Day One: September 11, 1996

Session One: Questions and Answers

After each session, there was an opportunity for questions and answers and group discussions pertaining to the speakers' presentations.

Q (Maurice Zeeman, U.S. EPA, Office of Pollution Prevention and Toxics): I heard Gil Veith talk this morning and I would like to comment briefly on a point he made. At a gut level, I believe issues related to bioaccumulation and sediment assessment are important issues. I would like to seek reactions from this panel or others to the following comments. Most of us are scientists. We deal with a Cartesian system where we have divided things up into little discrete parts, because they are too complex to try to handle as a group. The risk assessment paradigm is a perfect example. The key issue is how and when to start putting the parts back together to make some sense out of it. Throughout the conference, I would like to ask that you focus again and again on what Gil Veith said in his presentation where he got that "So what?" reaction from a fairly knowledgeable group of people to the statement that 29 percent of the sediments in major estuaries were being impacted. As we go through our methods and our models for bioaccumulation and sediment assessment, try to think about what additional research will provide essential data to help the public, lawmakers, and regulatory agencies make sound decisions and take sensible actions.

Betsy Southerland:

We are looking for ecological significance. What we are going to try to do, as we go through the next couple days of this conference, is look at the tissue residues from bioaccumulative pollutants and see what impact they have on aquatic life, what impact they have on wildlife consumers of that aquatic life, and their potential for human health impacts. We already have some field studies conducted by Rick Swartz that show a significant impact on populations of other benthos if there is acute toxicity in the amphipod test species. That certainly gives us an indication that at least the acute toxicity tests have ecological significance. What I want to hear today, which was one of the prime drivers for organizing this conference, is what information is available on bioaccumulation tests and tissue residue measurements to make equally important interpretations of ecological significance. Interpretation is the key. We can all do laboratory tests, but then we have to determine what the tests mean. Would anyone else care to comment?

Peter Landrum:

I will add a few comments. It has been my observation that whenever a particular group studies some aspect of the lower end of the food web, there is generally less interest in the work than if it involves fish or fish-consuming wildlife and birds. There seems to be a general lack of recognition of the supporting ecosystem required to maintain the reproduction and the productivity of the higher levels of the food chain. I do not know if it is within the purview of this group to be able to drive that point home. This point applies to studying phytoplankton productivity or contaminants at the lower end of the food web. The issue remains that the connections between the lower food web and the upper food web get lost, particularly when you move into the regulatory realm of lawmakers responsible for environmental legislation.

Q (Peter Chapman, EVS Environment Consultants): I would like to address a question to any of the panel members. We have talked about bioaccumulation in terms of relating it to toxicity effects we already see occurring. One of the attractions bioaccumulation has to me is the possibility of anticipating things before they occur. At this point, do we have any examples where bioaccumulation data have really enabled us to predict impacts, and, if not, how much further do you think we have to go before we can do that?

Henry Lee:

One example would be the work Rick Swartz did on the sum PAH model. Since these are neutral narcotics, we discussed bypassing sediment concentration and just going directly to the tissue residues. That would eliminate the issue of bioavailability. I think that is possible, if we can get good relationships between tissue residues and some

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ecologically significant effects. I would also like to add to some of Peter Landrum's earlier comments. Peter, I think the onus is on us to show that these changes in the benthos are important enough that they have affected fisheries populations or wildlife. The public is not primarily interested in amphipods, oligochaetes, or clams, but in fisheries and wildlife. If we cannot protect these populations, then we have failed.

Q (Norm Rubinstein, U.S. EPA, Office of Research and Development): The session this morning really focused on the issues of measuring bioaccumulation and predicting bioavailability. I imagine as the meeting progresses, we will get into the other side of the issue, which is the corresponding effects. But I am curious to see how you gentlemen feel about our current ability to predict bioavailability and bioaccumulation, either thermodynamically or kinetically. Do you have a sense of confidence in our ability to identify bioavailable fractions and go on from there to identify the corresponding ecological effects?

Peter Landrum:

My feeling about that, Norm, is that if we are talking about neutral organic compounds from sediments, we can probably predict bioaccumulation within a factor of ten to twenty, if that is adequate. I think trying to get any better predictions than that right now is not possible because, as I pointed out earlier, we do not have a complete understanding of how contaminants partition among sediment size fractions and the degree of feeding selectivity by the organisms. Without this information, we really do not know how much contaminant an organism is exposed to.

Chris Ingersoll:

We have been focusing quite a bit this morning on the nonpolar organics, but what you have not seen today is some of the work that has been done relative to metals and acid volatile sulfides (AVS). Some of the metals are able to be predictive of bioaccumulation that we are seeing. A series of papers on AVS and metal bioavailability will be published in the December 1996 Society of Environmental Toxicology and Chemistry (SETAC) journal. That issue will also include a good review article by Gary Ankley and others for a variety of studies to address the question of whether or not we can predict bioaccumulation relative to AVS and SEM (simultaneously extracted metal). He found that the SEM/AVS approach offers a more reliable and predictable tool than what is currently available.

Q (Steve Bay, Southern California Coastal Water Research Project): Peter Chapman listed coupling tissue residues with toxicity responses as one of the key issues for this session. But I was wondering about how the recommendations to often use insensitive or hardy test animals will impact this issue. Are we going to end up with a really nice data set, but no ability to couple residue levels with toxicity responses because we do not understand how to predict the responses from uptake residues in insensitive organisms? Are we in danger of that, or will we be able to figure that out once we get a good data set together?

Peter Landrum:

I think we could be somewhat in danger because these insensitive organisms are going to tell us that they can accumulate more than some of the sensitive organisms will. The sensitive animals will pass the toxicity threshold and produce a response. Insensitive organisms should give you some idea of what the maximum amount that could accumulate in an organism would be. This would allow you to at least define the level that you would have to drive down to protect against responses in other organisms.

Chris Ingersoll:

We, as toxicologists, need to develop adequate designs for studies involving water or sediment to measure toxic effects and bioaccumulation in the same exposures. I am really looking forward to hearing from some of the panelists later this afternoon about their databases. Some data are available, but you really have to search the literature to find those kinds of data sets.

Peter Chapman:

I have found it useful in toxicity tests, where the chemicals and organisms are appropriate, to measure bioaccumulation and toxic effects. Among other things, it can help me sort out what may be causing any effects I see. We are trying to move in that direction, but it is a very valid concern.

Q (Joe Greenblott, U.S. EPA, Office of Research and Development): I would like to ask a question about how models fit into the experimental work within the decision-making and risk assessment framework. What level of attention is being given to developing and researching these models and developing laboratory data to the conservative level required for decision-making, in light of the variability in laboratory data and the large uncertainty associated with the predictive models?

Henry Lee:

I will give you a different perspective. We are working now to determine ecosystem responses and cumulative effects. The variability is even greater for this work than for the data you saw here. I think you have to go to a risk aversion philosophy or a more environmentally protective approach. We cannot accurately predict a dose response, a stressor response, but we know it is bad to lose prey in a system or to lose a wetland. The direction we are going to have to go is to a risk aversion strategy. That is where comparative risk factors in, so we can determine how important one risk is versus another. Even if we cannot be quantitative, we can at least rank the risks.

Q (Eric Rifkin, Rifkin & Associates): A number of the panelists today referenced BSAFs, biota-sediment accumulation factors, and how they varied based on whether or not you were using lipid-normalized organisms and carbon-normalized sediment. Could the panelists comment on whether BSAFs can be used in a generic context or whether they need to be used in a site-specific context? I would also be interested in hearing how they can be used in developing sediment quality criteria in general.

Henry Lee:

It comes down to how well you need to know the answer. I agree with Peter that we can predict bioaccumulation within an order of magnitude. If that is good enough for the neutral organics, then you are home free. If you need a better answer, then you have a problem. The better the answer you need, the more expensive and more site-specific it gets. But I am comfortable working within the order of magnitude range.

Q (Lynn McCarty, L.S. McCarty Scientific Research and Consulting): I would like to raise a question related to an earlier question. The question is about sensitivity. I am always concerned that we do not really define what sensitivity is. Are we referring to sensitivity defined according to exposure-based tests or, since we have been discussing tissues residues, are we referring to sensitivity on a residue basis or received dose effect? In fact, as I will show this afternoon, you can consider these things for the same set of data and come to quite different conclusions. Most of the differences in sensitivity that I have seen in the literature can be readily expected and predicted from differences in modifying factors such as the size of the organism, the metabolic activity, and temperature. Until we clearly define what sensitivity means, we should not be making comparisons about which animals are more sensitive than others. In sediment testing, as we have seen from some of the discussions this morning, the variability that results from the differences in media and conditions of those media dramatically affects the accumulation rates and amounts of accumulation. Therefore, sensitivity is a confusing factor that needs to be clearly identified before we make some final pronouncements about it.

Peter Landrum:

I think if you go back and look at the toxicological literature, sensitivity had to do with differences between species. Sensitivities were usually determined, at least in the mammalian literature, with a defined dose approach. A known dose of something was given to two different species by the same route. There is no doubt that the route of exposure is going to alter the response that you expect, particularly for sediment where there are a lot of confounding factors that can influence the dose received. You can take one animal and move it from sediment to sediment with the same compound and get a change in response, because factors change that influence the bioavailability and, therefore, the received dose. If we are going to talk about sensitivity between organisms, we need to talk about the sensitivity based on the received dose.

Henry Lee:

I would like to add a comment on sensitivity. When Chris and I talk about sensitivity, we are referring to a value that is empirically derived. That was the basis for the sensitivity Chris showed in his diagram during his talk. In determining the sensitivity of potential test animals, we need to find animals that will survive for 28 days or however long it takes to reach steady state.

Q (Tom O'Connor, NOAA, National Ocean Service): Two of you have agreed with each other that the predictions based on equilibrium partitioning are good to within one order of magnitude. How would you assess the imprecision of extending the equilibrium partitioning methodology to body burdens in fishes?

Henry Lee:

In general, the imprecision is greater. We derived BSAFs for two fish that have limited home ranges in our DDT Superfund study. These BSAFs turned out to be relatively close to the values we derived for *Macoma* and other benthic organisms. However, these values are more variable for fish like flatfish that have extensive home ranges. But I think we can at least determine a maximum value for demersal fishes.

Q (Tom O'Connor): So the imprecision is in the range of the fish, not in the equilibrium between a given sediment and a fish?

Peter Landrum:

I think you have to consider the routes of exposure as well. If you have a pelagic fish and it is not feeding on things that are well connected with the sediment, then it is inappropriate to try to make a connection between the sediment and that fish. Whereas if you have an organism like a flatfish that is feeding on benthic organisms, it might be easier to make the connection. But you still have additional routes of exposure that you need to consider.

Q (Tom O'Connor): Yes, but equilibrium does not matter for this.

Peter Landrum:

But that is assuming the process is passive. All the models I talked about imply a passive process. When we look at fish in particular, we may no longer be considering a passive process as the sole driving force. So, the thermodynamics that you are trying to consider in terms

of sediment to the fish are no longer applicable. If feeding is taking place and the benthic organism is in equilibrium with the sediment, we could make the assumption that there is a connection between fish and sediment. But if feeding and digestion are taking place, they are active processes that may change the thermodynamics that apply to a particular fish.

Q (Tom O'Connor): So, the answer to my original question is that you cannot apply equilibrium at all to extrapolate the fish.

Peter Landrum:

Not unless they are eating the sediment.

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