASA has asked the National Academy of Sciences National Research Council to assess the transport of persistent organic pollutants into and out of the U.S. and that should be done by next year.

DR. DELORME: One of the things that I noticed you didn't factor is the scope of use or the scale of use of the chemicals. I think that's also an important variable you have to consider. If you have a chemical that's in use in the millions of tons, it doesn't necessarily have to be persistent in order to find in areas where it's not applied. There are examples I think you see in Canadian data air monitoring where, during the spring we see stuff that is not applied in Canada up there, but only for the fact that tons and tons of the stuff are being applied. It doesn't matter that it's not particularly volatile.

DR. KHAN: That's what Dr. Bradbury was alluding to because that's what will qualify a decision as a component of that and that's what we'll be looking into in the next SAP.

DR. HEERINGA: Dr. Maddalena?

DR. MADDALENA: I'm still a little unclear, when you use the OECD tools, all four

chemicals have been flagged as of concern. What else do you need to know? Where do you go from there?

DR. KHAN: Well that's what I said. You can go more deeper than that. You just take those data and then again, there's a lot of other components in the model. Like for example, I was showing that the chemical flow, as a transport of air particulates, and Dr. Bradbury was explaining that there are lots of things, because we're taking it at a constant flow rate, you know.

But that doesn't happen all the time. So those, and also like precipitation, like every day we don't have precipitation. So those are the things we need to think about it, before we start leveling something, like this is a persistent, this has a long range transport.

DR. MADDALENA: A number of these multi-media models do have intermittent rain now and the long term particles deposition, you're right it's very simplified. But I think what I'm getting at more was the last comment. Do you need to know what the mass use is? Can you put a cap on that? Is that even a policy decision that you could do? The chemical is clearly going to end up in the north pole if you start using it, even if it's a hypothetical scenario. You

might want to think about how much you can release.

DR. KHAN: That's the management decision Dr. Bradbury can answer.

DR. BRADBURY: And without dodging the question, I think your discussions tomorrow or the next day when we get to the charge questions will be very helpful. We're starting to talk about it now. So with the screening level models, I view the personally as part of the problem formulation. It's certainly probably useful to say, here's a pathway you may need to dig in deeper and get insights as to what that exposure potential is so we can do a better job of quantifying risk or estimating what the risk is.

Based on that outcome, potentially or theoretically providing scenarios to put out. But I think what will be helpful tomorrow is as you all talk about this with respect to describe what levels of certainty or uncertainty quality can you expect from current tools. What are some reasonable forecasts for the future, in terms of tool development, in terms of what kind of certainty can they be associated with.

With what's on the horizon, no pun intended. So you can start to get at some of the questions you're asking. So there's flexibility if you will in terms of risk management decisions, but it's

highly dependent on what's the state of the science and the quality of the science that helps inform that risk management decision.

And again, as I said before, some of these chemicals are in the decision making process now, so we are balancing that with what's today's best available science, what are the strengths and limitations of today's best available science, through an articulated decision. And that gets into how you define the uncertainties and certainties.

DR. HEERINGA: Dr. Bidleman?

DR. BIDLEMAN: If you look into these transport models, the results are hugely dependent on whether the chemical is released into the air or released into the soil or released into the water. That greatly affects the outcome.

In the case of pesticides, we know where they're applied. They're applied to soil for the most part. But even there, it depends greatly on whether it's applied on the surface, whether it's a foliar spray, whether it's granular, whether it's soil incorporated and this gets into the emissions scenario, which isn't really treated in these long range transport models. And it's not treated in the OECD screening tool.

But you have to put the whole package together, and to evaluate atmospheric transport, you need to start back at the source and also consider how these chemicals are getting out of the soil and into the atmosphere and at what rate.

DR. HEERINGA: Well at this point, it looks as though we have no additional questions. At this moment, Dr. Khan I'd like to thank you and also Kristina earlier for excellent presentations. My own opinion is the material has been extremely well organized, at least to set up the discussions and our review of the charge questions. I thank everyone for that. Panel members and public, I think we're at 10:03 on my watch. The company clock has 9:57 but we'll take a 20 minute breeak and we'll be back at 10:20 then.

DR. HEERINGA: Welcome back everyone. We're on our second day in the second half of our morning session, FIFRA science advisory panel on selected issues associated with risk assessment process for pesticides with persistent bio-accumulative and toxic characteristics. And at this point, we hear the last sequence of presentations, two more this morning. I'd like to welcome Brian Anderson on the topic of evaluation of aquatic toxicity of persistent bio-accumulative pesticides. Brian?

MR. ANDERSON: Thank you very much. I'll be talking about evaluating aquatic toxicity in persistent and bio-accumulative chemicals. I'd like to start by acknowledging a few people who aren't presenting but have made significant contributions to the white paper and to this presentation.

Dr. Tom Steger and Michael Hoffman have both made significant contributions and I just wanted to acknowledge them. In addition to the rest of the team as well. We're going to talk about three basic things during this presentation. I'm going to give some background information on toxicity data that we typically get to evaluate aquatic toxicity.

We're going to look at some types of studies that are particularly important for persistent bio-accumulative chemicals. And we're also going to look at some methods we use to evaluate aquatic toxicity where dietary exposure is the predominant exposure route. And we're going to look at total residues of concern.

Both how we choose toxicity values to describe the total residues of concern and we're also going to look at some information that was presented yesterday. When three methods were presented to estimate total exposure residues. And we're going to

see how each of those methods relates to a potential changes in risk conclusions. We'll look example chemicals one and two for that.

So data that we typically get for an aquatic toxicity assessment includes acute chronic studies, which are typically water only exposure studies. Acute studies are 2-4 days in duration. The endpoint we use for risk quotients is mortality or immobilization. The toxicity value that we use in our risk quotients is an LC-50 or an EC-50, that's a 50% effect level. For product studies or longer duration, from about 3 weeks to 6 months or more, depending on the organisms being tested and the type of study, the endpoints include growth, reproduction, and mortality. The toxicity value that we use in our risk quotient for chronic risk quotients is a no observed adverse effect concentration.

So the highest concentration does not result in an adverse effect. A couple studies that are particularly relevant for bio-accumulative chemicals include sediment toxicity studies, which is the case because chemicals that tend to bio-accumulate tend to have high, long KOW's, high KOC's so they tend to partition in the sediment, so they can persist for an extended period of time. Therefore, obtaining sediment

toxicity studies, both acute and chronic duration, can be important. In addition, we have two types of studies we can get to evaluate the chronic toxicity in fish, the early life stage study and a life cycle study.

The life cycle study has the potential to account for maternal transfer, which is transfer of lipid rich compounds to developing eggs with the associated contaminants. And the early life stage study does not have that capability. So obtaining the life cycle study can be particularly useful for chemicals that bio-accumulate. In addition, there are some other studies that we get on a case by case basis.

For example, chemical one, you'll see we've updated on degradative concerns. For example, chemical four, a number of studies are available. They characterize both accumulation and toxicity for multiple exposure routes.

In addition, we can get microcosm, mesocosm studies although those are less commonly submitted, but those are less commonly submitted, but those are available on a case by case basis. There are some challenges with our typical data sets with respect to persistent and bio-accumulative chemicals.

One of these challenges is study

duration. Chemicals that have high KOW's take longer to reach steady state and steady state is not achieved during the duration of the study. That could result in an under-estimation of toxicity. For example, example chemical 4, during the duration of the acute studies, 10% of studies say it was expected to be achieved. But even if you go to the longer duration studies, even 30 or 60 days, steady state still isn't expected to be reached.

That's based on the kinetics that were observed in the bio-concentration studies. So in addition, the data that we think we have are water exposure toxicity studies and in cases where dietary exposure is particularly important, then the water exposure studies may not fully express or evaluate the toxicity of the chemical, as we'll see with example chemical 4. So looking at example chemical 4 as the example, it has a very high KOW, very high KOC, high bio-concentration factor and very low solubility.

I expect it to be persistent in both water and sediment. And what this means is when a chemical enters the water, it's not probably going to stay in the water phase, but it's going to tend to partition to the sediments and to the biota and therefore dietary exposure would be expected to be a

predominant exposure pathway.

And that means that doing a water based risk assessment, meaning comparing water-based EEC's to a water based toxicity value may not be particularly meaningful or not be expected to fully evaluate the risks of this chemical. And therefore an alternative method would be useful in evaluating this chemical. And we're going to look at the critical body residue approach to do that.

The critical body residues in this case, we're defining that to mean the level of pesticide in an organism that corresponds to a defined effect. So in other words, what we're doing is instead of expressing toxicity in terms of concentration exposure media, we're expressing toxicity with respect to the assessed organism.

Yesterday, during the bio-accumulation assessment, Keith Sappington presented methods that allow organisms that, hypertropic level organisms that consume aquatic organisms that bio-accumulate the chemical to be assessed, meaning how we assess risk to birds or mammals that consume aquatic wildlife.

This is a way to evaluate potential risks for the organisms that are actually accumulating the chemical. So the critical body residue is a

toxicity value that can be compared to results from the accumulation assessment to calculate a risk quotient using methodology that we would otherwise typically use except for the EC and toxicity value are now in terms of milligrams per kilogram organisms as opposed to, for example, milligrams per liter water or milligrams per kilogram sediment.

And our standard acute and chronic LOC's would apply. The levels of concern range from .05 to .5 for acute effects and 1 for chronic effects. A major advantage to this approach is that it does allow for multiple exposure routes. It addresses the issue of lack of steady state and bio-availability, which is a key issue for chemical 4. It's also an established approach, it's not new, it's been around for some time.

Some assumptions with this approach, one is that toxic effect is indeed related to body burden. There are some chemicals where that wouldn't be the case. Strong irritants are surface acting types of chemicals. But this is an assumption that can be tested with measured data and it's also an assumption that's reasonable for most interorganic chemicals. In addition, another assumption is the potency is equivalent across exposure routes.

What that means is the CBR would be the

same, regardless of whether or not dietary exposure is occurring or water exposure. In addition, the critical body residue for a given endpoint like mortality is not time dependent. What that means is that the critical body residue would be the same, regardless of whether or not it's resulting from high intensity exposure for short duration of time or low intensity exposure for a longer duration of time.

That would be the constant critical body residue approach. So ideally, what we would want for a critical body residue would be a measured study, meaning a measured dose response where the exposure is expressed in terms of body burden and the other typical responses that we would measure in an acute and chronic toxicity study.

However, those data typically aren't available for our risk assessments. In the absence of such data, there might be a need to estimate a critical body residue and we'll see an example of how we have done that. Using example chemical #4, which did not have any measured and critical body residues available in fish, so we tried to make best use of the data that we had to estimate the critical body residue.

Using this simple first order equation that relates concentration in an aquatic organism to

the concentration in water, uptake and elimination, kinetic parameters from the bio-concentration study and time. So when the concentration in the water is equivalent to an LC-50 or a NOAC or whatever toxicity value that we're using, the concentration in the organism becomes an estimate of the critical body residue.

Listed here are some of the parameters we used in the estimate. So plugging in the numbers to the equation, we end up with a range of critical body residues for this chemical. Around 160 to 970 milligrams per kilogram. We give a range just due to some of the certainties with respect to the toxicity study and some of the bio-availability issues and that correlates to, on a molar basis, to around .3 millimoles per kilogram to 2 millimoles per kilogram.

When comparing to some of the range of values that have been reported for neutral organic narcosis range from around 2 millimoles to 8 millimoles per kilogram. This range is kind of on the lower side of that.

So there are number of uncertainties in this estimates, particularly in the differences in bio-availability, species, life stages across the various studies. Meaning we often have a

bio-concentration study, one species, a remote sensitive species in toxicity studies might be a different species under different conditions and different life stages that are also tested.

This results in considerable uncertainty, I think. Particularly, in this case when we have use of co-solvents throughout. And so one thing we can do to try to ground through this estimate is look at what other data we might have that's available to us that has measured residue.

The only other study that's typically submitted to us that does measure body residues would be bio-concentration studies and although not designed to evaluate toxicity, when fish start dying, that's typically recorded in a spot report, toxic effects that are observed. So in this case, we have a bio-concentration study that did measured effects after about 2 milligrams per kilogram and no report of anything dropping dead. This at least gives us some idea that our 160 value isn't overly conservative.

We would have an inconsistent estimation versus our measured data if our estimate was below this 2 milligrams per kilogram value. That was for an acute estimation for chronic effects. It's a little more difficult I would say. An attempt to estimate a

chronic CBR based on a water concentration and the duration of the study resulted in a chronic critical body residue that's greater than the acute body residue.

What that would mean is our estimate of mortality is lower than an estimate of body burden where no effects are occurring. That would be inconsistent. So we would want to see a measured data to help better characterize that critical body residue for chronic effects.

So since we're bringing this into a risk picture here this really what we're concerned about. Our acute risk quotient then could be calculated based on results in bio-accumulation assessment and compare that to our critical body residue. So we just pulled in some examples from the accumulation assessment and the results and our cue is around .05 to .2. That would mean that our endangered species LOC would be exceeded.

Given the uncertainties in the estimate, we would still want to see a measured value to confirm these conclusions here. We also note that the conclusions would be highly sensitive to the assessment methodology, both on the accumulation side and the toxicity side as well and our chronic risk would be

answered. So going through the fish, the same approach would be used for invertebrates as well.

We would look to see what measured data we have that characterize the critical body residues, estimate one if it's needed, and then we can calculate ours using the PRZM scale and compare those with the LOC's. And I think there's some utility in looking at some of the data for chemical 4 because we have a number of studies that are not typically available to us, but since these data are not usually available, then we might need to make best use of the data we have to try and estimate a critical body residue for invertebrates as well. So we're going to present methods we've used to estimate a CBR and compare that to our measured values and see where that leaves us.

So our most sensitive species for this chemical was acelas, which is a small isopod. We had a measured CBR of somewhere around .1 to .5 as a mesocosm study. So basically, what we did was took our fish kinetic data and applied that to the exposure levels that were in the study and the time durations when effects started to be observed to the estimated critical body residue.

Again, with acknowledging the considerable uncertainties going from one taxonomic

group to another, it results in an estimated value that's really within the range of the measured value here. I'm just going to look at 3 species, we'll look at choronomids as well. In this case, we actually have a preliminary study that's been submitted that measured body burden and toxicity. We also have the same mesocosm studies.

And again, applying our fish kinetic information and duration and exposure values. We would estimate the range of somewhere between 20 and 300 milligrams per kilogram and that's a much bigger range than what we saw for the last organism. And then for daphnids, again what we have is a bio-concentration study which is still in review that suggests that critical body residue would be somewhere between 6 and 30 milligrams per kilogram. And again, taking our fish kinetics data, we can estimate the critical body residue around 4.5 milligrams per kilogram, which is within that range.

Close to being in that range; it's slightly outside that range. So just to give an example of how an RQ would then be calculated based on these values, again these are just for example purposes only. We took the low end measured value, compared that to just a representative EEC from the

accumulations assessment to calculate risk quotients.

And you see our RQ's range from around .7 to around 400 and then on the sensitivity of the organism and the accumulation potential as well. So to conclude then, the critical body residue approach is being considered for evaluation. Persistent bio-accumulative chemicals in cases where dietary exposure might be important and when steady state will not be achieved during the duration of toxicity studies.

Ideally, this will be based on a measured critical body residues but there are ways to make best use of the data that we have to estimate a value needed. Changing gears here, we're going to look now at soil residues of concern. We're going to look at three basic things, provide a very rich summary of soil residues of concern, describe how toxicity values might be chosen to characterize soil residues of concern and then look at the sensitivity, the risk conclusions, both of the methods that are used to evaluate exposure, for example chemicals 1 and 2 and toxicity for those chemicals as well.

So for some background information, our total residues are typically the parent pesticide plus degradates but it's not limited to that. Sometimes

there are contamination byproducts or other types of substances that might be included. But typically you're looking at the parent, plus its degradates. The potential total residues of concern are first identified in the environmental fate studies and the degradates then are evaluated to determine whether or not there are toxicological concerns.

It can be done either with submitted data. Some might have studies that have been submitted either. Rates are offered in the study, but it might be an open literature study. Or it will be based on professional judgement as typically done through structural analysis. So the toxicity value that we can use to describe the total residues depends on a number of factors that depend on the availability of fate and toxicity data. Let's go ahead and look at our examples to illustrate these.

For chemical 1 in this case, we have a parent and we have a degradate. Toxicity of the parents and the degradates are similar. LC-50's for fish are within a factor of 2 of each other. Invertebrates, about a factor of 6 within each other. Sabatodes, their toxicities are similar. The structures are also fairly similar, so we would expect to see a similar or same mode of action. EEC's were

calculated for this chemical using three methods. Total residues, residue summation, and formation defined kinetics and we'll look at how EEC generated formation methods and how the affect risk.

And we're only going to go with the acute fish analysis, just for time constraints but the same time of analysis can be done for invertebrates or for chronic effects as well. So, what we figure then, these are the EEC's, the toxicity value and the risk quotient for the various three methods that were used to estimate exposures. We EEC's that range from about 30 to 50 micrograms per liter and pairing those to our lowest LC-50, just for the parent compound, results in risk quotients around 40 to 60.

That can be interpreted to mean that the EEC is about 40 or 60 times higher than the most sensitive LC-50. So then, some questions we can ask is what is the predominant component of the total residue. What are the differences in toxicities of the components. And then how would that affect risk. And we're going to look at one way to do that. So we took the formation decline kinetics, EEC's, and we broke that down into parent and degradate. And what becomes pretty clear is that the predominant component of the EEC is the degradate and not necessarily the parent.

So we can look at the difference in toxicity and the difference in the exposure from the total residues and the parent to look at how consideration of these factors might affect the risk quotient. So you look individually at the parent and the degradate and you get RQ's of around 1120. And we can simply add those up, assuming adaptivity, to get an RQ of around 31. And this is essentially the same thing as a toxic equivalency factor where an EEC is going to be weighted based on the various potencies of the two chemicals.

And that's a lot easier if you just add the two numbers together. I just wanted to conclude with a slide. So basically, what this means is our RQ, even considering the difference in toxicity and the EEC is still pretty close to the RQ's that were calculated originally using the three different methods. It's a little bit lower, but still the risk conclusions are the same. The risk conclusions are not sensitive either to the choice of toxicity value or to the choice of the exposure method.

So now looking at chemical #2, the parent lists four residues of concern. We see similar toxicity in the fish values, the LC-50's were within a factor of around 2.5. Invertebrates, we see some

greater variability. EC-50's range from about 12 to 300. The EEC's calculated for parent only and the total residues of concern using the total residue approach only. Sufficient data weren't available to calculate EEC's using the other two methods, the FD or the RS.

Again we're only going to look at the acute fish assessment, but methodology for looking at other species or chronic effects would be equivalent. So when we start looking at the parent only for this chemical, EEC of 18 micrograms per liter for that to be an LC-50 of 100 micrograms per liter, you get an RQ of .18.

So if you consider the degradate, then the EEC only goes up very modestly from 18 to 19 micrograms per liter. Given the similar LC-50's of fish, that results in an RQ range, depending on whatever toxicity value we choose, somewhere between .14 and .18. So again, the conclusion would be that the risk conclusions would not be sensitive to the choice of an LC-50 or the method used to estimate exposure.

Now given the similar or the low contributions of the degradate to the parent and the similar toxicity of the degradates and the parent, it's almost silly to further characterize it. Meaning what

is the contribution of the degradate to the EEC, what is the difference in the toxicity and how does that affect the RQ. But the idea here is that even if we don't have enough information to estimate our EEC's using the FD or RS approach, we can still further characterize our RQ's if necessary to account for differences in contributions of the parent versus degradate.

Particularly if there are differences in toxicity observed. And we could do that using the same basic approach. In this case, we just make an inference that the degradate, EEC, is the total residue minus the parent only. So that would result in a degradate EEC around one microgram per liter. So you can use the toxicity value lowest across the degradates, which is 56 micrograms per liter, to calculate the individual RQ and you can add those up the same way that we did for the last chemical.

And I'll acknowledge there's a mistake here too on the equation. The numerator and the denominator got mixed up, that should be 100 divided by 56. I apologize for the mistake. So the conclusion here is that risk conclusions aren't sensitive to choice of TRV or of the EEC. The toxic value or the exposure value. Methodology- RQ's range from about .14

to .34. The peak EEC is predominantly parent in this case, but even if you look at the chronic EEC's as well, 21 day chronic EEC is what we use to evaluate invertebrates, about 90% parent. But the contribution of degradate versus the parent increases as duration increases.

That may be something that's important to consider in risk characterization. So, to conclude, for chemical #1, the predominant component of the TROC was the degradate. The acute risk conclusions weren't sensitive to the choice of toxicity value or the exposure value after. For chemical #2, the predominant component of the total residue of concern was the parent chemical in this case. Including toxicity, the degradate had little acute effects but it could have greater toxicity on the chronic effects. And use of the parent LC-50 to represent the TROC was reasonable. And that is all that I have for this presentation.

DR. HEERINGA: Thank you very much, Brian. At this point, Dr. Schlenk has a question.

DR. SCHLENK: Yes, actually I have several. The white paper is a little bit different from your presentation. Actually, I was a little confused a bit on some of the things you went over. In the white paper, there's a discussion of an ECOSAR and

that is supposed to generate estimates of toxicity according to the text. I'm curious how that does that.

MR. ANDERSON: ECOSAR, that's a program that estimates toxicity based on KOW and chemical class. It has a number of chemical classes in it. We typically use that as more of a screen to tell us whether or not we might have a concern or not. We typically don't use it to satisfy data requirements or anything like that. Basically what it is, it's just a QSAR that relates toxicity to KOW.

DR. SCHLENK: And how is toxicity defined?

MR. ANDERSON: Toxicity is defined as LC-50, EC-50 or...

DR. SCHLENK: So acute lethality...

MR. ANDERSON: Acute lethality. But it also has QSAR's in there for chronic effects as well, that's right.

DR. SCHLENK: I'm sorry, what did you say?

MR. ANDERSON: There are chronic QSAR's available as well now. There are acute and chronic QSAR's.

DR. SCHLENK: So that's included in that sort of initial analysis?

MR. ANDERSON: That can be one tool that we use to identify a concern, that's right.

DR. SCHLENK: And what's ECIWIN?

MR. ANDERSON: ECIWIN? Ecostar is called a sub-component of ECIWIN. ECIWIN, there are basically a suite of different programs that are used to estimate various things with respect to environmental fate, bio-concentration and toxicity. They're all QSAR's that are based on different types of things. But they're just a way to estimate fate and toxicity parameters based in chemical structure.

DR. SCHLENK: And those assessments, in terms of mode of action, are all primarily, I'm guessing narcosis based? Is that the idea?

MR. ANDERSON: There are a number of different chemical classes. I'm not exactly sure how many.

DR. SCHLENK: Well, for PBT's. Is the assumption acute narcosis?

MR. ANDERSON: I would say that would depend on what data you have.

DR. SCHLENK: For these pesticides that you used in this white paper, would that, it seems like you're using acute narcosis as your toxicity estimate. Is that accurate?

MR. ANDERSON: I would say, when you're looking at the critical body residue, for example, what we saw was critical body residue that was based on the estimate that was lower than what had been recorded for narcosis. And that could suggest excess toxicity.

DR. SCHLENK: The trouble I'm having here is in terms....there's several statements in there that, you know, the assumption of adaptivity is reasonable.

And I'm, looking at the data provided, I'm having a hard time seeing how that conclusion was made and there's another thing basically you've got the LC-50's values in table 7.3 were relatively similar and the statement there is that there's similar toxicity. And I guess I'm really struggling kind of with how you're defining acute toxicity because I think it's pertinent to how you assess exposure. Which is what this critical body burden thing is really more of an exposure assessment. It's not necessarily a toxicity assessment.

I mean, you're actually looking at an amount of a compound in an organism and I'm having a hard time relating that exposure assessment to an effects assessment. Does that make sense? And it really boils down to the assumption of adaptivity

because in order to assume adaptivity, you assume the same mode of action and if you backed up in order to assume mode of action, you're using QSAR to assume. So I see a tremendous amount of uncertainty for making that first assumption all the way down to an assumption of adaptivity.

MR. ANDERSON: I just want to make sure I understand what you're saying. So you're saying if we're looking at a CBR for multiple residues of concern, for multiple chemicals. Typically I would say we would look at the various structures of the chemicals of concern. How similar are they? What do we know about the toxicity of the chemicals? Is there a possibility it's kept intact? Or is it not? Is there something fundamentally different about the chemical that would affect it's toxicity?

DR. SCHLENK: And you mean toxicity, again, acute lethality, right? It seems like all the data that you're presenting is primarily acute lethality derived.

MR. ANDERSON: That's true.

DR. SCHLENK: And that you're using the degradate with the parent in terms of an additive approach based on acute lethality. Is that accurate?

MR. ANDERSON: When we presented the

TROC analysis we only presented the acute assessment. But we would typically do this, depending on what data we have, we would probably make the same assumption on chronic effects.

MR. SAPPINGTON: Just to go back. When we're looking at the degradates, we would carefully look at the structure of those and compare them. Obviously you have to exercise some judgement in terms of similarity and potential modes of action. Recognizing of course, that the acute mode of action may differ from the chronic mode of action. The preference here is to actually generate, actually have measured values to we can explicitly consider the toxicity differences between the parent and degradate.

When we do not have that information, sometimes we don't have the luxury of waiting until those tests are generated and we're coming here and suggesting what ways might we, what set of assumptions might be reasonable.

So we would be bounding this context of adaptivity with information the best that we could gain from mode of action of this compound. The other source of this information may come from our sister division, HED, where they have a standing committee called the residues concern committee which is designed to

evaluate the formation of degradates as well as their mode of action.

So that information will be brought to bear as well. Before it was just an automatic, default assumption.

DR. SCHLENK: Okay, that's what I needed to know because that's not spelled out in the white paper. The process that you go through to actually make the assumption of adaptivity and then the assumption....so that's primarily what I was after.

DR. HEERINGA: Dr. Chambers.

DR. CHAMBERS: I had a question about the third assumption that you had in the CBR approach, that for a given endpoint it is not time dependent. So are you ignoring that; any kind of physiological adaptation? Any kind of down regulation or up regulation of receptors or something like that? That can really make it time dependent, couldn't it?

DR. ANDERSON: There are some approaches, as in the literature that have looked at time dependence of the critical body residue--I'll defer to my colleague.

MR. SAPPINGTON: Yes, time dependence has been, of the CBR has been shown to be-- to occur for some compounds. And there are different models out

there to evaluate that such as damage repair mechanism, actually treat that as a kinetic process. I think I would add that with regards to the assumption of time dependence and the CBR, I would be uncomfortable stretching that assumption all the way from acute to chronic effect, but within the realm of exposure durations, it might be considered acute and within the realm of exposure durations it might be considered chronic or subchronic.

That that may be where that function, you know, would be placed. I would again put some bounds on that, because once you get into chronic toxicity your modes of actions can differ and the ability of the organism to evoke their repair mechanism; if they are not being as overwhelmed as they would be in acute exposure.

The other thing that comes into play there is different life stages of the organism, particularly if we are talking about a life cycle path. And we know that different life stages have different abilities to biotransform chemicals. So, I would again--I think what Brian is pointing out is that by using a CBR you are making that assumption. However, when we're applying it, I don't think we would use necessarily an acute CBR for a full life-cycle type

assessment.

DR. HEERINGA: Dr. Norstrom?

DR. NORSTROM: My question is on the same topic, actually. It just happened, I was reading a paper last night by Simon Harmons where they reviewed some acute, in this case, toxicity native to things like chlorobenzenes in fish and concluded there can be up to a factor of four difference, depending on time, of exposure. So even in acute exposure phenomenon, I think you might consider that there can be that kind of an error in it, simply because there seems to be different mechanisms operating depending on whether the fish dies within the time frame were talking about here is one day or two days versus three days, that kind of thing. It's not universally true that it will only be one value for acute.

DR. HEERINGA: Dr. Schlenk?

DR. SCHLENK: Yeah, just one other thing I forgot to ask the last time. So again, the assumption of the mode of action is equivalent between all species, or is that taken into account as well? In terms of acute lethality?

MR. ANDERSON: Yeah, I think that would really have to be considered, particularly if you have an insecticide or something like that.

DR. SCHLENK: Right, so that choice is made in the sort of problem formulation meeting or counsel that you have before you actually deciding to go to CVR? Is that accurate?

MR. ANDERSON: My understanding of that committee is mainly mammalian based. However, in some of the QSAR models that are available, they are stratified, if you will, by broad taxonomic grouping. For example, we'll have, I believe we have outputs predicted for algae, for daphnia type planktonic invertebrates, as well as for fish.

And the other output that comes from that is the assumption, based on the structure of the chemical, of the mode of action to use to generate that prediction. So all of this ultimately gets back to the structure of the compound, but also the data sets that are used to validate those predictions. So yes, we would look at differential modes of action depending on the receptor species.

DR. SCHLENK: I guess that, for example, if I had an insect specific receptor antagonist that was DVT in fish, but obviously because it could survive in fish, if you were trying to use an invertebrate, you couldn't because it would kill it so fast you'd never have a chance of getting a body residue. So you are

looking at that?

MR. SAPPINGTON: And that would be the difference in the target versus non-target, put that around, the same thing occurs with an herbicide and if you look at the algal SAR predictions, they're nowhere near close to what we've actually observed, because they're based on more baseline type toxicity and not a specific mode of action that has been developed for that particular herbicide.

DR. SCHLENK: So I would assume then that this program that's utilized then differentiates that or is that done just more on a committee basis?

MR. SAPPINGTON: The SAR Program that I mentioned does give you the output of highly broad taxonomic classes, yes.

But I will add that this is an area that, internally, we are working on and that is to link up with our eco-toxicity data information on mode of action that is specific to different species. Because you have the mode of action, you may have that information for the target species provided by the registrant.

But we're charged with protecting the whole gamut of species out there, so to the extent that there are information available for other species, we

actually are starting a project this January to try to collect that and incorporate it into our database for this exact purpose.

DR. HEERINGA: Dr. Bradbury?

DR. BRADBURY: This will be a little more background on the context of some of the dialogue that's been going on and Dr. Doucette may be able to help during the deliberations as well.

Some of the QSAR modeling systems that Keith and others have been referring to have been developed by the sister part of the agency that deals with the Toxic Substances Control Act. And they've developed a QSAR system that's actually a function of work with ORD and the toxics program that has algorithms that take a look at the potential mode of action, looking at chemical structure.

It's not a plug and chug sort of operation, we use expert judgment as well as going through SAP review and OECD, it gets part of the OECD toolbox, QSAR tools. So that's just an example of one of the tools, one of the approaches to be used to help provide some insights into how to interpret the toxicological potential of a structure and clearly mode of action is a big part of that deliberation.

So I just wanted to clarify that. In

OPP, we're accessing other parts of the agency, but I'm using tools that have gone through extensive peer review in terms of both how they're built and the proper application of those tools in the context, for example, of mode of action and related issues that you've been raising.

DR. HEERINGA: Dr. Hickie?

DR. HICKIE: I wonder if you could go to slide sixteen? My impression is CVR is essentially independent of time, correct? And yet, when I look at that equation, that suggests that CVR should be dependent on time.

MR. ANDERSON: That's going to be a function of the toxicity study, meaning that's the 48 hour LC-50 study. That's estimating concentration in the organisms at the time where effects occurred. So that would the concentration in the organism associated like an LC-50.

DR. HICKIE: So that's a time-dependent LC-50 that's plugged in there?

MR. ANDERSON: Yes, that's right. It's the specific duration of the study.

DR. HICKIE: So, in this case, it would be a 72 hour study? It took three days?

MR. ANDERSON: Yeah, that's right. It's

a four day test and we took values from day three, but yes, that's right.

DR. HICKIE: It's not quite clear then. I was wondering what happens if you plug in two days or four days and particularly since the depuration half life is substantially longer than the testing period.

MR. ANDERSON: Yeah, that's right, that's right.

MR. SAPPINGTON: I think this is designed to estimate the concentration in the organism that's associated with the LC-50 that's observed in a particular test.

DR. HICKIE: So that should be, probably the location there should be, concentration in water should be a time dependent LC-50 as opposed to....

MR. SAPPINGTON: Yes, I mean that's one of the issues we brought up with these compounds. They're not at steady state during these tests. But the notion of time dependence, if that assumption is reasonably valid, would be that a CVR from a longer term exposure should be....the LC-50 associated with a longer-term exposure should be lower, but the CVR, because it's longer term exposure, would be close to the CVR you would get here. So that's the idea.

DR. HICKIE: It's just not entirely

clear in the white paper.

MR. SAPPINGTON: Okay, okay.

DR. HEERINGA: Dr. Delorme?

DR. DELORME: Just following along with Brendan's line of questioning here, there is a another uncertainty in that you're assuming that the death take place at ninety-six hours. In fact, for an LC-50, you're looking at the sum over that time period, so if the organisms all die significantly earlier then you're actually probably over-estimating your critical body residue. Because you're allowing longer time for updates.

MR. SAPPINGTON: I've also seen in the literature, issues about which organisms you measure, in terms of the residues, alive or dead and I think Peter Landern's group has published some information on that.

So yes, advice that you could give us on constraints, limitations, things to really pay attention to with this approach would be useful and recently there was a pellston workshop that Jim Meador chaired and I was involved with that workshop on the tissue residue base approach in general. And so information from that could also be brought in.

DR. HEERINGA: Thank you. Dr. Hickie?

DR. HICKIE: I should send you my PHD from years ago, which was focusing on pulse exposure toxicity and CVR's.

DR. HEERINGA: Dr. Hickie, we can certainly cite that and provide the references and if you want to provide it earlier, that would be fine. Include it in the docket for the meetings. Dr. Schlenk?

DR. SCHLENK: Just one other comment. You had mentioned earlier you are interacting with your sister agencies and I'm just curious, has there been interaction with, in terms of Duluth with John Nichols and PBK type aspects, particularly related to this in terms of critical body.

MR. SAPPINGTON: Yes, we work closely with the Duluth lab on a number of fronts. One of which would pertain to this effort is the Astor program which is another useful toxicity estimation procedure, that's with Chris Russell, indoor, that's being released out on the web, but also to update information in the Astor with some of the newer pesticide ingredients so we can bring the estimation procedures up to par with newer modes of action and newer compounds.

With regards to John's work, his work

with trout PBTK modeling and he's also working on PBTK modeling and in birds, namely a kestrel model with methyl mercury, that's an obvious extension that could be pulled in here. In fact, I'm working to bring him down here in December to give us a seminar on some of that to see where that plays in.

DR. SCHLENK: I think in terms of determining target organ specifically within the animal is pretty critical in this. I mean, I think this is, you know a good approach in terms of a first step, but you really want to refine it, it's determining where the compound is actually going within the organism I think is a very important step in that regard. His methodology would allow you to do that. At least in fish, anyway.

DR. HEERINGA: Yes, Dr. Meador?

DR. MEADOR: I may have missed it did you speculate why bacillus is so much lower on the CVR than those other two invertebrates?

MR. ANDERSON: I don't know why. I don't know why. I don't know if anybody else has any....it seems to be more sensitive, I haven't....

DR. MEADOR: One possibility, I mean that was a ninety-eight day test versus two forty-eight hour tests, so based on that KOW, it could have been

really slow internal re-distribution kinetics.

MR. ANDERSON: The issue is though that we saw effects pretty quick in that study, about six days into the study we started seeing effects.

DR. MEADOR: Even though it went out ninety-eight days?

MR. ANDERSON: That's right, that's right. But it seemed to be a short, possibly an acute effect. It wasn't, didn't appear anyway to be a result of slow accumulation over a period of time.

DR. MEADOR: I mean internally, a re-distribution. That takes quite a while, a KOW like that.

MR. ANDERSON: That's right, but we did see effects occurring within the first week.

DR. MEADOR: Did you consider the fish residue approach for those other two pesticides, one and two?

MR. SAPPINGTON: We did not apply a tissue residue approach to those. We did look at bio-accumulation, but only some aspects of diet and terrestrial organisms.

So as a way to estimate exposure to the diet. But not directly to the organisms themselves. The example chemicals we provided in the white paper

really do reflect a number of assessments done over a several year time period and some of the models we've used in the earlier ones have changed slightly to the next, to the next.

So it does reflect a pretty significant range in terms of the time and year that the assessment was done.

DR. MEADOR: So would you consider those pesticides in another assessment? I mean, would you look at the tissue as a new approach?

MR. SAPPINGTON: Well, for pesticide one, the KOW range would probably suggest that the diet isn't a dominant exposure route. Three and four, the diet, which is located around five and obviously we did it for four.

Those could be considered. But again, one of the issues here is addressing uncertainties with regard to estimating the CVR, which we've been talking about some aspect of that here versus actually verifying that with measured data and I think one of the potential outcomes from this meeting may be well when we have a chemical that meets a certain profile and if we get an indication of that early on, then perhaps we can be more pro-active in trying to get the type of information at the onset that would help us to

reduce the uncertainty. Otherwise, we will be in a mode of basically having to estimate CVR using whatever information we have from water-only exposures.

DR. MEADOR: Yeah, I'd recommend it for any bio-accumulative compound, that really doesn't matter if it's diet or water. The dominance for the route exposure. So it has utility for lots of different compounds.

MR. SAPPINGTON: Oh yeah, the approach is, yes. It's just that right now, the tools we have for compounds that are in the low hydrophobicity range seem to be adequate in terms of addressing the route of exposure as well as addressing the issue of steady state. It's when we start rubbing up against the steady state issue and the dietary problem.

Now the other aspect of CVR that wasn't mentioned but is particularly useful is this whole issue of mixtures and dealing with mixtures with similar modes of action. So that's another potential application, regardless of the other two aspects.

DR. HEERINGA: Dr. Oris?

DR. ORIS: Jim Oris, Miami University. One of the things I think you need to be very clear on are all of the assumptions that you are making.

And some of the assumptions can make

this work or make it fall apart really fast and so I'd encourage you to be very clear on the details of some of your assumptions.

For example, in the slide that's up there now, this is a concentration based pharmaco-kinetic model and as the mass of the organism changes or the concentration of the compound changes during the exposure, then the model is not valid. And so those aren't assumptions that are laid out in what I've read and need to be very explicit about those kinds of things.

So for example in maybe the invertebrate studies, you may not be able to make this assumption of a constant or no growth assumption in the four day test, for example. And if it's a static test, then you may not be able to make the assumption of constant concentration in the exposure.

So those are things that need to be I think explicitly laid out. And then in your list of assumptions for the CVR approach, if you back up to slide thirteen, I think there's some caveats you need to put on some of these to again, make them more clear. So potency is equivalent across exposure routes only if there's no first path metabolism.

CBR is not time dependent if there's no

repair. And an assumption of no growth also has to be put in, so that starts making this more utility in terms of acute toxicity, but if you lay those assumptions out explicitly than the utility that's for more chronic effect, then you need to be very careful how you apply that. So I just encourage you to be very explicit about how you lay out the assumption.

DR. HEERINGA: Thank you Dr. Oris and we'll make sure too that we bring that forward to our response to the charge question. At this point in time, I think I would like to thank Brian Anderson for the presentation and move on to the final presentation and conclusion which is going to be offered by Keith Sappington. Keith?

MR. SAPPINGTON: Thank you. I'm going to be discussing conclusions and some of our early thoughts regarding a path forward with respect to the issues associated with PBT-like chemicals. When we were, certainly after we put the white paper together and we were considering different presentations and the wide scope of these presentations, we thought it would be useful to try to have a wrap up at the end because we're covering quite a lot of territory.

So that will be the first half of my talk and the second half will be focusing on where

might we go from here. Obviously, that's going to be pending a lot of discussions that are going to continue this week and your recommendations and your report. But we thought it would be good to put out a few ideas that we have and get some specific feedback on that. So in terms of broad points, as outlined in the white paper and as presented over the last two days, we've encountered a number of scientific challenges and ecological risk assessment of pesticides that have varying PBT-like characteristics and those are listed here and these follow the topics in the white paper as well as the presentation.

And what we seek in this SAP is input on how we have addressed these issues in past assessments, so sort of backward looking. But also considering where we go from here in the future, because we'd like to have a systematic process, if you will, in the problem formulation phase where we can identify these issues and know when to address them with specific tools and understand the limitations of those tools and their strengths.

We are particularly interested in your feedback because we need that to help focus our resources on the most problematic issues in terms of uncertainty in our assessments and those that are more

tractable in the near term, as well as those in the long term. And lastly, we're looking for recommendations of steps we can take, both in the near term as well as the long term.

We do work closely with the office of research and development and do have opportunities to develop tools but as Dr. Bradbury and Dr. Brady mentioned, we have quite a rigorous schedule we maintain for conducting our ecological risk assessments. The steps that we can take in the near term would be very helpful as you're formulating your recommendations.

Environmental persistence, just to recap here, regarding consideration of parent degradants, again, it's not a question of if but it's a question of how. We presented three methods with the formation of the Klein method being the preferred method, however the question here is when this method is not feasible because of limitations and available data. All of the other two methods, reasonable approximation or do we need to consider other approaches or modifications to these approaches. And the actual selection of these methods will ultimately depend on the available information we have, these or other methods, and the toxicity data and associated uncertainties.

We talked a lot about the solubility issue and the fact that with these types of compounds, when you include them in our aquatic exposure assessment models, we can on occasion predict, estimate environmental concentrations that exceeds the solubility limit report in laboratory studies. We also discussed two different ways in which this issue has been addressed. One is to assume the laboratory based solubility, to actually cap concentrations at that limit.

The other is to cap them but assume that the precipitate that is hypothetically formed is allowed to dissolve when concentrations go below solubility. I think one of the big issues here that would be particularly helpful is your input on the uncertainty of extrapolating our laboratory-derived solubility values to the field.

It's not entirely clear to us what level of uncertainty that entails, we heard about some aspects of materials in the field that may enhance solubility and so with both of these approaches, they make this assumption and I think that's a pretty critical step that would be useful to receive your advice.

Degradation rates, with specific regard

to aquatic metabolism studies, our understanding is that these studies are not designed currently to distinguish between degradation processes that would be occurring in water and sediment, through a two phase system.

We do not interpret the partitioning from the water column to the sediment as a degradation process. And in these cases where our models do account for this partitioning process, for example with KOC, we have demonstrated the use of a total system half life as a way to represent the biotic degradation in the system and that this is used as a degradation component for the model and the partitioning process is handled separately.

We talked also quite a bit about the issue of long term pesticide accumulation and we think this is an issue with these compounds for year to year carryover. The white paper does provide information based on the prism model with respect to predicting long term concentrations of pesticides in soil.

This would be an obvious starting point for some of the terrestrial bio-accumulation issues we just talked about earlier this morning. This is not typically done in our assessments, but the capability is there. And so we are considering this as a logical

starting point for those types of assessments and would appreciate feedback on that. And then the combination of two models can also provide a long term estimates and sediments of pore water.

The issue of sediment dynamics, I think it's very clear from the presentations that how you treat the notion of sediment dynamics, that is resuspension, burial, and deposition and other related processes. How you treat those in your water quality model can have a substantial impact on the risk estimates by changing exposure concentration.

We recognize that the issue of varial, that is assuming a permanent loss, is not a conservative assumption and we sort of have kind of two extremes captured here, one in which varials are seen to be permanent and the other in which it's not addressed explicitly. And so your input on that would be especially useful.

Also, in the context of the spatial scale, because we are talking about a pond field scale, and to the extent that there is uncertainty when extrapolating to other types of water bodies, and keeping in mind that our program is at a national scale, I would offer those as considerations as well. Dr. Ambrose and Dr. Gobis and Dr. Mackay and also Dr.

Parker, provided a summary of a variety of models for addressing the issue of sediment dynamics and it's potential effect on pesticide bio-availability and it's very clear that these can vary from relatively simple processes to highly complex and the question here is how complex do we really need to go and so that's sort of the main question we have in front of us.

Bioaccumulation for these types of compounds we are concerned about other exposure routes and similar to other Agency programs we're considering using a suite of methods, not just one model or a particular type of field study. We believe that the various methods have strengths and they also have limitations and they complement one another in terms of an overall bio-accumulation process and we'd like feedback on that.

With regards to bio-accumulation and terrestrial food web this is not routinely evaluated but we recognize that this may be important for some pesticides and we suggest that we need some tools to evaluate when in the problem formulation process we need to be concerned about this and again, I can't emphasize enough the notion of in the problem formulation process focusing our resources on those pathways that are likely most important to a problem.

Given the volume of these it's tough, but we have to do.

Long range transport , we believe that this issue is relevant, not only for the historical use but also for the current use pesticides and we have summarized our current methods which largely rely on retrospective assessment, that is, looking at monitoring information at remote locations and I've summarized some of the limitations for this.

Dr. Khan also presented information on one tool that could be used to characterize long range transport potential, but we still in terms of ultimately getting to the risk question, we're struggling with linking our near term field loadings to far field concentrations and again this could be the issue of scale in that we're largely addressing what I would call the near field type risk and with a field scale model both in terrestrial and aquatic risk assessment, and so we are particularly interested in your recommendations of how we might put the rest of the spatial scale with relative temporal scale issues.

Regarding aquatic toxic toxicity of these compounds, we have highlighted limitations of our existing methods with respect to multiple exposure routes and toxicity in space eight.

We think the tissue residue approach has some utility in addressing some of these limitations and as we just discussed, as Brian Anderson presented, it is important to understand the function of this approach, but also in the context of the data that we traditionally get.

We think it's preferred to use measured residue effect in relationship and it would be nice to identify this up front where we might need this information and so we could gather that information early in the assessment process but we're also interested in your feedback on the use of estimated residue effect relationship.

We combined toxicity of parent and degradate mixtures. One approach there is to conduct a screen by using the most sensitive toxicity value for the individual components of the mixture but then refining this approach if need be depending on the results of the discussion.

As we just discussed the assumptions regarding mode of action, time dependent, fate and all are integral components to that assessment. Okay, I'm going to shift gears a little bit and talk about the thoughts, these are very early thoughts on path forward and again to reiterate this review represents just a

first installment on this process, at least from our perspective of integrating new methods into address PBT related issues and I've put new in quotations, because it may be new to pesticide programs but a number of these methods have been used in other programs and have been vetted quite rigorously in those programs.

Pending this review our thinking is that the established national and international criteria for classifying compounds according to their persistent bio-accumulative and toxic characteristics might be used as an initial screen in the problem formulation process for identifying when these risk assessment issues should be addressed.

We have listed these criteria here, and the ranges reflect different values used by different institutions and I would add that potentially with regards to bio-accumulation, we might need another bullet here addressing KOA.

These are the criteria summarized even further with regard to the long range transport and toxicity and then just to suggest visually how, the question is how would the problem formulation process differ for these compounds?

I think the overall framework is the same, but the type of information that you would be

really keying in on might be different and the types of questions that you're going to need to address may differ for these types of compounds and so this figure is basically suggesting a possible way of approaching this in problem formulation. The top part of the figure really represents the integrating available information box that I showed in the framework yesterday.

That's where information on the chemical property, its potential for long range transport, persistence, bioaccumulation and toxicity are brought together in conjunction with available screening criteria to ask the question are these related risk assessment issues likely?

This may not be all three of the combinations, there may be one for persistence, for example, or it may be two or it may be all three. If these aren't likely according to either these or a modified set of screening criteria then the problem formulation would likely proceed. It would typically for most of our compounds. However if the issues are flagged then there's a series of questions, appropriate questions, that we just provide some early examples and I'll walk through those.

If we focus on the slides. It's part of

the problem formulation process and the answers to these questions, we think these and other questions as they are developed would help inform the selection of that conceptual endpoint, the conceptual model, and ultimately the blueprint of the risk assessment itself.

Some of the questions again, I put these up here as examples, they're not intended to be exhaustive. They all focus on the PBT issues, so there are I would suggest many other kinds of generic questions that would also be part of the problem formulation process, but with regards to the PBT type questions, obviously understanding which environmental compartment the pesticide is likely to persist and the formation of degradates and their fate and toxicity is also highly relevant.

For compounds that have low solubility we need to ask the question and be careful about our predicted concentrations that may exceed solubility and how, be very explicit in how the bioavailability of these compounds is addressed in the assessment.

It's also important to understand the relationship between dissipation processes, that is movement as well as the degradation of a compound and interpret these in conjunction with the way our environmental models are using them and right now those

processes are used separately, the partitioning and the degradation processes are represented separately.

Understanding the potential for long term accumulation, that's important in basically interpreting the temporal scale of the assessment as well as the utility of both single season exposure estimates and toxicity estimates and then understanding how sensitive the results are to assumptions regarding sediment dynamics and the impact of that on pesticide bioavailability would be another potential question in this process.

These are questions regarding...related to the bioaccumulation assessment and actually we went over these questions yesterday and just in a nutshell I think some of the key questions here are understanding the role of metabolism, particularly when we're using some of the food web models and having an understanding about steady state, how long does the pesticide take to reach steady state in different organisms.

And then of course recognizing the limitations and how to integrate the results from the different types of approaches and with regard to threshold bioaccumulation, particularly as we move forward and potentially develop or evolve or apply

existing tools in this area, that would be an obvious part of the problem formulations process that is not currently rigorously addressed.

Questions regarding long range transport, I think these are already part of our processes in terms of looking at monitoring data but it would be nice especially for new compounds to have some way to flag reliably long range transport potential and understanding aspects of the methods and models that might do this, understanding the norms for that so that we aren't getting too many false positives or false negatives.

Regarding toxicity, a key question here is in understanding the importance of multiple routes of exposure for affecting the sensitivity of the organism, the exposure of the organism and ultimately toxicity to the receptors of concerns and also understanding the potential differential toxicity of the parent and degradate compound and how likely they are to have the same mode of action which is getting back to our earlier conversation.

And we talked about various tools that we could use to try to pull them in in expert judgment, that we can use to try to assess this question and understanding that mode of action can vary across

issues.

It's also important to recognize limitations of our existing standard protocols with regards to steady state and knowing when we need to request data for even longer term exposures if steady state isn't reached even in our, some of our current climate tests like the early life stage tests which would not get at the potential form of eternal transfer.

And another important aspect is understanding the variability and toxicity information that we observe, how likely is that to be actual differences in species sensitivity versus differences in bioavailability perhaps, that's observed in these tests and this becomes I think more relevant when comparing results from a laboratory to the field, but when you're looking at compounds of KOW ranges, of log KOW ranges upwards to eight, bioavailability is extremely important.

And that's what we have in terms of just some questions that we think would be good to have in front of a risk assessor if they're addressing these types of comments. Thank you very much.

DR. HEERINGA: Thank you very much, Mr. Sappington, for that overview. Questions of

clarification from the panel? Dr. Simonich.

DR. SIMONICH: This is certainly more of a policy question but in the real evaluation of current use pesticide, do you consider the regulatory status of that pesticide in other parts of the world?

MR. SAPPINGTON: It's something in a new chemical program and in an existing chemical program, there is a big emphasis for global work sharing and global partnering and undertaking risk assessments, new chemicals and old chemicals.

We have a longstanding collaboration with PMRA in Canada and part under NAFTA which we try to work together to use common tools and common insights driving risk management because you can vary between countries based on the benefits of the compound. In addition notwithstanding we have partnerships with the European Union and Australia and through OECD they look again to try to develop and harmonize approaches.

DR. HEERINGA: Dr. Bucher.

DR. BUCHER: John Bucher. In the White paper there was something that the concept called levels of concern and I'm not sure if I may have missed the discussion of that but I was, as part of the risk characterization that I believe it is there's some,

what could be fairly broad levels of concern category that, categories that it looks like the Agency uses, and my question really is are these categories broad, so broad that a lot of the things that we've talked about today are really refinements that aren't going to affect the overall level of concern?

MR. SAPPINGTON: The, let me first explain the level of concern. They rec...they, when the risk estimate is derived it's basically taking its own estimated exposure and dividing it by an estimate of the toxicity of the compound and that produces a risk quotient.

Levels of concern for acute effects, that toxicity value is something like a LD 50, so there is a, basically an adjustment, if you will, to that risk quotient to reduce that down to more of a threshold level effect so for example, the acute LOC is a value of .5 so an RQ of .5 would raise, or higher, would raise concern regarding potential for acute effect because obviously if you're killing half the organism at an RQ of one that's a pretty severe effect level and there is a gradation of those LOCs depending on some aspects of where different label language might be included and even endangered species, so the LOC is just an adjustment on the magnitude of the RQ that the

Agency uses as a flag.

I think one aspect of this characterization is that may not be carried through or captured just by the magnitude of that RQ or something like the duration, and what we're talking about here are potentially long duration, even after management actions might be taken, so that's another aspect of this characterization to be evaluated. And it's really the ability of the ecosystem to recover after exposure may be adjusted in some way. I'm not sure if that clarifies your question or not, but that's how the LOCs are used and then they are not compound specific right now. They're part of all compounds.

DR. HEERINGA: Dr. Bradbury has some additional...

DR. BRADBURY: If I could just expand a little bit on what Keith said, the levels of concern aren't pass/fail value if you will. As the presenters discussed over the last few days, it goes through a series of tiers in the risk assessment process, iteration of the risk assessment process, and the LOCs can be very helpful if you don't exceed a LOC then our basic assumption is that the likelihood of adverse effects, that use is low enough that you can proceed forward with a regulatory decision and be reasonably

comfortable at a low likelihood of ecological risk.

If the LOC is exceeded it may just be a reflection of the assumptions in that first tier of the risk assessment that has led to those exceedances, if you will, and so it's getting an LOC and many times the, you kind of roll up your sleeve and go to potentially greater levels of refinement of the risk assessment to try to better understand what the risk potential is and try to better quantify the risk potential.

So, that as best we can we move toward a more refined assessment and then you've got to take a look at what that predicted risk is in the context of the benefits of the subject product between ecological applications. So it isn't a pass fail value, it's a value that helps decide, do we need to focus and in what context do we need to focus.

DR. HEERINGA: At this point in time it looks as though everybody is at least satisfied with questions of clarification. I would like to again thank EPA scientific staff, EFED staff for these presentations that we've heard over the past day and a half.

I think it's, again, been highly organized in my experience and I'd let the panel

members comment for themselves but we very much appreciate the way it's been handled and organized all the way from the organization of the packet in sequence, instead of a pile of papers dropped here in various arrays and permutations, I very much like that and we move on.

I think we'll give the panelists a little extra time over lunch if they have any other questions of clarification for after the period of public comment. I mean just return briefly for a few questions of clarification before we launch into the charge question, but at this point in time I'd like to bring this segment of the meeting to a close with a few notes.

Again for panel members, I think there was a call again from Keith Sappington which I think we ought to pay attention to in terms of both near term and long term recommendations because of resource and the need to move ahead. I think it's valuable to be brought in at the planning process at this point.

I think maybe if we can work to sort of differentiate that and not to limit our recommendation but to sort of differentiate the practicality and potentially the other implications in near term and long term recommendations.

For public commenters, we'll enter the period of public comment after our lunch break. There are three scheduled public commenters and I want to make sure that all of the audience is aware that they do have the opportunity to make public comment, at this point it would be limited to a short comment of five minutes.

If you've not already contacted Myrta Christian and registered as a public commenter, you have the opportunity to do so over lunch and we'll add you. For the public commenters who have preregistered and have presentations that we'd like to ask that during the lunch break if you'll take a moment to have your presentations loaded on the lap top and Keith, would it be possible to use that lap top for the public, I guess, Myrta says it is, the DFO says it is, so if you have it on a memory stick or transfer, we would appreciate it if you would load your presentation over the lunch hour so that we'll be ready to go and move steadily through the public comments.

We are going to break for lunch now, we're at a few, about eight minutes short of twelve o'clock and I'd say that we take the time until 1:00 p.m. and we reconvene for the afternoon session at 1:00 p.m. Thank you.

(WHEREUPON , the proceedings were concluded at 11:52 a.m.)

DR. HEERINGA: Good afternoon, everybody, and welcome back to the afternoon session of our second day of our FIFRA Science Advisory Panel meeting on the topic of Selected Issues Associated with Risk Assessment Process for Pesticides with Persistent Bioaccumulative and Toxic Characteristics.

At this point in the process we have completed the series of presentations by the EPA scientific staff, staff of the environmental fate and effects division and had a chance for questions of clarification and we're now entering the period of public comment and we have had three public commenters who have approached the SAP staff and DFO Myrta Christian for permission to make public comment. If there's anyone else that would like to offer a short public comment you can see either Joe Bailey or Myrta directly here and we can try to get you on the schedule but that should happen immediately.

For the scheduled presenters, the first that we have is Dr. Jay Obermeyer who is representing Crop Life America. Dr. Obermeyer. Please feel free to introduce yourself, you have the floor.

DR. OBERMEYER: Good afternoon, again,

I'm Jay Obermeyer, presenting on crop protection. Today I speak on behalf of Crop Life America and I just want to give a few comments on the risk assessment process for pesticides with PBT characteristics.

Just a general comment to start out with, we appreciate the inclusion of the following formulation steps in this area of evaluation of chemicals as P, B, or T, showing support for the use of risk assessment for pesticides with PBT like characteristics. We also believe that screening criteria that EPA cites from Wyckert on page 18 serves as a first step in PBT evaluation. The direct share is exceeded for all three aspects of PBT, just in case that is a tail PBT related problem formulation be conducted.

We also believe there are good modeling approaches available that allow higher tier refinement of exposure risk assessments for reputed PBT chemicals. Recently refined risk assessment will allow EPA to more clearly define potential for risk so that it can be appropriately compared with benefits as required under FIFRA.

We commend EPA for promoting the use of these tools during this SAP. Some additional comments or general comments, we agree upon and understand the

criteria for each component of the classification. We also need to agree upon and understand test methods and calculations. For example, if we're going to use DT 50 or half life as a criteria for determining if a chemical is persistent, then we need to know exactly how we're supposed to measure the DT 50 or half life, whether we're supposed to use lab data or higher tier methods to predict these values.

Also threshold values should trigger further investigation and not trigger restrictions or bans. I really believe that a tiered approach should be used such that higher tier studies can be used to, need to be incorporated such that if lower tier studies cause exceedances of LOCs we can look at all data available to make a better judgment about risk.

We believe that a chemical must exceed all reasonable criteria to trigger further PBT evaluation and guide the problem formulation. Some specific comments on assessing persistence and some of these are redundant but just to reiterate we need agreement on methods for calculating segregation of dissipation kinetics for soil, water and sediment that are based on best available science. We need to understand if a P trigger would be based on lab data or tempered by more realistic higher tiered studies and

field data.

Registrant and regulators need a clearer understanding of P triggers to further evaluate the flow of water and sediment. Precision is only relevant to risk assessments for the target organisms where exposure will occur. For example, here pelagic organisms will not receive chronic water fate exposure from compounds which very rapidly dissipate from the water column into sediment and thus organism behavior and chemical transport conceptual models must be matched in the problem formulation step.

Another one of those that doesn't fit under P, we do want to address that, we need an agreement on methods and that's the exposure concentrations for total residues of concerns. So specific comments on assessing toxicity there are multiple guidelines today that generate toxicity end points. These end points can be used to assess toxicity risk of compounds that may bioaccumulate or bioconcentrate.

Again we want to stress we feel it's more appropriate to use a risk based approach when assessing toxicity and sort of propose that the level of accumulation of various trophic levels can be compared to known effects levels for focus species in

the food chain and this is gone, getting back to the idea of looking at bioaccumulation through aquatic food web such that forage fish and piscivorous fish which are then eaten by piscivorous birds or mammals and then those residue levels are transferred into the terrestrial system.

The comparison there between what's transferred from the aquatic to the terrestrial comparing that to LOELs and NOELs that are derived or produced for terrestrial organisms need to be clearly thought through and investigated more thoroughly before those locations are used.

Roughly that refinements should be allowed. We're thinking perhaps a chemical has multiple crop uses but with refinement it can be shown that only one of the crop uses that may have twice the application rate or maybe it's used four times in the application where another one might only have two applications, those type of adjustments can be used looking at LOC exceedances.

And we talked about stressing a tiered approach for PBT evaluations and risk assessments, got more detail here. Again tier one studies are basically guideline studies with modeling leading to comparison data with extreme criteria. If we get exceedances with

the Tier 1, we can go to higher tiered studies which is Tier 2, which is a simple nine guideline study with modeling typically incorporating additional media such as instead of just water by itself with water and sediment.

Tier 3 study is a little bit more complex, nine guidelines, modeling and again, in addition to the multimedia, we're also looking at multi-trophic, we're not looking at just one species but multiple species within the system.

With Tier 4 looking at some of the data plus probabilistic modeling approaches and the data here is we can get a weight of evidence approach such that we incorporate all the data that we have to make appropriate decisions.

We heard quite a bit about probabilistic model in yesterday's discussions. Just to briefly go over these. This type of modeling allows especially different crops, water bodies and water conditions in aquatic food webs. It allows the evaluation of mitigation practices such as reducing off charted spray drift and vegetative filter strips.

Food web modeling allows the exploration of the water in sediment compartments into value potential impacts to different trophic levels.

Compartment specific data can be incorporated into model assessments through fine exposures and resulting risks.

Here's a...to give an example of how higher tier studies can be used. The Tier 1 are a basic bio concentration study here done with fish. It was brought up yesterday that this was probably not a very good test to use for these PBT like chemicals because these chemicals have high log KOWs are more likely going to dissipate out in the water and sediment so really as Dr. Gobas mentioned measuring the chemical properties of the fish as opposed to, the properties of the fish as opposed to the properties of the chemical.

So we don't feel that data obtained from this type of study really is useful for PBT like chemicals. If we go to a higher tier study with probabilistic modeling such that we have a field model, a water body model, food web model, we can look at potentially with more detail how the chemicals are interacting within the food web. There's obviously more places for the chemical to be taken up or absorbed to such as the sediment in the different points in the food web.

And we think that this model here can give us a better indication of what the final

concentration might be for these types of chemicals and this would be a much better predictor than say for instance looking at standard multiplication values for different trophic levels so we're looking at a whole system here as opposed to just factoring in concentration factors with increase in trophic position.

Some comments on log KOW and dietary exposure for assessing bioaccumulation. Log KOW is a good screen since all substances known to bioaccumulate have a log KOW greater than five. However, not all substances that have a log KOW of 5 actually do bioaccumulate. There are many factors that determine whether a substance will bioaccumulate.

You need to relate it to the chemical as well as to the organisms. Obviously log KOW is an important component, but there's also other factors such as PKA or molecular size that from a chemical standpoint could affect bioavailability and bioaccumulation. And from the organism things such as size or the sex of the organism can affect bioaccumulation.

The series from food and media is a more complex process, made by things such as assimilation from the sea as well as metabolism. This graph here

illustrates the comparison with log KOW with bioaccumulation from food and this graph here represents a literature review of 656 studies.

The main thing I wanted to point out here that if you look at the individual log KOW values especially around, like a KOW of around 7, you see that there's quite a bit of variation within the data there showing that in some instances a chemical with a log KOW of 7 would bioaccumulate and others that indicate that it will not bioaccumulate.

So the main thing here is just to point out that strictly using log KOW as our indicator for criteria may not be that great but again it's definitely a component but other processes and parameters should be also considered.

As far as terrestrial assessments and long range transport. We do not address terrestrial evaluation but would like to mention that there are some models of interest that Gobas et. al. have developed that might be useful and again support using models with a SWAT type approach to assist them. As far as long range transport we do see the use of models as helpful for this area of regulation. The available models should be fully explored again using a SWAT type of approach to start.

So in summary Crop Life America recommends a tiered approach to characterization of chemicals as P, B, or T. CLA supports a multi tier approach to ecological risk assessment of pesticides. Crop Life America recommends a weight of evidence approach to risk assessment based on multiple tiers of data and modeling.

CLA commends EPA for continuing to support the use of risk assessment in this area of evaluation. We believe this area is more technically difficult to assess than other evaluations EPA currently utilizes for regulatory purposes. We believe there are good tools available that allow refinement in this area of risk assessment which will allow EPA to more clearly define risk versus benefit under FIFRA law. We commend EPA for knowing the use of these tools during this SAP. That's all I have. Thank you.

DR. HEERINGA: Thank you, Dr. Obermeyer. Are there questions, clarification for Dr. Obermeyer on his presentation? I don't see anything, thank you very much. Our next public commenter is Dr. Lynn McCarty who is representing Valent USA and Dr. McCarty, I'll let you introduce yourself.

DR. MCCARTY: Great, thank you. My name is Lynn McCarty. Just by way of background, I'm

an ecotoxicologist with a longstanding interest in residue based toxicity approaches which I think is quite appropriate for this meeting.

My co-author is John Arnot who is an environmental modeler with considerable experience and expertise in fate and exposure modeling to a wide variety of organic chemicals. You may recognize his name as the lead author on the bio food web model that's been used in the assessments here. You may also know of his association with Don Mackay.

I want to just point out that although this study was funded by Valent and the focus and clarity benefitted substantially by input from Valent staff, the report represents independent view opinions and the best professional judgments of the authors.

So what we're going to do is a very technical approach. We have been asked to do an ecological risk assessment for Pyridaben. This is chemical four in the White paper in case you're interested so that you can use, feel free to use any information to compare and contrast.

So just a quick overview of the presentation, it's pretty standard. We're going to have a background of Pyridaben's properties, some key points and some uncertainties I can just highlight of

the data and risk exposure modeling and the toxicity evaluation, a risk quotient analysis and some conclusions.

Now my co-author John Arnot made me promise that I would bring this up. It's in our report, and by the way the companion report for this presentation is a 101 page report which is on the public docket and I think the panel members can have it, probably have it already and anybody else who...it is a public document.

DR. HEERINGA: Panel members do have it and have seen it, thank you very much.

DR. MCCARTY: Mainly John wanted me to mention that in there we mention that we used a holistic modeling approach, and I am sure that I see people having visions of John and I sitting in front of our computers wearing our sandals, our hemp cloth shirts and eating or getting granola bars but that's not really what it means in this sense. What we meant by holistic modeling was that we used a comprehensive internally consistent treatment of the model systems as a whole rather than simply a group of various parts from pieces of information and various sources.

We recognize that the data comes from a variety of sources and may require interpretation or

adjustment for optimum use in any particular model. For example, we use, and this is one thing that John has spent his Ph.D. on, a part of it at least is using elementric adjustment for metabolic rates so that we, if we found something for an organism we made sure that if we used it for another phase that the, it was scaled properly to fit in so that's just an idea and we spent a great deal of time making sure everything fit properly and was, reflected our best judgment what the information should do.

So just by way of background Pyridaben is an insecticide effective on laparodopolis larva and frips. It has recently been approved for the use in the USA in enclosed greenhouses.

It has been the subject of some previous risk assessments which have concluded that health, human health risk is very low. It also was fairly good evidence to suggest, experimental evidence that suggests, available information that Pyridaben does not biomagnify in aquatic food chains and the key thing that we're going, the background issue here is that in a recent revised updated USEPA risk assessment for this chemical, they specifically requested more data evaluation and uncertainty reduction for body residue effect relationships for selected representative

aquatic species in particular daphnia carotenoids in fish which would be used to finalize the outdoor use registration process.

So just a, for the chemists in the crowd and I know there are many of you, we just have a slide here with some of the basic chemical properties of this substance. The chemical structure, molecular weight, et al. I get to point out for the log Koc at the bottom there 6.1 was the log Koc that was used by EPA and available in previous studies.

We looked at that. EPA classified this particular study this number came from as a secondary level quality data. We looked at this and John thought that the particular study wasn't quite what he had hoped for, it was, they used soil instead of sediment, they used was a low Koc content in the soil and it was a short term test so he went to the literature, Seth, et al., 1999 which is actually an update of Parakov1981 which is a review of the relationship between KOW and Koc and using that he decided that we might want to look at using a log Koc of 7.6.

As I'll mention later, we used this only for the aquatic phase not for the soil runoff part of the modeling. So the key points are that the key, the PRZM/EXAMS ACROBAT modeling assumption is steady state

bioaccumulation which is based on peak water and sediment levels and we are seriously worried about this as you, and you've heard this repeated a number of times before as high KOW compounds, we believe it may not be appropriate for pulse applications of pseudofibrophotic chemicals.

Water solubility in Pyridaben is very low, as you've seen here 0.15 micrograms per liter and a point that the toxicologists would probably jump on is that solvent is commonly used in toxicity accumulation experiments to increase the bioavailable concentration. It is my contention that the use of solvents could be found in toxicity and bioaccumulation test interpretation and we'll get into that in a little bit.

So we listed some uncertainties here since uncertainties is what we're trying to reduce here in this particular case, but we note that despite any uncertainties we might do there are a number of other uncertainties that remain, that are characteristics of the site. We've been talking about the nature of the pond and whether that was appropriate.

Label use versus model assumption. Sometimes the modeling that goes into the registration analysis is, pushes the limits of what the label might

suggest was appropriate. We have some uncertain chemical physical properties. It is a super hydrophotic chemical and whatnot.

I mean the last one I'll bring up, attention to is the assumption, we used the assumption that the total radio labeled residue, the equivalent to the total mass for Pyridaben in the organism was part of the effective dose so the measured data that we used, we had radio labeled material rather than traditional chemistry analysis.

However, we find out that these and other uncertainties and variabilities are typically encountered in pesticide risk assessment. They're not unique. Some of the, some of the emphasis may be a little different depending on what the KOW and the chemical is but I mean these are the, we could count on these all the time.

But our objective in this particular study is to reduce the uncertainty in fate exposure modeling and residue based toxicity variables as suggested in the most recent EPA risk assessment. So now you heard Donald Kise talk the other day and I believe the word essential came up with regards to using time dependent exposure. I think Don must have been really thinking very hard because we were

obviously channeling him and we did this having two models here without his input.

We used, in this thing we ran two models in parallel. The PRZM/EXAMS ACROBAT model that EPA's been using and the PRZM/AGRO model which is QUASI and ACROBAT which Don and Frank Gobas talked about. We used this in three tiers of increasing sophistication. In Tier 1 we didn't have any biotransformation information included, so zero transformation, biotransformation.

We used the log Koc value that was being used and no time dependent exposure and we ran both models. In Tier 2 we ran, we included now estimates of biotransformation. There's substantial and good evidence that this material is biotransformed and we selected a number of estimates of that to include in the model. We used the same log KOC, no time dependent exposure.

It is not impossible or extremely difficult to get PRZM, or to get EXAMS to do all sorts. For three B we only use AGRO, the AGRO model, we continue to use PRZM at the bottom as we mentioned, but we can't do kind of an exposure but we also did two steps and the first part we included the biotransformation, we used the log Koc of 7.6 for

aquatic modeling and 6.1 for transfer runoff but no time dependent exposure and then in the most sophisticated tier, 3B 2 we turned on time dependent exposure and we're going to look at that and we'll be able to compare all these things and give you an idea of the influence of these various parameters as, if you will, a bit of a sensitivity not so much analysis but view.

So the exposure modeling that John carried out, we did seven of the outdoor agricultural use scenarios as used by USEPA in 2008 and permitted by the proposed Pyridaben label and we used the USEPA fate modeling inputs unless we note otherwise. We used the actual water solubility of the chemical in the modeling and as you'll see later on, we're in the risk quotient analysis, I'm not going to present the details of that data. Those are in the report.

Models A and B produced similar exposures for Tier 1 and Tier 2 so both of those models in these situations using the same thing, but give similar results of our model. The model B, AGRO, gives a little, tends to be a little bit lower but pretty close, pretty close.

As a result of this we identified the worst, the worst case highest exposure situation which

was for North Carolina apples treated, using the ground application scenario. This was consisted of 60.4 pounds for active ingredient per acre applications 14 days apart.

I just want to point out that that's not typical of what this pesticide would be used for, it tended to be used in three treatments for growing season and in this area they have two growing seasons so you wouldn't typically put them, run six applications in a row but anyway, we used that anyway because that was what the previous assessment used.

So what we have here is a graphical output summarizing some of this information. I'll take a second to explain it. The first thing I want to point out is that the bulk water in blue is in nanograms per liter and the sediment concentration's in nanograms per gram so they're a thousand times different.

The log scale on the side is you just have to drop the units appropriately, we did that so that the graph would be a little more trackable because otherwise we'd have to shrink everything down so I think most of the people here can resolve that, but you can see comparing the steady state application exposure assumption versus a time dependent assumption that

there's quite a difference.

This is a 25 year run, and I think two things that you want to take away here is that the first that there's a major difference between the steady state and the time dependent exposure modeling for water concentration and sediment concentrations and I think you also want to take away there's no evidence of year to year increases in environmental levels of Pyridaben based on 25 years of six times per year app... six applications per year.

This is a similar sort of slide we've included here the study, we've taken out the water and sediment concentrations for the study's sake, but we have now included at the top and the horizontal lines a steady state estimated biological concentrations for azul plankton bred invertebrates and fish eating fish.

You can see here and the rest of it for the time independent exposure we have a complete thing, water sediment and those three groups of organisms you can see the exposure happening, the very rapid change that occurred there and everything here in the season. This is a two year period, if you ran we've stretched it, we've gone into the two years so you can see that in a bit more detail the trend lines that are happening here and the big differences between steady state and

exposure and time dependent exposure but if you ran, you know, it looks very much the same as the previous graph if you ran it over, if you showed all thirty years.

And again in this case you have to be careful with the water is in nanograms per liter and the remaining materials in nanograms per gram. So that was how we got to, how we developed some exposures and I'll talk a little bit later about the numbers we used for the organisms but that's the basis of the exposure.

The toxicity assessment, we used the body residue rates alternative approach. This is agreed to as applicable by both USEPA and Valent so there was no disagreement on that at all. However, we, John and I did this, decided to do this independent and from scratch So we started from, break from all the data that was available and made our own decisions on this.

So we really evaluated all of the available calculated and experimental residue data. The calculated residue data was judged to be uncertain due to the use of solvent in most of the experimental testing. I still cannot understand how people put solvent in things because if you put chemicals like

this in a water exposure immediately they don't cause any toxicity.

If you put sediment, if you put solvent in, they cause toxicity but you have modified the system dramatically by increasing the bioavailability so of course that's true and of course the toxic effect does occur, but it means you can't compare that result against tests where you didn't do, where you didn't have solvent in them because you haven't quantified the influence of the solvent and you have to know that it's changed things.

So it's not that solvent is necessarily unacceptable, it's that you must quantify the influence. I mean if I did this test at two degrees and a standard test was at twenty degrees, that wouldn't be allowed because we know that temperature has a difference. Well, solvent has the same sort of modifying factor on toxicity and unless it's quantified I don't believe that you can reliably use it and raise a great deal of uncertainty.

So anyway, so we were left with an experimental body residue data which was measured. I went through a detailed analysis of that and there was a detailed justification for the selection of that data and it's in the appendix of the report if you want to

see it. I'll give you my conclusions here in a second.

So we determined the estimated residue, effect residue for the representative aquatic species, as we needed to do something, we needed to do the risk quotient analysis, so that value was set at 0.04 millimoles per kilogram based on the highest no effect residue for the most extensive aquatic specifies which is daphnia.

And this just gives you an idea of what this, and it's a fairly detailed slide, I'll take a second on it. The, this part right here, this is the, this would be where the denominator, this is the value here that I chose the denominator and risk quotient analysis, but I have a bunch of organisms and groups here, and I have effect and no effect and you can see here I put narcosis up here just as a reference for organic chemicals.

This is the range that, of the calculated, EPA calculated values for fish minor, some effects and no effect levels here of that typical range. I think that was mentioned this morning. I'm not making much of it, but I don't have any other acute data but this is all no effect levels for fish.

This is effect level for daphnia and

these are no effect levels for daphnia and these are again measured data and this is effect levels for carotenoids and no effect levels for carotenoids, and some additional material here for other organisms.

So what happens, this allows me to get an idea of where the border between effects and no effects occur on a residue basis and so again as a more detailed analysis of how we did this, but this .04 is the basis for your highest, just below the highest no effect level and again these are, I went into details of why I've done that.

So we've chosen that as representative of all the organisms and that is, my est...that is actually an estimated steady state value for accumulation.

So here's a summary of what we did. I talked about earlier we have the application models, the three organisms, remembering that we're using the daphnia effect level, critical effect level for all of the organisms so in Tier 1A and Tier 2B which is essentially the two different models doing exactly what EPA did, you can see that the risk goes into slightly above one.

When you include biotransformation here in model, tier 2A and B in the two different models you

can see it neatly drops substantially below one so biotransformation is a significant determinant in the exposure of this organism and if you don't include it and it does happen if you don't include, it's likely that you'll overestimate the risk.

And now we weren't able to use the EXAMS model here so Tier 3B is out of the AGRO model only. In Tier B1 the only difference is we changed the Koc to 7.6 for the aquatic fate determination and see, that lowers it a modest amount. It's not trivial but it's not huge either, but we think that was a refinement worthwhile and given the uncertainty in this we felt that that was a, reflected a better decision.

However, when we turn on the time dependent exposure in Tier 3B2 you can see that the risk quotient drops dramatically, so, and the risk quotient is based on the 90th percentile of the model exposure residue for the species divided by that environmental effect residue of 0.04.

So I haven't given you, I mean the model numbers for the actual body residues are available in the report , and you can, this is simply the risk quotient so you can, you can figure out matters if you want to look at the report and get the exact ones if you're interested.

Just a visual of this. Here's what it looks like. Here's, here's the organisms. They all put raw data. These are the numbers that we used to try to find some endpoint of interest but it was, came out without the model, and here are the organisms and here is the model organism residues for all the key organisms up here.

We used the highest fish one, not the lowest one, so you can see there's a big difference between where things might happen and where the best estimate, the best model, most refined model estimates would suggest the residues would be.

So conclusions. Two sets of conclusions, one is for exposure. For exposure assessment mass balance modeling was refined with measured laboratory and mesocosm bioaccumulation data and the inclusion of biotransformation is important in exposure modeling as it can significantly reduce exposure and the tiered modeling approach allows the effects of refinement to be followed and it's quite clear that the realistic time dependent exposure generates much lower residue concentrations than steady state exposure. I don't think I'm saying anything new there, we've heard that repeatedly in the previous talks.

So the toxicology conclusions. Environmental effect residue of 0.04 millimoles per kilogram was derived from measured experimental body residue effect data for the most sensitive species. We believe that the measured body residue effect values reduced toxicity uncertainty for the USEPA identified organisms of concern, I think daphnia carotenoids in fish and that the worst case application risk quotient values for the most refined exposure modeling, Tier 3B2, using the daphnia EER are about forty to a hundred times or more below a risk quotient of one.

So I think, I think we, we believe that we've achieved our objective which is a substantial reduction in the uncertainly of both exposure and residue based toxicity and interpretation of Pyridaben in outdoor agricultural use. Thank you.

DR. HEERINGA: Thank you very much, Dr. McCarty. Questions? Dr. Simonich.

DR. SIMONICH: I have a few questions. What were the assumptions made for in terms of the environmental half lives for the various compartments?

DR. MCCARTY: Those are all documented in the book. To give you an idea of what we did is actually both John and I probably would have been on this thing, except we were working on this. And what,

I've been on previous ones and my objective in this was to prepare a report that would be like the report I would like to receive if I was on the committee. So if you look at our, our detailed report, there is a fairly extensive executive summary which summarizes all the details.

There's a main part of the report which gives you a more detailed approach and there is a, almost half of the report is an appendix which is a very detailed presentation of all the data that was examined, what we did with it, and all the assumptions, all the data values we used in the modeling, whatnot, and all of the, every decision that we made we justified that, so there's a table of contents in the appendix where you can go and look for any of those questions and it will be, it should be in the table of contents and so I know those answers are in, in the report.

DR. SIMONICH: But you can't give those to me now?

DR. MCCARTY: You have the report.

DR. SIMONICH: I don't have the report right here at this moment.

DR. MCCARTY: Well, I don['t have it, you know, I'm...

DR. SIMONICH: Okay, so you don't remember the values offhand?

DR. MCCARTY: No, I don't.

DR. SIMONICH: And then what is the major transformation reaction?

DR. MCCARTY: I'm not sure what you mean.

DR. SIMONICH: What are the...

DR. MCCARTY: Well, we, we don't...what we have is we have estimates of metabolic degradation. We don't know exactly what's happening, but the parent compound is, is, is changing.

DR. SIMONICH: Transformation reactions in the environmental compartment other than...

DR. MCCARTY: I'm not sure that we used, again, this is John's area of responsibility but again, the information is in the report. The main part, I'm not sure of whether we, exactly what was in it, but I know that there's for various organisms that were modeled, that's the main part of the metabolism we're considering, not, that's what we had data for which are fish invertebrates.

DR. SIMONICH: And what about the assessment of persistent degradation products?

DR. MCCARTY: We didn't do that.

DR. SIMONICH: Thank you.

DR. MCCARTY: The EPA has judged that the degradation products were not significant toxicological importance and so we tried the, I mean we are facing the same problem EPA has here in that we would like to have done more as well, just as they would have done, but you know, the result was a little bit about information that happens to be very fortunately more information for this particular chemical in that the work that was done used real label Pyridaben in experiments and so they were able to collect information on residues, whereas most of it, many chemicals don't have that sort of information so there's nothing. So we were very lucky to have something to work with when in fact typically you don't have very little.

DR. HEERINGA: Yes. Dr. Norstrom?

DR. NORSTROM: The, staying on the metabolite issue, do we actually know what the structure of the metabolites are?

DR. MCCARTY: There is some information on that, yes.

DR. NORSTROM: And they've been judged as not being persistent or not?

DR. MCCARTY: You'll have to check EPA's

thing. That's not within the scope of what we focused on.

DR. NORSTROM: So when you made those adjustments there, reducing whatever confirmed in the slide for the metabolism, is that based on radio label studies?

DR. MCCARTY: Yes.

DR. NORSTROM: So it's basically a decrease of the starting compound?

DR. MCCARTY: Yes.

DR. NORSTROM: And does this mean that the other radio labeled substances were missing? In other words they've been excreted or...

DR. MCCARTY: There's a variety of things that could have happened to them. Again this is, most of the information was not collected with the idea of having a detailed analysis of this type done so we've done the best that we could do with what we had.

DR. NORSTROM: I guess I'm asking what the experimental protocol for something like that was. Can we say whether or not any of the non-starting radio labeled stuff was still in fish, for example, or whether it was excreted or can we say that at all?

DR. MCCARTY: There is some information that indicates that and various things, it's a very

detailed thing which we did not address.

Again there's only so much uncertainty reduction we can do, but I understand your points and they're all well taken but I mean after a while, we can do a great deal more of sophisticated work but not really get any farther ahead and I think that's the situation you're in here.

We had enough information to improve the process but not to the level that you might like when you start to go down that improvement, but nonetheless it's better, I believe that this is much better, a much better evaluation and a much clearer picture than we would have had without those.

DR. NORSTROM: Yeah, I agree it's a great start. Thanks for providing the structure, by the way, which is where some of my questions are coming from. Some of the potential metabolites, just off the top of my head, could be persistent.

DR. MCCARTY: Well, we, our objective was not to do the per... I mean if you remember our objective, it was simply remove the uncertainty in the fate as we, fate and toxicity and the fate has to do with the toxicity part so we're not, I mean although this panel has a much broader mandate, what we did here was very narrowly focused and this chemical has been

under review for some time, it has received partial registration.

The, in the draft directive that we received from, that the company received from the EPA early this year is what we're addressing, so that the presumption that I would make is that the outer bounds have been dealt with.

Whether it's been to be satisfactory to your point of view or not I"m not sure, but that's not where again, we're not doing research here, we're trying to do focus on the regular activities but I want to make it very clear that we tried to put everything we possibly could in here to be.

So if you want, if you want to go into great detail and look at this, the material is there and it's clear as we could possibly make it and you should be able to find it easily and you may not necessarily agree with what we did, but you will know what we did which is I think one of the things that I always like to see and often don't get.

DR. NORSTROM: Thanks.

DR. HEERINGA: Just a note to the panel, too, the report on the Valent study, it was emailed by Myrta Christian to us, it was not part of the CD transmission.

Dr. Steenhuis?

DR. STEENHUIS: I would like to come back to the half life. We have pesticide four in the large....we have pesticide four in the, in our, in the white paper. It says half life ranging from 224 days 'til 1,110 days in the sediment or in the soil and your simulation shows that it disappeared in no time and I don't completely, I don't understand that. There's no carrying over effect that we can show.

DR. MCCARTY: Well, the information that you're talking about there is from a, and this relates to the previous question. This is a general metabolism by the micro biota in the field essentially. It will for all fate and we're talking, the metabolism we're including is simply the metabolism of the organisms that were, their exposure was being modeled for so we didn't substantially include that.

So the other thing is, that if you look at the mesocosm data which has been collected in this, the material disappears very quickly under the water column so those things, I think a lot of those estimates you may, it depends on who's estimating and what they're looking for. You can get quite a difference, but I mean, I know that, for example, in the mesocosm, the water column concentration is ten

percent of initial concentration after twenty four hours.

So I understand it's not the answer to your question, but there are some, this is actually one of the things I think we saw earlier that there's lots of, when we classified these things by these schemes, we use a bunch of assumptions. Those are models, very simple models and when the thing, if a thing meets assumption of the model then it's pretty good but this is a chemical that has some characteristics that fit into this category but there are some other things that modify that.

I think Dr. Bidleman was talking about some of those things for long range transport. I think there are some other things in terms of fate and persistence that may well apply so I can't answer all the...I can't answer the details of that question and I understand your confusion but we have our justification for what we did is in the report and we used the numbers, the numbers are there and you can review those and make your own conclusions.

DR. HEERINGA: Yes, Dr. Doucette.

DR. DOUCETTE: Lynn, given the, the other questions you brought up, this is probably minor, but I was interested in your choice of Koc going from

and I realize that soil with low organic carbon is different than sediment, but you chose an estimated value over an experimental value and Koc is supposed to be a constant and it worked to the advantage of lowering the quotient. How do you justify something like that?

DR. MCCARTY: Well, I relied on, John and I had the detailed discussion of that and he felt that that compilation of things for a wide variety of organic chemicals, the correlation, this is basically QUASAR, if you will, he felt that that QUASAR better reflected the estimate of this and he's an expert in this field as you well know, he's graded some of the...appropriated some of the models that are used here and so he felt it was not our concern to consider. However, you'll note very deliberately the way it was presented.

If you believe that and I understand your concern, you can subtract that out from the risk quotient analysis and it's very easy to do and you can still come up with it and I think you'll find it still doesn't make a big difference but we were very deliberate to try and make it very clear for people to do exactly that analysis.

I mean, there's nothing, I mean, again,

there's nothing here to hide, we wanted to be completely as open as possible so that people could follow it and make their own decisions and so I think that we've allowed you to do that and that's why I feel as if we've achieved exactly what we set out to do .

DR. HEERINGA: Dr. Hickie?

DR. HICKIE: I'd just like to make a comment, Koc should be a constant provided the organic carbon's the dominant absorption factor and that it's constant in composition between locations.

DR. HEERINGA: Dr. Maddalena?

DR. MADDALENA: That was a nice case study and, and report, thanks for providing that. I want to talk a little bit hypothetically here and allow you to step out of your role for Valent and just be... As a scientist if you had this product and you were going to register it, what area would it be applied on roughly and knowing that area and mass that's applied, what fraction leaves that area? So in other words is this for cumquats or is it for wheat and corn and I think that would make a very big difference in its ultimate fate.

DR. MCCARTY: Well, I mean, again, the scenarios were specified by the EPA for analysis and those are available in the Anderson, et al. 2008 report

on this so I...you're asking me to give a personal opinion about a regulatory decision, I don't feel very comfortable doing that.

I mean, we used, in fact, for example, we used six, six applications in a row, fourteen days apart which is normally spent to be three applications over two seasons so there would be quite a different exposure scenario as a result of that assumption. We use that assumption although I don't think it's realistic so simply because we're trying to be as close as possible to what EPA has done because then if we question all those things then we get into a great big discussion.

What we tried to do was to refine the estimates of these things as they specifically requested to try and show that perhaps there was a better way of doing it and that with very modest justifiable changes, we could demonstrate something different and actually I wasn't sure that that was going to happen. I mean we didn't have a preconceived outcome.

We did these things and this is literally what happened so I mean it could have been, it could have gone the other way and I might not be sitting here so you understand you're asking me a

question that really isn't in my purview to change.

DR. MADDALENA: Okay. I, I understand that...

DR. HEERINGA: Dr. McCarty, I agree on that too and let's make sure that we focus on the science and the report as it's presented, I don't think we need to cross examine.

DR. MADDALENA: Okay, I'm not cross examining. I appreciate it. It was a very good report. The point I'm trying to get at is whether we're looking for the keys under the street light. Where we're looking at it, this is an entirely different class of chemicals that you guys are having to deal with now and the fact that we did a really good job with the pond and with this assessment doesn't give me a lot of comfort in the long term and that is what I was asking you.

DR. MCCARTY: Okay, well, I understand, that's why the panel's here and I'm not sure I have the answer either on this, but I mean I think that this is the, this is the difficulty of being in transition here because this company is trying to register something when things are or the ground is shifting.

So I think that's why we focused very narrowly on answering the question, because I couldn't

anticipate what I need to do to try and assess that question but if you, if the panel comes up with something that decides you should do something different, I'm sure that the registrants will be, will do that when it's defined to be done.

I think you know, I mean, stepping outside my role and speaking for Valent here, I was very impressed that they were willing to put this on the public record

You know, this is pretty remarkable as far as I'm concerned in my experience and so, but they obviously have some confidence on this thing and they volunteered, they're being, as they say in government circles in Canada, at least open and transparent.

So I think that, you know, that to me is very, so they're trying to follow the rules as best they can and if the rules change I believe that these and other companies will try to do the same things so I think the opportunity here, I'm particularly pleased that the EPA is looking in this direction, I think it's the way to go, I think there's a lot of the devil's in the details but nonetheless the concepts, I don't have any problems with the concepts. Like you I'm not sure what the details will bring, but you know, let's see what happens.

DR. HEERINGA: Dr. Delorme?

DR. DELORME: Perhaps a minor point, Lynn, but with respect to your comments on solubility, one of the things we have to recognize is at the time of application there's a certain fraction that drifts into a water body possibly and that's in a formulated product which also often includes other things to keep it in solution. So completely dismissing the use of co-solvents I think is a little bit dangerous at times...

DR. MCCARTY: I didn't completely...being very careful there...

DR. DELORME: But there are situations where the compound may be more bioavailable...

DR. MCCARTY: Absolutely.

DR. DELORME: ...and the impact that that has, I'm not sure, it may be minor...

DR. MCCARTY: No, no, I'm sure you're right, but for what we were doing in trying to interpret a toxicity test where I do not know what the solvent did to mod...I mean I know the end result. They made something that when I put in the water normally was nontoxic that made it toxic, okay, so it clearly did something and I don't know how much of whatever it did, it did.

DR. DELORME: Right.

DR. MCCARTY: And so therefore I can't, I can't correct that, now fortunately if I had residues, if they had collected residues from this study when they were doing that, I could have, I could have figured out and fortunately there was some, there were a number of experimental, experiments carried out where they did, where they did this and I was able to do that and I did away with all that stuff.

And went strictly to the residue and tried to make sure it was that steady state so it was something I could, you know, the body residue was a steady state within the body compartment, and use that directly, but no, I mean to me they've done...when you use solvents in a toxicity testing it's only half the thing, because if you, you need to know what the effect is.

It's like saying, well, I did a test that, for a chemical physical property but I didn't correct the systemic temperature and pressure, nobody would do that and that's the equivalent of what we're doing here. We're doing something that we know is wrong and we're not collecting information to correct it, but I agree with you and that that's an issue.

In fact what you might end up doing in

toxicity testing is sometimes is actually collect that information with and without, so that you'll have an understanding of what the co-solvents and....

DR. DELORME: Or include the end use product as well.

DR. MCCARTY: Or test the product. In some cases the product was tested, but no, it is a good point, but again I didn't throw the baby out with the bath water, okay.

DR. HEERINGA: Okay, two more questions. Dr. Steenhuis and Dr. Simonich.

DR. STEENHUIS: Hi, my name is Thomas Steenhuis, I would like, I mean, I don't have time to read the whole paper in just five minutes. What you have here are sedimentation and deposition rates for the part, how did you determine that?

DR. MCCARTY: That I believe is EPA models. So sedimentation rate, that is AGRO, that's the AGRO model, and I'm told that that particular sedimentation rate is quite low as sedimentation rates go. Now this is, this is John's area of expertise but I asked Frank Gobas to be sure about that and Frank told me that, he looked at the number because I asked him that question just to reassure myself and he said, well, you know, and we've had this discussion about the

sedimentation rate in AGRO versus EXAMS so it is there, but it is low.

DR. STEENHUIS: I do agree with that, it's 1.5 centimeters when you calculate that, but how about you have also this type of sediment deposition and sediment resuspension range...

DR. MCCARTY: Again, I believe those are the model, what's in the model and you would have to ask John why he chose those if it's not there.

DR. STEENHUIS: I know, I didn't have time to read it.

DR. MCCARTY: Yeah. I understand, but I mean, again, we tried to provide that information, the details, so that if you, I can't always answer the question right away but it allows you to be able to ask the question.

DR. STEENHUIS: Absolutely, you're right.

DR. HEERINGA: Dr. McCarty, thank you very much, we appreciate the presentation. We have one more scheduled public presentation and it's by Dr. Stuart Cohen who is here representing, make sure I get this right, Amvac Chemical and let Dr. Cohen introduce himself.

DR. COHEN: Hi, my name is Stuart Cohen.

I'm with Environmental Turf Services in Maryland, excuse me, I'm representing Amvac Chemical.

First of all I want to start off by commending EPA to put together this package, it's such a complex subject, it's truly interdisciplinary to put all this together and I think Keith, you've probably been with pesticides less than two years and to do all this and present it in such a format to deal with a lot of cutting edge issues, is pretty commendable, and the White paper was put together very clearly and the presentations very, as the Chairman acknowledged, were very crisp and to the point.

The outline of my very brief presentation is I want to first talk about the time to reach equilibrium for food web modeling and then I want to address the need to address... I want to discuss the need to address metabolism in food web modeling and then talk about long range transport prevention and at the end I have a summary slide.

So in the past OPP has taken the single data, the upper 90th percentile of the worst day in the thirty year period for its bioaccumulation assessments. Okay, so that's a single day number, the upper 90th percentile, and has run that through food web models. Now this approach ignores the time for aquatic biologic

systems to reach equilibrium and this issue was addressed yesterday by Drs. Mackay and Gobas and just about everybody in between.

But in the White paper OPP presents the use of sixty day average concentrations which often allow to achieve a system equilibrium. OPP I don't think has said what the policy will be, but certainly by showing these two examples, that is preferable and we agree. Sixty is better than one for very high KOW and persistent chemicals. Greater than 60 days is probably even more appropriate for the system to reach equilibrium.

In fact if you run the CABAN model and you substitute in for the, you know, using the lumped first order rate constants for a case of metabolism in gill elimination et cetera, et cetera, I think you could find that for a Koc of, KOW, log KOW of around five or so, I think you'll find that it takes longer than even a hundred days to reach equilibrium.

But the point is that OPP with this White paper and with this new initiative is definitely on the right track and we totally support that. Now the need to address metabolism. OPP typically assumes no in vivo metabolism in its bioaccumulation assessments. This is mentioned in pages 105 and 110 of

the White paper.

Now of course this is incorporated, this is an integral part of the empirical data but in terms of modeling when it came, a Km metabolism rate constant has to be assumed it's assumed generally it's nulled out, it's assumed to be zero.

Data, in EPA's defense data on Km, metabolism rate constant, is almost always lacking in the studies that it seeks. On the other hand OPP generally gets depuration rates from the standard FIFRA guideline studies. So the K depuration is a summation of the four rate constants which we've seen Arnot and Gobas mention many times, it's just one of many documents that summarize what goes into that overall lumped rate constant, so Km is metabolism, let me get my glasses on so I can see that, okay, that's much better. Okay, Kb is fecal elimination, Kg is solution through growth and K2 is loss through respiration, mostly through the gills and I guess to some extent through the skin.

So one could back out of Km because, is there a pointer here, all right, so the CABAN model that EPA is putting forth, the Arnot and Gobas model, calculates these three parameters so if you get, and there is a reasonable data base on this and certainly

Frank Gobas could speak to this imminently better than I could, but I believe there's a reasonable data base underlying the calculations behind these so if you calculate this from that, you can get a Km, so let's talk about this a little bit.

In the pesticide four example, which you just had identified to you by the previous speaker, it examines, demonstrates the pitfalls ignoring metabolism. So initially calculating the kinetics based on the KOW presented some very, very high residues of pesticide four, Pyridaben, whatever it is, in fish but then when they went back in and put in the empirical data they showed much lower residues.

Okay, so even though that chemical has a log KOW of eight or so, whatever it is, you may think that's an extreme case. Pesticide two, that is PCNB, penta-chloro-nitro-benzine and you know, we're identifying that too, that has a log KOW of 4.6 and Km is also an issue there, not as dramatic an issue as for pesticide four but it's an issue as well.

So this plot, actually Dr. Norstrom, I believe you did some work with Derek Mure in this about ten years ago. You did a similar analysis, sorry for not citing you, but what this plot does, Fitz, Fitz was the lead author? Okay. What this plot shows is that

you have, and sorry for the PDF, it's right from the journal article.

The log of the depuration half life on the Y access plotted against the log at KOW, the equation that is presented for you there, the regression equation, the r squared is reasonable and what this shows is that if you've got a chemical, let's say the half life is up here, and the KOW is down here, let's say flat KOW four, and a half life of about twenty days.

All right, that's above the line. That means that that chemical is being actually bioformed, it's incorporated into the organism but if it's below the ninety-fifth percentile, ninety-five percent confidence limit down here, then that means you've got a chemical that's being metabolized, so even though EPA struggles, as it said in the White paper it struggles, in the examples it gave, it doesn't always give high credibility to all the depuration data and the problems with the kinetics, you can see here, you can use this as a semi-quantitative approach to determine when the Km, metabolism might be significant.

Vanderlindy did something analogous with a much broader base of chemicals and he looked at many taxa, he looked at insects, he looked at mollusks, this

is a fish based data base. So this could be a useful tool when specific metabolism kinetics data are lacking or equivocal and I'm not saying you have to use the Konwick et al paper, you know, look at everything, it's not best case or worst case, it's all cases.

You know, look at perhaps some of Fitz et als past work, as Dr. Norstrom was just whispering to me. Vanderlindy et al 's model with many taxa and also I suggest, I was talking to, you know, with Dr. Gobas yesterday about the Km data base underlying the development of his model with John Arnot and you know, he was saying that it was developed by and for chemicals that largely are not metabolized.

We did a little bit of playing around with it and we feel that Km doesn't have the right place in that model. We found that the bioaccumulation with Km zeroed out was reasonable so if we put Km into it, it would even show less bioaccumulation as indicated by empirical data. So I think the robustness of the Arnot and Gobas model should be tested with more chemicals that need to be metabolized when there's good data that either can be obtained directly or at a minimum backed out.

Next topic, long range transport potential. So here I'm going to be commenting on,

well, all my comments have to do with the process but it also has to do with not only the process today.

The last couple of days the focus has been on the processes. Now as Don Mackay said yesterday, don't be conservative on the processes, be conservative on the interpretation of the results.

I'm going to talk for the first time I think about input. You know, once you get the processes right then you've got to make sure you're doing a good job on the input, so I'm going to use PCNB, pesticide two, but this goes the way that OPP is overall applying a long range transport assessment. I'm also going to suggest an alternative model.

First right off the bat we cannot reproduce the results in the White paper that you saw earlier today for pesticide two. That, we use the same exact input parameters and I'll talk about those input parameters in a minute, we use the exact same input parameters, it's around page, I think it's around 162, I think it's table 6.1 and we use the same model that EPA used and for example, for transfer efficiency, EPA said that PCNB pesticide two transfer efficiency is 457 percent. I got 201 percent, I didn't change anything.

Earlier this morning you showed that a POV and overall persistence half life was 599 days, I

got 312 days without changing anything so there's some things, there's a little bit of a disconnect there and I don't know if that's just a simple matter of typos, you know, input error or if there's something more fundamental, but that needs to be looked at.

Now an alternative, so the screening level model, and OPP didn't present this as any more or less than it is. It's an OECD decision tool that is used for comparing chemicals. I'd like to propose that OPP consider something.

I'm not endorsing Vonya's model, I'm simply saying it goes to quantitation, because you can't have risk assessment without quantitation and just comparing one chemical against another is great for triage, it's great for priority setting, maybe setting priorities for monitoring if you're expending monitoring resources.

And you don't have, I'm going to talk a little bit about Vonya's model, global pop, but and you don't have to be wedded to this, what I'm saying is start considering at least a semi-quantitative approach. So Frank Vonya set forth the global pop model, initially in 2003 publication I think in ES&T, Environmental Science and Technology. Vonya and Mackay talked about the processes that go into this in 1999.

Fender Adele is mentioned once or twice in the White paper and again today by Dr. Kan as an overall comprehensive comparison of the models.

Vonya's global pop model estimates arctic contamination potential based on pesticide e-fate chemistry. It is a zonally averaged multi-media model designed for the global fate of pops, that's what it's designed for. So why do we care about arctic? Well, arctic is, obviously there's good ecological reasons to care about arctic, but also it's kind of like the ultimate long range transport, so I'm not endorsing it, I'm saying just evaluate it, consider it, and consider possibly other semi-quantitative or quantitative approaches.

Now for pesticide two globe pop predicts much less than one tenth of one percent of globally emitted PCMB when presented in arctic surface media over ten year period of emission and equilibration. Yet OPP, now I'm coming back to the comparison tool....yet OPP....oh, and to put that into perspective if you look at Vonya's work I would believe that for the ACP 10 the emitted fraction for a ten year period, a typical problem chemical would be in a couple percent range, okay.

So we're talking about PCNB would be

about two orders of magnitude lower than the more problematic chemicals. I didn't run lindane through the process but you know, I think Dr. Bidleman's published work in this area. You know, people know that lindane is out there and around, certainly in the DDT analogs and PCNB would be about two orders of magnitude lower than those.

Yet, when OPP presented its application of an OECD tool, it said that PCNB has much greater long range transport potential than DDT, aldrin and dieldrin, much greater. So when I saw that I said, huh, how did this happen? 'Cause I'm taking a common sense thirty thousand foot approach, how's that possible?

So it's an issue of apples and oranges so what OPP did was in, I'm not sure when Ron Parker gave his presentation yesterday, I'm not sure what came across in the specific input modeling guidance that OPP typically uses the upper 90th percentile for say the aerobic zone metabolism half life as an example, and that's been discussed and evaluated and people know that going in, that's been aired out, and it is implemented science policy.

For runoff modeling, for storm water runoff modeling, use as an example the upper 90th percentile of the aerobic zone metabolism half life.

Similar numbers go into aerobic aquatic metabolism but this is something different. This is long range transport potential assessment and all the papers I've seen and if you look at the actual OECD tool, what they talk about are reasonable numbers not the upper 90th percentile.

In fact I said in the beginning is it best case or the worst case, it should be all cases and I steal that line from a, I think his name was Dennis Kozlowski, he was a Dow scientist from the 1980s, a very top notch scientist, I may have his name wrong, but anyway so what the OECD tool does it presents, when it presents its reference chemicals. It doesn't present one number for water half life for aldrin, or one number for water half life for DDT. It presents a series of numbers. In fact it doesn't even present it for DDT. The reference chemicals it says it's supposed to be used wasn't used by OPP in its presentation or in its White paper.

The referenced chemicals for comparing something, for determining whether it's a PBT, are carbon tetrachloride, PCB analogs, hexachlorobenzine and lindane and its analog. Those weren't referred to, what was referred to are chemicals like aldrin isn't found out there but dieldrin is, there's an interesting

version. DDT is usually not found but DDE is and what the OECD tool says is you should use a range of input parameters and in fact it has a Monte Carlo module that you can run and that's the example. You do best case or do worst case, you do all cases.

So our concern is not with the OECD tool. Our concern is the way it was presented, applied in this White paper and I think the OECD tool could be very valuable for setting priorities and triage et cetera, but I would recommend that OPP also start going towards a semi-quantitative or quantitative risk assessment approach as well.

Now as Dr. Bidleman said earlier we also have concerns that in all of these models, in all these models, they don't consider, first of all they're not necessarily designed for pesticides, but they don't consider the pesticide application method, if it was soil incorporation versus air blasts in the orchard. It seems to me you have a lot different initial percent of emissions being kicked off into the atmosphere if you compare those two methods.

Also the volume, Dr. Maddalena I think it was, was questioning whether the volume used in here, production volume, there's a big difference in chemicals that only have a couple million pounds a year

globally versus those that are tens or hundreds of millions of pounds per year, so I'd recommend those refinements to this process.

Then picking, then picking up PCNB in reverse, it was said in today, earlier today's presentation that the presence of, there was two pesticides so they listed pesticide two and I don't remember whatever else, it was well defined. That's hardly the case, it couldn't be less the case. In the White paper it says one of the most common pollutants found in arctic snow was a metabolite of pesticide two slash PCNB.

This first appeared in an EPA document I think it was 2004. We immediately corrected EPA, EPA acknowledged in writing that this is wrong, acknowledged it and verbally and it's still appearing. A metabolite of PCNB has never been found in arctic snow. It was a table that had an abbreviation for an acronym for a different metabolite that's unrelated to PCNB. All you had to do was look at the footnote of the table or look at the text and you'd see it was a different chemical.

So not in arctic snow. Also it says in the atmosphere of areas in which pesticide was not used, I believe it's talking about a Saskatchewan study

maybe in China, I'm not sure but in the study, the PCNB target crops were in the area, sod, grass seed, grass for seed and cabbage, so it's not known that PCNB wasn't used in that area and in fact the way you use some of these models, there was a big assumption it's in the vapor phase.

In fact that was stated here and there was one study where the sampling points were a couple of miles away from where PCNB was used and the only, the only sample that had it was with a puff plug, a polyurethane phone plug, so it was trapping in this case because they also did air, but they were doing particular traps. The only PCNB residue was in one sample in a particulate filter.

So my point, for VAF assessment one day, no, many days, yes. OPP's initial examples are sixty days, that's a great start. Expand use of Km and evaluate the Arnot and Gobas model regarding this parameter. Consider the use of log Koa. I did not talk about this because this is human bioaccumulation, this is supposed to be an eco assessment meeting, but we are top predators and that's kind of like the ultimate in bioaccumulation.

Caruso et al, what they did was they studied mothers who were pregnant and following the

kids through about four longitudinal sites. They were about four years old or something like that, and they determined that chemicals with a log Koa greater than about eight was a concern for the study population. I know PCNB has a log Koa of about 6.3 or so. Anyway, log Koa was discussed earlier, I can't remember who, and the question from the panel and the response was it goes to clearance, the ability to clear out the chemical.

So I'm just chiming in to consider the use of log Koa either in human bioaccumulation potential or in mammalian terrestrial crafts. Evaluate global pop for potential use. Again, this is not an endorsement, it's a suggestion that you evaluate it or something analogous.

Integrate consideration for production by an application method into the LRTP assessments. Speaks for itself. And finally use appropriate model input, this meeting is focused on the processes and that's good, in a sediment burial et cetera, et cetera, but once you get a really good model, the output is only as good as the input. So you've got some references and we've got hard copies of that too. And I thank you for your time and I'd be happy to answer any questions.

DR. HEERINGA: Thank you very much, Dr. Cohen, and Dr. Simonich has an opening question.

DR. SIMONICH: Thank you for your presentation, and I appreciate your perspective. So am I to understand that you didn't run PDT eldrin or endron in global pop, and as you've shown, unfortunately, sometimes the use of models in the outputs might vary user to user so you did not run those, is that correct?

DR. COHEN: We did not run dieldrin, eldrin, endrin in global pop, no, but what Vonya did was run the perfect, they call them perfectly persistent chemicals, PPCs and to the extent that they, in global pop the PPCs come out at a few percent that the art of contamination potential for ten years that a few percent like between one and four percent of chemicals that would be in the range of not aldrin but probably dieldrin and probably DDE, that a few percent of what's admitted into the environment would...could then be deposited on an arctic surface and so that's in his paper.

DR. SIMONICH: Yeah, I'm aware of that paper.

DR. COHEN: I figured you were, all the research you've done.

DR. SIMONICH: But in fairness for direct comparison to EPA, they model DDT, eldrin and endrin.

DR. COHEN: Oh, now that's in the OECD tool.

DR. SIMONICH: Yes, I understand.

DR. COHEN: Oh, okay.

DR. SIMONICH: But I'm wondering where PCNB falls out compared to DDT, eldrin and endrin in global pop.

DR. COHEN: Oh, it was .0...oh, I ran that...oh, I'm sorry, we ran PCNB and it was .01 percent to .03 percent depending on the input parameters and no, did not, did not run that, I just, because Vonya had the perfectly persistent chemicals in there.

DR. SIMONICH: Which may not be DDT, aldrin and endrin.

DR. COHEN: Would be, right, not, certainly not, certainly not aldrin.

DR. SIMONICH So, also another question regarding your input parameters for PCNB and your critique of EPA's selection of parameters, what was the strategy you used to select your parameters?

DR. COHEN: I used their parameters,

well, to try to reproduce the work, I used the exact same parameters. Then and as an example of the transfer efficiency, let's see, what did I have the slide there...almost there, there we go, no, oh I know in the POV, the overall persistence, EPA reported today and I think in the White paper 599 days. When I used their input...

DR. SIMONICH: Just let me clarify, I mean with regard to global pops, what parameters did you select, it was the same as EPA's that was used....

DR. COHEN: Oh, oh, with global pop. When we ran what we think is reasonable input we came up with .01 percent but when we ran EPA's, definitely the aerobic soil metabolism half life that EPA first used for runoff modeling and I don't remember what we used for water. I think for water we used the same. We came up with .03 percent.

DR. SIMONICH: And how did you choose your reasonable values?

DR. COHEN: Based on, I mean this would take like two beers to explain the whole thing, but there has been a series of discussions back and forth, there is...

DR. HEERINGA: It sounds more reasonable with time.

DR. COHEN: Yes, there's, there's dispute...I mean this is a side issue here, but there's dispute about the appropriate aerobic flow of metabolism half life because some volatiles were lost during the study and EPA put them back in. When EPA put them back in, the half life went way back, way up and the R squared went way down so when you just let the volatile scope that we lost at the beginning, the R squared was very good.

The first order rate constant was very good and the half lives were short and there's been discussions back and forth and then there was a couple of studies and then so we took the mean, I don't remember if we took the mean or the worst one but then EPA takes the two, uses the C and T value and then derives the upper 90th percentile, separate issue.

It, both sides respectfully disagree but that's a separate issue so we ran it both ways, we ran global pop with what we thought was reasonable and with what EPA does in its runoff model.

DR. SIMONICH: Okay, so some of the differences between your discussion of the OECD tools and global pop, is that we're not doing an apple to apple, orange, orange comparison because we're not benchmarking the PCNB to DDT, aldrin, endrin. That

looks like was done in the OECD tool. We're also, the selection of the input parameters are also very different. I appreciate your points but...there's various aspects that don't make it a direct comparison.

DR. COHEN: Yeah, it's, that's right, so what we were trying...what we did, what OPP did was took the worst case examples of PCNB and like the DDT half life that...I'm sorry, I didn't mention this. The DDT half-life that was put in there, that is lower than I think USDA people would be aware of Don Walkups pesticide properties data base and Janice Chambers, you may even know about that.

There's a standard kind of like Bible for field half lives. The number that OPP used for the DDT soil half life was significantly lower than the number in the pesticide property data base, that half life. So here EPA took worst case for PCNB, compared it against reasonable or best case for DDT and that's the....I was simply trying to use that as an example of the input misrepresenting the results.

DR. HEERINGA: Other questions from the panel? Well, thank you very much, Dr. Cohen. Since there have been no other requests for public comment, I think we're ready to move on to the charge questions but before we do that I'd like to turn to the panel,

we've had a large number of presentations and we've had a chance to ask questions but maybe inadequate time or you formulated a question afterward, I'd just like to open the floor at this point to panel members to ask any other questions and I presume Dr. Brady, we could call up the relevant staff scientists to answer these. Are there any outstanding questions on the White paper, on the presentation material? Yes, Dr. Doucette? Maybe you could identify the topic area to us.

DR. DOUCETTE: The topic area would be degradation data that's submitted and I'm not sure who would be best to answer that but I've looked at the standard method and I'm still, I guess I would like to get a feel for when you receive a data package that looks at degradation and metabolism, what actually do you see and how comparable is that data from one study to another, how much flexibility is there in the data that's submitted, required to be submitted for the degradation part of it?

DR. HETRICK: I'm going to give you the short answer on this okay, number one, just to set the stage a little bit. When we see a....the registrants are normally just requested to submit one aerobic soil metabolism study to support a registration, one.

That doesn't give you a lot of leeway to

look at variations from one study to the other. Now in all fairness, more recently we've been getting more studies submitted to support registrations and that does help us to look at variations from soil to soil and across different matrices so you know, it can vary quite a bit. I've seen studies where we have half lives that range from 60 days up to 500 days and the tendency is that normally one would throw out that 500 days as an outlier.

I personally don't see that, I don't think that's appropriate to do that, to throw data out just for the sake of, it just doesn't fit the norm but there's that tendency to want to do that.

DR. DOUCETTE: And as a follow up, how about in terms of the transformation products that are identified or aren't identified?

DR. HETRICK: The, normally when you start looking amongst the different soils, I'm speaking just on a soils basis, normally you see pretty consistently the profiles look fairly consistent from soil to soil.

With the caveat that if there's a pH dependence type of, some type of process going on there that may create a unique soil product or hydrolysis product. That might differ with, according to the pH

of the soil so the point is that normally for, from a biological perspective the metabolites are generally fairly consistent from soil to soil.

DR. DOUCETTE: In what level do they...I mean there was the ten percent level is I mean do you see things ranging down to percent levels, I mean it depends...

DR. HETRICK: Yes, yes, and we can track that because we have radio labeled studies and in thank God for that because if we didn't have that capability we probably would miss quite a bit.

DR. DOUCETTE: And of those, and in the radio labeled studies that just gives you an indication that there is label there, are the specific metabolic products always identified?

DR. HETRICK: Normally the registrants go to great efforts to identify any residues that they extract and you know, I've got to give them kudos for they make a noble attempt to try to identify any residues that are extracted from soil.

DR. DOUCETTE: Thank you.

DR. HEERINGA: Other questions from the panel? We'll have the opportunity if you require clarification during the charge questions we can do that, but I prefer that when we enter the charge

questions that we have most of these clarifications out of the way. Yes, Dr. Delorme?

DR. DELORME: Jim, maybe you can answer the question. I was just looking for clarification on what, when you're looking at a whole system half life for an aquatic system, what impact does that have on your modeling?

DR. HETRICK: Well, actually when you start looking at the PRZM/EXAMS model when we, I'm going to break this down between PRZM and EXAMS because I think there's an important differentiation here. The model's fairly sensitive to the aerobic aquatic metabolism study, or half life, and what, the way we use the data in this case for our modeling purposes is we use the total system half life from the aerobic aquatic metabolism studies to represent the water phase degradation.

For the sediment phase degradation, we use the anaerobic aquatic metabolism total system half life and so therefore, we're not biasing that process either way by doing that. For the...and the degradation half life, the sensitivity of the PRZM/EXAMS modeling as far as the PRZM, the aerobic soil metabolism half life, is we make the assumption that again we're using a lump degradation half life

that represents the comparable metabolism in soil solution as well as what's on the soil colloid and that when we go back and do a sensitivity analysis.

We only...the only time you really see major differences in the output as far as the EECs in the pond, are only really have half lives that are fairly rapid on the orders of probably ten days and if we go from ten to twenty days you see some pretty big differences in your EECs but if you were to go from 100 days to 400 days, you might not see much difference.

Does that answer your questions?

DR. HEERINGA: Dr. Doucette?

DR. DOUCETTE: You mentioned ionizable chemicals, I've got that down on my list. Can you give me a feel for the percentage of ionizable chemicals or chemicals that would be ionized in environmentally relevant pHs that you deal with? Would it be 30 or half?

DR. HETRICK: No, I would say that they are probably about thirty percent.

DR. DOUCETTE: Thirty percent?

DR. HETRICK: Yeah, they're basically neutral organics that we're dealing with.

DR. DOUCETTE: Okay, for the ionizable compounds then how do you deal with Koc and sorption in

modeling especially those that are negative?

DR. HETRICK: That's a good question. We don't go through and do any speciation so we, that's generally not included in our modeling so we don't look at what's ionized versus what's not ionized. You know, so we don't really go through and do that speciation in the modeling.

As far as the Koc issue is concerned, we just do our Koc analysis on a range of soil types, making sure one of those soil types is actually a fairly low organic matter soil with a sand texture, either loamy sand or sand texture.

DR. DOUCETTE: So in a particular scenario if I've got an ionizable chemical that is let's say negatively charged at most environmentally relevant pHs, and I do that on a low organic carbon soil, probably isn't going to make any difference, it's probably not going to sorb either way?

DR. HETRICK: That's, that's correct.

DR. DOUCETTE: And so I still calculate a Koc based on that?

DR. HETRICK: Well, we have the capability of actually using a Kd or a Koc depending on what that relationship looks like. The first thing we normally do before we go through the Koc model,

partitioning model is we look to see if there's a relationship, a correlation between organic carbon and Kd and we don't go forward with that. If there is another relationship, we use a Kd type of estimate.

DR. DOUCETTE: And you'll use that all the way through PRZM and...

DR. HETRICK: Right, right.

DR. DOUCETTE: Thank you.

DR. HEERINGA: Dr. Gan?

DR. GAN: Just a little bit more clarification on question two, the persistence question. You just mentioned that for the water column half life, the members coming from the aerobic whole system half life for sediment it's going to be from the anaerobic...

DR. HETRICK: That's correct.

DR. GAN: So the whole system of half life approach will apply to both sediment and water?

DR. HETRICK : Right, right.

DR. GAN: Thank you.

DR. HEERINGA: Dr. Steenhuis?

DR. STEENHUIS: Can I ask a question about the AGRO model?

DR HEERINGA: Certainly.

DR. STEENHUIS: In order to evaluate the

model and which one is better, is it important to know what how the input parameters are determined and especially the sediment depth, the benthic zone depth and the resuspension rate? It seems that the results of these simulations are extremely sensitive to it, especially with regard to what we heard about pesticide four.

DR. HEERINGA: This goes back to your specific question to Dr. McCarty but we're talking now about the AGRO model in general?

DR STEENHUIS: The AGRO model how the input parameters are determined?

DR. HEERINGA: Keith, are you able to address that or if you would like to....

MR. SAPPINGTON: I've run the AGRO model and done some sensitivity analysis with it, but I think this question would be more appropriately directed to then developers of the model.

In terms of sensitivity it is, the runs that I've done it's sensitive to the rates of the deposition and the burial and resuspension and the other parameters that I mentioned in my presentation yesterday. I did not vary sediment depth so I don't know how sensitive it is as to the depth of the sediment. Organic carbon compound is quite sensitive

to that and what other parameters?

DR. STEENHUIS: Actually I mean how do you set those parameters? Do you know that? I mean they need to be set at some point and how will we make a choice, it seems one centimeter depth for the benthic zone or five centimeters like is used in EXAMS.

DR. HETRICK: I guess the question I have is are the deposition rates, resuspension rates in AGRO hard wired or can you change those?

MR. SAPPINGTON: You can definitely change those rates, and in fact the version that I used had constant rates and my understanding of the newer version uses PRZM to determine in some fashion those rates, so you would have a temporal variability as well as regional to that.

But in terms of defining the sediment layer at least in the comparisons that we did, we simply equated them to what we normally did in EXAMS so the information I was showing you about yesterday about EXAMS versus AGRO results, we kept as many of the parameters the same as we could including the sediment data.

DR. HEERINGA: Dr. Oris?

DR. ORIS: Yes, I was wondering if the EPA would like to respond to Dr. Cohen's comments on

input values or in the discrepancy between modeling that was done?

DR. RUHMAN: I have to check it myself to know what he's talking about, but these parameters that we have dealt with, we exactly use the same parameters the way we use for water modeling, we do not deviate from the water modeling but that is up to you and the panel how we, you know, proceed, you know, to use these parameters.

That was one of our questions but we use, this is for example purposes we did use exactly the same process of 90th percentile. We did some of the time we have five, six, you know, data points and how you use, you know, like Jim was talking about like we had a fifty and five hundred, you know, what do you use then? Do you use the average value or do you use the 90th percentile, and that's where we stayed with our normal procedures for the 90th percentile.

DR. HEERINGA: Dr. Oris, do you think that rectifying this discrepancy is important for your interpretation and response to the charge questions?

DR. ORIS: Well, I think that it would be helpful for some of the long range transport issues for sure, because there was a huge discrepancy there so, or, and comparison to values that came up for DDT

and dieldrin and eldrin I think that was the other thing that just seemed in the idea of comparing apples to apples and oranges to oranges I think. And I also wanted the EPA to have a chance to actually respond to that if they wanted to.

DR. HEERINGA: Sure.

DR. RUHMAN: Especially those, the values of the reference chemicals is already in the tool. I did not invent those. I just used the values from the tool.

DR. HEERINGA: Dr. Brady and Dr. Gan, I wonder if on this issue if you wanted to give it some thought, maybe even compare notes with Dr. Cohen if it is a matter of rectifying a discrepancy that's important. If it required a little time maybe to come back to us tomorrow or so if, you know, I don't want to put you on the spot with that but apparently I think it does come into play here.

DR. BRADY: Yeah, I think we'd like a little time...

DR. HEERINGA: Sure.

DR. BRADY: To do that and come back, thank you.

DR. HEERINGA: Yes, Keith Sappington.

MR. SAPPINGTON: I was just handed a

presentation of Drs. Mackay and Gobas yesterday where there was apparently a slide presented that compared the different outputs from different sediments.

DR. HEERINGA: Sediment depths, yes, I remember that chart, about five different sediment depths. Just a comment I think and going back to my interchange with Dr. Maddalena before, too. I don't want to cut people off with regard to these specific case studies, but my concern is that we are not reviewing risk assessments for these two chemicals, these case studies were inserted to demonstrate capabilities of the current system, the process of the current system.

And I recognize that it's very valuable to understand in the context of a specific chemical which you know much more about than certainly myself and many other people here, that they may inform the properties, they may inform the weaknesses of the approaches.

But I think we want to sort of back away a little bit from treating it as two known and two unknown specific risk assessments and that was my only concern there, so again, I don't want to restrict the panel's interpretation of that data but we want to keep it in the context of the four case studies rather than

four independent risk assessments and their validity or lack of validity at this point. It's just a comment. Again, you should feel free to make comments if you want to on each of those studies. I didn't mean to interfere that way. Any other questions? Clarification?

What I would suggest that we do is that we take a fifteen minute break now before we begin the process of responding to the charge questions and we're right on schedule, if not a little ahead of schedule, and we'll try to entertain charge questions number one and two after our break. So let's plan to reconvene at 3:00 p.m.

(WHEREUPON, a brief recess was taken.)

DR. HEERINGA: Okay, if we could move to get underway, please.

Welcome back, everyone, to the second half of our second day afternoon session, the FIFRA SAP meeting. At this point in the process we have, we're about to begin the panel's response to the charge questions that have been posed to us. Dr. Hickie, do you have....okay, we can, if any additional questions or clarification do come up as part of the discussion, we'll certainly permit it in this particular session so at this point in time I suggest that we move on to

charge question number one, and Don, can you read it into the record, please?

DR. BRADY: Absolutely. This is charge question one addressing exposure to parent and degradation products. When assessing the potential ecological risks of proposed pesticide uses, the Agency is charged with considering both parent compounds and any degradation products of concern.

In several of the case studies presented in the White paper, the Agency has illustrated three approaches for assessing the PBT characteristics and exposure to parent and degradation products.

When parent and degradates are considered sufficiently similar in their environmental base and toxicological properties and when these properties were unknown for the degradates the Agency has used the total residue method, i.e. the Agency model that combined parent and degradate using a common set of environmental fade and toxicological data.

In situations where the environmental fade and toxicological properties of the parent and degradate are available and considered sufficiently dissimilar, the Agency has modeled the environmental fate separately using the residue summation or formation degradation kinetics methods, i.e. modeling

individual residues from the parent and degradation products.

Please comment on the Agency's characterization of the strengths and limitations of these methods and the conditions under which each method should be applied. To what extent does the Agency's use of the total residue and individual residue methods reflect the current state of the science for assessing exposure to combined parent and degradate compounds?

DR. HEERINGA: I think there is one additional bullet.

DR. BRADY: Okay, please identify any methods the staff would recommend for addressing combined exposure to parent and degradate compounds based on the data typically available for pesticide ecological risk assessments as described in the White paper.

DR. HEERINGA: Dr. Doucette is our lead discussant and I'll leave it to him as to whether to I think address these three questions together or separately in order?

DR. DOUCETTE: We, our group of evaluators met last night and I was peer pressured into, I put together a couple of Power Point slides

with some discussion items to follow the lead, so it's on that machine and I'm happy to go over there and drive or sit here if somebody's willing...

DR. HEERINGA: I think Dr. Hetrick can drive for you.

DR. DOUCETTE: It's just in the folder under panel and with my name Doucette and there's only four or five and it's just to give us some discussion.

DR. HEERINGA: Give him a moment to bring them up then.

DR. DOUCETTE: Thank you, okay, we just went through reading the question and the discussants are listed there so you can go ahead to the first slide and I thought it was helpful for me to make sure that I understood the approach and I tried to summarize that as best I could. The total residue assumes that the, the total residue approach assumes that the parent and degradation products are equal in terms of fate and toxicity, and I'm paraphrasing and it is the approach that requires the least amount of data and at least in what was shown on the examples it's the most conservative.

The individual residue methods are divided in two categories, the residue summation or RS method and both parent and degradation product fate and

toxicity data are required, but in this particular case we know that the degradation products are there but we consider both the parent and degradation products applied at the same time and that eliminates the need for some of the kinetic transformation data through information that would be needed in the next approach and it through the examples, the effective environmental concentrations predicted were closer to the, I'm sorry, that should read FD method than the TR method.

So the final method is the formation degradation kinetics method which ideally should be the most in depth approach to solving this problem and again both parent and degradation product fates and toxicity data are required and it looks at the formation of the degradation products as the parent degrades, and hopefully I summarized that okay.

If I didn't, this would be a good time to correct me.

DR. RUHMAN: I think so, except that for the formation and decline methods you need to also to look at the transformation pathway, you need to have the transformation pathways, and sometimes the transformation pathway goes from one degradate to another. One degradate goes to the other and...

DR. DOUCETTE: Right, right, and I'm trying to paraphrase that, right, okay, thank you.

DR. RUHMAN: More complex. But what you said there is okay.

DR. DOUCETTE: Next slide, please. And hopefully this is just repeating the first question. It looks like my selection of fonts did not work out too well and this is our response, and again as we sat together as a group we felt that this was a tough question because really we thought that the White paper really answered the question, and by illustration, and we felt that the strengths and the limitations of the approach was really pretty well defined by the Agency in the White paper and we agreed that FD was more realistic than RS and TR and I think based on the examples it did seem that the TR approach, total residue approach, was most conservative but maybe less realistic.

And we also agreed that the data availability really does drive the choice and that's just the way it is and there was a question and one of the discussants brought up the point, well, why can't sufficient data always be obtained to allow the most realistic approach, and that's, you know, it's just a broad open question realizing that you don't get all

the data but you know, could more data be required in order to do that and we feel that that, the cost associated with obtaining the data that would allow the most realistic scenario is worth the cost and maybe that is on a chemical to chemical basis.

Okay, and I'm just going to try to go through these very quickly and then there's, I've got some discussion points that hopefully the panel can, the rest of the panel can chime in. This is the second question, how does this approach really reflect the current state of the science for both the parent and degradate?

We felt in terms of the transport part of it, treating them either way was probably appropriate since low concentrations of compounds generally, and certainly there are some exceptions to that, behave independently in terms of their transport. There was a lack of information on the approach used to, at least we didn't...we weren't able to get that information from the White paper on the approach used to assess the toxicity slash, you know, biological impacts of the mixture of exposure and I think that's an interesting and appropriate area to look at is, how do these things interact as a mixture in both those?

And then finally I don't know if that

last one got cut off, I've got it on my screen, it seems to show up fine.

Let me see here, response one, response two and down there there's a couple of bullets it just says the mixtures and then in order to do that there also needs to be consideration, and this was brought up a couple of times, in order to look at how similar the degradate is to the parent compound especially in terms of toxicity is getting back to that mode of action question.

And the process at least in my opinion and in general the members of our little group, maybe it isn't as transparent as it could be in other words, how do you determine how similar a degradate is to the parent and do you actually look at and based on the discussions I believe you do, it just was not apparent in the White paper, how do you determine mode of action and the difference between parent and degradate?

Okay, and then the third question is there and we can just read that and really our response is given the limitations sometimes associated with the availability of the data or at least checking the data, we were wondering if you've considered using some estimation tools like Cannonball which is a really interesting program and my understanding is since I was

on the epi suite review panel about a year ago is one that they are either in the process of strongly considering incorporating into the epi suite program and it predicts metabolic pathways, intermediates and half lives and again, it gives a range of probabilities, it also, I have some information there on the properties, of predicting the properties of those intermediates and so it may be a potential tool when data is lacking or at least in pre-screening some of these chemicals where you're waiting for data, you can start looking ahead of time, being proactive and looking at compounds that may actually be a problem or have problematic degradates.

Again, my understanding is that it is going to be combined eventually, at least most of it with epi suite, which is something that's available to everyone, and this one should also be. One of the discussants, I think it was Dan, mentioned that if metabolic data was not available from the traditional studies you might be able to use in vitro transformation assays that, you know, typically are conducted for the identification of persistent metabolites in human studies and I think as a group we felt fairly strongly that this whole idea of how things behave as a mixture is very important in an approach to

somehow incorporate that into the risk assessment is important.

And then finally there's some just I guess broader discussion points that I just threw out there because I didn't know how to put them in a category and again, it looks fine on my screen, I apologize for the font choice. And I think this was discussed several times in different charge areas but the idea of being able to improve and evaluate the current approach just by using representative slash benchmark chemicals where considerable data exists is a useful exercise and we felt as a group that it was. The impact of parality or stereo-selective processes, important consideration in terms of toxicity and that wasn't directly alluded to, or it was alluded to but not directly addressed.

I wasn't clear and this is my question, not necessarily one of the group, I noticed in the list of information that was required, there is information on the photochemical degradation products and I assume that those are taken through the whole process in terms of their fate. I didn't see anything on hydrolysis products. Are they also transferred through and I may have just missed that. Do you get information on specific hydrolysis products that are formed and follow

those then through the process?

DR. HETRICK: Yes, but normally, yes, we do.

DR. DOUCETTE: So my ignorance then or my misinterpretation. The other thing that was brought up briefly was the, you know and actually this has come up quite a bit in terms of pesticide formulations and its potential impact on the solubility and those sorts of things but being on several panels, several of us mentioned the fact that there's some micro-encapsulation or nano-particle distribution methods that are being discussed and how that might affect fate and transport and availability is something that may be coming down the road.

One of the themes that I have that kind of was recurrent through several of these was again the transparency, and there was a lot of discussion on assumptions and inputs associated with a lot of the models and I don't, based on the discussions that we heard from all the different staff members, I think there's a lot of discussion internally, but it didn't necessarily come across until everyone discussed it and it didn't seem to be clarified in the White paper and I think that would make things easier.

Estimated versus model versus measured

in range we had an example that one of the public members gave about the sensitivity of the input parameters, and I got the impression sometimes that measured values are nice, but if they didn't seem to fit a conceptual model then sometimes we used the model values and correct me if I'm wrong there but sometimes we flip back and forth between measured input and estimated input even though there might be measured values available.

For some reason we decided that the measured value was not necessarily reflective of what was going on, and I don't know that that's necessarily wrong but it you know, coming from a measurer I tend to prefer measured values and if the measured values deviate a lot then I think it's appropriate to look at ranges rather than, you know, selectively choosing one value or another.

Several brought up just the general concept of uncertainty which is really associated with the model output and the model input, and I think what we in general did a lot of times on our own was look at and tried to get a feel for sensitivity analysis, what properties or processes or inputs really drive the ultimate assessment and allow us to focus on those particular areas.

Do we look at a best case or a worst case of range, all those things were discussed, and it's more just topics and then I had mentioned, really hadn't discussed ionizable chemicals and how you actually deal with those in terms of pH and whether or not they're ionized or neutral and some of those I just, I admit I threw in on my own without discussing with the rest of the panel members, so those are discussion points.

DR. HEERINGA: Thank you very much, Dr. Doucette. I'd like to, I know that you had considerable input from associate discussants in formulating this, but I'd like to open it up now to the associate discussants. I'll just go through them in order, Dr. Gan, do you have additional comments that you'd like to add?

Dr. Donnelly.

DR. DONNELLY: I think that pretty well summarizes it. The only comment I have is...you know it is important to recognize that there are examples where the metabolizing degradation are both more soluble and more toxic than the parent compound. And I think the 3 methods that are available...the FD is probably the most accurate, but in a lot of situations, given the amount of data that can be available, almost

by default you end up using PR. I think that was pretty much covered in the discussion when we had that.

DR. HEERINGA: Danny? Tammo?

DR. STEENHUIS: This is not my speciality, but I do remember where Agent Orange, about chemical compounds which were much more toxic that the rest, and I think we should watch out for those.

DR. HEERINGA: That's reinforcing Dr. Donnelly's point. Other members of the panel who would like to weigh in on this particular question? Dr. Delorme.

DR. DELORME: As per usual, those of you that know me know I can't keep my mouth shut on these things. Just couple of points, and again, I come at this from a risk assessment perspective, not necessarily an expertise. I agree that the FD is probably the gold standard of what you want to take. I also want to mention that it's probably not only optimal to PPT's, you can use it for other chemicals as well.

I think you have to be careful, or we have to be careful, because obviously we do this kind of modeling too up in Canada, the same kinds of models. When you have differential transformation processes dominating in soil and water you could get formation of

different degradates in the soil or in the water. So when considering the TR's, the RS approach and subsequent modeling you've got to be careful.

If you have something that's primarily formed in water you shouldn't be putting that on the field, okay? You have to find a way of putting it just in the water. And we've actually run into a couple of cases where we've had to find a workaround. You have to be, you know, cognizant of that.

With respect to using the formation decline method, I agree with KT. It's going to be a challenge, especially when you get into the re-eval, where you may not have the database. And one thing you might want to do is compare results with a range of chemicals, get an idea of the variability of the uncertainty, so that when you are doing that you can characterize that. I think it's important to do.

But I think you probably have enough chemicals from recent stuff you can take a look across the race chem sheet and see what the implications are, see what kind of variability you've got, okay. So mine your data a little bit. The other thing is just a minor point. When using residue summation, you might want to consider a temporal offset when you are summing things up. So you can either do that in your

application files for PRZM EXAMs, or you can just offset it when you go do the additions.

DR. HEERINGA: Thank you very much. Yes, Dr. Norstrom.

DR. NORSTROM: I'm trying to compose a coherent statement here, I was going to read it. My suspicion is that usefulness of the GR method may be moot, because I suspect we don't often run into chemicals where the degradates have similar properties and toxicity to pesticides.

Most degradation has moved chemicals less to more polar compounds which can be eliminated or mineralized. This is especially true of PV compounds. Given that toxicity and mode of action are likely to change with functional group alteration, addativity can't be assumed in these cases anyway. That being said, if the properties, including toxic actions are the same that reasonably are supposed to additive, then the toxic equivalents approach could be taken.

Also if the degradates are not toxic, or if they have a very low bio-accumulation potential maybe they can just be ignored altogether. But I think I don't know, any idea what proportion of the pesticides you're dealing with actually have degradates that are very similar property to the starting

compound. I suspect pesticide 1, which I think I'd guess is probably an unusual case. Is that safe to say?

So I really honestly thing that it is almost a moot question. Most of the degradates are not going to be similar enough that you can actually use that approach, but maybe you can ignore them. I'm just guessing, that's just generic.

DR. HEERINGA: We'll certainly include your point of view in. Other comments from the panel in response to this particular question? Randy.

DR. MADDALENA: I was kind of hoping other people would talk so I would have a chance to formulate this a little more in my mind.

What I would like to see in this question is somewhat of a microcosm of what you are dealing with in the whole process. This is a choice between 3 different models, and varying from relatively simple to relatively complex. Easy to use, difficult to use, fairly easy to interpret, but not very relevant to the environment, sometimes hard to interpret, but you get my point.

And this process represents the bigger picture of what you are working to now, which is developing an overarching model on how to deal with OPS

or these PBTs. So what I would suggest is that you follow a pretty well developed pathway to do this, and counsel, the DBA counsel on regulatory, environmental models recently put a document through the Science Advisory Board that talks about this process in great detail.

Transparency, sensitivity analysis, uncertainty analysis, things that showed up on Dr. Gobas's final slide, as far as how to use the model once you develop the model. So I would strongly recommend that that be looked at, and that would save a lot of words in our report on how to use, how to go about this process. From these small choices or focus choices on specific pathways to the whole process of building a model and putting in the applications.

DR. HEERINGA: Thank you Randy. Other comments, with respect to charge question number 1? I'd like to turn to Dr. Grady to see if your team feels that this question has been addressed, or whether there are any clarifications or-

DR. HETRICK: I have one clarification. You mentioned Cataball and the ability to estimate half life. We have Cataball, we've played with Cataball. One of the issues that we have with it is that it's really based on a...you have to have populate it with

the appropriate data to be able to make those estimations, that's number 1. Number 2 is that I wasn't aware of the fact that you could actually estimate half lives out of it. If I'm mistaken on that I would appreciate some clarification on that.

DR. DOUCETTE: A point of clarification. I guess in terms of half life, it's something we've...that was discussed that it was going to be added. I'm not sure it's actually there yet. Right now it's just looking at biological oxygen demand and that's it. But there may be a way to actually use that information to give relative half life I think, based on an aerobic scenario. So I should have made that more clear.

DR. HETRICK: I want to just make sure I was clear on that.

DR. DOUCETTE: I have a question for you. What do you feel about the utility of a tool like that in a regulatory setting?

DR. HETRICK: We actually work with Yuranis, the person who actually designed Cataball, and I did an analysis of Cataball on probably about 10 different compounds and we looked at what we actually saw in the...what we found in the metabolism studies versus what Cataball predicted.

And really how you paramaterize and set that model up really determines what metabolites you see. So you have to be careful with that, and you really have to understand the nuances of that model before you start just go down the path of just thinking it's going to predict all the degradation products that you could possibly farm, that was our take home message.

I think it has a lot of promise, I think it's going to take a lot of work to get the data to build a reasonable database to be able to start to draw from. Right now it's using the Midi data - I think it's on sewage sludge - to make the predictions. We are trying to, we've been talking to Duluth, who is actually been working with Uvanus and I think in the future there is going to be an attempt to try to pull in metabolite maps from our aerobic soil metabolism data and feed it into the Cataball program.

DR. DOUCETTE: And that's an excellent point, because we talked about that in the International Qsar Foundation sponsored a review of Cataball, and that was the one thing that was mentioned. Yes, it uses that database, and that's interesting, but there is so much other data out there that we could use to incorporate into Cataball and add

those metabolic pathways, which would be great.

DR. HEERINGA: Any other items? Dr. Ruhman? Panel members, any last comments on this particular charge question? Again, we'll have a chance to revisit it at the end if there is anything that you...comes to mind as we proceed. Well at this point, Dr. Brady why don't we move on to charge question number 2.

DR. BRADY: Charge question number 2. Interpretation of aquatic degradation rates for persistent pesticides with high sediment absorption coefficients. Environmental fate of pesticides with high sediment coefficient, often influenced by dissipation processes rather than degradation processes. An aquatic metabolism study the absorption process can be a most important process in removing pesticides from the water column.

This removal process however is not considered that the degradation pathway, because the pesticide is simply transferred from the water column to the sediment. Therefor the total system half life of the pesticide in aquatic metabolism studies is used to represent the most accurate degradation rate in aquatic environments.

Considering the environment fate data

typically available to support pesticide registration decisions, please comment on the strengths and limitations of the Agency's approach of using total system half life for assessing pesticide persistence in aquatic metabolism studies.

DR. HEERINGA: Dr. Gan, our lead discussant on this question.

DR. GAN: Yeah, I have a few slides also. My slides are better I think.

DR. HEERINGA: We'll acknowledge that, without a doubt.

DR. GAN: Okay, my fellow discussants include Professor Willy Lick, Professor Bill Dorset, Professor Tammo Steenhuis, and Professor Louis Thibodeaux. And looking the question again, I think the keyword here is really total system half life. Coming from a University environment, it took me a while...trying to understand some of the problems that are being discussed here.

This is from the article I guess, I lifted from the PFD file I was given. I have seen a similar set up. I have used a similar set up. The key here is really, if you can click up one more time, you have a system basically that has a layer of sediment, which is covered with a layer of water, and we'll spike

the pesticide into the water to start your experiment.

And of course what happens, is at the beginning the pesticide will glume to the sediment, but you are taking samples from both water phase and sediment phase. And at the beginning the water phase contributions will be greatly influenced by that absorption process.

Again, you have seen this chart, the black line is the water phase completion/dissipation curve, and the red line is the pesticide concentration in sediment. And the green line is really the total system concentration. You add the sediment and water concentrations together and my understanding is you can potentially use the black curve to derive DG 50 or half life, apparently that's what's being done.

And of course, as I just mentioned, a couple of people mentioned, the first part was really inaccurate, because you have both processes going on, the face partitioning as well as degradation happening. I think mostly PBT chemicals, the partitioning will dominate for the first pure of time.

However, if you add these together, of course you get the green curve, and in my mind that really would reflect the half life, the whole system half life, next one, this slide really summarizes what

my understanding is, and maybe some of my fellow discussants understanding here, because we don't normally do this kind of work. Again, you know, try to understand this graphic.

So the original OECD protocol calls for constitution measurement in both water and sediment phases, and as I say, the water phase concentrations are greatly influenced by partitioning during the initial stage. The complication is that without the non-proven conditions...immediately after pesticide addition, the fraction of the composition should depend on the Koc, but with PBT that should be pretty significant.

The complication is that effect, in my mind of experimental design. Maybe we have inherited it from 20 years ago, right? And also, you can also quite misinterpretation of the data. You can also call a lack of purity in terms definition. I think one of the public commentors mentioned that, which I agree.

And really this boils down to last conclusion, that is the fact that this partition should be excluded from calculating half life. That's what the proposed approach is, we tend to agree with that. So this is to reaffirm this approach.

The proposed approach will effectively

eliminate any effect of absorption on the measured half life. This approach will look more closely at proximate, the true half life for the whole system of the PPB chemicals, because sediment is dominant. The whole system half life is a useful parameter for describing persistence in the whole system. And the proposed approach will better fit.

Because otherwise you have this so called heart-stick-shape, and the first water fit is not good. So you have a better quality of half life than that, in my mind.

And in my mind, compared to the current practice that poses the half life in a more conservative approach from water column exposures, because half life generated by this approach should increase significantly over the current data, most likely.

Okay, my last slide is just a few suggestions on confidence, because many of these points you guys have already considered. For example the first one is whole system half life should not be used for describing systems before the equipment has established. I think that's a valid comment.

The whole system half life should be used for describing the whole system persistence, but

really not for short term exposure through the water column. For example, right after the drift, I mean you do have this short period of time, that of non-premium. It is essential to introduce a mechanism based on conditioning to address the transient exposure scenario you have built.

I think that you guys mentioned that the current modeling practice has taken that into consideration. Has both phase petitioning and half life as the input parameters. And again, it's worth cautioning here, the best approach may be still to understand each individual process for information again from 1 study. For example, from the aquatic metabolism study, can be used for other scenarios. Well that's all I have, thank you.

DR. HEERINGA: Thank you very much, Dr. Gan. Let's go to the associate discussants to see if they would like to add comments, or thoughts. Dr. Lick, you're up.

DR. LICK: I'm very-

DR. HEERINGA: Your microphone.

DR. LICK: I've been looking at this experiment, and Im thinking of how I would possibly interpret the results, because you introduce the chemical into the water, somehow it defuses into

sediment and partitions into the sediment. For a very hydrophobic chemical, this is a very, very slow process.

And for very hydrophobic chemical, all chemicals would go into the sediment. And then, let's say there is very slow degradation, the slower in bottom sediments with a lot of water. You have this chemical sitting in the bottom for a very, very long time as slowly, by molecular diffusion, defusing into the overlying water, where it presumably degrades.

But this again is a very slow process, because you have to diffuse all of this chemical, a huge amount of chemical from the sediment into the overlying water. So the...I don't know what you would deduce from this. You can't...from that experiment alone, you can't get the degradation rate in the water. You can't get it in the sediment, and what you do get is very difficult to interpret.

DR. HEERINGA: Dr. Doucette.

DR. DOUCETTE: I tend to agree with Willy's assessment, but I also see the need for a standardized approach for generating the data to run the scenarios.

I did have a question in terms of...I don't remember, I quickly glanced through the procedure

on the choice of sediment in that particular study. How is that determined, or how is that made, or what is the standard scenario?

DR. GAN: Coming from a river system, or a lake system? It's real, real water.

DR. DOUCETTE: And what characterization of the solid phase do you get, other than organic carbons? Do you get everything? And have you looked at - and this is really related to my broad question that crosses a couple of groups on KOC, and absorption, - have you gathered enough data for a variety of soil or sediment types to start looking at cases where the...you know you mentioned that you have the ability to use KD rather than KOC. Have you go to the point where you've actually got enough data that you can look at other parameters, other than KOC to understand absorption?

DR. RUHMAN: Yes, all the time we looked at the clay ponds with the KD, what's the relationship, and also organic matter content, and we looked at the PH also.

DR. DOUCETTE: And do you now have internal relationships that look at other sort of properties, you know, to estimate or extrapolate out when you are starting to look at this now, other than

KOC?

DR. RUHMAN: We have all the soil properties. It has to be the right texture, organic matter, PH, everything about the soil. So we relayed the KOC, I mean the KD absorb to this for a meter.

DR. DOUCETTE: I guess maybe I didn't explain that very well. Do you have any other internal quasars that look at something besides organic carbon to predict absorption? It sounds like you have the data available.

DR. HETRICK: A little clarification here, some additional information. When we get a batch equilibrium study that comes in, normally there's probably about 4 or 5 soils, maybe 6 soils that are, that range in both organic matter and texture. And what normally happens, is that as I said before, we did a correlation between the KD and the organic carbon to see if there was any relationship there. But also there is additional statistical...some regress...some fairly basic regression to see if there is any kind of relationships between PH, clay and in some case they even do some specific surface. Not very often. And absorption.

But those are done on a...you know they're not done on the same set of soils, and you know

you have all kinds of issues there, but the point is that for each compound that's generally...those relationships are looked for. So that's a possibility to use that information to make some better predictions.

DR. DOUCETTE: And the final clarification, the 6 soils or sediments that you use, or you get information on for absorption are not necessarily or probably are not the soil that you use in the biodegradation?

DR. HETRICK: Yes, as Dr. Ruhman said, normally the sediment that is selected for these aquatic metabolism studies are natural sentiments, and normally we don't see the batch equilibriums, those are generally not conducted on those.

DR. RUHMAN: But sometimes we get also a sediment KOC.

DR. HEERINGA: Dr. Lick, we'll come back to you.

DR. LICK: I have one problem, that's the second part. I have another problem with this, and that is worrying about chemicals with different partition coefficients. The amount of sediment in the system will effect the results, and that effects absorption time.

So there again I don't know how to interpret this. I have now a question though. Why do that experiment? Why not do the degradation in the water, and do the degradation in the sediment. Why mix them together? You get 2 experiments, you get 2 results.

DR. RUHMAN: I think what we are trying to imitate is an aquatic system in time. I agree with Dr. Chambers.

DR. HEERINGA: James.

DR. HETRICK: I agree with Dr. Ruhman's assessment. I think we have to remember that the guidelines for this study were designed in 1982. Not that science just started in 1982, but the point is I think we progressed for longer than that. And actually in all fairness, back when this first started that was probably appropriate. But because the exposure models really weren't at the point that they are today, and so, good point.

DR. LICK: And for most partition coefficients that probably wasn't that bad a problem. But now when we get the high partition coefficients it's an enormous problem, and you're right, it's now 2008.

DR. HEERINGA: Let's move on to our next

associate discussant, Dr. Steenhuis.

DR. STEENHUIS: I can not add anything to that.

DR. HEERINGA: Satisfied with the previous comments? Dr. Thibodeaux?

DR. THIBODEAUX : I agree with all the previous discussants. In this methodology is the 7 to 1 ratio kept constant?

DR. HETRICK: Yes.

DR. THIBODEAUX: Is that apparatus that was shown typically used it's a bubble apparatus?

DR. HETRICK: That's correct, it normally...for anaerobic systems it bubbles nitrogen, for aerobic systems they are bubbling oxygen into the system.

DR. THIBODEAUX: What about vaporization? Is that somehow-

DR. HETRICK: Generally they are trapping the volatile.

DR. THIBODEAUX: And how is this information is in the model for example?

DR. HETRICK: As I eluded to earlier when that question was asked, we take the total system half life for the aerobic aquatic metabolisms study, which is the aerobic study, and we take that total system

half life and use that for our water column degradation rate.

DR. THIBODEAUX: In EXAMS?

DR. HETRICK: In EXAMS, that's exactly right. For the sediment degradation rate, we use the anaerobic aquatic metabolism total system half life. And that is for EXAMS.

DR. THIBODEAUX: For EXAMS?

DR. HETRICK: Right.

DR. THIBODEAUX: Thank you.

DR. HEERINGA: Other members? Back to Dr. Gan.

DR. GAN: You just mentioned that this protocol was invented in 1982, and just for an outsider, if you want, if EPA wants to modify something, does that mean you have to start from scratch again to reevaluate all the chemicals or how is this for you to change or modify some protocols?

DR. HETRICK: My experience has been that it's difficult. We actually have a revised OECD guidelines that we've adopted. But really it still falls within the same framework as this study design. It doesn't separate the water phase from the sediment. It mixes it and puts it into a total system. So your point is well taken.

DR. HEERINGA: Contributions from any other member of the panel? Okay, Peter, you're up.

DR. DELORME: Just a couple of points. Ultimately the problem here is that when you are calculating your EEC's and water or sediment you want to have something that's reasonably...that's reasonable, that reflects reality. So if you are using a whole system half life that's driven by sediment processes, then that might not be reflected in your EEC's, correct?

DR. HETRICK: Well one could argue that. We're making the assumption here that we have essentially a stratified re-dox situation in that environment. And normally if you look at the read-outs potential in these studies, the sediment is generally fairly reduced and the overlying water is more toxic, that's for sure.

DR. DELORME: I may be dating myself, but I seem to recall that there are protocols out there for water only, aerobic bio-transformation studies, so-

DR. HETRICK: No, I agree with you.

DR. DELORME: So one of the potential approaches that could be taken as to...is to get both studies. But to get away from the problem with the...that dynamic at the beginning, to use a site

sediment for example, when you are trying to get at degradation of sediment. That way, I understand from talking to Dirk earlier, that EXAMS can put in separate degradation rates, and you already do that.

So there are possible other things. That may be a longer term solution, but in the shorter term, if you do have a compound that goes to sediment, the reality is you may want to be focusing your risk assessment on sediment dwelling organisms as well. We need to make sure that you are picking the appropriate organisms. There are a few things to consider when you get into comparing toxicity.

DR. HEERINGA: Okay, Dr. Mehta.

DR. MEHTA: I was wondering what kind of sediment you used in the test.

DR. RUHMAN: From either river system or lake, and it's usually taken from an area where the pesticide is going to be used.

DR. MEHTA: There could be a wide range, even within a single river, and the permeability of sand, silt and shale is widely different. There are several orders of magnitude of karst that you...I was wondering if...how do you sample I guess you go to the nearest basin and sample it, or how do you do that?

DR. RUHMAN: I redesigned that myself, so

I sampled soils but there is no...there is no specific depth. You have to get a sample for the sediment, you have to get a sample for the water, then you have to characterize each. You get characterization of the sediment, that there is texture, clay, sand, silt, PH and organic matter. And sometimes there is oxygen, they also do that. So we get these data for those 2 compartments.

DR. HEERINGA: Dr. Norstrom.

DR. NORSTROM: Just a brief comment. I know we've talked...somebody mentioned it earlier, the whole business about bound residues. Is the Panel, did they consider that at all? What the importance of long term...if you're going to model these things over 10 years or something like that, you might have a portion of that residue that's locked up and not really bio-available.

DR. HEERINGA: Microphone, Dr. Lick.

DR. LICK: We are going to talk about that in question 3, which deals with a lot of these processes. And I think it makes more sense to do it all at once, if you don't mind.

DR. HEERINGA: Dr. Delorme.

DR. DELORME: I just had an additional point to make. With respect to doing a study with a

spiked sediment, if in the answer to question 3 we are going to, if you are going to start considering sediment dynamics, and actually brining in soil and have it go to the bottom, that might be a little bit more realistic as well. There are a couple of reasons why you might want to consider that.

DR. HEERINGA: Turn to Dr. Ruhman and Dr. Hetrick to see if you feel this particular charge question has been addressed? Or whether you have any clarifications that you would like to request?

DR. HETRICK: I'm fine.

DR. HEERINGA: Okay.

DR. RUHMAN: It's a very simple question.

DR. HEERINGA: A simple question, but the answer is a little bit difficult, that's the way it works. Okay, we are the point in the agenda where we should be at 6:00 p.m. and adjourning, but this is a floating agenda, and I would like to move on, if possible to charge question 3, but let me just first of all turn to Dr. Brady to make sure that since there is a published agenda, whether there is anyone on your team that is not here that should be here?

DR. BRADY: I think we're able to proceed, yes.

DR. HEERINGA: Okay, and just out of

courtesy, general public, is there anybody aware of somebody who is absolutely critical to hear this? They can obviously hear it or read it in the final report. Seeing nothing, I am prepared, and the panel agrees, to move ahead with charge question 3. Dr. Lick is the lead discussant, and he is ready to go. So why don't we plan to do that? Dr. Brady, if you would please read charge question number 3 into the record.

DR. BRADY: Charge question 3, sediment dynamics. As part of its baseline ecological risk assessment process OPP uses Environmental Fate and transport computer models to generate estimated environmental concentrations of a pesticide in surface water, pool water and sediment. The EEC's are generated using the EXAMs model parameterized to represent a static farm pond receiving pesticide mass in run off from a treated agricultural field simulated by PRZM.

It is assumed by OPP that EEC's generated from this scenario are conservative representations of expected pesticide concentrations, not only in this farm pond but also in small first and second order streams that receive run off containing pesticide residues from many fields. Currently, the OPP modeling approach accounts for movement of

pesticide mass between water column and benthic region, using a set of lumped parameters and a mass transfer co-efficient.

These parameters are intended to implicitly account for pesticide mass transfer due to processes such as diffusion, settling, re-suspension and other processes that tend to make the sediment layer with the water column. The current OPP modeling approach does not include inflow of sediment to the water body, which could lead to burial of sediment containing pesticide in deposition.

Please comment on the strengths and limitations of OPP's current approach for modeling pesticide transport between the water column and benthic region, which relies on the use of lumped parameters to represent multiple transport mechanisms to static ponds.

In the context of screening level and refined assessment, please comment on the strengths and limitations of simulating pesticide burial by sediment in static ponds as a process that renders pesticide permanently unavailable for biologic interaction. Please comment on the strengths and limitations of models described in the white paper with respect to modeling pesticide transport via sediment dynamic.

Which processes associated with sediment based pesticide transport, PG, sediment enrichment, settling, re-suspension, burial, bio-purgation, poor water diffusion, scour, bank erosion would be most important to consider in static ponds. Which processes would be most important in flowing water systems?

DR. HEERINGA: Thank you very much, Dr Brady. This is obviously a multi-part question, and Dr. Lick is the lead discussant on this, and I think you have a presentation that you want to.

DR. LICK: Well I think as you can see from the question itself, it's a very broad question. And before we actually got into the... answering specific parts of the question, I thought it would be worthwhile to talk about some of these processes, which we have eluded to over the last 2 days but never really talked about.

And if I could have the first slide, this is the description of the pond by standard...oops, well, that's okay. The part that we want to emphasize is the sediment water interactions, and here it has the re-suspension deposition.

Then there is this other thing on the left which is aquius mixing, which includes all the processes that effect the sediment water interaction,

including molecular diffusion, bio-turbation, and ground water flow. Two most significant ones there are molecular diffusion and bio-turbation. Ground water may not be important at some times.

The other thing, besides talking about those processes intimate to all those processes is the question absorption. And it's been assumed throughout, in all our talks and with all water quality models that absorption is fast, and therefore that we can assume equilibrium absorption. I want to question that assumption.

The other thing on the right there you see this sediment bed layer, benthic later. This is...the thickness of this layer is absolutely crucial to these water quality models. Very simply, if we turn off all the input into a pond, and then ask how long does it take for a pond to come to some clean state, we're going to get chemicals coming out of the bottom sediment.

If I assume this bottom benthic layer is 3 centimeters thick, I'm going to get one result. If I assume 6 centimeters I'm going to get twice that time, and if I assume 12 centimeters it's going to be 4 times that time. How the hell do you pick this number? That's a problem, and I've seen numbers anywhere from 3

up to 15 centimeters; that's a problem. Okay, next slide.

Just look at the bottom part. I apologize for these slides, this is a last minute thing and I threw together some stuff, and the staff was very willing to help me out and make, scan these things and put them in, but anyway. This is desorption of 3 different chemicals, 2 PCB's and hexachlorobenzene. MCB is a monochlorobiphenyl. HPCB is a PCB with six chlorines.

The partition coefficient for MCB is 1/10th of 1/3rd, for HCB is 10 to the fourth, HTCB is 5 times 10 to the fourth. This is a percent...we first equilibrated these things by letting them absorb for a long periods of time, like months, and then we desorbed. The first thing you noticed, desorption is slow. Eighty percent desorption is, oh 5 to 10 days for MCB, more like 30 days for HCB, and something like 150 days for HPCB. So these are slow processes. By slow, I mean by comparison with the transit time for particles.

In other words you take a particle and dump it into a pond, it will drop out in minutes to at most an hour or so. These are orders of magnitude longer than that time. So in that period of time the

chemical absorbed into the particle and just goes down to the bottom, and that's it.

The other thing I'd like to comment on is there is no indication of a labile, and non-labile or a labile and irreversible fraction, and it fact we've done dozens of experiments like that, and in every case the amount of chemical that has been absorbed also desorbs.

The process is reversible. All the chemical comes out if you wait long enough. I mean, this is a graduate student here. She waited a long time for this, but she, you know, she had 2 children, she had a job, so we quit after 200 days or so. But all the chemicals, except for that one, are absolutely reversible.

Okay, next slide. The other thing that's important, and I think is the most important process in most situations is the re-suspension or erosion deposition process, especially the erosion process. That's highly variable, depending on the sediment. We can have course sediments, we can have fine grain sediments which are consolidated, or we can have fine grain sediments which are non-consolidated. These erosion rates depend on the particle size, the mineralogy, the organic content, temperature, gas.

No one th th inks about this, but in any sediment that people in this room are concerned about, you probably have a lot of organic matter, means you have a lot of gas. The sediments, not the person, just the sediments. So you have to worry about that. You can not predict the properties of these sediments with theoretical consideration, but you can measure them, and these are some measured results.

What you see here is a sediment which has fairly uniform properties with depth. Never the less, it does consolidate, so the density of the sediment increases with depth. Erosion rates change from whatever the number is there, down by 2 orders of magnitude here. This is a long plot, it's not trivial. There are huge changes in erosion rates.

Next slide. That was from a core in the Detroit River, this is another core in the Detroit River. This is a layered sediment, so again you have erosion rates changing enormously from one layer to another and within, you know, 1 or 2 centimeters. It depends on how fine that interface is, next.

Just the tope one here. I just wanted to show this one, because this is 1 out of 32 cores that we took from the Kalamazoo River. Erosion rates are on the left, but in the middle is the...oops, no,

no, yeah, that's okay. Right there is the density as a function of depth, and the density is fairly constant in this top layer and then it decreases fairly rapidly. Erosion rates are fairly constant in this top layer, and then they decrease very rapidly.

This is the layer about 8 to 10 centimeters deep. This has nothing whatsoever to do with bio-turbation. There are no organisms in this core. What it is, is the fact that you have a huge storm in 1986, eroded a lot of sediment as the flood ended, the sediment dropped out and you've got this layer, 5 to 10 centimeters deep throughout the Kalamazoo River. This is fairly common.

While we're commenting on this, we have 32 cores and as we went through we weren't really looking for organisms, but we look at these cores as we go along. There were only 2 cores out of the 32 that had any organisms in them. Of those 2 cores, only possibly 1 did we say that there bio-turbated layer there. In other words there was a little thickness change in the density in 1 or 2 centimeters near the surface.

Next, yeah, all the way down. Most people will ignore molecular diffusion, because it's presumably not important. But we decided to do

experiments on the molecular diffusion of various organic chemicals, and this is Hexachlorabenzine, but we used other chemicals, all with partition coefficients that went from 5 meters per kilogram all the way up to 5 times 10 to the fourth meters per kilogram. Of course the answer does depend on the partition coefficient.

But the interesting thing here is the experiment ran for 512 days, another good graduate student here. But the Hexachlorabenzine, that's millimeters, in other words the Hexachlorabenzine that's centimeters?

No, no, that's millimeters, in other words the chemical only defused a few millimeters in a year and a half. And that's what we found with all the organic chemicals. The higher the absorption coefficient, the closer the chemical is bound to the interface. As the partition coefficient went down, then it was able to defuse into the interior. And it's fairly clear what's happening.

You have huge partition coefficients, so the chemical defuses into the sediment, it gets immediately sucked up by the particles, or the organic carbon in the sediment. So it can defuse until some more chemicals come in. But there are huge amounts of

chemical on the sediment, as compared to what's in the water. This the amount of chemical on the sediment.

Can we have next? We couldn't measure what was in the water, but we could calculate what was in the water. By the way, the solid lines in the previous result were the modeling result. From using that model, we calculated what the chemical concentration in the water was.

And first of all, if there were equilibrium, C sub S would be equal to KTCW. Obviously it isn't true. I mean we're off by almost an order of magnitude near the surface. And then this is the right scale here, it's centimeters, so it's millimeters where the chemical is absorbed. Next.

Okay, based on those results for, oh, a dozen chemicals, we developed a numerical model and this shows the flux or the mass transfer coefficient for these different chemicals. First, the tope line is the partition coefficient of 10 to the 6th, then 10 to the 5th, 10 to the 4th, and so on. The units there are 10 to the minus 6 centimeters per second. If we get it into the units of the white paper that's 10 to the minus 6, about a tenth of a centimeter per day.

So all these mass transfer coefficients run from about .1 centimeters per day, up to 1

centimeter per day. The white paper poses a transfer coefficient of .1, which is at the lower end here, so I think molecular diffusion is much larger than what is being used in the model.

And this is the lower limit, remember, anything else is going to raise the mass transfer. But molecular diffusion is always there, while the other processes may not be.

Okay, next, keep going. Okay, then we did some experiments with bioturbation to look at organisms, and how they distributed chemicals. And this is a problem where we had 3 different organisms, separate experiments. This is lumbriculus, which is a vertical feeder where we keep, feeds at that, passes the food up through the gut and deposits fecal pellets at the surface.

The others were more horizontal mixers, or just mixers in general. These are the chemicals at the surface, being transported down into the interior by a convection diffusion process. In other words, the passing of food through the gut and around is a convection process, but the organism also disturbs the sediment.

That's a mechanical diffusion process. Okay, so these are 90 day experiments, the solid lines

are the mathematical model, and everything else is the experimental results.

Next. We took that model, - that's fine, - and we decided to run it, to see how these organisms would behave over a very long time. So the experimental results are the 90 day results on the left, and then we see results from 1, 3, 5 and 10 years. Eventually you get something like a well mixed layer, which is what is universally assumed by my modelers. A well mixed layer of constant thickness.

Well I don't know how to define that, I don't think it's well mixed, and it certainly isn't a constant thickness. Because that one year it may be a little less than 2 centimeters, and it keeps changing until 10 years is about 3 centimeters. So, I'm sorry?

DR. BRADY: This one is centimeters, and not millimeters?

DR. LICK: Yes, this one is centimeters. Bio-turbation of course has...I mean it will generally mix or transport sediment roughly comparable to the depths of the organisms themselves.

The next slide is important, but you don't have it, so I'll tell you what it is. It's, based on this and the experiments, we calculated the flux. The flux of chemicals due to organisms is now up

to about 1 to 10 centimeters per day.

As compared to molecular diffusion, which is .1 to 1 centimeter per day. Now this is two of Fitz's, the vertical feeder, and at 10 to the 4th organisms per square meter.

Which is a fairly large density. Occasionally you'll find things up to several times, times ten to the fourth but more often than not in the Great Lakes, for instance, they'll tend to be between ten to the third, ten to the fourth. Sometimes in Lake Superior, it will be less than that and of course the flux decreases from that, so we're talking about organisms having mass transfer co-efficient usually between one to ten but certainly could be less than that and molecular diffusion between .1 and one.

I think that's all I have as far as description of the processes.

DR. HEERINGA: Thank you very much, Dr. Lick. Would you want to go on to actually take this and address the other question or shall we go to the associate discussant?

DR. LICK: I think it might be worthwhile if people had questions now.

DR. HEERINGA: Okay, well, let's, yeah...fair enough, that's, Dr. Parker and any others

on the material that Dr. Lick has presented, as background question three, any questions on....

DR. BRADY: Yes, I didn't understand the erosion rate coming from the sediment pores, how is that, so you're not talking about erosion rate coming off of an agricultural field.

DR. LICK: No, no.

DR. BRADY: Is it settling at the bottom?

DR. LICK: Bottom sediments. Okay, we have, I made, what we wanted to do was not only measure erosion rates of surficial sediments but because in big storms you can erode huge amounts of sediments, tens of centimeters quite often so we also wanted to know not only how fast sediments erode at the surface but after they're eroded how fast do they erode at ten, twenty, thirty, centimeters down.

DR. BRADY: So this is erosion from the bottom of a moving stream?

DR. LICK: Of a moving stream or near shore of a lake or something like that, so what we do is take coarse, put it into this plume and then erode the surface until we get down to a certain depth...well, we take measurements all the way along but we can go down to as much as a meter and measure

that.

DR. BRADY: Okay.

DR. HEERINGA: Additional questions, Dr. Parker, anyone else? Dr. Steenhuis.

DR. STEENHUIS: I understand the velocities, but your velocity is not for a mixed system, your velocity is I think for moving pollutants out throughout the whole.

DR. LICK: It's a mass transfer co-efficient. What it says is the rate at which mass is transferred is proportioned to the co-efficient times the difference in chemical concentration between two layers.

DR. STEENHUIS: That is actually, the concentration in the sediment is in the EXAM model is uniform over the top five centimeters, and what kind of concentration do you take?

DR. LICK: At the surface?

DR. STEENHUIS: You have not a mixed system, you have a system which has a gradient in it.

DR. LICK: Right, and these are, adds are from the experiment so we've normalized it with the concentration in the overlying water.

DR. STEENHUIS: What do you use for the concentration in the sediment?

DR. LICK: We don't. In other words, that's just the flux on the basis of the overlying water, that's why it decreases with time, because the flux does decrease with time, but we don't take into account, because I don't know how to define a chemical concentration in the benthic layer.

DR. STEENHUIS: I think we should talk after the meeting about it because I'm confused.

DR. LICK: Well, the problem this well mixed benthic layer really is something that was invented by modelers. It is not there, we have never observed a...well, except for the well mixed or mixed layer due to deposition, but otherwise we have never observed a well mixed layer, so I don't know how to define the well mixed layer, the thickness of the well mixed layer in the real world.

DR. STEENHUIS: Mathematically your problems can be just as well described as a mixed layer. It is a well defined mathematical problem I think. You can also calculate the diffusions of the top layer.

DR. LICK: I'm sorry, I didn't...

DR. STEENHUIS: I mean your, the problem you present without mixing can be described mathematically, too, just like you can describe a mixed

layer so you can just take, I mean as an alternative for this mixed layer you can also take your set of equations in order to predict the flux, too.

I mean you did not show the equation for calculating the fluxes, but there is a set of equations that you used to calculate the fluxes in the overlying layers which are different than the mixed layers.

DR. LICK: Oh, yeah.

DR. STEENHUIS: A set of equations, and simply what I'm trying to say is that your set of equations can be used to instead of this mixed layer and that gets you out of the problem of defining what the size of this mixed layer is.

DR. LICK: Yes, what we've used are continuum equations, we've described how chemicals are transported through the sediment column due to diffusion, convection modified by absorption. They are continuing, I mean they are differential equations rather than the mass transfer equations.

They, I mean at the very end of this talk what I would like to suggest is that we modify the EXAMS or similar water quality model by treating the sediment layer in several discrete levels and actually doing a one dimensional time dependent model which would treat each one of these processes independently

and that would give us a much better description of what's happening in this, in this, any system.

DR. HEERINGA: Dr. Lick, I think you've actually in your last comment moved on to actually responding to this question and I think if we could turn to that, I think you've made your first recommendation there.

DR. LICK: Well, okay, before we get there, I mean, there were several sub-questions here, the first one please comment on the strength and limitations and so on on the use of lumped parameters. I think we've sort of said something about the use of lumped parameters already.

The main thing there is each of these processes, erosion deposition, molecular diffusion, and bioturbation behave in a different way and therefore they have to be modeled in a different way and the reason you do that is that you can use a mass transfer co-efficient to, and calibrate it to your data, but if you're trying to actually predict something, the time dependence of the three different processes are different and they're different from what would be predicted by the mass transfer co-efficient.

So if I'm going forward in time, I can't really, I think it would be much better to deal with

each of these processes independently especially if you're looking at a variety of ponds where you may have organisms, you may not have organisms, you may have erosion, each one would act differently.

And the other thing was this parameter curve in, I think I sort of referred to that already but again, if you had very slow absorption, the chemical is going to stay with the particle, go from the surface, plot on the bottom, curve in as one, that's it. On the other hand, if I have low partition co-efficients, slow absorption, I mean fast absorption which goes along with low partition co-efficient, then all the chemical dissolves into the water, the particles goes down to the bottom but there's nothing on the particle any longer, and so in that case curve in is zero.

And you can make estimates of this, you don't have to assume 0.5 and that was the first sub-question so maybe there's questions at this point.

DR. HEERINGA: Dr. Hetrick.

DR. HETRICK: Getting back to your molecular diffusion co-efficients, that's going to be dependent on what compound you're talking about, right, so that's....

DR. LICK: Yes, the partition

co-efficient.

DR. HETRICK: Well, the partition...

DR. LICK: Primarily on the partition co-efficient.

DR. HETRICK: Yeah, okay.

DR. LICK: It's primarily the partition co-efficient and I think secondarily depending on the diffusion co-efficient or the molecular weight of the chemical.

DR. HETRICK: Okay, okay.

DR. LICK: Because I say I think because it's so overwhelmingly on the basis of a partition co-efficient.

DR. HETRICK: Okay, I was just...

DR. LICK: The rest of it is almost noise.

DR. HETRICK: I was just wondering if it's really, it becomes an issue then of this could actually be a variable that needs to be dependent on the properties of the pesticide that are being considered and do you recommend that type of analysis, that type of approach?

DR. LICK: On the basis of what we know now, or on the basis of these experiments, the first approximation would be just the partition co-efficient,

period, and then you could, I mean we have a formula for the, I mean again we have a mathematic model which we have used without changing, without fiddling with the co-efficient, in other words just using that expression and we've used it for, I don't know, six or eight different organic chemicals and it sits, okay, and it takes into account the partition co-efficient and the change in the molecular diffusion co-efficient of that chemical.

DR. PARKER: Actually I have another question as well.

DR. HEERINGA: Sure, Dr. Parker.

DR. PARKER: Looking at our two meter depth static pond, would the partitioning books say we have a pyrethroid coming in with spray drift. Would you expect the partition co-efficient itself would put most of the chemical on the sediment somewhat instantaneously in a two meter pond or does that take some time?

DR. LICK: A two meter pond only takes, you know, you know, minutes or maybe an hour for the very finest particles to drift down to the bottom. Particles settle out fairly rapidly.

DR. PARKER: So the PR ben doesn't really serve any purpose if it's, if the high KOC is

doing that by itself?

DR. LICK: I'll just talk about the science.

DR. PARKER: Okay.

DR. HEERINGA: Dr. Lick, I wonder if we could get your views on the second bullet then, that is the burial.

DR. LICK: Oh, yes, this is the fun one. Please comment on the strength and limitations of simulating pesticide burial by sediment in static ponds as a process that renders pesticides permanently unavailable for biological interaction.

I think it's fairly clear from all of the discussions that we've had that burial is absolutely essential if you, within the water quality model if you don't have burial, the chemical keeps concentrating in that layer and you get erroneous results. I mean think of a simple system where I have a sediment layer and I put in a pesticide with the runoff, mass particle runoff, that has a certain chemical concentration as it deposits on the bottom.

Later I have another storm and that layer, puts down another layer of the same concentration. The chemical concentration doesn't increase by all these processes, it stays the same

because the amount of pesticide increases at the same rate as the thickness of this layer. On the other hand, if I forced a constant layer of sediment, the water quality model wouldn't tell me that the pesticide concentration is increasing here, which is just not true and I think that showed up in a lot of the water quality models that have already been shown.

The other thing where it might come in is you have to, as far as fluxes are concerned, you have to compare the rate of deposition with the rate at which all these other processes behave. In other words you have deposition about let's say one centimeter per year or maybe a couple centimeters per year. Now that amount of deposition is certainly comparable to anything that molecular diffusion or bioturbation can have so that would dominate the whole flux process, and so again you have to consider this not only as far as burial is concerned but as far as the flux processes.

DR. HEERINGA: So on this third point that asks about these processes and their impact in static ponds and then separately in flowing water, your view would be that the sedimentation burial process dominates some of these other processes in terms of the modeling impacts?

DR. LICK: Well, what our experience,

I'm aware of a lot of the work that goes on in the Passaic and some of these other large rivers which are heavily contaminated with PCBs in the sediments, I mean, don't worry about stuff coming in, it's the PCBs in the bottom sediments that are important. If you look and estimate all the processes that might be important, you figure out that if you really wanted a first approximation all you would care about is absorption rate and erosion deposition, period.

You wouldn't worry about volatilization, bioturbation, molecular diffusion, all of that is irrelevant. I would get a really good answer if I just knew erosion deposition absorption.

DR. HEERINGA: You feel that would apply both in static and flowing systems in the Detroit River or Trenton Channel that's moving right along.

DR. LICK: Well, the river, yeah, the Trenton Channel, the Passaic River is there, you know, they move. No, a pond would be different, but you would still have the deposition problem, you might not have the erosion problem.

DR. HEERINGA: Right. Okay. What I'd like to do is I'd like to give the associate discussants a chance to weigh in here and the first associate discussant is Dr. Mehta.

DR. MEHTA: And what I'm going to do is go through these three comments and one of the, there are two parts to the transport problem. One is forcing and one is response and the models at present and the one that was presented by Arnot and Gobas, they look at the response but in fact we know that when you have, if you have a static pond as Dr. Lick said, you wouldn't have any resuspension, so if you are dealing with resuspension in a static pond then it is not a static pond.

Am I right or how would you get resuspension if it's a static pond, you would just think you'd have deposition.

DR. BRADY: Well, you wouldn't have dep... you wouldn't have resuspension through flow, we've considered that you might have some bioturbation that would continuously cause some mixing of the chemical with the benthic layer.

DR. MEHTA: But the erosion deposition function that having put up have to do with some soil factor on the bottom and you know I've never seen a pond that doesn't have it, it always does and you also have wind, so wind dries the surface water as you all know, but it introduces vorticity so as a result of that you have a stress on the water and when you have

even the slightest amount of wind driven net current, it actually completely modulates the effect of the waves below.

So it surprises me that, you know, we would consider even distant stress level models that don't include some causative factors of resuspension other than bioturbation.

DR. LICK: Well, let me...I look at farm ponds. What is quite often the case is that you have shallow, in very shallow areas and banks and your waves can erode sediments in these shallow areas and you get muddy water in there which diffuses out into the interior. And occasionally some bank erosion, but that's relatively small.

DR. MEHTA: Which one, the bank erosion ?

DR. LICK: Yeah.

DR. MEHTA: So the other thing is that on the response side, you know, that was why I asked the question about what kind of sediment is being used to test the absorption and you know, there is sand that is silt and that is clear, as Dr. Lick said, and they are quite different as we know. Sand has a high permeability and there is poor water motion or there can be. In silt less so and in clays it fluctuates.

There is very little poor water motion because of permeability of ten to the power minus nine and so on so any model that simply creates, calls sediment sediment, you know, I don't see the meaning of doing any particular kind of transport simulation because I think that the result would be totally spurious in that sense.

Now there are one V models available. Dr. Lick mentioned one V vertical model that had been used for the screen testing and I was a little bit surprised that they're not being used here at EPA even at level next to the one that was presented like EXAM and so on because I think even the no and robust model is deficient in terms of forcing and in terms of what is resuspense?

For example, as he said resuspension rates can vary over five or six orders of magnitude, but there are reasons for it. There can be a density effect or organic sediment or all kinds of other things. There can be gas in the sediment.

Similarly, the settling rate velocity can vary over five orders of magnitude because it can have cloth or you can have individual particles or if you have sub-micron particles then they simply don't settle basically. They stay in suspension. So the

other comment I had was that when I look at the high end model as it is called, if you look at the presentation, all the relationships mentioned there are from the 1980s and early 1990s.

Now in the last ten years a substantial additional amount of work has been done in improving all of those relationships with the effect of an interaction between the turbulence or the boundary effect and settling boundary and erosion and so on and so forth.

So I think that even the high end model is, it requires a substantial upgrading and finally I think that just a couple of comments I had is that the way burial was presented was in the following way.

That if you have a pulse of contaminant going into water and then you have no pulse beyond that, then burial does have an effect became you reduce the concentration, but of course if you have and that was, for example, what happened in San Francisco Bay has a lot of contaminations from the ships and oil and petrol chemicals but the sediment that arrives, clean sediment from the Sacramento, San Joaquin delta, there is a sediment and surprisingly the rate at which the sediment has been coming in that way is also the rate at which sea level is rising so the depths of water

have remained more or less the same but you have this burial effect which causes sediment to get buried.

But on the other hand, burial doesn't mean that the sediment will be buried forever, you could have a big hundred year or a thousand year storm and it could get resuspended. One example is the burial of DDT by Los Verdes as a result of the outfall and then they started, they reduced the amount of clean sediment in the outfall and there was erosion and the DDT started coming up.

So that is an example where burial has to be tracked, and burial is not just a static phenomenon where you can think of it as something that's disappearing from the water column because there are decisive biological effects that are consolidation effects.

So one has to track the density and the strength of the soil with time and for example, if a soil stays somewhere for a very long time, it would harden to a point where it doesn't resuspend it to. The effect of waves has really not been treated even in the high end model.

And next month there is an EDU chaplain meeting that will be dealing with really very high level modeling of waves and sediments and that is not

to say that all of that needs to go into testing procedures at this point in time but I was a little bit surprised by the fact that water waves are not considered and in a static pond with waves, if you go into a static pond, you'll find that as a result of wave action there's always a fluff layer at the bottom and that being the case, sometimes the flux of constituents from the sediment into the water column increases quite a bit more than molecular diffusion because of heating of this material.

And it turns out that that flux can be related to a dimensionless equation to the wave height and so on and so forth, but I think that when, for doubling, you look at subvariant going to Florida Bay, and we do have, this is the bay, and this static pond idea simply does not cover all that situation.

And finally I'd like to say that in this, in the modeling of sediment transport, the United States is far behind some of our colleagues in Europe. One of two other countries that have very advanced modeling is especially Holland and Denmark and you find that while Government agencies in the U.S. use some of their own models, most of the consultants actually do not, they use a mic 3 and mic 21 in doing another model.

And I think the reason for that is that these models have not seriously been looked at in terms of upgrading their technology, the technology we use in this model is fairly old. We can see that not only from the citations that were given, but also the proteins that are being used right here in EPA. So I think that those are my comments so far.

DR. HEERINGA: Thank you very much, Dr. Mehta. I'd like to go on to the other associate discussants. What I may do is go through the associate discussants this afternoon and then adjourn for the day and maybe we'll pick up some final discussion on this tomorrow morning before we begin charge question four, but right now Dr. Steenhuis is our next associate discussant. Your mike, would you pull it really close to you, too, Tammo, we've got this huge fan that's running here and I've got a little white noise problem.

DR. STEENHUIS: And I have an accent, too. In order to try to better understand what was going on with sedimentation rate, I tried to simulate it, and I would like to show that and I have some interesting aspects.

(WHEREUPON, there was a pause in the proceedings.)

I have my, I call it the modern EXAM slide because it doesn't have all the processes. The

next one.

And although after Dr. Lick I would have changed my model significantly, this other model is in instant equilibrium. Instead of all the processes on top I added the same amount and that gets, the advantage of that is you can exactly see what happens to the sedimentation rate and instead of a mass rate, I assumed actually between the water phase and I only assume degradation in the benthic, lake benthic area. Next slide.

It is more or less the same font model, it's the same effects. I assumed the ten hector pew, the one hector font, out of that two meters, five centimeters benthic zone is the same as the EXAMS model and initially I said the half life of a thousand days of sediment which is somewhat equal to pesticide four but there are partition coefficients of hundred kilograms per liter, really doesn't have an effect but absorption partition coefficient case and the concentration changes by number but the effects are the same and I have some sedimentation rate of zero, 0.6 and 1.2 centimeter per year and applied monthly and two applications per year of 100 grams. Next one.

And like you see over here is the sediment concentrations for different application

rates, the depth below the pond so that from zero to five centimeters that is the mixing or the benthic zone and in EXAM it is completely mixed so we see the same concentration and by adding sediment, the pesticide will be pushed down, so the blue line is after ten years of simulation and you see the sediment to 16 centimeters depth. And for 0.6 centimeters after ten years it is approximately of ten centimeters depth and, but there's no sediment concentration, you see that the sediment stays in the top five centimeters.

If you integrate another curve, if you see how much area is below the curve, you see that there's much more pesticide in the layer for the, where there is sediment concentration, where there is the highest rate of sedimentation. The lowest amount of sediment, the lowest amount of pesticide in the sediment is no sedimentation.

If you look to the bar, the next slide, the concentration of the water of course is opposite the highest sedimentation rate. The blue line has the lowest concentration in the sediment and the no sediment has the highest concentration water and is exactly consistent to what you saw before.

And on the conclusion based on this is the next one, is that increasing sediment concentration

rate indeed gets lower concentration in the water. All these models are completely consistent about that.

What he didn't...what is different or what I did not, what I didn't understand yesterday, but after modeling, the higher the sediment rate, the more pesticide are in the total body of water and that has an effect on if it is being remobilized. It's sediment, when it's on the bottom, it's not out of mind out of sight, it was kept mixed up like anything, a cow in the stream. What we see in New York, all kinds of cows in this thing, they will mix it up, people in the stream but also getting the flow system is still available.

Next one. Actually this was done out of pure, pure interest because before with previous sediment, with previous panels you didn't have any of the actions against that the sediment was not included. And the previous panel quite concerned about pesticide within a very short degradation life so I did the same analysis for a pesticide with a half life of thirty days, which one, which is aldecarb or something like that. You're talking partition coefficient would be lower, but it really doesn't affect and again sedimentation rate of zero centimeters up through 1.2 centimeters.

And the next slide we see the

concentration in the water and the rest is for, it's for 1.2 centimeters per year and the other concentration was exactly the same is for zero centimeters per year and if you, and the next one is the same slide for high application rate, with a thousand days you see a big difference.

Next. So essentially if you had, if what was done before the approach of a static pond without concealing the sediment concentration mixing layer absolutely completely okay, but if you want to simulate pesticide with high index with lower degradation rate, you need to have a different modeling approach as the one before.

Next one, this is essentially to show the effect of half life of concentration of water and concentration of sediment, so I assume the sedimentation of 1.2 centimeters per year and then essentially we see what happens if you change the concentration of the half life. Next one.

This is the concentration in the water and of course the lower, the higher, the red one is 2000 day half life and you find the higher concentration, which is completely logical, but why you find the concentration in the bottom of the sediment and not in the top of these two but there are really

sedimentation increases total amount of pesticides in the soil, anyway you look at it. That is, contaminated sediment. If you have clean sediment like you were talking before then it is the opposite effect, but it is adding dirty sediment and pesticide and the concentration of the water is the next one.

The pesticide concentration of course is the highest, that is the red line, for the lowest, for the highest half life which makes completely sense, and also the interesting thing is they all got to equilibrium and the same amount disappear each year in each half year independent of the half life in the sense that that is the way it works out. I mean, in equilibrium we have to remove the same amount as you add, so that is correct what you see over here. The squiggles are left in the beginning and the squiggles at the end are exactly the same.

Next one, despite a concentration of water, greater half life, greater concentration of all the sediments, the amount of sediment added has no effect in pesticide concentration of water for non pest, persistent pesticides but I would like to add to by changing the mass flux rate I can actually get a completely different answer if I mean I didn't have time before that because we do it right now what I

would have done is tonight after this panel, is changing the assumptions about the mass flux rate and the depth of the mass flux rate, and I can get different results.

So the point is that it's the assumptions about mass flux and the depth of the layer, depth of the benthic layer, it can affect. I mean, although I didn't do it and we saw that before too and in extremely sensitive parameter and we need to know the, we need to know the parameters in order to simulate this realistically.

DR. HEERINGA: Thank you, Dr. Steenhuis. I'd like to ask Dr. Thibodeaux to weigh in on this question.

DR. THIBODEAUX: Yes, turning to the first question, the strengths and limitations of OPP's current approach. I think you alluded to it indirectly when you, and here I'm talking about prism's coupled to exams, that we're on our way to a different methodology when you invited Dr. Mackay to present parko and I think that is a step in the right direction. The transport of pesticides between a water column and as Dr. Lick has pointed out so nicely, is a very complicated process. It changes with time, it changes with layers of the sediment.

He and I are very aware of the high end models that are currently being used to assist this assigned release of PCB releases from the northern rivers. Those high-end models have a lot to say and we have learned a lot over the last ten years about this. It's very clear from the model that Dr. Mackay has begun to come off of that one month parameter that's trying to represent all of these mechanisms. And of course, that's a very good first step.

You have to realize, since the exam was created, which was several years back, and it has been modified, with it's time for a vision, I think the article moves in the right direction. It should be an option of the operator to select processes that more nearly fit the receiving strength. I would recommend the pond be replaced by three options. The pond being one, but another being the flowing stream and possible a third which was an estuary. It seems to me if we use the model as an option for screening of other models, depending on the locale of the pesticide use and where it may end up, then that might be an important first step.

Dr. Lick was correct in the vertical, I think that layers should be created, there should be a, I don't like to use the word completely mixed, because

I agree with Dr. Lick, I don't think there's a completely mixed layer. There is enhanced mixing in the surface layer in the top, say roughly 10 centimeters, but as you go down, the process changes. That mixing occurs for a lot of reasons. Bio-turbation being one.

So I think that's one thing that the structure of the model and the transport processes would be a good first step and we've learned this from these more advanced models and I think it's time to start. And I think ARGO is a big first step in bringing this into the system. Of course, we want to retain the ability to look at transit conditions. I think that has been very clear through this presentation that steady state is sort of passe and we really need to look at the variations with time. The application and the response. So that pretty well covers my first point, chairman.

The next thing, the context of screening level and refined assessment. Please comment on the screening question#2 with simulated pesticide in varial and static ponds and processes that move the pesticide permanently, if you look at the overall fate balance and neglect the varial process, then there's something wrong with your model. In the case of ponds,

and again that's why I think we need 3 different possibly similar scenarios. Ponds, streams and marine estuaries. Very clearly, we would not have a contaminated sediment problem that's been brewing for the last half-century in the United States, particularly with PCB's in the Northern rivers, if it were not for varial.

So varial is a process that has to be in the model. Particularly in some of those models that we know where deposition overrides resuspension. So there's no doubt it. You'd have to consider that part. So I guess my answer for that one is back to my recommendation for #1 is. On question #3, I guess the answer is yes. Which process is, of course all of these, sediment enrichment, sediment resuspension, varial, bio-turbation, -- that's what the soil scientists called it.

Polar diffusion, scour and bank erosion cannot be present in all aquatic systems. Bank erosion, for example, is something quite common in streams because of the meander. So I think user should be given the choice of some of these options as well. So what I'm saying, for uses for screening at this level, I think it's time to make changes and I think from what we've learned in the last 10 or 20 years on

sediments, should allow you to start to develop compartment models, I think would be the right word. And that's what ARGO is.

Realizing that number is in your cases with site specific studies, in which we're looking at a generic type of application. I think even those can retain a lot of the realism that Dr. Lick talked about. And also the sediment transport. Can we put anything in a more realistic fashion than it is now? That's my comments, Mr. Chairman.

DR. HEERINGA: Thank you very much Dr. Thibodeaux. At this point, what I'd like to do today, is to, Will Doucette is the last associate discussant on this question and I'd like to give him a chance to give his comments and then I think I'll wrap up for the day. I know that Staci has one thing she wants to introduce. We'll do that before the end of the meeting. But Will, if you want to do your comments?

DR. DOUCETTE: It's easy going last, there's not much left, especially at this point in the day. I agree with the previous discussion of best not to use lump parameters because they vary both with site and chemical properties. I also agree with the absolute need to consider varial, so I really don't have anything more to add other than reiterating the

importance of what's been said.

DR. HEERINGA: What I'd like to do to make sure that those who have to travel locally can make their commuting connections and so on, I'd like to adjourn for today and we'll pick up again tomorrow morning and we need just a charge or request of the discussants on this.

Thinking about this whole issue about first priorities and longer term, I very much, the discussion that we heard is very valuable and there's been a lot of contribution here, but maybe when we return tomorrow morning, give this some thought, if you have a set of priorities, not only on the compartments, but on the mechanisms, how would you prioritize them out or develop them? And I think that would be valuable.

We'll give everyone on the panel a chance to comment on charge question# 3 tomorrow morning too. Staci. Dr. Simonich?

DR. SIMONICH: This is with regard to the discussion we had earlier today regarding the long range transport potential of pentachloronitrobenzene and I just realized this over the last few hours. But I wanted to call to Dr. Cohen and EPA's attention the recent publication by Trevor Brown and Frank Wania,

it's Environmental Science & Technology 2008, issue 42, pages 5202 through 5209, screening chemicals for the potential to be persistent organic pollutants, a case study of arctic contaminants.

Are you aware of that? Okay. So I would point out that in this publication that Dr. Wania used global pop to estimate the elevated arctic contamination and bio-accumulation potentials of over 100,000 distinct industrial chemicals. And 120 of those 100,000 chemicals were determined to have an elevated arctic contamination and bio-accumulation potential. And in that list is pentachloronitrobenzene and also in that list is --DDT.

So I would like to point that out, that Dr. Wania's use of global top suggests an elevated arctic contamination and bio-accumulation potential for pentachloronitrobenzene. I'd also like to point out to EPA that in this list includes other registered current use pesticides that might be of interest.

DR. HEERINGA: Thank you very much, Dr. Simonich. Dr. Lick?

DR. LICK: If I could have one minute.

DR. HEERINGA: You certainly can have one minute, use your microphone.

DR. LICK: I'd like to make two proposals

to the panel and to EPA. One is to develop a one-dimensional, time dependent model of these processes. One with your idea of what's important, what's not important. The second thing is, how the hell are we going to verify this? Suppose we do some field studies with ponds, different types throughout the country which have different characteristics.

DR. HEERINGA: I appreciate you bringing up the Kalamazoo River, I grew up in Kalamazoo, you brought me home.

Dr. Parker?

DR. PARKER: If I could throw out a couple more issues for the panel to think about, if we could muddy the waters a little bit if that's appropriate. I might also ask that the discussants think about tiering, in terms of a simple system that can be run quickly and easily and efficiently that gives us some bang for our buck without going with something entirely complex.

DR. HEERINGA: Well you've got another dimension. It's not only prioritization and the difficulty of development, but how it would fit into a tiered system of assessment. Dr. Bradbury?

DR. BRADBURY: If I can buy a minute. To follow up on Dr. Lick's concepts of how would you

evaluate whether or not the models were working and Dr. Mackay brought that up yesterday as well. I'm wondering if there isn't some semi-quantitative if not qualitative ways we might think about some pesticides of the past, psychodynes for example that we think had moved around past the site of application, be it long range transport through the air or maybe sediments moving around was our way to do a mental exercise of how this would all work.

If we put in dieldrin, even if we don't know what dieldren did, what would we conclude with the current models we have or what some of the iterations of the future may hold. Would they predict that aldrin is ending up in places that are far removed from where they were applied in cornfields in the midwest. Could it have been long range transport, could it have been movement of sediments to different basins or different rivers and systems.

It seems as if our constructs aren't sort of doing what we knew happened with those older pesticides, they'd give us some insights into what we're getting right and what we're getting wrong as we go forward. So one other way is to use old data to give us insights to know where we are and where we're heading.

DR. HEERINGA: Okay. I think we'll have a chance to revisit this all tomorrow morning, maybe a little refreshed. I'd like to turn then to Myrta Christian, the Designated Federal Official to see if she has any last minute announcements for today.

MRS. CHRISTIAN: No, we don't have any new announcements, but I do expect to see everyone tomorrow morning at 8:30.

DR. HEERINGA: Thank you very much everybody, have a great evening, and we'll see you tomorrow morning.

(WHEREUPON, the MEETING adjourned at 5:15 p.m.)

CAPTION

The foregoing matter was taken on the date, and at the time and place set out on the Title page hereof.

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SUBMITTED ON OCTOBER 29, 2008

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